EFFECTS OF POWER AND RADIO FREQUENCY ELECTROMAGNETIC FIELDS ON HUMAN PERFORMANCE

I : Power Frequency Effects on Melatonin levels, Attention and Memory

II : Radio Frequency Effects on Melatonin Levels, Cardiovascular Parameters, Aural Temperature, Melatonin levels, Attention and Memory

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DEDICATION

This thesis is dedicated to my mother, Olive Mobberley.

ABSTRACT

Humans have been exposed to manmade electromagnetic fields (EMFs) since electricity was first harnessed in the 1800s. This exposure has accelerated in the last few decades with the widespread use of electrical appliances producing 50/60 Hz power frequency EMFs. With the advent of cellular phones (900 or 1800 MHZ) exposure frequencies have increased and wavelengths shortened. With this exposure has come a public concern over possible health effects from increasing exposure to EMFs, particularly in the radio frequency bands.

Experiment One exposed 29 subjects to an EMF of 50 Hz, 100μT, pulsed (one second on/one second off). Each subject attended at 12 p.m. and 12 a.m. on two consecutive days, a total of two control and two exposure sessions. Effects on salivary melatonin levels, and the cognitive parameters of working memory and attention were studied. Experiment Two exposed 50 subjects to almost the same protocol and experimental conditions. An additional control sample of saliva was obtained before each of the four sessions. There was no significant effect on attention or auditory working memory in either experiment. In Experiment One the presence of the EMF had no effect on salivary melatonin levels, including the subgroup of those with naturally low levels. In Experiment Two, a significant drop in melatonin levels was found for the day session, but only when compared to the same day control. This raised questions as to the reliability of using a separate day as a control due to excessive within-subject variation over the two days. There were no significant differences between male and female subjects in response to the EMF.

Experiment Three investigated possible effects from a commercially available digital 900 MHz band cellular phone on melatonin levels, aural temperature, blood pressure, heart rate and the cognitive parameters of attention and memory. Forty-three subjects attended a single evening session. Two exposure sessions were surrounded by three control sessions. This was done to compensate for the natural change that occurs in melatonin levels, temperature and cardiac readings during an evening. Results were averaged for control sessions then compared to the average for the exposure sessions. A statistically significant rise in aural temperature was noted in both ears when the phone was operating. The difference was significantly greater on the side the phone was held. Scores in an attention test were also significantly lower when the phone was in use. The reduced attention level has implications for the safety of using of cellular phones whilst driving. Heating of the head may have biological consequences depending on the depth of penetration. There was no perceived effect on melatonin levels, no gender effects or effects on those with naturally low melatonin levels. Neither cardiac readings or numerical working memory were effected by exposure to a cellular phone.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

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CHAPTER ONE
OVER VIEW OF THE THESIS

Public exposure to EMFs (electromagnetic fields) has steadily increased over the last few decades. The number of frequencies to which the public is exposed has also increased, especially in the higher, or microwave, frequency bands. With this exposure has come a growing public concern as to whether everyday exposure to such fields is safe. It has long been acknowledged by the scientific community that the ionising radiation found in nuclear devices has the energy to break chemical bonds and kill or injure. Non-ionising, ultra-high frequency fields (microwaves) can also kill or injure due to their heating effect on tissues. Until recently it was thought that EMFs that could not ionise or heat tissues could not affect living systems. However, there is a growing body of scientific opinion which suggests that this may not be the case.

Initially, research focused on frequencies which do not have a heating effect, such as the low and extremely low frequency (ELF) fields associated with power lines, VDUs (visual display units) and domestic appliances. Early epidemiological studies found associations between power frequency EMFs and cancer, especially childhood leukaemia (Wertheimer & Leeper, 1979, 1982). These results led to a large number of laboratory-based studies, primarily on animals, that investigated the associations found, and sort to elicit a possible mechanism by which EMFs could affect living systems. The suppression of melatonin by EMFs was one such mechanism proposed (Stevens, 1987). Many studies reported significant results but often at fields intensities much higher than those to which humans are exposed. The most frequently used animal model has been rodents which are considerably smaller than humans and questions have been raised as to the validity of the extrapolation of these results to humans. There is a growing recognition that a body of laboratory research on human subjects is necessary.

Following the introduction of microwave ovens in the 1970s attention was focused on possible effects from exposure to microwave radiation. Initially research focused on effects from 2.45 GHz fields and most studies utilised an exposure distance of 1 metre or more from the source with field intensities high enough to cause significant heating effects. Research is now beginning to be focused on the UHFs associated with cellular phones and their associated base stations. Cellular phones are held close to the head and have the potential to affect the function of brain structures in close proximity to the phone. There is a growing public concern

as to possible carcinogenic effects (The Royal Society of N.Z. 1999 a, 1999 b) of cell phone use and, more recently, concern as to whether such use could impair the cognitive function of the user (BBC News, 1999). Such equipment is often used in association with the operation of machinery and complex decision-making situations.

The aim of this thesis was to investigate the possibility of an interaction between human subjects and EMFs. The frequencies of 50 Hertz and 900 MHz were chosen as they are the frequencies to which the public is most commonly exposed. Fifty Hertz is the mains power frequency in New Zealand and hence the frequency used by most domestic and office appliances. A flux density of 100 μT was chosen as it is the maximum continuous public exposure permitted in New Zealand (N.Z. conforms to international standards). The 900 MHz band is the frequency band used by many cellular phones which are now in common use. The aim was to model normal cellular phone use, as much as possible, whilst controlling for extraneous variables that could confound the results.

Dependant variables studied were salivary melatonin, attention and working memory for the 50 Hz EMF and salivary melatonin, attention, working memory, cardiovascular response and aural temperature for the 900 MHz EMF. Melatonin was studied as a possible mechanism of interaction between EMFs and living systems. These variables were chosen as there have been calls for more research in these areas, particularly in the 900 MHz band (IEGMP, 2000).

The thesis follows the format set out below. Chapter two provides information on the nature of electromagnetic fields. This is followed by a chapter providing background information on the physiology of melatonin, cardiac control mechanisms and a discussion on the cognitive parameters of attention and memory. Chapter four provides a review of the EMF literature in the areas relevant to the thesis and concludes with a review of possible mechanisms of interaction between EMFs and living systems. Chapters five and six contain the two experiments on the 50 HZ EMFs. Chapter seven contains the experimental work on the 900 MHz EMFs. Chapter eight contains a summary relating the findings of this research to the research literature and final conclusions. Appendix one contains the screening questionnaire relating to the 50 Hz experiments. Appendix two contains additional information relating to experiment two. Appendix three contains the screening questionnaire and additional data relating to experiment three.

CHAPTER TWO
BACKGROUND INFORMATION ON ELECTROMAGNETIC FIELDS

The purpose of the following section was to provide basic background information on electromagnetic fields as an aid to reading the research presented.

The Nature of Electric and Magnetic Fields

Electric fields

"An electric field is the space surrounding an electric charge within which it is capable of exerting a perceptible force on another electric charge" (Pitt, 1977. p.121). The electric field, E, so produced is a vector quantity and the force, F, experienced by a charge in the field is given by:

$$F=qxE$$

(where q is the magnitude of the charge placed in the field in coulombs and x is the vector product)

Electric field strength is a vector quantity and is measured in volts per metre (Tenforde and Kaune, 1987).

Magnetic fields

"Magnetic fields are produced by electric charges in motion" (Tenforde and Kaune, 1987. p.586). The force on an electric charge moving with velocity, v, through a magnetic field is given by:

$$F=qv\ B$$

(where B is the component of the field perpendicular to the direction of movement of the charge).
Magnetic fields are, for example, also produced around a wire carrying a current.

Magnetic fields have two descriptors, magnetic flux density and magnetic field strength. Magnetic flux density (B) is measured in Tesla or Weber per square metre (Wb/m^2) and is the density of lines of magnetic flux per unit area (Grissom, 1995). The old unit of gauss is also often seen in the literature (100 μT = 1 gauss). Magnetic field strength, H, is measured in Oersteds or amperes per metre. It is a vector quantity with the direction of the force being perpendicular to the direction of the field and movement of the particle (Chou et al, 1996).
1 gauss is equal to 10^{-4} tesla = $10^{-4}\ Wb/m^2$.

Whilst electric fields can be shielded, magnetic fields will pass through most matter. This means the magnetic component of an electromagnetic field from a wire carrying a current affects not only the room the wire is in but will pass through walls, ceiling and floors into adjacent areas.

Electromagnetic Field

An EMF is a "combination of a time-varying electric field and a magnetic field at a point in space" (Chou et al., 1996. p.196).

The Geomagnetic field

The earth's magnetic field is caused by electric currents circulating within the earth. Lines of magnetic flux cover the globe and are perpendicular to the surface at the poles and parallel with the surface at the equator. The angle between the ground and the flux line at any point on the globe is known as the local magnetic dip. The geomagnetic field is approximately 100 μT or 1 gauss at the earth's surface (Bueche, 1977).

Electromagnetic induction

When a conductor is moved across the field lines of a magnetic field a potential difference is induced in the conductor. Current flows when there is a potential difference between two connecting areas of a conductor. Likewise when the conductor is stationary, but the flux lines vary, a potential difference will also be induced in the conductor. The magnitude of this potential difference depends on the rate at which the flux changes (Faraday's Law). The direction of induced current acts to oppose the change producing it (Lenz's law)(Bell, 1988). In simple terms, a wire carrying a current will induce current in conductors close by when, either the wire is moved, or the current is changing. In other words when the current is turned on or off, or changing direction. In domestic power supplies the current is constantly changing direction (i.e. alternating). This means the magnetic field surrounding a wire carrying an alternating current has the ability to exert a force on charges moving within the field. This includes charged molecules within the human body.

The Electromagnetic Spectrum

Exposure to manmade EMFs has been increasing steadily since the first power stations began generating electricity in the 1880s. Initial sources produced static or extremely low frequency fields. Frequencies now range from ultra-low frequency fields through mains power frequencies, radio and T.V. frequencies, to the

extremely high frequencies of microwaves. Concerns about possible health effects from exposure to EMFs have also been long-standing. Initial concerns were related to effects from ELF EMFs. More recently there has been public concern relating to exposure from T.V.s and VDUs, microwave ovens and cellular telephones. EMFs have also been used therapeutically from early times. Some examples of current therapeutic uses are TENS machines in physiotherapy and as an aid to healing bone fractures.

There is an inverse relationship between frequency and wavelength. As wavelengths decrease in length, frequency increases. As the wavelength decreases the theoretical potential for interaction with biological systems increases.

Power Frequency Fields

The term power frequency fields is used to denote the frequency used for mains power. This is 50 Hz in New Zealand and most of the world and 60 Hz in the U.S.A.. Consequently it is the frequency most domestic and office appliances use and hence the most common frequency members of the public are exposed to.

In the ELF range the electric and magnetic fields are uncoupled and can be considered separately. Wavelengths are large (about 5000 km at 60 Hz) and do not 'radiate' away from their source (N.R.P.B, 2001).

Direct vs Alternating Current

Electricity is usually transmitted as an alternating current. An alternating current doesn't flow steadily in one direction, as a direct current does, but oscillates backwards and forwards. The number of times the current oscillates (or cycles) per second is called the frequency. One cycle per second has the unit of hertz (Hz). A direct current produces a static EMF whereas an alternating current produces an EMF that changes direction (National Radiation Laboratory, 1996).

Field strengths with examples

In New Zealand, power lines carry voltages ranging from the 230 volts of the normal household supply to transmission lines of up to 220,000 volts. All, except the main feeder line between the South and North Islands, are alternating current supplies operating at a frequency of 50 Hz. Examples of typical magnetic fields levels commonly experienced by the general public range from; 0.1-.05 μT above an electric blanket, 0.05-9 μT near an electrical appliance, to 0.5-80 μT beneath transmission lines (National Radiation Laboratory, 1996)

Safe limits

Whether 50/60 Hz EMFs have an adverse effect on humans has yet to be determined conclusively. The following National Radiation Laboratory guidelines on safe limits have been set on the basis that electric currents induced in the body by 50/60 Hz fields should not exceed those that occur naturally in the body. For the general public, continuous exposures may not exceed 100μT but may be as high as 1000 μT for a few hours. Occupational exposures are limited to 500 μT for the work day but may reach 5000 μT for short periods (National Radiation Laboratory, 1996). Unlike radio waves, ELF fields do not propagate away from their source, but remain in close proximity to it.

Detection of electric and magnetic fields

ELF electric fields may be detected by humans due to the vibration of body hair or spark discharges to grounded objects. Vibration of body hair occurs at 5-10 kV/m (Sagan, 1992). At 50-60 Hz thresholds for perception are around 7-23 kV/m, but some people may detect fields as low as 3-5 Kv/m. At 15-20 kV/m vibration of body hair may reach annoyance levels. The threshold for detection of spark discharges is lower and may be as low as 0.6-1.5 kV/m in some individuals. Spark discharges are less well tolerated than hair vibration and cause irritation in the region of 2-3.5 kV/m (Sienkiewicz et al., 1991).

Sagan (1992) stated that humans can detect magnetic fields only if they are intense enough to produce magnetophosphenes. Magnetophosphenes are faint, visual flickering sensations produced by an interaction of the induced electric current with cells in the retina. They can reliably be produced at 20 Hz above 5 mT and 50 Hz above 15 mT (Lovsund et al, 1979). Perception of magnetophosphenes at power frequency EMFs is generally regarded as unlikely (IARC Monograph, 2002).

Interaction of EMFs with biological materials

The interaction of EMFs with humans and animals is dependant on their size and shape and the electrical properties of the living tissues. At ELF frequencies animals and humans are a great deal smaller than one wavelength. The electrical permitivity of a body is related to the water and electrolyte content. Tissues such as brain, C.S.F. and heart have a higher conductivity than lungs and fat due to their higher water content (Gandhi et al, 2001). Most biological tissues have a permeability similar to that of a vacuum with the exception of those tissues containing minute amounts of biogenic magnetite (Kirschvink et al., 1992).

Radio Frequency Radiation

Radio frequency radiation covers the frequencies of 3 KHz to 30 MHz. Wavelengths range from 1m (at 30 MHz) to 100 km (at 3 KHz). Microwaves cover the area of the electromagnetic spectrum between 30 MHz (wavelength 1 m) and 300 GHz (wavelength 1 mm). Radio Frequency waves have the ability to propagate away from their source and exhibit properties similar to refraction and reflection (Saunders et al., 1991). A RF wave that is used for communication is called a carrier wave. The message it carries is added to this wave in a process called modulation (IEGMP, 2000).

The rest of this section will concentrate on those radio frequencies used by cellular phones.

Cellular Phone Frequencies

Currently available mobile phones use frequencies in the range 800-2200 MHz. Actual frequencies differ between countries. In Asia, frequencies are either in the band 810-935 MHz or 1895-1910 MHz. European countries occupy the 890-960 MHz and 1710-1880 MHz bands and North American bands fall between 824-849 MHz and 1850-2200 MHz. Maximum power outputs are in the 600-1000 mW range for the 800 MHz bands and 10-25 mW for the 1800-2000 MHz bands (Lin, 1997).

Analogue vs Digital Transmission

Cellular phones may use either analogue or digital methods of transmission. As the systems are different their potential for interacting with living systems also differs. The older analogue systems or TACS (Total Access Communication System) are being replaced by digital systems or GSM (Global System for Mobile Communication) in most parts of the world. A third generation of cellular phones, called UMTS (Universal Mobile Telecommunications System) is currently under development and will be available soon. The UMTS system will operate at frequencies of 1885-2010 MHz (IEGMP, 2000).

"In an analogue system the signals applied to the transmission media are continuous functions of the message waveforms. The amplitude, the phase or the frequency of a sinusoidal carrier can be continuously varied according to the voice or the message. In digital transmission systems, the transmitted signals are discrete in time, amplitude, phase or frequency, or in any combination of two of these parameters."(Lee, 1995. p.427.) First generation analogue phones have a power output of 600 mW. The newer digital phones can have a power output as

low as 10 mW.

Cellular Networks

Cellular phones send and receive information via local base stations. The maximum distance a GSM phone can currently operate over is about 35 km so base stations need to be fairly close together. In large cities they may need to be only a few hundred metres apart due to the interference produced by buildings. To increase the number of users that can operate via one base station the system uses a Time Division Multiple Access System (TDMA) that allows each channel to be used by 8 phones at once. Signals are compressed into 4.6 ms parcels and sent in pulses 0.58 ms long. The result is a 217 pulse modulated signal (IEGMP, 2000).

Cell Phones vs Base Station Emissions

The maximum power permitted from a GSM phone is 2W (900 MHz band) and 1 W (1800 MHz band). As the phone only transmits for one eighth of the time the average power is much less. The maximum intensity in the main beam of a base station, 50 m from a 10 m tower is about 5 V/m and 0.02 μT or about 50 to 100 times smaller than that produced from a cellular phone with a 2.2 cm antennae (IEGMP,2000).

SAR (Specific absorption ratios)

Radio Frequency waves penetrate living tissues. Such penetration is measured in SAR. The extent of penetration decreases as the frequency of the wave increases (IEGMP, 2000). Because high intensity RF waves can damage living tissue, a considerable amount of research has been undertaken to identify the absorption ratios in an effort to promulgate safe guidelines for exposure. Core body temperature rises significantly at whole body averaged SAR above 1-4 W/Kg (Elder & Cahill, 1984). Whole body averaged SAR is a single SAR value that represents the size of the SAR averaged over the whole animal. The local SAR represents the SAR in a small portion of the animal (Chou et al., 1996). Measuring absorption in living humans is difficult and research has concentrated on the identification of suitable models.

Lin (1997) used anatomical phantoms to predict SARs from a source with a power output of 600 mW. Power absorbed depended on frequency and antennae length. The higher frequency phones (1800 MHz band) produced a greater absorption rate (Peak SAR of 2.88 W/kg). In the 900 MHz band phones brain absorption rates were typically 1.13 W/kg with peaks of 1.38. The new generation phones, shortly

to be introduced, will operate at even higher frequencies and so produce even greater SARs.

Balzano (et al, 1995) illustrated a typical absorption pattern from a cellular phone held against the ear. The model used was constructed using internal materials with dielectric properties similar to human brain tissue. The energy absorbed was measured inside the head of the model to represent the level of absorption expected in the human brain . The maximum level was recorded just below the ear, 4 cm inside the skull and was 1.1 W/kg with the antennae extended and 1.8 W/Kg withit withdrawn.

The SAR varies greatly according to such factors as; the orientation between the source and the animal, whether the animal is moving, the frequency of the source, and the proximity of the source to the animal. This makes assessment of exposure difficult. Also SAR is a scalar quantity and so can not provide directional information. Direction may be important if the orientation of an induced field is an important factor in producing a biological effect (Chou et al. ,1996).

Microwave Auditory Phenomenon

Humans can hear microwave radiation at an average power density of 1 mW per square cm. It is experienced as a buzzing or clicking sound in, or behind, the head (Lin, 1994). This has implications for those attempting to design a double blind experiment.

New Zealand Exposure Standards

For people exposed to RF EMFs in an occupational setting the SAR limits averaged over the whole body are 0.4 W/kg. Localised SAR are 10 W/kg for the head and trunk and 20 W/kg for the limbs. For the general public the limits for whole body average SAR is 0.08 W/kg with localised SAR being 2 W/kg for the head and trunk and 4 W/kg for the limbs. The exposure limits are higher for occupational exposure as those groups are usually aware of the risk and are trained to minimise it . The general public lacks the training and may even be unaware the exposure is occurring (Standards New Zealand, 1999).

CHAPTER 3
BACKGROUND PHYSIOLOGY AND PSYCHOLOGY

The purpose of this brief section was to orientate the reader as to the physiology and psychology of the parameters that were assessed in this research. It was not the intention to provide a comprehensive review of the literature in these areas.

The Pineal Gland and Melatonin

The Pineal Gland

The pineal is a small organ weighing approximately 50-150 mg in humans and 1 mg in rats and is located in the diencephalon. In mammals the pineal is purely secretory in nature, but in birds and reptiles it also has a photo reception function (Arendt, 1988). The pineal has a rich blood supply. In animals the pineal may increase in size, relative to body mass with increasing distance from the equator (Arendt, 1988). The main hormones detectable in pineal tissue are melatonin and arginine-vasopressin, with the main functions of the gland being linked to melatonin (Cavallo, 1993).

Melatonin

Melatonin is very old phylogenetically and is produced by most animal species (Reiter, 1993). It was discovered in 1958 by Lerner who coined the name melatonin and characterised it as N-acetyl-5-methoxytryptamine (Arendt, 1988)

Melatonin is found in all body fluids. It has a daily rhythm, with levels being high during the hours of darkness and low during daylight hours. The rhythm is entrained by the light/dark cycle but is still maintained in the absence of light, in which case it tends to run to a 25 hour cycle in humans (Wever, 1986). Although amplitudes are lower than in the blood stream similar circadian melatonin rhythms have been noted in the C. S. F., saliva, seminal fluid, eye, ovarian and amniotic fluid (Arendt, 1985).

Although melatonin receptors have also been located in the retina, gonads and uterus, the main binding sites are in the hypothalamus in humans and the hypothalamus and pituitary in animals (Cavallo, 1993). In humans, binding sites exist in the suprachiasmatic nucleus of the hypothalamus but not in the pars tuberalis of the pituitary. Although receptors for melatonin have been found on the surface of cells it is lipophillic so it can freely enter cells through the cell

membrane. Melatonin may execute its effects through calcium or calmodulin.

Functions of Melatonin

Melatonin' s main functions are to provide information about the phase and strength of the day/night cue to organs that are not light-sensitive and also provides seasonal clues as day length alters during the year (Reiter, 1993). It's subsequent effects relate to the importance of light on physiological and behavioural rhythms (Arendt , 1995). These include sleep, temperature and reproductive rhythms. Melatonin provides an important cue for animals that breed seasonally. In some mammals melatonin acts on the hypophysis to inhibit the secretion of luteinising hormone (LH) and follicle stimulating hormone (FSH). This occurs by the inhibiting the release of luteinising hormone releasing hormone (LHRH) from the hypothalamus and also by inhibiting the actions of LHRH on the pituitary (Smith et al, 1983). The anti-gonadotrophic effects described above have not been demonstrated in humans. In young amphibia melatonin acts within melanocytes to produce lightening of the skin (Smith et al., 1983).

The pineal gland works in close association with the immune system and has an imuno-enhancing effect. A review by Maestroni (1993) described a series of experiments illustrating the links between the immune system and melatonin. In mice, the administration of a beta blocker in the evening depressed primary antibody and mixed lymphocyte response to a T-dependent antigen. They concluded that the "immunoenhancing and anti stress action of melatonin appear to be restricted to responses against T-dependant antigens "(p.3). Pinealectomised mice showed a reduced ability to mount a humoral response against sheep red blood cells. This effect was reversed by administration of melatonin. T-helper lymphocytes are target for melatonin. These cells produce gamma interferon and Interleukin 2 , the secretion of which has been reported to be effected by melatonin. They conclude that these properties of melatonin and the sensitivity of the pineal to immunologic substances such as cytokines or thymic hormones suggests an interaction between the pineal and the immune system.

A less well established function is a proposed oncostatic role in that melatonin is involved in the scavenging of cancer promoting free radicals. Carcinogenesis has been shown to be suppressed by antioxidants that scavenge free radicals. The most toxic of the free radicals is the hydroxyl radical, one of the oxygen-based free radicals. Although free radicals occur as normal products of aerobic metabolism they damage cells and tissues and modify DNA. Defence mechanisms developed

by the body to combat free radicals include antioxidant enzymes, tocopherol, uric acid, ascorbate, mannitol, glutathione and melatonin. The ability of melatonin as a hydroxyl radical scavenger depends on the acetyl group on the side chain and the methyl group located at position 5 on the indole section of the molecule (Reiter et al., 1994). For this reason the precursors of melatonin lack the hydroxyl scavenging ability of melatonin (Poeggler et al., 2002; Bromme et al, 2000)

Melatonin has been found to inhibit the growth of breast cancer cells in vitro. It has also been shown to influence tumour growth in many other cancers such as melanoma, and cancers of the lung, prostate, colon, ovary, bladder and uterus (Blask, 1993). Ram et al (2000) ascribed a differential response of MCF-7 human breast cancer cells to the growth inhibitory effects of melatonin and concluded that the primary growth inhibitory effects of melatonin occur via the membrane associated G-protein coupled mt1 melatonin receptor.

Circadian rhythm of melatonin

The circadian rhythm of melatonin is generated by the suprachiasmatic nucleus of the hypothalamus and entrained by the light/dark cycle (Arendt, 1985). Mammals exhibit a night time increase in blood and pineal levels of melatonin of 5 to 20 fold or more over day time levels, depending on the species (Reiter,1993). In humans about 70% of melatonin is produced in an eight hour period from 10 p.m. to 6 a.m.(Smith et al., 1983). This high night/low day pattern occurs regardless of whether the species is nocturnal or diurnal. Also in most species, except humans and pigs, the length of the dark phase shows a positive correlation with night length (Arendt, 1988). The secretion rate of melatonin into saliva in humans is approximately 0.5-0.9 pmol per hour in the daytime and 1.0-2.4 pmol per hour during the night (Vakkuri, 1985). Blood melatonin levels vary from approximately 7-14 pg/ml during day light to 20-85 pg/ml at night (Waldhauser and Dietzel, 1985).

The normal human melatonin rhythm is intra individually consistent in amplitude and circadian profile, "rather like a hormonal finger print" Arendt, 1995, p.209). In humans, blood melatonin concentrations rise slowly from the onset of darkness to reach a peak in the middle of the night. From this point, levels fall slowly until they reach daylight levels at about dawn. Waldhauser and Dietzel's (1986) description of the circadian pattern of melatonin concentrations in the bloodstream of 8 young men is typical of levels reported elsewhere in the literature. Samples were taken at 20 or 60 minute intervals over 24 hours. Levels

were generally around 10 pg/ml during the day with a rapid rise occurring between 9 and 11 p.m., coinciding with lights out. This rise peaked at 2-3 a.m. with a blood concentration of 50-60 pg/ml., then fell slowly to 6 a.m., after which they dropped rapidly to daylight levels.

Although this describes the general trend, considerable inter-individual variability exists. A person may have a pattern that varies considerably from the norm. Levels may be much higher or lower and the night peak may be phase advanced or phase delayed in comparison with reported norms (Waldhauser and Dietzel, 1986). Bergiannaki et al (1995) identified high and low melatonin excretors. The distinguishing point separating the two groups was given as 0.25 nmol/l. of urinary melatonin. This inter-individual variability means that where possible subjects should be used as their own controls or profiles should be matched for controls and experimental subjects in studies where variation is likely to effect the research outcome. There has been little research on stability of the profiles of melatonin in humans. Recently Honma et al. (1997) investigated the stability of the melatonin profile in 4 young females. They concluded that a shift of the melatonin peak can be regarded as a phase shift, rather than a day-to-day variation when it is greater than 1.4 hours. The ascending phase was described as less stable than the descending phase.

Synthesis, Release and Metabolism of Pineal Melatonin

Melatonin is produced in the pinealocytes from tryptophan in a multistep process. Tryptophan is first converted to 5-hydroxytryptophan and then to serotonin. Serotonin is converted into melatonin via N-acetyl serotonin. Melatonin is released into the capillaries of the pineal and from there it enters the bloodstream. Secretion into the blood occurs passively, the amount being determined by the rate of synthesis (Smith et al., 1983, Trinchard-Lugan, 1989). Secretion into saliva also occurs (Vakkurri et al., 1985). Serotonin NAT is the rate-limiting enzyme and it's activity increases 30 to 70 fold at night (Arendt, 1988). HIOMT (hydroxyindole-o-methyltransferase) is also more active at night but to a lesser extent.

Control of the synthesis of melatonin

Post-synaptic sympathetic ganglia release noradrenalin which binds to Beta-adrenergic receptors on the pinealocyte membranes. This binding triggers the activation of the membrane-bound Gs proteins which in turn activates adenylate cyclase. This results in a large intracellular increase in cyclic AMP which, in turn, causes the enzyme cascade in which serotonin is converted to melatonin. The

process is mediated by the rate limiting enzyme N-acetyl transferase (Figure 3.1).

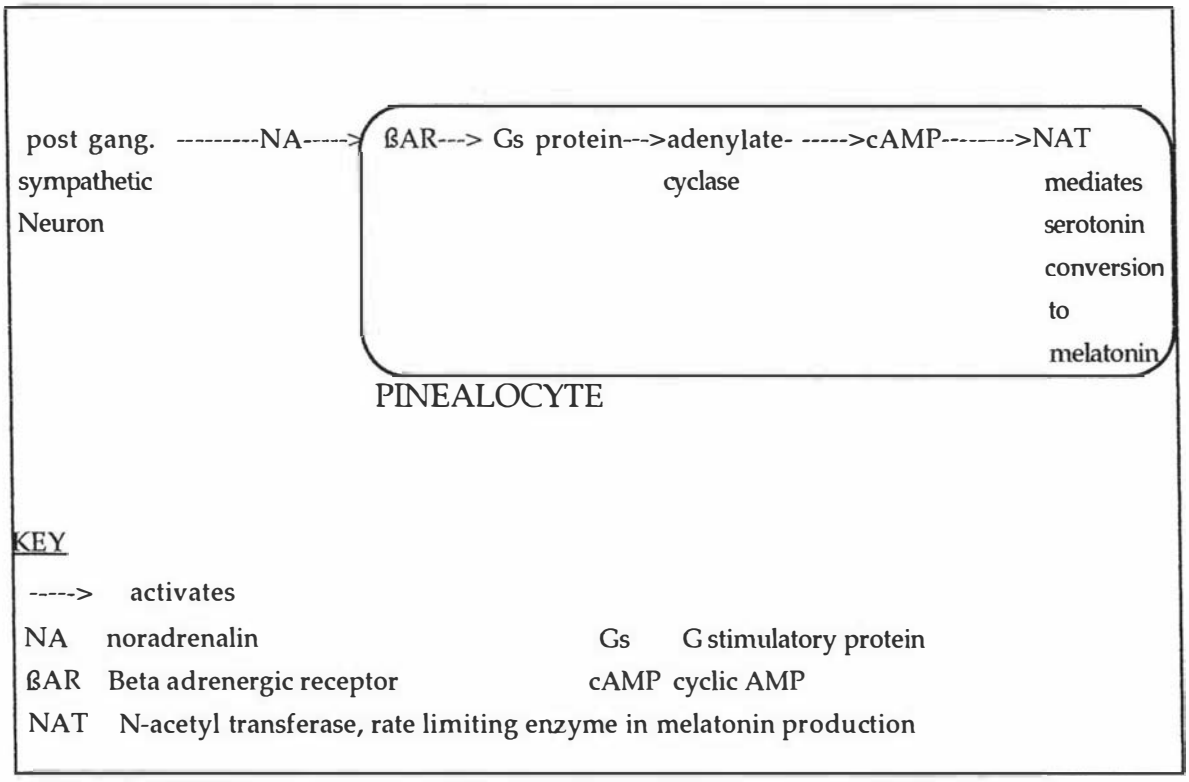


Figure 3.1 : Control of the synthesis of melatonin in the Pineal

Metabolism of Melatonin

Melatonin is metabolised in the liver. Hydroxylation occurs at position 6 and conjugation occurs with sulphate and sometimes glucuronic acid (Smith, et al., 1983). Most (99%) is excreted as 6-hydroxymelatonin sulphate, while about 1% is excreted as melatonin. Vakkurri et al. (1985) described the half life of orally-administered melatonin as 41 minutes for serum and 38 minutes for saliva.

Mediation of the circadian rhythm of melatonin

The circadian rhythm of melatonin release is mediated neurally via the retinohypothalamic tract. Light entering the retina has an inhibiting effect on the suprachaismatic nucleus of the hypothalamus. Multisynaptic pathways link the hypothalamus with the pineal (see Fig. 3.2)(Reiter, 1993). Retinal receptors send information to the suprachiasmatic nucleus and paraventricular nucleus of the hypothalamus. Links go to the upper thoracic, preganglionic fibres, post sympathetic neurons in the superior cervical ganglia then to the pinealocytes in the pineal gland (Reiter, 1993).

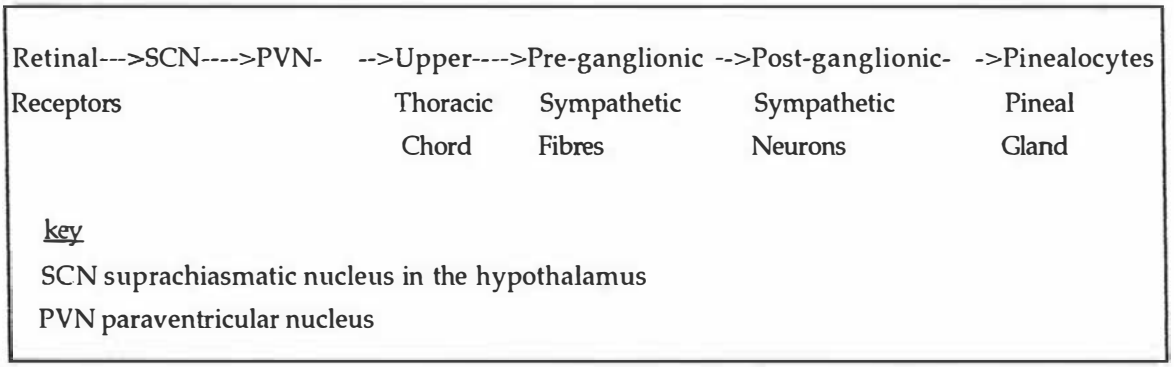


Figure 3.2 : Neural pathways linking the retina with the pineal

During the day the release of melatonin from the pineal is inhibited due to the deactivation of this pathway. At night the inhibiting effect of light is removed and pineal and blood melatonin concentrations rise (Reiter, 1993).

Factors Altering Melatonin Production

Light

As described previously, light acts as a zeitgeber (or time keeper) entraining the circadian rhythm of melatonin secretion. In the absence of light human circadian rhythms tend to follow a pattern slightly longer than 24 hours (Wever, 1985).

In 1972, Klein and Weller reported that exposing rats to light at night produced a drop in melatonin synthesis with the mechanism being the rate-limiting enzyme N-acetyl transferase. These findings were repeated by several other research teams. However, attempt to replicate the findings in humans failed. In 1985, Reiter et al. concluded that ordinary night lighting had little effect on human melatonin levels. Light needed to be two to three times normal room light levels to inhibit the nocturnal rise in melatonin. Lewy et al. (1980) found light of 2500 lux suppressed nocturnal melatonin whereas light of 500 lux, well above the intensity of a normal bulb produced no effect. Wever (1985) considered the threshold for suppression to be even higher at 3000 lux. In 1989, McIntyre and colleagues managed to produce significant reductions in plasma melatonin during exposure to light of 400 or 600 lux intensity between 2400 to 0300 hours. Maximum suppression was obtained after one hour with no further increase recorded. They were unable to replicate their prior research in which they produced suppression at 200 lux. Nathan et al. (1999) described a 12 to 18 % suppression of plasma melatonin by a white light of 200 lux. Exposure took place between midnight and 1 a.m. Following nocturnal suppression of melatonin by exposure to light some

people exhibit a rebound increase in melatonin levels (Beck-Friis et al. 1995).

Some animal species show more sensitivity to the effects of light and suppression occurs at lower intensity levels. For example, in rats 50% suppression occurs at 10 lux and 500 lux results in complete suppression (Minnemen et al., 1974). However, in all mammals the melatonin peak occurs during the dark phase regardless of whether the animal is day or night active. In rodents even a brief period of light in the dark phase will result in a steep decline in melatonin levels within the space of 2-3 minutes (Reiter, 1987).

The particular wavelengths of light producing the greatest inhibitory response on melatonin secretion varies between species. Brainard (et al., 1985) researched wavelengths causing the greatest suppression in humans. Using three subjects they gave the respective depression of plasma melatonin as; -12%(448 nm), 26% (476 nm), 64% (509 nm), 20% (542 nm), 16% (574 nm) and -8% (604 nm) . Morita et al. (1997) exposed 4 subjects to red, green or blue light of 1000 lux or 2500 lux in the morning. They reported a suppression of melatonin and rise in core temperature using a green light but only when the intensity was high (2500 lux). They reported no suppression at other wavelengths for 1000 or 2500 lux and concluded that the behaviour of melatonin and core temperature differs according to the wavelength of the light. The intensity of light needed to produce the effect was different for morning compared to evening administration. At night 1000 Lux promoted an increase in core temperature and a decrease in melatonin levels where as it required 2500 lux to achieve that effect between 4 and 9 in the morning for green light.

Thapan et al. (2001) observed the effect of light at varied wavelengths between 424 nm and 548 nm. They concluded that at each wavelength suppression increased with increasing light intensity. They also suggested a non-rod , non-cone photoreceptor having a primary role in light indued melatonin suppression. This suggestion is controversial and other possibilities have been suggested such as the S-cone pigment providing input into the circadian system (Rea et al. 2002).

The normal circadian pattern can be phase delayed using a changed light/dark cycle. Deacon and Arendt (1994) exposed six subjects to a light/dark cycle that was phase delayed by two hours a day for 3 days. Light of 1200 lux was on from 2000-0200 hours, then 2200-0400 hours, then 2400-0600 hours. Each period of light was followed by 8 hours of darkness. They were able to induce phase delay shifts of

2.67, 2.35 and 1.97 hours for urinary melatonin metabolites, plasma melatonin and salivary melatonin concentrations, respectively. In a similar study by Lewy et al. (1985) dusk was phase advanced for one week then dawn was phase delayed for a week. Advancing the dusk advanced the melatonin rhythm while delaying the dawn delayed the melatonin rhythm.

Seasonal variation

Circannual melatonin rhythms have been noted by several investigators (Arendt, 1988). Blood concentrations are highest in summer and winter and lowest in spring and autumn. The nocturnal rise of melatonin is phase advanced in summer compared to winter (Matthews et al, 1991), with a time difference of 1-2 hours (Arendt, 1995).

Changes in melatonin levels with age

The diurnal rhythm in melatonin present in adults is not present at birth. In a study by Kennaway et al. (1992), babies didn't show a rhythm in the urinary metabolite 6-sulphatoxymelatonin until 9-12 weeks of age. Before that time melatonin levels were very low. Premature infants who had been in a constantly lit hospital nursery had a further 2-3 week delay before a rhythm appeared which may have been due to the inhibitory effects of the hospital lighting. By 24 weeks of age the urinary metabolite had reached 25% of adult levels, rising to 50% at one year. Initially the night rise did not begin until 2 a.m. but by 18 weeks the rise began at 8 p.m. lasting through until 10 a.m. According to Waldhauser and colleagues (1993) melatonin reaches its highest levels at 1-3 years then decreases markedly until adolescence. Following a fairly stable production of melatonin during adulthood, levels decline in old age.

In 1909, Marburg (cited in Waldhauser and Dietzel, 1985) hypothesised that the pineal produced a substance that inhibited human sexual activity in the prepubertal period. As the child grew a continued reduction in gland output led to diminishing inhibition and the eventual onset of puberty. Although there has been considerable research linking melatonin production and breeding status in seasonally breeding animals there is little evidence of such a link in humans. Tetsuo et al. (1982) found no correlation between melatonin urinary metabolite excretion rates and age in children, the rates being similar to those in adults. However, no allowance was made for the differing body weights between children and adults. Such an allowance would result in a much higher production

of melatonin per unit of body weight with a decrease in output per weight unit, with increasing age. Tetsuo and co-workers (1982) did find a significant increase in melatonin metabolite excretion at the onset of breast development in girls. No similar rise was observed in males. However, Silman et al. (1979) had previously described a much higher daytime level of melatonin in prepubertal boys which the study linked to suppression of gonadal activity. The previously reported occurrence of precocious puberty in boys with pineal tumours adds weight to the concept of a pineal gonadal interaction (Kity and Altechule, 1954. cited Arendt, 1988). Although total melatonin production may not change with age, in a study of children and young adults, Waldhauser and Dietzel (1985) noted a considerable decline in night time serum levels with increasing sexual maturity. Serum melatonin dropped from 210 ± 35 pg/ml in 1-5 year olds to 133 ±17 pg/ml in 5-11 year olds and 46 ±6 pg/ml in young adults. Nocturnal blood concentrations of melatonin were negatively correlated with changes in Luteinising hormone. Whether this decline continued in adulthood was not established. Morning melatonin levels did not alter with age. Although studies suggest melatonin levels decrease as sexual maturation approaches the link may not be causative but both variables may be responding to a third causative factor (Arendt, 1988). Ohashi et al. (1997) studied young and elderly groups and concluded there was no significant difference between the groups in terms of total melatonin produced per day.

Changes over the Menstrual Cycle

The role of melatonin in the female reproductive cycle varies between species. In humans, Wetterberg et al. (1976) noted a rhythm in melatonin concentration which coincided with the menstrual cycle. Lowest levels occurred at the time of ovulation with a subsequent increase in levels until the late follicular phase when levels dropped slightly. Levels rose again at the time of menstruation. Other studies (Brezezinsky et al., 1988) have not supported these findings with melatonin levels varying little as a function of stage in the menstrual cycle. However, it must be noted that the Brezeinsky et al. study used a small sample. They did, however, note a significant elevation of plasma melatonin in women with hypothalamic amenorrhea. In seasonally breeding animals, melatonin has a role in the onset of oestrus. For example, melatonin is fed to ewes to advance oestrus by 6-8 weeks resulting in earlier lambing (Kennaway et al., 1982).

Temperature

Core body temperature has a cycle which is inverse to that of melatonin i.e. when melatonin levels are at the peak, core body temperature is at its lowest . This inter-relationship may be causal as exogenous melatonin depresses body temperature and bright light causes an increase in body temperature but a decrease in melatonin (Arendt, 1995).

Changes with Activity and Rest

In a 1989 study, Rivest et al. found nocturnal melatonin levels were higher in men confined to bed (rest) than those who were active. Conversely, Arendt (1995) described a small but rapid reduction in levels on lying down.

Effects of Diet and Fasting

In 1989 Rojdmark and investigated the effect of a two day fast on serum and urine melatonin in seven normal subjects. They found a significant decrease (19%) in nocturnal serum melatonin but no alteration in urinary excretion as a result of fasting. It was suggested that pinealocytes may require a certain level of glucose to function normally. This was supported by a return to normal melatonin secretion in fasting subjects given glucose supplementation. Arendt et al. (1982) reported a small, non-significant reduction 24 hour melatonin concentration in subjects who had fasted for 14 hours from breakfast. Either fasting has to be for a longer period than 14 hours or the timing of the fast effected results.

Drugs

Various drugs have been reported as having an effect on the rhythm of melatonin production. Although the effects of ethanol on melatonin levels has not been extensively investigated in man the introduction of exogenous melatonin has been shown to induce ethanol drinking in rats (Murialdo et al.,1991). In humans, a study of chronic alcoholics found day melatonin levels were raised significantly (Murialdo et al, 1991). This was contrary to a reduction in 24 hour melatonin reported by Borg (1983, cited by Murialdo, 1991). A single dose of ethanol has been reported to blunt the nocturnal melatonin peak. In all the above studies subject numbers were low.

Arendt (1985) found the ß-adrenergic antagonist Atenolol abolished the 24 hour urinary melatonin rhythm. Ten human subjects were given a single 100 mg dose of atenolol and plasma and urine was tested for melatonin content for 24 hours.

This took place in July (Northern Hemisphere). Cowen et al. (1983) had previously found a similar abolition of the melatonin peak. Whether this reflected a change in the release or clearance of melatonin was not established. Other drugs acting on the CNS, such as antidepressants, alcohol and caffeine also effect melatonin secretion. Agents that stimulate melatonin release include isopropterenol and lindane.

Disease and Illness

Various illnesses have been reported as having an effect on melatonin plasma levels. The most frequently researched of these are malignancies. Touitou et al (1985) found abnormal daytime levels in 65% of their elderly cancer patients with most subjects showing a decrease in melatonin plasma levels. Elderly controls had a melatonin levels consistent with the general population. Higher levels of melatonin correlated with autopsy findings of "cancer, chronic renal failure, cardiovascular disease, biological inflammatory syndrome and diabetes. Low levels correlated with neurologic disease, tobacco or alcohol addiction"(p. 135). Pineal cancer has also been linked to elevated melatonin levels. Cavallo (1993) investigated the relationship between elevated melatonin levels and pineal tumours and found no relationship.

In Cushings syndrome alterations in melatonin rhythm and concentrations have been noted, but such changes have not been found in Cushings disease (Rivest et al ,1989). Thyroid disease also has been linked to abnormal melatonin levels. Thus, Rojdmark et al (1991) reported elevated nocturnal serum melatonin levels (both peak height and total) and greater urinary excretion in hypothyroid patients. On the other hand, hyperthyroid patients had normal levels but their peaks were phase-advanced. Decreased plasma melatonin levels were reported by Puy et al (1993) in patients with recurrent acute intermittent porphyria attacks.

Clinical depression is often accompanied by disturbances in biological rhythms. The finding of low melatonin concentrations in patients suffering depression has led to a suggestion that melatonin levels may be used as a clinical marker of depression (Claustrat et al 1984, Cavallo, 1987). However not all studies support such findings (Thompson, 1988). Most studies used small sample sizes and large inter-individual differences in melatonin concentration, coupled with the difficulty of clearly defining clinical depression, brings the validity of the results into question. This means the use of melatonin concentrations as a clinical tool for diagnosing depression is unlikely.

Crossing Time Zones

Crossing time zones produces a temporary desynchronisation of the biological clock, commonly known as jet lag. The effects are more severe when travelling in an easterly direction. For example, Fevre-Montange et al. (1981) studied 5 males who took a 7 hour time change in a westerly followed by the same time change a month later in an easterly direction. Westerly travel produced a significant decrease in both day and night melatonin levels. This produced 6 hours of sleep deprivation and partial adaptation occurred the following day. In the easterly travel, total desynchronisation of the melatonin rhythm followed with 33 hours of sleep deprivation. Subjects also reported an increase in depression and anxiety scores and considerable sleep disruption. The above knowledge has led to the successful treatment of jet lag with orally administered melatonin (Arendt et al,1986). Eight subjects took 5 mg of melatonin at 1800 local time for three days prior to their departure across eight time zones in a double blind experiment. Their jet lag was self-rated as significantly less severe than that of the controls. An in depth discussion of the effects of jet lag on melatonin concentrations is outside the scope of this review.

Electromagnetic fields

The effects of EMFs on the body's production of melatonin is discussed in a following section.

Attention and Memory

Attention

Attention is defined by Chaplin (1968. P 42) as "the process of preferentially responding to a stimulus or range of stimuli", although a universally accepted definition has yet to appear in the literature (Lezak, 1995). Attention is generally viewed as "a system in which processing occurs sequentially in a series of stages within different brain systems involved in attention "Lezak, 1995 p.39).

Five different types of attention have been identified. They are as follows:
Sustained attention which is the ability to maintain concentration on a task.
Focused attention which is the ability to attend to the task at hand.
Selective attention which is the ability to exclude distractions.
Divided attention which is the ability to respond to two or more tasks at once.
Alternating attention which is the ability to switch from one task to another (Lezak, 1995).

The neuroanatomy of attention is very complicated. The main cortical areas involved are the inferior parietal cortex which is involved in spatial selective attention plus the frontal cortex which has a role in response selection and control, and also in sustained attention (Lezak,1995). Sustained attention also involves the frontal and cingulate cortex and basal ganglia (Marrocco and Davidson, 1998). The sub-cortical thalamic nuclei have a function in selective attention. The arousal and activation functions of the mesencephalic reticular system are also important in attention. Limbic system structures involved include the amygdaloid and septal nuclei which help establish salience (Cohen et al., 1998).

The main neurotransmitters involved in attention are acetylcholine and noradrenalin. Other neurotransmitter systems implicated include dopamine, histamine and serotonin. Noradrenalin and acetylcholine play key roles in pure attentional tasks and tasks that don't include changes in arousal or perceptual processing (Marrocco and Davidson, 1998).

The attention task used in this research involves the ability to pick out a target among a page of distractors. This requires the abilities of sustained attention and visual search. Theories of visual search include the notion of 'pop-up' in which attention is suddenly drawn to a target. According to Nakayama and Joseph (1998) pop up is based on a distributed spread of attention over a whole array.

This spread of attention combined with a narrowing appears a contradiction but can be explained in that two distinct processes are occurring simultaneously. Easy visual search, such as the location of a number or word on a page, is comprised of a "global attentional allocation to the whole array (useful to do the rapid search task) followed by a narrowing of attention to the target (Nakayama and Joseph, 1998, p.291)

Memory

"Memory is a process that results in a relatively permanent change in behaviour. It is never observed and is always inferred" Kolb and Whishaw, 1985. p.476). There is no one brain 'memory centre' that houses memories. Memory is a function of several brain systems working together (Emilien et al., 2004). However, different parts of the brain have more importance for certain types of memory. For example, the inferior temporal lobe is important for facial recognition.

Lesion studies from humans and animals have provided information on brain systems involved in memory. The role of the hippocampus in memory has been well known. Damage to the hippocampus results in mild amnesia, but damage that includes the entorhinal and parahippocampal areas results in severe amnesia. Lesions to the temporal lobes produces deficits in long-term memory (Kolb and Whishaw, 1985). The medial temporal lobe and medial thalamic structures work together in the establishment of new memories (O'Connor and Morin, 1998). Parietal lobe lesions result in deficits in short-term memory, particularly the ability to recall digits. Frontal lobe lesions are not associated with specific memory deficits, but effect the ability to resist interference (Kolb and Whishaw, 1985). The prefrontal role in working memory is in the mediation of problem solving strategies. Tasks that require subjects to rehearse verbal material activates a part of the parietal cortex whereas different parietal areas are activated during visuospatial processing (Marrocco and Davidson, 1998).

There are five processes involved with memory. The first is attention. The second involves encoding the information so it can go into storage (the third process). Memories are stored in two primary formats; as declarative memory for language accessible information and as habits or procedural memory that is non-language based. Once memories are stored they are consolidated. The last process is retrieval (Emilien et al., 2004).

There are three types of memory. Registration or sensory memory holds large amounts of information for 1-2 seconds in sensory store. Short term memory lasts a few seconds to one hour and includes working memory. Long-term memory or secondary memory refers to the ability to store information and includes semantic, reference and episodic memory (Lezak, 1995; Emilien et al., 2004).

The tasks used in this research involve working memory. The concept of working memory includes a capacity for performing mental operations. The model was formulated in the 1970's by Baddeley and Hitch (cited Gazzaniga et al, 1998) and is constructed of a central executive that controls the two subsystems of a phonological loop and visuospatial sketch pad. The phonological loop is a system for processing language. The visuospatial sketch pad provides for visuospatial processing . Working memory is a form of declarative memory as the processing goes on in consciousness (Eichenbaum and Cohen, 2001). The central executive of Working Memory is associated with the prefrontal cortex which also has a role in problem solving, personality, effect, motor control and language processing (Eichenbaum and Cohen, 2001).

Central Nervous System Structures involved in the Control of Heart Rate and Blood Pressure

In the control of heart rate, tonic vagal discharge at rest is initiated by the nucleus ambiguus in the medulla (the Cardiac inhibitory centre). Tachycardia is due to noradrenergic sympathetic output and bradycardia to stimulation by cholinergic vagal fibres (Opie, 2004). Stimuli that increase the heart rate also increase blood pressure, those that decrease the heart rate also decrease blood pressure. Stimuli that increase heart rate include; emotional factors such as excitement or anger, exercise, hypoxia, thyroid hormones, noradrenalin and adrenaline, and fever. Stimuli decreasing heart rate include input from baroreceptors in the arteries, expiration, fear, grief and increased intracranial pressure (Ganong, 1985).

The main control of blood pressure (B.P.) is exerted by the vasomotor neurons in the medulla. The vasomotor area receives excitatory inputs from the cortex via the hypothalamus, from pain pathways and the carotid and aortic chemoreceptors. Inhibitory input comes from the cortex via the hypothalamus, the lungs and the carotid and aortic venous baroreceptors (Ganong, 1985). Hypoxia and hypercapnia directly stimulate the vasomotor area.

An increase in B.P. stimulates baroreceptors in the aortic arch and carotid sinus and this information is relayed to the nucleus solitarius in the brain. A signal is sent to the vagal nucleus and vagal stimulation and sympathetic inhibition act to decrease heart rate and force of cardiac contraction (Opie, 2004). Low B.P. results in baroreceptors activating an adrenergic response which causes vasoconstriction and an increase in heart rate.

The heart pumps against peripheral vascular resistance which is influenced by the autonomic nervous system (acetylcholine and nor adrenaline) and local messengers such as adenosine and nitric oxide (Opie, 2004).

Cardiac contraction and relaxation are caused by an intracellular calcium cycle. A small amount of calcium enters the heart cell during depolarisation. This triggers the release of more calcium from the sacrcoplasmic reticulum. A wave of depolarisation sweeps along the T tubule opening the calcium channel in the membrane allowing calcium ions to cross the membrane. The cytosolic calcium increases causing myocardial contraction. Relaxation occurs when the calcium

uptake pump of the sacroplasmic reticulum and activity of the sodium calcium exchanger act to decrease cytosolic calcium (Opie and Bers,2004)

CNS Structures involved in Body Temperature Control

The regulation of body temperature is largely under autonomic control. Body temperatures are maintained within narrow limits with heat production being balanced by heat losses (Stitt, 1993). Even small displacements from these limits induce large effector responses (Jessen, 2001).

In the body, heat is produced by metabolic processes, muscle action and the assimilation of food. Heat losses occur via radiation, conduction, waste elimination and the vaporisation of water from the skin and respiratory system. Thermoregulatory mechanisms include behavioural, endocrine, somatic and autonomic mechanisms (Ganong, 1985).

Reflexes activated by cold are controlled by the posterior hypothalamus which is mediated by serotonin. Temperature regulating mechanisms acting to increase heat production include shivering, an increase in hunger, increased secretion of noradrenalin and adrenaline and increase in activity of the animal. Actions reducing heat loss include; horripilation, curling up and vasoconstriction (Ganong, 1985).

Reflexes activated by heat are controlled from the anterior hypothalamus and are mediated by norepinephrine. Cutaneous vasodilation, sweating and increased respiration all increase heat loss. Decreased appetite and inertia cause a decrease in heat production (Ganong, 1985).

Other possible thermosensitive sites include the spinal chord and it is possible that thermo sensors are spread throughout the trunk. Spinal chord thermosensors are as responsive to warming as the hypothalamus but are less responsive to cooling (Jessen, 2001).

CHAPTER FOUR
A REVIEW OF THE LITERATURE

In 1896, D'Arsonval found that a time-varying current could stimulate the retina and produce phosphenes and flickering. Phosphenes appear as a flickering white or blueish light over the whole field of vision (Sienkiewicz, et al. 1991). Lovsund et al. (1979) exposed subjects to fields of 10-45 Hz at intensities of 0-40mT. Thresholds producing phosphenes were found to be frequency dependent with a minimum threshold of 10mT at 20 Hz.

The first modern studies on the effects of EMFs on humans were carried out in Russia in the 1960's. Asanova and Rakov (cited Gamberale, 1990) surveyed the health of 500 workers who were exposed to electric fields of up to 26 kV/m from high voltage switch yards, . They found functional problems in the nervous system and changes in haematological, EEG and ECG parameters. Workers also reported somatic complaints such as headaches, irritability, dizziness, nausea, sleep and libido problems, and loss of appetite. The research was successfully replicated by other Russian researchers but subsequent attempts at replication by western scientists failed. It was suggested that the Russian effects may have been caused by extraneous variables such as the extensive use of solvents in Russian switch yards. A number of U. S. studies followed the Russian reports. Epidemiological studies, such as those carried out by Wertheimer and Leeper (1979, 1982) and O'Connor (1993), raised concerns in the west regarding possible links between EMFs from power lines and increased rates of cancer, especially childhood leukemias. The Wertheimer and Leeper studies also highlighted an association between pre-menopausal breast cancers and exposure to power frequency EMFs.

ELF fields, such as those produced by 50 or 60 Hz A.C. household appliances, have also been linked to health problems. For example, electric blankets have been linked to increased rates of testicular cancer and miscarriages but it is uncertain whether those effects were due to heating effects or to the electromagnetic fields *per se*. Not all reported effects of EMFs have been negative. In human medicine, EMFs have been used to aid bone healing (Polk, 1994). Lai and Singh (1997) produced an increased rate of RNA transcription using 60 Hz pulsed fields. Enhanced regeneration of the sciatic nerve in rats was reported following their exposure to EMFs (Kanje et al., 1993).

The main focus of current research covers the areas of: the mechanism of interaction between EMFs and living cells and tissues; effects on the developing embryo; effects on neuroendocrine and nervous system functioning; EMF immune system interactions; regulation of cell growth and tumour production; alterations in gene expression; and possible beneficial therapeutic effects (Adey, 1993).

The following review summarised current findings in the literature on possible interactions between radio and power frequency EMFs on; the pineal gland and melatonin production; heart rate and blood pressure; and the cognitive parameters of attention and memory. Possible heating in the head consequent to cellular phone exposure was also explored. The review concentrated on laboratory studies but epidemiological studies have also been included. Possible mechanisms by which EMFs could effect living systems were explored.

The Effects of EMFs on the Pineal Gland and Melatonin Production

The Wertheimer and Leeper studies (1982) led to Stevens (1987) proposing his melatonin hypothesis. He postulated that ELF EMFs suppress pineal melatonin production which, in turn, leads to an increase in breast carcinogenesis. (The melatonin hypothesis is discussed more fully in section 1.3.) The possibility that melatonin could provide a link between power frequency EMFs and cancer stimulated further research in the area.

In 1993, Reiter made the statement "The circadian rhythm of melatonin production .....in the mammalian pineal gland is modified by visible portions of the electromagnetic spectrum,...and reportedly by extremely low frequency (ELF) electromagnetic fields as well as by static magnetic field exposure"(p.394). The suppression of melatonin production by light has certainly been well documented. Even brief bursts of light of less than a second have been demonstrated to inhibit the formation of melatonin in animals (Reiter, 1985). In humans, a light of 2500 lux is required to suppress melatonin production (Lewy et al., 1980), though in susceptible individuals suppression has been reported with levels as low as 200 lux (MacIntyre et al., 1989). The possibility that extremely low frequency EMFs may also suppress melatonin production is a much more tentative hypothesis.

Power Frequency EMFs

Studies on animals

Studies on the effects of power frequency EMFs on melatonin production in animals have produced conflicting results. Some studies have reported suppressive effects on the formation of melatonin in rats (e.g. Martinez et al., 1992; Kato et al., 1993 &1994; Loscher et al,1994; Selmaoui et al., 1995; Mervissen et al., 1996; Bakos et al., 1997; Rosen et al., 1998)(Table 4.1). Other similar studies on rats, often repetitions by the same research teams, have failed to find significant effects (e.g. Kato et al., 1994; Grota et al., 1994; Bakos et al., 1995; Loscher et al., 1998; John et al., 1998; Bakos et al., 2002)(Table 4.2). Studies on other species, ranging from hamsters to sheep and baboons, have also produced conflicting results (Table 4.3).

In an early study Wilson et al. (1981), investigated the effect on rats of chronic, long term exposure (over 3 weeks) to 60 Hz electric fields and reported an abolition of the nocturnal melatonin peak. The effect did not appear to have a

dose-response effect above the threshold of 2 kV/m. The rats returned to the normal melatonin cycle a few days after the fields were removed. This research was repeated by the group in 1986 with similar results. Yellon (1994) also reported a suppression effect on melatonin from exposing Djungarian hamsters to a 0.1 mT, 60 Hz horizontal magnetic field for 15 minutes.

Kato et al. (1993) exposed rats to a circularly polarised 50 Hz field for 6 weeks and found a significant reduction in melatonin for the groups exposed to a field of over 1 μT. Differences in levels were not found between the groups tested at 1, 5, 50 or 250 μT so the response did not appear to be dose-dependent. Dim red lights of <0.007 lux were used in the research though a reason was not given. Later experiments at 50 Hz, 1 μT for 6 weeks also demonstrated a reduction in nocturnal melatonin which returned to normal one week following cessation of exposure (Kato et al., 1994).

Selmaoui and Touitou (1995) reported a suppression on melatonin levels from exposure to a 100 μT, 50 Hz sinusoidal EMF but also found an effect with a 10 μT field. In contrast to Kato (1994), Selmaoui and Touitou concluded that both duration and intensity of exposure were important. They had found a reduction in pineal NAT and concluded that a sinusoidal EMF alters melatonin via an inhibition of pineal NAT activity. However, pineal HIOMT activity remained unaltered. Rosen et al. (1998) observed the effect of a 60 Hz, 50μT vertical EMF on isolated rat pineal glands with a view to ascertaining the mechanism by which the suppression may take place. They concluded the suppression they found was due to the EMF altering the structure of the ß- adrenergic receptor thus inhibiting binding of the ligand.

Other studies have not supported the above findings. Bakos et al. (1995) exposed 20 rats in metabolic cages to 50 Hz vertical, 5 or 500 μT fields for 24 hours. No change was found in the urinary metabolite of melatonin, either during, or, following exposure. In a later study, Bakos (2002) used a longer exposure period of 8 hours a day for one week with a field intensity of 50μT, or 100μT and obtained the same result. In an extensive study John et al. (1998) exposed rats for 20 hours a day for 10 days, or 6 weeks to a 60 Hz, 1 mT field and reported no effect on urinary aMT6s (Table 4.2).

An interesting study by Lee et al. (1993) looked at the effect of EMFs on melatonin patterns and puberty in Suffolk lambs. The aim of the study was to

reproduce the field strength found at the edge of the right-of-way under a 500 kV transmission line. Ten lambs were held for 10 months under a transmission line. A further study in 1995 replicated the earlier research with 15 ewe lambs. No effect was found on serum melatonin patterns or puberty in either study. The EMF at 'sheep level' was 60 Hz, 6.3 kV/m and 3.77 μT. In a rare study on primates, Rogers et al. (1995) exposed baboons to 60 Hz, 50 μT (6 kV/m) or 100 μT (30 kV/m) fields for 6 weeks and found no effect on melatonin levels. Brendel (et al, 2000) exposed isolated hamster pineal glands to a 50 Hz, 86 μT EMF for 8 hours and reported lower levels of melatonin in exposed glands compared with controls. They concluded that reported effects were caused by direct effects on the pineal gland rather than indirectly, such as via the eyes. (Table 4.3).

