Tobacco Smoking is Associated with Better Cognitive Performance in Smokers with Schizophrenia

A thesis presented in partial

fulfilment of the requirements

for the degree

of Masters in Science

in Psychology at

Massey University

David Raymond Galbraith

1999

Abstract

Past research argues people with schizophrenia (P/S) exhibit a specific cognitive deficit. This study argues dopamine deficiency and debilitating anxiety reduce cognitive functioning in P/S and over 80 per cent of P/S actively smoke to replenish dopamine and reduce debilitating anxiety, which relates to improved cognition. Comparing 18 smokers and 8 non-smokers with schizophrenia, with each other, and with 20 smoking and 20 non-smoking non-psychiatric people we used an independent samples between groups 2 by 2 correlational design to determine two main hypotheses: smokers with schizophrenia, after smoking one cigarette, in comparison to non-smokers with schizophrenia, will score fewer Wisconsin Card Sorting Test (WCST) errors: non-psychiatric people will perform better than P/S on the WCST. Results showed smokers with schizophrenia performed better on the WCST than non-smokers with schizophrenia. Smoking accounted for 11.2 % and facilitating-anxiety 41.3 % of the variance between schizophrenia groups. When controlling for facilitating-anxiety the significant difference dissolved. Smoking did not influence subjective affect or physiological arousal. Diagnosis did not influence cognitive functioning and the influence of smoking on cognitive performance did not depend on diagnosis. The discussion made four main conclusions: tobacco smoking and facilitating-anxiety directly related to cognitive performance in smokers with schizophrenia when completing the WCST, smoking does not influence subjective affect in smokers with schizophrenia when subjectively relaxed, schizophrenia does not exhibit a specific cognitive deficit, although smokers with schizophrenia did not gain the most from smoking clinical observation and literature review implies they actively smoke to obtain medicinal and psychological benefits.

Acknowledgments

One participant with schizophrenia asked, "What is schizophrenia?". I sat, thinking how to reply, for what seemed an inordinate amount of time before replying, "I do not know". He replied, "I do not know either". I firstly acknowledge the people with schizophrenia who participated in this study and thank you with my deepest gratitude for your unselfish generosity. Without your participation this study would not have been possible.

I completed this thesis for three reasons. First, to meet the requirements for a Masters Degree in Science. Second, to provide evidence showing P/S do not exhibit a specific cognitive deficit. Finally, to provide evidence showing P/S are more alike than unlike non-psychiatric people and thereby support a move towards normalising schizophrenia.

Thank you Dr Arnold Chamove for supervising my thesis and Kathy Macdonald for grammar checking the final draft. Thankyou to the professional staff at the Hastings Psychiatric Unit, and Taharoto Psychiatric Unit in Auckland, who participated in the non-psychiatric comparison groups, and to the staff who provided assistance during cognitive testing. Thank you to the Hastings Psychiatric Hospital manger Rose Shooter, and charge nurse Karen Grainger, and Taharoto Psychiatric Unit manager Robin Shearer for allowing completion of the research in your communities. Thank you to Dr Jim Rankin from Hastings and Dr John Tompkinson and Dr Falicity Plunkett from Auckland for your consultation on participant suitability. Thank you Dewke from the Hastings Community Health Centre and Andrea Broadhurst from the Napier Community Health Centre for access to your communities. Thank you to the Hawke's Bay Medical Research Foundation for providing Studentship Funding. HBMRF support of this research does not imply they support tobacco smoking. Special thanks to Judith Baxter for your time and effort during my application for the studentship grant.

Thank you to my family for their words of support and to Barbie Harding-Moller for your grammatical advice in the early stages of the thesis. Finally, thanks to my devoted partner Sarah-Jane for your love, analytical proof-reading and uncompromising support throughout the completion of this thesis. Without your spiritual richness and culinary delights my journey would have been far more arduous, lonely and hungry.

David. R. Galbraith

Table of Contents

List of Figures

List of Tables

List of Appendices.

Introduction

People with schizophrenia do not exhibit a specific cognitive deficit.

This next section introduces schizophrenia and an explanatory theory for the cognitive dysfunction associated with it. Research findings which conclude that P/S exhibit specific cognitive deficit in attention, information processing, and memory tasks are then reviewed, before directly challenging the validity of this conclusion. Critical analysis of these articles finds P/S do not differ from people with depression in selective attention and memory tasks. Cognitive performance in non-schizophrenia psychiatric and non-psychiatric people also decreases as task difficulty increases. Further, studies concluding P/S exhibit a specific cognitive deficit did not standardise cognitive tasks for discriminative power, misinterpreted results and exhibited unacceptable alpha levels. Together, evidence from the critical literature analysis places considerable doubt on the conclusion that P/S exhibit a specific cognitive deficit. Therefore this study argues P/S do not exhibit a specific cognitive deficit.

Schizophrenia is a psychiatric label applied to a person exhibiting abnormalities of perception, attention and thought (Kaplin, Saddock & Grebb, 1994). Schizophrenia is believed to cause a reduction in cognitive performance. Because P/S exhibit difficulty perceiving, attending to, and processing information from their environment, the reduction in cognitive performance is ubiquitous across cognitive functioning. For example, in comparison to non-psychiatric and non-schizophrenia[1] psychiatric people, P/S make more errors when attending to and filtering stimuli (Harris, Bennedict, &

[1] Due to the social stigma associated to the term schizophrenic, this study uses the noun schizophrenia rather than the adjective, despite this usage at times being grammatically incorrect.

Leek, 1990; Neale, 1971), take longer to process and recognise stimuli (Braff & Saccuzzo, 1981; Saccuzzo, Hirt & Spencer, 1974), require more practice sessions to improve response performance (Saccuzzo et al, 1974) and have difficulty recalling learnt material (Russel & Bechuis, 1976; Sengal & Lavallo, 1983). These previous articles argue that the reduction in cognitive performance is due to a cognitive deficit specific to schizophrenia. One theory explaining the specific cognitive deficit is response competition.

Response competition is the competition from a task-irrelevant response on a task-relevant or appropriate response (Broen, 1968). As task difficulty increases Broen argues P/S exhibit a corresponding increase in the probability that task-irrelevant responses will compete and interfere with task-relevant responses. Consequently, P/S should perform equally in comparison to non-schizophrenia psychiatric and non-psychiatric controls on simple cognitive tasks. They should also perform significantly worse than the same controls on difficult cognitive tasks, due to increased task-irrelevant interference. Before this study can challenge the conclusion P/S exhibit a specific cognitive deficit it is important to understand Broen's response competition model and why P/S may perform significantly worse than non-schizophrenia controls on most cognitive tasks. Research into filtering, information processing and memory concludes P/S exhibit a specific cognitive deficit and support Broen's response competition model.

Filtering is a cognitive attention process where a participant attends to, and selects, a stimulus for further information processing, based on a single physical feature (Broadbent, 1971) e.g. participants only listen to stimulus in the left ear in an auditory attention task (Harris et al, 1990). Pigeon holing is the cognitive process where a

participant formulates a response by attending to stimuli not distinguishable by any one physical characteristic (Broadbent, 1971) e.g. having participants listen for target words in ongoing speech to both ears (Harris et al, 1990). Because pigeon holing requires the selection of stimuli differing in more than one physical characteristic it is more complex than filtering. Therefore, according to Broen's theory P/S should, in comparison to non-schizophrenia controls, perform worse on pigeon holing tasks than filtering tasks; which literature shows they do (Harris et al, 1990). Therefore, the finding supports Broen's response competition theory and, because P/S performed worse than controls, the suggestion that the population exhibits a specific cognitive deficit.

Information processing is the global term given to sensory, perceptual and conceptual processes associated with the movement of information between the sensory register, short term and long term memory (Nuechterlein & Dawson, 1984; Loftus & Loftus, 1976; Massaro & Loftus, 1996, cited in Bjork & Bjork, 1996). The efficiency of the sensory register and short term memory is reduced by interfering stimuli (Pashler & Carrier, 1996, cited in Bjork & Bjork, 1996). Broen's response competition theory states that there is a corresponding increase in task-irrelevant interference in P/S as task difficulty increases. Therefore, based on Broen's theory, P/S should perform worse than non-schizophrenia people on information processing tasks as task complexity increases.

Backward masking is an information processing task which manipulates an interfering or task-irrelevant stimulus. It thereby varies the amount of time a stimulus is available in the sensory register for cognitive processing and subsequent transfer of information to the working memory (Saccuzzo et al, 1974). For example, introducing incomprehensible white noise (Chaplin, 1984) at various time intervals after target stimulus presentation varies the amount of time stimuli are available and consequently

interferes with information processing. Because masking of stimulus in information processing is analogous to Broen's response competition theory and task-irrelevant stimulus interruption, P/S should require longer periods of stimulus presentation than non-psychiatric people to process information.

Experiments using backward masking found P/S required significantly longer periods of stimulus presentation to identify the target stimulus than non-psychiatric and depressive subjects. Research also found P/S required more practice sessions to improve their identification performance than non-psychiatric and depressive people (Braff & Saccuzzo, 1981; Saccuzzo et al., 1974). These results infer P/S experience task-irrelevant interference earlier in information processing than controls and therefore require longer periods of stimulus presentation to process information. The findings support Broen's theory and, because P/S performed significantly worse than both control groups, support the argument P/S experience a specific cognitive deficit.

Memory or information recall research provides additional evidence that P/S exhibit a specific cognitive deficit. Recognition memory is based on stimulus familiarity and recall memory requires people recall information based on internal word to word associations (Bauman & Murry, 1968; Wickelgren, 1977). Recognising stimuli is less complex than recalling stimuli for two reasons. Firstly, recognition provides people with more retrieval cues to trigger memory. Secondly, recognition provides more information indicating the correct response and consequently reduces task ambiguity (Wickelgren, 1977). Because recognition tasks provide more information than recall tasks recognition tasks are less ambiguous, therefore less complicated. The comparison between recall and recognition memory is important. Considering Broen's theory the comparison infers

P/S will perform equally on recognition tasks and worse on recall tasks in comparison to non-schizophrenia psychiatric and non-psychiatric people.

People with schizophrenia perform equally on recognition memory compared to non-psychiatric subjects (Bauman & Murray, 1968; Nachmani & Cohen, 1969) and significantly worse on recall tasks compared to non-psychiatric and depressive people (Nachmani & Cohen, 1969; Russel & Bechuis, 1976; Sengal & Lavallo, 1983). These results support Broen's theory that an increase in task complexity corresponds with an increase in cognitive deficit exhibited by P/S. Further, because P/S performed worse than both non-schizophrenia psychiatric and non-psychiatric controls on recall tasks, it implies they exhibit a specific recall memory deficit.

In summary, as selective attention, information processing and memory task difficulty increased in complexity P/S performed significantly worse than non-schizophrenia psychiatric and non-psychiatric controls. Together these findings support Broen's stimulus complexity theory. Since P/S performed significantly worse than both control groups, the findings also support the argument that P/S exhibit a specific cognitive deficit. However, critical analysis of the previously reviewed articles places this summary in doubt.

Although schizophrenia cognitive performance worsens as task complexity increases, research in stimulus attention tasks (Allen, 1982; Harris et al, 1990) and information processing tasks (Saccuzzo et al, 1974) shows that the cognitive performance of non-schizophrenia psychiatric and non-psychiatric samples also worsens. For example, Allen (1982) found no difference in magnitude of change between P/S and non-psychiatric controls on attention tasks. Both groups found the tasks equally difficult. Further, P/S did not differ significantly from depressive controls

when completing selective-attention tasks (Caudrey, Kirk, Thomas & Ng, 1980; Hemsley & Zawada, 1976) and free-recall memory tasks (Russell & Beckhuis, 1976). These findings contradict Broen's response competition theory and imply P/S do not exhibit a cognitive deficit specific to their population. Further, articles initially considered to support Broen's theory exhibited poor methodology, including no standardising tasks for discriminative power, result misinterpretation and unacceptable inflation of alpha.

Discriminative power relates to the extent a test can distinguish between two groups of subjects, for example P/S versus non-psychiatric people (Chapman & Chapman, 1973; Klein, 1994). If one test is more discriminating than another test it will find greater cognitive performance deficit than a test with lower discriminative power. Three studies which initially appeared to support the theory that P/S exhibit a specific cognitive deficit did not standardise cognitive tasks for discriminative power (Bauman & Murray, 1968; Nachmani & Cohen, 1969) or incorrectly standardised tasks (Harris et al, 1990). For example, Harris et al reported matching tasks for difficulty based on non-psychiatric controls scoring 100 per cent accuracy for both filtering and pigeon holing tasks. However, because non-psychiatric people scored 100 per cent on both tasks does not guarantee standardised discriminatory power. Literature supports this study's argument and recommends maximising discriminative power for non-chance tasks using a 50 per cent criterion (Lord, 1952, cited in Chapman & Chapman, 1973). The previous three studies found P/S exhibited equal cognitive performance on the simple recognition and filtering cognitive tasks and significantly worse recall and pigeon holing tasks in comparison with non-schizophrenia controls. Subsequently the studies concluded P/S exhibit specific memory recall and attention deficit.

However, the results of the previous studies may have been due more to recall and pigeon holing tasks discriminating between P/S and comparison groups more than the recognition or filtering tasks. Correctly standardising all tasks for discriminative power before conducting the research may have found P/S performing significantly worse than non-schizophrenia psychiatric and non-psychiatric people on all tasks. Therefore P/S would not have appeared to exhibit specific cognitive deficit on the more difficult tasks, but more general cognitive deficit than comparison groups across tasks. Further, research standardising tasks for discriminative power found no difference in magnitude of change from simple to complex attention tasks between P/S and non-psychiatric people (Allen, 1982). Both groups found the change from simple to complex tasks equally difficult, inferring P/S do not exhibit a specific cognitive deficit.

Another important dimension in generating valid research is conducting accurate interpretations of statistical data. Research initially supporting the argument P/S exhibit a specific cognitive deficit (Neale, 1971) can be reinterpreted to provide support for the argument P/S do not exhibit a specific cognitive deficit. The latter interpretation adheres to the following theory. Unless P/S perform better than non-schizophrenia controls on any 'simple' cognitive task, and then significantly worse in the 'difficult' task, results do not infer P/S exhibit a specific cognitive deficit (Calav & Monk, 1982). Rather, the finding only infers P/S exhibit more cognitive deficit than controls in the difficult task. For example, Neale (1971) found no difference between P/S and non-schizophrenia psychiatric and non-psychiatric people on the simple level of a multi level perceptual span task. However, in conditions with multiple irrelevant competing letters present the perceptual span of P/S was significantly less than controls. Neale concluded P/S exhibit a specific perceptual span deficit in comparison to non-schizophrenia psychiatric and

non-psychiatric controls. The conclusion supported Broen's theory and the theory P/S have a cognitive deficit specific to their population. However, the research based its conclusion on a non significant difference in the simple non-interference condition and a subsequent significant difference between groups, with P/S performing worst on the more difficult tasks. People with schizophrenia did not perform better than controls on the 'simple' task, implying that P/S exhibit more perceptual span deficit in the difficult tasks, and not that they exhibit a specific perceptual cognitive deficit.

Alpha refers to the probability of a statistical test determining a significant difference between groups when no difference exists (Tabachnick & Fidell, 1989), referred to as type one error (Coolican, 1994). The risk of committing a type one error increases when conducting more than one statistical test in a single experimental design. Such an inflation in risk of committing type one error is known as experimentwise alpha and is defined as the probability of falsely rejecting the null hypothesis for at least one test when conducting multiple tests (Weinfurt, 1995). When conducting research utilising multiple tests, which most articles reviewed by this research do, it is vital to adjust the original or nominal alpha to compensate for alpha inflations. When research conducts multiple tests but does not alter alpha, alpha becomes unacceptably high and the validity of results must be questionable. For example, Braff and Saccuzzo (1981) conducted research which initially appeared to support the argument that P/S exhibit a specific cognitive deficit in comparison to people with depression. The study based its conclusions on five univariate t-tests and nine multivariate and covariate analyses. Because they gave no indication that they had adjusted their alpha level to compensate for conducting 14 tests, there is an implication that the study did not alter alpha levels. Consequently their research would have experienced inflation in alpha. Using the

equation provided in Weinfurt (1995) and an alpha of .05 (Bergin & Garfield, 1994) it appears Braff and Succuzzo generated an experimentwise alpha of .7. The .7 alpha level represents a 70 per cent chance of finding at least one test significant when it is not, an unacceptable risk of committing type one error. Consequently any results generated from Braff and Saccuzzo need interpreting with caution.

In conclusion, the introduction has shown people with schizophrenia do not exhibit a specific cognitive deficit. Despite this, research with acceptable methodology still found P/S performed worse than non-psychiatric people. If P/S do not exhibit a specific cognitive deficit but still perform worse than non-psychiatric people, perhaps another explanation exists for their reduced cognitive functioning.

Anxiety reduces cognitive functioning.

The next section shows anxiety can reduce and improve cognitive performance. People with schizophrenia are anxious people and exhibit anxiety which reduces their cognitive performance and causes their cognitive dysfunction. Further, this section shows P/S and non-psychiatric anxious people exhibit debilitating anxiety. Reducing debilitating anxiety in non-psychiatric anxious people improves cognition. Therefore, because P/S and non-psychiatric people share debilitating anxiety this section argues that reducing debilitating anxiety in P/S will also correspond with better schizophrenia cognitive performance.

Anxiety can reduce cognitive functioning. Anxiety which reduces cognitive performance presents as either physiological arousal, cognitive worry, or both (Kaplan et al, 1994). Arousal is a multi-dimensional construct including cognitive, affective and physiological responses (Gjerde, 1983; Neiss, 1988). Physiological responses or arousal

related to anxiety includes autonomic nervous system activity, for example, increased heart rate and increased blood pressure. Arousal to anxiety also includes central nervous system activity, for example, increase in cortical electrical activity (Kaplan et al, 1994; Bellack & Hersen, 1988). Cognitive responses include worry and nervousness, also known as task-irrelevant responses (Eysenck & Folkard, 1980; Gaeddert & Dolphin, 1981). It is important to understand the various responses associated with anxiety in order to distinguish between anxiety which reduces and improves cognitive functioning.

Anxiety which improves cognitive performance is by definition facilitating-anxiety and is characterised by the presence of subjective relaxation and increased physiological measures of arousal. Anxiety which reduces cognitive functioning is by definition debilitating anxiety and is characterised by an increase in arousal and the presence of task-irrelevant responses (Alpert and Haber, 1960; Sarason, Mandler, & Craighill, 1952). Applying the definition and characteristics of debilitating anxiety to non-psychiatric literature shows debilitating anxiety reduces cognitive functioning (Hammermaster, 1989; Hudesman, Loveday & Woods, 1984; Metzger, Miller, Cohen, Sofka & Borkovec, 1990).

People with schizophrenia are anxious people (Depue & Fowles, 1973, Kelher, Wilson, Muldawer, & Pathak, 1975; Mendick, 1958; Serban, 1975). They exhibit cognitive worry and nervousness, also known as task-irrelevant responses, and therefore by definition P/S experience debilitating anxiety (Alpert & Harber, 1960; Broen, 1968; Sarason, et al, 1952). Because debilitating anxiety reduces non-psychiatric cognitive functioning this study argues anxiety causes schizophrenia cognitive deficit. The idea anxiety influences schizophrenia cognitive functioning is not new (Depue, 1974; Gjerde, 1984). Depue found P/S and anxious non-psychiatric subjects performed equally when

discriminating between certain stimuli projected onto a visual screen. Further, both samples performed worse than non-anxious non-psychiatric samples, thereby supporting this study's argument that anxiety causes cognitive dysfunction in P/S.

People with schizophrenia and anxious non-psychiatric people also perform comparably on similar cognitive tasks. For example, highly anxious subjects performed better on multiple choice tasks than on essay tasks and took longer to learn class material than non-anxious controls (Benjamin, McKeachie, Lin, & Holinger, 1981). Comparably, P/S performed significantly worse than non-psychiatric samples on memory recall than on recognition memory tasks. People with schizophrenia also took longer to learn task procedure than non-schizophrenia psychiatric and non-psychiatric controls (Bauman & Murray, 1968; Saccuzzo, et al, 1974).

Recognition memory is the retrieval of information based on stimulus familiarity. Familiarity of stimulus and external cues also trigger multi-choice task responses (Bauman & Murry, 1968; Wickelgren, 1977). Memory recall and essay writing require an internal subjective word-to-word association in order to generate the required response (Wickelgren, 1977). The comparison between recognition memory and multi-choice tasks, and recall memory and essay tasks, is important because it shows these tasks involve the same processes and allows comparisons across populations. It infers P/S and anxious subjects performed comparably on these tasks for the same reasons.

People with schizophrenia and non-psychiatric anxious people perform poorly on cognitive tasks due to increased arousal (Gjerde, 1983) and because task-irrelevant responses interfere with task-relevant responses (Broen, 1968; Sarason, Mander & Craghill, 1952). For example, both samples exhibit internal preoccupation with their

physiological anxiety sensations and their thoughts of worry and nervousness (Bleuler, 1950; Eysenck & Folkland, 1980; Gaeddert & Dolphin, 1981; Hammermaster, 1989; Wine, 1971). Consequently both samples miss external information cues used for information processing, which results in poor cognitive performance. Both populations exhibited debilitating anxiety.

Reducing debilitating anxiety using systematic desensitisation in non-psychiatric subjects with high levels of test anxiety improved cognitive functioning (Cohen 1969; Donner & Guerney, 1969; Hudesman et al, 1984; Johnsen & Sechrest, 1968; MaManus, 1971). Lower levels of anxiety correspond with higher cognitive performance in non-psychiatric subjects, in comparison with high levels of anxiety (Hammermaster, 1989; Metzger et al., 1990; Pomerleau & Pomerleau, 1987; Wine, 1971). Because P/S and anxious non-psychiatric people exhibit debilitating anxiety this study argues reducing debilitating anxiety in P/S will relate to better cognitive performance. Anxiety is not the only phenomenon which is associated with reduced schizophrenia cognitive performance.

Dopamine deficit reduces cognitive functioning.

The next section shows dopamine deficit relates to reduced cognitive functioning. People with schizophrenia exhibit low concentrations of dopamine and increasing dopamine corresponds with improved schizophrenia cognitive performance. Dopamine antagonist medications prescribed to P/S reduce dopamine levels. Subsequently this study argues low dopamine levels relate to poor schizophrenia cognitive functioning and dopamine antagonistic medications exacerbate their cognitive dysfunction.

