BUILDING RELATED ILLNESS

A PROCEDURE TO DETECT SYMPTOMATIC BUILDINGS

A thesis

submitted in partial fulfilment

of the requirements for the degree

of

Master of Philosophy

in

The Department of Agricultural Engineering

at

Massey University

William Edward Sisk

1994

TABLE OF CONTENTS

ACKNOWLEDGEMENTS

The author wishes to express sincere gratitude to his supervisors, Dr. Gavin Wall, Head of Department (Agricultural Engineering) and Robyn Phipps. Their assistance and sound advice throughout this project were of immense assistance.

The author also wishes to acknowledge the assistance given by a number of other academics. Ron Garland of the Department of Marketing who located the personnel to conduct the telephone survey and who offered practical advice on the structure of such interviews. This assistance saved a great deal of time. Thanks to Kerry Chamberlain from the Department of Psychology who provided good advice on questionnaire design. Finally, Dr. Alastair Robertson of Occupational Lung Unit, East Birmingham Hospital who provided me with a copy of the U.K. Office Environment Survey.

A special thank you goes to Debra McCrae for formatting this document and generally sorting out my typing. Not an easy task.

I want to acknowledge a special debt to my late friend and colleague Jack Tyler. Jack never failed to inspire me with his courage and sense of humour. Jack went as he lived, with style. I truly hope that you found that final class of Dip. Horts., Jack.

Finally, I wish to acknowledge my family, my wife Jennifer and daughters Amanda and Suzanne. Their support take me always to the horizon of my purpose and allow me to look beyond it. I can never thank you adequately for that.

To all of these good people I dedicate this work.

ABSTRACT

This study examines the topic of building related or building supported illness in the context of a commercial office setting. Numerous reports from the United Kingdom, Scandinavia, Holland, Denmark, Canada and the United States of America, indicate that workers in some office buildings suffer a degree of discomfort and physical symptoms related to building occupancy.

The problem is examined in the context of a commercial office environment and the term 'Building Related Illness' (BRI) and it's sub-set 'the Sick Building Syndrome' (SBS) are defined. The illnesses or specific syndromes known to be associated with building related illness are identified. There is ongoing debate as to the valid inclusion of some viral diseases.

This study takes a symptomatic approach to the identification of the various syndromes of interest. The numerous elements or stressors known to cause particular symptoms are identified and discussed.

BRI is identified by an unusual or extraordinary frequency of certain physical symptoms being experienced by the occupancy of a particular building. However, the symptoms of interest are found in the general community at an unknown incidence rate.

The exact role a building and it's association with a symptom or cluster of symptoms is, more often than not, difficult to ascertain. There are a number of confounding elements which need to be considered and eliminated before the building itself can be implicated as a causal factor. This is because the general nature of the symptoms associated with BRI can be caused by other factors. Broadly, the other causative factors may be 'Job Related' or 'Ergonomically Related' (eg. poorly designed work stations). It is well documented that workers in menial or less interesting employment report a higher prevalence of BRI type symptoms.

The role of chronic diseases in relation to commercial buildings are discussed and the alternative paradigm to dealing with these stressors is examined.

Finally, a statistical method for identifying a 'problem building' is piloted on two dissimilar buildings and the results are analyzed. The prevalence of symptom reporting amongst certain cohorts is similar to a number of overseas studies.

It was concluded that the proposed model was successful in identifying symptom clusters amongst certain cohorts within the buildings surveyed. In this respect the piloted questionnaire was successful.

The questionnaire is critically reviewed and a number of amendments are suggested.

CHAPTER 1

OBJECTIVES

The objectives of this study are:

(1) To establish from available literature, whether *Building Related Illness* is a problem of sufficient magnitude to warrant further investigation.

(2) To research the available literature pertaining to 'building related illness' and to identify the various causes.

(3) To examine the feasibility of developing a practical procedure for answering the question *"is the building under investigation sick"*.

The measure of sickness in a building is *relative* rather than *absolute*. The procedure needs to be capable of establishing whether the occupants of a particular building are experiencing an **abnormal level** of physical symptoms.

For the purposes of this study, an *'abnormal level'* is defined as *'a disproportionate level over and above that which would be expected from similar cohorts within the general community'*.

(4) To identify areas requiring further investigation.

CHAPTER 2

THE SIGNIFICANCE OF BUILDING RELATED ILLNESS

WHAT IS BUILDING RELATED ILLNESS?

Building Related Illness (BRI) has not been adequately defined within the literature. The EPA (1988), states that if problems within a building result in clinically defined illness, disease or infirmity of the occupants, the building is said to manifest Building Related Illness.

Raw (1992), states that BRI includes the unspecific syndrome known as the Sick Building Syndrome (SBS) and other building related problems such as physical discomfort, infections and long term cumulative chemical hazards such as asbestos and radon.

CONFUSION OF TERMS

The name 'Sick Building Syndrome' (SBS) has become synonymous with all BRI in much of the literature. This is a misuse of the term as SBS refers to one particular syndrome.

According to Hodgson (1986) the American Thoracic Society perpetuates the misuse of the term and uses SBS to refer to all illnesses, including hypersensitivity pneumonitis, related to indoor air quality.

Sykes (1989), reports that various terms have been used to describe the phenomenon of sickness amongst office workers - *'building sickness'*, *'sick building syndrome'*, *'sick office syndrome'*, *'tight building syndrome'*, *'office eye syndrome'*.

SICK BUILDING SYNDROME DEFINED

The term 'Sick Building Syndrome' is an expression used to describe buildings with physical anomalies affecting health (Berglund et al., 1986). Like many medical conditions, the syndrome is defined according to symptoms and occurrence.

A feature of sick buildings is that their occupants appear to suffer a higher incidence of illness than would be expected. All the symptoms associated with the Sick Building Syndrome are present in the population at large (at unknown incidence rates) but have been shown to be more common in some buildings than others (Raw, 1992).

A number of studies have identified that large sealed buildings with many workers in open plan areas are particularly symptomatic (Sterling et al., 1983; Robertson et al., 1985).

The symptoms related to SBS are well established. The World Health Organisation (1986), defined SBS as manifesting the following symptoms:

TABLE 1 - SICK BUILDING SYNDROME SYMPTOMS

Sensory irritation in eye, nose and throat	• dryness • stinging • hoarseness
Skin Irritation	• reddening of the skin • itching • dry skin and dermatitis
Neurotoxic symptoms	• mental fatigue • impaired memory • lethargy and drowsiness • reduced concentration • headaches • dizziness and giddiness • nausea
Unspecific hyperreactions	• running nose and/or eyes • asthma type symptoms in non-asthmatics • respiratory sounds
Odour and taste complaints	• changed sensitivity • unpleasant odour or taste

POSSIBLE CAUSES OF SBS

In spite of extensive research internationally, no single cause has been identified.

Many researchers have attributed the genesis of the syndrome to the energy crisis in the early 1970s. The rapid rise in fuel prices encouraged designers to develop 'tighter'(ie. reduced air exfiltration through the building envelope), and more energy efficient buildings. Ventilation rates and occupant control over the environmental systems were decreased. Indoor air

quality was further compromised by the accelerated development and use of synthetic materials in building materials, furnishings and clothing, many of which contain toxic or irritant compounds.

The term *'tight building syndrome'* was coined in the United States, implying that the problem was simply an air quality issue. This has not been entirely supported by subsequent research.

Health problems in buildings may originate from any of the disciplines involved, including poor architectural design, commissioning, management, maintenance or use.

While causal factors may well, and probably do, overlap, it seems reasonable to assume that specific illnesses caused by toxic agents are often misdiagnosed as the non specific Sick Building Syndrome. Raw (1991), is of the opinion that SBS should be defined by a set of symptoms, not perceived discomfort without symptoms or clinically diagnosed illnesses.

BUILDING RELATED ILLNESS DEFINED

There is considerable disagreement amongst researchers as to which illnesses are building supported.

Broadly, all diseases can be generically grouped as either acute or chronic. Acute conditions arise suddenly, are of relatively short duration and are, usually, without lasting 'side effects'. Chronic diseases develop slowly and are often life threatening.

In relation to the indoor environment, potential cancer causing mechanisms have been identified as the chronic stressors of concern. These include radon, asbestos, electro-magnetism and, possibly, formaldehyde.

There is less consensus amongst researchers as to which acute syndromes are supported or exacerbated by building occupancy. For example, Anon (1991), reports Burge as stating that human pathogenic viruses are host dependent, requiring people in order to survive. There is a debate in the public health community as to whether viruses are transmitted principally by air or by physical contact. Many public health officials favour the latter, in spite of the fact that the air transfer model better explains empirical studies of infection patterns. Influenza viruses tend to be transferred almost exclusively in the air and adenoviruses by contact. Thus, if the 'contact model' is verified then the building's part in the equation is simply that of increasing occupational density and bringing more people in contact with each other.

In the same report Burge is quoted as stating that bacteria and fungi can support themselves on nonliving environmental substrates. The bacterial infections supported by buildings include Legionnaires Disease, Pontiac Fever and Hypersensitivity Pneumonitis. Fungi, can support themselves on environmental substrates and can cause a range of Mycotic infections, Allergic Rhinitis and Allergic asthma.

Hodgson et al., (1986) indicates that the following syndromes are building related; allergic respiratory diseases such as sinusitis, asthma, allergic tracheo-bronchitis, hypersensitivity pneumonitis, humidifier fever, and mucous membrane irritation; infections such as legionnaires disease, pontiac fever and Q fever (a form of zoonosis); SBS; dermatitis

including allergic dermatitis, photodermatitis, irritant dermatitis; and, reproductive complaints.

Anon (1991), produced a far more comprehensive list which included a number of relatively rare syndromes (refer Table 13).

This study accepts that the probable building related syndromes identified by Hodgson et al., (1986) are well supported by the available evidence. Descriptions of the more unusual or rare syndromes whose association with physical building structures is tenuous or unproven can be found in Appendix 9.

The symptoms of interest have been conveniently tabulated by Bernard (1988) as follows. It should be noted that to include all of the syndromes implicated by Hodgson et al., (1986), Q fever, sinusitis and pharyngitis, and allergic tracheo-bronchitis have been added.

TABLE 2 - SYMPTOM CLUSTERS OF CONCERN

Disease	Health Symptoms										Other	Causal Agent or Cause
	Fever	Headache	Malaise	Cough	Myalgia	Chills	Dypsnea	Chest Pain	Fatigue	Weight Loss		
Legionnaires Disease	*	*	*	*	*	*	*	*		*	Pneumonia Confusion Vomiting Diarrhoea	Legionella Pneumophilia
Pontiac Fever	*	*	*	*	*	*		*			Diarrhoea Nausea Sore Throat Dizziness	Legionella Pneumophilia
Hypersensitivity Pneumonitis	*	*	*	*	*	*	*		*	*	Lethargy	Various eg. M.*faeni* T.*vulgaris*
Humidifier Fever	*	*		*	*	*	*	*	*	*	Pain in joints, polyuria	Possibilities: N.*gruberi* *Acanthameobae* *Aureobasidium- Pullulans* *Flavobacterium* *Cephalosporium* B.*subtilis*

SBS		*					*		*		irritation of upper & lower respiratory tract, eye irritation, skin irritation, dizziness, concentration difficulties	Multiple causes implicated
Sinusitis & pharyngitis		*	*								Chronic post nasal drip, nasal drainage, sore throat	Contaminated peripheral window heating or cooling units, fungal growth in water damaged areas.
Allergic tracheo-bronchitis				*				*			Chest tightness, primary upper airway involvement	*Cladosporium*
Q Fever	*	*	*		*	*		*			Severe Fever, temperature may rise to 40°C and persist for 1-3 weeks.	C.*burnetii* R.*burnetii*

Possible Causes and Mechanisms

Occupants of office buildings are susceptible to any of the thousands of identifiable medical syndromes that affect all human beings.

Any of the tabulated symptoms can be caused by factors other than the physical building structure. In a wider sense, **occupational stress** is manifest by a set of physical symptoms similar to those above.

Stress is defined in the Oxford Dictionary as *'a constraining or impelling force'*. Therefore it is an outside force or stressor exerting an influence on the body which will react in some physical manner. Craig (1981), cites a number of stressors pertaining to office workers including; personal problems, bad lighting, noise, repetitive muscular movement, poor ventilation, pressure from management and poor organisation, lack of job satisfaction, lack of control over personal environment, etc. Indeed, any outside force that can induce a physical reaction could be described as a stressor. In addition, building occupants may be exposed to biological agents, organic and inorganic vapours and gases, radiation and other harmful elements.

Broadly, the above stressors can be categorised as building related, job related or ergonomically related.

Role of Ergonomics in Symptoms of Concern

Poor furniture and work top design and machine-user desynchronisation can cause a range of symptoms including referred headache, eye strain, back-ache and fatigue.

Nicholson et al., (1988) gives an example of where a computer might be installed in an office to remove some of the drudgery from a clerical task. The potential of the system allows for increased throughput and workers at either end are required to work at the pace of the machine, leading to mental and physical fatigue, probably lowered morale and certainly reduced efficiency.

Poor furniture design has been identified as a factor contributing to worker malaise. Musculo-skeletal complaints, particularly back pain, have been recognised as being connected with sedentary work. A number of researchers have shown that static, seated postures correlate with the onset of low back pain (Magora, 1972; Adams et al., 1983). Grandjean (1988), reports that sitting postures result in higher disc pressures than standing upright. Burge et al., (1987) note that many VDU operators sitting in relatively fixed positions report the most symptoms of all job categories. Wilson et al., (1987), reported that those who work 6-7 hours per day at a VDU experience more symptoms than those who work fewer hours.

Role of the Job in Symptoms of Concern

Burge et al., (1987), reported that clerical and secretarial workers complained of more symptoms than professional or technical workers who in turn, reported more symptoms than managers. According to this group researchers, the reason why managers and professionals have fewer symptoms than secretarial or clerical workers is not clear, but it may be related

to their better accommodation within the buildings or a greater degree of control over their job or their greater ability to have changes made to the running of the environmental systems.

According to a report by the joint ILO/WHO Committee on Occupational Health (1984), a vast amount of literature has demonstrated that psychosocial factors at work contribute to a wide range of health disorders. A large amount of evidence has been accumulated on the relationship between non-specific psychological, behavioral and somatic syndromes and stressful or unrewarding working conditions.

Individuals interact with their working conditions in a manner which is determined human capacities and needs. The crucial job factors in this interaction include task, the physical and social working environment, managerial practices and employment conditions.

In the work environment, a number of negative, potentially health related psychosocial factors have been identified in a number of studies. These include the under utilisation of abilities, work overload, lack of control, role conflict, inequity of pay, lack of job security, problems in relationships at work and shift work (Refer to psychological factors).

Interactions of the Causal Factors

The possible multiple sources of the symptoms of concern complicates investigation of a possible building related problem.

For example, assume that VDU operators in a building are complaining of an excess of eye and somatic symptoms. Possible sources of the problem are the nature of the task (job related), poorly designed work stations (ergonomically related), or say glare from the screen caused by a poorly located light fitting (building related),

It is probably fair to say that, in some cases at least, the building is not the cause of malaise amongst occupants.

Physiological Reactions to Stress

Molhave (1990) suggests that human response to stress may be related to a biological model consisting of three stages:

> *(a) Sensory perception of the environment.* The senses include odour, taste and chemical sense. Chemical sense refers to nerves in the mucosal membranes and skin that react to chemical stimuli. Activation of the senses leads to irritation and possibly a protective response, such as sneezing.
>
> *(b) Weak inflammatory reactions.* Inflammatory reactions are related to microbiological, metabolic, or immune system reactions and are generally considered to be a protective reaction to potential cell damage. Acute, reversible reactions seem to be relevant to low level pollutant exposure in nonindustrial environments.

(c) Environmental stress reactions. The constant effort needed to identify wanted and to override unwanted sensory information, and the efforts needed to maintain protective reflexes may cause secondary effects, such as headache.

In many cases the link between a stressor such as a particular noxious pollutant and a specific symptom are clear and direct. In other instances the cause-effect relationship is less obvious. For example, somatic symptoms as a result of, say, annoyance with superiors or subordinates is probably the result of the release of neuro-hormones.

Pennebaker (1982), demonstrates a link between particular emotions and specific physical symptoms.

THE IMPLICATIONS OF BRI

SUMMARY

Generally the acute symptoms of BRI may be regarded as being minor, in that they are 'rarely' life threatening . This, of course, is not so with more serious clinical illness such legionnaires disease, but the prevalence of the latter is relatively rare.

There is no published evidence as to the prevalence of chronic effects as these data are much harder to ascertain.

According to Raw (1992), the symptoms of BRI are not trivial to the people who experience them on a regular basis at their place of work. Although the basis of estimates are not perfect, most figures suggest 30-50% of new or refurbished buildings are affected and slightly older buildings are affected as much if not more.

Air conditioned buildings are generally associated with higher prevalence.

PREVALENCE AND ECONOMIC COST OF BRI

Sheldon et al., (1988), reports that 25% of American workers feel that the quality of their workplace affects their work adversely. Woods (as reported in Levin 1989), found that 20% of the office workers in the United States may be affected by the Sick Building Syndrome.

A study conducted by BOMA in the USA (BOMA, 1989), found that 23% of the respondents rated air conditioning as the biggest problem affecting work performance. Poor indoor air quality was also ranked as a major problem in the study. Productivity, according to the respondents, would increase by 18% if the air conditioning problems were resolved and by 21% if the indoor air quality problems were eliminated. While these estimates may appear to be ambitious, they are nevertheless supported by similar surveys carried out in other countries.

Harris Research Centre UK (1989-90) in a survey of public perception of indoor environment in central business districts of major cities in the UK, France, Singapore, Germany and Australia reported that 62% of office staff felt that their productivity would improve with

cleaner or fresher air. Furthermore 22% of the respondents indicated that they took one or more days off work each year from complaints such as eye irritation, sore throat, runny nose, headache and other upper respiratory problems which they feel are caused or exacerbated by the environment in which they work.

Bergs (1990), in a survey of office absenteeism in the Netherlands, found that 24% of the respondents called in sick because of work related complaints for an average of 2.5 days per year. Bergs estimated that this resulted in over 1 million work days lost by office staff in the Netherlands. The annual cost was calculated as begin close to NZ$1 billion per year.

In an earlier study into the economic costs of respiratory tract infections in the United States, Dixon (1985), claimed that over 150 million lost work days and US$15 billion of direct medical care costs annually in the USA could be attributed to symptoms identical to those revealed by Harris poll results.

In a poll of 4373 office workers in the United Kingdom, Burge et al., (1987), 57% reported lethargy, 47% reported blocked nose, 46% dry throat, 46% headache and 9% reported chest tightness and difficulty in breathing. The symptoms were more than twice as frequent in buildings with local and centrally supplied induction fan coil units compared with naturally ventilated buildings. Interestingly, amongst the 'all air' systems buildings with 'steam' humidification were both the healthiest and unhealthiest.

McDonald (1984), reports similar results in a survey of federal government offices in Quebec, Canada. In a poll of 5,300 public servants 49% experienced nose irritation, 46% eye irritation and 83% headaches. In another Canadian survey, Sterling et al., (1985), reported a building where 86% of the occupants experienced headaches, 53% reported dry throat, 51% dry nose and 44% dry skin and dermatological complaints.

Temporary or Permanent Illness

A distinction has been made between so-called temporarily sick buildings which are usually either newly constructed or remodelled buildings, and permanently sick buildings (WHO, 1983).

According to Hedge et al., (1989), in temporarily sick buildings, symptom onset is acute and declines over time with most problems ceasing within one year. They report that studies of temporarily sick buildings frequently identify volatile organic compounds as the likely source of the problem.

In permanently 'sick' buildings, symptoms cannot be attributed to any specific air contaminant, and symptoms persist over time. Buildings categorised as permanently sick almost invariably appear to be air-conditioned, energy-efficient structures with a sealed building envelope.

Keypoints: The prevalence of symptoms in office buildings in all parts of the world is reported as being high. The economic cost in lost working days and degraded activity is reported as being substantial. Symptoms are generally more prevalent in air conditioned buildings but naturally ventilated buildings can also have problems.

CHAPTER 3

LITERATURE REVIEW

AIR QUALITY

According to the EPA (1991), a healthy environment is one in which the surroundings contribute to productivity, comfort and a sense of health and well being. To achieve this objective, the indoor air needs to be free from significant levels of odours, dust and contaminants and it should circulate to prevent stuffiness without creating drafts. Temperature and humidity needs to be appropriate to the season and to the clothing and activity of the building occupants. The EPA (1991), definition of good Indoor Air Quality includes the following factors:

> *"The introduction and distribution of adequate ventilation air, control of airborne contaminants and the maintenance of acceptable temperature and relative humidity"*

VENTILATION

EVIDENCE OF THE EFFECT OF VENTILATION ON BRI

According to Raw (1992), the role of ventilation, particularly in relation to SBS symptoms, has been debated in two different but related terms; the ventilation system and the supply of fresh air.

Burge et al., (1987), reported that the lowest mean symptom prevalence in the U.K. were found in naturally ventilated and mechanically ventilated buildings. Because of the association between SBS and air conditioning, inadequate ventilation has been high on the list of causes proposed by researchers.

Raw (1992), believes that the evidence suggests that low ventilation rates are not a major determinant for SBS.

There is little doubt that increasing the ventilation rate or reducing the proportion of recirculated will air will normally reduce pollutants. Farant et al., (1990) found that with higher outdoor rates, concentrations carbon dioxide, nitrogen oxide and Volatile Organic Compounds (VOCs) were lower.

Raw (1992) states that, on the whole, existing ventilation standards are adequate and there is little advantage from increasing them further unless there is no alternative means of reducing pollution in a building. Most of the reports of inadequate fresh air stemmed from malfunctioning or mismanaged systems. In those cases the minimum standards were not being met.

VENTILATION OBJECTIVES

The main objectives of ventilation in a building are to replace 'old' contaminated air in the zone of occupation with 'new' fresh air as quickly as possible and to remove generated contaminants (Skaaret, 1986). An additional requirement is that the 'new' air should reach the zone of occupation as 'undiluted' (uncontaminated) as possible and without causing a draught.

McNall et al., (1984), state that ventilation in office buildings serves several purposes:

(1) To provide a healthy and comfortable air quality environment for the building occupants which will not be damaging to the building structure, furnishings or contents. It is generally assumed that if peoples needs are met, the building needs will be met as well. Exceptions would be archival storage and other special purpose areas whose contents require special consideration. It is assumed that the ventilation system provides air for comfort which can be defined as *air which is not subjectively objectionable to the occupants.*

(2) To provide for temperature control, ventilation air is usually heated or cooled, and introduced into the space. Internal circulation is also needed to provide mixing for adequate uniformity within the space. Practical limitations on temperature levels used in the heating, ventilating, and air conditioning systems (HVAC) usually require that the air flow quantities for temperature control be 4 to 8 ACH (Air Changes per Hour). This is almost always the largest amount of ventilation required and therefore becomes the design benchmark. McNall et al., state that ventilation for air quality rarely needs to be more than 1 ACH, although this is hotly disputed by other authors. Energy conservation considerations usually require that as much air as possible be recirculated, but recirculated air does not remove significant amounts of pollutants without special air treatment.

(3) To control humidity, moisture may be added to ventilation air using any one of a variety of humidifiers, and moisture may be removed by condensation on cooling coils, by chilled water sprays or chemical desiccants. Relative humidity levels are usually maintained below 60% to protect the building structure from condensation, rot, etc., and to limit deterioration and mould growth on building contents. People can be thermally comfortable at relative humidities well above 60% (ASHRAE,1980). In cold weather, relative humidity without humidification can be below 10% and humidification is desirable to minimize discomfort, static electricity, etc. Condensation on cold windows and walls often prohibits the maintenance of relative humidities above 20% in cold weather (Grot et al.,1983).

(4) Ventilation is required to remove excess heat, humidity, and contamination from the space. This is usually done by exhausting a portion of the air returned from the space before the balance is heated or cooled and returned to the space. In some cases special local exhaust systems are used. Generally, office buildings are not equipped with special systems to serve zones of high internal heat or contamination rates with some exceptions such as lavatories and computer rooms.

(5) To provide small pressure differences between zones or between indoors and outdoors to control air exchanges, such as in entrance lobbies.

(6) To provide pressure differences to confine or exhaust smoke, heat, and toxic fumes for fire safety. The HVAC system is being used in an increasing number of cases for fire safety. Special dampers and fans are programmed to respond to smoke and fire sensors in various locations so that when problems are detected, the ventilation system will not spread the products of combustion.

(7) To provide enhanced air motion in hot spaces for greater thermal comfort.

Fresh Air Ventilation Rates

The overall exchange of air in buildings with fresh outside air is the result of a combination of infiltration, natural ventilation, and mechanical ventilation (Refer Appendix 3). Even in new buildings, infiltration can be a significant contributor to the total building air exchange rates. This is particularly true in winter when the driving forces for infiltration are the greatest and the mechanical ventilation systems are operated with the minimum of outside air (Baechler et al.,1990).

Air Exchange Standards

Ventilation is often described in terms of air changes per hour (ACH), even though ventilation is most important to the human occupants of a building and is expressed for this purpose as a volume rate per person (cubic feet per minute per person, cfm/person or litres per second per person, Ls^{-1}/person).

The outdoor air for fresh-air ventilation assumes that levels of contaminants in the ambient air are relatively low and safe for building occupants. In many cases, outdoor air can be heavily contaminated hence it is no longer referred to as 'fresh air' but as 'outdoor air'. ASHRAE Standards 62-1973 and 62-1981 recommend that outdoor air used for ventilation meet ambient air quality standards or be treated so that it does. Unfortunately, this standard is infrequently adhered to (Goldish,1989).

In 1989, ASHRAE revised the ventilation standard in response to studies showing that a mechanical ventilation rate of 5 cfm was not sufficient to maintain acceptable levels of indoor pollutants. The new standard (ASHRAE 62-1989) increased the minimum airflow rate from 5 cfm to 15 cfm/person and combined smoking and non smoking rates into one recommendation. The purpose of this standard is to:

> *'specify minimum ventilation rates and indoor air quality that will be acceptable to human occupants and are intended to avoid adverse health effects.'*

TABLE 3 - RECOMMENDED AIRFLOW RATES (CFM/PERSON) ASHRAE STANDARDS

Building Type	Standard 62-1981		Standard 62-1989
	Non-Smoking	Smoking	
Office spaces	5	20	20
Retail stores	5	25	.02-0.30 cfm/ft^2
Classrooms	5	25	15
Dining rooms	7	35	20
Hotel Conference Rooms	7	35	20
Ballrooms & Discos	7	35	25
Spectator areas	7	35	15
Theatre auditoriums	7	35	15
Transport waiting rooms	7	35	15
Hospital patient rooms	7	35	25
Residences	10	10	0.35 ACH
Bars/cocktail lounges	10	50	30
Beauty shops	10	35	25
Smoking lounges	--	--	60
Office Conference Rooms	7	35	20

Studies Implicating Ventilation as a Causal Factor

Numerous reports indicate that mechanically ventilated buildings tend be more symptomatic than naturally ventilated buildings.

Baechler et al., (1990), indicate that ventilation problems experienced in mechanically ventilated buildings are often as a result of poor system operation and/or maintenance. Typical problems encountered are:

(1) Buildings operated with 100% recirculated air in an attempt to conserve energy.
(2) Low ventilation efficiencies so that only a small fraction of the ventilation supply air can thoroughly mix in the occupied space before being exhausted.
(3) System imbalance resulting in some space receiving more ventilation air than others.

In the United States a number of studies have been conducted to measure overall air exchange rates in commercial buildings. These studies were conducted in occupied buildings with the HVAC systems in their normal mode of operation. The results of these studies are summarised as follows.

In a study conducted in the Pacific Northwest, overall air exchange rate measurements were made in 38 commercial buildings (Turk et al.,1987). The buildings were in two distinctly different climatic regions. Ages of the buildings ranged from 0.5 year to 90 years. The overall average air exchange rate was 1.5 ACH with a standard deviation of 0.87 ACH with a range of 0.3 ACH to 4.1 ACH. The investigators concluded that:

> *".... no statistically significant relationship existed between ventilation rates and building height, building age, and number of stories above grade, due in part to the dominating influences of season, air handling equipment differences, and HVAC system operating policies. It appears that some of these ventilation rates may have been artificially high because of building operator actions during the monitoring period. In each instance of low ventilation rates (<0.5 ACH), it was found that the dampers were closed for energy conservation measures to reduce the cooling load in the summer. In one building the system operators were not sure of the damper location or the control mechanism."* (Turk et al.,1986).

Grot et al., (1986), measured air infiltration and ventilation rates between 0.28 ACH and 0.70 ACH in eight federal buildings using tracer gas . The results were as follows:

TABLE 4 - AVERAGE VENTILATION/INFILTRATION FOR EIGHT U.S. FEDERAL BUILDINGS

Location	Average ACH	ASHRAE Minimum ACH
Anchorage	0.28	0.26
Ann Arbor	0.70	0.47
Columbia	0.40	0.62
Fayetteville	0.33	0.32
Huron	0.20	0.13
Norfolk	0.52	0.62
Pittsfield	0.32	0.38
Springfield	0.50	0.55

Half of the buildings did not meet the minimum ventilation rates recommended be ASHRAE.

Studies by Walingford et al., (1986) have shown that 50% of complaints in buildings occur because of poor ventilation. Robertson et al., (1991), reports the results of a survey in which ventilation inadequacies are primarily the most identifiable cause of indoor air quality problems.

Is an Increase in Ventilation Rates the Answer?

There is a strong lobby recommending that the ventilation rates be increased back to the levels recommended in the early 1970s. Their catch cry is:

'The solution to pollution is dilution'

Whether this is the solution is highly debatable. Seppanen (1990) reported that in a controlled study of office workers in Finland, the outdoor air supply was reduced from 23 l/s per person to 6 l/s, using 70 per cent recirculated air. No significant increase in symptoms or environmental complaints were found.

It has been suggested by a number of authorities that poor maintenance of the ventilation system seems to be a more common reason for ventilation problems in buildings. Olson (1988), reports that of some 233 ailing buildings that ACVA Atlantic inspected over a five year period, no fresh air intake *whatsoever* was found in 35% of cases, and inadequate volumes of air in 64%.

According to Broadbent (1991), assuming ventilation effectiveness, there is still the question of actual ventilation rate, usually expressed as litres per second per person. From a health point of view, ventilation rates could be very much lower than currently accepted levels. However, in response to comfort complaints in relation to air which is stuffy, odorous or irritating, ventilation rates are being increased in regulations worldwide. The 1991 edition of AS 1668 calls for an increase of about threefold in the ventilation rate compared with its predecessor.

Kohloss et al., (1991), suggests that increasing ventilation rates may be simplistic. In the ventilation rate procedure outdoor air either of acceptable quality or cleaned to such a quality is supplied to various occupancies at a flow rate found to provide 'acceptable indoor air quality'. The ventilation air must be effectively distributed throughout the occupied zone and the breathing space of occupants which is roughly the first 2 metres above the floor.

In many cases an imbalance in the system prevents the outdoor air from reaching this 'breathing zone'. Olsen (1988), describes a condition known as *short circuiting* where the supply air is drawn to return vents before it has an opportunity to circulate. Kohloss et al., (1991), suggest that ventilating air must be distributed evenly throughout the occupied space to be effective. Typical air distribution is from ceiling diffusers. Particularly in Scandinavian countries there has been a trend toward what has been termed 'displacement ventilation' in which air is supplied from low wall outlets. This has proven satisfactory for relatively light cooling loads but has not been adequate for full cooling loads or for heating without supplementing the supply air introduced into the room from above or from high sidewall outlets.

Displacement and under floor ventilation (common in the U.K. & U.S.A.) tends to be effective since the ventilating air is supplied directly into the occupied zone. While conventional overhead distributions are effective if proper air distribution principles are followed in the design, there are frequent problems. Lightly loaded VAV systems can result in poor diffusion and poor ventilation effectiveness if the design does not consider the lowest air flow modes of operation. Even with proper air-conditioning system design, adequately

specified ventilation rates, and proper filtration, the buildings air quality might still be poor. Energy wastage is still possible unless the system has been properly installed, tested, balanced and the controls properly calibrated, adjusted and maintained.

ALTERNATIVE APPROACHES TO AIR QUALITY

Fanger (1990) is opposed to the current approaches to air quality on the basis that they do not work. Current standards are based on people being the main polluter. He cites three reasons why the present standards do not work:

(a) There is a misconception that the occupants are the major or exclusive polluters. Studies by (Fanger et al., 1988; Pejtersen et al., 1990; Thorstensen et al., 1990) have documented that the building itself including the furnishing, carpeting and even the air conditioning itself may be a more important polluter than the occupants.

(b) Most of the existing standards prescribe air quantity rather than quality.

(c) The supplied air quantity is the same whether the outdoor air has a high or low quality.

Fanger proposes two new unit measures of 'pollution' to be incorporated in system design. These are the 'Olf' unit which quantifies the source strength of air pollution and the decipol which is the olfactory quantification or perception of the pollutant.

Using the odour criteria applied to pollution from people, Fanger (1988), suggests that an additional 40 L/s could be necessary for pollution originating from the building. However, it is not clear from Fanger's work whether some of the pollution from the building actually originated from people but have been transferred to 'sinks' (pollutants deposited on surfaces) in the building and its services.

Fanger's theory has been opposed by other researchers and is currently the subject of debate.

KEYPOINTS: Adequate ventilation is needed for biological health, removal of contaminants and local cooling. Present standards using the number of building occupants as the measure of required ventilation load may be inadequate as furnishings and machinery may contribute to contaminant levels. The effectiveness of the ventilation system needs to be monitored. In many cases poor systems operation and control has been found to be the cause of poor air quality.

VOLATILE ORGANIC COMPOUNDS

EVIDENCE OF EFFECTS ON BRI

Volatile organic compounds (VOCs) are a group of carbon-based chemicals that evaporate easily at room temperature and thus give off vapours that can be inhaled. VOC's are ubiquitous and are off gassed from building materials, consumer goods, clothing, cosmetics, cleaning fluids, office equipment and a host of other products.

Effects of various compounds are known at their occupational threshold limits, however levels of individual compounds in office buildings rarely exceed these threshold limit values (TLVs). A criticism of TLVs is that the synergistic effect of combinations of a number of compounds (each in low concentration) is unknown. Molhave (1990), has reported some effects of Total Volatile Organic Compound (TVOCs) load and their ability to produce SBS type symptoms (refer Table 6).

There is some evidence that VOCs decrease over time following completion or refit of a building. In one case (Ekberg 1991) reported that levels decreased rapidly over the first three months and were within established guidelines after a year. Baldwin et al., (1990), state that levels normally decrease to a stable level after approximately one year.

In one interventional study Hellstrom et al., (1990) eliminated spontaneous complaints in a school by removing PVC flooring and enlarging a flue. There are a number of studies linking elevated formaldehyde levels (one of the more ubiquitous VOCs) with eye and upper respiratory symptoms.

Raw (1992), indicates that VOCs are always worth considering as a potential cause of symptoms in the case of new buildings.

VOLATILE ORGANIC COMPOUNDS

Wallace (1987), concluded that nearly every home and place of business contains materials that can cause elevated exposure levels of VOCs. Over 900 separate volatile organic compounds have been discovered in indoor air. A report prepared for the EPA by Spaite *et al.*, (1990), classified a large number of materials as sources of indoor air pollution.

Baechler *et al.*,(1989) has shown that paint is a substantial source of indoor pollution contributing high levels of toluene and xylene to the indoor air.

Sheldon *et al.*, (1988) found that air samples collected from new buildings had particularly high VOC levels and hence, the link has been made between VOC's and, so called, *temporarily sick* buildings. Further, these compounds were similar in two new buildings, suggesting that the findings may be generally true for many buildings. Materials emitting these compounds at the highest rates were surface coatings such as adhesives, caulking, and paints; wall and floor coverings such as moulding, linoleum tile, and carpet, and other materials such as telephone cable.

Berglund et al., (1981) lists 307 volatile chemical compounds detected in indoor air in different countries. Included are unexpected compounds such as chloroform *being released* from synthetic carpet.

Baechler (1990), suggests that organic compounds can be grouped according to structural similarities.

TABLE 5 - STRUCTURE AND SOURCES OF VARIOUS ORGANIC COMPOUNDS

Structure	Organic Compound	Possible Source
Aliphatic and oxygenated aliphatic hydrocarbons	α-pinene, n-decane, n-undecane, n-dodecane, propane, butane, n-butylacetate, ethoxy ethyl acetate octane	Cooking and heating fuels, aerosols propellants, cleaning compounds, paints, carpet mouldings, particle board, refrigerants, lubricants, flavouring agents, perfume base
Halogenated hydrocarbons	Chloroform, methyl chloroform, dichloromethane, polychlorinated biphenyl, 1,1,1-trichloroethane, chlorobenzene, dichlorobenzene, carbon tetrachloride	Aerosol propellants, fumigants, pesticides, refrigerants, adhesives, caulk, paint, linoleum tile, carpet, latex paint, and degreasing, dewaxing, and dry cleaning solvents
Aromatic hydrocarbons	Xylene, ethyl benzene, trimethylbenzene, ethyltoluene, propylbenzene, benzene, styrene, toluene	Paints, varnishes, glues, enamels, lacquers, cleaners, adhesives, moulding, insulation, linoleum tile, carpet
Alcohols	Ethanol, methanol	lacquers, varnishes, polish removers, adhesives
Ketones	Acetone, dimethyl ketone, methyl ethyl ketone	Lacquers, varnishes, polish removers, adhesives, cleaners
Aldehydes	Formaldehyde, nonanal	Fungicides, germicides, disinfectants, artificial and permanent-press textiles, paper, particle boards, cosmetics, flavouring agents, insulation

Physiological Reactions to the Various Structures

Solvents are a major source of 'organic pollution'. According to Seedorf et al., (1990), the 'solvent syndrome' has been recognised in Belgium as an occupational disease since 1976 and, as such, is a condition that brings entitlement to compensation.

The acute, narcotic effects of occupational exposure has been known for some time. Their chronic effects are not as well understood and have only been the subject of recent research (over the last decade or so). The first indication of a relationship between solvent exposure and the development of intellectual impairment was in Scandinavian studies in the 1970's, in which non-specific symptoms of Central Nervous System (CNS) dysfunction such as headache, forgetfulness, insomnia, abnormal fatigue and personality changes were described (Axelson et al., 1976; Arlien-Soborg et al., 1979).

It is interesting to note that some symptoms associated with BRI are similar to those associated with certain groups of organic compounds, particularly narcosis, mild central nervous system effects and local irritation, particularly of the eyes nose and throat. While the levels of compound exposure is generally below that of 'safe' occupational exposure, there is a growing body of opinion amongst researchers that suggests 'mild' symptoms may be experienced at relatively low exposure levels. While it is clearly beyond the scope of the present discussion, the general symptoms associated with the Baechler's (1990) groupings is informative (refer Appendix 4).

Problems Associated with Threshold Limit Values (also refer Appendix 5)

Considerable international research is being undertaken to gain a better understanding of the effects of specific organic compounds or specific mixtures. Findings, in many cases, have resulted in an alteration (usually downwards) of the recommended TLVs. The issues involved are complex and beyond the scope of this study. However an attempt will be made to give an overview of the problem. Firstly the scope of the problem is vast. If it were assumed, for instance, that the 900 or so organic compounds, currently recognised, were finite and that any two or more of these compounds could, potentially, mix to form a new compound with distinct physiologically reactive properties - the potential combinations are, for practical purposes, are incalculable.

Some evidence of sensitization to organic compounds has been reported. For example, Altmann et al., (1990) used neurophysiological and psychophysical measurements to reveal the effects of acute low-level organic solvent exposure in humans. Reactions to the organic solvent tetrachloroethylene (also called, Perchloroethylene) were studied. The results indicated that the visual function in healthy adult males was mildly affected by Perchloroethylene levels of 50 ppm, maintained over a period of 4 days for 4 hours daily. This exposure level corresponds to less than half of the maximally allowed workplace level in New Zealand which allows a concentration of 50 ppm for 8 hours over a working week of 5 days. A particularly interesting finding was that the Perchloroethylene concentrations of the subjects exposed to 50 ppm were highest on the last exposure days as were the impairments to visual functions. On the day after final exposure, visual function had not returned to normal. This seems to suggest that this is due to the remaining Perchloroethylene concentration in the body. The concentrations of the compound in the blood and in the expired air of experimentally exposed volunteers indicate that long periods of time (ie.

several days) were necessary to eliminate Perchloroethylene completely from the body (Stewart et al., 1970; Ward et al., 1988). The ability of some chemical compounds to remain stored in body tissues or the lack of ability of the organism to efficiently purge itself of these chemicals poses a major problem in selecting appropriate TLVs.

A second criticism of TLVs is that they are solely concerned with the avoidance of chronic effects. The levels encountered in most office type settings rarely reach the levels in occupational settings. It is clearly stated that levels suggested by *American Conference of Government Industrial Hygienists (ACGIH)* are not concerned with mild irritation of the eyes nose and throat.

Total Volatile Organic Compounds (TVOCs)

Because of the unmanageable number of compound combinations, research by Molhave and others into the physiological reactions to TVOCs appears to be a practical alternative.

Molhave et al., (1986) conducted a series of experiments into human reactions to 'low levels' of volatile organic compounds. The researchers exposed a group of 62 human subjects to a *'Standard Mixture'* of 22 volatile organic compounds. The group reacted acutely in their subjective intensity evaluations of air quality, odour intensity and feeling of dry mucous membranes. The acute complaints or symptoms were not provoked by odour intensity and showed no signs of adaption during exposure over a protracted period.

The physiological reactions to the various concentrations were reported as follows.

TABLE 6 - TENTATIVE DOSE RESPONSE RELATION FOR DISCOMFORT RESULTING FROM EXPOSURE TO SOLVENT-LIKE VOLATILE ORGANIC COMPOUNDS

Total Concentration mg/m^3	Irritation and Discomfort	Exposure Range
< 0.20	No irritation or discomfort	the comfort range
0.20-3.0	Irritation and discomfort possible if other exposures interact	the multifactorial exposure range
3.0-25	Exposure effect and probable headache if other exposures interact	the discomfort range
>25	neurotoxic effects other than headache may occur	the toxic range

The above approach has a certain appealing simplicity as it is relatively simple to measure TVOC loads with modern testing equipment. However, the ranges adopted by the researchers at each level of effect are extremely large so it is not possible to make direct attribution of effects on building occupants from measured TVOC levels.

VOC Mitigation

According to Baechler et al., (1990), source removal is the most effective way to control organic pollutant levels.

Increasing whole building ventilation causes a smaller decrease in the concentration of a reactive pollutant such as NO_2 than nonreactive pollutants (Mueller Associates, Inc., 1987).

Local exhaust ventilation in the vicinity of a pollutant source is an effective source specific ventilation control technique (Kandarjian, 1988).

Air cleaning devices and the use of new, less polluting technologies can be used to mitigate VOC loads (Meuller Associates., Inc. et al., 1985).

Sealing particleboard and maintaining low levels of relative humidity can be beneficial in reducing levels of formaldehyde.

Keypoints: Volatile Organic Compounds are ubiquitous in indoor air. The acute effects of a large number of the compounds is known. The synergistic effects of low levels of mixtures of these compounds is less well known. The levels of individual compounds in an office setting is unlikely to exceed the Threshold Limit Value as adopted by the New Zealand Health Department. Volatile organic compounds are implicated in Temporarily Sick Buildings and this condition is common in buildings which have been refurbished or refitted within the proceeding twelve months. Release of VOCs are accelerated by increased levels of humidity.

INORGANIC GASES

There are a number of inorganic gases of concern which are found in the indoor environment. The more common of these are:

(a) Carbon monoxide
(b) Carbon dioxide
(c) Sulphur dioxide
(d) Nitrogen dioxide
(e) Ozone & other photochemical oxidants
(f) Hydrogen sulphide

The first four of these are combustion gases and can be introduced into the indoor environment by a variety of internal and external sources. Cigarette smoking is a major source of 'combustion' pollution (Godish; 1989). More than 2000 gaseous compounds have been identified in cigarette smoke (Mueller Associates, Inc., et al., 1985;1987). Tobacco smoking is discussed under a separate heading.

EVIDENCE OF EFFECTS ON BRI

Many simple inorganic oxides and other gases can have irritant or toxic effects, and can be present in buildings. The primary source of these pollutants is as a result of combustion in or near the building, and less frequently as a result of the decay of building products.

Raw (1992), reports that high levels of CO_2 can contribute to headache and lethargy but that it is very unlikely that CO_2 levels commonly found in buildings is a major cause of SBS or other illness.

Carbon monoxide (CO) is a lethal gas if inhaled in sufficient quantity. Turk et al., (1986) found elevated levels in buildings with underground carparking. Some of the neurobehavioural effects of CO are similar to some BRI symptoms, but generally, levels measured in buildings have tended to be well within 'safe' limits.

Sulphur dioxide is a known mucous membrane and respiratory tract irritant. There have been no reports of elevated levels within commercial office buildings and exposure risk is generally thought to be insignificant.

Nitrogen dioxide has an effect on pulmonary function and exacerbates asthma symptoms. Outdoor levels of NO may be high in heavily populated areas and could be entrained with outdoor air. The primary indoor source of the gas is from faulty combustion and heating equipment. There are a number of published reports linking the gas with illness in children (COST, 1989). However, there is no published evidence linking BRI symptoms with the gas in commercial office buildings. The probable reason for this is that such buildings are unlikely to have unvented heaters.

Ozone from photocopiers is thought to be a cause of BRI symptoms. An investigation by Taylor et al., (1984) links ozone from a 'wet process' photocopier with BRI symptoms. Skov et al., (1989) reported symptoms associated with photocopier work. Sykes (1989), is of the opinion that ozone from photocopier work is unlikely to cause symptoms outside of the staff working in the immediate environment.

Laser printers were also considered to be a potential source of ozone. Sonnino et al., (1984), reported on a number of tests made on a variety of laser printers and deemed them to be safe.

Hydrogen sulphide is a lethal, naturally occurring gas. It is common in geothermal areas, so is not expected to be a risk factor outside of these geographic locations. Relatively low concentrations of the gas can cause eye irritation.

Physiological Reactions to Inorganic Gases

TABLE 7 - SOURCES AND EFFECTS OF INORGANIC GASES

Pollutant	Possible Source	Physiological Effect
Carbon Monoxide	Combustion appliances, underground parking garages	Extensive cardiovascular, neurobehavioural, fibrinolysis, perinatal effects and death
Carbon Dioxide	Human respiration, gas stoves and heaters, wood and tobacco smoke	Insignificant health effects but high levels indicate reduced oxygen in the occupied space.
Sulphur Dioxide	Combustion of fossil fuels.	Alterations in pulmonary function, slowed bronchial mucociliary clearance.
Nitrogen Dioxide	Fossil fuel combustion, vehicle emissions.	Pulmonary effects, respiratory effects.
Ozone	Photocopying processes Laser printers	Pulmonary and upper respiratory effects, increased mucous production, chest tightness, substernal pain, lassitude & nausea
Hydrogen Sulphide	Rayon production, wood pulp plants, oil refining, tanning and naturally occurring in geothermal areas.	Conjunctival irritation, serious CNS effects and death if received in sufficiently large doses.

POLLUTANT SOURCES

Combustion gases are introduced into the indoor environment from multiple sources, such as attached parking garages, cooking facilities within the building itself or they may be introduced from the outside air.

For intermittent sources, source emission rate, building volume, and rate of mixing are the primary factors affecting peak concentrations (Baechler et al., 1990).

For continuous sources, source emission rate and duration, the outdoor concentration, and air exchange rate are the primary factors affecting long-term average concentrations (Mueller Associates, Inc., et al.,1985).

Numerous studies do not offer clear evidence that combustion gases can reach concentrations substantially higher than ambient concentrations (Spengler et al., 1985).

Two major environmental factors that can influence the contribution of ambient (outdoor) levels of combustion gases to indoor contaminant levels are location and time (Mueller Associates, Inc, et al.,1985):

(1) The location of the building relative to major outdoor sources can affect indoor air quality. Depending on the emission rates, air pollutants from stacks, flues, vents and cooling towers can affect indoor air quality. Buildings located near major streets or highways may be affected by the gaseous pollutants generated from vehicles. In general, urban and industrialized areas have higher concentrations than rural areas. Hydrogen sulphide is a risk factor in geothermal areas and sites with ground sources of the gas.

(2) Temporal fluctuations in concentration of outdoor gaseous pollutants will also influence indoor concentrations. Diurnal or seasonal patterns and day-to-day variations in the weather and daily variations in emission rates all determine ambient concentrations.

POLLUTANT SOURCE AND HEALTH EFFECT

(A) CARBON MONOXIDE (CO)

CO is a colourless, odourless, tasteless gas that is slightly lighter than air. It reacts with haemoglobin to form carboxyhaemoglobin (COHb). The affinity of haemoglobin is more than 200 times higher for carbon monoxide than for oxygen.

Carbon monoxide is one of the most common and widely distributed air pollutants. It is the product of incomplete combustion of carbon containing materials, but may also be produced by some industrial and biological processes.

Occurrence In Air

Carbon monoxide is widely generated indoors by unvented combustion appliances, particularly if they are operated in poorly ventilated rooms. In the room air of kitchens short-

term concentrations of 11.5-34.5 mg/m^3 (10-30ppm) have been measured in the Federal Republic of Germany (Seifert; 1984).

In a Dutch study values of 57.5 mg/m^3 (50ppm) and higher were found in 17% of residential dwellings (Boleij et al.,1982).

In a study of 38 commercial buildings, only six had a time weighted concentration of CO greater than the minimum detectable level of 2ppm (Turk et al.,1986). For these six buildings, the mean concentrations ranged from 2.1ppm to 3.3ppm. Three of these buildings had underground parking.

Turner (1988) seldom found levels greater than 9ppm in buildings monitored in Australia, the United Kingdom, and the United States.

In a survey of CO concentrations in 25 individual stores in a large shopping centre in Honolulu, Hawaii, the one hour standard of 200ppm was not exceeded.

Health Effects (refer Appendix 6)

The EPA (Report #600/8-79-022) indicates that four types of health effects are associated with carbon monoxide exposures.

(a) Cardiovascular effects
(b) Neurobehavioural effects
(c) Fibrinolysis effects
(b) Perinatal effects.

(B) CARBON DIOXIDE

The single greatest contributor to indoor CO_2 is human metabolic activity. Significant quantities of CO_2 are also generated by gas stoves and portable kerosene and gas space heaters. Wood and tobacco smoke produce smaller quantities.

In a study of commercial buildings in the Pacific Northwest, thirty-nine 8-hour average CO_2 measurements were made in thirty-seven buildings. Concentrations were found to depend on occupancy levels. Only one 15-minute CO_2 reading in a crowded elementary classroom exceeded 1000ppm (1290ppm). Periods of low occupancy (recess and lunch) were evident in the time series data. Four buildings had instantaneous maxima over 800ppm. Eight hour averages ranged from 337ppm to 840ppm (Turk et al.,1986). General office area CO_2 levels have been found by Turner (1986) to range from 350ppm to 2000ppm.

There are no reports of levels above the occupational exposure limit (5000 ppm).

Health Risk Evaluation

Carbon dioxide is relatively harmless to human beings. However, high levels of the gas is used as an indicator of oxygen deficiency and an indicator of insufficient clean air. Carbon dioxide is also and efficient carrier of odour molecules, so high CO_2 levels may lead to complaints about unpleasant smells.

According to Raw (1992), high levels of CO_2 can contribute to headache and lethargy but it is unlikely that levels found in buildings would reach that critical level.

(C) SULPHUR DIOXIDE

Sulphur dioxide (SO_2) and particles derived from combustion of fossil fuels are a major source of air pollution in urban areas. Sulphur oxides (SO_x) make up a complex pollutant mixture. For clarity they may be divided into two categories:

Sulphur dioxide is a colourless gas that reacts on the surface of a variety of airborne solid particles. It is readily soluble in water and can be oxidized within airborne water droplets. Sulphur dioxide results from the combustion of sulphur-containing ores, and other industrial processes. Domestic fires can also produce emissions of sulphur dioxide.

Indoor concentrations of sulphur dioxide are generally lower than outdoor concentrations, since absorbtion occurs on walls, furniture, cloths and in ventilation systems.

Acid aerosol comprises sulphuric acid (H_2SO_4) which is a strong acid. It is formed from the reaction of sulphur trioxide gas (SO_3) and water. Sulphuric acid is strongly hygroscopic. As a pure material, it is a clear colourless liquid. Ammonium bisulphate (NH_4HSO_4) is less acidic than pure sulphuric acid and is a crystalline solid.

Health Risk Evaluation

The effects of 'acid aerosols' have been studied in animal experiments. The WHO (Report #23, 1987) indicates that the most commonly observed effects are alterations in pulmonary flow resistance and irritation of the mucous membranes. Animals exposed to higher concentrations exhibited slowed mucociliary clearance.

In human experimentation, asthmatics exhibited marked dyspnoea, wheezing and shortness of breath.

There are no reports of exposure to the gas in commercial buildings and, because of it's potential source, risk of exposure would probably be higher in domestic residences.

(D) NITROGEN DIOXIDE

Many chemical species of nitrogen oxides (NO_x) exist, but the species of most interest from the point of view of human health is nitrogen dioxide (NO_2). Nitrogen dioxide is soluble in water, reddish-brown in colour and a strong oxidant. Indoor levels of nitrogen oxides are primarily affected by gas stoves, unvented kerosene and gas space heaters, and, to a lesser degree tobacco smoking. Infiltration of NO from outdoor sources is significant in heavily populated, industrialized areas where fossil fuel combustion and vehicle emissions are a major outdoor source of the pollutant (Baechler et al.,1990).

Health Risk Evaluation (refer Appendix 6)

It should be noted that risk from NO exposure in commercial office buildings is considered to be low unless there are unvented cooking or heating appliances in use.

(E) HYDROGEN SULPHIDE

Hydrogen sulphide (H_2S) is a colourless gas, soluble in various liquids including water and alcohol. It can be formed under conditions of deficient oxygen, in the presence of organic material and sulphate. Hydrogen sulphide occurs around sulphur springs and lakes, and is an air contaminant in geothermally active areas. Saline marshes can also produce the gas (Steudler et al., 1984).

Human activities can release naturally occurring hydrogen sulphide into ambient air. For instance, some natural gas deposits can contain up to 42% hydrogen sulphide (WHO Report #19, 1981). In industry, hydrogen sulphide can be formed whenever elemental sulphur or sulphur-containing compounds come into contact with organic materials at high temperatures. Hydrogen sulphide is formed during coke production, viscose rayon production, waste water treatment plants, wood pulp production using the sulphate method, sulphur extraction, oil refining and tanning.

In and around Rotorua, New Zealand, average ambient hydrogen sulphide concentrations are 0.08 m/m^3 (0.05ppm). Near a pulp and paper mill in California peak concentrations of up to 0.20 m/m^3 (0.13ppm) were measured (WHO Report #19, 1981).

Routes of Exposure

The respiratory system is the main route of human exposure to hydrogen sulphide. A report by Ronk et al., (1985), criticizes the earlier belief that hydrogen sulphide can enter the body via tympanic membrane defects in workplace concentrations.

Health Risk Evaluation (refer Appendix 6)

(F) OZONE

Ozone (O_3) is one of the strongest oxidizing agents. In the troposphere, it is formed indirectly by the action of sunlight on nitrogen dioxide. There are no significant anthropogenic emissions of ozone into the atmosphere. Existing ozone has been formed by chemical reactions that occur in the air.

The presence of hydroxyl radicals and volatile organic compounds in the atmosphere, either of natural or anthropogenic origin, causes a shift in the atmospheric equilibrium towards much higher concentrations of ozone.

Apart from ozone, other compounds are formed during the photochemical processes, such as the oxidants peroxyacyl nitrates, nitric acid and hydrogen peroxide, as well as secondary aldehydes, formic acid, fine particulates and an array of short lived radicals.

The primary indoor sources of ozone in office buildings is from photocopiers.

Ozone exposure has been implicated as a cause of BRI symptoms by some commentators (Taylor et al., 1984, Skov et al., 1989).

Health Risk Evaluation (refer Appendix 6)

INORGANIC GAS MITIGATION

According to Baechler et al., (1990), pollutant reactivity can be as important as air exchange rates in reducing indoor combustion gas concentrations, especially when the air exchange rate is low.

Nitrogen dioxide is removed through chemical reaction.

Carbon monoxide may be adsorbed on indoor surfaces.

The rate at which reactions occur varies for each pollutant and is influenced by temperature, moisture, presence of other compounds, and types of surfaces found (Mueller Associates, Inc., et al., 1985).

Combustion byproducts may be removed from the indoor atmosphere by a number of mechanisms other than air exchange. Individual pollutants may be reduced by one or more of the following physical or chemical reactions: adsorption, absorption, conversion, and deposition.

KEYPOINTS: Outdoor levels of inorganic gases are usually higher than indoor concentrations. They can be introduced into the indoor environment from a number of internal and external sources. Venting of pollutant sources such as photocopiers and combustion appliances directly to the outside or into a stack is recommended. The location of air intakes in buildings is an issue. A number of problems associated with particular buildings have been sourced to the leakage of gases into the buildings from underground carparks. The health effects associated with some of the gases can be acute or chronic and most have been associated with upper respiratory symptoms. Generally, individuals do not acclimatize after being exposed to elevated levels of these gases and remain responsive.

RESPIRABLE SUSPENDED PARTICULATES (RSPS)

EVIDENCE OF EFFECTS ON BRI

There are many sources of non-viable particulates and aerosols in indoor air, including plants, animals, mineral fibres, combustion, home and personal aerosols (Owen et al., 1990).

It is now well accepted that RSPs play a pivotal role in a number of BRI symptoms. In a cross sectional study of three buildings in Washington DC, Wallace et al., (1991) identified airborne dust as the strongest correlate with BRI symptoms. In the Danish Town Hall study, Graverson et al., (1990), found that the presence of macromolecular organic dust was correlated with mucosal and general symptoms.

Bishop et al., (1985) attempted to link symptoms with insulating materials associated with building services although this remains unproven.

A confounding factor was identified by Harrisson et al., (1990). Adverse health effects are more commonly reported in buildings with HVAC systems than in naturally ventilated buildings, while levels of airborne particulates are generally lower in the former.

Some concern has been voiced as to the role of mineral fibres in eye and skin irritation although the linkages have not been proven empirically.

RESPIRABLE SUSPENDED PARTICULATES, SIZE AND BEHAVIOUR

The behaviour of inhaled particles is a highly technical subject and is beyond the scope of this study. However a basic knowledge of the mechanisms important, because:

(a) Some of the particles are intrinsically toxic because of their chemical or physical characteristics (eg, asbestos);
(b) Other substances are adsorbed by the particles which then act as a carrier (eg. viruses, radon). The size and shape of particles is a critical issue. Generally, the deeper into the lungs that foreign bodies penetrate, the more potentially serious the damage.

For detail refer Appendix 7.

THRESHOLD LIMITS AND FILTRATION

Threshold limit values (TLVs) of all hazardous substances are reviewed and published annually by the American Conference of Government Industrial Hygienists (ACGIH) and these standards are almost universally adopted by the New Zealand Health department. The TLV adopted does not indicate a definitive dust concentration to which it is *safe* to be exposed, and above which it is not. The TLV assumes that, if individual hazardous materials can be prevented from exceeding a certain empirically determined level at any time, then below that level, it is unlikely to cause disease.

The occupational exposure limits in New Zealand are based on two broad categories. These are fibrogenic dusts, which cause scar tissue to be formed in the lungs and 'nuisance dusts' which do not cause significant organic disease or toxic effect when exposures are kept under reasonable control. However, TLVs are usually based on occupational exposures and the 'reasonable level limit' may induce sneezing, mucous production, etc. Inert or 'nuisance particles' do not have an irritant effect on the upper respiratory tract. They do not cause fibrotic pneumoconiosis, although this may occur if fibrotic or radioactive particles are associated with them. Some inorganic examples are chalk, cement, corundum, emery, gypsum, iron oxides, limestone and silicon carbide.

TLVs are constantly under review. Potter (1988), suggests that current practice of basing limits on respirable mass concentration is a *poor* indicator of the number of particles present in air, particularly if those in the smaller range size predominate. The health concern is governed more by the size of the particles present rather than the quantity. The particle size most likely to be retained by the human body will be in the size range of less than 1 μm. Respirable particulates from smoking products are mostly in the small size range ($< 0.5\mu$m) and in common with many indoor air pollutants, no recommended limits, or dose related curves are available.

According to Potter (1988), the Japanese mandatory indoor environment limit of 150$\mu g/m^3$ is more realistic than the ACGIH level, and suggests that in general office environment an even lower threshold of 100$\mu g/m^3$ would be preferable.

Pollutant Source and Strength

Indoor sources of RSPs include combustion sources such as unvented gas appliances, asbestos construction materials, and common dust. Other incidental indoor sources include aerosol sprays, resuspension of dust from vacuum cleaning, and wear and tear of building materials.

In the office environment, photocopy dust may contribute to elevated levels of RSPs (Turk et al., 1986; Mueller Associates, Inc., et al., 1987).

Some RSPs are from outdoor sources and enter the building via the HVAC system or through natural ventilation and infiltration.

Tobacco smoke and asbestos are major contributors to overall levels but are considered under separate headings.

Fibreglass and cellulose fibres are a contributor to overall levels. Fibreglass particles can be released from the insulation used in ventilation ducting (duct board). Gamboa (1988) found that fibreglass fibres released from duct board and fibreglass duct liner were typically well below permissable exposure limits proposed by NIOSH in 1977. Nevertheless, these releases still resulted in fibre levels approximately twice the normal background limit.

MITIGATION

NASAL AIRWAYS

These are of great importance in removing particles, vapours and gases from inhaled air before they can gain access to the lower respiratory airways. Very few compact particles larger than 20 μm and only about half of those >5 μm pass the nasal filter during breathing at rest, and many smaller particles of smaller size are also removed (Procter et al., 1969). Deposition of many particles 5 μm or less in diameter is increased in transit due to humidification which causes them to grow in size; but if nasal breathing is impossible, as in the case of obstructive disease of the airways or the inhalation of tobacco smoke which bypasses them, the number of particles reaching the pulmonary airways and alveoli is much greater than would otherwise be the case. Habitual mouth breathing allows an increased number of particles to reach the lower airways (Albert et al., 1972).

Peak airflows through the nose are approximately 1.01 l/s. Therefore, factors which lower this rate, such as nasal disease or other causes of increased resistance to air flow, and work loads which demand high ventilation rates result in a change from nasal to mouth breathing (Procter et al., 1973). Resistance to air flow is increased by low ambient temperatures (Salman et al., 1971).

ARTIFICIAL MECHANISMS

Particles are removed from the air, generally, by filtration. The ability of filters to remove airborne matter from the air stream is characterized by two parameters, 'arrestance' and 'efficiency'. Arrestance is a measure of the total weight of dust captured in the filter. The weight of dust is expressed as a percentage of the total weight of dust entering the filter. Arrestance provides a good indication of a filters ability to remove larger, heavier particles and is used as a measure of the performance of the lower grade filters. The performance of the higher grade filters is rated by their 'efficiency'. This parameter is determined by the filters ability to remove the microscopically sized particles from the airstream. Potter et al., (1988) provides the following filter grade and use information.

TABLE 8 - FILTER GRADES AND EFFICIENCY

FILTER GRADES		
GRADE	ARRESTANCE (A)%	EFFICIENCY (E)%
1	A < 65	
2	65 < A < 80	
3	80 < A < 90	
4	A > 90	
5		40 < E < 60
6		60 < E < 80
7		80 < E < 90
8		90 < E < 95
9		E > 95

TABLE 9 - RECOMMENDED FILTER GRADES FOR SPECIFIC ENVIRONMENTS

RECOMMENDED FILTER GRADES FOR PARTICULAR ENVIRONMENTS	
GRADE	APPLICATION
2/3	Pre-filters and for systems serving areas not requiring any great degree of cleanliness such as toilet supply systems and light industrial applications
4	For application as main filters for low to moderate cleanliness
5	Main filters for general application where decor is not critical
6	As for 5, but with added decor protection. Intermediate filter to extend the life of a HEPA main filter
7	As for 5, but for use where protection of decor is particularly important
8	High protection from dust staining, suitable for computer rooms and other areas containing electronic equipment
9	For high quality filtration but where HEPA filters are not justified

In addition to the nine grades of filter, there is a further classification known as High Efficiency Particulate Air (HEPA) filters, sometimes known as absolute filters. They are designed to provide very high efficiency filtration of particles in the sub micron range. Typical applications include clean rooms, operating theatres, research laboratories, laminar flow cabinets and nuclear installations. For general office environments, grade 6 filters are recommended with grade 7 in more prudently controlled environments.

Commonly reported faults with regard to filters is that they are not changed and cleaned regularly or the filter housing becomes damaged resulting in the filter being by-passed by the air stream.

Vacuum Cleaners

Poor quality vacuum cleaners can exacerbate an RSP problem by resuspending previously settled dust.

The type of final filters fitted to vacuum cleaners can have a significant effect on the levels of particulates which are generated during cleaning activities. Smith et al., (1990), found that a standard vacuum cleaner emitted on average 9.1×10^{10} particles per minute in the range 0.01-10 μm during vacuuming.

Natural Mechanisms

Natural mechanisms for RSP removal include physical deposition, chemical transformation, and particle agglomeration. Chemical transformation of the non fibrous particulates increase with increasing temperature. Higher relative humidities encourage the agglomeration of some airborne RSP into larger particles, accelerating their settling rate, as well as converting them into particles that are less likely to be inhaled. When larger particles are inhaled, they are deposited in the nose/mouth and not deep in the lungs (Baechler et al., 1990).

Interactions with Energy Conservation Measures

Building/system components that most significantly influence indoor levels of pollutants include:

(1) Envelope construction material and technique.
(2) Infiltration and natural ventilation.
(3) Control and operation of the ventilation system.

Energy conservation measures (ECMs) that alter or affect any of the above components will, either directly or indirectly, affect indoor RSP levels. A direct effect is one in which the ECM either increases or decreases the source emanation rates. An indirect effect is one in which the pollutant concentrations are increased or decreased. For example, in the absence of a strong indoor source (eg. tobacco smoke), any ECM that results in a net reduction of infiltration will likely reduce indoor RSP concentrations due to the reduction of infiltration of outdoor particulates (ie. dust) to the indoor environment. On the other hand, when major sources are present, ventilation that reduces the rate of infiltration will result in greater indoor RSP concentrations because of reduced dilution with fresh air.

Building envelope - installation of additional insulation in the walls, ceiling, roof, foundation, or slab can directly increase the level of RSP fibres by increasing the source of fibreglass & cellulose material in the building.

HVAC system - the only HVAC system ECM that will affect indoor RSP/fibre levels will be the installation of duct insulation. One study suggests that the relative increase in fibre levels can be large, although normal background levels are generally low and it is unlikely any standards will be exceeded.

Ventilation - Recirculation of exhaust air using activated carbon filters has the potential for directly increasing RSP/fibre levels by increasing the opportunity for fibre release into the ducting. This same ECM, however, will also remove RSPs from the indoor air as they are recirculated through the ventilation system.

The installation of vortex hoods in the kitchen areas of restaurants will effectively remove much of the combustion byproduct RSPs generated by the gas burners, thereby reducing RSP levels in the remainder of the building. Ideally this air should be directly vented to the outside and dumped as it tends to be hot, damp, odorous and contaminated.

Summary of Mitigation Techniques

Mitigation techniques to control RSPs fall into three broad categories: source control, ventilation, and air cleaning. Mueller Associates., Inc., et al., (1987), suggest a number of specific measures:

- Limit or eliminate tobacco smoking indoors.
- Use electrostatic precipitators and high efficiency particulate (HEPA) filters as effective removal media.
- Change air filters regularly and ensure that they are fitted correctly. A common fault caused by a damaged housing results in a gap around the filter, resulting in the filter being bypassed.
- Vent combustion appliances directly outdoors.

Keypoints: RSPs play an important role in air quality and health in that they can either directly cause illness or can act as a raft for other organisms to penetrate the respiratory system. The size and shape of the particles is a pivotal issue. Generally, the smaller the particle the deeper its penetration and thus the greater its potential to cause damage. Current cleaning methods remove large, relatively harmless particles easily, but have little effect in removing the small particles.

TOBACCO SMOKE

EVIDENCE OF THE EFFECT OF SMOKING ON BRI

Cigarette smoke is a significant source of indoor pollution that can harm nonsmokers. Extensive toxicologic, experimental and epidemiologic data, largely collected since the 1950s have clearly established that active cigarette smoking is the major preventable cause of morbidity and mortality (U.S. Department of Health, report 79-50066, 1979). Involuntary exposure to tobacco smoke has only recently been investigated as a risk factor for disease in non-smokers. The evidence on involuntary smoking is more limited in scope than for active smoking, nevertheless there is some compelling statistical evidence to support the hypothesis that involuntary smoking also causes health problems.

Evidence of the role of tobacco smoke in BRI is ambiguous. Zweers et al., (1990), found nose and throat symptoms were higher among smokers but analysis did not detect a significant effect of passive exposure on symptom reporting.

Urch et al., (1990) showed that nasal symptoms and headache are correlated with exposure to smoke in non-asthmatics. In asthmatics there was additionally a correlation with eye and throat symptoms and fatigue.

In contrast, studies which have compared symptoms in buildings in which smoking is or is not allowed have generally shown null results (Hedge et al., 1991; Sterling et al., 1987; Taylor et al., 1984).

Nevertheless, although there are some contradictions, the great body of evidence is that tobacco smoke is a major contributor to morbidity and death within the general community.

For detail refer Appendix 8.

LEGAL POSITION

The present legal situation in New Zealand is in a state of change.

Current 'clean-air' legislation allows smoking in the workplace, provided that none of the smoker's co-workers object. Apart from the health risk to employees in the smoking zone, respirable particulates may be distributed outside the area by action of the HVAC system, affecting other building occupants.

Employers were traditionally insured against action by employees through the Accident Compensation Act. This Act has now been replaced by *Accident Rehabilitation and Compensation Insurance Act (1992)*. Section 7 of this Act specifically excludes personal injury attributable to air conditioning systems and passive smoking.

Judicial interpretation of this section of the Act under challenge, is awaited. If an employee suffers a chronic or debilitating disease, in the absence of state funded insurance, natural justice would suggest that a right to sue exists. Therefore, any employer or building owner

who allows smoking in the building or workplace may become a legitimate target for litigation.

The Australian federal court decision in February 1991, seems to smooth the way for damages claims in that country at least. Justice Morling ruled that *'passive smoking causes lung cancer, asthma and respiratory disease in children.'*

CONTROL STRATEGIES

In the absence of a ban on smoking there are a number of other control strategies available.

Increasing ventilation can reduce concentrations of some smoke constituents, but effective removal of pollutants such as CO probably requires unrealistic ventilation rates (U.S. Department of Health, Education and Welfare, report 79-50066, 1979).

There is some evidence that it is possible to manufacture cigarettes that produce less sidestream smoke. In one study (Naylor Dana Institute, 1973) of a cigarette treated with 'Colite', a substance to prevent fires caused by smouldering cigarettes, the sidestream smoke from the treated cigarettes contained 24% less particulate matter and 19% less nicotine than untreated cigarettes.

The compromise situation, to control the health effects of involuntary smoking is to permit nonsmokers to avoid exposure through the provision of separate, self ventilated areas for smokers. Repace et al., (1980), shows that such separation significantly reduces the level of particulate pollution in the air nonsmokers breathe.

KEYPOINTS: There is a compelling body of evidence that cigarette smoke is a health hazard to non smokers in the work environment. Tobacco smoke is a major contributor to RSPs and Organic & Inorganic pollutant levels. It is the single most avoidable source of pollution. There is some evidence linking environmental tobacco smoke with BRI symptoms but reported results are ambiguous. Recent changes to the Accident Compensation Legislation, makes employers a potential target of litigation if illnesses is contracted through passive smoking.

ENVIRONMENTAL COMFORT

THERMAL COMFORT

IMPLICATION OF HEAT IN BRI SYMPTOMS

Potter (1988) reports that there is a correlation between temperatures at and above 23°C and the prevalence of BRI type symptoms, principally during the heating season.

Valbjorn et al., (1984), reported temperatures above 23°C increased the occurrence of mucous membrane symptoms but not headache.

In a Canadian study McDonald et al., (1986) reported that high temperature in combination with low relative humidity and imperfect ventilation increased the incidence of BRI type symptoms, although the role of temperature was not identified.

BRI type symptoms are often correlated with the perception of hot or dry air (Wallace et al., 1991).

Wyon et al., (1973) reported that small, rapid fluctuations in the thermal environment decreased work rate and accuracy and caused sleepiness and fatigue.

THERMAL STANDARDS

Various standards have been set for optimum comfort of building occupants, the two most widely accepted standards are derived from Fanger's comfort equation and used as the basis for ISO 7730 (1984), and ASHRAE 55-81 (1981). Neither of these standards have a stated aim to provide comfort for everybody. The ASHRAE standard, for instance has a definition:

> *Acceptable thermal environment is an environment in which at least 80% of the occupants would find thermally acceptable.*

ISO 7730 (1984), sets an optimum temperature (air, radiant, and radiant symmetry) range for people at different metabolic rates and wearing different clothing. Although the standard is based on sensory perception it draws up complex equations to allow for the calculation of the 'operative temperature'. Recommended comfort requirements, from ISO 7730 (1984), are:

(a) Operative temperature of 20-24°C.
(b) Vertical air temperature difference between 1.1 and 0.1m (head and ankle height) less than 3°C.
(c) Floor surface temperature 19-26°C (29°C with floor heating systems).
(d) Mean air velocity less than $0.15 m.s^{-1}$.
(e) Radiant temperature asymmetry (due to windows etc.) less than 10°C.
(f) Radiant temperature asymmetry from a warm ceiling less than 5°C.

Asymmetric radiation may be a problem in the following instances:

(1) Local cooling - radiation exchange with adjacent cold surfaces such as single glazed windows.
(2) Local heating - radiation exchange with adjacent hot surfaces, such as heated ceilings, etc.
(3) Intrusion of short wave radiation such as solar radiation through glazing.

Ashley (1986), relates a case where a female clerk was complaining of cold, draught etc. Her complaints eventually led to challenges of a sick building. An engineer put a thermometer on her desk which read 23°C and then looked around to see piles of files stacked on top of a convector by the window. The clerk was sitting in the path of a downdraught and close enough to a window to be effected by radiant cold. Removing the files was sufficient to remedy the situation. The engineer argued, with some validity, that this was a design fault. Designers must expect users to be *totally ignorant* and to do stupid things with services. If the convectors had rounded tops, this would prevent files, plants etc., from blocking the outlets.

Predicted Mean Vote

The Predicted Mean Vote (PMV) and the Predicted Percentage of Dissatisfied (PPD) indices defined in ISO 7730 (1984) are an assessment procedure for the likely satisfaction rating of a particular environment. Their purpose are to predict conditions which are most satisfactory to most of the people for most of the time.

Sykes (1989), suggests that, 'ideal' conditions can vary from population to population (surveys have suggested optimum temperatures ranging from 17°C for English workers in winter to 37°C for Baghdad office workers in summer) and from person to person within the population. The different acceptable temperatures for various countries and seasons might be explained by the necessity to correlate indoor and outdoor air temperature to avoid thermal shock when entering and leaving the building and the need for body acclimatization. While the preferred temperature may be influenced by expectation and possibly by extreme outdoor temperatures, these studies show that no single thermal environment is ideal for everyone; even if conditions are 'ideal', a percentage of occupants will be dissatisfied.

Schneider (1988), indicates that sensitivity to heat varies considerably from one individual to another and may be influenced by factors such as clothing, body activity, age, health, gender, etc. He believes that excessively high temperatures (which differ individually and may be approached in a lower range of room temperature if physical work is involved) will cause a reduction in concentration and performance, increase in pulse rate, increase in skin moisture and temperature resulting in fatigue and indisposition. He goes on to state that, in connection with strenuous intellectual activity the air temperature should not be above 18°C. If the surface temperatures are sufficiently high (especially floor temperatures) optimum air temperatures are around 10°C (cool head - warm feet), although this 'extreme' temperature difference between head and feet seems to run contrary to the other authorities.

Dissatisfaction with the thermal environment appears to be a greater problem in large air conditioned buildings than in smaller and naturally ventilated buildings. The standards set in ISO 7730-1984 are complex and not easy to achieve. Whereas in a building with opening

windows and radiators the occupants are able to vary their thermal environment to some extent. On the other hand, if the air conditioning system in a 'tight' building fails to control the thermal environment, there is often little the occupants can do to improve the conditions. This may give rise to stress induced by a feeling of helplessness - a common complaint amongst workers in sealed buildings.

A sensation of 'stuffiness' can also contribute to dissatisfaction with the thermal environment. Bedford (1974), attributes stuffiness to a lack of stimulation, suggesting that a change in air velocity will stimulate the tactile nerve endings in the skin. This seems to fit in part with the theory by Berglund et al., (1984) that imperceptibly small stimuli may be to blame for some BRI symptoms.

Int Hout (1984), reports that it was possible to reduce the number of complaints of stuffiness by using individual fans to increase the air velocity from 0.05 to 0.6 m/s^{-1}.

Mathews (1985), reports the same result by reducing the air temperature from 23 to 21° C.

Summary of Control Strategies

Jones (1985), states for a person to feel comfortable the following conditions are desirable:

(1) The air temperature should be higher than the mean radiant temperature in summer, but lower in winter.
(2) The average air velocity in the room should not exceed 0.15 m/s^{-1} in an air conditioned room but higher velocities may be acceptable with air temperatures greater than 26°C.
(3) The temperature difference between feet and head should be as small as possible, normally not exceeding 1.5°C and never more than 3°C.
(4) The radiant temperature asymmetry should not be more than 5°C vertically and 10°C horizontally.

Keypoints: There is a strong correlation between the temperatures at or above 23°C and the prevalence of BRI symptoms. No single thermal environment is ideal for everyone, a percentage of occupants will always be dissatisfied. Thermal dissatisfaction in air conditioned buildings is frequently associated with occupants feeling that they lack control over their immediate environment. Air movement is an integral part of the thermal comfort equation.

RELATIVE HUMIDITY

IMPLICATION OF HUMIDITY IN BRI SYMPTOMS

The moisture content of the air is thought to be a pivotal factor in BRI symptoms. A delicate balance seems to be required.

If the moisture content is too high it supports the growth of viable particulates such as mould and dust mites and it accelerates the release of water soluble organic compounds such as formaldehyde. A study by Smith et al., (1991) indicates that raising humidification levels resulted in an increase of airborne micro-organisms.