Table 4.1: Studies Reporting Suppression effects on Melatonin from 50/60 Hz Exposure in Rats

Reference	Exposure Conditions	Result
Martinez et al. 1992	50 Hz; 5200 μT; 30 min./ day for 21 days	Sign.decr. in synaptic ribbons and serum MT after 15 days
Kato et al. 1993	Rotating 50 Hz; 5, 50, 250 μT; continuous for 6 weeks	sign. decr. in MT
Kato et al. 1994	50 Hz; 1 μT; circularly polarised chronic exposure; 6 weeks	sign. decr. in MT recovery after 1 week
Loscher et al. 1994	50 Hz; 0.3-1 μT for 91 days	sign. decr. in nocturnal MT
Selmaoui et al. 1995	50 Hz; 1, 10, or 100 μT; for 12 hrs.; or 18 hr.s/ day for 30 days	sign. suppression in MT peak MT & Pineal NAT; no effect on HIOMT in 30 day group; sign. decr. in 12 hr. group only under 100 μT
Mevissen et al. 1996	50 Hz; 10 μT; 24hr.s/ day for 91 days	sign. decr. in circulating MT
Bakos et al. 1997	50 Hz; 1 μT or 100 μT; vertical field; for 24 hr.	no sign. decr. during experiment but sign. decr. of aMT6s at 100 μT next day
Rosen et al. 1998	60 Hz; 50 μT; vertical AC & 0.06 μT DC fields	suppresses MT in isolated pinealocytes

(cont. = continuous; decr.= decrease; exp.=exposure; incr. =increase; sign. =significant; MT= melatonin; MF= magnetic field; aMT6s-=sulphatoxymelatonin, a urinary metabolite of melatonin)

Table 4.2: Studies Reporting No Effects on Melatonin from 50/60 Hz EMF Exposure in Rats

Reference	Exposure Conditions	Result
Kato et al. 1994	50 Hz; 1 μT; 6 weeks chronic; horizontal or vertical MF	No effect on pineal or plasma MT
Grota et al. 1994	60 Hz; 65 kV/m electric field 20 hrs./day for 30 days	No effect on nightly rise in pineal NAT, MT, HIOMT
Bakos et al. 1995	50 Hz; 5 or 500 μT; 24 hrs. vertical MF	No effect on aMT6s during or after exposure
Loscher et al. 1998	50 Hz; 100 μT; 13 weeks	No effect on nocturnal MT
John et al. 1998	60 Hz; 1 mT (rms); horizontal MF 1. 20 hrs./day for 10 days 2. 20 Hrs/day for 6 weeks 3. 1 hr. pulsed 1 min on/ 1 min off for for 2 hrs. 4. 20 hr.s/day for 2 days pulsed as for 3.	No effect on aMT6s
Bakos et al. 2002	50 Hz; 50 μT or 100 μT; vert. MF 8 hr./day for 7 days	No effect on aMT6s

(cont. = continuous; decr.= decrease; exp.=exposure; incr. =increase; sign. =significant; MT= melatonin; MF= magnetic field; aMT6s-=sulphatoxymelatonin, a urinary metabolite of melatonin)

Table 4.3: Studies On the Effects of Power Frequency EMFs on Melatonin Production

Species	Reference	Exposure Conditions	Result
Djungarian Hamster	Yellon et al. 1994	60 Hz;100μT; 15 min. 2 hr.s before lights off	sign.lower pineal MT content 5 hr.s after lights off
Djungarian Hamster	Truong et al. 1997	60 Hz; 10 or 100 μT; 1. for 15 min. 2 hrs. or 4 hr.s before dark or 4 hr.s after dark 2. intermittent for 15 min. or 60 min. 1 min. on/ 1min.off; 1-2 hr. before lights off	 No effect on nocturnal MT rise No effect on nocturnal rise of MT
Djungarian Hamster	Brendel et al. 2000	50 Hz; 86 μT, 8 hrs.; rectangular wave form; isolated pineal gland	Reduced max. MT production
Siberian Hamster	Yellon et al. 1998	60 Hz; 100 μT; acute for 15 min or chronic for 14 or 21 days	No effect on MT rise or circulating MT
Siberian Hamster	Wilson et al. 1999	linearly polarised; horizontal MF 1. 60 Hz; 0.1 mT; 15 min. 2. 60 Hz; 50 μT; 15 min. 3. 60 Hz; 0.1 mT; 15 min. pulsed (3 min.on/ off) then 30 min on for 1 hr./ day for 16 days 4. 60 Hz; 0.1 mT; 3 hrs/ day for 42 days	 Sign. decr. in MT 3 &5 hr.s after dark males only No effect on MT Decr. in MT 4 hrs. after dark No sign. effect
Sheep	Lee et al. 1993	500 kV transmission line; 60 Hz, 4 μT; 6 kV/ m; 8 months	No effect on MT
Sheep	Lee et al. 1995	500 kV transmission line; 60 Hz, 3.77 μT; 6.3 kV/ m; 8 months	No effect on MT
Baboon	Rogers et al. 1995	vertical EF, horizontal MF 1. 60 Hz; 6 kV/ m; 50 μT; 6 weeks 2. 60 Hz; 30 kV/ m; 100 μT; 6 weeks	 No effect on MT No effect on MT

cont.= continuous; decr.= decrease; exp.=exposure; incr.=increase; sign.=significant; MT= melatonin; MF= magnetic field; aMT6s-=sulphatoxymelatonin, a urinary metabolite of MT.

Studies on humans

While there is considerable research describing the effects of ELF fields on animals (mainly rodents) few studies have been carried out on humans. Results so far have been as inconsistent as those from animal studies.

The first study looking at the effects of EMFs on human circadian rhythms was done by Weever in 1979 (cited Arendt, 1988; Wever, 1992). He concluded that a 10 Hz square wave field of 2.5 V/m shortened a free-running circadian rhythm by an average of 1.2 hours. A large sample of 300 subjects was isolated from external cues in an underground facility. Weever found that shielding subjects from natural magnetic fields lengthened the circadian rhythm by 20 minutes (Gamberale, 1990). However, although shielded from electric fields emanating from the surface, equipment in the chambers would have added 10-100V/m and 1μT (Sienkiewicz et al, 1991). Weever suggested that humans may be able to detect weak electric fields. In 1992, Sagan noted that the research had not been replicated by Weaver or anyone else.

Research on the effects of power frequency EMFs on melatonin levels in humans has begun only recently and results have been inconsistent. Positive findings have been reported by Wilson et al., (1990), Wood et al., (1998), Karasek et al.,(1998), Burch et al., (1998), Graham et al., (2000) and Juutilainen et al., (2000) (Table 4.4). Those studies reporting significant results tended to be either, environmental studies, or those using square wave forms.

In 1990, Wilson et al. carried out a study looking for possible effects of 60 Hz EMFs on pineal function in humans. Forty-two volunteers used standard wired or modified continuous polymer wire (CPW) electric blankets for 8 weeks. The EMF was measured at the position of the subjects' head. A background EMF of 0.7 mG was recorded. The standard blanket produced a field of 2.4 mG; the CPW(AC) a field of 4.2 mG; and the CPW(DC) a field of 0.56 mG. The subjects were divided into 3 groups. Group 1 (n=14), spent 4-5 weeks on CPW(AC) blankets. Group 2 (n=14), spent 4-5 weeks on CPW(DC) blankets. After this, groups 1 & 2 switched power modes for another 4-5 weeks. Group 3, (N=14), slept for 7 weeks on standard AC blankets. Early evening and first morning voiding urine was taken for two weeks before exposure, during exposure, and for two weeks after exposure to measure the urinary melatonin metabolite 6-hydroxymelatonin. No changes were found in melatonin levels among subjects using the conventional electric blankets. There were also no

significant differences found between AC and DC exposures (using ANOVA). A significant difference in results was found (using a non parametric sign test) between AC and DC in group 1 and between DC and post-exposure field conditions. Significant differences were also found in group 2 between AC and post-exposure field conditions. This reflected differences in 7 individuals. The pattern was a sign decrease ($p<0.05$) during AC exposure compared to DC and a significant rebound effect after exposure. Group 1 had AC then DC CPW exposure while Group 2 had DC then AC CPW exposure. Wilson and colleagues attributed the differences to the fact that CPW blankets turn on and off more frequently and have a 50% stronger field. Wilson et al suggested that a reduction in the metabolite of melatonin in 7 out of the 28 subjects may reflect the presence of a susceptible subgroup. He also postulated that as electric blankets are turned on and off at the same time of day on a regular basis they may act as a type of zeitgeber (Wilson, 1992). Another possibility is that the effect noted was a heating effect. Hong et al. (2001) used a non-heating electric sheet to expose 9 males to a 60 Hz, 0.7 μT (at the head) EMF and failed to find a significant effect on urinary melatonin.
In an occupational study, Juutilainen et al., (2000) noted a decrease in nocturnal 6- hydroxymelatonin in female garment workers exposed to an EMF of approximately 0.15 μT.

Laboratory studies on the effects of EMFs on melatonin have generally produced negative results when using a sine wave field, which is the wave form common to power frequency fields. Significant results have been reported from square wave forms (Wood et al., 1998; Karasek et al., 1998). In some studies a subgroup of susceptible individuals has been identified. Those individuals have been identified as those with naturally low levels of melatonin. Studies using low flux density fields failed to find field effects (Selmaoui et al, 1996) (Table 4.4).

In a series of three studies Graham et al. (1996, 1997,2000) investigated possible effects from a circularly polarised 10 and 200 mG 60 Hz EMF. The 1996 study exposed 33 males to either; a sham, 60 Hz 10 mG, or 200 mG, intermittent field. The field was intermittent, being on for an hour then off for an hour. In addition, during the "on" hour the field was cycled 15 seconds on/15 seconds off. The experiment was carried out under double- blind conditions and exposure time was between 2300 and 0700 hours. Blood was taken hourly and tested for melatonin. No significant differences between the groups were found except in men with low pre-exposure levels of melatonin. They

responded with a significant drop of melatonin in the 60 Hz 200 mG field. A repeat of the above experiment in 1997 using a larger sample failed to replicate the results. A later study (2000) over 4 nights and at occupational exposure levels (28.3 μT) failed to find an effect on melatonin but suggested a possible cumulative effect over time. The study used subjects as their own controls. Previously, Graham et al had used matched controls.

The possibility of a susceptible subgroup was reinforced by Crasson et al, (2001) who failed to find a significant effect from a daytime exposure to a 100 μT, 60 Hz EMF which was either pulsed (15 seconds on/off), or continuos. They reported a smaller increase in the following night time melatonin rise in those with plasma melatonin levels below 55 pg/ml after exposure to the continuous field. Subjects acted as their own controls. Karasek et al. (1998) also used a day time exposure but at 2.9 mT. The exposure period was for 20 minutes per day, 5 days a week for 3 weeks. A significant depression of the nocturnal melatonin rise was observed in this study but it differed from others in that a square wave was used. Wood et al (1998) also reported a significant effect on the melatonin rise from a square wave, 200 μT EMF but found n smaller effect from a sine wave. In both these studies subjects acted as their own controls.

A small amount of research has been done on the effects of NMR (nuclear magnetic resonance) equipment on humans. NMR (or MRI) scanners have a 50 or 60 Hz field associated with the revolving part of the equipment and also a very high intensity static field in the diagnostic part of the equipment. A study by Schiffman (1994) using a 60 Hz, 1.5 T field at night failed to show any suppression in melatonin. Results from studies which use small sample sizes need to be treated with caution.

Table 4.4 Studies on the Effects of Power Frequency EMFs on Melatonin in Humans

Reference	Subjects	Exposure Conditions	Control	Outcome
Wilson et al. 1990	32 female 10 Male	Electric blanket CPW or conventional for 8 weeks	same control	sign.decr.in nightly 6.OHMS with CPW blankets only
Selmaoui et al. 1996	32 male	50 Hz; 10 μT linearly polarised; a 9hrs cont. exposure then 9hrs intermitt.	separate control	no effect
Graham et al. 1996	1. 33 male	60 Hz; 1 μT or 20μT circularly polarised overnight, intermitt.	separate control	1. no effect on MT levels. Sign. effect on low MT excretors
	2. 40 male	60 Hz; 20μT overnight	separate control	2. no effect on MT levels
Graham et al. 1997	40 male	60 Hz; 20 μT; overnight contin.	separate control	no effect on MT levels
Wood et al. 1998	30 male	50 Hz; 20 μT sine wave or square wave	same control	delay of MT rise more marked in square wave
Karasek et al. 1998	12 male	40 Hz; 2.9 mT square wave 20 min/day; 5 days/week for 3 weeks at 1000 or 1800 hrs	same control	sign. decr. in MT rise
Burch et al. 1998	electric utility workers	60 Hz;	epidemiol.	temporally stable MF cause reduced nocturnal 6-OHMS
Graham et al. 2000	30 male	60 Hz; 28.3 μT; 4 nights	same control	no sign. effect but poss. cumulative effect
Juutilainen et al. 2000	60 female garment workers	0.15 μT to 0.75 μT	epidemiol.	decrease in 6-OHMS
Crasson et al. 2001	21 Male	50 Hz, 100 μT, sham contin., intermitt. 30 min./day	double blind seper. control	no effect- plasma MT, or aMT6s, but reduced aMT6s in low excretors
Hong et al. 2001	9 Male	50 Hz; electric sheet 0.7 μT at the head 11 weeks night time exposure	same control	no effect

(cont. = continuous; decr.= decrease; exp.=exposure; incr. =increase; sign. =significant; MT= melatonin; MF= magnetic field; aMT6s-=sulphatoxymelatonin, a urinary metabolite)

Radio Frequency EMFs

With the wide spread use of microwave ovens and cellular phones, attention has been drawn to the possibility that radio frequency waves may effect melatonin production. As these microwaves are closer than power frequency EMFs to the wave length of natural light they could reasonably be expected to hold greater potential for effects than ELF fields. The small amount of research that has been done on possible effects from these radio frequency waves on melatonin production is outlined below.

Studies on Animals

Stark et al. (1997) reported a brief significant increase in salivary melatonin in dairy cattle exposed to a short wave radio transmitter (3-30 Hz). The effect was noted after the transmitter was reactivated, having been off for three days. The result was described as a delayed acute effect. As there were only 5 cows in the experimental group, the result was not robust. In a study on rats and hamsters, Vollrath et al. (1997) found no notable effect on melatonin synthesis. Exposures were for 15 minutes to 6 hours per day, at 900 MHz, unpulsed or pulsed at 217 Hz with an SAR of 0.06-0.36 W/Kg in rats and 0.04 W/kg in hamsters.

Studies on humans

Studies are just beginning to be published on the effects of radio frequency fields on melatonin production (Table 4.5).

An environmental study on humans living near a short wave transmitter showed no effect on 6-hydroxy melatonin sulphate in urine (Altpeter, 1995. in Stark et al., 1997).

The three studies that have looked at the effects on melatonin in laboratory controlled conditions have failed to find effects. However, two of the studies use exposure protocols which do not mirror actual cellular phone use. In a simulation of cellular phone use Mann et al (1998) failed to find an effect on melatonin secretion in 24 male subjects. Subjects slept in a special facility lined with absorbing material to avoid reflections. Melatonin profiles were observed from blood taken every 20 minutes from 2300 to 0700 hours. They were exposed to a 900 MHZ EMF, pulsed at 217 Hz (mean power density 0.02 mW/cm^2). The antenna, 40 cm away from the subjects head, was connected to a GSM phone via an amplifier. Radon et al. (2001) exposed 8 males to a 900 MHz signal pulsed at 217 Hz. They observe no field induced effects on salivary melatonin levels. Subjects were preselected on the basis of having melatonin rhythms that showed the least intra-individual variation. Subjects under went

twenty 4 hour sessions from midday to 1600 hours or 2200-0200 hours. Half the exposures were sham. Subjects were exposed in a seated position. As with Mann et al. (1998) a remote antennae was used. The antenna was 10 cm away from the subject. While the use of simulations such as this allow the experimental conditions to be carefully controlled and also permit a double blind they do not accurately represent the conditions under normal phone use. Cellular phones are normally operated touching the user's head. Conditions of use include environments where reflection of signals is likely. This type of simulation has been criticised in the literature for being too dissimilar to the situation it is modelling (Kuster and Schonborn, 2000).

In a study using commercially available cellular phones, de Seze et al (1999) exposed 38 male subjects to either, a 900 MHz GSM model or an 1800 MHz digital model. Exposures were for 2 hours per day, 5 days per week for 4 weeks. Four sampling session were carried out at 15 day intervals. Exposures were either between 1400-1600 or 1630-1830 hours. The melatonin profile was not disrupted, nor were peak levels level or timing changed in either the 900 or 1800 MHz studies.

A lot more research is needed in this area as the use of cellular phones has increased exponentially in recent years, especially among the young. Possible biological effects need to be identified.

Table 4.5 Studies on the Effects of Radio Frequency EMFs on melatonin

Reference	Species	Exposure Conditions	Outcome
Stark et al. 1997	dairy cattle	3 to 30 MHz radio antennae; 0.3 mA/M	no sign. differences in salivary MT over 5 nights; short term sign. recovery effect 4 days post exposure
Vollrath et al. 1997	Rats & Djungarian hamsters	900 MHz unpulsed or pulsed at 217 Hz; for 15 mins- 6 hrs. SAR 0.06 to 0.36 W/kg rats, 0.04 W/kg hamsters	no effect on pineal MT, or pineal synaptic ribbon profile
Mann et al. 1998	24 human male	900 MHz pulsed 217 Hz mean power density 0.02 mW/cm^2; antenna 40 cm away from subject Overnight	no sign. effect on MT at night
de Seze et al. 1999	38 human male	GSM 900 MHz or DCS 1800 MHz; for 2 hrs/day, 5 days/ week for 4 wks.	no effect on MT profile
Radon et al. 2001	8 human male	900 MHz pulsed 217 Hz; mean flux dens. 1W/m^2 1 W/m; antennae 10 cm behind subject 20 sessions of 4 hours	no sign. change salivary MT

(cont. = continuous; decr.= decrease; exp.=exposure; incr. =increase; sign. =significant; MT= melatonin; MF= magnetic field; aMT6s-=sulphatoxymelatonin, a urinary metabolite of melatonin)

Psychophysiological Effects

Although there are a number of studies on the effects of EMFs on reaction time, there is only a small body of research on other possible psychophysiological effects. Other parameters include effects on; attention, mood, subjective feelings, visual and auditory discrimination and working memory.

Power Frequency Fields

Studies on Animals

Studies on the behavioural effects of ELF EMFs have produced conflicting results. Power frequency fields have been reported to alter activity levels and produce rearing behaviour in rodents. Not all studies agree. Trzeciak et al. (1993) exposed male rats to static, 0.49μT or 50 Hz, 0.018μT fields for 2 hours a day for 20 days. They noted a decrease in irritability in the animals but found no changes in open field behaviour or locomotory activity. Stern et al. (1996) also failed to replicate a study of another research team. He found no changes in behaviour in rats exposed to a 60 Hz, 5×10^{-5} T EMF. In contrast, changes in social behaviour were reported by Easley et al. (1992) in baboons exposed to 60 Hz, 30 kV/m fields for 3 weeks. Increases in passive affinity, tension and stereotypy were noted.

Changes in learned response retention have been reported. Sienkiewicz et al. (1998) exposed mice to a 0.75 mT , 50 Hz EMF and found a reduction in the rate of learning in a maze task. An earlier group of 4 studies by the same author (Sienkiewicz et al.,1996) had failed to find an effect. The earlier study used fields of either 5μT, 50 μT, 0.5 mT or 5 mT. In a 2001 study, Sienkiewicz et al. exposed mice to an object recognition task to test non spatial working memory after a 45 minute acute exposure to a 7.5 μT, 75 μT or 0.75 mT 50 Hz EMF. No effects were found.

Studies of fields of involving higher intensities have often reported greater effects. It must be noted that high intensity fields are probably detectable by the animal. Smith et al. (1994) reported that in their experiments rats could learn to detect fields as low as 200-1900μT, although their methodology has been criticised by others in the field (Stern, 1995). Also as stated by Sienkiewicz et al. (1991) changes reported in weight and food and water consumption can be explained by micro-shocks when the animal touches drinking spouts or food containers. They noted that studies which had been designed to eliminate such

events found no significant effects from the fields. Changes in behaviour are also difficult to isolate from field effects.

Studies on humans

Public concerns have been raised about exposure to EMFs from high tension power lines. This has prompted the initiation of epidemiological studies looking for possible effects. Gamberale et al. (1989) assessed linesmen working on 400 kV lines for cognitive effects. They found no significant effect on reaction time, vigilance, short-term memory and a perceptual test. Beale et al.(1997) also studied possible effects from proximity to high tension lines. Results indicated performance on memory and attention tests were no different to controls but living in close proximity to 50 Hz high voltage lines was associated with chronic anxiety symptoms and poorer coding test performance.

Laboratory studies on humans have been few in number and have covered a large number of cognitive variables.

In 1973, Johansson et al. (cited Gamberale,1990) exposed subjects to a 20 kV/m, 0.3 mT, 50 Hz field and found no change in reaction time or other psychomotor and cognitive tasks.

Cook et al (1992) studied the effect of a 60 Hz, 9 kV/m, 20 μT field on task completion. They reported a decrease in errors on a choice reaction time task in 18 men exposed for 6 hours. It was suggested that results for the choice reaction time and interval production tasks may have been a consequence of the field effecting the subjects' internal sense of time.

A 45 Hz field of 1. 26 mT was reported to slow learning in 20 subjects in a visual discrimination task. A greater effect was shown for those in an intermittent EMF, pulsed 1 second/ on/ off (Lyskov et al. 1993) than for a continuous EMF.

Graham et al. (1994) found a 60 Hz, 6 kV/m, 10 μT field significantly lengthened reaction time but fields of 9 kV/m (20μT) and 12 kV/m (30 μT) had no effect on reaction time.

Trimmel et al. (1998) reported an immediate reduction in attention, perception and memory performance following exposure to a 50 Hz, 1 mT EMF

accompanied by 45 dB noise. Sixty six subjects were exposed to the conditions for 1 hour.

Preece et al (1998) also reported a significant reduction in measures of attention, working memory and episodic secondary memory from a 0.6 mT , 50 Hz EMF. This was reflected in a reduced ability to hold digits in working memory, a reduced ability to recall words and a reduction in accuracy on a choice reaction -time task. In all the aforementioned tasks it was the accuracy, not the speed, that was effected. The length of the exposure was not stated.

In two experiments, Crasson et al, (1999) used a number of tasks to assess possible effects from a 50 Hz, 100μT EMF. Subjects were exposed to a 30 minute sham, continuous or intermittent EMF (pulsed 15 sec. on/ off). Cognitive tests carried out included; the d2 attention task, Rey auditory verbal learning digit span, dichotic listening a test of selective attention. Ten subjects were exposed at 1.30 p.m. and 11 at 4.30 p.m. Results in the cognitive tasks did not support Lyskov's hypothesis of an effect on associative and long term memory. Crasson did find reaction time slowing in a visual task.

As part of a series of studies, Whittington et al. (1996a) and Podd et al. (2002) investigated the effect of a 50 Hz, 100 μT EMF on two-alternative, forced choice duration discrimination tasks with three levels of difficulty. The Whittington study was large with a 100 subjects. The exposure time was 9 minutes. The presence of the EMF significantly reduced reaction time on the hardest task level but did not effect percentage correct. The second study (Podd, 2002), on 80 subjects, failed to find an effect on reaction time and accuracy in the visual discrimination task but did note a reduction in recognition accuracy. Inconsistency of findings, even within the same research facility, is a feature of EMF research. The 2002 study included a recognition memory task in which a reduction in recall was reported. This task was completed after leaving the field. The total exposure time was 11 minutes.

Keetley et al. (2001) studied the effects of a circularly polarised, 50 Hz, 28 wT (resultant) EMF on 30 subjects. They demonstrated a reduction in the recall of a 15 word list after an interference list was given. A decrease in a trail making test (a test of alternate attention) was also observed and they concluded that the EMF had consequences for short term learning and executive functioning. Exposure lasted 50 minutes with the start of the tests 20 minutes after

commencement.

Findings have been varied, as table 4.6 indicates. The most common effects appear to be in reaction time tasks, including forced choice tasks. Delayed word recall and digit recall also appear to be areas requiring further investigation.

Studies using higher field intensities have not necessarily been the ones to detect effects. Graham et al. 1994, found an effect on reaction time and concluded that there appear to be particular 'windows' which produce positive results when frequency or intensity values close by produce no significant changes.
Studies using pulsed EMFs appear to report effects more than ones using continuous EMfs. Longer exposure times have not necessarily produced greater effects with studies as short as 9 minutes reporting effects while some longer ones didn't.

There have been a large number of cognitive variables studied which, when added to the differences in EMF variables used, makes comparison of studies difficult. For trends to become apparent a much larger research base is necessary.

Table 4.6 : The Effects of Power Freq. EMF s on Cognitive and Perceptual Tasks in Humans

Reference	Subjects	Exposure Conditions	Control	Tasks and Outcome
Gamberale et al. 1989	26 male	power line workers 400 kV line	same control	RT vigilance digit span digit symbol
Beale et al. 1997	540	Residents under high voltage power lines	separate control	digit symbol-decr. digit span memory trail making symbol digit--modality d2 attention test visual process memory selective reminding life changes Question. Gen. health question.
Trimmel et al. 1998	66	50 Hz, 1 mT plus 45 dB noise 1 hour		attent. task- decrease memory task -decr. perception task-decr.
Cook et al, 1992	18	60 Hz, 20 μT two 3 hour exposures	same control	choice reaction time- (decr. errors) reaction time time estimation interval production vigilance Wilkinson addition digit span memory various mood measures
Lyskov et al. 1993	10	45 Hz, 1.26 mT interm.(1 sec on/off) or continuous, 1 hr.	same control	RT- slower to learn if real exposure was 1st
Graham et al., 1994	18	60 Hz, 10 μT, 6 kV/m 20 μT, 9 kV/m 30 μT, 12 kV/m	same control	RT -sign decr. RT RT

Table 4.6 contd : The Effects of 50 HZ EMF s on Cognitive and Perceptual Tasks in Humans

Reference	Subjects	Exposure Conditions	Control	Tasks and Outcome
Whittington et al. 1996a	100	50 Hz, 100 μT , 9 min.	separate control	forced choice - discrimination task decr. RT on hardest task
Preece et al. 1998	16	50 Hz, 0.6 mT, continuous linearly polarised	same control	Immediate word recall picture presentation simple reaction time digit vigilance choice reaction time spatial working mem. numeric working mem. delayed word recall delay word recognition delay picture recogn.
Crasson et al,1999	21	50 Hz, 100μT,30 min. intermittent (15 sec on/off) and continuous 11.30 or 4 p.m.	same control	d2 attention Rey auditory digit span dichotic listening- RT- possible effect
Podd et al. 2002	80	50 Hz, 100 μT , intermittent (1 sec on/off) 11 minutes	separate control	forced choice - discrimination task recognition mem.- decr.
Keetley et al. 2001	30	circularly polarised 50 Hz 28 wT(resultant), 50 min.		trail making-decr. word recall-decr.

KEY An underlined task denoted a statistically significant effect

decr. =decreased.

RT= Reaction Time

mem.= memory recogn. =recognition

same control =each subject underwent the exposure and control session.

separate control= different subjects were used for the control and exposure sessions.

Radio frequency EMFs

Studies on Animals

Most of the studies on animals have been carried out at 2450 MHz which is the frequency relating to microwave ovens. For example, Wang and Lai (2000) reported that rats used a different learning strategy to locate a submerged platform after they had been exposed to a pulsed 2450 MHz EMF with a whole body SAR of 1.2 W/kg for one hour. There is an extensive literature on the effects of GHz microwave exposures on animals, often at high SARs. However, in a review of behavioural effects of microwave irradiation, D'Andrea (1999) concluded that effects on attention. learning, memory and discrimination tasks may occur at SAR levels far below those needed to cause work stoppage. They further conclude that the absorption of microwave energy is dependant on the frequency and is not uniformly distributed over the body. Therefore, it is questionable whether the results of these studies are relevant to the much lower frequencies used by cellular phones. Consequently, the following review is confined to cellular phone frequencies. There have been few studies on animals at these frequencies. In an unusual study, Bornhausen and Scheingraber (2000) exposed pregnant rats to a 900 MHz, 217 Hz pulse modulated EMF that approximated the EMF found near cell phone base station antennae. They found no difference in the learning ability of the off-spring.

Studies on Humans

As yet there have been few studies published on the effects of exposure to cellular phone frequencies. Usage of cellular phones has been linked to somatic complaints of headaches, dizziness, difficulties in concentrating and short-term memory loss (Wilen et al, 2003). Recent studies have suggested microwave exposure effects on attention, working memory and time perception, may occur at levels far lower those previously expected (D'Andrea, 1999).

Some studies report positive effects such as an improvement in attention (Lee et al., 2001), while others found no effects on attention and tasks of processing speed (Edelstyn and Oldershaw, 2002). Lee et al. (2001) administered tests of attention to 72 teenagers, 37 of which, used cellular phones. The teenagers who used mobile phones were better at tasks such as the Trail making test (a test of divided attention). However, it was possible that cellular phones users were naturally better at multi-tasking and the result had nothing to do with the phone use. Two other tests of attention were uneffected by phone use. In the

study by Edelstyn and Oldershaw (2002), 38 subjects were assigned to either a group in which the phone was activated or a control group in which it was switched off. The experimental group was exposed for 30 minutes. Attention was assessed prior to exposure, and 15 minutes and 30 minutes after exposure. Performance was enhanced in digit span forwards and spatial span backwards, which are tests of attention. Serial subtraction (a test of divided attention and processing speed was also enhanced). Uneffected were; digit span backwards, spatial span forwards and verbal fluency.

Preece et al. (1999) exposed 36 adults to a simulated analogue and digital cell phone scenario for 20-30 mins. and found subjects had a reduced ability to make visual choices. Ten cognitive tests were carried out (Table 4.7) and performance in most was uneffected by exposure to the RF EMF. A real cell phone was not used due to the cost of airtime and an inability to control the output. They also looked at possible interference due to the amount of sleep the subject had the night before and alcohol, tea and coffee intake. Half the group were assessed for intake of the afore mentioned substances and no effect on results was found.

In a series of three studies, subjects were exposed to a 902 MHZ digital cell phone, modulated at 217 Hz with a pulse width of 577 μsec.s (Table 4.7). In the first study (Koivisto et al., 2000a), 48 subjects were exposed for 60 minutes and a significant increase in a simple reaction time task and a decrease in false alarms in a vigilance task was found. Time taken to complete a subtraction task was also reduced. There was no effect on choice reaction time, visual object recognition or word recognition. The study was single blind with the loudspeaker removed. The phone was attached to the left side of the head. They concluded that GSM cellular phones may effect cognitive processing. In a second study using the same exposure system, Koivisto et al (2000b) observed the effect on a memory task in which subjects recalled if a letter had been seen previously. There was a graduated level of difficulty. Subjects had increased response times, but no change in accuracy, with the more difficult level when exposed to the field. Exposure time was 30 minutes.

In the third, much larger study (Haarala et al., 2003), 64 subjects (2 groups of 32) were exposed to a similar exposure system for 60 minutes. Two different labs were used. Discontinuous transmission mode was not activated in the third experiment. It was not stated whether this had been done in the earlier experiments. It was stated that there was no detectable temperature increase between the phone and the ear. Nine reaction time tasks were carried out plus

3 attention tasks. These were modified stroop tasks and tested for the ability in selective attention. There were no significant effects found on any of the tasks. They concluded that there was no immediate effect on cognitive functioning from exposure to a cellular phone. They noted the criticism of the IEGMP (2000) which stated the Bonferroni criteria should have been applied in the previous study due to the large number of similar tests carried out. This would have reduced the positive results from three down to one. They also suggest that an EMF generator should be used rather than an actual phone to ensure parameters are controlled.

Eulitz et al. (1998) exposed 13 men to a 917.2 MHz EMF,pulsed at 217 Hz and found an alteration in the brain's response during an auditory discrimination task. Krause et al (2000a) reported that exposure to an EMF significantly modified the brain EEG readings, in 16 subjects, during a working memory task. They concluded that exposure modified brain responses, rather than modifying the resting EEG. In a further study (Krause et al., 2000b), confirmed the effect with 24 subjects using a task in which subjects performed a 'number back'task with a differential load of 1, 2, or 3 items.

Research into this area is just beginning and there are too few results published to suggest a trend. However, it is an area of public concern and high public risk if adverse effects from cell phone use become a reality.

Table 4.7: The Effects of RF EMFs on Cognitive and Perceptual Tasks in Humans

Reference	Subjects	Exposure Conditions	Control	Tasks and Outcome
Lee et al. 2001	72	37 phone users 35 non users	epidem-iological	Improved at Trail making test no effect on 2 tests
Edelstyn et al. 2002	38	18 phone 18 control 900 MHz phone 30 min.s	separate control	sign diff. after 5 minutes on: digit span forw. spatial span back. serial subtraction no effect on: digit span backw. spatial span forw. verbal fluency
Preece et al. 1999	36	915 MHz ~1 W 915 MHz (217 Hz) 0.125 W 20-30 min.	same control	Immediate word recall picture presentation simple reaction time digit vigilance choice reaction time spatial working mem. numeric working mem. delayed word recall delay word recogn. delay picture recogn.
Koivisto et al. 2000 a	48	902 MHz (217 Hz) 0.25W mean power 60 min, left ear used	same control	RT incr. vigilance-increased
Koivisto et al. 2000 b	48	902 MHz (217 Hz) 2 W 30 min	same control	RT- incr. for harder tasks
Haarala et al. 2003	64	902 MHz (217 Hz) 0.25 W	same control	9 RT tasks-no effect
Krause et al. 2000a	16	digital 902 MHz(217 Hz)	same control	EEG altered during digit recall

Table 4.7: continued

Krause et al. 2000b	24	digital 902 MHz (217 Hz)	same control	EEG altered during digit recall
Eulitz et al., 1998	13	917 Hz (217 Hz)	same control	EEG altered during auditory discrim. task

Key

underline denotes a significant effect on a task

Forw=forwards backw.=backwards discrim.=discrimination

same control= the same subjects were in both the exposure and control groups.

separate control=different subjects were used in the exposure and control groups.

The Cardiovascular System

Power Frequency EMFs

Studies on humans

Research on the effects of EMFs on the heart have produced varied results. In an early study covering several years, Hauf (1974, cited Gamberale, 1990; 1985) exposed over 100 subjects to fields of 1-20 kV/m for up to 5 hours. No cardiac effects were found. The only changes were small changes in blood cell variables which were within the normal physiological range. In 1982, Sander et al. exposed subjects to a 20 kV/m, 5 mT field for 4 hours a day for a week and found no changes in blood pressure or ECG (cited, Gamberale, 1990). A study looking at the effect of EMFs on the cardiac recovery rate after exercise found no effect in the exercise group but a significant ($p=<0.05$) decrease in heart rate in the no exercise control group (Maresh et al., 1988).

In a series of studies, Korpinen exposed volunteers and transmission line workers to the EMF beneath 400 kV transmission lines. Korpinen et al (1993) reported a small decrease in heart rate after transmission line workers and were exposed to 50 Hz 1.02- 15.43 μT, 0.14-10.21 kV/m EMF. However, there were no signs of extra systoles or arrhythmias. In a repeat study in 1994, 26 male volunteers were measured in real and sham EMFs and compared to 15 subjects who were sham exposed. Subjects sat in the field/sham for one hour. No effects on heart rate were found. Field strengths were 1.4-6.6 μT and 3.5-4.3 kV/m. Blood pressure was also recorded and reported in a later study (Korpenin et al., 1996). No field effect on systolic or diastolic pressure were found.

In a laboratory study, Whittington et al (1996a) exposed 100 subjects to a 50 Hz, 100 μT EMF, pulsed one second on/off. They found no effect on heart rate or blood pressure. The exposure time was 9 minutes and the recordings were taken immediately before, or after, exposure.

In a series of studies at the Midwest Institute, results have indicated that field intensities and exposure patterns are important factors in possible interactions with living systems. A finding of a significant decrease in heart rate from a 60 Hz, 9 kV/m, 20μT field was reported by Cook et al. (1992). Cook et al. found the greatest effects were gained immediately after the field was switched on or off. Graham et al (1994) matched three groups of 18 men and exposed them to 60 Hz fields at low (6 kV/m, 10μT), medium (9 kV/m, 20 μT) or high intensities

(12 kV/m, 30 μT). Significant slowing of heart rate and EEG changes were found only in the medium intensity group. Sastre and colleagues (1998) found a significant decrease in heart rate variability using an intermittent 20 μT EMF in 77 men exposed overnight, but no effect was found when a continuous field was used. Analysis of the EEG showed a reduction in the low power band which is caused by an effect on the thermoregulatory and blood pressure control mechanisms mediated through the sympathetic nervous system. The increase found in the high power band reflected an effect on the respiratory control mechanisms mediated by the parasympathetic nervous system. They suggest that the reduced heart rate variability found could result in workers, with prolonged exposure to elevated electromagnetic fields, being more prone to arrhythmia-related disease and myocardial infarction.

A later study by the same team, using a much higher flux density of 127.3 μT failed to produce an effect (Graham, 2000). The aim had been to determine if precise timing of the field switching could reset the cardiac rhythm. They concluded it didn't and discounted this as a possible reason for heart rate variability seen in EMF research. They did however, suggested that earlier (1998) results may have been due to physiological arousal caused by the nurse collecting the blood sample causing alterations in heart rate variability.

Radio Frequency Fields

There have been very few studies on possible effects of RF EMFs on the cardiovascular system (Table 4.8). Radiofrequency radiation have the potential to effect the cardiovascular system by directly effecting blood vessels, or effecting the receptors in the carotid body. Alterations in circulating hormones caused by EMFs also has the potential to alter cardiovascular parameters (IEGMP, 2000).

Early reports from the Soviet Union suggested occupational exposure to RF fields can effect cardiovascular function. The most common observation was a reduction in blood pressure accompanied by an increase or decrease in heart rate (IEGMP, 2000). However, the lack of success in reproducing the results in the West has lead researchers to conclude the effects reported were the consequence of poor experimental technique or chance occurrence (Jauchem, 1997).

In 1997, Jauchem concluded that if heating does not occur during exposure then

current flow is necessary for cardiovascular effects to ensue. In a study on rats, at very high SARs (12 W/kg) at 1 GHz or 10 GHz heating occurred and blood pressure was initially increased, then decreased and heart rate increased (Jauchem et a l., 2000). Exposures at these intensities are not possible with human subjects.

Braune et al (1998) found an increase in capillary perfusion, and blood pressure that was significantly higher during a 35 minute RF exposure. The increase was a magnitude of 5-10 mmHg and they concluded that the result was due to an increase in sympathetic efferent activity. Ten subjects were used and measurements were taken supine, standing and during the valsalva manoeuvre. The exposure system was a GSM 900 MHz, 2 watt, phone with a 217 Hz pulse, operated by remote control. In a follow-up study, 40 males and females were exposed to a similar system as was used previously from a GSM - like signal from a phone attached to the right ear. Systolic and diastolic blood pressure showed significant increases during the protocol of 20 minutes supine rest, 10 minutes of 70 degree upright tilt on a tilt table, and 20 minutes of supine rest. However, analysis of the variance indicated that these changes were not due to the EMF exposure. They conclude that the findings don't support a non thermal EMF effect on the cardiovascular autonomic nervous system (Braune et al., 2002).

Huber et al. (2003) exposed 16 subjects for 30 minutes, prior to a 3 hour daytime sleep, to a RF EMF of 900 MHz, 1 w/kg SAR. The exposure resulted in a reduced heart rate, but only if exposure occurred before,not during, sleep. Twenty-four subjects exposed overnight exhibited no change in heart rate. Mann et al. (1998) had also found no significant effect on heart rate when subjects were exposed during sleep.

The early reports of effects on isolated cardiac tissue have not been substantiated by subsequent research on hearts in vivo (Black and Heynick, 2003). There is a need for more research on humans in this area.

Table 4.8 : The Effects of RF EMF s on Cardiovascular Parameters in Humans

Reference	Subjects	Exposure Conditions	Control	Tasks and Outcome
Braune et al. 1998	10	900 MHz, 217 Hz 2 watt, 35 min.	same control	incr. systolic and diastolic b.p..
Braune et al. 2002	40	900 MHz, 217 Hz 2 watt, 50 min.	same control	no effect on b.p..
Huber et al. 2003	24	900 MHz, 1 w/kg SAR	same control	no effect on H.R.. H.R.variability-power incr. in high freq. range
	Expt 1	8 hr. overnight		
	Expt 2			
	16	30 min. exposure prior to daytime sleep		H.R.. reduced H.R.variability-power incr. in high freq. range

Thermal effects of radiation from cellular phones

Studies on humans

A cellular phone gets warm during a call due to the resistance in the electrical components. The amount of increase depends on factors such as power level, DTX function and heat conductance conditions (Tornevik et al, 1998).

In a large survey of 11,000 cell phone users in Norway and Sweden in 1998, Hansson-Mild described a dose -dependent increase in heat felt in and behind the ear during cell phone use. Surveys and anecdotal reports have suggested heating of the tissues surrounding the phone but the depth of penetration has not been established in humans. There have been few laboratory studies looking at the rises in temperature during cell phone use in humans. (Table 4.9). There are a larger number of studies which used models to assess SARs and possible heating and RF effects. These are addressed in the following section on mechanisms of interaction with living systems. The study be Bernardi et al. (2000) has been included here as it used actual cellular phones and provided data on changes in aural temperature .

Paredi et al. (2001) exposed ten male subjects to a commercially available (900 MHz) cellular phone for 30 minutes. They reported a significant increase in skin temperature, in the region of the nostril and parietal area to the order of 2.3 degrees celsius. The maximum increase in temperature occurred after 6 minutes of exposure. This caused vasodilation and reduced minimum nasal cross sectional area. A higher level of nasal nitric oxide (NO) was also detected. These changes diappeared when an ear piece was used. There were also no changes in skin temperature and NO on the opposite side to the mobile phone. A conclusion was drawn that the exposure caused easily measurable biological effects. They suggested further studies look at the longterm effects of use and the relationship between NO production, vasodilation and temperature.

Wilen et al. (2003) reported warmth behind the ear, burning face and tingling which increased with increasing numbers of calls and increased calling time. The study used 2402 people selected from a larger epidemiological study. In contrast to that finding, Koivisto et al (2001) reported the absence of a significant difference in subjective symptoms between subjects exposed to a 902 MHz, 217 Hz pulse modulated phone and controls. They had removed the loudspeaker and deactivated the discontinuous transmission. The phone was placed in a leather case so it did not contact the skin. Exposure time was 60

minutes in experiment one and 30 minutes in experiment two. Symptoms reported on included; headache, skin redness and warmth, fatigue and itching. A thermocouple had been used to check the temperature between the phone and the subject for 4 subjects. With the phone off the mean temperature was 35.0 °C and with the phone operating it was 35.1 °C. They concluded that thermal clues about exposure conditions were unlikely. They suggest further studies look at possible effects after repeated use and in persons who report themselves to be sensitive to effects from cell phone use.

Bernardi et al. (2000) evaluated local SAR distributions and temperature increases in a model of the human head exposed to RF fields radiated by several models of cellular phone. They reported that the maximum temperature increases occur in the ear and are in the range 0.22 to 0.43 degrees celsius. The maximum in the brain was 0.08 to 0.19 degrees celsius. The rises were obtained after 50 minutes of exposure. The external part of the brain had an increase of 0.10- 0.16 degrees celsius per 1 W/kg of SAR, average over 1 g of brain tissue.

Table 4.9: The Effects of Cellular Phone use on Head Temperature in Humans

Reference	Subjects	Exposure time	Outcome	Temp. Change
Paredi et al. (2001)	10	30 min	nostril and parietal area incr. in nasal NO	incr. of 2.3°C
Bernardi et al., 2000		50 min.	max. incr. in ear brain surface	0.08-0.19 °C 0.10-0.16 °C
Wilen et al., 2003.	2402	epidemiol.	warmth behind the ear, burning face, tingling	
Koivisto et al., 2001	48	Expt.1. 60 min. no significant effect on redness or warmth Expt. 2. 30 min. no significant effect on redness or warmth		

Possible Mechanisms of Action with Living Systems

Power Frequency EMFs

External electric currents induce time varying surface currents on the body which are dependant on the shape and orientation of the body relative to the ground. Internal electric currents and fields are induced as a result of the surface currents and are dependant on frequency in a linear fashion. These internal fields are very low, about 10^{-4} to 10^{-7} the strength, of the outside field. Current density is highest in the thinnest body cross-sectional areas (Sienkiewicz et al, 1991).

Time-varying magnetic fields result in forces on charged molecules and particles which produce electric currents according to Faraday's law. The density of the currents depends on the rate of change of the flux density and tissue conductivity. In pulsed fields the current is produced at the time of current change. The current produced depends on the frequency and flux density amplitude (Sienkiewicz et al, 1991).

Fields strong enough to be able to directly stimulate peripheral nerves are unlikely to be encountered in the normal home or working environment. However, in the central nervous system small nerves have graded potentials rather than an all-or-none response. Small induced currents in these areas of the brain could add to inhibitory or excitatory post-synaptic potentials or effect spatial summations and theoretically alter processing and effect memory or reasoning. These induced fields are small enough to be masked by the local thermally generated electrical noise. (Sienkiewicz et al, 1991). The resulting currents, called eddy currents, circulate in closed loops in a plane perpendicular to the magnetic field (Tenforde and Kaune, 1987).

As just described both electric and magnetic components of EMFs induce currents in the fluid of the pericellular space. As the cell membrane forms a dielectric barrier, only a small amount of this induced current penetrates the cell surface. For this reason it is believed that most of the effect of EMFs occur at the cell membrane (Tenforde and Kaune, 1987). The pericellular currents produce electrochemical alterations in components at the cell membrane surface. These alterations are transduced through the membrane and produce biochemical and physiological changes within the cell (Tenforde and Kaune,

1987).

Possible mechanisms have been described as either long-range events in the matrix of glycoproteins and lipoproteins making up the membrane or as localised events at surface receptors or ion channels within the membrane.

Long Range phenomenon in cell membranes

Although the electric field induced in tissue by an external ELF EMF is small when compared to the trans-membrane potential it has been suggested that an amplification process may occur in cell membrane molecules. Oscillations established by the field are amplified by the collective excitation of patches of membrane molecules extending over the cell surface (Tenforde and Kaune, 1987). This collective excitation results in energy being released as chemical energy and used in enzymatic pathways or ion pump activation, or, results in the reorganisation of molecules within the membrane.

Tenforde and Kaune (1987) described several models developed by researchers to account for non-linear membrane phenomena produced by weak EMFs. In 1968, Frohlich proposed that membrane molecules having electric dipole moments could be excited into coherent oscillation. The dipolar modes of longitudinal oscillations could then be channelled into a single mode by a small amount of energy provided it exceeded a certain critical threshold. The results would be an amplification of the signal in a non-linear manner as predicted by the Lotka-Volterra equations. In 1977, Kaczmarek also proposed a model of non-linear oscillation, similar to the Van der Polk oscillator, in which membrane proteins moved between ground and excited states in a limited cycle. In 1976 Grodsky modelled the cell membrane as a lattice of phospholipid polar groups interspersed by negatively charged glycoproteins. A weak external field could induce configurational changes in the phosphgolipid head groups in a resonant manner which would release energy stored within the membrane.
Another possible mechanism by which weak EMFs could be amplified within cell structures is the theory of soliton excitation first proposed by Davydov in 1979. A soliton is a slowly dissipating wave which moves through cell structures, such as peptide groups in a protein, as the result of a collective oscillation.

Localised events in cell membranes

Pericellular currents produced by external EMFs could effect specific ligand

binding sites on the membrane surface such as hormone receptors or could result in changes in ion channels crossing the cell membrane.

Chiabrera et al.(2000) suggested that weak EMFs could induce a microelectrophoretic motion within the membrane which could influence the distance between charged ligands and receptors on the cell surface. This could reduce the life of the ligand receptor complexes so effecting such phenomena as the activation of lymphocytes by antigens. It could also effect gating mechanism controlling membrane transport of various ions including calcium.

As the currents produced by weak EMFs are much smaller than surrounding noise without some form of amplification weak EMF signals can not be recognised by cells. Amplification through the noise is one possibility and involves the pumping of energy from the broadband noise to the system at the driving frequency of a periodic external force. This can increase the signal-to-noise ratio and is termed stochastic resonance. An interesting feature of stochastic resonance is that it may occur only for weak signals as the coefficient of signal amplification decreases with increasing modulation strength. If there is some coupling between the separate receivers amplification may be much increased. The most likely sites for this to occur are the ion channels in the cell membrane. The well-like structures of these channels mean that if the strength of coupling between them is in the order of kT then a network of the channels would make a good amplifier for external signals (Kruglikov and Dertinger, 1994).

Resonance

Recently researchers have focused on the the possibility that ion resonance effects could be produced by an interaction between a static field comparable to the geomagnetic field and a AC ELF EMF of about the same magnitude and of a specific frequency. In a cyclotron, charges particles exposed to a static magnetic field and a perpendicular oscillating field will move in circular orbits at right angles to those fields when the frequency of the oscillating field matches the particle gyrofrequency (Adey,1993).

The specific frequency is proportional to the strength of the DC field(B) and the charge to mass ratio of the the ion. The intensity of AC field producing the greatest effect can be calculated from a Bessel function response curve (Yost and Lidburdy, 1992).

The cyclotron resonance frequency for a given ion is reproduced below.

$$f_c = 0.5 \pi (q/m) (B_{DC})$$

In 1985, Liboff applied the theory of cyclotron resonance to the binding of calcium to biological molecules. He proposed that the presence of the field may result in a frequency-specific absorption of electromagnetic energy by the calcium ions. This theory predicted a window effect in which only certain frequencies would lead to resonant absorption of energy by an unhydrated ion. At 50-60 Hz the resonant path was greater than 1 metre and dampening occurred by solvent molecules or active site binding (Grissom, 1995, Adey, 1993). Attempts to apply the model experimentally failed to produce significant results (Galt et al, 1993; Galt et al, 1995.)

Liboff's model was modified byLednev in 1991 to form the parametric resonance model. In Lednev's model a calcium ion is bound to a protein or inside a hydrate shell and acts as a degenerate three-dimensional oscillator (Adair, 1992). The presence of a resonant condition effects the probability that an ion will remain bound to its carrier protein and thus the probability that the ion will carry out its ion-dependant biological function (Liboff et al., 1995).

A further modification of the model was made by Blanchard and colleagues and referred to as the ion parametric resonance model. This new model extended resonance effect to ions other than calcium (Blanchard and Blackman, 1994). They also extended the model to whole animal systems (Blanchard et al., 1995). The new model has been supported by research by Blackman and colleagues on neurite outgrowth in PC-12 cells at resonance conditions for hydrogen ions. The EMF produced changes in neurite outgrowth occurred under resonant conditions (Trilloet al., 1996). Considerable debate has occurred in the literature on the relative accuracies of the models developed by Lednev and Blackman (Blanchard and Blackman 1994; Blanchard et al., 1995, Liboff et al., 1995) The main controversy appears to be about the inclusion of a factor of 2 in the calculations by Blackman. For an in-depth comparison the reader is referred to Engstrom (1996).

Most research has concentrated on calcium ions which should display resonance at 40 Hz but resonant conditions are also present for other ions, for example, K^+ (20 Hz), H^+ (760 Hz). The main criticism with resonance models is that they do not address the question of transductive coupling at levels below

the thermal energy of living tissues. Answers to that question are being sought in research into EMF effects on free radicals (Adey, 1993).

Recombination of radical pairs

Radical pair recombination is effected by magnetic spin effects. A magnetic field effect may be due to an exogenous magnetic field or the the endogenous field produced by a non-zero nuclear spin. Different isotopes have different nuclear magnetic moments which influences reaction rate and the distribution of isotopes in the product. Even an EMF of 1-10 mT can split the Zeeman energy of a radical pair and provide an alternative pathway with a different reaction rate or different distribution of product (Grissom, 1995).

EMFs can " increase the yield of some types of free radicals through homolytic cleavage, photo-induced processes, or random encounters" (Scaiano et al., 1994, p. 549). The radical pairs may recombine producing inert products or may diffuse away and react with nearby molecules or structures, especially lipid bilayers, micelles and molecules in the intracellular environment. In 75% of random radical-radical encounters the radical pair has a triplet configuration, with the unpaired electrons having parallel spins (an electron spin being the angular momentum of the unpaired electron). Triplet radical pairs may be generated by photolysis (Grissom, 1995; Scaiano et al, 1994). Triplet radical pairs can not react with each other unless intersystem crossing (spin evolution) leads to a singlet state. In a singlet state radical pairs are available for product formation. Moderate EMFs can influence intersystem crossing by Zeeman splitting of the triplet sub levels and so effect whether reaction occurs with the other partner in the radical pair or separation occurs and the radical is available for other interaction with other radicals. An oscillating field produces intensity windows causing triplets to return to singlet states that react with one another (Adey, 1993).

For a detailed description of radical pair recombination the reader is referred to Grissom (1995).

Biogenic magnetite

Biogenic magnetite($Fe_3 0_4$) may be responsible for magneto reception in animals, such as magnetotaxic bacteria and honey bees, which are sensitive to magnetic fields (Kirschvink et al., 1992). The mechanism of action may be the

torque effects produced by a fixed magnetosome when exposed to an external magnetic field or a change in the size of the ion granules due to repulsion or attraction of their poles in differing alignments (Grissom, 1995). Magnetosomes moving under the influence of earth-strength magnetic fields can open transmembrane ion channels (Kirschvink et al, 1992).

Iron containing molecules are wide spread in living tissues and include transferrins, and cytochrome P-450 enzymes. While the iron atoms are too scattered to form magnetic dipoles they may interact with molecules having paramagnetic properties (Grissom, 1995). The possibility of such interactions was disputed by Adair (1993) on the basis that effects of 60 Hz fields of 5μT(50 mG) or less are below effects caused by thermal agitation and as such can not be biologically significant. Adair's view was questioned by Polk (1994 b) on the basis that Adair's model was not suitable for living systems and contained incorrect assumptions.

Melatonin

Melatonin has been suggested as a possible mechanism by which EMFs could interact with living systems. Melatonin has been ascribed an anticancer function as it is a free radical scavenger so a reduction in melatonin levels due to EMF exposure could theoretically lead to a rise in cancer caused by free radicals. A drop in melatonin would also produce a rise in oestrogen with the accompanying increased turnover of breast cells which could also increase the possibilities for cancer. Melatonin is discussed in detail in a previous section.

Possible Mechanisms by which EMFs could effect Melatonin Production

Research on the effects of exposure of animals to static EMFs has suggested that an EMF could act as a zeitgeber (or time setter) in a similar manner to light. Welker et al. (1983) found a significant reduction in melatonin levels and NAT activity in rats subjected to an inversion of the horizontal component of the earth's magnetic field at night. Welker et al. postulated that the mechanism of action may be the reduction of the rate of the enzyme NAT which would result in the much reduced output of melatonin. Another similar study found that the greatest effect was gained near and just after the middle of the night (Yaga et al., 1994). Other researchers (Reus and Olcese, 1986; Olcese et al., 1985) have found similar results. With a 50 degree rotation of the earth's magnetic field the production of melatonin was suppressed but the effect was abolished in rats which had been blinded. This suggested that the magneto receptors are located

in the eyes not in the pineal as occurs in pigeons (Olcese et al. 1985). Reuss and Olcese (1986) reported that a weak red light was also required for a pineal response to magnetic fields. Bliss and Heppner (1976) studied the house sparrow and were able to effect a free-running circadian rhythm by changing the vertical component of the earth's magnetic field. They concluded that the weak (near zero gauss) magnetic field acted as a zeitgeber in the absence of light though it was a much weaker one than light. They also suggested the effect may be species-specific. Studies by Olcese et al. (1988) and Reiter and Richardson (unpublished , cited Reiter, 1991) reported a change in the circadian rhythm of melatonin in pigeons and guinea pigs when exposed to an artificial static magnetic field. The Guinea pig pinealocytes altered their firing rates when placed in a magnetic field. Interestingly, in pigeons, this did not require the eyes which suggested the pineal may be directly sensitive to a magnetic field in that species.

Power Frequency EMFs and melatonin

Direct suppressive effects have been found on isolated pineal glands in the hamster (Brendel et al, 2000). However, it must be noted that the pineal is much closer to the surface of the skull in rodents than in humans.

It has been postulated that the depression of melatonin by power frequency EMFs is due to the inhibiting effect of the field on the activity of the enzyme NAT activity. Reiter (1993) noted a drop in NAT and melatonin levels in rats and postulated the cause as an induction of eddy currents produced when rapid inversion of the field occurred. Other possibilities have been suggested. Grota, et al. (1994) exposed Sprague-Dawley male rats for 30 days to a 65 kV/m, 60 Hz electric field for 20 hours a day. They found that night time increases in pineal NAT, HIOMT and melatonin were not altered but that serum melatonin was reduced. They suggested that it is not the synthesis, but tissue uptake and degradation, of melatonin that is effected by the electric fields.

Studies in rats have suggested melatonin plays an inhibitory role in DMBA (7,12-dimethylbenzyl-a-anthracene)-induced mammary carcinogenity. Mevissen et al (1996) reported a possible dose-dependant effect with DMBA - induced mammary tumours with the EMF appearing to be a co-promoting agent. They concluded that the effects of EMF exposure appeared to be dose dependant as a 50 Hz, 100 μT EMF caused a significant increase in tumour production whereas 10 μT did not.

Whilst melatonin's role in scavenging free radicals may provide a possible link between EMFs and cancer, the link could also be a less direct one. For example, a reduction in melatonin allows oestrogen and prolactin to increase. Increased oestrogen results in a greater turnover of breast cells and hence an increased opportunity for cancer (Pinholster, 1993). In 1989, Wilson suggested it is likely that the pineal gland responds to to ELF exposure via neuronally mediated changes.

Steven's melatonin hypothesis

Steven's (1987) melatonin hypothesis requires the EMF to suppress pineal melatonin production as the necessary first step in a chain leading to increased breast carcinogenity. Reduced melatonin production suppresses pituitary prolactin production and ovarian oestrogen production. This leads to an increased turn over of epithelial stem cells in the breast which results in an increased risk of hormone dependant cancers. Only a small number of studies have been done on humans and they have produced variable results. It is by no means certain that EMFs actually effect melatonin production in humans. Research in this area is in its infancy and just how, or if, EMFs effect the production of melatonin is currently largely speculation.

Radio Frequency EMFs : Possible Mechanisms of Interaction with Living Systems

There are two main postulated mechanisms of interaction between RF EMFs and living tissues. They are direct effects between the RF EMF and living systems or secondary effects due to the heating of tissues.

Heat generation

A cellular phone gets warm due to electrical resistance in the components. The amount of increase in temperature depends on factors such as power level and heat conduction conditions. With phones that have SAR values below the RF exposure guidelines it is likely that the heat felt on the ear is due to conductive heating (Wilen et al., 2003).

Thermoregulatory responses to RF waves in humans were measured in two studies by Adair et al (1998, 1999). Exposures of the dorsal body for 45 min to either 450 MHz, or 2450 MHz, resulted in vigourous sweating which was related to the ambient temperature and applied power level but changes in deep body temperature were limited to 0.1 °C. They concluded that at SARs up to 7.68 W/Kg are mildly hyperthermic but are moderated by normal heat loss mechanisms.

In animal studies, it has been demonstrated that the depth of penetration is dependant on the frequency, being greater at lower frequencies (Jauchem et al., 2000). Heating at deeper levels could effect the central nervous system and endocrine systems, while heating at higher frequencies would effect tissue at the surface ad peripheral nervous system (Jauchem et al., 2000)

Using a realistic head model Van Leewen et al. (1999) calculated the use of a cellular phone would cause a maximum brain temperature rise of 0.11 percent for an antenna with an average emitted power of 0.25 W. This produced a maximum average SAR of 1.6 W per 10 g of tissue. A SAR of this value was assessed as being too small to have lasting effects. A similar study by Wainwright (2000) calculated a maximum temperature rise in the brain of 0.1 degree celsius. They studied 6 configuration of antennae, hand and head, based on a 900 MHz or 1800 MHz transceiver with a 1 W total power output.

Van de Kamer and Lagendijk (2002) reported average SAR distributions in an adult female head from a radiating vertical dipole antenna (915 MHz) as 1.72

W/Kg for 1 g of tissue and 0.98 W/Kg for 10 g of tissue for a cubic and for an arbitrary shape, 2.55 W/Kg(1 g of tissue), 1.73 W/Kg (10 g of tissue). They used high resolution modelling. Anderson and Joyner (1995) measured SARs in a phantom head exposed to an analogue phone and found similar SARS. In the brain closest to the phone SARs were 0.12-0.83 W/Kg.

There has been concern that children may differ in energy absorption to adults. Schonborn et al. (1998) compared models based on a child's head with that based on an adult and concluded there were no significant differences in the absorption of energy in near field sources.

In a detailed study, Bernardi et al. (2000) evaluated local SAR distributions and temperature increases in a model of the human head exposed to RF fields radiated by several models of cellular phones. They reported that the SAR limit of 1.6 W over 6 g of tissue was exceeded in all situations considered. The phone used was typical of analogue phones radiating 600 mW in free space. Digital phones have a lower radiated power of 250 mW so the SAR values would be less and within IEEE limits. The temperature increases calculated at 1.6 W/kg were 0.09 degrees celsius, which were 20 times below those at which thermal damage is likely to occur.

Nitric Oxide

The use of a commercially available cellular phone caused an increase in nasal nitric oxide (Paredi et al., 2001).

Melatonin

So far there is no evidence that melatonin levels are effected by RF EMFs. If RF EMFs are shown to effect melatonin production this could occur by acting directly on the pineal gland or by heating effects, or indirectly via other systems. As RF photon energies lie between ELF emfs and the visible parts of the Electromagnetic spectrum neither has the ability to cause suppression of pineal activity via the photo pigments in the eye in the manner light does (IEGMP,2000).

Demodulation of Amplitude Modulated RF Energy

Microwaves are frequently modulated by ELF EMFs. Litovitz et al., (1997) noted that microwave fields amplitude modulated by an ELF sine wave EMF at 50 or 60 Hz can induce a 2 fold increase in ornithine decarboxylase activity at

SARS of 2.5 W/Kg. They also found that the superposition of ELF noise can mitigate this enhancement in both ELF mediated effects and ELF amplitude modulated microwave EMFs.

Effects on the Cellular Morphology of the CNS

RF-induced changes in cellular morphology are not expected except at high intensity or prolonged exposure. Changes occurring at high intensities include haemorrhage, oedema and vacuolation in neurons (Lai,1994).

Metabolism in neural tissues

Studies on isolated neurons have produced conflicting results. Some studies report a decrease in spontaneous activity, an increase in membrane conductance and a prolonged refractory period (Hermann and Hossman, 1997). The effects were abolished when EDTA was used to chelate calcium suggesting a change in calcium homeostasis. Several studies have reported an increase in calcium release from neural tissue when it is exposed to RF fields.

Changes in Blood Brain Barrier Permeability

RF exposure levels that raise brain temperatures causes an increase in BBB permeability. This is consistent with an increase in permeability that occurs with a temperature increase regardless of cause (Schirmacher et al., 2000).

Disturbances in Blood flow

It is possible that RF fields may cause local disturbances in blood flow (Huber et al. ,2003). This is a likely cause for the feeling of warmth behind the ear. Wilen et al (2003) suggests that warmth behind the ear as opposed to in the ear, is more likely to be due to RF effects rather than from conduction.

Frohlich's Coherent Excitations

Hyland (1998) explained that the concept of coherent excitations relies on the prevalence of electric dipoles in living matter. Frohlich's model proposed that above a certain rate of energy supply, part of the incoming energy is channelled into the lowest frequency vibrational mode associated with the system of identical electric dipoles. Individual dipole units vibrate in phase so the macro system of dipoles oscillates as a single electric dipole. Possible sites for the occurrence of these macro dipoles are regions of the cell membrane associated with embedded proteins. "Any particular coherent excitation exists only within a certain power window"(p.265) The ability of external microwave

radiation of millimetre wavelengths to influence cellular (and even sub cellular) processes most likely arises from its transformation into various internal, coherent mechanical vibrational modes of sub cellular wavelengths" (p.267).

Zeeman-Stark Model of RF Interaction with Ligand Binding

Chiabrera et al. (2000) describe the effect of RF fields on ligand binding to hydrophobic receptor proteins. A ligand controls receptor function, as docking with the receptor completes the hydrophobic core and activates the receptor. This then directs the alignment of its secondary structural elements. RF fields may effect this process if the energy of the RF photon matches the depth of the potential energy well of the receptor protein. This effects ion binding probability.

RF-Induced Changes in Protein Conformation

Bohr and Bohr (2000) describe an example of non-thermal effects of RF fields in which a 2.45 GHz RF field enhanced the kinetics of the folding and unfolding of the protein ß-lactoglobulin.

Summary

Effects of Power Frequency EMFs on Living Systems

In humans physical effects of EMFs reported have included alterations in; heart rate, blood pressure, EEG tracings, skin temperature, enzyme and hormone production, reaction time and saccadian eye movements. Psychological variables said to be effected by EMFs have included memory, concentration, reasoning, and simple decision making.

In reviewing the literature the most salient point that emerges is the lack of consistency in effects shown. Even repetitions from the same research team, using the identical protocol in the same facility, may produce significant effects then fail to replicate at a follow-up study. There are a large number of variables to consider when setting up an EMF experiment. Exposure can vary in frequency, flux density, extraneous fields, wave pattern (sinusoidal, square wave) continuous or pulsed delivery, variation in pulse rate, how the field is polarised and many more. This may explain many, but not all, the discrepancies among results.

Effects on Melatonin Levels

Studies on the effects of power frequency EMFs on melatonin production in animals have produced conflicting results. Some studies have reported suppressive effects on the formation of melatonin in rats (e.g. Martinez et al., 1992; Kato et al., 1993 &1994; Loscher et al. 1994, Selmaoui et al., 1995; Mervissen et al., 1996; Bakos et al., 1997; Rosen et al., 1998). Other similar studies on rats, often repetitions by the same research teams, have failed to find significant effects (e.g. Kato et al., 1994; Grota et al., 1994; Bakos et al., 1995; Loscher et al., 1998; John et al., 1998; Bakos et al., 2002). Studies on other species, ranging from hamsters to sheep and baboons, have also produced conflicting results.

Studies on humans have largely produced negative results. A small number of environmental studies has suggested effects (Wilson et al., 1990; Burch et al., 1998; Juutilainen et al. 2000; Hong, 2001). Laboratory studies have generally failed to support these results (Selamoui et al.,1996; Graham et al., 1997, 2000.) A few studies found effects in a possible subgroup whose levels of melatonin were naturally low (Graham et al., 1996; Crasson et al., 2001). The two studies that reported significant effects found delays in the evening rise, rather than suppressions (Wood et al., 1998; Karasek et al., 1998).