Reductions in dopamine decrease cognitive functioning. Research investigating intellectual functioning, attention and reaction time in people with heroin addictions who had self administered 1-methyl-4-phenyle-1,2,3,6-tetrahydropyridine (MPTP) found these people developed MPTP induced parkinsonism. The participants performed worse than eight healthy heroin addicts on scores of general intellectual function, construction, category memory and frontal lobe functioning (Stern & Langston, 1985). The neural change which accompanies parkinsonism and the associated cognitive impairments involves selective destruction of the dopaminergic neurones in the pars compacta and substantia nigra cortical regions (Bernheimer, Birkmayer, Hornykiewicz, Jellinger & Seitelberger, 1973). The reduction in dopamine transmission best explains the cognitive functioning impairment associated with parkinsonism (Stern & Langston, 1985). The previous conclusion is not an isolated argument. Other studies have found reductions in dopamine correspond with cognitive deficit, in primate (Elsworth, Deutch, Redmond, Sladek & Roth, 1990), and in human subjects (Berger, van Hoof, van Spaendonck, Horstink, van den Bercken, Jaspers & Cools 1989; Stern, Tefrud, Martin, Kutner & Langston, 1990).

People with schizophrenia exhibit dopamine deficit. Traditional theory argues that excessive cerebral dopamine transmission causes schizophrenia (Davis, Kahn, Ko, & Davidson, 1991; Kaplan et al, 1994; Lewis & Akil, 1996). However, recent research found cognitive impairment associated with schizophrenia relates to dopamine deficit (Daniel et al, 1991; Davis et al, 1991; Dolan et al, 1995; Lewis & Akil, 1997). Therefore, this study argues, if dopamine deficit is associated with schizophrenia cognitive dysfunction, then increasing dopamine in P/S should relate to better cognitive performance. Daniel et al (1991) found the administration of amphetamine, a dopamine

agonist, increased dopamine levels and improved schizophrenia cognitive performance on the Wisconsin Card Sorting Test (WCST). Although some chemicals increase dopamine, traditional pharmacological therapy for schizophrenia reduces dopamine transmission by directly blocking movement of dopamine across the synaptic space.

Some forms of medication used to treat schizophrenia actively block and reduce dopamine synaptic transmission, for example, phenothiazine anti psychotics (Silverman, 1994). Further, drug literature warns 15 per cent of P/S prescribed anti psychotic medication such as chlorpromazine and haloperidol will develop parkinsonism syndrome (Data Sheet Compendium, 1981-82; Silverman, 1994). One symptom of parkinsonism syndrome is reduced cognitive functioning (Bernheimer et al, 1973). This study predicts 15 per cent of P/S who participate in this research and receive dopamine antagonist anti psychotics may exhibit medication related dopamine deficiency, which will exacerbate any pre-existing dopamine deficit related cognitive dysfunction.

Nicotine increases cognitive functioning, increases dopamine concentrations, reduces anxiety and provides a natural protection against anxiety.

The next section shows P/S exhibit the highest tobacco smoking rate in the western society. Nicotine directly increases cognitive functioning and dopamine in non-psychiatric people. Nicotine reduces debilitating anxiety and provides biological protection against anxiety and stress in smokers with schizophrenia. The section argues therefore, that smokers with schizophrenia actively smoke to increase dopamine and reduce anxiety.

People with schizophrenia exhibit the highest tobacco smoking rate in western society. Comparing the smoking habits of 277 psychiatric outpatients with 18440 non-

psychiatric subjects found that P/S exhibit the highest smoking rate in the United States of America; over 80 per cent of P/S smoked (Hughes, Hatsukami, Mitchell & Dahlgren, 1986). Other researchers have found corresponding findings where P/S exhibited smoking rates of higher than 80 per cent (Gopalaswamy & Morgan, 1985; Masterson & O'Shea, 1984). Some research suggests P/S smoke to reduce boredom and provide a life pleasure (Golalaswamy & Morgan, 1985). Others, however, suggest smokers with schizophrenia self medicate with nicotine (Glynn & Sussman, 1990; Goff, Henderson & Amico; 1992; Howard, 1996). Clinical investigations made by the primary researcher support the suggestion that smokers with schizophrenia smoke for a purpose.

In order to investigate the purpose for smoking the author informally questioned 20 P/S[2] about why they smoked. Smokers with schizophrenia believed smoking helped them relax. Twenty per cent ($N = 4$) of smokers with schizophrenia associated smoking with a reduction in schizophrenia positive symptoms such as auditory hallucinations. Patient self-report corresponded with objective clinical record of increased time spent reading and interacting with peers immediately after smoking. The findings implied smokers with schizophrenia actively smoke to obtain medicinal and psychological benefits. Psychiatric and non-psychiatric literature supports the clinical observations that smoking appeared to directly improve cognitive functioning required for social activity.

People with schizophrenia exhibit deficit in hippocampal nicotinic receptors relating to increased hypersensitivity (Freedman, Hall, Adler, & Leonard, 1995). The increased sensitivity appears related to an inability to shut-out or gate-off sensory input. Nicotine not only reduces distractability by providing more nicotine to the hippocampal

[2] The informal questioning took place when the principal researcher worked as a nurse aid at the Hastings Psychiatric Unit.

region but also reinstates the sensory gates for auditory (Adler, Hoffer, Grifith, Waldo and Freedman, 1992) and visual stimuli (Klein & Andersen, 1991). Logically then, research shows nicotine improves vigilance and rapid information processing (Wesnes & Warburton, 1983), detection accuracy and speed in signal detection tasks (Wesnes & Warburton, 1978). Nicotine improves ability to filter out task-irrelevant stimuli (Levin, 1992) and improves short-term verbal recall (Peeke & Peeke, 1984). These cognitive skills are important in successful interpersonal interactions. One explanation for the improved cognitive performance is that smoking is associated with increased dopamine transmission.

Nicotine increases dopamine release. Nicotine receptors exist in high concentration in cortical regions which release dopamine and play intrinsic roles in cognitive functioning. For example, receptors exist in the substantia nigra and ventral tangential and acute nicotine administration increases dopamine release from these areas (Grenhoff, Aston-Jones & Svensson, 1986; Imperato, Mulas & Chiara, 1986; Levin, 1992). Further, the administration of nicotine antagonists inhibits dopamine release, (Ahtee & Kaakkola, 1978) and the administration of nicotine agonists counteracts this effect, increasing dopamine concentrations (Levin & Rose, 1995). Together with previous evidence that P/S exhibit dopamine deficiency which relates to reduced cognitive performance, the findings that nicotine increases dopamine release imply that smokers with schizophrenia may actively self-administer nicotine to increase dopamine concentrations.

Nicotine also reduces anxiety. Verbal indications by smokers with schizophrenia (Glynn & Sussman, 1990) and non-psychiatric people who smoke (Ikard & Tompkins, 1973) suggest smoking is relaxing. The more nicotine non-psychiatric smokers inhale

the more pleasant, less anxious and less internally tense or worried they became (Ague, 1973). Further, research reported non-psychiatric subjects who smoked were less anxious when smoking than when not smoking (Nesbitt, 1973; Pomerleau, Turk, & Fertig, 1984). An increase in tension also corresponds with increased smoking levels in non-psychiatric people (Pomerleau & Pomerleau, 1987). One interpretation of these results is people smoke to reduce feelings of debilitating anxiety. This is, however, an interesting paradox relating to the effects of smoking which requires a more detailed interpretation.

When schizophrenia and non-psychiatric people smoke they report a subjective experience of relaxation, yet smoking itself induces physiological symptoms indicative of anxiety (Kaplan et al, 1994). Smoking increases autonomic nervous system activity, for example, heart rate and blood pressure (Nisbitt, 1973; Ray, Nellis, Brady, & Foltin, 1986; Soria et al, 1996). Smoking increases central nervous system activity, for example, electric cortical activity (Gilbert et al, 1989). These examples are important because they suggest a contradiction between the claim that, smoking is relaxing, and the evidence that smoking generates autonomic and central nervous system activity indicative of anxiety. However, another interpretation is plausible.

Debilitating anxiety presents as physiological arousal, increasing heart rate and blood pressure, cognitive worry and nervousness. It is understandable, therefore, that smokers experience subjective relaxation while exhibiting increased heart rate and blood pressure. Smoking reduces cognitive intrusion of irrelevant-task responses, for example, worry and nervousness, while the subject remains physiologically roused as a direct consequence of nicotine on the autonomic and central nervous system. Consequently they exhibit facilitating-anxiety which relates to improved cognitive performance

(Albert & Haber, 1960), implying smokers with schizophrenia may self-administer nicotine to reduce subjective feelings of debilitating anxiety.

Smoking also appears to provide a natural protection from anxiety. Howard (1996) argued P/S have low levels of an adrenal hormone called DHEA which appears to have an ameliorating influence on stress by directly counteracting cortisol effects. Logically, the more DHEA the body produces the more resistance it has to anxiety. Nicotine appears to directly influence the levels of DHEA present in the body by increasing the amount of DHEA sulphate, from which DHEA forms. The suggestion by Howard infers smokers with schizophrenia actively smoke to increase levels of DHEA and thereby increase resistance to stress and anxiety.

In summary, empirical evidence shows P/S do not exhibit a specific cognitive deficit. Rather, P/S exhibit dopamine deficiency and debilitating anxiety, which correspond with poor cognitive performance. Schizophrenia has the highest smoking rate in western society and smoking directly increases dopamine and reduces debilitating anxiety, which associate with improved cognitive performance. Therefore, this study formulates the following predictions: tobacco smoking will relate to better cognitive performance in smokers with schizophrenia in comparison to none smokers with schizophrenia; better cognitive performance in smokers with schizophrenia will associate either, directly with a nicotine related increase in dopamine, or indirectly with a nicotine related reduction in debilitating anxiety; because P/S exhibit debilitating anxiety controlling for debilitating anxiety will result in schizophrenia and non-psychiatric people exhibiting equivalent cognitive performance.

Other research investigating tobacco smoking and schizophrenia cognition.

This study appears to be original research investigating schizophrenia cognition in relation to the association between cigarette smoking, debilitating anxiety and dopamine. The rationale for this statement comes from an extensive PsycLit 1974-1997 and Med line 1966-1997 data base research review. This review located no articles investigating the immediate influence of cigarette smoking on schizophrenia cognition in relation to debilitating anxiety or dopamine. However, this review located one article investigating the effects of smoking on schizophrenia cognitive functioning.

Using the Mini Mental Status Examination (MMSE) to measure cognition, Sandyk (1993) investigated the influence of nicotine on cognition in people with chronic schizophrenia. The study found smokers with schizophrenia exhibited significantly less cognitive impairment than non-smokers with schizophrenia. It concluded smoking protects smokers with schizophrenia from developing cognitive deficits. However, Sandyk did not analyse anxiety levels, practice effects or whether smoking actually improves cognitive functioning at the time of cigarette consumption.

Because Sandyk did not measure anxiety it remains uncertain whether cigarette smoking or differing levels of anxiety influenced cognitive ability. In addition to anxiety influencing results, practice effects may also have influenced outcome. People with schizophrenia do learn (Bellack, Blanchard, Murphy & Podell, 1996; Vollema, Geurtsen & Van Voorst, 1995) and people with chronic schizophrenia have multiple hospitalisations in their life time (Kaplan et al., 1994). Subsequently, some subjects who may have had multiple hospitalisations may also have completed the MMSE on more occasions than other people in the study. In these circumstances practice effect may have confounded results. Sandyk did not investigate whether smoking actually

improved cognitive performance at the time of cigarette consumption. Rather, the article investigated whether smoking protected P/S from developing cognitive dysfunction. In summary, it remains uncertain, therefore, whether smoking improves cognitive functioning at the time of cigarette consumption, either directly by increasing dopamine concentrations or indirectly by reducing debilitating anxiety.

Schizophrenia, tobacco smoking, debilitating anxiety and the current study.

The current study believes past research investigating schizophrenia cognitive functioning has neglected the influence of debilitating anxiety on schizophrenia cognitive performance. Reviewed literature appeared to place P/S, anxious by character, into test environments which by their very nature increase anxiety, and concluded they exhibited specific cognitive deficit. The accuracy of results generated from such methodology must be questionable. Not only have such results provided artifactual and invalid indications of schizophrenia cognitive ability, they reinforced the stereotypic view that P/S are different to non-psychiatric people because they exhibit a specific cognitive deficit. Therefore the completion of this study is justified by its social and empirical importance.

Socially this research is important because it endeavours to directly challenge the stereotype P/S exhibit a specific cognitive deficit. Empirically this study is important because it is research into a new area of schizophrenia and provides new insight into schizophrenia cognitive functioning in relation to cigarette smoking, debilitating anxiety and dopamine.

Smoking is a potentially fatal habit (Kaplan et al, 1994). More than eighty per cent of P/S smoke. Smokers with schizophrenia exhibit the same or higher rates of

mortality due to smoking-related illness as non-psychiatric people (Ananth & Burnstein, 1977; Tsuang & Woolson, 1977). Therefore this research is morally important. Before developing cessation programs to reduce schizophrenia smoking-related mortality we need to explain empirically why over 80 per cent of P/S smoke. This study makes its contribution to schizophrenia research using two research designs.

The current study utilised a correlational, independent samples between groups design, to investigate the influence of tobacco smoking on schizophrenia WCST performance. A related within groups design determined subjective and physiological anxiety from pre- and post-cigarette consumption for smoking participants. Assignment of people to groups was based on smoking status (smoking and non-smoking) and diagnosis (schizophrenia and non-psychiatric). Participants in the smoking groups smoked a standard amount of a Benson and Hedges special filter, tailor made, tobacco cigarette immediately prior to Wisconsin Card Sorting Test (WCST) completion. The WCST provided a measure of cognitive performance. Two physiological measures (heart rate and blood pressure) and one self-report measure (Self Evaluation Questionnaire-SEQ) provided an indication of anxiety.

The aims and hypotheses.

There were six aims in this research: to determine if tobacco smoking relates to better WCST performance in smokers with schizophrenia in comparison with non-smokers with schizophrenia, either indirectly by reducing debilitating anxiety or directly by increasing dopamine concentrations; to determine if smoking influenced subjective anxiety in smokers with schizophrenia; to determine if subjective affect relates to schizophrenia WCST performance; to determine if P/S exhibit a specific cognitive

deficit; to provide schizophrenia care givers and health professionals with an empirical explanation for why over 80 per cent of P/S smoke. The study sought to answer these six aims using information generated from the following six experimental hypotheses:

1. Smokers with schizophrenia will score fewer WCST errors, immediately after smoking one tobacco cigarette, than non-smokers with schizophrenia.
2. Smokers with schizophrenia, immediately after smoking one tobacco cigarette, will self-report lower SEQ levels of debilitating anxiety than non-smokers with schizophrenia, immediately prior to completing the WCST.
3. People with schizophrenia will score more WCST errors than non-psychiatric people.
4. When controlling for physiological anxiety, P/S will score equivalent WCST errors than non-psychiatric subjects.
5. People with schizophrenia will score higher levels of SEQ self-report debilitating anxiety, immediately prior to WCST completion, than non-psychiatric subjects.
6. The influence of one tobacco cigarette on WCST errors will depend on diagnosis. Smokers with schizophrenia will gain the most benefit from smoking.

In conclusion

If nicotine associates directly with cognitive performance in smokers, in comparison with non-smokers, with schizophrenia by increasing dopamine levels there should be a significant correlation between the smoking variable and WCST performance. There should be no correlation between anxiety and WCST performance. Smokers with schizophrenia should not differ in physiological and subjective anxiety from pre- to post-cigarette. If nicotine relates indirectly to cognitive performance in

smokers, in comparison with non-smokers, with schizophrenia by reducing debilitating anxiety there should be a significant reduction in physiological and subjective anxiety between pre- and post-cigarette. There should be a significant correlation between debilitating anxiety reduction in smokers with schizophrenia and their WCST performance. If P/S exhibit a specific cognitive deficit non-psychiatric people should perform significantly better than P/S when completing the WCST; controlling for facilitating-anxiety should not influence outcome.

Method

Subjects

Sixty-six people participated in this research, 34 females and 32 males, ranging in age from 17 to 50 years old with an average age across gender of 35.18 years. The 66 people formed two dichotomous groups, 18 smokers and 8 non-smokers with schizophrenia, and 20 smoking and 20 non-smoking non-psychiatric people. Smoking status and diagnosis determined assignment of participants to groups. The 38 people in the two smoking groups smoked a standard amount of a Bensen and Hedges special filter cigarette, provided by the principal researcher, immediately before completing the WCST. Participants were either unknown to the principal researcher or known on a professional basis[3]. All subjects completed the WCST (Heaton, 1981), provided heart rate, blood pressure readings, completed the SEQ (Marteau & Bekker, 1992) and participated under conditions of written informed consent. Recruitment of both P/S and non-psychiatricpeople followed standard procedures and research methodology gained ethical approval from the Hawke's Bay and Auckland North Health Ethics Committees.

Recruitment of non-psychiatric participants.

The professional staff at the Hawke's Bay Psychiatric Unit and Auckland Waitamata Health Taharoto Psychiatric Unit provided the non-psychiatric participants for this research. Recruitment of staff volunteers followed standard procedures. The staff meetings held at each unit provided a forum to introduce the study, and any staff interested in participating arranged a time to meet with the primary researcher to discuss

[3] The principal researcher worked as a psychiatric nurse aide at the Hastings Psychiatric Unit prior to completing this thesis research and subsequently knew some participants on a professional level.

the research. They also signed informed consent and organised a time to complete the WCST. Recruitment of P/S also followed standard procedures.

Recruitment of people with schizophrenia.

Inpatient and outpatient settings provided the population base from which participants with schizophrenia volunteered. The Hastings Psychiatric Unit in Hawke's Bay and the Waitamata North Health Taharoto Psychiatric Unit in Auckland consituted the inpatient settings. Community Mental Health Centres in Hawke's Bay and the Continuing Care Team (CCT) at Waitamata Health in North Auckland constituted the outpatient settings.

Community meetings held weekly at the Hawke's Bay community health centres provided the forum to introduce the study to potential outpatient participants. Any potential participants interested in taking part in the study approached the primary researcher after these meetings to volunteer. Consultant psychiatrists and community centre managers ensured suitability of participants to participate in the research. Introducing the study to outpatient participants in Auckland differed from the procedure used in Hawke's Bay. Clients considered suitable for this research by CCT nursing staff, manager and a consulting psychiatrist received a letter about the study. Any people interested in participating in the study informed their community nurse and they arranged a time for these people to meet with the principal researcher.

The psychiatric hospital in Hastings held weekly community meetings which provided a forum to introduce the study. Due to the acute nature of their schizophrenia, however, recruitment of potential participants required a different strategy than used in the community setting. The first step in this process required consultation with

psychiatric unit psychiatrists and the formation of a provisional list of possible participants. The primary nurse of each potential participant conducted an initial approach to these people to determine their interest in participating in the study. Interested people then met with the principal investigator to discuss the study and sign informed consent to participate. For ethical reasons the first interaction by the principal researcher with male P/S and all interactions with female P/S took place in the presence of their primary nurse. Contact procedures with potential participants at the Taharoto Psychiatric Unit in Auckland followed the same guidelines. Conducting a stringent inclusion and exclusion assessment procedure of volunteers in both inpatient and outpatient settings attempted to make the groups of participants as homogeneous as possible. The research also conducted inclusion and exclusion assessment of non-psychiatric volunteers.

Inclusion criteria:

1. Age: Participants were required to be older than sixteen years of age to ensure they could provide informed consent without requiring approval from their immediate guardian.
2. Gender: Both males and females were permitted to participate in this study.
3. Diagnosis: Only people with an unequivocal diagnosis of schizophrenia, as prescribed by the DSM IV, participated in this research. Inpatient psychiatrists determined diagnosis and the principal researcher recorded diagnosis from participant files in the outpatient settings before conducting cognitive testing.

4. Smoking status: Because this research investigated the immediate effect of tobacco smoking on cognition the non-smoking group contained reformed smokers and non-smokers. Current smokers constituted the smoking group.
5. Current mental state: Careful assessment of people with schizophrenia by clinic psychiatrists, nursing staff, and clinic coordinators from inpatient and outpatient settings ensured suitability of participants before completing the WCST.

Exclusion criteria:

1. Organic history: The study excluded any non-psychiatric subjects who had a history of head injury and/or associated side effects. For example, if any person experienced memory or concentration difficulties post head injury, their participation ceased. The exclusion of participants with a history of organic injury or post accident symptomatology is important because research shows subjects with organic damage perform worse than P/S on the WCST (Malmo, 1974). Schizophrenia cognitive studies frequently use such selection criteria to ensure organic based dysfunction does not confound results (Caudrey et al., 1980; Harris et al., 1990; Succuzzo et al., 1974). Although this criterion initially applied to P/S, a number of participants expressed histories of sporting or traffic accidents. Because of the difficulty in reliably determining the extent of the head injury suffered, and the difficulty in locating subjects with schizophrenia, this exclusion criterion only applied to the non-psychiatric subjects.
2. Drug effects in non-psychiatric controls: Any non-psychiatric controls currently on prescribed medication were excluded from participating in the study. The

exclusion of participants on medication protects the study from the risk of unknown drug effects confounding results.

3. Psychiatric history: The research methodology excluded non-psychiatric subjects with a history of schizophrenia or who had first degree relatives with a history of schizophrenia. Excluding people with such genealogy ensured clear delineation between non-psychiatric subjects and P/S. Literature shows first-degree relatives of P/S and P/S share cognitive tendencies (Holzman et al., 1974; Holzman, Kringlen, Levy, & Proctor, 1978; Holzman Levy & Proctor 1976). Without exclusion non-psychiatric people who exhibited similar cognitive tendencies to P/S could confound results.

Other criteria considered:

1. Education: Although education level did not influence selection criteria it significantly correlates with cognitive performance (Calev, Venables & Monk, 1983; Saccuzzo et al., 1974). Therefore, this study recorded statistically analysed primary, secondary and tertiary training or education.
2. Medication: Medication used in the biological treatment of schizophrenia may improve their cognitive functioning. For example, chloropromazine, risperidone and clozapine have been shown to improve cognitive functioning in P/S (Meadow, Donlon & Blacker, 1975; Sawaguchi, Matsumura, & Kubota, 1988; Stone, Callaway, Jones & Gentry, 1969; Strip & Lussier, 1996; Williams, Ballie, Dickson & Dalby, 1993). However, chlorpromazine and risperidone are dopamine receptor antagonist drugs which block dopamine transmission across the synaptic space. Reduction in the

transmission of dopamine corresponds to reduced cognitive functioning (Berger et al, 1989; Elsworth et al, 1990; Stern & Langston, 1985). Further, Haloperidol and fluphenazine associate with reduced prefrontal cortex activity (Sawaguchi et al, 1988) and haloperidol with reduced cognitive functioning (Levin & Rose, 1995). Consequently it is uncertain whether medications prescribed will enhance or inhibit cognitive functioning in P/S. Therefore this research recorded and analysed medication levels standardised into chloropromazine units as is standard practice.