If the moisture content is too low it can lead to mucous membrane dryness and irritation (Reinikainen et al., 1988), nose bleeds and electrostatic shocks. In a follow up study Reinikainen et al., (1990) reported that increased humidification reduced symptoms of dry skin and dry eyes while irritation of the throat was not significantly decreased. There was a significant decrease in allergic type symptoms which consisted of nasal congestion and sneezing.

RELATIVE HUMIDITY

Humidity refers to the moisture in the air and is expressed as a percentage. The warmer the air the more moisture it can hold. In winter, the ambient moisture content in the air can be very low. Where humidity is to be controlled, current practice recommends a range between 40-60%. The C.I.B.S.E (1991) guide relating to ventilation and air conditioning requirements, states:

> *Recent experiments have shown that relative humidity of 30% can cause disease in rats. Moreover, it appears that most animals require a relative humidity of approximately 50%. For these reasons, winter humidification is now considered essential on a 100% outdoor air system and where precautions must be taken with the structure. Where rooms are not air conditioned no severe problems are found with summer temperatures in this country (ie. England), assuming a good air change rate.*
>
> *For precise experiments, humidity control of 50%±10% is normally considered satisfactory within the specified temperature limits.*

Thermal comfort in buildings will not be greatly affected by large changes in relative humidity and human beings can easily tolerate values outside the specified range without experiencing major discomfort.

An experiment by Hansen et al., (1990) shows that people are poor judges of the ambient moisture levels. The researchers varied the relative humidity between 33-43%. The subjects did not notice the change and simply reported feeling that the air was dry.

While relative humidity does not cause a feeling of comfort/discomfort unless it reaches extreme values, it is pivotal to the evaluation of building health because of its interrelationship with other factors as previously discussed.

In addition to the above factors, Mant et al., (1992), hypothesise that high humidity can cause clothing to become wet, leading to difficulty in maintaining suitable body temperature. However there is no direct evidence to support this hypothesis.

Summary Effects of High Relative Humidity

(1) Dust mites

Dust mites are a major biological allergin (refer biological contaminants for a detailed discussion). There is compelling evidence that mite numbers can be reduced by decreasing relative humidity (Mathews, 1978).

(2) Airborne microorganisms

Humidity may affect the survival of bacteria and viruses (Sale, 1972; Dimmick et al., 1969). Airborne micro-organisms are less likely to survive in relative humidity of the order 50% or less. Humidity levels over 70% can cause dampness which may encourage the growth of micro-organisms such as fungi (Green, 1984). Humidifiers and the water bath in some humidification systems are implicated in supporting growth of micro-organisms (Parkes, 1982).

(3) Acceleration of Organic Compound Release

A number of studies have demonstrated that formaldehyde emissions tend to increase as humidity levels increase. Kazakevičs et al., (1979), support Troughton's (1969), hypothesis that the long term emission of formaldehyde will be mostly made up of formaldehyde arising from hydrolysis of the urea formaldehyde resin. Their measurements indicated an increase in emission levels as humidity increased. The reason given for this was that at higher humidity, water storage and activity in the 'chipboard' increased, favouring hydrolysis of the resin. Gustafsson (1990) verified these findings and stated that the release of formaldehyde doubles with an increase in RH from 30% to 90% in a linear function.

Other researchers hypothesised that other volatile organic compounds, particularly water soluble compounds would behave in a similar manner. Clobes et al., (1991) investigated the effects of humidity on VOCs and conducted two chamber tests. In both experiments, a nonpolar hydrocarbon mixture of propane/butane (33/67%) was sprayed into a chamber. In the first experiment, the VOC level decreased, as relative humidity decreased. In the second experiment, VOC levels increased as humidity increased.

(4) Particle agglomeration

Higher humidity cause particle agglomeration through moisture adsorption (Green, 1984). These larger particles have less chance of penetrating the lower respiratory tract and thus have less chance of causing infection (refer section on Respirable Suspended Particles).

Summary Effects of Low Relative Humidity

(1) Dryness and irritation

Low indoor RH (below 40%) has been linked to an increased incidence of upper respiratory infections due to higher viral survival rates (Andersen et al., 1983). The same researchers also reported complaints of dryness or irritation of the skin, eyes, nose and throat. These seem to manifest as bleeding noses, and split finger nails. There is some evidence that very low relative humidity (ie. below 20%) causes both eye (Berglund et al., 1986) and skin irritation (Engen, 1982).

It was widely believed that dry air lead to mucostasis in the nasal cavity. The hypothesis was that during nasal respiration, the inspired air is humidified as it passes through the nasal cavity by moisture supplied from mucous. Drier air, required a greater volume of evaporated water from nasal mucus. Thus when dry air is inspired it decreases the mucus transport rate leading to mucostasis. As a result, the bodies first line of defence is degraded leading to an increased probability of attack through the respiratory route. A controlled experiment by Andersen et al., (1972), made it clear that a high relative humidity does not improve mucociliary clearance nor does low ambient humidity impair it. They concluded that the *'nasal humidification system was remarkably efficient'*. This finding was subsequently validated by Ahlström et al., (1986).

In another study, the prevalence of BRI symptoms were higher in a humidified building as opposed to one which did not have humidification (Berglund et al., 1986).

Arundel et al., (1988) found no correlation between reported symptoms and the relative humidity level. However they concede that the results do not disprove the possibility that a low RH causes symptoms and point out that the ability of a questionnaire based survey to detect an association was limited.

(2) Electrostatic shocks

People walking about in a room are constantly charging themselves up electrostatically through shoe contact with the carpet. Simultaneously this charge is leaking to earth through the shoes and carpet (depending on the material of both). Normally the electrical resistance of shoes and carpet are sufficiently low to prevent the electrostatic charge building up to a voltage high enough to create a shock (Brundrett, 1977).

The key environmental factors influencing the generation of electrostatic electricity often interact and cannot be taken in isolation. The major factors are relative humidity, carpet type, shoe material and walking speed. The key environmental factor, however, has been identified as the relative humidity of the air in the neighbourhood of the carpet. This appears to be independent of temperature. Higher moisture content decreases the electrical resistivity (Sereda et al., 1964; Hearle et al., 1960; Ramer et al., 1968).

The critical humidity needed to avoid electrostatic shocks varies with carpet material. Bulgin (1953), calculated that 65% RH was the minimum for cotton or rayon to be safe in a potentially explosive situation. Ellis (1974), found that people walking on carpets with leather soled shoes in an atmosphere of 50% RH did generate voltage, but it was insufficient to create a shock. At 40% RH, very sensitive people might experience a slight sensation when

walking on nylon carpet which could be discharged through contact with a metal object. At 30% RH, each of three carpets tested, namely wool, nylon and acrylic, could create shocks in the order of 3.0-3.6kV. This confirmed work by Martin et al., (1971) and Cusack (1972).

Wilson (1972) investigated the electrostatic properties of twenty-seven different modern carpets at two relative humidities, 15 & 40%, and with subjects wearing leather shoes and plimsolls. This study showed wide differences between carpets. At 40% RH a strolling person could reach 2kV in a hundred paces on seven of the twenty-seven carpets. Five of the seven problem carpets were wool, one was nylon and one acrylic. Even at 15% RH only nineteen of the carpets generated the threshold voltage of 2kV in the person during the walking trial.

According to Brundrett (1977), there was only a small difference between artificial fibres and natural fibres. However, he suggested that relative humidities of around 50% were required if the carpet fibre was nylon and 40% for most other carpets.

The exception to the rule relates to underfloor heating. In this case the carpet temperature is elevated above the room ambient temperature. This, of course, dries out the carpet reducing its resistivity. The equivalent humidity to avoid electrostatic voltage then depends on the temperature rise of the carpet. A typical elevation of 5°C in the carpet temperature requires a relative humidity in the room of no less than 55% RH to avoid shocks.

It should be noted that a carpets propensity to generate electrostatic discharges can be curtailed by the application of a humectant to the carpet. Although, humectants have not been shown to be particularly durable with an average life expectancy of five years or less.

KEYPOINTS: The recommended humidity range for office buildings is between 40% - 60% RH. People are poor judges of ambient moisture levels and except at extreme levels, RH is not a subjective 'comfort' issue except at extreme levels. However, humidity appears to be pivotal factor in the way that it interacts with other potential stressors. High levels of relative humidity promote the growth and numbers of dust mites, micro-organisms, and accelerate the release of water soluble organic compounds such as aldehydes. On the other hand it reduces airborne RSPs in that it promotes particle agglomeration. Low relative humidity causes dryness of the mucous membranes leading to irritation of the eyes, and respiratory tract. It also promotes the incidence of electrostatic shock. The issue is one of balance and it could be argued, with some validity, that current upper levels are too high and that RH levels should be kept below 50%.

LIGHT

IMPLICATION OF LIGHT IN BRI SYMPTOMS

Collins et al., (1989) reported an increased level of eye irritation in buildings where the lighting environment was considered as being too bright or too dim.

Robertson et al., (1986), considered glare as being a potential problem factor but rejected the hypothesis. However, a later study by Wallace et al., (1991) found glare to be one of the main environmental variables to be correlated with the symptoms of headache, eye irritation, fatigue and concentration difficulties.

A study by Robertson et al., (1989) of two adjacent buildings, one air-conditioned and one naturally ventilated, indicated a general dislike by workers for fluorescent lighting. There was a greater incidence of headache in the air conditioned building. The symptomatic workers in this study tended to have darker work positions and they had less control over the illuminance level. The windows in the air-conditioned building were smaller than those in the naturally ventilated building and were also tinted. The amount of daylight in the building was reduced. This study raises more questions than answers; were the headaches caused by poor illuminance or poor natural light or the psychological factor, lack of personal control?

Lack of access to natural light has been considered in a number of studies as an exacerbating factor. Raw (1992), reports that the greatest number of symptoms of concern are often found in buildings which have large interior spaces and have relatively little daylight. A number of studies in windowless buildings have found an excess of symptoms amongst occupants. Many of the symptoms reported are similar to those appearing under conditions of perceptual deprivation. Wilkins et al., (1989) report that the frequency of headaches decrease with the height of the office above the ground and suggest that the mechanism involved is increased access to natural light.

The flicker rate of fluorescent lights with conventional ballasts has been promoted as being a major factor in headaches and eyestrain. Wilkins et al., (1984) proposed a link based on neural inhibitory mechanisms. In an interventional study by Wilkins et al., (1989), the average incidence of headaches and eye strain was more than halved under lighting with high frequency ballasts.

According to Raw (1992), there is no particularly strong evidence that lighting is a major cause of BRI type symptoms but it is probably one of the lesser contributory factors.

PHYSIOLOGICAL ROLE OF LIGHT

According to an article by Gilkes (1979), the importance of light to human health cannot be underestimated. In the century between 1850 and 1950, inadequate light in dwellings was frequently reported by medical health officers in the United Kingdom as being a major cause of poor health.

Light has three effects on human beings:

- It influences 'body rhythms' such as sleep patterns, ovulation and hormone secretion.
- It affects performance, alertness and mood.
- It exerts a direct physiological effect on the skin including the synthesis of Vitamin D and certain wavelengths may cause epidermal damage at the cellular level.

Lighting in the Office Environment

Craig (1981) reports that, poor lighting in offices is a common cause of eyestrain, which in turn causes stress and can lead to accidents. She makes a plausible ergonomic linkage between poor lighting and muscular-skeletal pain, suggesting that a common reaction to inadequate lighting is for the subject to move nearer to the work surface causing postural misalignment. This in turn may lead to backache, headache and muscular fatigue.

She refers to the results of a survey undertaken by the Alfred Marks Bureau in 1975, in the United Kingdom. It was reported that 29% of office workers surveyed found the lighting inadequate for their needs. Over 10% reported frequent eye fatigue and 40% of the secretaries and shorthand typists in the survey complained of eyestrain due to that fact that they do *'close work'* in inadequate lighting.

The quality of office lighting has been assessed in a large number of studies (Hopkinson, 1963; CIBS, 1984; Cuttle et al., 1967; Tregenza et al., 1974; Saunders, 1969; Van Ireland, 1967). Recommended lighting levels in office buildings have steadily increased over the years. Currently, CIBS (1984), recommended illuminance levels for office buildings is 500 lux.

In various studies Kraemer et al., (1977) found little correlation between desk top illuminance and observer's assessment of comfort although they were exposed to a wide range between 400 and 1000 lux. Van Ireland (1967), found that a surprisingly large number of people were satisfied with low desk-top lighting levels. Saunders (1969) found that most observers who had been allowed time to adapt rated desks with 200 lux or less as poor.

Photochemical Smog

Sterling (1983), hypothesised that fluorescent lighting contributed to the symptoms associated with the 'Sick Building Syndrome' by creating a photochemical smog. This is supposed to be caused by the catalytic action of ultraviolet radiation on the organic pollutants in the indoor environment.

While this is an interesting theory, little has been offered by way of evidence to support the hypothesis.

Possible Links with Epidermal Complaints

A number of researchers have expressed concern about fluorescent lighting. Window glass does not transmit UVb light, but fluorescent lighting without styrene diffusers can produce

wavelengths down to 250nm, the range implicated in the production of potentially carcinogenic cell mutations. Beral (1981), observed a doubling of risk of *malignant melanoma* amongst women exposed to fluorescent light at work. There is considerable disagreement about the significance of Beral's findings. Rigel et al., (1983), suggest that the increased levels of *malignant melanoma* observed by Beral was as a result of intermittent sunbathing, which would seem to explain the higher prevalence of the condition amongst the upper socio-economic groups in Britain.

Griffiths et al., (1985) reports a number of instances of *light-induced dermatitis* caused by fluorescent tubes. Goldstein et al., (1967), report that a side effect of working under fluorescent light combined with ingestion of certain depressants, antibiotics or diuretics may be a spontaneous eruption of the skin.

PHOTOSENSITIVITY

It has long been recognised that flicker is a cause of epileptic fits (Cobb, 1947). The exact prevalence of photosensitive epilepsy is impossible to establish. According to Mant et al., (1986) malfunctioning fluorescent light fittings are a recognised as a precipitant of epileptic fits.

Correctly functioning fluorescent tubes with standard ballasts flicker at twice mains frequency of 100 cycles per second (100Hz). Research by Wilkins et al., (1984; 1988; 1989) has shown that the flicker, although not perceived, can contribute to headaches. They have shown that this can be ameliorated by the use of high frequency ballasts which convert the flicker to a much higher frequency (ie. between 25kHz & 40 kHz).

KEYPOINTS: Lighting has been established as one of the minor contributors to the symptoms of interest. Several different mechanisms have been implicated; inadequate light leading to eye strain; glare; conditioning of neural inhibitory mechanisms due to imperceptible flicker. Lack of access to natural light has been cited as a factor, the mechanism may be psychological or physiological. The psychological factor may be one of sensory deprivation, the possible physiological factors may be an upsetting of the normal bio-rhythms or reduced levels of hormone secretion. Photosensitivity has been recognised as a precipitant of epileptic fits, although this is not a symptom of interest in this study it seems to support Wilkins and other's hypothesis regarding the effect of flicker. The interesting hypothesis of an interaction of fluorescent light with chemical pollution causing a photochemical smog remains unproven at this point.

NOISE

THE IMPLICATION OF SOUND IN BRI SYMPTOMS

Noise can cause headaches and fatigue and affect concentration, by a combination of its intensity, frequency, location, predictability and acceptability. Some low intensity noise can contain some pure tones which may cause irritation (Molina et al., 1989).

People vary in their response to noise with some being more sensitive than others. It is highly probable that those noise sensitive people may react with irritation or annoyance to noise, manifesting as a set of physical symptoms.

Raw (1992), reports that there is no direct evidence on the role of noise in the symptoms of interest. Many workplaces are as 'noisy' as 'sick' buildings without displaying excess symptoms.

Noise, on the other hand, may contribute to overall stress levels in workers and exacerbate existing complaints. Noise levels may partly explain why BRI symptoms are more prevalent in open plan offices with 10 or more work stations.

It would seem that, of the evidence in the available literature, sound is an exacerbating factor rather than a causative factor.

ACOUSTICAL ENVIRONMENT

The acoustical environment within a building is the result of the noise entering the space from outdoors, from adjacent areas, the engineering services, as well as the noise generated within the space by people and equipment. Whether or not the resulting acoustical environment is disturbing or annoying to an occupant depends on numerous factors, which may include both physiological and psychological factors that vary from person to person.

In general, annoyance depends on the sound quality and its information content, on its source and duration, on its relationship to expectations, the need for communication, and on the activity of the individual, whether working, concentrating or relaxing (Potter, 1988). For example, a level and intensity of sound totally acceptable to an individual in a work setting would be most distressing to the same individual at rest. In the office setting, increase above the ambient noise level is important. For example, a telephone ringing in a silent library is perceived to be more disturbing than a telephone ringing in a crowded, open plan office.

THE EFFECTS OF NOISE ON THE HUMAN ORGANISM

One of the more obvious effects of noise is interference with speech communication. Such interference involves a masking process. As a result of noise, a person may hear only a few or none of the speech sounds necessary for satisfactory intelligibility.

In a set of experiments conducted in the United States in 1968 reported by (Thissen et al., 1968), sleeping subjects were exposed (several times a night) to a recording of truck noise played at a selected level. Most of the subjects reacted with elevated pulse rates and 'panic' type symptoms. There was an appreciable reduction in their ability to concentrate following these episodes.

The most obvious physiological effect of noise is the cumulative loss of hearing activity after long-term exposure to a high-level noise field. Community noise levels rarely reach the, so called, threshold of pain. However there is some scientific concern that exposure to urban noise levels may contribute to the loss of *high-frequency acuity* - a loss that was formerly considered to be part of the natural aging process. It has been reported by Berland;1970, that primitive African tribes who live in a background noise level of 30 dB do not experience such loss of high-frequency acuity. However, when such tribes move to a larger and noisier city their hearing abilities become impaired and, unexpectedly, the incidence of *heart disease* increases.

There has been some concern about other physiological effects associated with noise such as dilation of the pupils, increased flow of saliva and gastric juices (Welsh, 1970). However, there is little quantitative information available about the importance of such effects.

Craig (1981), claims that excessive noise affects sense of touch, clarity of vision, balance and co-ordination, but does not offer any evidence to support this assertion. She goes on to say that it contributes to fatigue, loss of sleep, headaches and irritability. Much of this seems to be supported by anecdote but little by way of empirical observation.

However, one group of researchers have claimed that even *low level noise can be stressful.* Stellman (1977) claimed that office workers exposed to 'moderate' noise have an increased incidence of circulatory, digestive, neurological and psychiatric problems.

Atherley et al., (1970), studied university staff with sedentary occupations and found that after three days of systematic exposure to typewriter noise and aircraft noise, the subjects complained of tiredness and irritability. The researchers claimed that the observed effect was a *'type of mild anxiety-depression syndrome'.*

Noise and the Hypothalamus

The hypothalamus is an area located at the base of the brain. Kubzansky et al., (1961) noted that stimulation of specific hypothalamic areas has been shown to influence the optic and auditory systems.

Duffy (1962) notes that stimulation of the hypothalamus in man has been shown to produce effects on blood pressure, pulse rate, respiration and other visceral functions.

Stellar (1954) indicated that sensory and cortical stimulation can exert excitatory and inhibitory influences on the hypothalamus. Thus, it seems reasonable to conclude that altered sensory input (including auditory) may affect hypothalamic mechanisms with consequent effects on the behavioral functions under its control.

Schneider (1988), claims that noise affects the entire organism. Sound is not only an acoustic perception but affects the entire level of consciousness. He claims that it reaches the vegetative nervous centres via the hypothalamus which directs the function of the inner organs and the blood circulation. Noise also stimulates the hypophysis, a gland whose secretions control (to an extent) blood pressure. While Schneider does not forward any proof for these assertions, Schultz (1965), gives some credence to the hypothesis.

Theory of Optimal Sensory Input

Craig (1981), indicates that open plan offices are both noisy and pose a security problem - other peoples conversations may be overheard. To combat this, many firms introduced background music, or 'white noise'. While this additional sound source may do little to increase the decibel level within the occupied space, it introduces yet another sensory input.

The concept of a level of stimulation appropriate to the functioning organism has been considered by several writers. For example; the degree of energy mobilization (Cannon, 1929; Duffy, 1941, 1951), the degree of arousal (Freeman, 1948), level of arousal (Hebb, 1955; Bindra, 1959), level of activation (Schlosberg, 1954). All refer to a dimension representing the energy level or excitation level of the organism; an arousal or sensory continuum with deep sleep or anaesthesia representing the lower end and epileptic seizure, extreme anger or fear, or panic behaviour representing the upper end of extreme sensory stimulation.

A sleeping organism represents the lower level of activation. In sleep, the cortex and sympathetic division of the autonomic nervous system are inactive and the muscles are relaxed.

Bindra (1959) reviews the autonomic, somatic, and neural changes that take place within the organism under different levels of stimulation and notes the existence of marked individuals in both base level and degree of reactivity of these physiological functions.

Bindra discusses three main points of difference between high and low states of arousal. First, the involuntary bodily processes function differently in the two states. Second, voluntary musculature activity is higher in high arousal states.

Bindra (1959), Hebb (1955), Leuba (1955), McReynolds (1956), Berlyne (1960), all have suggested that between the two extremes on the sensory continuum lies an area of *optimal level of stimulation* and consequent arousal which results in efficient response and learning capabilities on the part of the organism.

At too high an arousal level, the intense sensory bombardment may interfere with functions involved in the cue or guiding function (Hebb, 1955), perhaps due to 'blocking'. Thus intense stimulation may interfere with responses already in the organisms repertoire and may prevent the acquisition of new responses to adapt to the situation. Duffy (1962) reviews a large number of studies indicating a wide variety of performance tasks which are handicapped by a very high level of activation.

Schneider (1988), suggests that an overload of auditory input from multiple sources may induce psychosomatic effects which include:

- irregular breathing
- ulcers
- stenosis of blood vessels
- stress reaction as a result of continuous adrenalin release.
- reactions of glands with inner and outer secretion
- changing of electrical skin resistance.

KEYPOINTS: Whether noise is disturbing or annoying to an occupant depends on numerous elements which include physiological and psychological factors and vary from person to person. Excessive noise interferes with speech and communication but office noise rarely reaches the danger level of hearing loss as may be encountered in an occupational setting. Constant low level noise can be stressful and exposed individuals show an increased incidence of circulatory, digestive, neurological and psychological problems. Sound may stimulate the hypothalamus directly and thus stimulate the autonomic system. Given an excess of auditory input from multiple sources, it may interfere with the individuals ability to adapt, leading to fatigue, lethargy and headache.

VIBRATION

THE IMPLICATION OF INFRA-SOUND IN BRI SYMPTOMS

Field (1987), failed to find adverse effects of physiological and psychological effects caused by infra-sound (vibration) in buildings.

Ising et al., (1982), report that infrasound can cause dizziness and nausea. This is thought to occur at levels above 120 dB. Such effects are thought not to occur below 100 dB but psychological effects can occur at all ranges down to the level of perceptibility. These psychological effects include irritation and lassitude. Measured levels inside buildings have been considerably below the 'critical' physiological threshold levels.

INFRA-SOUND

This is the twin of acoustic pollution. From a physical point of view frequencies below 16Hz are vibrations (infra sound) and will be noticed by the entire human organism as tremor or shaking. According to Potter (1988) the human body is a very sensitive detector of vibration. Under certain circumstances a standing person can detect amplitudes as small as 1 micron, whilst amplitudes of 0.05 micron can be detected by the fingertips. The basic data concerning 'whole body' sensitivity to vibration is provided by the Reiher-Meister scale.

Knowledge of the physiological effects of infra-sound is not particularly extensive. Zoological observation suggests that animals, use vibration as a first level warning of approaching danger. This gives rise to the hypothesis that this 'genetic response' may trigger in humans the first level of heightened alertness (ie. the first level of the fight-flight response, to use Cannon's term). This, typically, gives rise to elevated blood pressure and a general feeling of discomfort. These, false alarms, may lead to the type of sensory overload.

Infra-sound measurements made in buildings by Kroeling (1987; 1988) were in the frequency range 10 to 100 Hz. Sounds in that range are not expected to have measurable physiological effects.

KEYPOINTS: Vibration is known as infrasound. The human body is a more efficient detector of low levels of infrasound than it is of sound in the auditory range. Infrasound seems to cause 'mini panics' and may result in the same symptoms as constant low level noise. Levels of infrasound measured in office buildings have been less than the 'critical' physiological level.

NEGATIVE IONISATION

IMPLICATION OF IONS IN BRI SYMPTOMS

There have been a number of published claims that high levels of positive ions leads to discomfort, tiredness, headache and a number of other symptoms of interest. Supporters of ionization theory claim that by raising the proportion of negative ions, BRI symptoms effectively disappear (Ball, 1982; Hedge, 1987).

However, the results from a number of investigations are inconclusive and contradictory. The claim that negative ion generators have a beneficial effect on BRI symptoms is largely based on work by Hawkins (1982).

A follow up study Hawkins et al., (1984) was unable to repeat his original findings. An investigation by Hedge et al., (1987) indicated that negative ions had no effect on mood and performance. Robertson et al., (1985) concluded that there was no evidence that ion concentrations differ between buildings which are symptomatic and those which are asymptomatic.

On the other hand, Suliman et al., (1974) successfully used ionisation to treat weather sensitive people for what has been called the *'serotonin release syndrome'*. It is clear that ionisation has it's uses and benefits, but whether it is of any benefit in the relief of BRI type symptoms remains unproven.

AIR IONS

Air ions are electrically charged forms of air gases. Although there is some dispute about the exact nature of air ions, the consensus of opinion is that the negative ion (carrying a negative charge) is predominantly CO_3^- while the positive ion is N^+ (Rosenthal et al., 1980). Low concentrations of negative ions are said to be associated with a wide variety of illnesses and complaints, while high concentrations are claimed to promote a feeling of alertness and wellbeing.

Nevertheless, the whole ionisation issue remains most contentious. The CIBSE Guide (1978) states that: *"Present evidence on ionisation is inconclusive and no design criteria can be evaluated"*. Jones (1985), also supports this contra approach: *"In the past it has been thought that air containing molecules with a high negative ion content exhibited indefinable qualities of freshness that some subjects found desirable, but subsequent work has failed to support this. Current, reputable, scientific evidence indicates that the ion content of air has no measurable effect on human beings in buildings. If any influence is produced by the ions present it must be exceedingly subtle."*

Nevertheless supporters of ionization theory hold equally strong views. With equal certainty Krueger (1972), stated that: *"There is no doubt that positive air ionisation - in contradistinction to the negative one - produces highly unpleasant reactions due to serotonin release"*.

Schneider (1989) makes a number of somewhat sweeping claims as to the benefits of negative ionisation, but, unfortunately, offers little in the way of proof.

(a) A perquisite for the maintenance of life is that, of all conditions, the oxygen in the air is charged. The presence of negative ions is necessary to enable the body to metabolize the oxygen.
(b) Predominantly positive air ionization leads to headaches, tiredness, lowered blood pressure and breathing difficulties. Breathing is made more difficult when the inhaled air has the same positive charge as that in the lungs.
(c) Negative ions have a healing effect, especially with high blood pressure, sinus disturbances, asthma, migraine and exhaustion.
(d) Ions regulate the formation of the neuro-hormone Serotonin. Dominantly positive ions cause an increase in the Serotonin secretion which leads to uneasiness, irritability, sleep disturbances, pains, cramps and states of anxiety. Reduced secretion of the hormone with negative ions beneficially influences the nerves, glands and breathing activities as well as cell growth.
(e) The movement of the cilia in the respiratory passages (for filtering air) is reduced around 25% with predominantly positive ions, and this movement is increased around 25% with a surplus of negative ions.
(f) Negative ionization improves the blood contents, stabilizes breathing, stimulates the metabolism and growth, helps motor skills and endurance. With animals in negatively ionized air, the aging process is strongly delayed.
(g) Extended experiments have shown that positive ions in an oversupply, are felt by the majority of people to be uncomfortable. These ions often call forth head and throat pain, dizziness, increased irritability, depression and anxiety conditions, lessen the immunity powers of the body, etc. In reverse, the small negative ions, when present in dominating numbers, bring about increased desire to work and the ability to perform; they increase the resistance of the body, especially against cold illnesses and work favourably on bronchial asthma, whooping cough, high blood pressure and brown lung.
(h) Negative ions also have a positive influence with allergic illnesses.
(i) In a certain geographic area under examination, a very close correlation was shown between the yearly course of atmospheric strength and recorded medical consultations.

Typical Ion Concentrations

The behaviour of ions is well established scientifically. Positive and negative ions are formed continuously in the atmosphere by loss or gain of electrons by neutrally charged molecules. This occurs under the influence of natural phenomena such as sunlight, cosmic rays, lightning, falling water, flames and radioactive elements in the soil. The ions recombine to form neutral particles or cluster around water droplets or particulate matter (RSPs). At a particular site, ion concentrations vary with the time of day and the weather (Hawkins, 1982). Typical ion concentrations for different types of outdoor environment are given in the following table.

TABLE 10 - OUTDOOR ION CONCENTRATIONS

OUTDOOR ENVIRONMENT	Ion concentration, per cm^3	
	Positive	Negative
Coastal	2000	1800
Clean country air	1200	1000
Lightly polluted urban atmosphere	800	700
Typical CBD atmosphere	500	300

The number of ions present in suburban and urban areas is lower than in the countryside because of the higher incidence of *condensation nuclei*. Negative ions have a greater tendency to bind to condensation nuclei (RSPs) than positive ions because they are smaller and more mobile than the latter. Thus the ratio of negative to positive ions reduces in polluted atmospheres.

The concentration of ions within buildings is even lower than outdoors. Typical concentrations are given in the following table:

TABLE 11 - INDOOR ION CONCENTRATIONS

TYPE OF BUILDING	Ion concentration, per cm^3	
	Positive	Negative
Rural house; no air conditioning	1000	800
Rural office; air conditioning	100	100
City office; air conditioning	100	50

There are a number of reasons for the lower concentration of ions within buildings;

(1) In steel framed buildings, charged particles readily find a route to earth.
(2) In buildings which have mechanical ventilation or air conditioning systems the earthed duct rapidly removes negative ions. This has been graphically demonstrated using an ion counter. With an ion generator placed upstream of a short length duct, it was observed that 97% of ions were removed within 13 centimetres of the entrance (David, 1982).
(3) Artificial fibres have a high electrical resistivity and are associated with electrostatic charges, especially when relative humidity is lower than 40% (Brundrett, 1977). The small, mobile negative ions are attracted to the positively charged surfaces where they are neutralised.
(4) VDU screens have a high positive charge (between 6 to 30 kV). Negative ions are attracted to the screen from the surrounding area, particularly the breathing zone of the operator.

(5) Tobacco smoke contains a large number of microscopic particles between 0.01 and 1 micron, which act as condensation nuclei for negative ions. The smoke particles impinge on the room surfaces removing ions from the air.

Health Claims Associated with Negative Ions

The discovery of air ions in 1900 by Elster and Geital appears to have been followed almost immediately by claims for their biological influence (Davis, 1963). Although the reports of biological effects appeared constantly in literature since 1902, the work of Yaglou and Benjamin at Harvard probably did most to put the subject on a respectable footing (Yaglou et al., 1933; Yaglou & Benjamin, 1934).

Their hypothesis that air ions could influence behaviour and the state of human wellbeing began to loose credibility in the intervening decades. Hawkins (1981), believed that this was due to the over enthusiastic and unjustified claims made by manufacturers hoping to exploit air ionisation, and partly due to poorly controlled, unrepeatable and statistically weak experiments conducted by many researchers. By the mid-1950s the American Food and Drug Administration prohibited the sale of ionizers for medical application and research into the subject waned. One notable exception was the work of Kornbleuh in Philadelphia who continued to study air ionization throughout the 1950s and, despite considerable opposition at the time, maintained that air ions affected the electroencephalogram and were of clinical value in the treatment of headache, respiratory complaints and skin burns.

Modern Ionization Research

The modern revival of interest in air ions comes from the work of Suliman in Israel and Krueger in the United States.

Suliman et al., (1970), observed a medical condition suspected as being associated with *weather sensitivity*. Weather sensitive people report certain unpleasant physiological reactions and become irritable and anxious before the arrival of thunderstorms or other meterological phenomena such as the Sharav (Khamsin), a hot dry wind in the middle east. It is reported that such people may be subject to heart oppression, palpitations, dyspnoea and migraine. Others complain of asomnia, tension, oedemata, rheumatic pain, scar aching, precordial pain, flushes with sweat or chills, vasomotor rhinitis, hyperistalsis and polakisuria.

Suliman et al., (1975) conducted intensive clinical and biochemical studies of *Sharav* patients and were able to show that these symptoms were provoked by the release of *serotonin*. They designated the cluster of symptoms as the *"Serotonin Irritation Syndrome"*. The complaints were successfully treated by inhalation of air containing large numbers of negative ions or the administration of serotonin-blocking drugs.

The results of animal experiments by Kreuger et al., (1974), supported Sulman's findings in humans that high levels of positive ions increase the body tissue levels of a number of hormones (adrenaline, noradrenaline, thyroxine and 5-hydroxtryptamine).

However, other research results are ambiguous.

Hedge et al., (1982), critically reviewed much of the previous research. They indicated that a number of studies gave inconclusive or null results or contradicted other studies. They claimed that comparison between experiments is often difficult because of differences in experimental design, some experiments lacked a 'control' group and in other studies, statistical treatment of results was plainly wrong. These criticisms particularly applied to studies of the physiological effects of ions.

Fishman (1981), studied the effect of ionized air treatment on patients with bronchial asthma. No statistically significant evidence of patient response was found. The author concluded that it was unlikely that negative ions would be of significant benefit to the *majority* of patients with asthma.

Sharpe (1983), reported the effects of enhanced air ionization in an urban office environment. The trials took place in the typing pool of a modern, open plan air conditioned office building. Results indicated that in 1982 there was a reduction of 35% in casual sickness in the ionized area compared with the same period for 1981, while in the non-ionized area there was a reduction of only 11%. The figures for 1983 compared to 1981 showed an increase of casual sickness of 2% in the ionized area and 23% in the non-ionized area. In both years therefore, there was about 20% less casual sickness in the ionized area compared to the control areas, and Sharp concluded that it was reasonable to attribute this improvement to the ionizer.

Hawkins studied staff response to ionization in a variety of modern air conditioned locations. Ionisers were switched on and off on a weekly basis according to a random schedule in what Hawkins calls a *double-blind* (ie.neither the subjects or investigators analyzing the results knew when the ionizers were in operation). The occupants were asked to complete a subjective questionnaire of aspects of thermal comfort and health. Hawkins found a statistically significant reduction in the incidence of headache, nausea and dizziness when the ionizers were working (Hawkins, 1979; 1981; 1982). However in a more recent experiment he found that *lethargy* was the only complaint of those measured that improved significantly with an increase in negative ion concentration. He wrote that other results of his earlier work could not be repeated. He concluded that the prevalence of ill health reported in some buildings is caused by a variety of factors and that ionization was unlikely to be the total answer (Hawkins et al., 1984).

In a change of direction, Hawkins is investigating the extent to which negative air ionization clears room air of suspended matter. If ionizers can significantly reduce the amount of dust and pollen in a room, this may explain the reduced incidence of asthma and cross-infection of colds.

Health Dangers Associated with Ionization

A paper by Reinet (1979), warns of possible health risks associated with corona ionizers. His research concluded that these ionizers were responsible for producing high concentrations of large air ions. These are produced in an intense electrical field by the settlement of small ions on nuclei suspended in the air. The resulting electrically charged dust settles down in the lungs to a greater extent than electrically neutral particles.

In addition, corona ionizers produce a considerable amount of toxic gases, particularly ozone and nitrogenous compounds. Therefore, he concludes that their permanent use in living rooms is undesirable.

Sulman et al., (1978), contradict these findings. In more modern ionizers they claim that production of nitrous oxides and ozone is kept to a minimum and could not be detected by a *draeger tube* at more than 10 centimetres from the ionizing spikes. In addition, none of the patients treated by continuous negative air ionization suffered any adverse effects. Earlier observations of health effects by Rivolier et al., (1975), found no adverse affects amongst subjects treated with continuous ionization over a period.

Nevertheless, it should not be overlooked that Reinet's caution may point to possible long-term chronic effects rather than acute effects as looked for in the Suliman & Rivolier experiments.

CONCLUSION

If a conclusion can be drawn, it is one of confusion. While certain effects of negative ions have been demonstrated and reproduced in a number of studies, such as the biocide effect on micro-organisms or physiological changes in weather sensitive people, other research has been inconclusive if not downright contradictory.

The quality of the indoor environment is dependent on many factors which interact in a complex way, making it difficult to identify causal variables. This is a possible explanation as to why some studies found that negative air ionization was of benefit while others reported null results.

KEYPOINTS: The effects of ions on weather-sensitive people is well established scientifically. Negative ions may also have some biocidal effects. Misleading claims by some ion equipment manufacturers in the 1950-60s, lead to the US FDA (Food and Drug Administration) ordering the withdrawal of some nine brands from the market. The backlash from the academic community effectively consigned ionisation research to the scientific wilderness for over a decade. It may take more time yet before it is treated as a serious subject. There may be some potential chronic risk associated with the use of ionizers depending on the nature of the RSPs present in the occupied space. An associated benefit with ionisers is that they seem to accelerate the precipitation of particles out of the air, thus acting as a quasi or supplementary air cleaner.The status-quo supported by ASHRAE and CIBSE is that the benefits associated with ionization are insufficiently proven to warrant the expense of fitting the devices to buildings. The situation warrants continued investigation.

PSYCHOLOGICAL FACTORS

IMPLICATION OF PSYCHOLOGICAL FACTORS IN BRI SYMPTOMS

The large body of evidence suggests that psychological factors play a central role in symptom reporting. It is often difficult to tell whether the causative factor was physiological or psychological, as both are often present at the same time.

The highest level of symptom reporting comes from within air conditioned buildings and a common complaint from building occupants is that they lack control over their environment. For example, Robertson et al., (1989) indicated that symptomatic staff in a building complained that they had no control over the lighting. Building occupants expressing a feeling of helplessness or an inability to control their environment is a common theme throughout the literature.

Symptom reporting appears to be consistent amongst groups of people. Hedge et al., (1986) compared two groups of people within the same building with no obvious differences in their environment. The group of female clerical workers reporting low job satisfaction had a far higher incidence of reporting than the, largely, male professional group reporting high job satisfaction. This seems to support the hypothesis that a proportion, at least, of symptoms attributed to buildings are other than building related.

The most consistent characteristic influencing BRI type symptom reporting is gender, with females reporting the most symptoms (Wilson et al., 1987; Skov et al., 1987). This is not unique to BRI symptoms and is consistent in most health surveys (Pennebaker, 1982).

Guidotti et al., (1987) reported an outbreak of mass psychogenic illness amongst telephone exchange operators when 81 members of staff were taken ill. Air sampling results were negative and the outbreak was eventually attributed to mass hysteria manifesting as physical symptoms. Smith et al., (1978) describes three outbreaks of mass psychogenic illness in industrial plants. The symptoms were similar to BRI type symptoms. The outbreaks were found to affect workers in a predominantly female workforce who were under some physical or psychological stress.

According to Raw (1992), if BRI is not primarily psychological in origin, it has psychological consequences that may be magnified due to perceptions of the environment, the response of other building occupants and suggestion due to hearing about BRI. Psychological factors are generally more likely to affect the reporting of symptoms rather than the cause symptoms themselves. *In short, the most simple model is that the environment causes symptoms and the reporting of those symptoms is dependent on psychological factors.*

LINK BETWEEN EMOTIONAL STATE AND PHYSICAL SYMPTOMS

A number of symptoms associated with BRI have been described as 'classic psychosomatic symptoms'. According to Pennebaker (1982), perceptions that emotions and symptoms are closely intertwined indicates that there may be a physiological link. Additional credence must be given to this idea based on the large number of theories and findings indicating a link

between emotion and measurable physiological activity (Ax, 1953; Libby et al., 1973; Schachter, 1957; Wolf et al., 1943). As the result of an experiment with 177 students, Pennebaker (1982) reported strong correlations between specific emotions and symptoms as follows:

TABLE 12 - PHYSICAL SYMPTOMS RELATED TO SPECIFIC EMOTIONS

PHYSICAL SYMPTOM	EMOTION					
	Happy	Tense	Angry	Jealous	Sad	Guilty
Headache	*	*			*	*
Watering Eyes					*	*
Racing Heart	*	*			*	*
Congested Nose			*			
Tense Muscles	*	*	*	*	*	*
Flushed Face		*	*	*		*
Short Breath		*				*
Cold Hands						*
Warm Hands		*				
Dizziness						
Lump in Throat						*
Upset Stomach	*	*	*		*	*

PHYSIOLOGICAL, BEHAVIORAL & PSYCHOLOGICAL EFFECTS OF STRESS

The physiological effects of stress, particularly job related stress, is central to the investigation of BRI type complaints. The reason for this is that they, probably, most often confuse the issue.

When investigating complaints of a 'sick building', in most instances, symptoms attributable to job related stress need to be eliminated before the physical building structure can be implicated.

The stressful psychosocial factors in the working environment may be many and varied. They include physical aspects, some aspects of the organisation and system of work, and the quality of human relations in the enterprise. According to an ILO/WHO report (1984), all of these factors interact and may affect the psychological climate in the enterprise and the physical and mental health of workers. Psychosocial stressors at work and in working conditions are frequently long standing, whether they occur continuously or from time to time. In spite of the many ways in which people are equipped to cope with these situations, the demands put on them may exceed their resources, making them unable to cope, or, in the long run, causing new problems.

The psychosocial environment in which people work can have a major bearing on their health.

Levi (1972) defines psychosocial stimuli as those which originate in social relations. A report by the joint ILO/WHO Committee on Occupational Health (1984) defines psychosocial factors at work as:

> *"... interactions between and among work environment, job content, organisational conditions and workers' capacities, needs, culture, personal extra-job considerations that may, through perceptions and experience, influence health, work performance and job satisfaction."*

According to the same report, a vast amount of literature has demonstrated that psychosocial factors at work contribute to a wide range of workers' health disorders. A large amount of evidence has been accumulated on the relationship between non-specific psychological, behavioral and somatic syndromes and stressful or unrewarding working conditions. Individuals interact with their working conditions in a manner which is determined by working conditions and by human capacities and needs. Crucial job factors in this interaction include task, the physical and social work environment, managerial practices and employment conditions. In the working environment, a number of negative, potentially health related psychosocial factors have been identified in a number of studies. These include the under utilisation of abilities, work overload, lack of control, role conflict, inequity of pay, lack of job security, problems in relationships at work, shift work and physical danger.

(a) *Physical Work Environment* - In surveys concerning the working conditions of various occupational groups, workers' complaints often emphasise noise and temperature. Additionally, vibration and chemical exposure are mentioned (Cox, 1980).

(b) *Intrinsic Job Factors* - Work overload is characterised as being either quantitative (ie. too much to do) or qualitative (ie. work being too difficult). Various types of

behavioral malfunctions and perceived symptoms have been associated with job overload (Cooper et al., 1979; Kasl, 1973).

There appears to be a relationship between quantitative overload and cigarette smoking. Kroes et al., (1974) found that job overload was associated with such stress related symptoms as lowered self-esteem, low work motivation and escapist drinking.

In a study of 100 coronary patients, Russek et al., (1958) found that 25 per cent had been working two jobs and an additional 45 per cent had jobs that required 60 or more hours per week.

Some research suggests that both qualitative and quantitative overload produce different symptoms of psychological and physical strain, including job dissatisfaction, job tension, lowered self-esteem, feelings of threat and embarrassment, high cholesterol levels, increased heart rate and increased smoking (Cox, 1980).

The effects of underload are often aggravated by lack of control over the work situation (Gardell, 1976). Underload may also be a problem related to the application of new technologies. For example, most of the operators time in nuclear power plants is shown to be spent on monotonous rather than stimulating tasks. In certain jobs, such as policing and operating nuclear power plants, periods of boredom have to be accepted, along with the possibility that the mediocrity may be suddenly disrupted by an emergency situation (Davidson et al., 1980). This can cause sustained arousal in the employee's physical and mental state which may have subsequent detrimental effects (Bosse et al., 1978).

(c) *Arrangements of Work Time* - Daily work hours, as well as weekly, monthly, annual, and lifelong work time, to a large extent structure the way of life of the working population. The arrangements of work time is relevant to sleep-wakefulness patterns, social participation and general lifestyle.

Shift work is known to affect biological rhythms, such as circadian variations of body temperature, metabolic rate, blood sugar levels, mental efficiency and work motivation. The effects in daily life can be seen in sleeping behaviour, eating habits, family life and social activities.

A study by Cobb et al., (1973) on air-traffic controllers showed four times the prevalence of hypertension as well as a higher incidence diabetes and peptic ulcers than their control group of second-class airmen. Although other job stressors were important in the causation of these stress-related problems, a major job stressor was shift work.

Nevertheless, most investigations agree that shift work becomes physically less stressful as work schedules are better organised and individual workers are helped by various means of social support. Restriction of social participation is a common complaint among shiftworkers.

(d) *Job Clarity* - A person's role at work has been shown to be a major source of occupational stress when it involves role ambiguity (a lack of clarity about tasks), role conflict (conflicting job demands) and conflicts stemming from organisational boundaries (Cooper, 1976). It has been indicated that organisational stressors stemming

from role ambiguity and conflict can be associated with a risk of cardiovascular disorders (Eden et al., 1973). Workers in managerial, clerical and professional occupations are especially prone to occupational stress related role conflict (WHO report # 56, 1984).

According to a review by Kasl (1979), correlations between role conflict, ambiguity and job satisfaction are strong, while mental health measures tend to be weak. Personality traits are an important determinant of how an individual reacts to role conflict.

(e) *Personal Responsibility* - Having the responsibility for people and their safety has the potential to be an occupational stressor. The pressure on nuclear power operators, for example, may be caused by their responsibility for the safety of workers and the community when faced with unusual situations. Having this responsibility is a potential stressor among police and prison personnel (Kalimo, 1980). The responsibility for people's lives and safety was found to be a major occupational stressor among air-traffic controllers (Cooper et al., 1980). Also, there is evidence that stress is linked to the level of responsibility; the more responsibility, the greater the probability of risk factors or symptoms of cardiovascular disease.

(f) *Worker's Participation* - Organisational structure and climate, including such factors as office politics, lack of effective consultation, lack of participation in the decision-making process and unjustified restrictions on behaviour, make up a complex set of factors which, to a great extent, affect workers' well-being. Kroes et al., (1974) found that greater participation led to higher productivity, improved performance, lower staff turnover and lower levels of physical and mental health problems (including such stress-related behaviours as escapist drinking and heavy smoking).

(g) *Interpersonal Relationships* - The nature of relationships and social support from colleagues, supervisors and subordinates have been related to job stress (Payne, 1980). Poor relationships with other members of the group have been shown to increase stress while strong social support from peers relieved that stress and also moderated the effects of job stress on physiological functions and on smoking habits. In the case of air traffic controllers, more meaningful social support was provided by friends and colleagues than by those in supervisory positions.

(h) *Technological Change* - Psychological stress and related health problems associated with automated work process and the application of microelectronics are of concern to a number of researchers. Investigations have been carried out on ergonomic and hygienic work conditions and improvements in automated work processes (Cakir et al., 1979) and in visual display terminal (VDT) operations (Gunnarson et al., 1977). Reports on work with VDTs have demonstrated that the cognitive content of work is of crucial importance in building up the mental workload and stress reactions (Cakir et al., 1979). Data-entry work is repetitive and lacks variety and challenge. Employees in these tasks have reported more stress reactions and health complaints, than their co-workers in jobs with more variation, including, for example the correction of errors and dealing with clients (Cail et al., 1980; Cohen et al., 1981). Perceived complaints of mental load and stress have been confirmed in physiological indicators (eg. increased cortisol levels in VDT operators).

Changes in the qualification level of a job, which occurs with the transition into automated work, also affects workers (Cakir et al., 1979). When the qualification level was lowered, workers complained of monotony, even though the work was not repetitive. On the other hand, Gunnarson et al., (1977) reported that 60 per cent of such VDT operators who had previously been in clerical work complained about monotony and fatigue, although their current work content was similar to that of their previous tasks.

Delays and breakdowns in the computer systems were perceived by workers as being strongly disturbing. The lack of ability to anticipate such breakdowns and delays, and thus control the workload, tended to be the main causes of irritation and helplessness. On the other hand, even when the perceived mental workload was diminished to a certain extent, the period of introducing the change was perceived as being stressful by a majority of employees (Huhtanen, 1983).

In spite of the problems involved in the application of computer technology in certain jobs, computerisation may bring additional challenge and variety to the most routine and repetitive traditional ways of working. For example, it was found that text-setting workers using VDTs in the printing industry were more satisfied, reported fewer complaints of long-term daily stress and showed fewer signs of negative problems such as stress and inappropriate behaviour. It seems that the when problems of stress and inappropriate workload arise in computerised work settings, the main causes are a lack of variety and a lack of control over the job.

(i) *Unemployment or Threat of Unemployment* - Unemployment and underemployment relate to psychosocial factors at work. In many industrialised countries, the employment situation has deteriorated in recent years (WHO, 1984). The instability of employment affects workers' well-being. From observation, they become more ready to accept work under poor working conditions. They are often subjected to hard, long working hours, and a poor quality of work. The threat of losing the job adds to the job-related tension of workers.

Psychological Effects of Air Pollutants

Research into the specific psychological and behavioral responses to particular indoor pollutants is sparse (Evans et al., 1983). Using a Pavlovian conditioning paradigm, Russian investigators have reported that low-level exposures to such agents as nitrogen dioxide and sulphur dioxide may alter human visual processes, lengthen reaction time, and increase dysynchronization of alpha rhythms (Izmerov, 1971).

Evans et al., (1983) suggests that the irritating effects of oxidants and carbon monoxide may heighten anxiety and depression, producing an overall decline in mood state and psychological functioning. Indirect support for this proposal is presented by Strahilevitz et al., (1979), who reported moderate positive correlations (0.22,0.20) between ambient nitrogen dioxide levels in the St. Louis area and admissions of patients to psychiatric hospitals with alcoholic and organic brain syndromes.

Lagerwerff (1963) exposed subjects to concentrations of ozone ranging from 0.2 to 0.5 ppm. Apart from the physical reactions (refer section on inorganic gases), Lagerwerff also reported

obvious lethargy in many of his subjects, persisting for as long as a day or two after the exposure period.

In discussing psychological responses to air pollution, it is important to distinguish between the toxicologic characteristics of a pollutant and its averse properties (Colligan, 1981). The odour of an element, for example, may produce drastic alterations in mood state and demeanour, independently of neuropathic effects associated with acute exposures. Rotten et al., (1979), exposed subjects to either a moderately offensive odour (ethyl mercaptan) or an extremely offensive smell (ammonium sulphide), and found that the moderately offensive odour, but not the extremely offensive one, increased aggressive tendencies relative to a non-exposed control group. The authors suggest that the failure of the highly obnoxious odour to heighten aggressive responses may have been due to a stronger drive to escape from the setting entirely. They also cite other research indicating that malodorous pollution depresses mood states, decreases liking for other people, and generally devalues the surroundings. It is suggested that dividing the world into smokers and nonsmokers seems entirely justified in light of recent research indicating that nonsmokers feel more anxious, irritated, and generally fatigued when subjected to cigarette smoke and may evidence increased feeling of aggression (Jones, 1978).

It appears, therefore, that in addition to immediate neurological and physiological responses to specific pollutants, complex psychological reactions involving mood state, motivation, and interpersonal relations may also be triggered (Colligan, 1981). Odour, visibility, taste, and similar nontoxic yet aversive qualities of airborne pollutants are often a more immediate and obvious concern to an exposed individual than probabilistic statements about the potential long-range effects to chronic exposure. Selye's study of the "pharmacology of dirt" represents an attempt to understand this process in terms of the body's overall response to insult of any type. The individual, of course, is not impervious to these changes and recognises that something is wrong. Decrements in sensormotor performance or intellectual functioning, mood changes and increased irritability, or feelings of mental and physical fatigue and general malaise provide feedback to the individual that his/her system is under stress. This is the psychological correlate of Selye's general adaptive syndrome, and its study is referred to as the "psychology of dirt." Anxiety, tension, arousal, indeed the term "stress" itself, have been used to describe the vague psychological response that accompanies the awareness that the system is under challenge. The way that these altered physiological and psychological states are interpreted, in terms of their presumed cause and significance have important consequences for how different individuals cope with fluctuations in these internal states.

KEYPOINTS: BRI symptoms are similar to and may be described as classic psychosomatic symptoms. In many instances, it is likely that psychological reactions are the effect rather than the cause. Human emotions can be manifest as physical symptoms. Lower level functionaries are more symptomatic than management. Odour may cause stress and increases aggression and decreases environmental satisfaction. Causes of job related stress include poor interpersonal relationships, lack of job satisfaction, monotony, inability to function at the required level, insecurity, shift work, role conflict, role ambiguity, personal responsibility, poor management practices. Mass hysteria has sometimes been implicated in outbreaks of building related illness but usually need a unifying triggering event. Women tend to be represented in such outbreaks. Psychological factors are thought to be pivotal in the incidence of symptom reporting.

CLINICALLY DIAGNOSABLE SYNDROMES (CDS)

IMPLICATIONS FOR BRI SYMPTOMS

There is some evidence linking micro-organisms with SBS type symptoms.

Mainville et al., (1988) specifically attributed symptoms of extreme fatigue in a Canadian hospital to mycotoxins from mould growing where water penetration had occurred.

Schiefer (1990), attributed mycotoxins to a range of symptoms similar to those of SBS.

Stroem et al., (1990) reported that micro-organisms trapped in material and do not become airborne can release VOCs which could have effects on the health of building occupants.

IDENTIFICATION OF CDS

The literature is not clear as to how symptoms other than those associated with SBS should be diagnosed.

The generic diagnostic approaches available to the investigator are:

(1) epidemiological;
(2) direct medical diagnosis, when questionnaires are generally inappropriate, and;
(3) cause-effect linkages.

Each approach is useful depending on the underlying circumstances.

(1) The epidemiological approach is probably most useful in identifying an excess of allergic respiratory diseases, Pontiac, Q-fever, Humidifier fever and general flu-like symptoms. The reason for this is that most allergic respiratory diseases are present in the population at large so excesses can be diagnosed provided there are known benchmarks. Pontiac, Q-fever and Humidifier fever all have symptoms similar to influenza. However, influenza tends to be seasonally epidemic while the former are not. Thus, unseasonal clusters of symptoms should, in theory, be obvious.

(2) Direct medical enquiry is probably more appropriate in diagnosing serious diseases such as Legionnaires disease, or when small numbers of the building occupants are reporting unusual symptom clusters. Anon (1991), reports Burge as stating that examination of medical records may be the most appropriate sampling strategy in these instances.

(3) Diseases such as work related asthma may only affect a small sub-group within a building. An epidemiological (statistical) approach is worthless in these instances. Statistics are generally not useful in identifying 'one-off' events. In these cases an investigation may be initiated at the request of one person or a small sub-group of symptomatic persons.

For example, if one person or a small sub-group of occupants are affected on an occupationally mediated basis, it probably suggests the presence of a sensitizing agent. In this event a cause-effect study, such as patch testing, would probably yield the best results.

BUILDING RELATED VIABLE CONTAMINANTS

Numerous biological contaminants that can cause human disease are present in indoor air. They are either living organisms such as viruses, fungi and bacteria, or the byproduct of living organisms.

(1) *Viruses* - These organisms are usually transmitted through the air from person to person via airborne droplets or by airborne particle exchange (ie. rafts of human dander). Human pathogens require a human host to survive. Whether viruses are strictly building supported is a matter of debate. Arguably, a buildings role in facilitating viral spread is simply one of bringing more people in contact with each other. On the other hand there is some evidence that air conditioning systems facilitate the spread of measles and hives.

(2) *Bacteria* - The best known bacterium, in the building related sense, is *legionella pneumophilia* which is unusual among bacterial pathogens. It exists in natural outdoor reservoirs and infection is possible through inhalation of contaminated outdoor air. Cordes et al., (1980) suggests that the familiar mode of spread of the bacteria involves air modifying equipment that becomes contaminated with outdoor dust and produces concentrated bacterial aerosols. Ideal conditions for bacteria to proliferate is in the presence of moisture and a temperature range of 25-45°C, although they can survive outside of these parameters.

Lewis et al., (1969), suggests that the primary source of bacteria in the indoor environment is the human body. Approximately, 7 million skin scales are shed every minute per person, each fragment with an average of 4 viable bacteria.

Air conditioning units are now clearly identified as sources of intramural bacterial aerosols (Cordes et al., 1980). Crowley (1936), and Rozenzweig (1970), identified cool-mist vaporizers and nebulizers as a potential source of heavily contaminated aerosols. Smith (1977), attributed cool mist vaporizers as sources of acinetobacter infection.

Rylander et al., (1974), discussed carpeting as a focus for bacterial contamination but concluded that carpeting acted as a 'sink' trapping microbe laden particles within the pile. Bacteria can grow continuously in damp wool and become airborne during remodelling, with epidemics of respiratory disease resulting (Johnson et al., 1981).

(3) *Actinomycetes* - Thermophilic actinomycetes are not common in outdoor air but may be extremely abundant in interiors where plant materials are handled (Burge et al., 1980). According to Lacey (1977), levels of these organisms in barns and cotton mills can exceed 30,000/m^3 of air.

Domestic sources of actinomycetes are less clearly identified. Thermophilic actinomyctes have been recovered from humidifier fluid in air conditioners (Burge et al., 1980, Fink et al., 1971, Seabury et al., 1973), and Marinkovich et al., (1975) recovered the organisms from an evaporative cooler.

(4) *Fungi* - Fungal spore levels in domestic interiors have ranged from 1 to 6000/m^3 with normal levels below 1600/m^3. Burge et al., (1978), have shown that sources in nature are the major contributor to indoor air levels, with levels ranging from 20 to 300% of those outdoors.

Rose et al., (1979), has shown that filtration substantially lowered Hospital spore levels. Villaveces (1971), showed that a lowering of the relative humidity may also be a contributory factor to the reduction of spore levels.

Contamination of interiors commonly involve outdoor fungi that flourish on specific substrates. Burge et al., (1978) and Kozak et al., (1978), showed that water disasters often produce abundant mould growth indoors with attendant elevation of airborne spore levels. Any organic material will support mould growth when wet. Damp walls may acquire abundant Cladosporium cladosporioides and Aureobasidium (Liebeskind, 1971; Lumpkins, 1976), while damp leather, cotton and paper readily support Penicillium and Aspergillus spores. In one study by Swaebly et al., (1952), concentrations from 10^3 to 10^6 of fungal spores per gram of house dust were noted. Studies by Richards (1954), and Maunsell (1952), clearly showed that dust raising activities increased indoor air suspended spore levels. Staib et al., (1978; 1978), implicated house plants as sources of A. fumigatus in hospital rooms.

(5) *Human Pathogens* - Aspergillus fumigatus is a ubiquitous fungus and can be present even where minimal organic material provides a suitable substrate (Raper, 1965; Llamas et al., 1978).

The incidence of invasive aspergillosis in the population is low even amongst those exposed to high concentrations of spores (Halweg et al., 1978; Horejsi et al., 1960). The risk of hypersensitivity disease, however, appears to be significant.

Blastomyces, Cryptococcus, Coccidioides, and Histoplasma are all known human pathogens and exist in saprophytic natural reservoirs usually associated with bird and animal droppings (Ajello, 1967; D'Alessio et al., 1965). Levels of contamination achieved within interior situations by these fungi are unknown, however all enter the body by a respiratory route, and Coccidioides and Histoplasma are known to be highly infective (Swinne-Desgain, 1975). Cryptococcus has been isolated from air and from an air conditioner contaminated with bird droppings and feathers (Botard et al., 1969).

(6) *Pollens* - The presence of pollens in indoor air generally reflects incursions from outdoor sources. However Burge et al., (1984), found that with large indoor plants, pollens of cyclamen and impatiens have been found to reach several hundred grains per m^3.

Pollen is a common antigen to susceptible individuals, being one of the primary causes of perennial rhinitis. Nelson et al., (1936), and Freidlander et al., (1954), showed that electrostatic precipitators were of benefit to pollen-sensitive patients. Zwemer et al., (1973), claimed the benefit of HEPA filtration units in excluding pollen from the occupied space. It would seem that filtration is beneficial in reducing pollen levels.

(7) *Protozoa* - Protozoa has been recovered in culture from free air by several investigators as summarized by Schlichting (1969). Indoor fluid collections such as aquaria and humidifier reservoirs are among the sites of potential colonization by protozoa. MRC Symposium, (1977), cast suspicion on protozoa as the causative agent in 'Humidifier Fever'. In at least one outbreak Edwards et al., (1976), discovered a profusion of ciliates and amoebae on the humidifier baffle plates and accumulated dust. Sera from affected persons were shown to react with amoebae from the site, though not with ciliates. Reactivity was especially pronounced with antigens of *Naegleria gruberi.*

(8) *Acarids* - Largely interest in this group centres around pyroglyphid mites which contribute sensitizing materials to house dust. According to Mathews, (1978), house dust is poorly characterized and variable substance which is a recognized causative agent of allergic rhinitis and asthma. Cunningham et al., (1968), recovered *Dermatophagoides pteronyssinus* and D. *farinae* from air in indoor air. Allergic reactivity to these agents and to house dust has been described by Miyamoto et al., (1969) and Mitchell et al., (1969). Hewitt et al., (1973) suggested a role for mites in urticaria and other skin disorders, but on limited evidence.

Dust mites require relative humidity (RH) levels of around 75% to avoid death by desiccation. They can, however, enter a state of minimal bodily function and Sundell et al., (1990) has shown that they can survive the extremely low RH levels of the Swedish winter.

(9) *Human Dander* - Gregory, (1973), reports that epidermal scales of human origin are the most abundant, microscopically distinctive, component of indoor dusts. These structures readily serve as 'rafts' for aerial transport of bacteria and other microorganisms. The role of human dander as a distinct antigen for allergic subjects is controversial. However Voorhorst, (1977), reported antibody responses to this material.

SYNDROMES ASSOCIATED WITH BIO-AEROSOLS

Anon (1991), attributed the following range of syndromes to bio-aerosols. There is no compelling evidence in the literature linking many of these syndromes to the occupancy of commercial buildings. Where these links are tenuous or contentious, reference to the diseases may be found in Appendix 9.

TABLE 13 - DISEASES RELATED TO BIOAEROSOLS

	Transmission	Agent	Risk factor	Test Protocol
Contagious Diseases: influenza & some colds	Human to human (only)	Viruses growing on inanimate surfaces; filtration is not effective	Lack of specific immunity; Dose; Virulence	Disease ID; Epidemiology; Note: Air sampling is useless
Rickettsial Infection	Outdoor reservoirs or housed animals	Q-fever; Epidemic typhus; Brill-zinners disease; Endemic typhus; Rickettsalpox	Lack of specific immunity; dose; virulence	Disease ID; Sampling is generally not useful
Mycotic Infections Bacterial Diseases	Outdoor Reservoirs and birds and animals	Aspergillosis; Histoplasmosis; Coccidioidomycosis; Blastomycosis; Cryptococcosis Sporotrichosis Anthrax	Lack of specific immunity; dose; virulence	Disease ID; sampling is generally not useful except air sampling for aspergillus
Opportunistic infections from environmental sources: Legionellosis, Pontiac Fever	Cooling towers, compost, water reservoirs	Legionella pseudomonas	Immuno-deficiencies, dose, virulence	Source sampling of reservoirs
Disorders caused by Organic Agents	Exposure to antigens	Hyper- sensitivity Pneumonia; Humidifier fever; Extrinsic allergic alveolitis; PVC lung disease;	Genetic; Exposure patterns; Antigenicity	Disease ID; Site observationSource sampling; Air sampling
Hyper-sensitivity Atopic Diseases	Exposure to antigen	Allergic rhinitis - Hay fever; Anaphylaxis;	Exposure patterns; Antigenicity	Perennial or seasonal pattern
Occupational asthma; Byssinosis	Exposure to sensitizing agent;	Numerous including organic, inorganic compounds, animals, fungi, metals, dyes, drugs & enzymes, isocyanates	Genetic predisposition; exposure patterns	Site observation & source sampling

BUILDING SUPPORTED SYNDROMES

VIRAL DISEASES

Viruses are small parasites, several hundred species of which may infect man. Many have only been recognised recently, so their clinical effects are not fully understood. The viruses occurring primarily in man are spread chiefly by man himself mainly via respiratory and enteric excretions. Such viruses (refer Table 47; Appendix 9) are found in all parts of the world, their spread being limited to inborn resistance, prior immunizing infections or vaccines, sanitary and other public health control measures.

Many viruses pursue their biological cycle chiefly in animals, man being only a secondary or accidental host. The zoonotic viruses in contrast to the specifically human agents, are limited to those geographic areas and environments able to support their extrahuman natural cycles of infection (vertebrates, arthropods or both).

It is highly debatable whether viruses are building supported although there is some evidence that air conditioning systems can assist the spread of measles, hives and influenza (Anon., 1991).

VIRULENT INFECTIONS FROM ENVIRONMENTAL SOURCES

(1) RICKETTSIAL DISEASES

Rickettsial diseases are a zoonosis requiring an animal or avian reservoir and an insect vector (usually arthropod) to infect humans. A variety of illnesses manifested by sudden onset, a course of fever of one to several weeks, headache, malaise, prostration, peripheral vasculitis, and, in most cases, a characteristic rash.

Rickettsioses comprise four groups, however only one group is of interest in the building related sense.

Q-fever has been identified as a building supported syndrome in a number of studies.

Q-fever is a zoonosis caused by Coxiella burnetti. C. burnetii is maintained in nature through an animal tick. Various arthropods, rodents, other mammals, and birds are naturally infected and may play a role in human infection.

The disease may take several forms, from a mild, acute, influenza-like illness to pneumonias or endocarditis. The incubation period after exposure varies from 9 to 28 days. Onset of symptoms is abrupt, with fever, severe headache, chilliness, severe malaise, myalgia, and chest pains. Fever may rise to 40 C (104 F) and persist for 1 to 3 weeks. Mortality is less than 1% in untreated patients. Hepatitis may occur in about one-third of patients.

A number of reported outbreaks were associated with research facilities housing infected animals (Bayer, 1982; Dritz et al., 1979). Contamination of the air intakes with bird

droppings led to entrainment of the infectious agent in the ventilating system and dissemination of the disease to the building occupants.

As the symptoms are not specific and the disease is rare, the disease is frequently mis-diagnosed. One building related outbreak was noted solely because the local health department routinely tested for Coxiella markers in the blood of persons with obscure disease (Dritz et al., 1979).

(2) Mycotic Infections

These diseases are caused by fungi. The major diseases of interest are:

(a) Aspergillosis
(b) Histoplasmosis
(c) Coccidioidomycos
(d) Blastomycosis
(e) Cryptococcosis
(f) Sporotrichosis

Refer Appendix 9.

Opportunistic Infections from Environmental Sources

(1) Legionnaires Disease

Perhaps, of all the building supported illnesses, legionnaires disease attracts the greatest level of public attention. While the disease can be fatal in about 15% of cases, it's incidence is rare. The bacterium which causes the disease, however, is ubiquitous. The disease has been studied in depth and knowledge of it's epidemiology and pathology is extensive.

Background and Incidence

The first identified outbreak of Legionnaires disease was in Philadelphia in 1976. It is now clearly established that the offending organism was *Legionella pneumophilia*. These are freshwater organisms that have been found worldwide. They multiply in water at temperatures between 25 - 42° C but are able to survive a wider temperature for long periods. In survey by the DHSS in Britain of large buildings, Bartlett et al., (1988), reported that in 53% of hotels and 70% of hospitals sampled L. *pneumophilia* was present in at least one sample. Hot water was most often contaminated (hospitals 68%, hotels 49%) than cold (hospitals 24%, hotels 9%).

In spite of its ubiquity, the incidence of infection is low. The reasons for this are not fully understood but some are suggested. L. pneumophilia has been divided into 12 serogroups of which serogroup 1 strains are responsible for more than 90% of illnesses. Watkins et al., (1985), subdivided serogroup 1 strains into three further subgroups - Pontiac, Olda and Bellingham.

Sykes et al., (1988) summarises what is known about the disease and its causes as follows:

(i) Legionnaires disease is a form of pneumonia which also has effects on organs other than lungs. It is relatively rare when compared with other causes of pneumonia. Kurtz, (1988), reports the figure of community acquired pneumonias in Britain due to the organism to be about 2%. Victims of the disease are usually particularly susceptible for one reason or another - underlying disease, drug treatment, smoking or middle age in men. Because of this, most of the major outbreaks occur in places where people who are particularly susceptible congregate, such as rest homes and hospitals. Fit people are unlikely to get Legionnaires' disease even if infected with L. *pneumophilia*.

(ii) The disease is caused by inhaling water droplets and particles containing the causative bacterium. According to Kurtz, (1988), the infection does not spread from person to person but is contracted from the environment.

(iii) L. *pneumophilia* is very widespread and readily enters building water services and water systems. Many, if not most, water systems are contaminated.

(iv) Legionella proliferates in the presence of sludge, scale, rust, algae and most importantly in water of temperatures in the range 25 - 42° C.

Sources of the Bacterium

Various sources of Legionnaires disease have been identified. These include:

(a) hot water systems, especially pumped systems at high pressure. Showers, spray taps and bath taps are all believed to have caused cases of the disease, especially in hospitals;

(b) cooling towers and associated water systems have caused outbreaks not only in the building they serve but also over a wide area. An example of this was the BBC studio outbreak in London where the L.*pneumophilia* aerosol remained viable for over 500m from it's source and infected passers by in the street. Clearly, this has implications for adjacent buildings where the possibility of cross contamination exists;

(c) commercial spas.

Risk Assessment and Mitigation

Before deciding on precautionary measures it is necessary to assess the danger. For the disease to occur, three factors are necessary:

(i) heavily contaminated water
(ii) a means of dissemination
(iii) a potential victim (ie. a susceptible person)

If one or more of these factors is missing, then Legionnaires disease is unlikely. If all three factors are present, then the object must be to remove one or more of them.

The British Health and Safety Executive's Guidance Note EH 48, recommends the following measures:

(a) the avoidance of temperatures in the range 20 - 50o C. At temperatures below 20° C *Legionella* will not multiply, so that even if *Legionella* is present it will be at low concentrations and the risk will therefore be small. At temperatures over 50° C Legionella will be killed, especially at 60° C or more, so it may be eradiated by storing water at 60° C. But in many establishments occupied by old people, mental patients and children this will be unacceptable because of the increased danger of scalding;

(b) the avoidance of conditions which allow water to stand undisturbed. Kurtz, (1988), suggests that pipe runs should be as short as possible, and hot pipes should not run so close to cold ones that warm water results. Dead-legs and other areas where water may be stagnant should be eliminated.

(c) the avoidance of materials that can harbour bacteria and recontaminate water.

(d) regular cleaning and disinfection to remove *Legionella* and potential nutrients. The frequency of cleaning and disinfection will depend on the type of system and the more frequently these take place the better, especially for systems serving cooling towers which may require cleaning every 6 months or less, and;

(e) water treatment. This is a complex subject; it is not sufficient to add a bit of biocide and then forget about the system. Water treatments include scale or corrosion inhibitors, dispersants to prevent sediment from settling, biocides to kill bacteria and other organisms, and disinfectants. The effectiveness of many of these chemicals depends on their proper use, and in some cases they may react with each other or the system, causing corrosion.

(2) PONTIAC FEVER

Pontiac fever is a self limiting from of disease attributed one of the strains of legionella bacterium. It has also been associated with cooling towers, steam turbines, whirlpools, and, in one case, contamination of an oil cutting fluid in an automobile plant.

BACKGROUND AND INCIDENCE

The term *Pontiac fever* refers to an illness that occurred at a county health department facility in Pontiac, Michigan in 1968. It was described as an explosive epidemic of an acute febrile illness (Glick et al., 1977). The illness characterized principally by fever, chills, headache, and myalgia, affected at least 144 people, including 95 of 100 persons employed in the health department building. At the time the bacterium was found to be similar to that associated with legionnaires disease but, unlike the latter, was unassociated with pneumonia and/or mortality. The attack rate, as opposed to that of legionnaires disease is extremely high, approaching 100% in some cases (Hodgson et al., 1986).

Pontiac fever is caused by a sub-strain of the legionella bacterium. Although the Pontiac subgroup only accounted for 11% of the serogroup 1 strains isolated in Oxford in 1985-1986 (c.f 64% Olda and 25% Bellingham) from sources not associated with the disease (Tobin et al., 1986), it is responsible for about 90% of cases of disease. This suggests that Pontiac strains are more virulent.

Symptoms and Signs

A relatively uniform, self-limiting illness of chills, fever, headache and myalgia and lasting two to five days. The incubation period from first exposure to the agent appeared to be about thirty-six hours. In the Pontiac outbreak, the etiologic agent persisted in the building for a minimum of two weeks and existed in parts of the air-conditioning ducts or in the machine room for at least five weeks after its first appearance. The flu like symptoms could easily lead to mis-diagnosis.

The interesting factor about Pontiac Fever is that it is not spread from person to person, rather it can only be transmitted from an environmental source. This factor may be used as a diagnostic technique in that the disease will be limited to the building occupants and users and will not be transmitted to their families or associates.

Building Supported Hypersensitivity Syndromes

(1) Hypersensitivity Pneumonitis

Background and Possible Causes

Hypersensitivity pneumonitis, also known as extrinsic allergic alveolitis is a well described reaction, studied in detail among occupational groups (Parkes, 1982).

Generally, the syndrome is an allergic response to one of a variety of inhaled organic dusts. For instance farmer's lung is associated with repeated inhalation of dusts from hay containing thermophilic actinomycetes. The number of specific organic dusts known to be capable of causing hypersensitivity pneumonitis is increasing. The dust is generally either a microorganism or a foreign animal or vegetable protein inhaled in considerable amounts.

The disease is considered to be immunologically mediated. Precipitating anti-bodies to the offending antigen are usually demonstrated.

Only a small proportion of exposed persons develop symptoms, and then only after the considerable period of exposure required for induction of sensitization. Chronic parenchymal disease may result from continuous or frequent low-level exposure to the antigen. A history of previous allergic disease (eg., asthma, hay fever) is uncommon and is not a predisposing factor.

Symptoms and Signs

Symptoms consist of cough, chest tightness, breathlessness, fever, chills and muscle aches. Typically, it is related to the work week with resolution on weekends. Episodes of fever, chills, cough, and dyspnea occur in a previously sensitized individual, typically appearing 4 to 8 hours after re-exposure to the antigen (ie. return to work). Anorexia, nausea, and vomiting may also be present.

(2) Air Conditioner Disease (Humidifier Fever)

Background and Possible Causes

Parkes (1982), argues that hypersensitivity pneumonitis and humidifier fever are two separate diseases. The major distinguishing characteristics are thought to be an attack rate of 30-70% in humidifier fever as compared with much lower rates in hypersensitivity pneumonitis. Humidifier fever has been attributed to amoebae, thermophilic bacteria, fungi and endotoxin, whereas hypersensitivity pneumonitis is usually attributed to thermophilic actinomyces.

According to Parkes (1982), 'humidifier fever' was described by Pestalozzi in 1959 and by HM Chief Inspector of Factories (1969) among printers who had been exposed to water mist sprayed into the workroom from humidifiers contaminated with algae and bacteria. Respiratory and constitutional symptoms consistent with extrinsic allergic alveolitis have also been reported in relation to air conditioning systems contaminated with micro-organisms. Banaszac et al., (1970) originally attributed the disorder to M.*faeni* spores released into the atmosphere of an office from a contaminated air conditioning system.

Subsequent episodes of the disease have been associated with air conditioning systems in homes, offices, operating theatres and factories, especially in those factories which required carefully controlled humidity, such as printing, stationary and the manufacture of textiles (Fink et al., 1971; Sweet et al., 1971; Weiss et al., 1971; Hodgesnet al., 1974; Friend et al., 1977).

The illness is generally associated with heavy contamination by a mixed growth of bacteria and fungi of water recirculated through humidifiers (hence it's name).

While specific organisms have not been clearly identified, antibodies against *Naegleria gruberi* and Acanthamoeba have been found in affected workers when these amoebae are present in the humidifier systems (Edwards et al., 1976) and the precipitin to extracts of B.*subtilis* were constantly present in a group of badly affected workers.

Symptoms and Signs

Humidifier fever has been called 'Mondayitis' or 'Monday morning fever' because of its particular behaviour. It is an acute illness consisting of malaise, fever, myalgia, cough, tightness in the chest and breathlessness on exertion which resolves itself in 24 hours. A feature of the syndrome is that the symptoms are worse at the beginning of the week and improve during the rest of the week, but recur on re-exposure after an absence from work. This symptom pattern - apart from clear asthmatic features and recurrence on delayed re-

exposure - is similar to that of 'mill fever'. Investigation of four factories in Britain showed that only a minority of workers were affected but their symptoms were worse on Mondays.

Parkes (1982) reports that the incidence of the illness tends to be higher in the winter months due, possibly, to greater recirculation of heated air during this period.

Specific Mitigation Techniques

Parkes (1982) suggests that the prevention of growth of organisms in humidifier systems may be the answer. A rich flora grows readily in recirculated water reservoirs and some systems may release very small droplets into the air. The growth of organisms can be suppressed or prevented by input water filters or by steam injection of water which avoids recirculation or pooling. Heating of storage tanks to 60 or 70 °C and frequent cleaning (though difficult) may reduce the growth of organisms. Disinfectants are contra-indicated as they encourage the growth of some species of bacteria and are themselves likely to be inhaled (Medical Research Council Symposium, 1977).

(3) Sinusitis and Pharyngitis

Symptoms consist of chronic post-nasal drip, nasal drainage and sore throats. Although the mechanism of these complaints have not been defined, they do occur in epidemic settings of hypersensitivity pneumonitis and humidifier fever and by themselves. They have been associated primarily with contamination of peripheral window heating or cooling units or with improperly maintained water-damaged spaces. Cleaning or removing the implicated source has usually resolved the issue (Hodgson et al., 1986).

(4) Allergic Tracheo-Bronchitis

This is an inflammation of the upper airways. Symptoms consist of cough, chest-tightness, and chest pain. Onset is related to the work week with resolution on weekends (Hodgson et al., 1986). It may be distinguished from asthma only because of the primary involvement of the upper airways. One outbreak of the syndrome was attributed to contamination of an air-handling unit filter with *Cladosporium* (Kreiss et al., 1984).