Effects on Heart rate and Blood Pressure

Early environmental Russian studies pointed to an effect on heart rate and blood pressure but these studies were unable to be replicated in the West. The few laboratory studies publishes point to a possible slowing of heart rate and increase in heart rate variability when medium intensity, intermittent fields were used.

Effects on Cognitive Processes

A variety of tasks covering memory, attention reaction time, executive functioning, and discrimination have been carried out. Effects have been reported most often in reaction time tasks, with or without an attention component. There are still only a few studies published (about a dozen) and results have been equivocal.

Possible Mechanisms of Interaction with Living Systems

While tentative links have been made between EMFs and their biological effects work is just beginning on possible mechanisms which could explain such a link. Such possibilities include the induction of eddy currents, interaction of EMFs with intracellular magnetite, interference with the release of calcium and hence the second messenger cascade, or the effects of higher intracerebral calcium on the release of inhibitory neurotransmitters such as GABA (Reiter et al, 1994). Stevens (1987) proposed a melatonin hypothesis which suggested the link between EMFs and cancer was via a suppression of melatonin. In 1993, Lidburdy reported a 60 Hz 11.95 mG field blocked melatonin's oncostatic action. If melatonin is 'turned off' by EMFs this could provide a link between these fields and cancer. However, so far studies on EMF effects on melatonin do not support this hypothesis.

Radiofrequency EMFs

Research is just beginning to be published in the cell phone frequency range. Little replication has as yet been done. The IEGMP (2000) identified the following areas of research need; effects on brain function, consequences of exposure to pulsed signals (such as those produced by digital phones), psychological and sociological studies, possible health effects of cellular and sub cellular changes, epidemiological and humans volunteer studies.

Effects on Melatonin Levels

There have been few published studies on the effects of RF frequencies on melatonin. No effects have been found from the one animal study (Stark et al., 1997) and 4 human studies (Vollrath et al., 1997; Mann et al., 1998; de Seze et al., 1998; Radon et al., 2001).

Effects on Cognitive Processes

The nine studies published so far have covered reaction time, memory, attention, verbal fluency. Significant effects have been reported in reaction time, vigilance, numerical memory.

Effects on Heart rate and Blood Pressure

Very few studies have been published in this area. One indicated an increase in blood pressure on exposure but this was not confirmed in a follow-up study. One study reported a decrease in heart rate and heart rate variability with power increased in the high frequency range of the ECG.

Thermal Effects

The small number of studies on humans have indicated a heating effect from the cellphone with a significant increase in temperature in and around the ear, and side of the face. The increases reported range from 0.08- 2.3 °C.

Possible Mechanisms of Action with Living Systems

The main effect of RF fields has been attributed to a heating effect. Other possible mechanisms of interaction include; suppression of melatonin, demodulation of amplitude modulated fields, direct effects on neural tissue such as increased calcium release, changes in blood brain permeability, increased capillary perfusion, coherent excitation of electric dipoles, and changes in protein conformation or receptor proteins.

CHAPTER 5
EXPERIMENT ONE
An Investigation into Possible Effects from a Pulsed 50 Hz, 100 μT_{rms} EMF on Salivary Melatonin Levels and the Cognitive Parameters of Attention and Aural Working Memory

For the last two to three decades there has been public concern regarding possible health effects from exposure to EMFs. Epidemiological reports have suggested a link between exposure to power frequency EMFs and the occurrence of cancers such as leukaemias and breast cancer (Wertheimer and Leeper, 1979; 1982, 1995). A need existed to identify possible biological mechanisms to explain such a link. In 1987, Stevens proposed his melatonin hypothesis which suggested that melatonin production may be suppressed by exposure to EMFs in the same manner as natural light suppresses melatonin production. As melatonin is a free radical scavenger, if such a suppression occurred, it could theoretically result in an increase in malignancies.

There have been several occupational or environmental studies reporting suppression of melatonin levels consequential to chronic exposure to ELF EMFs (Wilson, 1990, Burch et al, 1998; Juutilainen et al, 2000, Hong, 2001). However, the possibility exists that the results in occupational studies may have been due to other factors in the environment, such as high light intensity or the use of chemical solvents.

There is a substantial body of laboratory research reporting reductions in the levels of the brain hormone, melatonin, in rodents exposed to EMFs (Martinez et al., 1992; Kato et al., 1993, Loscher et al., 1994; Selmaoui et al, 1995; Mevissen, 1996; Rosen et al, 1998). Although, other studies on rodents have failed to find effects (Grota et al., 1994; Bakos et al., 1995 & 2002; John et al., 1998). Studies on other animals, such as sheep and baboons, have also failed to find effects (Lee et al. 1995; Rogers et al., 1995). Field intensities have often been higher than would occur in a normal environment. Exposure times tend to have been for periods of several weeks.

There have been few laboratory studies on humans. Wilson et al. (1990) reported a decrease in the urinary metabolite of melatonin during a chronic (8 week) exposure to electric blankets, but only for the group on a type of blanket that switches on and off frequently. Other studies have reported no effects on melatonin levels (Selmaoui et al., 1996; Graham et al., 1997, 2000;) but these studies used low intensity fields of 10 μT to 28.3 μT. Two studies, (Graham et al. 1996; Crasson et al. 2001) reported effects, but only in people with naturally low levels of melatonin. The study by Graham (1996) had found that effect using a 1 μT continuous, or 20 μT intermittent field, overnight. It may be that those subjects were more susceptible to the effects of EMF exposure. This raises the question as to whether a higher flux density may have produced an effect in the other subjects. A follow-up study by Graham (1997) failed to replicate this result but the field used in the follow-up was continuous rather than intermittent. Findings have suggested that frequent switching on and off may be an important factor mitigating response to an EMF (Lerchl et al., 1991; Wilson et al. 1990; Cook et al., 1992).

All the above mentioned studies used only male subjects, ostensibly to avoid possible confounders produced by variations in the female menstrual cycle. Though studies have not been done to assess whether this is, in fact, a problem. Confining subjects to males only, excluded half the population and it can not be assumed that male data can be extrapolated to include the female population. If hormonal differences can be considered important enough to be a confounder then such differences could also be a potential coagent in any possible effects from exposure to EMFs. Just as subjects with low naturally occurring levels of melatonin may be more susceptible to the effects of EMFs (Graham et al., 1996) then females may by more susceptible at times in the menstrual cycle when melatonin levels are lower.

Tests of attention and working memory are a central focus of neuropsychological assessment as they are sensitive to disruption by brain injury (Gordon et al. 1997). Consequently, they may be the most likely cognitive parameters in which to find effects from exposure to EMFs.

Tests on attention and memory were included in this study because the literature contained little research on possible effects on these parameters in humans. An epidemiological study by Gamberale (1989) had found no effect on reaction time,

vigilance, numerical memory and digit symbol (which is a task of executive functioning) in workers exposed to a 400 kV power line. Cook et al. (1992) reported no effect on vigilance, addition and numerical memory but had found a decrease in errors on a choice reaction time task on exposure to a 50 Hz, 20μT EMF. Several other studies had been carried out on possible effects on reaction time (Lyskov, 1993; Graham et al., 1994; Cook et al.,1992, Whittington et al., 1996a). Whilst the current study was underway three papers were published. Beale et al (1997) found no effect on attention and memory but did find a decrease in digit symbol in people living under high voltage transmission lines. Preece et al. (1998) found significant field effects for numerical working memory, delayed word recall and choice reaction time in subjects exposed to a 0.6 mT EMF (length of exposure not stated). Trimmel et al. (1998) reported a reduction in visual attention, perception and memory as a consequence of exposure to a 50 Hz, 1 mT EMF. The subjects were also exposed to a 45 dB noise which will have made the task more difficult.

It has been suggested that EMF effects may be found only on relatively difficult tasks (Cook et al., 1992; Graham et al. 1994; Whittington et al., 1996a). For example, Whittington et al. (1996a) found a decrease in reaction time only on the hardest level of a visual discrimination task during exposure to a 50 Hz, 100μT EMF. Consequently, it is important to ascertain if possible effects from an EMF increased with the difficulty of the task. It is also possible that this effect may be further magnified by circadian effects. This possibility has not been explored.

If an EMF does effect attention or working memory then there could be important safety consequences. For example, the ability to discriminate between letters presented in morse code is a vital skill for pilots as navigational aids are identified by their morse code ident. A task similar to the working memory task presented to the subjects in this experiment. Disturbances in attention could have serious consequences for operators of machinery.

Experiment One was carried out in 1997/1998. The aim was to investigate possible acute effects from a 50 Hz, 100 μT pulsed EMF on the dependant variables of melatonin levels, attention and working memory.

Fifty hertz was chosen as it is the frequency used by most domestic and office appliances and hence the frequency to which the public is most commonly exposed. The flux density of 100 μT was chosen as the aim was to ascertain

whether the partial effect found by Graham et al. (1996) using 20 μT could be expanded using a higher field intensity. It is also the recommended maximum continuous public exposure permitted under National Radiation Laboratory (1996) guidelines and is a common level of exposure when using appliances, such as hairdryers.

Two times of day were chosen, midday and midnight, to allow for possible circadian effects on the susceptibility to an EMF. The use of midday and midnight allowed investigation of the possibility that effects of the EMF may be greater when melatonin levels are naturally low as well as the possibility of the EMF suppressing or delaying the evening melatonin rise. The design allowed for the possibility of EMF exposure enhancing melatonin production, though this has not been reported in the literature. It also allowed for possible circadian effects on attention and memory. It may be that an EMF may be more likely to show an effect at night, when a person is tired and working closer to their limits.
The EMF was pulsed as it may be the change in field condition that produced effects. Wilson (1990) had attributed the significant effects found in that study to the electric blankets frequently switching on and off. The one second on/ off timing of the pulse was chosen as it had previously been associated with field effects (Lyskov, 1993).

The length of exposure was 30 minutes. Lewy et al. (1980) had reported that the change in melatonin levels as a response to changing light intensity is quite rapid, within 30 minutes. If EMF exposure effects melatonin levels by the same mechanism as light does then an exposure time of 30 minutes should be enough to produce an effect.

A primary aim of the study was to examine the effects of an EMF on melatonin levels at night when levels are high and in the daytime when they are low. The possibility that those with naturally low levels of melatonin form a subgroup of people who may be susceptible to an EMF, was investigated. Possible differences in gender responses to exposure were also investigated.

Neuropsychological studies have suggested that "deficits in memory are secondary to attentional and problem solving deficits" (Eichenbaum and Cohen p.475, 2001). Consequently it was considered important to include a simple attention test in this study. The attention task was similar to that subsequently used by Beale et al. (1997) and Crasson et al., (1999) and was a test of attention

and visual scanning.

The aural memory task was a discrimination task with three levels of difficulty. Using the same task but varying the difficulty was done to reduce the possibility of adding confounders by changing the task. The current study was carried out to add to the research in the important areas of attention and memory adding a circadian factor not present in the above studies.

MATERIALS AND METHOD

Experimental Design

The study was a repeated measures design comprising two exposure and two control sessions. The sessions were: an on-field exposure and an off-field control beginning at either 1200 or 1300 hours, and an on-field condition and an off-field condition beginning at 2400 hours. Each session lasted 30 minutes.

The experiment used a randomised block design with a counterbalanced order. (Whether a group was in a day or night session first, or an exposure or control condition first, was randomised using random number tables). To control for practice effects half the subjects received a control session first and half an exposure session. Alternative forms of the tasks were also used to reduce practice effects.

Each individual subject completed their four experimental sessions over a consecutive two day period to reduce fluctuations that may occur due to the menstrual cycle in female subjects, or variations in health status. It also reduced the possibility of changes in circumstance that may affect cognitive performance.

The protocol was double-blind to the extent that the field status was unknown to both the subjects and the technicians carrying out the RIA or marking the tasks. During initial trials a computer ran the experiment and the experiment was fully double-blind with the experimenter also being unaware of the field status. However, a computer fault turned off the field with subsequent loss of data and so to ensure exposure occurred when it was supposed to, the field was switched on manually by the experimenter. Subjects were told they would always be in a field but that it would be varied. (This was a slight subterfuge as the ever present field in the control sessions was the earth's magnetic field). Up to four subjects undertook the experiment at any one session.

Each subject acted as their own control. The use of subjects as their own controls increased the sensitivity of the experiment. This was considered advisable as melatonin levels show large inter-individual variations and cognitive performance can also vary widely between subjects.

To control for time of day effects, the control sessions were on a different day at

the same time of day as the exposure session. It was generally accepted in the literature that normal human melatonin rhythm is intra individually consistent in amplitude and circadian profile, "rather like a hormonal finger print" provided environment and behaviour remain relatively constant (Arendt, 1995, p.209). Consequently it was considered a separate day control was acceptable.

To control for possible confounding variables, subjects were screened and eliminated from the study if they had health problems, used medication, smoked, or participated in activities that could affect the production of melatonin (e.g. were on anti-hypertensives, or had epilepsy). They were asked to limit activities prior to each session which could effect the research.

Instructions were either read from a sheet or played on an audio tape to ensure a standardised delivery.

Subjects

Subjects were volunteers recruited from amongst the staff and students at Massey University. The aim was to recruit 80 subjects which would have provided a power level of 0.8 for a small effect size using p=0.05 (Cohen, 1969). Graham et al (1997) had estimated 40 subjects was required for a power level of .80 but they assumed a medium reduction of 25-30 % in melatonin levels, a figure they had based on animal studies. As humans are not as sensitive to light as animals (especially rats, which are the most common experimental model), it could be expected that any reduction in melatonin due to EMF exposure would also be less than that reported for animals.
Unfortunately the research space was lost and the study had to be truncated at 29 subjects. Each subject attended two day and two night sessions, with the exception of subject nine who missed the day sessions. For the night sessions there were a total of 29 subjects, 8 males and 21 females. During the day there were 28 subjects, 20 female and 8 males. Ages ranged from 18 to 53 years, with a mean age of 23.7 years.

Subject screening

Prior to the first session, subjects were given an information sheet explaining the research and their rights as participants. They then completed a health screening questionnaire and signed a consent form (see Appendix 1.1). Any subject

responding in the affirmative to the screening questions was thanked for their time but excluded from the study. Smokers were not included in the study. To control for possible confounding variables, volunteers were asked to refrain from moderate or excessive exercise and taking alcohol for 6 hours prior to each session. They were also asked to restrict coffee, tea and coke intake to not more than one cup in the 4 hours prior to the experiment. They were also asked to avoid bright light prior to the night session.

Ethical approval

The research was approved by the Massey University Human Ethics Committee in 1996.

Exposure System

Location

The equipment was located in a room 5.5m by 4.5 m. There was no shielding in place. It was on the top floor of a three story building at Massey University. The exposure system was essentially the same as used by Kazantzis et al. (1998).

Positioning of the coils and seating

Four booths were formed by two sheets of particle board, 1.8m square, standing at right angles (Figures 5.1 and 5.4). Pairs of Helmholtz coils were suspended from PVC piping attached to the top of each booth. Subjects sat in a resin chair facing inwards and were not visible to other subjects. Heads were kept in the correct position, parallel to the coils, by a wooden head rest. No metal parts were used in the construction.

Lighting and Temperature

The room containing the equipment had no windows so contained no natural light. Lighting was provided by a clear glass 40 W bulb which was directed towards the ceiling and shaded from shining directly on the subjects. It was located on the crosspiece of the particle board (Figures 5.1 and 5.4). When a subject was looking at the page of tasks they received a reflected illumination of 226 lux, but this rose to 540 lux if the subject looked directly at the light source. The luminance was 0.8 cd/m^2 if looking towards the light source and 0.6 cd/m^2 when looking at the page of tasks (Measurements were taken using a Hagner Universal Photometer Model S3). The subjects were asked not to look directly at the light and the position of the head rest made it very difficult for them to do so.

Generation of the EMF

A function generator created a 50 Hz sine wave which went through a zero crossing switch thence to a 300 W amplifier. The amplifier produced a current of 0.18 A (rms) which flowed through the four pairs of coils in series (Figure 5.2). The resistance across the coils and connecting wires was 13 Ω giving a power of 0.4 W dissipated by the coils, neglecting any power radiated by the coils. Each Helmholtz pair created a field of 100 μT_{rms} between the coils. This was confirmed at each session using a custom-made Hall effect probe attached to a multi meter.
The EMFs produced by each coil pair were undetectable with the Hall effect probe at a distance of 0.3 m from the coils, in the direction of the adjacent coils. Consequently interference between coils was unlikely. There was no detectable heat, vibration or sound produced by the coils, but the amplifier contained a fan which was audible. For this reason the amplifier was switched on during both control and experimental sessions to protect the blind nature of the experiment.

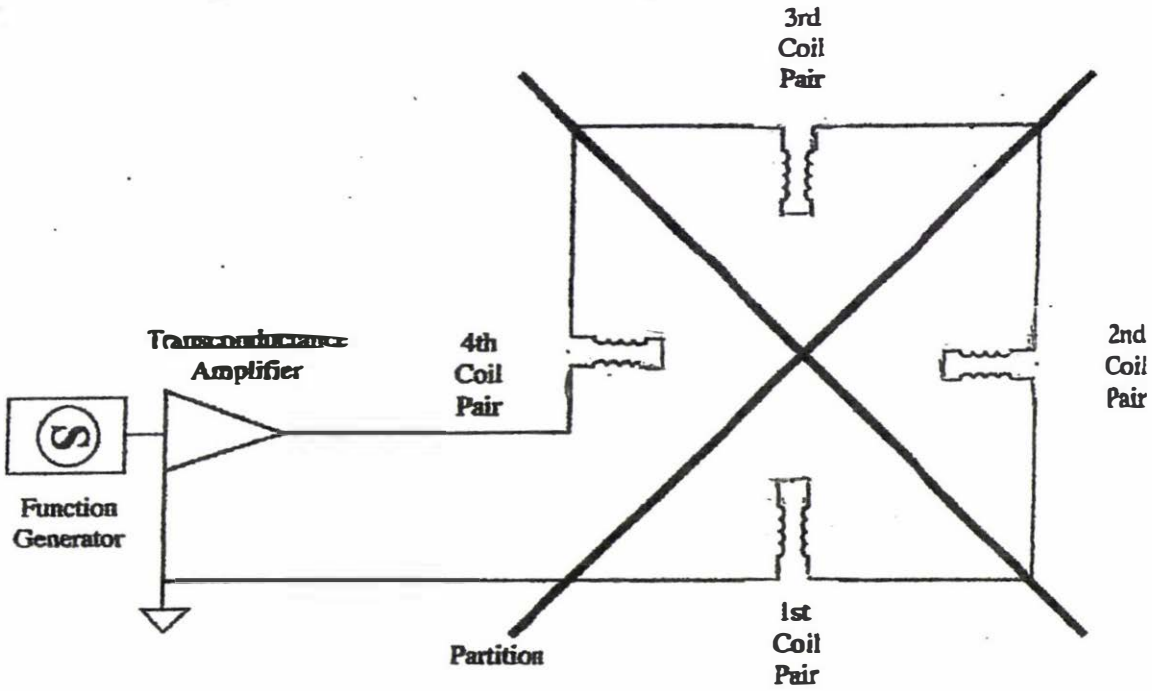


Figure 5.1 Electrical system used to generate the 50 Hz EMF

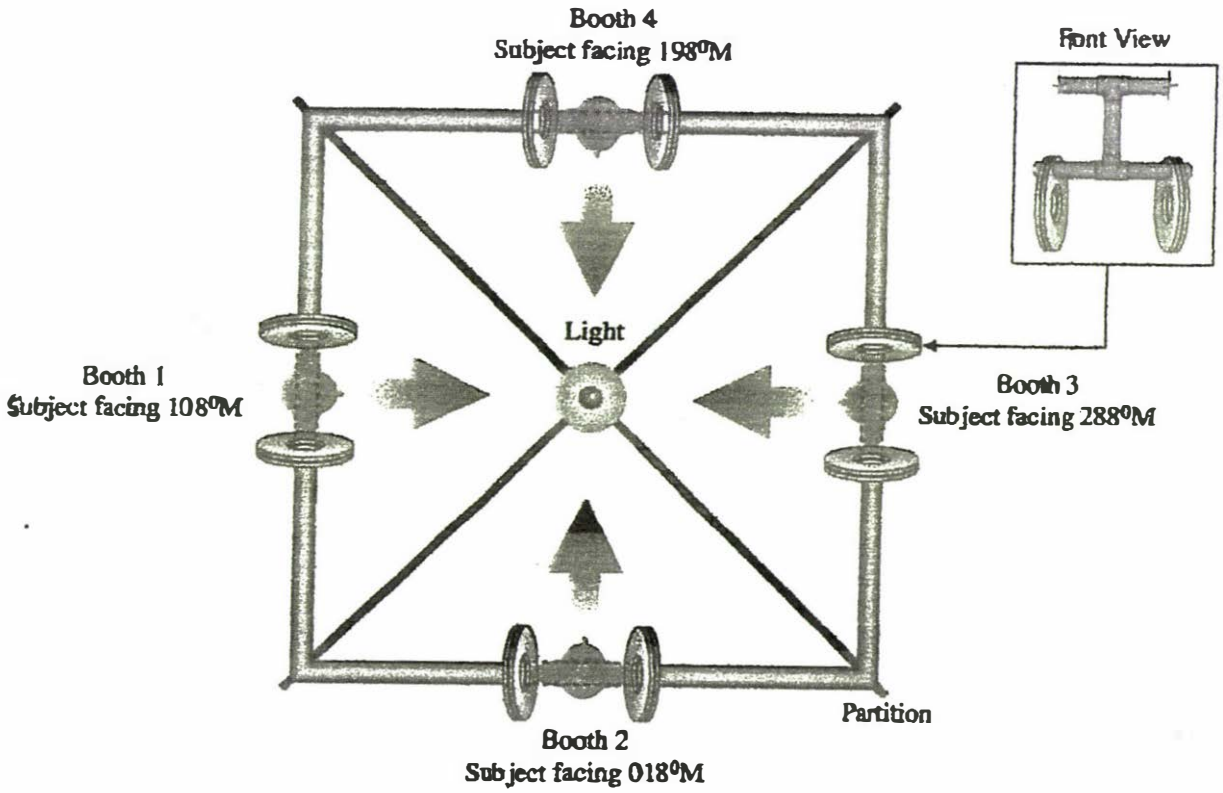


Figure 5.2 Top view of the booth configuration

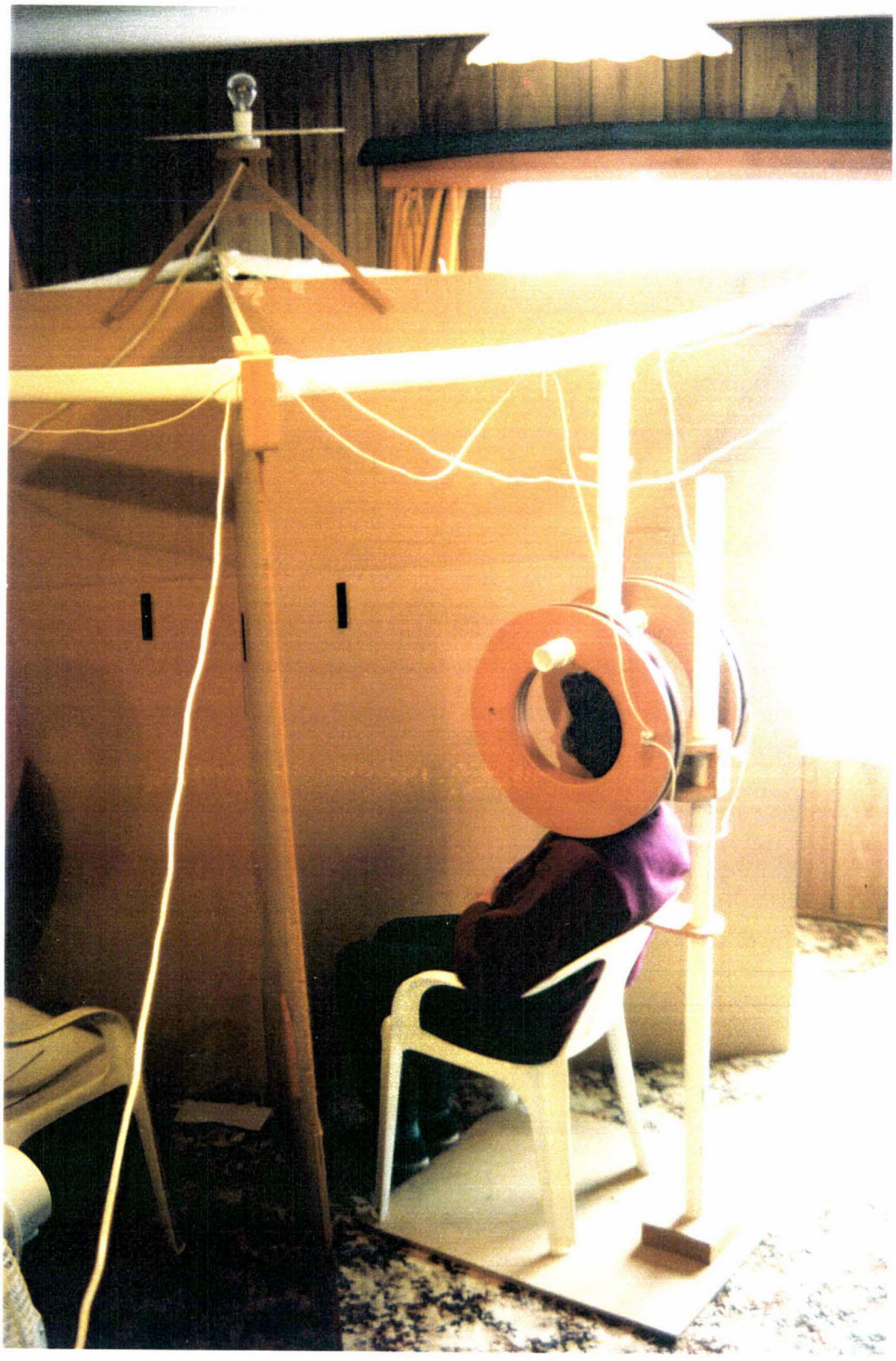


Figure 5.4: Subject seated in position between the coils.

(This photo was taken during experiment 2. All natural light was blocked for the experiment.)

Characteristics of the EMF produced

The EMF has been described following the protocol laid down by Valberg (1995).

Exposure intensity and timing

Subjects were exposed to a flux density of 100 μT_{rms} for 30 minutes per session. Each subject was exposed to four sessions: One control and one experimental session beginning at 1200 hours, and one control and one experimental session beginning at midnight.

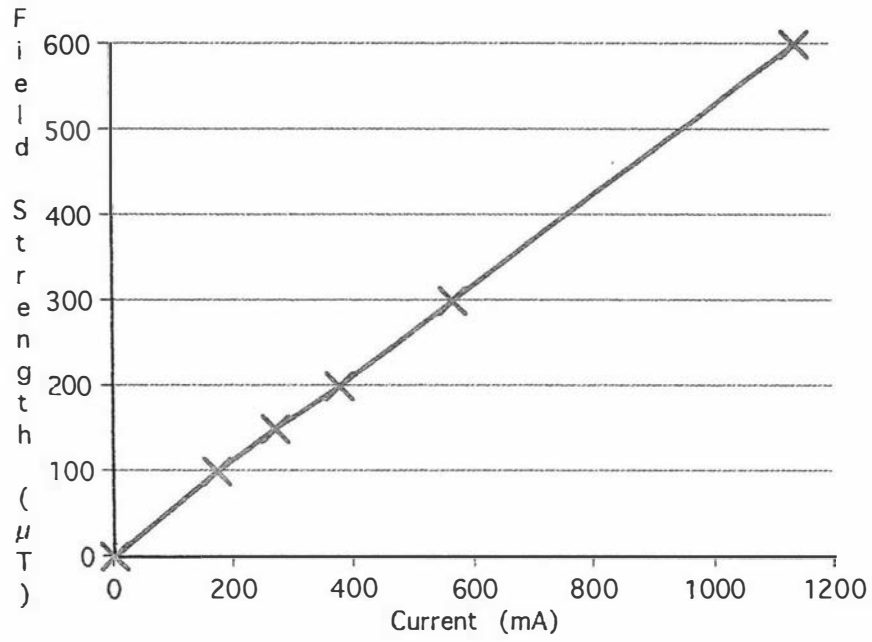
Exposure frequency-domain characteristics

The field was purely sinusoidal at a frequency of 50 Hz and was presented in a one second on, one second off pattern. The current was passed through a zero crossing switch to avoid the occurrence of spikes.

Exposure geometric (spatial) characteristics

The field was produced using a Helmholtz configuration, giving a linear polarisation. Coils were made from 0.001 m mean diameter copper wire with 120 turns per coil. The coils were 0.39 m in diameter and were placed 0.19 m apart. The field was homogeneous to +/- 10% of the nominated value.
A regression analysis of the six measuring points in the field between the coils produced a correlation coefficient of r^2=0.999423. The coil constant was 189.5 mA/Gauss (Figure 5.3) .

Measuring Point	I (mA)	B (μT)
1	0	0
2	173	100
3	271	150
4	378	200
5	563	300
6	1140	600



r^2 for all data was 0.999423.

The coils constant was 189.5 ma/100 μT

Figure 5.3 Helmholtz Coils Calibration Curve and Regression Analysis

Parameters Tested and Treatment of Data and Samples

Subjects carried out an attention task and an aural working memory before providing a saliva sample for the determination of salivary melatonin levels. Each subject acted as their own control which increased the sensitivity of the design. The paired t-test was chosen for the melatonin level analysis and memory task as the inter-individual variation could be expected to be larger than may be produced by a small field effect. The attention task was analysed using a multivariate ANOVA as two parameters (time and accuracy) were tested.

Attention task

Neuropsychological studies have suggested that deficits in memory are secondary to attentional and problem solving deficits" Eichenbaum and Cohen (p.475, 2001). Consequently, it was considered important to include an attention test in this study. The task chosen has several variations and is commonly used in N.Z. for the assessment of neuropsychological deficits in attention. It "is a measure of several inter -related functions which include sustained attention, visual scanning, response inhibition and rate of information processing. "Gordon et al. 1997. p.325. The task was chosen as these functions are very sensitive to the effects of brain injury so they may also be susceptible to possible interference by EMFs. Variants of the task have been used by other researchers studying the effects of EMFs (e.g. Beale et al, 1997; Crasson et al., 1999).

The attention task involved placing a vertical line through each number '6' on a page of numbers obtained from a random number table. Numbers were arranged in groups of 5. Two different pages of numbers were used. Page A for the first and third sessions and page B for the 2nd and 4th sessions. Page B differed from page A in that numbers had been shifted within groups, but not between groups. Groups had been shifted within, but not between lines. Alternating the pages reduced the possibility of subjects remembering the position of the '6's.

The response sheets were graded by a person unfamiliar with the protocol who was trained by the experimenter for the task. Results provided data on the number of '6's correctly crossed, number missed, inaccurate answers and time taken for each of the four sessions. The score was number of 6s crossed minus number of 6s missed. No subjects crossed incorrect numbers but if they had these would also have been subtracted from the score. The difference between exposure

and control session scores was evaluated using a multivariate analysis of variance.

Aural working memory task

Memory can be assessed using a variety of tests aimed at particular parts of memory. However, many tasks were considered unsuitable for use in a situation where the person must sit with their head between two coils. As some of the subjects were psychology students it is also likely that many would have been familiar with such tasks, introducing a possible confounding variable. It was decided to design a novel task that was expected to tax subjects. None of the subjects were familiar with Morse code. The task was graded with 3 levels of difficulty. Using the same task but varying the difficulty also reduced the possibility of a change in task introducing a confounding variable. Signal detection theory suggests that effects may be more likely to be detected as limits of performance are approached (McNicol, 1972).

The aural memory task consisted of the subject listening to an audio tape containing groups of letters in Morse code. Groups contained either two, three or four letters. There were 66 groups of letters. Whether a group had any letters repeated was selected using random number tables (RNT). Whether a group contained 2, 3 or 4 letters was also determined by RNT. Subjects were asked to decide if any of the letters in the group was repeated and responded by ticking either the 'yes' or 'no' column.

The use of the tape recorder ensured a standardised delivery of the instructions at each session. There was no audible pickup on the tape recorder from the operation of the coils.

This task took 25 minutes. Data generated included; the percentage correct for each level of difficulty (2, 3 or 4 letter groups) and also the total correct for each of the four sessions. The response sheets were graded by a person unfamiliar with the protocol who was trained by the experimenter for the task. The difference between exposure and control sessions was evaluated using the student's paired t-test.

Determination of salivary melatonin concentrations

In order to determine whether EMFs effected melatonin secretion, saliva samples were collected at the end of each experimental session. Due to the difficulty of providing a sample whilst seated between the coils the subjects were told to save saliva for the last few moments of the experiment and provide the sample immediately on leaving the EMF. During the trial runs the subjects had been able

to provide the necessary 0.25-0.3 ml in less than 3 minutes.

The decision to use saliva samples to measure melatonin levels was made as it provided a non-invasive measure of melatonin levels and did not require the qualified personnel or special equipment that blood sampling would have required. The provision of a saliva sample was also more acceptable to the subjects than invasive methods such as blood sampling. The collection of urine for the analysis of melatonin levels would have provided a longer sampling interval but was not practical for this experiment.

The use of salivary melatonin as an indicator for plasma melatonin has been validated by Nowak et al. (1987), who concluded that saliva sampling was a reliable non-invasive method for studying melatonin levels in humans. They further noted that the diurnal profile present in plasma was also present in saliva, but the concentration of melatonin in saliva was about 30% of that found in plasma. This is because only the 30% of melatonin not bound to albumin is free to enter saliva. Using salivary melatonin had the disadvantage of being less sensitive in detecting the presence of melatonin, especially at times of day when levels in the body were lower. Radon et al. (2001) also concluded there is ample evidence that melatonin can be reliably determined in saliva.

Melatonin has a wide inter-individual variation so subjects needed to be their own controls. Melatonin levels also vary seasonally and over the menstrual cycle for women, so individual sampling needed to take place over as short a time as possible. To reduce any possible effects from these variations each subject completed their four sessions within a two day period.

Low naturally occurring levels of melatonin were defined as below 55 pg/ml of peak value melatonin in plasma by Graham et al. (1997) and later by Crasson et al. (2001). Graham reported the peak occurred at 2 a.m. Data provided by Graham et al. (1997) gave the midnight mean level of melatonin as 30 pg/ml. in blood. As only one third of circulating melatonin enters saliva (Nowak et al.,1987) a blood value of 30 pg/ml equates to a value of 10 pg/ml in saliva. This value was used as the level at, or below, which subjects were classified as having low naturally occurring levels of melatonin for this experiment. The subjects' melatonin level on the night control session was the criteria used to select which subjects would be included. Subjects with a zero melatonin value in the control session were not

included.

Subjects provided a saliva sample at the end of each session. Samples were collected in 5 ml capped plastic tubes, either without stimulation, or by stimulation from the chewing of parafilm. Samples were frozen within an hour of being collected and remained frozen until they were assayed. According to Vakkuri (1985) samples can be stored at -20 °C for months or even years, without deterioration. A standard saliva pool of known melatonin concentrations was assayed at the same time as the samples. Due to the high number of samples multiple assays were required but all samples relating to each individual subject were assayed in the same batch.

RIA was chosen as the method for determining melatonin levels as, according to Arendt (1995), there is reasonable consensus that either RIA or gas chromatography-mass spectrometry are the appropriate methodology for the determination of melatonin levels in body fluids. RIA was chosen as a RIA laboratory was already set up in the department, run by a trained RIA technician. The technician tested the samples as this meant the testing was done by someone unfamiliar with the conditions of the experiment to remove a possible source of bias.

Differences between melatonin levels collected during control and exposure sessions were evaluated using the student's paired t-test. The paired t-test was chosen as it had been used frequently in the literature and enabled subjects to be their own controls. This was important as there is wide inter-individual variation in melatonin levels.

Radioimmunoassay for the detection of melatonin concentrations in saliva

Materials

1.Tricine Buffer

250 ml of buffer was made up using 2.25g NaCl (Reidel-de- Haen), 0.25g gelatine (BDH), 25 mg NaN_3 (all AnalaR), and 4.48g Tricine (Sigma Chemical Company, St. Louis, USA). Buffer was stored at 4° C after adjusting to pH 7.7 with 10 N NaOH. (Buffer was discarded after week).

2. Tracer

(O-methyl-^{3}H) melatonin (250 μ Ci) (TRK798, Amercontrol International, Little Chalfont, England) was stored at -20 °C at a dilution of 1% in ethanol. A working solution was made fresh each day by drying the

dilution under nitrogen to provide approximately 4000 cpm when dissolved in buffer. Prolonged exposure to heat or light was avoided.

3. Antiserum

Sheep anti-melatonin antiserum Guildhay G/G/704-8483 was used at appropriate dilutions. Partial dilutions in buffer were stored frozen in assay-sized aliquots and thawed as required.

4. Saliva for the standard curve

Saliva for the standards was collected from a single individual during the day when melatonin levels were very low or undetectable.

5. Standards

Standard curves were made by diluting a melatonin stock standard solution (1 mg/ml in absolute ethanol) with buffer to give a top standard of 1000 pg/ml, then serial dilutions in buffer to 7.8 pg/ml., plus buffer alone as a zero standard.

6. Saliva samples

After thawing, samples were vortexed then centrifuged at 3000 r.p.m. at 4 °C for 25 minutes, to remove any solids.

7. Activated charcoal

0.4% activated charcoal (Sigma Chemical Company, St. Louis, Mo, USA) with 0.004% Dextran T70 in pH 7.7 Tricine buffer was continuously stirred at 4 °C. Buffer was prepared one day ahead of use.

8. Scintillation fluid

Toluene/triton X100 (15g PPO and 500 mg POPOP) were dissolved in 3.334 L of toluene and 1.667 L of triton X100 was added. 8 ml was added to each tube supernate. (PPO= 2,4,5 Diphenyloxazol, Sigma Chemical Co. Mo. USA) (POPOP= 1,4-bis[5-Phenyl-2-oxazolyl]-benzene;2,2'-p-phenylene-bis[5-phenyloxazole]) Sigma Chemical Co. St. Louis, Mo 63178 U.S.A. Lot 35401160).

9. Organic Solvents

Absolute ethanol (May & Baker Australia Ltd., Victoria, Australia)
Toluene (Shell Chemicals, New Zealand Ltd., New Zealand)
Triton (Rohm and Haas N.Z. Ltd., Auckland, New Zealand)

Assay protocol

(After that of Fraser, S.; Cown, P; Franklin, M; Franey, C and Arendt, J.. In Clinical Chemistry, 1983, 29(2) 2, 396-7.)

Disposable plastic tubes (10 x 75mm) were used with Blohit & Gilson pipettors

with polyprop tips and Eppendorf Multipelte 4780 and tips. Duplicate tubes were set up for totals, NSB (non-specific binding), standards and samples. Assay drift was checked with beginning and end zeros and quality control samples for inter-assay and intra-assay variation estimations.

Procedure

1. Initially 500 μl of sample or standard was added to the assay tubes.
2. 200 μl of antiserum was then added to the tubes, vortex mixed, then incubated for 30 minutes at room temperature in the dark. (Tubes for Totals and NSB received 200 μl of buffer instead of antisera).
3. Tritiated melatonin was prepared, mixed gently and 100 μl was added to each tube. Tubes were then vortex mixed and incubated for 18 hours at 4 °C. Foil was used to cover the tubes to avoid prolonged exposure to light.
4. The antibody-bound melatonin (supernate) was separated from the free fraction by incubation for 20 minutes at 4 °C with 500 μl dextran-coated charcoal (Buffer was used in the totals). Addition was done quickly from a constantly stirred slurry using a multi-pipette. The tubes were vortexed immediately, then remained still for the rest of the incubation period.
5. The supernatant was centrifuged at 1500g for 15 minutes at 4 °C.
6. The supernatant was decanted into scintillation vials, 8 mls of scintillation fluid was added and the mixture mixed thoroughly. Samples were left standing for 12 hours until clear.
7. Vials were counted at a constant temperature by a Wallac 1409-411 liquid scintillation counter.
8. Melatonin concentrations were determined from the dose response curve using the Prism computer programme.

Experimental Protocol

The tasks were explained to the subjects and a practice trial of the Morse code task was carried out. This was repeated until the subjects were satisfied they knew what to do. This initial phase took approximately 20 minutes during which time the subjects became accustomed to the level of lighting and temperature. Subjects were then seated in their allocated booth with the coils positioned on either side of the subject's head. The subject used the same booth for each session. The function generator (low frequency oscillator) and transductance amplifier were then turned on for each exposure session or the amplifier alone was turned on if it was a control session. The reason for the amplifier being turned on for the

control sessions was that it contained a fan which was audible to the subjects. However, with the function generator turned off no current passed through the coils and hence no field was produced. The afore mentioned equipment was not visible to the subjects.

Format followed for each individual session

1.The subjects first carried out a task to test attention, in which the subject was told to put a vertical line through each six on a page of random numbers. The task was timed. All subjects began at the same time and as each subject finished their time taken was written on their sheet.

2. A memory/forced-choice task was then undertaken in which the subjects listened to a tape consisting of groups of letters in Morse code. There were either 2, 3, or 4 letters in a group. Subjects were asked to tick either the 'yes' or the 'no' column in response to the question, "were any of the letters in the group repeated?" Subjects were asked to guess if they were not sure.

3. Each subject provided a saliva sample at the end of each session .

Each of the four sessions lasted up to 30 minutes. Thus the time spent exposed to the EMF field was 30 minutes per exposure, up to 60 minutes in total for the two exposure sessions, with the remaining time being the two sham exposures. The status of the field was checked with a Hall probe at each set of coils during each session. This was done for both control and experimental sessions.

RESULTS

Melatonin Levels

Day sessions

Exposure to the EMF failed to produce a consistent effect on melatonin levels among the subjects. On exposure, salivary melatonin levels were reduced in 14 subjects and raised in 13 subjects, compared with the control session. In one subject the levels remained identical between the sessions (Table 5.1). The level of melatonin ranged from 0 to 47 pg/ml in the control sessions and from 0 to 38 pg/ml for the exposure sessions.

The difference between exposure and control sessions did not reach statistical significance using a paired t-test (-3.8 $\pm$ 2.959 pg/ml, mean $\pm$ SEM, t_{27}=1.284, at p>0.05).

Night sessions

Twenty-nine subjects attended the night sessions. Fifteen subjects demonstrated reduced levels of melatonin production when exposed to the EMF, 12 had an increase in levels and two subjects showed no change (Table 5.1). Changes ranged from a decrease of 40 pg/ml to a rise of 22 pg/ml. Control night values ranged from 0 to 78 pg/ml.

Over all subjects there was no statistical difference between the exposure and control sessions (-1.2 $\pm$2.140 pg/ml, mean $\pm$ SEM, t_{28}=0.561, at p>0.05), following analysis with a paired t-test.

Subgroup of subjects with naturally low levels of melatonin production

There were 8 subjects (4 males and 4 females) who fitted the category of those with naturally low levels of melatonin (Table 5.1).

Day sessions

Exposure to the EMF produced no significant difference between the control and exposure salivary melatonin levels for this group (-1.2 $\pm$7.957 pg/ml, mean $\pm$ SEM, t_7=-0.151, at p>0.05,) on analysis by a paired t-test.

Night sessions

Most subjects in the 'low' group had increased levels of melatonin, two showed no change and only one had reduced levels in the EMF (Table 5.1). In those subjects

with naturally low levels of melatonin production the difference between control and exposure night sessions was not significant.
(3.8 ± 1.709 pg/ml, mean $\pm$ SEM, $t_7 = 2.224$, at $p>0.05$.).

Gender Effects

The possibility of one gender being more susceptible than the other, to the influence of an EMF was investigated but no differences were found. There were 21 females in the night session and no significant field effects were found between control and exposure sessions using a paired t-test (-0.190 ± 2.149 pg/ml, mean $\pm$ SEM, $t_{20} = 0.0884$, at $p>0.05$). An unpaired t-test, comparing 8 males and 21 females, failed to find a difference in response to the EMF ($t_{28} = -0.6707$, at $p>0.05$) (Appendix 1.2).

Assay Sensitivity Levels

During the day, 11 out of the 29 subjects had salivary melatonin levels which were close to, or below, the sensitivity levels for the assay. The sensitivity levels for the RIA are given in table 5.2.

Table 5.2 Assay sensitivity

Subject	Sensitivity (pg/ml)
1-17	6.9
18-29	3.2
mean	5.0

Table 5.1: Expt. One Effects of Power Frequency Fields on Salivary Melatonin (pg/ml)

		Day			Night	
Subject	Control	Exposure	Difference	Control	Exposure	Difference
1	32	31	-1	52	41	-11
2	47	5	-42	18	19	1
3	17	11	-6	16	14	-2
*4	45	9	-36	8	8	0
*5	2	1	-1	5	9	4
*6	47	0	-47	6	14	8
7	2	10	8	21	18	-3
8	33	38	5	23	13	-10
9	-	-	-	0	17	17
*10	8	3	-5	3	4	1
11	6	24	18	18	16	-2
*12	1	1	0	9	8	-1
13	14	18	4	40	35	-5
14	2	5	3	36	24	-12
15	15	19	4	36	31	-5
16	5	2	-3	57	49	-8
*17	0	5	5	2	15	13
*18	4	5	1	6	11	5
19	22	4	-18	49	71	22
20	11	4	-7	54	52	-2
21	4	2	-2	0	1	1
22	4	5	1	24	35	11
23	0	4	4	32	37	5
24	12	6	-6	18	3	-15
25	8	5	-3	18	13	-5
26	10	11	1	78	85	7
27	8	30	22	65	25	-40
*28	9	10	1	10	10	0
29	12	5	-7	12	4	-8
Mean	13.6	9.8	-3.8	24.7	23.5	-1.2
SD	14.537	10.156	15.659	21.557	20.419	11.371
N	28			29		
SEM			2.959			2.140
t=			-1.284			-0.561
* low excretors-10 pg/ml or below in night control sessions (N=8)						
mean	14.5	4.2	-1.2	6.1	9.9	3.8
s.d.	19.705	3.732	22.505.	2.800	3.523	4.833
SEM	6.967	1.319	7.957.	0.990	1.246	1.709
t=			-0.151			2.224

Aural Working Memory

There were three categories of difficulty; 2 letter group (easiest), 3 letter group (moderate difficulty) and 4 letter group (hardest level).

Day sessions

In an analysis using a paired t-test there was no significant difference between the control and exposure sessions. The details of the results from the paired t-tests relating to each level of difficulty are set out below. All levels of difficulty produced null results. However, the hardest level of difficulty (4 letter group) showed the greatest difference between control and exposure sessions.

2 letter group (0.0 $\pm$ 0.381, mean difference $\pm$SEM, t_{27}=0.000, at p>0.05)
3 letter group (0.14 $\pm$0.418, mean difference $\pm$SEM, t_{27}=0.3352, at p>0.05)
4 letter group (-0.214 $\pm$ 0.498, mean difference $\pm$SEM, t_{27}=-0.430, at p>0.05)
(Tables 5.3 --5.5)

Night sessions

This was also the finding in the night sessions. No level of difficulty produced a statistically significant difference between the control and exposure sessions. The details of the results from the paired t-tests relating to each level of difficulty are set out below. As with the day sessions, the hardest level of difficulty (4 letter group) showed the greatest difference between exposure and control sessions (Tables 5.3 -5.5).

2 letter group (-0.24 $\pm$ 0.327, mean difference $\pm$SEM, t_{28}=0.7341, at p>0.05)
3 letter group (- 0.10 $\pm$ 0.403, mean difference $\pm$SEM, t_{28}=0.2484, at p>0.05)
4 letter group (-0.621 $\pm$ 0.619,mean difference $\pm$SEM, t_{28}=-1.003, at p>0.05)

The difference between control and exposure sessions was larger at night.

Table 5.3: Experiment One. Effects of Power Frequency Fields on Aural Memory

Two Lette (Score out of 22)

		Day			Night	
Subject	Control	Experiment	Difference	Control	Experiment	Difference
1	19	21	2	18	20	2
2	20	21	1	22	19	-3
3	22	18	-4	18	20	2
4	19	17	-2	19	22	3
5	20	18	-2	18	19	1
6	18	21	3	20	20	0
7	21	21	0	20	21	1
8	21	22	1	21	20	-1
9	-	-		21	22	1
10	17	14	-3	14	18	4
11	22	21	-1	21	22	1
12	20	21	1	20	19	-1
13	19	19	0	20	19	-1
14	21	22	1	21	22	1
15	20	22	2	22	19	-3
16	21	21	0	22	22	0
17	22	21	-1	22	21	-1
18	21	22	1	22	22	0
19	22	17	-5	22	22	0
20	21	22	1	22	22	0
21	21	22	1	22	22	0
22	22	22	0	21	22	1
23	22	22	0	22	22	0
24	21	21	0	20	21	1
25	22	21	-1	19	22	3
26	22	22	0	21	22	1
27	21	20	-1	22	21	-1
28	15	19	4	20	19	-1
29	19	21	2	21	18	-3
MEAN	20.4	20.4	0.0	20.4	20.7	0.2
SD	1.71	1.99	1.98	1.80	1.42	1.70
N	28			29		
SEM			0.37			0.32

Table 5.4: Experiment One. Effects of Power Frequency Fields on Aural Memory

Three Letter Group (score out of 22)

		Day			Night	
Subject	Control	Experiment	Difference	Control	Experiment	Difference
1	16	13	-3	16	14	-2
2	16	16	0	18	13	-5
3	13	15	2	13	13	0
4	14	9	-5	15	14	-1
5	13	16	3	14	16	2
6	14	13	-1	14	15	1
7	15	18	3	15	17	2
8	15	13	-2	13	14	1
9	-	-		15	16	1
10	11	9	-2	13	13	0
11	12	16	4	14	14	0
12	13	12	-1	11	13	2
13	15	15	0	15	13	-2
14	16	16	0	13	15	2
15	13	14	1	13	15	2
16	12	14	2	16	10	-6
17	17	16	-1	16	17	1
18	16	14	-2	17	16	-1
19	15	12	-3	17	15	-2
20	15	16	1	17	17	0
21	15	16	1	15	14	-1
22	17	18	1	16	18	2
23	16	18	2	18	18	0
24	17	16	-1	15	15	0
25	14	15	1	15	17	2
26	17	14	-3	15	15	0
27	13	15	2	12	15	3
28	14	12	-2	12	15	3
29	17	16	-1	16	15	-1
Mean	14.5	14.5	-0.1	14.8	14.9	0.1
SD	1.72	2.32	2.17	1.80	1.76	2.13
N	28			29		
SEM			0.41			0.40

Table: 5.5. Experiment One. Effects of Power Frequency Fields on Aural Memory

Four Letter Group (score out of 22)

		Day			Night	
Subject	Control	Exposure	Difference	Control	Exposure	Difference
1	14	11	-3	18	17	-1
2	15	18	3	17	10	-7
3	11	13	2	10	12	2
4	16	12	-4	17	14	-3
5	13	15	2	15	14	-1
6	12	16	4	16	13	-3
7	9	15	6	14	9	-5
8	12	12	0	14	12	-2
9	-	-		13	13	0
10	14	9	-5	10	12	2
11	14	15	1	16	11	-5
12	13	14	1	13	12	-1
13	14	12	-2	7	14	7
14	13	14	1	13	10	-3
15	12	13	1	13	14	1
16	13	12	-1	14	9	-5
17	17	14	-3	18	13	-5
18	14	13	-1	15	17	2
19	12	13	1	15	17	2
20	13	16	3	16	11	-5
21	13	15	2	16	17	1
22	18	17	-1	14	18	4
23	17	18	1	18	19	1
24	16	13	-3	13	14	1
25	17	14	-3	17	16	-1
26	13	12	-1	11	15	4
27	11	13	2	14	15	1
28	12	13	1	11	12	1
29	10	12	2	11	11	0
MEAN	13.5	13.7	0.2	14.1	13.5	-0.6
STDEV	2.20	2.07	2.59	2.72	2.72	3.28
N			28			29
SEM			0.51			0.61

Attention

On assessment with a multivariate ANOVA the difference between the control and exposure conditions did not reach statistical significance for either the day sessions ($p>0.05$)(Table 5.6) or the night sessions ($p>0.05$)(Table 5.7). However, the number of correct figures was skewed with a definite maximum. A lot of subjects had scored full marks or close to full marks and the marks of people who scored less than full marks had more scope to vary. A logit transformation was done for the number correct i.e. logit (No. Correct)=log(No. Correct/No. Wrong). This improved the residuals, but the difference between the control and exposure sessions was still not significant ($p>0.05$ in the day sessions, $p>0.05$ at night). There appeared to be little difference between day and night in response to the EMF.

Table 5.6: Expt. One Effects of Power Frequency Fields on Attention.

score out of 116

			Day					
		Control			Exposure			
Subject	No. Correct	Time (sec)		No. Correct	Time (sec)		Diff. no	Diff. time
1	110	142		114	147		4	5
2	109	180		110	194		1	14
3	113	153		115	161		2	8
4	113	143		116	134		3	-9
5	116	166		111	140		-5	-26
6	114	131		115	131		1	0
7	113	147		114	132		1	-15
8	113	227		113	160		0	-67
9	-	-		-	-			
10	106	161		106	173		0	12
11	109	142		109	137		0	-5
12	112	153		111	159		-1	6
13	115	141		114	139		-1	-2
14	115	211		113	211		-2	0
15	110	114		113	125		3	11
16	115	126		113	150		-2	24
17	113	140		113	158		0	18
18	108	156		112	195		4	39
19	115	110		115	134		0	24
20	113	161		111	173		-2	12
21	114	157		115	143		1	-14
22	116	120		116	111		0	-9
23	115	150		116	130		1	-20
24	113	154		115	160		2	6
25	116	162		116	151		0	-11
26	-	-		-	-			
27	99	173		114	153		15	-20
28	112	146		116	149		4	3
29	115	156		116	151		1	-5
					N=		28	
					MEAN		1.1	-0.8
					STDEV		3.46	20.08

Table 5.7: Expt. One Effects of Power Frequency Fields on Attention.

score out of 116

			Night				
		Control		Exposure			
Subject	No. Correct	Time (sec)	No. Correct	Time (sec)		Diff. No.	Diff. time
1	111	159	112	148		1	-11
2	110	187	111	193		1	6
3	116	167	115	153		-1	-14
4	115	135	116	145		1	10
5	111	141	110	132		-1	-9
6	114	135	114	128		0	-7
7	113	143	112	132		-1	-11
8	111	169	113	172		2	3
9	114	139	114	159		0	20
10	109	167	92	167		-17	0
11	108	144	107	139		-1	-5
12	113	152	110	157		-3	5
13	109	137	112	136		3	-1
14	115	223	114	195		-1	-28
15	106	122	111	115		5	-7
16	112	132	113	125		1	-7
17	116	130	112	141		-4	11
18	111	149	107	178		-4	29
19	116	105	115	128		-1	23
20	110	149	108	165		-2	16
21	116	171	112	140		-4	-31
22	112	128	115	130		3	2
23	114	159	116	150		2	-9
24	110	175	114	150		4	-25
25	116	156	116	148		0	-8
26	115	165	115	141		0	-24
27	113	150	116	139		3	-11
28	116	157	116	187		0	30
29	116	146	115	148		-1	2
				MEAN		-0.52	-1.8
				S.D.		3.92	15.86

DISCUSSION

Salivary melatonin levels

Day time levels

Acute exposure to a pulsed 50 Hz, 100T EMF does not appear to suppress day time melatonin levels. However, the number of subjects was small and many subjects had melatonin levels close to the sensitivity level of the assay. The small size of the sample means it is unlikely an effect could have been detected even if it did exist. (Rearranging the t-test formula and using the mean and standard deviation found in the study, for p=.05 and t=2, a total of 68 subjects would have been required.)

While, as expected, most subjects had very low levels of melatonin, the range was from 0 to 47 pg/ml. A similar range of melatonin levels has been widely reported in the literature (e.g. Graham, 1997, Vakkuri, 1985). As the presence of bright light is a known confounder the subjects were tested in a low light environment (226 lux). At the end of the session some subjects had commented on feeling the effects of the low light levels (e.g. feeling slightly sleepy). It may be that the light levels were low enough to cause an increase in melatonin levels in some subjects. Six subjects had midday levels over 20 pg/ml in saliva which would equate to a blood level of over 60 pg/ml. This is quite high for day time levels. According to Lewy et al.(1980) the change in melatonin levels as a response to changing light intensity is quite rapid, within 30 minutes. Lewy's study reported a drop in melatonin levels within 30 minutes of exposure to bright light and a recovery of levels within 30 minutes of cessation of exposure. This study took 30 minutes plus a 20 minute acclimatisation period pre-experiment. The saliva sample for analysis of melatonin levels was collected at the end of the session. If the higher control and exposure melatonin levels was due to the low light levels then the presence of the EMF didn't suppress this rise. There were only 3 subjects that showed a marked drop in levels for the exposure session.

Night Time Levels

The data suggested that an exposure to a pulsed 50 Hz, 100μT, sinusoidal EMF has no acute effect on night melatonin levels in humans. Using a larger field intensity (100 μT) did not expand the result found by Graham et al (1996) in a sub group exposed to 20 μT, to a wider number of subjects. However, the result was consistent with that reported by other studies investigating acute exposure to

sinusoidal power frequency EMFs (Graham et al., 1996, 1997; Selmaoui et al., 1996). Those studies used 20 μT and 10 μT respectively and longer exposure times. However, the truncation of the study at 29 subjects meant the ability to detect an effect was small and the chance of a type 2 error (assuming no effect when one may have occurred) was high. An analysis of the mean and standard deviation indicated a greater number of subjects would have been required to detect an effect if one existed.

At midnight the range of salivary melatonin was from 0 to 78 pg/ml during the control session and 1 to 85 pg/ml during the exposure session. This range is similar to that reported in the literature. Wood et al. (1998) reported a range at the start of the evening melatonin rise (10 .p.m. $\pm$ 0.2 Hr.) of 1.9-26.9 pg/ml and a peak range of 15.3- 163 pg/ml.

As well as a large inter-subject variation there was also a fairly large intra -subject variation. This was unlikely to be due to assay irregularities as all were assayed in duplicate and all samples relating to a specific subject were assayed in the same batch. The assay was done in a designated RIA lab by a technician who ran all the assays for the department. It is more likely to be due to confounders being present. However, subjects were pre-screened for known conditions that effect melatonin i.e. illness, nightshift work, lack of regular sleep patterns, use of medication and drugs (including alcohol and tobacco). They were also told to restrict alcohol, tea, coffee and other caffeine sources, exercise and exposure to bright light prior to each session. The subjects were not pre-screened for melatonin levels and sensitivity to light as had been done in the Mid West Institute studies (Graham et al., 1996, 1997; Cohen et al., 1992; Cook et al., 1992). This experiment used an unshielded room on the third floor of a university building. Whilst the experiment was the only activity during the night, there may have been extraneous EMFs from electrical looms in the walls or ceilings. During the day there were a large number of computers in adjacent rooms. Whilst ELF EMFs do not propagate away from their source some interference is still possible.

Low excretors

In the current study, the subgroup of individuals with low naturally occurring levels of melatonin did not appear to be effected by exposure to this EMF. However, there were only 8 subjects in the group which was too small for any meaningful conclusions. Discussion of this has been left to experiment two where the subject numbers were greater.

Gender Effects

There were no apparent differences between males and females in response to the the EMF. It must be noted that there were considerably more females than males in this study which could have effected the results. The standard deviation of each group was also very different (appendix 1.2). There were also no significant differences between the control and exposure sessions for females. However, the subject numbers were low with only 21 females and 8 males. Too low to make any conclusions. Gender effects are discussed more fully in experiment two.

In Summary

The results of the current study did not support Stevens's (1987) melatonin hypothesis of a possible link between cancer and exposure to EMFs via a suppression of melatonin production. However, the exposure time was short and did not rule out the possibility of effects from chronic exposure. Also, in the present experiment it is possible that the length of time some subjects took to provide a saliva sample could have affected the results. Some subjects took as long as ten minutes after leaving the EMF to finish providing their saliva sample. This was much longer than the 2-3 minutes in the pre-experimental trials. It was considered this may have effected the results. Also, as the study was truncated at 29 subjects the chance of a type 2 error was high.

Attention

The exposure to a 50 Hz, 100 μT EMF had no observable effect on attention either at midday or at midnight. However, the task in the present study was too easy for the group which resulted in many subjects getting close to full marks. This skewed the analysis. The subject numbers were also low.

The fact that some subjects reported effects from the low light levels in the midday sessions may have effected performance in both this and the memory task. However, the conditions were identical for the control and exposure sessions which were the objects of comparison.

This result in the present study was contrary to the paper of Trimmel and Schweiger (1998) who reported an immediate, significant reduction in attention and memory as a result of exposure to a 1 mT, 50 Hz EMF. They used 66 subjects, half of whom participated in an hour long exposure to an EMF and 45 dB noise with an hour break between the exposure to a control. The details of the memory test used could not be obtained for comparison.

Preece et al. (1998) also noted a significant reduction in attention following exposure to a 0.6 mT, 50 Hz EMF. However, they did not indicate the length of exposure so it may have been longer than in the present study. Also, their task was slightly different in that it required a comparison between a target digit and randomly presented digits. Each occurrence required a discriminatory choice. The subject couldn't just scan a page and pick out the required digit. A subsequent study by Crasson et al. (1999) found no effects on an almost identical task to the one in the present study. They used the same 100 μT field intensity and exposure period of 30 minutes as was used in the present study. Their study was conducted during the afternoon. The main difference was a 15 second on/off pulse compared to the 1 second on/off in the present study. As with the study by Graham et al. 1996 and 1997, in their studies on melatonin, the pulsing of the EMF seems to be important.

Aural Working Memory

The presence of a 50 Hz, 100 μT_{rms} EMF had no significant effect on aural working memory either at midday or at midnight. However, the harder level task (4 letter group) did show a greater difference between control and exposure sessions than the easier tasks. The greater difference between the easy task and harder task may indicate that performance in a task that was harder still may be affected by exposure to an EMF. Alternatively it may be that the number of subjects was too low to detect an effect. Calculations using the mean and standard deviations and a value of t=2, suggest the subject numbers were nowhere near large enough.

Whittington et al. (1996) found a significant difference on a visual forced choice task for the hardest level of task only. They used similar exposure parameters to the present study and an exposure time of only 9 minutes. However, they used 100 subjects. An analysis of the mean and standard deviation in the present study suggests that at p=.05 for t=2, 120 subjects would be required to gain a significant result for the night session and a higher number for the day session.

These results don't support those reported by Preece et al., (1998) who found a significantly decreased ability to maintain digits in working memory. However, the task in the present study was different to that employed by Preece and may have been easier. Memory is very complicated, involving many different brain areas. Different memory tasks use different parts of the brain. Very little research has been published on the cognitive effects of EMFs on humans. A lot more research is needed to identify which areas of memory may be susceptible.

CHAPTER 6
EXPERIMENT TWO
An Investigation into Possible Effects from a Pulsed 50 Hz, 100 μT $_{rms}$ EMF on Salivary Melatonin Levels and the Cognitive Parameters of Attention and Aural working memory: A Follow-up Study

Experiment One had to be truncated when the research space became unavailable. Experiment Two was a follow-up study carried out in 1998/99 using the same experimental parameters and equipment as Experiment One but in a completely new location with a new set of subjects. The opportunity was taken to change some elements of the protocol that had proved unsatisfactory. The changes have been described in the Materials and Methods section.

MATERIALS AND METHOD

Location

The room was in a private residence and was semi-underground. It was maintained at 18 degrees celsius. The location gave greater potential for control over possible extraneous EMF sources than the unshielded room at the University used for experiment one.

Subjects

Subjects were volunteers recruited from amongst the staff and students at Massey University. Each subject attended two day and two night sessions, with the exception of subject 48, who missed both day sessions and subject 24 who missed the control day session. There were a total of 50 subjects (19 males and 31 females) in the night sessions and 48 subjects (19 males and 29 females) in the day sessions. Ages ranged from 18 to 42 years, with a mean age of 21.4 years.

Exposure System

The equipment used was the same as was used for Experiment One (for details refer to experiment one) but the equipment had to be moved to a different location. Orientations and background EMF characteristics were therefore different to Experiment One.

Lighting and Temperature

The room used for Experiment two contained windows but these were blocked out with black polythene resulting in a complete absence of natural light. Light was provided by the same equipment as for Experiment One.

Parameters Tested and Treatment of Data and Samples

As for Experiment One, subjects carried out an attention task and auditory working memory task before providing a saliva sample for the determination of salivary melatonin levels. Data was analysed using the same techniques as for Experiment One.