Materials

The Wisconsin Card Sorting Test.

The Wisconsin Card Sorting test (WCST) measured cognition and consisted of two packs of 64 cardboard cards measuring 73 millimetres square, and four stimuli or key cards. Subjects were required to sort each of the 128 cards, matching them with one of the key cards. All cards displayed a single systematic configuration: triangle, circle, cross or star. Each configuration differed in colour (red, green, yellow or blue) and number (1,2,3 or 4). Maintaining card sequence from one to 64 in each pack of cards for each participant ensured standard order. The principal researcher recorded responses onto a WCST score sheet with blue or black ballpoint pen or pencil. Performance pressure was minimised by the absence of time restraint, and no research finding the WCST aversive was identified by this study; making the WCST an ethical choice.

Self Evaluation Questionnaire.

The Self Evaluation Questionnaire (SEQ) designed by Marteau and Bekker (1992) measured subjective anxiety and consisted of six questions: 'I feel calm, I feel tense, I feel upset, I feel relaxed, I feel content, I am worried'. A likert scale from 1-4 provided the method for participants to rate subjective anxiety: not at all, somewhat, moderately, very much (refer appendix E). Participants responded on the SEQ score sheets using blue or black ballpoint pen or pencil.

Blood pressure and heart rate.

Blood pressure was recorded from the radial artery located on the inside of the elbow joint. There are two types of blood pressure, systolic and diastolic pressures. The systolic pressure is from blood pumped into arteries and the diastolic blood pressure is from when the heart relaxes and allows itself to fill with blood. The systolic pressure is sensitive to temporal changes in blood pressure and indicates internal anxiety (Bellack & Hersen, 1988). Therefore, this research recorded systolic blood pressure to indicate level of physiological anxiety. A systolic blood pressure reading greater than 120 indicates high blood pressure and under 100 indicates low blood pressure (Jefferies, 1979).

The pulse from the radial artery located on the underside of the wrist provided the location to measure heart rate, and a wrist watch provided a method to measure heart beats per minute. Jefferies (1979) reports that more than 72 beats per minute is a high heart rate. The principal researcher recorded heart rate and blood pressure using either a blue or black ballpoint pen.

Design

Research design summary.

A within subjects design was preferable but not possible for the following reasons. Using a within subjects design would have required subjects complete at least two tasks and therefore required added commitment from them, making participation in the study less attractive. Because this study predicted difficulty in recruiting potential participants with schizophrenia, both in community and psychiatric settings, a within subjects design was not possible. This study also predicted a within subjects design was not possible for another reason: although we could control for order effect, the research could not prevent boredom or disheartenment if P/S performed poorly on the first task. People with schizophrenia may perform worse on the second task regardless of order because they were bored, rather than because of differing levels of cognitive ability, thereby confounding results. There was also concern that schizophrenia mental stability may alter between administrations of cognitive tasks and confound results. Further, because psychiatrists screened participants for stability to participate many P/S were also ready for discharge. Consequently, this study could not guarantee subjects availability to participate in a second test administration.

Because a within subjects design was not possible, this study used an independent samples between groups correlational 2 by 2 design, thereby removing the above stated concerns associated with not using a within subjects design for this research. This research also used a related within groups design, with repeated measures of anxiety for smoking participants only.

Independent Variables.

This study utilised two independent variables. The first independent variable was a standard Bensen and Hedges Special Filter cigarette which complied with government regulations, requiring nicotine concentration not exceeding 1.2-1.6 milligrams. Smoking status had two levels, smoking or non-smoking (between groups) with the cigarette being administered only to the smoking groups. Because of large variations in the time taken by subjects to smoke one cigarette it was impossible to standardise the amount of nicotine inhaled based on time. Consequently, participants smoked a predetermined, standardised amount of cigarette, stopping at the manufactures red ink label 'special filter', 5mm above the filter. The cigarette measured, from the ignitable end, 55mm to the red label and 60mm to the filter.

The second independent variable in this study was diagnosis, a categorical independent variable with two levels, people with schizophrenia, as determined by DSM IV diagnosis, and non-psychiatric people (between groups). This study used two dependent variables to determine any association between them and the independent variables.

Dependent variables.

The first dependent variable used in this study was cognition, measured using the WCST, a measure of abstraction which determines the ability to sort cards according to a principle of class membership (Mitchell, 1985). The WCST is an appropriate test for this research because it provides a quantifiable score of cognitive ability and schizophrenia cognitive research frequently uses the same test (Metz, Johnsen, Pliskin & Luchins, 1994; Stratta, Manari, Mattei, Casacchia & Alessandro, 1994).

Cognition was operationalised by recording the number of errors made by participants when completing the WCST and, generated ratio data (Dane, 1990). Each incorrect or error sort incurred one mark. A participant completed an incorrect sort if the card placed did not match the current sort. For example, if a participant placed a yellow circle with a red star when the correct sort was colour, they had completed an incorrect sort. When scoring errors made on the WCST van den Broek, Bradshaw and Szabadi (1993) noted that the scale is invalid unless participants complete all 128 card sorts. When measuring proficiency, therefore, based on the number of errors completed, it is important to have the subjects complete all possible sorts, or the same number of sorts. For example, a person may not complete all 128 card sorts and score more errors than somebody who does complete all 128 card sorts. Therefore, this research scored the first 64 cards, ensuring comparability across subjects. However, the scoring of only the first 64 cards occurred after a number of subjects had completed the test in less than 128 sorts. Therefore, to ensure standard procedure for all participants standard instructions remained the same. Consequently, all subjects received an initial request to sort the 128 cards. After completing the first 64 cards the principal researcher provided the participants with an opportunity to cease sorting, or to continue sorting in order to complete the six sorts, or the remaining 64 cards.

The second dependent variable was anxiety. People often report anxiety feelings differently than physiology indicators imply they feel. Different types of physiological indicators of anxiety also correlate poorly. Therefore, it is important to measure anxiety using a multi-dimensional assessment method (Bellack & Hersen, 1988). The multi-dimensional anxiety assessment method used in this research included two physiological measures (blood pressure and heart rate) and one self-report measure

(SEQ). Systolic blood pressure and heart rate operationailsed physiological indicators of anxiety. Higher blood pressure and heart rate indicated higher physiological anxiety (Kaplan et al, 1994; Bellack & Hersen, 1988). Blood pressure and pulse recordings produced ratio data (Dane, 1990).

The SEQ provided a quick, valid and reliable (Bellack & Hersen, 1988; Marteau & Bekker, 1992) method to operationalise state anxiety of participants immediately before they completed the WCST. Marteau and Bekker define state anxiety as how a person feels in the moment they complete their anxiety scale. People with schizophrenia are anxious by nature (Depue & Fowles, 1973; Kellner et al, 1975; Serben, 1975). Because the SEQ is brief and requires little time to complete it is an ethical choice to use in this research, thereby minimising possible discomfort. Further, the scale has few items, a corresponding low risk that items remain unanswered, and generated categorical data (Dane, 1990).

Covariables.

In addition to independent variables and dependent variables this study also measured and analysed covariables, that is, any variable different from the dependent variable directly under investigation (Tabachnick & Fidell, 1989). Subject allocation to groups took place based on smoking status and diagnosis, not random allocation. In order to reduce theoretical concern of extraneous variables influencing results covariables were operationalised, recorded and analysed. However, because this study measured a finite number of covariables, a risk remains that other covariables which were not measured may influence dependent variables. This research recorded the following covariables: age, cigarettes consumed per day, education, gender and

medication levels for P/S. All covariables provided ratio data, except gender (Dane, 1990).

In summary, the research design incorporates three types of variables, independent and dependent variables and covariables. This study used four groups of subjects to determine any association between the independent variables and the dependent variables.

Groups used in this study.

This study utilised two dichotomous nominal variables, smoking status and diagnosis. Subsequently, this research assigned participants to groups based on smoking status and diagnosis variables and separated each variable into a dichotomy, smokers and non-smokers and P/S and non-psychiatric people. The fact that these groups were independent of each other influenced the type of statistical design suitable for this research.

Choice of statistical analyses.

Data analysis for this research used parametric and non parametric tests.

Parametric tests.

Parametric tests were used because they exhibit more statistical power than non parametric tests. This study applied only those variables generating ratio data to parametric computation: blood pressure, cigarettes consumed per day, cognitive performance, education, heart rate and medication levels.

Independent groups t-test.

The independent groups *t*-test was used to determine whether there is a significant difference in the number of WCST errors between smokers, immediately after smoking one cigarette, and non-smokers with schizophrenia (Coakes & Steed, 1996; Coolican, 1994). This research met all assumptions required before completing a *t*-test calculation[4].

The independent groups *t*-test considered the following experimental hypothesis: People with schizophrenia who smoke will score fewer cognitive errors on the WCST, immediately after smoking one cigarette, than P/S who do not smoke.

The independent groups *t*-test provides a means to answer the following question: Is nicotine associated with better WCST performance in smokers with schizophrenia compared to non-smokers with schizophrenia?

One way Analysis of Variance.

The one way Analysis of Variance or ANOVA design allowed this study to determine whether a significant difference existed between P/S and non-psychiatric people when completing the WCST (Coakes & Steed, 1996; Coolican, 1994). The research met a number of assumptions before conducting an ANOVA.

The one-way ANOVA considered the following hypothesis: People with schizophrenia will score more errors on the WCST than non-psychiatric people.

Using the one way ANOVA also allowed *F* and *p* value comparison between hypothesis three and four: When controlling for the level of physiological anxiety P/S

[4] Because this study uses a number of analyses, and a number of assumptions must be met before their computation, an assumptions section is included in the results section.

will score significantly fewer cognitive errors completing the WCST than non-psychiatric subjects.

The one way ANOVA provided the means to answer the following questions: Do P/S and non-psychiatric people differ in cognitive performance when completing the WCST? Is physiological anxiety associated with reduced schizophrenia WCST performance in comparison to non-psychiatric people?

Multivariate analysis of variance.

The Multivariate Analysis of Variance or MANOVA is an extension of the ANOVA and provided this research with a method for investigating the influence of multiple covariables and reducing the risk of committing type one error (Tabachnick & Fidell, 1989; Weinfurt, 1996). This research meets statistical assumptions required to complete a MANOVA.

The MANOVA provided a means to answer the following question: Do any covariables, or the dependent variables heart rate and blood pressure, differ significantly across groups?

Factorial analysis of variance.

A 2 by 2 factorial design with four cells was computed to determine the interaction effect between smoking status and diagnosis on WCST performance. The first factor is smoking status, with two levels or conditions, smoking and non-smoking. The second factor is diagnosis, also with two levels, P/S and non-psychiatric people. This research meets the statistical assumptions required to complete a factorial analysis (Minium, King, & Bear, 1993).

The factorial design considered the following experimental hypothesis: the influence of one Bensen and Hedges cigarette, on cognitive errors made whilst completing the WCST, will depend on diagnosis.

The factorial design also provided a means to answer the following questions: Do smokers with schizophrenia gain the most benefit from cigarette smoking, in comparison to non-psychiatric people who smoke? Why do more than 80 per cent of P/S smoke?

Analysis of covariance.

The Analysis of Covariance or ANCOVA design was used to investigate the influence anxiety has on schizophrenia cognition in comparison to non-psychiatric people, between smokers and non-smokers with schizophrenia, and to determine if any significant covariables influenced results. This research met the statistical assumptions required to complete an ANCOVA analysis.

The ANCOVA considered the following experimental hypothesis: when controlling for anxiety level P/S will score significantly less cognitive errors when completing the WCST than non-psychiatric subjects.

The ANCOVA provided a means to answer the following questions: Is physiological anxiety associated with reduced schizophrenia WCST performance in comparison to non-psychiatric people? Does physiological anxiety influence the *t*-test between smokers and non-smokers with schizophrenia when completing the WCST? If any covariable differs significantly among groups, do they influence the number of errors scored on the WCST by P/S and non-psychiatric people?

Non parametric tests.

Although non parametric data is less powerful than parametric data, non parametric computations allow maximum use of data sets generated in this research. This study used non parametric analysis for nominal data sets. By administering the SEQ scale to smoking and non-smoking subjects immediately prior to the WCST this study generated unrelated nominal data (Coolican, 1994).

Mann-Whitney U Test.

The information generated by smokers and non-smokers with schizophrenia when completing the SEQ is unrelated and nominal. Therefore, the Mann-Whitney U test is an appropriate test to determine whether any significant difference existed between groups on tests of state anxiety (Coolican, 1994).

The Mann-Whitney (U) test considered the following experimental hypotheses: People with schizophrenia who smoke, immediately after smoking one cigarette, will self-report lower levels of anxiety than P/S who do not smoke when completing the SEQ, immediately prior to completing the WCST. People with schizophrenia will score higher levels of self-report anxiety when completing the SEQ immediately prior to completing the WCST than non-psychiatric subjects.

The Mann-Whitney (U) test provides a method to answer the following questions: Does debilitating anxiety influence schizophrenia WCST performance? Do P/S exhibit higher levels of debilitating anxiety, immediately prior to completing the WCST, in comparison with non-psychiatric people?

Chi-square Test.

A Chi-square analysis determined whether any significant gender difference existed between P/S and non-psychiatric people and between P/S who do and do not smoke. This research also used a Chi-square test to analyse how P/S scored each question within the SEQ. Gender and the SEQ generate nominal data, thereby justifying the use of Chi-square analysis (Coolican, 1994).

The Chi-square design answered the following questions: Do P/S and non-psychiatric groups differ in gender composition? Do schizophrenia groups differ in gender composition? Do people with schizophrenia differ on levels of subjective affect?

Wilcoxon T Signed Ranks Test.

A Wilcoxon (T) Signed Ranks Test determined whether smoking a single cigarette influenced subjective anxiety level for P/S who smoked at pre- and post-cigarette consumption. Pre- and post-tests generate related data and the SEQ generates nominal data, thereby justifying the use of the Wilcoxon computation (Coolican, 1994). The Wilcoxen design answered the following question: Does smoking a single cigarette influence subjective affect in people with schizophrenia who smoke?

Pre-research statistical power analysis.

A pre-experimental power analysis determined statistically acceptable alpha level or significance level, sample size, power coefficient and effect size. This study conducted the following power analysis. First, this research consulted six articles investigating schizophrenia information processing and attention performance and computed an effect size for power calculations ($ES = 0.72$, $N = 6$). However, an effect

size of .72 was too strong and associates with a corresponding risk of finding a smaller effect size non significant (Cohen, 1977). Therefore, based on consulted literature this study used a medium effect size .50 (Bergin and Garfield, 1994; Lipsey, 1990), an alpha or significance level of .05 (Bergin & Garfield, 1994) and a power coefficient of .80 (Cohen, 1977) for power calculations. The subsequent power calculation indicated this research required 50 subjects per group or 200 subjects in total to generate acceptable power (P = .80). Because of time limitations this was an unrealistic expectation for this research. Therefore, this study made alterations to the values in the power computation, reducing sample size to 20 subjects per group, reducing statistical significance to .20 (one tailed) and fixing effect size at .50. The altered power computation generated an acceptable power coefficient of .78 (Cohen, 1977). Because of limited research time and access to P/S, sample size needed to be relatively small. Without good statistical power research is not justifiable (Weinfurt, 1996), therefore this study considered acceptable power paramount.

One tailed significance criterion.

Based on reviewed literature this study predicts smokers with schizophrenia will perform better when completing the WCST in comparison to non-smokers with schizophrenia. Literature indicates that reducing anxiety in non-psychiatric samples increases cognitive performance, and also that P/S and anxious non-psychiatric people appear to exhibit a cognitive dysfunction related to anxiety. Smoking reduces anxiety in non-psychiatric people, and P/S exhibit the highest smoking rate in western society (Ikard & Tompkins, 1973; Huges et al, 1986). Further, P/S exhibit a dopamine deficit (Lewis & Akil, 1997), increasing dopamine associates with better cognitive

performance (Daniel et al, 1991), and smoking increases dopamine (Levin, 1992). Because this research predicts direction of outcome it is acceptable to use a one tail design for the critical *t*-test between smokers and non-smokers with schizophrenia when completing the WCST. Computing a one tailed test improves statistical power by lowering probability this research will commit type one error for the crucial *t*-test (Coolican, 1994). However, because this study uses multiple analyses there is a corresponding inflation of alpha or probability of committing type one error for the remaining statistical computations in this research (Weinfurt, 1996).

Bonferroni Inequality.

The Bonferroni Inequality theory provides a method to determine the maximum value of alpha or probability of committing type one error for a series of tests. The alpha value for a set of tests is equal to or less than the sum of the alpha levels associated with each test (Weinfurt, 1995). Consequently, the more tests a study conducts the higher the alpha level becomes and the greater the risk of committing type one error. Two alpha which apply to statistical analysis in this study are familywise and experimentwise alpha. Because this study conducts multiple tests and comparative analyses inside some of these computations it compensates for both alpha by doing two things.

To compensate for elevations in alpha this study determined the maximum number of tests required to achieve research aims and adjusted alpha to compensate for elevations in probability of committing type one error. This research required sixteen computations, four univariate, seven multivariate and five non parametric tests, and altered the alpha using the following equation: alpha divided by the number of comparisons made (Huberty & Morris, 1989). Applying the nominal alpha from power

analyses (alpha = .20) and the number of tests conducted ($N = 16$) generated a new and statistically acceptable alpha (alpha = .013 to two decimal places). This study uses the .013 alpha for all computations, except the crucial *t*-test between smokers and non-smokers with schizophrenia when completing the WCST. Because the *t*-test computation between schizophrenia groups on WCST performance is predicted this research evaluates the univariate analysis using the nominal alpha (Coolican, 1994).

Further, alterations to the .013 alpha were made to ensure familywise alpha does not inflate when conducting comparative analyses inside ANOVA designs (Tabachnick & Fidell, 1989; Weinfurt, 1996). This study makes alterations to alpha separately for each analysis based on the number of comparisons required, and displays the altered alpha with critical test values in the results section.

Procedure

People who smoke are most nicotine-deficient immediately after waking from sleep (Issac & Rand, 1972). Therefore, this study attempted to conduct all testing of smoking subjects after lunch to reduce nicotine deficiency influence on cognitive testing. In the inpatient setting the principal researcher and primary nurse for all P/S accompanied them to the experimental rooms. In the community settings the primary researcher accompanied male participants to an appointed study room and a female nurse or health clinic manager accompanied the principal researcher in all contact with female participants. In all instances, despite using various study rooms to conduct the research, all testing took place in quiet, well lit and ventilated rooms with standardised seating arrangements. The primary researcher sat either to the left or right shoulder of

the participants, depending on which chair the participant chose, and gave participants standardised instructions after they sat comfortably.

The standardised instructions covered two areas. First, the principal researcher reviewed the study and what the subject's participation involved, and answered any questions at this time. The participants then tossed a standard playing die six times to generate a random number. Using a random number ensured confidentiality of all information generated during testing procedures. The participants then answered 5-6 demographic questions and completed the SEQ - once if there were non-smokers and twice if they were smokers. In hospital settings primary nurses recorded the participant's blood pressure and heart rate, and in the community settings the principal researcher recorded these. All non-smoking subjects followed this procedure.

The smoking subjects completed the same procedure. After this they left the experiment room to consume a cigarette, either in the unit's smoking lounge or in a space provided outside the building. Smoking participants stopped smoking after consuming the standard amount of a Bensen and Hedges cigarette and immediately repeated the SEQ, on a new score sheet, and had their blood pressure and heart rate re-recorded.

The second part of the standard instructions for the research included the directions for the WCST. These instructions followed directly upon completion of the SEQ, heart rate, blood pressure and cigarette consumption. Test instructions followed the standardised form presented in the WCST Manual (Heaton, 1981). The researcher then arranged the sorting cards in a standard order with the first card, single red triangle configuration, at the participant's left side. The principal researcher informed the participants there were correct and incorrect sorting procedures. When the participants

sorted incorrectly the researcher replied 'incorrect', and 'correct when they sorted correctly. The WCST Manual suggests using the words right and wrong. However, staff consultation determined the term wrong generated a more punitive connotation than incorrect. Participants were then asked to sort all the cards.

Before completing the WCST, and after reading the instructions, the researcher paused to ensure that subjects understood, and answered any questions from the participants about the study. The principal researcher was fully responsive to any conversation leading up to the card sort. However, during the sort the principal researcher only indicated the correctness of sort. Although some subjects asked questions during the sorting process the primary researcher politely requested they discuss these at the end of the test. This procedure occurred at all times except when subjects scored more than two incorrect 'other' responses and had spread their cards in a step-wise fashion under the stimulus cards. Heaton (1981) defines an 'other' response as a response not matching the stimulus or key card on any dimension. In such situations the researcher reminded participants to place cards into piles under the stimulus cards, sorting according to stimulus cards and not by cards already sorted. Participants also receive this instruction prior to beginning the WCST. After completion of the task the researcher provided all subjects time to discuss the test. Finally the researcher reminded participants of test-sort confidentiality and thanked them for their participation.

Results

This study found smokers with schizophrenia performed better than non-smokers with schizophrenia when completing the WCST, immediately after the smokers had consumed a single cigarette. The average smoker with schizophrenia performed 29.1 per cent better than the average non-smoker with schizophrenia scoring, 7.03 fewer WCST errors. The smoking variable ($p < .05$) and facilitating-anxiety ($p < .001$) were significantly related to WCST performance. The smoking variable accounted for 11.2 per cent and facilitating-anxiety 41.3 percent of the significant difference between schizophrenia groups when completing the WCST. Smoking did not influence affect or arousal from pre- to post- cigarette consumption in smokers with schizophrenia. People with schizophrenia and non-psychiatric people performed equally when completing the WCST; controlling for facilitating-anxiety did not alter the non significant finding. No interaction effect existed between diagnosis and smoking status. Smokers with schizophrenia smoked significantly more cigarettes (39%) per day than non-psychiatric people who smoked. Non-psychiatric people had more years education than P/S. Schizophrenia groups had more males than females and equivalent medication regimes. All four groups had equivalent measures of age and blood pressure.