Occupational Asthma

Background and Possible Causes

Underlying the disease is hyperreactivity of the airways to a particular agent. Symptoms consist of a cough, wheezing and chest tightness. Asthma is thought to occur as an immediate (within one hour) or a delayed (within six to eight hours) reaction to an allergic stimulus (Parkes, 1982). Where asthma has developed individuals may react, not only to the specific substance to which they have become allergic but to other pulmonary irritants. Additionally, 'cross sensitization' may occur, so that individuals react more easily to more than one agent at the same time. Because of the non-specific hyper-reactivity and cross-reactivity, the office

environment may lead to exacerbation of pre-existing asthma or induce attacks even when the original offending agent has been removed.

There are any number of potential sensitizing agents in a typical office environment. Burge (1985), published a report associating asthma with exposure to a humidifier. Morey (1984), associated asthma with exposure to dust trapped in upholstered material. A number of office machines use chemicals associated with causation of asthma (Parkinson, 1986).

Background

Although there is no generally agreed definition of asthma, that based on the recommendation of the Ciba Guest Symposium Report (1959) appears to be widely accepted. It is as follows:

> *'Asthma is a disorder of function characterized by widespread partial obstruction of the airways which varies in severity, it is reversible either spontaneously or as a result of treatment, and is not due to cardiovascular disease.'*

Asthma falls into two subgroups: *'extrinsic asthma'* due to specific external allergens; and *'cryptogenic (or intrinsic) asthma* in which no external agency is evident. Occupational asthma, therefore, is caused by some specific extrinsic agent or agents in the form of dust, fume or vapour in an industrial environment. Byssinosis fulfils this definition of extrinsic asthma.

While it is impossible to give an overall indication of the prevalence of occupational asthma, it has been estimated that about 2% of all cases are occupationally related (Introna, 1966). In Japan, some 15% of asthma in adult males is believed to be occupational (Kobayashi, 1974).

Typical Development Pattern of Occupational Asthma

An allergic reaction does not occur on first exposure. The latent interval during which sensitization occurs varies from a few weeks to many years. When asthma first develops some years after an employee enters an industry, there is a high probability that an occupational origin of the disease will be overlooked (Parkes, 1982).

Physiological Patterns of Occupational Asthma

Four patterns of asthmatic reactions have been defined which are, apparently, largely determined by the cumulative effect of repeated exposure and the time taken for recovery.

(a) *Progressive deterioration throughout the working week.* Symptoms and reduction in ventilation are more severe at the end of the week than at the beginning of the week and recovery takes one to three days. Provided that recovery is substantial within two or three days the weekly pattern is regular but if it takes three days and a late reaction occurs on the first day back to work the record (PERF value) is the best of the week (the Monday best pattern). A morning dip may not develop until late in the week or it may be present throughout the week and on Sundays.

(b) *Similar deterioration on each work day.* Symptoms develop during each working shift but improve rapidly on leaving work so that recovery is virtually complete before the next day.

(c) *Progressive deterioration week by week.* This develops if the recovery period takes more than three days and the individual returns to work at the beginning of each week while lung function is still reduced. A gradual decline in PEFR occurs until a state of 'fixed' airflow obstruction is reached. On withdrawal from exposure recovery may not begin for about ten days and, on occasion, may last for as long as three months. This pattern appears to be particularly associated with asthma due to wood dusts and isocyanates but also occurs with other substances.

(d) *Maximal deterioration on the first day of the week.* This pattern in which recovery occurs during the remainder of the week is only occasionally encountered in occupational asthma. It is a specific feature of 'humidifier fever' proper, polymer fume fever and byssinosis.

DERMATITIS

Dermatitis may occur in buildings through allergies, phytotoxicity, and irritation. The diagnosis of work-related skin disease may be made through patch-testing (ie, exposing the skin to the putative agent and demonstrating a specific reaction).

(1) ALLERGIC DERMATITIS

Allergic reaction has been described with copier and toner fluids, hydroquinone, and carbonless copy-paper (Marks et al., 1980; 1984; NIOSH, 1979; Kleinman, 1982). Such disease can present either as classical eczema (scaling and weeping dermatitis), as urticaria (hives), or as simple dry, itching skin. Compilation of substances used in offices or for cleaning demonstrates a large number of chemicals previously associated with allergies (Parkinson et al., 1986).

(2) PHOTODERMATITIS

There have been some reports of dermatitis associated with the use of VDUs (Feldman et al., 1985; Nilsen, 1982; Rycroft et al., 1984; Tjonn, 1984). They consistently suggest that erythema could be provoked by light challenge. Although phytotoxicity from UVb light is well described, patch testing with UVa and UVb light has consistently failed to reveal sensitivity. Mounting an anti-glare screen which reduced the visible light spectrum relieved the problem in a number of cases (Rycroft et al., 1984).

(3) IRRITANT DERMATITIS

Irritant dermatitis has been attributed to fibres, decreased relative humidity, and irritants in use in offices. The first are a common cause of epidemics of rash and itching in office workers (Kreiss et al., 1984). A characteristic finding is relief of symptoms after showering. Fibres deposited on flat surfaces have allowed identification of the problem, whereas air-sampling has generally not proven helpful as fibres were always well below the permissable

exposure limit and usually not measurable. Molhave (1985), has described skin irritation from volatile organic compounds in an experimental setting although at a level well above that usually found in offices.

REPRODUCTIVE COMPLAINTS

(refer to Acute Implications of EMF)

CHRONIC HAZARDS

Investigation of chronic disease requires a different approach to that of acute disorders. The WHO report # 78, (1982), suggests that the pathway in dealing with chronic exposures is preventative rather than curative. The logical progression is as follows:

Expert concern
⇓
Descriptive studies
⇓
Generic analysis
⇓
Exposure and health effects studies
⇓
Epidemiology and impact estimates
⇓
Generic corrective measures

The major concern in occupational settings are those materials and elements that may be carcinogenic.

RADON AND DAUGHTERS

IONIZING RADIATION AS A CARCINOGEN

High fatality rates from lung cancer were observed amongst miners as early as the sixteenth century. However, it was not until the 1950s that excessive lung cancer rates occurring amongst uranium miners in the United States and Canada were linked to exposures to ^{222}Radium decay products.

Uranium gives rise to decay chain products of which uranium-238 is the first member through a series of solid elements to radium-226 which decays to the gas radon-222 which, in turn, gives rise to other isotopes - radon daughters. The important carcinogenic members are those which emit α-particles; namely, radon-222 (half life 3.8 days) and three radon daughters, polonium-218 (half life 3.05 minutes), polonium-214 (half life 26.8 minutes) and polonium-210 (half life 19.4 years).

Thorium is also fairly abundant in the Earth's crust. It decays into thoron gas (radon-220), thorium-A (polonium-216), thorium-B (lead-212) and thorium-C (bismuth-212). Of these thoron, thorium-B and thorium-C are α-emitters.

When first formed, the decay products are single ionized atoms but they readily attach themselves to molecules of water vapour or to dust particles as 'cluster ions' (Chamberlain et al., 1956). In this aerosol state, they can, on inhalation, penetrate the trachea, bronchi and beyond and be retained in the lungs.

α-Particles are positively charged helium nuclei with two protons and neutrons which have a greater mass and kinetic energy than other radiation particles. Owing to their large mass and positive charge, they have only feeble penetrating power. They cause dense ionization along their traverse paths which is maximal when their energy is nearly spent; that is less than 2 MeV (MeV = million electron volts. One electron volt is the energy acquired by an electron when accelerated through a potential difference of 1 volt). This occurs when they have passed through the bronchial mucosa and reached the basal cells. The average penetrability is about 50 μm (Lea, 1955). β-Particles, being electrons, have greater penetrating capacity but less ionizing power. It widely believed that ionization is the cause of malignant change in living cells.

For this reason inhaled α-particles are more important than β-particles - although high doses of the latter may induce lung tumours in experimental animals - there is strong evidence that exposure of man to radon-222 and α-emitting radon daughters is responsible for a significantly increased risk of developing carcinoma of the lung. The radiation dose to the lungs of radon and thoron appears to be approximately similar (Albert, 1966).

Characteristics of Radon and Its Decay Products

The principal characteristic of radon that gives it more radiological significance than earlier members of the uranium (and thorium) decay chains is the fact that it is a noble gas. As such, once formed in the radium-bearing material, a radon atom is relatively free to move, provided it first reaches the material's pore space (typically by recoil from the parent radium atom's emission of an α-particle). Once in the pore space, macroscopic transport of radon is possible, either by molecular diffusion or by flow of the fluid in the pore space.

Secondly, radon decays to radionuclides that are chemically active

Non-Occupational Exposure to Radiation

According to Nazaroff et al., (1988), the radiation dose from inhaled decay products of Radon-222 is the dominant component of natural radiation exposures of the general population in the United States. Monitoring in various countries yields average residential ^{222}Rn concentrations ranging from 10 to 100 Bq/m^3. To put this in perspective, in the United States with an average exposure of around 40 Bq/m^3, the average lifetime risk of lung cancer caused by exposure to radon decay products is estimated to be about 0.3%, causing in the order of 10,000 cases of lung cancer annually among the U.S. population of 235 million. In the USA, it is estimated that the radon dose exceeds by a factor of 10 to 100 the average doses from nuclear power or weapons testing.

A survey by the National Radiation Laboratory in New Zealand, (Robertson et al., 1988) report average residential ^{222}Rn concentrations ranging from 31.2 to 59.6 Bq/m^3.

From the above survey it is apparent that the natural gamma dose rate and peak radon concentrations in New Zealand are low relative to measurements in other countries. A feature

in New Zealand is that there is a higher proportion of wooden houses than in almost any other country used for comparative purposes.

A second feature of note is that the large proportion of the population live in the Auckland area, which has an exceptionally low terrestrial gamma dose rate.

A significant feature of the New Zealand survey is that while the average radon concentrations are lower than those prevailing in some other countries, the maximum concentrations detected were lower than the maxima in all the other countries used for comparative purposes. The New Zealand maximum was equivalent to 94 Bq/m^3 of the radon parent, but levels above 1000 Bq/m^3 have been measured in the UK with one measurement of 8000 Bq/m^3. In one area of Finland, 4% of houses had average concentrations exceeding 2000 Bq/m^3.

It should be noted that the above surveys were limited to residential dwellings. There are no figures available for concentrations in commercial buildings where different construction materials may contribute to a higher radiation dose.

Sources of Indoor Radon

The NRPB (1987) report states that there are five sources of radon in dwellings - natural gas, tap water, fresh air, building materials, and the subadjacent ground. Radon arises from trace concentrations of radium in the earth's crust, and indoor concentrations depend on ingress of this to the building interior. Radon can enter directly from soil or rock which is still in the crust, via utilities such as water and natural gas, or from crustal materials that are incorporated into the building structure in the form of concrete, rock and brick. The relative importance of these pathways depends on the circumstances, but it has become clear that direct ingress from the soil ordinarily dominates the higher indoor concentrations observed in domestic dwellings (Nazeroff et al., 1988).

Indications of this arose in early investigations of radon in U.S. houses, when it was found that measurements of radon emanating from structural materials could not account for observed indoor concentrations, based on estimates of the air exchange rate (Gesell et al., eds., 1981).

Building Material

Indication of the potential significance of radon in the general building stock came with the realization in the 1970s of the health implications of very high concentrations in Swedish homes using lightweight concretes incorporating alum shale as an aggregate (Nazeroff et al., 1988). This shale has an extremely high radium content, causing high radon emanation rates from the concrete, which together with low ventilation rates prompted by the desire to reduce energy, resulted in high airborne concentrations in this segment of housing stock.

In many cases, such as single-family residences in the Nordic countries and the United States, the soil and the bedrock beneath the houses appeared to be the main sources of indoor radon (Standen et al., 1984; Swedjmark et al., 1984; Castren et al., 1984). In larger structures, the building materials may contribute a greater share to the indoor concentration, but the absolute concentration is usually small (Nazeroff et al., 1988). Certain materials have

been found to contribute unusually large amounts of radon, and in such cases the building materials may be the source of unacceptably high indoor radon concentrations.

As early as the 1950s, Hultqvist reported high ^{226}Ra concentrations in alum based shale (Hulqvist, 1956). The use of alum shale based concrete in Sweden was later banned, but it is estimated that about 10% of Swedish houses have this type of concrete as the main building material. Other natural materials containing enhanced levels of ^{226}Ra include granites, some clay bricks and tuff (Kolb, 1974; Soratin et al., 1984; Krisuk et al., 1974; Sciochetti et al., 1983).

In addition, wastes from different industries have been used in building products. Fly ash from coal-fired power plants has been used as an additive in cement, and by-product gypsum from the phosphate industry has been used in plasterboard and in concrete (Pensko et al., 1980; O'Riordan et al., 1972). These wastes can contain a higher than average concentration of ^{226}Ra, and in some cases, these building materials are a significant source of indoor radon.

Nazeroff et al., (1988), reports the following radon concentrations in natural materials and in some building materials.

TABLE 14 - RADIUM CONCENTRATION - NATURAL MATERIALS

Material	Radium concentration ($Bq\ kg^{-1}$)
Concrete	10 - 80
Clay Brick	20 - 200
Cement	10 - 50
Granite	100 - 200
Tuff	100 - 600
Natural Gypsum	5 - 20
Alum-shale	300 - 2500

TABLE 15 - RADIUM CONCENTRATION - SOME BY-PRODUCTS USED AS BUILDING MATERIALS

Material	^{226}Ra Concentration ($Bq\ kg^{-1}$)
By-Product Gypsum	500 - 2000
Fly ash	50 - 300
Calcium silicate slag	1000 - 2000

Factors Affecting Indoor Concentrations

The indoor concentration of radon and its decay products, or of any other airborne pollutant, depends on three factors:

(1) The entry or production rate from various sources;
(2) The ventilation rate;
(3) The rate of chemical or physical transformation or removal.

Because of the relatively long half-life and lack of chemical activity, ^{222}Rn acts much like a stable pollutant whose indoor concentration is determined only by two factors, the rate of entry and the ventilation rate (Nazaroff et al., 1988). In contrast, the behaviour of the decay products is much more complex, depending on the radon that is present, the ventilation rate, and the interplay among radioactive decay, chemical reactivity, particle concentrations, and the nature of the boundary layer between the indoor atmosphere and the surfaces that enclose it. Nonetheless, for practical purposes, the decay-product concentration is indicated approximately by the radon concentration, which is determined by the source and ventilation characteristics.

Both excessive entry rates and decreased ventilation rates appear to be important causes of high concentrations found in Swedish homes, which constituted a signal that scientists in other countries should investigate radon concentrations in their own building stocks.

The importance of source strengths in determining indoor radon concentrations became clear from early studies of homes in the United States. Earlier work had already indicated significant concentrations of ^{222}Rn and its decay products in the U.S. housing stock (George et al., 1980), and subsequent work conformed that the radon concentration could be varied as the inverse of the ventilation rate (Gesell et al., eds., 1981). However, results from simultaneous ^{222}Rn and ventilation rate measurements in several group homes showed no apparent correlation between these two parameters.

Basic Mitigation Methods

The fundamental mitigation methods fall into four categories (Nazeroff et al., (1989).

(1) *Source removal.* This option is only practical in cases where the source has been introduced into the local environment by human intervention.

(2) *Sealing.* Increase the resistance of the building fabric to soil gas entry.

(3) *Ventilation.* Increases the rate of removal of radon from the building by increasing the structure ventilation rate.

(4) *Soil ventilation.* Reduce the soil gas flow by reducing the pressure differential between the building and the soil. The last method is probably the most important for the case of radon concentrations occurring naturally from the soil and rock.

Soil ventilation is often supplemented by sealing. Soil ventilation has a variety of forms. These often involve depressurisation or pressurisation of the layer of material immediately

below the building structure. Pressurisation of this layer is similar to creating a pressure barrier between the bulk of the house and the soil.

Depressurisation usually consists of a perforated pipe network beneath or around the building that is maintained at a pressure lower than the building pressure, either by a small fan or a passive vented stack. The local reversal of the pressure difference causes the air to flow out from the building through the soil to the collection system thus effectively preventing the entry of soil gas through the house-to-soil connections. The radon free air is drawn into the soil from both the building and atmosphere which also dilutes the radon concentration adjacent to the building (Scott, 1979). This system has been applied in a number of locations in Canada, Sweden (Ehdwall, 1980; Ericson, 1980; Ericson et al., 1984), and the United States (Nitschke et al., 1984; Henschel et al., 1986).

KEYPOINTS: The overall mortality assessment from exposure to radon and its daughter products is low. There are five sources of radon in dwellings - natural gas, tap water, fresh air, building materials, and the subadjacent ground. In single-family residences in the Nordic countries and the United States, the soil and the bedrock beneath the houses appeared to be the main sources of indoor radon. In larger structures, the building materials may contribute a greater share to the indoor concentration, but the absolute concentration is usually small. The fundamental mitigation methods fall into four categories (1) Source removal; (2) Sealing; (3) Ventilation; (4) Soil ventilation. Soil ventilation is probably the most important for the case of radon concentrations occurring naturally from the soil and rock. Exposure to natural radiation in New Zealand is relatively low but measurements have not been taken in commercial buildings.

ASBESTOS

Asbestos is a natural fibrous mineral that is found in many parts of the world. It occurs in three main forms: chrysolite (white asbestos), amosite (brown asbestos) and crocidolite (blue asbestos). Asbestos is chemically strong and resistant to heat and chemical attack. It is commonly used as reinforcing for cement and plastics. Some of the more common uses in buildings is as a lagging for boilers and pipes, insulation and as asbestos cement roofing.

DISEASES ASSOCIATED WITH ASBESTOS

According to British Department of the Environment publication (1986), the principal diseases known to be caused by exposure to asbestos are asbestosis, lung cancer and malignant mesothelioma.

Asbestosis - fibrosis or scarring of the lung in which the tissue becomes less elastic making breathing progressively more difficult. The process is irreversible and may progress even after cessation of exposure. Asbestosis is an industrial (occupational) disease arising from exposure to high levels of airborne dust. According to the British DOE, there is no risk of contracting this disease from normal levels of environmental exposure.

Lung Cancer - an increased incidence of lung cancer has been found amongst people who work with asbestos. The increase in risk depends on the degree of exposure and is very much greater for smokers than for non-smokers. All three types of asbestos fibre can cause cancer, but crocidolite (blue asbestos) and amosite (brown asbestos) are thought to be more dangerous.

Mesothelioma - is a cancer of the inner lining of the chest and abdominal wall. The incidence in the general population is very low and most cases are attributable to working with asbestos. Crocidolite and probably amosite are much more likely to cause mesothelioma than chrysolite.

EXPOSURE RISK FACTORS

The risk of contracting an asbestos related disease depends on a number of recognised factors. These appear to include the cumulative dose to which an individual has been exposed (although there is a contrary argument - refer to the 'one-hit' theory), the time since the first exposure, and the type and size of the asbestos fibres.

The status quo is an assumption that the risk of cancer is proportional to total exposure, but according to Doll et al., (1985), the risk of mesothelioma is strongly related to the time since first exposure. They report a common latency period of 10 - 20 years between first exposure and the onset of symptoms for asbestos related diseases. In the case of lung cancer, the period of latency may be up to 40 years or more.

The fibre size and shape are thought to be important variables in determining risk. Longer fibres with a length of greater than 200 μm (1 μm = one millionth of a metre) are generally cleared from the nasal passages but shorter fibres with a diameter of less than about 2 μm may penetrate deep into the lungs. Doll et al., (1985) report as follows:

"Laboratory evidence suggests that the hazard is greatest with fibres between 5 and 10 μm in length and less than 1.5 and 2 μm in diameter. There are, however, no sharp boundaries between hazardous and non hazardous configurations. Short fibres less than 1 or 2 μm in length may not be hazardous at all; but there is no evidence of any minimum diameter to hazardous fibres which may be carcinogenic even when the diameter is so small that they cannot be seen by the optical microscope." (refer Appendix 7 Behaviour of Inhaled Particles).

One Hit v Multiple Exposure School

According Stoke (1991), the *'one hit hypothesis'*, assumes that one molecule of a carcinogen or one fibre can damage biological material and change a cell into a cancerous cell. This one cell then multiplies and eventually causes cancer.

The *'threshold hypothesis'*, on the other hand recognises the possibility of repair of lesions in the cells' DNA. It assumes the existence of a complex defence mechanism in the body which copes with individual deviate cells, such as cancer cells, which may spontaneously appear throughout a lifetime. Cancer can only develop when the defence system is overcome by large numbers of such cells created by a large number of *'hits'*. In other words, cancer can only occur when the *'threshold of tolerance'* is overstepped.

The results of epidemiological studies appears to support the *'threshold hypothesis'*, in that all of the studies to date suggest that at lower levels of exposure, no excess cancers have been found.

The argument is an important one in that, if the *'one hit hypothesis'* was accepted, nothing less than total removal of the product would be acceptable.

Public Exposure Risk

Murray (1990), claims that no single subject in the field of occupational health has elicited more emotional response than asbestos. He blames the media for sensationalising the issue and the legal profession and asbestos removal contractors for profiting from the general hysteria. Strong words, perhaps, but not entirely without foundation.

According to the British DOE report (1986), most of the health effects of exposure to asbestos has been derived from studies of workers occupationally exposed to asbestos fibres at concentrations many times higher than those encountered by the general public. Estimates of the risk of low level of exposure have been based on extrapolation from occupational exposure levels, and the range of uncertainty in such estimates is large. Murray (1990), claimed that the first extrapolation took place with the guidance notes to the Asbestos Regulations of 1969. The figures of British Occupational Hygiene Society with regard to asbestosis were related to mesothelioma without sound scientific basis. He claims that a factor of ten was effectively 'plucked from the air', because nobody really knew.

The DOE report states that the risk of mesothelioma is thought to increase rapidly with time since first exposure and it is therefore likely that children will be at more risk than adults from similar exposure. Smoking and asbestos (refer section on smoking) appear to act synergistically in causing lung cancer, the smokers have a much greater additional risk of contracting lung cancer than non-smokers similarly exposed.

Doll et al., (1985) reached the following conclusions:

> *"The review of published studies by the Ontario Royal Commission (1984) and measurements made in British buildings on behalf of the department of the Environment suggest that exposure to true asbestos fibres of regulated sizes within asbestos containing buildings is seldom more than 0.0005 rf/ml (ie. 0.5 regulated fibres/litre) above background (as seen by optical microscopy). Exposure at this level for a working week in an office for 20 years in adult life or 10 years or so at school, or to lower average levels for more prolonged times at home is calculated to produce a life-time risk of death of 1 in 100,000. If 20% of the population experience such exposure, this would imply that one death in a year was caused in the whole country"* (note: Britain's population 57.087 million).

These estimates were based on the assumption of a linear exposure-risk relationship at low levels of exposure with no threshold or 'safe' levels of exposure to asbestos.

Doll & Peto's risk estimates were based on exposure to chrysolite asbestos and they pointed out that the risk could be appreciably greater for exposure to crocidolite and possibly amosite. There was a lack of quantitative data on the biological effects of exposure to crocidolite and amosite, but the researchers estimated that the risk of lung cancer and mesothelioma resulting from exposure to crocidolite might be 6 to 10 times greater than the risk from similar exposure to chrysolite. They also pointed out that the risk from exposure to amosite will probably be less than this but point out that there is no scientific evidence on which to base a specific figure.

The United States National Research Council Committee on Non-occupational Health Risks of Asbestosiform Fibres (1984), calculated the following lifetime risks arising from continuous exposure to asbestos:

> *"If a person were to inhale air containing asbestos at an average of 0.0004 fibres/cm^3 (0.4 fibres/litre) throughout a 73-year lifetime, the committee's best estimate is that the lifetime risk of mesothelioma would be approximately nine in one million (range 0 to 350 per million, depending on assumptions regarding the relationship of dose to risk). The corresponding lifetime risk for lung cancer would be about 64 in a million for male smokers (range 0 to 290), 23 in a million for female smokers (range 0 to 110), and 6 and 3 in a million respectively for male and female non-smokers.*

The DOE report (1986), states that the risk arising from typical levels of exposure to asbestos in buildings found in the United Kingdom is very small, especially compared to other common risks such as accidents. Nevertheless, they state that there is no known threshold level for exposure to asbestos below which there is no risk. Therefore, it is advisable to reduce exposure to a minimum that is reasonably practicable.

Asbestos in Drinking Water

A study of asbestos in drinking water was carried out by the Water Research Centre in the United Kingdom (Conway et al., 1984). Results from 144 samples suggested that there were low levels of asbestos in water supplies. There was some evidence that asbestos-cement pipes can release fibres into the distribution system.

The Committee on Medical Aspects of the Contamination of Air Soil and Water was asked to advise on the implications of the previous findings. The Committee concluded that:

> *"The only potential risk from the presence of asbestos in drinking water has been suggested that is at all plausible, is that of certain forms of cancer. The committee has considered the substantial body of research findings relevant to this question; it has found no convincing evidence which indicates that the concentrations and forms of asbestos in drinking water (in the UK), including those derived from the use of asbestos-cement pipes according to current practice, represents a hazard to the health of the consumer. The information assessed by this Committee suggests that, if there is any carcinogenic risk to the consumer to asbestos in drinking water, it is of an extremely low order and is not detectable by methods currently available".*

BUILDING RISK ASSESSMENT

According to the DOE report, once as the presence of asbestos material within a building has been established, the potential for fibre release must be assessed so that appropriate remedial measures may be taken. Systems of assessment based on assigning weighted values to factors such as accessibility, friability, damage and the like are sometimes used.

Potential for Fibre Release

The potential for fibre release from an asbestos material is determined by three factors:

(1) *The type of material* - the composition of an unknown material can be established by bulk sampling. For general purposes of assessment, the types of asbestos material are listed in approximate order of ease of fibre release (ie. highest risk first):

- sprayed lagging;
- insulating boards, insulating blocks, and composite products;
- ropes, yarns and cloth;
- millboard, paper and paper products;
- asbestos-cement (AC) products;
- bitumen roofing felts, damp proof courses, semi-rigid asbestos-bitumen products and asbestos-bitumen coated metals;
- asbestos-paper backed vinyl flooring;
- unbacked vinyl flooring and floor tiles;
- textured coatings and paints containing asbestos;
- mastic sealants, putties and adhesives;
- asbestos reinforced PVC and plastics.

The hazard presented by these materials is related to their hardness or toughness and the ease with which fibres may be released. The performance of the material is, to some extent, determined by the type of asbestos from which it is manufactured. Products containing crocidolite and amosite tend to be more friable and decay with age.

(2) *Integrity of the material* - for a material to be classified as being in good condition it must be intact, not broken, cracked or fractured and not bearing evidence of abrasion.

(3) *Position of the material* - readily accessible material is likely to be vulnerable to damage arising from vandalism, impact by people, vehicles or objects. In some instances, subject to damage arising from maintenance and repair work. Other sources of damage are vermin (rats, mice, birds) and water, which is particularly damaging to pipe and boiler lagging and sprayed asbestos.

Remedial Measures and Management

Interestingly, the DOE's guiding principle is:

> *"asbestos materials which are sound, undamaged and not releasing dust should not be disturbed."*

According to Stoke (1991), removing asbestos may be more hazardous than leaving it alone in that interference with the material may increase the airborne fibre concentration.

The DOE report (1986), suggests the following remedial measures:

(a) leave the material in place without sealing and introduce a management system;
(b) leave the material in place, seal or enclose, and introduce a management system;
(c) remove and dispose of the asbestos.

(a) Managing the material is the suggested option when the material is sound and undamaged. It involves recording the location of the material, periodic inspection and notification of maintenance personnel.

(b) Sealing (encapsulation) requires application of some form of coating. Asbestos materials may also be enclosed with sheet material sealed at the corners and edges.

(c) Removal is only an option when it is not possible to seal the material effectively and it is likely to release dust. Removal may lead to a short term elevation of dust levels. It is normally necessary to evacuate the building and monitor dust levels prior to the building being re-occupied.

Risks Associated with Man-Made-Mineral Fibres (MMMF)

A group of substitute materials for asbestos are mineral fibres. Saracci., (1985), offers epidemiological evidence to support that MMMF may have been a cause of lung cancer amongst workers involved in early slag wool/rock wool production. Other large studies by Enterline et al., (1984) and Simonato et al., (1986), showed some excess risk of cancer amongst rock wool/slag wool production workers.

A World Health Organisation meeting in 1986 reached the following conclusions:

> (1) An increased lung cancer risk has been documented in the production and maintenance workers employed in the early days of the rock/slag wool and glass wool industry. The increase amounts to a total excess of some 50% above expected

numbers during the period beyond 30 years after first employment. Although proportionally small, the added risk is numerically substantial because lung cancer is a common cause of death.

(2) The risk has been greater in the rock/slag wool sector of the industry than in the glass wool sector. No increased risk has been found after full introduction of dust suppressing technology.

(3) The increased risk may not necessarily have been caused by the fibres alone because documented carcinogens occurred in the factory environment, especially during the early production years, and contributed to the hazard.

(4) In the absence of a detailed dose-response relationships, the lung cancer risks associated with various degrees of fibre exposure cannot be estimated. However, current exposures to mean levels of 0.2 respirable fibres per ml or less seem unlikely to cause any detectable excess in lung cancer rates.

(5) It seems unlikely that exposure to MMMF, to the extent that has been prevalent in the industry over the past 30 years, can have caused any clinically material damage to the respiratory system, other than cancer.

(6) No other significant hazard has been demonstrated.

KEYPOINTS: The problems associated with asbestos are exaggerated and it has become a media and political football. Asbestos related diseases are more likely to be contracted in an occupational setting. Removal of asbestos from buildings is likely to increase rather than decrease the hazard, management and/or encapsulation may be preferable to removal. There is no single substitute for asbestos because of its unique chemical and mechanical properties. Research into the biological effects of man-made-mineral fibres (MMMF), noted that there was no clear evidence of increased mortality from cancer or other diseases associated with occupational exposure to MMMF in a manufacturing plant. Nevertheless, MMMF has not received an entirely clean bill of health.

ELECTRO-MAGNETIC FIELDS (EMF)

POSSIBLE ACUTE IMPLICATIONS OF EMF

EMF have been associated with adverse reproductive outcomes such as spontaneous abortions. Clusters of such adverse outcomes have led to several investigations. X-ray, ultra violet, visible and infra-red frequency, and micro-wave and radio wave frequency radiation have been measured and found to be less than existing standards (Cox, 1985). Some animal studies indicate that magnetic fields may disturb embryonic development (Delgado et al., 1985). Weak fields have been measured around VDUs, but these were considerably less than existing standards. Most of the 'adverse reproductive outcome' clusters have been attributed to a statistical phenomenon called *'expected unexpected clustering'*. Several studies were unable to document a reproductive hazard (Ericson et al., 1986; Kurppa et al., 1984).

BACKGROUND

Electromagnetic radiation is a form of energy that can be transmitted across a vacuum. It ranges from high frequency (short wave), ionizing radiation (eg. X-rays & gamma-rays), to lower frequency (longer wavelength), non-ionizing radiation. Non-ionizing radiation includes ultra-violet, visible light, infra-red, microwave, television and radio transmissions. Below this is the extremely low frequency (ELF) range which extends from a frequency of zero to 300 Hertz (Hz), and includes the electromagnetic fields generated by alternating currents. The sources of these currents include the electrical transmission grid itself and directly from electrically powered equipment. Electricity in New Zealand is transmitted at a frequency of 50 Hz.

There are substantially different physical properties and biological implications as frequency increases from 0 Hz (static fields, exemplified by the Earth's own magnetic field), through the ELF range of frequencies, radio and television transmission (in the range 10^5 - 5 X 10^8 Hz), radar and microwaves (range 5 X 10^8-10^{11} Hz), visible light (around 10^{15} hz), and ionizing radiation (about 10^{16} Hz and higher). The distinctive biophysical effects of ionizing radiation (which has sufficient energy to break chemical bonds) and non-ionizing radiation are well recognised, but there are different effects from different frequencies within the range of non-ionizing radiation as well (Savitz et al., 1989).

UNITS OF MEASUREMENT

Electric field strength is measured in units of volts or kilovolts per meter. Magnetic fields in air and biologic materials can be measured using either the magnetic field strength (amperes per meter (A/m)) or, more commonly, the magnetic flux density (units of gauss (G) or telsa (T)). For exposures of interest, the unit of milligauss (mG) is more practical, with 1mG equal to 0.1 microtelsa (μT). In all environments lacking magnetic properties, a field strength of 1 A/m can be translated into a magnetic flux density of 12.6 mG.

DISTINCTION BETWEEN ELECTRIC & MAGNETIC FIELDS

The difference between electric and magnetic fields is worthy of note. Electrical fields are produced by an electrical charge on the surface of a conductor, the strength being related to the conductor's voltage. Magnetic fields are present only when an electrical current flows.

As an illustration of the distinction, an electric blanket can generate electric fields even when turned off (and is plugged in), whereas it can generate magnetic fields only when it is drawing current to produce heat.

Experimental and epidemiologic evidence suggest that electrical and magnetic fields may produce distinct biologic effects. Both kinds of fields share the ability to induce currents in biological tissue, but may have independent actions as well (Carstensen, 1987). A primary difference between the fields is their susceptibility to shielding. Virtually all electrically conductive materials (buildings, trees, humans) distort and shield electric fields, whereas all but a few materials are transparent to magnetic fields. In other words, electric fields are sharply attenuated by buildings and other structures while magnetic fields can easily penetrate buildings and human tissue. For this reason, residential exposures to electric fields are not predicted by external power lines. Therefore, studies of the effects of residential exposure from external power lines are more reasonably interpreted as reflecting the effects of magnetic rather than electric fields (Savitz, 1987; Kaune et al., 1987).

Sources of Electromagnetic Fields

All human beings are exposed to a wide range of electromagnetic fields, both natural and artificial (Stuchly, 1986; Aldrich et al., 1985). The main naturally occurring magnetic field is the earth's static field. It has a field strength of about 0.5 Gauss. Time varying magnetic fields also exist, but at frequencies associated with the transmission of electricity, these natural fields are very weak. At 50 Hz, the natural magnetic field is between 10^{-9} and 10^{-8} Gauss, compared to 10^{-4} to 10^{-2} Gauss or more associated with artificially created magnetic fields, a difference of several orders of magnitude (Polk, 1974).

Electric power lines are but one of many sources of exposure. Many electric power lines produce localized fields of greater strength. According to Savitz et al., (1989), the most important sources for human exposure are difficult to define at this time for the following reasons:

> (a) the prevalence of appliance use and the distributions of residential and occupational exposures from these sources are still largely unknown; and
> (b) if prolonged exposure is the basis for purported health effects, the duration of exposure from these different sources would be of paramount importance. For example, hair dryers, which are used only briefly, would be of less concern than electric blankets, which are used over prolonged periods.

These multiple sources of exposure have several implications for epidemiologic studies. Although a completely unexposed group does not exist in industrialised societies, gradients in degree of exposure can be examined. Several individual sources of exposure, each of which provides a sizable contribution to the total, are suitable for epidemiologic study. Residential, occupational, and appliance-based electromagnetic fields are all potentially important contributors to total *'dose'*, although their relative contributions are unknown at the present time. Small children and housebound adults would tend to receive more of their total exposure from the home. Because of these multiple sources, there is probably sufficient identifiable exposure variation in low-level field exposure within most populations to allow for examination of health consequences (Kaune et al., 1987; Stuchly, 1986).

CANCER AND ELECTRO-MAGNETIC FIELDS (REFER APPENDIX 10)

KEYPOINTS: It has been hypothesised that EMF may have acute effects with regard to adverse reproductive outcomes. These remain unproven at this stage. A number of literature reviews concerning the possible role of electromagnetic fields in the etiology of cancer have drawn similar conclusions (Coleman et al., 1988; Aldrich et al., 1987; Ahlbom, 1988). There have been a number of suggestions that prolonged magnetic field exposure in residences and occupational exposure to these fields may increase the risk of leukaemia and brain cancer. Nonetheless, these studies seem to fall short of providing conclusive indications for a causal association (Savitz et al., 1989). The internally generated magnetic field strengths in commercial buildings remains to be investigated, particularly the emanations from the ubiquitous Video Display Units.

CHAPTER 4

HYPOTHESIS

INTRODUCTION

An objective of this study was to develop and trial a procedure to answer the question:

> *"Do the occupants of the building under investigation report an abnormal level of specific symptoms."*

Whilst an affirmative answer to the above does not answer the question: *"Is this building sick?"*, it provides *prima fasciae* evidence that a more thorough investigation is warranted. Justification for this statement may be found in the definition of a 'sick building', which is one in which the occupants suffer certain physical symptoms at greater frequency than would be expected from similar cohorts within the general community.

An extensive survey of the literature failed to identify other studies attempting to answer the fundamental question: *"Is this building sick"*.

There are numerous reports of comprehensive and expensive building diagnostic investigations having been undertaken, which failed to uncover any evidence of a 'building related' problem.

It is suggested that some of these diagnostic exercises may have been premature without stronger evidence of a problem. Subjective complaint from building occupants is probably sufficient cause to make an initial investigation but, arguably, it is not sufficient to trigger a thorough and expensive audit.

This study attempts to develop a procedure which bridges the gap between the initial complaint phase and the comprehensive building audit.

To answer the question, as posed it must be established that the occupants of the building under survey are experiencing an 'extraordinary' level of symptoms.

Simple comparison of reported symptom frequencies between occupants of different buildings is complicated by job, psychological and other factors. There is an extensive body of literature discussing the tendency of certain groups to report more physical symptoms than others. The tendency to report physical symptoms seems to be related to gender, marital status, socio-economic status, job type, age and so on.

The reasons why different groups report more or less symptoms than others is not fully understood. However, it is suggested that recognition of this tendency is central to the development of a procedure to achieve the stated objective.

It is known, for instance that groups like VDU operators complain of symptoms more frequently than managers. It is difficult to establish whether the symptoms being reported are job related or building related. It is logical to assume that some symptoms are building related but, probably not all. Thus, to examine whether VDU operators are suffering an extraordinary level of symptoms it makes sense to compare them to other VDU operators occupying other buildings.

HYPOTHESIS

It is hypothesised that:

> *"if a sufficiently large group of randomly selected people from a variety of buildings can be surveyed, some average of 'symptom prevalence' will be established. If a representative group of the cohorts from a building under survey are compared with this 'average group', it will give an indication whether the occupants of that particular building are experiencing 'an above average' level of symptoms".*

This will answer the question as posed:

> *"Do the occupants of the building under investigation report an abnormal level of specific symptoms."*

It should be noted that, the procedure does not directly establish that a particular building is sick, as building sickness tends to be relative rather than absolute. Rather it gives an indication of excess symptoms.

Further, there is no suggestion that if a building is better than average, no further attempts should be made to improve environmental conditions.

Other, equally valid benchmarks might be suggested, for example the adoption of the symptom average from a building *'believed'* to be healthy.

Finally, a 'failing grade' from the proposed model is not a definitive statement of a building related problem, rather it is *prima fasciae* evidence that a more thorough audit should be undertaken.

CHAPTER 5

LITERATURE REVIEW - SURVEY DESIGN

APPROACHES TO THE INVESTIGATION OF BRI

A number of approaches have been adopted to investigate building related epidemics. The two major approaches are the industrial hygiene and the epidemiologic approach.

The industrial model described by Bernstein et al., (1983) has been found to be of limited use. This is not surprising as, in general, hazardous substances and chemicals found in an office setting are lower than those found in an occupational setting and certainly less than the allowable 'threshold limit values'. Nevertheless measurements of key chemical indicators can give important clues to the various environmental factors. Taylor (1980), and Salisbury et al., (1982) have shown that carbon dioxide, innocuous in its own right, was a good indicator of ventilation efficiency and air quality. Hollowell et al., (1982) successfully used gas chromatography to identify organic compounds of concern in a building under investigation. Nevertheless, standards designed to protect workers in occupational settings from single chemicals may well be inadequate in work environments with numerous low-level exposures which may act additively or synergistically.

The second major investigative approach is epidemiologic. Epidemiology is the study of health and disease in populations. According to Walsh et al., (1984), for persons who have seen physicians, a medical record review may provide objective information. McLellan (1983), suggests that a deeper appreciation of office work will come as more sensitive epidemiological markers of disfunction are used, (eg., turnover rates, absenteeism, incidence of stress related disease and syndromes.)

The epidemiologic approach is the most commonly used method in relation to the investigation of acute syndromes, the industrial hygiene approach being more appropriate in respect of the chronic stressors.

Referring back to the definition of 'abnormal or extraordinary' level of symptoms as being *'a disproportionate level over and above that which would be expected from similar cohorts within the general community'*. The epidemiologic or disease oriented investigation of BRI is therefore relative rather than absolute. The investigator is seeking to identify 'abnormal clusters' of specific symptoms.

REVIEW OF SURVEY METHODS

A number of approaches to diagnosing Building Related problems have been used by various researchers. Broadly, the methods which have been used or suggested can be categorised as follows:

(1) **Questionnaire survey**. Raw (1992), reports that a questionnaire survey of the building is a standard diagnostic tool. The questionnaire is valuable as a preliminary to remedial measures, to establish the nature and number of complaints, whether the complaints come

from throughout the building or only certain parts, and what may be causing them. He suggests that the questionnaire may also provide a means of determining whether any subsequent remedial measures have been effective.

A major limiting factor with the questionnaire survey and face-to-face interviews is that they may not be valid if there is a change in the individuals or organisations occupying the building.

(2) **Face-to-face interviews**. Raw (1992), suggests that the face-to-face interview is as valid as the questionnaire option. However, it is more costly and time consuming. Wilson et al., (1987), report that higher prevalences of symptoms are typically reported in questionnaire surveys than in face-to-face interviews. The response rate from telephone surveys is not reported.

(3) **Medical records**. Burge (1990), recommends careful study of medical reports for identifying symptoms, accompanied by a visual inspection of the building. Medical records and/or walk-through inspection may reveal information requiring follow-up sampling. Walsh et al., (1984) express a similar opinion and suggests that a review of medical records may provide objective information.

(4) **Physical sampling.** Gammage et al., (1989), report a number of empirical guideline figures for screening purposes. These include 1000 ppm of carbon dioxide to screen for outdoor air supply, 500 colony forming units per cubic meter of micro-organisms to screen for sanitary conditions and 1 ppm of VOCs to screen for active sources of chemicals.

Burge (1990), states that the first rule of thumb of sampling is *'avoid it'*. The problems with sampling, especially air sampling, are multiple. Sampling for bio-aerosols is particularly complicated and different equipment will produce different results. Even if sampling is done properly, there are no quantitative baseline values or recommended threshold limits for airborne microorganisms. The levels of organic and inorganic gases which may cause minor irritation is also unknown and is not an issue contemplated in setting threshold limit values.

(5) **Independent observation.** Raw (1992), discusses a theory in which it was proposed to use a panel of independent observers to occupy a building for some time and report their symptoms. Unfortunately, they might have to spend 6-12 months in a building before giving an accurate report. Using sensitive individuals ('human canaries') who would react more quickly might overcome time lag problems, but there may be ethical difficulties.

(6) **Interventional studies**. A number of researchers have undertaken interventional studies. These, typically are related to expert concern about some aspect of the internal environment. For example, micro-biological growth on cooling pans, air handling units, drainage pans and the like. However, interventional studies are unlikely to be undertaken without symptomatic concern. It is highly improbable that an interventional study would be a first step in a *building* diagnostic investigation.

QUESTIONNAIRE DESIGN

Questionnaires have been used extensively to diagnose BRI. There has been no attempt to standardise these questionnaires. A number of research groups are using different scales and, in some cases, canvassing different symptoms. In order to remedy this, the CIBS Task Group has been set up in the U.K. to design a questionnaire which will be valid internationally.

The Task Group has examined and compared four major questionnaires. Their analysis of the scales, time periods used, and the symptoms included provide some guidelines for questionnaire development. The following questionnaires were examined:

- The Danish Town Hall Study Questionnaire (DICSW)
- Agricultural University of Wageningen (AUW)
- Building Use Studies Ltd (BUS)
- MM-Questionnaire (MM).

SYMPTOMS INCLUDED

According to Raw (1989), the symptoms vary among the questionnaires and appear to be dependent on the specialist interests of the investigators. For instance MM concentrates on skin symptoms whereas AUW is more interested in respiratory disorders. According to the same author, a standard questionnaire should include the symptoms common to all the questionnaires but leave scope for individual researchers to include further questions.

The symptoms which, by definition, should be included are referred to in Table 2.

In addition, Hedge (1990), compared eight questionnaires used in major international studies of 'sick' buildings. The comparison of the main symptoms solicited were presented in tabular form as follows:

TABLE 16 - QUESTIONS ASKED IN 8 MAJOR QUESTIONNAIRES

SYMPTOMS		QUESTIONNAIRES							
		A	B	C	D	E	F	G	H
EYES:	Dry	*	*			*	*	*	*
	Itching/tearing	*	*		*	*	*	*	
	Strained			*		*			
	Focusing difficulty			*		*			*
	Irritation/redness			*		*			*
	Burning				*		*		*
NOSE:	Stuffy/congested	*	*	*		*	*	*	*
	Runny	*	*		*	*	*	*	*
	Irritated/itchy			*				*	
	Sneezing					*	*		
	Nosebleeds								*
THROAT:	Dry	*	*		*	*		*	*
	Sore/irritated			*	*	*	*	*	*
	Scratchy								*
	Cough				*	*	*		*
	Hoarseness				*	*		*	
SOMATIC:	Headache	*	*	*	*	*	*	*	*
	Lethargy/tiredness	*	*		*	*	*	*	*
	Sleepiness			*		*			*
	Fatigue			*		*			
	Concentration difficulty			*	*	*		*	*
	Memory difficulty					*		*	
	Dizziness/faintness			*		*			*
	Nausea			*	*	*			
FLU-LIKE:	Chills					*			*
	Flu-like	*	*	*		*		*	
	Fever			*		*			*
	Weakness			*			*		*
	Shivering				*				
CHEST:	Breathing difficulty	*	*	*	*	*	*	*	*
	Chest tightness	*	*	*		*		*	*
	Wheezing			*		*			*
SKIN:	Dry			*	*	*	*	*	*
	Itching			*		*	*		*
	Rash				*		*	*	*
	Blotches						*		*
	Flaking								*
	Number of Symptoms	10	10	20	15	28	16	17	27

QUESTIONNAIRES COMPARED:

A:U.K. Office Environment Survey 1
B:U.K. Office Environment Survey 2
C:Office Work environment Survey
D:Danish Town Hall Study Survey
E:Environmental Protection Agency
F:Technical University/Institute Helsinki
G:Building Research Establishment
H:Indoor Air Quality Questionnaire

While the number of questions in each category differ, they all solicit symptoms in the same generic symptom groups. In spite of the variety of questions, there is clear consistency in the syndrome being described. The World Health Organisation (WHO, 1986), defined SBS as manifesting the following symptoms:

-irritated, dry or watering eyes (sometimes described as itching, tiredness, smarting, redness, burning, difficulty wearing contact lenses);

-irritated, runny or blocked nose (sometimes described as congestion, nosebleeds, itchy or stuffy nose);

-dry or sore throat (sometimes described as irritation, oropharyngeal symptoms, upper airway irritation, difficulty in swallowing);

-dryness, itching or irritation of the skin, occasionally with rash (or specific clinical terms such as erythema, rosacea, urticaria, pruritus, xeroderma; sometimes assessed by use of moisturiser or lipstick);

-less specific syndromes such as headache, lethargy, irritability and poor concentration.

SBS, is considered to be limited to these symptoms. A number of the above major studies, in particular the U.K. Office Environment Survey (1 & 2), the Office Work Environment Study and the Danish Town Hall Study, have been referred to by their authors as studies into the Sick Building Syndrome. In fact they are not. Inclusion of questions on flu-like symptoms and chest (pulmonary symptoms), canvasses a wider range of symptoms than, just, SBS. In fact all eight questionnaires canvass BRI symptoms as earlier defined.

MEASUREMENT AND SCALE SELECTION

According to Pennebaker (1982), symptom reporting can be measured in dozens of ways. For example, one can simply ask the subject to what degree he or she is currently experiencing such symptoms as a racing heart or a headache. Although, such a symptom checklist is more apposite as a state measure, it can also be used to tap the trait of, symptom reporting. This is because summing the items on such a scale yields a total symptom scale that is *internally consistent.* In addition to checklists that seek to learn how the subject is feeling at a given point in time, a number of symptom inventories have been devised that tap the frequency of occurrence of a number of physiological and psychological symptoms. Although some of these scales specify a time frame (eg. within the last year, month) many do not. Scales of this nature include the Cornell Medical Index (Abramson et al, 1965), the Hopkins Symptom

Checklist (Derogatis et al., 1942) and the Somatic Perception Questionnaire (Shields et al., 1979).

An interesting quality of each of these scales is that each is particularly consistent, meaning that a person who reports any one particular symptom is likely to report others. The tendency to report symptoms, then, can be viewed as a stable unidimensional construct.

Pennebaker suggests that a simple 7-point bi-polar scale, is appropriate for this type of 'medical survey', for example:

Right at this moment, I am experiencing:

---- ---- ---- ---- ---- ---- ----
No headache Headache

---- ---- ---- ---- ---- ---- ----
No cold hands Cold hands

In scoring this type of scale, the total symptom score can be obtained by summing all of the symptom items together. The mean score can be used to develop an index. The Office Environment Survey in the U.K. (Wilson, et al., 1986), used this technique. The standard questions about dry eyes, throat, headache and the like were canvassed. From the responses, the person symptom index and the building symptom index were derived. The person symptom index was the number of building-related symptoms reported by individuals in the study. The building symptom index was the average reported level of symptoms for each building.

Hedge et al., (1989) used the same technique in the major office health survey in Great Britain. In this particular survey, for three of the scales, the poles were reversed to counteract any response-set bias.

Selection of an appropriate scale is an important issue for two main reasons:

(a) Analysis - the scale chosen determines the appropriate statistical procedure.
(b) Clarity - inappropriate intervals in scales can confuse respondents.

(a) Scales typically consist of ordinal or interval variables (Aday, 1989). Ordinal variables permit ranking or ordering of responses. Ordinal measurements assume an underlying continuum along which respondents can be ranked on a characteristic of interest from high to low, excellent to poor, and so on. Ordinal scales make no assumptions about the precise distances between the points along the continuum.

Interval or ratio scales assume that the underlying quantitative continuum on which the study variable is based has intervals of equal length or distance, similar to the divisions of a ruler. The main difference between interval and ratio scales is the absence of an absolute zero point. Ratio scales have an absolute zero point while the former do not. Examples of interval scales are measures of intelligence or temperature.

The importance of the distinction between the types of scales are the appropriate statistical tests which can be used to analyze the results. Interval scales allow the use of parametric statistical tests while ordinal scales require the use of less powerful non-parametric tests.

(b) In analyzing the scales used in the four major questionnaires, Raw et al., (1989) makes the following comments.

MM asks if the symptoms occur 'often', 'sometimes' or 'never'. The scale is possibly too open to interpretation and does not offer the respondent any time scale for guidelines.

BUS used the scale 'daily', 'most days', 'most weeks', 'most months', 'less often'. The last category does not seem to follow on from the previous ones, but would probably not cause problems for most respondents.

AUW used the scale 'nearly daily', 'weekly', 'now and then', 'never'. The main comment about the use of 'never' in an ordinal scale is that a substantive zero point does not exist (Aday, 1989).

DICSG uses the scale 'daily', 'a couple of times per week', 'a couple of times per month' 'never/rarely'.

None of the scales uses completely exclusive categories, and some of the categories may be ambiguous. As a result, some respondents may have difficulty knowing how to report frequencies. For example, how would three times per week be coded, or once every few weeks.

Symptom Frequency - Severity

None of the four major questionnaires gave any guidance as to how severe symptoms should be before they are reported. Instead, the severity of BRI was judged by the number and frequency of symptoms. The validity of this is implicitly assumed rather than proved. According to Raw et al., (1989), it may not be practical to rate severity in addition to frequency, but there is scope for empirical testing here.

Time Period of Symptoms Canvassed

The time period for which the respondent is required to report symptoms needs to be considered. BUS and AUW ask respondents to use a 12 month recall period. According to Raw et al., (1989), using a yearly time period has an advantage in that it reduces seasonal effects, but the disadvantage is that the respondent is unlikely to have perfect recall over this period.

MM asks if they have had symptoms over the last three months.

DICSG consists of three questionnaires which relate to morning, afternoon and winter symptoms. The design enables the researcher to specify the time period. This has been criticised on the basis that it lacks consistency.

Aday, (1989) states that the time period canvassed in 'health surveys' is important in that it may lead to under reporting or over reporting errors. Over reporting arises when the

respondent 'telescopes' or includes events from outside the time period being asked. Under reporting occurs when the respondent omits events that should have been included. Under reporting is a function of time and increases as a function of the time horizon.

Relating Symptoms to Work

The symptoms of interest are common amongst the general population at unknown incidence. Clearly, proving that the physical building structure is the causative factor is central to the exercise. Of the four questionnaires examined, each included a question requesting this information. The questions asked were as follows:

DICSG *"Have you this winter suffered discomfort as a result of symptoms which you feel are due to the climate inside (these) offices"*

AUW *"Do these complaints normally diminish or disappear at home?"*

BUS *"... was this better on days away from the office?"*

MM *"Do you believe that (this symptom) is due to your work environment?"*

In the case of DICSG, the question is used as a filter. Respondents who replied "yes" were then asked "what symptoms". The other questionnaires determine first what symptoms are experienced at work, then whether each symptom experienced at work is reduced when away from work.

Background Information

Age, gender, job type, work place, and smoking habits were recorded by most questionnaires. Least popular of the questions asked were: the amount of tea and coffee drunk; wearing glasses/contact lenses; and, level of education. The AUW and MM asked about health background (eg. history of allergies). At least one U.K. questionnaire included questions on alcohol during the working day.

Raw et al., (1989) recommended the inclusion of questions on conditions such as asthma and hay fever.

Questionnaire Length

There is a large body of literature dealing with the length of questionnaires. Respondent fatigue is a commonly observed problem. When this is encountered, the validity or truth of the responses must come into question.

Generally, interviewer-administered questionnaires can be longer than self-administered ones.

Dillman (1978), recommends that self administered questionnaires should be no longer than twelve pages.

For topics not especially salient to respondents the questionnaire should be much shorter. Sudman et al., (1982), recommended that they be no more than two to four pages in length.

Aday, (1989), states that questionnaires can be longer if the respondents are aware that the interview is going to take place. She calls these 'warm' calls. Cold calling, on the other hand, suggests that brevity needs to be stressed.

MANAGEMENT OF THE SURVEYS

DELIVERY METHOD

The delivery method has not been an issue in the other reported surveys. All have consisted of either face-to-face interview or self-completed questionnaire. Consideration of delivery mode, mail survey, personal interview or telephone survey did not have to be addressed.

SAMPLING FRAME

Selection of a representative group from within a building is reasonably well established. Benard (1988), points out that a high response rate is necessary otherwise interpretation of the results becomes difficult.

Gamble et al., and Hedge et al., (1989), recommend that the target sample size at each office site should be a minimum of 50 workers. To achieve this target, workers were sampled using a probability proportional to size design in which the sample fraction was varied as a function of the total number of occupants in an organisation in a building (Moser et al., 1971).

Hedge et al., (1989) reported a response rate approaching 92%. Their survey was based on the sample fraction for each site with every n^{th} workstation being surveyed. Workers were excluded from the sample if they were on vacation, on maternity leave, worked part time, were temporary staff, or if they worked in the building for less than one month. Workers absent due to illness or who were away from the building for other reasons were included in the sample; for these respondents, a questionnaire and explanatory letter were left for subsequent collection. All the questionnaires were individually distributed and collected on the same day, at which time they were checked for completeness. Incomplete documents were followed up with the respondents at this point.

To obtain an unbiased sample of workers from each building Hedge et al., (1989) used the following methodology. If the building contained fewer than 120 workers, all were sampled. If more were present a sample was taken to include 70-100 workers. In some buildings this was done by studying workers on alternate floors, and in other buildings by sampling at an interval of every 2-10 work stations, taking care to obtain a sample across the depth of the building and between the faces of the building.

SAMPLE BIAS

Gamble et al., (1988) indicates that reporting bias in this type of survey is most likely to occur in two forms.

(1) It can occur on an investigation of a building in response to a complaint. There is already a perceived problem (by somebody), and thus there is the probability of an increase in the occurrence of complaints reporting.

(2) A second form of reporting bias can be characterized as epidemic psychogenic illness. Symptoms in this instance occur after learning of either a suspected exposure or the fact that somebody is ill (Boxer et al., 1984). Many of the complaints of office workers are similar to stress reactions and the impact of psychosocial factors in the office environment (Baker, 1984)

In a private communication Robertson (1993), suggested that the *Hawthorne Effect* may be a problem in that the respondents may be conditioned into giving certain responses because of the presence of an investigator.

However, Wilson et al., (1987), report lower symptom reporting in personal interviews. This may go someway towards overcoming some of the 'Hawthorne Effect'.

QUESTIONNAIRE ANALYSIS

The tendency to report symptoms appears to be influenced by a large number of variables including age, gender and job description. Raw (1992), suggests that the most simple model is that the environment causes symptoms and the reporting of those symptoms is dependent on psychological factors. Nevertheless, this phenomenon complicates analysis of these questionnaires. For example, the situation described by Hedge (1986), two groups of people sharing identical environmental conditions reported very different symptom rates. The people with low job satisfaction, mainly female clerical workers, reporting a far higher prevalence of symptoms. If such symptoms were not 'building related', were they 'job related' or 'psychosomatic'? Similar confounding factors are common place throughout these surveys.

Pennebaker (1982), defines a symptom as; *"a perception, feeling or even a belief about the state of our body."* The sensation is often, but not always, based on physiological activity. Above all, a physical symptom or sensation represents information about internal state. Sensations can be both causes and effects of behaviour. They often initiate activity as well as signal that an activity has occurred. In effect, the processes of noticing and reporting symptoms are highly adaptive and functional for an individual.

Symptom reporting is subject to a large number of perceptual biases and distortions. Thus it can be stated that physical symptoms are private and subjective experiences. A basic tenet of psychophysics states that *"equal stimulus ratios produce equal sensation ratios"* (Stevens, 1975). According to Pennebaker, this approach which is based on the assumption that all other variables are constant, is rarely met in the 'real world'. Indeed, it may indicate why two individuals exposed to the same stimuli may be affected differently.

DEMOGRAPHIC AND CULTURAL CUES TO SYMPTOM REPORTING

The prevalence of symptom reporting may vary with age, sex, martial status, socioeconomic, racial and cultural differences. In the United States, the National Centre for Health Statistics have gathered a large amount of data on this phenomenon.

Age. Among national surveys in the United States, older individuals report more symptoms than younger (NCHS, 1970). The pattern is consistent for physician visits (NCHS, 1979b), weekly aspirin use (NCHS, 1979c), and days of restricted activity due to illness (NCHS, 1979a). Within college samples, however, the trend is reversed. First-year college students report the greatest number of symptoms and final-year students the least (Comstock et al., 1973; Greenley et al., 1976; Moos et al., 1977).

Using increased/decreased productivity as a surrogate for the debilitating effects of building related illness Raw et al., (1989), reported that age had an irregular effect on productivity. There was a decrease in productivity in the 20-35 age group. Those over 35 'self reported' an increased level of productivity. The reasons for this could only be a matter of speculation, for instance the improvement in the over 35 group may be a result of maturity or improved workskills.

Job Status. Burge et al., (1987), indicated that the effect of age was more complex with those aged between 21-40 reporting more symptoms than either younger or older workers. The greater proportion of older workers being male may have explained some of the improvement in this age group (Refer to Gender).

Gender. Virtually all studies indicate that females report more symptoms than males. Similarly, women tend to have higher rates for aspirin use (NCHS, 1979c), physician visits (NCHS, 1979b; 1980a), days of restricted activity due to illness (NCHS, 1979a), and expenditures for both prescribed and nonprescribed medication (NCHS, 1967). These data are consistent across all ages with the exception of people under 15 years of age.

Comparable sex differences emerge among college samples employing retrospective symptom reports (Comstock et al., 1977; Pennebaker et al., 1977). An intriguing finding, however, is that when asked to report the degree to which they are *currently* experiencing 12 common symptom items, no consistent sex differences emerge (Pennebaker et al., 1978).

Wilson et al., (1986) and Raw, (1989) both indicate that women typically report more building related symptoms than men but there is no overall difference in productivity as a result. Skov et al., (1989), Nelson et al., (1991) and Burge et al., (1987), all concur that females tend to report a significantly higher number of symptoms than males.

Marital Status. The NCHS national surveys and college samples have consistently found that unmarried persons report more symptoms, take more aspirin, and perceive themselves to be in worse health than married. This finding is especially pronounced for individuals who have been formerly married (divorced, separated, widowed). Interestingly, those who never married tend to be more similar to married than formerly married respondents (NCHS, 1970; 1979a; 1979b; 1979c; Wan, 1976).

Occupational Status. Whether or not a person is employed, keeping house (or otherwise not in the labour force), or currently unemployed is also related to perceived symptoms, poor health, and aspirin use. Individuals who are employed report fewest symptoms. However, individuals who are currently unemployed but looking for work report being in better health and than those not in the labour force (NCHS, 1970; 1979c; Wan, 1976).

Burge et al., (1987), indicated that clerical and secretarial workers reported more symptoms than professional or technical workers who in turn, reported more symptoms than managers. According to this group of researchers, the reason why managers and professionals have fewer symptoms than secretarial or clerical workers is not clear, but it may be related to their better accommodation within buildings or to a greater degree of control over their job or their greater ability to have changes made to the running of the air conditioning system.

Chamberlain (1993), in a private communication suggested that the lower prevalence of symptoms amongst the management group may be related to *'executive hardiness'* which is a sort of organizational natural selection process.

Hedge et al., (1989), reported that those using V.D.U.s for more than six hours per day reported more job stress than those using the devices for less than six hours per day.

Socioeconomic Status. Indicators of socioeconomic status (ie. income & education) have a close linear relationship to symptoms and health. The higher the socioeconomic status related to fewer symptom reports (NCHS, 1970), perception of better health (Wan, 1976), and less aspirin use (NCHS, 1979c). However, higher SES individuals visit physicians more regularly (NCHS, 1979b). The latter finding may be related to the economic cost of access.

Racial and Subcultural Differences. In the United States, the majority of studies indicate that the American Africans report symptoms to a higher degree (Wan, 1976), report more days of restricted activity due to illness (NCHS, 1979a), and take more aspirin (NCHS, 1979c) than caucasians. As with the SES data, however, the latter visit physicians more often than the former (NCHS, 1979b; 1981a).

Among the American samples, large differences in symptom reporting and responses to pain have been found among ethnic groups. Zola (1966), for example, found that Irish-Americans were more likely to seek medical attention than Italian-Americans. Further, the symptoms for which they sought medical attention were different. Similarly, large differences in response to pain has been found between American-born Jews, Italians, Irish and Americans of British descent (Sternbach et al., 1965; Tursky et al., 1967; Zborowski, 1969).

Cross-cultural Differences. Anthropological studies indicate a tremendous variability in symptom reporting from culture to culture. Raper (1958) initially assumed that peptic ulcers were a rare occurrence among members of the African tribe he was studying, since tribal members never noted or complained of accompanying symptoms. Later autopsies revealed that peptic ulcers were present in the tribe in virtually the same proportions as in England. Apparently the African tribesmen were perceiving their internal sensations in different ways to their Anglo-Saxon counterparts. An interesting corollary is that Israeli immigrants from different cultures differ radically as regards the types of complaints for which they take their infants to hospital (Harlap et al., 1975).

Statistical Tests Used

Pennebaker (1982), reports that internal consistency of such medical questionnaires can be measured by examining the mean inter-symptom correlations to test whether a person reporting one symptom is likely to report others. A measure such as the Cronbach alpha coefficient could be used to test reliability across samples.

Croenbach's alpha measures the internal consistency of the scale. It is based on the internal consistency of a test (Norušis, 1990). That is, it is based on the average correlation of items within a test, if the items are standardised to a standard deviation of 1; or on the average covariance among items on a scale, if the items are not standardised. It is assumed that the items on a scale are positively correlated with each other because they are measuring, to a certain extent, a common entity.

Croenbach's alpha has several interpretations. It can be viewed as the correlation between this test or scale and all other possible tests or scales containing the same number of items, which could be constructed from a hypothetical universe of items that measure the characteristic of interest. Another interpretation of Croenbach's alpha is the squared correlation between the score a person obtains on a particular scale (the observed score) and the score that would have been obtained if questioned on all of the possible items in the universe (the true score).

Since α can be interpreted as a correlation coefficient, it ranges in value from 0 to 1. Negative α values can occur when items are not positively correlated among themselves and the reliability model is violated.

CHAPTER 6

OFFICE ENVIRONMENT SURVEY

Before deciding on which survey method to use, the advantages and disadvantages of the six approaches, discussed under the heading 'survey methods', were considered.

In deciding which method would yield the desired result, the probable skills of the end user had to be taken into consideration. It was considered that the most likely end user of the procedure would come from one of the technical disciplines (ie. air-conditioning engineer, technician, property manager, etc.).

Cognisant of the end-user's likely ability the 'medical record' option was discarded for a number of reasons:

(a) The personnel applying the procedure in the first instance would, probably, not have medical expertise;
(b) There might be a privacy and ethical issues involved, and medical records might not be available;
(c) It is unlikely that 'minor acute symptoms' associated with BRI would be recorded on medical records. In most cases, the symptoms are too minor for a 'sufferer' to consult a General Practitioner.
(d) The epidemiology and incidence of SBS and the prevalence of hypersensitivity syndromes is unknown. Thus, valid interpretation of results is difficult.

Physical sampling was rejected on the basis that it was inappropriate as a 'first step' option. Sampling is an expensive exercise. Raw (1992), indicated that the approach is wasteful if complaints within the building are localised or occur only in certain occupational groups or at certain times of the day or week. Additionally, even if samples were obtained, interpretation of the results would be extremely difficult as the dose-effect relationships are not really known.

The *human 'canary'* theory was rejected on an ethical basis. In addition, there is some doubt as to whether it would work. It was discounted by Grande et al., (1988), because allergic people's reactions are governed by a wide set of circumstances, not just the indoor environment.

Interventional studies were not considered to be a valid option at this 'first stage' of the investigation process. Logically, there seems no good reason to intervene before the problem has been established.

This left the choice of a questionnaire or a face-to-face survey. It was decided that both were valid and would achieve the desired results. The advantages of face-to-face interview is that the response rate is reported to be higher (Burge, 1987). However, the time and cost of such interviews is proportionately greater. The marginally higher response expected from face-to-face interviews was outweighed by time and budgetary constraints.

For the above reasons, a questionnaire survey was selected.

SURVEY DESIGN

The stated purpose of the survey was *to devise a suitable procedure to test whether the occupants of a building under survey were experiencing an 'extraordinary' level of symptoms compared to similar cohorts in the general population.*

An additional consideration was that the model had to be of practical assistance to the end user.

To achieve the stated objective two methods were considered:

(1)The status quo as used in most surveys was to ask the question: *"are the symptoms work related"*. This was asked in a number of ways, for example, *"was there an improvement on days away from the office?"*.

The comparative results in these surveys were given a relative score and compared to other buildings yielding an overall 'mean' score.

While the above method may answer the question as posed, there are both advantages and disadvantages associated with the approach:

The primary advantage is that the system has been tried and tested in a number of other studies. It is understood and accepted by other researchers.

However, there are also a number of disadvantages in respect to the stated objective:

(a) A large number of buildings would need to be surveyed to obtain a meaningful result.
(b) An underlying objective (as stated) is to provide a 'tool' for use by the 'non-expert. This above method implies open access to information which may be commercially sensitive. Access to such information may not be possible.
(c) If access is available, there still exists the problem of a *'pass mark'*. At what level does the building appear to have a problem? Suggested failing grades may be an index greater than the mean, or a score in the upper quartile or any number of arbitrary measures. It is suggested that such measures would be difficult to defend.

(2) A second method considered was to obtain base-line data from the general population of office workers. This is a normative approach to the problem. If it is accepted that the symptoms of interest are common amongst the general population (at unknown incidence), provided that a representative sample is selected, excess symptoms amongst a cluster of people with a common factor of a particular building may suggest that the excess is building related.

The method has a number of disadvantages:

(a) A large sample size would, probably, be required at the base line.
(b) A separate base line might be required for the different geographic regions because of differing externalities associated with different locations. For example, the area of the subject survey around Palmerston North is reputed to be detrimental to asthmatics and people suffering from perennial rhinitis because of high, seasonal, pollen counts.

The advantages are:

(a) The baseline, once calculated, is unlikely to be commercially sensitive and thus more accessible to the target users.
(b) The method avoids the respondent to subjectively assess whether there is an improvement or symptom diminution on weekends and days off. This is likely to be a major source of bias. In a private communication with Chamberlain (1993), he confirmed that, in the majority of studies, people claimed to feel better on their days off. A feeling of depression on Monday mornings and elation at the end of the week is a commonly observed condition.

The suggested method avoids the need to ask this particular question, because, once as the base line data is obtained, the only differentiating factor between the groups 'should be' the building under review. Thus the answer to 'improving health' or otherwise may be obtained through implication.

QUESTIONNAIRE DESIGN

The method chosen was the base-line approach. This influenced the length of the questionnaire (refer Appendix 11). The symptoms areas which needed to be canvassed were established. Table 2, established the general symptoms associated with the established building supported illnesses. With the exception of weight loss, diarrhoea, and chronic post nasal drip, the symptoms included in Table 20 were incorporated in the questionnaire.

The following *Nominal* variables were also included:

(a) Job type
(b) Age group
(c) Gender
(d) Smoking habit
(e) Whether smoking is allowed at the workplace

In addition to frequency, questions on severity of symptoms were included. It was believed that an indication of symptom severity would yield useful information. For example, a building with a high frequency of severe symptoms would have a greater problem than one with a high frequency of very mild symptoms.

BASELINE SAMPLE SELECTION

True baseline data, most probably, does not exist. The baseline suggests a level of symptoms uncontaminated by the occupancy of a building. There are a number of possible, if theoretical, approaches available to collect the required data.

(1) Sample the occupants of a perfect, or near perfect building, in which building related symptoms have been eliminated. For practical purposes, such a building probably does not exist at this time.

(2) Limit the sample to people who *do not* occupy office buildings.

There are many problems associated with this approach. The confounding issues are multitude and might include; occupational exposures, socioeconomic, employment status and other differences which affect symptom reporting.

(3) Sample a random selection of office workers from a variety of buildings.

This is not a baseline in the true sense, rather it identifies an average level of symptoms experienced by such workers.

For practical purposes, the last approach was chosen, with full realisation of its' limitations. The rationale is that if a large enough sample of data were collected, the extremes would be cancelled out giving a true average against which specific buildings might be measured. The result then is not that a building is sick but the level of symptoms experienced by the occupants are more or less than average.

BASELINE SAMPLE SIZE

The sample size at the base line was a problematic issue. Because of the demographic influences previously discussed, a large sample would seem to be indicated. Unfortunately, for our pilot study, the eventual sample size was dictated more by time and budgetary constraints. A sample size of around 250 valid responses was eventually aimed for and achieved.

Probability sampling seldom, if ever, provides statistics exactly equal to the parameters that they are used to estimate. Probability theory, however, permits the estimation of the degree of error to be expected for a given sample design (Babbie, 1992). A sample size of 250 is expected to give a sampling error of about 6.2% from the following formula:

$$\sqrt{\frac{p \times q}{n}} \times CONFIDENCE\ INTERVAL$$

$$\sqrt{\frac{0.5 \times 0.5}{250}} \times 1.96$$

$\therefore$ Estimated Sampling Error = 6.198%

Where:

p = a percentage
q = 1 - p
n = sample size

SAMPLING METHOD

To achieve the high rate of response required the support of the personnel manager in Building 2 and the General manager in building one were enlisted. These individuals distributed the questionnaires and collected them on the same day.

Workers were excluded from the sample if they were on vacation, on maternity leave, worked part time, were temporary staff, or if they worked in the building for less than one month. Workers absent due to illness or who were away from the building for other reasons were included in the sample; for these respondents, a questionnaire and explanatory letter were left for subsequent collection.

All the questionnaires were individually distributed and collected on the same day, and were checked for completeness. Incomplete documents were followed up with the respondents at this point.

Responses from the occupants in the two buildings the study were as follows:

(1) Building 1 - 100% response.
(2) Building 2 - 92% response.

In Building 2, 7% of the occupants had been there for less than one month. Therefore, the response rate from eligible occupants was close to 100%.

Discussion and Results

Basic Methodology

The approach involved identifying the number and severity of symptoms experienced by each nominal group and comparing each cohort with the equivalent baseline cohort.

For example, the frequency of headaches suffered by, say, VDU operators in a building under investigation, is compared to the frequency of headaches suffered by VDU operators in the baseline group. It is suggested that this approach circumvents the problems of symptom reporting tendencies common to differing groups.

It is unknown for instance, if the high level of symptoms commonly reported by VDU operators is as a result of 'job stress', psychological or physiological factors. It is submitted that the advantage of the proposed approach is that the reporting mechanism is unimportant as the factors should cancel each other out.

Of course, there may be other confounding factors at play, although in this survey at least the groups seemed to be relatively homogenous. For instance, VDU operators tended to be mostly female and under 40 years of age. Other groups displayed their own homogenous characteristics.

Scale Reliability

The Croenbach α for the subject scale was 0.9331 or 93.31% indicating that the scale is quite reliable.

Statistical Test Used

The data collected were ordinal rather than interval data. For these type of data *'nonparametric statistical tests'* are deemed to be more appropriate.

Such statistics refer to hypothesis tests about population probability distributions, but not about specific parameters of the distributions.

An answer to the following question was sought:

"Is there a statistically significant difference in symptom reporting between like cohorts".

The Mann-Whitney U test, also known as the Wilcoxon Rank Sum W test was considered to be appropriate. This test provides a procedure for testing whether two populations are identical when *independent* random samples are selected from two populations (Ott et al., 1985).

Data Reduction

Analyzing all variables within each sub-group meant that some 672 variables had to be examined. Some consideration was given to reducing the number of variables by grouping them in some manner. Two options were considered.

(1)The variables might be combined into their eight logical groups and averaged. For instance the three 'eye' questions were combined into a single 'eye' variable and the scores averaged. This reduced the number of variables to be analyzed from 672 to 128. This method gave plausible results although it was less discriminatory than the 'fuH' model.

The Croenbach α reduced from 0.9331 to 0.9099 indicating a small reduction in the reliability of the scale.

(2)The variables might be combined into their eight logical groups and the maximum score for included variables was adopted. For example, if dry eyes scored 0, irritated eyes scored 1 and strained eyes scored 4, the variable 'eyes' had a score of 4. This method gave the weakest results.

The α coefficient was reduced to 0.86 or 86.04%.

On balance, it was decided to adopt the full model. However, this does not rule out combining the frequency and severity scales into a single logical scale in the future.

BUILDING ONE

This building was an EDP installation. It was an older concrete block structure, with a CAV air conditioning system and four 'stand alone' localized cooling units in the machine rooms. These were separately plumbed and were not part of the main air conditioning system.

The installation operated on shifts for five days and nights each week and was closed down at weekends.

There had been a number of BRI type complaints from staff.

Firstly, all of the symptoms in the subject building were compared against the base-line group. All of the differences that were statistically significant at the 95% level and above were included, the remainder were disregarded. The results are reported in the following tables. In the tables, negative differences indicates that the group under study were *worse-off* than the base-line group, and positive differences means that they were better off.

Secondly, the sub-groups were compared against like subgroups. The reason for this is to be found in the different levels of symptom reporting as previously discussed. For instance, if the symptoms remained undifferentiated, a building with a predominance of, say, V.D.U. operators would be unduly penalised against the base-line. Analysis of the sub-groups yielded useful secondary information as became clear from examination of the results (also refer Appendix 12).

TABLE 17 - SUMMARY BUILDING 1 UNDIFFERENTIATED SYMPTOM COMPARISON

	# Occupants	# Statistically Significant Negative Differences	# Statistically Significant Positive Differences	Average # Symptoms Per Occupant
Total	27	24	0	0.88

TABLE 18 - SUMMARY BUILDING 1 BY JOB DESCRIPTION

	Male	Female	# Statistically Significant Negative Differences Reported	# Statistically Significant Positive Differences Reported
Management	3	0	4	0
Professional	0	0	N/a	N/a
Clerical/secretarial	1	11	7	0
V.D.U. operators	2	10	7	0
TOTAL	6	21	18	0
Negative Symptom Average: Managers (1.33), C/S (0.58), V.D.U (0.58)				

TABLE 19 - SUMMARY BUILDING 1 BY AGE COHORT

	Male	Female	# Statistically Significant Negative Differences	# Statistically Significant Positive Differences
Under 30	4	6	16	0
30-39	1	10	10	0
40-49	0	3	7	0
50-59	1	2	5	0
60-60	0	0	N/a	N/a
TOTAL	6	21	38	0
Negative Symptom Average: Under 30 (1.60), 30-39 (0.90), 40-49 (2.33), 50-59 (1.66)				

TABLE 20 - SUMMARY BUILDING 1 BY GENDER

	Male	Female	# Statistically Significant Negative	# Statistically Significant Positive Differences
Female	0	21	16	0
Male	6	0	7	0
TOTAL	6	21	23	0
Negative Symptom Average: Male (1.16) Female (0.76)				

TABLE 21 - SUMMARY BUILDING 1 BY TOBACCO SMOKING HISTORY

	Male	Female	# Statistically Significant Negative Differences	# Statistically Significant Negative Differences
Smoker	0	4	12	0
Ex-smoker	0	2	8	0
Non-smoker	6	15	18	0
TOTAL	6	21	38	38
Symptom Average: Smokers (3.00), Ex-Smokers (4.00), Non-S (1.20)				

TABLE 22 - SUMMARY BUILDING 1 BY ENVIRONMENTAL SMOKING

	Male	Female	# Significant Negative Differences	# Significant Positive Differences
Smoking	1	5	12	
Non-smoking	5	16	20	
TOTAL	6	21	32	0
Negative Symptom Average: Smoking (2.00), Non-Smoking (0.95)				

The results did not fit the expected pattern as reported in other surveys of this nature.

Managers reported a higher number of symptoms than the clerical/secretarial and V.D.U. groups.

The 40-49 age group reported the highest number of symptoms which also ran against the expected trend.

Males reported a higher number of symptoms than females.

For some inexplicable reason, ex-smokers reported a higher number of symptoms than smokers. It is speculated that an explanation for this is that there is tendency for smokers to underreport. Other social studies have found that there is a common tendency for people to underreport alcohol and tobacco consumption.

To further investigate these results the generic groups of symptoms by sub-group were tabulated as follows.