Experimental Protocol

The following changes were made to the protocol used for Experiment One.

During testing the subjects remained seated with their heads supported by the headrest. Subjects remained seated between the coils whilst providing a saliva sample for the analysis of melatonin levels. Although this was more difficult for the subjects, this new protocol was necessary as some subjects in Experiment One had taken as long as ten minutes to provide the saliva sample after they had left the field. It was considered this delay may have been long enough to effect the results. This also meant the subjects remained fairly still during testing and supplying the saliva sample which was considered important as a change in posture could effect melatonin levels (Arendt, 1995).

An additional control saliva sample was introduced from subject 12 onwards, prior to both the control and exposure sessions. After data collection had commenced an article appeared in the literature which suggested that exposure to an EMF may cause a delay in the evening melatonin rise (Wood et al., 1998). Consequently, from subject 12 onwards an additional saliva sample was obtained prior to the start of each session. The extra sample allowed the observation of the amount of change in melatonin levels between the start and finish of the experiment. A comparison of the size of the evening melatonin rise between the control and exposure sessions could then be made. The additional control sample also allowed observation of the within-subject consistency of the salivary melatonin levels between one day and the next. It provided information on the reliability of using a separate night as a control.

The start of the evening session was changed from 12 a.m. to 11 p.m.. This was done because it had been very difficult to obtain subjects for an experiment starting at midnight. It was recognised that the change moved the experiment an hour further away from the melatonin peak of 2 a.m.-3 a.m. which was less desirable but was necessary if the experiment was to continue. The midday session remained at the same time as for Experiment One.

RESULTS

Melatonin Levels

An extra control, produced prior to each session, was instituted from subject 12 onwards. Thus there were two controls for comparison with the exposure session: a control sample taken on a different day but the same time of day; and the extra control sample taken before each session. Comparisons between the control and exposure sessions were made using a paired t-test.

Day Sessions

Fifteen subjects had reduced levels of melatonin in the exposure session and 18 had higher levels compared to the control sessions. The remaining 15 subjects either showed no change, or had levels that were undetectable. Most subjects had very low levels of salivary melatonin in the day sessions (Table 6.1).

When the control session (control on a different-day) was used in the comparison the difference was not significant (mean difference = 0.6 pg/ml+ SEM = 1.021, t_{47}=0.5876, p>0.05) (Table 6.1). When the same-day control sample was used in the comparison the difference was statistically significant (mean difference= -2.2 + 0.1625 SEM, t_{37}=-3.522, at p<0.001) (Table 6.2).

Night Sessions

Of the 50 subjects in the night session, 19 had lower salivary melatonin levels in the exposure session, compared to the control session and 25 had increased levels. At this time of night it could be expected that melatonin should be rising in most subjects (Table 6.1).

Using the control session from the different night as the comparison, there was no difference detectable from the presence of the EMF (mean difference =-0.4$\pm$ 2.77 SEM, t_{49}=0.144, p>0.05 (Table 6.1). There was also no significant difference between the control and exposure sessions when the control sample was taken prior to the session (mean difference =0.8 pg/ml$\pm$1.184 SEM, t_{38}=0.6755, p>0.05)(Table 6.2)

The evening melatonin rise

The addition of an extra saliva sample prior to the start of each experimental session permitted the comparison of the size of the evening melatonin rise between control and exposure sessions. This extra sample was collected from subject 12 onwards providing a total of 39 subjects (Table 6.3).

On the control night the mean salivary melatonin levels were 15.2 pg/ml at the beginning of the experiment and 16.8 pg/ml at the conclusion, giving a mean rise of 1.6 pg/ml. Under exposure to the EMF the pre-exposure mean was 16.2 pg/ml with the mean at the conclusion of exposure being 17 pg/ml, a rise of 0.8 pg/ml (Table 6.3). The difference in rise between the two sessions was also 0.8 pg/ml which did not reach statistical significance (mean difference=0.8 pg/ml$\pm$2.936 SEM,t_{38}=0.2725, p>0.05)

Subgroup of subjects with naturally low levels of melatonin production

Day Sessions

There were 12 subjects (9 male, 11 female) in this category. The difference between the control and exposure sessions was not significant (mean difference = 2.0 pg/ml +SEM = 1.524, t_{19}=1.345, p>0.05) for this subgroup on analysis by a paired t-test. (table 6.1 and appendix 2).

Night Sessions

There were 21 subjects (9 male, 12 female) with pre-exposure levels of melatonin below 10 pg/ml. Differences between control and exposure sessions in the low excretor group did not reach statistical significance. (mean difference=1.3 $\pm$4.474, t_{20}=1.3170 p>0.05)(Table 6.1 and appendix 2)

Gender Effects

The possibility of one gender being more susceptible than the other, to the influence of an EMF was investigated but no differences were found (appendix 2). There were 31 females in the night session and no significant field effects were found between exposure and control sessions using a paired t-test (-0.032 $\pm$ 3.719 pg/ml, mean $\pm$SEM, t_{30}= 0.0884, at p>0.05). An unpaired t-test, comparing 19 males and 31 females, failed to find a gender difference in response to the EMF (t_{49}= -0.1736, at p>0.05) (Appendix 2).

Table 6.1: Expt. Two Effects of Power Frequency Fields on Salivary Melatonin

(pg/ml) (Control Session was on a different day at the same time)

		Day			Night	
Subject	Control	Exposure	Difference	Control	Exposure	Difference
1	4	1	-3	30	31	1
2	6	2	-4	112	157	45
3	9	1	-8	41	12	-29
4	2	16	14	53	11	-42
5	8	17	9	31	31	0
*6	0	0	0	1	1	0
7	5	10	5	17	9	-8
8	10	14	4	51	50	-1
9	12	2	-10	29	33	4
*10	15	40	25	8	19	11
*11	3	1	-2	2	11	9
12	3	2	-1	24	28	4
*13	0	0	0	3	4	1
14	34	9	-25	131	130	-1
15	3	13	10	16	52	36
16	2	2	0	13	2	-11
17	0	0	0	20	23	3
18	0	0	0	36	32	-4
19	5	10	5	12	13	1
20	0	0	0	99	16	-83
21	0	2	2	60	43	-17
*22	1	0	-1	6	3	-3
23	0	0	0	14	29	15
24	-	-		0	8	8
25	0	3	3	17	49	32
*26	0	2	2	2	0	-2
*27	2	5	3	8	16	8
*28	0	2	2	3	3	0
29	0	0	0	0	0	0
30	0	0	0	0	0	0
*31	0	0	0	9	13	4
*32	0	0	0	2	0	-2
33	2	0	-2	15	25	10
34	2	1	-1	22	49	27
*35	2	13	11	3	4	1
*36	0	4	4	8	5	-3
*37	0	5	5	2	5	3
*38	2	5	3	5	3	-2
39	3	1	-2	26	23	-3
*40	2	4	2	8	14	6

Table 6.1 continued

Subject	Control	Day Exposure	Difference		Control	Night Exposure	Difference
41	3	0	-3		0	4	4
*42	0	5	5		4	0	-4
43	0	0	0		16	26	10
*44	0	0	0		6	10	4
*45	11	3	-8		2	3	1
*46	0	0	0		5	0	-5
47	0	0	0		0	0	0
*48	-	-			4	1	-3
*49	10	0	-10		2	5	3
50	5	1	-4		55	7	-48
Mean	3.5	4.1	0.6		20.7	20.3	-0.4
SD	5.9	7.1	7.0		28.7	29.7	19.4
N	48				50		
SEM			0.990				2.744
t=			0.6061				0.1458
* low excretors-10 pg/ml or below in night control sessions (N=21)							
mean	2.0				1.3		
s.d.	6.985				4.474		
SEM	1.524				0.976		
N=	20				21		
t=	1.3450				1.3170		

Table 6.2: Expt. Two: Effects of Power Frequency EMFs on Salivary Melatonin, same day control

(pg/ml)		Day			Night	
Subject	Control	Exposure	Difference	Control	Exposure	Difference
12	5	2	-3	20	28	8
13	3	0	-3	6	4	-2
14	16	9	-7	138	130	-8
15	21	13	-8	68	52	-16
16	4	2	-2	1	2	1
17	8	0	-8	22	23	1
18	2	0	-2	16	32	16
19	20	10	-10	14	13	-1
20	0	0	0	19	16	-3
21	11	2	-9	50	43	-7
22	1	0	-1	0	3	3
23	2	0	-2	24	29	5
24	2	0	-2	3	8	5
25	4	3	-1	60	49	-11
26	0	2	2	6	0	-6
27	4	2	-2	22	30	8
28	6	2	-4	5	3	-2
29	2	0	-2	0	1	1
30	0	0	0	1	0	-1
31	0	0	0	12	13	1
32	1	0	-1	0	0	0
33	1	0	-1	31	25	-6
34	2	3	1	32	49	17
35	21	13	-8	0	4	4
36	0	4	4	1	5	4
37	5	5	0	0	5	5
38	7	5	-2	3	3	0
39	7	1	-6	20	23	3
40	3	4	1	3	14	11
41	2	0	-2	3	4	1
42	0	5	5	0	0	0
43	0	0	0	9	26	17
44	0	0	0	10	10	0
45	1	3	2	3	3	0
46	0	0	0	1	0	-1
47	0	0	0	0	0	0
48	-	-		1	1	0
49	11	0	-11	5	5	0
50	1	1	0	23	7	-16
Mean	4.6	2.4	-2.2	16.2	17.0	0.8
SD	6.008	3.530	3.760	26.003	24.094	7.302
N=37			SEM=0.618	N=38		SEM=1.184
			t=-3.5459			t=0.6754

Table 6.3 Effects of a 50 Hz EMF on the Salivary Melatonin Rise (pg/ml)								
	Control Evening				Exposure Evening			
Subject	Before	After	Differ.		Before	After	Differ.	C-E
12	32	24	-8		20	28	8	-16
13	5	3	-2		6	4	-2	0
14	148	130	-18		138	130	-8	-10
15	7	16	9		68	52	-16	25
16	55	12	-43		1	2	1	-44
17	14	20	6		22	23	1	5
18	17	36	19		16	32	16	3
19	12	12	0		14	13	-1	1
20	13	99	86		19	16	-3	89
21	64	60	-4		50	43	-7	3
22	0	6	6		0	3	3	3
23	6	14	8		24	29	5	3
24	0	0	0		3	8	5	-5
25	10	17	7		60	49	-11	18
26	3	2	-1		6	0	-6	5
27	4	8	4		22	30	8	-4
28	5	3	-2		4	3	-1	-1
29	0	0	0		0	1	1	-1
30	1	0	-1		1	0	-1	0
31	0	9	9		12	13	1	8
32	0	2	2		0	0	0	2
33	23	16	-7		31	25	-6	-1
34	20	22	2		32	49	17	-15
35	1	3	2		0	4	4	-2
36	7	8	1		1	5	4	-3
37	3	2	-1		0	5	5	-6
38	10	5	-5		3	3	0	-5
39	36	26	-10		20	23	3	-13
40	11	8	-3		3	14	11	-14
41	3	0	-3		3	4	1	-4
42	0	0	0		0	0	0	0
43	12	16	4		9	26	17	-13
44	4	6	2		10	10	0	2
45	0	3	3		3	3	0	3
46	4	5	1		1	0	-1	2
47	0	0	0		0	0	0	0
48	5	4	-1		1	1	0	-1
49	2	2	0		5	5	0	0
50	55	55	0		23	7	-16	16
Mean	15.2	16.8	1.6		16.2	17.0	0.8	0.8
SD	27.1	27.0	16.6		26.0	24.1	7.3	18.1
N	38				38			
SEM			2.692				1.000	2.600

Assay of Melatonin salivary samples

With 50 subjects, each providing 8 samples, it was not possible to assay all samples in the same batch. The assay sensitivities for each batch are listed in Table 6.4.

Table 6.4 Radio Immuno Assay sensitivities

Subject	Sensitivity (pg/ml)
1-17	4.4
18-33	2.1
34-40	6.5
41-50	5.5
Mean Sensitivity	4.6

COMBINED RESULTS FROM EXPERIMENTS ONE AND TWO

As experiments one and two used the same equipment and protocol it was decided to analyse the combined results to investigate whether the increase in subject numbers would change the outcome. It is acknowledged that there were some experimental differences. For the melatonin comparison the control used for experiment 2, was the control sample taken on the different day.

Combining the result from experiment one and two made no difference to the outcome of the research. There were no significant differences between control and exposure levels of melatonin for either the day or night samples (Appendix 2).

Day= -1.0 $\pm$ 1.25 (mean $\pm$ SEM),t_{76}= -1.25, at p>0.05

Night= -0.7 $\pm$ 1.889 (mean $\pm$ SEM),t_{78}= -0.37, at p>0.05

There were also no differences in the 'low excretor' subgroup (Appendix 2).

Day=-1.06$\pm$2.318 (mean $\pm$ SEM),t_{28}= -0.6900, at p>0.05

Night= -0.3 $\pm$1.86 (mean $\pm$ SEM),t_{29}= 0.1612, at p>0.05

Gender differences (Appendix 2)

There were no significant effects on melatonin levels in females and results for males and females were similar. Results given are for the night session.

Females= 1.12 $\pm$ 2.3629 (mean $\pm$SEM), t_{51}= -0.4739, at p>0.05

Males =-1.81$\pm$ 3.1846 (mean $\pm$SEM), t_{26}= -0.5683, at p>0.05

Attention

As with experiment one the 'number correct' figures were clearly skewed with a definite maximum. A logit transformation was carried out using a logit analysis of the number correct. Differences between the control and exposure conditions were not significant for the night sessions ($p>0.05$)(Table 6.6). The day session results were different with a marginal effect which would have been significant at $p=0.10$ but not at 0.05. When exposed to the field subjects tended to complete the task slightly quicker but got slightly fewer correct (Table 6.5).

It was not considered appropriate to analyse combined results from experiments one and two as this would be unlikely to add information given the results had a definite maximum.

Table 6.5: Expt. Two Effects of Power Frequency Fields on Attention. Day

score out of 116	Control		Exposure			
Subject	No. Correct	Time (sec)	No. Correct	Time (sec)	DIFF.NO.	DIFF.TIME
1	116	134	114	135	2	-1
2	114	206	114	205	0	1
3	114	228	108	240	6	-12
4	116	156	115	140	1	16
5	116	143	114	135	2	8
6	114	188	103	172	11	16
7	116	250	116	175	0	75
8	116	133	114	189	2	-56
9	115	142	113	134	2	8
10	112	172	112	147	0	25
11	113	178	116	162	-3	16
12	115	190	114	172	1	18
13	112	160	114	157	-2	3
14	111	182	112	167	-1	15
15	115	127	115	137	0	-10
16	116	129	114	233	2	-104
17	114	178	115	157	-1	21
18	112	183	115	157	-3	26
19	100	150	105	136	-5	14
20	113	130	112	130	1	0
21	114	137	116	135	-2	2
22	113	200	114	184	-1	16
23	111	187	112	177	-1	10
24	-	-	-	-	-	-
25	116	141	114	142	2	-1
26	116	193	110	186	6	7
27	116	205	116	211	0	-6
28	105	121	109	131	-4	-10
29	116	174	116	167	0	7
30	116	190	116	164	0	26
31	114	126	114	134	0	-8
32	115	136	110	152	5	-16
33	113	148	116	125	-3	23
34	110	165	111	185	-1	-20
35	114	109	113	112	1	-3
36	113	175	106	185	7	-10
37	113	159	114	208	-1	-49
38	116	134	114	165	2	-31
39	116	142	115	155	1	-13
40	114	136	116	151	-2	-15
41	114	154	112	201	2	-47
42	114	137	113	139	1	-2
43	113	183	109	144	4	39

44	115	162	115	144		0	18
45	115	177	115	161		0	16
46	115	193	115	177		0	16
47	113	179	113	161		0	18
48	-	-	-	-		-	-
49	111	185	110	171		1	14
50	114	162	107	158		7	4
N	48						
mean	113.6	163.9	112.8	162.6		0.8	1.3
s.d.						3.008	27.141

Table 6.6: Expt. Two Effects of Power Frequency Fields on Attention.							Night
score out of 116		Control	Exposure				
Subject	No. Correct	Time (sec)	No. Correct	Time (sec)	DIFF.NO.	DIFF.TIME	
1	114	140	104	130	-10	-10	
2	114	223	115	302	1	79	
3	107	225	107	255	0	30	
4	114	163	114	150	0	-13	
5	116	138	113	139	-3	1	
6	112	183	104	173	-8	-10	
7	114	158	111	165	-3	7	
8	115	143	113	140	-2	-3	
9	109	133	116	147	7	14	
10	111	156	108	151	-3	-5	
11	114	186	112	175	-2	-11	
12	114	183	112	187	-2	4	
13	115	146	112	173	-3	27	
14	109	172	111	173	2	1	
15	113	153	114	128	1	-25	
16	115	167	115	144	0	-23	
17	116	152	116	164	0	12	
18	116	148	113	161	-3	13	
19	110	140	97	136	-13	-4	
20	114	133	114	130	0	-3	
21	116	150	115	155	-1	5	
22	115	190	110	187	-5	-3	
23	113	185	115	186	2	1	
24	116	144	113	169	-3	25	
25	113	147	112	188	-1	41	
26	114	206	114	145	0	-61	
27	113	207	112	126	-1	-81	
28	109	137	111	144	2	7	
29	116	192	116	160	0	-32	
30	116	168	114	164	-2	-4	
31	113	124	114	114	1	-10	
32	113	132	114	140	1	8	
33	115	134	111	115	-4	-19	
34	110	160	112	158	2	-2	
35	116	130	115	112	-1	-18	
36	110	187	114	183	4	-4	
37	112	174	115	168	3	-6	
38	115	158	114	137	-1	-21	
39	113	158	114	141	1	-17	
40	114	158	116	144	2	-14	
41	110	145	113	173	3	28	
42	111	154	114	143	3	-11	
43	110	154	111	135	1	-19	

44	115	159	115	134	0	-25	
45	111	146	114	167	3	21	
46	116	181	116	194	0	13	
47	112	145	114	139	2	-6	
48	114	145	112	139	-2	-6	
49	110	149	109	167	-1	18	
50	115	173	113	187	-2	14	
mean	113.2	160.7	112.5	158.7	-0.7	-1.9	
N	50		50				
s.d.					3.394	24.158	

Aural Working Memory

Tasks were organised into 3 levels of difficulty, the two letter group being the easiest and the four letter group the hardest. The mean score for the 2 letter group was 95%, the three letter group, 77%, and the four letter group, 64 %.

Day Sessions

In an analysis using a paired t-test there was no significant difference between the control and exposure sessions. All levels of difficulty produced null results. However, the hardest level of difficulty (4 letter group) showed the greatest difference between control and exposure sessions. (Tables 6.7-6.9).

2 letter group= - 0.10±0.175 (mean difference ±SEM), t_{47}=-0.5666, at p>0.05)
3 letter group=- 0.15±0.253 (mean difference ±SEM), t_{47}=0.5928, at p>0.05)
4 letter group=- 0.35±0.330 (mean difference ±SEM),t_{47}=-1.0601, at p>0.05)

Night Sessions

In the night sessions there was a small effect from the EMF exposure but it did not reach statistical significance for any level of difficulty. The details of the results from the paired t-tests relating to each level of difficulty are set out below. As with the day sessions, the hardest level of difficulty (4 letter group) showed the greatest difference between exposure and control sessions (Tables 6.7-6.9).

2 letter group = -0.32 ±0.187 (mean difference+SEM), t_{49}=-1.7112 at p>0.05)
3 letter group= - 0.08±0.243 (mean difference ±SEM), t_{49}=0.3295, at p>0.05)
4 letter group= - 0.44±0.301 (mean difference ±SEM), t_{49}=-1.4618, at p>0.05)

COMBINED RESULTS FROM EXPERIMENTS ONE AND TWO

For the 4 letter group results still did not reach significance when experiments one and two were combined to give a larger sample.

Day= -0.14 ±0.278 (mean ± SEM),t_{76}= -0.5035, at p>0.05
Night= -0.05 ±0.297 (mean ± SEM),t_{78}= -0.1683, at p>0.05

Table 6.7: Experiment Two. Effects of Power Frequency Fields on Aural Memory

Two Letter Groups

(score out of 22)

		Day			Night	
Subject	Control	Exposure	Difference	Control	Exposure	Difference
1	21	20	-1	19	17	-2
2	19	21	2	21	17	-4
3	17	20	3	19	15	-4
4	22	22	0	22	22	0
5	22	22	0	21	22	1
6	20	18	-2	22	18	-4
7	22	21	-1	22	22	0
8	22	22	0	22	22	0
9	22	22	0	20	22	2
10	19	22	3	18	19	1
11	22	22	0	22	21	-1
12	22	21	-1	22	21	-1
13	20	20	0	21	19	-2
14	22	22	0	22	22	0
15	22	19	-3	20	20	0
16	20	20	0	20	20	0
17	22	22	0	22	22	0
18	21	21	0	21	22	1
19	17	17	0	18	16	-2
20	22	21	-1	21	22	1
21	21	22	1	21	22	1
22	22	21	-1	20	22	2
23	22	21	-1	22	21	-1
24	-	-		22	21	-1
25	22	21	-1	22	22	0
26	20	20	0	21	21	0
27	22	22	0	22	22	0
28	22	21	-1	22	22	0
29	22	22	0	22	21	-1
30	22	22	0	22	22	0
31	22	22	0	22	22	0
32	21	22	1	21	21	0
33	21	22	1	21	22	1
34	22	21	-1	22	22	0
35	21	20	-1	22	22	0
36	22	21	-1	22	22	0
37	22	21	-1	19	20	1
38	22	22	0	22	21	-1
39	22	22	0	22	22	0
40	21	22	1	22	22	0
41	22	22	0	22	22	0

42	22	21	-1		21	21	0
43	22	22	0		22	22	0
44	22	20	-2		22	22	0
45	21	22	1		22	22	0
46	20	22	2		22	19	-3
47	20	22	2		22	22	0
48	-	-			22	22	0
49	22	20	-2		21	21	0
50	20	20	0		20	20	0
mean	21.2	21.1	-0.1		21.2	20.9	-0.3
s.d.	1.25	1.13	1.21		1.12	1.72	1.32
N	48				50		
SEM			0.175				0.187

Table 6.8: Experiment Two. Effects of Power Frequency Fields on Aural Memory

Three Letter Groups

(score out 0f 22)

		Day			Night	
Subject	Control	Exposure	Difference	Control	Exposure	Difference
1	13	15	2	16	14	-2
2	14	11	-3	12	11	-1
3	12	16	4	15	9	-6
4	18	18	0	18	18	0
5	15	17	2	16	16	0
6	13	16	3	13	13	0
7	14	16	2	16	16	0
8	16	15	-1	14	15	1
9	13	16	3	13	16	3
10	16	14	-2	13	16	3
11	14	17	3	16	16	0
12	15	15	0	16	16	0
13	15	13	-2	16	14	-2
14	17	18	1	18	17	-1
15	17	17	0	16	17	1
16	13	16	3	16	14	-2
17	17	16	-1	14	15	1
18	18	17	-1	18	18	0
19	12	12	0	13	10	-3
20	16	17	1	15	15	0
21	16	17	1	14	15	1
22	18	15	-3	17	16	-1
23	15	16	1	16	14	-2
24	-	-		16	16	0
25	18	17	-1	17	17	0
26	13	13	0	15	13	-2
27	15	15	0	15	15	0
28	15	15	0	14	15	1
29	16	16	0	15	15	0
30	18	18	0	16	18	2
31	17	17	0	18	18	0
32	17	16	-1	16	17	1
33	15	16	1	14	15	1
34	18	14	-4	16	16	0
35	17	15	-2	17	18	1
36	17	17	0	16	17	1
37	16	13	-3	16	14	-2
38	16	16	0	17	17	0
39	17	18	1	17	17	0
40	15	13	-2	15	18	3
41	16	16	0	16	17	1

42	15	17	2		14	13	-1
43	16	16	0		16	15	-1
44	16	17	1		15	17	2
45	16	16	0		14	12	-2
46	16	17	1		17	15	-2
47	16	18	2		18	17	-1
48	-	-			16	18	2
49	15	15	0		14	17	3
50	14	13	-1		14	13	-1
mean	15.6	15.7	0.1		15.5	15.4	-0.1
s.d.	1.64	1.66	1.75		1.49	2.09	1.70
N	48				50		
SEM			0.253				0.240

Table 6.9: Experiment Two. Effects of Power Frequency Fields on Aural Memory

Four Letter Groups

(score out of 22)

		Day			Night	
Subject	Control	Experiment	Difference	Control	Experiment	Difference
1	8	12	4	13	13	0
2	15	10	-5	7	12	5
3	12	11	-1	10	8	-2
4	15	17	2	14	17	3
5	14	12	-2	14	13	-1
6	13	12	-1	12	16	4
7	14	15	1	14	13	-1
8	16	9	-7	13	14	1
9	10	13	3	10	12	2
10	11	10	-1	13	12	-1
11	14	14	0	15	14	-1
12	15	15	0	15	15	0
13	15	15	0	14	16	2
14	15	15	0	19	15	-4
15	15	16	1	15	17	2
16	13	14	1	12	10	-2
17	17	15	-2	16	14	-2
18	15	15	0	11	12	1
19	12	8	-4	9	14	5
20	13	15	2	12	14	2
21	16	14	-2	14	14	0
22	15	14	-1	15	14	-1
23	11	17	6	13	13	0
24	-	-		13	16	3
25	16	16	0	17	15	-2
26	11	15	4	15	14	-1
27	17	16	-1	14	10	-4
28	14	12	-2	13	14	1
29	14	16	2	15	16	1
30	17	16	-1	17	17	0
31	16	17	1	18	17	-1
32	13	14	1	13	14	1
33	16	15	-1	17	15	-2
34	17	15	-2	16	17	1
35	18	15	-3	15	19	4
36	17	15	-2	17	18	1
37	13	14	1	12	14	2
38	16	16	0	14	15	1
39	16	17	1	17	18	1
40	15	13	-2	17	17	0
41	15	17	2	18	16	-2

42	13	14	1		9	11	2
43	13	12	-1		12	15	3
44	16	15	-1		16	16	0
45	14	13	-1		13	14	1
46	16	15	-1		17	16	-1
47	15	15	0		15	16	1
48	-	-			17	13	-4
49	13	12	-1		9	11	2
50	16	11	-5		10	12	2
mean	14.4	14.0	-0.4		13.9	14.4	0.4
s.d.	2.06	2.17	2.35		2.70	2.27	2.13
N	48				50		
SEM			0.330				0.301

DISCUSSION

Salivary Melatonin levels

Daytime Levels

The range in salivary melatonin for the control session was from 0 to 34 pg/ml. The mean was 3.5 pg/ml and standard deviation, 5.9 pg/ml. This is similar to levels reported in the literature. Radon et al (2001) reported a mean of about 13 pg/ml with a standard deviation of about 10 pg/ml for their control sessions. Waldhauser and Dietzel (1986) reported levels of about 10 pg/ml during the day.

Exposure to a 50 Hz, 100 μT_{rms} EMF had a significant suppression effect on midday salivary melatonin levels. However, this only occurred when the comparison was made using the control sample was obtained just before the exposure session. No effect was found when the exposure session was compared to the separate day control. The significant result could have been an order effect but, as levels are uniformly low during the day, there was no reason why an order effect should occur. Analysis of the exposure data using a separate day as a control did not produce a significant effect so it is unlikely to be due to carry over from the night exposure session in those subjects who had an exposure 12 hours prior to the day session. The positive finding may indicate a real field effect or, more likely, may be an aberration due to the fact that many subjects were producing results which were close to the sensitivity level of the radioimmunoassay. The fact that about half the subjects had an increase in salivary melatonin levels when exposed to the field and half showed a decline would suggest this is not a real trend. A study published after the present study was carried out (Crasson , 2001) failed to find a significant effect from an exposure of 50 Hz, 100μT applied during the afternoon. Although that study used an intermittent or pulsed (15 sec. on/off) EMF.

Night Time Levels

At night the range was from 0 to 131 pg/m with mean of 20.7 pg/ml and a standard deviation of 28.7 pg/ml during the control session and a range of 0 to 157 pg/ml with a mean and standard deviation of 20.3 and 29.7 pg/ml respectively during the exposure session. This range is similar to that reported in the literature. Radon et al. 2001 reported a mean of 21 pg/ml (s.d.= 12 pg/ml) at 1100 hours and

28 pg/ml (s.d. =15 pg/ml) at midnight during their control session. Wood et al. (1998) reported a range at the start of the evening melatonin rise (10 .p.m. $\pm$ 0.2 Hr.) of 1.9-26.9 pg/ml and a peak range of 15.3- 163 pg/ml.

Effect on the evening rise

The current study could find no significant effect on the evening melatonin rise from an evening exposure to a 50 Hz, 100μT_{rms} EMF. As half the subjects had a daytime exposure before the evening session this result was also contrary to Karasek (1998) who previously reported a significant effect on the evening melatonin rise following daytime exposure at 2.9 mT. However, there was a difference in field intensity between the two studies.

At night, when melatonin levels are naturally rising it was expected that the exposure sample taken 30 minutes after the control would be significantly higher. The fact that there was no significant difference could indicate a blunting or delay of the rise. However, the difference in amount of the rise suggested this was not the case. As many subjects had very low levels of melatonin, it is likely that the timing of the experiment was too early to pick up the rise in many subjects. Waldhauser and Dietzel (1985) had described the rise as typically occurring between 9 p.m. and 11 p.m..

Wood et al, (1998) and Karasek et al. (1998) looked at the effect of daytime exposure on the evening melatonin rise. Both concluded a daytime exposure significantly delayed the evening melatonin rise. Wood et al. used both square and sine waves at 200 μT , 50 Hz and concluded the effect was greater with a square wave. Karasek et al. used 2.9 mT, 40 Hz square wave, 20 minutes per day, 5 days per week for 3 weeks. Although the field intensity in the present study was different to Wood et al.'s study it raises the question as to whether there were effects on the night sessions from exposure 12 hours previously. In the present studies 50 % of the subjects would have had a night exposure session following a daytime exposure. The others would have been sham exposed. In looking at the data in the light of the exposure order it appears this did not occur. Half the subjects in the present study who had a daytime exposure will have had this followed by a night time exposure and still no effect was found on melatonin levels.

Intra-individual variability in melatonin levels

It was noted there was a difference, in some subjects, between melatonin levels in the pre-session saliva samples between one night and the next for the same subject (Tables 6.1 & 6.2, Appendix 2). This difference was generally not great and most subjects had fairly stable melatonin levels between one day and the next. A few subjects had levels that were markedly different between the two day or night control sessions. This could be due to a delayed response to the EMF but also could indicate that profiles are not as stable from one day to the next as they reported in the literature. If so this would reduce the reliability of using a separate night as a control in those subjects. Graham et al. (1996, 1997) had pre-tested the profiles of subjects and removed any considered 'atypical'. Consequently, it appears that some subjects have profiles that are not considered 'normal'. However, an inconsistency between one control and the next at the same time of day could also indicate that some of the subjects had not followed the pre-session advice with regards to limitation of exposure to confounding variables. The settling in time in a low light environment before each session should have been enough to counteract any light effects prior to arrival at the testing room but there may have been other confounders present but not admitted to by subjects. This is always a difficulty when working with human subjects.

The subjects were not pre-screened for melatonin levels and sensitivity to light as had been done in the Mid West Institute studies (Graham et al.; Cohen et al.). Consequently, some subjects who had no recordable levels of melatonin were included. A lack of detectable melatonin does not necessarily mean they didn't produce any but may metabolise it faster than the other subjects. de Seze et al. (1999) in a study of 38 men had one subject with a flat melatonin profile so the occurrence is not that unusual. They excluded the person from the study as they considered the profile abnormal. In the present study, there were insufficient funds to explore the melatonin profiles of subjects before the experiment was carried out . Although it may have been better to remove those subjects with zero levels of melatonin from the analysis it would not have altered the result. It is debatable whether subjects with 'abnormal' profiles should be excluded from experiments as they may form a susceptible sub population. However, if included, subject numbers would have to be large enough to be able to distinguish a field effect from normal variation in melatonin levels. This would add considerably to the cost

of studies, especially where highly controlled expensive facilities such as those at the Mid West Institute are used.

Low excretors

In the current study, the subgroup of individuals with low naturally occurring levels of melatonin did not appear to be affected by exposure to this EMF. This result was contrary to that reported by Graham et al. (1996) who found that those with a natural melatonin level of less than 60 pg/ml peak levels responded to the EMF with reduced melatonin levels. However,the field parameters were different. The present study used 100 μT, with a 1 second on/off pulse. Subjects were not supine and the exposure time was only 30 minutes per session. A mix of males and females were used. Exposure in the study by Graham et al. was overnight. The field in Graham et al.'s (1996) study was alternated one hour on/off and pulsed 15 seconds on/off during the on phase. The follow-up study by Graham et al. (1997), failed to replicate their previous results. However, they had changed the protocol to a continuous EMF. A much later study by Crasson et al. (2001) used the same intensity as the present study (100 μT) but pulsed it 15 seconds on/off. They also used a 30 minute exposure but it was during the day. Urine was tested for a melatonin metabolite which provides a more sensitive measure than saliva as it is collected over a longer time period. They found a reduction in melatonin levels, but only in the 'low excretor' group.

Gender Effects

There were no apparent differences between males and females in response to to the EMF. There were also no significant differences between the control and exposure sessions for females. The females were not effected by exposure to the EMF nor were there apparent changes in melatonin levels due to the menstrual cycle. It may be concluded that the habit of confining subjects to males only may not be justified. Had a difference been found it would have then been necessary to conduct further research to separate field effects from changes due to the menstrual cycle.

Summary

It was concluded that a 50 Hz EMF at an intensity of 100 μT_{rms} did not cause a significant suppression of melatonin. The current study adds information about a

flux density not used in other studies but one to which the public may be chronically exposed. This result does not support the Steven's (1987) hypothesis. These results were consistent with those of experiment one. The few laboratory studies published on the effects of power frequency EMFs on humans have mostly reported null results (Selmaoui et al, 1996; Crasson et al., 2001; Graham et al., 1997, 2000). However, more research will be required before any conclusive statements could be made. Graham (1996) did identify a possible subgroup of 'low excretors' who responded to EMF exposure with a suppression of melatonin levels. Results from the current study did not support his finding. Graham et al. (1997, 2000) were unable to replicate their earlier result in later studies. There were no differences in response for female subjects, indicating the practise of excluding females from being subjects may not be justified.

Attention

There was no significant effect from exposure to the EMF during the night but during the day a slight, but non significant effect, was noted. As this effect was marginal and did not occur in Experiment One it may be a chance finding, however, subject numbers were larger in Experiment 2.

This result was contrary to that of Trimmel and Schweiger (1998) who reported a significant reduction in attention and memory as a result of exposure to a 1 mT, 50 Hz EMF. They used 66 subjects, half of whom participated in a hour long exposure to an EMF and 45 dB noise with an hour break between the exposure to a control. The details of the memory test used could not be obtained for comparison.

Preece et al (1998) also noted a significant reduction in a similar attention task However, their task was different in that it required a comparison between a target digit and randomly presented digits. Each individual occurrence required a discriminatory choice. The subject couldn't just scan a page and pick out the required digit. following exposure to a 50 Hz EMF. By contrast, the task in the present study allowed the subject to use a visual search technique. Easy visual search, such as the location of a number or word on a page, is comprised of a "global attentional allocation to the whole array (useful to do the rapid search task) followed by a narrowing of attention to the target (Nakayama and Joseph, 1998, p.291) Preece also did not indicate the length of exposure so it may have been

longer than in the present study.

Subsequent research by Crasson et al. (1999) found no effect on a very similar attention task to the present study but did report a slight influence on reaction time under conditions of sustained attention. They used a 30 minute daytime exposure to a 50 Hz, 100μT EMF, pulsed 15 seconds on/off.

A better protocol for the test in the current study would have been to provide a certain time to cross out the 6s but allow insufficient time for anyone to finish. Then only the number correct would have been the factors tested. The current design, which allowed all to finish, with time and accuracy both being variables may not have been discriminating enough.

The exposure time was short (5 minutes) as this was the first task. This was probably too short an exposure and the task should probably have been done after the memory task. However, Whittington et al. (1996) had found an effect on reaction time with an exposure of 9 minutes using a similar field parameters.

Some subjects reported effects(e.g. feeling sleepy) at midday from the low light levels and this may have effected results in both this and the memory task. However, it was the daytime where the greater effect was noted.

Auditory working memory

As with experiment one, the presence of a 50 Hz, 100 μT_{rms} EMF had no significant effect on working memory. However, the harder level task (4 letter group) did show a greater difference between control and exposure sessions than the easier tasks. The mean score for the 2-letter task was 20.7 correct whereas it was only 13.5 correct for the 4-letter task. Since the task had a 'yes' or 'no' format, a score of 11 should have been possible by chance alone. A mean of 13.5 indicated the task was difficult but may not have been quite difficult enough.

According to calculations in Experiment Two (using the mean and standard deviation and a t=2 value) it would have required a subject number of 138 to detect an effect in the daytime and 113 at night to detect an effect. Using the data from the combined results the subject pool would have been large enough to detect an

effect during the daytime but not for the night session when 106 subject would have been required to detect an effect should one have been present.

The results of the present study don't support those reported by Preece et al. (1998) who found a significantly decreased ability to maintain digits in working memory. However, the task in the present study was different to that employed by Preece. Preece's task involved numbers whereas the present study used different lengths of tone. Preece also used a 60 μT EMF which was lower than the 100 μT in this study. Podd et al. (2002) found a delayed effect on working memory. However, the task was different. Different memory tasks use different parts of the brain.

Very little research has been published on the cognitive effects of EMFs on humans. A lot more research is needed in this area, especially on attention and memory. Replications of previous studies reporting effects are needed.

CHAPTER SEVEN
EXPERIMENT THREE
An Investigation into Possible Effects from Exposure to a Cellular Phone On Heart Rate, Blood Pressure, Aural Temperature, Salivary Melatonin Concentrations and the Cognitive Parameters of Attention and Memory

There is a growing public concern as to whether the use of cellular phones is harmful to health. Anecdotal reports have linked phone use to brain cancers, such as acoustic neuromas, and to subjective complaints such as headaches, tingling, fatigue and problems with concentration and memory. Cellular phones transmit at an ultra high frequency in the 900 MHz band or 1800 MHz band, and as most users hold the phone next to the head, the antenna transmits an EMF which passes through the skull.

Research is just beginning to be published on the effects of exposure to EMFs in the cellular telephone frequency range. The IEGMP (International Expert Group on Mobile Phones(2000) has identified the following areas of research need; effects on brain function; consequences of exposure to pulsed signals; psychological and sociological studies; possible health effects of cellular and subcellular changes; possible mechanisms of interaction with living tissue; epidemiological and human volunteer studies. The group particularly identified a need for laboratory studies on humans as they noted that there are considerable anatomical and circadian pattern differences between animals and humans negating the effective use of animals as models. They further recommend the use of "realistic exposure conditions relevant to mobile phone technology" (IEGMP, 2000, p.8). The WHO specifically suggested the need to determine if low level RF exposure causes changes in melatonin synthesis (Repacholi, 1998). If melatonin was effected it could influence the immune system by inducing the release of cytokines from lymphocytes (Maestroni, 1993).

Cell phones heat up in use. Although it is difficult to measure depth of heat penetration in humans, even a small, localised increase in temperature has the potential to effect brain function. When the present study was carried out, anecdotal reports had been published suggesting heating effects from cellular phone use, but studies published quantifying these effects had been confined to

those using models of the human head. Subsequent to this study commencing , Paredi et al. (2001) reported a significant increase in skin temperature (2 .3 °C) over the nose and parietal areas, from exposure to a GSM phone. They found no difference on the non-phone side. Koivisto et al. (2001) had failed to find a difference in subjective symptoms of skin warmth and redness but their study used a phone with the loudspeaker removed and the discontinuous transmission deactivated. A leather case around the phone kept the phone out of contact with the skin.

A finding of a significant decrease in heart rate from a 60 Hz, 9 kV/m, 20μT field was reported by Cook et al. (1992). Cook et al. found the greatest effects were gained immediately after the field was switched on or off. Sastre and colleagues (1998) found a significant decrease in heart rate variability using an intermittent 20 μT, 60 Hz EMF in 77 men exposed overnight, but no effect was found when a continuous field was used. Little has been published on the effects of cellular phone use on blood pressure. A recent study by Braune et al. (1998) had noted a brief increase in blood pressure following a 35 minute exposure to a GSM phone. A follow up study in 2002, using a larger number of subjects and a longer exposure time (50 minutes), did not confirm the result. Huber et al. (2003) found a reduction in heart rate following a 30 minute daytime exposure, but not an 8 hour overnight exposure. It may be that a longer exposure period allows time for some sort of compensatory mechanism to be activated.

As melatonin is a free radical scavenger, an EMF-induced suppression of melatonin levels could, theoretically, affect a person's susceptibility to cancer and hence provide a possible link between cell phone use and cancer. Most studies using 50 Hz had reported no effect from an EMF on melatonin levels but Wood et al. (1998) and Karasek et al. (1998) had reported delays in the rise. There have been few studies looking at the effects of RF EMFs on melatonin production. Studies by Mann et al. (1998) and Radon (2001) failed to find an effect on melatonin from an exposure to a cellular phone. However in the study by Mann, the antenna and phone were remote from the subject. The antenna was 40 cm away from the vertex of the head in a supine subject and produced an average power density of 0.02 mW/cm^2. The room was electrically shielded and insulated to protect from reflections. This is not the normal situation of use for a cell phone. The study by Radon et al. (2001) had the subjects sitting 10 cm from an antennae that was linked via an amplifier to a

digital car phone. A study by de Seze et al. (1999) exposed subjects to a 900 MHz or 1800 MHz commercially available phone for 2 hours per day, 5 days per week for 4 weeks. They reported no alteration in the melatonin profile. Additional studies in this area are needed that realistically reflect the conditions of use. All the studies published have used only male subjects. de Seze (1999) noted a need to assess female subjects. The possibility of a subgroup of susceptible individuals as reported by Graham et al. (1996) has also not been addressed in RF EMFs.

Little research has been carried out on the effects of RF EMFs on attention and memory. Brain structures associated with the cognitive functions of memory and attention are adjacent to a cellular phone in use. Working memory is associated with the prefrontal cortex which is located close to a cell phone in use. Memory also involves areas such as the temporal lobe and temporal stem. Attention cannot be localised to one area of the brain but involves the orbito-frontal areas, inferior parietal cortex, inferior and medial temporal cortex and structure in the limbic system (Snyder & Nussbaum, 1998).

Most studies carried out on attention have included other factors such as reaction time or working memory. For example, Koivisto et al. (2000a) found an increase in vigilance on exposure to a 915 MHz EMF. The task involved identifying the letters l, m, or y in a presentation of detractor letters. Reaction time was measured as part of the task. Beale et al . (1997) and Crasson et al., (1999) used a task that was closest to pure attention in studies on possible cognitive effects from 50 Hz EMFs. There is a need for studies on possible effects from cellular phone use on attention as there could be safety consequences in activities such as driving which people often combine with talking on a cellular phone.

Preece et al. (1998) had reported disruptions in accuracy from a 50 Hz EMF on numerical working memory. In 1999, Preece et al. used the same task in an experiment exposing subjects to a simulated cellular phone but failed to find an effect but conditions were different to that found in a normal cell phone in use.

Studies on 50 Hz EMFs had pointed to an effect on delayed word recall (Keetley, 2001) where 15 words were given then, after a detractor list was given, the words were asked to be recalled. There was a significant field decrease in the number of words recalled. Preece et al. (1998) also found a

significant effect on delayed word recall from a 50 Hz EMF. A further study by Preece et al. (1999) using a 915 MHz RF EMF failed to find an effect in a delayed word recall task but the study used an exposure protocol that was different to a real phone in use. Preliminary studies on memory have suggested RF EMFs may have greater effect on more difficult memory tasks (Koivisto et al. 2000).

Experiment Three was carried out in 2000-2001. It was a pilot study whose aim was to address some of the above research issues. The parameters in the study were chosen as they were the areas where very few studies had been published. The aim of the study was to meet the requirement for 'realistic exposure conditions' whilst exercising as much control as possible over confounding variables relating to the parameters tested.

A digital phone was chosen as it was becoming the most common phone, with analogue phones expecting to be phased out.
An evening exposure time was chosen as it allowed an investigation into the effects of the phone on the evening melatonin rise.
The length of exposure was two 15 minute calls, a total exposure of 30 minutes. The two calls were made about an hour apart. Two calls were used as shorter, repeated calling is a feature of cell phone use, as airtime is expensive. This airtime expense was also a factor for the experiment.

Two possible mechanisms of interaction between RF EMFs and humans were also investigated. These were an increase in head temperature, and the possibility of a suppression of melatonin levels.

This research aimed to determine whether use of the cellular phone caused heating in the head and to quantify any change in temperature found. Temperature in the right ear (phone side) was taken to quantify the amount of heating which may be caused by RF fields or by conduction. Temperature was taken in the left ear (non-phone side) to investigate possible RF effects.

Possible effects from cell phone use on the evening melatonin rise were investigated. This is a time during which melatonin may be more susceptible to effects as EMF effects during this time is the most consistent effect reported in the literature (Karasek et al., 1998; Wood et al., 1998), although this effect has been reported for 50 Hz not RF. There have been few studies on possible

effects of cellular phone use on melatonin (Radon et al.,2001, Mann et al., 1997; de Seze et al., 1999) None have reported effects, but two of the studies used remote antenna and shielded rooms which don't reflect normal cell phone use (Radon et al.,2001, Mann et al., 1997). There is a need for more studies using a realistic exposure situation. The possibility that those with naturally low melatonin levels formed a susceptible sub group was investigated as was possible gender differences.

A possible EMF effect on the central nervous system control of heart rate and blood pressure was investigated. Few studies have been published on possible cardiovascular effects from cellular phone exposure in humans. Braune et al. (1998) found an increase in blood pressure in ten subjects exposed to a GSM digital phone for 35 minutes. Subjects were tested standing, supine and during the valsalva manoeuvre. Ten subjects were used. The present study aimed to investigate possible effects on heart rate and blood pressure using a larger number of subjects.

For the reasons stated in experiment one it was considered important to include a test of attention as attentional deficits show up as deficits in tasks on reaction time, forced choice, digit memory and word recall. All of the afore mentioned tasks have been reported to be effected by exposure to RF EMFs (Lee et al, 2000; Preece et al., 1999; Koivisto et al., 2000). Previous studies on attention have mainly included other elements such as reaction time or memory. The attention task used for experiment three was the same as that used in experiments one and two. It was similar to that used by Beale et al. (1997) and Crasson et al., (1999) in studies on 50 HZ EMFs It was a test of attention and visual scanning, which did not contain a memory component.

Preece et al. (1998) also found a significant effect on delayed word recall and numerical working memory tasks from a 50 Hz EMF. A further study by Preece et al. (1999) using a 915 MHz RF EMF failed to find an effect on the same tasks but the study used a simulated exposure rather than a real phone. The tasks need to be repeated using a cellular phone that has all the normal features users are exposed to. Both numerical and Word recall were assessed as they use different brain areas so may be effected differently.

MATERIALS AND METHOD

Design of the Experiment

The study was a repeated measures design comprising two exposure and three control sessions. During each of the five sessions the same tasks and measurements were completed. The order followed was; control, exposure, control, exposure, control.

The two experimental sessions were surrounded by three control sessions to compensate for the natural tendency of physiological variables to change over the course of an evening. Normally melatonin concentrations rise during the evening, whereas heart rate, blood pressure and temperature fall. Attention and memory can also decrease in acuity as the evening gets later.

The total experimental time was approximately 2 hours and 30 minutes. Each session took approximately 15 minutes. The actual time taken by each subject depended on the time taken to provide the saliva samples. It was important the subject remained in the field while the sample was provided as melatonin is released directly into the bloodstream as it is produced.

The subject had a 30 minute break following each exposure session to reduce fatigue and increase the time between exposures. This ensured the time between saliva samples was over 30 minutes. Lewy et al (1980) reported a drop in melatonin levels within 30 minutes of exposure to bright light and a recovery of levels within 30 minutes of cessation of exposure. If EMFs effect melatonin levels via the same mechanism that light does, a recovery to normal levels should have occurred before the next session. It was recognised that a 15 minute exposure time may be a bit short but much longer exposure times are unlikely in normal cellphone use. Also, a 30 minute exposure time, as used in experiments one and two, would have been prohibitively expensive in airtime with the number of subjects envisaged.

Each experiment was planned to begin 2 hours after sunset. This timing allowed melatonin levels to rise sufficiently to be detectable in saliva by RIA .

Each subject attended for a single evening. A single experimental time was chosen to reduce variability that can occur over longer time periods. This was

especially important for the cognitive variables. Subjects attended one at a time.

Subjects acted as their own control. The use of subjects as their own controls increased the sensitivity of the experiment. This was considered advisable as cardiac measures and melatonin levels show large inter-individual variations and cognitive performance can also vary widely between subjects.

The experiment was not carried out blind, as it was not practical and there is evidence that people can detect pulse-modulated radio frequency and microwave frequency radiation between 200 MHz to 6.5 GHz (Saunders et al., 1991).

The cellular phone was attached to the right ear as most people are right handed and this would be the most common ear used.

Instructions were read from a sheet to ensure a standardised delivery. Different versions of the tasks were used at each session to avoid practice effects. The set was used for a particular session differed between subjects and the order was randomised.

Subjects were screen before the study and eliminated if they were likely to introduce confounders (e.g. smoked). Subjects that participated were asked to refrain from activities prior to the experiment that may effect the results.

Subjects

Subjects were volunteers recruited from amongst the students of the Palmerston North campus of Massey University. A total of 43 subjects, 16 males and 27 females participated. Ages ranged from 18 to 41 years with a mean age of 22.6 years. University students were chosen as subjects, not only because they comprised a readily accessible pool of subjects, but also because, as a group of prospective professionals, they would be likely to be high users of cell phones in the near future. The aim was to use 80 subjects but the study was truncated at 43 due to the difficulty accessing subjects within a reasonable time period.

Subject screening

Prior to the first session, subjects were given an information sheet explaining the research and their rights as participants. They then completed a health

screening questionnaire (Appendix 3.1) and signed a consent form. Any subject responding in the affirmative to the screening questions was thanked for their time but excluded from the study. Smokers were not included in the study. To control for possible confounding variables, volunteers were asked to refrain from moderate or excessive exercise and taking alcohol for 6 hours prior to the session. They were also asked to restrict coffee, tea and coke intake to not more than one cup in the 4 hours prior to the experiment. They were also asked to avoid bright light prior to the session.
Details were collected on prior cell phone use.

Subject prior cellular phone use

Twenty of the subjects had not previously used a cellular phone and a further 13 had used one less frequently than once a week. Only 6 subjects had a daily frequency of use. Phone types used covered both digital and analogue models.

Ethical approval

The research was approved by the Massey University Human Ethics Committee (MUHEC 99/165).

Exposure System

Location

The experiment was carried out in a private residence in a residential area. There were no cell phone base stations in the local area. There were no other RF sources in the house. The room was a study measuring 2.65 m x 3. 65 m.

Digital Cellular Phone

The cellular phone used in this study was a commercially available cellular phone (Alcatel One Touch Easy, model OTE HF; manufactured by Alcatel in Europe). The phone had not been modified except for the addition of a band of velcro on the back. This was used to attach the phone to a plastic headband which held the phone in position on the subject's head (Figure 7.1). A 'placebo' cover was in place over the subject's left ear to produce the normal reduction in heat loss that occurs when an ear is covered. The ear muff was made of plastic and was similar in size to the cellular phone.

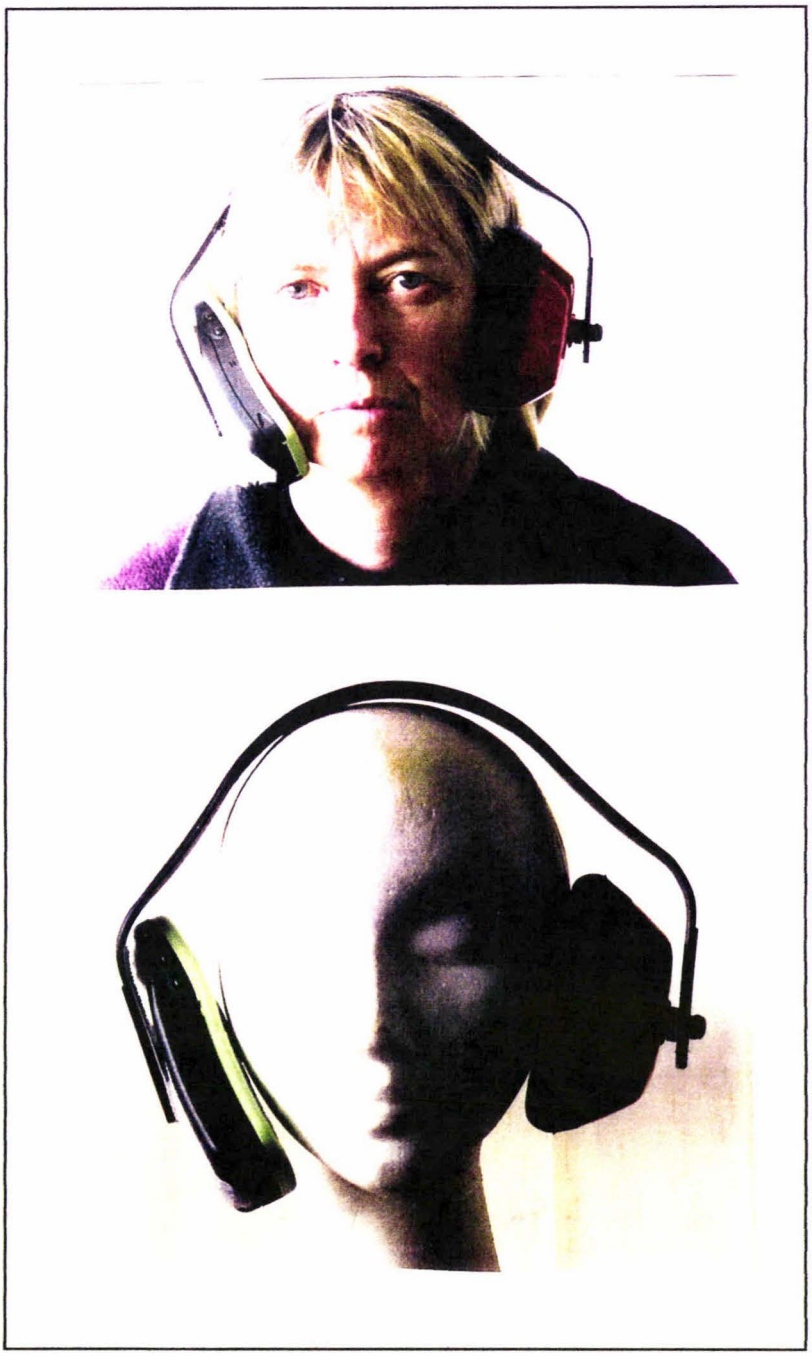


Figure 7.1: Photograph of the Cell Phone in Use

Characteristics of the EMF produced

Exposure intensity and timing

The mean output of the phone was 0.25 W time averaged power output. The measurement was the same whether the antennae was up or down. The field intensity varies according to the position of the phone relative to the closest station and the condition of the battery. Thus the intensity routinely varies over the course of a normal phone conversation. As the aim was to mimic the normal use of a phone as much as possible, no attempt was made to produce a consistent signal output. However, the battery was fully charged prior to each use and the phone was operating for the same length of time for each subject so the exposure parameters would have similar for each subject. A consistent location and seating position was used by all subjects. The output of a digital cellular phone is produced in pulses. GSM phones typically transmit only one-eighth of the time and send bursts of 577μs duration with 217 bursts per second (Linde and Mild, 1997).

Exposure frequency-domain characteristics

The frequency emitted was in the 900 MHz band (890-950 MHz)(Figure 7.2, 7.3). It was a typical digital phone which conformed to the New Zealand Standard. Van Leeuwen et al. (1999) described a typical GSM phone as having a 915 MHz dipole antennae with a time-averaged output of 0.25 W and a SAR of about 1.6 W/kg. Facilities to measure SAR for this phone, were not available.

Changes in output when transmitting speech and during periods of silence

The output of the phone changed when the person was talking compared to when speech was absent. When a person was talking the band width was about 890-950 MHz with a multi-modal pattern (Figure 7.2). When the person was silent the band width remained the same but the pattern changed to a bimodal pattern with a 50 % greater amplitude (Figure 7.3). The change was immediate on the cessation of speech.

Exposure geometric characteristics

Equipment was not available in New Zealand to measure the field geometric characteristics of this phone, but it was expected characteristics would be similar to those reported in the literature. For more information on typical field geometric characteristics the reader is referred to Chou et al. (1996), Bernardi et al. (2000).

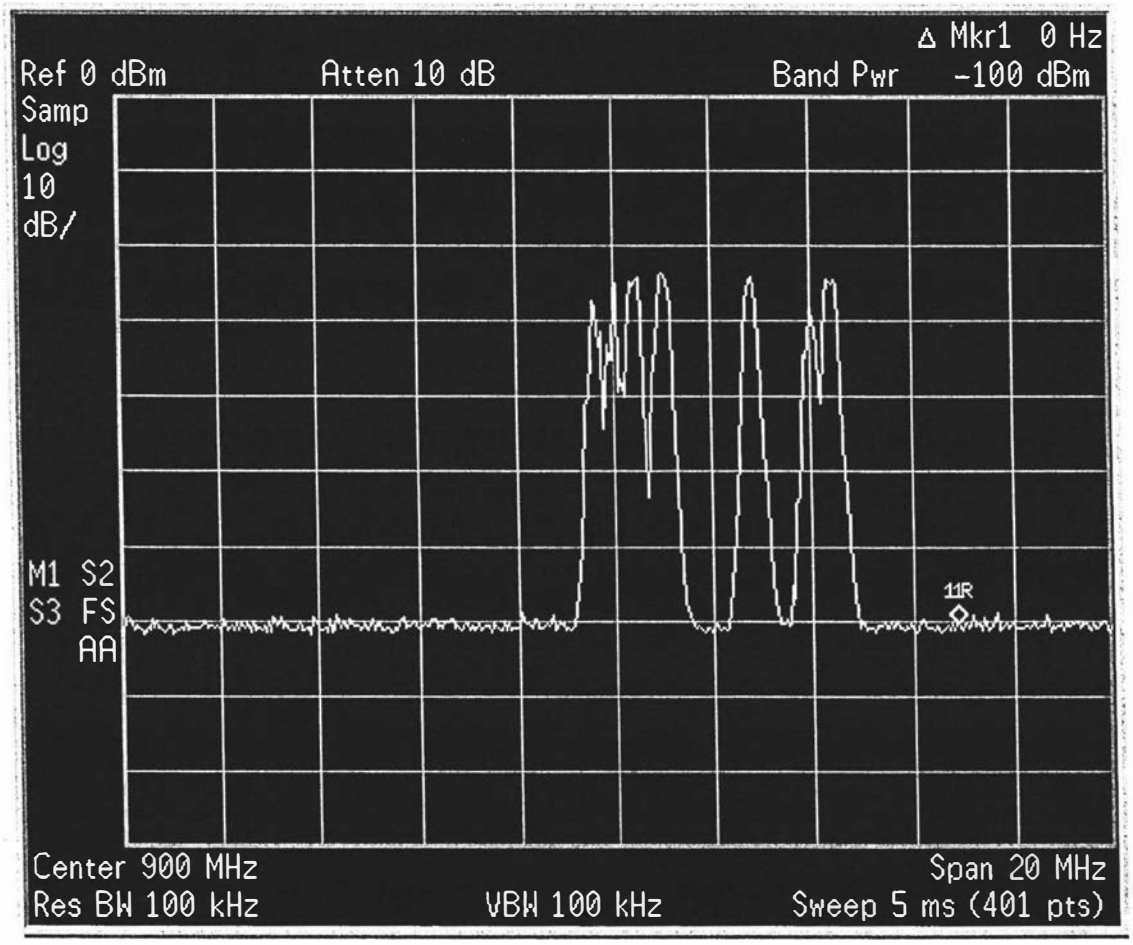


Figure 7.2: Oscilloscope Tracing of the Cell Phone Output-Subject Talking

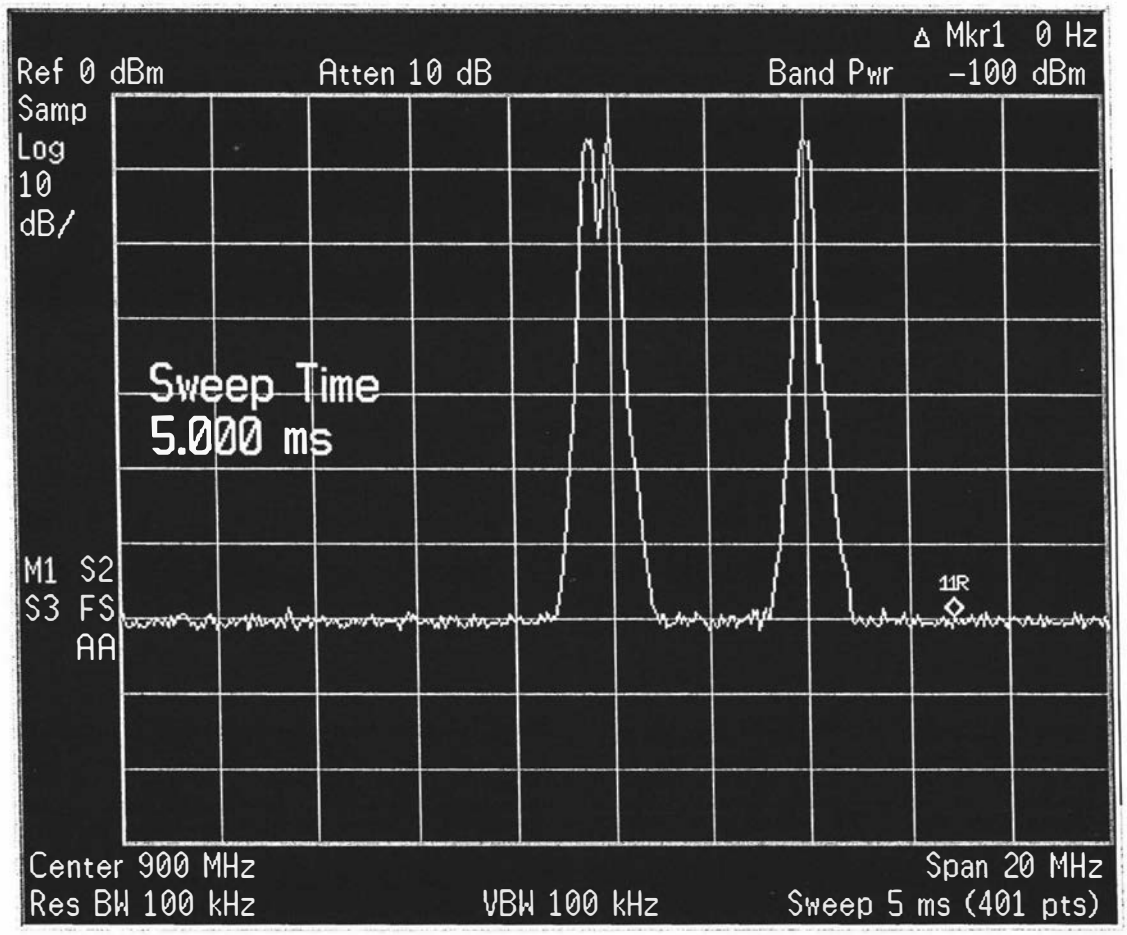


Figure 7.3: Oscilloscope Tracing of the Cell Phone Output-Subject Silent

Lighting

Lighting was provided by an overhead 40 watt (226 lux) clear glass bulb prior to the experiment commencing, then by a 15 watt lamp (150 lux) placed behind the subject during the actual experiment. This kept light levels well below those reported to suppress melatonin production in humans (Lewy et al., 1980; Reiter, 1991; Wever, 1985).

Seating

The subject sat in a chair with the phone positioned at the right ear and the blood pressure cuff on the left arm. The same seating position was used for all subjects. The chair was a padded wooden-framed construction.

Monitoring Equipment used and Measurements Taken

Cardiovascular measurements

Blood pressure and heart rate were initially monitored by a Rossmax Medical RM-4000 (Rossmax International, Germany) monitor attached to the left wrist, which provided a digital readout of the time, blood pressure and heart rate. The cuff automatically inflated on pressing a button and produced a reading in 60 seconds. Unfortunately this unit failed and was unable to be replaced so subjects 23-43 had their blood pressure taken by a Nissei ES-12G sphygmomanometer with the cuff placed on the left upper arm. Pulse rate was taken at the wrist and timed using a stopwatch.