This study utilised two dichotomous nominal independent variables, diagnosis (schizophrenia and non-psychiatric people) and smoking status (smoking and non-smoking). This study manipulated one physical independent variable, a Bensen and Hedges cigarette, and examined two dependent variables, cognition and anxiety. This

study measured a number of covariables: age, cigarettes consumed per day, education, gender, medication and years unwell. Participants were assigned to groups based on diagnosis and smoking status. This research evaluated parametric and non parametric statistical computations using an alpha of .013 and made comparisons inside ANOVA designs based on alterations to the .013 alpha (Bray & Maxwell, 1982; Weinfurt, 1994). The crucial one tailed *t*-test between smokers and non-smokers with schizophrenia on WCST errors was evaluated using the nominal .2 alpha.[5] Means and standard deviations for dependent variables and covariables appear in table form throughout the results.

Smokers with schizophrenia, immediately after consuming a single cigarette, performed better than non-smokers with schizophrenia when completing the WCST.

If smoking is associated with better cognitive functioning in smokers with schizophrenia, either directly by increasing dopamine or indirectly by reducing anxiety, then smokers after consuming a single cigarette should score fewer WCST errors than non-smokers with schizophrenia. Smokers with schizophrenia, immediately after smoking a single cigarette, scored significantly fewer WCST errors than non-smokers with schizophrenia.

An independent samples one tailed *t*-test was conducted on WCST errors. The categorical independent variable was smoking status with two levels, smoking and non-smoking. Eighteen smokers with schizophrenia smoked a single cigarette. The results from 26 participants were applied to the *t*-test, which yielded a significant difference

[5] Adjusted alpha appear with the critical values generated from each analyses. All data is applied to analyses with outlying data removed and any transformations completed. Statistical assumptions met for all analyses appear towards the end of the results section.

between smokers ($M = 27.72$, $SD = 9.88$, $N = 18$) and non-smokers ($M = 34.75$, $SD = 7.52$, $N = 8$) with schizophrenia. Ninety five per cent confidence intervals (CI) and effect size (ES) were computed ($t = 1.74$, $df = 24$, $p < .05$, $CI = -1.3$ to 15.36, $ES = .56$). Post hoc power analysis found the t-test computation generated acceptable power ($P = .72$) (Cohen, 1977; Boniface, 1995; Lipsey, 1990). Therefore this study rejects the possibility type one error occurred (Tabachnick and Fidell, 1989). Figure one shows the significant difference between smokers and non-smokers with schizophrenia for mean WCST errors, immediately after smokers consumed a single cigarette.

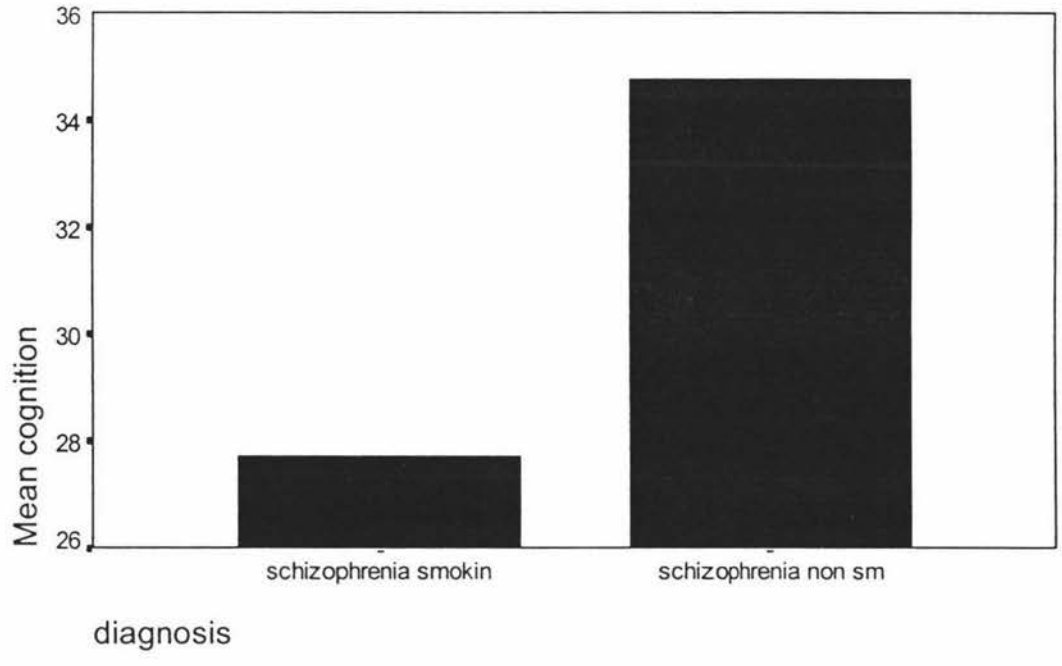

Figure 1. **Showing the mean WCST errors scored by smokers and non-smokers with schizophrenia immediately after smokers consumed a single cigarette.**

The p value tells the reader little about the size and reliability of the significant WCST group difference between smokers and non-smokers with schizophrenia. Therefore 95 per cent confidence intervals and effect size were computed to provide

more meaningful results. Because the *t* value lies between the confidence intervals the test is reliable (Everitt & Hay, 1992). The difference between smokers and non-smokers with schizophrenia WCST performance generated an effect size of .56, a medium difference between groups (Cohen, 1977). Standard deviations directly compare to effect size and represent the area under the normal distribution curve. Because the cognition variable was approximately normal the effect size could be transformed into a *z* score and the *z* score converted into a percentage. Smokers with schizophrenia performed 29.1 percent better than the average non-smokers with schizophrenia (Bergin and Garfield, 1994; Coolican, 1994). However, it remains uncertain how much of the significant difference between schizophrenia groups is accounted for by smoking.

Consequently this study computed a one tail Pearson's r correlation for the smoking variable ($r = .34, p < .05$). Converting the correlation into a percentage shows the smoking variable accounted for 11.2 per cent of the WCST difference between smokers and non-smokers with schizophrenia. However, it remains uncertain whether the smoking variable directly or indirectly accounted for 11. 2 per cent of the WCST variance between schizophrenia groups.

Smoking did not influence affect or arousal from pre- to post-cigarette consumption in smokers with schizophrenia.

If smoking directly associated with better WCST performance in smokers than in non-smokers with schizophrenia by increasing dopamine rather than indirectly reducing anxiety, affect and arousal should be equivalent from pre- to post-cigarette consumption in smokers with schizophrenia. Smokers with schizophrenia experienced

equivalent subjective affect and physiological arousal from pre- to post-cigarette consumption.

A related data Wilcoxen Signed Ranks Test was conducted for SEQ measured subjective anxiety. A single cigarette was the independent variable administered to smokers with schizophrenia. The results from 18 participants were applied to analyses, which found smoking a single cigarette did not influence subjective affect in smokers with schizophrenia from pre- to post-cigarette. The Wilcoxen test yielded a non significant difference in the following responses: I feel calm, content, relaxed, tense, upset or worried (T = -.29, -2.3, -.28, -.33, -1.13, -.09, respectively all ns).

Two related data t-tests were conducted from pre- to post-cigarette consumption for blood pressure and heart rate in smokers with schizophrenia. Assumptions required to conduct the computations were met and results from 18 participants were applied to each respective t-test. Both analyses yielded non significant differences between pre- and post-smoke consumption for blood pressure (t = 2.20, df = 17, p = .04) and heart rate (t = 1.67, df = 17, p = .11).

In summary, tobacco smoking related directly, rather than indirectly, to better WCST performance by smokers, in comparison with non-smokers, with schizophrenia. Further, the smoking variable only accounted for 11.2 per cent of the significant difference between schizophrenia groups. Therefore, another variable must also relate to the significant WCST difference between schizophrenia groups. The introduction stated facilitating-anxiety is anxiety which facilitates or improves cognitive functioning. Perhaps facilitating-anxiety directly related to better WCST performance by smokers in comparison with non-smokers with schizophrenia.

In comparison with non-smokers, smokers with schizophrenia exhibited higher facilitating-anxiety.

If facilitating-anxiety directly associated with better WCST performance in smokers with schizophrenia, in comparison to non-smokers with schizophrenia, then the smokers should exhibit subjective relaxation and higher blood pressure and heart rate than non-smokers, immediately before WCST completion. Smokers and non-smokers with schizophrenia exhibited equivalent subjective relaxation. However, smokers exhibited significantly higher heart rate and 8.5 milligrams higher blood pressure. Therefore smokers exhibited higher facilitating-anxiety than non-smokers (Alpert & Haber, 1960).

One tailed Mann-Whitney non parametric U tests found no significant difference between smokers (post-cigarette) and non-smokers with schizophrenia on SEQ scores of subjective affect, immediately before completing the WCST. The results from 26 participants were applied to the Mann-Whitney test, which yielded non significant differences for each of the following responses: I feel relaxed, calm, content, upset, tense, worried (U = 47, 43, 51.5, 61.5, 43 & 52, respectively all ns). Because there was no difference between schizophrenia groups for subjective affect this study computed a Chi-square analysis to determine what type of subjective anxiety schizophrenia participants experienced.

A Chi-square analysis was conducted for each of the SEQ individual questions answered by 26 P/S, irrespective of smoking status. People with schizophrenia were significantly 'not at all tense (X^2 = 12.46, df = 3, p = .006), 'not at all upset' (X^2 = 38.31, df = 3, p = .0001) and 'not worried' (X^2 = 15.54, df = 3, p = .0014) and they almost

reached significance for 'somewhat content' ($X^2 = 10.62$, $df = 3$, $p = .014$) and 'somewhat relaxed' ($X^2 = 10.61$, $df = 3$, $p = .014$).

A one way between groups MANOVA was conducted in the descriptive statistics and found a significant main effect for heart rate ($F = 8.70$, $df = 3, 62$, $p < .001$). A post hoc student Newman-Keul paired comparison found smokers exhibited significantly higher heart rate than non-smokers with schizophrenia at the .05 significance level. Although there was no significant difference between groups for blood pressure ($F = 1.86$, $df = 3, 62$, $p = .15$) smokers with schizophrenia exhibited blood pressure 8.5 milligrams higher than non-smokers.

Although smoking did not influence anxiety in smokers with schizophrenia, smokers exhibited greater facilitating-anxiety than non-smokers with schizophrenia immediately before completing the WCST. Therefore, it appears facilitating-anxiety directly associated with the significant WCST difference between schizophrenia groups.

Table 1 shows all four groups' means and standard deviations for the following variables: heart rate per minute, systolic blood pressure, WCST errors. Although blood pressure and heart rate increase for both schizophrenia and non-psychiatric smokers the increases are non significant. There is no difference between groups on blood pressure. Smokers with schizophrenia recorded higher heart rate than all other groups. There is an interesting similarity in cognitive errors made and blood pressure readings for both smoking groups.

Table 1. Means and Standard Deviations for Schizophrenia and Non-Psychiatric Smokers and Non-Smokers Dependent Variables: Heart Rate Per Minute and Systolic Blood Pressure, WCST Errors.

	Cognitive Errors	Blood Pressure (systolic)	Heart Rate (per minute)
P/S who smoke (N=18)	27.72* 10.20	120.50→125.39 13.98→12.40	93.33→99.77** 17.94→14.74
P/S who don't smoke (N=8)	34.75 7.52	116.88 13.97	84.37 6.63
Non-psychiatric who smoke (N=20)	27.20 11.90	122.85→126.50 11.94→15.76	79.85→86.30 10.04→8.39
Non-psychiatric who don't smoke (N= 20)	21.50 9.87	131.75 17.63	80.80 13.46
Average	27.79 9.87	125.13 14.94	87.81 10.81

Note;

(a.) Pre cigarette measurement → Post cigarette measurement.
(b.) Top numbers are mean scores and bottom numbers standard deviations.
(c.) All values rounded to two decimal places.
(d.) Smokers heart rate and blood pressure are post smoke measurements
* $p < .2$
** $p < .05$.

Controlling for facilitating-anxiety dissolved the original significant WCST difference between smokers and non-smokers with schizophrenia.

If facilitating-anxiety directly associated with better cognitive performance in smokers, in comparison with non-smokers, with schizophrenia, then controlling for physiological arousal should see the WCST significant difference between schizophrenia groups reduce or dissolve. Results show controlling for blood pressure and heart rate found a non significant difference between smokers and non-smokers when completing the WCST.

A one way ANCOVA was conducted for the number of errors made while completing the WCST. The categorical independent variable was smoking status with two levels, smokers and non-smokers with schizophrenia, the covariables were heart rate and systolic blood pressure. Statistical assumptions required to complete the computation were met. The results from 26 participants were applied to the ANCOVA, which yielded a non significant difference between the smokers and non-smokers on the number of errors made when completing the WCST ($F = .960$, $df = 1, 25$, $p = .34$). Although it appears that facilitating-anxiety when completing the WCST accounts for more variance between smokers and non-smokers with schizophrenia than the smoking variable, it remains uncertain how much more.

Facilitating-anxiety accounts for 41.3 percent of the variance between smokers and non-smokers with schizophrenia when completing the WCST.

If facilitating-anxiety accounts for more of the significant difference between schizophrenia groups completing the WCST than the smoking variable, then conducting Pearson's correlations should find physiological arousal accounts for more variance. Facilitating-anxiety accounted for 41.3 per cent of the significant WCST difference between schizophrenia groups. Considering the smoking variable accounted for 11.2 per cent of the WCST difference between groups, facilitating-anxiety accounts for 30.1 per cent more variance between schizophrenia groups.

One tail Pearson's r correlations were conducted for blood pressure ($r = .29$, $p = .74$) and heart rate ($r = .57$, $p < .001$). Converting the correlations into percentages showed blood pressure accounts for 8.5 per cent and heart rate 32.8 per cent of the variance in the significant difference between smokers and non-smokers with

schizophrenia when completing the WCST. Facilitating-anxiety accounted for 41.3 per cent of the WCST variance between schizophrenia groups.

In summary, tobacco smoking and facilitating-anxiety directly associated with better WCST by smokers, in comparison with non-smokers, with schizophrenia. The next section shows P/S, irrespective of smoking status, and non-psychiatric people perform equally when completing the WCST.

People with schizophrenia and non-psychiatric people perform equally when completing the WCST.

If P/S exhibit a specific cognitive deficit in comparison to non-psychiatric people then they should perform worse when completing the WCST. However, results show P/S and non-psychiatric people performed equally when completing the WCST.

A one way between groups ANOVA was conducted on errors made while completing the WCST. The categorical independent variable was diagnosis with two levels, schizophrenia and non-psychiatric people. The statistical assumptions required to conduct an ANOVA were met. The results from 66 participants were applied to the ANOVA which yielded a non significant difference between schizophrenia ($M = 29.88$, $SD = 9.88$, $N = 26$) and non-psychiatric people ($M = 24.61$, $SD = 11.23$, $N = 40$) ($F = 4.23$, $df = 1, 64$, $p = .04$. $ES = .46$). Figure 2 shows the mean number of errors scored by P/S and non-psychiatric people when completing the WCST, irrespective of smoking status.

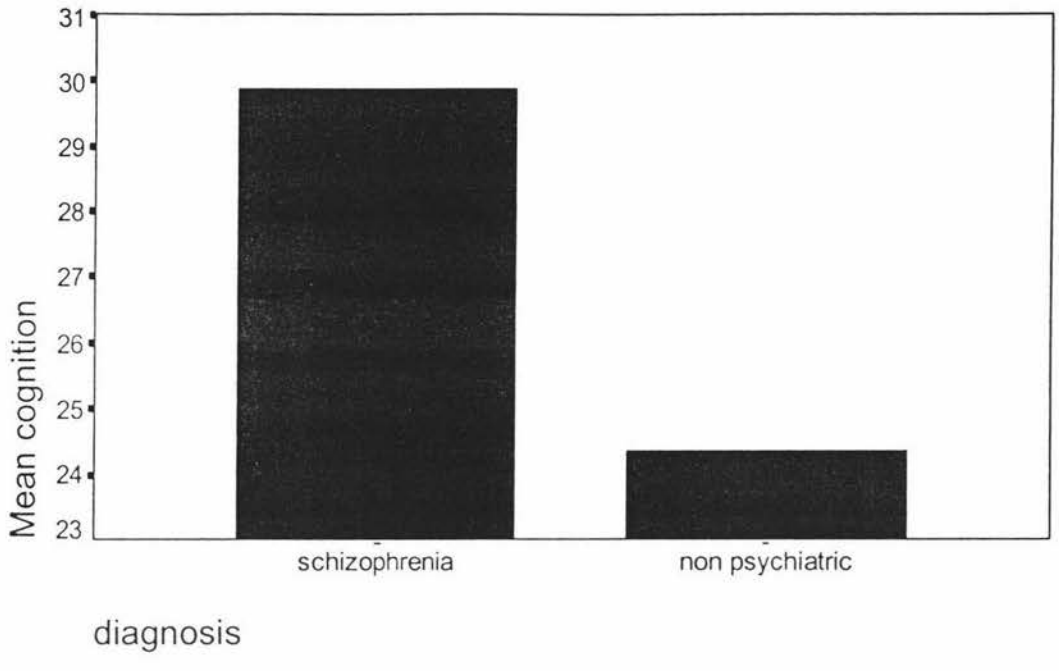

Figure 2. **Shows the mean WCST errors scored by P/S and non-psychiatric people.**

Although schizophrenia and non-psychiatric people performed equally on the WCST it is uncertain whether anxiety influenced cognitive performance. Therefore the *p* value was shown in order to make direct comparisons with the *F* value when holding systolic blood pressure and heart rate constant. The next section determines anxiety levels and anxiety type exhibited by both schizophrenia and non-psychiatric people immediately before completing the WCST.

Both P/S and non-psychiatric people exhibited facilitating-anxiety.

If facilitating-anxiety is related to the equivalent WCST performance between schizophrenia and non-psychiatric people then both populations should exhibit equivalent facilitating-anxiety. People with schizophrenia exhibited higher facilitating-anxiety than non-psychiatric people.

One tailed Mann-Whitney non parametric U tests conducted for the six questions in the SEQ between schizophrenia and non-psychiatric people found non-psychiatric people significantly more content ($U = 36.5, p = .0001$) and relaxed ($U = 62.5, p = .004$) than P/S. However, results also show a non significant difference between non-psychiatric people and P/S on scores of calmness, tenseness, worry and upset ($U = 80.5$, 91, 123.5, 120, respectively all ns).

An earlier presented Chi-square analysis conducted for each of the individual questions of the SEQ answered by P/S, irrespective of smoking status, showed P/S were significantly 'not at all tense ($X^2 = 12.46, df = 3, p = .006$), 'not at all upset' ($X^2 = 38.31$, $df = 3, p = .0001$) and 'not worried' ($X^2 = 15.54, df = 3, p = .0014$). Because schizophrenia people were not tense, worried or upset, and did not differ from non-psychiatric people on levels of subjective affect, both populations exhibited subjective relaxation.

Table 2 shows means and standard deviations for smoking and non-smoking schizophrenia and non-psychiatric people: age, heart rate per minute, systolic blood pressure, WCST errors, years education. People with schizophrenia recorded higher heart rate per minute than non-psychiatric people. Non-psychiatric people had more years education than P/S. There is no difference between schizophrenia and non-psychiatric people on WCST errors.

Table 2. Means and Standard Deviations for Schizophrenia and Non-Psychiatric People and Smoking and Non-Smoking People Dependent and Covariables: Age, Education, Heart Rate Per Minute, Systolic Blood Pressure, WCST Errors.

	Age	Cognitive Errors	Blood Pressure (systolic)	Education (Years)	Heart Rate (per minute)
P/S	35.00	29.88	121.88	11.73	95.40*
(N = 26)	9.57	9.88	12.70	2.03	11.03
Non-psych	35.30	24.61	129.73	14.77**	84.10
People	9.24	11.23	16.18	2.58	9.37
(N= 40)					
Smoking	35.05	27.45	125.97	13.22	92.82
people	9.64	10.98	14.09	2.86	11.08
(N=38)					
Non-smoking	35.36	25.29	127.50	14.05	81.82
people	8.97	10.97	17.78	2.68	11.90
(N=28)					

Note;

(a). Top numbers are mean scores and bottom standard deviations.

(b). All values rounded to two decimal places.

* $p = 0.001$

** $p < 0.001$

A one way between groups MANOVA was performed in the descriptive statistics on four variables for schizophrenia and non-psychiatric people: age, education, heart rate, systolic blood pressure. The results from 66 participants were applied to calculations and found P/S exhibited significantly higher heart rate ($M = 95.4$, $SD = 1.03$, $N = 26$) than non-psychiatric people (($M = 84.10$, $SD = 9.37$, $N = 40$) ($F = 12.80$, $df = 1, 64$, $p = .001$)). There was a non significant difference between these two groups for blood pressure ($F = 2.7$, $df = 1, 64$, $p = .12$).

People with schizophrenia experienced subjective relaxation and higher heart rate than non-psychiatric people. Therefore they exhibited higher facilitating-anxiety than non-psychiatric people, immediately before completing the WCST.

When controlling for facilitating-anxiety the difference between non-psychiatric people and P/S on WCST error remains non significant.

If facilitating-anxiety associated with improved cognitive performance in schizophrenia, resulting in equivalent WCST performance with non-psychiatric people, then controlling for physiological arousal should result in non-psychiatric people scoring significantly fewer WCST errors. However, controlling for physiological arousal finds schizophrenia and non-psychiatric people still performing equivalently on the WCST.

A one way between groups ANCOVA was performed on number of errors made whilst completing the WCST. The categorical independent variable was diagnosis with two levels, schizophrenia and non-psychiatric people. Two covariables were used in the computation, heart rate per minute and systolic blood pressure. Assumptions required for conducting an ANCOVA were met. The results from 66 participants were applied to the analysis, which yielded a non significant difference between schizophrenia and non-psychiatric people ($F = 6.24$, $df = 1, 64$, $p = .015$).

In summary schizophrenia people did not differ from non-psychiatric people when completing the WCST. Further, anxiety did not influence WCST outcome. The next section explores whether smokers with schizophrenia gain the most cognitive benefit from tobacco smoking in comparison to non-psychiatric smokers.

The influence of smoking a single cigarette did not depend on diagnosis.

If P/S smoke to help themselves think and gain more cognitive benefit from tobacco smoking than non-psychiatric people, then the influence of smoking a cigarette on WCST performance should depend on diagnosis. When considering the graph of the interaction effect (refer figure, 3) there appears to be a full interaction, however the effect is not significant.

