School of Psychology

The Causal Predominance of Psychotic Experience

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This thesis is presented for the degree of
Doctor of Philosophy
of
Curtin University of Technology
Western Australia.

November 2003

Declaration

This thesis contains no material which has been accepted for the award of any other degree or diploma in any university.

To the best of my knowledge and belief this thesis contains no material previously published by a person except where due acknowledgment has been made

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ACKNOWLEDGEMENTS

I would like to acknowledge my family particularly my parents, Brian and Mary Preston. They always encouraged their children to be inquisitive about themselves and the world around them. This ethos has been instilled in me and as such this thesis is a culmination of their support and love.

I would also like to thank my twin brother Terry Preston for his support throughout the research, and Rose Tassoni for reading the first drafts of the manuscript. Thanks also to Professor David Hay for his support and encouragement throughout the thesis.

I would also wish to acknowledge Professor Patrick McGorry and Dr Susan Harrigan for allowing me to have access to their early episode psychosis data. Their faith in my research interests is much appreciated.

Appreciation is also extended to Professors Peter Bentler (Psychology Department University of California Los Angeles) and Andrew Mackinnon (Mental Health Research Institute University of Melbourne) for taking the time to examine the thesis. Their expertise in offering valuable suggestions for improving the science of the research has greatly enhanced the quality of the thesis.

Finally, I would like to thank with utmost sincerity Dr Peter Sevastos my Supervisor and Mentor. Our long conversations in psychology, science, theology and music have given me a greater appreciation for the dedication he has to being a very giving person who is always concerned with the development of his students. His professional support and friendship is deeply cherished.

ABSTRACT

The present study investigated the causal predominance of cognition on anxiety, depression, paranoia, phobia and somatic concern over three time waves of self reported data measured every six months over one year, of 145 cases experiencing their first episodes of psychosis. In turn the symptoms of anxiety, depression, paranoia, phobia and somatic concern were examined for their cross-influential effects on cognition. Cognition was examined under a causal predominance hypothesis as the lead symptom because of its influence recognised in the literature under the neurodevelopmental hypothesis. These longitudinal effects were examined using structural equation modelling. Prior to this investigation, the research was able to demonstrate a stable 6-factor measurement model with these symptoms between two independent samples of early psychosis cases that met guidelines of treatment under the Australian national early psychosis treatment guidelines. This measurement model demonstrated good internal reliability and construct validity. Most symptoms over each time wave had a "domino effect" where the symptom prior to the next wave of assessment had an influence. This is known as a mediation effect. Somatic concern and depression demonstrated a "snow ball" or direct effect where the extent of the condition at time one influenced directly the condition at time three. Structural models, which examined the cross-influential effect between cognition and the other symptoms, demonstrated an effect between paranoia and cognition. This effect demonstrated that paranoia at Time 2 (i.e., 6 months after stabilisation of symptoms), had a cross-influential effect on cognition at Time 3 (ie, 12 months after stabilisation of symptoms). It was argued that poor thinking styles that lead to distortion in feelings of mistrust evident in the paranoia symptom, in turn led to deterioration in cognition. Other symptoms did not demonstrate a cross influential effect. Previous research suggesting that symptoms act independently of each other over time supports the results of independence of the other symptoms. Further research was suggested by linking different levels of psychosis research of the aetiological factors (e.g. genetic factors), neuropathology (e.g., reduced synapse density) and phenomenology (e.g., positive and negative symptoms) into an integrative framework. It was suggested that structural equation modelling as exemplified in the thesis could be used as a technique to examine how these differing levels could be investigated under a unified theory of psychosis based upon the neurodevelopmental hypothesis.

CHAPTER I

INTRODUCTION

Psychosis is defined by the Oxford English Dictionary as "severe mental illness, derangement, or disorder involving a loss of contact with reality" (Brown, 1993, p. 2402). More specifically, psychosis indicates " ...a severe mental disorder characterised by gross impairment in reality testing, typically shown by delusions, hallucinations, disorganised speech or disorganised ...behaviour" (Edgerton, 1994, p. 175). As a result of conflicting usage however, " ...there is no single acceptable definition of what psychosis is" (Campbell, 1996, p. 585). It is because of this usage that it is difficult to understand what exactly psychosis is and what causes it to arise. It is possible that this is largely due to the different historical notions of psychosis throughout recorded history. This means that psychosis as a construct in general and schizophrenia in particular, suffer from socio-historical influences which influence the understanding of psychosis to the present day. It is with this in mind that the literature on the subject of psychosis must be examined within a philosophical and socio-historical context. The thesis will often refer to the term *psychotic experience.* This refers to the way in which psychosis is understood in the socio-historical and philosophical milieu that it is placed within. It is to connote that the psychotic experience is not indeed fixed, but changes to fit the worldviews of the time. In reference to the body of work in this thesis, psychotic experience refers to the self-reported and observational measurement of signs and symptoms placed within a scientific and medical paradigm. Since the research is performed on cases experiencing their first episodes of psychosis, psychotic experience will refer to those

individuals who have a range of psychotic diagnoses although the majority of cases in the study have been given a diagnosis of schizophrenia.

The following review of the literature in psychosis will be divided into two chapters. Chapter I will explore the philosophical and socio-historical context of psychosis. This is done in order to provide the reader with a contextual framework by which the Western notions of psychosis as a construct have arisen from the current philosophical views held in the wider society. Chapter II will focus more specifically on the scientific development of psychosis and in particular schizophrenia. This chapter will trace the development of Kraepelian notions of dementia praecox in the early 19th century to the multi factorial models of recent times. It concludes by providing a description of the model of psychosis used in the empirical investigations in the thesis.

Before exploring the history of psychotic experience, some attention is required to explain the structure of the thesis. The thesis is divided into three major sections. The first section Chapters I and II, consists of the literature review. Each chapter will provide the framework by which the notions of psychotic experience have been developed in both a socio-historical and empirical context. The first chapter is to simply provide an exploration into the notions of psychosis from the pre-Hellenic periods to the end of the 18th Century. The purpose is to give the reader a background understanding of the major philosophical tenants of each period and how this has influenced the understanding and treatment of insanity both then and today. The second chapter explores the recent scientific and empirical construct of psychosis. This chapter will form the basis of the measurement model used in the second section of the thesis. Section II of the thesis concerns the empirical and statistical

investigation of the data used. Section II consists of Chapters III to VI. Chapter III explains the methodology used in the research to investigate the causal predominance of psychotic experience amongst cases experiencing their first episodes of psychosis. Chapter IV proposes the measurement model used to investigate the model of psychosis. Chapter V investigates the structural model that investigates the causal linkages between the symptoms proposed in Chapter IV over a three time-wave period. Chapter VI examines whether the causal linkages investigated in Chapter V are stable over time for correct causal influences to be made. Each of the chapters in Section II act as separate research papers in their own right. This means that they are structured to have an introduction, methods, results and conclusions. Each chapter attempts to logically flow from one to the next in a series of investigations. The final section of the thesis consists of Chapter VI which attempts to interpret the meaning of the results presented in Section II and addresses the limitations of the study. Suggestions for further research to advance the understanding of causal mechanisms in psychosis are also presented. By following this format of the three sections, it is intended that the reader will find the thesis a logical progression into the investigations purported within.

The history of Western notions of psychosis

The notions of psychotic experience has a rich and detailed history and is inextricably linked to the dominant philosophical paradigms of the time. It is almost impossible to separate the notions of psychotic experience from the worldview in which they are interpreted. Since psychosis is a sociological phenomenon as much as anything else, it is important to describe the link with the philosophical tenets of the

time and how psychotic phenomena are understood. This Chapter embeds the psychotic experience within its socio-historical and philosophical context focussing particularly on the Western notions of psychosis. Since current or modern understandings of psychosis are largely influenced by Western philosophical thought, it is important to understand the development of Western philosophy and its influence on the theory of psychosis, since it difficult to divide the two.

History of psychosis: ancient demonology

As societies developed out of the rise of improved agricultural techniques, man understood the world to be governed by both natural and supernatural forces. Good and bad harvests as much as good and bad health was explained in terms of an interaction between natural and supernatural forces. Behaviour of those outside the expected norms of society were often explained in supernatural terms, particularly those who were said to be possessed by spirits. These possessions were explained as influences external to a person, coming from without rather than from within. Aberrant behaviour was explained in terms of individuals being in discordance with the community, the environment, or with the gods (Birzer, 1993). Even with such aberrations, man has attempted to alleviate others who they believe to be inflicted with such phenomena. Archaeological evidence has shown that stone age cultures performed rather crude surgical operations called *trephining*, where a hole was cut into the head to allow a passage by which evil spirits could pass. It is quite possible that such individuals were suffering from some kind of psychotic illness since most psychotic phenomena are explained in a physical sense within the range of the head

and brain. It may have been reasoned at the time, that placing a hole in the head would allow for physical entities such as evil spirits to escape.

In the civilisations of Egypt and the Near East, mental illness was attributed to " ...the magical forces of malevolent deities, and the main therapists were priests who used religious and magical rites to counter those forces" (Colp, 2000, p. 3301). These evil deities were personified later in Christian cultures as demons, however the notion of evil demons prevailed in many of the first societies. As cultures became more sophisticated in their understanding of the world, credence in demons would fluctuate in complex ways. With increasing cultural sophistication came more elaborate ways of treating psychotic experience. Other treatments were used in such times that included ritual exorcisms. These ranged from mild activities such as prayer, chanting and making and drinking of various potions, to more extreme forms including submerging people in water, whipping and forced starvation in order to make the body less comfortable for evil spirits to stay (Birzer, 1993).

The emphasis in this explanation of mental illness is that the sufferer was indeed inhabited by evil or demonic spirits. Such spirits were seen to have both physical and metaphysical qualities. The alleviation of such afflictions were sought through encouraging these entities to leave the possessed body by both physical and spiritual means (Birzer, 1993).

The Greeks and the rise of rationalism

During the fifth century B.C., great Western philosophers such as Hippocrates, Socrates and Plato brought forward the concept of "discovery of the mind". This discovery was spearheaded by the use of rationality and logic to explain phenomena.

Such a technology asserted that through rational and logical thought processes, man could understand the truth and the nature of things. Within Greek culture, treating mental illness was the domain of religious cults and schools of philosophy. Most schools of philosophy at the time viewed madness as either psychological or somatic in origin or a mixture of both psyche and soma, and these three causes are still influential in explaining mental illness to the present day (Colp, 2000). Socrates, the father of Greek philosophy, began with a discussion of ethical terms and the search for absolute definitions. Socrates was concerned not only with what makes man a moral agent, which is often a metaphysical notion, but also how the world can be unified and made intelligible through universals which exist in the sensible world. For the first time in Western thought, a framework emerged that linked the philosophers task of encompassing both moral and scientific dimensions (Tarnas, 1998). This was achieved largely by making a causal connection between the existence of the physical world and its impact on the world of ideas.

Plato's "Ideas" are important to the emergence of psychosis in that the idea of psychosis could have only arrived if there was the discovery that universal Ideas survive beyond the particular. It is as if Plato allowed Western thought to entertain these notions and entities of reality, untarnished by temporality and spatial dimensions. The observation of psychotic phenomenon in the Greek worldview was allowed, since such an entity could exist beyond its particular observation. Such a radical notion also allowed psychotic phenomena to now be explained in both metaphysical and physical terms, due to Socrate's assertion that making sense of the sensual world is fundamental to understanding its impact on the world of ideas.

Plato divided the soul into three parts, rational, appetite (lusts and greed), and spirited affective. Plato argued that madness occurred when the appetitive soul lost the influence of the rational soul or when divine disturbance of the soul produced either inspired or destructive behaviour (Colp, 2000). Plato's assertion of both inspired and destructive behaviour allowed for madness to have both a "good madness" (Screech, 1985, p. 25) and a discordant madness. This allowed for some experiences of the human condition to be prophetic, inspirational and functional within the society, while others to be discordant both for the individual and the community. For these reasons Plato advocated a verbal dialectic between the " ...patient and a philosopher or a physician, a question-and-answer dialogue that enabled the patient to alleviate an illness by developing a state of philosophical knowledge" (Colp, 2000, p.3301).

While Plato advocated for a metaphysical explanation of madness, Plato's contemporary Aristotle believed mental illness occurred when the soul was subjected to changes in temperature, black bile and the emotions. This theory brought in the notions of physical mechanisms such as black bile interacting with the meta-physical world to cause disruptions within individuals. Aristotle was the first Western thinker to describe the affections of desire anger, fear and courage (Colp, 2000).

As the fifth century B.C. advanced, the shift from metaphysical explanations of the world to a physical explanation of the world, gained acceptance. The bold scientific speculations of Anaxaoras and Democritus all extended the Hellenic worldview that the world can be comprehended through naturalistic causes. By 410 B.C., Hippocrates laid the foundations of ancient medicine by proving to his fellow countrymen that mental disturbance (among other physical phenomena) was not a

supernatural phenomenon. He achieved this by classifying the disturbances he observed in strict definitional terms. Consequently, in his writings there are descriptions of mental disorders such as phobia and epilepsy. In most respects, Hippocrates was the first empirical clinician adopting Platonic notions of Ideas, along with the synthesis of the sensual world with the metaphysical (moral) world of Socrates. The world in relation to mental and physical illness, could be understood by sensual observation and not through divine rapture or spiritual insight. Through cold inhuman observation of the world, Hippocrates could act within the moral edicts of Socratic philosophy by his advocacy of treatment for mental illness. Like Aristotle, Hippocrates propounded that the human body contained four essential humours, - phlegm yellow bile, black bile and blood. He argued that these humours were secreted by different organs of the body and possess different qualities that vary with the seasons. The brain was considered the seat of life and normal human functioning required balancing the humours. Too much of one humour over another, would produce a certain state or mood. For example large excesses of phlegm cause a form of dementia, and black bile cause melancholia. Small excess of the humours would contribute to enduring temperaments and personalities such as the phlegmatic, choleric and sanguine personalities (Colp, 2000). Treatment consisted of balancing the humours; for example Hippocrates treatment of melancholia involved rest, exercise, a bland diet and abstinence from sex and alcohol (Birzer, 1993).

The Greek worldview through the introduction of logic as a technology of thinking, asserted that phenomena can be explained by both the purity of Platonic ideas and the empirical observation of the sensual world. As the Hellenic period developed, the empirical worldview developed further by allowing the introduction of mathematics (particularly geometry) to enter into scientific technology and

observations of the physical world. As such, all phenomena including psychotic phenomena were increasingly being explained in physical terms. Such a shift in emphasis led to less drastic treatments as those conducted in the ancient worlds such a trephining, with a greater focus on physical ameliorations, rather than shamanic rituals and religiose cures.

Decline of the Greek mind and the rise of the Judeo Christian worldview

By the end of the classical Greek period, the ideas of Western thought became increasingly under the influence of Roman hegemony. As the Greek cultural influence headed west to the Mediterranean, and south east to central Asia, the later classical Greek sensibilities were exposed to enormous multiplicity of viewpoints (Russel, 1961). No longer could the Greek worldview hold to its dominance in explaining the phenomenal world. The shift moved from Athens as the seat of cultural inheritance to the Byzantine world dominated then by the emergence of the Christian world. From the Byzantine world, the emergence of astrology into popular discourse of the time led explanations of the world away from the Greek physical empirical explanations, to one of celestial other worldly bodies. From this position developed an emergent schism between rational philosophies of the Hellenistic tradition and the mystery religions of the east. How this schism developed and the ultimate triumph of spiritual and religious explanations of the world would profoundly effect the explanations of psychotic experience for the next 1000 years. Bridging this period were the neoplatonists. In philosophers like Plotinus, the search for truth began by placing an emphasis on "the flight from the body" by placing Plato's Forms within the divine mind (Tarnas, 1998, p. 84). With this came an

increasing concern with evil and its relation to matter. Increasingly the understanding of psychotic experience moved away from rational philosophy, and gave way to the religious spirit and "suprarational mysticism" (Tarnas, 1998 p. 84). The neo-Platonists attempted to bridge this schism by placing Platonic Forms and Ideas within a spiritual context away from the empirical physical world. Matter no longer could be inferred from cold observation but was infused with spirit. With reference to the physical world, understanding matter was often relegated to a lower form of knowledge often infused with darkness and evil. The quest for truth leaned towards divinity and celestial understandings of the world and hence empiricism was relegated as a poorer cousin to divine inspiration. By the second century B.C. and the conquest of the Greek world by Rome, the Hellenic worldview was fading and was " ...displaced by the more Oriental view of human subordination to the overwhelming powers of the supernatural" (Tarnas, 1998, p. 87).

Prior to the dominance of the Judeo-Christian worldview on the Western world, a great deal of Roman views on madness were largely informed by classical Greek thinking. The Stoic and Epicurian notions of simplified moral living informed Roman thinking (not the current popular view of Epicurianism being one of sensual indulgence) (De Botton, 2000). For example mental illness was believed to arise when the passions and unsatisfied desires act on the soul to produce division in an uncontrollable way. Treatment was delivered by trying to control thoughts and conduct to achieve a mental state of *ataraxia* or calm self-composure. Such a treatment goal is still influential today where some modern tranquillisers have been classified as ataractics (Colp, 2000).

Two hundred years prior to the birth of Christ saw the rise of Galen, one of the greatest of Roman physicians. He argued that the soul could be divided into three parts, reason and intellect, courage and anger, and carnal appetites and desire. Galen was able to synthesise the nature and dispositional factors together to assert a picture of mental illness. He believed that diseases " ...are caused by adverse external influences (e.g., bad diet and bad air) acting on an existing predisposition and that psychological disorders cause physical disorders and vice versa' (Colp, 2000, p. 3302). Treatment under Galen's terms was to counterbalance what was unnatural within the disease such as cooling a fever or warming someone with a cold.

The treatment of the insane in Greco-Roman cultures was largely the responsibility of the family and not state institutions. The only exceptions to these were Roman soldiers who were admitted to military hospitals. Their families kept most of the seriously insane under restraint at home. Those who could not be "contained" by their families were often feared and hated, beaten, imprisoned or driven away from communities (Colp, 2000). Only those who were deemed as divinely inspired could be effectively welcomed into the community.

The stamp of the Judeo-Christian worldview on the Western world came during the early fourth century, with the Roman Emperor Constantine who had converted to Christianity and committed his imperial power on the propagation of the Christian faith throughout Europe. By the end of the fourth century Christianity became the official state religion of the Roman Empire (Tarnas, 1998). By this time, the then known world was once again described in spiritual terms where the luminous and numinous aspects of everyday life held precedence over the mere physical realities of everyday existence. With this re-focus to a world of metaphysical

qualities, came a resurgence of the divine and evil entering within the everyday behaviour of man. The experience of insanity did not escape this new paradigm of thinking, where aberrant behaviours were explained either as divine revelation or possession of demonic/evil spirits. Often this determination was made on the possessor's standing within the community and indeed their gender.

The profound influence of Christian thought on explanations of psychosis can be largely sourced to the revelatory aspect in Christian canonical law that God became flesh. This meant that the spiritual and divine had now through the birth, death and resurrection of Christ became human or *corpus christus* the body of Jesus. This meant that resurrection was possible for those who believed in the essence of the Christian faith and eternal damnation for those who did not believe. Psychotic experience in this context was not just explained solely as a demonic entity, external to that of the individual, but in the early Christian context it also allowed for divine revelation to be included in this explanation. While the Greek view of psychosis and such phenomena allowed for a more pluralistic explanation, the emergence of the Judeo-Christian worldview, narrowed the focus of psychotic phenomena to a monolithic and indeed a monotheistic origin based on spiritual morality (Tarnas, 1998).

The medieval era and the transformation of psychosis

The medieval era was a time of great turmoil. In the midst of this chaos, insanity and in particular psychosis started to develop a more sinister form and understanding. By this time the barbarian migrations from the West, and Islamic

expansion from the East, had destroyed a number of systems of civil authority, inherited from the early Hellenic days of republicanism (Tarnas, 1998). By this stage and in uncertain times, the Christian faith had absolute primacy over secular concerns. Such primacy discouraged any extensive involvement in classical thought and culture, and hence the Hellenistic empirical worldview of psychosis had largely dropped from public consciousness altogether. In place was a strict doctrinally and canonically led view of reality. Demands on keeping spiritually clean for the next world meant that temporal interest in nature, science, history and literature were forsaken. In this environment, the empirical understanding of psychotic experience was lost since temporal matters of the here and now was of little importance. This resulted in the flourishing of a prejudiced and discriminating view of psychosis. The spiritualised fervour of the times led to an explanation of psychotic experience to be largely demonic in nature. As such, mental illness did not derive from within the physical, sensual and temporal world, but instead came from without, from the metaphysical, spiritual and transcendental world. Explanations of possessions flourished greatly during this time and cures for such afflictions were met with religious rituals and treatments. In mild cases of possession, subjects were treated to rituals including praying, the sprinkling of holy water and pilgrimages to holy shrines. However as the Christian church became more prescriptive in its dealings with the faithful, those who demonstrated strong aberrant behaviour were subjected to forced starvation, being submerged in water, chained or flogged (Birzer, 1993). During this time some often-observed psychiatric reactions included symptoms characteristic of being possessed by the devil that was supposed to choke and throw victims about. Another was termed dance mania, which was characterised as an irresistible urge to dance and make noises. The third reaction is a form of spiritual depression known as *acedia*

which occurs when an individual develops distressful doubts in their ability to lead a religiously meaningful life (Colp, 2000).

Most notable at this time was the rise of evil possession of women namely those accused of witchcraft. Injury inflicted by supernatural devices was stimulated by the publication of *Malleus maleficarum* (*Witches' Hammer*) by Henry Kramer and James Sprenger in 1486. These two theologians had been designated by the Pope to act as papal inquisitors into witchcraft. In a Christian worldview dominated by a patriarchal system, it was believed that females who demonstrated aberrant behaviour, whether its genesis was a mental illness or not, had accusations of witchcraft levelled at them by the powerful and all pervasive clergy. *Malleus* described witches as mainly women who showed psychotic or hysterical symptoms and sexual delusions. It prescribed inquisitorial tortures that would force confessions of guilt from those who were accused. Such institutional madness led to the execution of many thousands of people, (mostly women) that went unabated for well over 150 years (Colp, 2000).

St. Thomas Aquinas and the return of human reason

With the dominance of Christian thought firmly held in the West, the intellectual rigidity that characterised the medieval period started to abate. With the doctrinal dominance of the Christian Church over pagan worship, the attitude towards secular learning had relaxed (Russel, 1961). At this nexus came the re-vitalisation of scholastic thought, spearheaded by the genius of St. Thomas Aquinas, who was able to make a link between the revelatory power of the Christian gospels on one hand and

the natural world and human reason on the other (Tarnas, 1998) This important synthesis led to the re-emergence of the classical Greek thought back into dominant Western thought. Ironically, the only institutions that kept any resemblance of classical Greek thought were the cloistered monasteries that flourished in the medieval period. It is from these texts and inspired by the revelatory nature and indeed experience of the gospels that St. Thomas Aquinas sought a new synthesis between the two dialectics. Aquinas argued that the Word of God was not a remote truth removed from the everyday experience of human life, but was in fact directly related to the immediate and temporal particulars of human experience. This was a great shift in Christiandom and hence for the explanation of psychosis, as it paved the way for man to return to a more empirical worldview of the "truth", and therefore revitalised the intellectual pursuit towards empirical knowledge and observation. This new synthesis questioned the notions of superstitious knowledge as a foundation to determine insanity and for sufferers to undergo ritualistic cures. Consideration of the earthly presentation of insanity was now open for interpretation from early somatic traditions largely espoused within the Hellenic period.

The emergence of the modern worldview of psychosis: critical scholasticism and Ockam's Razor

Throughout the later part of the medieval period and into the 14th century, the Western world flourished with scholastic endeavour. The Scholastic's exhaustive critical dialogue emerged with brave conjectures with daring alternative hypotheses, thereby challenging the authority of the Church. Such an environment led to a new

spirit in intellectualism with an era pregnant with scepticism and open to fundamental change. Such a period led to severing between the empirical worldview and the mystical divine revelatory experience espoused by Aquinas. This was led mainly by Ockham, a British philosopher who ironically was a priest well versed in the sensibilities of the Church. The notion of "Ockham's Razor" was that only concrete experience could serve as a basis for knowledge, and explanations of reality should not go beyond what is necessary to sever the ties between empiricism and mystical revelation (Tarnas, 1998). Such a tenet had a profound impact on Western thought and a schism once again emerged in Western culture, this time between empirical science and the Church's canonical teachings. This opened the way for psychotic experience to be studied in an empirical sense and allowed for the phenomenon to be grounded and explained in physiological terms (Tarnas, 1998). It followed that for this development to take shape, some time had to elapse before the modern explanations of psychosis could emerge. Within this time in history, a battle ensued on the most appropriate explanation of psychotic phenomena, that is between one of physiological empirical determinism on the one hand, and divine or evil spiritual possession on the other.

The Renaissance

Although most people may regard the Renaissance period as the return of religious iconography and the ascent of artistic man, the Renaissance period could more accurately be described as a sheer diversity of expression. For example, the Renaissance saw in a single generation the development of the genius of Di Vinci,

Michelangelo and Raphael, as well Columbus discovering the New World. Luther also rebelled against the Catholic Church and Copernicus discovered that the earth revolved around the sun. It was also the time when the printing press was invented enabling the Scientific Revolution and the Reformation both dependent on universal communication to take root substantially in Western society. Without these developments, the modern view of psychosis would not have emerged.

During this period of Western endeavour, human life was reaffirmed to hold an immediate temporal value that eventually displaced the medieval forces of other worldly spiritual destiny. Phenomena that were difficult to explain like psychotic experiences were continually returning to more temporal physiological explanations. Both the influences of the celestial world assumed under the Byzantine world, and the somatic world proposed by Hellenic philosophers came together under the Swiss physician Paracelsus. Medieval philosophy, astrology, magic and myths influenced his medical ideas. Rejecting the prevailing humoral theories of insanity, Paracelsus postulated that man consisted of both divine spirit and base animal instincts (lust, covetousness and the passions of the soul). Paracelsus believed that it was the stars stimulating the primal instincts and overwhelming the spiritual world that caused insanity. As a form of diagnosis the physician was required to identify the driving instinct that was dominating the persons' character and the star that corresponded to it. Treatments included psychotherapy, trephining and the administration of sulfur. Sulfur was administered because of its sedating effects to promote the healing powers of sympathy. Paracelsus was deemed one of the first "psychosomaticists" because he believed that emotions caused physical illness (Colp, 2000).

During the Renaissance period, another Swiss physician, Felix Plater, developed the classification of known diseases at the time in a book called *Observations of Diseases Injurious to Body and Mind* (1614). In this work, Plater accurately described mental handicap to have different phenomenological manifestations, where some individuals can be quite disturbed (as being psychotic), or others quite brilliant in one skill and handicapped in others (as in the modern notion of the savant). Plater advanced the process of separating psychological knowledge from philosophy and theology (Colp, 2000). He argued that phenomena should be observed in its own rights and in its own terms, similar to notions of Ocham's Razor.

The discovery of syphilis by Girolamo Fracastoro during the 15th Century brought together the notions of social morality and physical illness causing mental disease. The discovery that advanced syphilis led to a form of dementia prompted physicians to conclude that contracting disease through sexual activity could cause mental illness. The concept of sexual contagion was new and issues of morality entered into the debate towards the contraction and consequence of mental illness. Despite this great achievement of understanding the relationship between physical conditions and mental illness, the Renaissance period also suffered from the black plague where explanations of such an infliction were attributed once again to moral and spiritual behaviour.

The Birth of the Scientific Revolution

The Scientific revolution could also be seen as the Copernican revolution. Although Copernicus lived during the height of the Renaissance, it was his discovery

that the earth revolved around the sun that contributed to man's elevation as the centre of the universe, to come crashing down which then brought on the era of unbridled scientific enquiry. It was Copernicus's belief that nature was ultimately comprehensible in simple and harmonious mathematical terms, that pressed and guided him to such a radical confirmation of the earth's relationship to the sun. With a re-emergence of the humanist perspective from the Renaissance, the Christian church was under fire to explain its paradigm of reality. This division to this day effects how phenomena, particularly those once within the remit of theological and doctrinal discussion such as psychotic experience, were now open for question and indeed inquiry by scientific minds.

The main feature of the Scientific Revolution lay within its philosophical tenet that the physical world could be understood in geometrical and arithmetical terms. Copernicus, Keppler and Galileo all used such neo-Platonic techniques by prescribing and objectifying the world into reductionist parts. This proved to be a very useful technology that led to many advances from science. Such a technique would push religion off the centre stage in explaining psychotic experience and paved the way for seemingly impassioned observation and quantification of psychotic phenomena. The scientific paradigm was now open to be applied not only to corporeal aspects of the world but also to those experiences that exist primarily in the psyche.

Descartes famous datum *Cogito, ergo sum* – "I think, therefore I am" allowed for the final division between the mind, the body and spiritual / metaphysical explanations of the world. Although some authors argue that Descartes' division between the mind and the body has been somewhat overstated (Brown, 1985). Despite this, such a "philosophic thunderbolt" allowed for the removal of

metaphysical qualities into the explanation of mental illness and paved the way towards impassioned observation of psychotic phenomena free of metaphysical posturings (Russel, 1961; Tarnas, 1998).