Aural Temperature readings

Temperature readings were taken using an IRT 3020 Thermoscan aural thermometer (Kronberg, Germany). The thermoscan produced a reading in one second and was accurate to 0.1 degree Celsius.

Aural temperature was used as it was a non-invasive measure which was able to be carried out quickly. Other methods (such as the use of temperature sensitive tape, as is used on the foreheads of children, to provide continuous readings), were considered but rejected as not accurate enough.

During initial testing of the equipment, the signal from the phone had interfered with the thermoscan. The thermoscan beeped and didn't provide a reading if placed in the ear alongside the phone. This was why, initially the aim to test the temperature on the phone side as well as the non-phone side was abandoned. However, with subsequent trials it was found that if the phone was

angled back slightly for the 1-2 seconds it took to take the reading, a reading could be obtained. A check against a non-electronic thermometer confirmed the readings were accurate. If the probe picked up interference from the phone the thermoscan would beep and not provide a reading. This meant there was a check to ensure the thermoscan was in the correct position relative to the phone and was reading correctly. If the thermoscan beeped during the experiment the reading was redone. That is why temperature recordings for the right ear start from subject 21. The thermoscan never beeped when readings were taken from the left ear.

Salivary Melatonin levels

A single saliva sample was collected from each subject at the end of each of the 5 sessions, a total of 5 samples per subject. Samples were collected in 5 ml plastic tubes and immediately frozen. They were later assayed for melatonin concentration using the RIA procedure described in experiment one. A full discussion of the reasons for choosing the method of collection and assay for the determination of melatonin levels has been given in experiment one. The cut off value for the 'low excretor' group was set at 8 pg/ml or below. This was the same protocol as was used for experiments one and two, adjusted for a slightly earlier start time. Classification was made on the basis of the first control session.

Cognitive Tasks

The following cognitive tasks were used as they are common tests used in psychological assessment and have been extensively validated. Tasks were chosen to assess effects on simple attention, working memory for words and working memory using digits. Two memory task were chosen as manipulation of words in the brain uses a different area to that required for the manipulation of digits.

Digit span working memory task

This was a test for working memory involving numbers. The test is one commonly used to test for damage to the parietal cortex, which lies adjacent to a phone held against the ear.

Following the protocol reported by Preece (1998) subjects were read a series of 5 two-digit numbers to hold in working memory. After a one minute break, they were presented with a probe number and asked to indicate if that number was in the original list. There were 30 probe numbers, presented

individually. Responses were recorded on to a form by the experimenter. The score was the number correctly identified as being in the original list.

Delayed word recall

This test was chosen as it is commonly used in psychology and forms part of several common neuropsychology test batteries. The task was used by Preece et al. (1998) in assessing possible effects from a 50 HZ EMF and in a 1999 study assessing possible effect from a RF EMF.

At the start of each session a set of fifteen words was read to the subject to hold in working memory. Words were given at two second intervals. At the end of the session the subject was given one minute to recall as many of the words as possible. To avoid practice effects a different set of words was used for each session (sets A to E). To ensure the same set of words was not used for a particular session across all subjects, the selection of which order the sets were presented in for each subject was randomised. Responses were recorded by the experimenter with the score being the number of words correctly recalled.

Attention Task

The attention task was one that is commonly used in psychology and several forms are available. This was the same task used in experiments one and two. It was considered necessary to include a task that didn't contain a memory component and was as close as possible to a pure attention task. It is a timed task and "is a measure of several inter-related functions which include sustained attention, visual scanning, response inhibition and rate of information processing" (Gordon et al., 1997. p.325). Variants of the task have been used by other researchers studying the effects of EMFs (e.g. Beale et al, 1997; Crasson et al., 1999).

This was a paper and pencil test in which a page of numbers is supplied and the subject was told to put a vertical line through each six on the page. Numbers were arranged on the page in groups of 5. Subjects were allowed to choose whether they scanned down the columns or across the rows, but were required to use the same method each time. The time allowed was 60 seconds. There were 5 pages of numbers, a different one for each session. Each page contained the same numbers but differed in that numbers had been transposed within lines and the order of lines of numbers on the page differed. This was done to reduce practice effects. Responses were recorded by the subject. The attention

task was introduced to the protocol from subject 3 onwards.
Score was number of 6s correctly crossed minus number missed or incorrect numbers crossed.

Experimental Protocol

Format of the experiment

First the procedure was explained in detail. Any questions were answered and the consent form was signed. Subjects were asked to remove their watch and any metal objects such as jewellery which may have affected the electromagnetic field. A blood pressure cuff was placed on the left arm and the phone placed on the head with the phone against the right ear. The cell phone was attached by velcro to a headset which kept the phone in position. This also freed the subject to complete written tasks and handle the tube to provide the saliva sample for the measurement of melatonin levels. The subject then completed a practice trial to become familiar with the tasks and the taking of cardiac and temperature readings. The time taken also allowed the subject to adapt to the lower light levels and any effects of bright light exposure before the session to wear off. Once the subject's blood pressure and pulse had stabilised the experiment began.

Format followed for each individual session

A repeated measures design was used to reduce noise produced by the variability of individual pulse and blood pressure readings. Five readings were taken per session of blood pressure, temperature and pulse rate. These readings were then averaged to produce one result per session.

Each session was identical and followed the format set out below.

1. The 15 words to remember were read out.
2. Cardiac and temperature readings were taken.
3. The first group of 5 digits to remember was read out.
4. The second set of cardiac and temperature readings was taken.
5. The first set of 30 probe digits was read individually and the subject asked to indicate if each was/ wasn't in the group of 5 given a minute previously.
6. The third set of cardiac and temperature readings was taken.
7. The second group of 5 digits was given to remember with an instruction to forget the first set.
8. The fourth set of cardiac and temperature readings was taken.
9. The second set of 30 probe digits was read individually and the subject asked to indicate if each was/ wasn't in the group of 5 given a minute previously.

10. The fifth set of cardiac and temperature readings was taken.
11. The subject carried out the attention task.
12. The subject was asked to recall the 15 words given at the start of the session.
13. The subject provided a saliva sample for RIA melatonin assay.

During exposure sessions the cell phone dialled a land line held by the experimenter. This enabled the experimenter to check the link remained open. Instructions were given down the land line and the subject responded into the cell phone. This was done to simulate a normal conversation. Cell phones reduce power if there is no sound. During the control sessions exactly the same procedure was followed but with the phone switched off. The experimenter sat beside the subject for all sessions.

Data analysis

Two methods of analysis were carried out. A paired t-test was used to compare the mean of the three control sessions with the mean of the two experimental sessions for each parameter tested. The paired t-test was chosen as inter-individual variation for some parameters could be expected to be larger than may be produced by any possible field effects. Thus each subject acted as their own control. This method increased the sensitivity of the design. In addition to the t-test, each parameter was subjected to a repeated measures ANOVA which analysed the trend of the scores over the five sessions to determine whether there was a deviation from the expected score when the cellular phone was transmitting. For the ANOVA analysis, subjects were the main unit and measurement occasions were the subunits. In addition, the melatonin data were assessed using a log scale, as variance was unequal with subjects with high melatonin concentrations also having a high variance.

Pre-analysis treatment of data

Prior to carrying out the t-test the multiple readings taken in each session were averaged to produce one result per parameter, per session. The scores for the control sessions were then averaged and the same was done for the exposure sessions. The differences between the control and exposure sessions were calculated for each subject then analysed using a paired t-test.

For the ANOVA the multiple readings were averaged to produce one reading per parameter, per session then the ANOVA was carried out on the session results in the order in which the experiment was carried out. Data were analysed using SPSS, version 9 for windows software.

RESULTS

Aural Temperature

The aural temperature was higher in both ears when the phone was operating, with the temperature increase being more marked in the right ear to which the phone was attached. This is demonstrated in Figures 7.4 and 7.5 which show the mean temperature for each session for the left and right ears respectively. The control sessions demonstrated the expected slow decline in temperature over the evening.

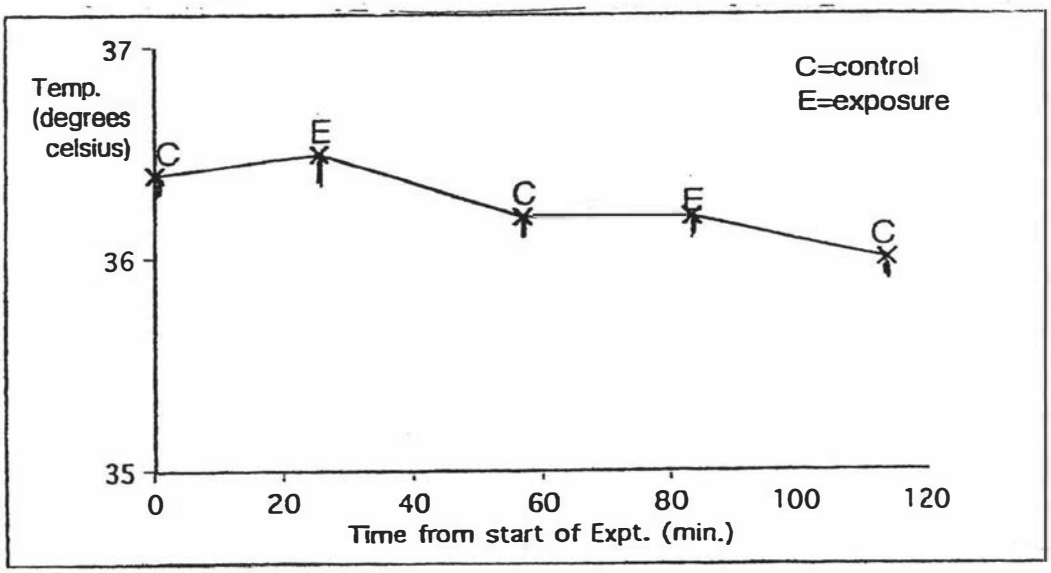
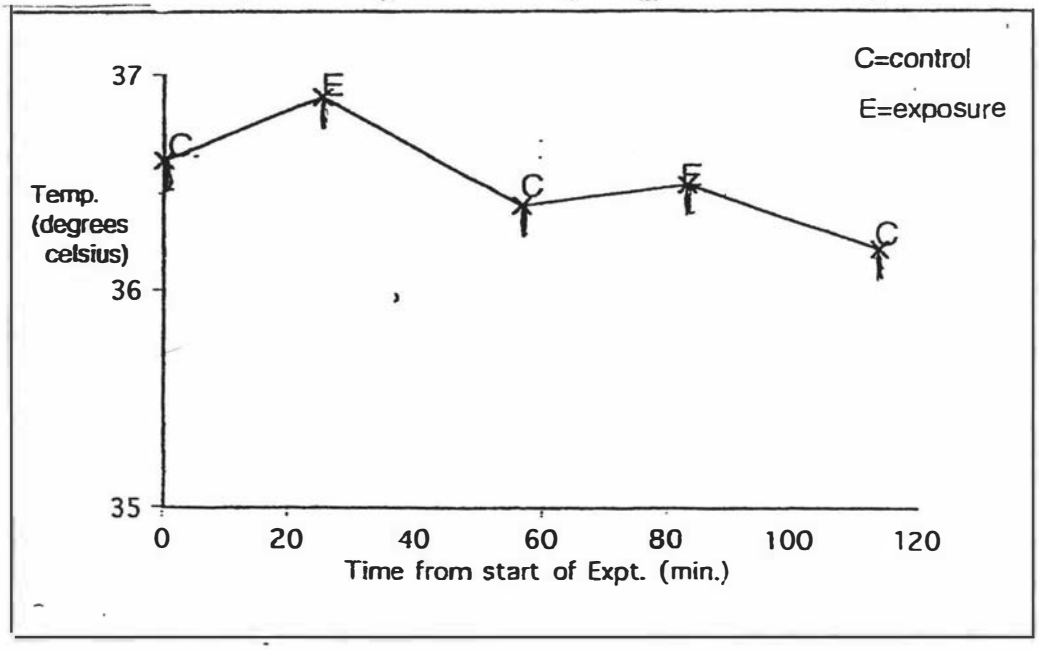


Figure 7.4: Mean (+SEM) aural temperature over all subjects during the experiment Left Ear



7.5: Mean (+SEM) aural temperature over all subjects during the experiment-Right Ear

Difference in aural temperature between Exposure and Control conditions

A paired t-test analysis indicated a significant rise in aural temperature in both the left ear (0.1±0.043, mean ± SEM, t_{42}=-2.1739, p<0.05)(Table 7.2) and right ear (0.3 ±0.079, mean ± SEM, t_{22}=-3.8030, p<0.001)(Table 7.3) when the phone was transmitting compared to when it was off. The rise in temperature was much more marked in the right ear to which the phone was attached.

An ANOVA confirmed the t-test results with the right aural temperature being significantly higher when the phone was transmitting (F=37.257 on 1.85 df. p<0.00) than when it was off. The Left (non-phone ear) was marginally hotter when the phone was transmitting than when it was off. In a comparison of the confidence intervals the SE difference was 0.067, so 36.36-36.24/0.067= 1.79. giving 0.05<p<0.10 (Table 7.1).

Table 7.1 : ANOVA analysis of the effects of cell phone use on aural temperature

source	df	mean square	F	Sig
side	1.000	2.899	37.257	.000
side * field	1.000	0.353	4.537	.036
error	85.000	0.078		

(side=left ear vs. right ear. side*field= left vs. right ear, by field on/off.)

Estimated marginal means for side*field effect

field	side	mean	SE	95% C.I.
off	right	36.140	0.042	36.326-36.494
off	left	36.242	0.043	36.156-36.328
on	right	36.694	0.051	36.593-36.796
on	left	36.362	0.052	36.258-36.466

Difference in aural temperature between ears

The right (phone side) aural temperature was significantly higher than the left aural temperature when the phone was transmitting (0.3 ±0.094, mean ± SEM, t_{22}=3.1915, p< 0.005)(Table 7.4).

An ANOVA confirmed this result (F=37.257 on 1,85 df. p<0.01).

The right (phone) ear was also significantly warmer than the non-phone ear when the phone was not transmitting, when analysed using a t-test (0.2 ± 0.6, mean ± SEM, t_{22}=3.3502, p< 0.05). A marginal difference was noted using ANOVA (F=4.537 on 1,85 df. p<0.05. (Table 7.1).

Table 7.2: Effects of Cell Phone Use on Aural Temperature

(degrees Celsius)				Left Ear				
	Pre. Control	Int. Control	Post. Control	Control Mean	First Exposure	Second Exposure	Exposure Mean	Mean Diff.
Subject 1	36.06	36.08	35.58	35.9	36.1	35.9	36.0	0.1
2	36.96	36.68	36.58	36.7	37.22	36.8	37.0	0.3
3	35.76	35.76	36.14	35.9	35.76	35.66	35.7	-0.2
4	36.62	36.7	36.5	36.6	36.84	36.68	36.8	0.2
5	36.54	36.64	36.34	36.5	36.66	36.4	36.5	0.0
6	36.82	36.26	35.84	36.3	36.68	36.28	36.5	0.2
7	36.48	36.16	35.72	36.1	36.4	35.96	36.2	0.1
8	35.84	35.86	35.9	35.9	35.9	35.98	35.9	0.1
9	36.1	35.98	35.9	36.0	36.54	36.12	36.3	0.3
10	36.46	36.14	35.74	36.1	36.14	36.18	36.2	0.0
11	36.02	35.58	34.9	35.5	35.92	35.6	35.8	0.3
12	35.54	35.84	35.78	35.7	35.9	36.14	36.0	0.3
13	37.24	36.94	36.92	37.0	36.92	36.92	36.9	-0.1
14	36.28	35.42	35.38	35.7	35.84	35.26	35.5	-0.1
15	36.82	36.36	35.94	36.4	36.32	36.42	36.4	-0.0
16	37.12	36.6	36.12	36.6	36.92	36.22	36.6	-0.0
17	36.58	36.78	36.08	36.5	36.9	36.48	36.7	0.2
18	36.4	36.56	35.7	36.2	36.42	35.9	36.2	-0.1
19	36.48	36.64	36.6	36.6	37	36.56	36.8	0.2
20	36.86	36.36	36.12	36.4	36.74	36.26	36.5	0.1
21	35.54	35.1	35.6	35.4	35.74	35.56	35.6	0.2
22	35.84	36.18	35.44	35.8	36.24	36.04	36.1	0.3
23	35.58	35.32	34.82	35.2	35.14	35.24	35.2	-0.1
24	35.96	35.1	35.52	35.5	35.04	35.72	35.4	-0.1
25	36.16	36.2	35.6	36.0	36	35.7	35.9	-0.1
26	36.42	36.66	36.5	36.5	36.6	36.42	36.5	-0.0
27	36.48	36.92	36.38	36.6	36.88	36.8	36.8	0.2
28	36.38	35.1	34.84	35.4	35.96	35.2	35.6	0.1
29	36.08	36.34	36.54	36.3	36.52	36.66	36.6	0.3
30	36.46	36.68	35.88	36.3	37.34	36.3	36.8	0.5
31	37.2	37	36.58	36.9	37.16	36.72	36.9	0.0
32	36.62	36.22	36.28	36.4	36.5	36.42	36.5	0.1
33	36.34	36.32	35.62	36.1	36.36	35.38	35.9	-0.2
34	37.28	37.14	37.08	37.2	37.62	37.26	37.4	0.3
35	37.2	36.68	36.56	36.8	37.52	37.38	37.5	0.6
36	36.58	36.32	35.98	36.3	37.68	37.26	37.5	1.2
37	36.4	36.04	36.8	36.4	36.5	36.68	36.6	0.2
38	35.86	35.58	34.96	35.5	35.32	35.12	35.2	-0.2
39	37.2	36.82	36.52	36.8	37.26	37.06	37.2	0.3
40	35.06	34.98	34.72	34.9	35.26	35.02	35.1	0.2
41	37.14	36.9	37.1	37.0	36.88	36.08	36.5	-0.6
42	36.6	36.68	36.78	36.7	37.2	37.16	37.2	0.5

43	35.92	35.9	36	35.9	36	35.66	35.8	-0.1
Mean	36.4	36.2	36.0	36.2	36.5	36.2	36.3	0.1
SD	0.53	0.57	0.61	0.53	0.66	0.62	0.62	0.28
SEM	0.076	0.915	0.915	0.076	0.107	0.076	0.076	0.043
							t=	2.1739

Table 7. 3: Effects of Cell Phone Use on Aural Temperature

(degrees Celsius)				Right Ear				
	Pre.	Int.	Post.	Mean	First	Second	Mean	Mean
Session	Control	Control	Control	Control	Exposure	Exposure	Exposure	Difference
Subject 21		35.4	35.44	35.4	36.45	35.92	36.2	0.8
22	35.86	35.92	35.22	35.7	36.2	35.9	36.0	0.4
23	35.94	35.22	35.18	35.4	35.72	35.66	35.7	0.2
24	36.5	35.46	35.8	35.9	36	36.58	36.3	0.4
25	35.56	36.08	35.38	35.7	36.44	35.86	36.1	0.5
26	36	37.06	36.68	36.6	37.24	37.38	37.3	0.7
27	37.14	36.58	36.88	36.9	36.88	36.68	36.8	-0.1
28	36.22	36	35.24	35.8	36.62	35.44	36.0	0.2
29	36.5	36.34	36.62	36.5	36.66	36.24	36.5	-0.0
30	37.26	37.3	36.34	37.0	37.38	36.72	37.0	0.1
31	37.28	36.98	36.8	37.0	37.36	37	37.2	0.2
32	36.96	36.48	36.32	36.6	37.18	36.78	37.0	0.4
33	36.52	36.24	35.54	36.1	36.32	35.32	35.8	-0.3
34	37.8	37.18	37	37.3	37.38	37.28	37.3	0.0
35	36.84	36.82	36.62	36.8	37.88	37.16	37.5	0.8
36	36.66	36.26	36.1	36.3	37.44	36.64	37.0	0.7
37	36.28	36.3	36.62	36.4	36.52	36.62	36.6	0.2
38	36.48	36.52	35.98	36.3	36.86	36.1	36.5	0.2
39	36.88	37.2	36.74	36.9	37.6	37.44	37.5	0.6
40	35.12	35	35.24	35.1	36.06	36.58	36.3	1.2
41	37.56	37.02	37.14	37.2	37.24	37.28	37.3	0.0
42	36.48	37.02	36.68	36.7	37.52	36.96	37.2	0.5
43	36.66	36.66	36.68	36.7	36.5	36.28	36.4	-0.3
N=23								
Mean	36.6	36.4	36.2	36.4	36.9	36.5	36.7	0.3
SD	0.64	0.66	0.66	0.62	0.59	0.62	0.57	0.37
SEM	0.125	0.138	0.138	0.129	0.123	0.129	0.119	0.079
							t=	3.803

Table 7.4: Temperature Difference Between Ears (degrees Celsius)						
Session	Control Left	Control Right	Control Difference	Exposure Left	Exposure Right	Exposure Difference
Subject 21	35.4	35.4	0	35.7	36.2	0.5
22	35.8	35.7	-0.1	36.1	36	-0.1
23	35.2	35.4	0.2	35.2	35.7	0.5
24	35.5	35.9	0.4	35.4	36.3	0.9
25	36	35.7	-0.3	35.9	36.1	0.2
26	36.5	36.6	0.1	36.5	37.3	0.8
27	36.6	36.9	0.3	36.8	36.8	0
28	35.4	35.8	0.4	35.6	36	0.4
29	36.3	36.5	0.2	36.6	36.5	-0.1
30	36.3	37	0.7	36.8	37	0.2
31	36.9	37	0.1	36.9	37.2	0.3
32	36.4	36.6	0.2	36.5	37	0.5
33	36.1	36.1	0	35.9	35.8	-0.1
34	37.2	37.3	0.1	37.4	37.3	-0.1
35	36.8	36.8	0	37.5	37.5	0
36	36.3	36.3	0	37.5	37	-0.5
37	36.4	36.4	0	36.6	36.6	0
38	35.5	36.3	0.8	35.2	36.5	1.3
39	36.8	36.9	0.1	37.2	37.5	0.3
40	34.9	35.1	0.2	35.1	36.3	1.2
41	37	37.2	0.2	36.5	37.3	0.8
42	36.7	36.7	0	37.2	37.2	0
43	35.9	36.7	0.8	35.8	36.4	0.6
N=23						
Mean	36.2	36.4	0.2	36.3	36.7	0.3
SD	0.63	0.62	0.28	0.77	0.57	0.45
SEM	0.131	0.129	0.059	0.161	0.119	0.094
		t=	3.3502		t=	3.1915

Cardiovascular Parameters

Heart Rate

Heart rate generally decreases during the evening in a resting subject. Such a decrease was noted when the mean heart rate over the 43 subjects was plotted for each session (Figure 7.6). The use of the cellular phone appeared to have little effect on this decrease.

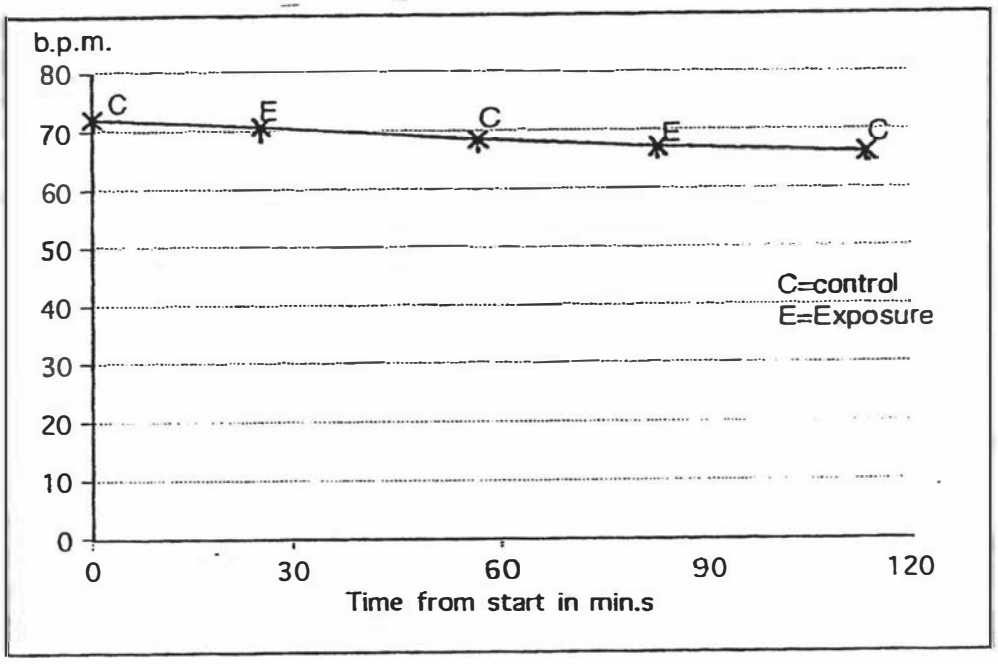


Figure 7.6 : Change in mean (+SEM) heart rate over all subjects during the experiment

This lack of an effect was confirmed by an analysis of individual differences using a two-tailed, paired t-test (Table 7.6). Results did not show a statistically significance difference (0.15 ± 0.3858, mean $\pm$ SEM, $t_{42}=0.3887$, $p>0.05$). An ANOVA also confirmed the result ($F=.376$ on 1,169 df, $p>0.05$)(Table 7.5).

Table 7.5: ANOVA analysis of the effects of cell phone use on heart rate

source	df	Mean square	F	Sig	
field	1.000	3.222	.376	.541	8.569
Error	169	8.569			

Estimated marginal means for field effect

field	mean	SE	95% C.I.
off	69.314	0.259	68.803-69.826
on	69.064	0.316	68.441-69.687

Table 7.6: Effect of Cellular Phone Exposure on Heart Rate (b.p.m.)

Subject	Pre. Control	Interval Control	Post Control	Mean Control	1st Exposure	2nd Exposure	Mean Exposure	Difference
1	83.4	73.2	69.2	75.3	78.4	75.2	76.8	1.53
2	84	79.4	72.8	78.73	80.6	74	77.3	-1.43
3	84.2	79.4	79.6	81.07	83.8	79.4	81.6	0.53
4	82.4	78.6	74.4	78.47	83.8	76.2	80	1.53
5	70.2	74.2	71.8	72.07	70.4	67	68.7	-3.37
6	78.2	70.8	64.6	71.20	75.4	68.6	72	0.80
7	79	72	66.4	72.47	82	70.6	76.3	3.83
8	88.8	81.4	78.6	82.93	82.6	79.6	81.1	-1.83
9	62.8	62.6	65.6	63.67	62	57.75	59.88	-3.79
10	86	78	80.2	81.40	89.8	85.8	87.8	6.40
11	78.8	71.6	68.8	73.07	76.4	72.4	74.4	1.33
12	69.8	68.4	61.8	66.67	68.8	69.8	69.3	2.63
13	68.2	65.8	62.2	65.40	65.6	64.8	65.2	-0.20
14	67.4	63.8	58.4	63.20	69	63.4	66.2	3.00
15	65	60.8	51.6	59.13	63	54.4	58.7	-0.43
16	71.8	68	58	65.93	70.2	56.6	63.4	-2.53
17	72	68.8	69.2	70.00	69.2	64.8	67	-3.00
18	62.6	59.2	58.6	60.13	59.2	59	59.1	-1.03
19	82.4	74.4	73	76.60	78.6	69	73.8	-2.80
20	71.6	63.2	61	65.27	63.6	64	63.8	-1.47
21	69.4	64.6	61.4	65.13	65.4	60.8	63.1	-2.03
22	72.2	71.2	-	71.70	67.6	65.5	66.55	-5.15
23	74.2	73	78	75.07	73.8	68.8	71.3	-3.77
24	50.4	48	44	47.47	50.4	47.2	48.8	1.33
25	64	64	68	65.33	64.8	70.4	67.6	2.27
26	68	67.2	64.8	66.67	68	67.2	67.6	0.93
27	80	76.2	69.8	75.33	75.2	74.6	74.9	-0.43
28	60.8	56	53.6	56.80	58.4	55.2	56.8	-0.00
29	84.8	79.2	80.8	81.60	84.8	84.8	84.8	3.20
30	70.4	67.2	69.6	69.07	73.6	72.8	73.2	4.13
31	72	76.2	80	76.07	74.4	77.2	75.8	-0.27
32	76.8	72	72	73.60	72.8	69.6	71.2	-2.40
33	68	67.2	66.4	67.20	66.4	62.4	64.4	-2.80
34	72.8	74.4	76.8	74.67	72.8	76.8	74.8	0.13
35	74.4	71.2	63.2	69.60	76	72	74	4.40
36	75.2	64.8	61.6	67.20	69.6	63.2	66.4	-0.80
37	78.4	77.6	78.4	78.13	82	73.6	77.8	-0.33
38	65.6	63.2	63.2	64.00	66.4	64	65.2	1.20
39	78.4	68	64.8	70.40	72	65.6	68.8	-1.60
40	69.6	60.8	58.4	62.93	59.2	59.2	59.2	-3.73
41	69.6	64.8	60.8	65.07	68.8	62.4	65.6	0.53
42	59.2	60	56	58.40	60	56.8	58.4	0.00
43	56.8	57.6	51.2	55.20	58.4	50.4	54.4	-0.80

								162
Mean	72.5	68.8	66.4	69.3	71.0	67.3	69.1	-0.1
s.d.	8.46	7.35	8.84	7.81	8.59	8.66	8.40	2.53
SEM	1.290	1.121	1.348	1.191	1.310	1.321	1.281	0.386
							t=	0.2591

Blood Pressure

Systolic Blood Pressure

A visual analysis of the trend in expected blood pressure fall over the evening did suggest the fall was halted at the times the cell phone was transmitting (Figure 7.7). This was investigated using a repeated measures ANOVA but did not reach statistical significance (F=.455 on 1,169 df, p=0.501)(Table 7.7).

A two-tailed, paired t-test analysis of the difference between control and exposure conditions did not reach statistical significance at the p=0.05 level (-0.5 ±0.569, Mean± SEM), t_{42}=0.8787, p>0.05)(Table 7.8).

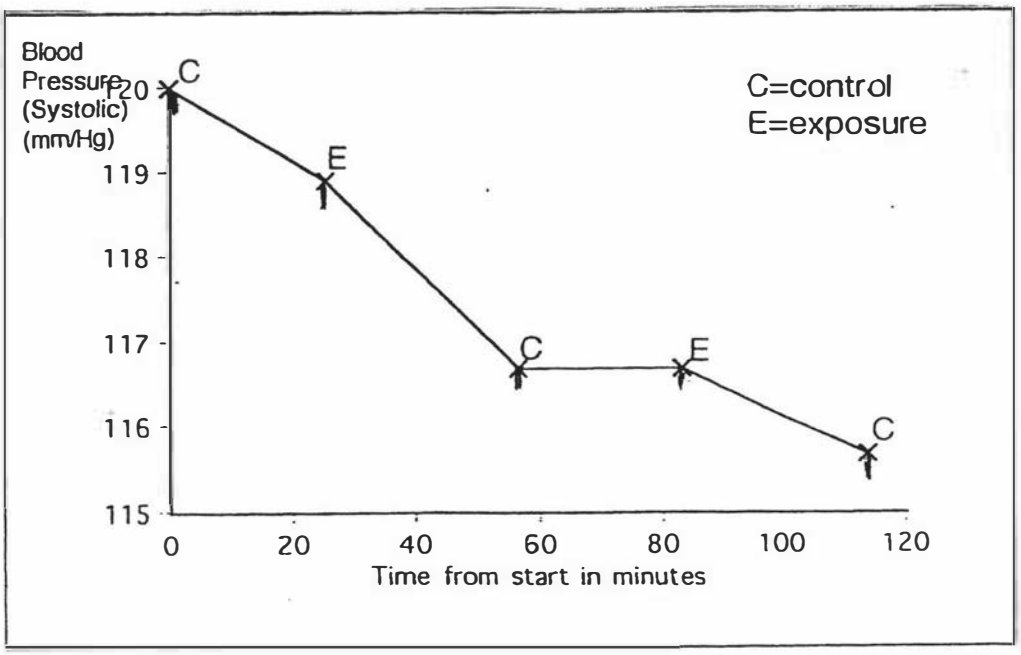


Figure 7.7 : Mean (+SEM) systolic blood pressure over all subjects over the course of the experiment

Table 7.7: ANOVA analysis of the effects of cell phone use on systolic blood pressure

source	df	Mean Square	F	Sig
field	1.000	.455	.501	32.609
error	169	32.609		

Estimated marginal means for field effect

field	mean	SE	95% C.I.
off	118.334	0.505	117.336-119.331
on	117.796	0.616	116.581-119.012

Table 7.8Effects of Cellphone Use on Blood Pressure (Systolic))mmHg)

Subject	Pre.	Int.	Post.	Mean	First	Second	Mean	
	Control	Control	Control	Control	Exposure	Exposure	Exposure	Difference
1	107	107.6	99.2	104.6	101.4	100.8	101.1	-3.5
2	119.6	104.6	99.2	107.8	109	107.8	108.4	0.6
3	99.6	100.6	102.4	100.9	101	102.8	101.9	1.0
4	90.2	91.2	88.2	89.9	83.6	90	86.8	-3.1
5	101.4	84.2	92.2	92.6	91.4	92.6	92.0	-0.6
6	147	136.6	133.2	138.9	142.2	138.8	140.5	1.6
7	123.6	128.2	122	124.6	128	122.6	125.3	0.7
8	124.8	110.2	118.2	117.7	114	115.2	114.6	-3.1
9	98.2	101	114	104.4	115.4	104.5	110.0	5.5
10	121.8	120	119	120.3	131.6	131.6	131.6	11.3
11	128.6	118.2	117.6	121.5	117.2	112.4	114.8	-6.7
12	96.2	95.4	98.8	96.8	99.6	96	97.8	1.0
13	122.2	124.6	111.6	119.5	119.6	112.4	116.0	-3.5
14	109	119.4	111.6	113.3	118.2	112	115.1	1.8
15	107.6	121	126.2	118.3	119.6	117	118.3	0.0
16	88.2	84.8	86.4	86.5	86.4	86.2	86.3	-0.2
17	145.6	139.4	147	144.0	146	134	140.0	-4.0
18	102.4	106.8	119.2	109.5	103.8	113.8	108.8	-0.7
19	114.2	99.8	110	108.0	106.4	99.6	103.0	-5.0
20	113	92.4	113.2	106.2	109.2	100.8	105.0	-1.2
21	99.8	98.4	103.2	100.5	92.6	93.6	93.1	-7.4
22	97.2	108.4	0	102.8	95.6	98.5	97.0	-5.8
23	101.8	104.6	125.6	110.7	103	125.6	114.3	3.6
24	145.2	134.8	140	140.0	134.8	134.4	134.6	-5.4
25	111.2	140.8	141.6	131.2	139.2	138.8	139.0	7.8
26	110.4	104.4	106.4	107.1	104.4	105.2	104.8	-2.3
27	120.4	113.5	117.2	117.0	118.4	118.4	118.4	1.4
28	127.6	126	117.2	123.6	124	111.6	117.8	-5.8
29	128.8	134.4	132.8	132.0	132.8	127.6	130.2	-1.8
30	107.6	111.6	109.2	109.5	111.6	111.2	111.4	1.9
31	134.8	128.4	144.4	135.9	134.8	132.4	133.6	-2.3
32	134	116	119.2	123.1	120	118.8	119.4	-3.7
33	139.2	136.8	134.4	136.8	137.6	133.2	135.4	-1.4
34	124.4	119.2	120.8	121.5	123.6	118.4	121.0	-0.5
35	129.6	127.6	120.8	126.0	126.8	127.6	127.2	1.2
36	132.4	124.8	123.6	126.9	131.2	124	127.6	0.7
37	114.4	104.8	102.8	107.3	106.8	103.2	105.0	-2.3
38	148.8	148	144.8	147.2	152.8	149.6	151.2	4.0
39	120	104.8	104.8	109.9	107.2	108.4	107.8	-2.1
40	137.6	129.2	126	130.9	134.4	129.2	131.8	0.9
41	172	152.4	146.4	156.9	165.5	152.8	159.2	2.2
42	148	140.4	140	142.8	143.2	140	141.6	-1.2
43	124	122.8	124.4	123.7	128	124.4	126.2	2.5

Table 7.8 Contd.								
Mean	120.2	116.7	115.7	118.3	118.9	116.7	117.8	-0.5
SD	18.33	17.00	24.00	16.21	18.38	16.28	17.10	3.73
SEM	2.795	2.592	3.66	2.472	2.803	2.483	2.608	0.569
							t=	0.8787

Pulse Pressure

Over the 43 subjects, there was little difference in mean Pulse Pressure when the phone was transmitting compared to when it was not transmitting (Figure 7.8).

A two-tailed, paired t-test indicated no significant difference between control and experimental conditions (0.7+ 0.4575, mean + SEM, t_{42}= 1.5317, $p>0.05$) (Table 7.9).

An ANOVA confirmed the t-test result (F=1.197 on 1,169 df, $p>0.05$)(Table 7.10)

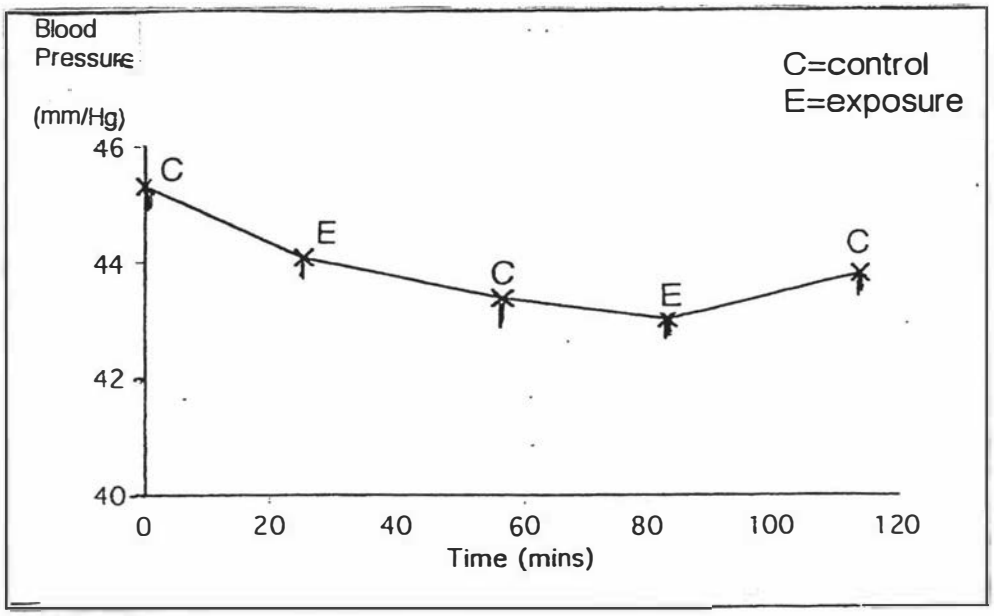


Figure 7. 8 : Mean (+SEM) pulse pressure over all subjects during the course of the experiment

Table 7.10 :ANOVA analysis of the effects of cell phone use on pulse pressure

source	df	Mean Square	F	Sig
field	1.000	35.178	1.197	.276
Error	169	29.397		

Estimated marginal means for field effect

field	mean	SE	95% C.I.
off	44.348	0.480	43.401-45.295
on	43.520	0.585	42.366-44.675

Table 7.9:Effects of EMFs on Blood Pressure (Pulse Pressure)

Subject	Control Pre.	Control Int.	Control Post.	Control Mean	Exposure First	Exposure Second	Exposure Mean	Mean Difference
1	40.4	51.2	46.8	46.1	34	43.6	38.8	-7.3
2	57	52.4	49.4	52.9	49.2	57.4	53.3	0.4
3	28.6	31.4	28.6	29.5	32.2	28.2	30.2	0.7
4	38	37.6	34.2	36.6	30.4	37	33.7	-2.9
5	38.2	21.4	31.4	30.3	32.2	31.8	32.0	1.7
6	62.6	69.4	59.6	63.9	57	61.8	59.4	-4.5
7	37.6	40.2	47.6	41.8	42.6	43.6	43.1	1.3
8	54.8	34.2	44.2	44.4	40.4	45.6	43.0	-1.4
9	29	27	32.4	29.5	43.8	32	37.9	8.4
10	38.8	40.4	36	38.4	42.2	42.2	42.2	3.8
11	40.2	45.6	44.4	43.4	44.4	41.4	42.9	-0.5
12	29.2	25.4	35	29.9	34.2	30.6	32.4	2.5
13	40	46.4	34.2	40.2	42.8	33.6	38.2	-2.0
14	35.6	57.4	54.6	49.2	41.8	46.2	44.0	-5.2
15	47.8	66.4	72.4	62.2	54.8	70.2	62.5	0.3
16	29.6	23.4	27.6	26.9	20.6	27.8	24.2	-2.7
17	44.4	41.6	37.2	41.1	46.2	34.8	40.5	-0.6
18	36.4	27.2	47.2	36.9	33.2	39.6	36.4	-0.5
19	40.8	31.2	45	39.0	37.2	33.6	35.4	-3.6
20	37	30.2	48.8	38.7	31.2	40.4	35.8	-2.9
21	35.6	26.2	36	32.6	28	30.6	29.3	-3.3
22	17.8	14.4	0	16.1	15.4	13	14.2	-1.9
23	42.2	44.6	59	48.6	45.2	40.6	42.9	-5.7
24	66	51.6	55.6	57.7	58.4	52	55.2	-2.5
25	38.4	51.2	48.4	46.0	52	49.6	50.8	4.8
26	32	27.6	25.6	28.4	27.6	24.4	26.0	-2.4
27	46.8	43	40	43.3	44.4	41.2	42.8	-0.5
28	55.2	55.6	44	51.6	52.4	41.6	47.0	-4.6
29	50	61.2	55.6	55.6	57.6	52	54.8	-0.8
30	30.4	29.2	32	30.5	33.6	30.4	32.0	1.5
31	61.6	57.6	63.6	60.9	61.2	58.8	60.0	-0.9
32	53.2	38.8	39.2	43.7	41.6	42.4	42.0	-1.7
33	60.8	60.4	64.8	62.0	60.4	61.6	61.0	-1.0
34	52	47.6	53.2	50.9	52.4	49.6	51.0	0.1
35	54.8	64	52.6	57.1	61.6	58.4	60.0	2.9
36	50.8	48.4	40	46.4	47.6	44	45.8	-0.6
37	41.6	37	32	36.9	37.2	34.8	36.0	-0.9
38	58.8	55.2	53.2	55.7	61.8	58	59.9	4.2
39	50.8	39.6	35.6	42.0	41.2	35.6	38.4	-3.6
40	52	47.2	41.6	46.9	45.2	50	47.6	0.7
41	82	58	49.6	63.2	73.2	55.2	64.2	1.0
42	60.8	55.2	51.6	55.9	54.8	52	53.4	-2.5

Table 7.9 contd.								
43	46.8	52.4	52	50.4	51.6	52	51.8	1.4
Mean	45.3	43.4	43.8	44.3	44.1	43.0	43.5	-0.7
SD	12.5	13.5	12.8	11.4	12.2	11.8	11.5	3.0
SEM	1.906	2.059	1.952	1.738	1.86	1.799	1.754	0.457
						N=43	t=	-1.5317

Salivary Melatonin Levels

When observations for each session were averaged over the 43 subjects there was little deviation from the expected steady evening rise in melatonin levels over the course of the experiment (Figure 7.9).

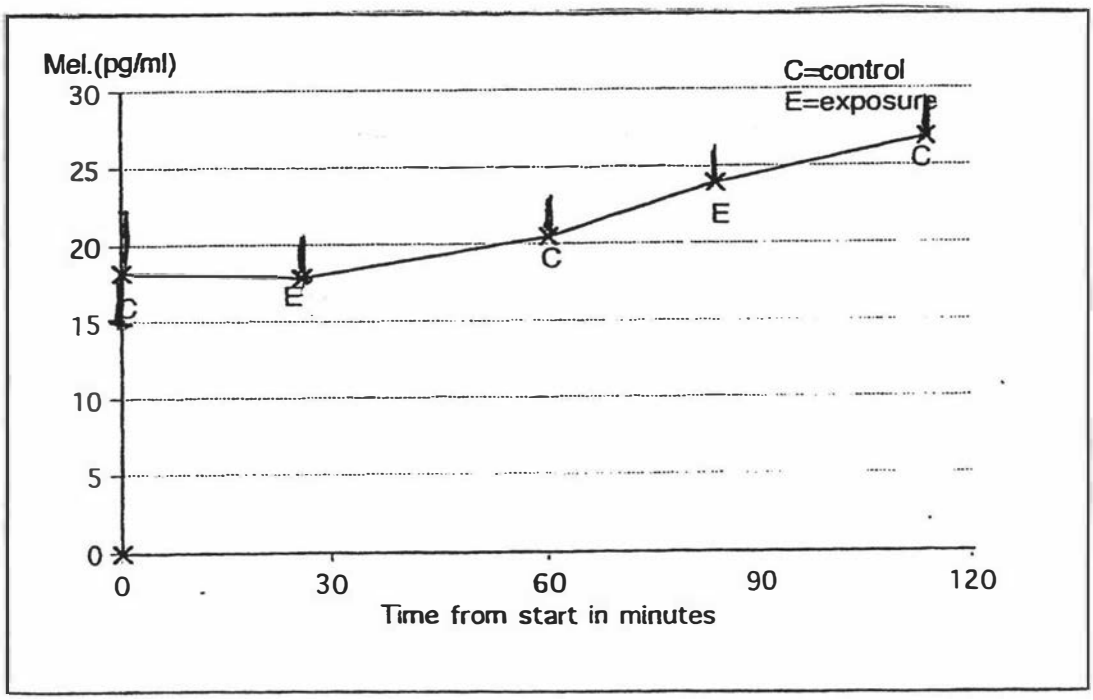


Figure 7.9 : Mean (+SEM) salivary melatonin levels over all subjects during the experiment

An analysis comparing the control and exposure sessions was carried out using a two-tailed, paired t-test (Table 7.11). Differences between control and exposure sessions did not reach statistical significance (-1.0 $\pm$0.625, mean$\pm$ SEM, $t_{42} = -1.6000$, $p > 0.05$).

A further analysis using ANOVA also did not reach statistical significance at the 0.05 level (F=.266 on df=1,169, p>0.05)(Table 7.12).

Data was assessed using a log scale as the variance was unequal, with subjects with high melatonin levels also having a high variance.

Table 7.12: ANOVA Log analysis of the effects of cell phone use on salivary melatonin

source	df	Mean square	F	Sig
field	1.000	0.012	.266	.607
Error	169	0.045		

Estimated Marginal Means

Field	Mean	SE	95% CI
Off	1.143	0.019	1.106-1.180
On	1.128	0.023	1.082-1.173

Subjects with low naturally occurring levels of melatonin

There were 13 subjects in this category. There was no difference between control and exposure sessions for this group (Table 7.11). (-1.38±0.6706, mean±SEM, t_{12}=2.053).

Gender Effects

There were no significant differences between control and exposure sessions for the female subjects (-0.500±0.900, mean ±SEM, p_{26}=0.5555). However, a significant difference was found for the male subjects (-1.77±0.720, mean ±SEM, p_{15}=-2.4569)(Appendix 3.5).

Radio-Immmunoassay

Samples were assayed in three batches (subjects 1-17, subjects 18-30, subjects 31-43). The maximum inter-assay variability in sensitivity was 1.65 pg/ml (Table 7.13).

Table 7.13: RIA sensitivity (2 SD)

subject	MT(pg/ml)
1-17	4.25
18-30	5.9
31-43	5.58
mean sensitivity	5.24

Table 7.11. The Effects of Cellphone Frequency EMFs on Salivary Melatonin

(pg/ml)		CONTROL			EXPOSURE			
Subject	Pre-Exp.	Interval	Post-Exp.	Mean-Con	Exposure	Exposure	Mean-Exp.	Difference
*1	3.0	10.0	14.0	9.0	5.0	10.0	7.5	-1.5
2	13.0	23.0	31.0	22.3	13.0	21.0	17.0	-5.3
3	10.0	20.0	23.0	17.7	18.0	19.0	18.5	0.8
4	13.0	11.0	5.0	9.7	11.0	5.0	8.0	-1.7
5	21.0	36.0	40.0	32.3	27.0	36.0	31.5	-0.8
6	34.0	35.0	47.0	38.7	29.0	43.0	36.0	-2.7
7	19.0	19.0	19.0	19.0	17.0	19.0	18.0	-1.0
8	9.0	5.0	5.0	6.3	21.0	18.0	19.5	13.2
9	0.0	9.0	20.0	9.7	4.0	15.0	9.5	-0.2
10	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
11	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
*12	7.0	23.0	28.0	19.3	12.0	29.0	20.5	1.2
13	25.0	32.0	37.0	31.3	29.0	34.0	31.5	0.2
14	25.0	21.0	15.0	20.3	21.0	13.0	17.0	-3.3
*15	8.0	15.0	11.0	11.3	8.0	15.0	11.5	0.2
*16	5.0	4.0	12.0	7.0	0.0	8.0	4.0	-3.0
*17	8.0	17.0	19.0	14.7	10.0	22.0	16.0	1.3
18	51.0	9.0	11.0	23.7	19.0	11.0	15.0	-8.7
19	57.0	67.0	77.0	67.0	66.0	78.0	72.0	5.0
20	26.0	31.0	34.0	30.3	34.0	26.0	30.0	-0.3
*21	6.0	8.0	25.0	13.0	4.0	12.0	8.0	-5.0
22	27.0	19.0	28.0	24.7	21.0	27.0	24.0	-0.7
23	13.0	16.0	21.0	16.7	13.0	18.0	15.5	-1.2
*24	2.0	2.0	10.0	4.7	2.0	5.0	3.5	-1.2
25	16.0	24.0	28.0	22.7	18.0	25.0	21.5	-1.2
26	27.0	44.0	61.0	44.0	41.0	56.0	48.5	4.5
27	34.0	12.0	22.0	22.7	16.0	18.0	17.0	-5.7
*28	2.0	4.0	14.0	6.7	0.0	9.0	4.5	-2.2
29	21.0	32.0	37.0	30.0	27.0	42.0	34.5	4.5
30	48.0	19.0	31.0	32.7	35.0	16.0	25.5	-7.2
*31	0.00	11.00	23.00	11.3	2.00	16.00	9.0	-2.3
32	17.00	27.00	37.00	27.0	20.00	26.00	23.0	-4.0
33	10.00	27.00	35.00	24.0	17.00	39.00	28.0	4.0
*34	1.00	19.00	35.00	18.3	2.00	24.00	13.0	-5.3
*35	2.00	2.00	11.00	5.0	0.00	7.00	3.5	-1.5
*36	5.00	20.00	51.00	25.3	8.00	39.00	23.5	-1.8
37	30.00	45.00	56.00	43.7	36.00	45.00	40.5	-3.2
38	0.00	0.00	2.00	0.7	0.00	0.00	0.0	-0.7
39	30.00	53.00	59.00	47.3	33.00	57.00	45.0	-2.3
40	17.00	29.00	48.00	31.3	28.00	45.00	36.5	5.2
41	0.00	0.00	0.00	0.0	0.00	0.00	0.0	0.0
42	137.00	74.00	55.00	88.7	91.00	64.00	77.5	-11.2
*43	6.00	7.00	18.00	10.3	10.00	17.00	13.5	3.2

Table 7.11 contd.								
Mean	18.3	20.5	26.9	21.9	17.9	23.9	20.9	-1.0
SD	23.5	17.2	18.5	17.7	18.2	18.2	17.4	4.1
SEM	3.584	2.623	2.821	2.699	2.775	2.775	2.653	0.625
(= subjects with naturally low melatoni .N=13)(x=-1.38,s.d.=2.418)*							N=43, t=	1.6000

Cognitive Parameters

Attention

The use of the cell phone resulted in a significant drop in attention levels. The expected rise in competency due to practice effects showed a noticeable dip at the times the cell phone was being used when mean results over all subjects was viewed on a session by session basis (Figure 7.10).

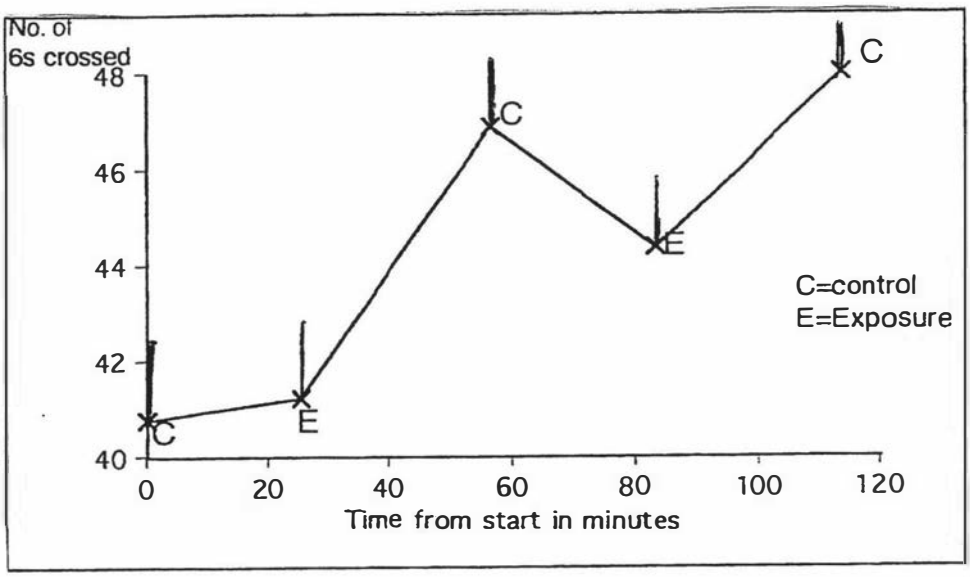


Figure 7.10 : Mean (+SEM) attention scores over all subjects during the course of the experiment

In a two-tailed, paired t-test analysis the drop in levels of attention was statistically significant (-2.5 $\pm$0.4685, mean difference $\pm$ SEM, t_{40}=5.3418, $p<0.001$)(Table 7.15). This result was confirmed using an ANOVA (F=20.77 on 1,162 df, $p<0.001$) (Table 7.14). There were 2 data sheets missing so the number was 41 for this task.

Table 7.14: ANOVA analysis of the effects of cell phone use on attention levels

source	df	Mean Square	F	Sig
field	1	302.8	20.77	<0.001
Error	162	14.6		

Estimated Marginal Means

Field	Mean	SE	95% CI
Off	45.300	0.344	44.620-45.980
On	42.818	0.422	41.985-43.651

This result was due to an increase in speed rather than accuracy as the mean number of '6's missed was the same for the control and exposure sessions.

Table 7. 15: Attention Task (NUMBER OF 6S CROSSED IN ONE MINUTE)

Subject	Pre.exp control	Interval control	Post-exp. control	Mean control	1st Exposure	2nd Exposure	Mean exposure	Difference
1								
2								
3	31	39	45	38.33	35	49	42	3.67
4	38	43	47	42.67	38	46	42	-0.67
5	33	37	43	37.67	38	34	36	-1.67
6	32	42	42	38.67	32	39	35.5	-3.17
7	45	44	44	44.33	44	51	47.5	3.17
8	41	46	45	44.00	35	33	34	-10.00
9	47	54	50	50.33	50	49	49.5	-0.83
10	29	38	44	37.00	26	37	31.5	-5.50
11	53	54	51	52.67	47	45	46	-6.67
12	36	42	40	39.33	36	35	35.5	-3.83
13	59	58	59	58.67	56	55	55.5	-3.17
14	31	52	46	43.00	40	43	41.5	-1.50
15	37	32	39	36.00	31	34	32.5	-3.50
16	33	42	44	39.67	35	35	35	-4.67
17	28	39	43	36.67	31	38	34.5	-2.17
18	45	48	47	46.67	46	41	43.5	-3.17
19	61	58	50	56.33	57	59	58	1.67
20	63	66	65	64.67	64	65	64.5	-0.17
21	29	43	47	39.67	33	35	34	-5.67
22	30	40	44	38.00	30	38	34	-4.00
23	45	48	47	46.67	44	46	45	-1.67
24	28	39	44	37.00	31	37	34	-3.00
25	55	61	53	56.33	60	64	62	5.67
26	43	51	49	47.67	43	46	44.5	-3.17
27	28	39	42	36.33	29	35	32	-4.33
28	30	38	40	36.00	32	34	33	-3.00
29	29	49	39	39.00	32	38	35	-4.00
30	64	72	71	69.00	64	68	66	-3.00
31	30	47	45	40.67	43	40	41.5	0.83
32	29	41	37	35.67	31	32	31.5	-4.17
33	34	42	46	40.67	32	41	36.5	-4.17
34	29	41	46	38.67	30	41	35.5	-3.17
35	40	46	47	44.33	41	43	42	-2.33
36	42	42	47	43.67	38	39	38.5	-5.17
37	38	49	44	43.67	42	40	41	-2.67
38	31	48	34	37.67	41	37	39	1.33
39	52	62	54	56.00	56	57	56.5	0.50
40	42	46	46	44.67	40	39	39.5	-5.17
41	33	42	45	40.00	31	37	34	-6.00
42	55	60	61	58.67	49	46	47.5	-11.17
43	35	44	45	41.33	35	34	34.5	-6.83

Table 7.15 contd.								175
Mean	39.0	46.3	46.7	44.0	39.8	43.1	41.5	-2.5
S.D.	11.2	8.8	6.9	8.5	10.4	9.6	9.8	3.0
SEM	1.749	1.374	1.078	1.327	1.624	1.499	1.531	0.468

Delayed Word Recall

There was no noticeable trend in the mean number of words recalled per session over the 43 subjects (Figure 7.11). Few subjects recalled more than 4 or 5 out of the 15 words given in any of the sessions (Table 7.16).

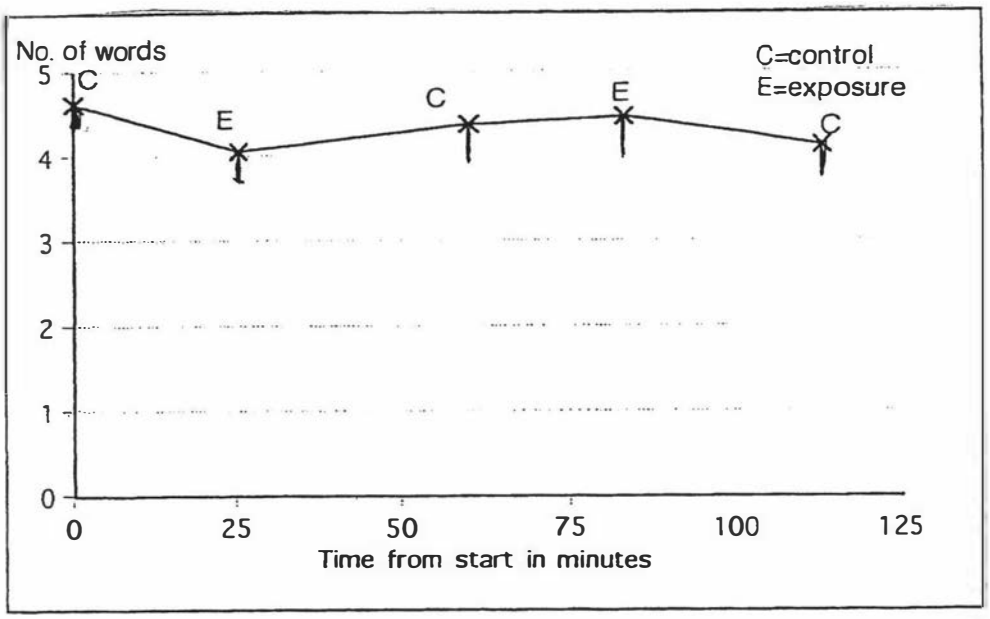


Figure 7.11 : Mean (+SEM) of words recalled over all subjects during the course of the experiment

A two-tailed, paired t-test confirmed this as the results did not reach statistical significance (0.2 $\pm$0.244, mean difference $\pm$ SEM, t_{42}=0.8197, p>0.05)(Table 7.16). An ANOVA also confirmed the result (F=.125 on 1,169 df, p=0.724)(Table 7.17).

Table 7.17: ANOVA analysis of the effects of cell phone use on delayed word recall

source	df	Mean Square	F	Sig
field	1.000	.403	.125	.724
Error	169	3.214		

Estimated Marginal Means

Field	Mean	SE	95% CI
Off	4.3595	0.158	4.407-4.670
On	4.270	0.195	3.885-4.654

Table 7.16 : THE EFFECTS OF CELL PHONE USE ON DELAYED WORD RECALL

(Number of words)		CONTROL			EXPOSURE			
SUBJECT	Pre-exp.	Interval	Post-exp.	MEAN-CON	Exposure1	Exposure 2	MEAN-EXP	DIFF.
1	4	5	6	5.0	4	4	4	-1.0
2	5	4	4	4.3	5	3	4	-0.3
3	3	4	2	3.0	5	0	2.5	-0.5
4	4	7	8	6.3	5	9	7	0.7
5	5	5	5	5.0	3	5	4	-1.0
6	4	4	5	4.3	0	4	2	-2.3
7	3	2	2	2.3	3	1	2	-0.3
8	6	6	7	6.3	3	7	5	-1.3
9	5	8	5	6.0	6	8	7	1.0
10	0	8	0	2.7	6	7	6.5	3.8
11	5	4	6	5.0	1	3	2	-3.0
12	4	1	2	2.3	6	2	4	1.7
13	7	4	4	5.0	1	6	3.5	-1.5
14	6	6	7	6.3	6	7	6.5	0.2
15	4	1	3	2.7	2	1	1.5	-1.2
16	7	5	7	6.3	4	7	5.5	-0.8
17	4	2	1	2.3	0	4	2	-0.3
18	10	0	3	4.3	5	1	3	-1.3
19	7	6	5	6.0	5	4	4.5	-1.5
20	4	4	7	5.0	6	5	5.5	0.5
21	4	8	6	6.0	4	6	5	-1.0
22	5	8	4	5.7	7	5	6	0.3
23	7	7	4	6.0	4	8	6	0.0
24	4	1	2	2.3	1	0	0.5	-1.8
25	6	1	3	3.3	4	1	2.5	-0.8
26	4	2	5	3.7	4	6	5	1.3
27	1	1	3	1.7	4	3	3.5	1.8
28	5	6	5	5.3	5	5	5	-0.3
29	4	7	6	5.7	3	6	4.5	-1.2
30	5	12	8	8.3	12	13	12.5	4.2
31	7	5	1	4.3	2	1	1.5	-2.8
32	5	6	3	4.7	3	5	4	-0.7
33	5	7	8	6.7	9	7	8	1.3
34	7	6	5	6.0	5	5	5	-1.0
35	6	4	2	4.0	4	5	4.5	0.5
36	5	3	2	3.3	5	7	6	2.7
37	3	4	4	3.7	1	3	2	-1.7
38	4	0	2	2.0	3	2	2.5	0.5
39	3	6	3	4.0	4	4	4	0.0
40	3	1	3	2.3	3	4	3.5	1.2
41	4	6	5	5.0	6	3	4.5	-0.5
42	1	0	2	1.0	2	2	2	1.0
43	4	1	2	2.3		3	1.5	-0.8

Table 7.16 contd.								178
MEAN	4.6	4.4	4.1	4.4	4.1	4.5	4.2	-0.2
S.D.	1.8	2.7	2.1	1.6	2.3	2.8	2.2	1.6
SEM	0.274	0.412	0.32	0.244	0.351	0.427	0.335	0.244

Digit span Working Memory

The use of the cell phone did not appear to result in a difference in numerical working memory on first analysis. The mean number of correct recalls was 29 for both the control and experimental sessions (Table 7.18). There was little change in mean responses over the course of the experiment (Figure 7.12). Most of the subjects gained almost full marks which made the analysis difficult as the variance showed a 'pinched' effect . A t-test analysis was not the appropriate measure in the circumstances. An ANOVA using logit analysis found a marginal drop in recall when the phone was transmitting ($F=4.545$ on 1,170 df, $p<0.05$) (Table 7.19).