TABLE 23 - UNDIFFERENTIATED SYMPTOM COMPARISON

Building One - Undifferentiated	
FREQUENCY	SEVERITY
Eyes Nasal Throat Somatic Skin	Eyes Nasal Throat Somatic Back Chest Skin

TABLE 24

Building One - Symptoms Reported by Job Description		
Title	FREQUENCY	SEVERITY
Management	Eyes	Eyes Throat Somatic
Clerical/Sec	Nasal Throat	Nasal Throat
V.D.U.	Nasal	Nasal Back (Muscular) Chest

TABLE 25

Building One - Symptoms Reported by Age		
Age Group	FREQUENCY	SEVERITY
Under 30	Eyes Nasal Throat Chest	Eyes Nasal Throat Somatic Chest
30-39	Throat Chest	Eyes Nasal Throat Somatic Back Chest
40-49	Eyes Chest	Eyes Nasal Back (Muscular)
50-59	Eyes Nasal	Eyes

TABLE 26

Building One - Symptoms Reported by Gender		
Gender	FREQUENCY	SEVERITY
Male	Eyes	Eyes Throat Somatic Chest
Female	Eyes Nasal Throat	Eyes Nasal Throat Somatic Back (Muscular) Skin

TABLE 27

Building One - Symptoms Reported by Smoking Habit		
	FREQUENCY	SEVERITY
Smoker	Eyes Nasal Throat Somatic	Eyes Nasal Throat Somatic Back
Ex-Smoker	Nasal Throat Somatic Skin	Nasal Throat Skin
Non-Smoker	Eyes Nasal Throat Somatic Chest	Eyes Nasal Throat Pulmonary

TABLE 28

Building One - Symptoms Reported by Environmental Smoking		
Env Smoke	FREQUENCY	SEVERITY
Yes	Throat Somatic Skin	Nasal Throat Somatic Back Skin
No	Eyes Nasal Throat Chest	Eyes Nasal Throat Somatic Back Skin

The most ubiquitous symptoms were nasal and throat symptoms, with eye complaints being the next most common. The next most commonly reported groups of symptoms were back (muscular) complaints from the V.D.U. operators. The least common complaints were chest/pulmonary, somatic and skin complaints.

Detailed analysis of the responses showed that single individuals or a very small number of individuals complained of the latter symptoms. This highlights a potential problem when dealing with such small sub-groups (as in this case). One individual suffering from a particular ailment can have an undue influence on the results.

This does not weaken the model as this influence can be analyzed out. Rather it posts a warning to analysts that care must be taken when dealing with smaller groups.

One suggested way of dealing with the above problem is that when an individual or very small numbers of occupants complain of particular symptoms which are not reported by the larger groups, the symptomatic individuals should be interviewed separately, to ascertain whether there is a medical condition present. A number of the other questionnaires address this problem by soliciting information on allergies.

It can be concluded, the occupants of the subject building report a statistically significant number of symptoms.

Reasons for the unusual results may be explained by analysis of the subject organizational structure.

(1) The manager has a separate office, other supervisory staff share the same work space as subordinates.
(2) None of the occupants, including management, have much control over environmental conditions.
(3) A number of the males are shift computer operators. There is some evidence that shift-workers complain of symptoms more often than day workers.
(4)The age of the occupants does not have a bearing on seniority or promotional prospects, there being a very flat organizational structure.

Nevertheless, it can be concluded that the building appears to have a problem and that a more detailed audit is justified.

BUILDING TWO

This is a modern building with a basement and ten floors. It is fully air conditioned with a VAV system and separate air handling units on each floor. Two of the floors have 'stand alone' air conditioning systems. One of these is a computer room and the other is ground floor legal offices.

The standard of accommodation is amongst the best available in Palmerston North, although it would not be 'head-office' standard in a larger commercial centre.

However, the building is not without problems. Since the building was commissioned there have some complaints regarding BRI symptoms.

The same analysis was undertaken as on Building One and the results tabulated as follows (also refer Appendix 13).

TABLE 29 - SUMMARY BUILDING 2 UNDIFFERENTIATED SYMPTOM COMPARISON

	# Occupants	# Statistically Significant Negative Differences	# Statistically Significant Positive differences	Average # Symptoms Per Occupant
Total	81	8	0	0.09

TABLE 30 - SUMMARY BUILDING 2 BY JOB DESCRIPTION

	Male	Female	# Statistically Significant Negative Differences Reported	# Statistically Significant Positive Differences Reported
Management	9	1	0	2
Professional	16	8	0	0
Clerical/secretarial	7	32	8	0
V.D.U. operators	0	8	8	0
TOTAL	32	49	16	0
Negative Symptom Average: Managers (0.00), Professional (0.00), C/S (0.20), V.D.U (1.00), Overall (0.19)				

TABLE 31 - SUMMARY BUILDING 2 BY AGE COHORT

	Male	Female	# Statistically Significant Negative Differences	# Statistically Significant Positive Differences
Under 30	6	27	2	0
30-39	8	7	2	0
40-49	9	11	2	1
50-59	9	4	0	1
60-60	0	0	N/a	N/a
TOTAL	32	49	6	2
Negative Symptom Average: Under 30 (0.06), 30-39 (0.13), 40-49 (0.10), 50-59 (0.00), Overall (0.07)				

TABLE 32 - SUMMARY BUILDING 2 BY GENDER

	Male	Female	# Statistically Significant Negative	# Statistically Significant Positive Differences
Female	0	49	8	0
Male	32	0	1	0
TOTAL	32	49	9	0
Negative Symptom Average: Male (0.03) Female (0.16)				

TABLE 33 - SUMMARY BUILDING 2 BY TOBACCO SMOKING HISTORY

	Male	Female	# Statistically Significant Negative Differences	# Statistically Significant Negative Differences
Smoker	3	5	0	0
Ex-smoker	12	5	0	0
Non-smoker	17	39	11	0
TOTAL	32	49	11	0
Symptom Average: Smokers (0.00), Ex-Smokers (0.00), Non-S (0.19)				

TABLE 34 - SUMMARY BUILDING 2 BY ENVIRONMENTAL SMOKING

	Male	Female	# Significant Negative Differences	# Significant Positive Differences
Smoking	1	1	2	0
Non-smoking	31	48	10	0
TOTAL	32	49	12	0
Negative Symptom Average: Smoking (1.00), Non-Smoking (0.13)				

The above results were more in line with other research. It can be speculated that this was because of the larger population sampled and the more conventional organizational structure within the building as a whole.

Managers and professionals had no significant negative differences and, indeed, were less symptomatic than the base-line group in two areas (nasal & backache). This may possibly be a reflection of superior accommodation.

Clerical workers reported some complaints with the V.D.U. operators reporting most.

The results from the age group analysis were less predictable but in line with other research. There was a reduction in the 40-49 group and zero complaints from the 50-59 age group. This may be as a result of the hierarchical structure within the building and/or a greater percentage of males represented in the latter groups.

Females reported a far higher number of symptoms than males although examination of the statistics show a far higher proportion of females in the lower level jobs.

Smoking-history yields vexing results with non-smokers reporting a higher number of symptoms than smokers and ex-smokers, although this might be explained by the higher proportion of non-smokers being female.

Those exposed to environmental smoke yields a somewhat amusing result. The exposed group which has only two members complain of more symptoms than non-smokers. As the building is a non-smoking zone, it is speculated that they may be self-reporting their own smoking.

TABLE 35

Building Two - Undifferentiated	
FREQUENCY	SEVERITY
Eyes Nose Throat Somatic	Eyes Nose Somatic

TABLE 36

Building Two - Symptoms Reported by Job Description		
Title	FREQUENCY	SEVERITY
Management	None	None
Professional	None	None
Clerical/Sec	Eyes Nasal Throat	Eyes Nasal Throat
V.D.U.	Nose Throat Chest	Eyes Nose Chest

TABLE 37

Building Two - Symptoms Reported by Age		
Age Group	FREQUENCY	SEVERITY
Under 30	Nasal	Nasal
30-39	Eyes Throat	None
40-49	Nasal Throat	None
50-59	None Nasal	None

TABLE 38

Building Two - Symptoms Reported by Gender		
Gender	FREQUENCY	SEVERITY
Male		Somatic
Female	Eyes Nasal Throat Somatic	Eyes Nasal

TABLE 39

Building Two - Symptoms Reported by Smoking Habit		
	FREQUENCY	SEVERITY
Smoker	None	None
Ex-Smoker	None	None
Non-Smoker	Eyes Nasal Throat Somatic	Eyes Somatic

TABLE 40

Building Two - Symptoms Reported by Environmental Smoking		
Env Smoke	FREQUENCY	SEVERITY
Yes	Eyes Somatic Skin	
No	Eyes Nasal Throat	Eyes Nasal Somatic

Clearly building two is far superior to building one, which was not unexpected.

However there is a disturbing cluster of symptoms amongst the clerical/secretarial workers and the V.D.U. operators, the most ubiquitous being eye, nasal and throat symptoms.

Some of the V.D.U. operators also report chest related symptoms. The group which has eight members is sufficiently small that a follow up interview might be appropriate before accepting the complaint as a valid symptom.

Finally, there is a ubiquitous level of somatic complaint reported by both male and female workers.

It can be concluded from the evidence that, there may a problem amongst certain groups which warrants further investigation.

SURVEY LIMITATIONS

Initially the length of the questionnaire was considered to be an issue and brevity was stressed. A number of possible questions were omitted because of this. A number of these should be included in future as they yield useful information. These include:

> *Nosebleeds* - which are a useful indicator of 'low' humidity. While 'low' humidity is not a problem in the North Island of New Zealand where the questionnaire was piloted, it may be a problem in South Island, increasing with distance from the equator.
>
> *Chronic post nasal drip* - is a useful indicator of sensitization.
>
> *Dyspnea* - breathlessness, without undue excretion is a useful indicator of a number of the clinically diagnosable illnesses of interest.

The inclusion of questions on conditions such as asthma and hay fever may provide a useful filter.

A major omission in the questionnaire was that of a question on shift work. In this respect, Building 1 may have been unduly penalised. This question should be included in subsequent questionnaires. Also suggested for inclusion in subsequent questionnaires is martial status - which has been identified as a potential confounding variable.

Potentially, the most serious limitation with the subject survey was that the delivery method may have introduced a measure of bias. The base line data were collected by telephone interview and the data from the buildings under investigation were collected by self-completed questionnaire. Although, there are no studies relating to the symptom reporting level from telephone surveys, 'personal interviews' are consistently reported as producing lower incidence rates. The use of management in the subject buildings may also have influenced the workers' response.

CHAPTER 7

CONCLUSIONS

(1) Building related illness has been identified as a potentially serious concern in most developed countries.

A number of prevalence studies reported that the syndromes affect between 20-30% of the office population .

Several studies have estimated that the economic cost of building related illness in terms of absenteeism and degraded productivity, is high. Most of the productivity figures are self-reported and must be questioned. Nevertheless, there is ample evidence that BRI is a problem of global proportions.

(2) A huge variety of building supported stressors have been identified as being capable of producing the wide range of symptoms associated with BRI. Some job related and psychological stressors can also produce the same range of physical symptoms. Before the problems can be attributed to the building, these confounding sources need to be investigated and eliminated.

(3) The stressors associated with the symptoms of interest and that can be directly attributed to the building itself include:

(a) Inadequate ventilation (including poor quality outdoor air);
(b) Volatile organic compounds from a variety if indoor sources:
- new furniture, painted surfaces
- carpets and glues

(c) Inorganic gases from basement car-parks and ozone from photocopiers. Although the ozone problem reduces as a function of distance from the source.
(d) Airborne particles from a variety of sources have been strongly linked to symptoms. The particles themselves can be downright toxic or can act as a raft for viable entities such as viruses. The shape and size of the particles dictate where the viable entity is deposited in the body. Generally, the deeper that these penetrate into the RI tract and lung, the more damage they are capable of inflicting.
(e) Tobacco smoke has been identified as a problem but it's role in BRI is ambiguous. Some studies have found a link between active and passive smoking and a variety of symptoms, while other reports have had null results. Nevertheless, there is overwhelming evidence that active and passive smoking causes a variety of other diseases.
(f) There is evidence of a link between thermal discomfort and the onset of symptoms. A common complaint from building occupants is that they lack control over the thermal environment, so the thermal environment may also be a psychological stressor.

(g) Humidity appears to be a pivotal factor in symptom reporting. While humidity, in the normal range, does not appear to be a stressor in itself, it interacts with other factors. If humidity is too high it supports the growth of viable particulates such as moulds and dust mites and it facilitates the release of water soluble compounds such as formaldehyde. If the moisture level is too low, it facilitates electrostatic shocks and dry mucous membranes manifesting as nose-bleeds.
(h) The role of natural and artificial light in the indoor environment is also considered to be a contributory factor. Artificial light is sometimes inadequate or poorly placed in relation to the work surfaces, causing reflection and glare. The flicker of fluorescent lights has been associated with headaches. Insufficient access to natural light has been associated with feelings of sensory deprivation.
(i) Noise and vibration from building services have been identified as a potential building stressor.
(j) There is some evidence linking a high relative proportion of positive air ions, commonly found in commercial buildings, with headaches and lethargy. The literature relating to the subject is inconclusive.
(k) Psychological factors seem to dictate the way people perceive and report symptoms. Some links have been made between BRI symptoms and their lack of control over the thermal and lighting environment. In this respect, such psychological reactions could be said to be building supported.
(l) There are a number of clinically diagnosable illnesses which are building supported. Dirty humidifiers, drip trays, faulty plumbing and cooling towers have been associated with outbreaks of Legionnaires Disease, Pontaic fever and Humidifier Fever.
(m) Air intakes contaminated with bird droppings have been associated with outbreaks of Q Fever.
(n) Various dirt sinks have been associated with asthma and various hypersensitive reactions.
(o) Airborne dust may facilitate the transfer of some viruses from person to person, although the transfer mechanism is not fully understood.

(4) There are a variety of stressors which are not 'strictly' building related but which are capable of causing similar symptoms to those associated with BRI.

(a) Job related stress associated with ambiguous work requirements, poor supervision, lack of responsibility and participation in the decision making process, lack of personal responsibility and control, threat of redundancy and a variety of other interpersonal stressors can lead to dissatisfaction manifesting as physical symptoms.
(b) Poorly designed work stations and work schedules can lead to 'task related' stress, resulting in backache, sore eyes and headache.
(c) Toxic substances introduced into the working environment by the tenant. For example, the printers ink on consignments of, say, annual reports or advertising pamphlets can cause short term increases of VOC levels in the immediate environment.

Arguably the ventilation system should be able to cope with these kinds of 'short-term' loadings.

These cannot be attributed to the building itself but are often confused with BRI.

(5) Chronic illnesses were considered under three headings. The pathway in dealing with these stressors is preventative rather than curative.

(a) Asbestos is probably best left undisturbed unless it is friable and poses an immediate danger. Removing asbestos can often pose more of a hazard than leaving it undisturbed as it, frequently, causes short term elevations in the airborne particle counts.
(b) Radon is a naturally occurring radiation from geological sources and from building materials. Radon is identified by measurement and there are a number of methods of dealing with 'ground sourced' radon when it occurs. The dose-response relationship to radon is established.
(c) Naturally occurring ground radiation in New Zealand is considered to be low by world standards.
(d) Electro-magnetic fields from electrical configurations is a potential cancer causing agent. The dose-response relationship has not been established and is still the subject to ongoing investigation.

(6) From the experiment it may be concluded that excess symptoms can be identified by comparing similar cohorts.

The approach circumvents some of the problems associated with symptom reporting tendencies and certain 'job related' characteristics. The rationale for this statement is that if certain symptoms are related to the job itself, all workers in that occupation will be affected to a greater or lesser degree by the same stressers.

From the evidence presented, it is concluded that the procedure answers the question as posed. Additionally, it identifies clusters of symptoms that may give an indication of possible problem sources.

CHAPTER 8

FUTURE RESEARCH RECOMMENDATIONS

In common with much social research, this study provides an answer to the question which it set out to answer but it also raised new issues and questions. It remains for future researchers to test the model on a larger sample and on a wider group.

It is recommended that a longitudinal study be undertaken on a larger population to establish whether:

(1) There are geographical influences on symptom reporting.
(2) There are seasonal factors involved.

In conjunction with the above, causation studies need to be undertaken on selected populations combining information gained from baseline comparison with physical measurements.

The latter seems to be the next logical step, as the questionnaire data is unlikely to be of use in a legal context unless it is validated by physical measurements.

APPENDIX 1

APPENDIX 1. GLOSSARY OF ABBREVIATIONS USED

AAV	-	Adenoassociated virus
AC	-	Asbestos Cement or Alternating Current (context)
ACH	-	Air Changes Per Hour
ADPI	-	Air Diffusion Performance Index
AFRD		- Acute Febrile Respiratory Disease
AH	-	Absolute Humidity
APC	-	Pharyngonconjunctival Fever
APCF	-	Acute Pharyngonconjunctival Fever
ARD	-	Acute Respiratory Disease
ASHRAE	-	The American Society of Heating, Refrigeration and Air Conditioning Engineers
BMJ	-	British Medical Journal
BRE	-	Building Research Establishment
BRI	-	Building Related Illness
CAV	-	Constant Air Volume
CIBS	-	Chartered Institution of Building Services
CIBSE	-	Chartered Institution of Building Services Engineers
CNS	-	Central Nervous System
dB	-	Decibel
DHSS	-	Department of Health and Social Security
DOE	-	Department of the Environment (U.K.)
ECM	-	Energy Conservation Measure
EKC	-	Epidemic Keratoconjunctivitis
ELF	-	Extremely Low Frequency
EMF	-	Electromagnetic Field
EPA	-	United States Environmental Protection Agency
ETS	-	Environmental Tobacco Smoke
GI	-	Gastro Intestinal (tract)
HMSO	-	Her Majesty Stationary Office
HSE	-	Health and Safety Executive
HVAC	-	Heating, Ventilating and Air Conditioning
Hz	-	Hertz
IAQ	-	Indoor Air Quality
JAMA	-	Journal of the American Medical Association
kV	-	kilovolt
Lm	-	Lumen
mG	-	Milligaus
MMMF	-	Man-Made-Mineral Fibres
NASA	-	United States National Aeronautics and Space Administration
NIOSH	-	National Institute of Safety and Health
NKB	-	Nordic Committee on Building Regulations
PERF	-	Peak Respiratory Function
PMV	-	Predicted Mean Vote
PPD	-	Predicted Percentage Dissatisfied
PVA	-	Polyvinyl Acetate
PVC	-	Polyvinyl Chloride
PVP	-	Polyvinylpyrolidone
RH	-	Relative Humidity

RH	-	Relative Humidity
RI	-	Respiratory Infection
RSP	-	Respirable Suspended Particles
RSV	-	Respiratory Syncytial Virus
SBS	-	Sick Building Syndrome
T	-	Telsa
TVOC	-	Total Volatile Organic Compounds
UFFI	-	Urea Formaldehyde Foamed Insulation
URI	-	Common Cold
USFDA	-	United States Food and Drugs Administration
VAV	-	Variable Air Volume
VCM	-	Polyvinyl Chloride Monomer
VDT	-	Visual Display Terminal
VDU	-	Visual Display Unit
VLF	-	Very Low Frequency
VOC	-	Volatile Organic Compounds
WHO	-	World Health Organisation

APPENDIX 2

APPENDIX 2. GLOSSARY

ABSOLUTE HUMIDITY	Complete saturation at a given temperature.
ABSORPTION	To soak or suck up (Liquids)
ACANTHAMOEBA	*Acanth(o)* - comb like form; *Amoeba* - a microscopic one-celled animal of the protozoa family.
ACARUS (ACARID)	A parasite of the genus Arachnida, which includes many varieties of ticks and mites (es. A. Scabei)
ACETYLCHOLINE	Chemical produced in the nerve endings & essential for the transmission of nerve impulses.
ACNEFORM	Resembling acne
ACTINOMYCOSIS	Long lasting disease caused by a microorganism (Actinomyces israelii) that is normally present in the mouth and throat.
ACUITY	Keenness or acuteness
ACUTE	(Disease) Arising suddenly and manifesting intense severity or of relatively short duration.
ADDISON'S DISEASE	Deficient secretion from the adrenal cortex, causing electrolytic upset, diminution of blood volume, lowered blood pressure, marked anaemia, hypoglycaemia and pigmentation of the skin.
ADENOID	(1) Overgrowth of lymphoid tissue in the nasopharynx; (2) Resembling a gland.
ADENOVIRUS	(From *Aden = Gland*) a variety of types of virus. Some causing upper respiratory infection, others pneumonia, others tumours, others epidemic keratoconjunctivitis.
ADRENAL NECROSIS	*Adrenal* - pertaining to the adrenal glands; *Necrosis* - mortification, localized death of tissue.

ADRENAL CASTS

Adrenal - pertaining to the adrenal glands; *Cast(s)* - fibrous material and exudate which has moulded to the form of the cavity or tube in which it has collected.

ADRENALINE

Hormone secreted by the *medulla* of the adrenal gland - that increases heartbeat & blood pressure in response to stress & anxiety.

ADRENOCORDIAL AXIS

Adrenocortex - part of the adrenal gland which secretes mineral and glucocorticoids which control the chemical constitution of the body fluids, metabolism and sexual characteristics; *Axis* - the imaginary line passing through the centre.

ADRENOHYPOPHSEAL

Hypophysis cerebri - small oval shaped gland lying in the pituitary fossa of the sphenoid bone and connected to the under surface of the brain by a stalk; the pituitary gland.

ADENOVIRUS

Viruses found in the lymphatic tissue.

ADSORPTION

To undergo or cause to undergo a process in which a substance, usually gas, accumulates on the surface of a solid forming a thin film.

ANAEMIA

A disorder due to a deficiency in the number of red blood cells or of their haemoglobin content, or both. The clinical features may include pallor, easy fatigue, and breathlessness, palpitation and loss of appetite.

AEROSOL

A colloidal dispersion of solid or liquid particles in a gas.

AETIOLOGY

(Variant spelling of etiology) (1) study of causation; (2) study of causes of disease; (3) the cause of disease.

AFEBRILE

Without fever

AGGLOMERATION

To accumulate or collect in a mass.

ALBUMINURIA

Existence of protein in the urine which is usually in the form of albumin. Can be a sign of kidney disease or failure.

ALLERGIN

A substance (antigen) to which the body reacts with unusual sensitivity.

ALVEOLUS	A small sac-like body cavity. The term is usually used to describe the millions of microscopic air sacs (alveoli) of the lung tissue.
AMBIENT	Surrounding
AMINO-ACID	One of the basic nitrogen-containing substances that go into the making of proteins in living matter.
AMMONIA	Colourless pungent gas used in the manufacture of fertilizers and as a refrigerant or solvent.
AMOEBA	A microscopic one-celled animal of the protozoa family.
AMPHIBOLE	Any of a large group of minerals consisting of silicates of calcium, iron, magnesium, and aluminium, which are common constituents of igneous rocks.
ANAPHYLAXIS	Extreme sensitivity to an injected antigen following a previous injection.
ANGINA	Any disease marked by painful attacks of spasmodic choking.
ANGINA PECTORIS	Specific condition that involves pain from the heart. The pain occurs because insufficient oxygen reaches the heart muscle, especially following exercise.
ANGIOSARCOMA	Abnormal growth of tissue formed by a group of small blood vessels. If it occurs in the brain it can lead to subarachnoid haemorrhage or stroke.
ANOREXIA	Psychiatric disorder characterized by a reduction in food intake.
ANTHRAX	A contagious disease of cattle which may be transmitted to man by inoculation, inhalation and ingestion causing malignant pustule.
ANTIBODY	A substance that is produced by the body in response to an infection. It combines with an antigen (such as a virus) and neutralizes it.
ANTIGEN	Any substance which, under favourable conditions, can simulate the production of antibodies.

ARRHYTHMIAS	An irregularity of the heartbeat that produces a variation in the pulse rate.
ASEPTIC	The state of being free from living pathogenic organisms.
ASPERGILLOSIS	Any infection caused by any species of *Aspergillus*.
A. FUMIGATUS	A genus of fungi often found in soil, manure and various grains.
ASTHMA	A chronic respiratory disease in which the person experiences difficulty in breathing accompanied by wheezing and 'tight chest'.
ASYMPTOMATIC VIRAEMIA	(*Asymptomatic* = symptomless) (*Viraemia* = the presence of virus in the blood)
ATAXIA	Condition characterized by loss or imparement of normal co-ordination, especially co-ordination of voluntary movements of the muscles.
ATOPIC	Hereditary tendency toward allergic reaction (eg. hay fever, asthma, eczema etc.)
ATTENUATION	To weaken or become weak.
AUSCULTATION	A method of listening to the body sounds for diagnostic purposes, particularly the heart, lungs and foetal circulation.
AUTONOMIC	(1) Occurring spontaneously; (2) relating to the autonomic nervous system that controls involuntary actions of the smooth muscles (ie. heart & glands)
BACTERIA	A group of micro-organisms also called the *'schizomycetes'*. They are typically small cells of about 1 μm in transverse diameter. Structurally there is a protoplast, containing cytoplasmic and nuclear material within a limiting cytoplasmic membrane and a supporting cell wall. Other structures such as flagella, fimbriae and capsules may be present. Individual cells may be spherical, straight or curved rods, or spirals; they may form chains or masses, and some show branching with mycelium formation. They may produce various pigments including chlorophyll. Some form endospores.

Reproduction is chiefly by binary fission. They may be free living, saprophytic or parasitic; some are pathogenic to man, animals and plants.

BASAL (1) At, of, constituting a base; (2) of or constituting a basis; fundamental.

BASOPHIL (1) Of cells or cell contents - easily stained by basic dyes; (2) a basophil cell (eg. leucocyte)

BIOAEROSOL *Bios* - living; *Aerosol* - atomized particles suspended in air.

BIOCHEMICAL Chemical compounds, reactions etc., occurring in living organisms.

BIOLOGICAL Of or relating to living organisms.

BIOPSY Examination of tissue from a living organism to determine the cause or extent of a disease.

BRADYCARDIA Slow rate of heart contraction, resulting in a slow pulse rate. In febrile states, for each degree rise in body temperature, the expected increase in pulse rate is 10 beats per minute. When the latter does not occur, the term *'relative bradycardia'* is used.

BRONCHIOLE Any of the narrow tubes *(bronchi)* that carry air to and from the lungs.

BRONCHITIS Inflammation of the bronchi; may be primary or secondary, acute or chronic.

BRONCHOCONSTRICTION Swelling or narrowing of the bronchi - restricting the air flow.

BRONCHOPNEUMONIA Small areas of the lungs are consolidated and coalesce but do not have a lobular or lobar distribution. Complication of many medical conditions including measles and whooping cough, More common in infancy and old age.

BRUCELLOSIS A generalized infection resulting from one of a species of *Brucella.* there are recurrent attacks of fever and mental depression. The condition lasts for months.

BYSSINOSIS	Form of pneumonoconiosis due to inhalation of cotton or linen dust.
CANDIDA ALBICANS	A genus of fungi. Yeast-like cells that form some filaments. *C.Albicans* is a commensal of the mouth, throat, vagina, gut, and skin.
CARBACHOL	parasympathetic nervous system stimulant similar to acetylcholine but active orally. Given in postoperative retention of urine and intestinal atony and as eye drops for glaucoma.
CARBOXYHAEMOGLOBIN	A stable compound formed by the union of carbon monoxide and haemoglobin; the red blood cells lose their respiratory function.
CARCINOGEN	Any substance that is known to cause cancer.
CARDIOVASCULAR	System that comprises both the heart & blood vessels that circulate blood throughout the body.
CATARACTS	An opaque (non transparent) area in the lens of the eye.
CATECHOLAMINE	Secretion of human tumours. Estimation of C. in urine being carried out in research on hypertension and mental disorders. Experimental evidence suggests that C. plays an important role in mood regulation.
CELLULAR	(1) Of, relating to, or resembling a cell; (2) consisting of or having cells or small cavities.
CELLULOSE	A substance which is the main constituent of plant cell walls and is used in making paper, rayon & film.
CERVICAL ADENOPATHY	(*Cervical* = pertaining to; (1) the neck; (2) the cervix or neck of an organ) (*Adenopathy* = any disease of a gland especially a lymphatic gland).
CHEMOSIS	An oedema or swelling of the bulbar conjunctiva (adj. chemotic).
CHRONIC	(1) Continuing for a long time; constantly recurring; (2) disease which develops slowly, or is of long duration.

CIRRHOSIS	A type of permanent and progressive liver damage consisting of fibrous scars and nodules on the liver.
COGNITIVE	Mental process by which knowledge is acquired, including perception, intuition and reasoning.
COHORT	Statistically or demographically similar member or group.
COMA	State of unconsciousness from which a person cannot be aroused.
CONDUCTANCE	The ability of a system to conduct electricity, measured by the ratio of the current flowing through the system to the potential across it.
CONGENITAL	Existing from birth or before; born together with.
CONJUNCTIVA	Delicate mucous membrane that covers the eyeball and under the surface of the eyelid.
CONJUNCTIVITIS	Inflammation of the conjunctiva.
CONTAGIOUS	Communication of disease from body to body.
CORNEAL OPACITY	A non-transparent or cloudy membrane forming part of the anterior outer coat of the eye.
CORTEX	Outer layer of any organ or part such as the grey matter in the brain that covers the cerebrum (ceribal cortex).
CORTICOSTEROIDS	A steroid hormone that is produced in the outer layer (cortex) of the adrenal glands.
CORTISOL	One of the corticosteroids - also called hydrocortisone which promotes the synthesis and storage of glucose & regulates fat distribution in the body.
CORUNDUM	A hard mineral containing aluminium oxide, used as an abrasive.
CORYZA	An acute upper respiratory infection of short duration due to a filterable virus; highly contagious; attacks produce only temporary immunity.
COTININE	Metabolite of nicotine

D,

First isolated at Coxsackie N.Y. One of the three groups included in the family of enteroviruses. Divided into groups A & B. many of group A appear to be non-pathogenic. Others cause aseptic meningitis and herpangina. Those in group B also cause aseptic meningitis and Bornholm disease.

CREPITANT RÂLES

(*Crepitant* = crackling sound heard via stethoscope in lung infections). (*Râle* = abnormal sound heard on auscultation of lungs, when fluid is present in the bronchi).

CROUP

A household term for a group of diseases characterized by swelling and partial blockage of the entrance of the larynx. Occurs in children and is characterized by crowing respiration.

CUTANEOUS

Belonging to the skin.

CYANOSIS

A bluish discoloration of the skin caused by lack of oxygen in the blood.

CYSTITIS

Inflammation of the bladder.

CYTO-

A prefix meaning something connected with a cell or cells.

CYTOMEGALIC

(*Kytos* = Cell; *Megas* = large). See cytomegalovirus

CYTOMEGALOVIRUS

A group of viruses belonging to the herpovirus group. They are so-called from the swollen appearance of infected cells. They are responsible for the condition of cytomegalic disease in newborn. The disease may be characterized by jaundice and enlarged liver and spleen. In those who recover from this severe form of disease, there may be permanent retardation.

CYTOPATHEOGENIC

Cyto - pertaining to a cell; *Pathogenic* - a disease producing agent, usually restricted to a living agent.

DANDER

Skin scales

DECREMENT

(1) Act of decreasing; diminution; (2) *Mathematics* - a negative increment.

DEPRESSION Mental state characterized by feelings of guilt, lack of hope, melancholy and the general feeling that life is not worth living.

DERMATITIS Inflammation of the skin.

DIABETES Name given to two metabolic disorders: (1) *Diabetes insipidus* -kidneys are overactive or unable to reabsorb water passed to then by the blood; (2) *Diabetes mellitus* - sugar diabetes in which the body cannot make use of sugars in the normal way.

DIURNAL Happening during the day or daily.

DYSPNEA Breathlessness, the essential characteristic is that shortness of breath can occur without undue physical exertion.

ECHOVIRUS There are more than 30 types that occur in all parts of the world. The full name is *Enteric Cytopathogenic human Orphan*. They are responsible for outbreaks of meningitis, common-cold-like illnesses, gastrointestinal infections and infections of the respiratory tract.

ECZEMA Is a reaction of the skin to a wide range of irritants or stimulants. Two classical criteria of eruption in eczema are it itches and that causes vesication of the skin. The second stage is the formation of papules. If the condition persists, the skin tends to become thickened and starts to scale off.

EDEMA Localized or general swelling caused by a build up of fluids in the body tissues.

ELECTROCARDIOGRAPHY Recording of the electrical activity of the heart.

ELECTROLYTE A solution that conducts electricity.

ELECTROSTATIC Electricity that is not dynamic or flowing in a current.

EMERY A hard, greyish-black mineral consisting of corundum with either magnetite or haematite; used as an abrasive or polishing agent.

ENCEPHALITIS — Inflammation of the brain. It is usually caused by a virus infection, and may occur as a complication of the common infectious diseases including measles.

ENCEPHALOMYELITIS — Inflammation of the substance of both the brain and the spinal cord.

ENDEMIC — A term applied to diseases which exist in particular localities or among certain races.

ENDOCRINE SYSTEM — Network of ductless glands of internal secretion. The endocrine glands produce and/or store various hormones, which are secreted directly into the blood stream.

ENDOTOXIN — A toxic product of bacteria which is associated with the structure of the cell and can only be obtained by destruction of the cell.

ENTERITIS — Inflammation of the intestines.

ENTERIC — Relating to the intestines.

ENZYME — The name applied to a chemical ferment produced by living cells.

EOSINOPHIL — Any cell in the body with granules in its substance that stain easily with the dye eosin. About 2% of the white blood cells are eosinophils. Eosinophil cells are also present in the pituitary gland.

EPIDEMIC — Term applied to a disease which affects a large number of people in a particular locality at one time. In a sense, it is the opposite to endemic, which means a disease always found in a locality.

EPIDEMIOLOGY — The scientific study of the distribution of diseases.

EPIDERMAL — Relating to the epidermis, the external non-vascular layer of the skin; the cuticle. Also known as the *'scarf skin'*.

EPIDYDMIS — Name applied to an oblong body attached to the upper part of each testicle composed of convoluted vessels and ducts. It is liable to be a seat of tuberculosis and other inflammation.

EPIGASTRIC DISTRESS — Upset or inflammation of the region lying in the middle of the abdomen over the stomach.

EPILEPSY — Falling sickness, a term applied to a nervous disorder characterized by a fit of sudden loss of consciousness attended with convulsions.

ERYTHEMA — A general term signifying several conditions in which areas of skin become congested with blood, consequently a red eruption appears. The eruption is accompanied by tingling and, often, itching and pain.

ERYTHEMA INFECTIOSUM — Slapped cheek disease. Is characterized by a fiery red rash on the cheeks. The rash spreads to the rest of the body and lasts about a week. It is highly infectious.

ERYTHEMA NODOSUM — Thought to be a manifestation of tuberculosis. Children and young adults, especially women may suffer from a more severe form which begins as red blotches on the hands and spreads up the arms to the body, produces lumps and vesicles, or even large blebs of fluid.

ETIOLOGY — (Aetiology - alternative spelling). Means a group of conditions which form the cause of any disease.

EUSTACHIAN — (Tubes). Are passages, one each side of the face leading from the throat to the middle ear.

EXACERBATION — Exacerbate - make worse.

EXANTHEMATA — An old name used to classify the acute infectious diseases distinguished by characteristic eruptions.

EXUDATION — (1) Process in which some of the constituents of the blood pass slowly through the walls of the small blood vessels in the course of inflammation. (2) *Exudate* - accumulation resulting from the previous process.

FAUCES — Name given to the somewhat narrowed opening between the mouth and throat. It is bounded above by the soft palate, below by the tongue and on either side by the tonsil. In front of and behind the tonsil are two ridges of mucous membrane, the anterior and posterior pillars of the fauces.

FEBRILE — Feverish; accompanied by fever.

FEMUR — The thigh-bone; the longest and strongest bone in the body.

FEVER — An elevation of body temperature above normal. (syn., *pyrexia).* designates some infectious conditions.

FIBROBLAST — A cell which gives rise to connective tissue.

FIBROGENIC — *Fibro* - fibre, fibrous tissue; *Genic* - formation, origin.

FIBROSIS — Formation of fibrous or scar tissue, which is usually either due to infection or deficient blood supply.

FIBROTIC PNEUMOCONIOSIS — General name given to a chronic form of inflammation of the lungs which is likely to affect workers who constantly inhale irritating particles at work.

FOLLICLE — A name applied to a very small sac or gland (eg. small collections of adenoid tissue in the throat and small digestive glands on the mucous membrane of the intestine.

FORMITE — A term used to include all articles which have been brought into sufficiently close contact with a person sick from some infectious disease to retain the infective material and spread the disease.

FULMINANT — Developing quickly and with equally rapid termination.

FUNGUS — A low form of vegetable life including many microscopic organisms capable of producing superficial and systemic disease in man.

GASTROENTERITIS	Inflammation of mucous membranes of stomach and small intestine; although sometimes the result of dietetic error, the usual cause is bacterial infection.
GASTROINTESTINAL	Pertaining to the stomach and intestine.
GENE	The factor in the chromosome responsible for the transmission of hereditary characteristics.
GENETICS	The science which deals with the origin of the characteristics of an individual or the study of heredity.
GLYCOSURIA	Presence of sugar in urine. By far the most common cause of glycosuria is *diabetes mellitus*, but it may occur in exophthalmic goitre, certain other glandular disturbances, and occasionally following emotional stress.
GRAM-NEGATIVE BACTERIA	Bacteriological stain for differentiation of germs. Those retaining the stain are Gram-Positive (+), those unaffected are Gram-Negative (-).
GRANULATING	The outgrowth of new capillaries and connective tissue cells from the surface of an open wound.
GRANULOCYTOPENIA	Disease of granulocytes (polymorphs) not sufficient to warrant the term agranulocytosis.
GRANULOMA	The term applied to a tumour or new growth made up of granulation tissue. This is caused by various forms of chronic inflammation such as syphilis and tuberculosis.
GRIPPE	An alternative name given to influenza.
GYPSUM	Plaster of Paris; selenite.
HAEMOPOIESIS	The formation of blood (adj. haemoporetic)
HAEMOGLOBIN	Is the colouring material which produces the red colour of blood. It is a chromoprotein, made up of a protein called *globin* and the iron containing pigment called *haemin*. It's main function is to transport oxygen to the tissues.
HAEMOPTYSIS	The coughing up of blood.
HAEMORRHAGIC	The escape of blood from a vessel.

HAWTHORNE EFFECT — A term coined in reference to a series of productivity studies at the Hawthorne plant of the Western Electric Company in Chicago. The researchers discovered that their presence affected the behaviour of the workers being studies.

HEPATIC CALCIFICATION — Calcification of the liver

HEPATITIS — Inflammation of the liver.

HEPATOMEGALY — Enlargement of the liver.

HERPANGINA — A short febrile illness in childhood, in which minute vesicles or 'punched-out' ulcers develop in the posterior parts of the mouth. It is due to infection with Group A Coxsackie viruses.

HERPES — Vesicular eruption due to a virus infection.

HERPES LABIALIS — Form of Herpes Simplex, relating to blisters which appear around the mouth (cold sores).

HILUM — A depression on the surface of an organ where vessels or ducts enter and leave.

HISTAMINE — A naturally occurring chemical substance in the body tissues which, in small doses, has profound and diverse actions on muscle, blood capillaries and gastric secretion. Sudden excessive release is believed to cause the main symptoms and signs of anaphylaxis.

HOMOLOGUE — Corresponding in origin and structure.

HUMIDIFIER — Device to increase the moisture content in air.

HYGROSCOPIC — Tendency to absorb water from the air.

HYPERPLASIA — An overgrowth of cells that results in an increase in the size of an organ.

HYPERPNOEA — Rapid deep breathing; panting, gasping.

HYPERREACTIVITY — A state of unduly heightened reaction to a stimulus or allergin.

HYPERSENSITIVITY — Abnormally sensitive to an allergen, drug or other agent.

HYPERTENSION Abnormally high blood pressure.

HYPERTROPHY Increase in the size of tissues or structures, independent of natural growth. It may be congenital (pyloric stenosis), compensatory, complementary or functional.

HYPOPLASIA Defective or incomplete development of an organ or tissue.

HYPOTHALAMUS A neural control centre at the base of the brain concerned with hunger, thirst, satiety and other autonomic features.

HYPOTHESIS Suggested explanation for a group of facts or phenomena, either accepted as a basis for further verification or as likely to be true.

HYPOXIA Deficiency in the amount of oxygen delivered to the body tissues.

IGNEOUS Rocks produced by fire or heat.

IMMUNOSUPPRESSION That which prevents the occurrence of an immune reaction.

INDOMETHACIN A drug that is used in the treatment of gout and rheumatoid arthritis. It is also used in the treatment of a congenital heart abnormality known as *patent ductus arteriosus*.

INDUCTION The act of bringing on or causing to occur.

INFLUENZA An acute viral infection of the nasopharynx and respiratory tract which occurs in epidemic and pandemic form.

INSECT Small invertebrate segmented animal having a head, thorax, abdomen and three pairs of thoracic legs, usually with one or two pairs of thoracic wings.

INTUSSUSCEPTION A condition in which one part of the bowel slips into (invaginates) the lower part, causing an intestinal obstruction.

IONIZATION Breaking up of a substance in solution into its constituent ions.

ISCHEMIC HEART DISEASE	*Coronary thrombosis* - also coronary artery disease. Caused by narrowing of the arteries until they are unable to carry enough blood supply to the myocardium to function efficiently.
ISOLATE	Freed a substance from its combinations.
ISOMETRIC	Grouped in combination, of equal measure.
JAUNDICE	A yellow discoloration of the skin due to the deposition of bile pigment in the deeper layers. Infective jaundice is commonly due to a virus or infective hepatitis.
KERATOCONJUNCTIVITIS	Conjunctivitis due to inflammation of the cornea in front of the eye.
LABILE HYPERTENSION	Unstable; readily changed hypertension.
LACRIMATION	The outflow of tears; weeping.
LARYNGITIS	Inflammation of the mucous membrane of the larynx, may be either acute or chronic.
LARYNX	The organ of voice situated below and in front of the pharynx and at the upper end of the trachea.
LASSITUDE	Weariness, languor, disinclination to exert or interest oneself.
LATENT	Hidden, concealed, dormant.
LESION	Pathological change in a bodily tissue.
LEUCOCYTE	Term applied to the white blood cells.
LEUCOCYTOSES	Temporary condition in which the polymorphonuclear leucocytes in the blood are increased in number. It forms a valuable means of diagnosis in certain diseases.
LEUCOPENIA	A condition in which the white corpuscles of the blood are greatly reduced in number.
LEUKAEMIA	A disease in which the number of white corpuscles in the blood is permanently increased. The disease is characterized by a great enlargement of the spleen, changes in the bone marrow and enlargement of the lymph

glands all over the body. The condition may be acute or chronic according to the type of corpuscle present.

LIVER LOBULES These are about the size of a pin head and form each in itself a complete secreting unit. The liver itself is built up of many hundred thousands of exactly similar lobules.

LONGITUDINAL STUDY A study design involving the collection of data at different points in time, as contrasted with a cross sectional study.

LYMPHADENOPATHY Any disease of the lymph nodes.

LYMPHOCYTES A variety of white blood corpuscle produced in the lymphoid tissues of the lymphatic glands of the body.

LYMPHOCYTOSIS Lymphocythaemia - excess of lymphocytes in the blood.

LYMPHOID Collection of tissues in the body consisting of the thymus, marrow of the bone, spleen, lymph nodes and other lymphoid tissue.

LYMPHOSARCOMA Malignant cancerous growth connected with the lymphoid tissue. The symptoms are similar to those of lymphatic leukaemia.

MACROPHAGE A phagocytic cell, which plays an important part in the organization and repair of tissue.

MALAISE Feeling of unease, mild sickness or depression.

MALARIA A tropical disease caused by one of the genus *Plasmodium* and carried by infected mosquitos of the genus *Anopheles*.

MARROW The soft, pulpy substance present in the bones. *Red marrow* is present in the cancellous tissue of the bones and is concerned with blood formation. *Yellow Marrow* is a fatty substance present in the shafts of long bones.

MAST CELL Any of a number of cells in the connective tissue that release heparin, histamine, and serotonin during inflammation & allergic reactions.

MENINGES	The surrounding membranes of the brain and spinal cord. There are three in number: (1) the dura mater (outer); (2) arachnoid (middle); (3) pia mater (inner).
MENINGITIS	Inflammation of the meninges.
MENINGOENCEPHALITIS	Inflammation of the meninges and the underlying brain matter. One form is caused by *Naegleria Fowleri* and is contracted through bathing in contaminated water.
MENISUS	(1) A semi-lunar cartilage; (2) Curved upper surface of a column of liquid.
MENORRHAGIA	Mensuration that involves greater than normal blood flow that usually lasts longer than normal.
MESENTERIC ADENITIS	Inflammation of a gland or lymph node.
METABOLIC	Sum of the chemical and physical processes that occur within the body. It includes repair or replacement of tissues and the production of energy.
METABOLITE	Any produce of metabolism. *'Essential Metabolite'* is any substance necessary for proper metabolism (eg. vitamins).
METHACHOLINE	A derivative of acetylcholine. It's main use is in the treatment of *paroxysmal atrial tachycardia.*
MICA	Group of minerals consisting of hydrous silicates of aluminium, potassium etc., in monoclinic crystalline form occurring in igneous & metamorphic rock.
MICRO-ORGANISM	Any organism of microscopic size such as a virus.
MICRON	A unit of length equal to 10^{-6} metre. It has largely been replaced by the *micrometre* the equivalent SI unit.
MILIARY	Resembling a millet seed. *'M.Tuberculosis'* a form of TB in which, TB nodules are widely disseminated throughout the organs and tissues of the body.

MOLLUSCULUM	A soft tumour. *'M. Contagiousum'* an infectious type of wart which appears on the skin as a waxy papule.
MONOMER	A compound whose molecules can join together to form a polymer.
MONONUCLEOSIS	An increase in the number of circulating monocytes (mononuclear cells) in the blood. *Infective Mononucleosis* is a virus infection with M. as a clinical sign.
MUCOCILIARY	*Cilia* - microscopic hair-like projections from certain epithelial cells. Membranes containing such cells are known as ciliated membranes (eg. those lining the trachea).
MUCOPURULENT	Containing mucus and pus.
MUCOSTASIS	*Muco* - mucus; *Stasis* - stagnation, cessation of movement.
MUCOUS MEMBRANE	Mucus-secreting membrane that lines the cavities or passages that are open to the external environment.
MUMPS	An acute, specific inflammation of the parotid glands, caused by a virus.
MYOCARDITIS	Inflammation of the myocardium (heart).
MYCOBACTERIUM	Small slender rod bacteria. Gram-positive and acid fast, both to a varying degree. Saprophytic, commensal and pathogenic species. *M. Tuberculosis* causes TB, *M.Lepra* causes leprosy.
MYCOPLASMAL PNEUMONIA	A small organism intermediate in size between viruses and bacteria.
MYCOTIC	Disease caused by any fungus.
MYOCARDIAL INFARCTION	Heart attack caused when a section of the heart is suddenly deprived of its blood supply.
MYOCARDITIS	Inflammation of the heart muscle.

MYXOVIRUS	Includes the influenza viruses A, B & C and para-influenza viruses types 1-3; and respiratory syncytial virus which is an important cause of respiratory disease in the early years of life.
NARCOSIS	Unconsciousness usually induced by narcotics or anaesthetics.
NASOPHARYNGITIS	Inflammation of the nasopharynx.
NEBULIZER	An apparatus for converting a liquid into a fine spray.
NEONATE	A newborn baby up to one month old.
NEONATORUM	Pertaining to the newborn.
NEOPLASIA	Literally the formation of new tissue. By custom it refers to the pathological process in tumour formation
NEPHROSIS	Any disorder of the kidney caused by a degenerative process other than infections.
NEURAL	Relating to a nerve or nervous system.
NEURALGIA	Pain in the distribution of a nerve.
NEUROLOGY	Study of the anatomy, physiology and diseases of the nervous system.
NEUROSES	A relatively mild mental disorder characterized by hysteria, anxiety, depression & obsessive behaviour.
NICOTINE	Poisonous alkaloid extracted as an oily liquid from tobacco.
NODULAR	(*Node)* a protuberance or swelling.
NODULE	A small node.
NORADRENALINE	Is a precursor of adrenaline in the medulla of the suprarenal glands. It is also present in the brain. It's main function is to mediate the transmission of impulses in the sympathetic nervous system. It has a transmitter function in the brain and also has an intense vasoconstrictor action.

OBSESSIVE BEHAVIOUR — Two types: (1) Obsessive compulsive thoughts; constant preoccupation with recurring morbid thought which cannot be kept out of the mind, and enters against the wishes of the patient who tries to eliminate it. The thought is almost always painful and is out of keeping with the persons normal personality. (2) Obsessive compulsive actions; consists of a felling of compulsion to perform repeatedly a simple act (eg. hand-washing, touching door knobs, etc). Ideas of guilt frequently form the basis of an obsessional state.

OLFACTORY — Pertaining to the sense of smell.

OPTIC — Pertaining to sight.

ORCHITIS — Inflammation of the testicle.

ORGANIC — Pertaining to an organ. associated with life. *O.Disease* is one in which there is a structural change.

OROPHARYNX — (1) That portion of the pharynx which is below the level of the soft palate and above the level of the hyoid bone; (2) pertaining to the mouth and pharynx.

OSTEITIS — Inflammation of the bone. *'O.Fibrosa'* excessive parathyroid secretion and absorption of calcium from the bone.

OXIDIZED — The process of converting a substance into an oxide by addition of oxygen.

PHARYNGITIS — Inflammation of the pharynx.

PHARYNX — The cavity at the back of the mouth. It is cone shaped, about 3 inches long and is lined with mucous membrane, at the lower end is the oesophagus.

PANDEMIC — An infection spreading over the whole country.

PAPILLAE — Multiple nipple-shaped eminence. *Circumvallate P.* a large papillae at the tip of the tongue. *Fungiform P.* is a papillae shaped fungus, found on the dorsocentral area of the tongue. *Renal P.* the summit of one of the renal pyramids.

PAPOVIRUS	A group of viruses one of which is responsible for warts.
PAPULE	Pimple.
PAPULOPUSTULE	Pus filled pimple.
PARAINFLUENZA	Caused by the parainfluenza virus all of which cause infection in the respiratory system.
PARALYTIC	Loss of muscular power due to interference with the nervous system.
PARANASAL PASSAGE	Near the nasal cavities, as the various sinuses.
PARESIS	A state of slight, temporary paralysis.
PARAESTHESIA	Any abnormality of sensation.
PAROTITIS	Inflammation of the paratoid gland (ie. salivary gland situated in front of and below the ear).
PATHOGEN	A disease producing agent, usually restricted to a living agent.
PATHOGENESIS	The origin and development of disease.
PATHOLOGY	The science which deals with the causes of, and changes produced in the body by disease.
PEPTIC ULCER	A non-malignant ulcer in those parts of the digestive tract which are exposed to the gastric secretions; hence usually in the stomach and duodenum.
PERICARDITIS	Inflammation of the outer serous covering of the heart. It may or may not be accompanied by an effusion and formation of adhesions between the two layers.
PERIORBITAL	*Peri* - around; *Orbit* - bony socket containing the eyeball and its appendages (adj. orbital).
PERENNIAL	Lasting through all seasons of the year.
PETECHIAE	A small haemorrhagic spot.
PHLEGM	The secretion of mucus expectorated from the bronchi.

PHOSPHATE	A salt of phosphoric acid.
PHYSIOLOGICAL	In accordance with the natural processes of the body.
PLATELET	Blood platelets, or thrombocytes are small spherical bodies which play an important part in the process of blood coagulation. Normally there are about 500,000 per cubic millilitre of blood.
PLEURA	Is the name of the membrane on either side of the chest and it forms a covering for one lung. The two pleura are distinctive though they touch one another for a short distance behind the breast bone.
PLEURACY	Inflammation of the pleura.
PLEURODYNIA	Intercostal myalgia or muscular rheumatism (fibrositis). It is a feature of Bornholm disease.
PNEUMONIA	Inflammation of the lung with production of alveolar exudate.
POLIOMYELITIS	Infantile paralysis. An epidemic virus infection which attacks the anterior motor neurones of the anterior horns of the brain stem.
POLIOVIRUS	Virus which causes poliomyelitis.
POLLEN	Microscopic spores formed in flowering plants and conifers. Implicated in allergic reactions amongst a portion of the population.
POLYMER	Compound formed by chemical addition from a number of identical molecules each which consists of a number of identical units.
POLYMERIZATION	Formation of a polymer by simple addition of a number of identical smaller molecules.
POLYNEURITIS	Inflammatory condition of the nerves in various parts of the body.
POLYP OR POLYPUS	A pedunculated tumour arising from any mucous surface (eg. cervical, uterine, nasal, etc.). usually benign but may become malignant.

PORPHYRIA	An inborn error in porphyrin metabolism, probably hereditary, causing pathological changes in the muscular and nervous tissue.
LYMPHADENOPATHY	Any disease of the lymph nodes.
PROSTRATE	A small conical gland at the base of the male bladder and surrounding the first part of the urethra.
PROTEIN(S)	Nitrogen compounds found in all animal and vegetable tissues. They are built up of amino-acids and are essential for growth and repair of body tissues.
PROTEOLYSIS	The braking down of proteins into simpler substances (adj. proteolytic).
PROTOZOA	The smallest type of animal life; unicellular organisms.
PRURITUS	Itching. *P.Ani* and *P.Vulvae* are considered to be psychosomatic conditions, except for a few cases where a local cause can be found.
PSYCHODYNAMIC(S)	The science of the mental processes, especially the causative factors in mental activity.
PSYCHOLOGY	A branch of science that deals with the mind and it's methods of working.
PSYCHOSOMATIC	Illness where emotional factors produce physical symptoms.
PULMONARY	Relating to the lung.
PULMONARY GRANULOMATOSIS	*Pulmonary* - pertaining to the lungs; Granulomatosis - a tumour formed of granulation tissue.
PURPURA	A disease characterized by the occurrence of purple-coloured spots on the surface of the body due to extravasations of blood in the skin, associated occasionally with haemorrhages from mucous membranes.
PURULENT	Pertaining to or resembling pus.

PYREXIA Fever; elevation of the body temperature above normal.

RADON The gaseous inert element (Rn-222) in the U-238 decay series. The immediate parent of Po-218 (RaA).

RADON DAUGHTERS The four short-lived elements which succeed radon in the U-238 decay series. These include Po-218 (RaA), Pb-214 (RaB), Bi-214 (RaC), and Po-214 (RaC^1).

RÂLES Abnormal sound heard on auscultation of lungs, when fluid is present in the bronchi.

RAYNAUD'S DISEASE A condition in which the circulation becomes suddenly obstructed in outlying parts of the body. It is predominantly a disease of young women and may result in cyanosis of fingers and toes, ending in gangrene.

REACTIVITY (1) Produce reciprocal or responsive effect; (2) respond to stimulus, undergo change due to some influence (react, reactive).

RELATIVE HUMIDITY Amount of moisture in the atmosphere as compared to that of complete saturation at a given temperature.

REOVIRUS *Respiratory enteric orphan virus.*

RETINA The light sensitive internal coat of the eyeball, consisting of 8 superimposed layers, seven of which are nervous and one pigmented.

SARCOMA A malignant tumour arising in the connective tissue.

RHEUMATOID SPONDYLITIS A condition characterized by ossification of the spinal ligaments and ankylosis of the sacro-iliac joints. It occurs chiefly in young men.

RHINITIS Inflammation of the mucus membrane of the nose.

RHINORRHEA Nasal discharge.

RICKETTSIA Small pleomorphic parasitic microorganisms which have their natural habitat in the cells of the gut of arthropods. Some are pathogenic to

mammals and man, in whom they cause the typhus group of fevers. They are smaller in size than bacteria and larger than viruses. Many of their physiological characteristics resemble bacteria, bit like viruses they are obligate intercellular parasites.

RIGORS — A sudden chill, accompanied by severe shivering. The body temperature rises rapidly and remains high until perspiration ensues and causes a gradual fall in temperature.

ROSEOLA INFANTUM — An acute infectious disease of infancy with an incubation period of 10-15 days.

SALMONELLA — A genus of bacteria. gram negative rods. Parasitic in many animals and man in whom they are often pathogenic. Usual mode of entry is through contaminated food.

SAPROPHYTIC — Free living micro-organisms obtaining food from dead and decaying animal and plant tissue.

SCLERODERMA — A disease in which localized oedema of the skin is followed by hardening, atrophy, deformity and ulceration. Occasionally it becomes generalized producing immobility.

SEROLOGIC — The branch of science dealing with the study of sera.

SERUM — A fluid which separates from blood, lymph and other body fluids when clotting takes place in them.

SINK — Relating to buildings - an object which captures pollutants and re-releases them back into the atmosphere when conditions are favourable (eg. carpet, porous wall lining, etc.)

SINUSITIS — Inflammation of the sinuses; used exclusively for the paranasal sinuses.

SOMATIC — Pertaining to the body as opposed to the mind. *'S.Nerves'* control the activity of the striated skeletal muscle.

SOMNOLENCE — *'Somnambulism'* sleep walking.

SPLEEN	A lymphoid, vascular organ immediately below the diaphragm, at the tail of the pancreas, behind the stomach. It is enlarged in severe infection and is not essential to life.
SPLENOMEGALY	Enlargement of the spleen.
SPORE(S)	A phase in the life cycle of a limited number of bacterial genera where the vegetative cell becomes encapsulated and metabolism almost ceases. These spores are highly resistant to environmental conditions such as heat and desiccation. The spores of important species such as *Clostridium tetoni* and *Cl. botulinum* are ubiquitous so that sterilization procedures must ensure their removal or death.
SPUTUM	Matter which is expectorated from the lungs.
STENOSIS	A term applied to a condition of unnatural narrowing of any passage or orifice of the body. The term is especially used in connection with the four openings of the heart at which the valves are situated.
STRAIN	Pull, stretching force, tension, demand upon or force that tries cohesion or strength or stability or resources, exertion required to meet such demand, injury or change of structure resulting from such exertion.
STRESS	Constraining or impelling force.
STROKE	Apoplexy resulting from a vascular accident in the brain, resulting in hemiplegia.
STUPOR	A state of marked imparement but not complete loss of consciousness.
SUB MICRON	(Particles) less than 1 μm in diameter.
SUBACUTE	Moderately severe. Often the stage between the acute and chronic phases of a disease.
SUBCUTANEOUS TISSUE	Beneath the skin.
SUBSTERNAL	Beneath the breast bone.
SYMPATHETIC NERVOUS SYSTEM	A portion of the autonomic nervous system. It is composed of a chain of ganglia on either side of

	the vertebral column in the thoracolumbar region and sends fibres to all plain muscle tissue.
SYNERGY	The harmonious working together of two agents, such as drugs, microorganisms, muscles, etc.
SYSTEM	A collection of organs in performance of a common duty (adj. systemic).
TEMPORAL	Relating to the temple.
TERMINAL BRONCHIOLES	Portion of the bronchi which terminates in the alveoli or the air sacs of the lungs.
THERMOPHILIC ACTINOMYCETES	*Thermophilic* - heat resisting. *Actinomyces* - a genus of parasitic fungus having a radiating mycelium. Also called a 'ray fungus'.
THORAX	The chest cavity.
THORON	Rn-220 the gaseous inert element in the Th-232 decay series. The immediate parent of Po-216 (ThA), the isotope of radon.
THORON DAUGHTERS	The short lived daughters of Rn-220, part of the Th-232 decay series. Only ThB, ThC, and ThC^1 are of consequence in the potential health hazard. ThB, although not an alpha transmitter itself, has a half life of 10.6 hours, and is therefore the most abundant source of atoms available for decaying through the alpha emitting successors, ThC and ThC^1.
TIBIA	The shin bone; the larger of the two bones in the lower part of the leg; it articulates with the femur, fibula and talus.
TISSUE CULTURE	The development of micro-organisms on tissue under ideal conditions of growth.
TONSILS	The small bodies on each side, covered by mucous membrane, embedded in the fauces; composed of about 10-18 lymph follicles (adj. tonsillar).
TOXAEMIA	A generalized poisoning of the body by products of bacteria and damaged tissue.

TOXIC-IDIOSYNCRATIC	*Toxic* - poisonous; *idiosyncratic* - peculiar variation of constitution or temperament. Unusual individual response to certain proteins, drugs, etc.
TOXICOLOGY	The science dealing with poisons.
TRACHEA	The windpipe.
TRACHEITIS	Inflammation of the trachea.
TRANSPLACENTAL	Through the placenta.
TROPOSPHERE	Layer of atmospheric air extending about 7 miles upwards from the earth's surface, in which the temperature falls with height.
TUFF	Kinds of volcanic fragmentary rock.
TUMOUR	A mass of abnormal tissue in a structure which resembles the normal tissue but fulfils no useful function and grows at the expense of the body.
TYMPANIC MEMBRANE	Eardrum.
UBIQUITOUS	Omnipresent; being everywhere or in an indefinite number of places at the same time.
ULCER	A breach of the surface of the skin or the surface of the membrane lining of any cavity within the body which does not heal quickly.
URAEMIA	A clinical syndrome due to renal failure resulting from either disease of the kidneys themselves or from disorder or disease elsewhere in the body which induces kidney disfunction, and which results in gross biochemical disturbance in the body, including retention of urea and other nitrogenous substances in the blood. Depending on the cause it may or may not be reversible. The fully developed syndrome is characterized by nausea, headache, hic-cough, weakness, dimness of vision, convulsions and coma.
URTICARIA	Nettle rash or hives. An allergic skin eruption.
VARICELLA-ZOSTER	Chickenpox.

VASOACTIVE An agent which acts on the blood vessels. *Vasoconstrictor* - an agent which causes a narrowing of the lumen of blood vessels. *Vasodilator* - agent which causes widening of the lumen of the blood vessels.

VASOMOTOR NERVES Nerves which cause changes in the calibre of the blood vessels.

VENTRICULAR Relating to the *Ventricle* - one of the two lower chambers of the heart

VERRUCAE Non-venereal warts on the genitals.

VIRULENCE Infectiousness; the disease producing power of the microorganism; the power of the microorganism to overcome host resistance.

VIRUS An ultra-microscopic, filter-passing microorganism, parasitic within living cells, and of which many forms cause disease in human beings. It uses the cell enzymes to manufacture more viruses. The cell ruptures, liberating these. They invade and destroy more cells. They are classified into eight groups: (1) *Poxviruses*, which cause skin lesions; (2) *Myxoviruses*, so named because of their affinity to mucus; (3) *Herpesviruses*, named from the creeping lesion of shingles; (4) *Adenoviruses,* first found in lymphatic (adenoid) tissue; (5) *Reoviruses*, named because they cause respiratory and enteric infections; (6) *Arborviruses*, because they are arthropod-bourn; (7) *Picornaviruses*, meaning very small, includes enteroviruses and rhinoviruses; (8) *Papovaviruses*, includes Groups 2, 5, 6, 7 R.N.A. containing viruses. Groups 1, 3, 4, 8 D.N.A. containing viruses.

VISCERA The internal organs.

VOLATILE Evaporating rapidly.

VULVOVAGINITIS Inflammation of the vulva and vagina.

XYLENE A clear inflammable liquid resembling benzene.

ZOONOTIC Disease in man transmitted from an animal vector.

APPENDIX 3

APPENDIX 3. VENTILATION

Natural Ventilation

Natural ventilation is a combination of intentional and unintentional air flow through the building.

Unintentional air flow is called infiltration. It is the flow of air through cracks, interstices, and unintentional openings in the building envelope.

Intentional natural ventilation is under the manual control of the building occupants. It occurs through operable windows, doors, skylights, roof ventilators, stacks, and other planned inlet and outlet openings. It can be classified as 'controlled' infiltration/exfiltration.

Natural ventilation is more likely to occur during periods of moderate to warm outdoor weather conditions. During these periods, infiltration is at a minimum and building occupants may open windows and other intentional openings to reduce indoor temperatures or create air motion to increase thermal comfort (Goldish, 1989).

The quantity of air flowing through openings depends on the dynamic pressure of the wind and buoyancy forces resulting from indoor/outdoor temperature differences. The interaction of these forces are summarised as follows.

The wind effect is due to the airflow around the building creating regions in which the static effect is either greater (positive) of less than (negative) the pressure inside the building. Simplistically, wind pressures are positive on the windward side of the building and negative on the leeward side. Negative pressures also exist across the top of the building. Positive pressure differentials result in infiltration and negative pressure differentials result in exfiltration.

The buoyancy force, or stack effect, is a result of temperature differences between inside and outside of the building. During the heating season, the warmer air inside is less dense and rises and exits the building near its top. It is replaced by cooler outside air, which flows in near the bottom of the building. The stack effect increases with increasing building height and can be exceptional in tall buildings with vertical passages such as elevator shafts and stairwells.

The degree of air exchange due to infiltration or natural ventilation is dependent on the magnitude of the two driving forces. Infiltration is greatest during the colder months when indoor/outdoor temperature differences are greatest. During warm weather periods, when natural ventilation is most likely to be used, the indoor/outdoor temperature differences are smallest, and the stack effect is minimal and the wind effect is the dominant infiltration mechanism. Buildings that rely on natural ventilation to supply their ventilation air are at risk during periods of stagnant conditions.

Mechanical Ventilation

Mechanical ventilation is the forced movement of air by fans into and out of a building. The primary purpose of the mechanical ventilation system is to provide a healthy and comfortable indoor environment for building occupants.

Most HVAC systems are designed to operate on a minimum of outside air averaging 10% to 20% of total airflow. Through intentional control of the dampers, or even failure of the damper control, the percentage of outside air introduced to the ventilation system can range from 0% to 100% (Goldish,1989).

The two most commonly used HVAC systems are the constant-air-volume (CAV) system and the variable-air-volume (VAV) system. In the CAV system, the supply air is distributed to spaces requiring ventilation and conditioned air through a series of ducts with inlet fans in the ceiling. Returned air is drawn from the conditioned space through ceiling outlets into a large return air plenum. Dampers can be operated to control the amount of intake and exhaust air and the percentage of air that is recirculated.

Temperature of the occupied space is controlled by regulating the temperature of the supply air. Proper operation of the dampers/damper systems is critical for supplying sufficient ventilation air.

VAV systems, regulate temperature in the occupied space by varying the amount of air supplied to the space. This type of system is attractive because of the potential energy savings. However, it has two major drawbacks. First, it is difficult to ensure that minimum outdoor ventilation air rates for each zone are being satisfied. Second, the system is designed/operated to fully close dampers when the desired temperature is reached. Consequently complaints of poor indoor air quality frequently arise (Goldish,1989).

APPENDIX 4

APPENDIX 4. VOLATILE ORGANIC COMPOUNDS

The following chemical information has been referenced from Sax (1974).

Baechler (1990), stated that the various volatile organic compounds can be placed in six groups according to structural similarities. These are as follows:

(1) Aliphatic and oxygenated aliphatic hydrocarbons;
(2) Halogenated hydrocarbons;
(3) Aromatic hydrocarbons;
(4) Alcohols;
(5) Ketones;
(6) Aldehydes.

(1) ALIPHATIC AND OXYGENATED ALIPHATIC HYDROCARBONS

This group is sometimes referred to as *open chain hydrocarbons*. Aliphatic hydrocarbons are generally biologically and chemically inert. That is, they produce no detectable functional subclinical alterations. Except for CH_4 and C_2H_6 the gaseous lower members of the series have anaesthetic properties.

Alicyclic hydrocarbons *(cyclohexane; methyl cyclohexane; etc.)* resemble aliphatic hydrocarbons toxilogically. They are generally CNS depressants and anaesthetics with a low order toxicity. They do not tend to accumulate in body tissue and so it is considered that cumulative toxicity from repeated exposure to low concentrations is improbable.

The *olefins* or unsaturated aliphatic hydrocarbons do not differ greatly from paraffins insofar as their toxic effect is concerned. C_2H_4 and some of its homologues occur in manufactured and C gases. C_2H_4 can be used as an anaesthetic, and when inhaled in sufficient quantity, it can be an asphyxiant. Prolonged or repeated exposures to high concentrations of various olefins have caused certain toxic effects in animals, such as liver damage and hyperplasia of the bone marrow but no corresponding effects have been evident in humans. The diolefins, C_4H_6 and C_5H_8, are more irritating than the paraffinic olefins or monoolefins of the same volatility. In general, it may be stated that the olefins are *comparatively* innocuous materials.

(2) HALOGENATED HYDROCARBONS

These materials from a chemical standpoint are hydrocarbons in which one or more of the hydrogen atoms have been replaced with a halogen, generally CI or Br, but this could include I or F as well. Toxilogically, the halogenated hydrocarbons are generally considered to be more toxic than the nonhalegonated hydrocarbons. The toxic effects are relatively varied and a sample of some of the specific members of the group is considered:

Chloroform $CHCl_3$ (Synonymn: *trichloromethane)* is a colourless, heavy liquid with a distinctive odour. It causes irritation of the conjunctiva. Upon inhalation, it causes dilation of the pupils with reduced reaction to light, as

well as reduced intraocular pressure. The material is a well known anaesthetic. In the initial stages there is a feeling of warmth in the face and body, then an irritation in the mucous membranes and skin followed by nervous abberations. Prolonged inhalation will bring paralysis accompanied by cardiac and respiratory failure and finally death.

It has been widely used as an anaesthetic. However, due to its toxic effects its use has been abandoned. The harmful chronic effects are narcosis and damage to the heart. Also prolonged administration as an anaesthetic may lead to such serious effects as profound toxaemia and damage to the liver, heart and kidneys, as well as the fact that it is a suspected carcinogen.

Experimentally, prolonged but light anaesthesia in dogs produces a typical hepatitis. Inhalation of concentrated chloroform vapour results in irritation of the mucous surfaces exposed to it. The narcosis is ordinarily preceded by a stage of excitation which is followed by loss of reflexes, loss of sensation and finally loss of consciousness.

Dichloromethane CH_2Cl_2 (Synonym: *Methylene Chloride)* is a colourless, volatile liquid. It is very dangerous to the eyes and has the property of inducing narcosis. Its narcotic effects are quiet strong. It has been used in Europe as an anaesthetic and is still used as a local anaesthetic. At a level of 2300 ppm for one hour, there was no feeling of dizziness, but nausea was noted in subjects after 30 minutes. The limit of perception by smell is between 25-50 ppm. Prolonged skin contact can cause dermatitis.

Polychlorinated biphenyls (Synonym: *Arochlors)* are mostly used as electrical insulators, plastisizers and in printing for microencapsulation of dyes in carbonless duplicating papers. The acute toxicity of PCB is relatively low. The materials do have significant toxicity, however, on repeated exposure. The subacute and chronic effects include edema formation, microsomal enzyme induction, porphyrogenic action, estrogenic activity, and immunosuppression.

Trichloroethylene $CHClCCl_2$ (Synonym: *Ethinyl trichloride; ethylene trichloride)*. Inhalation of high concentrations causes narcosis and anaesthesia. A form of addiction has been observed in people who are continually exposed. prolonged inhalation of moderate concentrations causes headaches and drowsiness. Fatalities following severe, acute exposure have been attributed to ventricular fibrillation resulting in cardiac failure. There is some question as to the damage to liver or other organs from chronic exposure.

Chlorobenzene C_6H_5Cl (Synonym: *Chlorinated benzene; monochlorobenzene; phenyl chloride)* is a clear, volatile liquid with an almond-like odour. it has a low acute local or chronic toxicity. It is moderately toxic systemically by ingestion, inhalation or skin absorption. It is a fairly strong narcotic and possesses slight irritant qualities. acute exposures result in somnolence, loss of consciousness, twitching of the extremities, cyanosis, deep rapid respiration and a small irregular pulse. The substance has carcinogenic implications.

Carbon tetrachloride CCl_4 (Synonym: *Perchloromethane; tetrachloromethane)* is a colourless liquid with a heavy, ethereal odour. On a systemic basis it is both acute and chronic. The material is highly toxic by ingestion, inhalation and skin absorption, and on a chronic basis it is an irritant.It has a narcotic action resembling that of chloroform, though not as strong. Following exposure to high concentrations, the victim may become unconsciousness, and if exposure is not terminated, death can follow from respiratory failure. In the cases of narcosis that recover, the after effects are more serious than those of delayed chloroform poisoning, usually taking the form of damage to the kidneys, liver and lungs. Exposure to lower concentrations, insufficient to produce unconsciousness usually results in severe gastro-intestinal upset and hepatic damage. The kidney lesion is an acute nephrosis; the liver involvement consists of an acute degeneration of the central portions of the lobules. Where recovery takes place, there may be no permanent disability. Marked variation in individual susceptibility to this material exists, some persons appearing to be unaffected by exposure which seriously poison fellow workers. Alcoholism and previous liver and kidney damage seem to render the individual more susceptible. Concentrations in the order of 1,000 to 1,500 ppm are sufficient to cause symptoms if exposure continues for several hours. Repeated daily exposure to such concentrations may result in poisoning.

The common form of poisoning is usually one of gastrointestinal upset, which may be followed by renal damage, cases have been reported in which the CNS has been affected, with production of polyneuritis, narrowing of the visual fields and other neurological changes. Prolonged exposure to small amounts of CCl_4 has also been reported as causing cirrhosis of the liver.

Locally, a dermatitis may be produced following repeated contact with the liquid. The skin oils are removed, the skin becomes red, cracked and dry. The effect of CCl_4 on the eyes, either as a vapour or as a liquid, is one of irritation, lacrimation, and burning.

Industrial poisoning is usually acute, with malaise, headache, nausea, dizziness, and confusion, which may be followed by stupor and sometimes loss of consciousness. Symptoms of liver and kidney damage may follow later, with the development of dark urine, sometimes jaundice and liver enlargement, uraemia may develop and cause death. Where exposure has been less acute, the picture is one of headache, dizziness, nausea, vomiting, epigastric distress, loss of appetite and fatigue. Visual disturbances (blind spots, spots before the eyes, a visual 'haze' and restriction of the visual fields) secondary anaemia and slight jaundice may occur. Dermatitis may be noticed on exposed areas.

The material is sometimes used as an insecticide.

Dichlorobenzene $C_6H_4Cl_2$ (Synonym: *Paracide; PDB)* is an organic solvent with a strong characteristic odour. On an acute basis this material is a mild irritant and allergin. It is moderately toxic by inhalation or ingestion. On an acute systemic basis it is moderately toxic by ingestion. On a chronic local

basis it is mildly allergenic and moderately irritant. On a chronic systemic basis it is toxic by ingestion and inhalation. Liver injury, pulmonary granulomatosis, anaemia, granulocytopenia and mucous membrane irritation has been reported. It is suspected of producing lenticular cataracts in humans. The material is sometimes used as an insecticide to kill moths, cockroaches, etc.

(3) AROMATIC HYDROCARBONS

Hydrocarbons of the aromatic classification are biochemically active. The vapours are much more irritating to the mucous membranes than equivalent concentrations of aliphatic or alicyclic hydrocarbons, and systemic injury can result from exposure to them.

Ethyl Benzene $C_6H_5C_2H_5$ (Synonyms: *Ethylbenzol; phenylethane*) is a colourless liquid with an aromatic odour. As a liquid, it is an irritant to the skin and mucous membranes. A concentration of 0.1% of the vapour in air is an irritant to the eyes of humans, and concentration of 0.2% is extremely irritating at first, then causes dizziness, irritation of the nose and throat and a sense of constriction in the chest. Exposure of guinea pigs to 1% concentration has been reported as causing ataxia, loss of consciousness, tremor of the extremities and finally death through respiratory failure. The pathological findings were congestion of the brain and lungs, with edema. No data are available regarding the effect of chronic exposure. Erythema and inflammation of the skin may result from contact with liquid. The irritant properties are sufficient to cause those exposed to be unable to remain in an atmosphere containing as little as 0.5% of the vapour.

iso-Propyl Benzene $c_6H_5CH(CH_3)_2$ (Synonyms: *Cumene; 2-Phenyl propane*). This compound has a potent narcotic action characterized by a slow induction period, although the effects are of long duration. It is a CNS depressant and the minimum lethal concentration for mice is 2,000 ppm. The long duration of its action indicates a possible slow rate of elimination, meaning that possible cumulative effects must be considered. It is thought to have greater acute toxicity than benzene or toluene.

Styrene Monomer $C_6H_5CHCH_2$ (Synonyms: *Vinyl benzene; phenyl ethylene; Cinnamene*). This material is moderately irritating and moderately toxic by ingestion and inhalation. It can cause irritation, violent itching of the eyes, lacrimation, and severe human eye injuries. Its toxic effects are usually transient and result in irritation and possible narcosis. It is not considered to be a particularly toxic material, because under ordinary conditions it does not vaporize sufficiently to reach a concentration that can kill animals. Experimentally, it has been found that 10,000 ppm was dangerous to animal life in from 30-60 minutes, 2,500 ppm was dangerous to life in 8 hours, while 1,300 ppm was the highest amount which was found to cause no systemic disturbances in 8 hours. however, all animals exposed to these amounts did evidence eye and nasal irritation, while those exposed to 2,500 ppm or more

showed varying degrees of weakness and stupor, followed by incoordination, tremors and unconsciousness. from a study to determine the chronic effects of the material, it was discovered that rats exposed to 1,300 ppm for a 7-8 hr/day, 5 days a week, for 26 weeks showed evidence of definite signs of eye and nasal irritation.

Toluene $C_6H_5CH_3$ (Synonyms: *Methylbenzene; phenylmethane; toluol*) is a colourless liquid with a benzol-like odour. It is derived from coal tar, and commercial grades contain small amounts of benzene (see benzene) as an impurity. Acute poisoning, resulting from exposure to high concentrations of the vapours are rare. Inhalation of 200 ppm of toluene for 8 hours (see further discussion on toluene elsewhere) may cause impairment of coordination and reaction time. With higher concentrations, up to 800 ppm, these effects are increased and are observed in a shorter time. In a few cases of acute toluene poisoning reported, the effect has been that of a narcotic, the victim passing through a stage of intoxication into one of coma. Recovery following removal from exposure has been the rule. There has been an occasional report of chronic poisoning describing an anaemia and leucopenia with biopsy showing a bone marrow hypoplasia.

o-Xylene $C_6H_4(CH_3)_2$ (Synonym: *o-Xylol*) is considered to be of low order toxicity by irritation or contact, inhalation, or skin absorption. Some narcotic effects have been reported (see further discussion regarding this compound).

Toluene and xylene are ubiquitous in indoor air. Both are used as carriers for paint pigment.

Benzene C_6H_6 (Synonyms: *benzol; phenyl hydride*) is a clear, colourless liquid with an aromatic odour. On an acute local basis, benzene is moderately irritating but only slightly toxic by inhalation or ingestion. On an acute systemic basis, it is moderately toxic by inhalation, ingestion, and skin adsorption. On a chronic systemic basis, benzene is considered highly toxic by all modes of entry to the body.