Romanticism and the Enlightenment Period

The Romantic and Enlightenment sentiment could be said to operate a complex interplay in constituting the modern sensibility. Indeed it is during this period that both Sigmund Freud and Oscar Wilde were able to propose their understandings of the human condition. Both the Romantics such as Goth and Nietzsche and the Enlightenment proponents of Darwin and Freud, argued the humanist perspective on reality with high estimates of man's powers (such as Nietzche's "Beyond Good and Evil") and concern with man's place within the universe (such as Darwin's theory of evolution). While the Enlightenment-scientific mind had a view of nature as an object of impassioned observation, experiment and theoretical explanation and technological manipulation, the Romantics emphasised in contrast nature as " ...a live vessel of spirit, a translucent source of mystery and revelation" (Tarnas, 1998, p.367). Caught in the middle of this was the notion of insanity where it could be equally seen as a physical ailment or active participation in moral decrepitude. By the eighteenth century, persons with all manner of mental affliction were housed in large buildings called asylums. Notably the English hospital of St. Mary of Bethlehem had patients laying howling in chains while the curious public bought tickets to watch them perform as a form of "public entertainment" (Scull, 1993, p. 51). The hospital's name became truncated to be known as Bedlam

which is now synonymous with any madness displayed by a mob of people (Birzer, 1993). Such conditions were often an indication of the battle between moral fortitude espoused by the Romantics and the impassioned scientific investigation of the Enlightenment period scientists.

Around the same time in France an edict by the French monarch created a new hospital administrative organisation for Paris that consisted of asylums for the insane. The administrators held considerable power since they were allowed to lock people up for considerable periods of time without recourse or challenge With inaccurate nosologies, a whole range of persons deemed undesirable by society were also incarcerated in asylums. These included orphans, prostitutes, homosexuals, aged persons and those chronically physically ill. The emphasis of treatment or retention moved away from the responsibility of the family to the state.

By the later part of the eighteenth century it became apparent that locking people up in appalling conditions did not in any way aid in their recovery, and the inhumanity of their treatment was questioned. Reforms particularly in France led by Phillip Pinel, saw the unchaining of patients (Hirschmüller, 1999). Therapies such as bleeding and cupping (blistering the skin with small hot cups) were done away with. For the first time in many centuries the return of a scientific explanation of mental illness and resulting therapies began to dominate (Birzer, 1993). Pinel was influenced and somewhat indebted to the French Enlightenment, and as such developed a rational method of observation of psychiatric diseases. During this time the asylum became the principal method of treatment (Hirschmüller, 1999).

In Germany, at the same time psychiatric phenomena during the Romantic period " ...was characterised by a fundamental debate on the nature of mental

disease" (Hirschmüller, 1999, p. 399). This debate developed a cleavage with German psychiatrists with the "Psychiker" on one hand, arguing mental disease as a " ...disease of the incorporeal soul's or as God's punishment for sins, or caused by intemperate passions" (Hirschmüller, 1999, p. 399). On the other hand the so-called "Somatiker" group argued that mental illness was caused by general bodily afflictions namely the brain and the nervous system.

It was only in the last 30 years of the nineteenth century that psychiatry as a subject of serious academic research and teaching was in fact established. Concordant with this was the movement of psychiatric research away from asylums and into universities (Hirschmüller, 1999).

By the early nineteenth century, treatments thought to relieve blood pressure on the brain were used to reduce the effects of mental illness. To relieve the pressure in the blood vessels, Benjamin Rush, a famous American Psychiatrist, believed in terrifying the patient by strapping them into a device called the 'tranquilliser" or by dropping them in ice-cold baths. These procedures were not devised as tortures but were a genuine attempt to relieve the suffering of patients. Along with these methods, doctors were encouraged to bring little presents to their patients, and employ well educated staff who would read to the patients and engage in conversations (Birzer, 1993).

On both sides of the Atlantic, scientific psychology emerged. Rebelling from Kantian notions that inner experience of ones self provides an adequate means of psychological perception, the nineteenth century founders of modern psychology adopted mathematics and experiments to be suitable instruments for psychological research (Hirschmüller, 1999). William Wundt started the first institute of

experimental psychology in Leipzig in 1879. Shortly after this, Ernst Kraepelin founder of one of the classical models of schizophrenia (Jablensky, 1999), worked at Wundt's institute and has been largely influential in German clinical psychiatry.

By the start of the 20th century the battle between the metaphysical and physical notions of psychosis had shifted towards a more impassioned deterministic, reductionistic and physiological explanation. The physical worldview through its hardened observation of facts and rigorous testing of hypotheses with mathematical technologies, lead to the dominance of physiological explanations of psychotic experience. This worldview of psychosis was not in isolation to the zeitgeist of the times, since other areas of human endeavour were confident in the discoveries forged by the scientific revolution. By the early part of the 20th century, Adolf Meyer in an article in the *American Journal of Insanity* fundamentally argued that neither a purely somatic nor a psychological approach would suffice by itself but

> ...that all mental activity must have its physiological side and its anatomical substratum ... combinations of nerve cells ...A disease of these cells ...means at the same time a physiological and psychological disorder; destruction of these cells, a destruction of physiological function (Colp, 2000, p. 3307).

Mid way through the 20th century the bio-psychological model of mental illness had taken the imagination of researchers. The introduction of chemical compounds such as chlorpromazine to reduce the effect of psychotic symptoms and lithium to better manage mood disorders became new areas of investigation. Treatment up to the present day, has largely been sought by examining the complex interplay between the bio-chemistry of the brain and signs and symptoms expressed

by distressed individuals with psychotic experience. This interplay requires researcher to define what is psychopathological in an empirical sense and to link it with biochemical markers or processes.

Summary

The history of psychotic experience has largely been shaped by the dominant philosophical underpinnings of the time. Throughout time, these views have moved from purely somatic or physiological explanations to numinous and metaphysical explanations. Usually when great schisms occur in current cultural thinking some synthesis of thought arises which shapes a new way of understanding psychosis both as phenomenological experience and as a cultural construct. Psychosis or aberrant behaviour has often been used to explain qualities that are consistent with spiritual forces which are either diabolical in nature or divinely inspired. Each interpretation appears to have some role to play in how psychosis is understood and how it is treated. For example, externalised forms that shape or govern the destiny of individuals are often interpreted as demonic possession, while internal forces devoid of metaphysical qualities, are described as unfortunate biological predispositions which interplay in a complex manner within the individual alone. Either position argues over the current paradigms of understanding the world not only within an epistemological sense, but also within the ontology of treatment and legislature.

The following chapter will explore the current formulations of the construct of psychosis and how these formulations will be used to generate the model by which the current study is conducted.

CHAPTER II

THE CONSTRUCT OF PSYCHOSIS

In this chapter the construct of psychosis and in particular schizophrenia will be explored. Formulations of theory from the beginning of the last century to the latest developments in confirmatory factor analysis will be highlighted. The purpose is to give the reader a background on the historical and current models of psychosis as a syndrome. In this context a syndrome describes " ...groups of symptoms that tend to coexist" (Peralta, Cuesta, & de Leon, 1994, p. 727). These coexisting symptoms have been postulated both from clinical observation and empirical studies using exploratory and confirmatory factor analysis. As such, the purpose of the review is to assist the study in building a model of psychosis consistent with the literature as well as particular to the early psychosis population of interest. Since nearly all models within the literature are concerned with schizophrenia, the focus on this chapter will be on models of schizophrenia and not other types of psychosis. The approach for the following chapter is to explore the different models of schizophrenia within a developmental time line in order to provide a picture of an emergent construct surrounding psychosis.

Kraepelin and the development of Dementia Praecox

It is true to say that "schizophrenia is one of the major diagnostic categories to emerge from nineteenth century psychiatric practice and thought" (Barrett, 1998a, p. 617). It is difficult however to explicate the common worldview of the early 19th century that mental illness among many things was a degenerative development

(Barrett, 1998a). Moral decrepitude as much as physiological problems were often seen as an equal contributor to the course of mental illness. The development of dementia praecox within a mental illness framework was very much seen as an end point. Kraepelin's theories of psychosis were largely influenced by Morel's *Clinical Studies* of 1852. By 1860 Morel introduced the notion of démence précoce. This notion did not refer to a disease entity but as a particular course or evolution of mental illness. The psychiatric century of the 1800s culminated with Kraepelin's 1899 text where dementia praecox ("premature dementia") was first elevated to the status of a major disease. Some argue that this was not done in a systematic empirical effort but largely as a process of a lineage of ideas (Barrett, 1998a). This is exemplified by Barrett, (1998a) in the following table tracing Kraepelin's formulation of dementia praecox as an illness.

Table 2.1
Excerpts from Kraepelin's Successive Classifications [1]

Date	Category		Subcategory
1883	States of psychological weaknesses	a.	Idiocy, cretinism, feeblemindedness, homosexuality
		b.	Moral insanity, litigious insanity
		c.	Neuraesthenic states, obsessions, phobias, impulsions
		d.	Senile dementia
		e.	Secondary weakness states
1887	Acquired states of weakness	a.	Senile dementia
		b.	Mental weakness resulting from organic brain diseases
		c.	Secondary states of mental weakness
1891	Psychic degeneracy processes	a.	Dementia praecox
		b.	Catatonia
		c.	Dementia paranoides
1896	Acquired mental diseases metabolic diseases	a.	Myoedema
		b.	Cretinism
		c.	Dementifying processes
		i.	Dementia Praecox
		ii.	Catatonia
		iii.	Dementia paranoides
1899	Dementia Praecox	a.	Hebrephrenia
		b.	Catatonic form
		c.	Paranoid form
1909-1915	Endogenous conditions with evolution towards deterioration dementia praecox	1.	Hebephrenia
		2.	Depressive form
		3.	Catatonia
		4.	Paranoid form
		5.	Schizophasic form

Kraepelin describes dementia praecox in such a way that we would understand it in contemporary nosology as consisting fundamentally of negative symptoms (Andreasen, 1997). Kraepelin describes dementia praecox to consist of abnormalities in cognition and emotion, avolition, anhedonia, affective blunting and problems with attention. These symptoms are descriptive of the nosological structure of negative symptoms. In Kraepelin's description of the disease it was the loss of cognitive, affective, volitional and attentional function of the sufferer that were cardinal to the classification of dementia praecox (Andreasen, 1997). The more florid descriptions of schizophrenia, namely symptoms which describe hallucinations and delusions (now known as positive symptoms and which will be discussed in detail later) came much

[1] *Note.* Table from Conceptual foundations of schizophrenia: I Degeneration by R.J. Barrett, 1998,

later in the 20th Century. These descriptions dominated the theories of psychosis and schizophrenia as cardinal to the description of the illness.

In Kraepelin's own words with regards to observing psychotic phenomenon he states:

> The result of this highly morbid process is emotional dullness, failure of mental activities, loss of mastery over volition, or endeavour, and ability for independent action ...The near connection between thinking and feeling, between deliberation and emotional activity on the one hand, and practical work on the other is more or less lost. Emotions do not correspond to ideas (Kraepelin, 1919, p. 74).

Although Kraepelin did describe a broad range of symptoms including hallucinations and delusions which continue to be correctly classified within current understandings of schizophrenic illness (Jablensky, 1997; Jablensky, 1999), it is the deficit of the above mentioned symptoms that characterises Kraepelin's understanding of the illness (Andreasen, 1997). It is understood by some reviewers that Kraepelin's early formulation of dementia praecox expresses a very pessimistic view of the course and outcome of the disease (Barrett, 1998a; Barrett, 1998b; Riecher-Rössler & Rössler, 1998). Indeed this pessimism may reflect the overwhelming state of large asylums and university clinics of the time that were overburdened with chronic protracted cases of mental illness. Some theorists argue that Kraepelin, under the terms of dementia praecox lumped several clinical pictures into the one unitary condition (Peralta et al., 1994). This paved the way for later theorists such as Bleuler

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Australian and New Zealand Journal of Psychiatry, 32, p. 622.

to postulate whether schizophrenia is a heterogenous group of disorders (Bleuler, 1950).

Bleuler and the development of Schizophrenia

By the mid 20th Century Kraepelin's nosological description of psychosis through his dementia praecox theory was becoming increasingly reified. Even when Kraepelin himself tried to creatively reapraise the field of study in psychoses, the fundamental notions of his theories became entrenched and institutionalised in psychiatric research (Jablensky, 1997). By the end of his career in the 1920s, Kraepelin was prepared to question the very foundations of his own work in psychosis, suggesting areas of personality development rather than particular pathological processes as possible processes that unfold to reveal psychoses (Jablensky, 1997). Despite this apparent retraction, Kraepelinian notions of psychosis persisted as a diagnostic concept of schizophrenia within psychiatric research. By the mid part of the 20th century some clinicians and theorists felt Kraepelin's nosological structure to be too narrow in its definition. While Kraepelin was more concerned with the deficit syndromic areas of psychoses, Eugen Bleuler (1950) on the other hand tried to classify the groups of schizophrenias by considering underlying fundamental abnormalities. He did this by dividing the symptoms of schizophrenia into two broad categories of *fundamental* and *accessory* symptoms (Andreasen, 1997). In order to uniquely classify schizophrenia as a disorder, Bleuler argued that the fundamental symptoms were particular to schizophrenia while the accessory symptoms could occur in a variety of disorders. Bleuler identified six fundamental symptoms of schizophrenia, namely autism, ambivalence, loss of continuity with associations, loss

of affective responsiveness, loss of attention and loss of volition (Andreasen, 1997; Bleuler, 1950). Fundamental symptoms were characterised by abnormalities in cognitive processing and emotional processes. Accessory symptoms on the other hand included such phenomena more popularly characteristic of schizophrenia in particular and psychosis in general. These include such phenomena as delusions and hallucinations. Bleuler concluded that 'besides the specific permanent or fundamental symptoms, we can find a host of other, more accessory manifestations such as delusions, hallucination or catatonic symptoms ...' (Bleuler, 1950, p. 13). Some writers suggest that these symptom descriptions of Bleuler closely match those which are now referred to as basic defects in cognitive processes such as volition, ability to think abstractly, initiation of thoughts and language, and the ability to attribute affects to experiences (Andreasen, 1997). Similarities however cannot be ignored between Kraepelin and Bleuler's theory of psychosis and schizophrenia. Both stressed the importance of a loss of cognitive, affective, volitional and attentional functioning in schizophrenia. Where they differed in their theories is that while Kraepelin thought that such deficits in functioning were the central symptoms which led to the disorder, Bleuler stated that they were pathognomonic. By the end of the twentieth century Bleuler's perspective on the illness has dominated with clinicians who are taught to define and diagnose schizophrenia based upon Bleuler's fundamental symptoms such as loosening of associations and blunting of affect (Andreasen, 1997).

Schneider's First Rank symptoms

By the 1960s and 1970s inspired by improving diagnostic precision and reliability, the nosological structure of schizophrenia presented by both Bleuler and Kraepelin became problematic. The problem with Kraepelinian and Bleulerian

notions of schizophrenia was that the phenomenology in which they describe psychosis and in particular schizophrenia, could also be present in a myriad of different disorders. This made research into schizophrenia problematic since it was difficult to identify what disorder the subjects actually had when other mental disorders could also express anhedonia, avolition and others. This was most notable with the emergence of depressive illness that could equally express these symptom clusters. It is because phenomena such as hallucinations and delusions provided clearer phenomenological boundaries that these *florid* symptoms were steadily given greater prominence as defining schizophrenia (Andreasen, 1997).

Through the 1960s and 1970s British Psychiatry was increasing in influence. Coupled with Karl Jasper's philosophical influence on Kurt Schneider, the phenomenological emphasis turned to psychotic experiences that in Jasper's argument were 'nonunderstandable' that is symptoms " ...that a normal person could not readily imagine or experience' (Andreasen, 1997, p. 107). Like Bleuler, Schneider wanted to identify symptoms that could be fundamental and in some ways a unique identifier of schizophrenia. Schneider argued that symptoms that caused an inability of the sufferer to distinguish boundaries between self and not-self, and a loss of a sense of personal autonomy could be classified as "first-rank" symptoms. Such symptoms described a loss of autonomy, insertion of other's thoughts or delusions of being controlled by outside forces. According to Schneider, the presence of one or more first-rank symptoms in the absence of any organic disease, is indicative of schizophrenia (Sims, 1997). These symptoms are exemplified in Table 2.2. Schneider argued that these symptoms are not comprehensive, and in order for symptoms to be regarded as first rank they (a) must occur with reasonable frequency, (b) not occur in conditions other than schizophrenia and, (c) must not be too difficult

to determine whether they are present or not (Sims, 1997). Schneider's first rank symptoms are not a diagnostic checklist; in that having seven first-rank symptoms does not determine greater psychopathology than a subject who has three.

Table 2.2
Schneider's First Rank Symptoms of Schizophrenia [2]

Schneider's First-rank symptoms
Delusion
1. Delusional precept
Auditory hallucinations
2. Audible thoughts
3. Voices arguing or discussing
4. Voices commenting on the patients action
Thought disorder: passivity of thought
5. Thought withdrawal
6. Thought insertion
7. Thought broadcasting (diffusion of thought)
Passivity experiences: delusion of control
8. Passivity of affect ('made' feelings)
9. Passivity of impulse ('made' drives)
10. Passivity of volition ('made' volitional acts)
11. Somatic passivity (influence playing on the body)

With such clear operational definitions of schizophrenia, researchers have adopted the Schneiderian model of schizophrenia into measuring and diagnosing schizophrenic symptoms. The most notable example is the Present State Examination developed by Wing, Cooper, and Sartorius, (1974). The Present State Examination provides researchers with a means of ascertaining which symptoms and syndromes are present. Some argue that the emphasis on Schneiderian first-rank symptoms satisfies the fundamental need to find an anchor point in the ever changing phenomenological landscape of schizophrenia (Andreasen, 1997). This shift away from deficit symptoms first described by Kraepelin and Bleuler allows schizophrenia as a notion to remain stable albeit within an artificially and statistically derived

framework. The success of such a technique has allowed the Schneiderian first-rank symptoms to be cardinal to the understanding and diagnosis of schizophrenia, and has indeed been introduced into other diagnostic instruments including the Schedule for Affective Disorders and Schizophrenia (SADS) (Endicott & Spitzer, 1978), and the Research Diagnostic Criteria (RCD) (Spitzer, Endicott, & Robins, 1978). Such an endorsement of florid symptoms has led schizophrenia and indeed other psychotic states to be defined by primary hallucinatory and delusionary symptoms. This may have evolved out of the desire to see nosological stability within a disorder that is notoriously unstable. Equally, such an emphasis on first rank symptoms could have been championed in an attempt to reduce over diagnoses of schizophrenia from the presentation of "mild Bleulerian negative symptoms" (Andreasen, 1997, p. 108). Not so surprisingly, the development of new ways of conceptualising schizophrenia and psychosis sought a compromise between both the deficit syndromes and the florid symptoms into a new synthesis – the theory of positive and negative symptoms.

Crow's positive and negative symptoms and the emergence of a two factor theory

In 1980, Crow published an influential article proposing two types of schizophrenia known as Type I where positive symptoms are present (i.e., hallucinations, delusions, and thought disorder) and Type II where negative symptoms predominate (i.e., affective flattening, poverty of speech, loss of drive) (Crow, 1980). Crow argued that the Schneiderian first rank symptoms were too restricted and although they may have increased the reliability of the diagnosis, they may not have necessarily increased the prediction of outcome. The proposal of Type I and II

[2] *Note*: Adopted from *Symptoms in the mind: an introduction to descriptive psychopathology*: Second Edition (p. 149), by A. Sims London: W.B. Saunders Company Ltd.

schizophrenia was based upon not only the differing presentations of patients but also the different prognosis between the types. This is expressed in Table 2.3. Type I was seen as acute schizophrenia where the condition was likely to be reversible, responsive to neuroleptic medication and hence a good prognostic outcome. On the other hand, Type II is characterised by a more chronic condition, and may not be receptive to neuroleptic medication and more enduring as a presentation. It was questionable whether the condition in Type II is reversible.

Table 2.3
Two Syndromes in Schizophrenia [3]

	Type I	Type II
Characteristic symptoms	Hallucinations, delusions, thought disorder (positive symptoms)	Affective flattening, poverty of speech, loss of drive, (negative symptoms)
Type of illness in which most commonly seen	Acute schizophrenia	Chronic schizophrenia, the "defect" state
Response to neuroleptics	Good	Poor
Outcome	Reversible	? Irreversible
Intellectual impairment	Absent	Sometimes present
Related pathological process	Increased dopamine receptors	Cell loss and structural changes in the brain

This notion of positive and negative symptoms as distinctive entities was not new in the literature. Indeed, Kraepelin (1919) posited that there are two main processes underlying schizophrenia: "the weakening of those emotional activities which permanently form the mainspring of volition" and "the loss of the inner unity of the activities of intellect, emotion and volition" (Kraepelin, 1919, p. 75). These two processes are rather similar to the current negative and disorganisation syndromes posited by later theorists (Peralta et al., 1994). In addition Hughlings-Jackson, in 1931, argued that negative symptoms represented a loss of function while positive

symptoms represent an exaggeration of normal functioning. Hughlings-Jackson describes positive functions of hallucinations and delusions as release phenomena that describe how such symptoms are often an extension of normal functioning processes. What Crow added to the dichotomous theory of schizophrenia was to regain renewed interest in the pathophysiological aspects of schizophrenia. Crow did this by arguing that changes in the dopaminergic receptors within the brains of people with schizophrenia led to the expression of psychotic symptoms. Neuroleptic medication which blocked dopamine receptors appear to be limited to positive symptoms characteristic of acute schizophrenia. The more chronic condition where negative symptoms were more predominate particularly in institutionalised patients is more likely to be less receptive to drug treatment (Crow, 1980)

The pathological processes which underlie Types I and II are assumed to be independent of one another, but may coexist in the same patient (Crow, 1980; Peralta et al., 1994). Such a dichotomous model predicts that positive and negative symptoms exemplified in Table 2.3 weigh in different independent factors. This theoretical proposal pathed the way for more increasingly complex models of schizophrenia which used the bed rock of the positive and negative dichotomy (Andreasen, 1997).

The positive and negative syndrome scale and other measures of the two factor model

With a renewed interest in the positive and negative dichotomy Kay, Fiszbein, and Opler (1987), developed the Positive and Negative Syndrome Scale (PANSS). This scale was an amalgam of 18 items from the Brief Psychiatric Rating Scale

[3] Note. Table from Molecular pathology of schizophrenia: more than one disease process? By T.J. Crow, 1980, *British Medical Journal, 280*, p. 67.

(BPRS) (Rhoades & Overall, 1988) and 12 items from the Psychopathology Rating Scale (PRS) (Singh & Kay, 1975). The PANSS operates within a semi-structured interview format where each item is placed on a seven point scale from 1 = absent to 7 = extreme. The PANSS is scored into three scales, these being a seven-item scale for positive symptoms, a seven item scale for negative symptoms, and a sixteen item general pathological symptom scale. The following table describes the PANSS instrument.

Table 2.4
Items Within the Positive and Negative Syndrome Scale

	Scale		Scale
	Positive Scale		General Scale
P1	Delusions	G1	Somatic concern
P2	Conceptual disorganisation	G2	Anxiety
P3	Hallucinatory behaviour	G3	Guilt feelings
P4	Excitement	G4	Tension
P5	Grandiosity	G5	Mannerisms & posturing
P6	Suspiciousness/Perception	G6	Depression
P7	Hostility	G7	Motor retardation
	Negative Scale	G8	Uncooperativeness
N1	Blunted affect	G9	Unusual thought content
N2	Emotional withdrawal	G10	Disorientation
N3	Poor rapport	G11	Poor attention
N4	Passive/apathetic social withdrawal	G12	Lack of judgement
N5	Difficulty in abstract thinking	G13	Disturbance or volition
N6	Lack of spontaneity & flow of conversation	G14	Poor impulse control
N7	Stereotyped thinking	G15	Preoccupation
		G16	Active social avoidance

The PANSS attempted to address the current limitations in the research, such as the lack of a standardised instrument, poor construct validity and longitudinal reliability, by developing a comprehensive measure of symptoms particular to schizophrenia and related disorders (Kay, Opler & Fizbein, 1986a). Kay, Lewis and Fizbein (1986a) found that previous empirical investigation into the syndromes of schizophrenia had inadequate definition of symptoms and poor operational criteria for

ratings. Also they had little information on reliability and validity and a lack of standardised procedure in securing data (Kay et al., 1986a). With the advent of the PANSS the literature was moving away from clinical observation as the basis of theoretical model development of schizophrenia to one of empirical validation. Through their validation studies using the PANSS, Kay and colleagues found that positive and negative symptoms are negatively correlated and suggested that these symptoms are mutually exclusive syndromes (Kay et al., 1987; Peralta et al., 1994).

Other instruments were also developed to investigate the positive and negative syndromes. Notably, the Schedule for the Assessment of Negative Symptoms (SANS) (Andreasen, 1984b) and the Schedule for the Assessment of Positive Symptoms (SAPS) (Andreasen, 1984a). These three instruments (PANNS, SAPS and SANS) became the standard procedure of measuring schizophrenic syndromes. Researchers using these instruments were also able to confirm the positive negative symptom dichotomy (see Table 2.5). Levine, Fogg and Meltzer (1983) were able to identify that delusions and hallucinations were considered part of the positive symptom dimensions and formal thought disorder considered part of negative symptoms. Meanwhile Mortimer, Lund and McKenna's model using exploratory factor analysis (Mortimer, Lund, & McKenna, 1990) included positive thought disorder into positive symptoms and the "bizarre symptoms" were divided between both the negative and positive syndromes. However, what became increasingly evident in the literature was the emergence of a " ...third 'disorganisation' syndrome in schizophrenia" (Mortimer et al., 1990, p. 46) characteristic of cognitive deterioration.

Table 2.5 provides the reader with a synopsis of the major studies conducted in the modelling of psychosis. The purpose is to provide a chronological history of the varying competing models and the empirical evidence that supports or disconfirms their existence within the literature. As such, the reader may find that the empirical evidence does not follow a smooth narrative where one study improves on the next. What appears is a general progress where more complex models emerge from the research as larger studies and more sophisticated analytical techniques are employed.

Three factor theory: emergence of disorganisation

The first empirical studies into the structure of schizophrenia were characterised by two elements (Bilder, Mukhergee, Rieder, & Pandurangi, 1985; Kay, Opler, & Fiszbein, 1986b; Kulhara & Chandiramani, 1990; Levine et al., 1983; Mortimer et al., 1990). The first was the influence of the dichotomous model proposed by Crow (1980) and the second was low sample size and rudimentary exploratory factor analysis. By the early 1990s, sample sizes increased along with the sophistication of statistical analysis employing confirmatory factor analysis. It is through these developments that more sophisticated models of schizophrenia emerged.