A 2 by 2 Factorial ANOVA was performed on one dependent variable, WCST errors. The categorical independent variables were diagnosis and smoking status, both with two levels: schizophrenia and non-psychiatric, smoking and non-smoking people. The factorial ANOVA was conducted on 66 participants and assumptions required to conduct the analysis met. The interaction effect is the only effect of interest for this research. Therefore, main effects are not reported. The analysis provided a non significant interaction effect ($F = 5.34$, $df = 1, 65$, $p = .024$).

Figure 3 shows what ostensibly appears to be a full interaction effect where the association between smoking status and cognitive functioning depends directly on diagnosis (Coolican, 1994). Smokers with schizophrenia appear to gain the most from cigarette smoking.

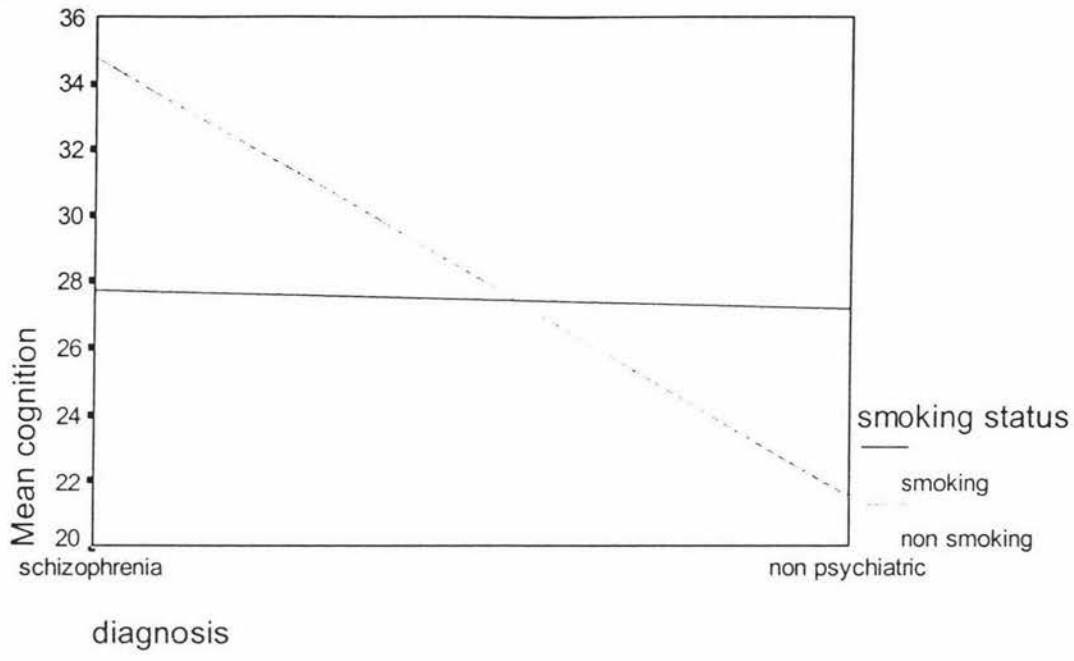

Figure 3. **Interaction effect of mean WCST errors made by both smoking and non-smoking schizophrenia and non-psychiatric people. The non-smokers line runs from 34.75 to 21.50, and the smokers line from 27.72 to 27.2.**

Descriptive statistics.

The next section summarises the descriptive analysis conducted in this research. This research completed descriptive analyses utilising data generated from both dependent variables and covariables.

A one way between groups MANOVA was performed for schizophrenia and non-psychiatric people on four variables: age, education, heart rate and systolic blood pressure. The categorical independent variable was diagnosis with two levels, schizophrenia and non-psychiatric people. The results from 66 participants were entered into calculations and the four univariate ANOVA designs conducted inside the MANOVA were evaluated using an alpha of .003. The analysis yielded a non significant difference between schizophrenia and non-psychiatric people for age

($F = .02$, $df = 1, 64$, ns) and blood pressure ($F = 2.7$, $df = 1, 64$, $p = .12$). Results showed non-psychiatric people had significantly more years education than P/S ($F = 25.77$, $df = 1, 64$, $p < .001$) and P/S had a significantly higher heart rate than non- psychiatric people ($F = 12.80$, $df = , 64$, $p = .001$). Education did not influence WCST performance between schizophrenia and non-psychiatric people.

A post hoc one way ANCOVA was conducted for number of errors made while completing the WCST. The categorical independent variable was diagnosis with two levels, P/S and non-psychiatric people, and the covariable was years education. Assumptions were met and the analysis completed at a .013 alpha level. The results from 66 participants were applied to the ANCOVA, which yielded a non significant difference between the two groups ($F = .98$, $df = 1, 64$, $p = .33$).

A second one way between groups MANOVA was performed for smokers and non-smokers with schizophrenia on four variables: age, education, medication and years unwell. The categorical independent variable was smoking status with two levels, smoking and non-smoking. The data from 26 participants were applied to calculations and the four univariate F tests conducted inside the MANOVA were evaluated using an alpha of .003. The analysis yielded a non significant difference for age ($F = .03$, $df = 1, 24$, ns), education ($F = .06$, $df = 1, 24$, ns), medication ($F = 1.05$, $df = 1, 24$, $p = .32$) and years unwell ($F = .04$, $df = 1, 24$, ns).

Post hoc Chi-square analysis using an altered alpha of .012, to avoid inflation in experimentwise alpha, found no significant difference for types of generic medication prescribed to P/S ($X^2 = 5.48$, $df = 7$, ns). Chi-square analysis found no significant difference between dopamine antagonistic medication and medication which does not block dopamine transmission for non-smokers ($X^2 = 2.79$, $df = 1$, ns) and smokers with

schizophrenia ($X^2 = 3.1$, $df = 6$, ns). Table 3 presents medications prescribed to schizophrenia participants in this research.

Table 3. Generic Medication Names Prescribed to Smokers and Non-Smokers with Schizophrenia, Frequency of Prescription and Dopamine Receptor Blocking Status.

Generic name	Smoke	Do not Smoke	Total Cases	Dopamine receptor blocker
Clozapine	3	2	5	No
Olanzopine	4	0	4	No
Resperidone	2	0	2	No
Chlorpromazine	4	1	5	Yes
Fluphenthixol	4	1	5	Yes
Haloperidol	2	3	5	Yes
Thioridazine	1	1	1	Yes
Trifluoperazine	0	1	1	Yes
Total	20	8	28	

A third one way between groups MANOVA was conducted on four variables: age, blood pressure, education and heart rate. The categorical independent variables were diagnosis and smoking status each with two levels: smokers and non-smokers with schizophrenia, smoking and non-smoking non-psychiatric people. The results from 66 participants were applied to computations. Four univariate ANOVA were conducted inside the MANOVA design and evaluated using an altered alpha of .003. The analyses found a non significant difference for age ($F = .016$, $df = 3, 62$, ns) and for blood pressure ($F = 1.86$, $df = 3, 62$, $p = .15$). However a significant difference was found for education ($F = 8.41$, $df = 3, 62$, $p < .001$) and heart rate ($F = 8.70$, $df = 3, 62$, $p < .001$). Using post hoc student Newman-Keul paired comparisons found both non-psychiatric groups had significantly more years education than the two schizophrenia groups and

smokers with schizophrenia had significantly higher heart rate than the other three groups.

An independent samples one tailed *t*-test was conducted on number of cigarettes smoked by smoking participants on a daily basis. The categorical independent variable was diagnosis with two levels, schizophrenia and non-psychiatric people. The results from 38 participants were applied to analysis, which found smokers with schizophrenia smoked significantly more cigarettes per day (39%) than non-psychiatric smokers ($t = 3.70$, $df = 36$, $p < .005$).

Chi-square analysis indicated that irrespective of smoking status, there were significantly more females (78.6 %) in non-psychiatric people than P/S ($X^2 = .8.9$, $df = 1$, $p < .01$). There were more males (77. 8 %) than females ($X^2 = 7.54$, $df = 1$, $p = .006$) with schizophrenia. Pearson's product-moment correlation found a negative low non significant correlation between gender and WSCT performance in P/S ($r = -.29$, $p = .16$).

Table 4 shows the means and standard deviations for variables used in the descriptive statistics computed in this research for smoking and non-smoking schizophrenia and non-psychiatric participants: age, cigarettes consumed per day, education, gender, medication levels and years unwell. Both non-psychiatric groups had more years education than both schizophrenia groups. There was a clear female gender difference between diagnostic groups, with more females in the non-psychiatric group than the schizophrenia group, irrespective of smoking status. There was also a clear gender difference within the schizophrenia group, with both smokers and non-smokers with schizophrenia consisting of more males than females. Smokers with schizophrenia

smoke more cigarettes per day than non-psychiatric smokers. The groups were evenly distributed for age.

Table 4. Covariable Means and Standard Deviations for Schizophrenia and Non-Psychiatric Smokers and Non-Smokers: Age, Education, Gender, Medication Levels, Smokes Consumed Per Day, Years Unwell.

	Age	education (in years)	gender	medication (CPZ) (c)	smokes	years unwell (SQRT) b)
P/S: smoke	35.41	11.67	16(m)***	21.12	22.78 ****	3.23
(N=18)	10.19	2.06	2(f)	5.42	7.78	1.29
P/S: don't smoke	35.50	11.88	7(m)***	18.51	----	3.1
(N=8)	8.45	2.10	1(f)	8.94	----	1.53
Non psychiatric	34.77	14.63 *	5(m)	-----	13.85	----
who smoke	9.72	2.80	15(f)**	-----	7.08	----
(N=20)						
Non psychiatric	35.30	14.92 *	10(m)	-----	----	----
who don't smoke	9.37	2.40	10(f)**	-----	----	----
(N= 20)						
Average	35.25	13.28		19.82	18.28	3.17
	9.43	2.34		7.18	7.43	1.34

Note;
(a.) Top numbers are mean scores and bottom numbers standard deviations.
(b.) All values rounded to two decimal places.
(c.) Years unwell was transformed using a square root transformation (SQRT).
(d.) Medication levels standardised into chloropromazine (CPZ) units and transformed using square root transformations (SQRT).
(e.) (m) refers to male gender and (f) female gender.
* $p < .05$
** $p < .01$
*** $p = .006$
**** $p < .001$

General statistical assumptions.

Certain statistical assumptions were met for each dependent variable and covariable before this research computed parametric analyses. All tests required assumptions of variable normality, linearity and homogeneity of variance.

All parametric analyses used in this research required univariate normality. With-out normality the risk of type one error increases and statistical power decreases (Tabachnich & Fidell, 1989; Coakes & Stead, 1996). The probability of discovering a significant difference between groups diminishes and the confidence with which the study draws conclusions weakens (Coolican, 1994). Using both statistical (skew, kurtosis and Lilliefors statistics) and graphical (expected normal probability plots) techniques this study considered variable normality within each of the four participant groups, and for each variable individually. Normality was acceptable for the three dependent variables and four covariables: blood pressure, heart rate, cognitive errors, age, cigarettes consumed, education, medication levels. However normality did not exist for medication levels and years unwell (see transformation section), both of which required square root transformation to ensure acceptable normality.

Linearity, or the assumption that a straight line relationship exists between variables within each group, is an important requirement for multivariate analysis of variance statistics (Tabachnick & Fidell, 1989). It is important because multivariate statistical computation requires linear variable combinations to generate accurate results. Therefore, the more violated the assumptions of linearity the more distorted the results, and the more misrepresentative the interpretations of these results might be. A bivariate scatter plot was conducted for heart rate and cognition, the two variables exhibiting the least normal distribution. A linear relationship existed between these variables and consequently linearity is assumed in the other variable combinations.

Homogeneity of variance is met when there is a non significant difference between samples' variances (Coolican, 1994). Homogeneity of variance is important because multivariate analysis bases computations on variance within groups or cells and

between groups. Therefore, the more difference between samples' variances the higher the chance of committing type one error and the lower statistical power. Consequently, there is less confidence in rejecting any null hypothesis. The Multivariate analyses of variance provide robust protection for deviation from homogeneity of variance if no outliers exist and if sample sizes do not differ (Tabachnick & Fidell, 1989). This study removed outliers before variable transformation. Because samples did differ in size certain parameters had to be adhered to in order to ensure homogeneity of variance.

The ratio between the largest and the smallest groups had to be less than 4:1. Cell variance (standard deviation squared) between largest and smallest variance should not exceed 20:1 (Tabachnick & Fidell, 1989). The ratio between the largest group sample and the smallest group sample equalled 2.5 :1 and the ratio between the largest and smallest variance was well under 20:1. The ratio of blood pressure to heart rate equalled 2.7:1, blood pressure to cognition 2.3:1, and heart rate to cognition equalled 1.9:1. In summary, homogeneity of variance for all variables and covariables was not violated.

Statistical assumptions specific to each test design.

In addition to the general assumptions stated above more specific assumptions had to be met for each analysis before their respective computation. These assumptions were met and appear in the following section.

Assumptions required to complete an ANOVA design were met. Samples were drawn from normally distributed populations. Research reviewed in the introduction of this current study uses multivariate analyses for both schizophrenia and non-psychiatric people (Braff & Succuzzo, 1981; Saccuzzo et al, 1974). Therefore the assumption that

samples would have been drawn from a normal population is met. Statistical and graphical methods also indicate sample normality exists, inferring population normality exists. Homogeneity of variance exists as indicated by Levene's test for homogeneity of variance.

The one way ANCOVA analyses conducted in this research adhered to certain statistical assumptions before computation. The individual scores on both the dependent variable and covariables were independent of each other and the covariables exhibited normality. Oval scatter plots indicated linear relationships between the dependent variable and covariables so results can be confidently interpreted (Coakes & Steed, 1996). The research measured blood pressure and heart rate using techniques frequently used in modern medicine. Therefore, reliability of measurement methods is assumed.

Three one way between groups MANOVA were conducted in this research. All MANOVA designs met required statistical assumptions before computation. There were more cases in each cell than dependent variables. Univariate normality and linearity were proven for dependent and covariables. Degrees of freedom exceeded 20, even with uneven sample sizes, therefore, multivariate normality is assumed (Tabachnick & Fidell, 1989). Univariate normality was proven within groups and because both univariate and multivariate normality was proven, linearity is also assumed. All three variables exhibited homogeneity of variance and covariance as indicated by a non significant Box's M test. Outliers were screened using linear regression and Mahalanobis distances; no outlying data was discovered for any variable. Because log determinants were greater than -9.21034, singularity of variables was assumed.

In summary, this research met statistical assumptions required for computation of the parametric analyses used. Therefore, interpretations of the results and

generalisations to other schizophrenia research, using the same methodology, can be made with confidence.

Removing univariate and multivariate outlying data.

Outlying data is defined as cases with extreme values on one variable or a combination of variables which unduly influence statistics (Tabachnick & Fidell, 1989). This research removed univariate outlying data before considering data transformations for two reasons: Tabachnick and Fidell conduct the same procedure when screening grouped data and removing the impact of outlying data improves normality. Consequently transformations may not be required. Avoiding transformations is beneficial because there is a corresponding limitation of the study's results to other schizophrenia research when transforming data. There is also an increase in result complexity, making interpretation harder. In summary, outliers were removed first in an attempt to avoid the need for data transformation.

This study used the following procedure to reduce the influence of outlying data: First, a check was made to ensure outlying data had been correctly entered into the data set. Second, a check was made for redundant variables. Because dependent variables did not correlate highly (refer correlation matrix, appendix F) it was not possible to delete any unnecessary variables exhibiting outlying data. Outlying data existed in both schizophrenia and non-psychiatric people and because participants with schizophrenia were more difficult to obtain than non-psychiatric participants their cases were not deleted. Consequently, outlying data was retained and altered according to the procedures in Tabachnick and Fidell. This study re-valued the outlying data point to equal one unit more or less than the variable's next most extreme point. Non-psychiatric

volunteers were easier to locate for control groups, therefore this study deleted non-psychiatric cases exhibiting outlying data. Refer appendix A for list of cases re-valued or deleted.

Removing Multivariate outliers for grouped data took place after removal of univariate outliers and consideration of data transformations, as recommended by Tabachnick & Fidell (1989, p. 106) when screening grouped data. Because multivariate outliers also unduly influence results, as do univariate outliers, they must be located and their influence reduced. This research used Mahalanobis distances to determine whether any multivariate outliers existed both in dependent variables and covariables. The Mahalanobis distance is the distance which any one case lies from the centroid or centre point created from the mean of all variables (Tabachnick & Fidell, 1989). Using Mahalanobis distances, a Chi-square critical value of 13.8, and an alpha of .001, no multivariate outliers were located for dependent variables cognition, heart rate and blood pressure, or for covariables age, cigarettes consumed per day, education, medication levels and years unwell. Locating no multivariate outliers was expected because all variables except medication prescribed and years unwell exhibited good normality, linearity and had univariate outliers already removed.

In summary, because this research uses meaningful units of measurement for age, cognition, blood pressure, cigarettes consumed per day, education and heart rate it was beneficial to avoid transformations. It was important to avoid transformations although they may improve normality, because they increase complexity of result interpretation and would have made results somewhat meaningless to lay people. Consequently univariate outliers were screened for and removed in an attempt to avoid

having to transform both dependent variables and covariables. However, because normality was not perfect in all the variables, this study considered transformations in order to see if they provided any benefit to variable and group normality.

Transformations.

Transforming variables may improve normality and linearity. Therefore accuracy of analysis, statistical power and confidence in rejecting the null hypothesis also improves (Tabachnic & Fidell, 1989). Benefit from transformations is not always guaranteed. An increase in result complexity, and consequently result interpretation, can negate improvements made when using transformations (Tabachnick & Fidell, 1989). For example, people understand blood pressure if presented as systolic over diastolic blood pressure. However, if presented as square roots, logarithms, or a combination of reflections and transformations of these units the results becomes more ambiguous. Further, transforming data limits possible generalisation from this study to other schizophrenia research exhibiting the same methodology. This is important because this study wishes to generalise results to past schizophrenia cognitive research. Therefore it is important to consider any benefit gained from transformation against subsequent increase in interpretation difficulty and generalisation limitation. Determining if transformations are necessary requires the consideration of both graphical and statistical information (skew, kurtosis and Lilliefors statistic). The closer to zero skew and kurtosis lie the better the normality (Coakes & Steed, 1996).

There is no standard procedure to determine excessive skew or kurtosis. However, using guidelines from Tabachnick and Fidell (1989) this research experimented with square root and logarithm transformations, and reflecting with square

root and logarithm transformations. Transformations were conducted for two reasons: removing outlying data did not generate perfect normal distributions for dependent variables or covariables and Tabachnick and Fidell recommended that in most cases transformations improve data analysis. The decision to transform data was based on three criteria:

1. Moderate to severe skew and kurtosis had to be present. Transforming data exhibiting mild positive skew is unbeneficial because it merely reflects values and creates mild negative skew (Tabachnick & Fidell, 1989).
2. Non significant Lilliefors statistics.
3. Poor visual indicators of normality as displayed in expected normal probability plots.

Dependent variables' transformations.

Blood pressure for all groups exhibited low to mild skew and kurtosis, significant Lilliefors statistics and minor deviations from normality in expected probability plots. Therefore normality was assumed, transformations not attempted and raw data applied to statistical computation.

Heart rate per minute exhibited mild to moderate positive and negative skew and kurtosis. The four groups exhibited the following values: for smokers with schizophrenia skew = .77 and kurtosis = -.32, for non-smokers with schizophrenia skew = .57 and kurtosis = -.62, for non-psychiatric smokers skew = .248 and kurtosis = -.90, and for non-psychiatric non-smokers skew = -.08 and kurtosis = .42. Smoking schizophrenia and non-psychiatric people exhibited non significant normality as indicated by Lilliefors statistic, mild to moderate skew and expected normal probability

plots slightly deviated from normal. Non-smoking schizophrenia and non-psychiatric people exhibited significant normality and mild skew and similar expected normal probability plots to the two smoking groups. Because non significant Lulliefors statistics existed for the two smoking groups transformations were attempted to improve normality. Although logarithm and square root transformations improved skew slightly they made kurtosis worse and did not generate significant Lilliefors statistics for the two smoking groups. Nor did they improve visual indicators of normality for any group; expected if deviations are due to random error. Taken together these findings infer transformation of heart rate does not improve normality enough to warrant the increase in result complexity. Therefore this research applied raw heart rate data to analyses.

Cognitive errors made whilst completing the WCST exhibited low to mild positive and negative skew and kurtosis, significant Lilliefors statistic and visually comparable normality plots to blood pressure. Consequently, transformations were not considered necessary for this variable and raw cognitive errors made on the WCST were applied to statistical analyses.

Covariable transformations.

Although this research did not use covariables in the six main experimental hypotheses investigated, they play an important methodological role. Measuring the following covariables: age, cigarettes consumed per day, education, medication levels, and years unwell, ensured these particular variables did not influence this studies main findings.

Age exhibited normality with low to mild skew and mild to moderate kurtosis, significant Lilliefors statistic and satisfactory expected normal probability plots. Consequently, raw age data was applied to analyses.

Cigarettes consumed per day by smoking subjects only generated mild to moderate skew, significant Lilliefors statistics and satisfactory expected normal probability plots. Therefore, transforming cigarettes smoked per day is unbeneficial and raw data of cigarettes consumed per day was applied to analyses.

Education generated moderate skew and kurtosis. The four groups provided the following values: for smokers with schizophrenia skew = -.64 and kurtosis = -1.11, for non-smokers with schizophrenia skew = .09 and kurtosis = -1.2, for non-psychiatric non-smokers skew = -.85 and kurtosis = .82, and for non-psychiatric smokers skew = -.79 and kurtosis = .96. Although skew appears poor in comparison to other variables already considered, only P/S who smoke exhibit non significant normality as indicated by Lilliefors statistic. Further, all four groups exhibited expected normal probability plots with acceptable distribution. Transforming data using logarithm, square root and reflection transformations failed to improve significance level in smokers with schizophrenia, nor did transformations improve visual indicators of normality. Consequently raw education data was applied to statistical analyses.

Years unwell exhibited moderate to severe skew and kurtosis. People with schizophrenia exhibited the following data: for smokers with schizophrenia skew = 1.1 and kurtosis = .46 and for non-smokers with schizophrenia skew = .55 and kurtosis = -1.12. Although both groups generated significant Lilliefors statistics and acceptable expected normal probability plots, transformations were attempted in order to reduce the severe skew. Square root transformations drastically improved skew in

both groups, increased significance of Lilliefors statistics and improved expected normal probability plots. Transforming the variable 'years unwell' will not unduly complicate results because it is not considered in this study's main hypotheses. Therefore square root transformation of 'years unwell' was applied to statistical analyses.