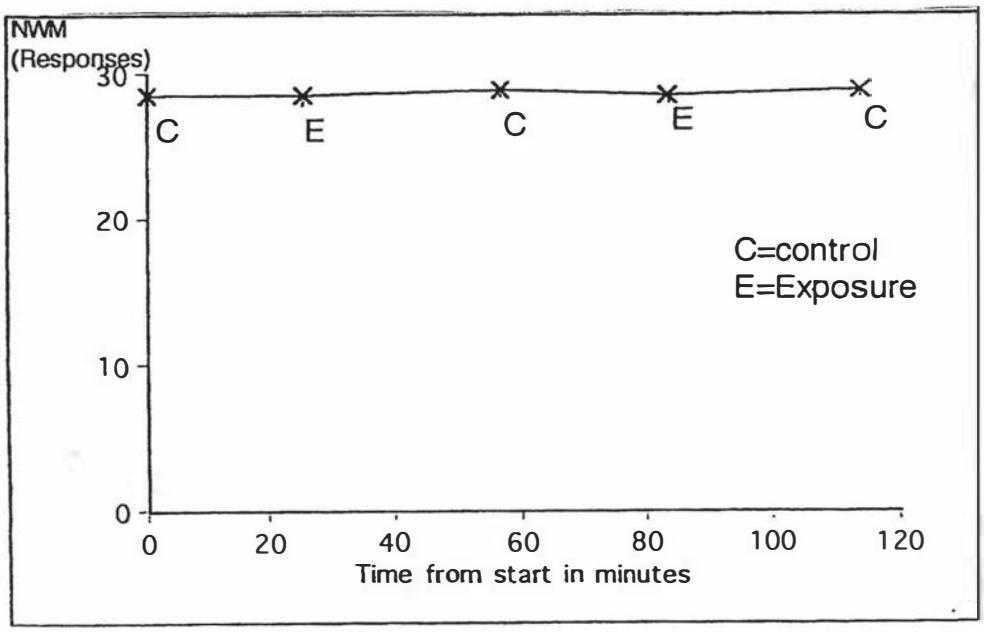


Figure 7.12 : Mean (+SEM) of digits correct over all subjects during the course of the experiment

Table 7.19: ANOVA logit analysis of the effects of cell phone use on working memory

source	df	Mean Square	F	Sig
field	1.000	0.138	4.544	.034
Error	170	0.030		

Estimated Marginal Means (all logit scales)

Field	Mean	SE	95% CI
Off	1.211	0.015	1.180-1.241
On	1.159	0.019	1.122-1.196

Table: 7.18 THE EFFECT OF CELL PHONE USE ON NUMERICAL WORKING MEMORY

(Number correct)

	CONTROL							EXPOSURE					
	Pre-Expos.		Interval		Post-Expos.		Mean	Expos. 1		Expos. 2		Mean	Diff.
Subj.	Set 1	Set 2	Set 1	Set 2	Set 1	Set 2		Set 1	Set 2	Set 1	Set 2		
1	28	30	28	28	29	30	29	29	30	30	29	30	1
2	28	29	29	29	30	30	29	30	30	30	29	30	1
3	30	29	29	29	30	29	29	26	26	28	29	27	-2
4	30	28	28	30	27	29	29	28	26	30	29	28	-0
5	30	30	30	30	29	29	30	30	30	30	27	29	-0
6	29	30	29	29	29	30	29	30	27	29	29	29	-1
7	29	29	30	28	30	30	29	30	30	30	28	30	0
8	25	28	30	28	29	30	28	30	28	29	28	29	0
9	30	30	30	30	30	29	30	30	30	30	29	30	-0
10	29	25	24	26	28	29	27	26	30	30	30	29	2
11	30	30	30	29	28	29	29	30	29	30	28	29	-0
12	28	30	26	30	29	30	29	28	30	28	30	29	0
13	30	30	30	30	29	30	30	29	30	30	28	29	-1
14	29	30	30	28	30	29	29	29	28	30	29	29	-0
15	24	24	28	26	29	29	27	27	23	26	27	26	-1
16	29	30	30	29	28	30	29	30	30	30	30	30	1
17	29	28	30	26	28	30	28	28	22	27	27	26	-2
18	28	30	30	30	29	30	30	30	30	29	29	30	0
19	30	30	30	30	30	30	30	29	29	30	29	29	-1
20	29	30	30	29	30	29	30	28	30	30	29	29	-0
21	28	30	30	30	30	30	30	30	29	29	29	29	-0
22	30	29	29	30	28	30	29	30	25	30	29	28	-1
23	29	27	30	30	29	30	29	30	29	30	29	30	0
24	28	30	30	29	29	30	29	29	29	30	30	30	0
25	30	29	30	29	29	29	29	30	30	28	29	29	-0
26	30	30	30	30	30	29	30	30	30	30	29	30	-0
27	22	24	23	18	29	28	24	25	27	23	26	25	1
28	28	30	30	30	29	30	30	28	30	29	29	29	-0
29	28	27	29	30	30	30	29	30	30	29	27	29	0
30	30	30	28	30	29	30	30	30	30	30	29	30	0
31	28	30	28	30	28	29	29	28	26	30	30	28	-0
32	27	29	30	26	29	29	28	27	27	28	26	27	-1
33	29	30	28	30	30	30	30	30	29	29	29	29	-0
34	20	30	27	27	29	26	26	27	26	25	26	26	-0
35	30	28	30	30	29	30	30	29	30	29	30	30	0
36	29	25	30	29	29	30	29	30	30	29	29	30	1
37	30	29	30	29	29	30	30	28	29	28	27	28	-2
38	25	28	29	29	26	26	27	28	29	28	28	28	1
39	27	29	29	29	27	30	28	27	26	29	27	27	-1
40	29	30	30	30	30	30	30	30	30	30	28	30	-0
41	29	30	29	30	30	30	30	30	30	24	29	28	-1

Table 7.18 contd													
42	28	28	28	30	29	30	29	27	30	29	30	29	0
43	29	24	29	28	29	29	28	29	26	29	27	28	-0
MEAN	28	29	29	29	29	29	29	29	28	29	28	29	-0

DISCUSSION

Aural Temperature

The temperature profile showed the expected decline over the evening but this was noticeably reversed at the times the cell phone was operating (Figures 7.4, 7.5).

The use of the cellular phone produced a significant rise (mean increase of 0.1 °C) in aural temperature in the ear to which the phone was attached. The opposite ear was also significantly warmer (mean increase 0.3 °C) when the phone was transmitting which suggested the temperature rise may not be simply local.

It was not possible in the present study, to determine whether the rise in temperature in the right (phone) ear was caused by conduction from heating due to the electrical internal resistance of the cell phone or as a consequence of the EMF it produced. A better design may have been to place something between the subject and the phone, such as a leather case as Koivisto (2001) had done.

In the left ear, the temperature rise was less likely to be due to conduction. If it was caused by the RF field acting on the probe then that field would have had to pass right through the brain to get to the probe in the left auditory canal. This is contrary to the generally held stance that RF fields do not penetrate any depth. Although Bernardi et al.(2000) reported temperature increases in the brain in a model head exposed to different cellular phones.

Subsequent research has suggested that aural temperature may be effected by by the temperature of venous blood returning from the scalp and face (Jessen, 2001). If so, the readings may not accurately reflect core temperature but may be effected by localised heating produced by the phone heating up surface tissue.

Surveys and anecdotal reports have suggested heating of the tissues surrounding a cellular phone in use. In 1998, Hansson-Mild described a dose - dependent increase in heat felt in and behind the ear during cell phone use. No laboratory studies have apparently been published on the effects of cellular phone frequencies on aural temperature in humans. However, Paredi et al.

(2001) reported a significant increase (2.3 degrees Celsius) in the skin temperature in the nostril and parietal regions on the side a GSM was operating. This was higher than the 0.3 °C found in the present study, which is to be expected as their exposure time was 30 minutes, double that of the present study. Paredi et al. found no temperature difference in the non-phone ear, whereas the present study found a significant increase in temperature, with a mean increase of 0.3 °C. Conversely, the results do not support those of Koivisto (2001) who failed to find an increase in skin temperature. However, in that study the phone used was in a leather case to reduce heat transfer and had the speaker removed and discontinuous transmission inactivated. The rise in temperature found in the present study could be due to heating as a consequence of electrical resistance or it may be hypothesised that it could be in some way initiated by the discontinuous transmission normally present in a GSM phone. The Koivisto study may not have found a rise in temperature because this feature, normally present in a cellular phone, was deactivated.

The temperature difference in the phone-ear was commensurate with that reported by Bernardi et al. (2000) who found a maximum of 0.08-0.19 °C in the ear of a model head exposed to several models of cellular phone. However, the temperature increase in the non-phone ear was twice as large as predicted by the Bernardi modelling. The largest temperature difference they found was in the ear. They concluded that the rise was way below that required to produce thermal damage. However, a lack of thermal damage does not equate with a lack of biological effects.

The difference between exposure and control sessions could also be due to the RF interfering with the thermoscan. However, in practice trials the thermoscan beeped if it was encountering interference from the phone. Trials indicated the readings were accurate if it wasn't beeping. It didn't beep at any stage when on the non-phone side.

When the phone was not transmitting the phone ear was still significantly warmer than the non-phone ear but the actual difference was only 0.1 °C, which is the accuracy level of the thermoscan. This increase was not due to the difference between the thermal insulation properties of the cell phone compared to the placebo cover used on the other ear as the difference in temperature between ears was not present in the first control session. It only occurred following the first exposure to the cell phone transmission. Thus it

was produced by the cell phone transmission. This could indicate a carry over in heating from the exposure sessions into the subsequent control sessions which could conceivably affect the other parameters tested if effects found were due to a heating effect. It may also be why the mean difference was higher in the non-phone ear. This could suggest the time between the exposure session and the following control (30 minutes) may have been too short. However, the mean temperature in the three control sessions followed the expected steady decline over the course of the evening. If there was a carry over into the following control session it would be expected that this decrease would not occur.

The findings of the present study quantify the increase in aural temperature consequent to a fairly normal, extended cellular phone conversation. It should be noted that, although the increases were statistically significant the actual amount of the rise was less than the normal physiological change in temperature during an evening. Both ears showed a steady decline of 0.4 °C over the two and a half hours of the experiment. By contrast, the rise in the left ear was 0.1 °C and the right ear 0.3 °C. The finding of a temperature increase in the non-phone ear was interesting and needs further exploration.

Cardiovascular Effects

Blood pressure normally declines in the evening. The use of the cellular phone did produce a temporary halt in the expected evening fall in systolic blood pressure at the times the phone was operating but this did not reach statistical significance. This temporary halt may have been due to the fact that the subjects knew the phone was on, and hence may be the result of a slight anxiety effect. However, if this were the case it would be expected that the effect would also be present in the heart rate data. The pulse pressure showed a steady decline until the last control when it rose. The reason for this rise is not clear but, it was only 0.8 mm Hg, which is within the SEM (1.9 mg) so is not notable. There was no effect on heart rate, which demonstrated the expected steady fall over the evening.

These results do not suggest cardiovascular effects occur from cellular phone use. However, the statistical power level was such that very small effects would be unlikely to be detected. Analysis of the results suggests a sample of 73 for pulse pressure, 219 for systolic pressure and 1137 for heart rate would

have been required to detect an effect using t=2, p=0.05. This and the marginal effect found at 43 subjects, suggests that, for blood pressure, a repeat study using a larger sample may have produced different results. Alternatively, an exposure time of 15 minutes may have been too short. However, longer phone calls are not usual, so extending exposure times may not be valid. There is also no evidence to suggest any effect may be dose-dependent.

These result did not support the findings of Braune et al., (1998) who found an increase in capillary perfusion and blood pressure consequent to exposure in 10 males exposed to a GSM digital phone for 35 minutes. A follow up study in 2002, using a larger number of subjects (N=40) and a longer exposure time (50 minutes), did not confirm the result. They concluded that earlier effects were due to environmental factors, rather than the EMF. Huber et al. (2003) found a reduction in heart rate following a 30 minute daytime exposure, but not an 8 hour overnight exposure. The studies were small, using 24 and 16 subjects, respectively. It may be that a short exposure results in effects but a longer exposure time allows for some sort of compensatory mechanism to arise. Until more studies are done such comments are purely spectulatory. In the study by Huber, subjects were supine and readings were taken during sleep or rest. In the present study, subjects were seated and undertaking adjacent cognitive tasks, a protocol that more accurately mirrors the normal situation accompanying cell phone use. However, it must be acknowledged that this may cause problems with regard to the control of confounding variables.

In the present study, the profile showed a steady decline in heart rate and blood pressure over the evening which suggested the experimental protocol taken to limit the effects of confounders was sound. However, it must be noted that the design, which aimed to reduce the effect of intra-subject variability, may have reduced the ability to pick up a small effect. Taking a number of readings then averaging them underestimates the variability. Analysis of the full data (Appendix 3.3, 3.4) suggests this does not appear to have effected results in this research.

There have been too few studies done to reach a conclusion with regards to possible RF EMF effects on the cardiovascular system, but results from the current study suggest possible effects on systolic blood pressure may become apparent with a larger sample. The currently published studies used small samples, one as few as ten subjects. More research is needed on humans using

a larger number of subjects, to pick up small, acute effects. Research is also lacking on the chronic cardiovascular effects of cellular phone usage. Also, most studies, including the present one, used healthy young subjects. This has provided little knowledge about susceptible populations, such as the very young, very old, or those with cardiac problems.

Salivary Melatonin Levels

Salivary Melatonin levels were unaffected by the short term use of a GSM cellular phone. This result supported findings reported by Mann et al. (1998), de Seze et al. (1998) and Radon (2001) in human studies and Vollrath et al. (1997) who studied the effects of 900 MHz EMFs on rats. The studies by Radon and Mann differed from the current study in that both used antennae located a distance from the subject to negate a possible heating effect (10 cm behind a seated subject, and 40 cm from the vertex of a supine subject, respectively). Such a placing of the antennae would result in a different signal intensity than that experienced by a person using a cellular phone placed against the ear. The experimenters also used a shielded chamber which prevented the reflection of signals that commonly occur and thus the conditions did not model normal cell phone use. The present study more closely reflected normal cellphone usage and had a larger number of subjects. However, the study may not have been large enough. Analysis of the results suggested a sample of 67 subjects was required to reach a t-value of 2 at p=0.05. Although the study had to be truncated at 43 , it was still the largest study so far.

In the present study, there was no field effect found in the subgroup identified as 'naturally low excretors'. However, there were only 13 subjects in the group. This may be too small to detect an effect so the possibility of a type 2 error is high. This is the first study to assess this in RF EMFs. In power frequency studies, two groups (Graham et al., 1996 and Crasson et al, 2001) had reported suppressed melatonin levels on exposure to EMFs in subjects with naturally low melatonin levels.

Analysis of the results by gender produced an interesting result. Females had been excluded from prior published research on the basis that variations in melatonin with changing phases of the menstrual cycle may add a confounding variable. In the present study, there was no significant difference between control and exposure sessions for the female subjects but there was a significant decline in melatonin levels in the males. However, there were only 16 males in

the study, compared with 27 females. Females showed almost no difference between control and exposure sessions (mean difference= -0.50 pg/ml) whereas male subjects had a -1.77 pg/ml mean difference. The results suggest there is no reason to exclude females from being subjects. This was also the conclusion for the 50 Hz studies in experiments one and two. The significant difference for males was probably due to one higher score distorting the results. A similar score occurred in the females but the effect was reduced by the larger number of subjects (Appendix 3.5).

Four subjects had zero or close to zero levels of melatonin. A prior screening would have seen these subjects eliminated from the study but would have increased the cost. Inclusion of the subjects did not alter the results. As explained in experiment two, such results are not uncommon and studies often report excluding subjects in the pre-screening selection on the basis of non-standard profiles, or undetectable melatonin levels. For example, Radon et al. (2001) exposed 8 males to a 900 MHz signal pulsed at 217 Hz. They observed the effects on salivary melatonin levels. Subjects were preselected on the basis of having melatonin rhythms that showed the least intra-individual variation. This may help in removing possible confounders but may also select out the very subjects who may be most likely to show an effect as they are likely to be the most sensitive to environmental stimuli affecting melatonin.

An exposure time of 15 minutes may not be long enough to effect melatonin levels. Lewy et al. (1980) found exposure to bright light caused a suppression of melatonin well within 30 minutes. If an EMF effect used the same mechanism then it could take longer than the 15 minutes used in the present study.

The present study measured melatonin levels in saliva. The concentration of melatonin in saliva is around 30% that of serum. Thus a study using this method may not be as sensitive to small changes in melatonin levels as a study using serum. Radon (2001) also measured melatonin levels in saliva. Using a specially designed shielded chamber , in seated subjects, the melatonin levels reported were very similar to the present study, rising from 12 pg/ml at 2200 hours to about 28 pg/ml at midnight.

Stevens (1987) proposed a melatonin hypothesis in which an EMF-induced suppression of melatonin was linked to an increase in rates of breast cancer.

Research on humans, including the present study, has not supported this hypothesis. However, the number of studies published is very small and all looked at acute effects. Research is required on the possible long term effects of cellular phone use on melatonin. Digital phones have been in common use for only a short period of time. Most of the subjects used in this study had little prior exposure to cellular phone frequencies. It should now be possible for studies to begin looking for effects from longer term use.

Cognitive Parameters

Attention

The use of a digital cellular phone produced a significant drop in levels of attention. The result was due to an increase in time taken, rather than an increase in errors as the mean number of errors was 1.4 per subject for both the exposure and control conditions. This could have implications for activities such as the use of cellular phones whilst driving or operating machinery. However, research in this area is sparse and the study would need to be replicated before any firm conclusions could be drawn.

The only studies using a similar test in the literature were in the 50 Hz frequency range (Beale et al., 1997; Crasson et al., 1999). Both reported null results. No effect was found for this task in experiments one and two but the frequency was different and the exposure time was shorter as the task was done at the commencement of exposure. In experiment 3, the test was done at the end of the experiment after 15 minutes of exposure.

Attention was the only parameter, apart from temperature, to produce a strongly significant result. It is interesting to note that the pattern of the output of the phone during this task, was different to that for other cognitive tasks as this task was done in silence (Figures 7.2, 7.3). The difference in wave pattern may be significant, in that the brain may be more susceptible to that particular wave pattern, or it may be a coincidence. The statistically significant result for attention may need to be treated with caution as it would be expected to get 1 in 20 results sign by chance at $p=0.05$. However, it would still be significant if a Bonferroni correction was applied. As the cognitive parameters and physiological measurements are likely to be independent of each other it is debateable whether this correction is necessary.

The results found in the present study could be due to heating effects as the phone heats up considerably in use. It could also be a result of direct simulation by the EMF of the orbito-frontal areas, inferior parietal cortex or medial temporal cortex. A further study could be designed to answer this question with the cell phone replaced during the control sessions with an identical case containing a heating system that did not require an EMF. If the difference still remained then it could be reasonably assumed that the EMF was not the cause of the decline in attention and it was simply due to the application of heat to the head.

Memory

Delayed Word Recall

The use of the cellular phone did not have an acute affect on delayed word recall. This was contrary to findings in studies using 50 Hz EMFs, however, the possible mechanism for interactions with living systems is thought to be different for RF fields. These findings are consistent with Preece et al. (1999) who, using a 915 MHz RF EMF, failed to find an effect in a delayed word recall task. However, that study used a simulated exposure rather than a real phone.

The scores in this task were lower than expected, considering university students were the subjects. Even for the first control the mean score was only 4.6 words recalled. This is much lower than the 10-12 expected. As the first control was low it can't be a field effect carrying over to the other sessions. It may be due to the number of tasks that were done before the words were asked to be recalled but it is common to do several other tests before recalling the words when this task is used to assess brain injury in a clinical setting.

More research could be done using a larger number of subjects to investigate possible small effects. However, analysis of the mean and standard deviation in the present study suggests 400-500 subjects would be required for a t- value to reach 2 at p=0.05. So, it is likely that there is no effect from the exposure on delayed word recall. But, as the phone was attached to the right side of the head, a study with the phone on the left side may produce different results as the language centres are usually located in the left hemisphere. Possible chronic effects on memory have yet to be investigated.

Digit Span Working Memory

A marginal drop in recall was found when the phone was transmitting but this did not reach statistical significance. It could be that the effect was small and a larger sample may yield different results. This result was contrary to the effect found by Preece et al. (1998) for an identical task on exposure to a 50 Hz EMF. It did, however, concur with their findings when the same task was carried out under exposure to a 915 MHz simulated cellular phone signal. Their exposure time was 25- 30 minutes which was longer than the 15 minutes used in the present study. However, the phone differed from a normal cell phone. In their simulation of an analogue and a digital phone they used a power output that was constant. In a real phone the power output varies. It has been demonstrated in 50 Hz studies that it is the presence of variability that has produced effects (Lyskov, 1993). This has also been identified as a factor in RF EMFs.

Other similar tasks have been used in studies on RF effects with varying results reported. Kovisto et al. (June, 2000b) found accuracy of recall was uneffected but speed of recall was reduced in the harder task in a recall of numbers task. They found no effect for easier tasks so, as with the 50 Hz studies, effects appear to be occur when the subject is working close to capacity. Speed of recall was not assessed in the present study, so it is not known if this effect was also present. A study published subsequent to the present research (Edelstyn and Oldershaw, 2002) also reported a significant facilitation of performance. The tests they used (digit span forwards and spatial span backwards) differed from the test used in this study but both required the recall of digits. Exposure was for 30 minutes, with 38 subjects tested. Testing was carried out after exposure had occurred rather than during exposure. They reported effects for some tasks e.g. digits forwards, whilst other similar tasks, e.g. recall of digits in reverse order showed no effects. This may be because these tasks require different parts of the brain. The recall of digits in reverse order requires the subjects to remember the last number in the series and is a task of divided attention as well as memory. It may be that such a task requires storage into deeper areas of the brain, such as the temporal stem, which may be deeper than the RF field can penetrate.

Digit recall has been associated with other effects under RF exposure. Krause et al. (2000 a, 2000b) reported EEG changes when the subjects were undertaking a digit recall task. Eulitz et al.(1998) also reported EEG changes during an auditory discrimination task.

There have only been a small number of studies carried out on RF effects on cognitive performance. The present study is the only one that has replicated a task previously reported in RF research and although the phone characteristics were somewhat different the outcome was the same as in the original study (Preece et al., 1999).

Summary

The finding, in this study, of a significant reduction in attention has implications for the use of cellular phones while driving. This result needs replicating by other research teams. The use of the cell phone did not appear to effect verbal memory but the marginal drop in numerical working memory requires further investigation. It is interesting to note that the one significant result took place when the person was silent and the output from the phone was different to that in the rest of the cognitive tests, which all required talking. This may be a coincidence or it may be that the brain is more susceptible to this wave pattern.

Heart rate appeared to be uneffected by exposure to a 900 MHz digital signal. Melatonin levels were also uneffected and this result was consistent with the few other studies that have been done. The present study provided information on gender effects and RF effects on 'low excretors' in the assessment of melatonin, that had previously been unavailable in the literature. The practice of confining subjects to males only appears unwarranted. The pause in the expected decline in blood pressure needs further exploration with a larger number of subjects (200-300). The number of subjects used in the study, while greater than most reported in the literature, was still too small to have a reasonable chance of picking up a small effect for many parameters, when the means and standard deviations were analysed. Thus it can only be said that if an effect exists it must be small and further research, using a greater number of subjects is necessary. It should also be stated that a percentage of the population may be susceptible to effects, a situation which would not show up in the form of analysis used in this study and prevalent in the literature.

CHAPTER EIGHT
FINAL DISCUSSION AND CONCLUSIONS

POWER FREQUENCY EMFS

Melatonin

Epidemiological studies have suggested a link between exposure to 50 Hz EMFs and decreased nightly melatonin levels (Wilson et al.,1990; Burch et al, 1998, Juutilainen et al, 2000). Laboratory studies have generally failed to support these findings (Selmaoui et al, 1996; 2000; Hong et al., 2001). A few studies have reported a decrease in melatonin levels but only in those subjects with naturally low levels (Graham et al, 1996; Crasson et al, 2001). This effect not been able to be replicated. An attempt at replication by Graham et al. (1996, 1997) failed, however in that replication, the field conditions were altered from an intermittent to a continuous exposure. Two studies reported a reduction in the nightly melatonin rise following daytime exposure using a square wave form (Wood et al, 1998; Karasek et al, 1998). Wood et al. also used a sine wave in one study. These were the first studies on humans reporting a delayed effect.

The present study found no effects on melatonin levels from an acute exposure to a 50 Hz, 100μT EMF with a one second pulse. The results were consistent between the initial and replication study. The results were the same for daytime and night time exposure so no circadian effects were noted. At 50 subjects experiment two was the largest study so far. The significant result found during the day in experiment two was considered to be an aberration due to the very low levels of melatonin that were close to the sensitivity level of the assay. The levels of melatonin found were consistent with most reported in the literature, so it is unlikely that confounding variables, such as light levels, effected the result.

The current study could find no significant effect on the evening melatonin rise from a 30 minute evening exposure to a 50 Hz, 100μT_{rms} EMF. This was contrary to findings by Karasek et al., 1998 and Wood et al., 1998. However, the flux density and waveforms were different to that used in the present study. Wood et al. used both square and sine waves at 200 μT , 50 Hz and concluded the effect was greater with a square wave. Karasek et al. used 2.9 mT, 40 Hz square wave, 20 minutes per

day, 5 days per week for 3 weeks. It appears a square wave elicits a greater field effect.

The current study is the first that has looked for possible gender effects in response to EMF exposure. Previously, subjects had been confined to males. There was no significant differences between males and females in either experiment. On the basis of these results there is no evidence for the exclusion of females as subjects.

Those subjects with naturally low levels of melatonin were uneffected in the present study. Using a higher field intensity did not extend the results found by Graham et al. (1996) to a greater proportion of the subjects. However, the exposure times in the current study were much shorter. Exposure in the study by Graham et al. was overnight. Their field was alternated one hour on/off and pulsed 15 seconds on/off during the on phase.

Results from the present study and those currently in the literature do not support Steven's (1987) melatonin hypothesis. Exposure to power frequency fields do not appear to suppress melatonin levels in laboratory studies. Consequently, it could be hypothesised that if field effects do exist, they must be either small, or exist in small section of the population. However, the effects reported from occupational exposures could reflect the effects of chronic exposure that acute laboratory studies are unable to detect.

The variability of the results reported in the literature may reflect the difficulty in providing controlled conditions in humans whose melatonin patterns are effected by their normal daily tasks and environment prior to going to the laboratory. Research on humans using longer exposure periods needs to be carried out but this will be a difficult task logistically. As more studies are published covering a wide range of flux densities, exposure times and pulse patterns, a frequency/intensity window of susceptibility may emerge. The current body of research has covered only a small number of the possible permutations. Consequently it is not prudent to make definitive statements as to whether melatonin levels are effected by exposure to power frequency EMFs. More research is required, especially replications of significant findings using the same fields characteristics.

Cognitive Effects

The most common parameter tested has been reaction time, often with a cognitive variable, such as attention or memory, as a component. Some studies have reported significant effects (Graham et al., 1994; Preece et a l., 1998). Others have not (Gamberale, 1989). Significant results have been reported for attention and memory (Trimmel et al, 1998; Preece et al., 1998; Keetley et al, 2001). However, the research base is still very small.

Attention

In the present study there was no EMF-related effect found for the attention task but the task was too easy, with most subjects getting close to full marks. Consequently, it is difficult to draw reliable conclusions from this study. It would have been better to have given a time for the task which wasn't long enough for subjects to finish, then compare scores and errors made. As the task was done at the beginning of the session, the exposure time was also short (5 minutes) so the task would have been better done at the end of the session. However, Whittington et al. (1996) had found an effect on a reaction time/ forced choice task with an exposure of only 9 minutes using similar field parameters. An almost identical attention task was used by Beale et al. (1997) in an epidemiological study. Crasson et al. (1999) also used the task in a 30 minute daytime exposure using the same frequency and intensity as the present study, but a longer pulse rate. Neither study reported an effect. The inclusion of a circadian factor in the present study did not alter results. Consequently, the type of attention and visual scanning measured in this task does not appear to be effected by a 50 Hz EMF but there have been only three studies done, using different exposure parameters. More duplication of this test is required before conclusions can be drawn.

Aural Working memory

The presence of a 50 Hz, 100 μT_{rms} EMF had no significant effect on aural working memory either at midday or at midnight. However, the harder level task (4 letter group) did show a greater difference between control and exposure sessions than the easier tasks. Whittington et al. (1996) found a significant difference on a visual forced choice task for the hardest level of task only. They used similar exposure parameters to the present study and an exposure time of only 9 minutes. However, they used 100 subjects. Analysis of the mean and

standard deviation in the present study, suggests a subject number between 110 and 140 would have been required to reach a t-score of 2. So, although larger than most in the literature, the sample was not large enough. The use of subjects as their own controls did help counter the individual variability that occurs in this type of task, however, any effect size must be fairly small.

It is difficult comparing results across studies as most have used different tasks which may assess different brain areas and processes. While it is important to broaden the research base it is also important to replicate significant results using the same tests. Currently there are too few studies on possible cognitive effects of EMF exposure to make definitive statements but all the reported studies have found significant effects in some of the tests they have used. However, it must be noted that in some cases multiple tests have been carried out on parameters that may be inter-dependant without the use of the Bonferoni correction, so some significant results can be expected by chance.

Possible Reasons for the Inconsistency in Findings

In reviewing the literature the most salient point that emerges is the lack of consistency in effects shown. Repetitions from the same research team, using a similar protocol in the same facility, may produce significant effects then fail to replicate at a follow-up study. There are a large number of variables to consider when setting up an EMF experiment. Changing even one factor, such as whether the field is pulsed or continuous, appears to effect the result.

EMFs have a range of field characteristics that could be important in determining an effect on melatonin levels and cognitive parameters. Such characteristics include; frequency, flux density, extraneous fields, wave form (sinusoidal, square wave) continuous or pulsed delivery, variation in pulse rate, type of polarisation, the timing of the pulse, field direction, length of exposure, time of day of exposure, order effect, extraneous EMFs and individual sensitivity.

In addition to the above are the large number of parameters tested, which may be differentially sensitive to the effects of EMFs, and the necessity to control confounders particular to each parameter.

Frequency-Intensity windows

A dose-response relationship is usually required to demonstrate causality in toxicology research. While a small number of studies have provided limited evidence of this effect, most do not. It may be that a dose response model is not appropriate in EMF research. It may be that some biological effects are 'windowed' Morgan and Nair (1992).

Graham et al (1994) provided some evidence of this. They noted that the relationship between field strength and response is not linear and that a field strength producing changes in one variable may not produce an effect in another variable. They concluded there appear to be 'windows' of intensity which effect responses to particular variables (e.g. heart rate). For example, Graham et al (1994) matched three groups of 18 men and exposed them to 60 Hz fields at low (6 kV/m, 10μT), medium (9 kV/m, 20 μT) or high intensities (12 kV/m, 30 μT). Significant slowing of heart rate and EEG changes were found only in the medium intensity group. Significant reductions in reaction time and accuracy in a performance on a time estimation task were found only in the low intensity group. It appears different parameters are differentially sensitive to particular field intensities.

Waveform

Wave form appears to be an important factor. Greater effects have been reported using a square wave rather than a sine wave. Wood (et al., 1998) reported a delay in the nocturnal rise in melatonin with a square wave field (20 μT, 50 Hz) but also reported a smaller effect from a sine wave. The significant depression in the melatonin rise reported by Karasek et al. (1998) was following a chronic, daytime exposure using a 2.9 mT, 40 Hz, square wave, bipolar field. He used 12 male subjects with exposures of 20 minutes per day, 5 days per week for three weeks. Results using square waves do not have the same implications for the public as exposure to this wave form is not as common as a sine wave form.
In studies on cognitive effects significant results have been reported for sine waves. No studies appear to have used square waves.

Polarisation

Studies using circular polarisation have reported effects more often than those

using linearly polarised EMFs for melatonin (e.g. Graham et al. 1996). For cognitive effects significant results have been reported using both circularly polarised fields (Keetley et al., 2001) and linearly polarised fields (Preece et al. 1998).

Pulsed or intermittent vs continuous EMFs

Studies using pulsed EMFs more commonly report effects than those using continuous EMFs (e.g. Lyskov et al., 1993; Crasson et al., 1999; Podd eta l., 2002.) In one study, a depression in melatonin levels in 'low excretors' was found using an intermittent EMF but in a replication using a continuous EMF the effect did not reoccur (Graham et al., 1996; 1997).

Reflections and Transients

It could be that the lack of ability to reproduce epidemiological results in the lab is not due to the presence of confounding variables in the parameters tested in the environment, but the fact that lab studies do not reflect real life exposure parameters of the EMF. Most laboratories use shielded rooms to remove the possibility of reflections. It may be that the removal of reflections and transients from the lab exposure protocol, with the aim of producing a controlled EMF exposure, may be removing the very factors that are responsible for the interaction between EMFs and living systems. In the present study, while the EMF was controlled, the possibility for reflection in the environment existed. The possibility for the EMF to vary in intensity, however, was not present.

Time of day of exposure

At the time the present studies were carried out there were no studies looking at the effects of EMFs on melatonin levels in the day time. This may have been due to the expected low levels during the day making assay difficult. Although a significant suppression effect was found on melatonin levels in experiment two, this only occurred using the same day control. Levels of melatonin were close to the sensitivity levels of the assay so the result can not be relied upon. Two studies have been carried out on the effects of daytime exposure on night levels of melatonin (Karasek et al, 1998; Wood et al., 1998) and both reported delayed effects on the evening melatonin rise. Crasson et al. (2001) found a smaller increase in night time urinary aMT6s after intermittent afternoon exposure.

Studies on the effects of EMF on cognitive parameters have not previously included a circadian factor. The present study appears to be the first to do so. There were no differences found between day and night sessions in response to the EMF. It was thought that attention may be more susceptible to disturbance at night but this was not the case. In fact, in experiment two a small non significant effect was found in the day time. Although the field intensity was different in the present study to that used by Karasek et al. and Wood et al., their findings raises the question as to whether there may have been effects on the night sessions from exposure 12 hours previously in the present studies. Fifty percent of the subjects would have had a night session following a daytime exposure. It is possible this could have effected night time levels.

Length of Exposure

Exposure time has also been variable between studies. However, a longer exposure does not correlate with greater effects. Studies as short as nine minutes have reported effects (e.g. Whittington et al., 1996), while longer ones didn't .

Body fluid tested for melatonin levels

Although the use of saliva samples to test for melatonin has been well validated (Nowak et al. 1987) and has been used by other studies (Stark et al., 1997; Radon et al., 2001) it is acknowledged it is a less sensitive measure than using blood testing. Testing for the urinary metabolite provides even greater sensitivity as collection covers a greater period of time. However, the gains in sensitivity must be measured against cost and the necessity for specially trained assistance. When using a large volunteer subject base the acceptability and practical issues relating to the collection of blood and urine samples made those options impractical in the present study.

Selection of Cognitive tests

There have only been a few studies carried out on possible EMF effects on cognition. Different studies have utilised different tests, each of which is sensitive to a different brain function. Consequently, the comparison of results is almost impossible. The current study used the attention test which had previously been used in two published studies for this reason. The results were the same as for the published studies. The EMF was the same as for Crasson et al (1999) except

Crasson's pulse rate was 15 seconds on/off instead of the 1 second on/off in the present study. The other study (Beale et a l., 1997) was an epidemiological study under high tension lines.

Control of Confounding variables

Measures taken to reduce confounders in the present study included; exclusion of subjects that engaged in activities likely to confound results, limitations on the pre-test activities, control of lighting and temperature, control of distractors such as noise, and the use of subjects as their own controls to control for intra subject variability. Subjects remained seated as changes in posture may have effected melatonin levels. Subjects were given time to adjust to the experimental conditions before testing and were given practice sessions to reduce practice effects. Sessions were in counterbalanced order and different versions of the tests were used for each session. These were alternated. However, the use of a counterbalanced order did mean it was difficult to look for order effects in the results as had been reported by Lyskov et al. (1993).

It was not possible to control humidity and in experiment one it was possible there were extraneous EMFs as the room was unshielded and located in a university building. In experiment two, control was easier as it was in a private residence and all appliances such as freezers (which were located in the room above the experiment) were turned off.

Establishing base level profiles and pre-screening of subjects

In studies on melatonin, the larger laboratories pre-screened subjects and eliminated them from the study if they had atypical profiles or had melatonin levels too low to be recorded. This was not done in the present study as it was too expensive. Consequently, it was noted there was a difference, in some subjects, between melatonin levels in the pre-session saliva samples between one night and the next for the same subject. This difference was generally not great and most subjects had fairly stable melatonin levels between one day and the next. A few subjects had levels that were markedly different between the two day or night control sessions. This may simply reflect the individual's normal pattern or may be due to confounding variables. As stated above, attempts were made to control confounding variables in the sessions, especially exposure to light. An

inconsistency between one control and the next at the same time of day could indicate that some of the subjects had not followed the pre-session advice with regards to limitation of exposure to confounding variables. The settling in time in a low light environment before each session should have been enough to counteract any light effects prior to arrival at the testing room but there may have been other confounders present but not admitted to by subjects. This is always a difficulty when working with human subjects.

Although it may have been better to remove those subjects with zero levels of melatonin from the analysis it would not have altered the result. It is debatable whether subjects with 'abnormal' profiles should be excluded from experiments as they may form a susceptible sub population. However, if included, subject numbers would have to be large enough to be able to distinguish a field effect from normal variations in melatonin levels. This would add considerably to the cost of studies, especially where highly controlled expensive facilities such as those at the Mid West Institute are used.

Subjects used as own controls

Crasson et al. (2001) stated "Inter-individual differences in pineal production of melatonin, however, have to be taken into account in further studies" p.234 Most studies have used the same subjects for both the exposure and control sessions but have not paired the data. This means some opposing changes among subjects could have cancelled each other out. The use of a paired analysis in the present study increased the sensitivity of the design. This is important when studying parameters such as melatonin and cognitive variables where intra-individual variation is likely to be larger than any EMF effects.

Number of Subjects

The number of subjects in the published laboratory studies on melatonin ranges from 9 to 33. That is smaller than the 50 subjects used in experiment two of the present study. Small subject numbers increase the possibility of a larger variability (or stress event, such as an exam) effecting the outcome of the research. The likelihood of a type 2 error (accepting the null hypothesis when it is incorrect) is increased when subject numbers are low. The present study reported the number of subjects required to reach $t=2$, when $p=0.05$ (from the mean and standard

deviation of each of the parameters), to aid the interpretation of results.

Double Blind

The use of a double blind design would have been desirable in the present study but was not possible with the equipment available. The compromise developed meant the subjects were unaware of the status of the field and the use of coding for result sheets and saliva sample tubes meant those assessing the results would have been blind to the field condition. The experimenter, however, was aware of the status of the field.

Order Effects

Graham et al. (1994) also found that the order subjects were exposed to, either sham or exposure first, effected whether there would be changes produced in heart rate but not in EEG or performance. Those exposed to the real field then the sham field showed greater effects than those exposed the other way round. Thus not only does field frequency and intensity effect results but also the order in which the subjects are sham or real field exposed. The possibility of order effect was acknowledged, but designed out of the present experiment by using a counterbalanced block design, as it was considered too difficult to include another experimental variable.

The timing of the control

The timing of the control appears to be critical. For example, in experiment two a significant result was obtained on the same data using same day control figures for the comparison but not when the different day control figures were used. In most studies the control was at least 24 hours from the exposure session. While this reduces possible carry over from the exposure session it increases the likelihood of confounding variables effecting results. This is especially so for cognitive variables that are likely to be more prone to variation due to external influences.

Statistical Power

Most studies used small samples, some less than 10 subjects. Whittington et al. (1996a) analysed 19 experiments using human subjects and concluded the chance of detecting a small effect was only 8%. It is no wonder that results appear inconsistent and that attempts at replication often fail. Some studies which fail to

replicate results conclude an effect does not exist. For example, Graham et al.(1997) determined that 40 subjects would provide a power greater than .8 at p=.05. but they estimated a 20-30% change in melatonin levels based on results from studies on rodents. It is likely that the extrapolation of data from rodent studies to humans is not valid due to the vastly differing size and hence potential for EMF interference on the pineal.

The Use of Animal Models

Wilson et al. (1999) postulated that parameters such as the neuroendocrine status of the animal or artificial changes made during the research may effect how an animal responds to an EMF. Animals that are seasonal breeders or that hibernate may be sensitive to EMF effects at some times but not at others. Studies frequently reported using animals with a circadian rhythm that had been artificially altered for the convenience of the researcher. Artificial light has also often been used. These factors may alter the sensitivity of the animal to EMFs or produce other factors that may confound the results.

Most research on rats has been carried out on adults. Reiter et al. (1988) replicated their research on sexually immature rats and found the reduction in nocturnal melatonin to be less than for adult rats. They used 10, 65 and 130 kV/m and the reduction was less in juveniles at all doses.

Effects of EMFs also appear to differ according to the sex of the animal. Wilson et al (1999) found a significant decrease in melatonin production in male Djungarian hamsters exposed to 60 Hz, 0.1 μT EMFs but not in the females identically exposed. As most studies use male animals this raises questions as to the ability to generalise from results, even within species.

Susceptibility to the effects of EMFs may also vary between species. Some rodents, e.g. the Syrian hamster, have melatonin rhythms that are uneffected by electromagnetic fields (Reuss and Olcese, 1986). Djungarian Hamsters use magnetic orientation to locate their nests so may be more sensitive to EMFs than other rodents (Wilson, 1999). However, the species is not uniformly susceptible to EMFs as Truong (et al., 1997) reported.

Sagan (1993) noted that in many of the studies published, exposure levels were

high enough for the animal to detect the electric field which could have been responsible for the responses reported. Modes of detection could include micro-shocks or vibration of body hair and would cause stress to the animal which would have consequences for the endocrine system, particularly during long term exposure. This also raises the question as to how valid such studies are as models for consequences for humans when field intensities used are many times above what humans are exposed to.

Results reported across studies have been inconsistent and the conclusion made by Reiter in 1993 is still valid. He stated that "In recent years, replication of the studies using sine wave electric fields have not confirmed their ability to inhibit nocturnal melatonin formation in adult rats"(1993. p.397). The same can be said of other animal species tested.

Although findings from animal studies have been inconsistent a greater proportion have produced significant effects on melatonin production than has been reported for studies on humans. It could be that the distance between the EMF source and the Pineal gland is too great for an effect to occur in humans. This could account for the disparity between results on human subjects and the reported suppression effects of EMFs on melatonin in rodents where the pineal is much closer to the surface of the skull. It may be that the use of animals as models is not a reliable tool for the assessment of effects on melatonin in humans.

CELLULAR PHONE FREQUENCY EMFS

Experiment three was designed as a pilot study to address some areas identified by the IEGMP (2000) as needing research. The group particularly identified a need for laboratory studies on humans as they noted that there are considerable anatomical and circadian pattern differences between animals and humans negating the effective use of animals as models. They further recommend the use of 'realistic exposure conditions relevant to mobile phone technology" IEGMP, 2000, p.8). The WHO specifically suggested the need to determine if low level RF exposure causes changes in melatonin synthesis (Repacholi, 1998, 1999).

Temperature Effects

Most of the research published has concentrated on the assessment of SAR, using models of the head. There have only been a few studies published on humans. In a large survey of 11,000 cell phone users in Norway and Sweden in 1998, Hansson-Mild described a dose -dependent increase in heat felt in and behind the ear during cell phone use. Surveys and anecdotal reports have suggested heating of the tissues surrounding the phone but little laboratory research has been done on humans.

In the present study, the use of a cellular phone significantly increased aural temperature on both sides of the head. It is not known whether this was due to conduction or the RF field. The increase was 0.1 °C on the phone side and 0.3 °C on the non-phone side. This is the first study quantifying the effects of cell phone use on aural temperature. The results of the current study are commensurate with those reported in a similar study by Paredi (2001) who found a significant increase in skin temperature (2.3±0.2 °C) over the nostril and occipital area on the same side as the phone. However, unlike Paredi et al., a significant increase in temperature was also found in the non phone ear. Conversely, the results do not support those of Koivisto (2000) who failed to find an increase in skin temperature. However, in that study the phone used was in a leather case to reduce heat transfer and had the speaker removed and discontinuous transmission inactivated. The finding of an increase in the non-phone ear in the present study was intriguing and requires further research. It could be a RF effect on the probe or due to conduction. However, conduction of heat from one side of the head to the other is less likely due to the circulatory pattern of the head. The thermoscan was effected by the

phone when it was very close to it. It beeped during those times. Trials indicated that if the phone was not beeping the readings were accurate. The thermoscan never beeped when readings were being taken on the non-phone side and had not given inaccurate readings during trials at that distance from the phone. For the thermoscan probe to be effected by the phone, the field would have had to pass through the skull, or brain, to reach the probe deep in the opposite ear. If the field could penetrate to do so then it is possible for an effect to occur. This is contrary to currently held beliefs about penetration depths of RF fields. Although studies using models of heads suggest such penetration is possible (Bernardi, 2000).

It should be noted that, although the temperature increases were statistically significant, the actual amount of the rise was less than the normal physiological change in temperature during an evening. Both ears showed a steady decline of 0.4 °C over the two and a half hours of the experiment. By contrast, the rise in the left ear was 0.1 °C and the right ear 0.3 °C.

It was not possible to determine whether the rise in temperature found in the present study extended to the brain. Techniques to enable the measurement of small changes in brain temperature need to be developed. It is difficult to measure depth of heat penetration in humans. The current study did not address this issue but the rise in temperature in the non-phone ear could suggest a general increase in head temperature, at least at the surface. If such an increase in head temperature resulted in even a small increase in hypothalamic temperature it could produce alterations in the level of secretory products of the endocrine glands. Such changes in hormone secretion have been reported as a consequence of small chemical or electrical stimuli (Mann et al., 1997). Models produced by Wainwright (2000) and Van Leeuwen et al. (1999) predicted brain temperature rises of 0.1 °C. Small changes in temperature in the hypothalamus and medulla cause large responses in all thermoregulatory effector mechanisms (Jessen, 2001). Temperature changes also effect blood-brain barrier permeability (Schirmacher et al, 2000).

Cardiovascular Effects

Results from Soviet sources in the 1960s and 1970s suggested chronic, long term exposure to radio frequency EMFs caused hypotension associated with

bradycardia or tachycardia (Jauchem, 1997). However, these early studies have been criticised as being poorly controlled (Jauchem, 1997; IEGMP, 2000). In 1993, the WHO concluded that RF EMFs do not cause cardiovascular effects at intensities below those which cause heating, unless biologically significant electric currents are present.

There have been very few laboratory studies on the effects of RF EMFs on humans. A recent study by Braune et al. (1998) reported a slight reduction in heart rate and an increase in blood pressure following a 35 minute exposure to a GSM digital phone in 10 subjects. However, this study has been criticised for faults in both design elements and statistical analysis. In a 2002 follow-up study, Braune et al. found no effect on blood pressure, using a larger sample of 40 subjects. They concluded, earlier effects were due to environmental factors, rather than the EMF. Subjects were tested standing, supine and during the valsalva manoeuvre. Huber et al (2003) reported a reduced heart rate following a 30 minute daytime exposure, but not an 8 hour overnight exposure. The studies were small, using 24 and 16 subjects. The finding of an effect for a shorter but not longer period may indicate some sort of compensatory mechanism arises. Until more studies are done such comments are purely spectulatory.

The present study represented a repeated, acute exposure in young subjects. A small non-significant effect was noted in systolic blood pressure. An analysis of the data suggested 73 subjects were needed to detect an effect so the present study was not quite large enough. Alternatively, at 15 minutes, the exposure time may have been too short. However, longer calling times are not a feature of normal phone use. A larger sample may have produced different results for the blood pressure study. There were no effects from the RF exposure on heart rate or pulse pressure. An analysis of the data suggested a larger sample would not have altered these results. Longer exposure times may have changed the results but results from the few published studies available do not support this view. The presence of a normal profile showing a steady decline in heart rate and blood pressure over the evening suggested the experimental protocol taken to limit the effects of confounders was sound. However, it must be noted that the design, which aimed to reduce the effect of intra-subject variability, may have reduced the ability to pick up a small effect.

Possible modes of interaction between RF EMFs and the cardiovascular system could be via a temperature rise in the brain effecting cardiac control centres, or by direct stimulation of these centres by the EMF. Such an interaction has been proposed by Sastre et al. (1998) in which power frequency fields have been linked to an increase in arrhythmia-related diseases among electricity workers after prolonged exposure. So, far laboratory studies have not provided the evidence to support such proposals.

There have been too few studies done to reach a conclusion with regards to possible RF EMF effects on the cardiovascular system. More research is needed on humans using a larger number of subjects, to pick up small, acute effects. Possible effects from chronic usage and the possibility of susceptible populations, such as the elderly or those with cardiovascular problems needs to be investigated.

Effects on Melatonin

Stevens (1987) proposed a melatonin hypothesis in which an EMF-induced suppression of melatonin was linked to an increase in rates of breast cancer. Similar statements have been made in relation to cell phone use and other cancers (Carlo et al., 2000). The few studies that have been published do not support the Stevens hypothesis. It could be that, in humans, the pineal gland is either not susceptible, or is too deep a structure to be effected. In some rodents the interaction between an EMF and melatonin suppression appears to occur via the retina. This pathway does not appear to be a viable mechanism for such an interaction in humans, although the placement of a cellular phone by the ear would certainly put the retina within the field produced by the antennae.

Results in the present study, the largest so far, confirmed those of Mann et al. 1998 that pulsed RF EMFs (900 MHZ, pulsed 217 Hz) have no effect on evening melatonin levels. Radon et al. (2001) found no effect from day and night exposure. In the longest study published, de Seze et al. (1999) exposed subjects to a 900 MHz or 1800 MHz commercially available phone for 2 hour per day, 5 days per week for 4 weeks. They reported no alteration in the melatonin profile. The WHO specifically suggested the need to determine if low level RF exposure causes changes in melatonin synthesis (Repacholi, 1998). The conclusion would appear to be that it doesn't but the number of published studies is still very small. A larger

number of studies would be needed before any definitive statement could be made. Such studies need to have a large subject base and exposure conditions that are relevant to actual phone use.

Of the three published studies on humans, two had the antennae removed some distance from the head (Mann et al, 1997; Radon et al., 2001). Such a placing of the antennae would result in a different signal intensity and pattern than that experienced by a person using a cellular phone placed against the ear. Mann et al. used a metal free chamber lined with absorbing materials that suppressed possible reflection of the RF waves. This was done to enhance reproducibility. However, signal reflection is a normal property of cell phone use which could conceivably be a causative factor in any possible interaction with living systems. The present study was carried out in a private residence with no close sources of RF interference. The normal possibilities for reflection of the signal were present, as the aim was to be as close as possible to normal conditions of use. There were, however, protocols in place to control for possible confounding variables in the parameters tested. These proved adequate as melatonin levels found were in line with those reported in the literature.

With the exception of the current study, all studies used young, healthy males. The current study provided information relating to females and is the only study thus far to do so. Results suggested that the practice of limiting subjects to males only may not be necessary.

The small number of studies so far published looked at acute effects although the study by de Seze (1998) covered a period of 4 months. There is a need for studies looking at the effects of chronic usage. None of the studies, including the present study, included possible risk populations such as the young or very old, or those with health problems. The inclusion of such subjects carries special ethical risks which would make ethical approval difficult to obtain. However, these may be the sections of the population at greatest risk from possible effects of exposure to RF EMFs.

Cognitive Effects

The main cognitive finding reported in the literature has been a shortening of reaction times, particularly associated with tasks that have an attention or working memory component (Hermann and Hossmann, 2003). There have been a small number of studies investigating effects on attention, memory and judgement. Tasks used have differed between studies, making the comparison of results difficult. For this reason, in the present studies, tasks chosen were those that had been used by other studies, although the shortage of RF studies meant the attention task was one used in studies on 50 Hz EMFs.

Attention

The use of a digital cellular phone produced a significant drop in levels of attention. The result was due to an increase in time taken, rather than an increase in errors. This result was not consistent with the other results in the study but attention is independent of the other parameters (although memory has an attention component). The aim was to use a task of attention that didn't contain a memory or judgement component.

The attention task was the same one that was used in the 50 Hz experiments. The effect was not apparent at 50 Hz but the method was slightly different. In experiments one and two, subjects were allowed to complete the sheet and the errors and time taken were assessed. In experiment three subjects got only one minute to do the task. This made data analysis easier as it meant subjects couldn't get close to full marks. The task was carried out at the beginning of the exposure in the 50 Hz experiments whereas in the cell phone experiment it was carried out at the end of 15 minutes of exposure. This meant the exposure time was longer in the cell phone experiment.

This was the only task done in silence. When the user was not talking the pattern of the output from the phone changed. The output was reduced, as seen by the number of spikes on an oscilloscope, but the amplitude of those spikes increased. The brain may be more susceptible to this pattern. It would be interesting to investigate whether tasks in other studies reporting significant results were done in silence. This would only apply to those using real cell phones rather than simulations that produced a steady output.

The same task has not been used in RF studies but was used by Beale et al. (1997) and Crasson et al. (1999) in 50 Hz studies. In both cases results were not significant. However, the postulated mechanism of interaction between RF fields and living systems differs from that proposed for power frequency EMFs. The finding of a significant decrease in attention with cell phone use has implications for activities such as the use of phones while driving.

Memory

The delayed word recall and digit span working memory tasks used in the present study were the same as those used by Preece et al (1998) and Keetley (2001) in studies on 50 Hz EMFs. They reported significant field effects. A subsequent study by Preece et al. (1999) on 900 MHz EMFs used the same tasks but failed to find significant effects.

In the present study, there were no field effects on delayed word recall but the scores were lower than expected in both the control and exposure sessions. It was not known why this occurred but it is likely to be a result of the number of tasks done in between receiving the list and recalling it. A less likely cause would be a field effect carrying over into the control sessions. As the phone was attached to the right side of the head, which doesn't contain the language centres, it is possible that different results may have been found if the phone was on the left side.

A marginal effect was found for the digital working memory task on the ANOVA (P=0.03). It would be worth doing a replication with a larger sample. As memory contained an attention component and a significant effect was obtained for attention, it may be this result was due to the attention part of memory task. Both tasks required attention to numbers rather than letters or words.

Studies on RF effects on other similar cognitive tasks have reported varying results. Kovisto et al. (June, 2000b) found accuracy of recall was uneffected but speed of recall was reduced in the harder task in a recall of numbers task. Edelstyn and Oldershaw (2002) reported a significant facilitation of performance in a digits forwards recall task, but a similar task (recall of digits in reverse order) showed no effects. Research into the effects of cell phone use on memory is just beginning and a lot more is required. Particularly replication of significant results.

Possible reasons for the inconsistency in findings in the Literature

As with the power frequency literature, there has been a variability in results reported. As the number of studies published on the parameters covered by the present study is very small this is not unexpected. Possible reasons for this variability are proposed below.

Selection of a Control

In some studies in the literature completely different subjects were used for the control and exposure sessions. This is a problem when testing cognitive parameters as it is very difficult to control for confounders, such as personality traits, with small subject numbers. In the present study it was recognised that the variability between people is quite large in terms of cardiovascular measures, melatonin levels and cognitive parameters. This was countered by using subjects as their own controls and blocking the experiment. The anova also took this into account statistically.

Double blind

The present study was not carried out double blind. A double blind design would have been harder to arrange with the cell phone as subjects can detect a phone in use. To use a remote antennae or to modify the system in other ways, as has been done in other studies, would alter the nature of the field to the point that it would be questionable as to whether results could then be related to those expected from normal cell phone usage. This type of modification has been criticised in the literature as being inadequate (Kuster and Schonborn, 2000).

Choice of cognitive task

Studies on cognition have covered different tasks and hence differing brain processes so it is difficult to make comparisons. With the possible mechanism of interaction unknown it is difficult to make rational choices about which area of human cognition to study (Podd et al., 2002). Consequently, there have been a large number of cognitive variables studied which, when added to the differences in EMF variables used, makes a very large number of potential variables. For trends to become apparent a much larger research base will be needed. It would be useful if there is replication done of tasks reporting effects and if successfully replicated studies could then extend into similar tasks to check the extent of such

an effect. The present study sought to do this by using tasks in which effects had already been reported (at least from the 50 Hz literature).

Control of Confounding Variables

It is difficult to control for the large number of possible confounders in psychological variables and still maintain a viable number of subjects in each category, or a sample size that is not too costly. Such confounders can include age, gender, health status, stress levels, personality factors, occupational factors and many others.

In the present study, measures taken to reduce confounders included; lack of close RF sources, exclusion of subjects that engaged in activities likely to confound results, limitations on the pre-test activities, control of lighting and temperature, control of distractors such as noise, taking of multiple readings for cardiac and temperature parameters to reduce variability, and use of subjects as their own controls to control for intra subject variability. In addition to this it was decided to do all the sessions in one evening as the subjects were university students and stress and cognitive loading can change considerably over the course of a day or two. To cope with the expected decline in attention and increasing fatigue over the evening, the control sessions were placed before, between and following the exposure sessions. This also controlled for the normal evening change in melatonin, temperature and cardiovascular parameters.

Exposure times

Cellular phone conversations are generally short. The present experiment used an exposure time of 15 minutes which, although considered short for some cognitive tasks, represents a longish phone call. Most studies used 30 or 60 minutes which would be rather long for a cell phone call. Shorter, repeated calls are more common.

Acute vs chronic exposure

Cellular phones have only been in common public use for a relatively short time. At the time the present study was carried out (2000-2001) few of the students owned one. This has changed dramatically in the past couple of years. It should now be possible to investigate the medium term effects of regular cell phone use.

Highly controlled vs realistic exposure scenarios

SAR varies greatly according to such factors as; the orientation between the source and the person, whether the person is moving, the frequency of the source, and the proximity of the source to the person.

Some studies have been criticised for using simulations that were deemed too different from a phone in use. The IEGMP particularly identified a need for laboratory studies on humans as they noted that there are considerable anatomical and circadian pattern differences between animals and humans negating the effective use of animals as models. They further recommend the use of "realistic exposure conditions relevant to mobile phone technology" (IEGMP, 2000, p.8).

Some studies have removed speakers or deactivated the discontinuous transmission (Koivisto, 2000; Haarala, 2003). In 50 Hz studies, it was the pulsing of the EMF that was more often associated with field effects. It is reasonable to suggest that it may be an intermittent signal that is more likely to produce an effect in RF exposure. The removal of variations in the normal signal, in favour of producing a standardised exposure, may be removing the very parameter that causes an effect. Studies have often tried to isolate a possible heating effect from an RF effect and so simulations used have differed from a normal phone in use.

The use of shielded rooms also prevent reflection of signals which would normally occur and may be a factor in any possible effect. In the study by Huber et al. (2003), subjects were in a shielded room and were supine and asleep or resting. Whilst this may be sound from the point of view of controlling confounding variables, it doesn't reflect the normal usage pattern and may remove factors such as reflection or head movement which may change the phone output and so may contribute to a potential effect. Results from studies that are very highly controlled have greater scientific value than less well controlled studies but if the exposure conditions vary greatly from the conditions that the research was designed to model, then it has very little validity or practical use. For example, the testing of subjects whilst supine and asleep may help reduce the effects of confounding variables in cardiovascular measurements but how valid is this when relating to normal phone use?

The aim of the present study was to meet the requirement for 'realistic exposure conditions' whilst exercising as much control as possible over confounding variables. The present study was carried out in a private residence so it was possible to control RF sources in the immediate proximity. A balance was sought between the need for control of the experimental conditions and the necessity for the results to be valid to normal usage. Subjects were seated and were carrying out cognitive tasks prior to and after readings were taken. Whilst subjects had to remain seated and hence fairly still, the exposure more closely reflected normal operating conditions for phone use.

The power will have varied with the state of the battery just as it does in normal use. However the battery was fully charged before each use and the time length of exposure for each subject was identical. Thus each subject would have been exposed to a similar power level for each similar session, e.g. subject one and subject 43 would each have been exposed to a similar power level in the second exposure session. The use of three control sessions, placed around the exposure sessions, compensated for the decline in power over the evening. That is an additional reason why results from the three exposure sessions were averaged and compared to the two exposure sessions.

The difference in profile of the cell phone transmission when talking and silent was interesting. The one significant result occurred during a period of silence when the profile was different to that for the other parameters. This change in profile would not be present in a simulation of a cell phone where a steady output is used. If the significant result was related to this different profile then it would not be found in a simulation study.

Use of subjects as their own controls

The use of subjects as their own controls was an important feature which increased the sensitivity of the design in the present study. This was particularly important with the parameters tested as the inter-individual variation in melatonin concentrations, cognitive measures and cardiovascular measurements was likely to be greater than those which could be expected from a small field effect.

Small sample sizes and statistical Power

Statistical power is the probability of detecting an effect if one exists (Lipsey, 1990). To date, research into the effects of electromagnetic fields have produced results that are variable and many attempts at replication have failed. Some have interpreted the inconsistency of results as an indication that EMFs have little or no effect on biological systems. However, the inconsistency of results and failure to replicate may be a consequence of a lack of sensitivity in experimental design. In particular, most studies have used small sample sizes. As effects from electromagnetic fields are likely to be small in magnitude, a small sample size leads to low statistical power and therefore very little chance of finding a significant result even where a field effect exists. The main way of increasing statistical power is to increase sample size. Sample sizes in the literature surveyed range between 10 and 64 with most in the 30-40 range. While this is enough to detect a medium effect it is too small a number to detect a small effect. Therefore some studies are concluding there are no field effects when the sample is too small to detect one.
In the present study, the parameters studied appeared to fall into three categories; those for which there is clearly an effect (aural temperature and attention), those for which more subjects could alter the null results (melatonin and blood pressure) and those that would require a very large sample to get a result or basically there is no effect (heart rate, delayed word recall).

Future Research Needs

The IEGMP (International Expert Group on Mobile Phones)(2000) identified the following areas of research need; effects on brain function; consequences of exposure to pulsed signals; psychological and sociological studies; possible health effects of cellular and sub cellular changes; possible mechanisms of interaction with living tissue; epidemiological and human volunteer studies. The group particularly identified a need for laboratory studies on humans as they noted that there are considerable anatomical and circadian pattern differences between animals and humans negating the effective use of animals as models. They further recommend the use of 'realistic exposure conditions relevant to mobile phone technology" IEGMP, 2000, p.8). The WHO specifically suggested the need to determine if low level RF exposure causes changes in melatonin synthesis (Repacholi, 1998).

More laboratory-based research on humans

There has been little laboratory-based research focusing on humans, especially in the area of cellular phone frequencies. The extensive literature on rodents has produced results that have been unable to be replicated in humans, this has led to the conclusion that animal anatomy is sufficiently different from humans for their use as models for humans to be unreliable in relation to EMF effects (IEGMP, 2000). It is also noted that the laboratory-based research done on humans thus far tended to use young healthy males who form only a small proportion of the total population. Future studies need to include a balance of sexes. Different age groups need to be studied, as it may be that older age groups may be more susceptible to EMF effects just as they are more susceptible to many cancers.

Replication of statistically significant results

Research that has reported statistically sign results needs to be replicated using the same experimental conditions. For this to occur, researchers need to provide detailed, accurate information on experimental conditions and design. This has been absent from many published studies. The present study followed the protocol suggested by Valberg (1995) which set down the parameters by which authors should describe the characteristics of the 50 Hz EMF used in their studies as an aid to researchers trying to replicate results. A similar protocol would be helpful for the cell phone frequency ranges.

Identification of possible Frequency-intensity windows in power frequency EMFs

Studies have covered a wide range of frequencies, flux densities, field phases duration, wave forms, and repetition rates. The inconsistency of results in the published research may be due to the possibility that EMF effects occur in certain frequency-intensity windows. Such windows could differ between species and parameters tested. As the body of research grows patterns may become clear.

Identification of possible susceptible sub populations

There may be a sub-population of individuals who are susceptible to EMF effects just as there are groups who are much more sensitive to other environmental variables than the rest of the population. For example, it could be that those who have been reported as sensitive to much lower levels of light (<200 lux) in suppressing melatonin could also be a group who may be susceptible to EMF

effects. This requires further research. If melatonin is found to be suppressed by EMFs , those with depression, alcoholism, family history of hormone dependent cancers could constitute a susceptible population (Wilson et al, 1982).

Laboratory-based research on chronic effects

There needs to be a way found to look at chronic effects of EMFs on humans, especially for RF fields. This would be difficult to set up in a controlled manner.

Investigations into possible mechanisms

There needs to be more research into possible mechanisms, particularly in relation to RF EMFs and the separation of possible EMF effects from heating effects. The role of ELF modulation in RF effects needs to be further explored.

Studies need to increase statistical power

Statistical power depends on sample size, population variance, the magnitude of the expected effect, significance level and the directionality of the test (Zimmerman and Williams, 1986). Statistical power is greater when the sensitivity of the design is high (by controlling unwanted variability and using a large number of subjects), when effect sizes are large, and when alpha is more lenient (Murphy and Myors, 1998). Whittington et al. (1996) and Podd et al.(2002) have argued for the relaxing of alpha to 0.10. Their argument being that it is tradition rather than sound scientific reasoning behind the selection of 0.05 and that the consequences of making a type 2 error make it prudent to err on the side of caution.