Table 2.5

Empirical Research on the Models of Schizophrenia

Authors	Sample Size	Diagnosis, source of patients	Sample Characteristics	Rating Scales	Models Tested	Best Fitting Model [1]	Analysis	Results
(Kay et al., 1986b)	101	DSM III R schizophrenia	69.3% male age: 36.81	PANSS	3 factor model	3 factor model	Multiple Regression	Positive $R^2 = 0.72$ Negative $R^2 = 0.79$ General $R^2 = 0.68$
(Levine et al., 1983)	89	Research Diagnostic Criteria schizophrenia	76.4% male age: 28.9	SADS-C NOSIE	3 factor model	Positive cognitive-affective negative symptoms social withdrawal	Rasch modelling	See paper
(Bilder et al., 1985)	32	Research Diagnostic Criteria schizophrenia	Age: 32.5	SADS		Disorganisation of thought, blunting of affect and volition, florid psychotic features	Exploratory factor analysis	Principle components analysis
(Liddle, 1987)	40						Exploratory factor analysis	
(Kulhara & Chandiramani, 1990)	98	ICD-9 schizophrenia	55.6% male 52 up to 30 years 27 30 years +	SANS SAPS		Positive, negative, bizarre behaviour	Exploratory factor analysis	Principle components analysis First FA 84.53% variance Second FA 68.89% variance
(Mortimer et al., 1990)	80	Research Diagnostic Criteria schizophrenia	Age: 45	SANS	2 factor solution	Positive, negative	Exploratory factor analysis	Procedure not mentioned 62.2% variance
(Phillips et al., 1991)	446	DSM III R schizophrenia	56% male age: 31	SANS SAPS		3 factor solution negative symptom, bizarre behaviour/formal thought disorder hallucinations/delusions	Exploratory factor analysis	Principle components analysis with varimax rotation 70.1% variance
(Peralta, de Leon, & Cuesta, 1992)	115	DSM III R schizophrenia	69% age: 35.7	SANS SAPS		3 factor solution negative, delusions hallucinations, disorganisation	Exploratory factor analysis	Principle components analysis with varimax rotation 70% variance
(Peralta et al., 1994)	253	DSM III R schizophrenia	65% male age: 36 onset: 25.3	SANS SAPS	1, 2, 3 and 4 factor models	4 factor model positive, disorganisation, negative, relational	Confirmatory factor analysis	AGFI = 0.919 χ^2 =60.5 df = 29

Authors	Sample Size	Diagnosis, source of patients	Sample Characteristics	Rating Scales	Models Tested	Best Fitting Model [1]	Analysis	Results
(Brekke, DeBonis, & Graham, 1994)	193	Research Diagnostic Criteria 66% schizophrenia 44% schizoaffective disorder	73% male age: 33	BPRS	1 2 & 3 factor models	3 factor model positive, negative and disorganised	Confirmatory factor analysis	GFI = 0.91 χ^2 =130.3 df = 62
(Cuesta & Peralta, 1995)	100	DSM-III R schizophrenia	69% male age: 35.8 onset: 25.0	PANSS	2, 3 and 4 factor models	4 factor model positive, negative, disorder of relating, disorganisation	Confirmatory factor analysis	AGFI = 0.827 χ^2 =56.54 df = 38
(Lindenmayer, Berstein-Hyman, Grochowski, & Bark, 1995)	240	DSM III R	2 groups young: 22.64 old: 54.95	PANSS		5 factor solution negative, cognitive, positive, excitement, depression/anxiety	Exploratory factor analysis	Principle components analysis with equamax rotation 57.5% variance
(Peralta & Cuesta, 1995)	253	DSM III R 100% schizophrenia	67% male age: 36	SANS	1 to 7 factor models	5 factor model affective flattening anhedonia-asociality attention avolition apathy alogia	Confirmatory factor analysis	GFI = 0.828 χ^2 =471 df = 142
(Mellers, Sham, Jones, Toone, & Murray, 1996)	114	DSM III R schizophrenia	89% male age: 28.1	PSE		4 factor solution disorganisation syndrome psychomotor poverty hallucinations delusions	Exploratory factor analysis	Not mentioned 58% variance
(White, Harvey, Opler, & Lindenmayer, 1997)	1 233	Research Diagnostic Criteria schizophrenia schizoaffective disorder	59% male age: 47	PANSS	20 models	5 factor pentagonal model positive, negative, dysphoric mood, activation and autistic preoccupation	Confirmatory factor analysis	CFI = 0.917 χ^2 = 725.3 df = 235
(Peralta & Cuesta, 1998)	253	DSM III R 100% schizophrenia	67% male age: 36	SAPS	1 to 5 factor models	5 factor model: hallucinations, non-Schneiderian delusions, Schneiderian delusions, bizarre behaviour, positive formal thought disorder	Confirmatory factor analysis	GFI = 0.844 χ^2 =605.9 df = 265
(Smith, Mar, & Turoff, 1998)	683	Schizophrenia DSM III R or RDC schizophrenia	Meta-analytic review of 28 samples	SAPSS/SANS BPRS PANSS	1 to 4 factor model	3 factor model positive negative, disorganisation	Confirmatory factor analysis	GFI = 0.94, NNFI 0.82 RMSEA = 0.10

Authors	Sample Size	Diagnosis, source of patients	Sample Characteristics	Rating Scales	Models Tested	Best Fitting Model [1]	Analysis	Results
(Lançon, Aghababian, Llorca, & Auquier, 1998)	205	DSM IIIR 100% schizophrenia	60% male age: 36.2	PANSS	Forced 5 factor model	5 factor model negative positive excited, depressive cognitive components	Exploratory factor analysis	Principle-components analysis with varimax rotation 57% variance
(Long & Brekke, 1999)	193 (intake)	Schizophrenia 62.69% Schizoaffective 37.30%	73.05% male age: 33.18	BPRS	4 factor model	4 factor model thought disturbance, anergia, affect, disorganisation	Confirmatory factor analysis	GFI = 0.91 χ^2 =987.71.54 df = 129
(Lançon, Auquier, Nayt, & Reine, 2000)	340	DSM III R Schizophrenia	118 acute 224 chronic	PANNS	Forced 4 factor model	5 factor model negative, positive, excitation, depression, cognition	Exploratory factor analysis	Principle components analysis with varimax rotation 62.1% variance
(Lykouras et al., 2000)	258	DSM III R Schizophrenia	80.2% male age: 31.2	PANSS		5 factor solution negative, excitement, depression, positive, cognition	Exploratory factor analysis	Principle components analysis with varimax rotation 66.8% variance
(Loftus, DeLisi, & Crow, 2000)	103	DSM III R Schizophrenia Schizoaffective disorder	62% male age: 36.7	SADS		2 factor model factor 1: thought withdrawal, insertion and broadcasting factor 2: third person voices, thought echo and running commentary	Exploratory factor analysis	Principle components analysis with varimax rotation 67% variance
(Ehman, Holliday, MacEwan, & Smith, 2001)	165	DSM III R Schizophrenia 68% Affective disorder (9%) Schizoaffective disorder (16%) Other diagnosis (7%)	66.6% male age: 34.5	RAPP		5 factor solution Aggression, positive symptoms, negative symptoms, somatisation/anxiety, organic/disorganisation	Exploratory factor analysis	Maximum likelihood method with varimax rotation
(Dudgeon & Mackinnon, 2001)	221	DSM-IIIR schizophrenia	Not mentioned	PANSS	3 factor model	3 factor model positive, negative and disorganised	Confirmatory factor analysis	(not confirmed) χ^2 =127.9 df not supplied RMSEA = 0.091

Note: SADS-C = Schedule for Affective Disorders and Schizophrenia-Current, NOSIE = Nurses' Observation Scale for Inpatient Evaluation, RAPP = Routine Assessment of Patient Progress, PANNS = Positive and Negative Syndrome Scale, SANS = Schedule for Assessment of Negative Symptoms, SAPS = Schedule for Assessment of Positive Symptoms, BPRS = Brief Psychiatric Rating Scale, SPQ = Schizotypal Personality Questionnaire, PSE = Present State Examination. [1] Factors presented in order of highest to lowest loading.

Previous developmental work like Strauss et al. (1974) argued that in addition to positive and negative symptoms another syndrome, denoting disorders in relating, became evident as a separate factor. The authors suggested that each of the symptom clusters have different pathological processes. The disorders in relating would have their own longitudinal history different from those of the positive and negative symptoms. Coupled with this, the emergence of not only poor social relations but deteriorated cognitive states started to emerge as a separate syndrome.

Bilder and co-workers (Bilder et al., 1985) used exploratory factor analysis on a small sample of patients with schizophrenia (n = 32) and suggested a factor which describes "disorganisation of thought independent of current definitions of the positive / negative symptom construct" (Bilder et al., 1985, p. 409). To strengthen their assertion they performed neuropsychological tests that correlated with each of the symptom clusters which appeared " ...consistent with a process characterised by failure in the development of a normal repertoire of cognitive abilities" (Bilder et al., 1985, p. 409). They suggested that the "defect state" often characterised by negative symptoms typical of the Type II class described by Crow " ...may not be a monothetic construct" (Bilder et al., 1985, p. 409). Such a conjecture despite the methodological limitations of the study paved the way for more expansive exploration of the schizophrenia construct.

Liddle (1987) also argued for a three factor solution by using some of the items from both the SAPS (Andreasen, 1984a) and SANS (Andreasen, 1984b). By using exploratory factor analysis on 40 schizophrenia patients with persistent symptoms, the syndrome of positive symptoms (delusions and hallucinations), disorganisation (positive formal thought disorder, inappropriate affect and attention disturbances) and negative symptoms (excluding attention disturbances) were

interpreted. In other small exploratory studies Kulhara & Chandiramani (1990) described two exploratory factor analyses using the SAPS and SANS. The first involved 98 patients with schizophrenia as diagnosed by the International Classification of Diseases (ICD-9). The second involved a follow-up of 79 of these patients ranging from 18 to 30 months. In both analyses, a 3-factor model was confirmed with negative symptoms being described by affective flattening, anhedonia (i.e., lack of pleasure from life), and attentional impairment. The second factor described positive symptoms of hallucinations, delusions and bizarre behaviour. Alogia (i.e., amount of conversation and conversation initiation), avolition (i.e., curiosity, motivation and purpose) and thought disorder were described as the *thought disordered* syndrome. The only differences between the two analyses are that bizarre behaviour loaded in different factors between positive symptoms and thought disorder.

By 1991 the first substantial empirical investigation into the underlying symptomatological structure of schizophrenia was conducted by Phillips and colleagues (Phillips et al., 1991) on 446 schizophrenia patients in China. Using the SAPS and SANS via a principal components analysis with varimax rotation, they found results were similar to Liddle's study (Liddle, 1987) confirming a 3 factor solution (see Table 2.5). The first factor describes negative symptoms while the second factor identifies bizarre behaviour and formal thought disorder as a unitary construct. The final factor describes hallucinations and delusions. Phillips et al. (1991) argues that central to Crow's (1980) positive and negative symptom dichotomy, is the tenet that they are relatively independent of each other. Since the Phillips et al. (1991) study performed exploratory factor analysis on both intake and discharge at four inpatient settings, they were able to examine the relationship

between the two symptoms at two stages of the schizophrenic condition. They found that although the models confirmed a dichotomy between the two symptoms, the intake condition produced a negative relationship ($r = -0.20$, $p < 0.001$) between positive and negative symptoms while at discharge the relationship was positive ($r = 0.23$, $p < 0.001$). They argued that different and conflicting associations between positive and negative symptoms, found in previous studies (Andreasen, 1982; Rosen, Mohs, Johns, Small, & Kendler, 1984) were due mainly to the stage that subjects were assessed within their treatment. Phillips et al. (1991) concurred with Kay et al. (1988) that the disorganisation factor associated with cognitive deficits of attention are in fact broken in two; one that relates to positive symptoms the other with negative symptoms. A 1992 study conducted by Peralta, de Leon and Cuesta (1992) on 115 subjects with schizophrenia supported Liddle's three factor model, suggesting that the positive symptoms are not as homogenous as first postulated by Crow (1980). With larger studies a more complex picture was emerging in the literature as to the underlying structure of schizophrenia. Indeed Peralta, de Leon and Cuesta (1992) fortuitously argued that as a body of research " ...we are far from a valid classification of schizophrenia symptoms and the positive-negative dichotomy appears to be an oversimplification" (Peralta et al., 1992, p. 335).

Confirmatory factor analysis and increasing model complexity

By the mid 1990's, researchers were starting to employ confirmatory factor analytic techniques into schizophrenia research. Confirmatory factor analysis is the technique where the underlying structure of observed variables are assumed to covary within an *a priori* framework. In other words, the model is imposed on the data and

fit indices are provided to determine whether the model fits the data. In most instances fit indices above 0.90 are considered to indicate a model being a good fit to the data (Bentler, 1990). Exploratory factor analysis is largely an interpretive procedure where factor structures are generated by the data, generally on an atheoretical basis and post hoc.

With increasing sample sizes and more sophisticated statistical techniques the number of factors underlying the schizophrenic condition was expanding. At the forefront of this development that continued throughout the decade, were the Spanish researchers of Peralta and Cuesta. Their seminal work in 1994 (Peralta et al., 1994) introduced confirmatory factor analysis to 253 inpatient subjects with schizophrenia by testing one, two, three and four syndrome models to the data. The fourth dimension these researcher examined was the notion of relational deficits. This brought a sophistication to the modelling procedure since they had moved away from simple expressions of symptoms to the interpersonal relationships held between subjects with schizophrenia and the world in general. By using confirmatory techniques Peralta, Cuesta and de Leon were able to test the competing models of the two-syndrome models posited by Levine and Kay (Kay et al., 1987; Levine et al., 1983), and the three-syndrome model of Phillips (Peralta et al., 1992; Phillips et al., 1991; Strauss & Estroff, 1989). In addition, the researchers introduced a new syndrome coined "the relational syndrome" (Peralta et al., 1994, p. 732). The modified 4 factor theory (the bizarre behaviour item was dropped from the disorganisation syndrome due to very high standard errors) proved a reasonable fit to the data ($\chi 2 = 23.7$ df 21, $p = 0.307$ AGFI = 0.956, NNFI 0.995, NFI 0.968; note indices above 0.90 are determined to be a good fit to the data). The four factor model proposed positive symptoms (hallucinations and delusions), disorganisation (formal

thought disorder and inappropriate affect), negative symptoms (affective flattening, alogia and avolition) and relational syndrome (intimacy, closeness, and relationships).

In the same year Brekke, DeBoni and Graham (1994) performed a confirmatory factor analysis with six competing models. Three instruments were used, the first being the Brief Psychiatric Rating Scale (BPRS) (Overall & Gorham, 1988) that measured both positive and negative symptoms, two items from the Community Adjustment Form (CAF) (Test, Knoedler, Allness, Burke, & Brown, 1991) which were included in the negative symptoms that measures alogia, and four items from the Quality of Life Scale (QLS) (Heinrichs, Hanlon, & Carpenter, 1984) that measures anhedonia and avolition. Model 1 proposed a model in which two 2-factor models were tested. One model based on Crow's positive and negative symptom model (Crow, 1980) where the factors were held independent, and the other model where the factors were allowed to covary. Two 3-factor models with a disorganised syndrome were included in the modelling procedure. Once again the first 3-factor model proposed independence amongst the factors, while the second model proposed a covarying structure between the symptom dimension. The results showed that the 3-factor model where the factors were allowed to covary demonstrated a modest fit to the data (χ^2 = 130.3 df 62, GFI = .91 AGFI = 0.87 T-L = .89). Such a result indicated in a confirmatory sense that the factors were correlated and not arbitrarily or uniquely independent of one another. When examining the relationship between the factors it was found that the disorganised factor (most closely aligned to poor cognitive performance) was " ...more strongly related to both the positive symptoms (r = .29 p < .01) and negative symptoms (r = .38 p < .01)" (Brekke et al., 1994, p. 252). This research suggests that the underlying structure of schizophrenia was less orthogonal than originally postulated and that the different

symptomatological structure related to each other in more complex ways particularly with the disorganised factor 'binding' or acting as a 'glue' between the positive and negative symptoms.

Similar results were found in the Cuesta and Peralta study (1995) by using the PANSS (Kay et al., 1987) on 100 subjects with schizophrenia. Proceeding from previous research, Cuesta and Peralta tested the two dimensional model of Kay (Kay et al., 1986b) and the three dimensional models of Peralta et al. (1992) and Strauss et al. (1974). In addition to these models they examined the Peralta and Cuesta model mentioned above (Peralta et al., 1994) along with other 4-factor models. The 4-factor models proposed the addition of an excited factor added to the positive and negative symptoms and the syndrome of disorganisation. The other four-factor model proposed by Peralta et al. (1994) suggested the syndrome of poor relatedness as the fourth factor in the model. Another 4-factor model proposed by Kay's 'pyramidical model' which includes the dimensions of positive, negative, excited and depressive dimensions, was also tested. All models were tested using confirmatory factor analysis. The researchers concluded:

> the present data do not support the positive-negative bidimensional construct of schizophrenia symptoms. The models composed of three or four PANSS underlying psychopathological dimensions obtained better goodness of fit that null, one, or two-dimensional models. Moreover, better overall fit was obtained when the underlying psychopathological dimensions incorporated in the positive and negative construct were disorder of relating and / or disorganisation (Cuesta & Peralta, 1995, p. 478).

Increasingly researchers were beginning to reject the bidimensional positive-negative models first proposed by theorists in the 1980's (Crow, 1980; Kay et al., 1986b). Indeed Kay and Sevy (1990) were aware of these criticisms, and argued that " ...the positive-negative construct had "validity" but not "sufficiency" to explain all the psychopathological expressions of the schizophrenic domains (Cuesta & Peralta, 1995, p.479).

By the mid 1990's, new models were emerging that explored the possibility that schizophrenia had more affective components to the expression of the condition. This notion expanded the borders of clinical nosology away from "hard" signs and symptoms to more "softer" and subtle aspects of the human condition. These included the notion of interpersonal relatedness, depression and anxiety, beyond the stronger phenomenology determined in the negative syndrome. Lindenmayer et al. (1995) argued that previous models of schizophrenia were too simplistic in the sense that most people with schizophrenia present a mixed syndrome where the construct of positive and negative symptoms are variable. As the negative symptoms started to break into more complex syndromes that included cognitive deterioration and disorders in relatedness it was difficult to distinguish as Lindenmayer et al. (1995) argued between primary and secondary negative symptomatology. To address these issues the Lindenmayer et al. (1995) study used principal components analysis on 240 subjects with schizophrenia. By examining the scree plot a 5-factor solution was retained and examined. The 5-factor solution is described in the following table.

Table 2.6
Factor Loadings of the Lindenmayer et al. (1995) Study Using Principal Components Analysis of the PANSS with Equamax Rotation

Symptom Dimension	Symptom Dimension
F1 Negative component	F2 Excitement component
N2 Emotional withdrawal	P4 Excitement
N4 Passive/apathetic withdrawal	G14 Poor impulse control
N6 Lack of spontaneity	P7 Hostility
N3 Poor rapport	G4 Tension
N1 Blunted affect	
G16 Active social withdrawal	
F3 Cognitive component	F4 Positive Component
P2 Conceptual disorganisation	P1 Delusions
G10 Disorientation	G9 Unusual thought content
N5 Difficulty in abstract thinking	P5 Grandiosity
G5 Mannerisms and posturing	P6 Suspiciousness/persecution
G11 Poor attention	P3 Hallucinations
F5 Depression component	
G2 Anxiety	
G3 Guilt feelings	
G6 Depression	
G1 Somatic concern	
G15 Preoccupation	

In this study the negative syndrome is broken into two with the "harder" symptoms of emotional withdrawal etcetera loading on the negative symptoms factor. The "softer" symptoms that describe experiences held by other mental health conditions such as depression and anxiety have loaded on the depression factor. This model is argued by the researchers to be a more accurate representation of schizophrenic psychopathology since the model explains " ...several separate but coexisting domains of psychopathology" (Lindenmayer et al., 1995, p. 31). Other researchers supported similar claims (Mellers et al., 1996). With such a model the construct of psychosis was entertaining more expansive models of the psychotic experience beyond bidimensional boundaries.

Two confirmatory factor analytic studies by Peralta and colleagues independently examined negative symptomatology using the SANS (Peralta &

Cuesta, 1995) and positive symptoms using the SAPS (Peralta & Cuesta, 1998). Both studies demonstrated poor fit to the data. The study examining negative symptoms found that a 5-factor theory with the dimensions of affective flattening, alogia, avolition-apathy, anhedonia-asociality and attentional impairment explained the data the best on 253 subjects with schizophrenia. The results however demonstrated poor fit to the data (χ^2 = 471 df 142, GFI = .828 NFI = 0.849). Despite this the authors claimed that the study " ...provides evidence of the factorial validity of the SANS" (Peralta & Cuesta, 1995 p. 1455). The authors recognised that the 5-factor solution demonstrated high intercorrelation with each other identifying the dimensions within the negative symptoms as not being orthogonal. They conclude that in particular to negative symptoms, " ...schizophrenic negative symptoms seem to constitute a strong cohesive syndrome within which some differentiated dimensions are possible" (Peralta & Cuesta, 1995, p. 1456). Despite these claims, an alternative view was taken in the study examining positive symptoms using the SAPS on the same cases in the 1995 study. The authors argued that increasing multi-dimensional models of positive symptoms explained the data with a 5-factor solution of hallucinations, non-Schneiderian delusions, Schneiderian delusions, bizarre behaviour, and positive formal thought disorder fitting the data. This 5-factor solution of positive symptoms like its negative symptom counterpart demonstrated poor fit to the data (χ^2 = 605.9 df 265, GFI = .844). Such results suggested greater complexity of structure even within the positive and negative symptom dichotomy. Peralta and Cuesta therefore posited that relying on a unidimensional measure of positive symptoms as suggested by the original scoring of the SAPS instrument is likely to be misleading.

By the late 1990s some confusion surrounding the exact dimensionality of schizophrenia became evident. This confusion appeared largely as a result of using

two differing instruments with small studies and focussing on only one aspect of the psychotic experience. The White et al. study published in 1997 was the first study to use a large multi centre sample (5 sites) of 1 233 subjects with schizophrenia. The authors correctly stated that different solutions that ranged from 3 to 7 factors were often dependent " ...on the scale and analytic method" (White et al., 1997, p. 264). They also recognised, as was emergent in the literature previously (Peralta & Cuesta, 1995), that solutions were often dependent on extraneous variables such as medication status and overall clinical state (psychotic vs residual vs remitted). In addition length of illness, age and gender were also seen as confounding variables that may contribute to differences in factor analytic solutions (White et al., 1997). The exploratory factor analytic studies previously conducted were always at risk of providing results that would be " ...idiosyncratic to the sample involved" (White et al., 1997, p. 264) since they could not impose a model structure in an *a priori* sense. Like most instruments in psychiatry the item numbers are often larger than the samples of interest. The original PANNS (see Table 2.4) is a 30-item instrument. Most factor analytic studies should operate a "rule of thumb" where a minimum of 10 to 20 observations per item is required for effective factor solutions (Tabachnick & Fiddell, 1996). This means that a critical sample size between 300 to 600 cases is required for optimal validity of the procedure. By observing the studies summarised in Table 2.5 only one study (Phillips et al., 1991) prior to the White et al. (1997) met this criteria.

Armed with these criticisms, 20 alternative models (most of them presented in this chapter) were examined for their factorial validity in the White study (White et al., 1997). When each of the 20 models were entered into the data, none of them met the minimum acceptable fit criteria of 0.90. Even with the Robust Confirmatory Fit Index (RCFI) which corrects for non-normal distribution amongst the items (Bentler,

1990; Byrne, 1994) the highest fitting index was 0.862. Due to the poor results of previous models, the research stratified the data into half according to site, sex and age. An exploratory factor analysis was performed using maximum likelihood principal factors analysis with varimax rotation. The number of factors extracted was determined using the scree plot. The resultant 5-factor model from the exploratory procedure was then evaluated using Confirmatory Factor Analysis (CFA). The Lagrange Multiplier (LM) test for adding parameters to the model was used as a respecification procedure if the model proved a poor fit to the data. The result of the CFA initially demonstrated a poor fit to the data (χ^2 = 2 175.96 df, 365 RCFI = 0.746). The LM test for additional parameters found 5 items loaded on more than 2 factors and 1 item (conceptual disorganisation) suggested a complex uninterpretable loading. The 5 items (lack of judgement, suspiciousness, active social avoidance and disorientation) were dropped from a subsequent model and re-entered as a new model. This procedure was to examine the correlated error between the items. The result of this CFA showed continuing poor fit to the data (χ^2 = 1 010 df, 260 RCFI = 0.863). The LM suggested five variable pairs had highly correlated error terms (not mentioned in the paper which errors were correlated). These pairs were freely calculated and this model entered into the data for analysis. The results showed an adequate fit to the data (χ^2 = 725.3 df, 235 RCFI = 0.917) and no subsequent modifications were made to the data. The 25 item 5-factor solution described a pentagonal model describing positive, negative, dysphoric mood, activation and autistic preoccupation. Tests for invariance between age, symptom severity and chronicity of illness did not differ in their overall symptom structure.

This study to date was the most comprehensive in investigating the construct of psychotic experience of those subjects with schizophrenia. The model suggests a

more complex relationship between the different syndromes than previously thought. The contribution of this study to the literature suggested a 5-factor model that was stable across the stages of the illness, and was not influenced by poor sample size, age or gender effects. In all probability the White study (White et al., 1997) provides one of the most accurate descriptions of the underlying structure of the psychotic condition.

Smith, Corrine and Turoff (1998) employed a meta-analytic confirmatory factor analysis by combining 28 independent samples. A correlation matrix was constructed by performing z' transformation of each study independently. They then summed each of these transformations, and transformed this quotient back into an *r* for entry into a final correlation matrix for analysis using confirmatory factor analysis. The data consisted of 683 subjects with schizophrenia. Four models were tested including a unidimensional severity-liability hypothesis. This hypothesis suggests that all schizophrenic symptoms are " ...associated with a single latent factor and their intercorrelations can be accounted for by their joint relations with this single factor" (Smith et al., 1998, p.62). A dual-process hypothesis was also postulated where two factors are said to be unrelated to each other, where each " ...gives rise to a characteristic set of manifest symptoms" (Smith et al., 1998, p. 63). In addition to these hypothesis a 3 factor model based upon Liddle's work (Liddle, 1987) posits intercorrelations among three symptoms; psychomotor poverty, disorganisation and reality distortion. Since this model is silent with respect to anhedonia-asociality and bizzare behaviour, the anhedonia-asociality symptom was placed on the psychomotor poverty factor and the bizarre behaviour on the disorganised factor. Results of the confirmatory factor analysis, supported the three factor model of Liddle's description (Liddle, 1987) (GFI = .0.94 RMSEA = .10). Despite the model showing reasonable

fit based upon the fit indices, the Root Mean Square Error of Approximation (RMSEA) was poor with values higher than .10 demonstrating poor fit to the data (Browne & Cudeck, 1993). Smith et al. (1998) concluded that the best fitting model was still a poor approximation of the data and suggested that a 4-factor model may be a better representation of the dimensionality of schizophrenia. This 4-factor model argues for the splitting of the positive symptoms into those encompassing reality distortion and those encompassing disorganisation. They conclude the study by stating correctly that "progress toward more adequate models, however, begins with insight into the inadequacy of the current one" (Smith et al., 1998, p. 68). Such an admission points towards a more complex model structure arguing a 4- or 5-factor dimensionality.

While most studies to date have been concerned with the underlying factor structure of schizophrenia, the Long and Brekke (1999) study investigated whether these structure were invariant over time. This was done by taking the 18 item Brief Psychiatric Rating Scale (BPRS) (Overall & Gorham, 1988), and administering it to 193 subjects with schizophrenia every six months over a 3 year period. The concern was to test configural invariance of the 18 and 16 items versions of the BPRS over each time wave. A 4-factor oblique model proposed by Mueser, Curran, and McHugo (1997), was tested. The four-factor model consisted of thought disorder, anergia, affect and disorganisation within both a 16 and 18 item measure. Invariance was only found at the configural level with the 16 item version demonstrating the best fit to the data (χ^2 = 3,718.24 df, 658 CFI = 0.91). The study found that although the configural aspect of the model proved invariant (i.e., the same factor structure) the magnitudes of the item-factor and factor-factor relationships varied over time. At the more refined metric and phi level of measurement, each model proved variant over time. The

authors recognised that the study suffered from a small sample that was highly non-normal in distribution. In short, the conclusion that can be drawn from the study shows that although the basic structure remains the same, the strength of the associations between the factors and between items and factors remain variant. This is a problematic issue in time series analysis and structural modelling when linkages between the latent factors over time are examined for their causal influence. These matters will be discussed in detail in Chapter VI.

A five-factor structure was calculated in a study by Lançon et al. (2000) on 340 subjects with schizophrenia. Using exploratory factor analysis with the PANSS a more expansive model was proposed. The factors included negative, positive, excitation, depression and cognition factors. This model supported the result of the 1998 Lançon et al. study. In addition they were able to report a similar factor structure at both the acute and progressive phase of the illness. The limitations of the study are evident when taking into consideration the advanced techniques in structural invariance using confirmatory factor analysis similar to the technique employed by Long and Brekke (1999). Although a similar structure was reported, the use of principal components analysis is subject to the limitations cited by the White et al. (1997) study. For example, the fact that both samples come from the same study and methods of collection, it is likely that the results would be similar. Even if they are similar, principal components is an exploratory technique that does not use *a priori* hypothesis testing and in a sense is only a projective interpretative technique.

Lykouras and colleagues (2000) used a 2-step process where they first examined the underlying factor structure of 258 patients with schizophrenia assessed using the PANSS under principal components analysis. Using the scree plot criterion, a 5-factor model that described negative symptoms, excitement, depression, positive

symptoms and cognitive impairment was entered into a CFA model. The fit indices showed a very poor fit to the data (χ^2 = 1283.54 df, 179 CFI = 0.611). Problem items in the model included unusual thought content and difficulty in abstract thinking. Although the authors suggested that the 5-factor model supports previous studies and demonstrated good internal consistency, particularly the negative symptoms factor, it is difficult to say that the model can give confidence to researchers that the underlying factor structure using the PANSS is stable. The largest study of its kind was conducted by White et al. (1998), who attested to the fact that the underlying dimensionality of schizophrenia is still largely unknown. Lykouras at al. (2000) argued that the factor structure found in their study may be more due to " ...the factor structure of the PANSS rather than the illness" (Lykouras et al., 2000, p.99). Little is mentioned in the literature that the PANSS scoring procedure places the factor structure in order of scoring. In other words, items within the instrument are ordered already under prescribed factors. This ordering effect can naturally bias results particularly with observational instruments since unnatural covariation can occur. For a factor analytic procedure to be truly valid the items must be randomly allocated throughout the measuring instrument. This is deliberately done in order for items to naturally covary together without the influence of common method variance and response bias. Without this procedure it is difficult to examine items which are deliberately placed in order of a prescribed factor structure and expect the analysis to perform any other way.

The pyramidal model of schizophrenia

While most recent studies in schizophrenia suggest a more complicated structure than the simple positive negative symptoms dichotomy, few researchers have examined a structural theory surrounding the syndromes. With confirmatory factor analysis, the notion that affective "soft" states such as anxiety and depression can exist as part of the syndrome of schizophrenia as much as the "hard" negative symptoms of alogia and affective flattening became increasingly accepted. Although researchers have argued that these two states can be separately measured (Addington, Addington, & Atkinson, 1996; Addington, Addington, & Maticka-Tyndale, 1993a; 1993b; 1994; Addington, Addington, Maticka-Tyndale, & Joyce, 1992), it is really difficult to say that at the experiential level negative symptoms can be separated from states that people without schizophrenia would describe as depression or anxiety (Preston & Harrison, 2003). It is quite possible that symptoms described along the negative spectrum may not be experienced any differently than internal states of depression and poor relatedness. If such symptomatology is separate (i.e., depression from negative symptoms) they are in any rate likely to be highly correlated. With the emergence of the third factor of disorganisation or cognitive distortion from the positive and negative symptom dichotomy, it is likely that these symptoms would play a complex interactional role with one another to influence the outcome and course of the psychosis.