Amount of medication prescribed exhibited mild skew and severe kurtosis for non-smokers with schizophrenia and low skew and moderate kurtosis for smokers with schizophrenia. Because of unacceptable normal probability plots and poor kurtosis square root transformations were used. After transformation, probability plots improved, skew and kurtosis improved (smokers with schizophrenia: skew -.10, kurtosis -1.3; non-smokes with schizophrenia: skew .01, kurtosis -1.52) and Lilliefors statistics were significant. Because the amount of medication prescribed is not considered in the main experimental hypotheses using transformed data will not unduly complicate results. Therefore, this research analyses square root transformations of 'medication prescribed'.

Discussion

Literature shows over 80 per cent of P/S smoke (Huges et al, 1986) and research suggests they smoke to alleviate boredom and provide a life pleasure (Golalaswamy & Morgan, 1986; Neergraad, 1997). Although P/S may smoke to give themselves something to do, they may have other reasons. With over 80 per cent of the schizophrenia population smoking it is arguable they actively smoke to gain other benefits. Research shows nicotine administered via tobacco smoking reduces anxiety in both schizophrenia (Glynn & Sussman, 1990) and non-psychiatric populations (Ague, 1973) and increases dopamine concentration (Levin and Rose, 1995). Reducing anxiety in non-psychiatric people (Hammermaster, 1989) and increasing dopamine in P/S (Daniel et al, 1991) correspond with improved cognitive performance. People with schizophrenia and non-psychiatric anxious people exhibit comparable debilitating anxiety and P/S exhibit dopamine deficiency. Therefore this research suggested smokers with schizophrenia actively smoke to reduce anxiety and increase dopamine levels, which relate to better cognitive performance. This study located no research investigating the immediate influence of tobacco smoking on schizophrenia cognitive functioning at the time of cigarette consumption. This research was an initial investigation into the belief that smoking associates with better cognitive performance in smokers, in comparison with non-smokers, with schizophrenia. We predicted smoking would be related to better schizophrenia WCST performance, either directly by increasing dopamine or indirectly by reducing anxiety, immediately after smokers consumed a single tobacco cigarette.

Smokers with schizophrenia performed better than non-smokers with schizophrenia when completing the WCST.

The first main result of this study allowed us to accept the first experimental hypothesis. Smokers with schizophrenia scored significantly fewer WCST errors (M = 27.72 errors, SD = 9.88) immediately after smoking one cigarette, compared to non-smokers with schizophrenia (M = 34.75 errors, SD = 7.52). Perhaps the most obvious explanation for this significant difference is that tobacco smoking was related to better cognitive functioning in P/S who smoke, either directly by increasing dopamine or indirectly by reducing anxiety.

The t-test computation between smokers and non-smokers with schizophrenia is analogous to a correlation analysis with a dichotomous nominal variable, smokers and non-smokers with schizophrenia (Coolican, 1994). Because the t-test computation is correlational a number of explanations are possible for the significant difference between smokers and non-smokers with schizophrenia when completing the WCST. We may, for example, have committed type one error; we may have computed a fluke correlation; an extraneous variable may have influenced outcome; smoking status or smoking a single cigarette may have been associated with the significant difference. Subsequently this research can not make a causal statement about the relationship between tobacco smoking, schizophrenia and cognitive performance. However, literature and clinical observations imply tobacco smoking rather than smoking status influenced schizophrenia cognitive performance. Further, the possibility this research committed type one error, a fluke correlation, or extraneous variables influenced outcome, can be excluded.

The possibility of committing type one error and falsely rejecting the null hypothesis is confidently rejected, based on post hoc power computed for the crucial *t*-test. The power of the *t*-test conducted between smokers and non-smokers with schizophrenia on the number of errors made when completing the WCST equalled .72. The power level is above .7 suggested by Boniface (1995) and subsequently the risk of committing type one error is acceptable.

The *t*-test computation in this study used a dichotomous nominal variable. Therefore, saying that there is a significant difference between cognitive performance and the two schizophrenia groups is the same as saying there is an association between smoking status and cognitive performance. Further because the *t* value can be transformed into a point biserial correlation (Coolican, 1994) the suggestion a fluke correlation was computed is rejected. The *t* value generated from the parametric computation lay inside the 95 per cent confidence intervals (-1.3 to 15.36). Consequently, the *t* value, and therefore the biserial correlation, was reliable and not a fluke (Evitt & Hay, 1992) at the 95 per cent confidence interval.

This study considered the influence of the following schizophrenia covariables on the outcome of the first main result: age, education history, medication prescribed and years unwell. Covariables were equivalent across smokers and non-smokers with schizophrenia, except gender, with more males than females with schizophrenia. Gender did not influence WCST performance, and because the other covariables were equivalent across schizophrenia groups, the variables measured would not have influenced statistical analysis. However, this study did not randomly assign subjects to groups, therefore other extraneous variables, not measured, may have influenced outcome.

Although a within subjects design was preferable it was not possible in this research and this study discussed explanations for the impossibility in the method. Consequently this study used an independent samples between groups design. It is impossible, therefore, to discern whether smoking status or smoking a single cigarette influenced cognitive performance. It appears the two most probable explanations for the significant difference between smokers and non-smokers when completing the WCST are either smoking status, or the possibility that smoking a single cigarette may be associated with cognitive functioning. Clinical observations and literature-based reviews suggest smoking a single cigarette, rather than smoking status, may be associated with better cognitive performance by smokers, in comparison to non-smokers, with schizophrenia.

Clinical observations by the primary researcher[6] prior to this study found smoking appeared to improve cognitive functioning in twenty smokers with schizophrenia, by indirectly reducing self-reported anxiety immediately after smoking a single cigarette. Every person with schizophrenia who smoked reported smoking helped them to concentrate, feel relaxed and 20 per cent ($N = 4$) reported that smoking reduced schizophrenia positive symptoms, for example, auditory hallucinations. Together the clinical observations corresponded with an objective increase in time spent reading and interacting with peers. The information gathered from clinical observations and discussions with smokers with schizophrenia implied nicotine administered via a tobacco cigarette corresponded with improved cognitive functioning and general well-being at the time of cigarette consumption.

Literature shows smoking reduces debilitating anxiety (Ague, 1973) and also that a reduction in debilitating anxiety improved cognitive functioning in non-

psychiatric people (Hammermaster, 1989; Hudesman et al, 1984; Metzger et al, 1990; Wesnes & Warburton, 1983). Literature reports P/S are generally anxious and find life more stressful than non-psychiatric people (Depue & Fowles, 1973; Kelher et al, 1975; Medick, 1958; Serban, 1975). Because both P/S and non-psychiatric anxious people exhibited cognitive worry, nervousness and the presence of task-irrelevant responses, it appeared that both populations experience debilitating anxiety (Bleuler, 1950; Eysenck & Folkland, 1980; Gaeddert & Dolphin, 1981; Wine, 1971). The facts that smoking reduces anxiety in non-psychiatric people, P/S and non-psychiatric people exhibit the same anxiety and over 80 per cent of P/S smoke support clinical observations made by the primary researcher that smokers with schizophrenia appear to smoke to reduce anxiety.

Literature also supports the argument that smoking may be associated with schizophrenia cognitive functioning by directly increasing dopamine levels at the time of consuming a single cigarette. Despite traditional theory arguing schizophrenia was caused by excessive dopamine, literature now links cognitive impairment in P/S with dopamine deficit (Daniel et al, 1991; Davis et al 1991; Dolan et al, 1995; Lewis & Akil, 1997), and argues schizophrenia has dopamine deficit in cortical areas vital to cognitive functioning (Grenhoff et al, 1986; Imperto et al, 1986; Levin, 1992). Research shows nicotine increases dopamine transmission in these cortical areas (Grenhoff et al, 1986; Imperato et al, 1986), over 80 per cent of P/S smoke and increases in dopamine correspond with improved schizophrenia WCST performance (Daniel et al, 1991).

In summary, although this research's design is correlational and between groups, clinical evidence and literature review suggest smoking rather than smoking status best

---

[6] Observations made whilst working as a nurse aide in the Hastings Psychiatric Hospital.

explains the significant difference between smokers and non-smokers with schizophrenia when completing the WCST. It remains uncertain, however, whether the better cognitive performance by smokers with schizophrenia on the WCST was associated directly to nicotine related increase in dopamine levels, or indirectly to nicotine related reductions in debilitating anxiety. The following section aims to determine whether smoking is directly or indirectly associated with WCST performance in smokers, in comparison to non-smokers, with schizophrenia.

If better cognitive performance by smokers with schizophrenia was directly related to increasing dopamine levels, rather than indirectly to reducing anxiety, then affect and arousal should have been equivalent from pre- to post-cigarette consumption. Results from this research showed no significant difference in smokers with schizophrenia pre- and post-cigarette for measures of subjective affect and physiological arousal. Further, literature shows when increasing dopamine in P/S there is a related improvement on the WCST (Daniel et al, 1991). Therefore the result provides initial evidence for the argument that smoking may have directly influenced cognitive performance in smokers with schizophrenia by increasing dopamine concentration. Further the result supports the argument that smoking rather than smoking status influenced cognitive performance in smokers with schizophrenia.

However, the finding that smoking did not influence affect or arousal is contradictory to literature-based evidence and therefore introduces doubt about the influence of nicotine on dopamine levels. That is, if nicotine failed to influence affect and physiology in P/S, when research clearly shows it does in non-psychiatric people, it is questionable how smoking is then able to influence dopamine concentration. Literature shows smoking a single cigarette increases heart rate and systolic blood

pressure (Nisbitt, 1973; Ray, Nellis, Brady, & Foltin, 1986; Soria et al, 1996), and smoking is subjectively relaxing for both smokers with schizophrenia (Glynn & Sussman, 1990) and non-psychiatric smokers (Ague, 1973). Therefore, this study expected smoking should have made smokers more subjectively relaxed, and increased heart rate and blood pressure in smokers with schizophrenia. Consequently, this study will consider other explanations for the non significant difference between measures of affect and arousal from pre- and post-cigarette consumption for smokers with schizophrenia, before concluding that smoking is associated with increased dopamine concentration.

Smokers with schizophrenia reported being already subjectively relaxed at pre-cigarette consumption with results showing them been significantly 'not at all, 'tense', 'upset' or 'worried'. Literature shows the frequency of smoking in non-psychiatric people increases as anxiety increases, and where stress causes the body to metabolise nicotine faster (Dobbs, Stickler & Maxwell 1981; Golding & Mangan, 1982; Pomerleau & Pomerleau, 1987; Schachter, Silverstein & Perlick, 1977; West & Lennox, 1992). The results from the previous literature imply that smoking only influences subjective affect when smokers are subjectively anxious. Therefore, smoking may not have influenced affect from pre- to post-cigarette because smokers with schizophrenia already experienced subjective relaxation. Further, smoking may not have influenced heart rate and blood pressure in smokers with schizophrenia because of the cumulative effect of tobacco smoking on nicotine blood plasma levels (Issac & Rand, 1972). This study did not record time between participants' last personal cigarette and their experimental cigarette. Therefore, it is possible participants' heart rate and blood pressure had already being influenced by previous smoking.

In summary, smokers with schizophrenia were already subjectively relaxed, consequently smoking did not influence subjective affect. Further, smoking failed to influence physiological arousal because previous cigarettes may have already influenced heart rate and blood pressure. Taken together, the previous two suggestions imply that smoking and better WCST performance are not indirectly related in smokers, in comparison with non-smokers, with schizophrenia in this study. Therefore this study initially concludes smoking and cognitive performance are directly related in smokers with schizophrenia due to associated increases in dopamine.

If smoking directly relates to cognitive performance in smokers with schizophrenia due to increased dopamine, then smokers and non-smokers with schizophrenia should have exhibited equivalent subjective affect and physiological arousal before completing the WCST. Although results showed smokers and non-smokers with schizophrenia exhibited equivalent subjective relaxation, smokers had significantly higher heart rate and 8.5 milligrams higher blood pressure. From this we can infer that smokers with schizophrenia exhibited facilitating-anxiety (Alpert & Haber, 1960), which improves cognitive functioning. Subsequently the finding that smokers exhibited greater facilitating-anxiety than non-smokers with schizophrenia places in doubt the initial conclusion that smoking relates directly to cognitive performance in smokers with schizophrenia due to associated increases in dopamine.

Facilitating-anxiety is characterised by improved cognitive performance, subjective relaxation and absence of cognitive worry or task-irrelevant responses (Alpert & Haber, 1960). Because smokers with schizophrenia exhibited subjective relaxation and higher physiological arousal, by definition they exhibited higher facilitating-anxiety than non-smokers with schizophrenia. If facilitating-anxiety relates directly with the

cognitive functioning of smokers with schizophrenia, then controlling for heart rate and blood pressure should dissolve the significant difference found between smokers and non-smokers with schizophrenia when completing the WCST. Results show controlling for heart rate and blood pressure did dissolve the significant difference. The dissolving of the significant difference infers facilitating-anxiety relates directly with cognitive performance in smokers, in comparison with non-smokers, with schizophrenia when completing the WCST.

In summary, the best explanation for smokers, relative to non-smokers, with schizophrenia, performing significantly better on the WCST is that facilitating-anxiety and smoking were directly associated with better cognitive performance. Smoking failed to influence affect or arousal inferring no indirect association between smoking and cognitive performance. It remains uncertain, however, whether the smoking variable or facilitating-anxiety accounted for most of the WCST variance between smokers and non-smokers with schizophrenia. Person's correlations computed for heart rate, blood pressure and the smoking variable showed the smoking variable accounted for 11.2 per cent, and physiological arousal or facilitating-anxiety accounted for 41.3 per cent of the significant WCST difference between smokers and non-smokers with schizophrenia. Considered together, the correlations infer facilitating-anxiety accounted for 30.1 per cent more variance than the smoking variable.

Conclusions from the first main finding; smokers with schizophrenia, immediately after consuming a single cigarette, performed better than non-smokers with schizophrenia when completing the WCST.

The first conclusion from the first main finding is that tobacco smoking was directly but not indirectly associated with better WCST performance of smokers, in comparison with non-smokers, with schizophrenia. The second conclusion from the first main finding is that facilitating-anxiety directly associated with better WCST performance of smokers, in comparison with non-smokers, with schizophrenia. The conclusion that tobacco smoking and facilitating-anxiety directly associate with better WCST performance by smokers, in comparison with non-smokers, with schizophrenia generates theoretical, practical and ethical implications. The next section summarises these implications and their importance in regard to this study and future research.

Theoretical implications of the first conclusion.

The first theoretical implication from the conclusion that tobacco smoking directly associates with better WCST performance in smokers with schizophrenia is that smokers may actively smoke to help themselves think. The inference made from the first conclusion supports the theory formulated in the introduction of this study that smokers with schizophrenia do not smoke merely to alleviate boredom. Rather, based on reviewed literature and clinical observations, this research argued that smokers with schizophrenia actively smoked to gain medicinal and psychological properties from nicotine. For example, this study argued smokers with schizophrenia smoked to obtain nicotine increased dopamine concentrations (Grenhof et al, 1986; Imperato et al, 1986; Levin, 1992) and reduce anxiety levels (Ague, 1973; Glynn & Sussman, 1990).

The second theoretical implication from the conclusion that tobacco smoking directly associates with the better cognitive performance in smokers with schizophrenia is that nicotine may be directly related to increased dopamine levels. Literature supports the implication smoking associates with increased dopamine levels. Nicotine increases dopamine (Grenhof et al, 1986; Imperato et al, 1986; Levin, 1992) and associates with better cognitive performance on the WCST in P/S (Daniel et al, 1991). The implication that smoking may have increased dopamine concentrations supports the theory introduced by this study that P/S exhibit dopamine deficiency, and may actively smoke to replenish deficient dopamine levels. The support for the theory P/S exhibit dopamine deficiency and smokers with schizophrenia actively smoke to replenish dopamine deficit implies that the traditional theory of schizophrenia inappropriately pathologised this population.

Traditional theory and research argue excessive dopamine causes schizophrenia (Kaplan et al, 1994; Lewis & Akil, 1996). Consequently, dopamine blocking or antagonising medication was historically used to treat schizophrenia. The implication smokers with schizophrenia actively smoke to replenish dopamine suggests traditional theory is questionable, and treatment based on the dopamine hypothesis would have exasperated the very symptoms considered diagnostic categories for schizophrenia. For example, administering dopamine antagonistic medication would have enhanced disorganised speech characterised by disordered thought content and process (Diagnostic and Statistical Manual of Mental Disorders, 1994). Such intervention would have generated symptomatic artefacts and cognitive deficit which past research has considered specific to schizophrenia, and together further pathologised people labelled with schizophrenia. Consequently, the implication that the traditional dopamine theory

of schizophrenia is questionable provides evidence towards normalising schizophrenia. This study argued P/S are more like, than unlike non-psychiatric people, and the implication that the theory of schizophrenia is invalid supports this study's position.

The last implication from the first conclusion that smoking is directly associated with better WCST performance in smokers, in comparison with non-smokers, with schizophrenia is P/S do not exhibit a specific cognitive deficit. The implication P/S do not exhibit a specific cognitive deficit provides initial validation of the argument introduced by this study that past research concluding P/S exhibit a specific cognitive deficit is invalid. For example, administering cognitive tests to P/S exhibiting dopamine deficit and who received dopamine antagonistic medications would have ensured P/S performed significantly worse than non-psychiatric people. The findings would have been due to dopamine deficiency rather than a specific cognitive deficit.

However, this study is unable to make firm conclusions because dopamine levels were not recorded from pre- to post-cigarette consumption. Consequently, it remains speculative that smokers with schizophrenia actively smoked to help themselves think by replenishing deficient dopamine levels. Further, this study recognises and acknowledges the complexity of the dopaminergic system. However, despite the speculation and complexity of dopamine transmission, this research has provided literature review and correlational inference supporting the argument people with schizophrenia may exhibit dopamine deficit and actively smoke to replenish dopamine. Together, the evidence provides initial support for the arguments introduced in the introduction of this study. Smokers with schizophrenia actively smoke, exhibit dopamine deficit and P/S do not exhibit a specific cognitive deficit. Finally, the traditional dopamine hypothesis of schizophrenia needs revision and the exploration of

the dopaminergic system and it's relationship with schizophrenia cognition must continue.

Ethical implications generated from the first conclusion.

Poor methodology discounts some studies showing P/S exhibit mortality rates lower than exhibited by the general population (Modrzewska & Book, 1979; Forest & Forest, 1974, cited in Tsuangi, Perkins & Simpson, 1983). For example, the previous studies generated mortality data using proportionate comparisons assuming the overall death rate in P/S to be the same as in the general population. The death rates between P/S and non-psychiatric people are not equivalent (Kaplan et al, 1994). Because these studies did not use absolute death rates the data is invalid. Research using absolute death rates found P/S exhibited mortality rates equal to or greater than non-psychiatric people (Ananth & Burnstein, 1977; Tsuang & Woolson, 1977). Smoking increases the risk of developing terminal illness and P/S have the highest smoking rate in western society (Kaplan et al, 1994; Hughes et al, 1986). Therefore, the conclusion that smoking is directly associated with WCST performance in smokers with schizophrenia, compared with non-smokers with schizophrenia, generates an important ethical consideration. Research should develop ways of reducing smoking rates in smokers with schizophrenia and develop safe alternative methods to obtain any dopamine related improvement in cognitive functioning, thereby reducing associated health risks. Perhaps one solution to reduce the health risks associated with cigarette smoking in smokers with schizophrenia, and to retain the nicotine related cognitive benefits, is to begin administering nicotine gum to them. Research shows nicotine gum improves cognitive functioning in non-psychiatric non-smokers (Provost & Woodward, 1991). Perhaps nicotinic gum or

patches would relate to increased dopamine level, and better cognitive functioning in P/S, providing a safer alternative to tobacco smoking?

The conclusion smoking may increase dopamine concentrations generates another ethical consideration. Administration of dopamine antagonistic medication to P/S, when schizophrenia is associated with dopamine deficit and dopamine deficit related to schizophrenia cognitive dysfunction, is unethical, especially considering non-dopamine antagonist medication is readily available. Reviewed literature shows dopamine deficit causes cognitive dysfunction (Grenhof et al, 1986; Imperato et al, 1986) and increases in dopamine correspond with improved cognitive functioning on the WCST in P/S (Daniel et al, 1991). Further, nicotine increases dopamine (Levin, 1992) and this study found a significant correlation between the smoking variable and cognitive performance on the WCST, immediately after smokers with schizophrenia smoked a single cigarette. Therefore, it is unethical to diagnose a person with schizophrenia based the presentation of disordered thought content and process and then administer medication which directly exacerbates those symptoms.

Further, if smokers with schizophrenia smoke to increase dopamine concentrations which, in turn correspond with better cognitive performance, then limiting access to cigarettes also becomes an ethical issue. Professional experience in psychiatric hospitals has shown that many smokers with schizophrenia, if left to their own devices, would chain smoke. Consequently psychiatric staff limit many smokers with schizophrenia to half hourly or hourly smoking regimes. It is questionable, ethically, to limit cigarettes for smokers with schizophrenia if they are self administering nicotine to increase dopamine levels and improve their cognitive functioning and general well-being.

Perhaps it is more ethically appropriate to administer nicotine gum to smokers with schizophrenia. Perhaps it is more ethically appropriate to limit dopamine antagonistic medications, or stop prescribing them all together, thereby increasing schizophrenia dopamine levels and reducing the biological need to smoke!

Future research to validate the first conclusion from the first main finding.

Future research needs to validate this study's conclusion that a nicotine related increase in dopamine associates with better WCST performance by smokers, in comparison to non-smokers, with schizophrenia. In order to validate the conclusion future research must record dopamine concentration at pre- and post-cigarette consumption and conduct within subjects experimental design. Recording dopamine level and using a within subjects design will allow causal statements about the influence of dopamine on schizophrenia cognitive functioning in relation to tobacco smoking. Because this study did not measure dopamine it is possible dopamine did not influence cognitive functioning. Further, this study did not conduct a within subjects design and subsequently it remains possible another variable, only present in smokers with schizophrenia, may have confounded the results. For example, it may be the act of smoking that improved cognitive performance, not a nicotine related increase in dopamine, which associated with better cognitive performance on the WCST.

Before developing successful smoking cessation programs research needs to determine whether the significant correlation between smoking and the cognitive performance of smokers with schizophrenia, associates with the act of smoking, an extraneous variable, or a nicotine related increase in dopamine. Future research could differentiate between the influence of the act of smoking and the influence of nicotine

by using methodology from non-psychiatric research investigating the act of smoking in comparison with the influence of nicotine. Non-psychiatric studies used unlit cigarettes (Nesbitt, 1973) and non tobacco smokes or 'shame cigarettes'(Pomerleau et al, 1984).