In chronic systemic poisoning, the onset is slow and early symptoms are vague and difficult to define. There is fatigue, headache, dizziness, nausea and loss of appetite, loss of weight, and weakness. Later, there is pallor, nosebleeds, bleeding gums, menorrhagia, petechiae and purpura may develop.

Following absorption of benzene, elimination is chiefly via the lungs when fresh air is breathed. The absorbed benzene is oxidized and the oxidation products are combined with H_2SO_4 and glycuronic acid and eliminated via the urine. This is often used as a diagnostic test. Benzene is a cumulative poison and is much more dangerous upon chronic exposure. benzene is a recognized carcinogen, a cause of leukaemia and possibly lymphosarcoma.

(4) ALCOHOLS

No general statement can be made as to environmental impact or human toxicity. Alcohol generally means ethyl alcohol, which is relatively nontoxic compared with methyl alcohol. To identify the toxilogical features of alcohol each type needs to be examined individually.

Ethanol C_2H_5OH (Synonyms: *Ethyl alcohol; methyl carbinol; spirit of wine; alcohol*). The material is a solvent and ingredient in insect repellents and screw-worm remedies.

Methanol CH_3OH (Synonyms: *Methyl alcohol; wood alcohol*) is a clear, mobile liquid. It possesses distinct narcotic properties. It is slightly irritant to the mucous membranes. Its main toxic effect is exerted on the nervous system, particularly the optic nerves and possibly the retinae.

(5) KETONES

These are organic compounds containing the chemical group CO derived from secondary alcohols by oxidation. Acetone, which is dimethyl ketone is the most familiar of this group of compounds. No general statement can be made regarding the toxicity of ketones. Some are highly volatile and hence may have narcotic or anaesthetic effects. Skin absorbtion as well as inhalation may be an important route of entry into the body. None of the ketones has been shown to have a high degree of chronic toxicity.

Acetone CH_3COCH_3 (Synonyms: Dimethyl ketone; ketone propane; propanone) is a colourless volatile liquid with the fragrant odour of mint. It is narcotic in high concentrations. It is a permitted food additive.

Methyl *iso*-Butyl Ketone $(CH_3)_2CHCH_2COCH_3$ (Synonyms: *iso*-methyl ketone; hexone; 4-methyl-2-pentanone; isopropylacetone). The material is irritating to the eyes and mucous membranes and narcotic in high concentrations.

Methyl Ethyl Ketone C_4H_8O (Synonym: 2-Butanone) is a colourless liquid with an acetone like odour. The material can produce local irritation and narcosis.

(6) ALDEHYDES

Aldehydes are usually acute local irritants via inhalation, ingestion or tissue contact. The general parenteral toxicity of aldehydes seems to be related to their irritant properties. Contact dermatitis is seen, but little or no cumulative toxicity. Thus, primary irritation appears to be of the mucous membranes of the eyes and upper respiratory tract. High concentrations can injure lungs and other organs of the body.

The unsaturated and halogenated aldehydes generally cause more noticeable irritation than saturated aldehydes. Aromatic and heterocyclic aldehydes generally cause less irritation than even saturated aldehydes. The lower molecular weight aldehydes act chiefly on the eyes and upper respiratory tract, while the higher molecular weight, less soluble ones penetrate more deeply into the respiratory tract and may affect the lungs. Certain aldehydes cause allergic reaction.
Most aldehydes possess some anaesthetic properties, which properties decrease with increasing molecular weight.

The pathology noted in animal experiments from high concentrations of aldehydes is mainly on the respiratory tract, however, pulmonary edema as well as liver, kidney and CNS damage was also noted.

Formaldehyde HCHO (Synonyms: Formalin; Methanal; BFV; Formic aldehyde)

The more common VOCs in indoor air are Xylene, Toluene and Formaldehyde. Of all the pollutants recognized as being important to the quality of indoor air, none has attracted as much attention from the public and researchers than formaldehyde. Formaldehyde is ubiquitous in modern environments. It is used as a preservative in cosmetics, toiletries and food contact substances.

Formaldehyde release from Urea-Formaldehyde Resin (UFR) bonded wood products is a well established cause of indoor air contamination (Anderson et al., 1975; Meyer, 1983; Meyer et al., 1983). The most widely used UF-bonded products are plywood, particleboard, medium density fibreboard (MDF), urea-formaldehyde insulation (UFI) and general adhesives.

The release of formaldehyde is proportionate to the total formaldehyde in the product and off-gassing decreases with age (Sundin, 1982). High ventilation rates are insufficient to combat formaldehyde release as indoor formaldehyde levels are also dependent on the temperature, the moisture content and the vapour pressure of the emitting material. It is often overlooked that the temperature of the material in the building envelope is usually different from that of the indoor air, as it is strongly influenced by diurnal insolation cycles, weather conditions and other seasonal as well as microclimatic factors (Meyer, 1983). Air humidity can affect the formaldehyde concentration in a complex way because wood is in slow equilibrium with moisture in the air at 20°C and 50%RH wood moisture is about 9 wt%. It increases at 90%RH to about 20 wt%, and decreases at 10%RH to 2.5 wt%. Thus, a change of 10%RH in a standard living room with particleboard flooring can lead to the transfer of 5 litres of water between wood surfaces and the air. Persistent high humidity may be detrimental to the wood and it is capable of slowly hydrolysing urea-formaldehyde resin thereby decreasing its strength and increasing its propensity for releasing formaldehyde.

***n*-Valeraldehyde $CH_3(CH_2)_3CHO$** (Synonyms: Valeral; Pentanal; Amylaldehyde; Valeric aldehyde). This material is a mild irritant by ingestion, inhalation and its systemic toxicity is low by ingestion or inhalation. It is considered a mild irritant and narcotic.

Crotonaldehyde $CH_3CHCHCHO$ (Synonyms: 2-Butenal; beta-Methylacrolein) is a water mobile liquid with a pungent suffocating odour. It is one of the group of unsaturated aldehydes which are several times more toxic than the saturated group. It is a very powerful irritant, allergin and poison by ingestion and inhalation. It is a lacrimating material which is very dangerous to the eyes. It can cause corneal burns and is irritating to the skin.

Propinolaldehyde C_3H_6O (Synonyms: Propanal; Methylacetaldehyde; Propyl aldehyde) is a volatile liquid with a suffocating odour. It is a moderate irritant and moderately toxic by ingestion or inhalation. It is a common air contaminant due, in a large part, to the operation of internal combustion engines.

APPENDIX 5

APPENDIX 5. THRESHOLD LIMIT VALUES

The threshold limit values adopted by the New Zealand Department of Health are, essentially, those proposed by the American Conference of Government Industrial Hygienists in 1984. According to the publication by the Occupational Health and Toxicology Branch of the New Zealand Division of Public Health;

> *" Threshold limit values refer to airborne concentrations of substances and represent conditions under which it is believed that nearly all workers may be repeatedly exposed day after day without adverse effect. Because of a wide variation in individual susceptibility a small percentage of workers may experience discomfort from substances at concentrations at or below the threshold limit; a smaller percentage may be affected more seriously by aggravation of a preexisting condition or by development of an occupational illness. "*

The ACGHI define three categories of threshold limit values:

(a) The Threshold Limit Value - Time Weighted Average (TLV-TWA). This is the time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek, to which *nearly all* workers may be repeatedly exposed, day after day, without adverse effect.

(b) Threshold Limit Value - Short Term Exposure Limit (D-STEL). This is the concentration to which workers can be exposed to for a short time without suffering from (1) irritation, (b) chronic or irreversible tissue damage, (3) narcosis of sufficient degree to increase the likelihood of accidental injury, impair self rescue or materially reduce work efficiency. It is not a separate independent exposure limit, rather it supplements the time-weighted average (TWA) limit where there are recognised acute effects from a substance whose chronic effects are of a chronic nature. STELs are recommended only where toxic effects have been reported from short-term exposures in either humans or animals.

An STEL is defined as a 15-minute time-weighted average exposure which should not be exceeded at any time during the work day even if the 8-hour time weighted average is within the D. Exposures at the STEL should not be longer than 15 minutes and should not be repeated more than four times per day. There should be at least 60 minutes between successive exposures at the STEL. An averaging period other than 15 minutes may be recommended when this is warranted by observed biological effects.

(c) Threshold Limit Value-Ceiling (D-C). This is the concentration that should not be exceeded even for an instant.

The main argument against TLVs, as they stand, is that the synergistic effect of different irritant mixtures is unknown. The present system relies on simple additivity. When two or more hazardous substances act upon the same organ system, authorities suggest that their combined effect should be given consideration. When the sum of the following fractions,

$$\frac{C_1}{T_1} + \frac{C_2}{T_2} + \ldots\ldots + \frac{C_n}{t_n} = 1$$

exceeds unity, then the threshold limit of the mixture should be considered as being exceeded. C_1 indicates the observed atmospheric concentration, and T_1 the corresponding threshold limit.

Example 1 If the air contains 400 ppm of acetone (D = 750 ppm), 150 ppm of sec-butyl acetate (D = 200 ppm) and 100 ppm of methyl ethyl ketone (D = 200 ppm):

$$\frac{400}{750} + \frac{150}{200} + \frac{100}{200} = 1.78$$

In this instance the threshold limit is exceeded.

Example 2 In the special case when the source contaminant is a liquid mixture and the atmospheric composition is *assumed* to be similar to that of the original material (ie. on a time-weighted average exposure basis, all of the liquid (solvent) mixture eventually evaporates).

The following formula is used:

$$\frac{1}{\left[\frac{f_a}{TLV_a} + \frac{f_b}{TLV_b} + \frac{f_c}{TLV_c} + \ldots. + \frac{f_n}{TLV_n}\right]}$$

For example consider the following:

Liquid contains (by weight) 50% heptane: D = 400 ppm or 1600 mg/m^3 (ie. 1 mg/m^3 = 0.25 ppm). 30% methyl chloroform: D = 350 ppm or 1900 mg/m^3 (ie. 1 mg/m^3 = 0.18 ppm) and 20% perchloroethylene: D = 50 ppm or 335 mg/m^3 (ie. 0.15 ppm).

D of the mixture

$$\frac{1}{\left[\frac{0.5}{1600} + \frac{0.3}{1900} + \frac{0.2}{335}\right]}$$

$$\frac{1}{[0.00031 + 0.00016 + 0.0006]}$$

$$\frac{1}{0.00107} = 935\ mg/m^3$$

Therefore of this mixture

50% of 935 = 468 mg/m^3 is heptane
30% of 935 = 281 mg/m^3 is methyl chloroform
20% of 935 = 187 mg/m^3 is perchloroethylene

These values can be converted to parts per million (ppm) as follows:

Heptane:	468 mg/m^3 Times 0.25 = 117 ppm
Methyl chloroform:	281 mg/m^3 Times 0.18 = 51 ppm
Perchloroethylene:	187 mg/m^3 Times 0.15 = 29 ppm

Therefore: D of mixture = 117 + 51 + 29 = 197 ppm, or 935 mg/m^3

APPENDIX 6

APPENDIX 6. INORGANIC GASES

Carbon Monoxide (Health Effects)

Hypoxia caused by carbon monoxide leads to deficient function in sensitive organs and tissues like the brain, heart, the inner wall of blood vessels and platelets.

With regard to cardiovascular effects, a decreased oxygen uptake capacity and the resultant decreased work capacity under maximal exercise conditions have been shown to occur in healthy young adults (Weiser et al.,1978). These cardiovascular effects may have health implications for the general population in terms of a potential curtailment of certain physically demanding occupational or recreational activities under circumstances of sufficiently high carbon monoxide exposure. However, of greater concern at more typical ambient carbon monoxide exposure levels are certain cardiovascular effects (eg. aggravation of angina symptoms during exercise) which are likely to occur in a smaller but sizable segment of the general population. This group, consisting of chronic angina patients is presently regarded as the most sensitive risk group in relation to carbon monoxide exposure effects, on the basis of evidence of an aggravation of angina patients at carboxyhaemoglobin (COHb) levels of 2.9-4.5% COHb (Anderson et al.,1973).

Statistically significant impairment of vigilance tasks has been described at carboxyhaemoglobin levels above 5% COHb (Putz et al.,1976). However, there is no reliable evidence to demonstrate decrements in neurobehavioural function in healthy young adults at levels of COHb below 5%. Much of the research at 5% COHb showed no effect, even when neurobehavioural functions similar to those affected in other studies were investigated (Haider et al.,1976; Winneke, 1974; Christensen et al.,1977; Beningnus et al.,1977). However, none of those studies which demonstrated significant effects on neurobehavioral functions examined carboxyhaemoglobin levels below 5%. Therefore, on one hand the empirical evidence indicates that carboxyhaemoglobin levels as low as 5% produce decrements in neurobehavioural function and, on the other hand, the possibility of decrements at carboxyhaemoglobin levels lower than 5% cannot be fully excluded.

Higher levels of carboxyhaemoglobin may lead to secondary effects, for instance, decrease in blood pH and changes in fibrinolysis (Kalmaz et al.,1980). Perinatal effects such as reduced birth weight and retarded postnatal development have also been described.

There is general agreement that from levels of 10-15% COHb upwards many of these malfunctions may occur, as well as subjective symptoms such as headache and dizziness.

TABLE 41 - HUMAN HEALTH EFFECTS ASSOCIATED WITH CO EXPOSURE

Carboxyhaemoglobin Concentration (%)	Effects
2.3 - 4.3	Statistically significant decrease (3-7%) in the relation between work time and exhaustion in exercising young healthy men
2.9 - 4.5	Statistically significant decrease in exercise capacity (ie. shortened duration of exercise before onset of pain) in patients with angina pectoris and increase in duration of angina attacks.
5 - 5.5	Statistically significant decrease in maximal oxygen consumption and exercise time in young healthy men during strenuous exercise.
< 5	No statistically significant vigilance decrements after exposure to carbon monoxide
5 - 7.6	Statistically significant impartment of vigilance tasks in healthy experimental subjects.
5 - 17	Statistically significant diminution of visual perception, manual dexterity, ability to learn, or performance in complex sensor motor tasks (eg. driving)
7 - 20	Statistically significant decrease in maximal oxygen consumption during exercise in young healthy men.

Nitrogen Dioxide (Health Effects)

Most studies of the relationship between residential exposure to NO_2 and health have focused on respiratory symptoms and illnesses and on the level of pulmonary function.

It was reported that, NO_2 may damage the lung directly through its oxidant properties or indirectly by increasing susceptibility to respiratory infections (National Research Council, 1976; Jakab, 1980).

In animal models, NO_2 reduces the efficacy of specific lung defence mechanisms, and effects on mucociliary clearance, the alveolar macrophage, and the immune system have been demonstrated (National Research Council, 1976; Dawson et al.,1979; Morrow, 1984).

Data on the health effects of NO_2 concentrations likely to be encountered by the general population are derived from experimental and epidemiologic studies. The results of some human exposure studies imply that levels comparable to those measured in homes may increase airways reactivity in some asthmatics, but the result of a number of studies are inconsistent (Morrow, 1984; Orehek et al., 1976; Bauer et al., 1976).

A number of epidemiologic studies have been carried out to assess the relationship between chronic effects and low level exposures. The majority of these investigations were cross-sectional surveys of school children. The investigators generally assessed current symptom status and retrospective illness histories, as obtained by parent completed questionnaire, and pulmonary function. Although NO_2 levels were measured in several of the investigations (Florey et al.,1979; Melia et al., 1982; Fischer et al., 1985) exposure was most often assessed by simple questions concerning type of fuel used for cooking. Consistent evidence of excess respiratory symptoms and illnesses in children exposed to gas stoves has not been satisfactorily demonstrated.

Early reports from two cross-sectional surveys of schoolchildren in Great Britain indicated that children from homes with gas stoves had a higher prevalence of respiratory symptoms than children from homes with electric stoves (Melia et al., 1977; Melia et al., 1979). When one of the survey groups was followed longitudinally, however, the relative risks associated with gas stove use became highly variable and tended to decrease as the children grew older (Melia et al., 1979).

The same British investigators surveyed a third group of 808 schoolchildren, and measured NO_2 concentrations in the homes of a small sample ($n=103$). The prevalence of respiratory symptoms was higher in children from homes where gas was used for cooking and increased with higher NO_2 concentrations, although both effects were of borderline statistical significance (Florey et al., 1979). However, NO_2 and respiratory symptoms were not replicated, when these same investigators subsequently studied another sample of 183 children (Melia et al., 1981).

Two prospective studies of infants in Great Britain also failed to demonstrate an association between the use of gas for cooking and respiratory illness (Melia et al., 1983; Ogston et al, 1985).

Data on children from the United States are similarly inconsistent. Two large cross sectional studies, one involving the Harvard Air Pollution Health Study (Speizer et al., 1980) and the other involving schoolchildren in Iowa (Ekwo et al., 1983) have demonstrated that reports of serious respiratory illness before two years of age and hospitalization for respiratory illness were more common among children from homes with gas stoves. When the original cohort in the Harvard Air Pollution Health Study was expanded, however, the odds ratio of 1.23 for serious respiratory illness before 2 years of age decreased to 1.12. In the study of Ekwo and associates, the effect of exposure to a gas stove varied strongly and inconsistently with parental smoking habits. The effect was absent in homes where one parent smoked, largest where both parents smoked, and indeterminate where neither smoked.

Schenker et al., (1983) found no association between type of cooking stove and current respiratory symptoms or previous illness history in a cross-sectional survey of 4,071 schoolchildren in Western Pennsylvania.

The relationship between stove type and respiratory illness has also been studied prospectively. Keller et al., (1979), in a study of 1,952 family members of all ages in Ohio, found that respiratory illness did not vary by stove type.

The findings on NO_2 exposure and respiratory illness indicate that the magnitude of the NO_2 effect at concentrations found in most buildings is likely to be small.

The World Health Organisation recommends nitrogen dioxide levels 400$\mu g/m^3$ (0.21ppm) and 150$\mu g/m^3$ (0.08ppm) as 1-hour and 24-hour guidelines respectively. The 1-hour guideline is based on the judgement that the lowest-observed-effect level in asthmatics (560$\mu g/m^3$, 0.3ppm) is not necessarily adverse and a guideline somewhat lower provides a further margin of protection.

Hydrogen Sulphide (Health Effects)

In its acute form, hydrogen sulphide acts on the nervous system.

At concentrations of 15 mg/m^3 and above, hydrogen sulphide causes conjunctival irritation (Savolainen, 1982). The following table outlines the dose-effect relationships.

TABLE 42 - DOSE-EFFECT OF HYDROGEN SULPHIDE

Hydrogen Sulphide Concentration mg/m^3	ppm	Effect	Reference
1400-2800	1000-2000	Immediate collapse with paralysis of respiration	(1)
750-1400	530-1000	Strong CNS stimulation, hyperpnoea followed by respiratory arrest	(1)
450-750	320-530	Pulmonary oedema with risk of death	(1)
210-350	150-250	Loss of olfactory sense	(2)
70-140	50-100	Serious eye damage	(2)
15-30	10-20	Threshold for eye irritation	(2)

References

(1) WHO Report #19 (1981)
(2) Savolainen (1982)

According to the WHO, the lowest-adverse-effect level of hydrogen sulphide is 15 mg/m^3, when eye irritation is caused.

In view of the steep rise in the dose-effect curve implied by reports of serious eye damage at 70 m/m^3, a relatively high protection factor has been recommended. In order to avoid substantial complaints about odour annoyance among the exposed population, hydrogen sulphide concentrations should not be allowed to exceed 7$\mu g/m^3$, with a 30-minute averaging period.

Ozone (Health Effects)

In a large number of controlled human studies significant imparement of pulmonary function has been reported. This is usually accompanied by respiratory or other symptoms (EPA 1986; von Nieding et al., 1979).

Changes in pulmonary function associated with 1-3 hours of ozone exposure in normal subjects during exercise have been reported for the following parameters:

- forced expiratory volume for 1 second ($235\mu g/m^3$)
- airway resistance ($470\mu g/m^3$)
- respiratory frequency increased ($470\mu g/m^3$)

The severity of respiratory and other symptoms parallels the imparement of pulmonary function both in magnitude and time scale (EPA report, 1986). Symptoms that have been reported are cough, throat tightness, thoracic pain, increased mucous production, râles, chest tightness, substernal pain, lassitude and nausea.

In addition to causing functional changes and symptoms, ozone is capable of inducing increased nonspecific airway sensitivity to acetylcholine, metacholine and histamine (Holtzman et al., 1979).

Functional recovery from a single exposure takes place in two phases: a 50% improvement within 1-3 hours and a return to pre-exposure values within 24-48 hours. Recovery of other regulatory systems does not parallel the functional recovery, and affects may persist for days (eg. airway hyperreactivity) (Holtzman et al., 1979).

Repeated daily short-term exposures show that the decrements in pulmonary function are maximal after the second exposure day; thereafter responsiveness to ozone is attenuated. Very small or no changes are observed after the fourth and fifth exposure days. Attenuation if respiratory symptoms has also been observed, lasting up to 3 weeks. Such attenuation apparently only occurs when the initial exposure is sufficient to cause functional changes, and is not long-lasting (EPA report, 1986; Horvath et al., 1981).

A number of studies of various sub groups such as patients with chronic obstructive lung disease and smokers showed that these were the same in their response to ozone as the normal population (Linn et al., 1983; Linn et al., 1979). Within the normal population there is a range of responsiveness to ozone that is reproducible, that is, an individual who is highly responsive remains highly responsive over the study period, often over months (McDonnell et al., 1985).

The recommended 1-hour exposure level is in the range of $150\text{-}200\mu g/m^3$ (0.076-0.1ppm)

The 8-hour guideline for exposure to ozone is $100\text{-}120\mu g/m^3$ (0.05-0.06ppm).

APPENDIX 7

APPENDIX 7. RESPIRABLE SUSPENDED PARTICULATES (RSPs)

Substances of diverse nature and physical form, once airborne, may reach the upper and lower airways of the lungs in inspired form. Their ability to do this depends on their physical properties. The more a material is broken down, finely divided and dispersed the more likely are its particles to be airborne and, therefore, capable of inhalation.

There are types of particulate clouds, other than those produced by attrition of rock, which have a wide range of particle size; everyday usage speaks of 'dusts', 'fumes', 'smokes' and 'mists', and it is important that these should be distinguished (Parkes et al., 1982).

> *Dust* refers to tiny particles which have settled on a surface, can be readily disturbed and are visible in a shaft of sunlight. It is more properly defined as consisting of solid particles dispersed in air (or other gaseous media) due to disintegration of materials, both organic and inorganic.
>
> *Fumes* consist of metal oxides formed by heating metals to their melting points. Particle sizes range from 0.1 μm to 1 μm diameter.
>
> *Mists* are liquid droplets formed by condensation of vapours or the 'atomization' of liquids around appropriate nuclei. Many mists are less than 0.1 μm diameter but can range up to 500 μm in diameter.

The term *aerosol* is commonly used to describe all of the above categories and includes dispersed particles and droplets. Potentially dangerous particles, vary in size from those just small enough to enter the upper respiratory tract down to small gas molecules.

TABLE 43 - COMPARISON OF PARTICLE SIZES

Comparison of Particle Sizes	
Material	Dimension Range
Sand grains	200 - 2000 μm diameter
Cement dusts	4 - 100 μm diameter
Pollens	10 - 100 μm diameter
Fungal spores	2 - 100 μm length 0.5 - 7 μm diameter
Actinomycete spores	0.6 - 2.5 μm length
Rock dusts	1 - 10 μm diameter
Tobacco smoke	0.2 - 2 μm diameter
Viruses	28nm - 0.2 μm diameter

Behaviour of Inhaled Particles

Aerosols inhaled into the respiratory tract closely follow the movement of air in which they are suspended, and the depth to which they penetrate into the lung depends not only upon their physical characteristics but also on the volume of each respiration. Once a particle comes in contact with the wall of an airway or an alveolus it cannot again become airborne. Because the majority of inhaled particles less than 1 μm in diameter are expelled in exhaled air, their concentration in inhaled air must be sufficiently high to enable some of them to be deposited in the lungs.

The composition of particles, whether in the solid or liquid state, does not influence their deposition. There are four ways in which solid particles are deposited - sedimentation, inertial impaction, interception and diffusion.

Sedimentation

Sedimentation is settlement influenced by gravity. It is determined by the density and diameter of particles (density $\times$ diameter^2). Under some circumstances the form of air flow in a tube may influence sedimentation but, for practical purposes, it has no effect upon particle sedimentation in the lungs which occurs predominantly at low velocities. Particles deposited in this way will have aerodynamic sizes of about 2μm or less. Deposition in the larger airways is chiefly due to sedimentation.

The free falling speed of fibrous particles is determined by the square of their diameter and is little influenced by their shape or length. Gravitational settlement of fibres occurs only in large airways and it limits the diameter of fibres that penetrate to small airways to less than 3μm (Timbrell, 1970). In the small airways, deposition of fibres is not determined by their falling speed but by interception.

Inertial impaction

When an airstream carrying fairly large particles has its direction changed by the curving or branching of airways (as in nasal cavities and large airways of the lungs) the particles tend to follow the original path in the airstream and, in consequence, impinge upon the walls. Impaction of particles in this way is related to their density $\times$ diameter^2, the diameter and change of direction of the tube and the rate of air flow in the tube.

This is the primary mechanism of deposition in the nose and is important in the large airways for compact particles larger than 10μm. Particles smaller than this are able to penetrate the small airways and alveoli.

Interception

This concerns particles of irregular shape (eg. *mica plates*) or of fibrous habit (eg.*asbestos)* in which the length and shape of the particles - their aerodynamic diameter - is more important than their falling speed.

The lower the length/diameter ratio of a fibrous particle the more its behaviour resembles that of a compact particle, but the longer a fibre the less likely it is to behave in this way. Hence, long fibres of small diameter (ie. $<$ 3 μm), unlike compact particles, avoid

sedimentation and impaction in the larger airways and are intercepted by collision with the walls of the terminal and respiratory bronchioles particularly at their bifurcations. This explains why asbestos fibres as long as 200μm may be found in this region.

Chrysolite (white asbestos) fibres are frequently curled, a property which increases their likelihood of collision with the walls of narrow airways, mainly at their bifurcations. Amphibole fibres, on the other hand, are always rigid, and this favours their orientation parallel to the axis of the airways by aerodynamic forces so that they penetrate deeper into the lung. Crocidolite (blue asbestos) fibres reach the periphery of the lung having suffered little sedimentation *en route* (Timbrell, et al., 1970).

The variable depth of penetration by different types of asbestos fibre may be significant in determining which types are likely to cause disease (Parkes et al., 1982).

Diffusion

This effect is exhibited by very small particles (less than 0.1μm diameter) and which is independent of their density, influences their deposition significantly in the region beyond the terminal bronchioles and also upon the wall of the trachea.

APPENDIX 8

APPENDIX 8. TOBACCO SMOKE

Walsh et al., (1984) reports that children of smoking parents are more likely to develop respiratory disorders than children of non-smoking parents, and these children have measurably impaired lung function associated with permanent damage to the small airways. Nonsmoking adults who work for several years in smoky offices have similarly impaired lung function, similar to that observed in light smokers.

Ambient tobacco smoke has been associated with an increased risk of lung cancer, non smoking wives of smoking husbands have 2-3 times the risk of lung cancer as nonsmoking wives of nonsmoking husbands.

In addition to producing disease in otherwise healthy nonsmokers, ambient tobacco smoke aggravates the condition of people with existing disease. For example, people with heart disease develop chest pain after shorter periods of exercise after breathing ambient cigarette smoke.

There is no consensus as to whether a true tobacco allergy actually exists. It does seem that those patients with a history of allergies to other substances are more likely to report the irritating effects of tobacco smoke (Epstein, 1977; Speer, 1968).

Researchers have quantified the commonly known short-term effects of tobacco smoke on nonsmokers, including burning eyes, nose, and throat, headache and nausea.

In short, cigarette smoke in concentrations commonly encountered in daily living may not only bother nonsmokers but can cause them physical harm.

Sidestream Smoke

Comprehensive discussions of the chemistry of sidestream and mainstream smoke are included in the 1979, 1984 and 1986 reports of the U.S. Surgeon General, in the report of the National Research Council on indoor air pollution (1981), and the report of the National Research Council on environmental tobacco smoke (1986).

The sidestream smoke often contains concentrations of toxins higher than mainstream smoke. This is because when the cigarette is smouldering, the temperature of the burning cone is lower, so there is incomplete combustion. However, dilution by room air markedly reduces concentrations inhaled by the involuntary smoker in comparison to the active smoker. Nevertheless, involuntary smoking is accompanied by many toxic agents generated by tobacco combustion (National Research Council, committee on indoor pollutants, 1981; Sterling et al., 1982).

Tobacco smoke is a complex mixture of gases and particles that contain a wide variety of chemicals (U.S. Department of Health and Human Services, report 84-50205, 1984). A typical cigarette smoker inhales mainstream smoke 8 to 10 times, for a total of 24 to 30 seconds out of a burn time of 12-minutes for a typical cigarette (Neurath, 1973). Hence sidestream smoke is produced during 96% of the total time a cigarette is lit, so sidestream smoke is the most important constituent of ambient tobacco smoke. Sidestream smoke is an

important source of indoor carbon monoxide (CO), nicotine, ammonia (NH_3), polycyclic hydrocarbons, acrolein, nitrogen oxides (NO_x), pyrene, benzopyrenes, formaldehyde, and hydrogen cyanide. Tar particle (chloroform extract) emissions from sidestream smoke are estimated to be as much as 30 to 40 mg per cigarette (Mueller Associates, Inc., et al., 1987).

The increase in ambient levels depends on the number of smokers, the intensity of their smoking, the ventilation rate and the use of air cleaning devices. In a study conducted in 38 commercial buildings in the Pacific Northwest, the geometric mean for respirable suspended particles in all buildings was 24 $\mu g/m^3$. The geometric mean for smoking areas and nonsmoking areas was 24 $\mu g/m^3$ and 15 $\mu g/m^3$ respectively. Fourteen of the 70 smoking sites and only one of the 106 nonsmoking sites exceeded the ASHRAE annual exposure guideline of 75 $\mu g/m^3$. The highest recorded level was of 308 $\mu g/m^3$. This occurred in a Portland office building cafeteria where smoking was allowed (Turk et al., 1986).

Baechler et al., (1990) suggests that even in buildings with high indoor RSP levels due to tobacco smoke, levels in nonsmoking areas of the same building can be quiet low. In the 'Turk' study, in only one building were RSP concentrations within a factor of 2 of the smoking area concentrations. Local exhaust, ventilation near smoking areas, dilution by larger building volumes, and removal mechanisms significantly reduce RSP concentrations in areas away from the source (Turk et al., 1986). However, in buildings with smaller volumes and poor ventilation, isolating smoking areas may have little effect on limiting the distribution of RSPs.

Passive Smoking - Possible Health Issues

The majority of research into passive smoking has focused on respiratory effect, however there has been some investigation into diverse health effects including non-respiratory cancer, ischemic heart disease, early age at menopause (Everson et al., 1986), sudden infant death syndrome and birth weight (Martin et al., 1986; Rubin et al., 1986).

(a) Respiratory Disorders

Long-term exposure to ambient tobacco smoke produces measurable deterioration in the lung function of otherwise healthy nonsmoking adults. White et al., (1980) carried out pulmonary function tests on 2100 middle-aged adults. They found that nonsmokers who worked in smoky environments had deteriorated lung function similar to that of light smokers and smokers that did not inhale. The deterioration reflected the damage to the small airways of the lung and the researchers concluded that it may be a precursor to more serious lung disease.

These results were consistent with the findings of Tager et al., (1979), in a study of lung function in children.

The presence of two independent studies both reaching the conclusion that long-term exposure to second hand smoke produces measurable damage to the lungs seems to support the conclusion that *occupational exposure to sidestream smoke can produce physical damage in otherwise healthy nonsmokers.*

(b) Exacerbation of Pre-existing Conditions

A number of studies suggest that sidestream smoke can aggravate existing medical conditions. Those at risk include people with heart disease, lung disease, and allergies to other substances.

Arnow, (1978), exposed 10 people who had coronary heart disease to second-hand smoke, then administered exercise tests to assess whether or not the smoke reduced their ability to exercise. He found that involuntary smoking increased resting heart rate, blood pressure, venous carboxyhaemoglobin, and reduced the length of time the people could exercise before developing chest pain. On average, the subjects could only exercise 240 seconds before developing chest pain when they breathed unpolluted air, but only 181 seconds after sitting in a well ventilated room while other people were smoking. Their ability to exercise dropped to 146 seconds when the room was not ventilated. A plausible explanation was that the CO in the cigarette smoke binds to the haemoglobin in the blood and prevents it from carrying adequate oxygen to the heart and other body tissues (Anderson et al., 1973; Arnow et al., 1977). There may also be a direct effect of the nicotine on the heart, acting to depress it.

Arnow et al., (1977), had similar results with people with lung disease. He exercised 10 patients with hypoxic chronic lung disease before and 1-hour after exposing them to 100ppm of CO. There was a significant reduction in how long these people could exercise, a reduction from 219 seconds to 147 seconds, before they developed shortness of breath.

(c) Cardiovascular Disease

While there are extensive data establishing cigarette smoking as a causal risk factor for cardiovascular diseases (U.S. Department of Health and Human Services, report 84-50202, 1983), only a few studies addressed involuntary smoking as a risk factor for these diseases. In the cohort of nonsmoking Japanese women, Hirayama (1983), found a small but statistically significant increased risk of death from ischemic heart disease associated with the spouses smoking. Garland et al., (1985), prospectively determined mortality from ischemic heart disease in nonsmoking older women in California. After adjustment for established risk factors, marriage to a smoking spouse was associated with a relative risk of 2.7 at a confidence level of 90%. Gilis et al., (1984), assessed the baseline prevalence of cardiovascular symptoms and major electrocardiographicy abnormalities in a population sample residing in Scotland and then determined cause-specific mortality for up to 10-year follow up. In the preliminary report, involuntary smoking was not associated with cardiovascular symptoms at baseline nor with cardiovascular mortality on follow up. A case control study in England *did not* show increased risk for ischemic heart disease or for stroke in nonsmokers married to smokers (Lee et al., 1986).

(d) Lung Cancer

In 1981, reports were published from Japan (Hirayama, 1981), and from Greece (Trichopoulos et al., 1981) that indicated increased lung cancer risk in nonsmoking women married to cigarette smokers. These studies, which purported to show a causal link between passive smoking and a potentially fatal disease had major ramifications for the tobacco industry and, indeed, employers who allowed smoking in the workplace. The significance of this was not lost on the tobacco industry who launched a major campaign in the United States to discredit the reports.

Hirayama (1981), examined the effect of involuntary smoking on lung cancer by observing 91,540 nonsmoking Japanese housewives aged 40 and above. During the 14 years in which his study tracked these women, 174 nonsmoking women died of lung cancer. He also examined the smoking habits of these women's husbands, taking into account such variables as the amount of smoking the husband did and whether the women lived in an urban or rural environment. He found that nonsmoking wives of men who smoked more than 20 cigarettes per day had 2.4 times the risk of developing lung cancer as nonsmoking wives of nonsmokers. He found an even higher risk of sidestream smoke exposure among wives of smoking agricultural workers, about 4.6 times the risk of women married to nonsmokers. There were similar, but smaller, increases in risk for women married to lighter smokers. Hirayama suggested that the lower risk attributable to a husband's smoking in the urban environment was due to the greater presence of other environmental carcinogens, which made it more difficult to single out the effect of cigarette smoke.

Trichopoulos et al., (1981), found similar results in a case-control study of Greek women. They located 40 nonsmoking female Caucasian residents of Athens who were admitted to hospitals between 1978-1980 with a final diagnosis of lung cancer. They selected 154 nonsmoking women as controls from a hospital specializing in orthopaedic disorders to avoid women with other diseases related to smoking. The women in the two groups were then questioned about the smoking habits of their husbands and themselves. Nonsmoking wives of men who smoked more than 20 cigarettes per day had 3.4 times the risk of developing lung cancer as nonsmoking wives of nonsmoking husbands. Similar, but smaller, risks were associated with being married to a light smoker or an ex-smoker.

Time trends of lung cancer mortality in nonsmokers has been examined with the rationale that temporally increasing the exposure to environmental tobacco smoke should be parallelled by increasing mortality rates. Enstrom (1979), calculated nationwide lung cancer mortality rates for 1914-1968 and concluded that a real increase had occurred in nonsmoking males after 1935. However, occupational and environmental exposure other than tobacco smoke could explain the apparent mortality rate increase in males.

Garfinkel (1981), published a study of American women which failed to detect a statistically significant increase in the risk of developing lung cancer among nonsmoking women of smoking husbands. Garfinkel's study tracked 469,000 nonsmokers for 12 years and measured the death rates from cancer. He located 176,739 nonsmoking women who were married; of these women, 143 died of lung cancer during the study. He found little increase in risk of lung cancer associated with being married to a smoker.

The timing of this report was a windfall for the tobacco industry. It directly contradicted the findings in the Japanese and Greek studies. However, Walsh et al., (1984), suggests that the results may not be as contradictory as they appear given the differences in lifestyle in Japan, Greece, and the U.S., and the 20-year latency period associated with lung cancer. Twenty years ago, few women in Japan and Greece smoked. Moreover, social customs encouraged these women to stay at home, further restricting a woman's exposure to smoke from sources other than her spouse. This situation contrasts with the then-current situation in the U.S. in two ways.

(1) Twenty years prior to the report, relatively more American women were smoking.

(2) American women were more mobile than their Japanese and Greek counterparts and hence were exposed to more smoke outside the home.

This additional exposure makes it difficult to accurately estimate the true exposure to sidestream smoke for the American woman and may obscure the fact that involuntary smoking is associated with an increased risk of cancer. Hirayama's observation of higher risk attributable to involuntary smoking among women living on farms compared to those living in cities is consistent with this view. Both Trichopoulos and Garfinkel comment on the difficulty of estimating true exposure. Given these methodological difficulties in assessing exposure in the American women compared with their Japanese and Greek counterparts, together with the fact that Garfinkel found a 10-30% increase in mortality associated with passive smoking, are not as incompatible as they first appeared. Hirayama (1981), later pooled the data from all three studies and found a significant increase in lung cancer among nonsmoking wives of smoking husbands.

In Germany, Knoth et al., (1983), described a series of 59 female lung cancer cases of which 39 were nonsmokers. Based on census data, they reported that a much greater than expected proportion of those nonsmokers lived in households with smokers. This report did not include an appropriate comparison series and the suitability of substituting census data was not addressed by the authors.

In spite of this variable epidemiologic evidence, environmental tobacco smoke has been characterized as a respiratory carcinogen. The International Agency for Research on Cancer of the World Health Organization ((WHO, vol 36, 1986) has concluded that *'passive smoking gives rise to some risk of cancer.'* The agency supported this conclusion in its monograph on tobacco smoking by citing the characteristics of sidestream and mainstream smoke, the absorption of tobacco smoke materials during involuntary smoking, and the nature of dose-response relationships for carcinogenesis. The National Agency for Research on Cancer argued on the basis of biological plausibility rather than on the basis of epidemiologic evidence.

The National Research Council (1986), and the U.S. Surgeon General (1986), have also concluded that involuntary smoking increases the incidence of lung cancer in nonsmokers. In reaching this conclusion, the National Research Council (1986), cited the biological plausibility of association between environmental tobacco smoke exposure and lung cancer and the supporting epidemiologic evidence. The report carefully considered the sources of bias that may have affected the epidemiologic studies. Based on a pooled analysis of the epidemiologic data and adjustment for bias, the report's authors concluded that the best estimate for the excess risk of lung cancer in nonsmokers married to smokers was 25%. The 1986 report of the Surgeon General, characterized involuntary smoking as a cause of lung cancer in nonsmokers. The conclusion was based on the extensive information already available on the carcinogenicity of active smoking, on the qualitative similarities between environmental tobacco, and on the epidemiologic data on involuntary smoking.

(e) Other Cancers

A number of reports suggest that environmental tobacco smoke exposure may increase the risk of cancer at sites other than the lung. One study found that in children, maternal exposure to environmental tobacco smoke during pregnancy was associated with increased risk of brain tumours (Preston-Martin et al., 1982), and in another study paternal but not

maternal smoking increased the risk of childhood rhabdomyosarcoma (Grufferman et al., 1982). Such effects might arise from smoking-induced changes in germ-cells of the parents or through transplacental exposure rather than as a direct effect of smoke inhalation (Everson, 1980; Grufferman et al., 1982).

In adults, involuntary smoking was linked to a generally increased risk at specific sites. Miller (1984), interviewed surviving relatives of 537 deceased nonsmoking women in western Pennsylvania concerning the smoking habits of their husbands. A significantly higher risk of cancer death (odds ratio = 1.94, $p < 0.05$) was found in women who were married to smokers and were also not employed outside their homes. The large number of potential subjects who were not interviewed and the possibility of information bias detract from this report.

Sander et al., (1985), concluded a case control study on the effects of childhood and adulthood exposures to environmental tobacco smoke on the risk of cancer. The 518 cases included all cancers other than basal cell cancer of the skin; the cases and the matched controls were between 15-59 years. For all sites combined, significantly increased risk was found for parental smoking (crude odds = 1.6) and for marriage to a smoking spouse (crude odds = 1.5); the effects of these 2 exposures were independent. Significant associations were also found for some individual sites; for childhood exposure, maternal and paternal smoking increased the risk of haematopoietic malignancy, and for adult exposure, spouse's smoking increased the risk of cancers of the female breasts, female genital system and the endocrine system. These findings are primarily hypothesis generating and require replication. Hirayama (1981), reported significantly increased mortality from nasal sinus cancers and from brain tumours in nonsmoking women married to smokers in the Japanese cohort.

These associations of involuntary smoking with cancer at diverse non-respiratory sites cannot be readily supported with arguments for biological plausibility. Increased risks at some sites, for example cancer of the nasal sinus and female breast, have not been observed in active smokers (U.S. Department of Health and Human Services, report 82-50179, 1982).

APPENDIX 9

APPENDIX 9. CLINICALLY DIAGNOSABLE SYNDROMES

The following has been referenced from the Merck Manual, fourteenth edition.

TABLE 44 - HUMAN VIRAL DISEASES; PERSON-TO-PERSON SPREAD

Viral Groups & Categories	Known Serotypes	Syndromes	Prevalence & Distribution
Respiratory Influenza A, B, C	3	Influenza; AFRD; acute bronchitis & pneumonia; croup	Epidemic, occasionally pandemic (A, B); endemic (C)
Parainfluenza	4	AFRI (children); acute bronchitis & pneumonia; croup	1: local epidemic; 1 & 3: widely in children
Mumps	1	Parotitis, orchitis, meningoencephalitis	Global; mostly in children; some adults
Adenoviruses	33	AFRD (children); ARD (adults); APCF; EKC; viral pneumonia; acute follicular conjunctivitis	1-3; 5-7: Children; 1, 7, 14, 21: Adults 8: Local EKC
Reoviruses	3	Mild RI	Widely in children
Respiratory syncytial	1 (3 sub types)	U & L RI (infants) mild URI (adults)	Paediatric clinics and hospital wards
Infectious mononucleosis	1	Infectious mononucleosis	Widespread; chiefly in young adults
Rhinovirus	Unknown	Common cold; acute coryza with or without fever	Universal; especially in cold months
Enteric Polioviruses	3	Poliomyelitis (paralytic); aseptic meningitis; AFRD (children)	Almost universal in warm months amongst the younger age group
Coxsackieviruses	30 (A's: 24; B's: 6)	Herpangina; epidemic pleurodynia; aseptic meningitis; myocarditis; pericarditis; AFRD paralytic disease; fever & exanthem	Varies with types; most persons infected; more prevalent in warmer months & children are more prone to infection

Echoviruses	31	Aseptic meningitis; fever and exanthem; meningoencephalitis with rash; diarrhoea neonatorum; paralytic disease; myocarditis; pericarditis; ARD	As for coxsackieviruses
Epidemic gastroenteritis	3 (possibly more)	Epidemic nausea & vomiting	Local epidemics in children particularly in the colder months and in neonates
Exanthems Rubeola	1	Measles; encephalomyelitis	Almost universal CNS involvement rare
Rubella	1	German measles	Universal; birth defects from infection during 1st trimester of pregnancy
Varicella-zoster	1 Same	Chickenpox Herpes zoster	Almost universal in children occasionally in adults Common in adults; reactivation or reinfection
Herpes Simplex	2	Herpes labialis; herpetic gingivostomatitis; eczema; keratoconjunctivitis; encephalitis; vulvovaginitis	Recurrent, almost universal
Roseola infantum	1 (perhaps more)	Rose rash, infants (exanthem subitem)	Widespread; early childhood
Variola	1	Smallpox	Formerly epidemic & endemic (unless vaccinated)
Erythema infectiosum	2 (perhaps more)	'Fifth' disease; rash, malaise	Sporadic outbreaks
Persistent (Latent) Cytomegaloviruses (salivary gland)	1	Congenital defects (cytomegalic inclusion disease); hepatitis (CMV mononucleosis)	Virus widespread; recognized disease uncommon

Hepatitis (1) Type A	1	Hepatitis A	Widespread; often epidemic
(2) Type B	1	Hepatitis B	Widespread; may follow use of whole blood & derivatives of contaminated equipment
(3) Non-A, Non-B	Unknown	Hepatitis C	Similar to Type B
Papovavirus	1	Warts (Verrucae)	Universal; common; often recurrent
Molluscum contagiosum	1	Molluscum tumours	Infrequent

GROUP I: CONTAGIOUS DISEASES

RESPIRATORY VIRAL INFECTIONS (RI):

(1) THE COMMON COLD

Symptoms

This is an acute, usually afebrile, catarrhal respiratory tract infection, with major involvement of any or all airways, including nose, paranasal passages, throat, larynx, and often the trachea and bronchi.

Onset is abrupt after a short (1 to 3 days) incubation period. Illness generally begins with throat discomfort, followed by sneezing, rhinorrhea and malaise. Characteristically it is an afebrile illness, but fever of 38 C to 39 C (100 to 102 F) can occur in children and infants. Pharyngitis, laryngitis, and tracheitis with substernal tightness and burning discomfort are variable symptoms related to the individual patient and the etiologic agent. Secretions thicken in the course of uncomplicated infection. Hacking cough lasts into the second week. severe tracheobronchial involvement with purulent sputum suggests primary or secondary cheobronchial involvement with purulent sputum suggests primary or secondary bacterial invasion. In the absence of complications, symptoms normally resolve in 4 to 10 days.

Background and Causes

Many viruses cause the common cold, including rhino-, influenza, parainfluenza, respiratory syncytial, corona, adeno-, certain echo-, and coxsackieviruses. The common cold has a striking seasonal relationship. Spring, summer and autumn colds are often picornal-virus infections; late fall and winter colds are most frequently paramyxo- or myxovirus infections.

Predisposing factors have not been clearly identified. Chilling of the body surface will not in itself induce colds, and susceptibility is not affected either by the persons health and nutrition or by upper respiratory tract abnormalities (eg. enlarged tonsils or adenoids). Infection may be facilitated by excessive fatigue, emotional distress, or allergic nasopharyngeal disorders and during the midphase menstrual cycle.

(2) Respiratory Syncytial Virus (RSV)

Symptoms

RSV is one of the more important causes of lower respiratory illness (Including bronchiolitis and pneumonia) in infants and young children, and can be fatal. The sudden death of a baby with respiratory disease is often believed to be due to RSV infection. In healthy adults, RSV causes mild upper respiratory illness, but it is also an important cause of influenza, bronchopneumonia, and exacerbation of chronic bronchitis. Elderly people and those with underlying pulmonary disease may be susceptible to infection with RSV.

The clinical manifestations of RSV infection are variable and, in general, differ with age and host factors. Dyspnea, cough and wheezing are the most prominent symptoms; fever and bronchiolitis are the most frequent findings. Crepitant rales are characteristic; bronchopneumonia is often apparent in x-rays.

In adults, infection may be unapparent or only an afebrile URI (common cold); however, RSV infections account for about 15% of hospital admissions for acute exacerbations of chronic bronchitis. Secondary bacterial pneumonia (more commonly pneumococcal) may be a more common complication than is thought.

Causes and Background

RSV is an RNA virus, classified as a paramyxovirus. RSV is associated with a sharp annual outbreak of acute respiratory disease occurring in late autumn or in winter. Like influenza, it increases morbidity and mortality from bronchitis and pneumonia. The annual recurrence of a single RSV serotype indicates that reinfection, with illness, occurs. Although about 70% of persons have serum antibody against RSV by age 5, infections continue to occur in all people of all ages.

(3) Influenza

Symptoms

A specific acute viral respiratory disease characterized by fever, coryza, cough, headache, malaise, and inflamed mucus membranes. It usually occurs as an epidemic in winter. Prostration, haemorrhagic bronchitis, pneumonia and sometimes death in severe cases.

During the 48-hour incubation period, transient asymptomatic viraemia may occur before infection localizes in the respiratory tract. Influenza A or B is sudden in onset, with chilliness and fever up to 39 to 39.5 C (102 to 103 F) developing over 24 hours. Prostration and generalized aches and pains (more pronounced in the back and legs) appear early.

Respiratory tract symptoms may be mild at first, with sore throat, substernal burning, non productive cough, and sometimes coryza; later the respiratory disease becomes dominant. Cough can be severe and productive. Usually after 2 to 3 days acute symptoms subside rapidly and fever ends, though fever lasting up to 5 days may occur without complications. Abnormal bronchociliary clearance and altered bronchiolar air flow are regularly present. Weakness, sweating, and fatigue may persist for several days or occasionally for weeks.

In severe cases, haemorrhagic bronchitis and pneumonia are frequent and can develop within hours. Fulminant fatal viral pneumonia occasionally occurs; dyspnea, cyanosis, haemoptysis, pulmonary edema, and death may proceed as soon as 48 hours after the onset of influenza. Such severe disease is most likely to occur during a pandemic caused by a new influenza A serotype and in persons at high risk.

Causes and Background

Influenza is caused by myxoviruses. Influenza A virus is the most frequent single cause of clinical influenza, which is also caused by influenza B, paramyxo-. and sometimes rhino- or echoviruses. Mode or spread is by person-to-person contact and airborne droplet spray contaminates articles with viruses and may infect people. Infection produces sporadic respiratory illness each year. Acute epidemics occur, on average, every three years, generally nationwide during autumn or early winter. A major shift in the prevalent antigenic type of influenza A virus has occurred about once in a decade, resulting in an acute pandemic. Persons of all ages are afflicted, but prevalence is highest in school children, and the severity is greatest in the very young, aged or infirm.

Epidemics often occur in two waves - the first in students and active members, the second mostly occurs in *'shut-ins' and persons in semi-closed institutions. Influenza B causes epidemics about every fifth year* and is much less often associated with pandemics. influenza C is an endemic virus that sporadically causes respiratory disease.

(4) PARAINFLUENZA VIRUSES

Symptoms

A group of viruses causing a number of respiratory illnesses varying from the common cold to influenza-like pneumonia, with febrile croup as their most common manifestation.

It is an acute febrile respiratory infection that is clinically indistinguishable from influenza or other respiratory virus infection occurring in the same age group. Onset is marked by fever and moderate coryza. The degree of the malaise is directly related to the height of the fever. In many cases the temperature does not exceed 38 or 39 C (101 or 102 F); in others it may peak several times at 40 C (104 F). Moderate sore throat and a dry cough usually develop early in the disease. Hoarseness and croup are prominent symptoms in many cases; this acute laryngotracheobronchitis is the most severe and dangerous manifestation of parainfluenza virus infections in children.

Fever may subside promptly or continue for 2 to 3 days. In some patients, particularly those who develop lower respiratory tract involvement, fever lasting a week or more may recur one or more times.

Background and Causes

The parainfluenza viruses are RNA paramyxoviruses and consist of four serological distinct agents categorized as Types 1, 2 , 3 and 4.

Infections with Types 1 and 3 are common early childhood; sharp localized outbreaks occur in nurseries, schools, paediatric wards, and orphanages. Widespread community epidemics are prevented by almost universal immunity in adults. Infection with each type produces different epidemiologic patterns. Parainfluenza infections occur in all seasons, but epidemic disease in autumn is more likely to be due to Type 1. The epidemic types, 1 and 2, tend to recur reciprocally every other year. Type 3 disease is endemic, highly contagious, occurs in all seasons and causes one-third of the infections.

Second and even third infections with the same strains of virus, particularly with Types 1 and 3, are not uncommon, though the partial immunity developed during previous episodes may reduce the spread and severity of subsequent infections.

(5) ADENOVIRUSES

Symptoms

A group of viruses causing a variety of acute febrile disorders characterized by inflammation of the respiratory and ocular mucus membranes and hyperplasia of the submucous and regional lymphoid tissue.

Acute febrile respiratory disease is the usual manifestation of known adenoviral infection in children. However, some adenoviruses are of possible interest in the building related sense.

ARD (Acute respiratory disease) is observed in military recruits during periods of mobilisation. ARD is characterized by malaise, fever, chills, and headache. Respiratory manifestations including asopharyngitis, hoarseness, and dry cough. The disease may resemble streptococcal pharyngitis with exudate on the faucial pillars and posterior phalangeal wall. Cervical adenopathy is present, but the nodes are not as tender as in streptococcal pharyngitis. Viraemia and viruria may occur and there may be a fine erythematous macular rash on the body. Fever usually subsides within 2 to 4 days.

APC (Acute pharyngconjunctival fever) produces the clinical triad of fever, pharyngitis, and conjunctivitis. Infection is sometimes waterborne. In a typical outbreak 50% or more of the patients have all 3 components, while others may have only 1 or 2. The conjunctivitis is initially unilateral and sometimes painful. Involvement of the lower respiratory tract may occur in addition to pharyngitis. The illness usually subsides within a week, but follicular conjunctivitis may persist for another week.

Conjunctivitis without constitutional symptoms appears to be a common manifestation of infection with several adenovirus serotypes. It occurs most often in young adults, chiefly parents of children with APC, and is self-limiting and benign. Symptoms and signs include a foreign body sensation in the eye, lacrimation, and focal erythema of the palpebral and

bulbar conjunctiva. The discharge is mucoid but not purulent. The other eye is subsequently involved in about half of the patients, usually less severely. A mild sore throat occasionally develops, often on the same side as the affected eye.

EKC *(Epidemic keratoconjunctivitis)* is a specific, sometimes severe, epidemic disease caused by adenovirus. Widespread epidemics have occurred in the USA, Europe and Asia. Onset is sudden one eye showing redness and chemosis followed by periorbital swelling, preaucular lymphadenopathy, and superficial corneal opacities. Systemic symptoms and signs are mild or absent. The illness usually lasts 3 to 4 weeks, though opacities may persist much longer and vision has sometimes been permanently impaired.

Background and Causes

Adenoviruses are DNA viruses 60 to 90 nm in size. Not infrequently, adenoviruses have another smaller DNA vitus associated with them, called adenoassociated virus (AAV). About 4 to 5% of clinically recognised respiratory illnesses are caused by adenoviruses.

Acute respiratory disease (ARD) occurs in military camps. Epidemic keratoconjunctivitis (EKC) is seen largely in industrial plants.

MYCOTIC INFECTIONS

(1) ASPERGILLOSIS

Symptoms

An 'opportunist' appears after antibacterial or antifungal therapy (to which it is usually resistant) in bronchi damaged by bronchitis, bronchiectasis, or tuberculosis. The 'fungus ball' (aspergilloma), a characteristic form of the disease, appears as a dense round ball, capped by a slim meniscus of air, in the cavity. Aspergillomas usually occur in old cavitary disease (eg., tuberculosis) or in patients with rheumatoid spondylitis. Symptoms (cough, sputum, dyspnea) and findings on the physical examination or chest film are usually those of the underlying disease.

Background and Causes

An infectious disease of the lung with occasional haematogenous spread, caused by various Aspergillus, especially *A.fumigatus*. A noninvasive pulmonary disorder may also occur as an allergic reaction to *A.fumigatus*.

(2) HISTOPLASMOSIS

Symptoms

There are three recognized forms of the disease. The primary acute form causes symptoms (fever, cough, malaise) indistinguishable in endemic areas from undifferentiated URI or grippe-like disease. The progressive disseminated form follows haematogenous spread from the lungs and is characterized by hepatomegaly, lymphadenopathy, splenomegaly, and, less

frequently, oral or GI ulceration. Addison's disease is an uncommon but serious manifestation. The lesions in the liver are granulomatous, show the intercellular fungus, and may lead to hepatic calcification. The chronic cavitary form produces pulmonary lesions indistinguishable, except by culture, from cavitary TB. The principal manifestations are cough, increasing dyspnea, and eventually disabling respiratory embarrassment.

Background and Causes

This is an infectious disease caused by Histoplasma capsulatum, characterized by a primary pulmonary lesion and occasional haematogenous dissemination, with ulceration of the oropharynx and GI tract, hepatomegaly, splenomegaly, lymphadenopathy, and adrenal necrosis.

Infection follows inhalation of dust that contains spores. Severe disease is more frequent in males.

(3) Coccidioidomycos

Symptoms

An infectious disease, caused by the fungus Coccidiodies immitis, occurring in a **primary form** as an acute, benign, self-limiting respiratory disease, or in a **progressive form** as a chronic, often fatal, infection of the skin, lymph glands, spleen, liver, bones, kidneys, meninges, and brain.

Primary pulmonary coccidioidomycosis, the more common form, may occur asymptomatically, as a mild URI, as an acute bronchitis, occasionally with pleural effusion, or as a pneumonia. Symptoms, in descending order of frequency, include pleural fever, cough, chest pains, sputum production, sore throat, and haemoptysis. Physical signs may be absent, or occasional scattered rales and areas of dullness to percussion may be present. Leucocytoses is present and the eosinophil count may be high. Some patients develop 'desert rheumatism,'a more recognizable form with conjunctivitis, arthritis, and erythema nodosum.

Progressive coccidioidomycosis develops from the primary form; evidence of dissemination may appear a few weeks, months, or, occasionally, years after primary infection. Symptoms include continuous low-grade fever, severe anorexia, and loss of weight and strength. Progressive cyanosis, dyspnea, and mucopurulent or bloody sputum are present in the pulmonary type. The bones, joints, skin, viscera, brain, and meninges may be involved as the disease spreads.

Background and Causes

Infection is acquired by inhalation of dust containing the spores of the fungus *Coccidiodies immitis*. The disease is endemic to certain areas.

(4) BLASTOMYCOSIS

Symptoms

An infectious disease caused by the fungus **Blastomyces dermatitides**, primarily involving the lungs and occasionally spreading hematogenously, characteristically to the skin.

Pulmonary form: Primary pulmonary blastomycosis frequently forms patches of bronchopneumonia that appear, on chest film, to fan out from the hilum like a neoplastic growth. Onset is usually insidious. A dry hacking or productive cough, chest pain, fever, chills, drenching sweats, and dyspnea are initial symptoms.

Systemic form: Sites of haematogenous spread include skin, prostrate, epididymis, testis, bone and subcutaneous tissue, and rarely, oral or nasal mucosa. The vertebrae, tibia, and femur are more commonly involved than other bones; swelling, heat and tenderness are present over the lesion. Genital tract lesions are characterized by painful swelling.

Skin lesions begin as papules, or papulopustules on exposed surfaces and spread slowly. Painless miliary abscesses, varying from pinpoint to 1 mm in diameter develop on the advancing borders. Irregular, wartlike papillae form on the surfaces. As the lesions enlarge, the centre heals with a typical atrophic scar. A fully developed individual appears as an elevated verrucous patch measuring 2 cm or larger with an abruptly sloping, purplish-red, abscess-studded border. Ulceration may occur if bacteria are present.

Background and Causes

B. dermatitides is a fungus of unknown natural source. Most reported cases are from the USA, chiefly in the southeastern states of the Mississippi River valley, and occur in man aged 20 to 40. A sufficient number of cases from widely scattered sites in Africa and precludes geographic limitation.

(5) CRYPTOCOCCOSIS

Symptoms

An infectious disease due to the fungus *Filobasidiella neoformans*, with a primary focus in the lung and characteristic spread to the meninges and occasionally to the kidneys, bone and skin.

Meningitis with headache is the most common form. The patient seeks medical care because of blurred vision or is brought to a physician because of such mental disturbances as confusion, depression, agitation, or inappropriate speech or dress.

Background

Distribution is worldwide. Most cases occur in adults aged 40 to 60, and in men more often than women. Individuals with Hodgkin's disease are particularly susceptible.

(6) SPOROTRICHOSIS

This is an infectious disease caused by the plant saprophyte Sporothrix, and characterized by the formation of nodules, ulcers, and abscesses, usually confined to the skin and lymph channels but occasionally affecting the lung or other tissues.

The most common form, cutaneous-lymphatic, occurs characteristically on the arm and hand. The primary lesion, usually on the finger, begins as a small, movable, non-tender, subcutaneous nodule that slowly enlarges, adheres to the skin, becomes pink and later necrotic, and finally ulcerates. In a few days or weeks, similar discoloured subcutaneous nodules appear along the course of the lymphatic channels. Local pain, heat and general symptoms are notably absent.

Inhalation of the microorganism can cause pneumonia. Symptoms are relatively mild.

GROUP IV: HYPERSENSITIVITY DISEASES

It is difficult to formulate a classification that adequately categorizes the gamut of diseases in which hypersensitivity reactions may play a role. These diseases range from hay fever (which results from hypersensitivity to an exogenous antigen, is limited to the respiratory and ocular mucosal surfaces, and lacks systemic morbidity) or SLE (a multi-system disease with significant morbidity.

TABLE 45 - CAUSES OF HYPERSENSITIVITY PNEUMONITIS

Disease	*Antigen*	*Source of Particles*
Farmer's lung	Micropolyspora faeni or Thermoactinomyces Vulgaris	Mouldy hay
Bird fancier's lung; pigeon breeder's lung; hen worker's lung	Serum proteins and droppings	Parakeets; pigeons; hens
'Air-conditioner (or humidifier) lung	M.*faeni*, T.*vulgaris*	Humidifiers, air conditioners
Bagassosis	M.*faeni*, T.*vulgaris*	Bagasse (sugar cane waste)
Mushroom worker's lung	M.*faeni*, T.*vulgaris*	Mushroom post-spawning compost
Suberosis (cork worker's lung)	Mouldy cork dust	Mouldy cork
Maple bark disease	*Cryptostroama cortical*	Infected maple bark

Malt worker's lung	*Aspergillus fumigatus or A.clavatus*	Mouldy barley, malt
Sequoiosis	*Pullularia pullulans* or *Graphium* species	Mouldy sawdust from redwoods
Cheesewasher's lung	*Penicillium species*	Mouldy cheese
Wheat weevil disease	*Sitophilus granarius*	Infested wheat flour
Pituitary snuff taker's lung	Bovine or porcine serum protein or pituitary antigens	Heterologous pituitary snuff
Fishmeal worker's lung	Unknown	Fish meal
Furrier's lung	Animal hair or dander	Animals pelts
Thatched roof worker's lung	Unknown	Straw, reed, etc., used as roofing
Coffee worker's lung	Coffee bean dust	Coffee beans

HYPERSENSITIVITY ATOPIC DISEASES

Disorders caused by Type I hypersensitivity, resulting from a release of vasoactive substances by mast cells and basophils that have been sensitized by the interaction primarily with IgE (*reaginic* or *skin-sensitizing* antibody). the terms hypersensitivity and allergy are often used synonymously to mean the exaggerated response to an antigen, leading to various types of tissue damage.

The most common human allergic disorders - hay fever (seasonal allergic rhinitis), asthma (particularly in children), infantile eczema, and some cases of urticaria and GI food reactions - are atopic diseases. Patients with atopic diseases have in common an inherited predisposition to develop hypersensitivity to substances in the environment that are harmless to 80% of people. Features similar to atopy have been identified in several mammalian species.

(1) ALLERGIC RHINITIS

Symptoms

A symptom complex including hay fever and perennial allergic rhinitis, characterized by seasonal or perennial sneezing, rhinorrhea, nasal congestion, pruitis, and often conjunctivitis and pharyngitis.

The roof of the mouth, pharynx, and eyes begin to itch gradually or abruptly after the onset of the pollen season. Lacrimation, sneezing, and clear, watery nasal discharge accompany or soon follow the pruritus. Frontal headaches, irritability, anorexia, depression and insomnia may appear. The conjunctiva is injected and the nasal mucous membranes are swollen and bluish red. Coughing and asthmatic wheezing may develop as the season progresses.

Background and Causes

Hay Fever is the acute seasonal form of allergic rhinitis, it is generally induced by wind-borne pollens. The spring type is usually due to tree pollens; the summer type to grass and weed pollens; and the autumn type to seed pollens (eg. ragweed). Occasionally, hay fever is due to airborne fungus spores. Important geographical regional differences occur.

(2) Perennial allergic Rhinitis

In contrast to hay fever, symptoms of perennial rhinitis vary in severity (often unpredictably) throughout the year. Extranasal symptoms such as conjunctivitis are uncommon, but chronic nasal obstruction is often prominent and may extend to eustachian tube obstruction. The resultant hearing difficulty is particularly common in children. The diagnosis of allergic rhinitis is supported by a positive history of atopic disease, the characteristic bluish-red mucosa, numerous eosinophils in the nasal secretions, and positive skin tests (particularly to house dust, feathers, animal danders, or fungi, and occasionally to foods).

Certain sufferers have chronic rhinitis, sinusitis, and polyps and often have negative skin tests. These people are not allergic but often have aspirin and indomethacin sensitivity. When patients suffer with mild but annoying chronic continuous nasal obstruction or rhinorrhea, have no demonstrable allergy, polyps, infection or drug sensitivity, a condition identified as vasomotor rhinitis is indicated.

(3) Anaphylaxis

Generalized anaphylaxis is an acute, often explosive, systemic reaction characterized by urticaria, respiratory distress, and vascular collapse and occasionally by vomiting and abdominal cramps. It occurs in a previously sensitized person who is further introduced to the sensitizing antigen.

Typically, in 1 to 15 minutes, the patient complains of a sense of uneasiness and becomes agitated and flushed. Palpitation, paresthesias, pruritus, throbbing in the ears, coughing, sneezing, and difficulty in breathing are other typical complaints.

The most common causative antigens are foreign serum and other proteins, certain drugs, desensitizing injections, and insect stings.

Occasional occurrences of anaphylaxis within buildings have been recorded (eg. reactions to fly sprays).

APPENDIX 10

APPENDIX 10. ADVERSE HEALTH OUTCOMES FROM ELECTRO-MAGNETIC FIELDS

The literature involving childhood cancers provides some evidence for an effect of electromagnetic fields, although the findings are inconsistent on the strength of the associations and the specific cancers involved (Bethwaite et al., 1991).

The sentinel study undertaken in the United States by Wertheimer and Leeper assigned all the households occupied by 344 subjects who died with childhood cancers and matched controls to one of four levels of EMF exposure (Wertheimer et al., 1979). The surrogate level of exposure was assessed on the arrangement of wiring configurations of powerlines and transformers and their distance from the home. Children dying from cancer were found to have lived in homes near high current configurations more often than controls. Increased risks of 2-3 times were produced for leukaemia, nervous system tumours and lymphoma for residence near high current configurations.

Fulton et al., (1980) attempted to reproduce Wertheimer and Leeper's study design in Rhode Island, for leukaemia but not other cancers. They studied a hospital series of 110 cases of leukaemia diagnosed in the period 1964-78 in people aged 20 or less, and compared them with 225 controls from the general population, matched to cases on year of birth. They assessed exposure for all sources within 46 metres of each address. They were unable to discover an association between leukaemia and degree of exposure. However it was argued that the selection of controls in this study created a bias towards urban residence. With re-analysis, after partial correction for bias, an association with residence near high current configurations in children under eight years of age was noted (Coleman et al., 1988).

A Swedish study by Tomenius et al., (1986), found that the relative risk of cancer in people living at addresses with field strength of 3 mG or more was 2.7 times, and when analysis was further restricted to subjects living at the same address since birth, the relative risk was 5.6 times. The relative risk of malignant disease for residence near 200 kV wires was higher in females than males, although not significantly so. The relative risk was 2.9 times for children under five. All the results were significant at a Confidence Interval of 95%.

Wertheimer et al., (1982), reported a second study of 1179 adults with cancer in the periods 1967-75, and 1179 controls matched for age, sex and socioeconomic level. Residential exposure data were obtained by a similar method as that in their first study. Again, they reported an increase in cancer risk in people living near high density wiring configurations. However, the methodology used in this study was widely criticized. It was claimed that controls were not selected in a uniform manner making interpretation of the results difficult.

In a British study, McDowall (1983), studied a cohort of 7613 people in East Anglia who at the time of the 1971 census lived within 30 metres of an overhead high-tension power line or within 50 metres of an electrical transformer substation, and determined the mortality compared to the general population mortality ratios, for the years 1971-1983. While there appeared to be an increase in cancers amongst the group, the results were not statistically significant.

A similar population-case control study in four London boroughs also failed to detect an excess risk from residential proximity to overhead powerlines and electricity substations (Coleman et al., 1985). However, reanalysis of the results showed that the relative risk of leukaemia rose slightly and non-significantly to 1.3 times within 25 metres of a substation.

When electricity distribution is via underground cables, the exposure cannot be assessed in the same manner as overhead power lines . The unassessed contribution to electromagnetic field exposure in likely to be small, since the helical configuration of conductors in such cables results in a small net field which decays very rapidly with distance - approximately as the inverse cube (Coleman et al., (1988).

Data from a small case control study by Savitz et al., (1990), has shown a small increase in the incidence of childhood cancers , including leukaemia, with prenatal and postnatal electric blanket exposure. However, studies of adult cancers and residential EMF exposures have produced inconsistent results.

Specific Cancers & Electrical Occupations

Effort has gone into investigating the risks of EMFs in the occupational setting, where exposure intensities may be several times greater than residential levels. Occupational studies have been easier to conduct with access to job titles on the tumour registry and death certificate data.

Milham (1982), reported an excess of leukaemia amongst electrical workers. The mortality ratio was especially high in power station operators, aluminium-reduction workers and motion-picture projectionists.

McDowall (1983), studied all deaths of men aged 15-74 in England and Wales, from 1970-72, among 10 electrical occupations. Four of the 10 occupational groups had higher than expected mortality, particularly from acute myeloid leukaemia. These groups included electrical and electronic engineers and telegraph operators.

Coleman et al., (1983), examined 125,887 incident tumours registered to males aged 15-744 in the South Thames Cancer Registry for which the occupation at registration was known. They looked at the same ten occupational groups as McDowall. Eight of the ten groups showed an excess of leukaemia, although these excesses were statistically significant only for electrical and electronic fitters and for radio and telegraph operators.

Calle et al., (1985), found no significant overall excess in proportional mortality for electrical workers between 1963-68 in Wisconson among white males aged 20 or more.

In a New Zealand study of cancer in electrical workers, Pearse et al., (1989) found an excess of leukaemia amongst eight groups occupationally exposed to electromagnetic fields. The two groups at most risk were electronic equipment assemblers and television repairmen.

In a large Canadian study Howe et al., (1983), reported a twofold increase in mortality from leukaemia and non-Hodgkin's lymphoma during the period 1965-79, amongst power and telephone linemen. No other electrical occupation showed a significant association with these two causes of death.

Milham, (1985), reported a twofold excess of leukaemia mortality in amateur radio operators for acute myeloid leukaemia. The excess mortality was independent of employment in electrical occupations (Wangler et al., 1985; Coleman, 1985).

Stern et al., (1986), investigated 53 leukaemia deaths at a naval nuclear shipyard in Maine. The principal exposures of interest were ionizing radiation and organic solvents. The three fold excess of leukaemia observed among electricians was an unexpected finding which persisted after an adjustment for radiation and solvent exposure.

Wiklund et al., (1981), used data from the Swedish Cancer Environment Registry to investigate a suggestion of increased leukaemia incidence among Telecommunication Administration employees. They found 12 cases of leukaemia, compared to the 11.5 expected, based on Swedish cancer rates.

Vagero et al., (1983), also used the Swedish Cancer Environment Registry to examine cancer incidence in the electronics industry. They found a small but significant excess in the incidence of all cancers (1.15 in men & 1.08 in women). There was a twofold risk in pharyngeal cancer, but no excess of leukaemia.

Olin et al., (1985), studied the mortality of 1254 electrical engineers who graduated from Stockholm in the period 1930-59. There were no excess cancer deaths as a whole, but three fatal melanomas were observed (0.9 expected), and two of those were in electrical transmission workers.

Swerdlow (1983), reported a significant excess of cancer of the eye amongst male electrical workers aged 15-74 in England and Wales.

Lin et al., (1985), reported a twofold risk of brain tumours in electrical workers, and a positive trend in risk with estimated electromagnetic field exposure. The risk was present for tumours specified as gliomas or astrocytomas, but not for brain tumours of an unspecified type.

Spitz et al., (1985), carried out a population based case-control study to assess the association of neuroblastoma with paternal occupation recorded at birth. When all the paternal occupations with potential electromagnetic field exposure were grouped together, the relative risk of neuroblastoma in children was 2.1; within the risk group, the relative risk for children of electronics workers was 11.75.

On the basis of these studies, an excess of leukaemia, especially acute myeloid leukaemia, is apparent in electrical workers. Although the evidence regarding brain cancer is less extensive, several observations of an association with electrical occupations has been reported. Given the methodological limitations of the studies, the consistency of the findings is notable (Savitz et al., 1989).

Risk of Non-Malignant Disease

Reichmanis et al., (1979), reported an association between addresses of people committing suicide and the estimated intensity of electric and magnetic fields resulting from transmission of electricity. The study developed from a clinical observation that depressive illness appeared to be more common in people who lived near high voltage transmission lines. The

outcome of the survey was inconclusive. However, Perry et al., (1981) reanalysed the same data and took physical measurements at the sites of the suicides. The residences of those who committed suicide had significantly higher magnetic fields than the control homes. The relative risk of suicide was estimated at 1.5 in homes with 'high' (1 mG or greater) 50 Hz fields. There was a significant trend in the risk of suicide with increasing field strength.

McDowall's study in East Anglia did not support Perry et al's findings, nor did it suggest that mortality from any other non-malignant cause of death was increased in people living in the vicinity of electrical transmission lines.

Foetal Risk, Birth Defect and Adverse Outcomes

Nordstrom et al., (1983), reported on 372 married couples of whom the father worked had worked for one of two Swedish power companies. The couples reported 880 pregnancies between 1953-79, excluding induced abortions. Perinatal deaths, congenital malformations, and spontaneous abortions were regarded as adverse outcomes. Men who worked in shipyards were exposed to the highest voltages and this group reported adverse outcomes more frequently (19% compared to 11%). The excess was due to a higher frequency of congenital malformation among their offspring (8% compared too 2%). There was no consistent pattern in the types of malformation reported.

Wertheimer et al., (1986), studied the use of electric over blankets and electrically heated waterbeds during pregnancy. The devices gave rise to a magnetic field exposure of 10-15 mG and 3-4 mG at power frequency respectively, if switched on while the bed was in use. The authors reported that births to couples who had used electrically heated beds showed a seasonal pattern: births conceived in the seasons when electrical bed heating would be in use, were of longer gestation but of lower birthweight than births conceived when the heating was not in use. Births to couples who did not report using electrically heated beds showed no such seasonal patterns.

APPENDIX 11

APPENDIX 11. QUESTIONNAIRE USED

The following 'anonymous questionnaire' was used in the building surveys and by the telephone interviewers to collect the base line data.

OFFICE ENVIRONMENT SURVEY

This questionnaire is trying to establish whether there is a relationship between the occupancy of office buildings and the number and severity of commonly experienced symptoms.

Please would you answer the following questions.

To ensure complete confidentiality, **DO NOT** write your name on the questionnaire.

BACKGROUND INFORMATION

---

1. How would you describe your main work activity....(Please circle one number)

Managerial? 1
Professional? 2
Clerical/secretarial? 3
V.D.U. Operator? 4
Other (Please specify)

---

2. To which age group do you belong....(Please circle one number)

Under 30 ? 1
30-39 ? 2
40-49 ? 3
50-59 ? 4
60 and Over? 5

---

3. What is your gender .....(Please circle one number)

Female? 1
Male? 2

---

4. Are you.....(Please circle one number)

A smoker? 1
A former smoker? 2
A non-smoker? 3

---

5. Do other people smoke in your immediate working environment? (Please circle one number)

Yes 1
No 2

SECTION 2. The following questions ask about your general well-being over the last 12 months. The majority of people have experienced most of these symptoms at one time or another.

We are interested in finding out how frequently you experience these symptoms.

0	1	2	3	4
Have never or almost never	Once every 2 - 3 weeks	1 or 2 days each week	3 - 4 days each week	Every day spent at work

Please circle only one number for each item. If you never experience a particular symptom circle 0. If you are bothered on a daily basis, circle 4 and so on.

Dry eyes	0	1	2	3	4
Irritated or Watering Eyes	0	1	2	3	4
Strained eyes	0	1	2	3	4
Stuffy or Congested nose	0	1	2	3	4
Running nose	0	1	2	3	4
Irritated or itchy nose	0	1	2	3	4
Sneezing episodes	0	1	2	3	4
Dry throat	0	1	2	3	4
Sore or irritated throat	0	1	2	3	4
Cough or hoarseness	0	1	2	3	4
Headache	0	1	2	3	4
Lethargy or drowsiness	0	1	2	3	4
Concentration or memory difficulties	0	1	2	3	4
Dizziness or faintness	0	1	2	3	4
Flu like symptoms*******	0	1	2	3	4
Aching limbs or backache	0	1	2	3	4
Difficulty in breathing	0	1	2	3	4
Feeling of chest tightness	0	1	2	3	4
Wheezing	0	1	2	3	4
Dry or itching skin	0	1	2	3	4
Skin blotches or raised areas of skin	0	1	2	3	4

******* Flu like symptoms = nausea, shivering, fever, weakness********

SECTION 3.

In the previous section we asked you how frequently you experienced a number of common symptoms. We are now interested in finding out how bothered by or how severely you experience these symptoms.

We will give you a new scale, again please only circle one number on each line. .....

0	1	2	3	4
Not at all	A little	Moderately	Quiet a bit	Extremely

So how bothered are you by.....