One of the best models to reflect a synthesis between syndromes that appear specific to schizophrenia and other symptoms and general symptoms of anxiety and depression is the von Knorring and Lindström (1995) five-factor pyramidal model (see Figure 2.1). In this model the authors argue that syndromes can be placed on an axis with positive and negative symptoms linked via depression and anxiety and the

cognitive states such as disorganisation can be tied together via an excitement or activation syndrome. The negative factor is described by symptoms such as blunted affect, lack of spontaneity, motor retardation, poor rapport, emotional withdrawal and passive social avoidance. The positive factor is described by symptoms such as delusions, unusual thought content, grandiosity and hallucinatory behaviour. The excited factor described such symptoms as hostility, uncooperativeness, poor impulse control and excitement. The anxious/depressive factor includes anxiety, depression, guilt feelings and somatic concern (von Knorring & Lindström, 1995).

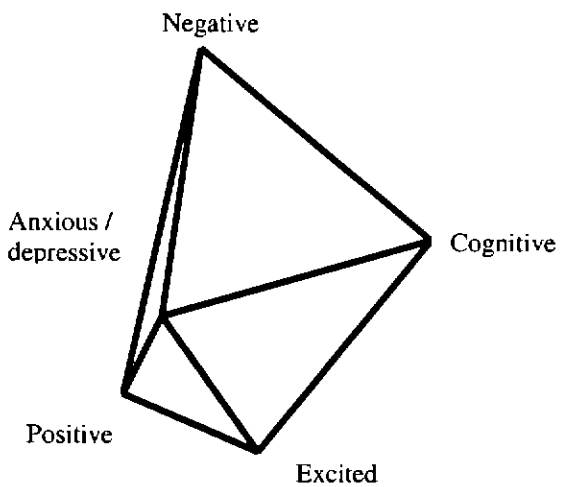

Figure 2.1[4]. Schematic representation of the five-factor pyramidical model of schizophrenia proposed by von Knorring and Lindström.

Such a model allows for symptoms that describe experiences that are specific to schizophrenia to be linked with symptoms that are common to the human condition such as anxiety and depression. Since the focus of this thesis is primarily on the subjective experience of psychosis, it is likely that such a model may exist in relation to the "internal state" of the experience in addition to what is observed by "expert

[4] Note: Figure from Principal components and further possibilities with the PANSS by L. von Knorring, and E. Lindström, 1995, Acta *Psychiatrica Scandinavica, 91(suppl 388)*, p. 7.

raters". It is also possible with the pyramidal model to include asociality or poor social relatedness as posited by Peralta et al. (1994) as an axis between negative symptoms and poor cognitive performance. While negative symptoms in their essence describe the affective states of social withdrawal, they do not often describe the active avoidance of social interaction as a "motivating state". In other words, it is possible that subjects with psychosis may be motivated to withdraw from social interaction as much as display poor social interaction skills. The inability to socially relate to others may be due to a poorer capacity to process complex information necessary for meaningful social interaction.

Summary of research in the construct of psychosis

The purpose of this chapter was two-fold. First, to provide the reader with a historical development to date of the empirical research into the construct of psychosis, most notably schizophrenia. The second was to derive a model of psychosis for the research in this thesis based upon the current models. Each will be discussed in turn.

It was Kraepelin (1899) at the turn of the 19th Century that argued the notion of dementia praecox (premature dementia) as a recognised disease process. Through the influences of both Morel's *Clinical Studies* of 1852 and through a series of his own clinical observations, Kraepelin proposed a disease which characterised a progressively deteriorating mental state leading to a poor outcome of social withdrawal and isolation (Barrett, 1998a). In modern nosology, Kraepelin's dementia praecox describes a condition consisting fundamentally of negative symptoms with abnormalities in thinking, emotion and volition of will. The more florid symptoms,

now described as part of the positive end of the spectrum, did not feature in Kraepelinean nosology. Kraepelin argued a "deficit syndrome" of poor outcome that may have reflected more the environment Kraepelin was observing his subjects rather than the condition itself. The pessimism that characterises Kraepelin's description of a likely irreversible state of deterioration may have reflected the overwhelming state of large asylums and university clinics of the time, as these institutions were often overburdened with chronic and protracted cases of mental illness.

By the end of Kraepelin's career in the 1920s, even he was quite prepared to question the very foundation of his work and the view that people with schizophrenia end their lives in an increasingly poorer deteriorating state of being. Despite Kraepelin's own retraction of his theories, the dementia praecox paradigm persisted in schizophrenia research (Jablensky, 1997). By the middle part of the 20th Century the Kraepelinian notion of schizophrenia was seen to be too narrow. By 1950 Bleuler tried to classify schizophrenic symptomatology into fundamental and accessory symptoms (Andreasen, 1997). In order to classify schizophrenia as a unique disorder, Bleuler attempted to divide the presentation of symptoms that are unique to schizophrenia and those symptoms that are common in other mental health conditions. The fundamental symptoms where characterised by abnormalities in cognitive processing and emotional processes (Bleuler, 1950). These symptoms still described those within the negative end of the spectrum of symptoms. Accessory to these symptoms, are those now described in the positive dimension that include delusions and hallucinations. Both Kraepelin and Bleuler had similarities in theory formation since both stressed the importance of a loss of cognitive, affective, volitional and attentional functioning as central to the description of symptoms of schizophrenia. Where their theories differed is that while Kraepelin thought that such deficits in

cognition, affect and social relations where central to the condition, Bleuler argued they were pathogonomic (Andreasen, 1997). Certainly to this present day, Bleuler's influence in clinical practice is evident where fundamental symptoms such as "the loosening of associations" (i.e., poor or incorrect attributional style) and blunted affect are central to the diagnosis and screening of schizophrenia.

By the 1960s and 1970s the theories of schizophrenia by both Bleuler and Kraepelin started to have their own inherent limitations. With improving clinical services in the Western world, the symptoms, which were said to describe the condition of schizophrenia, could also be evident in a myriad of other conditions. This became problematic for research in psychosis, since other disorders could express anhedonic and avolitional states; most notably within depressive disorders. It appears that because symptoms such as hallucinations and delusions provided such distinct phenomenological boundaries, that these florid symptoms were steadily given greater prominence in the defining of schizophrenia (Andreasen, 1997). Schneider reinvestigated positive symptoms as cardinal to the classification of schizophrenia since these symptoms were seen as 'nonunderstandable' and hence more particular to schizophrenic experience than other mental health conditions. The Schneiderian first rank symptoms describe a loss of boundary between self and non-self including a lack of personal autonomy, and were argued as being descriptive of schizophrenia. Symptoms that describe a loss of autonomy, or insertions of other's thoughts or delusions of being controlled by outside forces, were characteristic of first-rank symptoms. According to Schneider's nosology, the presence of one or more first-rank symptoms in the absence of any organic disease is indicative of schizophrenia (Sims, 1997). The emphasis with the diagnostic technique was that a condition could be recognised if it occurred in reasonable frequency, and other conditions could be ruled

out. The significance of Schneider's first rank symptoms was to rule out any other possible explanation of a person's presenting state. This nosological process appears more to find an anchor point in the ever-changing phenomenological landscape of schizophrenia (Andreasen, 1997). In other words, although a shift away from negative symptoms to positive symptoms as the cardinal description of schizophrenia was meant to improve diagnostic accuracy, it also restrained the construct of schizophrenia to an artificial and statistically derived framework (Andreasen, 1997). The contribution of Schneider's first rank symptoms to the construct of schizophrenia was to set up a debate on the importance of both positive and negative symptoms of psychosis. It is not so surprising that the concept of the positive and negative symptom dichotomy by Crow (1980) sought to bring the two "waring" factions together.

Crow argued that although the Schneidarian notions of schizophrenia may have contributed to increased diagnostic accuracy of schizophrenia it was limited in predicting outcome or the course of the illness. Crow sought to synthesise the Kraepelinian notions of the outcome of schizophrenia with the Schneidarian notions of definable symptoms particular or peculiar to schizophrenia. In other words the Type I and Type II model of schizophrenia was not only in terms of the difference in the presentation of schizophrenia but also the likely outcome of the presentation type. Type I was seen as acute schizophrenia where the condition was likely to be reversible and responsive to neuroleptic medication and hence a good prognostic outcome. On the other hand, Type II is characterised by a more chronic condition not as receptive to neuroleptic medication and therefore more enduring as a presentation. It was questionable whether the condition in Type II is reversible. This amalgamation of presentation and outcome set off a flurry of research into the positive and negative

dichotomy that has not noticeably abated to the present day. The positive and negative view of schizophrenia was not new to the literature and all the theorists previously mentioned described, to some extent, the existence of these two syndromes. What was unique in Crow's position was to link the symptom dimensions to the pathophysiology of the brain. It was noted by Crow that neuroleptic medication which blocked dopamine receptors in the brain appear to be limited to positive symptoms characteristic of acute schizophrenia. The more chronic condition where negative symptoms were more predominate, particularly in institutionalised patients is more likely to be less receptive to drug treatment (Crow, 1980). It became increasingly understood in the literature, that to understand the effect of neuroleptic medication on the treatment of schizophrenia, that the research community needed to gain a better understanding of the dimensionality of schizophrenia and how medication can effect these different syndromes.

Up to this point in the development of schizophrenia as a construct, most of the theorising was based upon clinical observation and case studies. By the mid 1980s, more emphasis was placed on deriving empirically validated notions of schizophrenia and relying more heavily on the empirical evidence as a way of progressing this construct.

The development of improved instrumentation specific to the phenomenon of schizophrenia and other psychosis became the focus of new interest in the modelling of this condition. The positive and negative syndrome scale (PANSS) (Kay et al., 1987), and the schedule for the assessment of positive symptoms (SAPS) (Andreasen, 1984a) and negative symptoms (SANS) (Andreasen, 1984b) became the mainstay of empirical measurement of schizophrenia and related conditions (see Table 2.5).

Early studies in the mid to late 1980s and into the early 1990s were characterised by small sample sizes using exploratory factor analysis. Even in this early period of investigation the positive and negative dichotomy was under question as an accurate representation of the syndromes of schizophrenia. Studies by Bilder et al., (1985); Kay et al. (1986b); Levine et al. (1983); and Phillips et al. (1991) demonstrated an emerging third dimension, which described a disorganisation factor. This factor was symptomatic of poor cognitive performance that often described symptoms of tangentiality, disorganisation and poverty of thought. While these were often classified under negative symptoms, the literature started to recognise that cognitive deterioration is central to the condition of schizophrenia and other psychotic conditions and may be separate to the other negative symptoms.

It was difficult for the research in the area to develop any substantial formulations as to the dimensionality of schizophrenia, due to the small power and exploratory techniques of these studies. By the mid 1990's multi-centre studies with larger samples sizes via the use of confirmatory factor analytic techniques provided the view that the dimensionality of schizophrenia was more complex than previously purported by the positive – negative dichotomy. Studies were conducted both across the positive negative divide (Brekke et al., 1994; Cuesta & Peralta, 1995; Peralta et al., 1994), and within positive symptoms (Peralta & Cuesta, 1998) and negative symptoms specifically (Peralta & Cuesta, 1995). Theorists were postulating not so surprisingly more complex models whether they looked specifically within a certain symptom dimension or across symptoms. Despite these investigations most of the studies showed poor fit to the data, thereby hinting at a difficult and complex structure that may be dependent on the scale and analytical method used as well as age, course of illness and medication effects of subjects.

The first major study that had a large sample size (N = 1 233) that tried to address these criticisms was that of White and colleagues in 1997. Using confirmatory factor analysis of the PANSS they tested 20 different models previously presented in the research literature. A finding of concern was that none of these previous 20 models tested from a unitary one-factor models to more complex 5-factor models could fit the data. By using re-specification techniques informed by the large data set, a 5-factor pentagonal model was proposed that fits the data within acceptable limits. This included positive symptoms, negative symptoms, dysphoric mood, activation and autistic preoccupation (White et al., 1997). Other studies such as Ehman et al. (2001); Lançon et al. (1998); and Long & Brekke (1999), found 5-factor theories with similar factor descriptions. What became more apparent in the literature was that the simple 2 or 3-factor models were inadequate in describing the dimensionality of schizophrenia. More importantly the emergence of symptomatalogical states that are evident in other mental health conditions were becoming increasing accepted as descriptors of psychosis. Most notably, conditions of depression (Lançon et al., 1998; 2000) and anxiety (Ehman et al., 2001) were seen as legitimate descriptions of the conditions of schizophrenia. In addition, relational states (Peralta et al., 1994) and active social avoidance were seen as other components of schizophrenia.

Finally the 5-factor pyramidal model of schizophrenia forwarded by White at al. (1997) and von Knorring & Lindström (1995) suggested an interactive dimensional model, where both symptoms specific to the schizophrenia condition (i.e., positive and negative symptoms) and shared by other conditions (i.e., depression, anxiety) appears the most likely dimensionality that underpins the psychotic experience. This is argued because it is unlikely that specific symptoms do not exist or are independent

to general symptoms that can be expressed in a number of mental health conditions. A synthesis of these two qualities appears to have emerged in the last ten years with the advent of more sophisticated models of schizophrenia and larger data sets to explore the dimensionality of different models.

Towards a model of the psychotic experience

It is with this integration of specific and general symptoms in mind that this current research will draw its modelling procedure. As stated before, all modelling procedures are largely dependent upon the type of instrumentation used and the focus of attention. Since this research focuses on psychotic experience from the perspective of the subjective as opposed to the objective experience, it is important for the reader to bear in mind that the emphasis on instrumentation is somewhat different to those investigated by the PANSS and similar instruments. Since the emphasis of the study is on the internal experience rather than the observational signs of psychosis, it is difficult to argue that the dimensions under investigation are in fact strictly symptoms. More accurately through the modelling procedure of structural equation modelling, latent factors will be explored which are more akin to psychological states or conditions. This moves the research away from classical medical paradigms of observational symptoms and into psychological processes and internal states. Since the psychological dimensions investigated in the study can be manifest in a variety of psychological conditions, it would be inaccurate to create a nomenclature of symptoms. This is because in the classical sense, symptoms are indicative of a particular condition, syndrome or illness. The emphasis in this study is the interplay between psychological dimensions in a particular cohort understood to have psychotic

experience. With this in mind the factors described in the modelling procedure will be referred to as dimensions or constructs, and items which indicate these dimensions as manifest variables.

As previously stated, it is possible that the subjective experience of psychosis may not possess the subtlety of description that observational instruments may measure. Differences in convergent and divergent discriminate validity may lie on the definitional differences between scientific and experiential notions of psychosis (Preston & Harrison, 2003). Since no instrument has been devised to test the divergence or convergence of psychosis between the two perspectives (e.g., observational versus experiential), the modelling procedure is proximal by nature. That is, the model proposed for this research will approximate previous models since there is no direct evidence within the literature that they are in fact measuring the same thing.

What can be put forward, however, is a self reported model that is likely to describe 5 major factors. The first is a factor that measures positive symptoms of psychotic experience. These symptoms could include delusional states such as paranoia or hallucinations. The second factor will describe some form of negative symptoms that are likely to be akin to the descriptions of depression such as low mood and poor motivation. A third dimension and what is increasingly being explored in the literature are notions of cognitive performance. These include thinking clearly and making correct associations. Since these are often linked to negative symptoms they are likely to be correlated with the depression factor. A fourth dimension will describe some excited or arousal state. From a self-reported point of view this is likely to be described as states of anxiety, such an internal agitation and startle responses. Related to this state will also be some concern for bodily health and well-

being often denoted as psychosomatic or somatic concern. The fifth dimension is likely something to do with asociality or social avoidance similar to the dimension described by Peralta et al. (1994). Such psychological states would include active social avoidance and withdrawal from meaningful social interaction.

The following chapters will describe a methodology by which these factors will be derived, tested and cross-validated. In addition, linkages will be made between these dimensions to examine their causal influences over time within the early stages of a psychotic condition. Finally, some interpretations of the findings will be presented in light of current theoretical formulations on aetiology and cause of psychosis, along with further directions on how to use this modelling technique to contribute to the knowledge of schizophrenia and other psychoses.

CHAPTER III

METHODOLOGY

In the previous chapter, a review of how psychosis is conceptualised and understood as a construct was presented. Key developments in theory construction, disagreement in construct development and discussion points were reviewed. The movement away from a more simplified two to three factor theory of schizophrenia into 5 and 6-factor models, where cognitive and relational states are expressed in the model, are gaining widespread acceptance in the literature. In this chapter, these models will be used to inform the process by which the data will be analysed. The samples, procedures, measures, analyses and statistical applications used to examine the dimensions and causal influences of symptoms over time will be described. The use of self reported data, exploratory factor analysis, confirmatory factor analysis and longitudinal causal modelling are the key elements of the research methodology.

Samples

Sample 1 Validation sample: EPPIC Study Melbourne Victoria

The Early Psychosis Prevention and Intervention Centre (EPPIC) served as the primary validation sample from which the constructs for the measurement of psychosis were derived. Given that the EPPIC sample had a larger number of subjects measured at baseline and followed up consistently over one year, this sample comprises the bulk of the analyses for this thesis. In addition, the patient characteristics of the EPPIC sample provided a greater representation of patients with

schizophrenia with this diagnosis therefore making up the bulk of studies in early psychosis.

The EPPIC program commenced operation in October of 1992. Its purpose is to provide " ...a comprehensive community-based service to adolescents and young adults experiencing the first onset of a psychotic illness and to provide ongoing care through the critical period" (McGorry, Edwards, Mihalopoulos, Harrigan, & Jackson, 1996, p. 309). This critical time period is thought to occur in the first two years of expressing a psychotic illness. EPPIC serves a catchment area of 800, 000 people covering the west metropolitan region of Melbourne, Victoria. This region has two public psychiatric hospitals and five community mental health centres (McGorry et al., 1996).

EPPIC operates on an outpatient case management system with full-time case managers carrying an individual case load of up to 40 patients. The age range for the service is between 16 to 30 years with a primary focus on young adolescents. Cases up to the age of 45 have also been accepted into EPPIC. Each subject used in the study signed a consent form. The consent form along with a letter authorising the release and use of the EPPIC data for the purposes of this doctoral thesis are found in Appendix I.

Sample 2 Cross validation sample: EPOES Study Perth Western Australia

Throughout 1995 to 2000 a number of metropolitan mental health services within Perth Western Australia developed specialised early psychosis intervention programs. Each of these programs was set up under the principles of the first national recommendations for establishing early psychosis intervention services (Whiteford,

1996). These principles included using a case management model with low patient to case manager ratios between 1 to 10 and 1 to 15. The interventions would operate primarily under a biopsychosocial model with the implementation of low dose atypical antipsychotic medication, with supported psychosocial intervention under a case management model. The basic principles that underpin the national guidelines, are expressed in the following table.

Table 3.1
Principles that Underlie the Australian Clinical Guidelines for Early Psychosis

Principles
▪ Identifying, monitoring and providing needs-based care during a potential prodromal phase in early psychosis are optimal.
▪ Mental Health Services are accessible and provide a timely assessment for people experiencing or significantly at risk of their first episode of psychosis and their families.
▪ Consumers and their carers receive a comprehensive, timely and accurate assessment and a regular review of progress.
▪ A case manager and treating psychiatrist should be allocated to each client upon entry to the service, and provide a range of services to meet the needs of the client and their family and carers.
▪ Psychopharmacological interventions are to be provided during the acute phase and ongoing management of recovery from psychosis.
▪ Psychological interventions are provided as part of the acute phase and ongoing management of recovery from psychosis.
▪ Family and Carers are involved in the assessment, treatment and recovery process in episodes of acute psychosis.
▪ Psychoeducation for clients and families is an essential component of the treatment process in early psychosis.
▪ A comprehensive range of group programs specifically tailored to the needs of people with early psychosis should be available.
▪ Clients will receive treatment in the least restrictive manner wherever possible.

The service sites recruited for the study included the Fremantle Hospital and Health Service Early Psychosis Program, Bentley Mental Health Service First Psychosis Liaison Unit, Joondalup Mental Health Service First Episode Psychosis Program, and the Rockingham Kwinana Mental Health Service Early Psychosis System of Care program. These four programs are located throughout the Perth metropolitan region with the Joondalup catchment area in the north, Rockingham

Kwinana in the south, and the Bentley catchment area in the east. The Fremantle catchment area could be considered a central metropolitan location. The total population of these catchment areas is 700, 000, which is comparable to the EPPIC catchment area in Melbourne, Victoria.

Since 1996, these four service sites have been meeting monthly under the auspices of the Early Psychosis Group (EPG). The EPG was set up primarily to inform the Department of Health Western Australia of the progress of implementing early psychosis intervention programs within the metropolitan health service of Perth. With this advice came the undertaking of evaluating treatment outcomes under these guidelines. This development led to the introduction of Early Psychosis Outcome Evaluation System (EPOES) study. EPOES is co-ordinated by the EPG and contributes to the bulk of the cross-validation data used in this study. In addition to the EPOES data, data was extracted from an initial early psychosis outcome study undertaken by the Bentley Mental Health Service between 1995 and 1998.

Sample Characteristics

Comparisons were made between the validation and cross validation sample on age, age at onset of psychosis, gender and DSM-III-R (APA, 1987) diagnosis. This examination was made to establish the homogeneity or heterogeneity of the two samples. Since both samples use different times, in which baseline measures were conducted, it is anticipated that the cross-validation group would have more schizophreniform disorder because the data collection was performed earlier on in the course of the illness than the validation sample. In addition, the validation sample has a younger age range for inclusion in the study (16 - 30), while the cross-validation

sample operate within an adult mental health service delivery model, where patients from 18 onwards are accepted for treatment. Patients younger than 18 in the Perth mental health clinics are directed to the child and adolescent mental health services.

Recruitment of subjects for both the validation and cross-validation samples operate under a naturalistic cohort design. In other words, subjects were not randomly assigned to different treatment modalities but were asked to participate in research as they were receiving treatment within specialised early psychosis programs. The emphasis for each sample was to monitor the progress of subjects on a range of clinical outcomes over time. Although age range, catchment population, and case load ratios differ between the two samples, both samples strive to operate under the national first episode psychosis treatment guidelines (Whiteford, 1996) and as such are comparable in terms of treatment focus and delivery models.

Procedures

Sample 1 Validation Sample: EPPIC Study Melbourne Victoria

Subjects were administered a battery of tests using the Royal Park Multi-Diagnostic Instrument for Psychosis (RPMIP). This instrument assesses prodromal symptoms in first-episode patients with demonstrable reliability and validity (McGorry et al., 1990) and was assessed at entry into the EPPIC service. After this initial assessment, subjects were assessed at the *stabilisation period* usually 6-8 weeks following the initial assessment (Edwards, McGorry, Waddell, & Harrigan, 1999). At this time, a battery of tests including the Brief Psychiatric Rating Scale (BPRS) (Overall & Gorham, 1988), Scale for the Assessment of Negative Symptoms (SANS) (Andreasen, 1984b), Beck's Depression Inventory (BDI) (Beck & Beck, 1972) and

the Symptom Checklist 90 (SCL-90) (Derogatis, 1993) were administered. These tests were subsequently re-administered on two more occasions every 6 months from the initial stabilisation period (Edwards et al., 1999). Subjects in the study consented to the testing procedures before assessments were made.

The present study only examines data from the SCL-90 and additional demographic data obtained from the RPMIP which include age, age of onset, DSM-III-R diagnosis and gender. Each of the subsequent waves of the 6 and 12-month follow-up periods is used in the longitudinal data. For comparisons with the cross-validation sample the SCL-90 items were converted into the Brief Symptom Inventory 53 (BSI) (Derogatis, 1982) which is a shorter form of the SCL-90. Item scaling and construct definitions of the two instruments remain unchanged.

All participants in both the validation and cross validation samples had to have a recognised psychotic diagnosis under the DSM-III-R (APA, 1987) marked by evidence of delusions, hallucinations, marked formal thought disorder, or grossly disorganised, or bizarre behaviour (Edwards et al., 1999). Exclusion from the samples included organic mental disorders, mental retardation, epilepsy and inadequate command of English.

Sample 2 Cross validation sample: EPOES Study Perth Western Australia

All data used in the cross-validation group consists of BSI data administered within 4 weeks of being formally accepted into any of the four early psychosis intervention services. In addition to the BSI data, demographic details including age, age of onset, DSM-III-R diagnosis, and gender were collected. Additional data is collected within the EPOES study but were not used in this thesis. The reader is

directed to Preston et al., (2003) for details of the data collection strategy for the EPOES study see Appendix IV. To summarise the data collection procedure, patients who were referred to the respective mental health services were assessed for their eligibility to receive treatment for early psychosis. This included being within the age range of 18-40, and having a recognised psychotic mental health disorder under the diagnostic guidelines of the DSM-III-R (APA, 1987). Within 4 weeks of being assigned to a case manager, patients were invited to participate in the EPOES study by participating in a semi-structured interview measuring psychopathology using the Brief Psychiatric Rating Scale (BPRS) (Overall & Gorham, 1988; Rhoades & Overall, 1988). Prior to the interview to reduce the effects of the interview influencing patient's self reported ratings, patients completed the BSI (Derogatis, 1982), and the Social Functioning Scale (Birchwood, Smith, Cochrane, Wetton, & Copestake, 1990). This data along with an interview measuring onset of illness and prodromal features and substance use was collated into EPOES. All service sites received ethics approval to conduct EPOES and all patients provided written consent to participate in the study (see Appendix II for Consent Form). This was signed only after a standardised information form was provided to the patient and the procedures and purpose of the study explained by the case manager.

The majority of assessments were performed in the clinic. Where appropriate assessments were made in the community during patient visits.

Measures

Psychometric properties of the Brief Symptom Inventory

To meet the assumptions of independence of measurement in factor analysis and causal modelling, the self reported measure of the Brief Symptom Inventory (BSI), as opposed to the observational measure of the BPRS, was used as the measure of psychopathology. Although the BPRS is specifically designed to measure psychotic phenomena, the instrument is subject to the observer effect where an observer can make many observations. This is called a nested effect where the phenomenon is filtered through the influence of one observer. This would mean that the autocorrelating effects of one assessor to many cases would have to be taken into account in the confirmatory factor analysis. Since the BPRS data was anonymised in both samples it was impossible to account for the nested effects within the data. In addition to these psychometric limitations, the thesis is concerned with the experience or internal state from the subject themselves. Any factor analytic research using observational instruments will always have some difficulty in substantiating whether the underlying constructs exist within the data as a natural co-occuring phenomena, or a social construction due to training of a given phenomenological event (Preston & Harrison, 2003). This is because training observers to see phenomena a certain way already imposes a model on what may not be naturally co-occuring phenomena.

Previous studies have reported that self reported instruments can have high concordance with observational instruments particularly with positive symptoms (Dixon & King, 1995b; Hamera, Schneider, Potocky, & Casebeer, 1996). The BSI allows for assessment not just specifically to psychosis, but other symptom dimensions that may surround the psychotic experience including anxiety, depression, somatisation, and social isolation.

The Brief Symptom Inventory is a well-known measure of general psychopathology and has had extensive validation (Broday & Mason, 1991; Francis,

Rajan, & Turner, 1990; Johnson, Murphy, & Diamond, 1996; Royse & Drude, 1984; Wood, 1987). The instrument has 53 symptoms which ask participants to rate on a five point scale from 0 *not at all* to 5 *extremely* on how they have "been distressed or bothered ... during the past 7 days including today" (Derogatis, 1982). The BSI is a shorter from of the Symptom Checklist – 90 (SCL-90) (Derogatis, 1993). As the originator of the instrument states, the BSI was " ...designed to reflect the psychological symptom patterns of psychiatric and medical patients as well as community nonpatient respondents" (Derogatis, 1982, p. 3). The BSI is scored and profiled across nine primary symptom dimensions and three global indices. These dimensions are somatisation, obsessive compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and psychotocism (Derogatis, 1982). These symptom dimensions have evolved through a combination of both clinical and empirical validation procedures (Derogatis & Cleary, 1977). These symptom dimensions will be used along with exploratory factor analysis on the validation sample to inform the measurement model proposed.

The BSI has been used extensively on a number of patient populations. Morlan has compared the BSI with the BPRS and has reported the BSI to be a good overall measure of psychopathology when compared to an observational measure. The BSI has been used on patients with schizophrenia and has found to be correlated with other measures of social functioning (Patterson et al., 1997). Other populations including patients with substance use (Royse & Drude, 1984), outpatient mental health patients (Wood, 1987) and those suffering from grief and loss (Johnson et al., 1996), have found the BSI to be a reliable and valid measure of psychopathology with good discriminant validity.

The BSI has reported good internal consistency amongst the 9 symptom dimensions ranging from .78 to .83 (Croog, Kong, Levine, Weir, & Baume, 1990), and acceptable levels of test-retest reliability with coefficients from .68 to .90 (Derogatis, 1982).

In addition to the psychometric properties of the BSI and extensive use in psychiatric populations, the symptom constructs of the BSI are able to describe similar structures to observational instruments like the PANSS and BPRS that are used specifically for psychotic subjects (Lançon et al., 1998; Preston & Harrison, 2003). As well as the ease of administration (i.e., 10 to 15 minutes to complete) and outpatient norming available, the BSI was justified as a good measure of psychopathology both for the validation and cross validation sample.