Theoretical, ethical and practical implications for conclusion two of the first main finding.

The conclusion that facilitating-anxiety directly related to better WCST performance in smokers with schizophrenia implies support for the validation of the argument P/S do not exhibit a specific cognitive deficit. The conclusion also implies that anxiety influences schizophrenia cognitive functioning and therefore supports this study's argument that past research has neglected the influence of anxiety on schizophrenia cognitive performance (Depue, 1974; Gjerde, 1983). Past research (Harris et al, 1990; Neale, 1971; Saccuzzo et al, 1974) appears to have created artefact support for the theory that P/S exhibit a specific cognitive deficit. The validation of the argument anxiety associates with schizophrenia cognition is important because it contradicts traditional belief, further normalises schizophrenia and allows the formulation of a new theory. The new theory is that P/S do not exhibit a specific cognitive deficit, rather anxiety influences their cognitive functioning.

The second theoretical implication from the conclusion that facilitating-anxiety directly associates with better cognitive performance is smokers with schizophrenia may actively smoke when exhibiting debilitating anxiety in order to reduce such affective states. Literature reviewed in this study logically supports the implication smokers with schizophrenia actively smoke to reduce debilitating anxiety. Tobacco smoking reduces debilitating anxiety in non-psychiatric people (Ague, 1973) and smokers with

schizophrenia (Glynn & Sussman, 1990). Debilitating anxiety reduces cognitive performance in non-psychiatric people (Hammermaster, 1989). The introduction showed P/S and non-psychiatric people exhibit debilitating anxiety. Further, clinical observations by the primary researcher found smokers with schizophrenia reported actively smoking to reduce symptoms of debilitating anxiety, which related to direct social benefits. Therefore, it remains plausible smokers with schizophrenia actively smoke to generate facilitating-anxiety or reduce debilitating anxiety.

In summary, the discussion has provided multiple inferential evidence that smokers with schizophrenia actively smoke to help themselves think, replenish dopamine deficit and possibly to reduce any debilitating anxiety present. Further, this study has also provided multiple evidence P/S do not exhibit a specific cognitive deficit, rather dopamine deficit and anxiety influences their cognitive functioning. The implication that anxiety influences schizophrenia cognitive functioning generates an important ethical consideration.

Thought, feeling and behaviour are not mutually exclusive, rather each influences the other (Beck, 1995). Therefore anxiety will, when present, influence schizophrenia cognitive functioning and behavioural presentation. Mental health providers having contact with P/S need to consider how anxiety influences schizophrenia thinking and behaviour, especially how debilitating anxiety negatively influences schizophrenia behaviour and thought. The failure to consider the detrimental influence of debilitating anxiety on schizophrenia cognitive functioning may lead to inappropriate and unethical treatment. For example, debilitating anxiety, if present, would confound schizophrenia cognitive performance on assessment scales such as the Mini Mental Status Examination. The MMSE assesses cognitive functioning and

general orientation to the environment, and is a standard instrument used in admission and assessment in psychiatric hospitals (Kaplan et al, 1994). Therefore, debilitating anxiety would confound assessment information gathered from the MMSE. Further, debilitating anxiety may also lead nursing staff in psychiatric hospitals to misinterpret ward schizophrenia behaviour. For example, a nurse may record that a person with schizophrenia exhibited intense agitation directly related to their 'psychotic state' and subsequently recommend their medication regime be increased and hospital admission extended. In reality, the person with schizophrenia may have been experiencing debilitating anxiety independent of any psychotic phase. Although both the scenarios described above are fictional, failure to consider the influence of debilitating anxiety could lead, unethically, to inappropriate medical regimes, nursing care and, ultimately, longer periods of inpatient hospitalisation.

This study's finding that facilitating-anxiety directly associated with better schizophrenia cognitive functioning on the WCST generates a practical implication. It implies that teaching practical ways of reducing debilitating, or increasing facilitating, anxiety should associate with better cognitive functioning in P/S. Learning such skills could improve quality of life, self esteem and provide vocational opportunity. Consequently P/S would become more empowered, less stigmatised and less a victim of their own circumstances and of the schizophrenia diagnosis. Future research should also investigate, longitudinally, the relationship between anxiety and general well-being or quality of life, vocation, self esteem and hospitalisation recidivism for schizophrenia. Research into these areas would validate this study's concern about the detrimental or negative impact debilitating anxiety has on schizophrenia general well-being and cognitive functioning.

Second main finding: Schizophrenia and non-psychiatric people performed equally when completing the WCST.

The second main result of this study allowed us to reject the third experimental hypothesis. People with schizophrenia (M = 29.9, SD = 9.9) did not significantly differ from non-psychiatric people (M = 24.6, SD = 11.2) on the number of WCST errors. Perhaps the most obvious explanation for the result is P/S and non-psychiatric people do not differ in cognitive ability when completing the WCST.

However, the explanation schizophrenia and non-psychiatric people do not differ in cognitive ability is inconsistent with past schizophrenia cognitive literature. Studies reported in the literature found P/S performed worse than non-psychiatric people on varied cognitive tasks (Allen, 1982; Harris et al, 1990; Succuzzo et al, 1974). Further, the non significant difference on WCST performance between schizophrenia and non-psychiatric people is contrary to clinical investigations finding P/S perform worse than non-psychiatric controls on the WCST (Heaton, 1981). This study, however, argues that the previous research ignored any influence anxiety had on schizophrenia cognitive functioning, and therefore did not provide an accurate indication of schizophrenia cognitive ability.

The finding schizophrenia and non-psychiatric people do not differ in cognitive performance on the WCST is consistent with literature where both populations exhibit equivalent anxiety. For example, Depue (1974) matched schizophrenia and non-psychiatric people for debilitating anxiety and found they performed equally on cognitive discrimination tasks. Depue's finding implies that schizophrenia and non-psychiatric people should perform equally on cognitive tasks, when their cognitive

functioning is measured in an environment standardised for debilitating and/or facilitating anxiety. Both schizophrenia and non-psychiatric people exhibited facilitating-anxiety immediately before completing the WCST.

Statistical analyses found schizophrenia and non-psychiatric people equivalently relaxed and both groups exhibiting blood pressure and heart rate above normal levels of 120 milligrams of blood pressure and 72 heart beats per minute. Therefore both populations appeared to exhibit comparable facilitating-anxiety. However, P/S exhibited significantly higher heart rate than non-psychiatric people and therefore significantly higher facilitating-anxiety immediately before completing the WCST. Despite exhibiting higher facilitating-anxiety, when controlling for blood pressure and heart rate the result remained non significant, implying that the difference in anxiety was insufficient to alter the second main finding. Therefore anxiety did not influence schizophrenia or non-psychiatric cognitive performance on the WCST. Consequently, the best explanation for the non significant difference between schizophrenia and non-psychiatric people on the WCST is that both populations exhibited equal cognitive ability not confounded by a differing type or level of anxiety.

Conclusion from the second main finding: People with schizophrenia and non-psychiatric people performed equally when completing the WCST.

The conclusion from the second main finding that schizophrenia and non-psychiatric people performed equally on the WCST is that both populations exhibited equal cognitive ability not confounded by differing anxiety levels. The conclusion generates important implications.

Implications of the conclusion from the second main finding.

The conclusion schizophrenia and non-psychiatric people performed equally on the WCST because both populations exhibited equal cognitive ability not confounded by differing anxiety levels implies additional validation for the argument P/S do not exhibit a specific cognitive deficit. Further, the implication provides additional support for the argument that past research concluding P/S exhibit a specific cognitive deficit was created by invalid representation of schizophrenia cognitive ability. One explanation for the invalid conclusions on schizophrenia cognitive functioning is past research ignored the influence of debilitating anxiety on schizophrenia cognitive functioning. Findings from this study support the argument past research neglected to consider the influence of anxiety on schizophrenia cognitive performance. For example, anxiety directly influenced cognitive performance on the WCST in smokers, in comparison to non-smokers, with schizophrenia. Secondly, both schizophrenia and non-psychiatric people exhibited facilitating-anxiety immediately before completing the WCST and performed equally. Further, literature also supports the notion that anxiety influences schizophrenia cognition. For example, Gjerde (1983) notes 'the performance (cognitive) of schizophrenics appears to be quite analogous to the performance of essentially normal but highly aroused subjects' (p. 68).

The implication P/S do not exhibit a specific cognitive deficit emphasises the need for future research to explore the impact anxiety has on schizophrenia cognition and subsequent care. This study has already discussed potential schizophrenia research at the tertiary care level. For example, future research could investigate the extent to which mental health professionals consider the detrimental influence anxiety has on schizophrenia presentation, assessment, subsequent treatment planning and length of

admission. Further, based on the findings in this research and reviewed literature future research must consider the influence of facilitating and debilitating anxiety on schizophrenia cognition in order to accurately determine schizophrenia cognitive functioning and determine accurate cognitive norms. Determining accurate normative samples for P/S is vital considering normative data bases existing for neuropsychological and psychological tests manuals and research may be confounded by anxiety. The aim of empirical research should be to create accurate norms for schizophrenia cognitive functioning which are not confounded by anxiety.

Smokers with schizophrenia did not gain the most benefit from tobacco smoking.

The third main result of this study allowed us to reject the sixth experimental hypothesis. The influence of one cigarette on cognitive errors made whilst completing the WCST did not depend on diagnosis. The best explanation for the non significant interaction effect is smokers with schizophrenia do not get the most medicinal or psychological benefit from tobacco smoking.

However, although the non significant interaction effect implies people with schizophrenia do not get the most benefit from tobacco smoking it does not mean they do not actively smoke. This research argued smokers with schizophrenia actively smoke to obtain medicinal and psychological properties, and a number of factors support this argument. First, this study demonstrated that while smoking and non-smoking non-psychiatric people did not differ on the WCST, smokers, in comparison to non-smokers, with schizophrenia performed significantly better. Second, the smoking variable accounted for 11.2 per cent of the variance between smokers and non-smokers with

schizophrenia when completing the WCST. Further, smokers with schizophrenia smoke significantly more cigarettes (39 %) per day than non-psychiatric smokers and literature shows smokers with schizophrenia have the highest smoking rate in western society (Hughes et al, 1989). Also, clinical observations imply smokers with schizophrenia obtain both medicinal and psychological benefit from smoking immediately after smoking a single cigarette. For example, smokers with schizophrenia reported a nicotine-related reduction in auditory hallucinations, and objectively they spent more time reading and interacting with peers after smoking. Fifth, literature shows P/S are generally anxious people (Depue & Fowles, 1973; Kelher et al, 1975) and exhibit dopamine deficiency (Daniel et al, 1991). Smoking reduces anxiety (Ague, 1973; Glynn & Sussman, 1990) and increases dopamine (Grenhoff et al, 1986; Imperato, et al, 1986; Levin, 1992). Increases in dopamine are important because they associate with improved cognitive functioning in P/S (Daniel et al, 1991). Finally, reductions in anxiety in non-psychiatric people correspond with improved cognitive functioning (Cohen, 1969, Donner & Guerney, 1969). Taken together the previous six factors imply that over 80 per cent of P/S actively smoke to gain medicinal and psychological properties from tobacco smoking.

Because smokers with schizophrenia actively smoke and experience equivalent smoking related mortality rates to non-psychiatric smokers (Ananth & Burnstein, 1977; Tsuang & Woolson, 1977) an ethical and moral implication becomes apparent. Research needs to investigate other ways of providing the medicinal and psychological benefits which smokers with schizophrenia attempt to access by smoking. Finding alternative methods to achieve the benefits provided by nicotine (such as reducing debilitating anxiety and increasing dopamine levels) will reduce smoking levels, and this reduce

smoking-related mortality rates. Before developing successful programs to reduce schizophrenia smoking rates, future research needs to replicate and validate this study's explanation for why over 80 per cent of P/S smoke.

Final conclusions.

The current research investigated WCST performance by smokers with schizophrenia, immediately after smoking a single tobacco cigarette, in comparison with non-smokers with schizophrenia. The research also compared schizophrenia WCST performance, irrespective of smoking status, with non-psychiatric people. This research made four main conclusions. First, tobacco smoking and facilitating-anxiety directly associated with better WCST performance in smokers with schizophrenia in comparison with non-smokers with schizophrenia. Second, because smokers with schizophrenia already exhibited subjective relaxation tobacco smoking did not influence subjective affect. Therefore tobacco smoking did not indirectly influence WCST performance in smokers with schizophrenia. Third, P/S do not exhibit a specific cognitive deficit and contrary conclusions reported from past schizophrenia cognitive research neglected the influence of anxiety on schizophrenia cognition. Fourth, although results showed smokers with schizophrenia did not gain the most from tobacco smoking, many factors support the argument that they actively smoke to obtain nicotine related medicinal and psychological benefits.

This study does not suggest P/S begin tobacco smoking. Despite the findings implying smokers with schizophrenia gain cognitive benefit from tobacco cigarettes. The important passion behind this research was the author's personal belief that P/S were more like, rather than unlike, non-psychiatric people. Consequently this study

sought to normalise schizophrenia by demonstrating that P/S do not exhibit a specific cognitive deficit, but their cognitive functioning is influenced by anxiety and low dopamine concentrations. The conclusion that facilitating-anxiety and tobacco smoking directly associate with schizophrenia cognitive performance immediately after cigarette consumption, appears original to this research. This study located no other research showing a relationship between facilitating-anxiety, per se, or tobacco smoking, and better schizophrenia cognitive performance for smokers, in comparison with non-smokers, with schizophrenia, immediately after smokers consumed a single cigarette. Therefore the findings from this research are exciting and satisfying because they are original, but most importantly because they further normalise schizophrenia.

References

Adler, L. E., Hoffer, L. J., Griffith, J., Waldo, M. C., & Freedman, R. (1992). Normalization by nicotine of deficient auditory sensory gating in relatives of schizophrenia. *Biological Psychiatry, 32*, 607-616.

Ahtee, L., & Kaakkola, S. (1978). Effect of mecamylamine on the fate of dopamine in striatal and mesolimbic areas of the rat brain: Interaction with morphine and haloperidol. *British Journal of Pharmacology, 62*, 213-218.

Ague, C. (1973). Nicotine and smoking: effects upon subjective changes in mood. *Psychopharmacologia, 30*, 323-328.

Allen, H. A. (1982). Dichotic monitoring and focused versus divided attention in schizophrenia. *British Journal of Clinical Psychology, 21*, 205-212.

Alpert, R., & Haber, R. N. (1960). Anxiety in academic achievement situations. *Journal of Abnormal and Social Psychology, 61*, 207-215.

Ananth, J., & Burnstein, M. (1977). Cancer: less common in psychiatric patients? *Psychosomatic, 18*, 444-446.

Bauman, E., & Murray, D. J. (1968). Recognition versus recall in schizophrenia. *Canadian Journal of Psychology, 22*, 18-25.

Beck, J. S. (1995). *Cognitive therapy: Basics and beyond*. New York: Guilford Press

Bellack, A. S., Blanchard, J. J., Murphy, P., & Podell, K. (1996). Generalization effects of training on the Wisconsin card sorting test for schizophrenic patients. *Schizophrenia Research, 19*, 189-194.

Bellack and Hersen (1988). *Dictionary of behavioural assessment techniques* (1st ed.). New York: Pegmon Press.

Benjamin, M., McKeachie, W. J., Lin, Y., & Holinger, D. P. (1981). Test anxiety: deficits in information processing. *Journal of Educational Psychology, 73,* 816-824.

Berger, H. J. C., van Hoof, J. J. M., van Spaendonck, K. P. M., Horstink, M. W. I., van den Bercken, J. H. L., Jaspers, R., & Cools, A. R. (1989). Haloperidol and cognitive shifting. *Neuropsychologia, 27,* 629-639.

Bergin, A. E., & Garfield, S. L. (1994). *Handbook of psychotherapy and behavior change.* New York: John Wiley and Sons.

Bernheimer, H., Birkmayer, W., Hornykiewicz, O., Jellinger, K., & Seitelberger, F. (1973). Brain dopamine and the syndromes of Parkinson and Huntington. *Journal of the Neurological Sciences, 20,* 415-455.

Bleuler, E. (1950). *Dementia praecox or the group of schizophrenias.* (J. Zinkin, Trans.). New York: International Universities Press Inc. (Original work published 1911).

Boniface, D. R. (1995). Experimental design and statistical methods for behavioural and social research. London: Chapman & Hall.

Braff, D. B., & Saccuzzo, D. P. (1981). Information processing dysfunction in paranoid schizophrenia: A two-factor deficit. *American Journal of Psychiatry, 138,* 1051-1056.

Broadbent, D. E. (1971). *Decision and stress.* London: Academic Press Inc.

Broen, W. E. (1968). *Schizophrenia, research and theory.* New York: Academic Press.

Calev, A., & Monk, A. F. (1982). Verbal memory tasks showing no deficit in schizophrenia: fact or artefact. *British Journal of Psychiatry, 141,* 528-530.

Cavlev, A., Venables, P, H., & Monk, A. F. (1983). Schizophrenic tendencies and memory deficits in normals. *Personality and Individual Difference, 4,* 89-94.

Caudrey, D. J., Kirk, K., Thomas, P. C., & Ng, K. O. (1980).Perceptual deficit in schizophrenia: A deficit in redundancy utilization, filtering or scanning? *British Journal of Psychiatry, 137,* 352-360.

Chaplin, J. P. (1984). *Dictionary of psychology* (10th ed.). New York: Laurel.

Chapman, L. J., & Chapman, J. P. (1973). Problems in measurement of cognitive deficit. *Psychological Bulletin, 79,* 380-385.

Coakes, S. J., & Steed, L. G. (1996). *SPSS for windows. Analysis without anguish.* New York: John Wiley & Sons.

Cohen, J. (1977). *Statistical power analysis for the behavioural sciences.* New York: Academic press.

Cohen, R. (1969). The effects of group interaction and progressive hierarchy presentation on desensitization of test anxiety. *Behaviour Research and Therapy, 7,* 15-26

Coolican, H. (1994). *Research methods and statistics in psychology* (2nd ed.). London: Hodder & Stoughton.

Dane, F. C. (1990). *Research methods.* California: Brooks/Cole.

Daniel, D. G., Weinberger, D. R., Jones, D. W., Zigun, J. R., Coppola, R., Handel, S., Bigelow, L., R., Goldberg, T. E., Berman, K. F., & Kleinman, J. E. (1991). The effect of amphetamine administration on regional cerebral blood flow during cognitive activation in schizophrenia. *Journal of Neuroscience, 11,* 1907-1917.

Data Sheet Compendium (1981-1982). London: Datapharm Publications Limited.

Davis, K. L., Kahn, R. S., Ko, G., & Davidson, M. (1991). Dopamine in schizophrenia: a review and reconceptualization. *American Journal of Psychiatry, 148*, 1474-1486.

Depue, R. A. (1974). The specificity of response interference to schizophrenia. *Journal of Abnormal Psychology, 83,* 529-532.

Depue, R. A., & Fowles, D. C. (1973). Electrodermal activity as an index of arousal in schizophrenia. *Psychological Bulletin, 79,* 233-238.

Diagnostic and Statistical Manual of Mental Disorders (4th ed.). (1994). Washington: American Psychiatric Association.

Dobbs, S. D., Strickler, D. P., & Maxwell, W. A. (1981). The effects of stress and relaxation in the presence of stress on urinary PH and smoking behaviours. *Addictive Behaviours, 6*, 345-353.

Dolan, R. J., Fletcher, P., Frith, C. D., Friston, K. J., Frackowiak, R. S. J., & Grasby, P. M. (1995). Dopaminergic modulation of impaired cognitive activation in the anterior cingulate cortex in schizophrenia. *Nature, 378*, 180-182.

Donner, L., & Guernery, B. G. (1969). Automated group desensitziation for test anxiety. *Behaviour Research and Therapy, 7,* 1-13.

Elsworth, J. D., Deutch. A. Y., Redmond, D. E., Sladek, J. R., & Roth, R. H. (1990). MPTP- induced parkinsonism: relative changes in dopamine concentration in subregions of substantia nigra, ventral tegmental area and retrorubral field of symptomatic and asymptomatic vervet monkeys. *Brain Research, 513,* 320-324.

Evitt, B. S., & Hay, D, F. (1992). *Talking about statistics. A psychologist's guide to data analysis.* London: Edward Arnold.

Eysenck, M. W., & Folkard, S. (1980). Personality, time of day and caffeine: Some theoretical and conceptual problems in Revelle, et, al. *Journal of Experimental Psychology, 109,* 32-41.

Freedman, R., Hall, M., Adler, L., & Leonard, S. (1995). Evidence in postmortem brain tissue for decreased numbers of hippocampal nicotinic receptors in schizophrenia. *Biological Psychiatry, 38,* 22-33.

Gaeddert. W. P., & Dolphin, W. D. (1981). Effects of facilitating and debilitating anxiety on performance and study effort in mastery based and traditional courses. *Psychological Reports, 48,* 827-833.

Gjerde, P. F. (1983). Attentional capacity dysfunction and arousal in schizophrenia. *Psychological Bulletin, 93,* 57-72.

Glynn, S. M., & Sussman, S. (1990). Why patients smoke. *Hospital and Community Psychiatry, 41,* 1027.

Goff, D. C., Henderson, D. C., & Amico, E. (1992). Cigarette smoking in schizophrenia: Relationship to psychopathology and medication side effects. *American Journal of Psychiatry, 149,* 1189-1194.

Golding, J., & Mangan, G. L. (1982). Arousing and de-arousing effects of cigarette smoking under conditions of stress and mild sensory isolation. *Psychophysiology, 19,* 449-456.

Gopalaswamy, K. A., & Morgan, R. (1985). Too many chronic mentally disabled patients are too fat. *Acta Psychiatrica Scandinavica, 72,* 254-258.

Grenhoff, J., Aston-Jones, G., & Svensson, T. H. (1986). Nicotine effects on the firing pattern of midbrain dopamine neurones. *Acta Physiologica Scandinavia, 128,* 151-158.

Hammermaster, C. S. (1989). Levels of performance and cognitive interference in test anxious subjects. *The Alberta Journal of Education Research, 35,* 164-170.

Harris, A. E., Benedict, R. H. B., & Leek, M. R. (1990). Consideration of pigeon holing and filtering s dysfunctional strategies in schizophrenia. *British Journal of Clinical Psychology, 29,* 23-35.