Dry eyes 0 1 2 3 4

Irritated or Watering Eyes 0 1 2 3 4

Strained eyes 0 1 2 3 4

Stuffy or Congested nose 0 1 2 3 4

Running nose 0 1 2 3 4

Irritated or itchy nose 0 1 2 3 4

Sneezing episodes 0 1 2 3 4

Dry throat 0 1 2 3 4

Sore or irritated throat 0 1 2 3 4

Cough or hoarseness 0 1 2 3 4

Headache 0 1 2 3 4

Lethargy or drowsiness 0 1 2 3 4

Concentration or memory difficulties 0 1 2 3 4

Dizziness or faintness 0 1 2 3 4

Flu like symptoms****** 0 1 2 3 4

Aching limbs or backache 0 1 2 3 4

Difficulty in breathing 0 1 2 3 4

Feeling of chest tightness 0 1 2 3 4

Wheezing 0 1 2 3 4

Dry or itching skin 0 1 2 3 4

Skin blotches or raised areas of skin 0 1 2 3 4

****** Flu like symptoms = nausea, shivering, fever, weakness*******

APPENDIX 12

SYMPTOMS Direct Undifferentiated Symptom Comparison BUILDING 1	STATISTICS Z-Score	2-tailed-P	$a^{.05}$	Direction
Frequency Dry Eyes	-1.8896	0.0588	No	
Frequency Irritated or Watering Eyes	-3.3134	0.0009	Yes	-
Frequency Strained Eyes	-0.9888	0.3228	No	
Frequency Stuffy or congested nose	-3.0976	0.0020	Yes	-
Frequency running nose	-3.5701	0.0004	Yes	-
Frequency irritated or itchy nose	-2.9849	0.0028	Yes	-
Frequency sneezing episodes	-2.2660	0.0234	Yes	-
Frequency dry throat	-4.0363	0.0001	Yes	-
Frequency sore or irritated throat	-3.7339	0.0002	Yes	-
Frequency cough or hoarseness	-0.1472	0.8830	No	
Frequency headache	-2.3316	0.0197	Yes	-
Frequency lethargy or drowsiness	-1.4777	0.1395	No	
Frequency concentration/memory difficulty	-0.7644	0.4446	No	
Frequency dizziness or faintness	-0.0220	0.9825	No	
Frequency flu like symptoms	-1.0131	0.3110	No	
Frequency aching limbs or backache	-1.9386	0.0526	No	
Frequency difficulty in breathing	-1.7123	0.0868	No	
Frequency chest tightness	-1.4208	0.1554	No	
Frequency wheezing	-1.1874	0.2351	No	
Frequency dry or itching skin	-2.5264	0.0115	Yes	-
Frequency skin blotches	-0.7082	0.4788	No	
TOTAL SIGNIFICANT DIFFERENCES			9	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**9/0**

SYMPTOMS Direct Undifferentiated Symptom Comparison BUILDING 1	STATISTICS		a.[05]	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-2.3956	0.0166	Yes	-
Severity Irritated or Watering Eyes	-3.2524	0.0011	Yes	-
Severity Strained Eyes	-0.6469	0.5177	No	
Severity Stuffy or congested nose	-3.0507	0.0023	Yes	-
Severity running nose	-3.1161	0.0018	Yes	-
Severity irritated or itchy nose	-2.7462	0.0060	Yes	-
Severity sneezing episodes	-1.8344	0.0666	No	
Severity dry throat	-4.2271	0.0000	Yes	-
Severity sore or irritated throat	-4.2480	0.0000	Yes	-
Severity cough or hoarseness	-2.2038	0.0275	Yes	-
Severity headache	-2.0681	0.0386	Yes	-
Severity lethargy or drowsiness	-2.4208	0.0155	Yes	-
Severity concentration/memory difficulty	-2.4280	0.0152	Yes	-
Severity dizziness or faintness	-1.5712	0.1161	No	
Severity flu like symptoms	-0.5819	0.5607	No	
Severity aching limbs or backache	-3.2552	0.0011	Yes	-
Severity difficulty in breathing	-2.2971	0.0216	Yes	-
Severity chest tightness	-1.3612	0.1734	No	
Severity wheezing	-1.5570	0.1195	No	
Severity dry or itching skin	-1.9661	0.0493	Yes	-
Severity skin blotches	-1.8742	0.0609	No	-
TOTAL SIGNIFICANT DIFFERENCES			14	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**14/0**

SYMPTOMS Job Description = Management BUILDING 1	STATISTICS		a.[05]	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-0.4990	0.6178	No	
Frequency Irritated or Watering Eyes	-2.7622	0.0057	Yes	-
Frequency Strained Eyes	-0.1691	0.8657	No	
Frequency Stuffy or congested nose	-0.9065	0.3647	No	
Frequency running nose	-0.9518	0.3412	No	
Frequency irritated or itchy nose	-1.2703	0.2040	No	
Frequency sneezing episodes	-0.2662	0.7901	No	
Frequency dry throat	-0.1366	0.8913	No	
Frequency sore or irritated throat	-0.5049	0.6136	No	
Frequency cough or hoarseness	-0.9060	0.3650	No	
Frequency headache	-1.5722	.01159	No	
Frequency lethargy or drowsiness	-0.2949	0.7680	No	
Frequency concentration/memory difficulty	-0.7623	0.4459	No	
Frequency dizziness or faintness	-0.4156	0.4459	No	
Frequency flu like symptoms	-0.7039	0.4815	No	
Frequency aching limbs or backache	-1.4406	0.1497	No	
Frequency difficulty in breathing	-1.4776	0.1395	No	
Frequency chest tightness	-0.4274	0.6691	No	
Frequency wheezing	-1.0988	0.2719	No	
Frequency dry or itching skin	-0.1658	0.8683	No	
Frequency skin blotches	-0.6040	0.5459	No	
TOTAL SIGNIFICANT DIFFERENCES			1	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**1/0**

SYMPTOMS Job Description = Management BUILDING 1	STATISTICS		a.[05]	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-1.5305	0.1259	No	
Severity Irritated or Watering Eyes	-2.2643	0.0079	Yes	-
Severity Strained Eyes	-1.4081	0.1591	No	
Severity Stuffy or congested nose	-0.0824	0.9343	No	
Severity running nose	-1.0462	0.2955	No	
Severity irritated or itchy nose	-0.0670	0.9466	No	
Severity sneezing episodes	-0.5849	0.5586	No	
Severity dry throat	-0.3100	0.7566	No	
Severity sore or irritated throat	-2.0382	0.0415	Yes	-
Severity cough or hoarseness	-0.2092	0.8343	No	
Severity headache	-2.0382	0.0415	Yes	-
Severity lethargy or drowsiness	-0.0799	0.9364	No	
Severity concentration/memory difficulty	-1.6469	0.0996	No	
Severity dizziness or faintness	-1.6388	0.1012	No	
Severity flu like symptoms	0.0000	1.0000	No	
Severity aching limbs or backache	-0.7077	0.4791	No	
Severity difficulty in breathing	-1.2229	0.2214	No	
Severity chest tightness	-0.5603	0.5753	No	
Severity wheezing	-1.0657	0.5753	No	
Severity dry or itching skin	-0.7325	0.4638	No	
Severity skin blotches	-0.4842	0.6083	No	
TOTAL SIGNIFICANT DIFFERENCES			2	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**2/0**

SYMPTOMS Job Description = Clerical/Secretarial BUILDING 1	STATISTICS		a.[05]	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-0.7706	0.4410	No	
Frequency Irritated or Watering Eyes	-1.8747	0.0608	No	
Frequency Strained Eyes	-0.7268	0.4673	No	
Frequency Stuffy or congested nose	-2.1627	0.0306	Yes	-
Frequency running nose	-2.9478	0.0032	Yes	-
Frequency irritated or itchy nose	-0.9949	0.3198	No	
Frequency sneezing episodes	-0.5740	0.5660	No	
Frequency dry throat	-3.9195	0.0001	Yes	-
Frequency sore or irritated throat	-3.5672	0.0004	Yes	-
Frequency cough or hoarseness	-0.7899	0.4296	No	
Frequency headache	-0.7430	0.4575	No	
Frequency lethargy or drowsiness	-0.4377	0.6616	No	
Frequency concentration/memory difficulty	-0.6319	0.5275	No	
Frequency dizziness or faintness	-1.1287	0.2590	No	
Frequency flu like symptoms	-0.3042	0.7610	No	
Frequency aching limbs or backache	-0.6498	0.5158	No	
Frequency difficulty in breathing	-0.3599	0.7189	No	
Frequency chest tightness	-0.9942	0.3201	No	
Frequency wheezing	-0.8374	0.4023	No	
Frequency dry or itching skin	-1.6548	0.0980	No	
Frequency skin blotches	-0.1037	0.0980	No	
TOTAL SIGNIFICANT DIFFERENCES			4	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**4/0**

SYMPTOMS Job Description = Clerical/Secretarial BUILDING 1	STATISTICS		a.[05]	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-0.7886	0.4304	No	
Severity Irritated or Watering Eyes	-1.5788	0.1144	No	
Severity Strained Eyes	-0.5377	0.5908	No	
Severity Stuffy or congested nose	-2.5277	0.0115	Yes	-
Severity running nose	-1.6824	0.0925	No	
Severity irritated or itchy nose	-0.9887	0.3228	No	
Severity sneezing episodes	-0.4919	0.6228	No	
Severity dry throat	-3.6758	0.0002	Yes	-
Severity sore or irritated throat	-3.6758	0.0002	Yes	-
Severity cough or hoarseness	-0.1614	0.8718	No	
Severity headache	-0.0606	0.9516	No	
Severity lethargy or drowsiness	-0.9761	0.3290	No	
Severity concentration/memory difficulty	-0.3176	0.7508	No	
Severity dizziness or faintness	-0.0410	0.9673	No	
Severity flu like symptoms	0.0800	0.9362	No	
Severity aching limbs or backache	-1.4473	0.1478	No	
Severity difficulty in breathing	-0.4177	0.6762	No	
Severity chest tightness	-1.4092	0.1588	No	
Severity wheezing	-0.1010	0.9196	No	
Severity dry or itching skin	-1.7450	0.0810	No	
Severity skin blotches	-1.2589	0.2081	No	
TOTAL SIGNIFICANT DIFFERENCES			3	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**3/0**

SYMPTOMS Job Description = V.D.U Operator BUILDING 1	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-0.9743	0.3299	No	
Frequency Irritated or Watering Eyes	-0.7264	0.4676	No	
Frequency Strained Eyes	-0.1099	0.9125	No	
Frequency Stuffy or congested nose	-1.7785	0.0753	No	
Frequency running nose	-1.7005	0.0753	No	
Frequency irritated or itchy nose	-2.4136	0.0158	Yes	-
Frequency sneezing episodes	-2.1619	0.0306	Yes	-
Frequency dry throat	-0.9608	0.3367	No	
Frequency sore or irritated throat	-0.7856	0.4321	No	
Frequency cough or hoarseness	-0.4820	0.6298	No	
Frequency headache	-0.8870	0.3751	No	
Frequency lethargy or drowsiness	-0.8002	0.4236	No	
Frequency concentration/memory difficulty	-0.9468	0.3437	No	
Frequency dizziness or faintness	-0.4869	0.6263	No	
Frequency flu like symptoms	-0.6008	0.5479	No	
Frequency aching limbs or backache	-1.1409	0.2539	No	
Frequency difficulty in breathing	-1.9110	0.0560	No	
Frequency chest tightness	-1.1717	0.2413	No	
Frequency wheezing	-1.6210	0.1050	No	
Frequency dry or itching skin	-0.8746	0.3818	No	
Frequency skin blotches	-0.8837	0.3769	No	
TOTAL SIGNIFICANT DIFFERENCES			2	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**2/0**

SYMPTOMS Job Description = Clerical/Secretarial BUILDING 1	STATISTICS		a.[05]	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-1.6658	0.0957	No	
Severity Irritated or Watering Eyes	-1.5235	0.1276	No	
Severity Strained Eyes	-0.7093	0.4781	No	
Severity Stuffy or congested nose	-1.9381	0.0526	No	
Severity running nose	-2.6950	0.0070	Yes	-
Severity irritated or itchy nose	-2.7679	0.0056	Yes	-
Severity sneezing episodes	-2.4637	0.0138	Yes	-
Severity dry throat	-1.1312	0.2580	No	
Severity sore or irritated throat	-0.8522	0.3941	No	
Severity cough or hoarseness	-2.9340	0.0033	No	
Severity headache	-1.1701	0.2420	No	
Severity lethargy or drowsiness	-1.9571	0.0503	No	
Severity concentration/memory difficulty	-1.7826	0.0746	No	
Severity dizziness or faintness	-1.0021	0.3163	No	
Severity flu like symptoms	-1.1507	0.2498	No	
Severity aching limbs or backache	-2.3692	0.0178	Yes	-
Severity difficulty in breathing	-2.0410	0.0412	Yes	-
Severity chest tightness	-1.1823	0.2371	No	
Severity wheezing	-1.8703	0.0614	No	
Severity dry or itching skin	-0.6329	0.5268	No	
Severity skin blotches	-1.3825	0.1668	No	
TOTAL SIGNIFICANT DIFFERENCES			5	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				5/0

SYMPTOMS Age = Under 30 BUILDING 1	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-1.5208	0.1283	No	
Frequency Irritated or Watering Eyes	-2.9057	0.0037	Yes	-
Frequency Strained Eyes	-0.6419	0.5210	No	
Frequency Stuffy or congested nose	-2.7337	0.0063	Yes	-
Frequency running nose	-1.1917	0.0284	Yes	-
Frequency irritated or itchy nose	-2.2022	0.0277	Yes	-
Frequency sneezing episodes	-1.9402	0.0524	No	
Frequency dry throat	-2.9063	0.0037	Yes	-
Frequency sore or irritated throat	-1.9052	0.0567	No	
Frequency cough or hoarseness	-0.8451	0.3981	No	
Frequency headache	-0.2059	0.8368	No	
Frequency lethargy or drowsiness	-0.4641	0.6426	No	
Frequency concentration/memory difficulty	-0.0309	0.9754	No	
Frequency dizziness or faintness	-0.1057	0.9159	No	
Frequency flu like symptoms	-0.0873	0.9304	No	
Frequency aching limbs or backache	-0.5329	0.5941	No	
Frequency difficulty in breathing	-2.0410	0.0412	Yes	-
Frequency chest tightness	-2.2448	0.0248	Yes	-
Frequency wheezing	-1.4252	0.1541	No	
Frequency dry or itching skin	-1.5188	0.1288	No	
Frequency skin blotches	-0.7487	0.4541	No	
TOTAL SIGNIFICANT DIFFERENCES			7	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				7/0

SYMPTOMS Age = Under 30 BUILDING 1	STATISTICS		a.^{05}	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-2.3772	0.0174	Yes	-
Severity Irritated or Watering Eyes	-3.0938	0.0020	Yes	-
Severity Strained Eyes	-0.0301	0.9760	No	
Severity Stuffy or congested nose	-3.0859	0.0020	Yes	-
Severity running nose	-2.3813	0.0173	Yes	-
Severity irritated or itchy nose	-2.5667	0.0103	Yes	-
Severity sneezing episodes	-1.4858	0.1373	No	
Severity dry throat	-2.7420	0.0061	Yes	-
Severity sore or irritated throat	-2.7865	0.0053	Yes	-
Severity cough or hoarseness	-1.5672	0.1171	No	
Severity headache	-0.6997	0.4841	No	
Severity lethargy or drowsiness	-1.2023	0.2293	No	
Severity concentration/memory difficulty	-1.9016	0.0572	No	
Severity dizziness or faintness	-1.9943	0.0461	Yes	-
Severity flu like symptoms	-1.2970	0.1946	No	
Severity aching limbs or backache	-0.7227	0.4699	No	
Severity difficulty in breathing	-3.6366	0.0003	Yes	-
Severity chest tightness	-2.3982	0.0165	Yes	-
Severity wheezing	-3.0981	0.0019	Yes	-
Severity dry or itching skin	-1.3229	0.1859	No	
Severity skin blotches	-1.4041	0.1603	No	
TOTAL SIGNIFICANT DIFFERENCES			11	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**11/0**

SYMPTOMS Age = 30-39 BUILDING 1	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-0.1487	0.8818	No	
Frequency Irritated or Watering Eyes	-0.1362	0.8917	No	
Frequency Strained Eyes	-0.6934	0.4880	No	
Frequency Stuffy or congested nose	-0.6322	0.5273	No	
Frequency running nose	-1.1411	0.2538	No	
Frequency irritated or itchy nose	-0.0378	0.9698	No	
Frequency sneezing episodes	-0.0683	0.9456	No	
Frequency dry throat	-1.9898	0.0466	Yes	-
Frequency sore or irritated throat	-1.9803	0.0477	Yes	-
Frequency cough or hoarseness	-2.1454	0.0319	Yes	-
Frequency headache	-2.1923	0.0284	Yes	-
Frequency lethargy or drowsiness	-1.3672	0.1716	No	
Frequency concentration/memory difficulty	-1.7088	0.0875	No	
Frequency dizziness or faintness	-0.9604	0.3369	No	
Frequency flu like symptoms	-0.5121	0.6086	No	
Frequency aching limbs or backache	-2.3524	0.0187	Yes	-
Frequency difficulty in breathing	-1.0594	0.2894	No	
Frequency chest tightness	-0.5244	0.6000	No	
Frequency wheezing	-1.0594	0.2894	No	
Frequency dry or itching skin	-1.6155	0.1062	No	
Frequency skin blotches	-0.0917	0.9269	No	
TOTAL SIGNIFICANT DIFFERENCES			4	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**4/0**

SYMPTOMS Age = 30-39 BUILDING 1	STATISTICS		a.^{05}	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-0.1949	0.8455	No	
Severity Irritated or Watering Eyes	-0.7261	0.4678	No	
Severity Strained Eyes	-0.7774	0.4369	No	
Severity Stuffy or congested nose	-0.4140	0.6788	No	
Severity running nose	-0.5010	0.6163	No	
Severity irritated or itchy nose	-0.1022	0.9186	No	
Severity sneezing episodes	-0.2291	0.8188	No	
Severity dry throat	-2.0561	0.0398	Yes	-
Severity sore or irritated throat	-1.5864	0.1127	No	
Severity cough or hoarseness	-0.3451	0.7300	No	
Severity headache	-1.7401	0.0818	No	
Severity lethargy or drowsiness	-2.0574	0.0397	Yes	-
Severity concentration/memory difficulty	-2.0399	0.0414	Yes	-
Severity dizziness or faintness	-0.5372	0.5912	No	
Severity flu like symptoms	-0.4379	0.6615	No	
Severity aching limbs or backache	-3.3770	0.0007	Yes	-
Severity difficulty in breathing	-1.4070	0.1594	No	-
Severity chest tightness	-0.9684	0.3329	No	
Severity wheezing	-1.3254	0.1850	No	
Severity dry or itching skin	-1.2328	0.2177	No	
Severity skin blotches	-0.4331	0.6649	No	
TOTAL SIGNIFICANT DIFFERENCES			4	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**4/0**

SYMPTOMS Age = 40-49 BUILDING 1	STATISTICS		a.[05]	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-2.6755	0.0075	Yes	-
Frequency Irritated or Watering Eyes	-2.1319	0.0330	Yes	-
Frequency Strained Eyes	-0.0357	0.9715	No	
Frequency Stuffy or congested nose	-0.0527	0.9580	No	
Frequency running nose	-0.7459	0.4557	No	
Frequency irritated or itchy nose	-1.6124	0.1069	No	
Frequency sneezing episodes	-0.3737	0.7087	No	
Frequency dry throat	-1.7366	0.0825	No	
Frequency sore or irritated throat	-1.7014	0.0889	No	
Frequency cough or hoarseness	-1.3459	0.1783	No	
Frequency headache	-0.8588	0.3904	No	
Frequency lethargy or drowsiness	-0.2435	0.8076	No	
Frequency concentration/memory difficulty	-0.7892	0.4300	No	
Frequency dizziness or faintness	-1.6077	0.1079	No	
Frequency flu like symptoms	-0.7726	0.4397	No	
Frequency aching limbs or backache	-0.3982	0.6905	No	
Frequency difficulty in breathing	-1.9879	0.0468	Yes	-
Frequency chest tightness	-0.5868	0.5573	No	
Frequency wheezing	-1.7535	0.0795	No	
Frequency dry or itching skin	-0.0291	0.9768	No	
Frequency skin blotches	-0.7220	0.4703	No	
TOTAL SIGNIFICANT DIFFERENCES			3	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**3/0**

SYMPTOMS Age = 40-49 BUILDING 1	STATISTICS		a.^{05}	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-2.5950	0.0095	Yes	-
Severity Irritated or Watering Eyes	-2.1483	0.0317	Yes	-
Severity Strained Eyes	-0.0721	0.9425	No	
Severity Stuffy or congested nose	-0.0260	0.9793	No	
Severity running nose	-0.5106	0.6096	No	
Severity irritated or itchy nose	-0.5661	057136	No	
Severity sneezing episodes	-0.7296	0.4656	No	
Severity dry throat	-2.1697	0.0300	Yes	-
Severity sore or irritated throat	-1.5405	0.1234	No	
Severity cough or hoarseness	-1.8075	0.0707	No	
Severity headache	-0.5904	0.5549	No	
Severity lethargy or drowsiness	-0.0858	0.9316	No	
Severity concentration/memory difficulty	-0.3667	0.7138	No	
Severity dizziness or faintness	-1.0145	0.3104	No	
Severity flu like symptoms	-0.9663	0.3339	No	
Severity aching limbs or backache	-2.2228	0.0262	Yes	-
Severity difficulty in breathing	-1.4994	0.1138	No	
Severity chest tightness	-0.6895	0.4905	No	
Severity wheezing	-1.3894	0.1647	No	
Severity dry or itching skin	-0.9052	0.3653	No	
Severity skin blotches	-0.6222	0.5338	No	
TOTAL SIGNIFICANT DIFFERENCES			4	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**4/0**

SYMPTOMS Age = 50-59 BUILDING 1	STATISTICS -		a.[05]	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-1.0085	0.2710	No	
Frequency Irritated or Watering Eyes	-2.2021	0.0277	Yes	-
Frequency Strained Eyes	-0.5879	0.5566	No	
Frequency Stuffy or congested nose	-2.2398	0.0251	Yes	-
Frequency running nose	-2.0166	0.0437	Yes	-
Frequency irritated or itchy nose	-2.6656	0.0077	Yes	-
Frequency sneezing episodes	-1.4050	0.1600	No	
Frequency dry throat	-0.2471	0.8048	No	
Frequency sore or irritated throat	-0.7121	0.4764	No	
Frequency cough or hoarseness	-1.0045	0.3151	No	
Frequency headache	-0.1736	0.8621	No	
Frequency lethargy or drowsiness	-0.2904	0.7715	No	
Frequency concentration/memory difficulty	-0.4120	0.6803	No	
Frequency dizziness or faintness	-0.0000	1.0000	No	
Frequency flu like symptoms	-0.5270	0.5982	No	
Frequency aching limbs or backache	-0.6434	0.5200	No	
Frequency difficulty in breathing	-0.2554	0.7984	No	
Frequency chest tightness	-1.5814	0.1138	No	
Frequency wheezing	-0.3649	0.7152	No	
Frequency dry or itching skin	-0.1025	0.9184	No	
Frequency skin blotches	-0.3715	0.7103	No	
TOTAL SIGNIFICANT DIFFERENCES			4	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**4/0**

SYMPTOMS Age = 40-49 BUILDING 1	STATISTICS		a.^{05}	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-1.2065	0.2276	No	
Severity Irritated or Watering Eyes	-2.4305	0.0151	Yes	-
Severity Strained Eyes	-1.0199	0.3078	No	
Severity Stuffy or congested nose	-1.2626	0.2067	No	
Severity running nose	-1.9125	0.0558	No	
Severity irritated or itchy nose	-1.6559	0.0977	No	
Severity sneezing episodes	-1.2626	0.2667	No	
Severity dry throat	-0.1942	0.8460	No	
Severity sore or irritated throat	-0.4841	0.6284	No	
Severity cough or hoarseness	-0.8010	0.4231	No	
Severity headache	-0.5825	0.5602	No	
Severity lethargy or drowsiness	-0.1382	0.8901	No	
Severity concentration/memory difficulty	-0.3169	0.7513	No	
Severity dizziness or faintness	-0.3650	0.7151	No	
Severity flu like symptoms	-0.9426	0.3459	No	
Severity aching limbs or backache	-1.0615	0.2885	No	
Severity difficulty in breathing	-0.4516	0.6515	No	
Severity chest tightness	-1.2706	0.2039	No	
Severity wheezing	-0.4518	0.6514	No	
Severity dry or itching skin	-0.1508	0.8801	No	
Severity skin blotches	-0.6797	0.4967	No	
TOTAL SIGNIFICANT DIFFERENCES			1	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**1/0**

SYMPTOMS Gender = Female BUILDING 1	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-1.5719	0.1160	No	
Frequency Irritated or Watering Eyes	-2.2496	0.0245	Yes	-
Frequency Strained Eyes	-0.7785	0.4363	No	
Frequency Stuffy or congested nose	-3.0043	0.0027	Yes	-
Frequency running nose	-3.3900	0.0007	Yes	-
Frequency irritated or itchy nose	-2.5189	0.0118	Yes	-
Frequency sneezing episodes	-1.5814	0.1138	No	
Frequency dry throat	-3.3712	0.0007	Yes	-
Frequency sore or irritated throat	-3.6820	0.0002	Yes	-
Frequency cough or hoarseness	-0.0152	0.9879	No	
Frequency headache	-1.6372	0.1016	No	
Frequency lethargy or drowsiness	-0.8294	0.4069	No	
Frequency concentration/memory difficulty	-0.4572	0.6475	No	
Frequency dizziness or faintness	-0.5347	0.5929	No	
Frequency flu like symptoms	-1.1302	0.2584	No	
Frequency aching limbs or backache	-1.7149	0.0864	No	
Frequency difficulty in breathing	-0.9348	0.3499	No	
Frequency chest tightness	-1.3736	0.1696	No	
Frequency wheezing	-0.4135	0.6793	No	
Frequency dry or itching skin	-1.4395	0.1500	No	
Frequency skin blotches	-0.4173	0.6765	No	
TOTAL SIGNIFICANT DIFFERENCES			6	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**6/0**

SYMPTOMS Gender = Female BUILDING 1	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-2.2167	0.0266	Yes	-
Severity Irritated or Watering Eyes	-2.2907	0.0220	Yes	-
Severity Strained Eyes	-0.0261	0.9792	No	
Severity Stuffy or congested nose	-2.5219	0.0117	Yes	-
Severity running nose	-2.6481	0.0081	Yes	-
Severity irritated or itchy nose	-2.4047	0.0162	Yes	-
Severity sneezing episodes	-1.1802	0.2379	No	
Severity dry throat	-3.5142	0.0004	Yes	-
Severity sore or irritated throat	-2.8899	0.0039	Yes	-
Severity cough or hoarseness	-1.4759	0.1400	No	
Severity headache	-0.7821	0.4342	No	
Severity lethargy or drowsiness	-2.1110	0.0348	Yes	-
Severity concentration/memory difficulty	-1.4444	0.1486	No	
Severity dizziness or faintness	-0.0379	0.9698	No	
Severity flu like symptoms	-0.1228	0.9022	No	
Severity aching limbs or backache	-2.9816	0.0029	Yes	-
Severity difficulty in breathing	-1.1918	0.2333	No	
Severity chest tightness	-1.1555	0.2479	No	
Severity wheezing	-0.7835	0.4334	No	
Severity dry or itching skin	-2.1206	0.0340	Yes	-
Severity skin blotches	-1.6746	0.0940	No	
TOTAL SIGNIFICANT DIFFERENCES			10	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**10/0**

SYMPTOMS Gender = Male BUILDING 1	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-0.5192	0.6036	No	
Frequency Irritated or Watering Eyes	-2.6509	0.0080	Yes	-
Frequency Strained Eyes	-0.1031	0.9179	No	
Frequency Stuffy or congested nose	-0.2986	0.7652	No	
Frequency running nose	-0.4498	0.6529	No	
Frequency irritated or itchy nose	-1.0696	0.2848	No	
Frequency sneezing episodes	-1.3012	0.1932	No	
Frequency dry throat	-1.2168	0.2237	No	
Frequency sore or irritated throat	-0.1898	0.8495	No	
Frequency cough or hoarseness	-0.3439	0.7309	No	
Frequency headache	-1.4153	0.1570	No	
Frequency lethargy or drowsiness	-0.5656	0.5717	No	
Frequency concentration/memory difficulties	-0.7870	0.4313	No	
Frequency dizziness or faintness	-0.9319	0.3514	No	
Frequency flu like symptoms	-0.1564	0.8757	No	
Frequency aching limbs or backache	-0.1258	0.8999	No	
Frequency difficulty in breathing	-1.9508	0.0511	No	
Frequency chest tightness	-1.0352	0.3006	No	
Frequency wheezing	-1.8227	0.0684	No	
Frequency dry or itching skin	-1.9073	0.0565	No	
Frequency skin blotches	-0.1567	0.8755	No	
TOTAL SIGNIFICANT DIFFERENCES			1	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**1/0**

SYMPTOMS Gender Male BUILDING 1	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-0.8405	0.4006	No	
Severity Irritated or Watering Eyes	-2.4081	0.0160	Yes	-
Severity Strained Eyes	-0.9095	0.3631	No	
Severity Stuffy or congested nose	-1.0446	0.2962	No	
Severity running nose	-0.6614	0.5083	No	
Severity irritated or itchy nose	-0.5018	0.6158	No	
Severity sneezing episodes	-1.2426	0.2126	No	
Severity dry throat	-1.3199	0.1869	Yes	
Severity sore or irritated throat	-2.5390	0.0111	Yes	-
Severity cough or hoarseness	-1.4719	0.1410	No	
Severity headache	-2.1204	0.0340	Yes	-
Severity lethargy or drowsiness	-0.4639	0.6427	No	
Severity concentration/memory difficulty	-2.2981	0.0216	Yes	-
Severity dizziness or faintness	-3.2071	0.0013	Yes	-
Severity flu like symptoms	-0.9573	0.3384	No	
Severity aching limbs or backache	-0.3748	0.7078	No	
Severity difficulty in breathing	-2.6775	0.0074	Yes	-
Severity chest tightness	-0.9655	0.3343	No	
Severity wheezing	-1.8486	0.0645	No	
Severity dry or itching skin	-0.4396	0.6602	No	
Severity skin blotches	-0.1339	0.8935	No	
TOTAL SIGNIFICANT DIFFERENCES			7	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**7/0**

SYMPTOMS Smoker BUILDING 1	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-1.2296	0.2819	No	
Frequency Irritated or Watering Eyes	-2.3569	0.0184	Yes	-
Frequency Strained Eyes	-1.2285	0.2193	No	
Frequency Stuffy or congested nose	-1.3710	0.1704	No	
Frequency running nose	-2.7680	0.0056	Yes	-
Frequency irritated or itchy nose	-0.8640	0.3876	No	
Frequency sneezing episodes	-0.7034	0.4818	No	
Frequency dry throat	-2.4349	0.0149	Yes	-
Frequency sore or irritated throat	-1.9923	0.0463	Yes	-
Frequency cough or hoarseness	-0.2448	0.8066	No	
Frequency headache	-2.3997	0.0164	Yes	-
Frequency lethargy or drowsiness	-1.0769	0.2815	No	
Frequency concentration/memory difficulties	-0.1905	0.8489	No	
Frequency dizziness or faintness	-0.7218	0.4704	No	
Frequency flu like symptoms	-0.9652	0.3345	No	
Frequency aching limbs or backache	-1.2631	0.2066	No	
Frequency difficulty in breathing	-0.1691	0.8657	No	
Frequency chest tightness	-0.6666	0.5050	No	
Frequency wheezing	-0.7996	0.4239	No	
Frequency dry or itching skin	-0.5638	0.5729	No	
Frequency skin blotches	-0.3118	0.7552	No	
TOTAL SIGNIFICANT DIFFERENCES			5	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**5/0**

SYMPTOMS Smoker BUILDING 1	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-1.4020	0.1609	No	
Severity Irritated or Watering Eyes	-2.5701	0.0102	Yes	-
Severity Strained Eyes	-2.2469	0.0246	Yes	-
Severity Stuffy or congested nose	-1.4972	0.1343	No	
Severity running nose	-1.4275	0.1534	No	
Severity irritated or itchy nose	-2.0812	0.0436	Yes	-
Severity sneezing episodes	-1.3442	0.1789	No	
Severity dry throat	-3.1914	0.0014	Yes	-
Severity sore or irritated throat	-2.8590	0.0042	Yes	-
Severity cough or hoarseness	-1.2720	0.2034	No	
Severity headache	-2.4488	0.0143	Yes	-
Severity lethargy or drowsiness	-1.6401	0.1010	No	
Severity concentration/memory difficulty	-1.5755	0.1152	No	
Severity dizziness or faintness	-1.1364	0.2558	No	
Severity flu like symptoms	-1.6108	0.1072	No	
Severity aching limbs or backache	-3.1632	0.0016	Yes	-
Severity difficulty in breathing	-1.7470	0.0806	No	
Severity chest tightness	-1.7230	0.0849	No	
Severity wheezing	-0.7006	0.4835	No	
Severity dry or itching skin	-1.4322	0.1521	No	
Severity skin blotches	-0.6806	0.4961	No	
TOTAL SIGNIFICANT DIFFERENCES			7	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**7/0**

SYMPTOMS Ex-Smoker BUILDING 1	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-1.6271	0.1037	No	
Frequency Irritated or Watering Eyes	-1.7850	0.0743	No	
Frequency Strained Eyes	-1.1941	0.2324	No	
Frequency Stuffy or congested nose	-2.0028	0.0452	Yes	-
Frequency running nose	-2.0227	0.0431	Yes	-
Frequency irritated or itchy nose	-1.7947	0.0727	No	
Frequency sneezing episodes	-0.7552	0.4502	No	
Frequency dry throat	-1.9421	0.0521	No	
Frequency sore or irritated throat	-2.3564	0.0185	Yes	-
Frequency cough or hoarseness	-0.8536	0.3933	No	
Frequency headache	-0.0249	0.9801	Yes	-
Frequency lethargy or drowsiness	-1.3652	0.1722	No	
Frequency concentration/memory difficulty	-0.6107	0.5414	No	
Frequency dizziness or faintness	-0.3395	0.7343	No	
Frequency flu like symptoms	-0.8098	0.4180	No	
Frequency aching limbs or backache	-0.9444	0.3450	No	
Frequency difficulty in breathing	-0.3395	0.7343	No	
Frequency chest tightness	-0.4463	0.6554	No	
Frequency wheezing	-0.3394	0.7343	No	
Frequency dry or itching skin	-2.3746	0.0176	Yes	-
Frequency skin blotches	-1.3563	0.1750	No	
TOTAL SIGNIFICANT DIFFERENCES			5	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**5/0**

SYMPTOMS Ex-Smoker BUILDING 1	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-1.7442	0.0811	No	
Severity Irritated or Watering Eyes	-1.6553	0.0979	No	
Severity Strained Eyes	-0.9786	0.3278	No	
Severity Stuffy or congested nose	-2.3841	0.0171	Yes	-
Severity running nose	-0.7330	0.4636	No	
Severity irritated or itchy nose	-0.8313	0.4058	No	
Severity sneezing episodes	-0.7940	0.4272	No	
Severity dry throat	-1.9932	0.0462	Yes	-
Severity sore or irritated throat	-1.5812	0.1138	No	
Severity cough or hoarseness	-0.7397	0.4595	No	
Severity headache	-1.3695	0.1708	No	
Severity lethargy or drowsiness	-1.5821	0.1136	No	
Severity concentration/memory difficulty	-0.7803	0.4352	No	
Severity dizziness or faintness	-0.3394	0.7343	No	
Severity flu like symptoms	-0.4366	0.6624	No	
Severity aching limbs or backache	-0.9034	0.3663	No	
Severity difficulty in breathing	-0.4934	0.6217	No	
Severity chest tightness	-0.5808	0.5614	No	
Severity wheezing	-0.4466	0.6552	No	
Severity dry or itching skin	-2.6376	0.0083	Yes	-
Severity skin blotches	-1.3380	0.1809	No	
TOTAL SIGNIFICANT DIFFERENCES			3	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**3/0**

SYMPTOMS Non-Smoker BUILDING 1	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-1.1800	0.2380	No	
Frequency Irritated or Watering Eyes	-2.3409	0.0192	Yes	-
Frequency Strained Eyes	-0.1092	0.1930	No	
Frequency Stuffy or congested nose	-2.4537	0.0141	Yes	-
Frequency running nose	-2.2975	0.0216	Yes	-
Frequency irritated or itchy nose	-2.4612	0.0138	Yes	-
Frequency sneezing episodes	-2.1121	0.0347	Yes	-
Frequency dry throat	-2.8356	0.0046	Yes	-
Frequency sore or irritated throat	-2.5910	0.0096	Yes	-
Frequency cough or hoarseness	-0.3454	0.7298	No	
Frequency headache	-0.0779	0.1682	Yes	-
Frequency lethargy or drowsiness	-0.5975	0.5502	No	
Frequency concentration/memory difficulty	-0.6518	0.5145	No	
Frequency dizziness or faintness	-0.4113	0.6808	No	
Frequency flu like symptoms	-0.5430	0.5872	No	
Frequency aching limbs or backache	-1.1387	0.2548	No	
Frequency difficulty in breathing	-2.0360	0.0417	Yes	-
Frequency chest tightness	-1.3205	0.1867	No	
Frequency wheezing	-1.8702	0.0615	No	
Frequency dry or itching skin	-1.8823	0.0598	No	
Frequency skin blotches	-0.6042	0.5457	No	
TOTAL SIGNIFICANT DIFFERENCES			9	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**9/0**

SYMPTOMS Non-Smoker BUILDING 1	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-1.6516	0.0986	No	
Severity Irritated or Watering Eyes	-2.2741	0.0230	Yes	-
Severity Strained Eyes	-0.4352	0.6634	No	
Severity Stuffy or congested nose	-1.9688	0.0490	Yes	-
Severity running nose	-2.7604	0.0058	Yes	-
Severity irritated or itchy nose	-1.9995	0.0456	Yes	-
Severity sneezing episodes	-2.5578	0.0105	Yes	-
Severity dry throat	-2.9236	0.0035	Yes	-
Severity sore or irritated throat	-3.0724	0.0021	Yes	-
Severity cough or hoarseness	-1.9970	0.0458	Yes	-
Severity headache	-1.6620	0.0965	No	
Severity lethargy or drowsiness	-1.5106	0.1309	No	
Severity concentration/memory difficulty	-1.7745	0.0760	No	
Severity dizziness or faintness	-1.0292	0.3034	No	
Severity flu like symptoms	-0.2118	0.8323	No	
Severity aching limbs or backache	-1.9581	0.0502	No	
Severity difficulty in breathing	-2.0018	0.0453	Yes	-
Severity chest tightness	-0.9398	0.3473	No	
Severity wheezing	-1.5355	0.1247	No	
Severity dry or itching skin	-0.7668	0.4432	No	
Severity skin blotches	-1.6478	0.0994	No	
TOTAL SIGNIFICANT DIFFERENCES			9	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**9/0**

SYMPTOMS People smoking in the immediate working environment BUILDING 1	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-0.9744	0.3298	No	
Frequency Irritated or Watering Eyes	-1.4850	0.1375	No	
Frequency Strained Eyes	-0.9744	0.3298	No	
Frequency Stuffy or congested nose	-1.4707	0.1414	No	
Frequency running nose	-0.9590	0.3376	No	
Frequency irritated or itchy nose	-0.7513	0.4525	No	
Frequency sneezing episodes	-0.5184	0.6042	No	
Frequency dry throat	-2.4535	0.0141	Yes	-
Frequency sore or irritated throat	-1.4820	0.1383	No	
Frequency cough or hoarseness	-0.0238	0.9810	No	
Frequency headache	-1.9817	0.0475	Yes	-
Frequency lethargy or drowsiness	-1.0770	0.2815	No	
Frequency concentration/memory difficulty	-0.6184	0.5363	No	
Frequency dizziness or faintness	-0.6208	0.5347	No	
Frequency flu like symptoms	-0.4603	0.6453	No	
Frequency aching limbs or backache	-1.8116	0.0701	No	
Frequency difficulty in breathing	-0.6206	0.5349	No	
Frequency chest tightness	-1.1368	0.2556	No	
Frequency wheezing	-0.6206	0.5349	No	
Frequency dry or itching skin	-3.0240	0.0025	Yes	-
Frequency skin blotches	-2.6148	0.0089	Yes	-
TOTAL SIGNIFICANT DIFFERENCES			4	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**4/0**

SYMPTOMS People smoking in the immedediate working environment BUILDING 1	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-0.5462	0.5849	No	
Severity Irritated or Watering Eyes	-1.8373	0.0662	No	
Severity Strained Eyes	-0.5979	0.5499	No	
Severity Stuffy or congested nose	-2.1216	0.0339	Yes	-
Severity running nose	-1.5188	0.1288	No	
Severity irritated or itchy nose	-1.8641	0.0623	No	
Severity sneezing episodes	-0.2060	0.8368	No	
Severity dry throat	-3.1179	0.0018	Yes	-
Severity sore or irritated throat	-2.3860	0.0170	Yes	-
Severity cough or hoarseness	-1.7989	0.0720	No	
Severity headache	-2.1421	0.0322	Yes	-
Severity lethargy or drowsiness	-1.9056	0.0567	No	
Severity concentration/memory difficulty	-0.7640	0.4449	No	
Severity dizziness or faintness	-2.0740	0.0381	Yes	-
Severity flu like symptoms	-0.0651	0.9481	No	
Severity aching limbs or backache	-2.3122	0.0208	Yes	-
Severity difficulty in breathing	-1.2305	0.2185	No	
Severity chest tightness	-0.4313	0.6663	No	
Severity wheezing	-0.0316	0.9748	No	
Severity dry or itching skin	-2.9120	0.0036	Yes	-
Severity skin blotches	-3.3921	0.0007	Yes	-
TOTAL SIGNIFICANT DIFFERENCES			8	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**8/0**

SYMPTOMS Non-Smoking working environment BUILDING 1	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-1.6943	0.0902	No	
Frequency Irritated or Watering Eyes	-2.8641	0.0042	Yes	-
Frequency Strained Eyes	-0.3466	0.7289	No	
Frequency Stuffy or congested nose	--2.6931	0.0071	Yes	-
Frequency running nose	-3.5187	0.0004	Yes	-
Frequency irritated or itchy nose	-3.0551	0.0022	Yes	-
Frequency sneezing episodes	-2.2203	0.0264	Yes	-
Frequency dry throat	-3.2083	0.0013	Yes	-
Frequency sore or irritated throat	-3.3238	0.0009	Yes	-
Frequency cough or hoarseness	-0.0995	0.9207	No	
Frequency headache	-1.4397	0.1499	No	
Frequency lethargy or drowsiness	-0.9843	0.3250	No	
Frequency concentration/memory difficulty	-1.1316	0.2578	No	
Frequency dizziness or faintness	-0.2875	0.7737	No	
Frequency flu like symptoms	-1.3590	0.1741	No	
Frequency aching limbs or backache	-1.0668	0.2861	No	
Frequency difficulty in breathing	-2.1979	0.0280	Yes	-
Frequency chest tightness	-2.2366	0.0253	Yes	-
Frequency wheezing	-1.6270	0.1037	No	
Frequency dry or itching skin	-1.1648	0.2441	No	
Frequency skin blotches	-0.6604	0.5090	No	
TOTAL SIGNIFICANT DIFFERENCES			9	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**9/0**

SYMPTOMS Non-Smoking working environment BUILDING 1	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-2.3074	0.0210	Yes	-
Severity Irritated or Watering Eyes	-2.6526	0.0080	Yes	-
Severity Strained Eyes	-0.1237	0.9016	No	
Severity Stuffy or congested nose	-2.1479	0.0317	Yes	-
Severity running nose	-2.4760	0.0133	Yes	-
Severity irritated or itchy nose	-2.0610	0.0393	Yes	-
Severity sneezing episodes	-2.1054	0.0353	Yes	-
Severity dry throat	-3.0188	0.0025	Yes	-
Severity sore or irritated throat	-3.3698	0.0008	Yes	-
Severity cough or hoarseness	-1.2272	0.2198	No	
Severity headache	-1.0739	0.2829	No	
Severity lethargy or drowsiness	-1.5535	1.1203	No	
Severity concentration/memory difficulty	-2.1027	0.0355	Yes	-
Severity dizziness or faintness	-0.4031	0.6869	No	
Severity flu like symptoms	-0.4595	0.6459	No	
Severity aching limbs or backache	-2.2552	0.0241	Yes	-
Severity difficulty in breathing	-1.8586	0.0631	No	
Severity chest tightness	-1.1604	0.2459	No	
Severity wheezing	-1.6810	0.0928	No	
Severity dry or itching skin	-0.5802	0.5618	No	
Severity skin blotches	-0.0560	0.9554	No	-
TOTAL SIGNIFICANT DIFFERENCES			10	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**10/0**

APPENDIX 13

SYMPTOMS Direct Undifferentiated Symptom Comparison BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-0.9128	0.3614	No	
Frequency Irritated or Watering Eyes	-3.3442	0.0008	Yes	-
Frequency Strained Eyes	-1.6349	0.1021	No	
Frequency Stuffy or congested nose	-2.4631	0.0138	Yes	-
Frequency running nose	-4.2744	0.0000	Yes	-
Frequency irritated or itchy nose	-1.4231	0.1547	No	
Frequency sneezing episodes	-1.7776	0.0755	No	
Frequency dry throat	-1.5116	0.1306	No	
Frequency sore or irritated throat	-3.2147	0.0013	Yes	-
Frequency cough or hoarseness	-1.1969	0.2313	No	
Frequency headache	-2.7452	0.0060	Yes	-
Frequency lethargy or drowsiness	-1.7490	0.0803	No	
Frequency concentration/memory difficulty	-1.5871	0.1125	No	
Frequency dizziness or faintness	-0.6504	0.5154	No	
Frequency flu like symptoms	-0.1167	0.9071	No	
Frequency aching limbs or backache	-1.0366	0.2999	No	
Frequency difficulty in breathing	-0.3921	0.6850	No	
Frequency chest tightness	-0.7060	0.4802	No	
Frequency wheezing	-0.2117	0.8324	No	
Frequency dry or itching skin	-1.3070	0.1912	No	
Frequency skin blotches	-1.3130	0.1892	No	
TOTAL SIGNIFICANT DIFFERENCES			5	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**5/0**

SYMPTOMS Direct Undifferentiated Symptom Comparison BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-0.3004	0.7638	No	
Severity Irritated or Watering Eyes	-2.5996	0.0093	Yes	-
Severity Strained Eyes	-1.8722	0.0612	No	
Severity Stuffy or congested nose	-1.6010	0.1094	No	
Severity running nose	-2.6013	0.0093	Yes	-
Severity irritated or itchy nose	-0.2473	0.8047	No	
Severity sneezing episodes	-0.8275	0.4079	No	
Severity dry throat	-0.8173	0.4137	No	
Severity sore or irritated throat	-1.1604	0.2459	No	
Severity cough or hoarseness	-0.6236	0.5329	No	
Severity headache	-1.9269	0.0540	No	
Severity lethargy or drowsiness	-2.4714	0.0135	Yes	-
Severity concentration/memory difficulty	-1.9323	0.0533	No	
Severity dizziness or faintness	-0.0306	0.9756	No	
Severity flu like symptoms	-1.0641	0.2873	No	
Severity aching limbs or backache	-1.3465	0.1782	No	
Severity difficulty in breathing	-1.1557	0.2478	No	
Severity chest tightness	-0.4939	0.6214	No	
Severity wheezing	-0.1713	0.8640	No	
Severity dry or itching skin	-1.2828	0.1996	No	
Severity skin blotches	-0.4541	0.6497	No	
TOTAL SIGNIFICANT DIFFERENCES			3	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**3/0**

SYMPTOMS Job Description = Management BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-0.2867	0.7743	No	
Frequency Irritated or Watering Eyes	-0.5747	0.5655	No	
Frequency Strained Eyes	-0.1457	0.8842	No	
Frequency Stuffy or congested nose	-0.4072	0.6839	No	
Frequency running nose	-0.3753	0.7074	No	
Frequency irritated or itchy nose	-1.7944	0.0727	No	
Frequency sneezing episodes	-1.3380	0.1809	No	
Frequency dry throat	-0.5760	0.5646	No	
Frequency sore or irritated throat	-0.7781	0.4365	No	
Frequency cough or hoarseness	-0.2312	0.8172	No	
Frequency headache	-0.9174	0.3589	No	
Frequency lethargy or drowsiness	-0.2018	0.8401	No	
Frequency concentration/memory difficulty	-0.6191	0.5359	No	
Frequency dizziness or faintness	-0.7573	0.4489	No	
Frequency flu like symptoms	-1.3801	0.1675	No	
Frequency aching limbs or backache	-1.6524	0.0985	No	
Frequency difficulty in breathing	-0.9943	0.3201	No	
Frequency chest tightness	-1.5494	0.1213	No	
Frequency wheezing	-1.0978	0.2723	No	
Frequency dry or itching skin	-0.4739	0.6356	No	
Frequency skin blotches	-1.0971	0.2726	No	
TOTAL SIGNIFICANT DIFFERENCES			0	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**0/0**

SYMPTOMS Job Description = Management BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-0.5283	0.5973	No	
Severity Irritated or Watering Eyes	-0.6717	0.5018	No	
Severity Strained Eyes	-0.5427	0.5873	No	
Severity Stuffy or congested nose	-0.4408	0.6594	No	
Severity running nose	-0.4073	0.6838	No	
Severity irritated or itchy nose	-1.9596	0.0500	Yes	+
Severity sneezing episodes	-0.9359	0.3493	No	
Severity dry throat	-0.3262	0.7443	No	
Severity sore or irritated throat	-0.9921	0.3213	No	
Severity cough or hoarseness	-0.4654	0.6417	No	
Severity headache	-0.7160	0.4740	No	
Severity lethargy or drowsiness	-0.7758	0.4379	No	
Severity concentration/memory difficulty	-0.6634	0.5071	No	
Severity dizziness or faintness	-0.8814	0.3781	No	
Severity flu like symptoms	-1.9521	0.0509	No	
Severity aching limbs or backache	-1.9974	0.0458	Yes	+
Severity difficulty in breathing	-1.0974	0.2725	No	
Severity chest tightness	-1.6317	0.1028	No	
Severity wheezing	-1.1949	0.2321	No	
Severity dry or itching skin	-0.4391	0.6606	No	
Severity skin blotches	-0.8813	0.3781	No	
TOTAL SIGNIFICANT DIFFERENCES			2	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**0/2**

SYMPTOMS Job Description = Professional BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-0.3757	0.7072	No	
Frequency Irritated or Watering Eyes	-0.6632	0.5072	No	
Frequency Strained Eyes	-0.5292	0.5967	No	
Frequency Stuffy or congested nose	-0.2737	0.7843	No	
Frequency running nose	-1.7953	0.0726	No	
Frequency irritated or itchy nose	-0.3417	0.7326	No	
Frequency sneezing episodes	-0.6265	0.5310	No	
Frequency dry throat	-1.2244	0.2208	No	
Frequency sore or irritated throat	-1.8594	0.0630	No	
Frequency cough or hoarseness	-0.2210	0.8251	No	
Frequency headache	-1.5071	0.1318	No	
Frequency lethargy or drowsiness	-1.0550	0.2914	No	
Frequency concentration/memory difficulty	-0.7363	0.4616	No	
Frequency dizziness or faintness	-0.2621	0.7933	No	
Frequency flu like symptoms	-0.3986	0.6902	No	
Frequency aching limbs or backache	-1.2698	0.2042	No	
Frequency difficulty in breathing	-0.3649	0.7152	No	
Frequency chest tightness	-0.1651	0.8689	No	
Frequency wheezing	-0.5759	0.5647	No	
Frequency dry or itching skin	-0.3024	0.7623	No	
Frequency skin blotches	-1.1070	0.2679	No	
TOTAL SIGNIFICANT DIFFERENCES			0	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**0/0**

SYMPTOMS Job Description = Professional BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-0.2681	0.7886	No	
Severity Irritated or Watering Eyes	-0.5841	0.5592	No	
Severity Strained Eyes	-0.9035	0.3662	No	
Severity Stuffy or congested nose	-0.4895	0.6245	No	
Severity running nose	-0.3385	0.7350	No	
Severity irritated or itchy nose	-0.5655	0.5717	No	
Severity sneezing episodes	-0.2660	0.7902	No	
Severity dry throat	-0.8229	0.4106	No	
Severity sore or irritated throat	-0.2104	0.8334	No	
Severity cough or hoarseness	-0.8405	0.4006	No	
Severity headache	-0.7985	0.4246	No	
Severity lethargy or drowsiness	-1.5353	0.1247	No	
Severity concentration/memory difficulty	-1.3735	0.1696	No	
Severity dizziness or faintness	-0.0917	0.9269	No	
Severity flu like symptoms	-1.5249	0.1273	No	
Severity aching limbs or backache	-1.7695	0.0768	No	
Severity difficulty in breathing	-0.1834	0.8545	No	
Severity chest tightness	-0.7114	0.4769	No	
Severity wheezing	-0.3217	0.7477	No	
Severity dry or itching skin	-0.9389	0.3478	No	
Severity skin blotches	-1.0534	0.2922	No	
TOTAL SIGNIFICANT DIFFERENCES			0	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**0/0**

SYMPTOMS Job Description = Clerical/Secretarial BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-0.6354	0.5252	No	
Frequency Irritated or Watering Eyes	-3.1975	0.0014	Yes	-
Frequency Strained Eyes	-2.4475	0.0144	Yes	-
Frequency Stuffy or congested nose	-1.3830	0.1667	No	
Frequency running nose	-3.6309	0.0003	Yes	-
Frequency irritated or itchy nose	-0.6466	0.5179	No	
Frequency sneezing episodes	-1.3399	0.1803	No	
Frequency dry throat	-1.1421	0.2534	No	
Frequency sore or irritated throat	-2.3195	0.0204	Yes	-
Frequency cough or hoarseness	-1.3200	0.1868	No	
Frequency headache	-1.6058	0.1083	No	
Frequency lethargy or drowsiness	-0.6036	0.5461	No	
Frequency concentration/memory difficulties	-0.4063	0.6845	No	
Frequency dizziness or faintness	-0.8197	0.4124	No	
Frequency flu like symptoms	-0.3090	0.7573	No	
Frequency aching limbs or backache	-0.2403	0.8101	No	
Frequency difficulty in breathing	-0.5721	0.5673	No	
Frequency chest tightness	-1.6458	0.0998	No	
Frequency wheezing	-0.9300	0.3524	No	
Frequency dry or itching skin	-1.7079	0.0877	No	
Frequency skin blotches	-0.6579	0.5106	No	
TOTAL SIGNIFICANT DIFFERENCES			4	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**4/0**

SYMPTOMS Job Description = Clerical/Secretarial BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-0.2455	0.8061	No	
Severity Irritated or Watering Eyes	-2.4784	0.0132	Yes	-
Severity Strained Eyes	-2.3645	0.0181	No	-
Severity Stuffy or congested nose	-0.7894	0.4246	No	
Severity running nose	-2.1909	0.0285	Yes	-
Severity irritated or itchy nose	-0.0111	0.9912	No	
Severity sneezing episodes	-0.1080	0.9140	No	
Severity dry throat	-0.8365	0.4029	No	
Severity sore or irritated throat	-1.3339	0.1822	No	-
Severity cough or hoarseness	-1.1798	0.2381	No	
Severity headache	-0.8052	0.4207	No	
Severity lethargy or drowsiness	-0.8478	0.3965	No	
Severity concentration/memory difficulty	-0.3991	0.6898	No	
Severity dizziness or faintness	-0.4796	0.6315	No	
Severity flu like symptoms	0.0309	0.9753	No	
Severity aching limbs or backache	-0.2646	0.7914	No	
Severity difficulty in breathing	-1.3638	0.1726	No	
Severity chest tightness	-1.5249	0.1273	No	
Severity wheezing	-1.0214	0.3071	No	
Severity dry or itching skin	-1.5724	0.1159	No	
Severity skin blotches	-0.0651	0.9481	No	
TOTAL SIGNIFICANT DIFFERENCES			2	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**2/0**

SYMPTOMS Job Description = V.D.U Operator BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-0.6534	0.5135	No	
Frequency Irritated or Watering Eyes	-1.5197	0.1286	No	
Frequency Strained Eyes	-1.1095	0.9128	No	
Frequency Stuffy or congested nose	-2.6500	0.0080	Yes	-
Frequency running nose	-1.5428	0.1229	No	
Frequency irritated or itchy nose	-2.6821	0.0073	Yes	-
Frequency sneezing episodes	-1.9476	0.0515	No	
Frequency dry throat	-0.1988	0.8424	No	
Frequency sore or irritated throat	-2.4721	0.0134	Yes	-
Frequency cough or hoarseness	-0.7126	0.4761	No	
Frequency headache	-1.0279	0.3040	No	
Frequency lethargy or drowsiness	-1.4793	0.1391	No	
Frequency concentration/memory difficulty	-0.5642	0.5726	No	
Frequency dizziness or faintness	-0.1362	0.8917	No	
Frequency flu like symptoms	-0.9067	0.3646	No	
Frequency aching limbs or backache	-0.2965	0.7669	No	
Frequency difficulty in breathing	-0.5259	0.5990	No	
Frequency chest tightness	-1.9661	0.0493	Yes	-
Frequency wheezing	-1.3724	0.1699	No	
Frequency dry or itching skin	-0.7121	0.4764	No	
Frequency skin blotches	-0.5257	0.5991	No	
TOTAL SIGNIFICANT DIFFERENCES			4	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**4/0**

SYMPTOMS Job Description = Clerical/Secretarial BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-0.9542	0.3400	No	
Severity Irritated or Watering Eyes	-2.2345	0.0254	Yes	-
Severity Strained Eyes	-0.1985	0.8426	No	
Severity Stuffy or congested nose	-3.0710	0.0021	Yes	-
Severity running nose	-1.7565	0.0790	No	
Severity irritated or itchy nose	-2.3098	0.0209	Yes	-
Severity sneezing episodes	-1.6914	0.0908	No	
Severity dry throat	-0.5477	0.5839	No	
Severity sore or irritated throat	-1.6205	0.1051	No	
Severity cough or hoarseness	-1.0376	0.2994	No	
Severity headache	-1.6003	0.1095	No	
Severity lethargy or drowsiness	-1.8195	0.0688	No	
Severity concentration/memory difficulty	-0.7687	0.4421	No	
Severity dizziness or faintness	-1.1646	0.2442	No	
Severity flu like symptoms	-0.8475	0.3967	No	
Severity aching limbs or backache	0.0000	1.0000	No	
Severity difficulty in breathing	-1.1803	0.2379	No	
Severity chest tightness	-2.1490	0.0316	Yes	-
Severity wheezing	-0.4840	0.6284	No	
Severity dry or itching skin	-1.1912	0.2336	No	
Severity skin blotches	-0.4855	0.6273	No	
TOTAL SIGNIFICANT DIFFERENCES			4	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**4/0**

SYMPTOMS Age = Under 30 BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-0.2500	0.8026	No	
Frequency Irritated or Watering Eyes	-0.9980	0.3183	No	
Frequency Strained Eyes	-0.6885	0.4912	No	
Frequency Stuffy or congested nose	-1.5387	0.1239	No	
Frequency running nose	-3.3253	0.0009	Yes	-
Frequency irritated or itchy nose	-1.9141	0.0556	No	
Frequency sneezing episodes	-1.3932	0.1635	No	
Frequency dry throat	-1.2033	0.2289	No	
Frequency sore or irritated throat	-1.5765	0.1149	No	
Frequency cough or hoarseness	-0.6645	0.5064	No	
Frequency headache	-1.2243	0.2208	No	
Frequency lethargy or drowsiness	-0.4541	0.6497	No	
Frequency concentration/memory difficulty	-0.3320	0.7399	No	
Frequency dizziness or faintness	-0.0489	0.9610	No	
Frequency flu like symptoms	-0.0294	0.9765	No	
Frequency aching limbs or backache	-0.9752	0.3294	No	
Frequency difficulty in breathing	-0.8993	0.3685	No	
Frequency chest tightness	-0.6436	0.5198	No	
Frequency wheezing	-1.3572	0.1747	No	
Frequency dry or itching skin	-0.0896	0.9286	No	
Frequency skin blotches	-1.8545	0.0637	No	
TOTAL SIGNIFICANT DIFFERENCES			1	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**1/0**

SYMPTOMS Age = Under 30 BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-0.3272	0.7435	No	
Severity Irritated or Watering Eyes	-0.9585	0.3378	No	
Severity Strained Eyes	-0.4798	0.6313	No	
Severity Stuffy or congested nose	-1.1805	0.2378	No	
Severity running nose	-2.3236	0.0201	Yes	-
Severity irritated or itchy nose	-0.8545	0.3928	No	
Severity sneezing episodes	-0.3025	0.7623	No	
Severity dry throat	-0.0499	0.9602	No	
Severity sore or irritated throat	-0.0356	0.9716	No	
Severity cough or hoarseness	-0.0104	0.9917	No	
Severity headache	-0.8979	0.3692	No	
Severity lethargy or drowsiness	-0.7805	0.4351	No	
Severity concentration/memory difficulty	-0.4733	0.6360	No	
Severity dizziness or faintness	-0.6387	0.5230	No	
Severity flu like symptoms	-0.0425	0.9661	No	
Severity aching limbs or backache	-0.5313	0.5952	No	
Severity difficulty in breathing	-1.0080	0.3135	No	
Severity chest tightness	-0.9636	0.3352	No	
Severity wheezing	-1.0034	0.3157	No	
Severity dry or itching skin	-0.5577	0.5571	No	
Severity skin blotches	-0.5543	0.5863	No	
TOTAL SIGNIFICANT DIFFERENCES			1	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**1/0**

SYMPTOMS Age = 30-39 BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-0.6524	0.5142	No	
Frequency Irritated or Watering Eyes	-2.6053	0.0092	Yes	-
Frequency Strained Eyes	-0.3261	0.7443	No	
Frequency Stuffy or congested nose	-0.7458	0.4558	No	
Frequency running nose	-0.9868	0.3237	No	
Frequency irritated or itchy nose	-0.2342	0.8148	No	
Frequency sneezing episodes	-0.7394	0.4596	No	
Frequency dry throat	-0.6353	0.5252	No	
Frequency sore or irritated throat	-2.0194	0.0434	Yes	-
Frequency cough or hoarseness	-0.2287	0.8191	No	
Frequency headache	-1.1824	0.2370	No	
Frequency lethargy or drowsiness	-1.3780	0.1682	No	
Frequency concentration/memory difficulty	-1.3864	0.1656	No	
Frequency dizziness or faintness	-0.2058	0.8369	No	
Frequency flu like symptoms	-0.1230	0.9021	No	
Frequency aching limbs or backache	-0.0214	0.9829	No	
Frequency difficulty in breathing	-0.4697	0.6386	No	
Frequency chest tightness	-0.6986	0.4848	No	
Frequency wheezing	-0.2941	0.7687	No	
Frequency dry or itching skin	-1.3474	0.1778	No	
Frequency skin blotches	-0.7102	0.4774	No	
TOTAL SIGNIFICANT DIFFERENCES			2	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**2/0**

SYMPTOMS Age = 30-39 BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-0.0676	0.9461	No	
Severity Irritated or Watering Eyes	-1.1870	0.2352	No	
Severity Strained Eyes	-0.1936	0.8465	No	
Severity Stuffy or congested nose	-0.3456	0.7296	No	
Severity running nose	-0.3074	0.7586	No	
Severity irritated or itchy nose	-0.3580	0.7204	No	
Severity sneezing episodes	-0.9175	0.3589	No	
Severity dry throat	-0.4647	0.6421	No	
Severity sore or irritated throat	-0.3039	0.7612	No	
Severity cough or hoarseness	-0.3205	0.7486	No	
Severity headache	-0.8070	0.4196	No	
Severity lethargy or drowsiness	-1.6245	0.1043	No	
Severity concentration/memory difficulty	-1.2832	0.1994	No	
Severity dizziness or faintness	-0.8515	0.3945	No	
Severity flu like symptoms	-0.8452	0.3980	No	
Severity aching limbs or backache	-0.5455	0.5854	No	
Severity difficulty in breathing	-0.3617	0.7176	No	
Severity chest tightness	-0.5904	0.5549	No	
Severity wheezing	-0.6987	0.4847	No	
Severity dry or itching skin	-0.9808	0.3267	No	
Severity skin blotches	-0.5536	0.5799	No	
TOTAL SIGNIFICANT DIFFERENCES			0	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**0/0**

SYMPTOMS Age = 40-49 BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-0.7115	0.4768	No	
Frequency Irritated or Watering Eyes	-1.7482	0.0804	No	
Frequency Strained Eyes	-1.2373	0.2160	No	
Frequency Stuffy or congested nose	-1.9731	0.0485	Yes	-
Frequency running nose	-1.8914	0.0586	No	
Frequency irritated or itchy nose	-0.0375	0.9701	No	
Frequency sneezing episodes	-1.2096	0.2264	No	
Frequency dry throat	-0.4943	0.6211	No	
Frequency sore or irritated throat	-2.2476	0.0246	Yes	-
Frequency cough or hoarseness	-0.2385	0.8115	No	
Frequency headache	-0.7573	0.4488	No	
Frequency lethargy or drowsiness	-0.2408	0.8097	No	
Frequency concentration/memory difficulty	-0.2485	0.8037	No	
Frequency dizziness or faintness	-0.4673	0.6403	No	
Frequency flu like symptoms	-0.6405	0.5218	No	
Frequency aching limbs or backache	-1.6542	0.0981	No	
Frequency difficulty in breathing	-1.3673	0.1715	No	
Frequency chest tightness	-1.5801	0.1141	No	
Frequency wheezing	-1.7605	0.0783	No	
Frequency dry or itching skin	-0.0678	0.9460	No	
Frequency skin blotches	-0.0324	0.9741	No	
TOTAL SIGNIFICANT DIFFERENCES			2	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**2/0**

SYMPTOMS Age = 40-49 BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-0.1766	0.8598	No	
Severity Irritated or Watering Eyes	-1.3621	0.1732	No	
Severity Strained Eyes	-1.2697	0.2042	No	
Severity Stuffy or congested nose	-1.1370	0.2555	No	
Severity running nose	-0.8461	0.3975	No	
Severity irritated or itchy nose	-0.1631	0.8705	No	
Severity sneezing episodes	-0.9420	0.3462	No	
Severity dry throat	-0.8984	0.3689	No	
Severity sore or irritated throat	-1.6897	0.0911	No	
Severity cough or hoarseness	-0.1684	0.8663	No	
Severity headache	-0.4103	0.6816	No	
Severity lethargy or drowsiness	-0.2042	0.8382	No	
Severity concentration/memory difficulty	-0.9213	0.3569	No	
Severity dizziness or faintness	-0.2790	0.7802	No	
Severity flu like symptoms	-1.3540	0.1757	No	
Severity aching limbs or backache	-2.4882	0.0128	Yes	+
Severity difficulty in breathing	-0.8393	0.4013	No	
Severity chest tightness	-0.6681	0.5041	No	
Severity wheezing	-0.6901	0.4901	No	
Severity dry or itching skin	-0.1214	0.9034	No	
Severity skin blotches	-0.1836	0.8544	No	
TOTAL SIGNIFICANT DIFFERENCES			1	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**0/1**

SYMPTOMS Age = 50-59 BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-0.9828	0.3257	No	
Frequency Irritated or Watering Eyes	-0.9947	0.3199	No	
Frequency Strained Eyes	-0.4008	0.6886	No	
Frequency Stuffy or congested nose	-0.1799	0.8572	No	
Frequency running nose	-0.3597	0.7190	No	
Frequency irritated or itchy nose	-1.0442	0.2964	No	
Frequency sneezing episodes	-0.3651	0.7150	No	
Frequency dry throat	-0.0323	0.9742	No	
Frequency sore or irritated throat	-1.0372	0.2996	No	
Frequency cough or hoarseness	-1.0371	0.2997	No	
Frequency headache	-0.7247	0.4686	No	
Frequency lethargy or drowsiness	-0.7654	0.4441	No	
Frequency concentration/memory difficulty	-0.4898	0.6243	No	
Frequency dizziness or faintness	-1.8811	0.0600	No	
Frequency flu like symptoms	-0.1515	0.8796	No	
Frequency aching limbs or backache	-0.4942	0.6212	No	
Frequency difficulty in breathing	-0.9332	0.3507	No	
Frequency chest tightness	-0.1679	0.8667	No	
Frequency wheezing	-0.4564	0.6481	No	
Frequency dry or itching skin	-0.0552	0.9559	No	
Frequency skin blotches	-1.8201	0.0687	No	
TOTAL SIGNIFICANT DIFFERENCES			0	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**0/0**

SYMPTOMS Age = 50-59 BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-1.3289	0.1839	No	
Severity Irritated or Watering Eyes	-1.3320	0.1829	No	
Severity Strained Eyes	-0.3957	0.6923	No	
Severity Stuffy or congested nose	-0.4147	0.6783	No	
Severity running nose	-0.3235	0.7463	No	
Severity irritated or itchy nose	-2.0337	0.0420	Yes	+
Severity sneezing episodes	-0.2628	0.7927	No	
Severity dry throat	-0.7009	0.4833	No	
Severity sore or irritated throat	-0.9340	0.3503	No	
Severity cough or hoarseness	-0.6965	0.4861	No	
Severity headache	-0.1637	0.8700	No	
Severity lethargy or drowsiness	-1.8664	0.0620	No	
Severity concentration/memory difficulty	-0.8771	0.3837	No	
Severity dizziness or faintness	-0.7584	0.4482	No	
Severity flu like symptoms	-1.3661	0.1719	No	
Severity aching limbs or backache	-0.2613	0.7939	No	
Severity difficulty in breathing	-0.1049	0.9164	No	
Severity chest tightness	-1.0912	0.2752	No	
Severity wheezing	-0.9371	0.3487	No	
Severity dry or itching skin	-0.5969	0.5505	No	
Severity skin blotches	-1.5985	0.1099	No	
TOTAL SIGNIFICANT DIFFERENCES			1	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**0/1**

SYMPTOMS Gender = Female BUILDING 2	STATISTICS -		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-1.5053	0.1323	No	
Frequency Irritated or Watering Eyes	-3.7651	0.0002	Yes	-
Frequency Strained Eyes	-1.6151	0.1063	No	
Frequency Stuffy or congested nose	-2.5019	0.0124	Yes	-
Frequency running nose	-4.5020	0.0000	Yes	-
Frequency irritated or itchy nose	-2.1549	0.0312	Yes	-
Frequency sneezing episodes	-1.9069	0.0565	No	
Frequency dry throat	-0.7558	0.4498	No	
Frequency sore or irritated throat	-2.5987	0.0094	Yes	-
Frequency cough or hoarseness	-0.6614	0.5083	No	
Frequency headache	-2.6770	0.0074	Yes	-
Frequency lethargy or drowsiness	-0.8294	0.4069	No	
Frequency concentration/memory difficulty	-1.4511	0.1467	No	
Frequency dizziness or faintness	-0.8654	0.3868	No	
Frequency flu like symptoms	-0.1519	0.8793	No	
Frequency aching limbs or backache	-0.2745	0.7837	No	
Frequency difficulty in breathing	-0.4212	0.6736	No	
Frequency chest tightness	-1.0443	0.2964	No	
Frequency wheezing	-0.2733	0.7846	No	
Frequency dry or itching skin	-1.2526	0.2104	No	
Frequency skin blotches	-0.7240	0.4690	No	
TOTAL SIGNIFICANT DIFFERENCES			6	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**6/0**

SYMPTOMS Gender = Female BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-1.3246	0.1853	No	
Severity Irritated or Watering Eyes	-3.2058	0.0013	Yes	-
Severity Strained Eyes	-1.8197	0.0688	No	
Severity Stuffy or congested nose	-1.4899	0.1363	No	
Severity running nose	-2.4627	0.0138	Yes	-
Severity irritated or itchy nose	-0.5236	0.6005	No	
Severity sneezing episodes	-0.2267	0.8206	No	
Severity dry throat	-0.3430	0.7316	No	
Severity sore or irritated throat	-0.1492	0.8814	No	
Severity cough or hoarseness	-0.1661	0.8681	No	
Severity headache	-1.5761	0.1150	No	
Severity lethargy or drowsiness	-0.8039	0.4215	No	
Severity concentration/memory difficulty	-0.9052	0.3654	No	
Severity dizziness or faintness	-0.3068	0.7590	No	
Severity flu like symptoms	-0.9104	0.3626	No	
Severity aching limbs or backache	-0.4293	0.6677	No	
Severity difficulty in breathing	-1.3118	0.1896	No	
Severity chest tightness	-1.1646	0.2442	No	
Severity wheezing	-0.1529	0.8785	No	
Severity dry or itching skin	-1.5810	0.1139	No	
Severity skin blotches	-0.4465	0.6552	No	
TOTAL SIGNIFICANT DIFFERENCES			2	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**2/0**

SYMPTOMS Gender = Male BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-0.8853	0.3760	No	
Frequency Irritated or Watering Eyes	-0.2972	0.7663	No	
Frequency Strained Eyes	-0.3553	0.7224	No	
Frequency Stuffy or congested nose	-0.5257	0.5991	No	
Frequency running nose	-0.7992	0.4242	No	
Frequency irritated or itchy nose	-0.7559	0.4497	No	
Frequency sneezing episodes	-0.1272	0.8988	No	
Frequency dry throat	-1.2949	0.1953	No	
Frequency sore or irritated throat	-1.7137	0.0866	No	
Frequency cough or hoarseness	-0.9469	0.3437	No	
Frequency headache	-0.6266	0.5336	No	
Frequency lethargy or drowsiness	-1.2091	0.2266	No	
Frequency concentration/memory difficulty	-0.7542	0.4507	No	
Frequency dizziness or faintness	-0.1527	0.8786	No	
Frequency flu like symptoms	-0.0251	0.9800	No	
Frequency aching limbs or backache	-1.5233	0.1277	No	
Frequency difficulty in breathing	-0.1209	0.9038	No	
Frequency chest tightness	-0.0768	0.9388	No	
Frequency wheezing	-0.6452	0.5188	No	
Frequency dry or itching skin	-0.2875	0.7737	No	
Frequency skin blotches	-1.4057	0.1598	No	
TOTAL SIGNIFICANT DIFFERENCES			0	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				0/0

SYMPTOMS Gender Male BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-1.2635	0.2064	No	
Severity Irritated or Watering Eyes	-0.0822	0.9345	No	
Severity Strained Eyes	-0.5324	0.5945	No	
Severity Stuffy or congested nose	-0.3999	0.6892	No	
Severity running nose	-0.6310	0.5280	No	
Severity irritated or itchy nose	-0.6789	0.4972	No	
Severity sneezing episodes	-0.9709	0.3316	No	
Severity dry throat	-1.7808	0.0749	No	
Severity sore or irritated throat	-1.5066	0.1319	No	
Severity cough or hoarseness	-0.7362	0.4616	No	
Severity headache	-0.7736	0.4392	No	
Severity lethargy or drowsiness	-2.7119	0.0067	Yes	-
Severity concentration/memory difficulty	-1.9388	0.0525	No	
Severity dizziness or faintness	-0.3989	0.6900	No	
Severity flu like symptoms	-0.5749	0.5653	No	
Severity aching limbs or backache	-1.9200	0.0549	No	
Severity difficulty in breathing	-0.1759	0.8604	No	
Severity chest tightness	-0.5957	0.5514	No	
Severity wheezing	-0.4624	0.6438	No	
Severity dry or itching skin	-1.3555	0.8922	No	
Severity skin blotches	-1.6606	0.0968	No	
TOTAL SIGNIFICANT DIFFERENCES			1	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**1/0**

SYMPTOMS Smoker BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-0.8103	0.4178	No	
Frequency Irritated or Watering Eyes	-1.5138	0.1301	No	
Frequency Strained Eyes	-0.3431	0.7315	No	
Frequency Stuffy or congested nose	-0.4544	0.6495	No	
Frequency running nose	-1.1562	0.2476	No	
Frequency irritated or itchy nose	-0.4911	0.6234	No	
Frequency sneezing episodes	-0.1282	0.8980	No	
Frequency dry throat	-1.0962	0.2730	No	
Frequency sore or irritated throat	-0.2410	0.8096	No	
Frequency cough or hoarseness	-0.9598	0.3372	No	
Frequency headache	-1.4408	0.1496	No	
Frequency lethargy or drowsiness	-0.3053	0.7602	No	
Frequency concentration/memory difficulty	-0.0855	0.9318	No	
Frequency dizziness or faintness	-1.0170	0.3092	No	
Frequency flu like symptoms	-0.0355	0.9717	No	
Frequency aching limbs or backache	-0.7526	0.4517	No	
Frequency difficulty in breathing	-1.3276	0.1843	No	
Frequency chest tightness	-1.0169	0.3092	No	
Frequency wheezing	-1.1256	0.2603	No	
Frequency dry or itching skin	-0.7819	0.4343	No	
Frequency skin blotches	-1.7874	0.0739	No	
TOTAL SIGNIFICANT DIFFERENCES			0	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**0/0**

SYMPTOMS Smoker BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-1.0958	0.2732	No	
Severity Irritated or Watering Eyes	-1.4291	0.1530	No	
Severity Strained Eyes	-1.1804	0.2379	No	
Severity Stuffy or congested nose	-0.8458	0.3977	No	
Severity running nose	-0.4023	0.6874	No	
Severity irritated or itchy nose	-0.8593	0.3902	No	
Severity sneezing episodes	-0.3775	0.7058	No	
Severity dry throat	-1.5813	0.1138	No	
Severity sore or irritated throat	-1.0157	0.3098	No	
Severity cough or hoarseness	-0.7884	0.4304	No	
Severity headache	-1.8760	0.0607	No	
Severity lethargy or drowsiness	-0.7821	0.4341	No	
Severity concentration/memory difficulty	-0.7133	0.4757	No	
Severity dizziness or faintness	-0.5339	0.5934	No	
Severity flu like symptoms	-0.4338	0.6644	No	
Severity aching limbs or backache	-0.6102	0.5417	No	
Severity difficulty in breathing	-0.2282	0.8195	No	
Severity chest tightness	-0.1662	0.8680	No	
Severity wheezing	-0.1408	0.8881	No	
Severity dry or itching skin	-0.5630	0.5735	No	
Severity skin blotches	-1.7885	0.0737	No	
TOTAL SIGNIFICANT DIFFERENCES			0	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**0/0**

SYMPTOMS Ex-Smoker BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-1.3727	0.1699	No	
Frequency Irritated or Watering Eyes	-0.6659	0.5055	No	
Frequency Strained Eyes	-0.1126	0.9104	No	
Frequency Stuffy or congested nose	-0.3787	0.7049	No	
Frequency running nose	-0.4375	0.6617	No	
Frequency irritated or itchy nose	-0.2553	0.7985	No	
Frequency sneezing episodes	-0.5202	0.6029	No	
Frequency dry throat	-0.6837	0.4942	No	
Frequency sore or irritated throat	-0.9807	0.3267	No	
Frequency cough or hoarseness	-0.4375	0.6618	No	
Frequency headache	-0.9015	0.0572	No	
Frequency lethargy or drowsiness	-1.9213	0.0547	No	
Frequency concentration/memory difficulty	-0.3201	0.7489	No	
Frequency dizziness or faintness	-0.8811	0.3783	No	
Frequency flu like symptoms	-0.9930	0.3207	No	
Frequency aching limbs or backache	-0.3494	0.7268	No	
Frequency difficulty in breathing	-0.8051	0.4208	No	
Frequency chest tightness	-1.3757	0.1689	No	
Frequency wheezing	-1.4945	0.1350	No	
Frequency dry or itching skin	-0.7577	0.4486	No	
Frequency skin blotches	-1.1811	0.2376	No	
TOTAL SIGNIFICANT DIFFERENCES			0	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**0/0**

SYMPTOMS Ex-Smoker BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-1.3260	0.1848	No	
Severity Irritated or Watering Eyes	-1.1088	0.2675	No	
Severity Strained Eyes	-0.1531	0.8783	No	
Severity Stuffy or congested nose	-0.0076	0.9939	No	
Severity running nose	-0.4399	0.6600	No	
Severity irritated or itchy nose	-0.5377	0.5908	No	
Severity sneezing episodes	-0.3578	0.7205	No	
Severity dry throat	-0.5175	0.6048	No	
Severity sore or irritated throat	-0.8273	0.4080	No	
Severity cough or hoarseness	-1.0083	0.3133	No	
Severity headache	-0.7377	0.4607	No	
Severity lethargy or drowsiness	-1.7147	0.0864	No	
Severity concentration/memory difficulty	-0.3580	0.7203	No	
Severity dizziness or faintness	-0.0169	0.9865	No	
Severity flu like symptoms	-0.3837	0.7012	No	
Severity aching limbs or backache	-0.4419	0.6586	No	
Severity difficulty in breathing	-0.6761	0.4990	No	
Severity chest tightness	-0.3212	0.7481	No	
Severity wheezing	-0.3912	0.6957	No	
Severity dry or itching skin	-0.1021	0.9187	No	
Severity skin blotches	-1.2101	0.2262	No	
TOTAL SIGNIFICANT DIFFERENCES			0	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**0/0**

SYMPTOMS Non-Smoker BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-1.6896	0.0911	No	
Frequency Irritated or Watering Eyes	-3.9544	0.0001	Yes	-
Frequency Strained Eyes	-2.0469	0.0407	Yes	-
Frequency Stuffy or congested nose	-3.0381	0.0024	Yes	-
Frequency running nose	-4.8757	0.0924	Yes	-
Frequency irritated or itchy nose	-1.6828	0.0924	No	
Frequency sneezing episodes	-2.3469	0.0189	Yes	-
Frequency dry throat	-1.9906	0.0465	Yes	-
Frequency sore or irritated throat	-3.4032	0.0007	Yes	-
Frequency cough or hoarseness	-1.8598	0.0629	No	
Frequency headache	-2.8284	0.0047	Yes	-
Frequency lethargy or drowsiness	-1.1350	0.2564	No	
Frequency concentration/memory difficulty	-1.6695	0.0950	No	
Frequency dizziness or faintness	-0.8469	0.3971	No	
Frequency flu like symptoms	-0.5302	0.5959	No	
Frequency aching limbs or backache	-1.0634	0.2876	No	
Frequency difficulty in breathing	-1.0344	0.3010	No	
Frequency chest tightness	-0.3752	0.7075	No	
Frequency wheezing	-0.0686	0.9453	No	
Frequency dry or itching skin	-1.6615	0.0966	No	
Frequency skin blotches	-0.0414	0.9670	No	
TOTAL SIGNIFICANT DIFFERENCES			8	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**8/0**

SYMPTOMS Non-Smoker BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-0.7942	0.4271	No	
Severity Irritated or Watering Eyes	-3.2912	0.0010	Yes	-
Severity Strained Eyes	-1.8989	0.0576	No	
Severity Stuffy or congested nose	-1.5708	0.1162	No	
Severity running nose	-3.2548	0.0011	Yes	-
Severity irritated or itchy nose	-0.2026	0.8395	No	
Severity sneezing episodes	-1.1906	0.2338	No	
Severity dry throat	-1.9085	0.0563	No	
Severity sore or irritated throat	-1.2031	0.2289	No	
Severity cough or hoarseness	-0.6410	0.5215	No	
Severity headache	-2.5085	0.0121	Yes	-
Severity lethargy or drowsiness	-1.7735	0.0761	No	
Severity concentration/memory difficulty	-1.8640	0.0623	No	
Severity dizziness or faintness	-0.3373	0.7060	No	
Severity flu like symptoms	-0.8760	0.3810	No	
Severity aching limbs or backache	-1.6230	0.1046	No	
Severity difficulty in breathing	-1.2607	0.2074	No	
Severity chest tightness	-0.4793	0.6317	No	
Severity wheezing	-0.3982	0.6905	No	
Severity dry or itching skin	-1.9254	0.0542	No	
Severity skin blotches	-1.1619	0.2453	No	
TOTAL SIGNIFICANT DIFFERENCES			3	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**3/0**

SYMPTOMS Smoking working environment BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-2.1185	0.0341	Yes	-
Frequency Irritated or Watering Eyes	-1.9997	0.0455	Yes	-
Frequency Strained Eyes	-1.3631	0.1728	No	
Frequency Stuffy or congested nose	-1.4334	0.1517	No	
Frequency running nose	-0.4463	0.6554	No	
Frequency irritated or itchy nose	-0.9937	0.3204	No	
Frequency sneezing episodes	-0.5157	0.6061	No	
Frequency dry throat	-1.1015	0.2707	No	
Frequency sore or irritated throat	-0.9818	0.3262	No	
Frequency cough or hoarseness	-0.9750	0.3296	No	
Frequency headache	-1.2891	0.1974	No	
Frequency lethargy or drowsiness	-1.1040	0.2696	No	
Frequency concentration/memory difficulty	-0.6941	0.4876	No	
Frequency dizziness or faintness	-0.3590	0.7196	No	
Frequency flu like symptoms	-0.7935	0.4275	No	
Frequency aching limbs or backache	-1.4725	0.1409	No	
Frequency difficulty in breathing	-0.3589	0.7197	No	
Frequency chest tightness	-0.6620	0.5080	No	
Frequency wheezing	-0.3589	0.7197	No	
Frequency dry or itching skin	-0.7499	0.4533	No	
Frequency skin blotches	-0.6619	0.5080	No	
TOTAL SIGNIFICANT DIFFERENCES			2	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**2/0**

SYMPTOMS Non-smoking working environment BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-1.3587	0.1742	No	
Severity Irritated or Watering Eyes	-1.4899	0.1363	No	
Severity Strained Eyes	-1.1334	0.2570	No	
Severity Stuffy or congested nose	-0.3883	0.6978	No	
Severity running nose	-0.2787	0.7805	No	
Severity irritated or itchy nose	-0.9137	0.3609	No	
Severity sneezing episodes	-1.0978	0.2723	No	
Severity dry throat	-1.0984	0.2720	No	
Severity sore or irritated throat	-1.2275	0.2196	No	
Severity cough or hoarseness	-1.0964	0.2729	No	
Severity headache	-1.4360	0.1510	No	
Severity lethargy or drowsiness	-0.0388	0.9690	No	
Severity concentration/memory difficulty	-0.0755	0.9382	No	
Severity dizziness or faintness	-0.5236	0.6005	No	
Severity flu like symptoms	-0.4353	0.6633	No	
Severity aching limbs or backache	-0.7144	0.4750	No	
Severity difficulty in breathing	-0.5233	0.6008	No	
Severity chest tightness	-0.7265	0.4675	No	
Severity wheezing	-0.5845	0.5522	No	
Severity dry or itching skin	-0.7760	0.4377	No	
Severity skin blotches	-0.5945	0.5522	No	
TOTAL SIGNIFICANT DIFFERENCES			0	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				0/0

SYMPTOMS Non-Smoking working environment BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Frequency Dry Eyes	-0.5302	0.5959	No	
Frequency Irritated or Watering Eyes	-3.2156	0.0013	Yes	-
Frequency Strained Eyes	-1.8136	0.0697	No	
Frequency Stuffy or congested nose	-2.2680	0.0233	Yes	-
Frequency running nose	-4.1332	0.0000	Yes	-
Frequency irritated or itchy nose	-1.1613	0.2455	No	
Frequency sneezing episodes	-1.9884	0.0468	Yes	-
Frequency dry throat	-1.6876	0.0915	No	
Frequency sore or irritated throat	-3.5161	0.0004	Yes	-
Frequency cough or hoarseness	-1.4920	0.1357	No	
Frequency headache	-2.8339	0.0046	Yes	-
Frequency lethargy or drowsiness	-1.6734	0.0942	No	
Frequency concentration/memory difficulty	-1.6660	0.0957	No	
Frequency dizziness or faintness	-0.6291	0.5293	No	
Frequency flu like symptoms	-0.1081	0.9139	No	
Frequency aching limbs or backache	-0.9542	0.3400	No	
Frequency difficulty in breathing	-0.3602	0.7187	No	
Frequency chest tightness	-1.0373	0.2996	No	
Frequency wheezing	-0.2007	0.8409	No	
Frequency dry or itching skin	-1.1708	0.2417	No	
Frequency skin blotches	-1.2680	0.2048	No	
TOTAL SIGNIFICANT DIFFERENCES			6	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**6/0**

SYMPTOMS Non-Smoking working environment BUILDING 2	STATISTICS		$a^{.05}$	Direction
	Z-Score	2-tailed-P		
Severity Dry Eyes	-0.1526	0.8787	No	
Severity Irritated or Watering Eyes	-2.4981	0.0125	Yes	-
Severity Strained Eyes	-1.9782	0.0479	Yes	-
Severity Stuffy or congested nose	-1.7561	0.0791	No	
Severity running nose	-2.9322	0.0034	Yes	-
Severity irritated or itchy nose	-0.4151	0.6781	No	
Severity sneezing episodes	-1.1817	0.2373	No	
Severity dry throat	-1.0254	0.3052	No	
Severity sore or irritated throat	-1.6551	0.0979	No	
Severity cough or hoarseness	-1.1039	0.2696	No	
Severity headache	-1.9585	0.0502	No	
Severity lethargy or drowsiness	-2.2660	0.0235	No	
Severity concentration/memory difficulty	-2.2660	0.0235	Yes	-
Severity dizziness or faintness	-0.1289	0.8974	No	
Severity flu like symptoms	-0.7362	0.4616	No	
Severity aching limbs or backache	-0.8735	0.3824	No	
Severity difficulty in breathing	-1.2576	0.2085	No	
Severity chest tightness	-0.7699	0.4413	No	
Severity wheezing	-0.4639	0.6427	No	
Severity dry or itching skin	-1.1831	0.2368	No	
Severity skin blotches	-0.4343	0.6640	No	-
TOTAL SIGNIFICANT DIFFERENCES			4	
TOTAL NEGATIVE/POSITIVE DIFFERENCES				**4/0**

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Abramson, J. H., Terespolsky, L., Brook, J.G., Kark, S.L. (1965). Cornell Medical Index as a health measure in epidemiological studies: A test of the validity of a health questionnaire. *The British Journal of Preventative Medicine,* 19, 103-110.