Statistical Programs

All analyses were conducted using SPSS Version 11.00 (SPSS, 2000) or EQS Version 5.7b (Bentler, 1995; Bentler & Wu 1995) SPSS was used to explore the data for both descriptive statistics and exploratory factor analysis (EFA). Although exploratory factor analysis has been traditionally the main method for examining underlying constructs within the social sciences increasingly, through the seminal work of Jöreskog and Sörbom in the 1970's and 1980's (Jöreskog, 1971a; 1971b; 1982; Jöreskog & Sörbom, 1979), confirmatory factor analysis (CFA) and structural equation modelling (SEM) have advanced to be the preferred methodology for developing scales and testing models. With the improvement in computer technology over the past 10 years, the use of CFA and SEM have gained in popularity in disciplines such as organisational and industrial psychology (Stone-Romero, Weaver,

& Glenar, 1995), addiction research (Newcomb & Bentler, 1986) and education (Byrne, 1991).

Despite some notable exceptions in the psychosis research (Cuesta & Peralta, 1995; Peralta et al., 1994), little has been done if at all in the way of using CFA and SEM on self reported data on psychotic experience. To date, no research has been performed on longitudinal research linking the symptoms clusters over time to examine causal interplays between the symptom clusters. Anderson and Gerbing (1988) attributed increased use of SEM in psychology and the social sciences to the potential it provides for the development, assessment and modification of theoretical models. It is for these reasons that CFA and SEM form the basis of the statistical methodology for the thesis in order to examine the underlying dimensional structure of psychosis, and how this structure cross-influences each other over the initial course of the illness.

It is important however to distinguish between the exploratory and confirmatory approaches to factor analysis. With EFA, often there is no *a priori* specification of the number of underlying constructs that may specify a given illness. Neither is it possible in EFA to be able to specify the factors on which indicators load. The difference in CFA is that items can be specified *a priori* to load on a given construct, where the researcher can predetermine where the items load and how many factors describe a given phenomenon (i.e., illness). CFA operates under a stricter positivist scientific paradigm where statements of truth are specified before the experiment is executed (Chalmers, 1976), and hence are more theory driven. Exploratory factor analysis requires the *post hoc* interpretation of item covariance and therefore is always at the mercy of interpretation after the data is analysed. In addition to this, CFA accommodates modelling of and correction for measurement

error which enables for greater accuracy in the estimation of model parameters (Saris, 1989).

Exploratory factor analysis is still a useful procedure in the assessment, development and modification of theoretical models. Gerbing & Hamilton (1996) argued that EFA is a legitimate procedure preceding CFA, because it enables the researcher to investigate how well items load onto alternative factors to those specified in a confirmatory factor analytic model. This provides information to the researcher on how items behave outside of their intended loadings (Hurley et al., 1997). In other words, the use of EFA can be seen as an " ...ordered progression" from CFA, by building theoretical models and examining how item loadings behave without constriction (Anderson & Gerbing, 1988 p. 412). As a practical application however, most CFA is both a confirmatory and exploratory procedure where the final model is often derived by re-specifying item loadings to achieve better model fit.

The stringency of CFA paradoxically has its own limitations. It is difficult to expect items to have a zero loading on other factors and expect this to reasonably occur in any given population. A good fit to the data may be too much to expect from " ...real scales" since this may be too much to ask from psychometric instruments in the first place (Hurley et al. 1997, p. 672). In other words, in an attempt to find statistical purity in a measurement model, generalisability of the model is inadvertently lost to other similar populations of interest.

Based upon these considerations, EFA will be used as a way of examining the relationship between items that make up the BSI as they occur in both the validation and cross validation sample. With this information in mind, the methodology will proceed within an ordered progression where certain items are specified in a confirmatory model. The EFA will be used to help define the constructs and to refine

them later in a confirmatory model. In addition to this, theoretical constructs that underlie psychosis and the constructs that approximate these models within the BSI will be considered in the measurement model. Since the BSI has been normed on psychiatric populations, the construct definitions suggested by the originator of the instrument (Derogatis, 1982) will also be considered in the model building.

As mentioned before, since the improvement in computer technology there are a number of statistical packages that are available to the researcher for CFA and SEM. Each package has its advantages and disadvantages depending upon the statistical emphasis of the developer. The most popular packages include LISREL and EQS. EQS has advantages over other confirmatory and structural equation programs including LISREL, because it accommodates violations of assumptions of multivariate non-normality - a pre-requisite for the application of maximum-likelihood (ML) procedure. Since most psychotic symptomatology is moderately to highly skewed, it is important that violations of non-normality are accommodated. Many data sets in social science are non-normal in distribution and researchers often do not account for these violations by using statistical procedures that account for these violations (Hu, Bentler, & Kano, 1992). The EQS program version 5.7b will be used since it accommodates multivariate non-normality through the maximum likelihood robust estimation procedure and produces a scaled chi-square statistic known as the Satorra-Bentler scaled χ^2 (Satorra & Bentler, 1988). Byrne (1995) argues "in contrast to LISREL, then, EQS uses an estimation method that assumes the data are multivariate normal but bases evaluation of model fit on a statistic that has been corrected to take non-normality into account" (p. 148). In addition to these considerations, EQS provides information on the multivariate skewness and kurtosis

of the entire model. This can inform the researcher on how much the model departs from normal distribution theory methods of structural modelling.

Although other researchers may suggest using asymptotic distribution free (ADF) methods as an estimation procedure to accommodate for departure in normality, Hoyle and Panter (1995) warn against this procedure since it may over estimate true model fit and lack generalisability to other samples. Given that EQS can accommodate both procedures, and that previous researchers recommend the program for its ease of use and appropriateness of its test statistics to the data (Byrne, 1995; Ullman, 1996), this package will be used for the present study.

Data Analysis

In this section, the research design in reference to the measurement and structural modelling is overviewed. The "two-step approach" to structural equation modelling is outlined (Anderson & Gerbing, 1988). In addition, the process of exploratory factor analysis informing the confirmatory factor analytic technique is also described. Issues of cross-validation between the validation and cross validation sample are also highlighted. The purpose of this section is to orientate the reader towards the systematical and analytical approaches used to identify an underlying measure of psychopathology and to test the interrelationships between the symptom dimensions longitudinally. Mediation effects will also be discussed in terms of the influence of symptoms within themselves over time.

Tests for sample characteristics

The demographic data of the validation and cross-validation samples will be examined for their sample characteristics. Chi-square analysis will be used to test for gender and diagnostic differences in proportional representation and independent t-tests will be used to examine differences in age and age of onset.

Two-step process of model building

Anderson and Gerbing (1988) argued that a two step approach is required for effective structural and causal modelling of data. The first step requires the research to propose a measurement model. The measurement model underpins the structural model by specifying the relationship between observed measures (symptoms within the BSI) to their proposed underlying constructs. These constructs once specified are also allowed to inter-correlate freely amongst themselves (Anderson & Gerbing, 1988, p. 411). The second step, which is the development of the structural model involves specifying the " ...causal relations of the constructs to one another, as posited by some theory" (Anderson & Gerbing, 1988, p. 411). Where measures and theories have not been tested previously, Anderson and Gerbing recommended that the measurement model prior to the simultaneous estimation of measurement model and structural sub-models be respecified in a separate estimation procedure. This is done in order to avoid the interaction effects between the measurement and structural model and allows for more accurate relationships to be observed (Hair, Anderson, Tatham, & Black, 1992).

The measurement model requires that each observed symptom load exclusively on its prescribed latent factor. This allows for the identifiers to unambiguously define the constructs of interest. This means that the measurement

model is unidimensional where alternative indicators have " ...only one underlying trait or construct in common .." (Anderson & Gerbing, 1988, p. 414). In other words to define a given construct two or more indicators are required to load specifically on a targeted construct and have 0 or near 0 loading on other constructs within the model. Jöreskog (1971b) referred to such unidimensional multiple-indicator measures as "congeneric" measurements. The use of multiple-indicators assists in improving construct definition, reliability and control of measurement error.

As mentioned before, the use of EFA in measurement modelling is useful since it allows the researcher to identify where symptoms (indicators) load on non specified constructs (factors). Anderson and Gerbing (1988) argued that "ideally, a researcher would want to split a sample, using one half to develop a model and the other half to validate the solution obtained from the first half" (p. 412). This procedure mimics the cross-validation procedure in the measurement model under confirmatory factor analysis. It is proposed for the present study to follow similar procedures but using the cross validation sample of the Western Australian subjects as the "split sample" instead of splitting the original Victorian validation sample. This has two advantages over the split method. First the Western Australian sample (n = 187) is larger than splitting the Victorian Sample (n = 144) and second it is essentially independent although similar in diagnostic characteristics. If similar construct patterns are found between the two independent samples then the modelling procedure will have greater external validity. With this technique, maximum likelihood extraction and oblimin rotation methods are preferred (Fabriagar, Wegener, MacCallum & Strathan, 1999).

Confirmatory factor analysis is the process by which a proposed model is tested against a data set with reference to a range of fit indices. These fit indices indicate the extent to which a proposed model fits the underlying covariance of items (indicators) in the data set. Chi-square is the most common indicator of model fit (Ullman, 1996). A small chi square relative to the degrees of freedom, in principle, suggests small disparities between actual and estimated matrices and therefore suggests a "good fit". The problem with chi-square however is that it is widely understood to be sensitive to sample size (Jöreskog & Sörbom, 1989) with large samples detecting small differences between matrices. Over the past two decades, a number of alternative incremental fit indices have been recommended (Bentler, 1990; Bollen & Long, 1993; Marsh, Balla & Mc Donald, 1988). These include the Bentler-Bonnet Normed Fit Index (NFI), Bentler-Bonnet Non-normed Fit Index (NNFI), the Comparative Fit Index (CFI) and the Robust Comparative Fit Index (RFCI). These indices compare the estimated model with a model that has no structure (a null model) to determine model fit. Point estimates greater than or equal to 0.95 suggest a good fit to the data (Hu & Bentler, 1999). The Root Means Square Error of Approximation (RMSEA) compares model discrepancy with population parameters and provides confidence intervals (rather than point estimates alone) to evaluate model fit. Narrow confidence intervals suggest greater precision in assessment of fit. As a rule, if the confidence intervals are below 0.05, the hypothesis of "not a close fit" can be rejected by which the corollary implies a close fit. When both points of the confidence intervals are above 0.05, the hypothesis of "a close fit" could be rejected. RMSEA values of 0.05 or less indicate "good fit" (Byrne, 1994b; Steiger, 1990), while values between 0.05 and 0.08 represent "moderate fit" or reasonable errors of approximation. RMSEA values above 0.10 indicate "poor fit" to the data (Browne & Cudeck, 1993).

It is important to consider that not one independent index of fit can determine adequate model fit. It is best to use a number of indices in conjunction with each other to determine whether a particular model is a good approximation to the data. With this in mind, results will be reported for the CFI, RCFI, RMSEA and the RMSEA confidence intervals as primary indices of model fit.

Although a model may be a good fit to a particular sample, it is important to assess whether this model can be cross-validated on a similar independent sample (Burke, Brief, George, Robertson, & Webster, 1989). Cole and Maxwell (1985) have argued that the evidence of construct validity in one sample does not guarantee construct validity in another group. To address the issue that modelling procedures may be idiosyncratic to the sample under investigation, structural invariance of the measurement model requires to be tested within a "multi-sample" procedure (Byrne, 1994b). The multi-sampling procedure requires one data set to be compared with another, where increasing restriction in model testing is imposed between two or more samples to identify whether the estimates are statistically invariant. This is done in a stepwise procedure.

The first requirement for determining invariance across samples involves assessing equivalence of "form" (Bollen & Long, 1993). It is understood that when two samples have the " same parameter matrices with the same dimensions and the same location of fixed, free and constrained parameters" then they are seen as structurally invariant (Bollen & Long, 1993, p. 356). Fit statistics such as the RCFI and RMSEA are assessed to determine that the same dimensions are common across samples. This is to test equivalence of form. If equivalence of form cannot be demonstrated, Bollen argued that it then "makes little sense to proceed" (Bollen & Long, 1993, p. 360). If however the form of the model is equivalent, increasingly

restricted tests are used to measure invariance between the loadings, error variances and construct covariances across the groups (Bollen & Long, 1993). The tests operate in a hierarchical fashion, with the equality of the item loadings being more important to establish than demonstrating equality between the error variances and covariances. Tests of equality between the error variances however have been argued as unduly strict (Bentler & Wu, 1995; Byrne, 1994). For the purposes of this study, invariance was determined at the level of form, loadings and construct covariances.

Base-line models are required for both the validation (Victorian) and cross validation samples (Western Australian). The next procedure is to test for invariance across both samples. This requires the loadings for each model to be equal across both samples. This model is nested within the base-line model where a new chi-square is calculated. With each loading constrained to be equal an increase in degrees of freedom also occurs (Bollen, 1989). A non-significant increase in the chi-square difference, relative to the degrees of freedom, suggests that the model is invariant across both samples (Byrne, 1994).

The next procedure is to maintain the equality constraints for the loadings, while in addition to these constraints, the covariances are also calculated to be equal across the samples. The procedure for significance testing is repeated with a non-significant increase in chi-square relative to the degrees of freedom suggests the model generalises across both data sets.

Having established invariance in the measurement model the second step, suggested by Anderson and Gerbing (1988), can proceed. This is where the hypothesised structural relations between the constructs can be examined for their associations. A number of competing structural models can be examined against competing theories to assess which fit the data the best. On the basis of the fit indices

described previously, assessments can be made to indicate which model provides the most parsimonious and theoretically plausible account of the data. It is important to note that these models can be modified on the basis of the loadings, standardised residuals and modification indices (Hair et al., 1992). However these modifications should only be made where sound theoretical reasons are provided for doing so.

Examining mediation effects

When more than one time wave is examined in a structural model, it is important to examine the mediation effects of constructs over time. This occurs when the structural relations between constructs (i.e., symptoms) may contain both direct and indirect effect. With the examination of symptoms this is an important consideration. It is important to assess whether the extent of a given symptom may have mediating effects across time and not only within time. For example, it is possible that the extent of depression at Time 1 may influence the extent of depression at Time 3. Such a result is indicative of a "snowballing" effect where the influence of symptomatology may extend beyond the time point previously measured. A mediating effect can be described as an effect where the " ...influence of an antecedent is transmitted to a consequence through an intervening mediator (James & Brett, 1984, p. 307). In reference to this study, mediation effects are considered within symptoms rather than across symptoms. In order to reduce structural complexity and examine the unique influence of symptoms within themselves over time, the mediation effects are examined in relation to the influence of Time 1 on Time 3 in the wave series. This can be expressed diagrammatically in the following

figure. The mediation effect occurs when the pathway leading from Time 1 to Time 3 is calculated to examine its mediating influence.

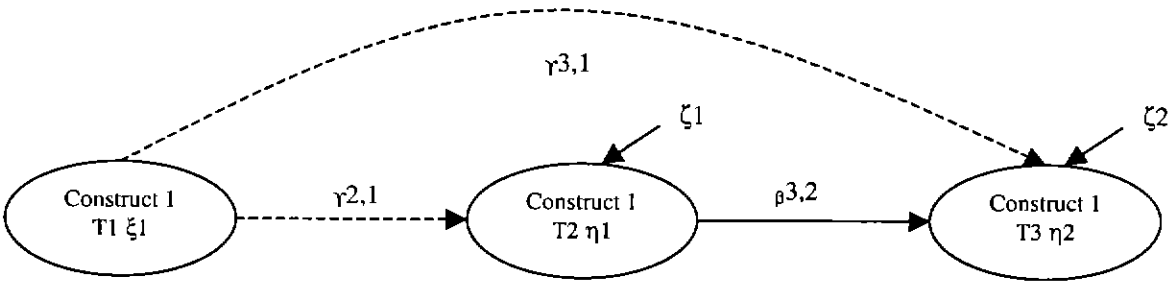

Figure 3.1 Mediation model of one latent construct over time.

Note: measured indicators, error terms, and disturbance terms not shown for ease of interpretation. T1 = Time 1, baseline: T2 = Time 2, 6 months from baseline: T3 = Time 3, 12 months from baseline.

Mediation effects are examined in a process recommended by Bollen (1989), where inclusion of the parameter in the mediation effect results in an increase in the degrees of freedom. Where there is no statistical difference in chi-square between the models (relative to the difference in degrees of freedom), the more parsimonious of the competing models is preferred. A more parsimonious model is one with superior fit and less parameter estimates to assist in model design. Where there is a statistical difference in the chi-square, the better fitting model is preferred. In relation to this study a mediation effect would indicate presence of an effect at Time 3 ($\beta_{3,2}$) as a consequence of influences at Time 2, but not at Time 1 ($\gamma_{3,1}$). This is demonstrated in Figure 3.1 with the full coefficient line running from Time 2 to Time 3. This is an important consideration since these mediation effects need to be taken into account when structural influence is examined between dimensions over time.

Causal modelling and cross-lagged effects

Once the measurement model is cross validated and the time series data are examined for mediation effects across time, the next step is to examine the relationships between dimensions over time. Apart from the mediation effects, the previous procedures concern cross sectional data from two samples at the initial stages of a psychotic illness. Glomb, Munson, Hulin, Bergman, and Drasgow (1999) argued that longitudinal research is "indisputably important in examining causal relationships, yet few researchers answer the perpetual call for such research in psychology" (p. 14). Longitudinal designs have advantages over cross-sectional designs. First, using the same subject over time controls for individual differences. Second causal relations among variables can be established.

Willett (1988) suggested that three data points are required to fully determine the patterns of relationships among the variables over time. It is with three data points the researcher can examine the reciprocal causation among variables (Padhazur & Pedhazur-Schmelkin, 1991), and the reason that the EPPIC data with substantial 3 wave data (n = 145) is used in the study. Although a great deal of research has been done to examine the underlying structure of psychotic illness (see Table 2.5 for summary of confirmatory factor analysis on schizophrenia instruments), few if any studies have been conducted to examine the causal linkages of the symptoms over time (Long & Brekke, 1999).

Due to the complexity of the illness and suggested four to five factor structures that underlie the illness, it is difficult with current technologies and sample sizes to examine all causal influences simultaneously. Preliminary analysis examining the cross influential effects of all symptoms within the model over time would require over 800 cases in each time wave, and computing memory larger than

those available to the researcher (Bentler, 2002). To accommodate this limitation (which is present in any time series research conducted on a low prevalence disorder such as psychosis), it was decided to pair the symptoms in a cross influential causal model. Instead of pairing all combinations of symptoms and increasing chance interaction effects, the research focused on the cross influential effects between cognition and all other symptoms. This was done to examine the influence of cognitive deterioration, evidenced as cardinal to schizophrenia and other psychotic disorders (Analysis, Lysaker, Bell, Bryson, & Kaplan, 1998; Chadwick, Birchwood, & Trower, 1997; Cutting & Murphy, 1988; Frith, 1992; Hoffman & McGlashan, 1993; Moritz et al., 2001; Perris, 1989; Rapoport et al., 1999; Ulug, 2002), and how this may be causally influenced by other symptoms, and in turn, how cognition may causally influence other symptoms in a reciprocal relationship. This reciprocal causal relationship is expressed diagrammatically in Figure 3.2.

In this example, the exogenous constructs of cognition and paranoia covary at Time 1, with the cross influential parameters leading from each time point in the wave. Although, not represented in the diagram, the errors from each indicator in the time series are also allowed to covary to take into consideration indication-specific variation (Ecob, 1987). To examine the unique influence of each cross influential pathway, one parameter is free to be calculated releasing one degree of freedom, followed by fixing of the same parameters. The difference between the nested models is then determined. A statistically significant increase in the chi-square is indicative of a cross influential effect. For example, in Figure 3.2, the parameter from Construct 2 at Time 1 can be examined for its cross influence on Construct 1 at Time 2 ($\gamma_{1,2}$). Alternatively the influence of Construct 1 at Time 2 on Construct 2 at Time 3 ($\beta_{4,1}$) can be examined over time. These coefficients are marked in full lines. Examination

of the standardised solutions in EQS indicates the strength of this relationship (Byrne, 1994).

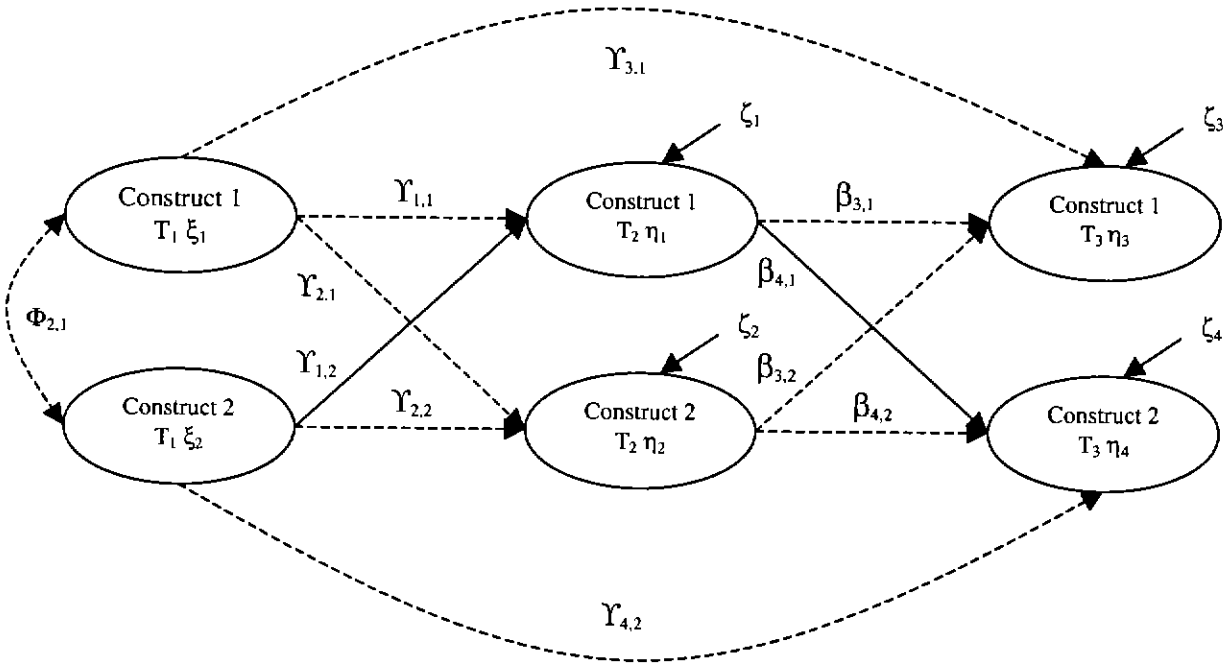

Figure 3.2 Model 1: Full cross-lagged model between two latent constructs over time.

Note: measured indicators, error terms, and disturbance terms not shown for ease of interpretation. T1 = Time 1 baseline, T2 = Time 2 6 months from baseline, T3 = Time 3 12 months from baseline.

Alpha, beta and gamma change in longitudinal analysis

When examining latent constructs over time, it is important to examine whether the constructs hold the same meaning amongst respondents. Before claims of causality are made the research must investigate whether the construct definitions remain in an alpha state, or change to a beta or gamma state (Golembiewski, Billingsley, & Yeager, 1976; Thomas, Cunningham-Snell, & Anderson, 1988). If respondents' interpretation of symptoms (the construct) remain substantially the same over time, changes in mean responses are considered an "alpha change". If respondents recalibrate the measurement scale, for example by re-evaluating their

psychotic illness relative to other sufferers, then "beta change" has occurred. However, if for example, the respondent reconceptualises the underlying meaning of a given construct through self-examination or through exposure to relevant literature, then the construct has undergone a definitional change. This change is known as "gamma change" The difficultly in measuring causality is that the construct must remain in an alpha state for "true" causal inferences to be made. In other words, the extent of the symptomatology can change over time for causal inferences to be made, but not when the construct is recalibrated or reconstructed. In this effect, interpretation of causal modelling becomes problematic, and statements of meaning must be given in light of these changes to the measuring instrument.

Confirmatory factor analytic techniques (Schmitt, 1982) and structural equation modelling techniques have been developed to determine alpha, beta and gamma change (Thomas et al., 1988; Vanderberg & Self, 1993). The procedure examines the invariance of the measurement model not between different samples, but over different time points within the same sample. The process is an important precursor to any form of longitudinal analysis (Lance, Vanderberg, & Self, 2000). The procedure requires using the multi-sampling procedure in EQS where Time 2, and Time 3 data are compared with Time 1 (Byrne, 1994). Following the suggestions of Bollen (1989), loadings at Time 1 are constrained to be equal with the loadings at Time 3. If the incremental fit indices are within acceptable levels it is inferred that the model suggests invariance over time. The procedure is also repeated from Time 2 to Time 3 and Time 1 to Time 3. This stage tests alpha change. Next a more restrictive test examining the paths are constrained to test for beta and gamma change. Once again if the resulting chi-square is not statistically significant it is inferred that the measurement model is invariant over time at the beta and gamma level. If by

constraining the disturbance (*psi* matrix) and construct covariances (*phi* matrix) to be equal, then the measurement model has some confidence in being structurally invariant over time (Bollen, 1989). Since the structural model examined paired cross influential paths (see Figure 3.2), each symptom was measured independently for alpha, beta and gamma change over time. In addition to this, latent mean analysis will be conducted to examine the trend of these dimensions over time.

Conclusion of analyses

The research design attempts to proceed in a logical flow with each step using both theoretical and empirical considerations. Each step will be subjected to rigorous statistical criteria. First, the construct validity of the measurement model will be considered using exploratory factor analysis, in addition to theoretical considerations to propose a model of psychosis within the limitations of the measuring instrument. This measurement model will then be validated and cross-validated on two independent samples of patients experiencing their first episodes of psychosis. Structural invariance of the measurement model will be determined against a number of indices described previously prior to examining the structural model. Tests for mediation effects for each symptom cluster will be examined prior to the causal modelling procedure. This is to examine whether certain symptoms have a "snow balling" effect in time wave data. Issues with beta and gamma change will be tested prior to the structural model, to examine whether the latent constructs that define the symptoms remain stable over time. Finally the causal models will be examined using a paired cross influential design pairing the symptom of cognition with the other symptoms. This is done to reduce chance interaction effects and to examine the

reciprocal causal influence of the state of cognition on the other symptom factors. Any cross influential effects will be interpreted in light of the strength of the results and current theories on the emergence of psychosis. All procedures will be determined against strict statistical criteria of structural equation modelling suggested in the literature.

In summary, this chapter has provided an overview of the samples, measures and procedures used to determine the causal predominance of psychotic experience in early psychosis subjects. The next chapter will describe the results of these procedures in relation to the measurement model.

CHAPTER IV

MEASUREMENT MODEL

Over the past five to ten years researchers have increasingly been interested in the underlying constructs which govern psychotic experience (Kay & Sevy, 1990; Lançon et al., 1998. Peralta et al., 1992; 1994). Researchers have mainly relied on observational instruments such as the Positive Negative Syndrome Scale (PANSS) (White et al., 1997), the Brief Psychiatric Rating Scale (BPRS) (Burger, Calsyn, Morse, Klinkenberg, & Trusty, 1997) and the Schedule for the Assessment of Positive and Negative Symptoms (SAPS) (Andreasen, 1984a) and (SANS) (Andreasen, 1984b). Many models have been proposed using both exploratory factor analysis (EFA) and confirmatory factor analysis (CFA). As previously reviewed (see Table 2.5 for summary of factor structures), most recent models identify between a four and five factor structure. Consistently the literature describes a structure that has symptoms that manifest cognitive deterioration; "positive symptoms" such as hallucinations, delusions and paranoia; negative symptoms such as anhedonia and blunted affect; social isolation, excitement or arousal states, and anxiety and depression. As mentioned before, these studies are commonly problematic, since the researcher performs CFA on observational instruments that are nested within the observer. No psychiatric research to date has accounted for the issue of independence of measurement and the nested effects of using one observer to make many observations. No research has tried to adjust for the intraclass correlating effects of nested models. In addition to this, no paper has addressed the issue that modelling pre-constructed instruments within a given professional framework such as psychiatry

will have a bearing on how a phenomenon like psychosis is observed, and consequently how it is rated.

Despite these limitations, the following research attempts to use these studies to guide the theoretical structure of psychosis in the measurement modelling procedure. Where this research differs from previous investigations is that the data is free of nested models and relies on the ratings of the sufferers themselves as a way of building the measurement model. The immediate advantage is that the research does not have to adjust for nested models from the observer effect, and allows for each observation to be independent of the next (a basic underlying assumption of any factor analysis). The disadvantage of such a technique is that the measurement of the construct relies on a person who is relatively "self aware" with cognitive faculties sufficiently intact to make sound judgements as to their symptomatology. The difficulty in measuring psychotic experience directly from the sufferers themselves is that the nature of the illness may destroy the very apparatus required for sound logical appraisal of mental state that is, the mind itself. In order to reduce the effect of "poor internal judgement", the samples used are from respondents at the early stages of a psychotic illness. This may help reduce any effects that a deteriorating course of psychosis such as in schizophrenia may reduce the ability of respondents to make sound judgments (Craig et al., 2000; Cuesta, Peralta, & Zarzuela, 2000b).