The "primary responsibility rests with the authors of articles reporting non-significant results to demonstrate the worth of the results by discussing the power of the tests" (Fagley, 1985. p.391). This is rarely done. This is why, in the present study, an analysis has been done to report on the ability of the given sample size to detect an effect in the light of the variability found in the data.

More Research on Cognitive factors

There is little research available on possible cognitive effects of EMFs, especially in relation to RF EMFs. More research is required to build up a knowledge base about possible effects on the very many different cognitive parameters and also to replicate those studies that have reported effects.

More research on chronic cell phone usage and new higher frequencies

Research is also urgently needed on the possible effects from chronic cell phone usage and the other cell phone frequency range of 1800-2200 MHz.
The introduction of third generation phones which operate at a much higher frequency is a new area that will require investigation.

Final Comments

Until recently, most people's exposure to EMFs came from power lines and domestic and office appliances. Now the public is being exposed to a greater number of EMF sources, covering an increasingly high frequency range. Portable devices mean this exposure is often carried around with the person. With this has come a concern as to the safety of such exposure, especially in the higher frequency ranges. That EMFs can effect living systems is clear. The use of EMFs in the medical setting, such as for the treatment of the non-union of bone fractures, has been well recognised. What is not clear is how far these effects extend and whether they can be harmful as well as beneficial. The mechanisms by which these effects occur has also not been established. However, there is enough evidence for some bodies to issue cautions with regards to exposure. For example, the IEGMP (2000) recommended that it was wise to limit children's exposure to cellular phones. In 2002, the IARC of the WHO concluded there is limited evidence for the carcinogenity of ELF EMFs in childhood leukaemia.

Effects reported in epidemiological studies have often failed to be replicated in the laboratory on humans. Research on animals frequently doesn't translate to humans so there is a need to concentrate on humans particularly in RF research where body size is an important component of the interaction (IEGMP, 2000). The same situation occurs in research using cells in vitro. Effects are reported in the lab that do not translate to effects in a living system, with its potential for the maintenance of equilibrium.

The area of Power Frequency laboratory research on humans is characterised by interesting findings that are often unable to be replicated. This may be due to differing field characteristics and the lack of enough detail in published studies to enable a true replication. Many studies report no effects on exposure to an EMF but then a study is published which reports an effect and stimulates further

research. The present study is an example in which two significant results were found among others that were not significant. Different parameters appear to be sensitive to different frequencies and intensities.

In the cell phone frequency range the research base is not large enough in the parameters discussed in this thesis for conclusions to be drawn. However, most studies on cognitive parameters report findings for at least some variables tested. This may indicate an effect in this area or it may be the result of a greater difficulty controlling confounders in this area. It is important that experimental designs reflect the spectrum of amplitude modulation to which the user is exposed. Studies that omit some of the characteristics such as utilisation of several channels, should be avoided (Kuster and Schonborn, 2000).

The field of EMF research is further complicated by the number of studies published that have insufficient statistical power given the variability of their data. However, it should be noted that even if a finding has statistical significance that does not necessarily equate with biological significance. For example, a small change in the permeability of the blood brain barrier could have considerable biological consequences, whilst failing to reach statistical significance. Conversely, significant changes in aural temperature may have few biological consequences. As there are many factors involved in any possible interaction between living systems and EMFs and many variables to test, it may take some years before enough literature is accumulated on all possible combinations of exposure for patterns to become clear. This is especially so for the area of cellular phone research. This scenario makes for an untidy conclusion and is not very reassuring for public concerns, but it is one that accurately reflects the current state of the EMF literature.

APPENDIX ONE
EXPERIMENT ONE

APPENDIX 1.1 SUBJECT SCREENING QUESTIONAIRE
APPENDIX 1.2 SUBJECT GENDER INFORMATION AND MELATONIN

The effects of electromagnetic fields on humans at different times of day.

Subject Screening Questionaire

Name _____

Please answer the following questions.

	Yes/No
1. Are you on any medication?	_____
2. Are you pregnant?	_____
3. Do you have any chronic health problems?	_____
4. Do you have any cardiovascular problems?	_____
5. Have you had any nervous system, or brain disorder, (for example; epilepsy, stroke, migraine, multiple sclerosis) ?	_____
6. Have you had a head injury or been knocked unconscious?	_____
7. Have you been confined to bed due to illness in the last 3 months?	_____
8. Are you having, or planning to have, psychotherapy?	_____
9. Have you participated in magnetic field research before?	_____
10. Do you wear any metal prosthesis or implanted electronic devices?	_____

The following things affect the body's production of melatonin so volunteers are asked to refrain from the following for 6 hours prior to each session.

Moderate or excessive exercise.
Any intake of alcohol or other drug affecting the brain.
Taking medication
Coffee/ tea/ coke intake needs to be restricted to not more than one cup in the previous 4 hours.

App. 1.2: Expt	Subject Gender Information				
Subject	Sex	Melatonin exposure-control (pg/ml) night			
1	F	Female	Female	Male	Male
2	M	Subject No.	Mel. Difference	Subject No.	Mel. difference
3	F	1	-11	2	1
4	F	3	-2	6	8
5	F	4	0	10	1
6	M	5	4	12	-1
7	F	7	-3	15	-5
8	F	8	-10	18	5
9	F	9	17	21	1
10	M	11	-2	27	-40
11	F	13	-5		
12	M	14	-12		
13	F	16	-8		
14	F	17	13		
15	M	19	22		
16	F	20	-2		
17	F	22	11		
18	M	23	5		
19	F	24	-15		
20	F	25	-5		
21	M	26	7		
22	F	28	0		
23	F	29	-8		
24	F	mean	-0.190	mean	-3.750
25	F	s.d.	9.852	s.d.	15.145
26	F	SEM	2.1499	SEM	5.355
27	M	t=	-0.0884	t=	-0.7003
28	F				
29	F				
	N=29		N=21 female		N=8 male
	Unpaired t-test				
	t28=-0.6707	s.d. pool =	12.7756		

APPENDIX TWO
EXPERIMENT TWO

Control Samples		Melatonin (pg/ml)			
				night	night
	diff day	same day		Control	Exposure Evenir
Subject	control	Control		Before	Before
12	3	5		32	20
13	0	3		5	6
14	34	16		148	138
15	3	21		7	68
16	2	4		55	1
17	0	8		14	22
18	0	2		17	16
19	5	20		12	14
20	0	0		13	19
21	0	11		64	50
22	1	1		0	0
23	0	2		6	24
24	-	2		0	3
25	0	4		10	60
26	0	0		3	6
27	2	4		4	22
28	0	6		5	4
29	0	2		0	0
30	0	0		1	1
31	0	0		0	12
32	0	1		0	0
33	2	1		23	31
34	2	2		20	32
35	2	21		1	0
36	0	0		7	1
37	0	5		3	0
38	2	7		10	3
39	3	7		36	20
40	2	3		11	3
41	3	2		3	3
42	0	0		0	0
43	0	0		12	9
44	0	0		4	10
45	11	1		0	3
46	0	0		4	1
47	0	0		0	0
48	-	-		5	1
49	10	11		2	5
50	5	1		55	23
Mean	2.5	4.6		15.2	16.2
SD	5.924	6.008		27.1	26.0
N	38	38		38	38

Expt 2 Low excretors of melatonin (10 pg/ml or less)							
		Day				Night	
Subject	Control	Exposure	Difference		Control	Exposure	Difference
*6	0	0	0		1	1	0
*10	15	40	25		8	19	11
*11	3	1	-2		2	11	9
*13	0	0	0		3	4	1
*22	1	0	-1		6	3	-3
*26	0	2	2		2	0	-2
*27	2	5	3		8	16	8
*28	0	2	2		3	3	0
*31	0	0	0		9	13	4
*32	0	0	0		2	0	-2
*35	2	13	11		3	4	1
*36	0	4	4		8	5	-3
*37	0	5	5		2	5	3
*38	2	5	3		5	3	-2
*40	2	4	2		8	14	6
*42	0	5	5		4	0	-4
*44	0	0	0		6	10	4
*45	11	3	-8		2	3	1
*46	0	0	0		5	0	-5
*48	-	-			4	1	-3
*49	10	0	-10		2	5	3
		mean	2.0				1.3
		s.d.	6.985				4.474
		SEM	1.524				0.976
		N=	20				21
		t=	1.3450				1.3170

Exper. TWO	Difference in Melatonin levels between Exposure and Control						
				Night			
				Female	Female	Male	Male
Subject	Sex	Subject	Sex	Subject No.	Mel. Diff.(p	Subject No.	Mel. Diff.(p
1	F	26	M	1	1	2	45
2	M	27	F	4	-42	3	-29
5	F	28	M	5	0	6	0
6	M	29	F	9	4	7	-8
7	M	30	M	10	11	8	-1
8	M	31	M	13	1	11	9
9	F	32	F	14	-1	12	4
10	F	33	M	15	36	19	1
11	M	34	F	16	-11	22	-3
12	M	35	F	17	3	26	-2
13	F	36	M	18	-4	28	0
14	F	37	F	20	-83	30	0
15	F	38	M	21	-17	31	4
16	F	39	F	23	15	33	10
17	F	40	F	24	8	36	-3
18	F	41	M	25	32	38	-2
19	M	42	F	27	8	41	4
20	F	43	F	29	0	47	0
21	F	44	F	32	-2	50	-48
22	M	45	F	34	27		
23	F	46	F	35	1		
24	F	47	M	37	3		
25	F	48	F	39	-3		
	N=50	49	F	40	6		
	MALES=19	50	M	42	-4		
	FEMALES=31			43	10		
				44	4		
				45	1		
				46	-5		
				48	-3		
				49	3		
				mean	-0.032	mean	-1.000
				s.d.	20.708	s.d.	17.436
				SEM	3.719	SEM	4.688
				t=	0.0086		-0.2133

Effects of Power Frequency Fields on Salivary Melatonin (pg/ml)-combined data

		Day			Night	
Subject	Control	Exposure	Difference	Control	Exposure	
Expt. One 1	32	31	-1	52	41	-11
2	47	5	-42	18	19	1
3	17	11	-6	16	14	-2
4	45	9	-36	8	8	0
5	2	1	-1	5	9	4
6	47	0	-47	6	14	8
7	2	10	8	21	18	-3
8	33	38	5	23	13	-10
9	-	-		0	17	17
10	8	3	-5	3	4	1
11	6	24	18	18	16	-2
12	1	1	0	9	8	-1
13	14	18	4	40	35	-5
14	2	5	3	36	24	-12
15	15	19	4	36	31	-5
16	5	2	-3	57	49	-8
17	0	5	5	2	15	13
18	4	5	1	6	11	5
19	22	4	-18	49	71	22
20	11	4	-7	54	52	-2
21	4	2	-2	0	1	1
22	4	5	1	24	35	11
23	0	4	4	32	37	5
24	12	6	-6	18	3	-15
25	8	5	-3	18	13	-5
26	10	11	1	78	85	7
27	8	30	22	65	25	-40
28	9	10	1	10	10	0
29	12	5	-7	12	4	-8
Expt. Two 1	4	1	-3	30	31	1
2	6	2	-4	112	157	45
3	9	1	-8	41	12	-29
4	2	16	14	53	11	-42
5	8	17	9	31	31	0
6	0	0	0	1	1	0
7	5	10	5	17	9	-8
8	10	14	4	51	50	-1
9	12	2	-10	29	33	4
10	15	40	25	8	19	11
11	3	1	-2	2	11	9
12	3	2	-1	24	28	4
13	0	0	0	3	4	1
14	34	9	-25	131	130	-1
15	3	13	10	16	52	36
16	2	2	0	13	2	-11

17	0	0	0		20	23	3
18	0	0	0		36	32	-4
19	5	10	5		12	13	1
20	0	0	0		99	16	-83
21	0	2	2		60	43	-17
22	1	0	-1		6	3	-3
23	0	0	0		14	29	15
24	-	-			0	8	8
25	0	3	3		17	49	32
26	0	2	2		2	0	-2
27	2	5	3		8	16	8
28	0	2	2		3	3	0
29	0	0	0		0	0	0
30	0	0	0		0	0	0
31	0	0	0		9	13	4
32	0	0	0		2	0	-2
33	2	0	-2		15	25	10
34	2	1	-1		22	49	27
35	2	13	11		3	4	1
36	0	4	4		8	5	-3
37	0	5	5		2	5	3
38	2	5	3		5	3	-2
39	3	1	-2		26	23	-3
40	2	4	2		8	14	6
41	3	0	-3		0	4	4
42	0	5	5		4	0	-4
43	0	0	0		16	26	10
44	0	0	0		6	10	4
45	11	3	-8		2	3	1
46	0	0	0		5	0	-5
47	0	0	0		0	0	0
48	-	-			4	1	-3
49	10	0	-10		2	5	3
50	5	1	-4		55	7	-48
N	77				79		
mean	7.2	6.2	-1.0		22.1	21.5	-0.7
s.d.	10.91	8.65	10.97		26.16	26.55	16.79
SEM			1.25				1.889
t			-1.25				-0.37

Table 6.2: Effects of Power Frequency Fields on Salivary Melatonin (pg/ml)Combined Da							
Low excretors		Day				Night	
Subject	Control	Exposure	Difference		Control	Exposure	Difference
Experiment One							
*4	45	9	-36		8	8	0
*5	2	1	-1		5	9	4
*6	47	0	-47		6	14	8
*10	8	3	-5		3	4	1
*12	1	1	0		9	8	-1
*17	0	5	5		2	15	13
*18	4	5	1		6	11	5
*28	9	10	1		10	10	0
Ex. Two*6	0	0	0		1	1	0
*10	15	40	25		8	19	11
*11	3	1	-2		2	11	9
*13	0	0	0		3	4	1
*22	1	0	-1		6	3	-3
*26	0	2	2		2	0	-2
*27	2	5	3		8	16	8
*28	0	2	2		3	3	0
*31	0	0	0		9	13	4
*32	0	0	0		2	0	-2
*35	2	13	11		3	4	1
*36	0	4	4		8	5	-3
*37	0	5	5		2	5	3
*38	2	5	3		5	3	-2
*40	2	4	2		8	14	6
*42	0	5	5		4	0	-4
*44	0	0	0		6	10	4
*45	11	3	-8		2	3	1
*46	0	0	0		5	0	-5
*48	-	-			4	1	-3
*49	10	0	-10		2	5	3
*50	5	1	-4		55	7	-48
Mean	5.8	4.3	-1.6		6.6	6.9	0.3
s.d.	11.82	7.63	12.70		9.52	5.41	10.19
SEM	2.158	1.393	2.3186		1.7381	0.9877	1.86
t=			-0.69				0.1612
N=29					N=30		
Exp. 1	N=8						
mean	14.5	4.2	-10.2		6.1	9.9	3.8
s.d.	19.70	3.73	19.70		2.80	3.52	4.83
SEM	6.965	1.3188	6.965		0.9899	1.2445	1.7076
t=			-1.4357				2.2253
Exp.2	N=22						
Mean	2.5	4.3	1.8		6.7	5.8	-1.0

s.d.	4.26	8.76	6.93		11.07	5.62	11.38
SEM	0.9082	1.8676	1.4775		2.3601	1.1982	2.4262
t=			1.2183				0.4071

Melatonin combined results (pg/ml) night			
Female	Female	Male	Male
Subject No.	Mel. Difference	Subject No.	Mel. difference
Expt. One 1	-11	2	1
3	-2	6	8
4	0	10	1
5	4	12	-1
7	-3	15	-5
8	-10	18	5
9	17	21	1
11	-2	27	-40
13	-5	Expt. Two 2	45
14	-12	3	-29
16	-8	6	0
17	13	7	-8
19	22	8	-1
20	-2	11	9
22	11	12	4
23	5	19	1
24	-15	22	-3
25	-5	26	-2
26	7	28	0
28	0	30	0
29	-8	31	4
Expt. Two 1	1	33	10
4	-42	36	-3
5	0	38	-2
9	4	41	4
10	11	47	0
13	1	50	-48
14	-1		
15	36		
16	-11		
17	3		
18	-4		
20	-83		
21	-17		
23	15		
24	8		
25	32		
27	8		
29	0		
32	-2		
34	27		
35	1		
37	3		
39	-3		
40	6		
42	-4		

43	10		
44	4		
45	1		
46	-5		
48	-3		
49	3		
N=	52		27
Mean =	1.12		-1.81
S.D.=	17.039		16.548
SEM=	2.3628		3.1846
t=	0.4739		0.5683

Combined Effects of Power Frequency Fields on Aural Memory

Four Letter Group (score out of 20)

		Day			Night	
Subject	Control	Exposure	Difference	Control	Exposure	difference
1	14	11	-3	18	17	-1
2	15	18	3	17	10	-7
3	11	13	2	10	12	2
4	16	12	-4	17	14	-3
5	13	15	2	15	14	-1
6	12	16	4	16	13	-3
7	9	15	6	14	9	-5
8	12	12	0	14	12	-2
9	-	-		13	13	0
10	14	9	-5	10	12	2
11	14	15	1	16	11	-5
12	13	14	1	13	12	-1
13	14	12	-2	7	14	7
14	13	14	1	13	10	-3
15	12	13	1	13	14	1
16	13	12	-1	14	9	-5
17	17	14	-3	18	13	-5
18	14	13	-1	15	17	2
19	12	13	1	15	17	2
20	13	16	3	16	11	-5
21	13	15	2	16	17	1
22	18	17	-1	14	18	4
23	17	18	1	18	19	1
24	16	13	-3	13	14	1
25	17	14	-3	17	16	-1
26	13	12	-1	11	15	4
27	11	13	2	14	15	1
28	12	13	1	11	12	1
29	10	12	2	11	11	0
1	8	12	4	13	13	0
2	15	10	-5	7	12	5
3	12	11	-1	10	8	-2
4	15	17	2	14	17	3
5	14	12	-2	14	13	-1
6	13	12	-1	12	16	4
7	14	15	1	14	13	-1
8	16	9	-7	13	14	1
9	10	13	3	10	12	2
10	11	10	-1	13	12	-1
11	14	14	0	15	14	-1
12	15	15	0	15	15	0
13	15	15	0	14	16	2
14	15	15	0	19	15	-4
15	15	16	1	15	17	2

16	13	14	1		12	10	-2
17	17	15	-2		16	14	-2
18	15	15	0		11	12	1
19	12	8	-4		9	14	5
20	13	15	2		12	14	2
21	16	14	-2		14	14	0
22	15	14	-1		15	14	-1
23	11	17	6		13	13	0
24	-	-			13	16	3
25	16	16	0		17	15	-2
26	11	15	4		15	14	-1
27	17	16	-1		14	10	-4
28	14	12	-2		13	14	1
29	14	16	2		15	16	1
30	17	16	-1		17	17	0
31	16	17	1		18	17	-1
32	13	14	1		13	14	1
33	16	15	-1		17	15	-2
34	17	15	-2		16	17	1
35	18	15	-3		15	19	4
36	17	15	-2		17	18	1
37	13	14	1		12	14	2
38	16	16	0		14	15	1
39	16	17	1		17	18	1
40	15	13	-2		17	17	0
41	15	17	2		18	16	-2
42	13	14	1		9	11	2
43	13	12	-1		12	15	3
44	16	15	-1		16	16	0
45	14	13	-1		13	14	1
46	16	15	-1		17	16	-1
47	15	15	0		15	16	1
48	-	-			17	13	-4
49	13	12	-1		9	11	2
50	16	11	-5		10	12	2
N			77				79
mean			-0.14				0.05
s.d.			2.437				2.640
SEM			0.278				0.297
t			-0.5035				0.1683

APPENDIX THREE

EXPERIMENT THREE

A study of possible effects of cellular phones on humans
Subject Screening Questionaire

Name _____

Age _____ Sex _____

Please answer the following questions.

Yes/No

1. Are you on any medication? _____
2. Are you pregnant? _____
3. Do you have any chronic health problems? _____
4. Do you have any cardiovascular problems? _____
5. Have you had any nervous system, or brain disorder, (for example; epilepsy, stroke, migraine, multiple sclerosis) ? _____
6. Have you had a head injury or been knocked unconscious? _____
7. Have you been confined to bed due to illness in the last 3 months? _____
8. Are you having, or planning to have, psychotherapy? _____
9. Have you participated in magnetic field research before? _____
10. Do you wear any metal prosthesis or implanted electronic devices? _____

Cell phone use

Do you use a cell phone _____ yes _____ no

if 'yes'

how long have you used one _____ weeks , _____ years (state how many)

frequency of use _____ several times a day, , _____ once or twice a day, ,

_____ several times a week, _____ once or twice aweek, _____ only occasionally

Please give the names and model of phones used if known._____

_____

The following things affect the body's production of melatonin so volunteers are asked to refrain from the following for 6 hours prior to the session.

Moderate or excessive exercise.

Any intake of alcohol or other drug affecting the brain.

Coffee/ tea/ coke intake needs to be restricted to not more than one cup in the previous 4 hours.

Effects of Cell Phone Use on Temperature (Degrees Celsius)

Subject	Session	1L	1R	2L	2R	3L	3R	4L	4R	5L	5R
1	Pre-Exposure	35.6		36		36.3		36.2		36.2	
	First Exposure	36.7		35.6		36.2		36		36	
	Interval	36		36.1		36		36.1		36.2	
	Second Exposure	36		36.1		35.9		35.6		35.9	
	Post Exposure	35.8		35.7		35.5		35.3		35.6	
2	Pre-Exposure	37.3		36.8		36.7		37		37	
	First Exposure	37.2		37.5		37.2		37.2		37	
	Interval	37		36.9		36.7		36.7		36.1	
	Second Exposure	36.9		37.1		36.4		36.8		36.8	
	Post Exposure	37.1		36.6		36.4		36.5		36.3	
3	Pr eExposure	36.3		35.8		36		35.2		35.5	
	First Exposure	35.7		35.7		35.7		36		35.7	
	Interval	35.3		36.3		35.2		35.9		36.1	
	Second Exposure	35.5		35.2		36.3		36.2		35.1	
	Post Exposure	35.9		36.4		36.1		36.1		36.2	
4	Pre-Exposure	36.2		36.8		35.9		37		37.2	
	First Exposure	36.8		36.5		37		36.9		37	
	Interval	36.9		36.6		36.3		36.9		36.8	
	Second Exposure	36.7		36.5		36.5		36.8		36.9	
	Post Exposure	36.1		36.6		36.6		36.9		36.3	
5	Pre-Exposure	36.4		36.6		36.3		36.9		36.5	
	First Exposure	36.7		36.5		36.5		36.8		36.8	
	Interval	36.6		36.8		36.7		36.6		36.5	
	Second Exposure	36.4		36.4		36.4		36.5		36.3	
	Post Exposure	36.5		36.3		36.3		36.1		36.5	
6	Pre-Exposure	37.2		37		36.6		36.7		36.6	
	First Exposure	36.9		36.5		36.9		36.8		36.3	
	Interval	36.3		36.2		36.3		36		36.5	
	Second Exposure	36		36.5		36.2		36.3		36.4	
	Post Exposure	35.8		36.2		35.5		36		35.7	
7	Pre-Exposure	36.4		36.7		36.6		36.8		35.9	
	First Exposure	36.5		36.3		36.6		36.2		36.4	
	Interval	36.1		36.3		36.1		36.4		35.9	
	Second Exposure	35.9		36.3		35.9		36		35.7	
	Post Exposure	36		35.4		35.9		35.7		35.6	
8	Pre-Exposure	35.7		36.2		35.8		35.5		36	
	First Exposure	35.6		36.2		35.9		35.7		36.1	
	Interval	35.8		35.9		35.7		36.2		35.7	
	Second Exposure	35.9		36.4		35.7		35.8		36.1	
	Post Exposure	36		35.9		35.8		36		35.8	
9	Pre-Exposure	36.4		36.2		36.1		36		35.8	
	First Exposure	36.3		36.4		36.7		36.4		36.9	
	Interval	35.8		35.9		35.8		36.1		36.3	

Subject	Mean L	Mean R	Mins
1	36.06		0
	36.1		24
	36.08		53
	35.9		72
	35.58		99
2	36.96		0
	37.22		17
	36.68		47
	36.8		67
	36.58		96
3	35.76		0
	35.76		30
	35.76		60
	35.66		84
	36.14		113
4	36.62		0
	36.84		24
	36.7		55
	36.68		75
	36.5		106
5	36.54		0
	36.66		23
	36.64		53
	36.4		70
	36.34		95
6	36.82		0
	36.68		23
	36.26		51
	36.28		67
	35.84		94
7	36.48		0
	36.4		20
	36.16		52
	35.96		75
	35.72		105
8	35.84		0
	35.9		19
	35.86		52
	35.98		72
	35.9		101
9	36.1		0
	36.54		26
	35.98		59

	Second Exposure	36.1		36		36.2		36		36.3	
	Post Exposure	35.9		36.1		35.8		35.9		35.8	
Subject	Session	1L	1R	2L	2R	3L	3R	4L	4R	5L	5R
10	Pre-Exposure	36.8		36.2		36.5		36.5		36.3	
	First Exposure	36.6		36		36.3		36.2		35.6	
	Interval	35.9		36.2		35.9		36.6		36.1	
	Second Exposure	36		36.2		36		36.4		36.3	
	Post Exposure	35.8		35.9		35.3		36.2		35.5	
11	Pre-Exposure	36.3		35.6		35.8		36.3		36.1	
	First Exposure	36.2		36.4		36		35.5		35.5	
	Interval	35.8		35.7		35.4		35.4		35.6	
	Second Exposure	35.6		35.7		35.8		35.4		35.5	
	Post Exposure	35.1		35.2		34.8		34.8		34.6	
12	Pre-Exposure	35.6		35.7		35.7		35.3		35.4	
	First Exposure	36.1		36		35.9		35.7		35.8	
	Interval	35.7		35.9		35.8		35.8		36	
	Second Exposure	36.4		35.6		36.3		36.1		36.3	
	Post Exposure	35.7		35.7		36		35.7		35.8	
13	Pre-Exposure	37.3		37.3		37.3		37.1		37.2	
	First Exposure	36.9		36.8		36.9		37		37	
	Interval	37		37		36.9		36.8		37	
	Second Exposure	37		36.9		36.9		36.8		37	
	Post Exposure	36.7		37.1		36.7		37.1		37	
14	Pre-Exposure	36.3		36.4		36.5		36		36.2	
	First Exposure	36.1		35.9		35.6		35.9		35.7	
	Interval	35.4		35.5		35.3		35.6		35.3	
	Second Exposure	35.4		34.9		35.2		35.2		35.6	
	Post Exposure	35.3		35.4		35.2		35.5		35.5	
15	Pre-Exposure	36.7		36.9		37		37		36.5	
	First Exposure	36.4		35.7		36.4		36.5		36.6	
	Interval	36.4		36.5		36.3		36.3		36.3	
	Second Exposure	36.5		36.2		36.6		36.2		36.6	
	Post Exposure	36.1		35.9		35.9		35.9		35.9	
16	Pre-Exposure	37.1		37		37.2		37		37.3	
	First Exposure	36.8		37.1		37		36.7		37	
	Interval	37		36.6		36.6		36.4		36.4	
	Second Exposure	36.5		36.3		36.2		36		36.1	
	Post Exposure	36.5		36.3		36.1		35.6		36.1	
17	Pre-Exposure	36.3		36.7		36.4		36.9		36.6	
	First Exposure	36.8		36.9		37		37		36.8	
	Interval	36.9		36.7		36.9		36.6		36.8	
	Second Exposure	36.6		36.5		36.5		36.4		36.4	
	Post Exposure	36.2		36.1		36.1		36		36	
18	Pre-Exposure	36.3		36.3		37		36.2		36.2	
	First Exposure	36.9		36.6		36.7		35.8		36.1	
	Interval	37		36.4		36.6		36.5		36.3	

	36.12		101
	35.9		131
Subject	Mean L	Mean R	Mins
10	36.46		0
	36.14		27
	36.14		55
	36.18		70
	35.74		97
11	36.02		0
	35.92		21
	35.58		49
	35.6		64
	34.9		88
12	35.54		0
	35.9		22
	35.84		52
	36.14		75
	35.78		108
13	37.24		0
	36.92		23
	36.94		57
	36.92		78
	36.92		108
14	36.28		0
	35.84		18
	35.42		43
	35.26		61
	35.38		87
15	36.82		0
	36.32		29
	36.36		59
	36.42		82
	35.94		112
16	37.12		0
	36.92		22
	36.6		51
	36.22		79
	36.12		109
17	36.58		0
	36.9		20
	36.78		52
	36.48		70
	36.08		103
18	36.4		0
	36.42		31
	36.56		75

	Second Exposure	36.5		35.3		36.1		36.1		35.5	
	Post Exposure	35.7		36.2		35.4		35.8		35.4	
Subject	Session	1L	1R	2L	2R	3L	3R	4L	4R	5L	5R
19	Pre-Exposure	36.7		35.9		36.4		36.8		36.6	
	First Exposure	37.2		36.8		37.1		36.9		37	
	Interval	36.8		36.4		36.8		36.6		36.6	
	Second Exposure	36.7		36.6		36.5		36.7		36.3	
	Post Exposure	36.5		36.6		36.8		36.6		36.5	
20	Pre-Exposure	36.8		36.9		36.9		36.8		36.9	
	First Exposure	36.8		36.7		36.7		36.9		36.6	
	Interval	36.3		36.4		36.4		36.3		36.4	
	Second Exposure	36.3		36.5		36		36.2		36.3	
	Post Exposure	36.4		36		35.9		36.2		36.1	
21	Pre-Exposure	35.7		35.7		35.2		35.4		35.7	
	First Exposure	35.8		35.8	36.5	35.5	36.4	35.4	36.1	36.2	36.8
	Interval	35.2	35.6	35.1	35.9	35.2	35	35.1	35.2	34.9	35.3
	Second Exposure	35	36.2	35.8	36.2	35.8	35.5	35.7	35.9	35.5	35.8
	Post Exposure	35.5	35.6	35.7	34.7	35.5	35.7	35.1	35.6	36.2	35.6
22	Pre-Exposure	35.9	35.8	36.1	35.6	35.9	36	35.6	35.7	35.7	36.2
	First Exposure	36.1	36	36.3	36.3	36.1	36.1	36.2	36.5	36.5	36.1
	Interval	36.3	35.7	36	36.1	36.3	35.5	36	36	36.3	36.3
	Second Exposure	36.1	36.1	36.7	36.1	36.1	35.9	35.6	35.7	35.7	35.7
	Post Exposure	35.4	35.4	35.6	35.3	35.3	35.1	35.4	35.1	35.5	35.2
23	Pre-Exposure	35.6	35.5	35.3	36.1	35.6	36.6	35.6	35.9	35.8	35.6
	First Exposure	35.2	36	34.9	35.9	35.7	35.3	34.9	35	35	36.4
	Interval	35.3	35.3	35.3	34.8	35.5	34.8	34.9	35.4	35.6	35.8
	Second Exposure	35.5	36.1	35.1	36.2	35.2	35	35.1	35.7	35.3	35.3
	Post Exposure	34.6	35	34.6	34.9	34.6	35.7	35.3	35.6	35	34.7
24	Pre-Exposure	36.4		36.7	37.6	35.8	36.3	34.9	35.5	36	36.6
	First Exposure	35	34.6	36.1	36.8	34.8	36.8	34.7	34.6	34.6	37.2
	Interval	35.2	34.7	34.3	34.6	34.4	35.4	35.4	35.9	36.2	36.7
	Second Exposure	35.5	36.5	35.4	36.2	34.8	35.7	37	37.5	35.9	37
	Post Exposure	36.6	35.8	35.9	37.3	35.5	34.5	34.5	35.2	35.1	36.2
25	Pre-Exposure	36.1	35.9	36.2	35.8	35.9	35	36.6	35.8	36	35.3
	First Exposure	35.2	36.2	35.7	36	36	36.8	36.8	36.8	36.3	36.4
	Interval	36.4	37	36.3	36.8	36.6	36	35.8	35.6	35.9	35
	Second Exposure	35.9	36.2	35.7	35.9	35.4	36.1	35.8	35.5	35.7	35.6
	Post Exposure	35.4	35.3	35.5	35.4	36	35	35.7	35.4	35.4	35.8
26	Pre-Exposure	36.8	36.4	36.4	35.6	36.9	36	35.9	36.4	36.1	35.6
	First Exposure	37	37	36.7	37.9	36.5	36.7	36.2	38.1	36.6	36.5
	Interval	36.1	36.8	37.3	37	36.7	37.3	36.5	36.8	36.7	37.4
	Second Exposure	36.1	37.2	35.9	37	36.9	37.6	36.8	37.9	36.4	37.2
	Post Exposure	36.3	36.8	36.4	36.2	36.8	36.5	36.1	36.9	36.9	37
27	Pre-Exposure	36.4	37.1	36.4	37.1	36.7	37	36.4	37.5	36.5	37
	First Exposure	37	37	36.9	36.8	37.2	36.8	36.8	37.3	36.5	36.5
	Interval	37.1	36.5	36.6	36.5	36.8	36.6	37.2	36.7		

	35.9		98
	35.7		125
Subject	Mean L	Mean R	Mins
19	36.48		0
	37		24
	36.64		51
	36.56		67
	36.6		94
20	36.86		0
	36.74		20
	36.36		31
	36.26		48
	36.12		74
21	35.54		0
	35.74	36.45	17
	35.1	35.4	46
	35.56	35.92	66
	35.6	35.44	91
22	35.84	35.86	0
	36.24	36.2	19
	36.18	35.92	54
	36.04	35.9	85
	35.44	35.22	124
23	35.58	35.94	0
	35.14	35.72	22
	35.32	35.22	57
	35.24	35.66	81
	34.82	35.18	108
24	35.96	36.5	0
	35.04	36	33
	35.1	35.46	77
	35.72	36.58	108
	35.52	35.8	147
25	36.16	35.56	0
	36	36.44	45
	36.2	36.08	82
	35.7	35.86	113
	35.6	35.38	147
26	36.42	36	0
	36.6	37.24	35
	36.66	37.06	78
	36.42	37.38	101
	36.5	36.68	140
27	36.48	37.14	0
	36.88	36.88	25
	36.92	36.58	60

	Second Exposure	37.1	36.5	36.2	36.6	36.2	37	37.2	37	37.3	36.9
	Post Exposure	36.8	37	37	36.8	36.1	36.8	35.9	36.7	36.1	37.1
Subject	Session	1L	1R	2L	2R	3L	3R	4L	4R	5L	5R
28	Pre-Exposure	36	37.1	35.9	36.6	36.8	36.3	36.4	36.1	36.8	35
	First Exposure	36.7	36.7	36.2	37.4	34.7	36.5	36.9	36.9	35.3	35.6
	Interval	35.9	34.7	35.1	36.1	35.2	37	34.7	35.8	34.6	36.4
	Second Exposure	35	35.1	35.7	35.6	35.6	35.9	35.3	35.1	34.4	35.5
	Post Exposure	35.5	35.1	34.4	35.2	34.9	35.5	34.6	35.4	34.8	35
29	Pre-Exposure	36	36.5	36.1	36.5	36.1	36.2	35.7	36.6	36.5	36.7
	First Exposure	36.3	36.2	36.9	36.7	36.2	37.1	36.7	36.5	36.5	36.8
	Interval	36.3	36.8	37.2	36.9	35.8	36.6	36.5	35.5	35.9	35.9
	Second Exposure	36.4	35.7	36.2	36.3	36.9	36.2	36.9	36.3	36.9	36.7
	Post Exposure	36.3	36.5	36.7	36.6	36.5	36.5	36.9	36.6	36.3	36.9
30	Pre-Exposure	36.4	37.1	35.6	37.4	37	37.2	37.3	37	36	37.6
	First Exposure	37.5	37.5	37.7	36.7	37.6	37.9	36.5	37.8	37.4	37
	Interval	37.4	37.5	37.4	37	36.8	37.4	36.4	37.5	35.4	37.1
	Second Exposure	37.1	37.3	36.1	35.6	36.6	36	36	37.5	35.7	37.2
	Post Exposure	36.4	36.1	35.6	37.3	36.3	36	35.8	36.3	35.3	36
31	Pre-Exposure	37	37.6	37.2	37.3	37.6	37.1	37.4	37.2	36.8	37.2
	First Exposure	37.3	37.6	37.7	37.4	37	37.3	37.5	37.2	36.3	37.3
	Interval	37.1	36.8	36.9	37.1	36.8	37	37.2	36.9	37	37.1
	Second Exposure	37.1	37.6	36.7	37.2	36.9	36.8	36.3	36.6	36.6	36.8
	Post Exposure	36.6	36.9	36.8	36.6	36.8	36.4	36.7	36.8	36	37.3
32	Pre-Exposure	36.8	37.3	36.5	37.2	36.4	36.8	36.3	36.7	37.1	36.8
	First Exposure	36.5	37.9	36.6	36.9	36.4	37.1	36.4	37	36.6	37
	Interval	36.2	36.6	36.2	36.5	36.1	36	36.2	36.7	36.4	36.6
	Second Exposure	36.5	37	36.7	36.6	36.2	36.8	36.4	36.9	36.3	36.6
	Post Exposure	36.6	36.2	36.2	36.4	36.1	36.4	36.2	36.3	36.3	36.3
33	Pre-Exposure	36.2	36.7	36.4	36.5	36.3	36.5	36.2	36.6	36.6	36.3
	First Exposure	36.8	36.6	36.3	36.4	36.7	36	36	36.6	36	36
	Interval	36.1	36.6	36.4	36.3	36.2	36.1	36.5	36	36.4	36.2
	Second Exposure	35.6	35.6	35.3	35.4	35.6	35.3	35	35.2	35.4	35.1
	Post Exposure	35.4	35.9	35.4	34.9	36	35.7	36	36.5	35.3	34.7
34	Pre-Exposure	37.6	38	37.5	37.5	36.3	37.6	37.6	38	37.4	37.9
	First Exposure	37.8	37.6	37.6	38	37.8	37	37.2	37.7	37.7	36.6
	Interval	37.3	37.3	36.8	37.3	37.2	36.8	36.9	37	37.5	37.5
	Second Exposure	37.9	37.5	37.2	37.3	37.5	37	36.9	37.3	36.8	37.3
	Post Exposure	36.9	37.1	36.7	36.8	37.4	36.9	37.1	37.2	37.3	37
35	Pre-Exposure	37	36.8	37	37.1	37.2	37.2	37.4	36.3	37.4	36.8
	First Exposure	37.5	36.9	37	37.4	37.9	38.9	37.7	37.2	37.5	39
	Interval	36.5	37	36.8	37	36.7	36.7	36.5	36.4	36.9	37
	Second Exposure	37.7	37.2	37.4	37.2	37.2	37.1	36.8	37.1	37.8	37.2
	Post Exposure	36.5	36.7	36.7	36.6	36.9	36.6	36.4	36.7	36.3	36.5
36	Pre-Exposure	37.7	37.4	36.2	37.4	36.8	35.9	36.1	36	36.1	36.6
	First Exposure	36.7	37.1	37.8	37.3	37.8	37.3	38.1	38	38	37.5
	Interval	36	36.2	36.6	36.3	36.5	36.3	36.3	36.1	36.2	36.4

	36.8	36.68	83
	36.38	36.88	116
Subject	Mean L	Mean R	Mins
28	36.38	36.22	0
	35.96	36.62	26
	35.1	36	61
	35.2	35.44	90
	34.84	35.24	121
29	36.08	36.5	0
	36.52	36.66	26
	36.34	36.34	56
	36.66	36.24	80
	36.54	36.62	110
30	36.46	37.26	0
	37.34	37.38	21
	36.68	37.3	51
	36.3	36.72	69
	35.88	36.34	97
31	37.2	37.28	0
	37.16	37.36	30
	37	36.98	69
	36.72	37	89
	36.58	36.8	117
32	36.62	36.96	0
	36.5	37.18	55
	36.22	36.48	108
	36.42	36.78	156
	36.28	36.32	188
33	36.34	36.52	0
	36.36	36.32	22
	36.32	36.24	57
	35.38	35.32	85
	35.62	35.54	128
34	37.28	37.8	0
	37.62	37.38	29
	37.14	37.18	74
	37.26	37.28	98
	37.08	37	138
35	37.2	36.84	0
	37.52	37.88	35
	36.68	36.82	83
	37.38	37.16	106
	36.56	36.62	140
36	36.58	36.66	0
	37.68	37.44	24
	36.32	36.26	56

	Second Exposure	37.8	36.8	36.8	36.9	37.3	37.2	37.6	36.4	36.8	35.9
	Post Exposure	35.9	35.9	36.2	36.4	35.8	36.2	35.9	36	36.1	36
Subject	Session	1L	1R	2L	2R	3L	3R	4L	4R	5L	5R
37	Pre-Exposure	36.3	36.6	36.6	35.7	35.8	36.5	37	36.4	36.3	36.2
	First Exposure	36.9	36.6	36.1	36.9	36.9	36	36.1	36.3	36.5	36.8
	Interval	35.9	36.1	35.8	36.5	36.4	36.6	36	35.9	36.1	36.4
	Second Exposure	36.5	36.6	37.3	37.2	37.2	36.2	36.1	36.3	36.3	36.8
	Post Exposure	36.8	36	36.6	36.6	36.8	36.5	37.2	37.1	36.6	36.9
38	Pre-Exposure	36.3	35.7	36.3	36.6	35	36.3	35.5	36.6	36.2	37.2
	First Exposure	35.6	36.8	35.5	36.8	35.2	37.4	35	36.6	35.3	36.7
	Interval	34.8	35.5	35.2	36.7	35.5	37	36.6	36.5	35.8	36.9
	Second Exposure	35.3	36.1	35.1	35.7	35.4	35.8	34.9	36.3	34.9	36.6
	Post Exposure	34.4	36.6	35.6	36.4	35.2	35.7	34.3	35.4	35.3	35.8
39	Pre-Exposure	37.1	37.1	37.2	37	37.4	36.1	37.4	37.4	36.9	36.8
	First Exposure	36.5	37.7	37.6	37.2	37.6	37.9	37.1	37.5	37.5	37.7
	Interval	36.9	37.8	37	36.9	37	37	37	37.2	36.2	37.1
	Second Exposure	37.7	37.7	37.4	37.5	37.2	37.6	36.6	36.9	36.4	37.5
	Post Exposure	36.6	36.8	36.1	36.2	36.3	37.1	36.9	36.9	36.7	36.7
40	Pre-Exposure	34.9	35.5	34.7	35.5	34.8	34.4	36	35.9	34.9	34.3
	First Exposure	34.3	35.6	35	36.8	36.1	36.8	35	35.7	35.9	35.4
	Interval	3 48	34.6	35.2	35.2	35	34.5	34.8	35.2	35.1	35.5
	Second Exposure	35.7	37.3	35.1	36.6	35	35.7	34.1	36.8	35.2	36.5
	Post Exposure	34.6	35.6	34.8	34.7	34.9	35.7	34.6	35	34.7	35.2
41	Pre-Exposure	37.3	37.7	38	38.3	37	37.6	36.6	36.9	36.8	37.3
	First Exposure	36.6	37.4	36.8	37	36.8	37	37.2	37	37	37.8
	Interval	37.3	37.3	37.5	36.9	36.5	36.9	36.7	36.8	36.5	37.2
	Second Exposure	37.5	37.4	37.1	37.2	35.8	37.1	36.9	37.2	36.7	37.5
	Post Exposure	36.9	37.1	37.1	36.9	37.1	36.8	36.8	37.3	37.6	37.6
42	Pre-Exposure	36.3	36.2	36.5	35.9	35.8	36.4	37.4	36.7	37	37.2
	First Exposure	36.9	38	37.2	37.4	37	37.7	37.6	37.2	37.3	37.3
	Interval	36.6	37	36.4	36.9	36.7	37.5	36.8	37	36.9	36.7
	Second Exposure	37.1	37.1	37.4	36.7	36.9	37.3	37	37.1	37.4	36.6
	Post Exposure	36.8	36.4	36.7	36.9	36.8	36.8	37	36.6	36.6	36.7
43	Pre-Exposure	36	36.4	34.4	36.9	36.5	36.2	36.1	37.1	36.6	36.7
	First Exposure	35.5	35.1	36.7	36.5	35.8	36.7	36.4	37	35.6	37.2
	Interval	36.6	36.9	36.6	37.1	35.7	35.7	34.6	36.5	36	37.1
	Second Exposure	36.2	35.9	35.9	35.7	34.8	36.9	35.4	35.9	36	37
	Post Exposure	36	36.9	36.7	36.7	36.1	36.7	35.4	36.7	35.8	36.4

	37.26	36.64	79
	35.98	36.1	103
Subject	Mean L	Mean R	Mins
37	36.4	36.28	0
	36.5	36.52	30
	36.04	36.3	69
	36.68	36.62	91
	36.8	36.62	121
38	35.86	36.48	0
	35.32	36.86	29
	35.58	36.52	63
	35.12	36.1	83
	34.96	35.98	115
39	37.2	36.88	0
	37.26	37.6	26
	36.82	37.2	67
	37.06	37.44	89
	36.52	36.74	124
40	35.06	35.12	0
	35.26	36.06	23
	34.98	35	62
	35.02	36.58	95
	34.72	35.24	135
41	37.14	37.56	0
	36.88	37.24	29
	36.9	37.02	64
	36.8	37.28	90
	37.1	37.14	120
42	36.6	36.48	0
	37.2	37.52	25
	36.68	37.02	58
	37.16	36.96	78
	36.78	36.68	113
43	35.92	36.66	0
	36	36.5	21
	35.9	36.66	57
	35.66	36.28	76
	36	36.68	105

Effects of Cell Phone Frequency EMFs on Blood Pressure.

Subj.	Sess.	Reading											
			1			2			3			4	
		Sys.	Dia.	PP	Sys.	Dia.	PP	Sys.	Dia.	PP	Sys.	Dia	PP
1	Pre.	101	75	26	110	65	45	113	64	49	109	64	45
	1st	104	71	33	99	73	26	87	58	29	100	74	26
	Int.	130	61	69	102	60	42	111	57	54	100	52	48
	2nd	98	66	32	98	57	41	102	53	49	109	50	59
	Post	105	46	59	106	54	52	100	55	45	94	54	40
2	Pre.	134	67	67	125	62	63	119	70	49	111	59	52
	1st	112	61	51	102	52	50	108	68	40	121	54	67
	Int.	102	59	43	112	49	63	112	49	63	92	55	37
	2nd	102	47	55	108	53	55	116	52	64	104	53	51
	Post	100	51	49	91	53	38	105	44	61	103	46	57
3	Pre.	106	74	32	104	71	33	97	70	27	102	71	31
	1st	95	71	24	112	70	42	104	67	37	97	65	32
	Int.	103	73	30	100	67	33	105	68	37	98	70	28
	2nd	103	67	36	108	94	14	104	72	32	99	69	30
	Post	102	79	23	99	70	29	109	71	38	98	72	26
4	Pre.	89	52	37	92	60	32	90	51	39	85	51	34
	1st	80	57	23	81	59	22	86	50	36	91	50	41
	Int.	99	63	36	90	49	41	84	50	34	87	52	35
	2nd	79	56	23	97	57	40	87	51	36	87	52	35
	Post	87	56	31	76	55	21	94	56	38	96	51	45
5	Pre.	101	64	37	89	61	28	99	62	37	113	70	43
	1st	96	64	32	98	62	36	98	65	33	92	50	42
	Int.	88	60	28	92	75	17	79	58	21	77	61	16
	2nd	103	65	38	99	64	35	90	57	33	92	60	32
	Post	98	71	27	92	59	33	90	55	35	91	59	32
6	Pre.	146	89	57	137	83	54	147	86	61	151	83	68
	1st	145	82	63	146	74	72	147	106	41	151	76	75
	Int.	119	74	45	136	65	71	142	66	76	136	67	69
	2nd	144	79	65	142	66	76	127	76	51	153	65	88
	Post	133	64	69	141	80	61	128	65	63	127	73	54
7	Pre.	123	80	43	118	82	36	146	99	47	117	86	31
	1st	118	99	19	125	86	39	111	83	28	134	74	60
	Int.	135	113	22	127	74	53	122	79	43	135	88	47
	2nd	124	76	48	118	78	40	127	82	45	117	87	30
	Post	123	78	45	130	75	55	116	74	42	120	65	55
8	Pre.	122	67	55	150	71	79	113	71	42	100	66	34
	1st	130	76	54	113	71	42	99	71	28	106	79	27
	Int.	112	71	41	130	105	25	104	71	33	98	67	31
	2nd	143	74	69	110	74	36	108	61	47	107	69	38
	Post	110	87	23	117	76	41	136	66	70	117	73	44

Subj.				Sess.			
		5			Mean	Mean	Time
	Sys.	Dia.	PP		Sys.	PP	Mins
1	102	65	37	Pre.	107	40.4	0
	117	61	56	1st	101.4	34	24
	95	52	43	Int.	107.6	51.2	53
	97	60	37	2nd	100.8	43.6	72
	91	53	38	Post	99.2	46.8	99
2	109	55	54	Pre.	119.6	57	0
	102	64	38	1st	109	49.2	17
	105	49	56	Int.	104.6	52.4	47
	109	47	62	2nd	107.8	57.4	67
	97	55	42	Post	99.2	49.4	96
3	89	69	20	Pre.	99.6	28.6	0
	97	71	26	1st	101	32.2	30
	97	68	29	Int.	100.6	31.4	60
	100	71	29	2nd	102.8	28.2	84
	104	77	27	Post	102.4	28.6	113
4	95	47	48	Pre.	90.2	38	0
	80	50	30	1st	83.6	30.4	24
	96	54	42	Int.	91.2	37.6	55
	100	49	51	2nd	90	37	75
	88	52	36	Post	88.2	34.2	106
5	105	59	46	Pre.	101.4	38.2	0
	73	55	18	1st	91.4	32.2	23
	85	60	25	Int.	84.2	21.4	53
	79	58	21	2nd	92.6	31.8	70
	90	60	30	Post	92.2	31.4	95
6	154	81	73	Pre.	147	62.6	0
	122	88	34	1st	142.2	57	23
	150	64	86	Int.	136.6	69.4	51
	128	99	29	2nd	138.8	61.8	67
	137	86	51	Post	133.2	59.6	94
7	114	83	31	Pre.	123.6	37.6	0
	152	85	67	1st	128	42.6	20
	122	86	36	Int.	128.2	40.2	52
	127	72	55	2nd	122.6	43.6	75
	121	80	41	Post	122	47.6	105
8	139	75	64	Pre.	124.8	54.8	0
	122	71	51	1st	114	40.4	19
	107	66	41	Int.	110.2	34.2	52
	108	70	38	2nd	115.2	45.6	72
	111	68	43	Post	118.2	44.2	101

9	Pre.	98	72	26	94	71	23	109	69	40	89	71	18	
	1st	110	68	42	95	71	24	127	69	58	103	79	24	
	Int.	114	71	43	101	81	20	92	78	14	94	68	26	
	2nd	95	62	33	91	68	23	116	80	36			0	
	Post	118	82	36	114	80	34	115	80	35	116	80	36	
Subj.	Sess.								Reading					
			1			2			3			4		
		Sys.	Dia.	PP	Sys.	Dia	PP	Sys.	Dia	PP	Sys.	Dia	PP	
10	Pre.	120	89	31	119	78	41	121	80	41	127	82	45	
	1st	122	83	39	132	92	40	131	92	39	138	89	49	
	Int.	111	83	28	125	83	42	122	80	42	119	78	41	
	2nd	132	96	36	130	85	45	135	87	48	131	88	43	
	Post	120	86	34	117	87	30	121	80	41	121	83	38	
11	Pre.	136	85	51	110	84	26	145	118	27	128	81	47	
	1st	114	81	33	118	73	45	115	70	45	119	67	52	
	Int.	121	75	46	125	75	50	111	73	38	113	70	43	
	2nd	118	78	40	113	72	41	108	70	38	115	63	52	
	Post	109	76	33	121	70	51	124	77	47	119	73	46	
12	Pre.	88	68	20	103	70	33	92	68	24	105	66	39	
	1st	91	70	21	96	68	28	94	65	29	104	61	43	
	Int.	82	72	10	94	72	22	83	72	11	114	66	48	
	2nd	88	71	17	109	63	46	110	61	49	82	66	16	
	Post	86	65	21	99	63	36	109	62	47	95	66	29	
13	Pre.	118	83	35	136	84	52	121	76	45	124	93	31	
	1st	125	83	42	139	74	65	119	74	45	113	77	36	
	Int.	123	84	39	120	80	40	118	76	42	145	78	67	
	2nd	124	84	40	113	77	36	118	75	43	99	78	21	
	Post	123	75	48	113	77	36	113	78	35	107	76	31	
14	Pre.	99	72	27	110	72	38	105	75	30	104	77	27	
	1st	118	65	53	145	112	33	120	69	51	105	74	31	
	Int.	118	67	51	112	60	52	117	64	53	123	59	64	
	2nd	118	68	50	101	72	29	96	66	30	123	58	65	
	Post	111	58	53	108	54	54	121	58	63	122	56	66	
15	Pre.	121	80	41	99	64	35	91	53	38	102	53	49	
	1st	119	50	69	152	95	57	123	48	75	79	53	26	
	Int.	120	62	58	134	48	86	154	55	99	101	59	42	
	2nd	111	48	63	115	48	67	123	48	75	118	44	74	
	Post	119	51	68	124	61	63	131	53	78	131	48	83	
16	Pre.	98	60	38	93	57	36	86	62	24	85	57	28	
	1st	96	69	27	82	63	19	87	67	20	84	62	22	
	Int.	86	61	25	88	65	23	88	62	26	82	56	26	
	2nd	89	65	24	93	59	34	78	53	25	84	56	28	
	Post	83	54	29	85	57	28	94	57	37	89	60	29	
17	Pre.	132	109	23	148	99	49	151	97	54	149	96	53	
	1st	135	100	35	152	101	51	153	102	51	138	94	44	

9	101	63	38	Pre.	98.2	29	0
	142	71	71	1st	115.4	43.8	26
	104	72	32	Int.	101	27	59
	116	80	36	2nd	104.5	32	101
	107	86	21	Post	114	32.4	131
Subj.				Sess.			
		5			Mean	Mean	Time
	Sys.	Dia	PP		Sys.	PP	Mins
10	122	86	36	Pre.	121.8	38.8	0
	135	91	44	1st	131.6	42.2	27
	123	74	49	Int.	120	40.4	55
	130	91	39	2nd	131.6	42.2	70
	116	79	37	Post	119	36	97
11	124	74	50	Pre.	128.6	40.2	0
	120	73	47	1st	117.2	44.4	21
	121	70	51	Int.	118.2	45.6	49
	108	72	36	2nd	112.4	41.4	64
	115	70	45	Post	117.6	44.4	88
12	93	63	30	Pre.	96.2	29.2	0
	113	63	50	1st	99.6	34.2	22
	104	68	36	Int.	95.4	25.4	52
	91	66	25	2nd	96	30.6	75
	105	63	42	Post	98.8	35	108
13	112	75	37	Pre.	122.2	40	0
	102	76	26	1st	119.6	42.8	23
	117	73	44	Int.	124.6	46.4	57
	108	80	28	2nd	112.4	33.6	78
	102	81	21	Post	111.6	34.2	108
14	127	71	56	Pre.	109	35.6	0
	103	62	41	1st	118.2	41.8	18
	127	60	67	Int.	119.4	57.4	43
	122	65	57	2nd	112	46.2	61
	96	59	37	Post	111.6	54.6	87
15	125	49	76	Pre.	107.6	47.8	0
	125	78	47	1st	119.6	54.8	29
	96	49	47	Int.	121	66.4	59
	118	46	72	2nd	117	70.2	82
	126	56	70	Post	126.2	72.4	112
16	79	57	22	Pre.	88.2	29.6	0
	83	68	15	1st	86.4	20.6	22
	80	63	17	Int.	84.8	23.4	51
	87	59	28	2nd	86.2	27.8	79
	81	66	15	Post	86.4	27.6	109
17	148	105	43	Pre.	145.6	44.4	0
	152	102	50	1st	146	46.2	20

	Int.	147	109	38	126	91	35	147	108	39	133	93	40	
	2nd	150	102	48	119	99	20	128	96	32	132	105	27	
	Post	152	106	46	147	113	34	149	113	36	135	112	23	
18	Pre.	102	75	27	104	70	34	106	63	43	102	59	43	
	1st	110	65	45	114	73	41	96	68	28	102	72	30	
	Int.	118	110	8	91	74	17	105	77	28	98	64	34	
	2nd	115	101	14	114	61	53	107	65	42	114	70	44	
	Post	137	71	66	119	70	49	112	73	39	127	70	57	
Subj.	Sess.								Reading					
			1			2			3			4		
		Sys.	Dia.	PP	Sys.	Dia	PP	Sys.	Dia	PP	Sys.	Dia	PP	
19	Pre.	123	73	50	121	79	42	117	78	39	93	63	30	
	1st	94	79	15	120	69	51	99	66	33	107	67	40	
	Int.	109	76	33	97	75	22	108	70	38	89	56	33	
	2nd	95	66	29	99	72	27	117	57	60	94	72	22	
	Post	122	68	54	100	68	32	113	61	52	108	66	42	
20	Pre.	125	103	22	121	63	58	97	72	25	113	72	41	
	1st	104	70	34	101	63	38	97	73	24	133	116	17	
	Int.	74	62	12	104	63	41	92	65	27	100	68	32	
	2nd	91	63	28	120	64	56	93	69	24	107	56	51	
	Post	107	55	52	137	60	77	133	96	37	96	55	41	
21	Pre.	109	70	39	88	60	28	99	62	37	87	60	27	
	1st	89	75	14	102	65	37	91	65	26	93	66	27	
	Int.	112	91	21	117	77	40	96	61	35	81	66	15	
	2nd	91	67	24	98	64	34	94	62	32	89	57	32	
	Post	117	74	43	93	63	30	100	71	29	102	63	39	
22	Pre.	95	85	10	100	76	24	97	84	13	88	76	12	
	1st	93	88	5	107	77	30	90	80	10	102	76	26	
	Int.	93	73	20	112	90	22	107	99	8	129	112	17	
	2nd	102	83	19	95	88	7			0			0	
	Post			0			0			0			0	
23	Pre.	105	59	46	90	57	33	129	57	72	96	61	35	
	1st	92	65	27	122	55	67	84	55	29	113	62	51	
	Int.	98	56	42	124	60	64	97	75	22	69	56	13	
	2nd	117	59	58	137	109	28	105	71	34	117	64	53	
	Post	150	67	83	110	68	42	147	57	90	104	66	38	
24	Pre.	152	78	74	154	80	74	138	78	60	142	80	62	
	1st	140	80	60	140	82	58	138	82	56	138	80	58	
	Int.	140	80	60	132	80	52	132	80	52	132	88	44	
	2nd	138	84	54	138	82	56	134	82	52	132	82	50	
	Post	148	90	58	140	80	60	130	80	50	142	90	52	
25	Pre.	110	72	38	110	72	38	110	74	36	118	74	44	
	1st	130	88	42	138	96	42	142	82	60	142	88	54	
	Int.	140	92	48	150	100	50	140	82	58	142	82	60	
	2nd	132	88	44	138	88	50	140	98	42	142	90	52	

	144	88	56	Int.	139.4	41.6	52
	141	94	47	2nd	134	34.8	70
	152	105	47	Post	147	37.2	103
18	98	63	35	Pre.	102.4	36.4	0
	97	75	22	1st	103.8	33.2	31
	122	73	49	Int.	106.8	27.2	75
	119	74	45	2nd	113.8	39.6	98
	101	76	25	Post	119.2	47.2	125
Subj.				Sess.			
		5			Mean	Mean	Time
	Sys.	Dia	PP		Sys.	PP	Mins
19	117	74	43	Pre.	114.2	40.8	0
	112	65	47	1st	106.4	37.2	24
	96	66	30	Int.	99.8	31.2	51
	93	63	30	2nd	99.6	33.6	67
	107	62	45	Post	110	45	94
20	109	70	39	Pre.	113	37	0
	111	68	43	1st	109.2	31.2	20
	92	53	39	Int.	92.4	30.2	31
	93	50	43	2nd	100.8	40.4	48
	93	56	37	Post	113.2	48.8	74
21	116	69	47	Pre.	99.8	35.6	0
	88	52	36	1st	92.6	28	17
	86	66	20	Int.	98.4	26.2	46
	96	65	31	2nd	93.6	30.6	66
	104	65	39	Post	103.2	36	91
22	106	76	30	Pre.	97.2	17.8	0
	86	80	6	1st	95.6	15.4	19
	101	96	5	Int.	108.4	14.4	54
			0	2nd	98.5	13	85
			0	Post	0	0	124
23	89	64	25	Pre.	101.8	42.2	0
	104	52	52	1st	103	45.2	22
	135	53	82	Int.	104.6	44.6	57
	152	122	30	2nd	125.6	40.6	81
	117	75	42	Post	125.6	59	108
24	140	80	60	Pre.	145.2	66	0
	136	76	60	1st	138.4	58.4	33
	138	88	50	Int.	134.8	51.6	77
	130	82	48	2nd	134.4	52	108
	140	82	58	Post	140	55.6	147
25	108	72	36	Pre.	111.2	38.4	0
	144	82	62	1st	139.2	52	45
	132	92	40	Int.	140.8	51.2	82
	142	82	60	2nd	138.8	49.6	113

	Post	140	92	48	140	92	48	142	90	52	142	96	46
26	Pre.	110	82	28	110	78	32	112	76	36	110	80	30
	1st	108	80	28	108	76	32	102	76	26	100	76	24
	Int.	106	80	26	102	78	24	104	78	26	104	74	30
	2nd	106	80	26	106	82	24	106	78	28	104	82	22
	Post	110	84	26	104	78	26	106	84	22	108	78	30
27	Pre.	120	74	46	124	72	52	120	72	48	118	72	46
	1st	118	72	46	118	82	36	118	74	44	118	70	48
	Int.	108	66	42	112	68	44	116	72	44	118	76	42
	2nd	112	76	36	120	78	42	118	78	40	124	78	46
	Post	114	74	40	116	76	40	116	76	40	122	78	44
Subj.	Sess.								Reading				
			1			2			3			4	
		Sys.	Dia.	PP	Sys.	Dia	PP	Sys.	Dia	PP	Sys.	Dia	PP
28	Pre.	122	74	48	128	70	58	128	72	56	128	72	56
	1st	124	72	52	116	70	46	130	76	54	122	70	52
	Int.	128	64	64	128	76	52	126	68	58	124	76	48
	2nd	118	70	48	110	72	38	108	70	38	110	70	40
	Post	114	72	42	120	72	48	118	72	46	122	78	44
29	Pre.	130	78	52	128	78	50	126	82	44	132	80	52
	1st	132	78	54	130	70	60	128	76	52	138	76	62
	Int.	138	78	60	136	70	66	132	68	64	136	72	64
	2nd	132	78	54	132	74	58	126	76	50	122	78	44
	Post	132	78	54	132	80	52	134	76	58	128	78	50
30	Pre.	106	80	26	110	78	32	108	76	32	106	76	30
	1st	112	78	34	114	78	36	108	78	30	114	78	36
	Int.	110	82	28	114	86	28	114	80	34	112	84	28
	2nd	112	84	28	112	84	28	112	78	34	108	80	28
	Post	112	82	30	108	74	34	110	76	34	110	76	34
31	Pre.	136	76	60	136	76	60	134	72	62	136	70	66
	1st	140	76	64	134	74	60	134	74	60	132	72	60
	Int.	136	78	58	128	72	56	124	68	56	128	68	60
	2nd	128	74	54	132	74	58	132	72	60	134	74	60
	Post	142	78	64	134	78	56	148	78	70	150	82	68
32	Pre.	138	80	58	128	80	48	132	84	48	136	80	56
	1st	126	80	46	120	76	44	120	76	44	118	80	38
	Int.	118	78	40	120	80	40	114	76	38	116	78	38
	2nd	122	74	48	120	74	46	112	78	34	122	78	44
	Post	122	84	38	120	80	40	116	80	36	118	78	40
33	Pre.	140	78	62	136	78	58	138	78	60	140	80	60
	1st	136	78	58	138	80	58	138	76	62	138	76	62
	Int.	130	78	52	138	76	62	138	76	62	140	76	64
	2nd	134	72	62	138	72	66	132	68	64	130	72	58
	Post	132	72	60	134	72	62	132	70	62	136	66	70
34	Pre.	126	78	48	126	72	54	124	72	52	124	70	54