2. Adama, M. H., W. (1983). The effect of posture on the fluid content of lumbar invertebral discs. *Spine,* 8, 665-671.

3. Aday, L. A. (1989). *Designing and conducting health surveys.* San Francisco: Jossey-Bass Publishers.

4. Ahblom A. (1988). A review of the epidemiologic literature on magnetic fields and cancer. *Scandinavian Journal of Work Environmental Health,* 14, 337-343.

5. Ahlstrom, R. B., B., Berglund, U., Lindvall, T. (1986). A comparison of odour perception in smokers, non-smokers and passive smokers.

6. Ajello, L. (1967). Comparative ecology of respiratory mycotic disease agents. *Bacteriol. Review,* 31, 6.

7. Albert, R. E., Lippmann, M. (1972). Factors influencing dust retention in the pulmonary parenchyma. *Annals New York Academy of Science,* 200, 37-45.

8. Albert, R. E. (1966). *Thorium: its industrial hygiene aspects.* New York: Academic Press.

9. Aldrich, T. E., C. (1985). Handbook of epidemiologic methods with special emphasis on ELF fields. *Publ. ORNL-6237.* Oak Ridge, Tennessee: Oak Ridge National Laboratory, Health and Safety Research Division.

10. Alfredsson, L. (1982). Job, psychosocial factors and coronary heart disease: Swedish prospective findings and U.S. prevalence findings using a new occupational interference method. In H. Denolin (ed.), *Psychological problems before and after myocardial infarction. Advanced Cardiology.* Bale: Karger.

11. Altmann, L. B., A., Wiegand, H. (1990). Neurophysiological and psychophysical measurements reveal effects of acute low-level organic solvent exposure in humans. *International Archives of Occupational and Environmental Health,* 62, 493-499.

12. American National Standards Institute/American Society of Refrigerating and Air Conditioning Engineers/Illuminating Engineers Society of North America. (1980). Energy conservation in new building design. (90A-1980). New York: ANSI/ASHRAE/IES.

13. Andersen, I. L., G.R., Proctor, D.F. (1972). Human mucosal function under four controlled humidities. *American Review of Respiratory Diseases,* 101, 438-449.

14. Andersen, I. B., Lundqvist, G.R., Proctor, D.F. (1973). Human perception under four controlled conditions. *Archives of Environmental Health,* 26, 22-27.

15. Andersen, I. L., G.R., Molhave, L. (1985). Indoor air pollution due to chipboard used as a construction material. *Atmospheric Environment,* 9, 1121-1127.

16. Anderson, E. W., Andelman, R.J., Strauch, J.M., Fortuin, N.J., Knelson, J.H. (1973). Effect of low level carbon monoxide exposure on onset and duration of angina pectoris. *Annals of Internal Medicine,* 79, 46.

17. Anderson, I. L., G.R., Molhave, L. (1975). Indoor air pollution due to chipboard used as a construction material. *Atmospheric Environment,* 9, 1121.

18. Annual report of the Chief Inspector of Factories and Workshops for the year 1898. (1898). Part II. London: HMSO.

19. Annual Report of the Chief Inspector of Factories and Workshops for the Year 1907. (1907). London: HMSO.

20. Anon. (1983). Electromagnetism and cancer. *Lancet,* 1, 224.

21. Anon. (1991). Understanding bioaerosols. *Indoor Air Quality Update,* 4(6), 1-5.

22. Anshelm Olson, B. G., F., Ingren, A. (1985). Coexposure to toluene and p-xylene in man: central nervous functions. *British Journal of Industrial Medicine,* 42, 117-122.

23. Arlidge, J. J. (1892). *The hygiene, disease and mortality of occupations.* London: Percival & Co.

24. Arlien-Soborg, P. B., P., Gyldensted, C., Melgaard, B. (1979). Chronic painter's syndrome. Chronic toxic encephalopathy in house painters. *Acta Neurol Scand,* 60, 149- 156.

25. Arnow, P. M., Fink, J.N., Schleuter, D.P. (1978). Early detection of hypersensitivity pneumonitis in office workers. *American Journal of Medicine,* 64, 236-241.

26. Arnow, W. S. (1978). Effects of passive smoking on angina pectoris. *New England Journal of Medicine,* 299, 21.

27. Aronow, W. S., Isbell, M.W. (1973). Carbon monoxide effect on exercise induced angina pectoris. *Annals of Internal Medicine,* 79, 392.

28. Aronow, W. S., Ferlinz, J., Glauser, F. (1977). Effect of carbon monoxide on exercise performance in chronic obstructive pulmonary disease. *American Journal of Medicine,* 63, 904.

29. Ashley, S. (1986). Sick buildings. *Building Services,* February, 25-30.

30. ASHRAE. (1977). ASHRAE Standard 62-1973, Standards for natural and mechanical ventilation. *Atlanta: American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc.,*

31. ASHRAE. (1981). ASHRAE Standard 62-1981. *Ventilation for acceptable indoor air quality. Atlanta: American Society of Refrigerating, and Air-Conditioning Engineers, Inc.,*

32. ASHRAE. (1989). ASHRAE Standard 62-1989, Ventilation for acceptable indoor air quality. *Atlanta: American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc.,*

33. Atherley, G. R. C. (1970). Moderate acoustic stimuli: The inter-relation of subjective importance and certain physiological changes. *Ergonomics,* 13, 536-545.

34. Auribault, M. (1906). Bulletin d'Inspection du Trevail Paris. 14, 120-132.

35. Ax, A. F. (1953). The physiological differentiation between fear and anger in humans. *Psychosomatic Medicine,* 15, 433- 442.

36. Babbie, E. (1992). *The practice of social research.* Belmont, California: Wadsworth.

37. Baechler, M. C., Ozkaynak, H., Spengler, J.D., Wallace, L.A., Nelson, W.C. (1989). Assessing indoor exposure to volatile organic compounds released from paint using the NASA data base. In *Proceedings of the Annual Meeting of the Air and Waste Management Association.* Anaheim, California. Air and Waste Management association, Pittsburgh, Pennsylvania.

38. Baechler, M. C., Hadley, D.L., Marseille, T.J. (1990). Indoor air quality issues related to the acquisition of conservation in commercial buildings. Richland, Washington: Pacific Northwest Laboratory.

39. Baelum, J. L., G.R., Molhave, L., Andersen, N.T. (1990). Human response to varying concentrations of toluene. *International Archives of Occupational and Environmental Health*, 62, 65-71.

40. Baelum, J. A., I., Lunqvist, G.R., Molhave, L., Pedersen, O.F., Vaeth, M., Wyon, D.P. (1985). Response of solvent- exposed printers and unexposed controls to six-hour toluene exposure. *Scandinavian Journal Work Environmental Health,* 11, 271-280.

41. Baelum, J. (1990). Toluene in alveolar air during controlled exposure to constant and to varying concentrations. *International Archives of Occupational and Environmental Health,* 62, 59-64. (Massey)

42. Bainbridge, L. (1974). Problems in the assessment of mental load. *Le Travail Humain,* 37, 279-302.

43. Baker, E. L., Seppainen, A.M. (1986). Human aspects of solvent neurobehavioural effects. *Neurotoxicology,* 7, 43-56.

44. Baker, E. L., Fine, L.J. (1986). Solvent neurotoxicity: The current evidence. *Journal of Occupational Medicine,* 28, 126- 129.

45. Baldwin, M. E., Farant, J.P. (1990). Study of selected volatile organic compounds in office buildings at different stages of occupancy. In *Proceedings of the 5th International Conference on Indoor Air Quality and Climate.* 2 Toronto, Canada. (pp. 665-670). Ottawa: Canada Mortgage and Housing Corporation.

46. Ball, D. J. (1982). A sceptic's view of air ionisation. *Building Services and Environmental Engineer,* 5(1), 21-22.

47. Bammer, G. B., I. (1987). A review of research on repetitive strain injuries (RSI). In P. W. Buckle (ed.), *Musculoskeletal Disorders at Work.* London: Taylor and Francis.

48. Bansazak, E. F., Thiede, W.H., Fink, J.N. (1970). Hypersensitivity pneumonitis due to contamination of an air conditioner. *New England Journal of Medicine,* 283(6), 271- 276.

49. Barbash, B. (1974). Job satisfaction and attitude surveys. (Document OECD-MS/IR/744.31). Paris: Organisation for Economic Co-operation and Development.

50. Bartlett, C. L. R., Dennis, P.J.L., Harper, D., Hutchinson, J.G.P., Kurtz, J.B., Tillett, H.E. (1988). Legionella in plumbing systems. *Journal British Occupational Hygiene Society,*

51. Bauer, M. A., Utell, M.J., Morrow, P.E., Speers, D.M., Gibb, F.R. (1984). 0.30 ppm nitrogen dioxide inhalation potentiates exercise-induced bronchospasm in asthmatics (abstract). *American Review of Respiratory Disease,* 129(Suppl: A151),

52. Bayer, R. A. (1982). Q fever as an occupational illness at the National Institutes of Health. *Public Health report,* 97, 58-60.

53. Bedford, T. (1974). Bedford's basic principles of ventilation and heating. London: Lewis.

54. Behrend, H. P., S. (1976). Absence and the individual: A six year study in one organisation. *International Labour Review,* 114, 311-327.

55. Beningus, V. A. (1977). Lack of effects of carbon monoxide on human vigilance. *Perceptual and Motor Skills,* 45, 1007-1014.

56. Benowitz, N. L., Kuyt, F., Jacob, P III, Jones, R.T., Osman, A.L. (1983). Cotinine disposition and effects. *Clinical Pharmacol Ther,* 34, 604-611.

57. Beral, V. (1982). Malignant melanoma and exposure to fluorescent lighting at work. *Lancet,* 2, 290-293.

58. Berglund, B. B., Berglund, U., Lindvall, T. (1986). Assessment of discomfort and irritation from indoor air. In *Indoor Air Quality '86. Managing Indoor Air for Health and Energy Conservation.* Atlanta, Georgia. (pp. 138-149). Atlanta: ASHRAE.

59. Berglund, B. B., Berglund, U., Lindval, T. (1984). Characterisation of Indoor Air Quality and Sick Buildings. *ASHRAE Transactions,* 90(1b), 1045-1955.

60. Berglund, B. (1982). A longitudinal study of air contaminants in a newly built preschool. *Environment International,* 8, 111-115.

61. Berglund, B. Berglund, U. (1982). Olfactory and chemical characterization of indoor air - towards a psychophysical model for air quality. *Environment International,* 8, 327-332.

62. Berglung, B. B., Lindval, T. (1986). Sensory reactions to sick buildings. *Environment International,* 12, 147-159.

63. Bergs, J. A. (1990). Survey of office absenteeism. *Netherlands Ministry for Housing and Construction,*

63b. Berkow, R., Fletcher, A. J. *The Merck Manual,* Merck Sharpe & Dohme Research Laboratories, Rathway NY.

64. Berlyne, D. E. (1960). *Conflict, arousal and curiosity.* New York: McGraw-Hill.

65. Bernard, J. M. (1986). Building associated illnesses in an office environment. *Proceedings Indoor Air '86. Managing Indoor Air Quality for Health and Energy.*

66. Bernstein, R. S., Sorensen, W.G., Garabrant, D., Reaux, C., Treitman, R.D. (1983). Exposures to airborne Penicillium from a contaminated ventilation system; clinical, environmental and epidemiological aspects. *American Industrial Hygiene Association Journal,* 40(3), 161-169. (Massey)

67. Berrens, L. (1966). On the composition of feather extracts used in allergy practice. *International Archives of Allergy,* 38, 166.

68. Bethwaite, P. P., N. (1991). Electromagnetic fields and cancer: an ongoing debate. *New Zealand Medical Journal,* June, 225-226.

69. Bindra, D. (1959). *Motivation: a systematic reinterpretation.* New York: Ronald Press.

70. Birren, F. (1950). *Colour psychology and colour therapy.* Secaucus: Citadel Press.

71. Birren, F. (1978). *Colour and human response.* New York: Von Nostrand Reinhold Company.

72. Bishop, V. L., Auster, D.E., Vogel, R.H. (1985). The sick building syndrome. What is it and how to prevent it. *National Safety and Health News,* 132, 31-38.

73. Bland, M. B., B.R., Pollard, V., Banks, M.H. (1978). Effect of children's smoking and parent's smoking on respiratory symptoms. *Arch. Dis. Child.,* 53, 100.

74. Bluyssen, P. F., P.O. (1990). Addition of olfs from different pollution sources. In *Indoor Air '90.* 1 Toronto., 569- 574.

75. Boleij, J. (1982). Indoor air pollution by carbon monoxide and nitrogen oxides. In K. Aurand (ed.), *Indoor Air Quality*. Stuttgart - New York: Gustav Fischer Verlag, 199-208.

76. BOMA. (1989). HVAC rated major concern of tenants. *ASHRAE*, July,

77. Bonham, G. S. (1981). Children's health in families with cigarette smokers. *American Journal of Public Health*, 71(3), 290.

78. Bonnell, J. A. (1982). Effects of electric fields near power- transmission plant. *Journal of the Royal Society of Medicine*, 75, 933-941.

79. Bonnell, J. A. (1983). Leukaemia and electrical workers. *Lancet*, 1, 1168.

80. Bonnell, J. A., Norris, W.T. (1984). Pylons, their fields, and man. *Lancet*, 2, 1343.

81. Bosse, R. (1978). Anxiety, extraversion and smoking. *British Journal of Social and Clinical Psychology*, 17, 269-273.

82. Botard, R. W., Kelley, D.C. (1969). A survey to determine the occurrence of Histoplasma capsulatum and Cryptoccus neoformans in air conditioners. *Mycopathol. Mycol. Appl.*, 37, 372.

83. Bouhuys, A. (1966). Asthma and byssinosis. *Rev. Allergy*, 22, 473-476.

84. Bouhuys, A. (1967). Response to inhaled histamine in bronchial asthma and byssinosis. *American Review of Respiratory Disease*, 95, 89-93.

85. Boxer, P. A., Signal, M., Hartle,. R.W. (1984). An epidemic of psychogenic illness in an electronics plant. *Journal of Occupational Medicine*, 26, 381-385.

86. Branton, P. O., D. (1979). A behaviourial study of anaesthetists at work. In D. Osborne (ed.), *Psychology and Medicine*. London: Academic Press.

87. Brett, J. M. (1980). The effects of job transfer on employees and their families. In C. L., Payne, R. Cooper (eds.), *Current Concerns in Occupational Stress*. Chichester, New York: John Wiley and Sons.

88. Broadbent, C. (1991). Sick buildings or just unwell? In *Engineering Solutions to the Sick Building Syndrome*. Sydney, Australia: BSFA, 7-16.

89. Broadbent, D. E., Gath, D. (1979). Chronic effects of repetitive and non-repetitive work. In T. M., C.J. Cox (eds.), *Response to Stress, Occupational Aspects*. Surrey: JPC Science and Technology Press.

90. Broadbent, D. F., Broadbent, M.H.P., Male, J.C., Jones, M.R.L. (1985). Health of workers exposed to electric fields. *British Journal of Industrial Medicine*, 42, 75-84.

91. Brundrett, G. W. (1977). A review of factors influencing electrostatic shocks in offices. *Journal of Electrostatics*, 2(4), 295-315.

92. Bulgin, D. (1953). Static electricity on rubber tyred vehicles. *British Journal of Applied Physics,* Suppl 2, 83-87.

93. Burge, H. P., Boise, J., Solomon, W.R., Bandera, E. (1978). Fungi in libraries; an aerometric survey. *Mycopathologia,* 64, 67.

94. Burge, H. A., Hodgson, M. (1988). Health risks of indoor pollutants. *ASHRAE Journal,* 30(7)

95. Burge, H. A. (1985). Indoor sources for airborne microbes. In Garnage & Kaye (eds.), *Indoor Air and Human Health, Proceedings of the Seventh Life Sciences Symposium.* Knoxville, Tennessee, 139-148.

96. Burge, H. A., Solomon, W.R., Boise, J.R. (1980). Microbial prevalence in domestic humidifiers. *Applied Environmental Microbiology,* 39, 840.

97. Burge, P. S. (1990). Building sickness - a medical approach to the causes. *Proceedings of the 5th International Conference on Indoor Air Quality and Climate.* 5 Toronto, Canada. Ottawa: Canada Mortgage and Housing Corporation, 3-14.

98. Burge, P. S., Finnegan, M.J., Horsefield, N. (1985). Occupational asthma in a factory with a contaminated humidifier. *Thorax,* 40, 248-254.

99. Burge, P. S., Hedge, A., Wilson, S., Harris-Bass, J., Robertson, A.S. (1987). Sick building syndrome: a national survey. *Annals of Occupational Hygiene,* 31, 493-504.

100. Burge, S. (1986). Office Environment Survey (questionnaire). *Occupational lung disease unit. East Birmingham Hospital. Birmingham B9 5ST. England,*

101. Burge, S. H., A., Wilson, S., Bass, J.H., Robertson, A. (1987). Sick building syndrome: a study of 4373 office workers. *Annals of Occupational Hygiene,* 31, 493 - 504.

102. Buzan, T. (1974). *Use both sides of your brain.* New York: E.P. Dutton.

103. Buzan, T. (1983). *Use both sides of your brain.* New York: E.P. Dutton.

104. Cail, F. (1980). Investigations in operators working with CRT display terminals: Relationships between task content and psychophysiological alterations. In *Ergonomic aspects of visual display terminals. Proceedings of the International Workshop.* Milan. (pp. 211-217). London: Taylor and Francis.

105. Cakir, A. (1979). *Visual display terminals.* New York: John Wiley & Sons.

106. Calle, E. S., D.A. (1985). Leukaemia in occupational groups with presumed exposure to electrical and magnetic fields. *New England Journal of Medicine,* 313, 1476-1477.

107. Cameron, P. K., J.S., Zaks, J.M., Wolfe, J.H., Tighe, G., Oslett, B., Stocker, R., Winton, J. (1969). The health of smokers and nonsmokers' children. *Journal of Allergy,* 43, 336.

108. Cannon, W. B. (1929). *Bodily changes in pain, hunger, fear, and rage.* New York: Appleton-Century.

109. Caplan, R. D. (1975). Job demands and worker health: Main effects and occupational differences. (HEW 75-160, NIOSH). Washington D.C.: United States Department of Health Education and Welfare.

110. Carpenter, C. F., Shaffer, C.B., Weil, C.S., Smyth, H.M. (1944). Studies on the inhalation of 1:3 butadiene; with a comparison of its narcotic effect with benzol, toluol, and styrene and a note on the elimination of styrene by the human. *Journal Industrial Hygiene and Toxicology,* 26, 69-78.

111. Carstensen, E. L. (1987). *Biological effects of transmission line fields.* New York: Elsevier.

112. Catren, O. W., K., Makelainen, I., Voutilainen, A. (1984). Radon measurements in Finnish houses. *Radiation Protection Dosimetry,* 7, 333.

113. Chaffin, D. B., Andersson, G.B.J. (1984). *Occupational Biomechanics.* New York: Wiley.

114. Chamberlain, A. C., Dyson, E.D. (1956). The dose to the trachea and bronchi from the decay products of radon and thoron. *British Journal of Radiology,* 29, 317-325.

115. Chamberlain, K. (1993). Department of Psychology, Massey University. [Private communication].

116. Chartered Institution of Building Services Guide Book (1978). *Section A1 environmental criteria for design* : CIBS.

117. Check, W. (1982). (Editorial) Sometimes getting into hot water solves a (legionella) problem. *JAMA,* 248, 2793-2794.

118. Cheever, E. A., C.K.D. (1978). *Beyond the basics, advanced kitchen design.* Sacramento: Cheever.

119. Cheliout, F. (1979). Rhythme theta posterieur au cours de la veille active chez l'homme. *Revue EEG en Neuropsychologie,* 9, 52-57.

120. Cherry, N. J., J.D., Venables, H., Waldron, H.A. (1983). The effects of toluene and alcohol on psychomotor performance. *Ergonomics,* 26, 69-78.

121. Cherry, N. G., D. (1990). Neurotoxic effects of styrene: further evidence. *British Journal of Industrial Medicine,* 47, 29-37.

122. Chrebtiw, I. (1991). Building Sickness - are engineering solutions the only answer? - What about chemicals? In *Engineering Solutions to the Sick Building Syndrome.* Sydney, Australia. (pp. 41-49). : BSFA.

123. Chrenko, F. A. ed (1974). *Bedford's basic principles of ventilation and heating.* London: Lewis.

124. Christensen, C. L. (1977). Effects of three kinds of hypoxias on vigilance performance. *Aviation, Space and Environmental Medicine,* 48, 491-496.

125. Chwat, M. M., R. (1963). Byssinosis investigations into cotton plants in Israel. In *14th International Conference in Occupational Health.* Int. Conf. Series No 62 Madrid. Amsterdam: Excerpta Medica.

126. Ciba Guest Symposium Report. (1959). Terminology, definitions and classification of chronic pulmonary emphysema and related conditions. *Thorax,* 14, 286-299.

127. CIBSE (1991). *The CIBSE guide.* London: CIBSE.

128. CIBSE (1984). *Code for interior lighting.* London: CIBSE.

129. Ciesielski, C. A., Blaser, M.J., Wang, W.-L.L. (1984). Role of stagnation and obstruction of water flow in isolation of Legionella pneumophilia from hospital plumbing. *Appl. Environ. Micro.,* 48, 984-987.

130. Clayton, Y. M., Midgley, G. (1971). Estimation of dermatophytes and Candida spores in the environment. *Journal Medical Microbiology,* 4, 3.

131. Cleary, P. D., Angel, R. (1984). The analysis of relationships involving dichotomous dependent variables. *Journal of Health and Social Behaviour,* 25, 334-348.

132. Clegg, C. W. (1983). Psychology of employee lateness, absence and turnover: A methological critique and an empirical study. *Journal of Applied Psychology,* 1, 88-101.

133. Clobes, A. 1., Ananth, G.P., Hood, A.L., Schroeder, J.A., Lee, K.A. (1991). *Human activities as sources of volatile organic compounds in residential environments.* Racine, Wisconsin: S.C. Johnson & Son, Inc.

134. Cobb, S. R., R.M. (1973). Hypertension, peptic ulcer and diabetes in air-traffic controllers. *JAMA,* 224(4), 489-492.

135. Cobb, S. (1947). Photic driving as a cause of clinical seizures in epileptic patients. *Archives of Neurology and Psychiatry,* 58, 70.

136. Cohen, B. G. F., Colligan, M.J., Wester, W., Smith, M.J. (1978). An investigation of job satisfaction factors in an incident of mass psychogenic illness at the workplace. *Occupational Health Nurs,* 26, 10-16.

137. Cohen, B. G. F. (1981). An investigation of health complaints and job stress in video display operations. *Human Factors,* 23(4), 387-400.

138. Coleman, M. P., Bell, J., Skeet, R. (1983). Leukaemia incidence in electrical workers. *Lancet,* 1, 983-983.

139. Coleman, M. (1985). Leukaemia mortality among amateur radio operators. *Lancet,* 1, 156.

140. Coleman, M. B., V. (1988). A review of epidemiological studies of the health effects of living near or working with electricity generation and transmission equipment. *International Journal of Epidemiology,* 17, 1-13.

141. Colley, J. R. T., Holland, W.W., Corkhill, R.T. (1974). Influence of passive smoking and parental phlegm on pneumonia and bronchitis in early childhood. *Lancet,* 2(7888), 1031.

142. Colley, J. R. T., Douglas, J.W.B., Reid, D.D. (1973). Respiratory disease in young adults: influence of early childhood lower respiratory tract illness, social class, air pollution, and smoking. *British Medical Journal,* 3, 195.

143. Colley, J. R. T. (1974). Respiratory tract symptoms in children and parental smoking and phlegm production. *British Medical Journal,* 2, 201.

144. Colligan, M. J. (1981). The psychological effects of indoor air pollution. *Bulletin of the New York Academy of Medicine,* 57, 1014-1026.

145. Collins, B. L., Fisher, W.S., Gillette, G.L., Marans. (1989). Evaluating office lighting environments: second level analysis. (NISTIR 89-4069). U.S.A.: National Institute Standards and Technology.

146. Collis, E. L. (1915). The occurrence of an unusual cough among weavers of cotton cloth. *Procedures Royal Society of Medicine,* 8(2), 108-112.

147. Comstock, L. S., C. (1973). A health survey of students 1: Prevalence of problems. *Journal of American College of Health Associations,* 22, 150-155.

148. Conway, D. M., Lacey, R.F. (1984). Asbestos in drinking water: Results of a survey. In *Technical Report TR 202.* U.K.: Water Research Centre.

149. Cooke, W. E. (1924). Fibrosis of the lungs due to inhalation of asbestos dust. *British Medical Journal,* II, 147.

150. Cooper, C. L. (1980). Investigating occupational stress: a methological approach. *Journal of Occupational Behaviour,* 1(3), 191-202.

151. Cooper, C. l., Marshall, J. (1976). Occupational sources of stress. A review of the literature relating to coronary heart disease and mental ill-health. *Journal of Occupational Psychology,* 49, 11-28.

152. Cordes, L. G., Wiesenthal, A.M., Gorman, G.W. (1981). Isolation of Legionella pneumophils from shower heads. *Annals of Internal Medicine,* 94, 195-197.

153. Cordes, L. G., Fraser, D.W., Skaliy, P., Perlino, C.A., Elsea, W.R., Mallison, G.F., Hayes, P.S. (1980). Legionnaire's disease outbreak in Atlanta, Georgia country club: evidence of spread from an evaporative condenser. *American Journal of Epidemiology,* 111, 425.

154. COST. (1989). Indoor pollution by NO_2 in European countries. *COST Project 613, Report 3.* : Commission of the European Communities Directorate General for Science, Research and Development Joint Research Centre - Institute for the Environment.

155. Coultas, D. B., Samet, J.M., Howard, C.A., Peake, G.T., Skipper, B.J. (1986). salivary cotinine levels and passive tobacco smoke exposure in the home (abstract). *American review Respiratory Diseases,* 132(Suppl:A), 157-158.

156. Cox, E. A. (1985). Radiation emissions from VDUs. In *Health Hazards of VDUs,* B. Pearse, ed. Chichester, New York: Wiley and Sons.

157. Cox, T. (1980). Repetitive work. In C. L., Payne, R. Cooper (eds.), *Current Concerns in Occupational Stress.* (pp. 23-42). New York: John Wiley and Sons.

158. Craig, M. (1981). *Office workers survival handbook. A guide to fighting health hazards in the office.* London: BSSRS Publications.

159. Crowley, T. P. (1936). Contaminated humidifiers. *JAMA,* 120,

160. Cunningham, A. M., Gregory, P.H. (1968). Mites in bedroom air. *Nature (London),* 217, 1271.

161. Cusack, J. A. (January 1972). Static control in carpets, Part 1. *Modern Textiles,* 66-70.

162. Cusack, J. A. (February 1972). Static control in carpets, Part 2. *Modern Textiles,* 25-29.

163. Cuttle, C. C., Valentine, W.B., Lynnes, J.A., Burt, W. (1967). Beyond the working plane. Procedures *Commissione Internationale d'Eclairage.* Washington DC. (pp. 471-482).

164. D'Alessio, D. J., Heeren, R.H., Hendricks, S.L., Ogilvie, P.H., Furcolow, M.L. (1965). A starling roost as a source of urban epidemic histoplasmosis in an area of low incidence. *American Review of Respiratory Disease,* 92, 725.

165. Damodaran, L. S., A., Wilson, p. (1980). *Designing systems for people.* Manchester: NCC Publications.

166. David, J. (January 1982). Negative response. *Building Services,*

167. Davidson, M. J., Veno, A. (1980). Stress and the policeman. In C. L., Marshall, J. Cooper (eds.), *White Collar and Professional Stress.* London: John Wiley and Sons.

168. Davies, C. N. (1967). In *Assessment of airborne radioactivity.* Vienna: International Atomic Energy Agency.

169. Davis, J. B. (1963). Review of scientific information on the effects of ionized air on human beings and animals. *Aerospace Medicine,* 34, 35-42.

170. Dawson, S. S. (1979). Health effects of inhalation of ambient concentrations of nitrogen dioxide. *American Review of Respiratory Disease,* 120, 281-292.

171. Delgado, J. M. R., Leal, J., Monteaoaguda, J.L., Garcia, M.G. (1982). Embryological changes by weak, extremely low frequency electromagnetic fields. *Journal of Anatomy,* 134, 533-5511.

172. den Tonklar, W. A. M, Rudolf, W. (1982). Air quality inside motor cars. In K. Aurand (ed.), *Indoor Air Quality.* (pp. 219- 233). Stuttgart - New York: Gustav Fischer Verlag.

173. Dennis, P. J. L. (1986). *Environmental factors affecting the survival and pathogenicity of Legionella pneumophilia.* [Council for National Academic Awards].

174. Department of the Environment (1986). *Asbestos materials in buildings.* London: HMSO.

175. Department of Health. Threshold limit values and short-term exposure limits. *Pamphlet.*

176. Departmental Committee on Compensation for Industrial Diseases. (1907). *Report.* London: HMSO.

177. Derogatis, L. R., Lipman, R.S., Rickels, K., Uhlenhute, E.H., Covi, L. (1974). The Hopkins Symptom Checklist (HSCL): A measure of primary symptom dimensions. *Psychological Measurement in Psychopharmacology,* 7, 79-110.

178. Dick, R. B., Setzer, J.V., Wait, R., Hayden, M.B., Taylor, B.J., Tolos, B., Putz-Anderson, V. (1984). Effects of acute exposure to toluene and methyl ethyl ketone on psychomotor performance. *International Archives of Occupational Environmental Health,* 54, 91-109.

179. Dillman, D. A. (1983). Mail and other self-administered questionnaires. In P. H., Wright, J.D., Anderson, A.B. Rossi (eds.), *Handbook of Survey research.* Orlando, Fla.: Academic Press.

180. Dimmick, R. L., Akers, A.B. (1969). *An introduction to experimental aerobiology.* New York: Wiley.

181. Dixon, R. E. (1985). Economic costs of respiratory tract infections in the United States. *American Journal of Medicine,* 78(Suppl b), 45-51.

182. Doig, A. T. (1949). Other lung diseases due to dust. *Postgraduate Med Journal,* 25, 639-649.

183. Doll, R. P., J. (1985). *Effects on health of exposure to asbestos.* Britain: Health and Safety Commission.

184. Doll, R. (1955). Mortality from lung cancer in asbestos workers. *British Journal of Industrial Medicine,* 12, 81-86.

185. Driken, J. M. (1973). Stress and cardiovascular health: An international co-operative study: II. The male population of a factory at Zurich. *Social Science and Medicine,* 7, 573-584.

186. Dritz, S. B., A., Hine, C., Spinelli, J., Morrish, R., Wade, R., Roberto, R. (1979). *MMWR,* 28, 333-334.

187. Dubios, R. (1969). The human environment. *Science Journal,* 5, 75-80.

188. Duffey, E. (1941). The conceptual categories of psychology: a suggestion for revision. *Psychological Review,* 48, 177-203.

189. Duffy, E. (1962). *Activation and behaviour.* New York: Wiley.

190. Duffy, E. (1951). The concept of energy mobilization. *Psychological Review,* 58, 30-40.

191. Easterby, R. S. (1967). Ergonomics checklists: an appraisal. *Ergonomics,* 10(5), 549-559.

192. Eden, D. (1973). Job stresses and risk factors in coronary heart disease among five occupational categories in kibbutzim. *Social Science and Medicine,* 7, 875-892.

193. Edwards, J. H., Barboriak, J.J., Fink, J.N. (1970). Antigens in pigeon breeders disease. *Immunology,* 19, 727.

194. Edwards, J. H., Griffiths, A.J., Mullins, J. (1976). Protozoa as sources of antigen in humidifier fever. *Nature (London),* 264, 438.

195. Edwards, J. H., Wagner, J.C., Seal, R.M.E. (1976). Pulmonary responses to particulate materials capable of activating alternative pathway of compliment. *Clinical Allergy,* 6, 155- 164.

196. Ehdwall, H. (1980). Tracing and dealing with dwellings with high radon and radon daughter concentrations. In *Third Workshop on Radon and Radon Daughters in Urban Communities Associated with Uranium Mining and Processing.* (report AECB-1161-3). Ottawa: Atomic Energy Control Board.

197. Eisdorfer, D. E., G.R. (eds) (1982). *Stress and human health.* New York: Springer.

198. Ekberg, L. E. (1991). Indoor air quality in a new office building. *Proceedings of IAQ '91 - Healthy Buildings.* Washington DC. Atlanta: ASHRAE.

199. Ekwo, E. E., Weinberger, M.W., Lachenbruch, P.A., Huntley, W.H. (1983). Relationship between parental smoking and gas cooking to respiratory disease in children. *Chest,* 84, 662- 668.

200. Ellis, V. S. (July 1974). Epitropics - third generation conductive fibres. *Textile Manufacturer,* 19-23.

201. Ellwood, P. C., Pemberton, J., Merrett, J.D., Carey, G.C.R., McAulay, I.R. (1965). Byssinosis and other respiratory symptoms in flax workers in Northern Ireland. *British Journal of Industrial Medicine,* 22, 27-37.

202. EML (1972). *Environmental Measurements Laboratory Procedure Manual; HASL-300.* : US Department of Energy.

203. Engen, T. (1982). *Perception of odors.* New York: Academic Press.

204. Engholm, G. E., A., Fletcher, A.C., Hallin, N. (1987). Respiratory cancer incidence in Swedish construction workers exposed to man-made mineral fibres and asbestos. *Annals of Occupational Hygiene,* 31, 663-675.

205. England, A. C., Fraser, D.W., Pilkaytis, B.D., Tsai, T.F., Storch, G., Broome, C.V. (1981). Sporadic legionellosis in the United States. *Annals of Internal Medicine,* 94, 164-170.

206. Enstrom, J. E. (1979). Rising lung cancer mortality among nonsmokers. *Journal of the National Cancer Institute,* 62, 755-760.

207. Enterline, P. E., Marsh, G.M. (1984). The health of workers in the MMMF industry. In *Biological Effects of Man-Made Fibres.* 1 (pp. 311-339). Copenhagen: WHO Regional Office for Europe.

208. EPA. (1989). Addressing indoor air quality at EPA headquarters facilities. US: Environmental Protection Agency. (Report to congress on indoor air quality, volume 1, federal programs addressing indoor air quality.)

209. EPA. (1991). Building air quality: A guide for building owners and facility managers. Washington DC: U.S. Government Printing Office.

210. EPA. (1988). Indoor air facts No.4. *Sick Buildings.* : U.S. Environmental Protection Agency.

211. EPA. (1991). Two indoor air exposure modelling studies: contam modelling results, and serial correlation effects. *NTIS Data Base,* PB91-159707, 1-34.

212. Epstein, N. (1977). The effects of tobacco smoke pollution on the eyes of the allergic nonsmoker. In J. G., W., Ball, K., Taylor, R.M. Steinfeld (eds.), *Proceedings of the Third World Conference on Smoking and Health.* Washington, D.C. : DHEW Publication number (NIH) 77-1413.

213. Ericson, A. K., B. (1986). An epidemiological study of work with video screens and pregnancy outcome: a registry study. *American Journal of Industrial Medicine,* 9, 447-457 & 459- 475.

214. Ericson, S. S., H., Clavensjo, B. (1984). Modified technology in new constructions, and cost effective remedial action in existing structures, to prevent infiltration of soil gas carrying radon. *Radiation Protection Dosimetry,* 7, 223.

215. Ericson, S.-O. (1980). Some remarks about remedial actions and research program in Sweden. In *Third Workshop on Radon and Radon Daughters in Urban Communities Associated with Uranium Mining and Processing.* (report AECB-1161-3). Ottawa: Atomic Energy Control Board.

216. Evans, G. W., Jacobs, S.V. (1981). Air pollution and human behaviour. *Journal of Social Issues,* 37, 95-125.

217. Everson, R. B., Sandler, D.P., Wilcox, A.J., Schreinemachers, D., Shore, D.L., Weinberg, C. (1986). Effect of passive exposure to smoking on age at natural menopause. *British Medical Journal,* 293, 792.

218. Everson, R. B. (1980). Individuals transplacentally exposed to maternal smoking may be at increased cancer risk in adult life. *Lancet,* 2, 123-127.

219. Experts Report (1985). *Metrology and monitoring of radon and thoron and their daughter products.* Paris: OECD Nuclear Energy Agency.

221. Fanger, P. O., Lauridsen, J., Bluyssen, P., Clausen, G. (1988). Air pollution sources in offices and assembly halls, quantified by the olf unit. *Energy and Buildings,* 12, 7-19.

222. Fanger, P. O. (1991). Designing for Good Air Quality in Air- Conditioned Buildings. *Institute of Refrigeration,* Pamphlet,

223. Fanger, P. O. (1988). Hidden olfs in sick buildings. *ASHRAE Journal,* 30(11), 40-42.

224. Fanger, P. O. (1988). Introduction to the olf and decipol units to quantify air pollution perceived by humans indoors and outdoors. *Energy and Buildings,* 12, 1-6.

225. Fanger, P. O. (1989). The new comfort equation for indoor air quality. In *The Human Equation, Proceedings of the ASHRAE/SEOG Conference IAQ 89.* San Diego, California. : American Society of Heating, Refrigerating, and Air- Conditioning Engineers, Inc., Atlanta, Georgia.

226. Fanger, P. O. (1989). The new comfort equation for indoor air quality. *ASHRAE Journal,* 33-38.

227. Fanger, P. O. (1990). New principles for a future ventilation standard. In *Indoor Air 90: Proceedings of the Fifth International Conference on Indoor Air Quality and Climate.* Toronto. : Canada Mortgage and Housing Corporation.

228. Fanger, P. O. (1988). Olf and decipol - the new units for perceived air quality. *Australian Refrigeration, Air Conditioning and Heating,* 56-61. (Massey)

229. Farant, J. P., Bedard, S., Tamblyn, R.T., Menzies, R.I., Tamblyn, R.M., Hamley, J., Spitzer, W.O. (1990). Effect of changes in the operation of a building's ventilation system on environmental conditions at individual workstations in an office complex. *Proceedings of the 5th International Conference on Indoor Air Quality and Climate.* 1 Toronto, Canada. (pp. 581-585). Ottawa: Canada Mortgage and Housing Corporation.

230. Feinberg, S. M. (1946). *Allergy in practice.* Chicago: Year Book Medical Publishing.

231. Feldman, L. R., Eaglestein, W.H., Johnson, R.B. (1985). Terminal illness. *Journal of the American Academy Dermatol.,* 12, 366.

232. Field, A. (1987). Infrasound and sick buildings. *Building Services,* January, 59.

233. Fine, L. P., L., Keyserling, W. (1987). An epidemiological study of postural risk factors for shoulder disorders in industry. In P. W. Buckle (ed.), *Musculoskeletal Disorders at Work.* London: Taylor and Francis.

234. Fink, J. N., Banaszak, E.A., Thiede, W.H., Barboriak, J.J. (1971). Interstitial pneumonitis due to hypersensitivity to an organism contaminating a heating system. *Annals of Internal Medicine,* 74, 80-83.

235. Fink, J. N., Resnick, A.S., Salvaggio, J. (1971). Presence of thermophilic actinomycetes in residential heating systems. *Applied Microbiology,* 22, 730.

236. Finnegan, M. J., Pickering, C.A.C. (1986). Building related illness. *Clinical Allergy,* 16, 389-405. (Massey)

237. Finnegan, M. J., Pickering, C.A.C. (1985). Work related asthma and humidifier fever in air conditioned buildings. In B. L., T., Sundell, J. Berglund (eds.), *3rd International Conference on Indoor Air Quality and Climate.* Vol 1: Recent Advances in Health Sciences and Technology Stockholm. (pp. 257-262). : Swedish Council for Building Research.

238. Fischer, P. (1985). Indoor air pollution and its effect on pulmonary function of adult non-smoking women, II. Associations between nitrogen dioxide and pulmonary function. *International Journal of Epidemiology,* 14, 221-226.

239. Fishman, D. S. (1981). Subjective comfort associated with negative air ions. *Heating Ventilating Engineer,* Oct, 13-15.

240. Florey, C. (1979). The relation between respiratory illness in primary schoolchildren and the use of gas for cooking, III: Nitrogen dioxide, respiratory illness and lung function. *International Journal of Epidemiology,* 8, 347-353.

241. Florida Electric Power Coordinating Group (1985). *Biological effects of 60-Hz power transmission lines.* Springfield, Virginia: NTIS.

242. Flynn, N. M., Hoperich, P.D., Rawacki, M.M., Lee., K.K., Lawrence, R.M., Goldstein, E., Jordan, G.W., Kundarge, R.S., Wong, G.A. (1979). An unusual outbreak of windborne coccidiodomycosis. *New England Journal of Medicine,* 301, 358.

243. Folinsbee, L. J. (1984). Pulmonary function changes after 1- hour continuous heavy exercise in 0.21 ppm ozone. *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology,* 57, 984-988.

244. Fontan, J. (1964). *Le dosage des radioelements gazeux donnant des produits de filtration. Son application a la mesure de la radioactivite naturelle de l'atmosphere, these de doctorat d'etat.* Toulouse, France.

245. Fontan, J. B., D., Bouville, A. (1962). Emploi d'un systeme de coincidences dans le dosage selctif du radon et du thoron atmospheriques. *Le Journal de Physics et le Radium Tome,* 23, 333.

246. Forthofer, R. N., Lehnen, R.G. (1981). *Public program analysis: a new categorical data approach.* Belmont, California: Lifetime Learning.

247. Fox, A. J., Tombleson, J.B.L., Watt, A., Wilkie, A.G. (1973). A survey of respiratory disease in cotton operatives. Part II Symptoms, dust estimations and the effect of smoking habit. *British Journal Industrial Medicine,* 30, 48-53.

248. Fox, G. A. (1971). Personnel selection, vocational guidance and job satisfaction. In *Proceedings of Seminar on Job Satisfaction.* New South Wales, Australia. : Division of Occupational Health and Pollution Control, Department of Health.

249. Frank, C. (1986). Eye symptoms and signs in buildings with indoor climate problems (Office eye syndrome). *Acta Opthalmologica,* 64, 306-311.

250. Fraser, T. M. (1983). Human stress, work and job satisfaction: A critical approach. In *Occupational Safety and Health Series No.50.* Geneva: International Labour Organisation.

251. Freeman, G. L. (1948). *The energetics of human behaviour.* Ithaca, New York: Cornell University Press.

252. Freidman, G. D., Petitti, D.B., Bawol, R.D. (1983). Prevalence and correlates of passive smoking. *American Journal of Public Health,* 73, 401-405.

253. Friedlander, S. F., A.S. (1954). Effectiveness of a portable electrostatic precipitator in the elimination of environmental allergins and control of allergic symptoms. *Annals of Allergy,* 12, 419.

254. Friend, J. A. R., Gaddie, J., Palmer, N.K.V., Pickering, C.A.C., Pepys, J. (1977). Extrinsic allergic alveolitis and contaminated cooling-water in a factory machine. *Lancet,* 1, 297-300. (Massey)

255. Froberg, J. (1970). Conditions of work: Psychological and endocrine stress reactions. *Archives of Environmental Health,* 21, 789-797.

256. Fucigna, J. T. (1967). The ergonomics of offices. *Ergonomics,* 10(5), 589-604.

257. Fulton, J. C., S., Preble, L. (1980). Electrical wiring configurations and childhood leukemia in Rhode Island. *American Journal of Epidemiology,* 111, 292-296.

258. Galer, I. E. (1987). *Applied Ergonomics Handbook (2nd edition).* London: Butterworths.

259. Gamble, J. M., Richards, T., Petersen, M., Castellan, R.M. (1986). Building - related symptoms: problems in identification. , 16-30.

260. Gamboa, R. R., Gallagher, B.P., Mathews, K.R. (1988). Data on glass fibre contribution to the supply airstream from fibreglass duct liner and fibreglass duct board. In *IAQ 88: Engineering Solutions to Indoor Air Problems.* : American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc., Atlanta, Georgia.

261. Gammage, R. B., Gupta, K.C. (1984). Formaldehyde. In C. D., P., Copenhaver, E. Walsh (eds.), *Indoor Air Quality.* 109-139 Boca Raton: CRC Press.

262. Gammage, R. B., Hansen, D.L., Johnson, L.W. (1989). Indoor air quality investigations: a practitioner's approach. *Environment International,* 15, 503-510.

263. Gandevia, B. M., J. (1965). Ventilatory capacity changes on exposure to cotton dust and their relevance to byssinosis in Australia. *British Journal of Industrial Medicine,* 22, 295- 304.

264. Gardell, B. (1978). Arbeitsgestaltung, intrinsische Abeitszufriedenheit und Gesundheit. In *Industrielle Psychopathologie* (Frese, M.) Berne: Verlag Hans Huber.

265. Gardell, B. (1976). Technology, alienation and mental health. *Acta Sociologica,* 19, 83-94.

266. Garfinkel, L. (1981). Time trends in lung cancer mortality among nonsmokers and a note on passive smoking. *Journal of the National Cancer Institute,* 66, 1061.

267. Garland, C. B., E., Saurez, L., Criqui, M.H., Wingard, D.L. (1985). Effects of passive smoking on ischemic heart disease mortality of nonsmokers. *American Journal of Epidemiology,* 121, 645-650.

268. Gellhorn, E. (1964). Motion and emotion: the role of proprioception in the physiology and pathology of emotions. *Psychol. Review,* 71, 457-472.

269. George, A. C., Breslin, A.J. (1980). The distribution of ambient radon and radon daughters in buildings in the New Jersey-New York area. In T. F., Lowder, W.M. Gelsell (eds.), *Natural Radiation Environment III.* (CONF-780422). Springfield, VA: Technical Information Centre Department of the Environment.

270. Gilkes, M. J. (1979). Eyes run on light. *British Medical Journal,* 1, 1681-1683.

271. Gill, C. I. C. (1947). Byssinosis in the cotton trade. *British Journal of Industrial Medicine,* 4, 48-55.

272. Gillis, C. R., Hole, D.J., Hawthorne, V.M., Boyle, P. (1984). The effect of environmental tobacco smoke in two urban communities in the west of Scotland. *European Journal of Respiratory Disease,* 65(Suppl 133), 121-126.

273. Gilmer, V. H. (1961). *Attitudes, job satisfaction and industrial morale, in Industrial Psychology.* New York: McGraw Hill Book Company.

274. Girman, J. R., Alevantis, L., Petreas, M., Webber, L. (1989). The bake-out of an office building: a case study. *Environment International,* 15, 449-453.

275. Glick, T. H., Gregg, M.B., Berman, B., Mallinson, G., Rhodes, W.W., Kassanoff, I. (1978). Pontiac fever. An epidemic of unknown etiology in the health department: Clinical and epidemiological aspects. *American Journal of Epidemiology,* 107, 149-160.

276. Godish, T. (1989). *Indoor air pollution control.* Chelsea, Michigan: Lewis Publishers, Inc.

277. Goldenthorpe, J. H. (1963). *The affluent worker: Industrial attitudes and behaviour.* Cambridge: The University Press.

278. Grande, L. B., Hanssen, S.O. (1988). Allergic persons, human sensors regarding indoor environment quality? In B. L., T. Berglund (eds.), *Proceedings of Healthy Buildings 88.* 3 Stockholm, Sweden. (pp. 561-564). Stockholm: Swedish Council for Building Research.

279. Grandjean, E. (1987). *Ergonomics of the computerised office.* London: Taylor and Francis.

280. Grandjean, E. (1988). *Fitting the Task to the Man: a Textbook of Occupational Ergonomics.* London: Taylor & Francis.

281. Graverson, S. S., P., Valbjorn, O., Lowestein, H. (1990). The role of potential immunogenic components of dust (MOD) in the sick-building-syndrome. *Proceedings of the 5th International Conference on Indoor Air Quality and Climate.* 1 Toronto, Canada. (pp. 9-13). Ottawa: Canada Mortgage and Housing Corporation.

282. Gray, M. (1978). *Neuroses.* New York: Von Nostrand.

283. Green, G. H. (1984). *The health implications of the level of indoor air humidity, Indoor Air Vol 1.* Stockholm: Swedish Council for Building Research.

284. Greenberg, R. A., Haley, N.J., Etzel, R.A., Loda, F.A. (1984). Measuring the exposure of infants to tobacco smoke: nicotine and cotinine in urine and saliva. *New England Journal of Medicine,* 310, 1075-1078.

285. Greenhow, H. (1860). Third report of the medical officer of the privy council. *Sir John Simon,*

286. Greenly, J. M., D. (1976). Social selection in seeking help for psychological problems. *Journal of Health and Social Behaviour,* 17, 249-262.

287. Gregory, P. H. (1973). *The microbiology of the atmosphere.* New York: John Wiley and Sons.

288. Griffiths, W. A. D., Wilkinson, D.S. (1985). *Essentials of Industrial Dermatology.* Oxford: Blackwell.

289. Grot, R. A., Persily, A.K. (1983). Air infiltration and air tightness tests in eight U.S. office buildings. Air infiltration reduction in existing buildings. In *4th Air Change Conference.* Elam. Switzerland.

290. Grot, R. A., Persily, A.K. (1986). Measured air infiltration and ventilation rates in eight large office buildings. In H. R., Lagus, P.L. Trechel (eds.), *Measured Air Leakage of Buildings, ASTM STP 904.* (pp. 151-183). Philadelphia: American Society for Testing and Materials.

291. Grufferman, S. D., E.S., Maile, M.C., Michalopoulos, G. (1983). Parents' cigarette smoking and childhood cancer. *Medical Hypotheses,* 12, 17-20.

292. Grufferman S., W. H. H., Delong, E.R, Kimm, S.Y., Delzell, E.S., Falletta, J.M. (1982). Environmental factors in the etiology of rhabdomyosarcoma in childhood. *Journal of the National Cancer institute,* 68, 107-113.

293. Guidotti, T. L., Alexander, R.W., Fedoruk, M.J. (1987). Epidemiological features that may distinguish between building associated illness outbreaks due to chemical exposure or psychogenic origin. *Journal of Occupational Medicine,* 29, 148-150.

294. Gunnarsson, E. O., O. (1977). *(English translation) Fysisk och psykisk arbetsmiljo i ett terminalsbaserat datasystem.* Stockholm: Arbetarskyddsstryrelsen, undersokningsrapport.

295. Haider, M. (1976). Effects of moderate CO dose on the central nervous system - electrophysiological and behaviour data, clinical relevance. In A. J., Duel, W.C. Finkel (eds.), *Clinical Implications of Air Pollution Research.* Acton, MA. (pp. 217-232). Publishing Sciences Group: Air Pollution Medical Research Conference, American Medical Association.

296. Halweg, H. K., P., Podsaid, O.B., Owczarek, J., Ponahajba, A., Pawlicka, L. (1978). Studies on air pollution by fungal spores at selected working posts at a paper factory. *Pneumonol. Pol.,* 46, 577.

297. Hansen, T. B., Andersen, B. (1986). Ozone and other air pollutants from photocopying machines. *American Industrial Hygiene Association Journal,* 47(10), 659-665.

298. Harlap, S. D., A., Haber, M., Samueloff, N., Rossman, H., Prywes, R. (1975). Ethnic group, immigration and infant morbidity in West Jerusalem. In L. Levi (ed.), *Society, Stress and Disease.* Vol. 2 London: Oxford University Press.

299. Harlap, S. D., A.M. (1974). Infant admissions to hospital and maternal smoking. *Lancet,* 1(7857), 529.

300. Harris Polls. (1989). Public perception of indoor environment. *Healthy Buildings International,* (Survey of building occupants in business districts of major cities; Singapore, Germany, United Kingdom, France and Australia).

301. Harris, T. R., Merchant, J.A., Kilburn, K.H., Hamiltion, J.D. (1972). Byssinosis and respiratory diseases in cotton mill workers. *Journal Occupational Medicine,* 14, 199-206.

302. Harrison, J. P., C.A.C., Faragher, E.B., Austwick, P.K.C. (1990). An investigation of the relationship between microbial and particulate indoor air pollution and the sick building syndrome. *Proceedings of the 5th International Conference on Indoor Air Quality and Climate.* 1 Toronto, Canada. (pp. 149-154). Ottawa: Canada Mortgage and Housing Corporation.

303. Hawkins L.H. (1981). Studying air ionisation and effects on health. *Building Services and Environmental Engineering,* (April)

304. Hawkins, L. H., Barker, T. (1978). Air ions and human performance. *Ergonomics,* 21(4), 273-278.

305. Hawkins, L. M., L. (1984). Air ions and the sick building syndrome. *Indoor Air,* 3, 197-200.

306. Hawkins, L. (1981). The influence of air ions, temperature and humidity on subjective wellbeing. *Journal Environmental Psychology,* 1, 279-292.

307. Hawkins, L. H. (1982). Negative air ionisation. *Building Services and Environmental Engineer,* April.

308. Hawkins, L. H. (1979). Problems of air ions and air conditioning. *Building Services and Environmental Engineering,* August.

309. Hearle, J. W. S., Peters, R.H. (1960). Moisture in textiles. *Textile Institute.*

310. Hebb, D. O. (1955). Drives and the CNS (conceptual nervous system). *Psychol. Review,* 62, 243-254.

311. Hedge, A. E., E. (1982). Air ionisation: an evaluation of its physiological and psychological effects. *Annals of Occupational Hygiene,* 25(4), 409-419.

312. Hedge, A. S., E.M., Sterling, T.D. (1986). Building related illness indices based on questionnaire responses. In *Proceedings of the ASHRAE conference, Indoor Air Quality '86.* Managing Indoor Air for Health and Energy Conservation (pp. 31-43).

313. Hedge, A. C. (1987). Do negative ions affect mood and performance? *Annals of Occupational Hygiene,* 31, 285-290.

314. Hedge, A. (1991). Healthy office lighting for computer workers: a comparison of lensed-indirect and direct systems. *Proceedings of IAQ '91 - Healthy Buildings.* Washington DC. (pp. 61-66). Atlanta: ASHRAE.

315. Hedge, A. S., Sterling, E.M. (1989). Indoor air quality and health in two office buildings with different ventilation systems. *Environment International,* 15, 115-128.

316. Hedge, A. B., Robertson, A.S. (1989). Work-related illness in offices: A proposed model of the sick building syndrome. *Environment International,* 15, 143-158. (Massey)

317. Hellstrom, B. P., Strom, G. (1990). Four buildings with SBS-symptoms. *Proceedings of the 5th International Conference on Indoor Air Quality and Climate.* 4 Toronto, Canada. (pp. 385-390). Ottawa: Canada Mortgage and Housing Corporation.

318. Helms, C. M., Massanari, M., Zeitler, R., Streed, S., Gilchrist, M.J.R., Hall, N., Hausler, W.J., Sywassink, J., Johnson, W., Wintermeyer, L., Hierholzer, W.J. (1983). Legionnaires disease associated with a hospital water supply: a cluster of nosocomial cases. *Annals of Internal Medicine,* 98,

319. Henschel, D. B., Scott, A.G. (1986). The EPA program to demonstrate mitigation measures for indoor radon: Initial results. In *Indoor Radon. Procedure APCA International Specialty Conference.* Philadlephia. Pittsburg PA: Air Pollution Control Association.

320. Hertzberg, F. (1966). *Work and the nature of man.* New York: World Publishing.

321. Hess, C. T., Weiffenbach, C.V., Norton, S.A. (1983). Environmental radon and cancer correlations in Maine. *Health Physics,* 454, 339.

322. Hewitt, M. B., G.I., Miller, D.C., Turk, F., Turk, S. (1973). Mites in the personal environment and their role in skin disorders. *British Journal of Dermatology,* 89, 401.

323. Hicks, J. B. (1984). Tight building syndrome: when work makes you sick. *Occupational Health and Safety,* January, 51- 56.

324. Hill, M. J. (1973). *Men out of work: A study of unemployment in three English towns.* Cambridge: The University Press.

325. Hills, N. E. (1979). *You are a rainbow.* Boulder City: University of Trees Press.

326. Hirayama, T. (1981). Non-smoking wives of heavy smokers have a higher risk of lung: a study from Japan. *British Medical Journal,* 282(183),

327. Hirayama, T. (1981). Passive smoking and lung cancer. *British Medical Journal,* 282, 1393.

328. Hirayama, T. (1983). Passive smoking and lung cancer: consistency of association. *Lancet,* 2, 1425-1426.

329. Hirsch, S. R. (1976). A one-year survey of mould growth inside twelve homes. *Annals of Allergy,* 36, 30.

330. Hiryama, T. (1983). Passive smoking and lung cancer, nasal sinus cancer, brain tumour and ischemic heart disease. In W. F., Frecker, R.C., Nostbakken Forbes (eds.), *Proceedings of the Fifth World Conference on Smoking and Health.* Ottawa. (pp. 137-141). : Canadian Council on Smoking and Health.

331. Hodges, G. R., Fink, T.N., Schlueter, D.P. (1974). Hypersensitivity pneumonitis caused by a contaminated cool- mist vaporiser. *Annals of Internal Medicine,* 85, 501-504.

332. Hodgson, A. A. (1985). *Alternatives to asbestos products.* : Anjalena Publications Ltd.

333. Hoffman, D. B., K.D., Adama, J.D., Haley, N.J. (1984). Indoor air pollution by tobacco smoke: model studies on the uptake by nonsmokers. In B. L. T., Sundell, J., eds Berglund, *Indoor air. Vol.2. Radon, passive smoking, particulates and housing epidemiology.* Stockholm. (pp. 313-318). : Swedish Council for Building Research.

334. Hoiberg, A. (1982). Occupational stress and illness incidence. *Journal of Occupational Medicine,* 24(6), 445-451.

335. Hollowell, C. D., Miksch, R.R. (1978). *Sources and concentrations of organic compounds in indoor environments.* New York: Academy Med.

336. Holtzman, M. I. (1979). Effect of ozone on bronchial reactivity in atopic and nonatopic subjects. *American Review of Respiratory Disease,* 120, 1059-1067.

337. Hopkinson, R. G. (1963). *Architectural physics: Lighting.* London, U.K.: H.M. Stationary Office.

338. Horejsi, M. S., J., Tomikova, A., Mecl., A. (1960). A syndrome resembling farmers lung in workers inhaling spores of asperglus and penicillia mould. *Thorax,* 15, 212.

339. Horvath, S. M. (1981). Adaption to ozone: duration of effect. *American Review of Respiratory Disease,* 123, 496-499.

340. Horvath, S. M. (1979). Pulmonary function and maximum exercise responses following acute ozone exposure. *Aviation, Space and Environmental Medicine,* 40, 901-905.

341. Howe, G. R., Lindsay, J.P. (1983). A follow-up study of a ten-percent sample of the Canadian Labour Force. I. Cancer mortality in males 1965-73. *Journal of the National Cancer Institute,* 70, 37-44.

342. Hultqvist, B. (1956). *Studies on naturally occurring ionizing radiations.* Stockholm: Almqvist & Wiksells Boktryckeri AB.

343. Hunt, J. W. (1971). Job satisfaction and organisation. In *Proceedings of Seminar on Job Satisfaction.* New South Wales, Australia. : Division of Occupational Health and Pollution Control, Department of Health.

344. Hydrogen Sulfide. (1981). Environmental Health Criteria No. 19. Geneva: World Health Organisation.

345. ILO (1985). *Visual display units: job content and stress at work.* Geneva: International Labour Organisation.

346. ILO/WHO. (1984). Psychosocial factors at work: Recognition and control. In *Occupational Safety and Health Series No.56.* Geneva: Joint ILO/WHO Committee.

347. Ingren, A. A., T., Anshelm Olson, B., Gamberale, F. (1986). Experimental exposure to toluene in combination with ethanol intake. Psychophysiological functions. *Scandinavian Journal Work Environmental Health,* 12, 128-136.

348. Ingren, A. (1986). Subjective and objective signs of organic solvent toxicity among occupationally exposed workers. *Scandinavian Journal of Work Environmental Health,* 12(269), 469-475.

349. Int Hout, L. (1984). Tight building syndrome: is it hot air? *Heating/Piping/Air Conditioning,* January, 99-103.

350. Introna, F. (1966). L'asma bronchiale allergica come malatta professionale. *Minerva Med,* 86, 176-181.

351. Ising, H. S., C. (1982). The effects of infra-sound on humans. *Zeitschrift fur Larmebekampfing,* 29, 79-82.

352. Ising, H. M., D., Shenoda, F.B., Schwarze, C. (1982). *Infra- sound effects on humans.* Dusseldorf: HdA-Reihe, VDI-Verlag.

353. Israel, H. (1940). *Radioaktivitat, I. Grunlagen und Messmethoden.* Leipzig: J. Ambrosius Barth.

354. Itten, J. (1970). *The elements of color.* New York: Van Nostrand.

355. Izmerov, N. F. (1971). Establishment of air quality standards: Definition of the first effects of air pollution. *Archives of Environmental Health,* 22, 711-719.

356. Jackson, C. (1980). *Color me beautiful.* New York: Ballantine Books.

357. Jackson, S. E., Maslach. (1981). The measurement of experienced burnout. *Journal of Occupational Behaviour,* 2(2), 99-113.

358. Jakab, G. I. (1980; 56: 847-856). *Nitrogen dioxide-induced susceptibility to acute respiratory illness: a perspective.* New York: Acad Med.

359. Jarvis, M. J., Russell, M.A. (1984). Measurement and estimation of smoke dosage to non-smokers from environmental tobacco smoke. *European Journal of Respiratory Diseases*,65(Suppl 133), 68-75.

360. Jarvis, M. J., Russell, M.A., Fyerabend, C. (1985). Passive exposure to tobacco smoke: saliva cotinine concentrations in a representative population sample of non-smoking schoolchildren. *British Medical Journal*, 291, 927-929.

361. Johnson, C. L., Bernstein, I.L., Gallagher, J.S., Bonaventre, P.F., Brooks, S.M. (1981). Familial hypersensitivity pneumonitis induced by Bacillus subtilis. *American Review of Respiratory Diseases*, 455, 1977.

362. Johnston, R. (October 1970). Another union. *Journal of Industrial Relations*, 30-43.

363. Johnston, R. (1973). Some characteristics of workers in the building industry. *Journal of Industrial Relations*, 15, 105- 107.

364. Johnston, R. C., J.C. (1970). Workers' attitudes to their jobs. *Economic Activity*, October, 37-47.

365. Jolly, J. R. (1985). Legionella and domestic water heaters in the Quebec City area. *Canadian Medical Association Journal*, 132, 160.

366. Jonassen, N. (1983). Determination of exhalation rates. *Health Physics*, 45, 2.

367. Jonassen, N. (1982). Radon daughter levels in indoor air, effect of filtration and circulation. *Progress report II.* : Laboratory of Applied Physics, Technical University of Denmark.

368. Jones, D. P., O'Connor, S.A., Colins, J.V., Watson, B. (1976). Effects of long-term ionised air treatment on patients with bronchial asthma. *Thorax*, 31, 428-432.

369. Jones, J. W. (1978). Adverse emotional reactions of nonsmokers to secondary cigarette smoke. *Environmental Psychological Nonverbal Behaviour*, 3, 125-127.

370. Kalimo, R. (1980). Stress in work: Conceptual analysis and a study on prison personnel. *Scandinavian Journal of Work Environment Health*, 6(Supplement 3), 148.

371. Kandarjian, L. (1988). Federal policy options for indoor air pollution from combustion appliances. J. R., Ed. Harper, *Transactions of an International Specialty Conference: Combustion Processes and the Quality of the Indoor Environment.* Niagara falls, New York, 310-332.

372. Kangere, A. (1969). *Colour and personality.* York Beach: Samuel Weiser, Inc.

373. Kasl, S. (1979). Epidemiological contributions to the study of work stress. In C. L., Payne, R. Cooper (eds.), *Stress at Work.* Chichester, New York: John Wiley and Sons.

374. Kasl, S. (1973). Mental health and work environment: An examination of the evidence. *Journal of Occupational Medicine,* 15(6), 509-518.

375. Kaune, W. T., Stevens, R.G., Callahan, N.J. (1987). Residential magnetic and electric fields. *Biomagnetics,* 8, 315-335.

376. Kay, J. P. (1831). Observations and experiments concerning molecular irritation of the lungs as one source of tubercular consumption; and on spinners phthisis. *North England Med. Surgical Journal,* 1, 348-363.

377. Kazakevics, A. A. R., Spedding, D.J. (1979). The rate of formaldehyde emission from chipboard. *Horzforschung,* 33, 155-1588.

378. Keller, G. F., K.H., Muth, H. (1983). Method for the determination of Rn-222 (radon) and Rn-220 (thoron) - exhalation rates using alpha-spectroscopy. *Rad. Prot. Dos. 3,* 1(2), 83.

379. Keller, M. D., Lanese, R.R., Mitchell, R.J., Cote, R.W. (1979). Respiratory illness in households using gas and electricity for cooking. I. Survey of incidence. *Environ Res,* 11, 495-503.

380. Kern, R. A. (1938). Asthma due to sensitivity to mushroom fly (Aphiochaeti agarici). *Journal of Allergy,* 9, 604.

381. Kingston, D. W., D.C. (1969). Isolation of amoebae from the air. *Journal Medical Microbiology,* 2, 27.

382. Kleinman, G. D., Horstman, S.W. (1982). Health complaints attributed to the use of carbonless copy paper. *American Industrial Hygiene Association Journal,* 43, 432-435.

383. Knoth, A. B., H., Schmidt, F. (1983). Passivrauchen als Lungenkrebsursache bei Nichtraucherinnen. *Med Klin,* 2, 66-69.

384. Kobayashi, S. (1974). Occupational asthma due to inhalation of pharmacological dusts and other chemicals with some reference to other occupational asthma in Japan. In Y. Yamamura (ed.), *Allergology.* (pp. 124-132). Amsterdam: Excerpta Medica.

385. Kolb, W. (1974). Influence of building materials on the radiation dose to the population. *Kernenegie Offentlichkeit,* 4, 18.

386. Kotlarska, H. (1956). Essential hypertension and profession (translation). *Polish Archives of Internal Medicine,* 26(497),

387. Kozak, P. P., Jr., Gallup, J., Cummins, L.H., Gillman, S.A. (1978). Factors of importance in determining the prevalence of indoor molds. *Journal of Allergy and Clinical Immunology,* 61, 185.

388. Kraemer, S. A. Partners (1977). *Open-Plan Offices. New Ideas, Experiences and Improvements.* London: McGraw-Hill.

389. Kreis, K.K., Hodgson, M.J. (1984). Building associated epidemics. *Indoor Air Quality,* Chptr 6, 87-106.

390. Kreiss, K. K., Hodgson, M.J. (1984). *Building associated epidemics.* Boca Raton, Florida: CRC Press. (87-106)

391. Kreiss, K. G., M.G., Conright, K.L., Scheere, A.R. (1982). Respiratory irritation due to carpet shampoo: two outbreaks. *Environment International,* 8, 337-341.

392. Kreuger, A. P. (1972). Are air ions biologically significant? A review of a controversial subject. *International Journal of Biometerology,* 16, 313-322.

393. Kreuger, A. P. (1973). Are negative ions good for you? *New Scientist (London),* 58(850), 668-669.

394. Kreuger, A. P., Reed, E.J. (1976). Biological impact of small air ions. *Science,* 193, 1209-1213.

395. Kreuger, A. P., Smith, R.F. (1960). Negative air ion effects on the concentration and metabolism of S hydroxtriptamine in the mammalian respiratory tract. *Journal of General Physiology,* 44, 269-276.

396. Krisiuk, E. M., Tarasove, S.I., Shamov, V.P., Shalak, N.I., Lisachenko, E.P., Gomelsky, L.G. (1974). A study of radioactivity in building materials. *National Committee on the Utilization of Atomic Energy, Leningrad,*

397. Kroes, W. H. (1974). Job stress: An unlisted occupational hazard. *Journal of Occupational Medicine,* 16(10), 659-661.