On the other hand, post-modern literature is critical of how psychosis is defined and from whose perspective (Marchall, 1995; Palmer, 2000). Construct definition particularly with observational instruments let alone self reported instruments are in themselves social constructions, with strictly defined parameters of meaning serving the scientific community and not necessarily those who "suffer from a given illness" (Gergen, 2001). Issues of insight and self awareness are hotly

debated in the literature (Amador, Strauss, Yale, & Gorman, 1991; Analysis et al., 1998; Carroll et al., 1999; Cuesta & Peralta, 1994; Pini, Cassano, Dell'Osso, & Amador, 2001; Selten, Wiersma, & van den Bosch, 2000a), and can have a major impact on both the theoretical and empirical implications of modelling psychosis. What can be said about this on an empirical point of view however, is that a deteriorating self awareness would lead to a random or error ladened response set with any self reported instrument. Such a manifest result in the data would make it impossible to plausibly test a parsimonious model of psychosis. If lack of self awareness brought on by madness is a random and error ladened event, then one could reasonably argue that it would not be possible to confirm a measurement model in any self reported data set. If however, experiences of psychosis are commonly held (within the limitations of the measuring instrument) between respondents, then the natural covariance of indicator items towards a given latent construct should be evident and thus testable. It is with this assumption in mind that the current research attempts to use previous research to inform the structure of psychosis within a four to five factor model described previously, and use self reported data to examine this model free of observational bias.

In this chapter, confirmatory factor analysis is used to establish the dimensionality of psychosis of an early psychosis cohort. Theoretical constructs defined in Chapter II will be used to guide the construction of a final measurement model in conjunction with empirical evidence from both the validation sample (Victorian EPPIC data) and the cross-validation sample (Western Australia Early Psychosis Group data). To assist the reader on the logical procedures by which the measurement model is constructed, some of the information from the previous chapter will be reiterated.

As stated previously, Anderson and Gerbing (1988) recommend a two-stage approach to construct validation. The first step in the procedure, which is the focus of this chapter, is to use confirmatory factor analysis to estimate a "measurement model". The purpose of the measurement model is to specify the relationship between indicator variables (symptoms expressed) and the underlying factor which drive the expression of a given number of common symptoms (Hair et al., 1992). Confirmatory factor analysis assesses the extent to which the specified relationships between the indicators (items describing a symptom) and the constructs (symptom clusters) are represented in a given set of data (Anderson & Gerbing, 1988). To test for construct validity, it is argued that an independent cross validation sample is used to test the structural invariance of the model (Byrne, 1994; Byrne, Shavelson, & Muthén, 1989).

Having established a viable measurement model replicable to another sample in structure and calibration, Anderson and Gerbing (1988) suggest the second step involve estimating the structural relations between the constructs of interest. In the instance of this research, time wave data is used to examine the reciprocal causal relationship between pairs of symptoms in the model.

This Chapter will focus on step one of the Anderson and Gerbing's approach, and will not discuss the structural relations between the dimensions. The focus is on the underlying experience in structural terms of psychosis, and whether this experience is generalisable across a relatively similar sample of respondents experiencing their first episode of psychosis. In order to examine this structure based on previous research some examination of each symptom cluster is required.

Dimensionality of Psychosis

As stated previously, most factor analytic research has described a four to five factor theory of psychosis (see Table 2.5 for construct definitions of factor analytic research). These factors can be described under five major dimensions. The first is positive symptomatology such as delusions, hallucinations and paranoia. The second is negative symptomatology such as blunted affect and anhedonia. The third dimension describes cognitive deterioration such as attention and memory performance. The fourth dimension describes social interaction states such as social isolation and avoidance (phobia). The final state describes states of high arousal such as anxiety and agitation, and low arousal in depression. In addition, psychosomatic concerns are also expressed by respondents who have experienced a psychotic illness.

Table 4.1
Dimensions of Psychosis Within the Literature

Dimension	Sign / State / Symptom
Positive Symptom	Hallucination Paranoia Delusions
Negative Symptoms	Blunted Affect Anhedonia
Cognitive Deterioration	Memory deficits Lack of concentration and attention Thought blocking
Social Interaction	Social isolation / avoidance Poor social skills
Arousal states	High arousal: anxiety agitation Low arousal: depression
Psychosomatic concern	Bodily complaints

Although these are highly specified expression of symptoms, it is difficult for any one measure to fully account for each dimension. The Brief Symptom Inventory (BSI) although a reliable and valid measure of psychiatric symptomatology (Broday & Mason, 1991; Derogatis, 1982; Dixon & King, 1995; Morlan & Tan, 1998; Piersma, Reaume, & Boes, 1994) it is not an instrument specifically designed for one psychiatric disorder, namely in this instance psychosis. It is important to bear in mind that items within the BSI may only approximate specific symptomatology of psychosis since the main purpose of the instrument is to measure psychopathology amongst all psychiatric conditions. In other words, in its attempt to be a general instrument of psychiatric symptomatology, it does not efficiently or specifically describe the phenomenology of a given disorder. Despite this limitation, the BSI is a better instrument to use amongst psychiatric populations because of its widespread application in the field of research compared with an unqualified instrument of unknown psychometric history. The measurement model examined in the study may not be specific to psychosis but to the cohort under investigation as one where all cases in the data set have had a recognised psychotic disorder. This means that the measurement model proposed and the structural relationships between the symptoms are only specific to psychosis, where by the population of interest are cases with psychosis. Construct definition of the measurement model as such only approximates the dimensions described in Table 4.1.

All psychiatric instruments including the BSI have their own internal dimensionality dependent on the construction of each item to express a given symptom or symptom cluster. This means that any exploratory factor analysis of the BSI is limited to how the items are constructed, and the likelihood of them being driven by a given construct from previous empirical research on the instrument. This

means that the measurement model must take into consideration the theoretical issues that surround a given phenomenon and the limitations of the instrument to measure these constructs. In terms of constructing the measurement model, the empirical evidence of the nine symptom dimensions described in the BSI (Derogatis, 1982) are also considered along with the theoretical tenants that underlie psychosis research. As stated previously, these nine dimensions include somatisation, obsessive compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and psychotocism (Derogatis, 1982). These will also be considered along with the theoretical research described above in relation to building a measurement model of psychosis. Before this is tackled however, a brief description of the current literature in each symptom dimension is warranted.

Positive and Negative Symptoms

The postulation of both positive symptoms (hallucinations, delusions and thought disorder) and negative symptoms (muteness, withdrawal, apathy and anergia) is largely an attempt to consolidate the two ends of the psychotic spectrum. This consolidation has arisen as much to resolve philosophical and nosological constructs of psychosis, as it has to achieve a "unifying theory" of symptoms and syndromes of psychosis. One of the earliest proponents of the positive-negative symptom paradigm was Hughlings-Jackson (1931), who argued that negative symptoms represented a loss of function while positive symptoms represent an exaggeration of normal functioning. Jackson describes positive functions of hallucinations and delusions as release phenomena that describe how such symptoms are often an extension of normal functioning processes. Crow (1980) postulated that there are two types of

schizophrenic syndromes with different mechanisms, aetiology and response to treatment. Type I is characterised by those with prominent positive symptoms that tend to be acute in nature, and have a high likelihood to respond to medication. The degenerative course of the illness can be reversed. Type II tends to possess prominent negative symptoms and have less likelihood of responding to medication. The course within Type II tends to become chronic with questionable improvements in long term outcomes.

Cognitive Deterioration

Recent literature has focused more intensely on the cognitive aspect of schizophrenia and other psychoses (Bilder et al., 2000; Cosway et al., 2000; Cuesta, Peralta, & Zarzuela, 2000a; Heaton et al., 2001). Some studies have identified poor cognitive processing skills (Ngan & Liddle, 2000), relationships to negative symptomatology (Penadés, Gastó, Boget, Catalán, & Salamero, 2001) and changes in brain morphology (Rapoport et al., 1999). Essentially these studies identify that the processing of information by people with psychosis is hindered as the illness progresses. This can lead to distortions in thinking such as delusions and paranoid states where external stimuli are mis-attributed as explanations of fancy (delusions) or threat (paranoia) (Bentall, Kinderman, & Kaney, 1994). In relation to a causal predominance theory, deteriorating cognitive states could lead to greater arousal or poorer arousal if the person decides to isolate himself or herself to cope with a fearful belief about the world, or to escape in ideas of fancy and delusion.

Social interaction

Most psychotic illnesses and in particular schizophrenia have a characteristically marked deterioration in relating within a social context. Often sufferers will under or over estimate social cues that interfere with effective relationships with other people (Russel et al., 2000). Social disability related to psychosis is well known in the literature, with longitudinal studies showing poor outcomes in the area of social functioning particularly among people with schizophrenia (Wiersma et al., 2000). Some earlier theorists believed that poor social learning skills were evident in the childhood development of people who later developed psychosis (Cameron, 1959). Lately, more research has been conducted on schizotypal personality traits amongst early adolescents to see whether these traits within individuals later express into a psychotic disorder (Bergman, Silverman, Harvey, Smith, & Siever, 2000; Miller et al., 2002; Reynolds, Raine, Mellingen, Venables, & Mednick, 2000). It is believed that suspicious or guarded individuals with social isolatory tendencies may have a higher risk in developing a psychotic illness (Miller et al., 2002).

High and Low Arousal States

Often psychotic disorders can be characterised with affective disregulation. This dimension is a difficult construct to define since it is very closely linked particularly to the notions of negative symptoms described earlier. Recently, research has been conducted to separate these two dimensions of depression and negative symptoms (Addington et al., 1992; 1993a; 1994; 1996; Addington, Addington, & Schissel, 1990). In reality however, these dimensions are likely to be highly

correlated and may not be distinguishable within the internal states of sufferers of psychosis (Preston & Harrison, 2003). It is difficult to examine whether states of anxiety are antecedent to psychosis, or are the outcome of other drivers in the psychotic experience such as cognitive deterioration and positive symptoms. Recent research has shown anxiety and depression to have a significant negative impact on the quality of life of people with schizophrenia (Huppert, Weiss, Lim, Pratt, & Smith, 2001). This could also be linked with some emergent theory that psychotic episodes can create post traumatic stress characterised by anxious and depressed states (Myin-Germeys, Krabberdam, Jolles, Delespaul, & van Os, 2002; Myin-Germeys, van Os, Schwartz, Stone, & Delespaul, 2001) which are linked to increasing deteriorating cognitive performance.

Psychosomatic Concern

Although not a major focus in the literature, psychosomatic concern of bodily well being has been identified as a key expression of schizophrenia. Most notably, Schneider in his postulation of "first rank symptoms of schizophrenia" included "somatic passivity", where the influence of external causes on the body were considered cardinal to signs of schizophrenia (Sims, 1997). Over concern for the health of one's body can be indicative of psychotic expression where these concerns are tied into delusional symptoms (Sims, 1997). Most instruments that measure psychotic phenomena include a line of questions about the concern of the person's bodily health (Kay et al., 1986a; Overall & Gorham, 1988). These are also included in the BSI (Derogatis, 1982).

Based upon the arguments made above, along with the review of the literature in Chapter II, a 6-factor model will be proposed. That is, the psychotic experience will be defined by symptoms that express cognitive deterioration (disorganisation), depression (negative symptoms), anxiety (arousal states), somatic concern, phobia (social avoidance) and paranoia (positive symptoms).

Consistent with previous theories on psychosis, the latent constructs examined will be considered in light of these six major constructs and the measurement model will be defined in relation to these latent constructs. The following hypotheses follow from the arguments presented above:

Hypotheses

$H_{4.1}$. A six dimensional measurement model of psychosis (consisting of cognition, depression, anxiety, somatic concern, phobia, and paranoia) will adequately summarise data drawn from the validation sample (Victorian EPPIC data).

$H_{4.2}$. The form, loadings, and construct covariances of the measurement model derived from the validation sample will generalise (be invariant) to data drawn from an independent cross-validation sample (Western Australian Early Psychosis Data).

Method

Sample Characteristics

Prior to examining Hypothesis 4.1, both the validation and cross-validation sample were examined for sample characteristics. Comparisons were made between age, age of onset, DSM-III-R diagnosis, gender and psychopathology. The intention of these comparisons is to provide the reader with a profile of the similarity and differences between the samples or patient demographics. Subsequent analysis of the measurement model between the two samples must be made in light of the patient characteristics between the two samples. If the samples are different in patient characteristics but similar in construct validity of the measurement model then one could argue for an even stronger sense of structural invariance of the psychotic experience. Although the structural invariance procedure is not a comparison between diagnostic groups, the structural invariance between the samples, despite differences in patient characteristics, would argue for the robustness of the model. In other words, if the measurement model holds invariant despite the differences in patient characteristics then the underlying structure of the measurement model is robust and not subject to great differences to the type of psychosis defined under diagnostic conventions.

Age of Onset

Age of onset of psychosis has attracted wide spread debate in the literature. Some researchers have been concerned about how the onset of psychosis and age interact to predict outcome (Lenior, Digemans, Linszen, De Hann, & Schene, 2001; Linszen, Dingemans, & Lenior, 1994; Maziade et al., 1996; Mc Miler, Lawrie, Byrne, Cosway, & Johnstone, 2002; Wieselgren & Lindstrom, 1996; Yung et al., 1999), while other researchers are interested in the neurological expression of psychosis and

its effects on the brain (Murray, O'Callaghan, Castle, & Lewis, 1999; Rapoport et al., 1999; Thompson et al., 2001). What is difficult with onset studies is how to define the onset of psychosis and what phenomenology is required to say with some confidence that the expression of a certain set of signs and symptoms are particular to psychotic illness (Maziade et al., 1996; Mc Miler et al., 2002; Yung & McGorry, 1996; Yung et al., 1999). How age at onset is determined for the validation and cross-validation group will be discussed in turn.

Data drawn from the validation sample which include demographic information are extracted from the Royal Park Multi-Diagnostic Instrument for Psychosis (RPMIP). This instrument assesses prodromal symptoms of patients experiencing their first episodes of psychosis and demonstrates adequate reliability and validity (McGorry et al., 1990). Prodromal features (i.e., clinical features which precede a psychotic episode) are assessed in terms of identifying particular psychosocial stressors. The interview assesses the presence of psychosocial stressors up to one year preceding the psychotic episode. Prodromal and residual symptoms are described in the following table. These symptoms are assessed to determine at what stage the psychotic illness emerged prior to treatment. This is calculated in months from the time of admission into hospital for treatment or the emergence of "frank" psychotic symptoms (Edwards et al., 1999). Age of onset is calculated by age in years and months from the patient's age to the emergence of psychotic symptoms.

Table 4.2
Assessment of Prodromal and Residual Symptoms (DSM III R Schizophrenia) Used in the RPMIP

Prodromal Symptoms	Description
Social isolation or withdrawal	Loss in contact with friends and family
Impairment in role functioning	Reduction in work or tasks and responsibilities including home, work and education duties
Peculiar behaviour	Odd or unusual change in lifestyle (eg collecting garbage, hoarding food etc)
Impairment in personal hygiene	Problems in maintaining personal hygiene
Blunted, flat or inappropriate affect	Problems maintaining eye contact, monotonous speech, "wooden" gestures, affect inappropriate to conversation
Digressive vague speech	Conversation difficult to follow, unnecessary detail, frequent changes of subject
Odd or bizarre ideation	Claims of special powers, interests in the occult or magic, paranoid ideation
Unusual perceptual experiences	Reports of visual, auditory, tactile, or body phenomena that can not be explained by rational processes
Marked lack of initiative	Hard to get motivated, lacks interest in things they previously liked to do, feelings of weakness or tiredness
Mood disturbance	Presence of substance use resulting in disturbances in mood

The cross-validation sample used a more simplified assessment for the onset of psychosis. The date of onset of psychosis is determined by "the commencement of positive symptoms which do not include the period of prodrome" (see Appendix II for EPOES Client Assessment Form). Positive symptoms are those determined as frank psychotic symptoms including hallucinations, delusions, and those described in Table 4.2, such as odd or bizarre ideation, unusual perceptual experiences, and peculiar behaviour. The approach with the cross-validation group does not use an epidemiological sampling frame, but rather a pathway to care paradigm, where help seeking behaviour determines when and what psychotic symptomatology is displayed.

This approach in determining the onset of psychosis is more akin to how most mental health clinics in Australia would operate, while the validation sample uses a determination of onset typical of a research institute. The cross-validation sample also calculates onset in time by years and months from when the patient's current age to the display of frank psychotic symptomatology.

Age of onset was assessed in terms of the patient displaying prodromal features prior to the onset of a psychotic episode. In addition to demographic information, patients were administered the Brief Psychiatric Rating Scale (BPRS) (Overall & Gorham, 1988), Scale for the Assessment of Negative Symptoms (SANS) (Andreasen, 1984b), Beck's Depression Inventory (BDI) (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) and the Symptom Checklist List (Derogatis, 1993). The data used in the measurement model involves the first wave of the time series data known as the "stabilisation period". The stabilisation period is defined as the period usually 6 to 8 weeks following the initial assessment of patients with their first episode of psychosis (Edwards et al., 1999). The initial assessment usually occurs when the patient is acutely unwell and is receiving inpatient treatment. Such a battery of tests would be difficult to administer at this earlier stage of the illness, and would probably only provide low yield in terms of reliable and valid data. Usually at the initial stages of a psychotic illness it is difficult to secure an accurate diagnosis since the patient can display signs and symptoms of psychosis that may develop into a number of manifestations. These may include schizophrenia, schizoaffective disorder or a drug induced psychosis (Bell, Dudgeon, McGorry, & Jackson, 1998; McGorry et al., 1995b; Yung & McGorry, 1996; Yung et al., 1999).

Diagnosis

Although both the validation and cross validation sample use the DSM-III-R (APA, 1994), differences at which the diagnostic assessment is made by the treating psychiatrist may result in differences in the type and profile of psychotic disorders. The cross-validation sample that consisted of four early psychosis programs in Perth, administered the initial assessment within the first 4 weeks of patients being formally received into their care. This could mean that the diagnostic assessments of the cross validation group might be unstable, since the assessment is likely to be made earlier on in the course of the illness. In addition to this, the motivation for providing a diagnosis was one of clinical treatment rather than of research interest, since it was the treating consultant psychiatrist that determine the diagnosis of the patient and not a research investigator.

The validation sample used a more thorough research assessment of diagnosis based upon DSM-III R criteria for psychotic disorder. The determination of diagnosis is one based upon research rather than a clinical paradigm, and so the motivations to place a diagnosis against the presenting signs and symptoms is not one based upon clinical care (as with the cross-validation group), but in reference to a nomenclature that can be justified under research purposes.

While it is well recognised that diagnosis is unstable at the early stages of psychotic illness (Azevedo et al., 1999; Bell et al., 1998; Maziade et al., 1996; McGorry et al., 1995b; Schwartz et al., 2000), the purpose of the comparison between the two samples is to provide a descriptive picture of how the two samples can be determined as similar or dissimilar in diagnostic type. It is important to emphasise that the limitations of accurately describing early psychosis cohorts is problematic due to the uncertain nature of psychotic illness to express a specific and predictable course

based upon known phenomenology. Where commonality can be said to exist between the two samples is based on the condition that both operate services designed to treat patients experiencing their first episodes of psychosis in accordance with the national early psychosis guidelines (Whiteford, 1996) (see Table 3.1).

Diagnoses were placed into four groups. The first being schizophrenia and schizophreniform disorder, the second psychosis with affective disorders, the third as substance induced and brief psychotic disorder, and the final group being psychotic disorder not otherwise specified. Each will be discussed in turn.

Schizophreniform disorder was included with schizophrenia since recent research suggests that the majority of patients with schizophreniform disorder develop into schizophrenia (Zarate, Tohen, & Land, 2000). Diagnosis as a nomenclature is problematic at the early stages of psychotic illness and the reader should be aware that the results of the diagnostic comparisons between the two groups should only be interpreted as a general descriptive of cases within each data set. It is difficult to say with any confidence if the diagnosis of a patient with psychosis is stable in the first two years of the illness (Azevedo et al., 1999; Maziade et al., 1996, McGorry et al., 1995b; Schwartz et al., 2000). Since both samples are describing patients within the first 6 months of their first episodes of psychosis a diagnosis is provisional at best.

The second group included psychotic disorders that have a major affective component to the presentation of the disorder. This included both active and passive affective states. In the active spectrum, schizoaffective disorder and bipolar affective disorders with psychotic features of a manic type were included. On the passive side, major depressive disorder with psychotic features were also included in this group. This group describes patients who display not only psychotic features but also

extreme affective states that may give rise to the expression of psychosis or exacerbate or complicate the psychosis with dis-regulatory behaviour.

The third group is those patients who have experienced drug-induced psychosis or a brief psychosis that is likely to be brought on by substance mis-use. This third group describes those cases who may go on to express a genetic based psychotic disorder evident possibly in schizophrenia (Burke, Murphy, Bray, Walsh, & Kendler, 1996), or have a brief episode of psychosis that may not return if the patient continues not to use substances (James & Castle, 2002). Since both the validation and cross-validation sample are relatively young in age and drug use in young adults is prevalent (Cantor-Graae, Nordstrom, & McNeil, 2001), it is important to consider this group of patients as a separate category of patients.

The final group is of those patients who have a psychotic disorder not otherwise specified. This is usually used for cases that do not fit the diagnostic requirement of the other groups or provisionally the diagnostician is not confident in providing a firm diagnosis of psychosis until they can generate a fuller clinical picture. This usually occurs by monitoring the symptoms and signs of psychosis over the following 6 months to determine what presentation the patient is likely to display.

Measure

Data from the validation sample that measured self-reported psychopathology consisted of the Symptom Checklist 90 item version (SCL-90) (Derogatis, 1993). The cross-validation sample had data consisting of the Brief Symptom Inventory (BSI) (Derogatis, 1982) (See Appendix III for item response set of the BSI). As described in Chapter III Methodology, the BSI (Derogatis, 1982) is a short form of the SCL-90.

Items from the SCL-90 that make up the BSI were transferred into the BSI item format so that both the validation and cross-validation samples could be directly compared. Not all 53 BSI items contributed to the original exploratory factor analysis (see Table 4.9 for description of items used in the measurement model). To reduce item redundancy, those items that described each of the six dimensions described earlier (cognition, depression, anxiety, social withdrawal, paranoia and somatic concern) were examined. These items were drawn from the original symptom dimensions described in the BSI scoring manual (Derogatis, 1982). The scale of 0 to 4 is the same between the SCL-90 and BSI and these anchors were re-coded from 1 to 5 for the EFA.

In order to compare the validation and cross-validation sample on symptomatology, each of the nine subscales (i.e., Somatisation, Paranoid Ideation, Psychoticism, Obsessive Compulsive, Anxiety, Depression, Phobic Anxiety, Hostility and Interpersonal Sensitivity) and the three global indices (ie, Global Severity Index, Positive Symptom Total and Positive Symptom Distress Index) of the BSI were compared. This analysis was done in order to provide a symptomatalogical profile between the two samples. Comparison was made by way of independent sample *t*-tests. To assist the reader with the meaning of each scale used in the BSI, the following table provides a brief description drawn from the BSI administration, scoring and procedures manual (Derogatis, 1982).

Table 4.3
Description of Scales Calculated Under the BSI

Scale	Description	Items in Scale
Somatisation	Distress arising from the perception of bodily dysfunction.	2 7 23 29 30 33 37
Obsessive-Compulsive	Symptoms that describe thoughts, impulses and actions that are experienced as unremitting and irresistible.	5 15 26 27 32 36
Interpersonal Sensitivity	Feelings of personal inadequacy and inferiority, particularly in comparison with others.	20 21 22 42
Depression	A range of symptoms including dysphoric mood, lack of interest and motivation in life.	9 16 17 18 35 50
Anxiety	Signs such as nervousness and tension as well as panic attacks and feelings of terror.	1 12 19 38 45 49
Hostility	Includes thoughts, felling or actions that are characteristic of anger and aggression.	6 13 40 41 46
Phobic Anxiety	Persistent fear response to specific person, place, object or situation that is irrational or disproportionate.	8 28 31 43 47
Paranoid Ideation	Characteristics of projective thought, suspiciousness, grandiosity and delusion.	4 10 24 48 51
Psychoticism	Items indicative of withdrawn, isolated schizoid lifestyle, first-rank symptoms are also included.	3 14 34 44 53
Global Severity Index	The average severity of all items within the BSI.	All items added / 53
Positive Symptom Total	The sum of items endorsed with a positive (nonzero response).	Sum of positive responses
Positive Symptom Distress Index	Calculated by dividing the sum of the item values by the PST.	PST / sum of items values

Analyses

The research proceeded in a number of steps. The first procedure was to reduce item redundancy by identifying items from the BSI that had the highest loadings on each of the 6 factors proposed for the measurement model. This involved performing separate exploratory factor analyses on each of the six latent factors prescribed in the measurement model. The scoring manual for the BSI (Derogatis,

1982) was used to identify the items which make up the six factors. For example items 9, 16, 17, 18, 20, 35 and 50 were used to measure Depression. Each analysis was examined to identify whether the scale fell under a single factor. The three highest loading items (cardinal items) on the factor were then chosen to describe that factor (e.g., items 17, 35 and 50 measure Depression) within the measurement model. This procedure was used to form the overall measurement model that was then tested on the validation sample. The oblique rotation was the option selected for interpreting the solution, since it "provides a more accurate representation of how constructs are likely to be related to one another" (Fabriagar et al., 1999, p 282), rather than the varimax (orthogonal) rotation that assumes independence among the constructs. In addition, EFA (known as common factor analysis), rather than principal component analysis (PCA), was deemed more appropriate in analysing the data because the intention of the analysis was to identify the underlying latent constructs, rather than obtaining a solution that summarises the relationship among manifest variables , as is the case with PCA. This procedure does not appear a viable model for describing psychological behaviours (McArdle, 1990).

Second, this procedure was repeated for the cross-validation sample for the same purposes. That is, to explore the relationships between items prior to any specification of latent constructs as well as to identify which items load highly onto a given factor. These high loading or "cardinal symptoms" would assist in informing the measurement model under confirmatory factor analytic conditions.

Third, these analyses would inform the measurement model by testing the model independently under confirmatory factor analysis for both the validation and cross-validation samples. Model fit was assessed using EQS version 5.7b (Bentler, 1995). Overall model fit was determined by assessing a number of "fit indices" (Bentler, 1990; Marsh, Balla, & Hau, 1996). The Comparative Fit Index (CFI) and

the Robust Comparative Fit Index (RCFI) were used to assess the overall fit of the model. Values of 0.93 and above for the RCFI and CFI suggest acceptable levels of fit (Browne & Cudeck, 1993).

These indices are considered conventional measures of model fit and are criticised because they are affected by sample size (Steiger, 1990). The root mean square error of approximation (RMSEA) was also used to indicate model fit. The RMSEA measures model discrepancy per degree of freedom with values of 0.05 or lower indicating very close fit. A RMSEA approaching 0.08 represents reasonable errors of approximation (Browne & Cudeck, 1993). In contrast to other fit indices that provide point estimates, the RMSEA has known distributional properties that allows for the calculation of confidence intervals that contain the true value of the index for the model in the population (MacCallum, Browne, & Sugawara, 1996). As stated in Chapter III, MacCallum et al. (1996) proposed a framework for testing hypotheses about model fit based upon confidence intervals. When the confidence interval range falls below 0.05 it is argued that there is strong support for rejecting the "not-close fit" hypothesis (i.e., accepting the hypothesis of "close fit"). When the range of the confidence interval is above 0.05, the hypothesis of "close fit" is rejected. When the range of the confidence interval "straddles" 0.05, neither the hypothesis of "close fit" nor the hypothesis of "not-close fit" may be rejected, allowing for both hypotheses to remain plausible (MacCallum et al., 1996).

In order to draw confident conclusions about model fit, adequate power needs to be demonstrated. Power is the "probability that the results of a significance test will lead to rejection of the null hypothesis when there is a true effect in the population" (Kline, 1998 p. 308). Power analysis for tests of model fit require specification of effect size (i.e., the extent to which the null hypothesis is incorrect).

The requisite sample size is calculated when the null hypothesis is less than or equal to 0.05 and the alternative hypothesis is equal to 0.08, using an alpha level of 0.05 (alpha) and a power of 0.80 (beta). In this study all model fit evaluations were based on the MacCallum et al. (1996) framework after demonstrating adequate power and sample size requirements.

In the fourth stage of the analysis, model respecification procedures were used to identify the items that most clearly captured the constructs of interest (Byrne et al., 1989). Not all models are well constructed the first time they are assessed against the data, and it is realistic to expect that within structural equation modelling " ...most often some respecification of the measurement model is required" (Anderson & Gerbing 1988, p. 416). Within a multiple indicator measurement model, one way of respecifying the model is to examine the residuals. That is, items that have not had sufficiently strong loadings on their target factors, or may contribute to excessive multivariate non-normality may be deleted from the model (Kline, 1998). Low factor loadings are those items with values less than 0.3 (Kline, 1998). One of the advantages of the EQS program is that it provides the Lagrange statistics. These statistics provide the contribution of each parameter estimate in terms of the chi-square. Parameters that are restricted (typically, set to zero) may have a large Lagrange Multiplier chi-square statistic, indicating that the restriction is unlikely to be true in the population and not consistent with the data. In such a case, releasing the restriction (typically, freeing a fixed zero parameter for estimation) will reduce the overall chi-square statistic of the model.