Heaton, R. K. (1981). *Wisconsin card sorting test manual.* Florida: Psychological Assessment Resources.

Hemsley, D. R., & Zawada, S. L. (1976). Filtering and the cognitive deficit in schizophrenia. *British Journal of Psychiatry, 128,* 456-461.

Holzman, P. S., Kringlen, E., Levy, D. L., & Proctor, L. R. (1978). Smooth eye pursuit movements in twins discordant for schizophrenia. *Journal of Psychiatric Research, 14,* 111-126.

Holzman, P. S., Levy, D. L., & Proctor, L. R. (1976). Smooth eye pursuit eye movements, attention and schizophrenia. *Archives of General Psychiatry, 33,* 1415-1420.

Holzman, P. S., Proctor, L. R., Levy, D. L., Yasillo, N. J., Meltzer, H. Y., & Hurt, S. W. (1974). Eye tracking dysfunction in schizophrenic patients and their relatives. *Archives of General Psychiatry, 31,* 143-151.

Howard, J. M. (1996). DHEA, melatonin, and schizophrenia. Located at, *http://www.shizophrenia.com/news/NEWS1.hhtml.*

Huberty, C. J., & Morris, J. D. (1989). Multiple analysis versus multiple univariate analysis. *Psychological Bulletin, 105,* 302-308.

Hudesman, J., Loveday, C., & Woods. N. (1984). Desensitization of test anxious urban community-college students and resulting changes in grade point average: a replication. *Journal of Clinical Psychology, 40 (1-3),* 65-67.

Hughes, J. R., Hatsukami, D. K., Mitchell, J. E., & Dahlgren, L. A. (1986). Prevalence of smoking among psychiatric patients. *American Journal of Psychiatry, 143,* 993-997.

Ikard, F. F., & Tompkins. S. (1973). The experience of affect as a determinant of smoking behavior. *Journal of Abnormal Psychology, 81,* 172-181.

Imerato, A., Mulas, A., & Chiara. D. (1986). Nicotine preferentially stimulates dopamine release in the limbic system of freely moving rates. *European Journal of Pharmacology, 132,* 337-338.

Issac, P. F., & Rand, M. J. (1992). Cigarette smoking and plasma levels of nicotine. *Nature, 236,* 308-310.

Jefferies, P. M. (1979). *Balliere's nurses dictionary* (19th ed.). London: Cassell Ltd.

Johnsen, S. M., & Sechrest, L. (1968). Comparison of desensitization and progressive relaxation in treating test anxiety. *Journal of Consulting and Clinical Psychology, 32,* 280-286.

Kaplan, H. I., Sadock, B. J., & Grebb, J. A. (1994). *Synopsis of psychiatry.* (7th ed.). Baltimore: Williams & Wilkins.

Kelher, R., Willson, R. M., Muldawer, M. D., & Pathak. D. (1975). Anxiety in schizophrenia. *Archives of General Psychiatry, 32,* 1246-1254.

Klein, C., & Andersen, B. (1991). On the influence of smoking on smooth pursuit eye movements of schizophrenics and normal controls. *Journal of Psychophysiology, 5,* 361-369.

Klein, P. (1994). *The handbook of psychological testing.* New York: Routledge.

Levin, E. D, (1992) Nicotine systems and the cognitive function. *Psychopharmacology, 108,* 417-431.

Levin, E. D., & Rose, J. E. (1995). Acute and chronic nicotinic interactions with dopamine systems and working memory performance. In L. G. Abood, & A Lajtha (Eds.), *Diversity of interacting receptors* (pp. 245-252). New York: Academy of Sciences.

Lewis, D. A., & Akil, M. (1997). Cortical dopamine in schizophrenia; Strategy for postmortem studies. *Journal of Psychiatric Research, 31*, 175-195.

Lipsey, M, W. (1990). *Design sensitivity statistical power for experimental research.* London: Sage Publications

Loftus, G. R., & Loftus, E. F. (1976). *Human memory, the processing of information.* New York: John Wiley and Sons.

MaManus, M. (1971). Group desensitisation of test anxiety. *Behavior Research and Therapy, 9,* 51-56.

Marteau, T. M., & Bekker, H. (1992). The development of a six-item short-form of the state scale of the spielberger state-trait anxiety inventory (STAI). *British Journal of Clinical Psychology, 31,* 301-306.

Massaro, D. W., & Loftus, G. R. (1996). Sensory and perceptual storage: data and theory. In E. C. Bjork & R. A. Bjork (Eds.), *Memory* (pp. 67-99). New York: Academic Press.

Masterson, E., & O'Shea, B. (1984). Smoking and Malignancy in Schizophrenia. *British Journal of Psychiatry, 145,* 429-432.

Meadow, A., Donlon, P. T., & Blacker, K. H. (1975). Effects of phenothiazines on anxiety and cognition in schizophrenia. *Diseases of the Nervous system, 36,* 203-208.

Mendick, S. A. (1958). A learning theory approach to research in schizophrenia. *Psychological Bulletin, 55,* 316-327.

Metz, J.T., Johnsen, M. D., Pliskia, W. H., & Luchins, D. J. (1994). Maintenance of training effects on the Wisconsin card sorting test by patients with schizophrenia or affective disorders. *American Journal of Psychiatry, 151,* 126-132.

Metzger, R. L., Miller, M. L., Cohen, M., Sofka, M., & Borkovec, T. D. (1990). Worry changes decision making: The effects of negative thoughts on cognitive processing. *Journal of Clinical Psychology, 46,* 78-88.

Minimum, E. W., King, B. M., & Bear, G. (1993). *Statistical reasoning in psychology and education* (3rd ed.). New York: John Wiley.

Mitchell. J. V. (Ed.). (1985). *The ninth mental measurements year book.* Nebraska: University of Nebraska Press.

Modrzewska, K., & Book, J. A. (1979). Schizophrenia and malignant neoplasms in a north Swedish population. *Lancet, 1,* 275-276.

Nachmani, G., & Cohen, B. D. (1969). Recall and recognition free learning on schizophrenics. *Journal of Abnormal Psychology, 74,* 511-516.

Neale, J. M. (1971). Perceptual span in schizophrenia. *Journal of Abnormal Psychology, 77,* 196-204.

Neergaard, L. (1997). Living with schizophrenia. *http://www.schizophrenia.com/news/News 1.html*

Neiss, R. (1988). Reconceptualizing arousal: psychobiological states in motor performance. *Psychological Bulletin, 103,* 345-366.

Nesbitt, P. D. (1973). Smoking, physiological arousal and emotional response. *Journal of Personality and Social Psychology, 25*, 137-144.

Nuechterlein, K. H., & Dawson, M. E. (1984). Information processing and attentional functioning in the developmental course of schizophrenic disorders. *Schizophrenia Bulletin, 10*, 160-203.

Pashler, H., & Carrier, M. (1996). Structure, process, and the flow of information. In E. L. Bjork & R. A. Bjork (Eds.), *Memory* (pp. 4-25). California: Academic Press.

Peeke, S. C., & Peeke, H. V. S. (1984). Attention, memory and cigarette smoking. *Psychopharmacology, 84*, 205-216.

Pomerleau, C. S., & Pomerleau, O. F. (1987). The effects of psychological stressor on cigarette smoking and subsequent behavioral and physiological responses. *Psychophysiology, 24,* 278-285.

Pomerleau, O. F., Turk, D. C., & Fertig, J. B. (1984). The effects of cigarette smoking on pain and anxiety, *Addictive Behaviors, 9,* 265-271.

Provost, S. C., & Woodward, R. (1991). Effects of nicotine gum on repeated administration of the Stroop test. *Psychopharmacology, 104*, 536-540.

Ray, R. L., Nellis, M. J., Brady, J. V., Foltin, R. W. (1986). Nicotine and caffeine effects on the task-elicited blood pressure response. *Addictive Behaviors, 11*, 31-36.

Russel, P. N., & Beckhuis, M. E. (1976). Organisation in memory a comparison of psychotics and normals. *Journal of Abnormal Psychology, 85,* 527-534.

Saccuzzo, D. P., Hirt, M., & Spencer, T. J. (1974). Backward masking as a measure of attention in schizophrenia. *Journal of Abnormal Psychology, 83,* 512-522.

Sandyk, R. (1993). Cigarettes smoking: Effects on cognitive functions and drug-induced parkinsonism in chronic schizophrenia. *International Journal of Neuroscience, 70,* 193-197.

Sarason, S. B., Mandler, G., & Craighill, P. G. (1952). The effect of differential instructions on anxiety and learning. *Journal of Abnormal Psychology, 47,* 561-565.

Sawaguchi, T., Matsumura, M., & Kubota, K. (1988). Dopamine enhances the neural activity in spatial short-term memory in the primate prefrontal cortex. *Neuroscience Research, 5,* 465-473.

Schachter, S., Silverstein, B., & Perlick, D. (1977). Psychological and pharmacological explanations of smoking under stress. *Journal of Experimental Psychology, 106,* 31-40.

Sengel, R. A., & Lavallo, W. R. (1983). Effects of cuing on immediate and recent memory in schizophrenics. *The Journal of Nervous Mental Disease, 171,* 426-430.

Serban, G. (1975). Stress in schizophrenics and normals. *British Journal of Psychiatry, 126,* 397-407.

Silverman, H. M. (Ed.). (1994). *The pill book* (6th ed.). New York: Bantam Books.

Soria, R., Stapleton, J. M., Gilson, S. F., Sampson-Cone, A., Henningfield, J. E., & London, E. D. (1996). Subjective and cardiovascular effects of intravenous nicotine in smokers and non-smokers. *Psychopharmacology, 128,* 221-226.

Stern, Y., & Langston, J. W. (1985). Intellectual changes in patients with MPTP-induced parkinsonism. *Neurology, 35,* 1506-1509.

Stern, Y., Tetrud, J. W., Martin, W. R. W., Kutner, S. J., & Langston, J. W. (1990). Cognitive change following MPTP exposure. *Neurology, 40,* 261-264.

Stip, E., & Lussier, I. (1996). The effect of risperidone on cognition in patients with schizophrenia. *Canadian Journal of Psychiatry, 41* (Suppl. 2), 35-40.

Stone, G. C., Callaway, E., Jones, R. T., & Gentry, T. (1969). Chlorpromazine slows decay of visual short term memory. *Psychonomic Science, 16,* 229-230.

Stratta, P., Manari, F., Mattei, P., Casacchia, M., & Alessandro, R. (1994). Information processing strategy to remedial Wisconsin card sorting test performance in schizophrenia; a pilot study. *American Journal of Psychiatry, 151,* 915-918.

Tabachnick, B. G., & Fidell, L. S. (1989). *Using multivariate statistics* (2nd ed.). New York: Harper Collins.

Tsuangi, M. T., Perkins, K., & Simpson, J. C. (1983). Physical diseases in schizophrenia and affective disorder. *Journal of Clinical Psychiatry, 44,* 42-46.

Tsuang, M. T., & Woolson, R. F. (1977). Mortality in patients with schizophrenia, mania, depression and surgical conditions. *British Journal of Psychiatry, 130,* 162-166.

Vollema, M. G., Geurtsen, G. T., & Van Aoorst, A. J. P. (1995). Durable improvements in wisconsin card sorting test in schizophrenia patients. *Schizophrenia Research, 16,* 209-215.

Van den Broek, M. D., Bradshaw, C. M., & Szabadi, E. (1993). Utility of the modified wisconsin card sorting test in neuropsychological assessment. *British Journal of Clinical Psychology, 32,* 333-343.

Weinfurt, K. P. (1995). Multivarite analysis. In L. Grimm & P. Yarnold (Eds.), *Reading and understanding multivariate statistics* (pp. 245-276). Washington DC: American Psychology Association.

Wesnes, K., & Warburton, D. M. (1983). Smoking, nicotine and human performance. *Pharmacological Therapy, 21*, 189-208.

Wesnes, K., & Warburton, D. M. (1978). Effects of nicotine smoking and tablets upon human attention. In R. E. Thornton (Ed.). *Smoking Behaviour: physiological and psychological influences* (pp. 131-147). Churchill Livingstone, Edinburgh.

West. R., & Lennox, S. (1992). Function of cigarette smoking in relation to examinations. *Psychopharmacology, 108,* 456-459.

Wickelgren, W. A. (1977). *Learning and Memory*. New Jersey. Prentice & Hall.

Williams, R., Ballie, P., Dickson, R. A., & Dalby, J. T. (1993). Cognitive and behavioural efficacy of clozapine in clinical trials. *Canadian Journal of Psychiatry, 38*, 522.

Wine, J. (1971). Test anxiety and the direction of attention. *Psychological Bulletin, 76,* 92-104.

Appendix A; Re-Valued Cases For All Groups.

Data re valued for groups, diagnosis and smoking status

1. P/S who smoke;
   - a. blood pressure - case 15, 100 to 107,
     - case 16, 123 to 114
     - case 20, 150 to 146,
     - case 25, 120 to 115,
   - b. Heart rate - case 12, 60 to 87,
     - case 14, 60 to 88,
     - case 15, 128 to 123,
   - c. smokes per day - case 10, 50 to 36,

2. P/S who do not smoke;
   - a. Heart rate -case 3, 64 to 77,
   - b. Cognition - case 7, 9 to 27,
   - c. Education - case 5, 22 to 15,
   - d. Blood pressure - case 9, 122 to 113,

3. Non smoking non psychiatric;
   - a. Blood pressure - case 42, deleted
   - b. Education - case 30, 19 to 18,
     - case 32, 19 to 18,
     - case 41, 9 to 10,
     - case 45, 9 to 10,
     - case 46, 19 to 18,
   - c. Heart rate - case 30, 64 to 71.
     - case 31, 60 to 72.
     - case 44, 52 to 73.
     - case 45 108 to 101.

Appendix B; Consent form

Consent form

Name of study: Does Smoking help Schizophrenics Think?"

I give my informed consent to participate in this study which investigates the effect of smoking on schizophrenic people to think. I consent to publication of study results so long as the information is anonymous and disguised so that no identification can be made. I further understand that although a record will be kept of my having participated in the experiment, all experimental data collected from my participation will be identified by number only.

I have read and understand the information sheet dated..... ..... for volunteers taking part in the study designed to investigate whether smoking helps schizophrenics to think. I have had the opportunity to discuss this study. I am satisfied with the answers I have been given.

I have been informed that there are no known expected discomforts or risks involved in my participation in this experiment. This judgement is based on a large body of research with people completing the same test as myself.

I have been informed that there are no disguised procedures in this experiment. All procedures can be taken at face value.

I understand that taking part in this study is voluntary (my choice) and that I may withdraw from the study at any time and this will in no way affect my future health care.

I have been informed that the researcher will gladly answer any questions regarding the procedures of this study when the experimental session is completed.

I understand that my participation in this study is confidential and that no material which could identify me will be used in any reports on this study.

I understand the compensation provisions for this study (form A).

I have had time to consider whether to take part.

I know who to contact if I have any questions about the study.

I wish to have a copy of the results, YES / NO.

I (full name) hereby consent to take part in this study.

This project has been approved by the Hawke's Bay and North Health Ethics Committee. This means that the Ethics Committee may check that this study is running smoothly, and has followed appropriate ethical procedures. Complete confidentiality is assured.

If you have any ethical concerns about the study, you may contact the Hawkes Bay Ethics Committee on 06 8440360 or the North Health Ethics Committee on 09 3574300 .

Date:

Signature:

Full names of Researchers: David. R. Galbraith.
Supervisor, Dr Arnold Chamove.

Contact Phone Number for Researchers: C/- Auckland University
0-9 373 7599

Appendix C; Information Sheet.

Information sheet, page one.

Does Smoking Help Schizophrenics to Think?

Researcher: David. R. Galbraith.
Phone number;

Research title, Does Smoking Help Schizophrenics to Think?

Principal Investigator or researcher

Name: David. R. Galbraith
Address; C/- Professional Psychology Unit.,
Human Sciences Building.,
Auckland University.,
Symonds Street.,
Auckland.

Phone; 09 373 7599

You are invited to take part in research interested in finding out whether smoking helps schizophrenics to think.

ABOUT THE STUDY

The aims of the study.

1. To see if smoking helps schizophrenics to think in comparison to non smoking schizophrenics and also in comparison to smoking and non smoking non-psychiatricpeople.

2. To see if smoking makes schizophrenics feel more relaxed.

3. To answer questions about why so many schizophrenics smoke.

How were people chosen to take part in this study?

1. People chosen for this study had to be either a smoking or non smoking schizophrenic person or a smoking or non smoking non-psychiatricperson.

2. You had to be over the age of 16 years old.

3. If you are a patient here at hospital your doctor had to agree to you taking part in the study.

How many other people will be in the study?

1. Hopefully 40 to 80 other people will also be in the study.

Where will the study be held?

1. The study will be held here at the Hastings Hospital.

What is the time frame for the study?

1. Your role in this study will require about 30 minutes of your time.

What will happen during the study?

1. I will explain all about the study and what is going to happen during the time taken to complete the test.

2. You need to sign a form, this form shows you know what will happen in the experiment and that you agree to take part in the study.

3. You and I will then sit down in a quiet room, a nurse will take your heart beat and your blood pressure. Then I will ask you 9 questions about how you are feeling, your age and how long you went to school.

4. Next I will show you a number of cards. You will need to sort the cards into which ever pile of cards you think is the right pile.

5. If you are a smoker, then just before completing the test you will smoke one cigarette.

6. Any information that you give me will be recorded under a coded number. This is your code during the study. No body will know who you are or who the number belongs to. The code will provide me a way knowing which group subjects belonged to. The number also allows me to correctly analyse age and gender, schooling history, medications, blood pressures and heart beats and test scores.

RISKS AND BENEFITS

1. There are no risks involved in this study, if at any time you feel uncomfortable with the test please tell me and we will stop the card sorting and you will not have to continue.

2. The only inconvenience of the study is that it requires some of your time.

3. The benefit of the study is that it is new research that may provide answers and generate more questions about how smoking influences schizophrenic thinking.

4. This research is not a part of any treatment that you may be currently undergoing.

5. The research will not cost you anything.

6. Because this is a clinical study, but does not involve medication, you need to be aware that if you suffer Physical injury as a result of your participation in this study you may be covered by Accident Rehabilitation and Compensation Insurance Corporation Coverage. However, it is important to remember that cover is not automatic and your entitlement to compensation is determined by a number of things. For example, whether you earn a wage or not. Further, it is important to remember that in most cases ARCIC provides only partial repayment of costs and there is no lump sum compensation payable under the current ARCIC rules. If you suffer only mental injury, there will be no ARCIC compensation available. You also need to be aware that if you have cover under the ARCIC legislation your right to sue the researcher (s) or anyone else involved in the study is extremely limited. If you have any concerns about the cover given by ARCIC and what you are entitled to, you should contact your nearest ARCIC branch office for further information before you consent to participate in this trial.

PARTICIPATION

1. Your participation in this study is entirely voluntary (it is your choice). You do not have take part in this study, and if you choose not to it will not effect any future care or treatment.

2. If you do agree to take part in the study you are free to withdraw from the study at any time, without having to give a reason and this will in no way effect your future health care.

GENERAL

1. What will happen at the end of the study?

At the end of the study we will spend a few minutes talking about how you found the test and how you are feeling after finishing the test. I will ensure that you are feeling Ok about how the test went for you. If you wish, a time will be arranged after all testing is complete for everybody to meet. At this meeting I will answer any questions anybody has specific to the test.

2. Where can you get more information about the study?

If you require more information about the study, you can get in contact with myself and we can arrange a time to answer your questions. If I am unable to give you that information required I will do my best to find somebody who can.

3. If you need an interpreter, can one be provided?

An interpreter will be made available if possible.

4. Do you have to answer every question?

You do not have to answer any questions you do not understand or feel do not apply to the study.

5. If you have any queries or concerns about your rights as a participant in this study you may wish to contact a Health and Disabilities Services Advocate, telephone;

CONFIDENTIALITY

No material which could personally identify you will be used in any reports on this study.

All information regarding this study will be stored in a locked filing cabinet in the main office until the study is completed. At this time all information will be transferred to

Auckland, where I am at university, and be stored in a coded file on a personal computer.

No individual will be identifiable from the data stored on these files as all results are allocated to a number not a name.

RESULTS.

If you would like a copy of results then I will arrange for a copy to be posted to you.

STATEMENT OF APPROVAL

This study has received ethical approval from the Manawatu-Whanganui Ethics Committee.

Please feel free to contact the researcher if you have any questions about this study.

Phone David. R. Galbraith

C/- Auckland University.

(09): 373 7599

Appendix D; Demographic Score Sheet.

Please answer all of the following questions.

Section A:

female/male (please circle)

1. How old are you?.....
2. How many years did you go to school?.....
3. How old were you when you first experienced your illness?.....
4. Have you ever completed the Wisconsin Card Sorting Test before?.....
   (If yes, please state how long ago you completed this test)
5. How many cigarettes do you smoke per day?

Appendix E; Self Evaluation Questionnaire.

The following is the Shortened version of the State Trait Anxiety Inventory , called the Self Evaluation Questionnaire (Y-6 item), designed by Marteau and Bekker (1992). Reproduced from the *British Journal of Clinical Psychology* .

Name.....date.....

A number of statements which people have used to describe themselves are given below. Read each statement and then circle the most appropriate number to the right of the statement to indicate how you fell right now, at this moment. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

	Not at all	Somewhat	Moderately	Very much
1. I feel calm	1	2	3	4
2. I am tense	1	2	3	4
3. I feel upset	1	2	3	4
4. I am relaxed	1	2	3	4
5. I feel content	1	2	3	4
6. I am worried	1	2	3	4

Please make sure that you have answered all the questions

Appendix F; Correlation Matrix of Dependent and Covariables used in this Study.

The following correlation matrix was calculated using a two tailed Pearson's product moment correlation, applying results from 66 participants to computation.

	AGE	B/P	COG	EDU	GEN	H/R	SMKS	Y/ILL
AGE		.072 P= .57	.183 P= .14	.053 P= .67	-.20 P= .14	-.123 P= .32	.09 P= .56	.763 P= .01
B/P			.063 P= .64	.102 P= .42	-.08 P= .51	-.139 P= .26	.106 P= .53	-.112 P= .59
COG				-.277 P= .02	-.207 P=.10	-.035 P= .78	.075 P= .65	.253 P= .21
EDU					.10 P= .43	-.225 P= .07	-.421 P= .01	.109 P= .59
GEN						-.127 P= .31	-.356 P= .03	-.586 P= .01
H/R							.115 P= .49	.031 P= .88
SMKS								.373 P= .127
Y/ILL								