398. Kroling, P. (1987). Untersuchungen zum 'Building Illness' - Syndrom in klimatisierten Gebauden. *Gesundheits Ingenieur Haustechnik Bauphysik Umwelttechnik,* 108,

399. Krueger, A. P., Reed, E.J. (1976). Biological impact of small air ions. *Science,* 193(24 September), 1209-1213.

400. Kubzansky, P. E., Leiderman, P.H., Mendleson, J., Solomon, P., Wexler, D. (1961). Sensory deprivation: an overview. In P. Solomon (ed.), *Sensory Deprivation.* (pp. 221-238). Cambridge, Mass.: Harvard University Press.

401. Kurppa, K. H., P., Rautala, K., Turminen, T. (1984). Birth defects and video display terminals. *Lancet,* ii, 1339.

402. Kurtz, J. B. (1988). Legionella Pneumophila. *Annals of Occupational Hygiene,* 32(1), 59-61. (Massey)

403. Kyerematen, G. A., Damiano, M.D., Dvorchik, B.H., Vessel, E.S. (1982). Smoking-induced changes in nicotine disposition: application of a new HPLC assay for nicotine and its metabolites. *Clinical Pharmacol Ther,* 32, 769-780.

404. Lacey, J. (1977). Microorganisms in the air of cotton mills (letter). *Lancet,* 2, 455.

405. Lagerwerff, J. M. (1963). Prolonged ozone inhalation and its effect on visual parameters. *Aerospace Medicine,* 34, 479-486.

406. Latham, R. E., ed. (1958). *The travels of Marco Polo.* London:-Penguin Books.

407. Lea, D. E. (1955). *Actions of radiations on living cells.* London: Cambridge University Press.

408. Lebowitz, M. D., Knudson, R.J., Burrows, B. (1984). Family aggregation of pulmonary function measurements. *American Review of Respiratory Diseases,* 129, 8-11.

409. Lebowitz, M. D., Burrows, B. (1976). Respiratory symptoms related to smoking habits of family adults. *Chest,* 69(48),

410. Lee, T. C., Stout, J.E., Yu, V.L. (1988). Factors predisposing to legionella pneumoplilia colonization in residential water systems. *Arch Environmental Health,* 43, 59- 62.

411. Leedom, J. M., Loosli, C.G. (1979). Airborne pathogens in the indoor environment with special reference to nonsomial infections. In R. Edmonds (ed.), *Aerobiology: The ecological systems approach.* (pp. Chapter 6). Stroudsburg, Pa.: Dowden, Hutchinson and Ross.

412. Levi, L. (1971). Society, stress and disease. In *The psychosocial environment and psychosomatic diseases.* 1 London: Oxford University Press.

413. Levi, L. (1972). Stress and distress in response to psycho- social stimuli. *Acta Medica Scandinavia,* 191,

414. Levin, H.E. (1989). Sick building syndrome: review and exploration of causation hypotheses and control methods. In *The Human Equation: Health and Comfort, Proceedings of ASHRAE/SOEG Conference IAQ '89.* San Diego, California. : American Society of Heating, Refrigeration and Air Conditioning Engineers, Inc., Atlanta Georgia.

415. Levin, H. E. (1989b). Specifying interior materials. *Indoor Air Quality Update,* 2(4), 5-11.

416. Lewis, H. E., Foster, A.R., Mullan, B.J., Cox, R.N., Clarke, R.P. (1969). Aerodynamics of the human micro-environment. *Lancet,* 1, 1273.

417. Libby, W. L., Lacey, B.C., Lacey, J.I. (1973). Pupillary and cardiac activity during visual attention. *Psychophysiology,* 10, 270-294.

418. Libenskind, A. (1971). Diagnostic value of culture procedures and provocation tests in suspected mould allergies. *Acta Allergol.,* 26, 106.

419. Lin, R. S., Dischinger, P.C., Conde, J., Farrell, K.P. (1985). Occupational exposure to electromagnetic fields and the occurrence of brain tumours: an analysis of possible associations. *Journal of Occupational Medicine,* 27, 413-419.

420. Linn, W. S. (1985). Effects of exposure to 4 ppm nitrogen dioxide in healthy and asthmatic volunteers. *Archives of Environmental Health,* 40, 234-239.

421. Linn, W. S. (1978). Health effects of ozone exposure in asthmatics. *American Review of Respiratory disease,* 117, 835- 843.

422. Linn, W. S. (1983). Response to ozone in volunteers with chronic obstructive pulmonary disease. *Archives of Environmental Health,* 38, 278-283.

423. Lowry, T. S., L. (1956). Silo-filler's disease - a syndrome caused by nitrogen dioxide. *JAMA,* 62, 153.

424. Lucas, H. F. (1957). Improved low-level alpha-scintillation counter for radon. *Rev. Sci. Institute,* 28, 68.

425. Luck, W. N., H. (1985). Nicotine and cotinine concentrations in serum and urine of infants exposed via passive smoking or milk from smoking mothers. *Journal of Paediatrics,* 107, 816- 820.

426. Lumpkins, E. D., Sr., Corbit, S. (1976). Airborne fungi survey. II. Culture plate survey of the home environment. *Annals of Allergy,* 36, 40.

427. Luscher, M. (1969). *The Luscher colour test.* New York: Random House.

428. Maslow, A. H. (1954). *Motivation and personality.* New York: Harper and Row.

429. Magora, A. (1972). Investigation of the relationship between low-back pain and occupation. 3. Physical requirements: sitting, standing and weight lifting. *Industrial Medicine,* 41, 5-9.

430. Malmstroem, T. G., Ahlgren, A. (1982). Efficient ventilation of office rooms. *Environment International,* 8, 401-408.

431. Mansfield, L. E., Nelson, H.S. (1982). Allergins in commercial house dust. *Annals of Allergy,* 48, 205.

432. Mant, D. C., Muir-Gray, J.A. (1986). *Building regulation and health.* UK: British Research Establishment.

433. Mant, D. C., Muir Gray, J.A. (1992). Building regulation and health. Garston: Building Research Establishment.

434. Margolis, B. L., Kroes, W.H., Quinn, R.P. (1974). Job stress: an unlisted occupational hazard. *Journal of Occupational Medicine,* 16, 659-661.

435. Markinovitch, V. A., Hill, A. (1975). Hypersensitivity alveolitis. *JAMA,* 231, 944.

436. Marks, J. G. (1980). Allergic contact dermatitis from carbonless copy paper. *JAMA,* 245, 2331-2333.

437. Marks, J. G., Trautelein, J.J., Zwillich, C.W., Demers, L.M. (1984). Contact uricaria and airways obstruction from carbonless copy paper. *JAMA,* 252, 1038-1040.

438. Martin, H. D., Radford, R.D., Lea, K.R. (1971). A shock free carpet system at 10% relative humidity. *Modern Textiles,* April, 76-79.

439. Martin, T. R., Bracken, M.D. (1986). Association of low birth weight with passive smoke exposure in pregnancy. *American Journal of Epidemiology,* 124, 633-642.

440. Mathews, K. P. (1978). Aerobiology and inhalant allergens. II. Other inhalant allergens. In E. J., Reed, C.E., Ellis, E.F. Middleton (eds.), *Allergy: Principles and Practice.* St. Louis: C.V. Mosby.

441. Matthews, R. (1985). Pollution begins at home. *New Scientist,* 108, 34-37.

442. Maunsell, K. (1952). Airborne fungal spores before and after raising dust. *International Archives of Allergy,* 3, 93.

443. McDonald, J. C., Arhirii, M., Armstrong, B., Bernard, J., Cherry, N.M., Cyr, D., Faran, J.P., McKenna, T., McKinnon, D. (1986). Building illness in a large office complex. In D. S. Walkinshaw (ed.), *Indoor Air Quality in Cold Climates: Hazards and Abatement Measures.* (pp. 7-22). Ottawa: ACPA.

444. McDonnell, W. F. (1985). Reproducibility of individual responses to ozone exposure. *American Review of Respiratory Disease,* 131, 36-40.

445. McDowall, M. E. (1986). Mortality of persons resident in the vicinity of electricity transmission facilities. *British Journal of Cancer,* 53, 271-279.

446. McLaughlin, J. P., Jonassen, N. (The effect of pressure drops on radon exhalation from walls). (1980). In *Natural Radiation Environment III, Volume 2.* (conf-780422). : Department of the Environment.

447. McLaughlin, J. P. (1982). A passive integrating method of measuring relative radon and thoron exhalation rates. In *Procedures of a Specialist Meeting on the Assessment of Radon and Daughter Exposure and Related Biological Effects.* Rome. University of Utah, USA: RD Press.

448. McLellan, R. K. (1983). The hazards of office work. *Toxic Substances Journal,* 5(3), 162-181.

449. McNall, P. E., Persily, A.K. (1984). Ventilation concepts for office buildings. *Annals of American Conference Government Industrial Hygienists,* 10, 49-58.

450. McReynolds, P. A. (1956). A restricted conceptualization of human anxiety and motivation. *Psychol. Reports,* 2, 292-312.

451. Mead, M. (1950). *Sex and temperament in three primitive societies.* New York: Mentor.

452. Medical Research Council Symposium. (1977). Humidifier fever. *Thorax,* 32, 653-663.

453. Meister, D. (1967). Applications of human reliability to the production process. In W. B. Askren (ed.), *Symposium on Reliability of Human Performance in Work.* report AMRI-TR-67- 88 Wright-Patterson Airforce Base: Aerospace Medical Laboratories.

454. Melia, R. J. W., Florey, C du V., Altman, D.G., Swan, A.V. (1977). Association between gas cooking and respiratory disease in children. *British Medical Journal,* ii, 149-152.

455. Melia, R. J. W., Florey, C du V., Morris, R.W. (1982). Childhood respiratory illness and the home environment. II. Association between respiratory illness and nitrogen dioxide, temperature and relative humidity. *International Journal of Epidemiology,* 11,

456. Melia, R. J. W. (1979). The relation between respiratory illness in primary schoolchildren and the use of gas for cooking, I. results from a national study. *International Journal of Epidemiology,* 8, 333-338.

457. Melia, R. J. W., Florey, C du V., Sittampalam, Y., Watkins, C. (1981). The relation between respiratory illness in infants and gas cooking in the U.K.: A preliminary report. In *Proceedings of the VI world congress on air quality.* Paris. : International Union of Air Pollution Prevention Associations.

458. Melia, R. J. W. (1983). The relation between respiratory illness in infants and gas cooking in the UK: a preliminary report. In *Proceedings of the VIth World Congress on Air Quality.* Paris.

459. Mella, D. L. (1981). *Colour power, the language of colour.* Albuquerque: Domel Artbooks.

460. Metzger, W. J., Patterson, R., Fink, J., Semerdjam, R., Roberts, M. (1976). Sauna-takers disease. *Journal of the American Medical association,* 236, 2209-2211.

461. Meyer, B. (1985). Formaldehyde release from building products. B. Berglund. Lindvall, T., Sundell, J., eds., *3rd International Conference on Indoor Air Quality and Climate.* Vol 3: Sensory and hyperreactive reactions to sick buildings Stockholm. (pp. 29-34). : Swedish Council for Building Research.

462. Meyer, B. (1983). *Indoor air quality.* Reading, MA: Addison- Wesley.

463. Milham, S. (1982). Mortality from leukaemia in workers exposed to electrical and magnetic fields. *New England Journal of Medicine,* 307, 249.

464. Milham, S. (1985). Silent keys: leukaemia mortality in amateur radio operators. *Lancet,* 1, 811.

465. Miller, G. H. (1984). Cancer, passive smoking and nonemployed and employed wives. *West Journal of Medicine,* 140, 632-635.

466. Miller, M. M., Patterson, R., Fink, J., Roberts, M. (1976). Chronic hypersensitivity lung disease with recurrent episodes of hypersensitivity pneumonitis due to a contaminated central humidifier. *Clinical Allergy,* 6, 451.

467. Miller, M. W. (1980). Re: Electrical wiring configurations and childhood cancer. *American Journal of Epidemiology,* 112, 165-167.

468. Mitchell, W. R., Wharton, G.W., Larson, D.G., Modic, R. (1969). House dust mites and insects. *Annals of Allergy,* 27, 93.

469. Miyamoto, T. O., S., Ishizaki, T,. (1969). Antigenic reaction between house dust and dust mite (Dermatophagoides farinae hughes, 1961) by a fraction method. *Journal of Allergy,* 44, 282.

470. Molhave L., B. B., Pedersen, O.F. (1986). Human reactions to low concentrations of volatile organic compounds. *Environment International,* 12, 167-175.

471. Molhave, L. B., B., Pedersen, O.F. (1985). Human reactions during controlled exposures to low concentrations of organic gases and vapours known as normal indoor air pollutants. In *Indoor Air: Sensory and Hyperactivity Reactions to Sick Buildings - Proceedings of the 3rd International Conference on Indoor Air Quality and Climate.* Stockholm. Swedish Council for Building Research.

472. Molhave, L. (1986). Indoor air quality in relation to sensory irritation to volatile organic compounds. *ASHRAE Transactions,* 92(1a), 306-316.

473. Molhave, L. (1987). The sick building - a subpopulation among problem buildings. In *Indoor Air '87, Proceedings of the 4th International Conference on Indoor Air Quality and Climate.* 2 Berlin. (pp. 469-473). : Institute for Water, Soil and Air Hygiene.

474. Molhave, L. (1985). Volatile organic compounds as indoor air pollutants. In R. B., Kaye, S.V. Gammage (eds.), *Indoor Air and Human Health, Proceedings of the Seventh Life Sciences Symposium.* Knoxville, Tennessee. (pp. 403-414). Chelsea, Michigan: Lewis Publishers, Inc.

475. Molhave, L. (1990). Volatile organic compounds, indoor air quality and health. In *Proceedings of the 5th Conference on Indoor Air Quality and Climate.* 5 Toronto, Canada. (pp. 15- 33). Ottawa: Canada Mortgage and Housing Corporation.

476. Molina, C. P., C.A.C., Valbjorn, O., de Bortoli, M. (1989). Sick building syndrome - a practical guide. *COST 613 Report No 4.* Brussels/Luxembourg: Commission of the European Communities.

477. Moos, R. V. Dort, B. (1977). Physical and emotional symptoms and campus health centre utilization. *Social Psychiatry,* 12, 107-115.

478. Morey, E. R. (1984). Environmental studies in mouldy office buildings: biological agents, sources and preventative measures. *Annals of American Conference Government Industrial Hygienists,* 10, 21-35.

479. Morey, P. R. (1988). Microorganisms in buildings and HVAC systems: a summary of 21 environmental studies. In *Engineering Solutions to Indoor Air Problems, Proceedings of the ASHRAE Conference IAQ '88.* Atlanta, Georgia. : American Society of Heating, Refrigeration and Air-Conditioning Engineers, Inc., Atlanta, Georgia.

480. Morey, P. R., Jenkins, B.A. (1989). What are typical concentrations of fungi, total volatile organic compounds, and nitrogen dioxide in an office environment? In *Proceedings of the ASHRAE/SOEG Conference IAQ '89.* San Diego, California. : American Society of Heating, Refrigeration and Air Conditioning Engineers, Inc., Atlanta, Georgia.

481. Morgan, A. (1969). Physical behaviour of radon and its daughters with particular reference to monitoring methods. In H. A. Shapiro (ed.), *Proceedings of the International Conference.* Johannesburg. (pp. 540-543). Cape Town: Oxford University Press.

482. Morgan, M. S., Camp, J.S. (1986). Upper respiratory tract irritation from controlled exposure to vapour from carbonless copy forms. *Journal of Occupational Medicine,* 28, 41-49.

483. Morrow, P. E. (1984). Toxicological data on NO2: an overview. *Journal Toxicol Environ Health,* 13, 205-227.

484. Moser, C. A., Kalton, G. (1971). *Survey methods in social investigation.* London: Heineman Educational.

485. Muchinsky, P. M. (1977). Employee absenteeism: A review of the literature. *Journal of Vocational Behaviour,* 10(3), 316- 340.

486. Mueller Associates, I. S. Corporation, and Brookhaven National Laboratory. (1985). *Indoor Air Quality Environmental Information Handbook: Combustion Sources.* (DOE/EV/10450-1). Washington, D.C.: U.S. Department of Energy.

487. Mueller Associates, I. A. National Laboratory, and Brookhaven National Laboratory. (1987). *Indoor Air Quality Environmental Information Handbook: Building System Characteristics.* (DOE/EV/10450-H1). Washington, D.C.: U.S. Department of Energy.

488. Murphy, J. P., Gellhorn, E. (1945). The influence of hypothalamic stimulation on cortically induced movements and on action potentials of the cortex. *Journal Neurophysiology,* 8, 341-364.

489. Murray, R. (1990). Asbestos: A chronology of its origins and health effects. *British Journal of Industrial Medicine,* 47, 361-365.

490. Murrell, K. F. H. (1965). *Ergonomics.* London: Chapman & Hall.

491. National Centre for Health Statistics. (1977). Acute conditions: Incidence and associated disability. *Series 10, Number 11* Washington D.C.: U.S. Government Printing Office.

492. National Centre for Health Statistics. (1979a). Acute conditions: Incidence and associated disability, United States, July 1977-June 1978. *Series 10, Number 132* Washington D.C.: U.S. Government Printing Office.

493. National Academy of Science (NAS) (1985). *Building diagnostics: a conceptual framework.* Washington, D.C.: National Academy Press.

494. National Centre for Health Statistics. (1981a). Patients' reasons for visiting physicians: National ambulatory care survey United States, 1977-1978. *Series 13, Number 56* Washington D.C.: U.S. Government Printing Office.

495. National Centre for Health Statistics. (1979b). Physician visits: Volume and interval since last visit, United States - 1975. *Series 10, Number 128* Washington D.C.: U.S. Government Printing Office.

496. National Centre for Health Statistics. (1967). Prescribed and nonprescribed medicines. (Series 10, Number 11). Washington D.C.: U.S. Government Printing Office.

497. National Centre for Health Statistics. (1970). Selected symptoms of psychological distress. (Report 11, Number 37). Washington D.C.: U.S. Government Printing Office.

498. National Centre for Health Statistics. (1979c). Use habits among adults of cigarettes, coffee, aspirin, and sleeping pills. *Series 10, Number 131* Washington D.C.: U.S. Government Printing Office.

499. National Research Council, C. O. Passive Smoking. (1986). Environmental tobacco smoke: measuring exposures and assessing health effects. Washington D.C.: National Academy Press.

500. National Research Council, Committee on Indoor Pollutants (1981). *Indoor pollutants.* Washington DC: National Academy Press.

501. National Research Council, Committee on Medical and Biologic Effects on Environmental Pollutants, Subcommittee on Nitrogen Oxides. (1976). Nitrogen oxides. Washington DC: National Academy of Sciences.

502. National Research Council, N. A. of Science. (1981). Indoor pollutants. Washington DC: National Academy Press.

503. Naylor Dana Institute. (1973). Analysis of mainstream and sidestream smoke of cigarettes treated with colite. *Internal report,* November,

504. Nazaroff, W. W., Nero, A.V., Eds. (1988). *Radon and its decay products in indoor air.* New York: Wiley.

505. Neal, P. A., Schneiter, R., Caminita, B.H. (1942). Report on acute illness among rural mattress makers using low grade, stained cotton. *Journal American Medical Association,* 119, 1074-1082.

506. Neill, M. A., Gorman, G.W., Gilbert, C., Roussel, A., Hightower, A., McKinney, R.M., Broome, C.W. (1985). Nosocomial legionellosis, Paris, France. *American Journal of Medicine,* 78, 581-588.

507. Nelson, C. J., Clayton, C.A., Wallace, L.A., Highsmith, V.R., Kollander, M., Bascom, R., Leaderer, B.P. (1991). EPA's indoor air quality and work environment survey: relationships of employees' self-reported health symptoms with direct indoor air quality measurements. *Proceedings of IAQ '91 - Healthy Buildings.* Washington DC. (pp. 22-32). Atlanta: ASHRAE.

508. Nelson, T. R., B.Z., Welker, W.H. (1933). The effect of air filtration in hay fever and pollen asthma. *JAMA,* 100, 1385.

509. Neurath, G. a. Horstmann, H. (1973). Einfluss des feuchtigkeitsgehaltes glutzonentemperatur. *Beitr. Tabak- forsch,* 2(93),

510. Nicholson, A. S., Ridd, J.E. (1988). *Health Safety and Ergonomics.* Great Britain: Butterworths.

511. Nilsen, A. (1982). Facial rashes among video-display unit operators. *Contact Dermatitis,* 8, 25-28.

512. NIOSH. (1979). Hazard evaluation and technical assistance report. (TA 78-69). Cincinnati, Ohio: National Institute for Occupation Safety and Health. January.

513. Nitschke, I. A., Wadach, J.B., Clarke, W.A., Traynor, G.W., Adams, G.P., Rizzuto, J.E. (1984). A detailed study of inexpensive radon control techniques in New York state houses. In B. Berglund, Lindvall, T., Sundell, J. (eds.), *Indoor Air: Radon, Passive Smoking, Particulates and Housing Epidemiology.* Stockholm: Swedish Council for Building Research.

514. Nordstrom, S. B., E., Gustavsson, L. (1986). Reproductive hazards among workers at high voltage substations. *Bioelectromagnetics,* 7, 91-101.

515. Norusis, M. J. (1990). *SPSS/PC+Statistics 4.0.* Chicago, Illinois: SPSS Inc.

516. Nyblom, L. (1980). Radon in the air in buildings where heat is accumulated in stone magazines. Geilo, Norway: Nordic Society for Radiation Protection.

517. Nyman, K. R., R. (1978). Absence from work and their determinants in Finland. (Reports of the Social security Institution, A14). Helsinki: Research Institute for Social Security.

518. O'Hanlon, J. F. (1981). Boredom: Practical consequences and a theory. *Acta Psychologica,* 49, 53-82.

519. O'Neill, J. B., R. (1984). The development of a relevant ergonomic checklist for designers of the new technology office. In E. Grandjean (ed.), Ergonomics and Health in Modern Offices. (pp. 141-145). London: Taylor & Francis.

520. O'Neill, M. (1961). *Hailstones and halibut bones.* Garden City: Doubleday and Company.

521. O'Riordan, M. C., Duggan, M.J., Rose, W.B., Bradford, G.F. (1972). *The radiological implications of using by-product gypsum as a building material.* (NRCB-R7). Harwell, Oxon: National Radiological Protection Board.

522. Oettingen, W. F., Neal, P.A., Donahue, D.D. (1942). The toxicity and potential dangers of toluene. *JAMA,* 118, 579- 584.

523. Ogsten, S. A. (1985). The Tayside infant morbidity and mortality study: effect on health of using gas for cooking. *British Medical Journal,* 290, 957-960.

524. Ogston, S. A., Florey, C., Walker C.H.M. (1985). The Tayside infant morbidity and mortality study: effect on health of using gas for cooking. *British Medical Journal,* 290, 957-960.

525. Olin, R. V., D., Ahlbom, A. (1985). Mortality experience of electrical engineers. *British Journal of Industrial Medicine,* 42, 211-212.

526. Oliver, T. (1902). *Dangerous trades.* London: Murray.

527. Olsen, E. S., L. (March 1990). Solvent Syndrome - Epidemiology Study. *Annals of Occupational Hygiene,* 379-386.

528. Olson, C. (1988). Remedies for the sick building syndrome. *Building Design and Construction,* 29(4), 100-104.

529. Orehek, J. (1976). Effect on short-term, low-level nitrogen dioxide exposure on bronchial sensitivity of asthmatic patients. *Journal of Clinical Investigation,* 57, 301-307.

529b. Ott, L., Mendenhall, W., (1985). *Understanding Statistics.* Boston: Duxbury Press.

530. Owen, M. K., Ensor, D.S., Sparks, L.E. (1990). Airborne particle sizes and sources found in indoor air. *Proceedings of the 5th International Conference on Indoor Air Quality and Climate.* 2 Toronto, Canada. (pp. 79-84). Ottawa: Canada Mortgage and Housing Corporation.

531. Ozkaynak, H. R., P.B. (1987). Sources and emission rates of organic chemical vapours in homes and buildings. *Indoor Air '87: Proceedings of the 4th International Conference on Indoor Air Quality and Climate.* Berlin. : Institute for Water, Soil, and Air Hygiene.

532. Parkes, W. R. (1982). *Occupational lung disorders.* London: Butterworths.

533. Parkinson, D. K., Grennan, M.J. (1986). Acute-onset sarcoidosis presenting as workplace-related asthma. *American Journal of Industrial Medicine,* 9, 243-246.

534. Patissier, P. (1822). *Traite des maladies des artisans.* Paris.

535. Pattishall E.N., S. G. L., Etzel, R.A., Helms, R.W., Haley, N.J., Denny, F.W. (1985). Serum cotinine as a measure of tobacco smoke exposure in children. *American Journal Dis Child,* 139, 1101-1104.

536. Payne, R. (1980). Organizational stress and social support. In C. L., Payne, R. Cooper (eds.), *Current Concerns in Occupational Stress.* New York: John Wiley and Sons.

537. Pearce, N. R., J., Fraser, J. (1989). Case-control studies of cancer in New Zealand electrical workers. *International Journal of Epidemiology,* 18, 1-5.

538. Pearson, J. J., G.E. (1966). Soil concentrations of emanating Ra-226 and Rn-222 from soils and plants. *Tellus,* 18, 655.

539. Pejtersen, J. B., P., Kondo, H., Clausen, G., Fanger, P.O. Air pollution sources in ventilation systems. In *CLIMA 2000.* Sarajevo. 139-144.

540. Pejtersen, J. O., L., Skar, S., Clausen, G.H., Fanger, P.O. (1990). Simple method to determine the olf load in a building. In *Indoor Air '90.* 1 Toronto. (pp. 537-542).

541. Peltz, L. R. (1980). *Fashion: Colour , line and design.* Indianapolis: Bobbs-Merrill Educational Publishing.

542. Pennebaker, J. W., Brittingham, G.L. (1982). Environmental and sensory cues affecting the perception of physical symptoms. In *Advances in environmental psychology.4,* Hillsdale N.J.: Earlbaum, 115-136.

543. Pennebaker, J. W., Burnham, M.A., Schaeffer, M.A., Harper, D. (1977). Lack of control as a determinant of perceived physical symptoms. *Journal of Personality and Social Psychology,* 35, 167-174.

544. Pennebaker, J. W., Skelton, J. (1978). Psychological parameters of physical symptoms. *Personality and Social Psychology Bulletin,* 4, 524-530.

545. Pensko, J. S., Z., Blaton-Albricka, K. (1980). Emanating power of radon-222 measured in building materials. In T. F., Lowder, W.M. Gesell (eds.), *Natural Radiation Environment III.* CONF-780422 (pp. 1407). : U.S. Dept of Energy.

546. Pepys, J. W., I.D., D'Souza, M.F., Greenberg, M. (1973). *Clinical and immunological responses to enzymes of Bacillus subtilis in factory workers and consumers.* Clinical Allergy 3.

547. Perry, F. S., Reichmanis, M., Mario, A.A., Becker, R.O. (1981). Environmental power-frequency magnetic fields and suicide. *Health Physics,* 41, 267-277.

548. Pestalozzi, C. (1959). Febrile gruppener krankungen in einer modellschreinerei durch inhalation von mit schimelpilzen kontaminiertem befeuchterwasser (befeucterfieber). *Schweiz Med. Wschr.*, 89, 710-713.

549. Pickering, C. A. C., Moore, W.K.S., Lacey, J., Holford- Strevens V.C., Pepys, J. (1976). Investigation of respiratory disease associated with an air conditioning system. *Clinical Allergy*, 6, 109-118.

550. Pohl, E. P., J. (1976). Determination of environmental or occupational Rn-222 in air and water and Ra-226 in water with feasible and rapid methods of sampling and measurement. *Health Physics*, 31, 343-348.

551. Polk, C. (1974). Sources, propagation, amplitude and temporal variation of extremely low frequency (0-100 Hz) electromagnetic fields. In J. G., Sances, A., Battocletti, J.H. Llaurado (eds.), *Biological and Clinical effects of Low Frequency Magnetic and Electric Fields*. Illinois: Charles C Thomas.

552. Porter, L. W., Steers, R.M. (1973). Organizational, work and personal factors in employee turnover and absenteeism. *Psychology Bulletin*, 80(2), 151-176.

553. Porter, R. W. (1953). Hypothalamic involvement in the pituitary-adrenocortical response to stress stimuli. *American Journal of Physiology*, 172, 515-519.

554. Potter, I. N. (1988). The sick building syndrome. (Technical Note 4/88). Berkshire, U.K.: Building Services Research and Information Association (BSRIA).

555. Preston-Martin S., Y. M. C., Benton, B., Henderson, B.E. (1982). N-nitroso compounds and childhood brain tumours: a case-control study. *Cancer Res*, 42, 5240-5245.

556. Proctor, D. F., Vaeth, M., Wyon, D.P. (1983). Human response to controlled levels of toluene in six-hour exposure. *Scandinavian Journal Work Environmental Health*, 9, 405-418.

557. Proctor, D. F., Anderson, I., Lundqvist, G., Swift, D.L. (1973). Nasal mucociliary function and indoor climate. *Journal of Occupational Medicine*, 15, 169-174.

558. Proctor, D. F., Swift, D.L., Quinlan, M., Salmon, S., Takagi, Y., Evering, S. (1969). The nose and man's atmospheric environment. *Archives of Environmental Health*, 18, 671-680.

559. Putz, V. R. (1976). Effects of CO on vigilance performance: Effects of low level carbon monoxide on divided attention, pitch discrimination and the auditory evoked potential. Cincinatti, OH: U.S. Department of Health, Education and Welfare.

560. Pye, A. M. (1979). Alternatives to asbestos in industrial applications. In L. C., S.S. Michaels (eds.), *Asbestos Volume 1: Properties, Applications and Hazards*. (pp. 339-373). : Wiley Interscience.

561. Pye, A. M. (1986). *Alternatives to asbestos products: A review.* : Health and Safety Commission.

562. Quackenboss J.J., K. M. S., Spengler J.D., Letz R. (1982). Personal monitoring for nitrogen dioxide exposure: methodological considerations for a community study. *Environ International,*x 8, 249-258.

563. Quinlan, P. (1989). Protocol for the comprehensive evaluation of building-associated illness. *Occupational Medicine: State of the Art Reviews,* 4, 771-797.

564. Ramer, E. M., Richards, H.R. (1968). Correlation of the electrical resistivities of fabrics with their ability to develop and hold electrostatic charges. *Textile Research Journal,* January, 28-35.

565. Raper, A. (1958). The incidence of peptic ulceration in some African tribal groups. *Transactions of the Royal Society of Tropical Medicine and Hygiene,* 152, 535-546.

566. Raw, G. J., Fox, T.A. (1990). Condensation, heating and ventilation in small homes. *Building Research Establishment - Pamphlet,* 2/90, (Massey)

567. Raw, G. J., Roys, M.S., Leaman, A. (1989). Further findings from the office environment survey, Part 1: Productivity. (BRE/109/1/9). U.K.: Building Research Establishment.

568. Raw, G. J., Roys, M.S., Leaman, A. (1990). Further findings from the office environment survey: Productivity. In *Indoor Air '90.* Toronto.

569. Raw, G. J., Leinster, P., Thomson, N., Leaman, A., Whitehead, C. (1991). A new approach to the investigation of the sick building syndrome. In *CIBSE National Conference.* Canterbury.

570. Raw, G. J. (1991). Sick Building Syndrome: An Integrated Approach. *Building Research Establishment,* EP236, 1-12. (Massey)

571. Raw, G. R. (1992). Sick building syndrome: a review of the evidence on causes and solutions. *HSE Contract Research report No 42/1992.* U.K.: HMSO.

572. Raw, G. J., Oseland, N.A. (1989). A standard questionnaire for the investigation of sick building syndrome. *Building Research Establishment,* Note N40/89,

573. Reichmanis, M. P., F.S., Marino, A.A., Becker, R.O. (1979). Relation between suicide and the electro-magnetic field of overhead power lines. *Physiol Chem Phys,* 11, 395-403.

574. Reinikainen, L. M., Jaakkola, J.J.K., Heinonen, O.P. (1988). The effect of air humidification on different symptoms in an office building. An epidemiological study. In B. Berglund, Lindvall, T. (eds.), *Proceedings of Healthy Buildings '88.* 3 Stockholm, Sweden. (pp. 207-125). Stockholm: Swedish Council for Building Research.

575. Reinnikainen, L. M., Jaakkola, J.J.K., Helenius, T., Seppanen, O. (1990). The effect of air humidification on symptoms and environmental complaints in office workers. A six period cross-over study. *Proceedings of the 5th International Conference on Indoor Air Quality and Climate.* 1 Toronto, Canada. (pp. 775-780). Ottawa: Canada Mortgage and Housing Corporation.

576. Repace, J. L., Lowrey, A.H. (1980). Indoor air pollution, tobacco smoke, and public health. *Science,* 208(464),

577. Reznikov, M. L., J.H., Dawson, D.J.H. (1971). Investigation by seroagglutination of strains of the Myobacterium intercellular - M.scrofulaccum group from house dusts and sputum in Southeastern Queensland. *American Review of Respiratory Diseases,* 104, 951.

578. Richards, M. (1954). Atmospheric mould spores in- and out-of- doors. *Journal of Allergy,* 25, 429.

579. Rigel, D. S. (1983). Malignant melanoma and exposure to fluorescent light at work. *Lancet,* 1, 704.

580. Rivolier, J. R., Harisson, Y., Zouloumian, P. (1975). Research on the possible action of negative over ionization on healthy humans. *Biometerology,* 6(Suppl. International Journal of biometeorology vol 19), 134.

581. Robertson J. (1991). Ventilation effectiveness: the key to a healthier environment. *Engineering Solutions to Building Sickness.* Sydney, Australia. (pp. 17-25). : BSFA.

582. Robertson, A. B., S. (1986). Building sickness - all in the mind? *Occupational Health,* March,

583. Robertson, A. S., McInnes, M., Glass, D., Dalton, G., Burge, P.S. (1989). Building sickness are symptoms related to office lighting? *Annals of Occupational Hygiene,* 33, 47-59.

584. Robertson, A. S., Burge, P.S., Hedge, A., Sims, J., Gill, F.S., Finnegan, M., Pickering, C.A.C., Dalton, G. (1985). Comparison of health problems related to work and environmental measurement in two office buildings with different ventilation systems. *British Medical Journal,* 291, 373-376.

585. Robertson, A. S., Roberts, K.T., Burge, P.S., Raw, G.J. (1990). The effect of change in building ventilation category on sickness absence rates and the prevalence of sick building syndrome. *5th International Conference on Indoor Air Quality and Climate.* 1 Toronto. (pp. 237-242).

586. Robertson, A. S., Burge, P.S., Weiland, A. (1985). Extrinsic allergic alveolitis due to antigen from a humidifier at 15 degrees C. *Thorax,* 40(3), 229 (abstr).

587. Robertson, A. S., Burge, P.S., Weiland, G.A., Carmalt, M.H.B. (1987). Extrinsic allergic alveolitis caused by a cold water humidifier. *Thorax,* 42(1), 32-37.

588. Robertson, G. (1991). Results of air quality surveys in six hundred and ninety five major buildings across USA, UK, Europe, Scandinavia, Australia, New Zealand and Hong Kong. *Healthy Buildings International,* March,

589. Robinson, N. D., F.S. (1963). The ionisation state of the atmosphere as a function of the meterological elements and of various sources of ions. *International Journal of Biometeorology,* 101-107.

590. Rock, I. (1975). *An introduction to perception.* London: McMillan.

591. Roebuck, J. A., Kroemer, K.H.E., Thomson, W.G. (1975). *Engineering anthropometry methods.* Wiley: New York.

592. Ronk, R. W., M. (1985). Hydrogen sulfide and the probabilities of "inhalation" through a tympanic membrane defect. *Journal of Occupational Medicine,* 27, 337-340.

593. Rose, H. D., Hirsch, S.R. (1979). Filtering hospital air decreases Aspergillus spore counts. *American Review of Respiratory Disease,* 119, 511.

594. Rosen, F. L. and Levy, A. (1950). Bronchial asthma due to allergy to tobacco smoke. *JAMA,* 144(620)

595. Rosenberg J., B. N. L., Jacob, P., Wilson, K.M. (1980). Disposition kinetics and effects of intravenous nicotine. *Clinical Pharmacol Ther,* 28, 517-522.

596. Rosenthal, T. B., E. (1980). Superoxide involvement in negative ion effects. *Nature,* 288, 739.

597. Rosner, B. (1986). *Fundamentals of biostatistics.* Boston: Duxbury Press.

598. Rotton, J. F., J., Barry, T. (1979). The air pollution experience and physical aggression. *Journal Applied Social Psychology,* 9, 379-412.

599. Royal Commission on Matters of Health and Safety Arising from the Use of Asbestos in Ontario. (1984). Toronto, Ontario: Ontario Ministry of the Attorney General.

600. Rozenweig, A. L. (1970). Contaminated humidifiers. *New England Journal of Medicine,* 283, 1056.

601. Rubin, D. H., Leventhal, J.M., Krasilnikoff, P.A., Weile, B., Berget, A. (1986). Effect of passive smoking on birth- weight. *Lancet,* 2, 415-417.

602. Russek, H. I., Zohman, B.L. (1958). Relative significance of hereditary diet and occupational stress in CHD of young adults. *American Journal of Medical Science,* 235, 226-275.

603. Russell, P. A. G. (1985). Indoor air pollutants - type, sources and control. *Canada Mortgage and Housing Corporation,*

604. Rycroft, R. J. G., Calnan, C.D. (1984). Facial rashes among VDU operators. In *Health Hazards of VDUs*(Pearse, B.) Chichester, New York: John Wiley and Sons.

605. Rylander, R. M., K.E., Verner-Carlson, B., Ohrstrom, M. (1974). Bacteriological investigation of wall-to-wall carpeting. *American Journal of Public Health,* 62, 163.

606. Rylander, R. H., P., Lundholm, M., Mattsby, I., Stenqvist, K. (1978). Humidifier fever and endotoxin exposure. *Clinical Allergy,* 8, 511-516.

607. Sale, C. E. (1971). Humidification during cold weather to assist perennial allergic rhinitis patients. *Annals of Allergy,* 29, 256-357.

608. Sale, C. S. (1972). Humidification to reduce respiratory illness in nursery school children. *Southern Medical Journal,* 65(7),

609. Salisbury, S. K., A., Miller, B.I., Roper, P. (1982). 101 Marietta Tower Building. (TA-80-122-1117). Cincinnati: NIOSH.

610. Salman, S. P., D.F., Swift, D.L., Evering, S.A. (1971). Nasal resistance: description of a method and effect of temperature and humidity changes. *Ann. otalar.,* 80, 736-743.

611. Samet, M. M., C., Spengler, J.D. (1987). Health effects and sources of indoor air pollution. *American review of Respiratory Diseases,* 136, 1486-1508.

612. Samuelsson, C. (1984). Rn-222 and decay products in outdoor and indoor environments. *Doctoral Thesis, Radiofyskia Institutionen, Lund University,*

613. Sandberg, M. S., M. (1983). The use of movements for assessing air quality in ventilated rooms. *Build. Environ.,* 18, 181-197.

614. Sander, D. P., Everson, R.B., Wilcox, A.J. (1985). Passive smoking in adulthood and cancer risk. *American Journal of Epidemiology,* 121, 37-48.

615. Saracci, R. (1985). Man-made mineral fibres and health. *Scandinavian Journal Work Environmental Health,* 11, 215-222.

616. Saunders, J. E. (1969). The role of the level and diversity of horizontal illumination in the appraisal of a simple office task. *Ltg Res. Technology,* 1, 37-46.

617. Savitz, D. W., H., Barnes, F.A., John, E.M., Tvrdik, J.G. (1988). Case control study of childhood cancer and exposure to 60-Hz magnetic fields. *American Journal of Epidemiology,* 128, 21-38.

618. Savitz, D. C., E. (1987). Leukemia and occupational exposures to electromagnetic fields; review of epidemiologic surveys. *Journal of Occupational Med,* 29, 47-51.

619. Savitz, D. J., E., Klecker, R. (1990). Magnetic field exposure from electric appliances and childhood cancer. *American Journal of Epidemiology,* 131, 763-773.

620. Savitz, D. P., N., Poole, C. (1989). Methodological issues in the epidemiology of electromagnetic fields and cancer. *Epidemiologic Reviews,* 11, 59-78.

621. Savolainen, H. (1982). Nordic expert group for TLV evaluation: 40; Hydrogen sulfide. *Arbeta Och Halsa,* 31, 1-27.

622. Sax, N. I. ed. (1974). *Industrial Pollution.* New York: Van Nostrand Reinhold Company.

623. Schacter, J. (1957). Pain, fear, and anger in hypertensives and normotensives. *Psychosomatic Medicine,* 19, 17-29.

624. Schenker, M. B., Weiss, S.T., Murawski, B.J. (1982). Health effects of residence in homes with urea formaldehyde insulation: a pilot study. *Environment International,* 8, 329- 363.

625. Schenker, M. B., Samet, J.M., Speizer, F.E. (1983). Risk factors for childhood respiratory disease: the effect of host factors and home environmental exposures. *American Review of Respiratory Diseases,* 28, 1028-1043.

626. Schilling, R. F. S. (1956). Byssinosis in cotton and other textile workers. *Lancet,* 2, 261-265.

627. Schilling, R. S. F., Letai, A.D., Hui, G.J., Beck, J.B., Schoenberg, J.B., Bouhuys, A. (1977). Lung function, respiratory disease, and smoking in families. *American Journal of Epidemiology,* 106, 274.

628. Schilling, R. F. S., Vigliani, E.C., Lammers, B., Valic, F., Gilson, J.C. (1963). A report on a conference on byssinosis. In *14th International Conference on Occupational Health.* International Conference No 62 Amsterdam: Excerpta Medica.

629. Schiochetti, G. C., G.F., Ingrao, G., Scacco, F. (1983). Results of a survey on radioactivity of building materials in Italy. *Health Physics,* 45, 385.

630. Schlicting, H. E. (1969). The importance of airborne algae and protozoa. *Journal of the Air Pollution Control Association,* 19, 946.

631. Schlosberg, H. (1954). Three dimensions of emotion. *Psychological Review,* 61, 81-88.

632. Schneider A. (Compiler). (1988). *Correspondence course baubiologie.* Neubeuern: Institut fur Baubiologie.

633. Schreincke, G. H., A., Kratzsch, J., Huber, B., Voigt, G. (1990). Stress-related changes of saliva cortisol in VDU operators. *International Archives of Occupational and Environmental Health,* 62, 319-321.

634. Schultz, D. P. (1965). *Sensory restriction. Effects on behaviour.* New York: Academic Press.

635. Seabury, J. J. (1982). California Occupation Safety and Health Administration Consultation Service. Sacramento.

636. Seabury, J. S., J., Domer, J., Fink, J., Kawai, T. (1973). Characterization of thermophilic actinomycetes isolated from residential heating and humidification systems. *Journal of Allergy and Clinical Immunology,* 51, 161.

637. Seamonds, B. C. (1982). Stress and its effects on absenteeism in a corporate employee group. *Journal of Management,* 24, 393-397.

638. Seedorf, L. O., E. (1990). Exposure to organic solvents - I. A survey on the use of solvents. *Annals of Occupational Hygiene,* 34, 371-378.

639. Seedorf, L. O., E. (1990). The use of solvents. *Annals of Occupational Hygiene,* March, 371-377.

640. Seifert, B. (1984). Air pollution in dwellings and other indoor areas. *Staub, Reinhaltung der Luft,* 44, 377-382.

641. Seifert, B. A., H.J. (1982). Indoor air concentrations of benzene and some other aromatic hydrocarbons. *Ecotoxicol. Environ. Safety,* 6, 190-192.

642. Seifert, B. U., D. (1987). Methodologies for evaluating sources of volatile organic chemicals (VOC) in homes. *Atmospheric Environment,* 21, 395-404.

643. Seifert, B. S., C., Steinbach, I., Ullrich D., Mailahn W. (1987). Seasonal variation of concentrations of volatile organic compounds in selected german homes. In Seifert B. (ed.), *Indoor Air '87.* 1 Berlin. (pp. 107-111).

644. Selye, H. (1960). The concept of stress in experimental physiology. In J. m. Tanner (ed.), *Stress and Psychiatric Disorder*(Tanner, J.M.) Oxford: Blackwell.

645. Selye, H. (1956). *The stress of life.* New York: McGraw-Hill.646. Selye, H. (1973). Stress and aerospace medicine. *Aerospacial Medicine,* 44, 190-193.

647. Selye, H. (1974). Stress without distress. *World Health Organization, Geneva,* December, 3-11.

648. Selye, H. (1976). *The stress of life.* New York: McGraw-Hill.

649. Seppanen, O. W., J.R. (1990). The fifth international conference on indoor air quality and climate. *ASHRAE Journal,* 32(10), 18-20.

650. Sereda, P. J., Feldman, R.F. (1964). Electrostatic charging on fabrics at various humidities. *Journal of the Textile Industry,* 55, 288-298.

651. Sexton, K. S., J.D., Treitman, R.D. (1984). Effects of residential wood combustion on indoor air quality: a case study in Waterbury, Vermont. *Atmospheric Environment,*

652. Sexton, K. L., R., Spengler, J.D. (1983). Estimating human exposures to nitrogen dioxide. An indoor outdoor modelling approach. *Environ Res,* 32, 151-166.

653. Shands, K. N., Meyer, R.D., Gorman, G.W., Edelstein, P.H., Finegold, S.M., Fraser, D.W. (1985). Potable water as a source of Legionnaire's disease. *JAMA,* 253, 1412-1416.

654. Sharpe, A. P. (1983). *An investigation into the effects of enhanced negative air ionisation in an urban office environment.* (Report to Portsmouth City Council). UK.

655. Sheilds, S. A., Stern, R.M. (1979). Emotion: The perception of bodily change. In P. P., Blankstein, K.R. Spigel (eds.), *Advances in the Study of Communication and Affect.* Vol. 5 New York: Plenum.

656. Sheldon, L. S., Handy, R.W., Hartwell, T.D., Whitmore, R.W., Zelon, H.S., Pellizzari, E.D. (1988). In *Indoor air quality in public buildings, Volume 1.* (EPA/600/S6-88/009a). Washington, D.C.: U.S. Environmental Protection Agency.

657. Shy C.M, G. J. R., Hackney, J.D., Lebowitz, M.D., Menzel, D.B. (1978). Health effects of air pollution. *ATS News,* (6), 1-63.

658. Simonato, L. F., A.C., Cherrie, J., Andersen, A., Bertazzi, P.A., Charnay, N., Claude, J., Dodgson, J., Esteve, J., Frentzel-Beyme, R., Gardner, J., Jensen, O.M., Olsen, J.H., Saracci, R., Teppo, L., Win. (1986). The man-made mineral fibre European cohort study: extension to the follow-up. *Scandinavian Journal Work Environment Health,* 12(Suppl 1), 34-47.

659. Singleton, W. T. (1975). *An introduction to ergonomics.* Geneva: World Health Organisation.

660. Singleton, W. T. (1979). Some conceptual and operational doubts about job satisfaction. In R. G., Shipley, P. Sell (eds.), *Satisfaction in Work Design.* London: Taylor and Francis.

661. Skaaret, E. (1986). Contaminant removal performance in terms of ventilation effectiveness. Environment International, 12, 419-427.

662. Skaaret, E. (1984). A survey of concepts of ventilation effectiveness. *Open Sintef Report.* (STF15 A84057). Trondheim: The Norwegian Institute of Technology.

663. Skaaret, E. M., H.M. (1983). Ventilation efficiency - a guide for efficient ventilation. *ASHRAE Transactions,* 89, 480-495.

664. Skov, P. V., O., Pedersen, B.V., and DICSG. (1989). Influence of personal characteristics, job related factors and psychosocial factors on the sick building syndrome. *Scandinavian Journal of Work Environment Health,* 15, 286-295.

665. Skov, P. V., O. (1987). The sick building syndrome in the office environment: the Danish town hall study. *Environment International,* 13, 339-349.

666. Skov, P.V. (1987). The sick building syndrome in the office environment: the Danish Town Hall Study. In *Proceedings of the 4th International Conference on Indoor Air Quality and Climate.* Berlin. Institute of Water, Soil and Air Hygiene.

667. Slavin, R. G. (1978). Antigens implicated in the pathegenesis of extrinsic allergic alveolitis. *Journal Allergy Clinical Immunology,* 61, 223.

668. Slawina, S. E. (1975). Methods of evaluation of heaviness and intensity of work. *Poznam, Polska,* (Translation),

669. Smiley, J. A. (1951). The hazards of rope making. *British Journal of Industrial Medicine,* 8, 265-270.

670. Smith, D. H., Lockhart, W., Mair, A., Wilson, W.A. (1962). Flax workers byssinosis in East Scotland. *Scottish Medical Journal,* 7, 201-211.

671. Smith, D. D., Donovan, R.P., Ensor, D.S., Sparks, L.E. (1990). Quantification of particulate emission rates from vacuum cleaners. *Proceedings of the 5th International Conference on Indoor Air Quality and Climate.* 3 Toronto, Canada. (pp. 647-652). Ottawa: Canada Mortgage and Housing Corporation.

672. Smith, J. W., B. (1991). Humidification: friend or foe? *Building Services,* 13, 45.

673. Smith, P. W. (1977). Room humidifiers as a source of Acinteobacter infections. *JAMA,* 237, 795.

674. Soratin, H. S., F. (1984). Natural radioactivity of building materials in Austria. *Radiat. Prot. Dosim,* 7, 59.

675. Sources of managerial and white-collar stress. (1979). In C. L., Payne, R. Cooper (eds.), *Stress at Work.* (pp. 81-106). New York: John Wiley and Sons.

676. Spaite, P. W., Stockton, M.B., McLean, J.S. (1990). Classification of materials as potential sources of indoor air pollution. Washington DC: U.S. Environmental Protection Agency.

677. Speer, F. (1968). Tobacco and the nonsmoker. *Archives of Environmental Health,* 16, 443.

678. Speizer, F. E. (1980). Respiratory disease rates and pulmonary function in children associated with NO2 exposure. *American Review of Respiratory Disease,* 121, 3-10.

679. Spengler, J. D., Cohen, M.A. (1985). Emissions from indoor combustion sources. In Garnage and Kaye (eds.), *Indoor Air and Human Health, Proceedings of the Seventh Life Sciences Symposium.* Knoxville, Tennessee. (pp. 29-31).

680. Spengler, J. D., Sexton, K. (1983). Indoor air pollution: a public health perspective. *Science,* 221(4605), 9-17.

681. Spengler, J. D., Dockery, D.W., Turner, W.A., Wolfson, J.M., Ferris, B.G., Jr. (1981). Long term measurements of respirable sulfates and particles inside and outside homes. *Atmos.Environ.*, 15(23),

682. Spiegleman, J. B., G., Freidman, H. (1963). Effects of central air conditioning on pollen, mould, and bacterial concentrations. *Journal of Allergy,* 34, 426.

683. Spitz, M. R., Johnson, C.C. (1985). Neuroblastoma and paternal occupation: a case-control analysis. *American Journal of Epidemiology,* 121, 924-929.

684. Staib, F. T., B., Theil, D., Blisse, A. (1978). Aspergillus fumigatus and Aspergillus niger in two potted ornamental plants, cactus (Epiphyllum truncatum) and clivia (Clivia miniata). Biological and epidemiology aspects. *Mycopathologia,* 66, 27.

685. Staib, F. F., U., Tompak., B., Abel, T., Thiel, D. (1978). A comparative study of antigens of Aspergillus fumigatus isolates from patients and soil of ornamental plants in the immunodiffusion test. *Zentral. Bakteriol.*, 242, 93.

686. Stellar, E. (1954). The physiology of motivation. *Psychological Review,* 61, 5-22.

687. Stellman, J. (1977). *Women's work, women's health: Some myths and realities.* New York.

688. Stellman, M. J., Klitzman, S., Gordon, G.C., Snow, B.R. (1985). Air quality and ergonomics in the office; Survey results and methodologic issues. *American Industrial Hygiene Association Journal,* 46(5), 286-293. (Massey)

689. Sterling, E. M., Sterling, T.D. (1984). Baseline data: health and comfort in modern office buildings. In *Proceedings of the 5th Air Infiltration Centre Conference: The Implementation and Effectiveness of Air Infiltration Standards in Buildings.* The Air Infiltration Centre. Berkshire, England: International Energy Agency.

690. Sterling, E. S., T. (1983). The impact of different ventilation levels and fluorescent lighting types on building illness: an experimental study. *Canadian Journal of Public Health,* 74, 385-392.

691. Sterling, E. S., T., McIntyre, D. (1983). New health hazards in sealed buildings. *Journal American Institute of Architecture,* 64-67.

692. Sterling, E. M., McIntyre, E.M., Collett, C.W., Sterling, T.D., Meredith, J. (1985). Sick buildings: case studies of tight building syndrome and indoor air quality investigations in modern office buildings. *Environmental Health Review,* 29, 11-19.

693. Sterling T.D., K. D. (1982). Indoor byproduct levels of tobacco smoke: a critical review of the literature. *Journal of the Air Pollution Control Association,* 32, 250-259.

694. Sterling, T. D., Sterling, E., Dimich-Ward, H.D. (1983). Air quality in public buildings with health related complaints. *ASHRAE Transactions,* 89(2a), 198.

695. Sterling, T. D., Sterling, E., Dimich-Ward, H. (1983). Building illness in the white collar workplace. *International Journal Health Serv.*, 13(2), 277-287.

696. Sterling, T. D., Collett, C.W., Sterling, E.M. (1987). Environmental tobacco smoke and indoor air quality in modern office environments. *Journal of Occupational Medicine,* 29, 57-62.

697. Sterling, T. D., Sterling, E.M. (1984). Investigations on the effect of regulating smoking on levels of indoor pollution and on the perception of health and comfort of office workers. *European Journal of respiratory diseases,* 65(133), 17-32.

698. Sterling, T. D., Collett, C.W., Ross, J.A. (1988). Levels of environmental tobacco smoking under different conditions of ventilation and smoking regulations. In J. P. Harper (ed.), *Transactions of an International Specialty Conference: Combustion Processes and the Quality of the Indoor Environment.* Niagara Falls, New York. (pp. 223-235).

699. Stern, F. W., R., Beaumont, J. (1986). A case-control study of leukaemia at a naval shipyard. *American Journal of Epidemiology,* 123, 980-992.

700. Sternbach, R. T., B. (1965). Ethnic differences among housewives in psychophysical and skin potential responses to electric shock. *Psychophysiology,* 1, 241-246.

701. Steudler, P. A., Peterson, B.J. (1984). Contribution of gaseous sulphur from salt marshes to the global sulphur cycle. *Nature,* 311, 455-457.

702. Stevens, S. S. (1975). *Psychophysics: Introduction to its perceptual, neural, and social prospects.* New York: Wiley.

703. Stewart, R. D., Baretta, E.D., Dodd, H.C., Torkelson, T.R. (1970). Experimental human exposure to tetrachloroethylene. *Archives Environ Health,* 20, 224-229.

704. Steyer, R. (1986). Sick-building syndrome. *Across the Board,* 23(12), December, 34-41.

705. Stoke, J. (1991). The indoor environment. *Occupational Physician,* September, 1-14.

706. Stout, J. Y., V.L., Vickers, R.M. (1982). Ubiquitousness of Legionella pneumophilia in the water supply of a hospital with endemic legionnaires' disease. *New England Journal of Medicine,* 306, 466-468.

707. Strahilevitz, M. S., A., Miller, J. (1979). Air pollutants and the admission rate of psychiatric patients. *American Journal of Psychiatry,* 136, 205-207.

708. Stranden, E. K., A.K., Lind, B. (1984). *The ETB-Dosemeter, a passive integrating radon dosemeter combining activated charcoal and TLD.* : Radiation Protection Dosimetry, Vol 5, No. 4.

709. Stranden, E. K., A.K., Lind, B. (1984). The influence of moisture and temperature upon radon exhalation. In *Procedures of the International Seminar on Indoor Exposure to Natural Radiation and Related Risk Assessment.* Capri, Italy.

710. Stranden, E. (1981). Radiation in houses utilizing stone magazines for heat accumulation. *Health Physics,* 41, 29.

711. Stuchley, M. A. (1986). Human exposure to static and time- varying magnetic fields. *Health Physics,* 51, 215-225.

712. Sudman, S. B., N.M. (1982). *Asking questions: a practical guide to questionnaire design.* San Francisco: Jossey-Bass.

713. Sulman, F. G., Levy, D., Lunkan, L., Pfeifer, Y. (1978). Absence of harmful effects of protracted negative air ionisation. *International Journal of Biometry,* 22(1), 53-58.

714. Sulman, F. G., Levy, D., Lewy, A., Pfeifer, Y., Superstine, E., Tal, E. (1975). Effect of the Sharav and Bora on urinary neurohormone excretion in 500 weather-sensitive females. *International Journal of Biometeorology,* 19, 202-209.

715. Sulman, F. G. (1974). Meterological front movements and human weather sensitivity. *Karger Gazette,* 30, 1-2.

716. Sulman, F. G., Danon, A., Pfeifer, Y., Tal, E., Weller, C.P. (1970). Urinalysis of patients suffering from climatic heat stress (Sharav). *International Journal of Biometeorology,* 14, 45-53.

717. Sundin, B. (1982). Formaldehyde emission from wood products; technical experiences and problem solutions from Sweden. In *Proceedings of the 3rd Medical Legal Symposium on Formaldehyde Issues.* Washington DC.

718. Swaebly, M. D., Christensen, C.M. (1952). Molds in house dust, furniture stuffing and air within homes. *Journal of Allergy,* 23, 370.

719. Swedjemark, G. A., Mjones, L. (1984). Radon and radon daughter concentrations in Swedish homes. *Radiat. Prot. Dosim.,* 7, 341.

720. Swedjmark, G. A., Mjones, L. (1984). Exposure of the Swedish population to radon daughters. In B. Berglund, Lindvall, T., Sundell, J. (eds.), *Indoor Air: Radon, Passive Smoking, Particulates and Housing Epidemiology.* 2 (pp. 37). Stockholm: Swedish Council for Building Research.

721. Sweet, L. C., Anderson, J.A., Callies, Q.C., Coates, E.O. (1971). Hypersensitivity pneumonitis related to a home furnace humidifier. *Journal Allergy Clin Immunol,* 48(3), 171- 178.

722. Swerdlow, A. J. (1983). Epidemiology of eye cancer in adults in England and Wales, 1962-1977. *American Journal of Epidemiology,* 118, 294-300.

723. Swinne-Desgain, D. (1975). Cryptococcus neoformans of saprophytic origin. *Sabouraudia,* 13, 303.

724. Syandberg, M. (1981). What is ventilation efficiency? *Build. Environ.,* 16, 123-135.

725. Sykes, J. M. (1989). Sick building syndrome. *Building Services Engineering research and Technology,* 10(1), 1-11.

726. Tager, I. B., Weiss, S.T., Rosner, B., Speizer, F.E. (1979). Effect of parental cigarette smoking on the pulmonary function of children. *American Journal of Epidemiology,* 110, 15.

727. Taylor, A. J. N. (1980). Occupational asthma. *Thorax,* 35, 241-245.

728. Taylor, P. R., Dell 'Acqua, B.J., Baptiste, M.S. (1984). Illness in an office building with limited fresh air access. *Journal of Environmental Health,* 47(1), 24-27. (ventilation/)

729. Taylor, R. (1974). Stress at work. *New Society,* 30, 139-143.

730. Theorell, T. K., R. (1984). Blood pressure variations at work in asymptomatic non-medicated males. *Journal of Human Stress,*

731. Thomas, A. E., Platts-Mills, M.D., Chapman, M.D. (1987). Dust mites: immunology, allergic diseases and environmental control. *The Journal of Allergy and Clinical Immunology,* 80(6), 755-775.

732. Thomas, D. C., McNeill, K.G., Dougherty, C. (1985). Estimates of lifetime lung cancer risks resulting from RN progeny exposure. *Health Physics,* 49, 825-846.

733. Thorstensen, E. H., C., Pejtersen, J., Clausen, G.H., Fanger, P.O. Air pollution sources and indoor air quality in school. In *Indoor Air '90.* 1 Toronto. (pp. 531-536).

734. Timbrell, V. P., F.D., Wagner, J.C. (1970). Characteristics of respirable asbestos fibres. In H. A. Shapiro (ed.), *Pneumoconiosis, Proceedings of the International Conference.* Johannesburg. (pp. 120-125). London and Capetown: Oxford University Press.

735. Timbrell, V. (1969). The inhalation of fibres. In H. A. Shapiro (ed.), *Pneumonoconiosis Proceedings of the International Conference.* Johannesburg. (pp. 3-9). London and Capetown: Oxford University Press.

736. Tjonn, H. H. (1984). Report of facial rashes among VDU operators in Norway. In B. Pearse (ed.), *Health Hazards of VDUs.* Chichester, New York: Wiley and Sons.

737. Tobin, J. O., Watkins, I.D., Woodhead, S., Mitchell, R.G. (1986). Epidemiological studies using monoclonal antibodies to Legionella pneumophilia serogroup 1. *Israel Journal of Medical Science,* 22, 711-714.

738. Tobin, J. (1983). Legionnaires Disease. *Oxford Textbook Medicine,* 1(5), 331-335.

739. Tolmenius, L. H., L., Enander, E. (1986). Electrical constructions and 50 Hz magnetic field at the dwellings of tumour cases (0-18 years of age) in the county of Stockholm. In *Procedures of the International Symposium on Occupational Health and Safety in Mining and tunnelling.* Prague.

740. Tomenius, L. (1986). 50-Hz electromagnetic environment and the incidence of childhood tumours in Stockholm county. *Biomagnetics,* 7, 191-207.

741. Tregenza, P. R., Romaya, S.M., Dawes, S.P., Heap, L.J. (1974). Consistency and variation in the preferences for office lighting. *Ltg Res. Technology,* 6, 205-211.

742. Trichopoulos, D. K., A., Sparros, L., MacMahon, B. (1981). Lung cancer and passive smoking. *International Journal of Cancer,* 27(1),

743. Troughton, G. E. (1969). Accelerated aging of glue-wood bonds. *Wood Science,* 1, 172-176.

744. Turk, A. (1983). Gaseous air cleaning can help maintain tolerable indoor air quality limits. *ASHRAE Journal,* 35-37.

745. Turk, B. H., Brown, J.T., Geisling-Sabotka, K., Froehlich, D.A., Grimsrud, D.T., Harrison, J., Revzan, K.L. (1986). Indoor air quality measurements in 38 Pacific Northwest commercial buildings. In *Proceedings of the 79th Annual Meeting of the Air Pollution Control Association.* Minneapolis, Minnesota. Pittsburg, Pennsylvania: Air Pollution Control Association.

746. Turk, B. H., Brown, J.T., Geisling-Sabotko, K., Froehlich, D.A., Grimsrud, G.T., Harrison, J., Koonce, J.F., Prill, R.J., Revzan, K.L. (1987). Indoor air quality and ventilation measurements in 38 Pacific northwest commercial buildings. Portland, Oregon: Bonneville Power Administration.

747. Turner, S. (1988). Environmental tobacco smoke and smoking policy. In J. P. Harper (ed.), *Transactions of an International Specialty Conference: Combustion Processes and the Quality of the Indoor Environment.* Niagara Falls, New York. (pp. 236-247).

748. Tursky, B. S., R. (1967). Further physiological correlates of ethnic differences in responses to shock. *Psychophysiology,* 4, 67-74.

749. Tuypens, E. (1961). Byssinosis among cotton workers in Belgium. *British Journal of Industrial Medicine,* 18, 117-119.

750. U.S. Department of Health, E. a. Welfare. (1979). Involuntary smoking, in smoking and health: a report of the Surgeon General. *DHEW Publication number PHS79-50066,* Washington, D.C.,

51. U.S. Department of Health, E. a. Welfare, Public Health Service. (1979). Smoking and health. *A Report of the Surgeon General.* (DHEW (PHS) 79-50066). Washington D.C.: U.S. Government Printing Office.

752. U.S. Department of Health and Human Services, P. H. Service. (1982). The health consequences of smoking: cancer. *A Report of the Surgeon General.* (DHHS (PHS) 82-50179). Washington DC: U.S. Government Printing Office.

753. U.S. Department of Health and Human Services, P. H. Service, Office on Smoking and health. (1984). The health consequences of smoking: chronic obstructive lung disease. *A Report of the Surgeon General.* (DHHS (PHS) 84-50205). Washington D.C.: U.S. Government Printing Office.

754. U.S. Department of Health and Human Services, P. H. Service, Office on Smoking and Health. (1986). The health consequences of involuntary smoking. *A Report of the Surgeon General.* Washington D.C.: U.S. Government Printing Office.

755. U.S. Department of Health and Human Services, P. H. Service. (December, 1983). Report on workshop on respiratory effects on involuntary smoke exposure: epidemiologic studies. M. D. Bethesda, : Public Health service, National Institutes of Health.

756. U.S. Environmental Protection Agency (EPA). (1988). Indoor air facts number 4: sick buildings. Washington, D.C.,

757. UNSCLEAR. (1977). United Nations Scientific Committee on the Effects of Atomic Radiation. *Sources and effects of ionizing radiation; report to the General Assembly.* (Publication number E.77.IX.1). : United Nations Publication.

758. UNSCLEAR. (1982). United Nations Scientific Committee on the Effects of Atomic Radiation. *Ionizing radiation: Sources and biological effects; report to the General Assembly.* (Number E.82.IX.8). : United Nations Publication.

759. Uragoda, C. G. (1975). A clinical and radiographic study of coir workers. *British Journal of Industrial Medicine,* 32, 66- 71.

760. Uragoda, C. G. (1977). An investigation into the health of kapok workers. *British Journal of Industrial Medicine,* 34, 181-185.

761. Urch, R. B., Silverman, F., Corey, P., Shephard, R.J. (1990). Acute symptom responses to passive smoke in asthmatic and nonasthmatic individuals. *Proceedings of the 5th International conference on Indoor Air Quality and Climate.* 1 Toronto, Canada. (pp. 337-342). Ottawa: Canada Mortgage and Housing Corporation.

762. Vaalbjorn, O. K., N. (1984). Headache and mucous membrane irritation. An epidemiological study. In B. Berglund, Lindvall, T., Sundell, J. (eds.), *Proceedings of the 3rd International Conference on Indoor Air Quality and Climate.* 2 (pp. 249-254). Stockholm: Swedish Council for Building Research.

763. Vagero, D. A., A., Olin, R. (1985). Cancer Mortality among workers in the telecommunications industry. *British Journal of Industrial Medicine,* 42, 191-195.

764. Vagero, D. O., R. (1983). Incidence of cancer in the electronics industry: using the new Swedish cancer environment registry as a screening instrument. *British Journal of Industrial Medicine,* 40, 188-192.

765. Vagero, D. O., R. (1986). Incidence of cancer in the electronics industry using the new Swedish Cancer Environment registry as a screening instrument. *British Journal of Industrial Medicine,* 40, 188-191.

766. Valbjorn, O. K., N. (1984). Headache and mucous membrane irritation. An epidemiological study. In B. Berglund, Lindvall, T., Sundell, T (eds.), *Proceedings of the 3rd International Conference on Indoor Air Quality and Climate.* 2 Stockholm, Sweden. (pp. 249-254). Stockholm: Swedish Council for Building Research.

767. Valentin, M. (1978). Trevail des hommes et savants oublies. *Paris: Editions Docis,* 177-184.

768. Vamplew, C. (1973). Automated process operators: Work attitudes and behaviour. *British Journal of Industrial Relations,* 11, 415-439.

769. Van Ireland, J. F. (1967). *Two thousand Dutch office workers evaluating lighting.* TNO, Delft, The Netherlands: Research Institute for Environmental Hygiene, Publication 283.

770. Vaughan, N. G., T. (1993). Healthy Office Environment Survey (questionnaire). *The Science and Engineering Research Council and the DTI,* Pearl Assurance House, Cardiff,

771. Vigliani, E. C., Parmeggiani, L., Sassi, C. (1954). Studio de un epidemia di bronchite asmatica fra gli operai di una tessitura di cotone. *Med Lav,* 45, 349-378.

772. Villaveces, J. W. (1971). The dehumidifier: Its indoor use in controlling molds and mould asthma: A personal case history. *Annals of Allergy,* 29, 93.

773. von Nieding, G. (1977). Acute effects of ozone on human lung function. *VDI-Berichte,* 270, 123-129.

774. Voorhorst, R. (1977). The human dander atopy. I. The prototype of autoatopy. *Annals of Allergy,* 39, 205.

775. Wagner, J. C. (1968). *Asbestos and cancer. Book 3.* Chicago: Abbott Universal Ltd.

776. Walingford, K. M., Carpenter, J. (1986). Field experience overview: investigating sources of indoor air quality problems in office buildings. *Proceedings of IAQ '86: Managing Indoor Air for Health and Energy Conservation.* Atlanta, Georgia. (pp. 4488-453). Atlanta: ASHRAE.

777. Wallace, L. (1987). Emissions of volatile organic compounds from building materials and consumer products. In *Indoor Air '87: Proceedings of the 4th International Conference on Indoor Air Quality and Climate.* Berlin. : Institute for Water, Soil and Air Hygiene.

778. Wallace, L. A., Hartwell, T.D., Perritt, K., Sheldon, L.S., Michael L., Pellizzari, E. (1987). The influence of personal exposure to volatile organic compounds. In Seifert B. (ed.), *Indoor Air '87.* 1 Berlin. (pp. 117-121).

779. Wallace, L. J., R., Sheldon, L., Pellizzari, E. (1987). Volatile organic chemicals in 10 public-access buildings. In B. Seifert (ed.), *Indoor Air '87: Proceedings of the 4th International Conference on Indoor Air Quality and Climate.* Berlin. : Institute for Water, Soil, and Air Hygiene.

780. Wallace, L. A., Nelson, C.J., Dunteman, G. (1991). Workplace characteristics associated with health and comfort concerns in three buildings in Washington DC. *Proceedings of IAQ '91 - Healthy Buildings.* Washington DC. (pp. 56-60). Atlanta: ASHRAE.

781. Wallingford, K. M., Carpenter, J. (1986). Field experience overview: investigating sources of air quality problems in office buildings. *ASHRAE conference.* Atlanta. : ASHRAE.

782. Walsh, P. J., Dudney, C.S., Copenhaver, E.D. (1984). *Indoor air quality.* Boca Raton, Florida: CRC Press Inc.

783. Wan, T. (1976). Predicting self-assessed health status: a multivariate approach. *Health Services Research,* 11, 464-477.

784. Wangler, R. B., Bradley, P.M., Clift, W.D., Davidson, D., Higgins, L., Sandstrom, K., Stephens, R. (1985). Leukaemia risk in amateur radio operators. *Lancet,* 1, 156.

785. Ward, R. C., Travis, C.C., Hetrick, D.M., Andersen, M.E., Gargas, M.L. (1988). Pharmacokinetics of tetrachloroethylene. *Toxicol Appl Pharmacol,* 93, 108-117.

786. Warren, W. P. (1972). Hypersensivity pneumonitis due to exposure to budgerigars. *Chest,* 62(2), 170.

787. Watkins, I. D., Tobin, J.O'H., Dennis, P.J.L., Brown, W., Newman, R., Kurtz, J.B. (1985). Legionella pneumophilia serogroup 1 subgrouping by monoclonal antibodies - an epidemiological tool. *Journal Hygiene Camb.,* 95, 211-216.

788. Weiner, J. S. (1982). The measurement of human workload. *Ergonomics,* 25(11), 953-965.

789. Weiss, N. S., Soleymani, D.A. (1971). Hypersensitivity lung disease caused by contamination of an air conditioning system. *Annals of Allergy,* 29, 154-156.

790. Welford, A. T. (1973). Stress and performance. *Ergonomics,* 16, 567-580.

791. Wertheimer, N. L., E. (1982). Adult cancer related to electrical wires near the home. *International Journal of Epidemiology,* 11, 345-355.

792. Wertheimer, N. Lepper, E. (1979). Electrical wiring configurations and childhood cancer. *American Journal of Epidemiology,* 109, 273-284.

793. Wertheimer, N. Lepper, E. (1986). Possible effects of electric blankets and heated waterbeds on foetal development. *Bioelectromagnetics,* 7, 13-22.

794. Westphal, J. (1987). *Colour: some philosophical problems from Wittgenstein.* Oxford: Blackwell.

795. White, J. R., Froeb, H.F. (1980). Small-airways dysfunction in nonsmokers chronically exposed to tobacco smoke. *New England Journal of Medicine,* 302(13), 720.

796. WHO. (1987). Air quality guidelines for europe. *European Series(26).* Copenhagen: WHO Regional Publications.

797. WHO. (1972) *Environmental influences on mental health; In: Health Hazards of the Human Environment.* Geneva.

798. WHO. (1982). Indoor air pollutants: exposure and health effects. *Report on a WHO Meeting at Norlingen.* Copenhagen: WHO Regional Office for Europe.

799. WHO. (1983). Indoor air pollutants: exposure and health effects. *EURO reports and studies,* 78, 23-26.

800. WHO. (1986). Indoor air quality research. *EURO Reports and Studies,* No. 103,

801. WHO. (1989). Indoor air quality: indoor pollutants. *EURO Reports and Studies(111).* Copenhagen: WHO Regional Publications.

802. WHO, I. A. for Research on Cancer. (1986). IARC monographs on the evaluation of the carcinogenic risk of chemicals to humans: tobacco smoking. (vol. 38). Lyon, France: IARC.

803. Wicke, A. P., J. (1983). Application of surface-barrier- detectors for measurement of environmental radon and radon daughters in air. In *Procedures of the International Meeting on Radon-Radon Progeny Measurements.* Report EPA 520/583/021 Montgomery, Alabama. Washington DC: US Environmental Protection Agency, Office of Radiation Programs.

804. Wiklund, K. E., J., Eklund, G. (1981). An application of the Swedish Cancer-Environment registry: Leukaemia among telephone operators at the telecommunications administration in Sweden. *International Journal of Epidemiology,* 10, 373-376.

805. Wilkening, M. H., Stanley, R.D., Clements, W.E. (1972). Radon flux measurements in widely separated regions. *Natural Radiation Environment III.* (CONF-780805). : ERDA Report.

806. Wilkening, M. H., Clements, W.E., Stanley, D. (1972). Radon- 222 flux measurements in widely separated regions. In J. A. S., Lowder, W.M., Gessell, T.F. Adams (eds.), *National Radiation Environment II.* (CONF-720805). Springfield, VA: National Technical Information service.

807. Wilkins, A. J., Nimmo-Smith, I., Slater, A.I., Bedocs, L. (1989). Fluorescent lighting, headaches and eyestrain. *Lighting Research and Technology,* 21, 11-18.

808. Wilkins, A. J., Nimmo-Smith, M.I., Slater, A., Bedocs, L. Fluorescent lighting, headaches and eye-strain. In *CIBSE National Lighting Conference.* Cambridge.

809. Wilkins, A. J., Nimmo-Smith, I., Tait, A., McManus, C., Della Salla, S., Tilley, A., Arnold, K., Barrie, M., Scott, S. (1984). A neurological basis for visual discomfort. *Brain,* 107, 989-1017.

810. Wilson, N. (1972). Assessment of static propensity of carpets. *Shirley Report,* SS1972,

811. Wilson, S. H., A. (1986). *The office environment survey, a study of building sickness.* London: Building Use Studies Ltd.

812. Wilson, S. H., A. (1987). *The office environment survey: a study of building sickness.* London: Building Use Studies.

813. Wilson, S. O., P., Hedge, A., Jones, P. (1987). *The sick building syndrome and environmental conditions: case studies in nine buildings.* London: Building Use Studies.

814. Winneke, G. (1974). Behavioral effects of methylene chloride and carbon monoxide as assessed by sensory and psychomotor performance. In C. Xintaras (ed.), *Behavioral Toxicology: Early Detection of Occupational Hazards.* DHEW Publication (NIOSH) No. 74-126 Washington DC. (pp. 130-144). : U.S. Department of Health Education and Welfare.

815. Wittgenstein, L. (1980). *Culture and value.* Oxford: Blackwell.

816. Wolf, S. W., H.G. (1943). *Human gastric function: An experimental study of man and his stomach.* New York: Oxford University Press.

817. Wood, B. (1984). *The healing power of colour.* New York: Destiny Books.

818. Woods, J. E., Morey, P.R., Rask, D.R. (1989). Indoor air quality diagnostics: qualitative and quantitative procedures to improve environmental conditions. In N. L., Harper, J.P. Nagda (eds.), *Design and Protocol for Monitoring Indoor Air Quality.* Philadelphia: The American Society for Testing and Materials.

819. Woods, J. E., Drewry, G.M., Morey, P.R. (1987). Office worker perceptions of indoor air quality effects on discomfort and performance. In B. Seifert (ed.), *Indoor Air '87.* 2 Berlin. (pp. 264-268).

820. Wright, D. S. (1980). Man made mineral fibres: a historical note. *Journal Society of Occupational Medicine,* 30, 138-140.

821. Wyon, D. P., Asgeirsdottir, T.H., Jensen, P.K., Fanger, P.O. (1973). The effects of ambient temperature swings on comfort, performance and behaviour. *Archives des Sciences Physiologique,* 27, 441-458.

822. Yanglou, C. P., Benjamin, L.C. (1934). Diurnal and seasonal variations in small ion content on outdoor and indoor air. *Heating, Piping, Air Conditioning,* 6, 25.

823. Yanglou, C. P., Brandt, A.D., Bengamin, L.K.C. (1933). Physiological changes during exposure to ionized air. *Heating, Piping, Air Conditioning,* 5, 423.

824. Yocom, J. (1982). Indoor-outdoor air quality relationships: a critical review. *Journal of the Air Pollution Control Association,* 32, 500-520.

825. Zborowski, M. (1969). *People in pain.* San Francisco: Jossey- Bass.

826. Zola, I. (1966). Culture and symptoms: An analysis of patients presenting complaints. *American Sociological Review,* 31, 615-630.

827. Zuskin, E. V., F., Bouhuys, A. (1976). Byssinosis and airway responses to exposure to textile dust. *Lung,* 154, 17-24.

828. Zweers, T. P., L., Brunkreef, B., Boleij, J.S.M. (1990). Relationships between health and indoor climate complaints and building, workplace, job and personal characteristics. *Proceedings of the 5th International Conference on Indoor Air Quality and Climate.* 1 Toronto, Canada. (pp. 495-500). Ottawa: Canada Mortgage and Housing Corporation.

829. Zwemer, R. J., Karibo, J. (1973). Use of laminar control device as adjunct to standard environmental control measures in symptomatic asthmatic symptoms. *Annals of Allergy,* 31, 284.