It is understood that two items are sufficient to define a construct (Bollen, 1989). It is the intention of the modelling procedure in this study, to use three or four of the highest loading items for each construct of interest. The use of three items or

more best describe a uni-dimensional construct, rather than a two item factor which is likely to describe a limited aspect of the construct (Hankins, French, & Horne, 2000).

The fifth procedure is to examine the convergent validity, discriminant validity and overall fit of the respecified model. Convergent validity was examined by assessing the parameter estimates (factor loadings) of the items on their specified dimensions. High and moderate loadings suggest convergent validity. Standardised values greater than 0.5 demonstrate reasonably high factor loadings (Kline, 1998).

In the sixth stage, the fit of the measurement model was examined in an independent sample. The independent sample is different to that of the split-half randomisation procedure where the same sample is randomly broken in two. In this instance, the Western Australian sample will act as the cross-validation group. Bollen (1989) argued that an examination of model fit in an alternative sample is a sensible precursor to conducting invariance analysis. This is where the model is first examined independently on the validation and cross-validation group.

In the seventh part of the procedure, having established the viability of the model in the two samples, the factor structure derived from the validation sample was examined directly against the cross-validation group. This procedure goes beyond establishing the measurement model in the validation sample since the same measurement structure is required in the cross-validation sample (Burke et al., 1989). Cole and Maxwell (1985) argued that the evidence of construct validity in one sample does not necessarily guarantee construct validity across similar groups. The multi-sampling procedure in EQS was used to "cross-validate' the statistical equalities between the two sets of data. Equivalence between the two samples is evidence for generalisabilty of the model.

To establish invariance, competing models with increasing levels of restriction are compared. To establish cross-validation the loadings, error variances and construct covariances are required to be equivalent across both samples (Bollen & Long, 1993). Some authors argue however that the testing of equivalence at the error variance level is unduly strict (Bentler, 1995; Byrne, 1994; 1994b). With this in mind, consideration of structural invariance was based on comparisons of form, loadings and construct intercorrelations. The error variances were not considered in the invariant analysis. Non-significant difference in the chi-square statistic at each subsequent level of analysis suggests that the model generalises across both sets of data.

The 'n' for all analyses was based on listwise deletion of cases. Missing data for each item was replaced with the gender mean once the data was assessed in terms of missing completely at random (Schafer & Olsen, 1998). No item in each sample had missing values higher than 5% allowing for good approximation of the data with replaced scores. Multivariate non-normality may result in inflated Type I error and inaccurate significance tests of individual parameters (West, Finch, & Curran, 1995). Although EQS accommodates violation of this assumption through a scaled chi-square statistic (Satorra & Bentler, 1988), this statistic is unavailable in the multi-sample test of invariance in the version used. Unfortunately EQS version 5.7b does not calculate the Yuan-Bentler (2000) correction, which adjusts for this non-normality. In addition, the study was unable to use the expectation-maximisation based method (Arbuckle, 1996; Jamshidian & Bentler, 2000) which could utilise all 244 subjects to calculate covariance matrices over the 3 time waves. All analyses were performed on the covariance matrices using the maximum likelihood estimation.

Results

Patient Characteristics

The validation sample consisted of 244 cases. Of these 72.5% were males. The cross validation sample consisted of 187 cases of which 68.4% were male. Proportional analysis using chi-square analysis revealed no differences in gender between the validation and cross validation samples ($\chi^2 = 1.24$, df = 1, $p = .264$) (see Table 4.4). Comparisons between DSM-III-R diagnosis revealed the validation sample to have higher proportional representation of subjects with schizophrenia (57%) than the cross validation sample (37.4%) and lower levels of subjects diagnosed with drug induced psychosis (0%) compared to the cross validation sample (20.32%) ($\chi^2 = 98.56$, df = 3, $p = .0001$) (see Table 4.4). These differences may best be explained in terms of when and how the diagnosis was made between the two samples. The validation sample diagnosis was made at the stabilisation period after admission into hospital some six to eight weeks after receiving initial treatment (Edwards et al., 1999). This longer period of time compared with the cross-validation sample, where the diagnosis is made within the initial stages of treating the patient, may have contributed to the differences. Since the validation sample diagnosis is made at a longer period after the initial presentation of psychosis a more accurate diagnosis of schizophrenia could be made. In addition, the diagnosis within the validation sample is made under research conditions with an assessment more thorough than those made in the Western Australian sample, which were made under normal mental health clinic conditions. Previous research has found clinic based diagnosis to be more varied in its accuracy than those made under research conditions (Forrester, Owens, & Johnstone, 2001).

Age and age at onset was also examined between the two samples. The independent *t*-test results indicated statistically significant differences with the validation sample being both younger in age (t = -7.27, df 1,429. p = .0001) and at age of onset (t = -7.80, df 1,429, p = .0001). This is most likely to be attributable to the catchment area age population differences between the two samples. The validation sample age range of 15 to 35 "straddles" adolescent and adult age groupings while the cross-validation sample only included subjects who were managed by adult mental health services (age range 18 to 45).

Onset was calculated in values of months by subtracting the age from the age of onset (since age was calculated in years and months). The analysis indicated the cross validation sample to have a lower onset in months of 2.1 months compared to 4.7 months with the validation sample (t = 2.93, df 1,429, p = .004). Although this is statistically significant the practical difference in terms of onset are most likely to be negligible.

Comparisons of psychopathology by using the three global indices and nine symptom dimensions of the BSI (Derogatis, 1982) indicated the validation sample to have lower levels of psychopathology on all measures compared to the cross-validation sample (see Table 4.4). Once again this could be explained in terms of when the assessments were made between the samples. Since the validation sample was assessed during a "stabilisation period" of the illness, it is expected that the cross-validation sample would have higher levels of psychopathology.

Table 4.4
Demographics of Validation and Cross-Validation Samples Used for Validating the Measurement Model

	Validation Sample		Cross-validation Sample				
	Victorian (N = 244)		Western Australian (N = 187)				
Patient Characteristic					Analysis	df	*p*
Gender					$\chi^2 = 1.24$	1	.264
Male	179		128				
Female	65		59				
Diagnosis ICD-10					$\chi^2 = 98.56$	1,3	.0001
Schizophrenia	139		70				
Affective Psychosis	90		26				
Drug Induced	0		38		$\chi^2 = 106.02$	1,3	.0001
Other Psychoses	18		53				
Onset		SD		SD			
Age	22.07	3.45	25.76	6.87	t =-7.27	1,429	.0001
Age of onset	21.60	3.47	25.55	6.84	t = -7.80	1,429	.0001
Onset in months	4.75	1.00	2.10	.81	t = 2.932	1,429	.004
Psychopathology							
Somatisation	.62	.69	.79	.74	t =-2.34	1,429	.019
O-C	1.02	.90	1.34	1.00	t = -3.45	1,429	.001
I-S	.97	.98	1.36	1.09	t =-3.94	1,429	.0001
Depression	1.11	1.01	1.34	1.03	t = -2.37	1,429	.018
Anxiety	.89	.85	1.30	1.01	t = -4.55	1,429	.0001
Hostility	.59	.69	.86	.85	t =-3.62	1,429	.0001
Phobic Anxiety	.69	.84	1.05	1.01	t = -4.01	1,429	.0001
Paranoid Ideation	.85	.88	1.19	1.02	t =-3.69	1,429	.0001
Psychoticism	.80	.86	1.13	.99	t = -3.62	1,429	.0001
GSI	.84	.71	1.15	.83	t =-4.12	1,429	.0001
PST	23.20	14.83	29.93	14.65	t = -4.69	1,429	.0001
PSDI	1.65	.71	1.82	.69	t =-2.45	1,429	.014

Note: O-C = Obsessive Compulsive; I-C = Interpersonal Sensitivity; GSI = Global Severity index; PST = Positive Symptom Total; PSDI = Positive Symptom Distress Index.

Exploratory Factor Analysis

The following table displays the separate factor analyses performed with each of the six factors extracted from the BSI scoring manual (Derogatis, 1982). The three highest loading items were selected considering both the validation and cross-

validation sample. Four items were chosen for cognition due to the strength of the loadings and the central role that cognition plays in the development and course of psychosis (Bilder et al., 2000; Cosway et al., 2000; Cuesta et al., 2000a; Heaton et al., 2001; Ngan & Liddle, 2000; Penadés et al., 2001; Rapoport et al., 1999).

Table 4.5
Item Loadings of Each Factor Within the BSI Used to Inform the Cardinal Items in the Measurement Model (n = 244)

	Factor Loadings		Factor Loadings
	Victoria		Victoria
Paranoia		Cognition	
BSI4	0.608	BSI5	0.721*
BSI10	0.697*	BSI15	0.672
BSI21	0.707*	BSI26	0.637
BSI24	0.693	BSI27	0.745*
BSI48	0.523	BSI32	0.730*
BSI51	0.718*	BSI36	0.754*
Anxiety		Depression	
BSI1	0.688*	BSI9	0.628
BSI12	0.651	BSI16	0.747
BSI19	0.720*	BSI17	0.773*
BSI38	0.692	BSI18	0.764
BSI45	0.785*	BSI20	0.711
BSI49	0.628	BSI35	0.754*
		BSI50	0.809*
Phobia		Somatisation	
BSI8	0.748*	BSI2	0.545
BSI28	0.718	BSI7	0.526
BSI31	0.774*	BSI23	0.541
BSI42	0.620	BSI29	0.690*
BSI43	0.775*	BSI30	0.642*
BSI47	0.619	BSI33	0.660*
		BSI37	0.617

Note: * cardinal items used in the measurement model for each factor

From this procedure these items were entered into an exploratory factor analysis (EFA) using the maximum likelihood extraction with promax rotation was conducted on the validation sample (n = 244) to test the dimensionality within the

items. The initial factor analysis of the validation sample yielded 3 factors after 5 iterations of the data with eigenvalues greater than one, collectively accounting for 51.32% of the variance. This solution produced a significant chi square ($\chi^2 = 323.308$ df = 117,$p < .0001$). The 3-factor solution could be interpreted as factor 1 depression and cognitive deterioration, factor two somatic concern and paranoia, and factor 3 phobia and anxiety.

Table 4.6
Pattern Matrix of Initial Maximum Likelihood Extraction with Promax Rotation for Validation Sample (n = 244)

Indicator	Factor 1	Factor 2	Factor 3
BSI35	.900		
BSI50	.894		
BSI27	.666		
BSI36	.656		
BSI17	.623		
BSI32	.504		
BSI29		.753	
BSI33		.715	
BSI30		.656	
BSI51		.556	
BSI5	.406	.465	
BSI10		.416	
BSI21		.371	
BSI1		.365	
BSI8			.883
BSI43			.817
BSI31			.648
BSI45			.582
BSI19			.364
$\chi^2 = 323.308$	df = 117	$p < .001$	51.32% Variance

Note: Item loadings < .3 are not reported in the pattern matrix

To test whether a 6-factor model proposed earlier which describes anxiety, cognition, depression, paranoia, phobia and somatic concern, was a superior fit to the data, a 6-factor model was entered into the maximum likelihood method. This model accounted for 60.12% an increase of 9% from the initial extraction. There was a

statistically significant reduction in the chi-square relative to the change in degrees of freedom from the initial factor analysis (difference χ^2 = 191.68 df = 45, $p < .05$). These results suggest that the 6-factor solution is superior to the initial 3-factor solution, where no factor number specification was made to the extraction process. Observation of the item loadings in Table 4.7 suggest that factor 1 describes depression, factor 2 phobia, factor 3 cognition, factor 4 somatic concern, factor 5 anxiety and factor 6 paranoia. Only BSI item 51 (feeling that people will take advantage of you if you let them) cross loaded on both the anxiety (.487) and paranoia (.538) factors.

Table 4.7
Pattern Matrix of 6-Factor Maximum Likelihood Extraction with Promax Rotation for Validation Sample (n = 244)

Indicators	Factor 1	Factor 2	Factor 3	Factor 4	Factor 5	Factor 6
BSI50	.901					
BSI35	.771					
BSI17	.702					
BSI8		.796				
BSI43		.794				
BSI31		.594				
BSI27		.367				
BSI5			.912			
BSI36			.584			
BSI32			.559			
BSI30				.742		
BSI33				.641		
BSI29				.537		
BSI45					.630	
BSI19					.609	
BSI1					.547	
BSI10						.760
BSI51					.487	.538
BSI21						.568

χ^2 = 131.626 df = 72 $p < .001$ Variance 60.12%
Note: Item loadings < .3 are not reported in the pattern matrix

Observation of the correlation matrix of the factors indicates moderate relationships between the factors. The highest correlation is between depression and cognition (r =

.694) which suggests that both factors may have an underlying relationship that describes negative symptomatology, since both factors describe both low arousal and poor cognition which are cardinal to negative symptomatology.

Table 4.8
Factor Correlation Matrix for Validation Sample (n = 244)

	Factor 1	Factor 2	Factor3	Factor 4	Factor 5	Factor 6
Factor 1	1.000					
Factor 2	.626	1.000				
Factor 3	.694	.576	1.000			
Factor 4	.500	.504	.594	1.000		
Factor 5	.608	.625	.557	.645	1.000	
Factor 6	.504	.533	.579	.529	.547	1.000

The exploratory factor analysis suggests that the 6-factor model was able to describe better the data rather than the initial 3-factor solution. With only one item having higher loadings on both anxiety and paranoia the model appears to be a good approximation to the data. Item description for each latent factor is shown in the table below.

Table 4.9
Items Used from the BSI that Describe the Measurement Model

Factor and Item Description	Factor and Item Description
Cognition	Paranoia
5. Trouble remembering things	10. Feeling that most people cannot be trusted
27. Difficulty making decisions	21. Feeling that people are unfriendly or dislike you
32. Your mind going blank	51. Feeling that people will take advantage of you if you let them
36. Trouble concentrating	
Anxiety	Social Phobia
1. Nervousness or shakiness inside	8. Feeling afraid in open spaces or on the streets
19. Feeling fearful	31. Having to avoid certain things, places or activities because they frighten you
45. Spells of terror or panic	43. Feeling uneasy in crowds, such as shopping or at a movie
Depression	Somatisation
17. Feeling blue	29. Trouble getting your breath
35. Feeling hopeless about the future	30. Hot or cold spells
50. Feelings of worthlessness	33. Numbness or tingling in parts of your body

The next procedure was to take this measurement model and examine the dimensionality of the model within a confirmatory factor analytic framework. In addition to this, the structural invariance of the model will be cross-validated against an independent sample.

Confirmatory Factor Analysis

Having established a measurement model via both the EFA process and theoretical considerations within the literature, the next step in the analysis was to perform a CFA on the validation sample. All 244 cases used in the EFA were used for the CFA. Although a number of the cases within the CFA contributed to multivariate non-normality, the range between these cases was not extreme enough to warrant their exclusion from the analysis (Byrne, 1994). Using EQS version 5.7b (Bentler, 1995), the six dimensional model of psychosis was examined on the validation sample.

As previously stated, Bollen and Long (1993) suggested that three items with the highest loadings from the EFAs are sufficient to measure a latent construct in a CFA. The only exception to this suggestion was with the cognition factor. Four items were chosen to improve construct definition since within the literature some attention has been focused on the cognitive aspects of the illness (Bilder et al., 2000; Cosway et al., 2000; Cuesta et al., 2000a; Heaton et al., 2001; Ngan & Liddle, 2000; Penadés et al., 2001; Rapoport et al., 1999). Thus 19 items, organised into 6 dimensions were used in the confirmatory factor analysis. Figure 4.1 shows the full measurement model where each of the observed indicators is hypothesised to load on its target construct (indicated by the lambda path λ from the construct ksi ξ), with the

constructs allowed to covary freely (indicated by phi Φ) between the constructs. The error terms (delta δ) are also displayed in Figure 4.1. The order in which the items are placed in the BSI are shown in Appendix III.

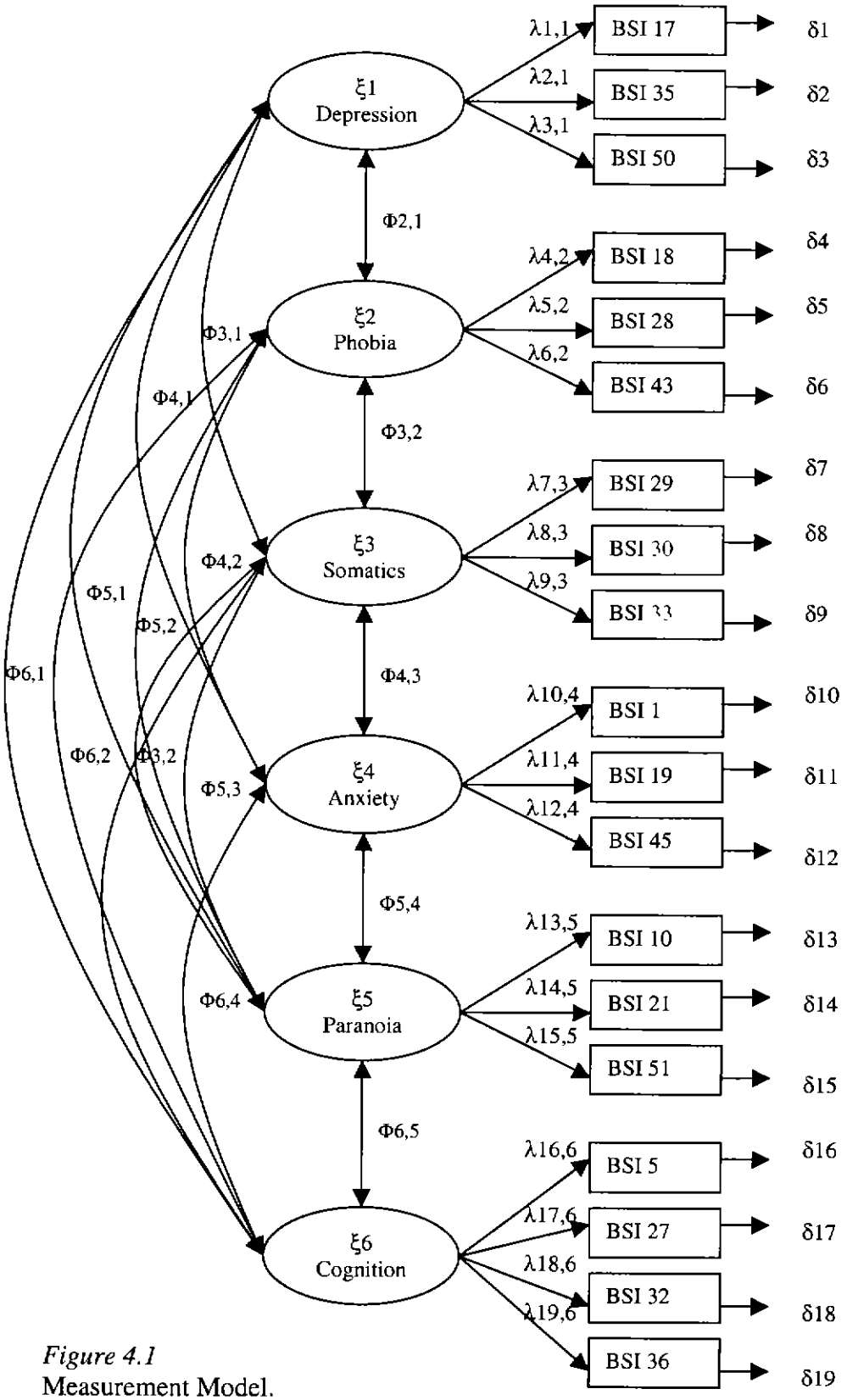

Figure 4.1
Measurement Model.

Table 4.10 shows the fit statistics for the six-factor measurement model. The null model where all indicators act as their own factor (Byrne, 1994) as well as a one-dimensional model (where all indicators are driven by one unitary construct) and three dimensional model from the original EFA are displayed. The null model acts as a "baseline model" where all indicators are independent or uncorrelated to each other. Bentler (1995) argued that "in large samples, the independence model is also a good baseline model against which other models can be evaluated for the gain in explanation they achieve" (p. 92). In other words a significant reduction in the chi-square from the baseline model to the proposed model indicates some order in the data beyond single unitary indicators acting independently of themselves. The one-dimensional model tests a similar notion against the measurement model, but instead whether a 6-factor model has better explanatory power against a 19 factor independent model (as in the baseline model), this procedure examines whether 6 factors better describe the data than where all indicators come from one unitary construct. The complete output of the 6-factor CFA analysis, including univariate statistics, is shown in Appendix V.

Table 4.10 shows that the 6-factor model was the only acceptable fit to the data. The model demonstrated high kurtotic distribution (Mardia kurtosis coefficient: 183.988) indicating poor multivariate normality. All the "fit" indices for the 6-factor model were above the acceptable criteria for each. The Comparative Fit Index (0.940) and the Robust Comparative Fit Index (0.970) were all above the 0.93 criteria. The RMSEA point estimate 0.065 was relatively close to Steiger's 1990 criteria for close fit (0.05) and well below MacCallum et al's., (1996) criterion for a "mediocre fit" between 0.08 and 1.00. The RMSEA confidence intervals (0.053 - 0.075) did not straddle 0.05 and, in addition demonstrated a narrow range, thus one could

confidently reject the model as a poor fit to the data and the confidence intervals suggest precision in the indicator of fit (MacCallum et al., 1996). Neither the null model nor the one-dimensional model provided acceptable fit to the data.

Table 4.10
Fit Indices of the 6-Factor Measurement Model of Psychosis Compared with the Null 1and 3 Factor Models (Validation Sample n = 244)

Model	χ^2	S-B χ^2	df	CFI	RCFI	RMSEA (CI)
Null	2515.941		171			
1 Factor	624.790	388.369	152	0.798	0.845	0.113 (0.104 - 0.122)
3 Factor	435.923	281.446	149	0.878	0.913	0.089 (0.079 - 0.099)
6 Factor	275.033	181.222	137	0.940	0.970	0.065 (0.053 - 0.075)

Note: S-Bχ^2 = Satorra-Bentler scaled statistic, CFI = CFI, RMSEA = Root Mean Square Error of Approximation, CI = Confidence Interval.

The same procedure was conducted independently on the cross-validation sample (n = 187) with the results reported in Table 4.11. The "fit" indices indicate the 6-factor model to be the only model within acceptable ranges of each fit indices. The CFI and RCFI resulted in scores above the 0.93 criterion of acceptable fit. The RMSEA point estimate was further away from the 0.05 criterion than the validation sample, although within acceptable limits. In comparison to the validation sample, the fit indices were slightly lower than those of the validation sample but well within the bounds of what would be considered in the literature as the model representing a "reasonable" fit to the data (MacCallum et al., 1996; Steiger, 1990). Like the validation sample, neither null nor the one or three-dimensional models provided acceptable fit to the data. A non-zero RMSEA indicates that some loadings may of cross-factored or error correlated higher than expected.

Table 4.11
Fit Indices of the 6-Factor Measurement Model of Psychosis Compared with the Null, 1 and 3 Factor Models (Cross-Validation Sample n = 187)

Model	χ^2	S-B χ^2	df	CFI	RCFI	RMSEA (CI)
Null	2237.790		171			
1 Factor	487.007	360.0942	152	0.818	0.838	0.109 (0.098 - 0.119)
3 Factor	439.148	324.9811	149	0.860	0.898	0.102 (0.091 - 0.113)
6 Factor	258.453	197.710	137	0.939	0.963	0.069 (0.056 - 0.082)

Note: S-Bχ^2 = Satorra-Bentler scaled statistic, CFI = CFI, RMSEA = Root Mean Square Error of Approximation, CI = Confidence Interval.

Invariance Analysis

Having demonstrated the 6-factor model's validity as a measurement model independently in both the validation and cross-validation samples, the next step is to proceed with testing the model simultaneously between the two samples. The purpose of cross validation of the measurement model lies in the strength by which the measurement model can be transferred to similar populations of interest (Bollen, 1989; Bollen & Long, 1993; Burke et al., 1989). This analysis proceeds by determining whether there is equivalence in form, loadings and construct covariances across independent samples (Bentler, 1995; Byrne, 1994). Results of the invariance analyses between the validation and cross-validation samples are shown in Table 4.12. The baseline model, in which the basic form of the model is compared between the two samples, provided a good fit to the data (CFI = 0.939, RMSEA 0.047, RMSEA confidence interval = 0.041 – 0.053). The first step was to constrain the loadings to be invariant (non-significant) or equal across the samples. This allowed for 19 degrees of freedom to be calculated with this constraint. Table 4.12 shows this

constraint resulted in a non-significant inflation of the chi-square relative to the baseline ($\Delta\chi^2 = 20.228$, 19 df, $p > 0.05$). In addition to these constraints (ie; maintaining equality for the loadings across the samples), the factor covariances were also constrained to be equal across the samples. This also resulted in a non-significant chi-square ($\Delta\chi^2 = 21.471$, 15 df, $p > 0.05$).

Table 4.12
Fit Indices of the 6-Factor Measurement Model and Invariance Analysis Investigating the Factor Loadings and Factor Covariances (Validation Sample n = 244)

Model	χ^2	$\Delta\chi^2$	df	Δdf	CFI	RMSEA (CI)
Form Baseline	533.484	-	274	-	0.939	0.047 (0.041 - 0.053)
Invariance of Loadings	553.712	20.228	293	19	0.939	0.046 (0.040 - 0.051)
Invariance of Loadings & Covariances	575.183	21.471	308	15	0.937	0.045 (0.039 - 0.050)

Note: $\Delta\chi^2$ change in chi-square, Δdf = change in degrees of freedom, CFI = CFI, RMSEA = Root Mean Square Error of Approximation, CI = Confidence Interval.

In short, the non-significant difference in the chi-square statistic, at each subsequent level of analysis, suggests that the model generalises across both sets of data. The results, therefore, show that the proposed model of psychosis successfully generalises across two similar samples of subjects with early psychosis.

Table 4.13 show the means, standard deviations and alpha reliabilities (α) across both samples for each dimension within the measurement model. As expected, from the analyses, all constructs had internal consistencies exceeding Nunnally's (1978) criterion ≥ .70. Higher scores for each symptom dimension denote higher levels in psychopathology. All symptom dimensions were statistically higher for the cross-validation sample compared to the validation sample.

Table 4.13
Descriptive Statistics for Psychosis Measurement Model Between the Validation and Cross–Validation Samples

Dimension	Validation Sample n = 244			Cross validation Sample n = 187			
	Mean	SD	α	Mean	SD	α	*t* value
Depression	1.10	1.10	.83	1.49	1.17	.86	-3.464 ***
Phobia	.78	.95	.82	1.18	1.14	.83	-3.998 ***
Somatic Concern	.54	.80	.72	.71	.84	.73	-2.047 *
Cognitive Deterioration	1.15	1.00	.83	1.47	1.09	.86	-3.145 **
Anxiety	.86	.92	.78	1.28	1.10	.83	-4.303 **
Paranoia	.90	.99	.77	1.30	1.14	.81	-3.901 ***

Note: * significant difference between samples at $p < .05$, ** = $p < .01$, *** = $p < .001$. Range 0 – 4 for all variables.

Correlations among the symptom dimensions across both the validation and cross-validation sample are shown in Table 4.14 All correlations in both samples were statistically significant ($p < .001$). The correlations in the validation sample ranged from .544 to .682. These indicate relatively moderate correlations. A similar strength in relationship existed between the symptom dimensions in the cross-validation sample with correlations ranging from .496 to .753. Such relationships between the samples suggest the six factor measurement model to have similar associations amongst the factors. Fisher's Z transformation of correlations indicated that correlations between depression and anxiety; and paranoia; and anxiety and social phobia are statistically different across both samples with the cross-validation sample showing higher associations. However after Bonferroni adjustment ($p = .004$), these correlations were rendered non-significant.

Table 4.14
Correlations Between the Dimensions of Psychosis in the Validation and Cross-Validation Sample

	1	2	3	4	5	6
1 Depression	1.00	.651 **	.496 *	.650 *	.732 **	.696 **
2 Social Phobia	.561 **	1.00	.484 *	.592 **	.753 *	.622 *
3 Somatic Concern	.414 **	.456 *	1.00	.558 *	.556 **	.548 *
4 Cognition	.682 **	.550 **	.533 **	1.00	.670 *	.582 **
5 Anxiety	.595 **	.660 *	.527 *	.589 *	1.00	.673 *
6 Paranoia	.530 **	.564 *	.544 **	.572 *	.602 **	1.00

Note: lower half of matrix is from validation sample (n = 244) upper half from the cross-validation sample (n = 187) ** $p < .01$, * $p < .05$.

Discussion

The study examined whether a 6-factor model of psychosis describing *cognitive deterioration, depression, anxiety, somatic concern, social phobia* and *paranoia* is a commonly held experience between two independent samples of subjects with early psychosis. This model was built by considering both theoretical debate in the literature as to the nature and structure of psychosis, as well as the pre-existing symptom structure prescribed in the Brief Symptom Inventory (Derogatis, 1982).