	144	96	48	Post	141.6	48.4	147
26	110	76	34	Pre.	110.4	32	0
	104	76	28	1st	104.4	27.6	35
	106	74	32	Int.	104.4	27.6	78
	104	82	22	2nd	105.2	24.4	101
	104	80	24	Post	106.4	25.6	140
27	120	78	42	Pre.	120.4	46.8	0
	120	72	48	1st	118.4	44.4	25
			0	Int.	113.5	43	60
	118	76	42	2nd	118.4	41.2	83
	118	82	36	Post	117.2	40	116
Subj.				Sess.			
		5			Mean	Mean	Time
	Sys.	Dia	PP		Sys.	PP	Mins
28	132	74	58	Pre.	127.6	55.2	0
	128	70	58	1st	124	52.4	26
	124	68	56	Int.	126	55.6	61
	112	68	44	2nd	111.6	41.6	90
	112	72	40	Post	117.2	44	121
29	128	76	52	Pre.	128.8	50	0
	136	76	60	1st	132.8	57.6	26
	130	78	52	Int.	134.4	61.2	56
	126	72	54	2nd	127.6	52	80
	138	74	64	Post	132.8	55.6	110
30	108	76	32	Pre.	107.6	30.4	0
	110	78	32	1st	111.6	33.6	21
	108	80	28	Int.	111.6	29.2	51
	112	78	34	2nd	111.2	30.4	69
	106	78	28	Post	109.2	32	97
31	132	72	60	Pre.	134.8	61.6	0
	134	72	62	1st	134.8	61.2	30
	126	68	58	Int.	128.4	57.6	69
	136	74	62	2nd	132.4	58.8	89
	148	88	60	Post	144.4	63.6	117
32	136	80	56	Pre.	134	53.2	0
	116	80	36	1st	120	41.6	55
	112	74	38	Int.	116	38.8	108
	118	78	40	2nd	118.8	42.4	156
	120	78	42	Post	119.2	39.2	188
33	142	78	64	Pre.	139.2	60.8	0
	138	76	62	1st	137.6	60.4	22
	138	76	62	Int.	136.8	60.4	57
	132	74	58	2nd	133.2	61.6	85
	138	68	70	Post	134.4	64.8	128
34	122	70	52	Pre.	124.4	52	0

	1st	130	76	54	128	70	58	120	72	48	122	68	54
	Int.	124	80	44	124	72	52	118	68	50	120	70	50
	2nd	118	78	40	120	64	56	118	68	50	118	68	50
	Post	122	64	58	122	70	52	116	64	52	122	72	50
35	Pre.	144	72	72	130	74	56	126	78	48	124	72	52
	1st	126	62	64	126	60	66	130	64	66	130	64	66
	Int.	126	58	68	130	62	68	124	66	58	128	66	62
	2nd	136	70	66	128	72	56	128	74	54	120	62	58
	Post	120	60	60	124	72	52	118	69	49	120	66	54
36	Pre.	138	80	58	136	80	56	132	82	50	126	86	40
	1st	132	82	50	130	84	46	132	86	46	136	84	52
	Int.	126	82	44	126	78	48	124	76	48	126	68	58
	2nd	128	78	50	128	78	50	122	82	40	120	82	38
	Post	122	86	36	122	86	36	126	82	44	124	82	42
Subj.	Sess.								Reading				
			1			2			3			4	
		Sys.	Dia.	PP	Sys.	Dia	PP	Sys.	Dia	PP	Sys.	Dia	PP
37	Pre.	112	74	38	118	74	44	112	74	38	120	72	48
	1st	104	70	34	108	70	38	110	70	40	106	70	36
	Int.	106	69	37	106	68	38	100	70	30	106	66	40
	2nd	102	70	32	104	68	36	102	68	34	106	68	38
	Post	102	68	34	102	68	34	102	68	34	104	78	26
38	Pre.	152	92	60	146	88	58	148	86	62	152	92	60
	1st	146	89	57	150	96	54	154	88	66	156	92	64
	Int.	142	92	50	150	92	58	148	92	56	150	92	58
	2nd	148	88	60	146	90	56	148	92	56	154	96	58
	Post	150	92	58	156	98	58	144	90	54	140	92	48
39	Pre.	122	68	54	122	72	50	118	70	48	120	68	52
	1st	106	70	36	112	68	44	112	66	46	106	66	40
	Int.	100	68	32	102	62	40	102	62	40	112	66	46
	2nd	108	70	38	108	74	34	114	74	40	106	72	34
	Post	112	74	38	100	64	36	102	68	34	100	70	30
40	Pre.	132	84	48	138	88	50	142	90	52	138	88	50
	1st	134	90	44	136	88	48	134	88	46	138	90	48
	Int.	128	82	46	130	78	52	134	88	46	130	84	46
	2nd	126	78	48	128	78	50	126	80	46	130	82	48
	Post	132	92	40	128	86	42	122	78	44	126	84	42
41	Pre.	178	90	88	170	98	72	176	92	84	168	82	86
	1st	166	92	74	166	94	72	168	88	80	162	96	66
	Int.	146	98	48	156	92	64	154	94	60	150	90	60
	2nd	158	96	62	148	96	52	154	98	56	156	100	56
	Post	148	98	50	148	96	52	146	98	48	142	96	46
42	Pre.	152	88	64	148	92	56	148	84	64	146	84	62
	1st	144	90	54	144	88	56	144	90	54	142	88	54
	Int.	136	84	52	144	88	56	138	86	52	142	86	56

	118	70	48	1st	123.6	52.4	29
	110	68	42	Int.	119.2	47.6	74
	118	66	52	2nd	118.4	49.6	98
	122	68	54	Post	120.8	53.2	138
35	124	78	46	Pre.	129.6	54.8	0
	122	76	46	1st	126.8	61.6	35
	130	66	64	Int.	127.6	64	83
	126	68	58	2nd	127.6	58.4	106
	122	74	48	Post	120.8	52.6	140
36	130	80	50	Pre.	132.4	50.8	0
	126	82	44	1st	131.2	47.6	24
	122	78	44	Int.	124.8	48.4	56
	122	80	42	2nd	124	44	79
	124	82	42	Post	123.6	40	103
Subj.				Sess.			
		5			Mean	Mean	Time
	Sys.	Dia	PP		Sys.	PP	Mins
37	110	70	40	Pre.	114.4	41.6	0
	106	68	38	1st	106.8	37.2	30
	106	66	40	Int.	104.8	37	69
	102	68	34	2nd	103.2	34.8	91
	104	72	32	Post	102.8	32	121
38	146	92	54	Pre.	148.8	58.8	0
	158	90	68	1st	152.8	61.8	29
	150	96	54	Int.	148	55.2	63
	152	92	60	2nd	149.6	58	83
	134	86	48	Post	144.8	53.2	115
39	118	68	50	Pre.	120	50.8	0
	100	60	40	1st	107.2	41.2	26
	108	68	40	Int.	104.8	39.6	67
	106	74	32	2nd	108.4	35.6	89
	110	70	40	Post	104.8	35.6	124
40	138	78	60	Pre.	137.6	52	0
	130	90	40	1st	134.4	45.2	23
	124	78	46	Int.	129.2	47.2	62
	136	78	58	2nd	129.2	50	95
	122	82	40	Post	126	41.6	135
41	168	88	80	Pre.	172	82	0
	166	92	74	1st	165.6	73.2	29
	156	98	58	Int.	152.4	58	64
	148	98	50	2nd	152.8	55.2	90
	148	96	52	Post	146.4	49.6	120
42	146	88	58	Pre.	148	60.8	0
	142	86	56	1st	143.2	54.8	25
	142	82	60	Int.	140.4	55.2	58

	2nd	138	86	52	142	90	52	134	86	48	142	86	56	
	Post	142	90	52	140	88	52	138	86	52	140	92	48	
43	Pre.	126	82	44	126	76	50	120	78	42	122	76	46	
	1st	126	74	52	130	80	50	128	78	50	126	76	50	
	Int.	124	72	52	124	68	56	120	68	52	126	74	52	
	2nd	122	70	52	124	70	54	130	70	60	124	76	48	
	Post	120	70	50	124	70	54	126	72	54	126	78	48	

	144	92	52	2nd	140	52	78
	140	86	54	Post	140	51.6	113
43	126	74	52	Pre.	124	46.8	0
	130	74	56	1st	128	51.6	21
	120	70	50	Int.	122.8	52.4	57
	122	76	46	2nd	124.4	52	76
	126	72	54	Post	124.4	52	105

Heart Rate (HR)							
				Reading			
Subject	Session	1	2	3	4	5	Mean
1	Pre-Exposure	86	86	78	81	86	83.4
	First Exposure	77	81	81	76	77	78.4
	Interval	70	72	78	73	73	73.2
	Second Exposure	77	80	74	71	74	75.2
	Post Exposure	69	73	70	70	64	69.2
2	Pre-Exposure	96	82	92	74	76	84
	First Exposure	87	79	79	77	81	80.6
	Interval	80	80	88	73	76	79.4
	Second Exposure	76	78	71	71	74	74
	Post Exposure	75	66	70	78	75	72.8
3	Pre-Exposure	86	81	84	83	87	84.2
	First Exposure	78	89	83	86	83	83.8
	Interval	80	83	82	76	76	79.4
	Second Exposure	89	83	78	70	77	79.4
	Post Exposure	80	79	76	80	83	79.6
4	Pre-Exposure	84	80	83	81	84	82.4
	First Exposure	88	82	81	81	87	83.8
	Interval	81	79	76	76	81	78.6
	Second Exposure	77	78	79	76	71	76.2
	Post Exposure	73	78	66	69	86	74.4
5	Pre-Exposure	73	60	71	70	77	70.2
	First Exposure	64	73	73	73	69	70.4
	Interval	71	76	79	76	69	74.2
	Second Exposure	67	68	60	71	69	67
	Post Exposure	72	67	66	73	81	71.8
6	Pre-Exposure	76	84	78	77	76	78.2
	First Exposure	76	75	76	75	75	75.4
	Interval	69	71	72	70	72	70.8
	Second Exposure	68	74	66	68	67	68.6
	Post Exposure	64	66	65	62	66	64.6
7	Pre-Exposure	83	82	80	78	72	79
	First Exposure	75	60	74	130	71	82
	Interval	71	76	68	71	74	72
	Second Exposure	70	70	75	67	71	70.6
	Post Exposure	67	70	64	66	65	66.4
8	Pre-Exposure	89	97	83	92	83	88.8
	First Exposure	83	71	83	93	83	82.6
	Interval	78	88	83	82	76	81.4
	Second Exposure	80	81	78	80	79	79.6
	Post Exposure	74	77	82	78	82	78.6
9	Pre-Exposure	63	76	63	54	58	62.8
	First Exposure	59	61	55	61	74	62

	Interval	67	61	61	68	56	62.6
	Second Exposure	55	60	66	-	50	57.75
	Post Exposure	64	65	64	65	70	65.6
Subject	Session	1	2	3	4	5	Mean
10	Pre-Exposure	89	90	83	86	82	86
	First Exposure	84	88	87	102	88	89.8
	Interval	81	76	76	80	77	78
	Second Exposure	89	89	80	84	87	85.8
	Post Exposure	78	82	82	80	79	80.2
11	Pre-Exposure	82	83	76	77	76	78.8
	First Exposure	76	78	75	78	75	76.4
	Interval	76	69	69	71	73	71.6
	Second Exposure	72	74	74	69	73	72.4
	Post Exposure	71	69	66	70	68	68.8
12	Pre-Exposure	70	66	70	73	70	69.8
	First Exposure	69	69	71	71	64	68.8
	Interval	70	69	78	63	62	68.4
	Second Exposure	71	70	68	71	69	69.8
	Post Exposure	56	64	63	60	66	61.8
13	Pre-Exposure	64	67	66	76	68	68.2
	First Exposure	61	69	67	67	64	65.6
	Interval	64	64	69	66	66	65.8
	Second Exposure	69	65	64	64	62	64.8
	Post Exposure	61	68	55	62	65	62.2
14	Pre-Exposure	70	64	68	69	66	67.4
	First Exposure	66	74	65	68	72	69
	Interval	63	66	66	63	61	63.8
	Second Exposure	71	62	61	62	61	63.4
	Post Exposure	56	62	60	57	57	58.4
15	Pre-Exposure	66	63	68	66	62	65
	First Exposure	62	64	62	64	63	63
	Interval	69	60	57	58	60	60.8
	Second Exposure	55	56	54	52	55	54.4
	Post Exposure	49	52	49	54	54	51.6
16	Pre-Exposure	71	71	71	77	69	71.8
	First Exposure	72	76	66	71	66	70.2
	Interval	71	65	64	75	65	68
	Second Exposure	58	56	53	58	58	56.6
	Post Exposure	56	56	60	56	62	58
17	Pre-Exposure	89	66	66	65	74	72
	First Exposure	69	74	78	58	67	69.2

	Interval	68	63	76	69	68	68.8
	Second Exposure	63	62	68	70	61	64.8
	Post Exposure	62	78	68	67	71	69.2
18	Pre-Exposure	62	73	68	56	54	62.6
	First Exposure	58	64	55	56	63	59.2
	Interval	51	59	70	65	51	59.2
	Second Exposure	56	52	58	76	53	59
	Post Exposure	69	48	56	55	65	58.6
Subject	Session	1	2	3	4	5	Mean
19	Pre-Exposure	78	86	90	76	82	82.4
	First Exposure	94	74	82	71	72	78.6
	Interval	77	75	73	71	76	74.4
	Second Exposure	56	68	72	72	77	69
	Post Exposure	75	76	70	78	66	73
20	Pre-Exposure	58	69	74	77	80	71.6
	First Exposure	61	70	57	59	71	63.6
	Interval	67	66	60	61	62	63.2
	Second Exposure	72	60	54	68	66	64
	Post Exposure	54	54	96	45	56	61
21	Pre-Exposure	70	68	68	66	75	69.4
	First Exposure	60	64	69	72	62	65.4
	Interval	67	63	64	61	68	64.6
	Second Exposure	60	60	64	59	61	60.8
	Post Exposure	58	66	60	61	62	61.4
22	Pre-Exposure	71	69	73	76	72	72.2
	First Exposure	67	68	63	72	68	67.6
	Interval	66	77	69	79	65	71.2
	Second Exposure	65	66	-	-	-	65.5
	Post Exposure	-	-	-	-	-	-
23	Pre-Exposure	69	71	82	86	63	74.2
	First Exposure	77	61	72	77	82	73.8
	Interval	81	79	68	68	69	73
	Second Exposure	76	77	52	77	62	68.8
	Post Exposure	77	83	60	78	92	78
24	Pre-Exposure	52	48	52	48	52	50.4
	First Exposure	48	52	48	52	52	50.4
	Interval	48	48	52	44	48	48
	Second Exposure	48	44	48	48	48	47.2
	Post Exposure	44	44	44	44	44	44

25	Pre-Exposure	60	68	64	68	60	64
	First Exposure	72	64	60	64	64	64.8
	Interval	72	60	64	64	60	64
	Second Exposure	80	68	68	68	68	70.4
	Post Exposure	64	72	68	68	68	68
26	Pre-Exposure	68	68	68	68	68	68
	First Exposure	68	68	68	68	68	68
	Interval	64	68	68	68	68	67.2
	Second Exposure	68	68	68	68	64	67.2
	Post Exposure	68	60	60	68	68	64.8
27	Pre-Exposure	84	84	84	72	76	80
	First Exposure	68	72	80	80	76	75.2
	Interval	76	76	84	68	77	76.2
	Second Exposure	77	68	84	72	72	74.6
	Post Exposure	61	72	76	60	80	69.8
Subject	Session	1	2	3	4	5	Mean
28	Pre-Exposure	60	64	60	60	60	60.8
	First Exposure	60	60	60	56	56	58.4
	Interval	56	52	60	56	56	56
	Second Exposure	60	60	52	52	52	55.2
	Post Exposure	52	56	52	56	52	53.6
29	Pre-Exposure	84	84	84	84	88	84.8
	First Exposure	80	84	88	92	80	84.8
	Interval	80	76	84	80	76	79.2
	Second Exposure	84	88	84	80	88	84.8
	Post Exposure	84	80	84	80	76	80.8
30	Pre-Exposure	72	72	72	68	68	70.4
	First Exposure	72	72	76	76	72	73.6
	Interval	64	72	68	68	64	67.2
	Second Exposure	76	76	68	72	72	72.8
	Post Exposure	64	72	76	68	68	69.6
31	Pre-Exposure	72	64	72	72	80	72
	First Exposure	76	76	64	80	76	74.4
	Interval	77	72	76	80	76	76.2
	Second Exposure	74	76	76	80	80	77.2
	Post Exposure	80	80	80	80	80	80
32	Pre-Exposure	76	76	80	76	76	76.8
	First Exposure	76	64	76	72	76	72.8
	Interval	72	68	72	72	76	72
	Second Exposure	68	68	68	72	72	69.6
	Post Exposure	72	72	72	72	72	72

33	Pre-Exposure	68	68	68	68	68	68
	First Exposure	68	68	68	64	64	66.4
	Interval	76	64	68	60	68	67.2
	Second Exposure	60	60	68	60	64	62.4
	Post Exposure	64	68	64	68	68	66.4
34	Pre-Exposure	76	72	76	72	68	72.8
	First Exposure	72	72	72	72	76	72.8
	Interval	72	72	80	76	72	74.4
	Second Exposure	76	76	76	80	76	76.8
	Post Exposure	76	84	76	76	72	76.8
35	Pre-Exposure	80	72	72	76	72	74.4
	First Exposure	76	84	76	72	72	76
	Interval	64	72	72	76	72	71.2
	Second Exposure	68	76	68	72	76	72
	Post Exposure	68	60	60	64	64	63.2
36	Pre-Exposure	76	72	76	80	72	75.2
	First Exposure	64	72	76	72	64	69.6
	Interval	64	68	60	68	64	64.8
	Second Exposure	64	60	68	60	64	63.2
	Post Exposure	60	60	60	60	68	61.6
Subject	Session	1	2	3	4	5	Mean
37	Pre-Exposure	80	76	76	80	80	78.4
	First Exposure	88	84	80	80	78	82
	Interval	80	76	80	72	80	77.6
	Second Exposure	76	76	72	76	68	73.6
	Post Exposure	80	76	80	76	80	78.4
38	Pre-Exposure	64	68	68	60	68	65.6
	First Exposure	72	68	68	60	64	66.4
	Interval	64	64	64	64	60	63.2
	Second Exposure	64	64	64	64	64	64
	Post Exposure	64	60	60	64	68	63.2
39	Pre-Exposure	76	80	84	76	76	78.4
	First Exposure	68	72	76	72	72	72
	Interval	64	68	68	72	68	68
	Second Exposure	68	60	68	64	68	65.6
	Post Exposure	64	64	60	68	68	64.8
40	Pre-Exposure	76	72	68	68	64	69.6
	First Exposure	60	60	60	60	56	59.2
	Interval	56	60	60	64	64	60.8
	Second Exposure	60	56	60	60	60	59.2
	Post Exposure	56	60	56	64	56	58.4

41	Pre-Exposure	76	68	68	72	64	69.6
	First Exposure	68	72	68	68	68	68.8
	Interval	64	64	64	68	64	64.8
	Second Exposure	64	60	60	64	64	62.4
	Post Exposure	60	60	60	64	60	60.8
42	Pre-Exposure	56	60	60	60	60	59.2
	First Exposure	64	60	60	60	56	60
	Interval	60	60	60	60	60	60
	Second Exposure	56	56	56	56	60	56.8
	Post Exposure	56	56	56	56	56	56
43	Pre-Exposure	56	56	56	56	60	56.8
	First Exposure	56	56	56	64	60	58.4
	Interval	60	60	56	56	56	57.6
	Second Exposure	56	40	56	52	48	50.4
	Post Exposure	48	48	52	56	52	51.2

Experiment Three: Gender and Melatonin Levels				
Difference in Melatonin levels between Exposure and Control				
Night				
Female	Female	Male	Male	
Subject No.	Mel. Diff.(pg/m	Subject No.	Mel. Diff.(pg.ml)	
1	-1.5	4	-1.7	
2	-5.3	6	-2.7	
3	0.8	7	-1.0	
5	-0.8	10	0.0	
8	13.2	11	0.0	
9	-0.2	14	-3.3	
12	1.2	15	0.2	
13	0.2	17	1.3	
16	-3.0	20	-0.3	
18	-8.7	24	-1.2	
19	5.0	28	-2.2	
21	-5.0	32	-4.0	
22	-0.7	35	-1.5	
23	-1.2	38	-0.7	
25	-1.2	41	0.0	
26	4.5	42	-11.2	
27	-5.7			
29	4.5			
30	-7.2			
31	-2.3			
33	4.0			
34	-5.3			
36	-1.8			
37	-3.2			
39	-2.3			
40	5.2			
43	3.2			
N=43				
Males=16				
Females=27				
Mean	-0.50		-1.77	
SD	4.677		2.879	
SEM	0.900		0.720	
t=	0.5555		2.4592	

The Effects of Cellphone Frequency EMFs on Salivary Melatonin

(concentration in pg/ml)

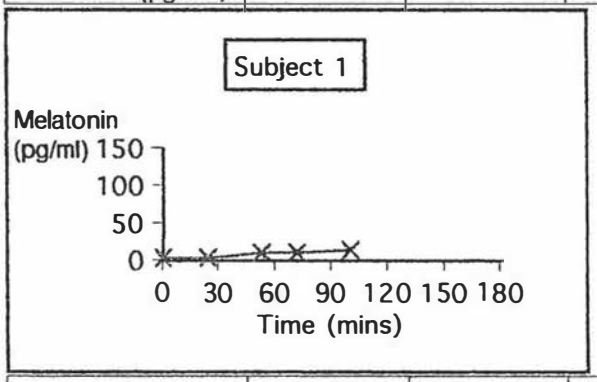
		Control Samples		Exposure Samples						
Subject	Time	Pre-Exp	Time	Exp.1	Time	Interval	Time	Exp. 2	Time	Pos-Exp
1	0	3.0	24	5.0	53	10.0	72	10.0	100	14.0
2	0	13.0	17	13.0	48	23.0	68	21.0	97	31.0
3	0	10.0	29	18.0	58	20.0	81	19.0	111	23.0
4	0	13.0	26	11.0	56	11.0	76	5.0	108	5.0
5	0	21.0	27	27.0	56	36.0	72	36.0	97	40.0
6	0	34.0	22	29.0	51	35.0	67	43.0	94	47.0
7	0	19.0	21	17.0	53	19.0	75	19.0	105	19.0
8	0	9.0	16	21.0	51	5.0	70	18.0	97	5.0
9	0	0.0	31	4.0	65	9.0	111	15.0	136	20.0
10	0	0.0	17	0.0	44	0.0	58	0.0	85	0.0
11	0	0.0	27	0.0	50	0.0	65	0.0	89	0.0
12	0	7.0	15	12.0	48	23.0	68	29.0	101	28.0
13	0	25.0	22	29.0	55	32.0	77	34.0	106	37.0
14	0	25.0	18	21.0	43	21.0	59	13.0	87	15.0
15	0	8.0	31	8.0	59	15.0	83	15.0	113	11.0
16	0	5.0	21	0.0	53	4.0	78	8.0	111	12.0
17	0	8.0	18	10.0	50	17.0	72	22.0	100	19.0
18	0	51.0	30	19.0	74	9.0	96	11.0	123	11.0
19	0	57.0	21	66.0	48	67.0	65	78.0	91	77.0
20	0	26.0	21	34.0	50	31.0	66	26.0	93	34.0
21	0	6.0	18	4.0	47	8.0	66	12.0	90	25.0
22	0	27.0	22	21.0	68	19.0	96	27.0	121	28.0
23	0	13.0	23	13.0	65	16.0	79	18.0	108	21.0
24	0	2.0	36	2.0	76	2.0	107	5.0	145	10.0
25	0	16.0	43	18.0	80	24.0	119	25.0	152	28.0
26	0	27.0	34	41.0	74	44.0	100	56.0	137	61.0
27	0	34.0	23	16.0	57	12.0	80	18.0	114	22.0
28	0	2.0	26	0.0	61	4.0	90	9.0	119	14.0
29	0	21.0	22	27.0	52	32.0	76	42.0	105	37.0
30	0	48.0	23	35.0	52	19.0	70	16.0	99	31.0
31	0	0.00	30	2.00	69	11.00	87	16.00	116	23.00
32	0	17.00	48	20.00	103	27.00	147	26.00	180	37.00
33	0	10.00	25	17.00	61	27.00	98	39.00	128	35.00
34	0	1.00	39	2.00	75	19.00	101	24.00	139	35.00
35	0	2.00	36	0.00	80	2.00	103	7.00	137	11.00
36	0	5.00	21	8.00	54	20.00	76	39.00	110	51.00
37	0	30.00	30	36.00	67	45.00	89	45.00	118	56.00
38	0	0.00	26	0.00	60	0.00	79	0.00	112	2.00
39	0	30.00	24	33.00	65	53.00	87	57.00	121	59.00
40	0	17.00	23	28.00	62	29.00	98	45.00	134	48.00

41	0	0.00	29	0.00	64	0.00	91	0.00	119	0.00
42	0	137.00	25	91.0	67	74.00	88	64.00	124	55.00
43	0	6.00	22	10.00	56	7.00	75	17.00	105	18.00
Mean	0.0	18.3	25.6	17.9	60.0	20.5	83.3	23.9	113.4	26.9
SD	0.0	23.5	7.1	18.2	11.8	17.2	17.6	18.2	19.6	18.5
RIA Sensitivities (2 SD)										
Subjects		MT (pg/ml)								
1-17		4.25								
18-30		5.90								
31-43		5.58								

The Effects of Cellphone Frequency EMFs on Salivary Melatonin (pg/ml)

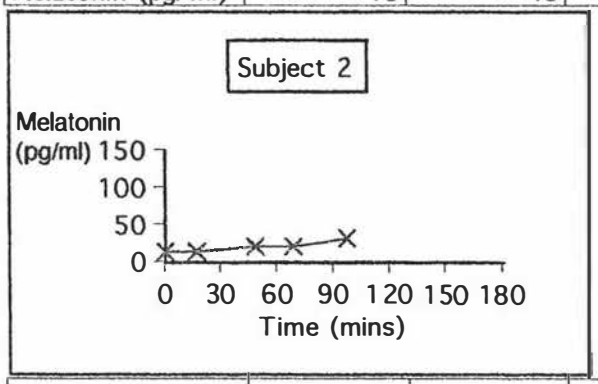
Subject 1

Time (mins)	0	24	53	72	100
Melatonin (pg/ml)	3	5	10	10	14



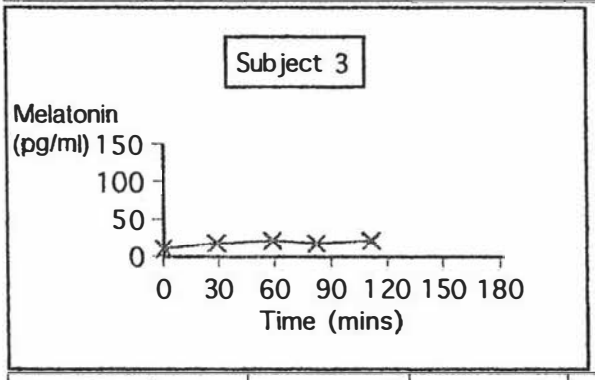
Subject 2

Time (mins)	0	17	48	68	97
Melatonin (pg/ml)	13	13	23	21	31



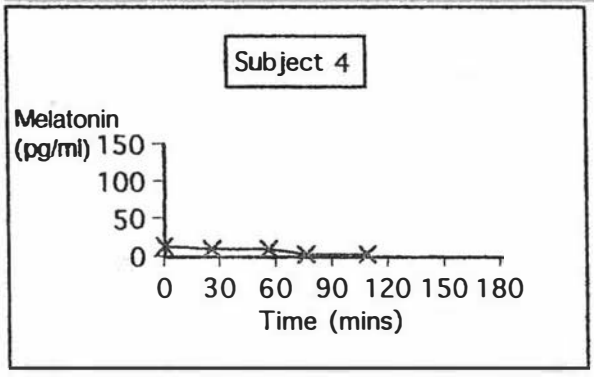
Subject 3

Time (mins)	0	29	58	81	111
Melatonin (pg/ml)	10	18	20	19	23



Subject 4

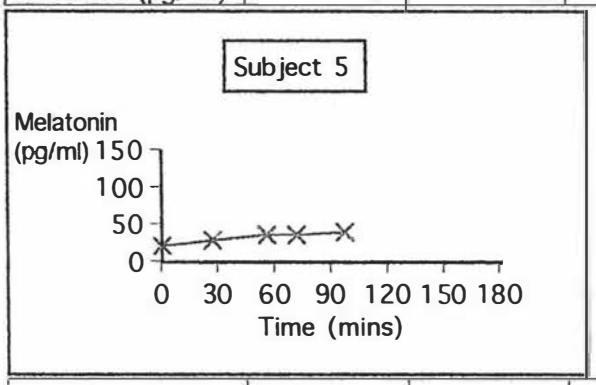
Time (mins)	0	26	56	76	108
Melatonin (pg/ml)	13	11	11	5	5



The Effects of Cellphone Frequency EMFs on Salivary Melatonin (pg/ml)

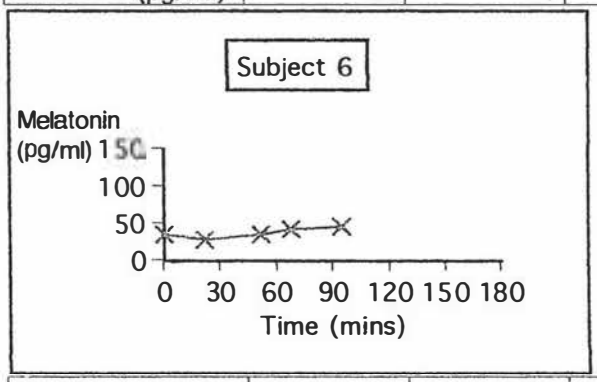
Subject 5

Time (mins)	0	27	56	72	97
Melatonin (pg/ml)	21	27	36	36	40



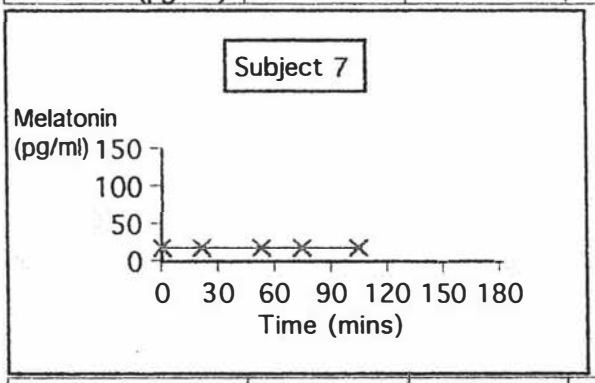
Subject 6

Time (mins)	0	22	51	67	94
Melatonin (pg/ml)	34	29	35	43	47



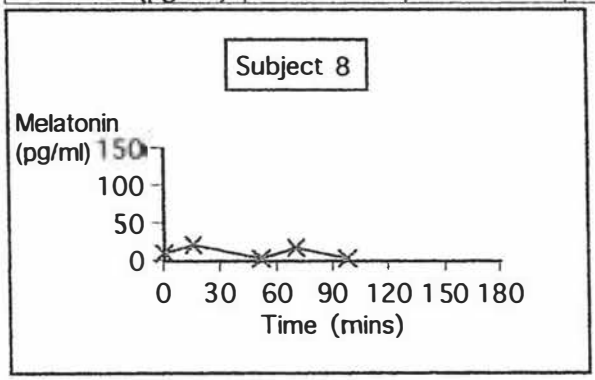
Subject 7

Time (mins)	0	21	53	75	105
Melatonin (pg/ml)	19	17	19	19	19



Subject 8

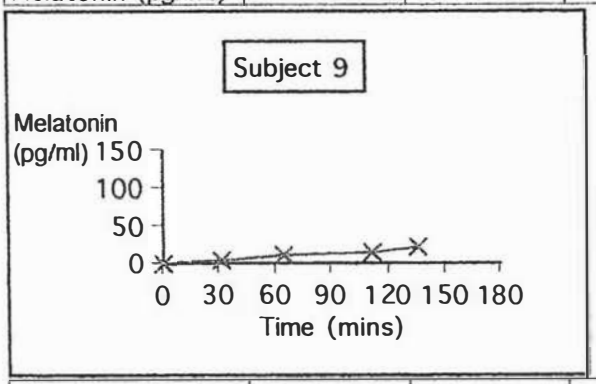
Time (mins)	0	16	51	70	97
Melatonin (pg/ml)	9	21	5	18	5



The Effects of Cellphone Frequency EMFs on Salivary Melatonin (pg/ml)

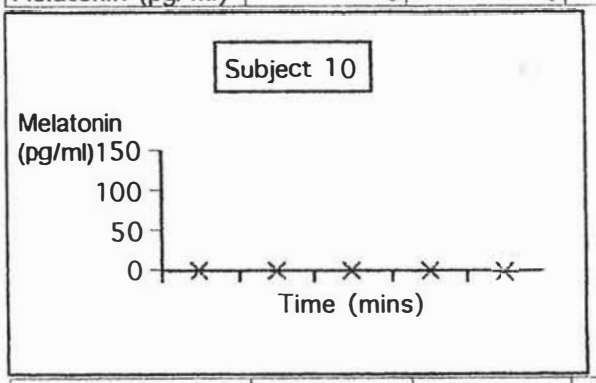
Subject 9

Time (mins)	0	31	65	111	136
Melatonin (pg/ml)	0	4	9	15	20



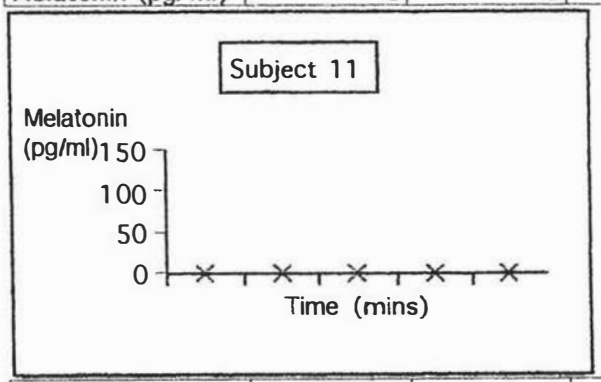
Subject 10

Time (mins)	0	17	44	58	85
Melatonin (pg/ml)	0	0	0	0	0



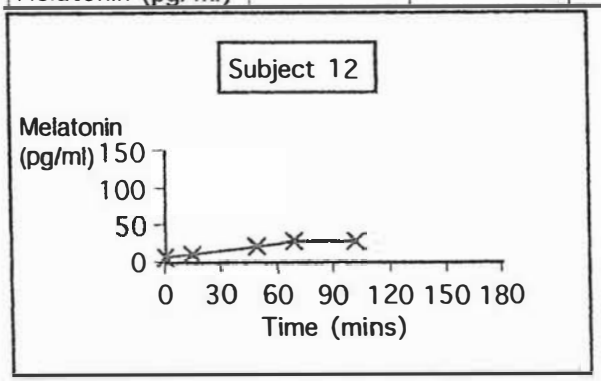
Subject 11

Time (mins)	0	27	50	65	89
Melatonin (pg/ml)	0	0	0	0	0



Subject 12

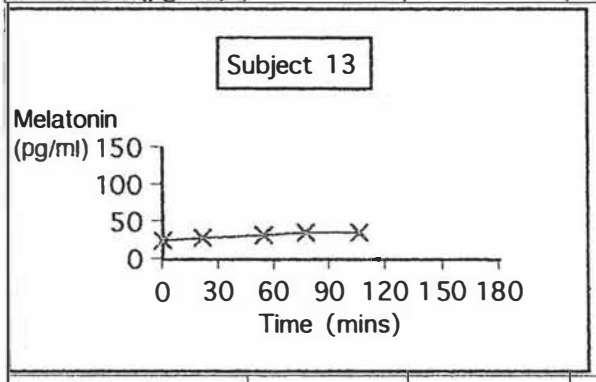
Time (mins)	0	15	48	68	101
Melatonin (pg/ml)	7	12	23	29	28



The Effects of Cellphone Frequency EMFs on Salivary Melatonin (pg/ml)

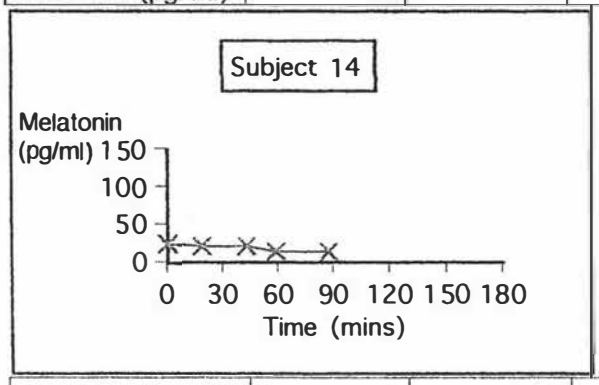
Subject 13

Time (mins)	0	22	55	77	106
Melatonin (pg/ml)	25	29	32	34	37



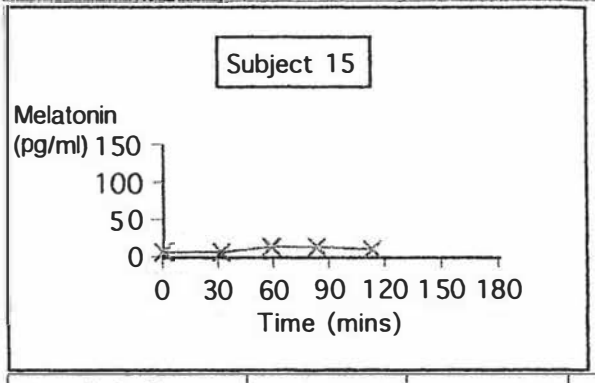
Subject 14

Time (mins)	0	18	43	59	87
Melatonin (pg/ml)	25	21	21	13	15



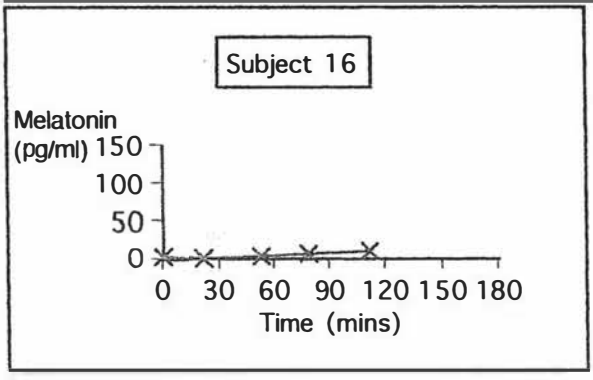
Subject 15

Time (mins)	0	31	59	83	113
Melatonin (pg/ml)	8	8	15	15	11



Subject 16

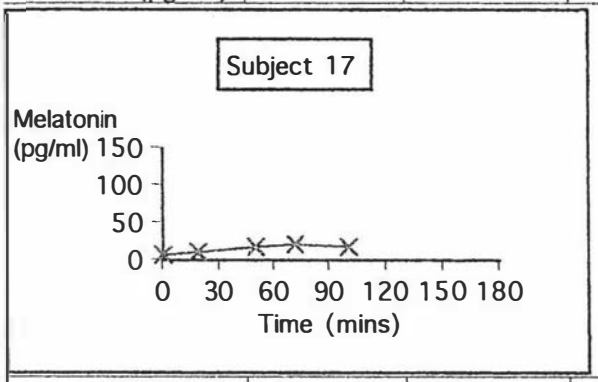
Time (mins)	0	21	53	78	111
Melatonin (pg/ml)	5	0	4	8	12



The Effects of Cellphone Frequency EMFs on Salivary Melatonin (pg/ml)

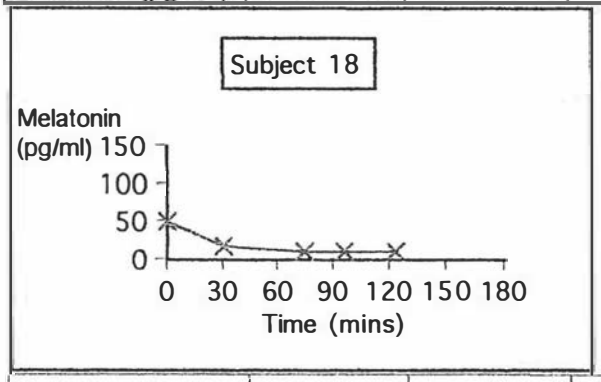
Subject 17

Time (mins)	0	18	50	72	100
Melatonin (pg/ml)	8	10	17	22	19



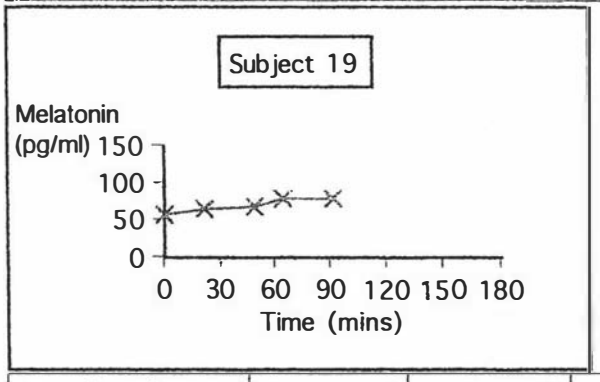
Subject 18

Time (mins)	0	30	74	96	123
Melatonin (pg/ml)	51	19	9	11	11



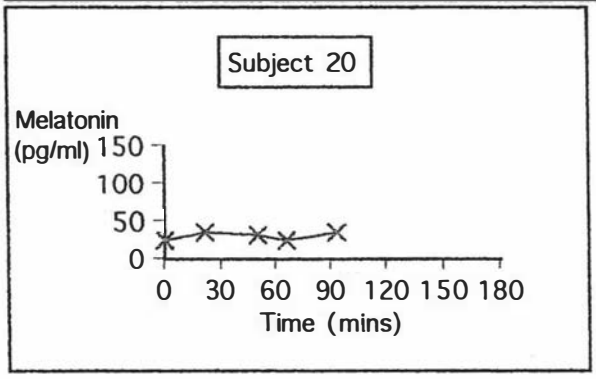
Subject 19

Time (mins)	0	21	48	65	91
Melatonin (pg/ml)	57	66	67	78	77



Subject 20

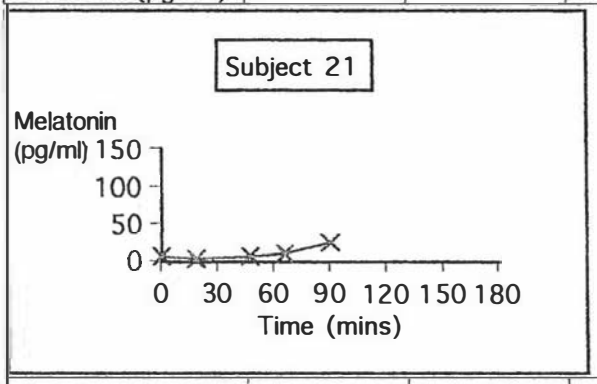
Time (mins)	0	21	50	66	93
Melatonin (pg/ml)	26	34	31	26	34



The Effects of Cellphone Frequency EMFs on Salivary Melatonin (pg/ml)

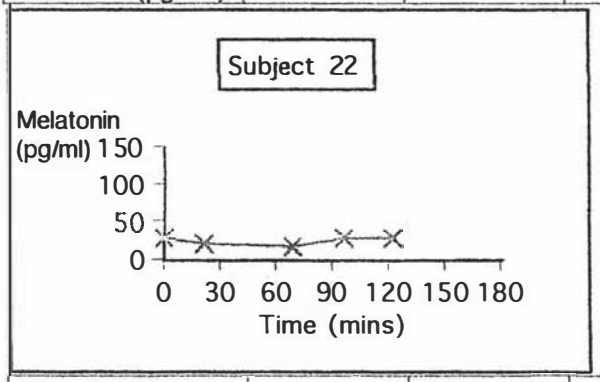
Subject 21

Time (mins)	0	18	47	66	90
Melatonin (pg/ml)	6	4	8	12	25



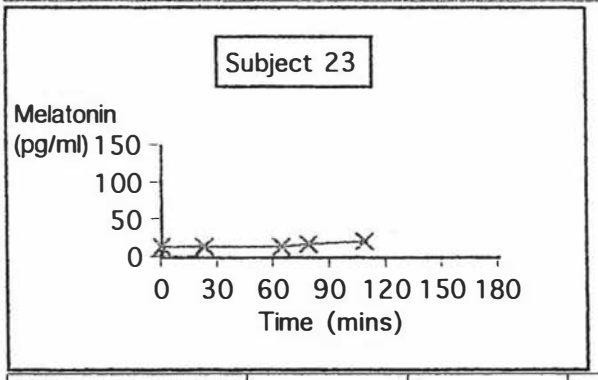
Subject 22

Time (mins)	0	22	68	96	121
Melatonin (pg/ml)	27	21	19	27	28



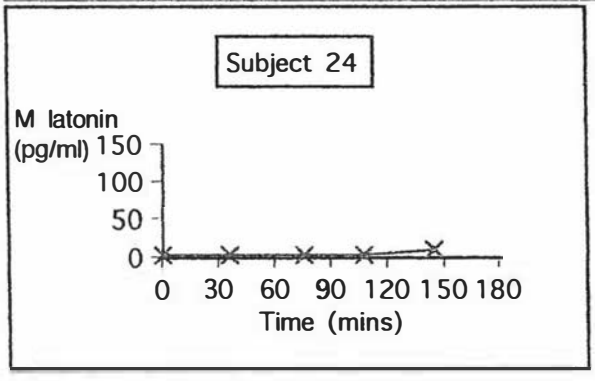
Subject 23

Time (mins)	0	23	65	79	108
Melatonin (pg/ml)	13	13	16	18	21



Subject 24

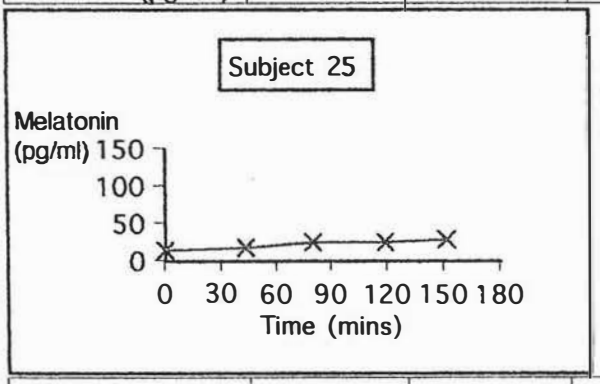
Time (mins)	0	36	76	107	145
Melatonin (pg/ml)	2	2	2	5	10



The Effects of Cellphone Frequency EMFs on Salivary Melatonin (pg/ml)

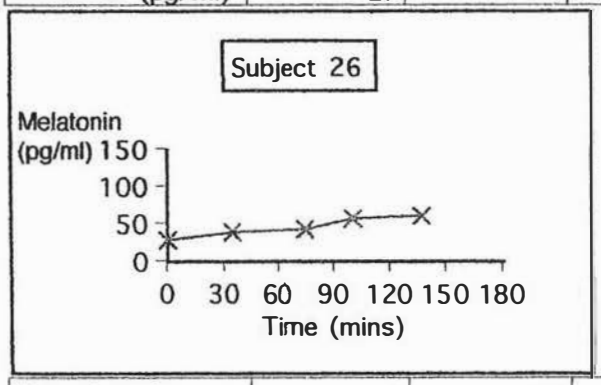
Subject 25

Time (mins)	0	43	80	119	152
Melatonin (pg/ml)	16	18	24	25	28



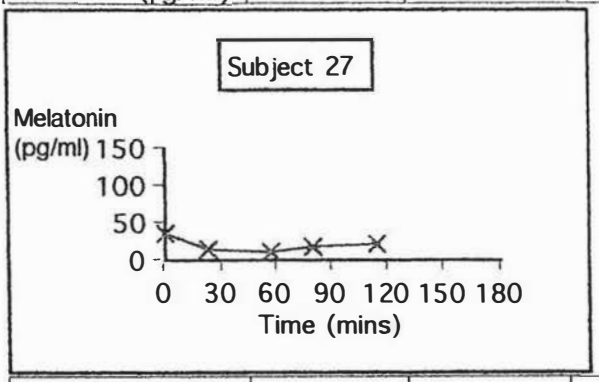
Subject 26

Time (mins)	0	34	74	100	137
Melatonin (pg/ml)	27	41	44	56	61



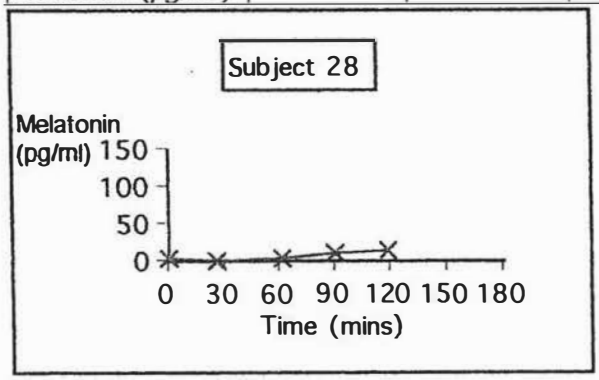
Subject 27

Time (mins)	0	23	57	80	114
Melatonin (pg/ml)	34	16	12	18	22



Subject 28

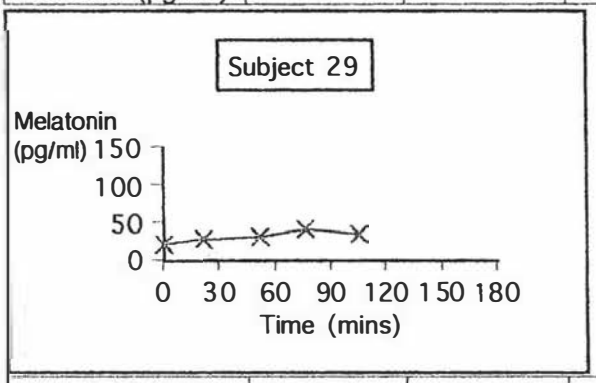
Time (mins)	0	26	61	90	119
Melatonin (pg/ml)	2	0	4	9	14



The Effects of Cellphone Frequency EMFs on Salivary Melatonin (pg/ml)

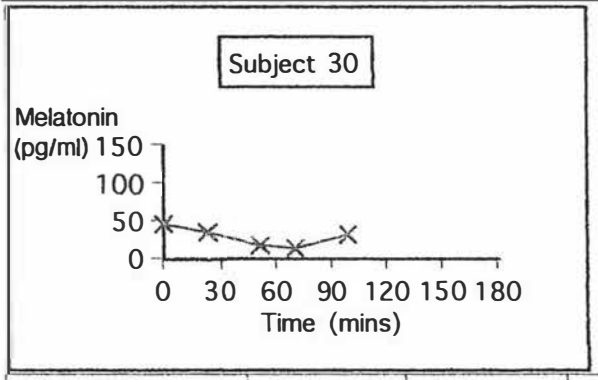
Subject 29

Time (mins)	0	22	52	76	105
Melatonin (pg/ml)	21	27	32	42	37



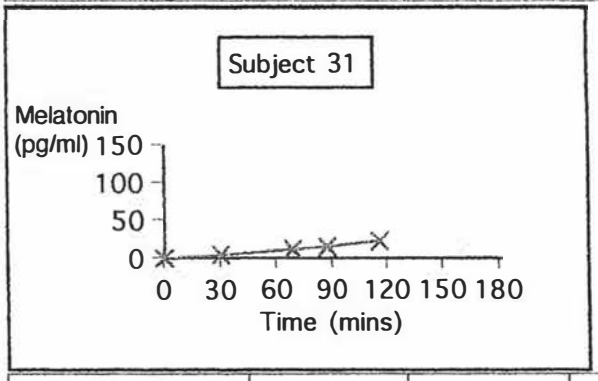
Subject 30

Time (mins)	0	23	52	70	99
Melatonin (pg/ml)	48	35	19	16	31



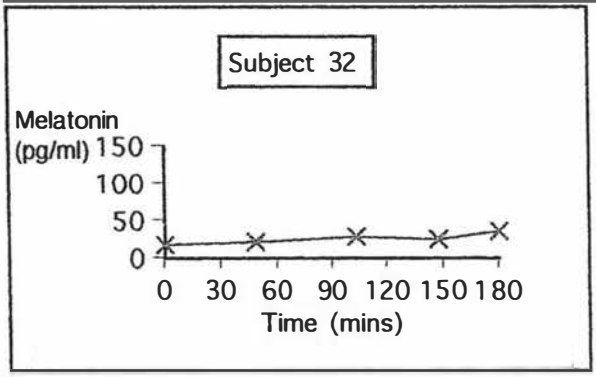
Subject 31

Time (mins)	0	30	69	87	116
Melatonin (pg/ml)	0	2	11	16	23



Subject 32

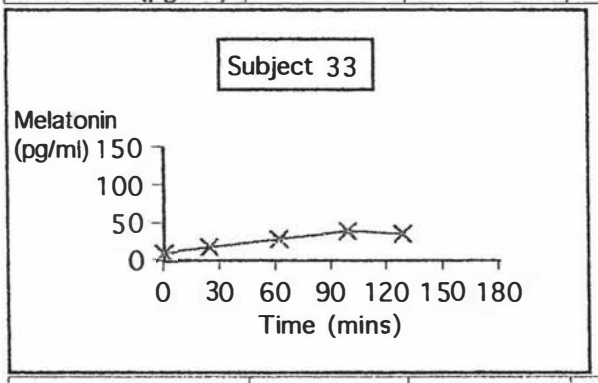
Time (mins)	0	48	103	147	180
Melatonin (pg/ml)	17	20	27	26	37



The Effects of Cellphone Frequency EMFs on Salivary Melatonin (pg/ml)

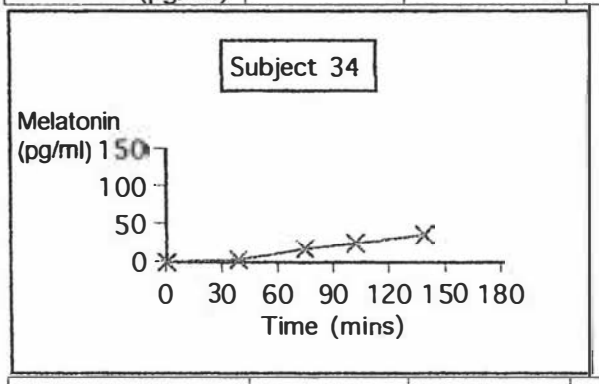
Subject 33

Time (mins)	0	25	61	98	128
Melatonin (pg/ml)	10	17	27	39	35



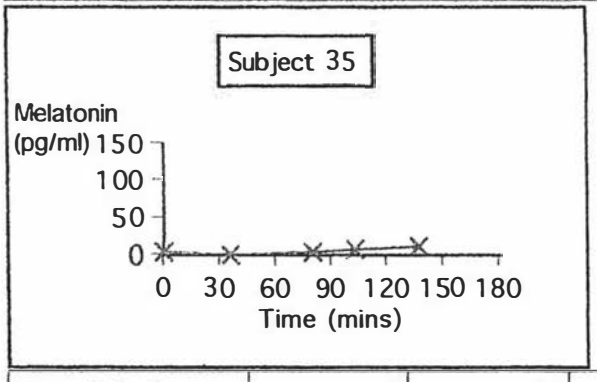
Subject 34

Time (mins)	0	39	75	101	139
Melatonin (pg/ml)	1	2	19	24	35



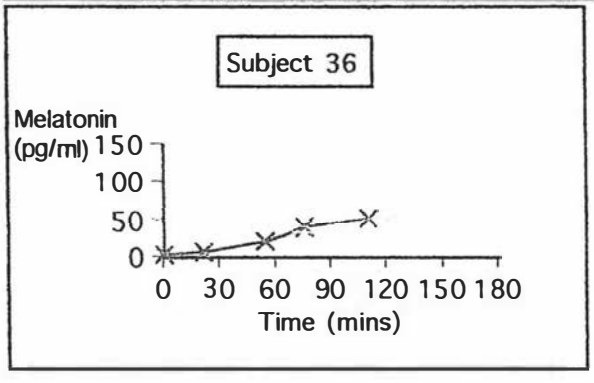
Subject 35

Time (mins)	0	36	80	103	137
Melatonin (pg/ml)	2	0	2	7	11



Subject 36

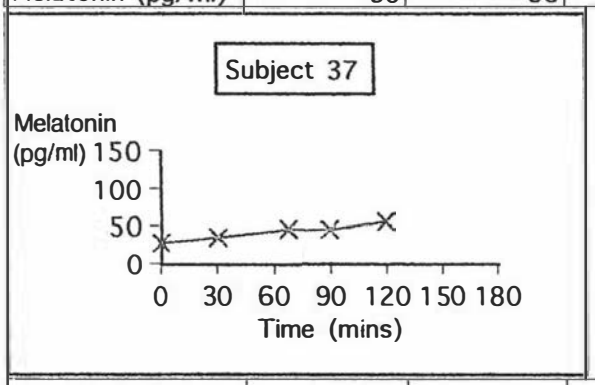
Time (mins)	0	21	54	76	110
Melatonin (pg/ml)	5	8	20	39	51



The Effects of Cellphone Frequency EMFs on Salivary Melatonin (pg/ml)

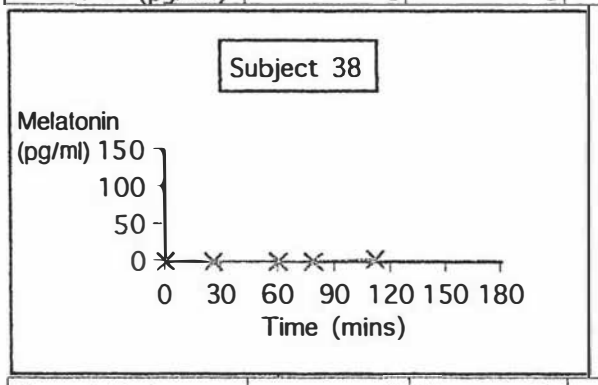
Subject 37

Time (mins)	0	30	67	89	118
Melatonin (pg/ml)	30	36	45	45	56



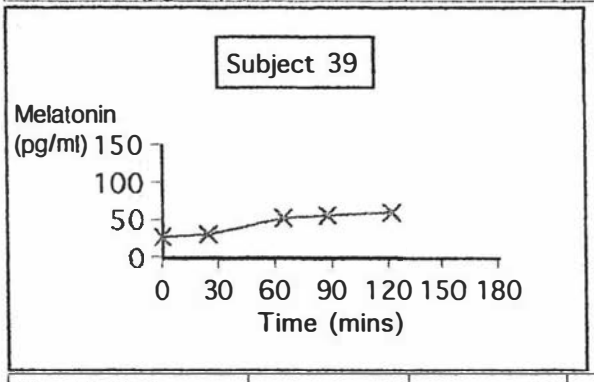
Subject 38

Time (mins)	0	26	60	79	112
Melatonin (pg/ml)	0	0	0	0	2



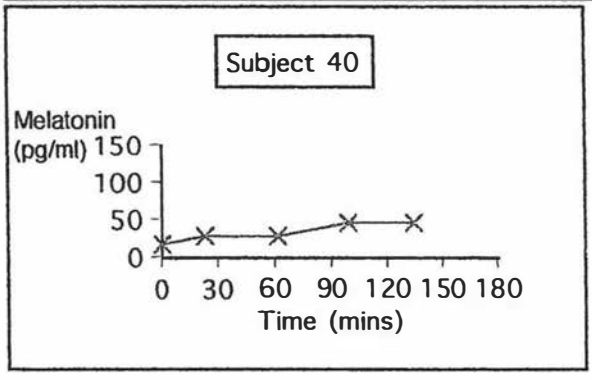
Subject 39

Time (mins)	0	24	65	87	121
Melatonin (pg/ml)	30	33	53	57	59



Subject 40

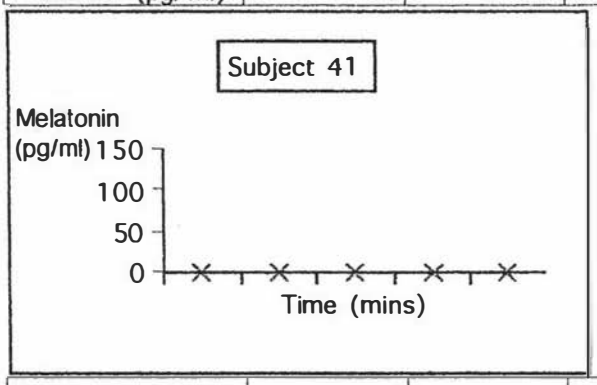
Time (mins)	0	23	62	98	134
Melatonin (pg/ml)	17	28	29	45	48



The Effects of Cellphone Frequency EMFs on Salivary Melatonin (pg/ml)

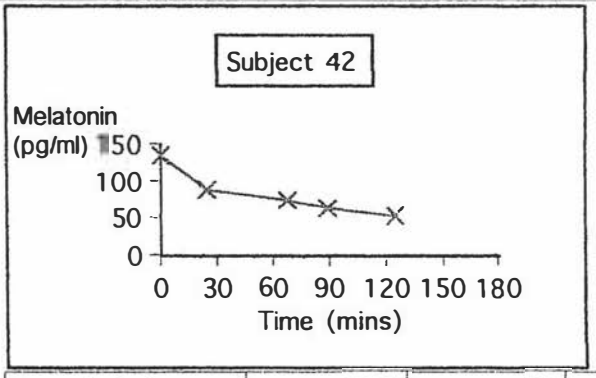
Subject 41

Time (mins)	0	29	64	91	119
Melatonin (pg/ml)	0	0	0	0	0



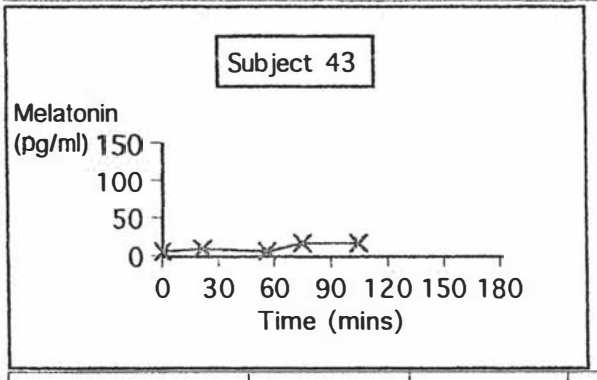
Subject 42

Time (mins)	0	25	67	88	124
Melatonin (pg/ml)	137	91	74	64	55



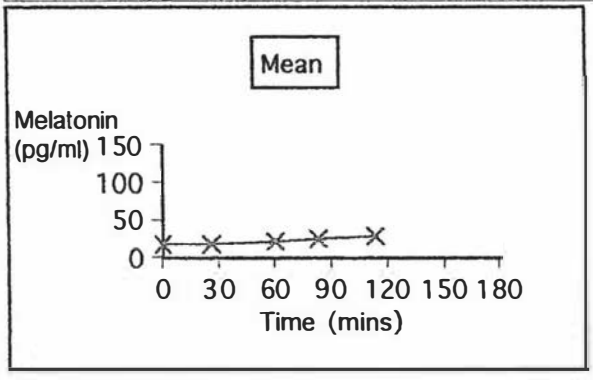
Subject 43

Time (mins)	0	22	56	75	105
Melatonin (pg/ml)	6	10	7	17	18



Mean

Time (mins)	0	25.6	60	83.3	113.4
Melatonin (pg/ml)	18.3	17.9	20.5	23.9	26.9



LIST OF ABBREVIATIONS AND BRIEF DEFINITIONS

AC Alternating Current

aMT6 the urinary metabolite of melatonin

b.p.m. beats per minute

cns central nervous system

ELF Extremely Low Frequency

EMF Electromagnetic Field

Function Generator. A Function Generator produces the frequency and waveform.

Frequency the number of periods of sinusoidal variation per unit time

GSM Global System for Mobile Communication

GHz Giga Hertz, a million hertz

Hall Effect Probe A device used to measure the magnitude of a magnetic field.

Helmholtz Coil Two coils of the same radius, carrying identical current in the same sense are arranged coaxially producing a highly uniform field.

Hz Hertz (cycles per second, a unit of frequency)

Internal fields are fields induced inside an animal by external fields

kHz kilo Hertz (1000 Hz)

MW Microwave

Power Frequency Fields (frequencies of 50 or 60 Hz, used around the world for mains power)

RF Radio Frequency

rms Root Mean Square (a kind of average)

SAR Specific energy absorption rate.

Transconductance Amplifier. This amplifies the waveform and powers the coil, using current as the mode of feedback.

Wavelength is the distance between two successive points of a periodic wave in the direction of propagation.

WHO World Health Organisation

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