The validation sample comprised of 244 cases of patients between the age of 15 and 35 who have recently experienced their first episodes of psychosis within the Early Psychosis Prevention and Intervention Centre (EPPIC) Melbourne. A cross-validation sample of 187 cases with ages between 18 and 45 was compared from four Western Australian early psychosis programs. Both samples were compared on age, age at onset, gender and DSM-III-R diagnosis. Tests for patient characteristics and levels of psychopathology revealed the validation group to be younger, have a longer length from age to age of onset of psychosis, and more likely to have a diagnosis of schizophrenia compared to the cross validation sample. Differences, demographically and psychopathologically between the samples can be described in terms of catchment area, age range, and when the assessments for diagnosis and psychopathology were made during the course of the illness. Both samples can be considered early psychosis intervention programs since both systems of care in Victoria and Western Australia operate under the National Early Psychosis Intervention Guidelines (Whiteford, 1996).

The study provides new insights into the structure and nature of psychosis since it is the first study of its kind to validate a measurement model of psychosis using self report data. Previous studies have used both exploratory and confirmatory factor analysis to examine the underlying symptomatological structure of psychosis but exclusively from an observational point of view (see Table 2.5 for review of factor analytic studies in psychosis). These studies were used to form the basis of the structure of the measurement model where commonly a 4 to 5 factor structure is reported (Cuesta & Peralta, 1995; Kay & Sevy, 1990; Lançon et al., 1998; 2000; Peralta & Cuesta, 1992; 1994; 1995). As highlighted earlier, the problem with previous studies in psychosis is that the researchers have relied almost exclusively on observational instruments. This appears problematic for any form of factor analysis since the models explored or confirmed are nested in the "observer effect". The observer effect is understood in this context as the "pre-constructed" notions of psychosis nested in the observers understanding of what he/she observes and rates as psychosis. While this may appear seemingly objective, it is problematic for factor analytic studies since the likelihood of the observer rating in a particular way (a response set bias), is increased if the observer is trained in a certain model of psychosis. Second, most instruments such as the Positive and Negative Syndrome Scale (PANSS) (Kay et al., 1987; Kay et al., 1986b) or Signs for the Assessment of Negative or Positive Symptoms (SANS, SAPS) (Karakula & Grzywa, 1999), suggest a pre-constructed dimensionality in the instrument itself due to the way they are administered. One major assumption of any factor analytic investigation is the requirement that each observation is independent of the other. To the author's knowledge, no study investigating the symptomatological structure of psychosis to date has accounted for the fact that their studies violate this assumption, nor have they

corrected for this serious limitation by using nested modelling techniques (Byrne, 1994; Byrne et al., 1989). In addition to this limitation, none of these studies have used a cross-validation sample independent of the original sample to test whether the model is replicable.

The present study overcomes these limitations by first allowing for each observation to be independent of the next. This was achieved by using an instrument that measures psychopathology through self-report. Second, a cross-validation sample was used to impose the same factor structure to test for structural invariance of the measurement model. Both samples although similar in terms of meeting the guidelines of providing early psychosis intervention strategies (Whiteford, 1996), were separated geographically where one sample can not contaminate nor influence the other. The measurement model underwent stringent tests of invariance (Anderson & Gerbing, 1988; Byrne & Shavelson, 1989; Byrne, 1994) to ascertain the likelihood of the measurement model to be a plausible explanation of the experience of psychosis from the cases themselves and not filtered through the observer effect.

Overall, the results support the proposed six dimensional model of psychosis (see Figure 4.1) experienced by those with their first episodes of psychosis. Given that the dimensions simulate previous studies in the area that use observational instruments (Cuesta & Peralta, 1995; Kay & Sevy, 1990; Lançon et al., 1998; 2000; Peralta & Cuesta, 1994; 1995; 1998), the results lend firm evidence that this 6-factor model is an adequate description of the psychotic experience from those who independently rate it.

It is important to keep in mind however, that the model is limited by its ability to accurately describe a given latent construct (eg depression). The Brief Symptom Inventory (BSI) although a well validated instrument on psychopathology (Broday &

Mason, 1991; Francis et al., 1990; Johnson et al., 1996; Royse & Drude, 1984; Wood, 1987), is not an instrument that measures specifically schizotypy (Bergman et al., 2000; Claridge et al., 1996; Mc Miler et al., 2002; Reynolds et al., 2000; Suhr & Spitznagel, 2001), nor does it claim to measure exclusively the experience of psychosis. In this instance, researchers are limited to the purpose of a given measuring instrument and how it may answer specific questions in their line of investigation. Although the covariation among the indicators for each construct is naturally driven by the construct itself, it does not mean that these latent constructs are specific to the psychotic illness itself. In other words, other psychiatric conditions can equally display levels of depression, anxiety, social phobia and the like. What is unique is not necessarily the symptomatological structure itself, but that the symptomatological structure can be confirmed on a given psychopathological phenomenon such as psychosis.

Correlations between the 6-factors demonstrate moderate associations between them. Although this does not indicate a higher order factor (where $r > 0.80$ is required), it does suggest some unitary element that may underpin or drive the 6 factors. This may be more particular between the factors that describe cognitive deterioration and depression (i.e., negative symptoms) and anxiety and social phobia (i.e., fear). To date, no research has been conducted investigating higher order latent structures that may drive the first order factor, so often reported in the literature.

Observation of the standardised loadings of each item on their symptom dimension prove that most items had acceptable loadings ($\geq .60$). Only item 43 on social phobia (*feeling uneasy in crowds, such as shopping or at a movie*) demonstrated a poor loading for the validation sample (0.526). Despite this limitation, most scales reported acceptable levels of internal reliability with alpha

coefficients higher than 0.70 (Nunnally, 1978). Indeed, the current factor structure could be used by other researchers as a parsimonious, stable, and reliable measure of psychopathology for psychotic cases with strong indicators for each symptom dimension. The brevity, reliability and stability of the instrument could therefore be of interest to other researchers. It is important to keep in mind that the BSI is a copyrighted instrument and use of the instrument must be in the prescribed format and on the original form provided by the publisher. The measure suggested in this study is only appropriate for use in psychosis research where many measures are used, and parsimonious indicators are sought.

Overall, however, the robustness of the results was evidenced by successfully cross-validating the model to an independent sample. Despite the results demonstrating heterogeneity between the samples, the measurement model still proved to be invariant suggesting the factor structure to be stable despite slight variations in sample characteristics. The significant differences in psychopathology based upon the symptom dimensions (see Table 4.4) between the samples, suggest that the measures are sensitive to differences in the organisation. The fact that the cross-validation sample had higher scores on all dimensions compared to the validation sample still provides good evidence for sensitivity of measurement, since this sample was measured earlier on in the course of the illness. The validation sample was assessed during a "stabilisation period" of the illness (Edwards et al., 1999) and had come from a younger catchment population and had shorter duration of onset of illness.

In summary, a theoretically sound and empirically robust measure of psychopathology of subjects with an early psychosis was developed. This measurement model proved stable between two independent samples. It is with

confidence the research moves to the next chapter where the structural relations between the six dimensions will be examined over time.

CHAPTER V

STRUCTURAL MODEL

In the previous chapter, the development of a 6-factor model of psychosis was proposed. In this chapter, a structural model is examined which identifies the structural relations between pairs of constructs from the measurement model. In order to propose a plausible structural model, both theoretical and empirical research needs to be considered (Byrne, 1994). A theoretically plausible model is considered if it provides a parsimonious explanation from the data. In other words, the proposed structural model is efficient in explaining the data and that alternative proposed models are inadequate both in terms of theoretical and empirical evidence.

Within the measurement model, some discussion was raised about the six separate components of the measurement model (i.e., anxiety, cognition, depression, paranoia, phobia, and somatic concern) and how they may contribute to separate theories surrounding the development of a psychotic illness. These symptoms were then tested as a 6-factor measurement model with each construct describing one aspect of the psychotic experience. According to Anderson and Gerbing (1988), the first procedure is to determine the overall fit of the model. The second, the focus of this chapter, is to examine the causal relationships between the constructs. In order to do this, both direct and mediating effects specified in the selected model are tested. These analytical concepts will be explored within the methodology.

Methodology

Structural Sample Characteristics.

Due to sample attrition over time, not all of the original baseline data of 244 cases used to develop the measurement model (see Chapter IV) could be used in the structural model. The concern could be that those cases that satisfy completion of each indicator item at each time point might be different from those cases that dropped out of the study. To test whether cases in the structural sample are the same as those who have dropped out of the study, comparisons will be made on their patient characteristics.

Independent *t*-tests will be performed on the continuous variables of age, and education (ie, 1 = some secondary, 2 = secondary, 3 = trade or technical diploma, 4 = tertiary diploma, 5 = tertiary incomplete, and 6 = tertiary degree). Chi-square tests for categorical variables using the Pearson chi-square will be performed on marital status (i.e., 1 = once coupled, 2 = never coupled) and living situation (i.e., living with parents yes/no; living with spouse yes/no; living alone yes/no). Country of birth categories will be collapsed into Australia and New Zealand, Asia, Europe and Other Countries. DSM-III-R diagnosis (APA, 1987) was collapsed with schizophrenia and schizophreniform diagnosis in one group, since previous research suggests that the majority of patients with schizophreniform diagnosis later develop the diagnosis of schizophrenia (Zarate et al., 2000). The second group encompasses other psychotic disorders which include schizoaffective disorder, delusional disorder, bipolar affective disorder with psychotic features, depressive disorder with psychotic features and psychotic disorders not otherwise specified.

In addition to patient demographics, duration of prodromal features, duration of prior psychotic symptoms, duration of hospital stay in days and maximum daily Chlorpromazine dose were compared between the sample used to test the structural model and excluded cases using independent *t*-tests. Chlorpromazine dose acts as an equivalent dose converter for the different neuroleptic medications prescribed to cases. By converting medication to a common substance, comparisons can be made on the amount of medication prescribed to patients. The conditions of prodromal features and duration of psychotic symptoms were determined under the Royal Park Multi-Diagnostic Instrument for Psychosis (RPMIP). This instrument assesses prodromal symptoms in first-episode patients with demonstrable reliability and validity (McGorry et al., 1990) and was assessed at entry into the Early Psychosis Prevention and Intervention Centre (EPPIC) service.

To test for equivalence in psychopathology the 6 dimensions were compared between the structural sample and the excluded cases at each time point using independent *t*-tests.

Measure

As described in the previous chapter, 19 items describing 6 factors were used for the measurement model. These factors include anxiety, cognition, depression, paranoia, phobia and somatic concern. As previously noted, Bollen (1989) and Kline (1998) suggested that a minimum of two items is sufficient to define a construct and meet the requirements for the identification of multi-factorial measurement and structural models. Bentler and Chou (1987) argued that three items better described a

latent construct. In this instance, three items were used to define each construct except cognition consisting of four items. Four items were used for the cognition factor due to each items high factor loadings, and particular interest in the literature on the cognitive aspects of psychotic experience. With model identification it is suggested by Kline (1998) that the number of observations should be equal to, or exceed, the number of estimated parameters and that each latent variable has a scale.

Table 4.9 identifies the items used from the Brief Symptom Inventory (BSI) (Derogatis, 1982) which consist of the measurement model. All constructs showed good internal reliability with alpha coefficient ranging from $\alpha = .72$ to $\alpha = .83$ for the validation sample and $\alpha = .73$ to $\alpha = .86$ for the cross-validation sample (see Table 4.13).

To test for internal consistency with the structural model, alpha coefficients for each of the 6 symptom dimensions were calculated over the three time series.

Direct and Mediating Effects

Researchers in psychology are recognising the importance of studying the influence of mediation effects (James & Brett, 1984). Mediation effects occur when " ...the influence of an antecedent is transmitted to a consequence through an intervening mediator" (James & Brett, 1984, p. 307). In the present case, the concern is whether a condition over time has a "snowballing effect". That is the effect, for example, of depression at Time 1 influences the effects of depression at Time 3, in addition to the effects at Time 2 on Time 3. This cumulative effect is suggestive of a

momentum in the psychological state that gathers pace through time. The purpose of this procedure is to examine the dynamical relationship of constructs over time. To assume that constructs operate in a linear "domino effect" where one time wave influences the next may be too simplistic. With structural equation modelling, it is possible to examine the influence of constructs over time from any time point in the sequence. By examining the effects of Time 1 on Time 3 the researcher can make some judgement as to whether cross influential effects (the influence of 2 or more constructs across time), need to be understood in relation to how a particular symptom develops over time. Some psychological states may have a domino effect, where the previous time influence the subsequent time wave, or they may in fact have an cumulative developmental effect where the constructs expressed at previous points in time continue to effect the expression of the construct. With the emergence of the neurodevelopmental hypothesis of schizophrenia (Murray et al., 1999), concerns of an cumulative effect of symptomatology over time is an important consideration in the research.

To test the mediating effects, a process recommended by Bollen (1989) was employed. The process involves comparing nested models, differing in the number of estimated parameters that are statistically compared with chi-square difference tests. The chi-square difference test takes into account the differences in degrees of freedom for competing models. In relation to constructs over a 3 wave time series, the parameter estimate from Time 1 to Time 3 is freed for calculation. Where there is no statistical difference in chi-square between the models relative to the difference in degrees of freedom (i.e., df = 1), the more parsimonious model is preferred. That is, the model with the fewer number of estimated paths is preferred. A non-significant chi-square from Time 1 (T1) to Time 3 (T3) ($_{\gamma}2,1$) indicates that Time 1 does not have

a direct effect on Time 3 but is mediated by Time 2 (T2) ($_{\gamma}1,1$). A significant chi-square would indicate that Time 1 has a direct influential effect on Time 3 in addition to the effects measured at Time 2. This is expressed in the following figure.

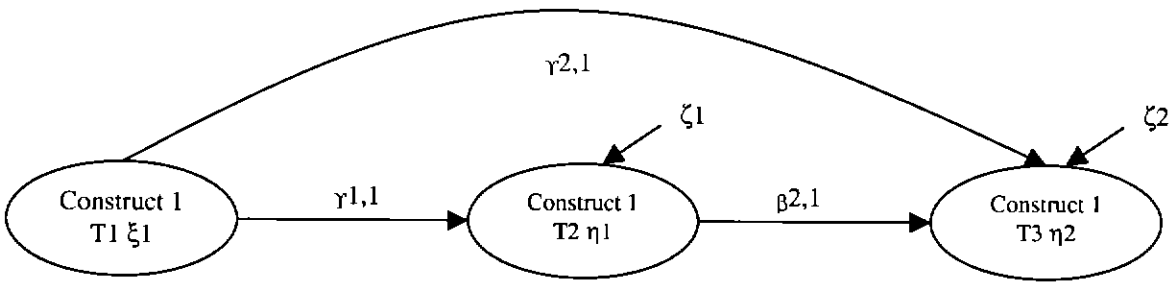

Figure 5.1 Model 1: Mediation model of one construct over time.

Note: measured indicators, error terms, and disturbance terms not shown for ease of interpretation. T1 = Time 1 baseline, T2 = Time 2 6 months from baseline, T3 = Time 3 12 months from baseline.

Each construct is examined individually over time. With the full model (saturated model) calculated first (demonstrated in Figure 5.1) all parameter estimates leading to T3 are set free (i.e. calculated). The saturated model serves as the baseline for the mediation test. First, path $\gamma_{1,1}$ is fixed to zero while path $\gamma_{2,1}$ is set free. If the difference test between the saturated model and the alternative model (1 df) is statistically significant it suggests that $\gamma_{1,1}$ is important to the model. Second, path $\gamma_{1,1}$ is set free while path $\gamma_{2,1}$ is fixed to zero. If the difference test between the saturated model and the second alternative model (with 1 df) is not statistically significant, it suggests that the $\gamma_{2,1}$ is not important in the model, confirming the mediation hypothesis.

To recapitulate, the first assessment (T1), is at the stabilisation period usually 6 to 8 weeks after the initial assessment of the subject being treated (Edwards et al.,

1999; McGorry et al., 1996). The subsequent assessments are 6 and 12 months following the stabilisation period. Only those cases that have been successfully followed up on all indicator items within the measurement model across the three time points will be examined. Those cases that do not satisfy these conditions are list wise deleted from the data set.

Cross-lagged Effects

The central focus of the thesis is to examine the dynamic relationship between psychological states expressed by subjects with a psychotic condition. More specifically, the intention is to investigate the causal predominance of cognitive symptoms over time. As stated before, much of the literature in recent decades has focused on the contribution of cognitive processing with regard to the progression and course of schizophrenia and other psychotic spectrum disorders (Goldberg & Weinberger, 1995; Heaton et al., 2001; Ngan & Liddle, 2000). The major model to have emerged in the literature has posited the "neurodevelopmental hypothesis" where it is argued that " ...subtle anomalous brain development occurs in utero which reveals itself symptomatically, years later, as the heterogenous symptoms of schizophrenia" (Kotrla, Sater, & Weinberger, 1997 p. 187). In this model, brain abnormalities occur at an early stage of foetal development and the psychotic experience can be triggered from both pre/perinatal and post-natal markers (Keshavan, 1997). Such pre and peri natal markers postulated have included famine, malnutrition, viral infection and obstetric complications most notably hypoxia (Jablensky, 1997).

The difficulty with such a hypothesis is that it is difficult to provide supportive evidence because in part " ...normal brain development is not amenable to direct investigation" (Kotrla et al., 1997 p. 187). What is known however, is that neuroimaging technologies consistently demonstrate abnormalities in the structure or function of multiple brain regions in schizophrenia, most notably in the temporal and frontal lobes (Kotrla et al., 1997). Other investigators have suggested that poor connectivity of the neural networks in the brain contribute to abnormalities that are expressed in symptoms typical of schizophrenia (Bullmore, O'Connell, Frangou, & Murray, 1997).

Although such studies suggest a neuropathological aetiology of psychosis, very little research has focused on the interactive nature of how such pathology expresses itself as symptoms within a dynamical system. The intention is to link the construct expression of cognitive deterioration with the other construct dimensions to model dynamically over time, whether these constructs have a cross influential effect. In other words, does cognition causally influence other psychological states over time, or in turn, do the other psychological states causally influence the expression of cognition over time.

Three time waves of data are required to truly examine a causal hypothesis (Willett, 1988). With two time waves of data, a researcher can only examine the link between one time sequence to the next. With three or more time waves, the researcher is able to examine mediation as well as direct effects that precede the previous condition of interest (i.e., the effects of Time 1 on Time 3). A reciprocal causation amongst variables can be established (Padhazur & Pedhazur-Schmelkin, 1991), since the influences of one dimension can be crossed or inverted in the next

time wave and so their cross interaction can be examined. Such a cross-lagged effect is best demonstrated in Figure 3.2. The analysis proceeds by creating a full cross-lagged model where direct gamma paths are lead from the exogenous variables at Time 1 to calculate their influence on the same dimensions at Time 2 ($\gamma_{1,1}; \gamma_{2,2}$). In addition, cross-lagged paths from Time 1 to Time 2 between the constructs are also examined ($\gamma_{2,1}; \gamma_{1,2}$). This process is repeated in the next time wave, but in this instance they are beta paths since the influence of the endogenous variables at Time 2 are examined for their influence at Time 3 within each construct ($\beta_{3,1}, \beta_{4,2}$) and across constructs ($\beta_{4,1}; \beta_{3,2}$). Finally the direct influential paths leading from Time 1 to Time 3 are also examined ($\gamma_{3,1}, \gamma_{4,2}$). Significant standardised path coefficients are examined for their direction (i.e., cumulative : + path, or suppressive – path) and strength. Based upon Kline's work, standardised path coefficients with absolute values less than 0.10 indicate a 'small' effect with values around 0.30 indicative of a 'medium' effect. Absolute values of 0.50 or more are considered 'large' effects (Kline, 1998).

With longitudinal modelling, it is likely that the measurement error of repeated measures covary and that to account for this, these error terms are correlated in the modelling procedure (Wheaton, Muthen, Alwin, & Summers, 1977). Correlated errors reflect measurement error in addition to indicator-specific variation (Ecob, 1987), and hence the measurement errors were set to covary with each time wave. This procedure does not affect interpretation of the results (Byrne et al., 1989; Hoyle & Panter, 1995; Milsap & Hartog, 1988). In addition synchronous covariation between constructs were modelled, as it was improbable that all covariances between the endogenous variables could be explained through the structural model.

Within Chapter II, some discussion was raised in relation to how the aetiological cause of psychosis leads towards brain pathophysiology that leads to the expression of phenotypic psychological states. It is proposed that the constructs expressed under the measurement model give rise to this symptomatic expression of psychosis. With research in psychosis pointing towards a neurodevelopmental hypothesis of brain pathophysiology, it seems appropriate to examine the causal influence of that psychological state which expresses brain pathophysiology the most; cognitive performance (i.e., *trouble remembering things, difficulty making decisions, your mind going blank* and *trouble concentrating*). By pairing this psychological state with others the attempt is two fold. First, arguments laid down in the thesis suggest that cognitive deterioration plays a cardinal role in the course of psychosis. Recent literature argues that this stems from the neurodevelopmental hypothesis (Keshavan & Murray, 1997), of a disorder that has its origin in pre and peri natal development (Kotrla et al., 1997), and that is later expressed in pathology in late adolescence and early adult hood (Carbone, Harrigan, McGorry, Curry, & Elkins, 1999; McGorry, Harrigan, Amminger, Norman, & Malla, 2001; McGorry et al., 1995a; Yung et al., 1999). It is possible that as these psychological states are expressed they may interact with each other in a causal dynamic relationship that patterns and drives the psychotic illness in a predominating fashion centrally around worsening cognitive states. It is with this argument in mind that the cross-lagged effects of cognition on the other constructs are examined within the structural model.

The sample size of the structural model does not represent robust conditions with a sample size lower than 250. A more appropriate "2-index presentation strategy" has been suggested by Hu and Bentler (1999) who recommended that " ...Comparative Fit Index (CFI) < .95 and standardised root mean squared residual

(SRMR) > .09 (or .10) may be more appropriate when N ≤ 250 if committing Type I error is less desirable" (p. 25). With this in mind, these indices will be presented for the cross-lagged structural models.

Hypothesis

The first series of hypotheses will consider the structural sample patient characteristics and the direct and mediating effects of psychological states over time. On the basis of the arguments presented, the following hypothesis are considered:

$H_{5.1}$: There will be no difference between the original baseline sample (n = 244) and the structural model sample (n = 145) on patient characteristics of age, gender, marital status, living situation, education, and country of birth.

$H_{5.2}$: There will be no difference between the original baseline sample (n = 244) and the structural model sample (n = 145) on duration of prodromal features, psychotic states, hospitalisation and maximum daily chlorpromazine equivalent dose.

$H_{5.3}$: There will be no difference between the original baseline sample (n = 244) and the structural model sample (n = 145) on psychopathology over each time series on anxiety, depression, cognition, paranoia, phobia and somatic concern.

$H_{5.4a}$: Anxiety at Time 1 has a direct influence on anxiety at Time 2.

$$\eta_1 = \gamma_{1,1}\, \xi_{1,1} + \zeta_1$$

Where the endogenous variable at Time 2 is denoted by eta η_1 and the path coefficient leading from Time 1 denoted by gamma $\gamma_{1,1}$ with the disturbance term expressed as zeta ζ_1.

$H_{5.4b}$: Anxiety at Time 1 and anxiety at Time 2 have direct influences on anxiety at Time 3.

$$\eta_3 = \beta_{3,1}\,\eta_1 + \gamma_{3,1}\,\xi_1 + \zeta_3$$

Where the endogenous variable at Time 3 is denoted by eta η_3 and the path coefficient leading from Time 1 denoted by gamma $\gamma_{3,1}$, and the path coefficient leading from Time 2 beta $\beta_{3,1}$ with the disturbance term expressed as zeta ζ_3.

$H_{5.5a}$: Cognition at Time 1 has a direct influence on cognition at Time 2.

$$\eta_1 = \gamma_{1,1}\,\xi_{1,1} + \zeta_1$$

$H_{5\,5b}$: Cognition at Time 1 and cognition at Time 2 have direct influences on cognition at Time 3.

$$\eta_3 = \beta_{3,1}\,\eta_1 + \gamma_{3,1}\,\xi_1 + \zeta_3$$

$H_{5.6a}$: Depression at Time 1 has a direct influence on depression at Time 2.

$$\eta_1 = \gamma_{1,1}\,\xi_{1,1} + \zeta_1$$

$H_{5.6b}$: Depression at Time 1 and depression at Time 2 have direct influences on depression at Time 3.

$$\eta_3 = \beta_{3,1}\,\eta_1 + \gamma_{3,1}\,\xi_1 + \zeta_3$$

$H_{5.7a}$: Paranoia at Time 1 has a direct influence on paranoia at Time 2.

$$\eta_1 = \gamma_{1,1}\, \xi_{1,1} + \zeta_1$$

$H_{5.7b}$: Paranoia at Time 1 and paranoia at Time 2 have direct influences on paranoia at Time 3.

$$\eta_3 = \beta_{3,1}\, \eta_1 + \gamma_{3,1}\, \xi_1 + \zeta_3$$

$H_{5.8a}$: Phobia at Time 1 has a direct influence on phobia at Time 2.

$$\eta_1 = \gamma_{1,1}\, \xi_{1,1} + \zeta_1$$

$H_{5.8b}$: Phobia at Time 1 and phobia at Time 2 have direct influences on phobia at Time 3.

$$\eta_3 = \beta_{3,1}\, \eta_1 + \gamma_{3,1}\, \xi_1 + \zeta_3$$

$H_{5.9a}$: Somatic concern at Time 1 has a direct influence on somatic concern at Time 2.

$$\eta_1 = \gamma_{1,1}\, \xi_{1,1} + \zeta_1$$

$H_{5.9b}$: Somatic concern at Time 1 and somatic concern at Time 2 have direct influences on somatic concern at Time 3.

$$\eta_3 = \beta_{3,1}\, \eta_1 + \gamma_{3,1}\, \xi_1 + \zeta_3$$

The second series of hypotheses concerns the cross-lagged effects between cognition and the other symptoms.

On the basis of the arguments presented, the following hypotheses are considered in the null position:

$H_{5.10a}$: There will be no cross-lagged effect between cognition and anxiety between Time 1 and Time 2.

$H_{5.10b}$: There will be no cross-lagged effect between cognition and anxiety between Time 2 and Time 3.

$H_{5.11a}$: There will be no cross-lagged effect between cognition and depression between Time 1 and Time 2.

$H_{5.11b}$: There will be no cross-lagged effect between cognition and depression between Time 2 and Time 3.

$H_{5.12a}$: There will be no cross-lagged effect between cognition and paranoia between Time 1 and Time 2.

$H_{5.12b}$: There will be no cross-lagged effect between cognition and paranoia between Time 2 and Time 3.

$H_{5.13a}$: There will be no cross-lagged effect between cognition and phobia between Time 1 and Time 2.

$H_{5.13b}$: There will be no cross-lagged effect between cognition and phobia between Time 2 and Time 3.

$H_{5.14a}$: There will be no cross-lagged effect between cognition and somatic concern between Time 1 and Time 2.

$H_{5\ 14b}$: There will be no cross-lagged effect between cognition and somatic concern between Time 2 and Time 3.

Results

Structural Sample Characteristics

To construct the structural sample, cases were drawn from the original sample of the 244 cases from the Victorian Early Psychosis Prevention and Intervention Centre (EPPIC) data (McGorry et al., 1996; Yung et al., 1999). To be eligible for inclusion in the structural sample, cases were required to have completed all 19 items within the measurement model (see Chapter IV) across the three time waves (i.e. stabilisation, six and twelve month follow-up assessments). Cases were dropped from the analysis if they failed to complete any of the 19 items across the three time frames.

The structural sample consisted of 145 cases that successfully completed all 19 items within the measurement model across the three time waves of data collection. These cases were compared for patient characteristics against the excluded cases (n = 121). Table 5.1 indicates that age, age at onset, and education distributions are invariant between cases used in the structural model and excluded cases. Equivalent proportions of cases of schizophrenia and schizophreniform disorder and other psychosis between the two samples were also observed, along with cases living with their parents, alone or with spouses. There was also no difference between the samples on country of birth. The excluded sample however had a higher proportion of cases that were married, separated or divorced (i.e., once coupled).

Table 5.1

Patient Characteristics of Cases Used in the Structural Model Compared to Excluded Cases

	Structural Cases (n = 145)	Excluded Cases (n = 121)	df	Analysis	*p*
Patient Characteristic				*t*	
Age	21.79 (± 3.57)	22.31 (± 3.58)	264	-1.218	.224
Age at onset	21.32 (± 3.37)	21.86 (± 3.63)	264	-1.243	.215
Educational Level	3.49 (± 1.95)	3.30 (± 2.28)	264	.740	.460
				χ^2	
Schizophrenia	79 (53.4%)	66 (55.9%)	1	.173	.678
Other Psychoses	69 (46.6%)	52 (44.1%)			
Male	106 (54.4%)	89 (45.6%)	1	.007	.934
Female	39 (54.9%)	32 (45.1%)			
Marital Status					
Once Coupled	12 (8.3%)	28 (23.1%)	1	11.407	.001
Never Married	133 (91.7%)	93 (76.9%)			
Living Status					
Not living with parent	41 (28.3%)	104 (71.7%)	1	3.327	.068
Living with parent	47 (38.8%)	74 (61.2%)			
Not living alone	134 (92.4%)	107 (88.4%)	1	1.229	.268
Living alone	11 (7.6%)	14 11.6%)			
Not living with spouse	136 (93.8%)	105 (86.8%)	1	3.813	.051
Living with spouse	6 (6.2%)	16 (13.2%)			
Country of Birth					
Australian or New Zealand	129 (89.0%)	98 (81.0%)	3	4.349	.226
European	4 (2.8%)	7 (5.8%)			
Asia	7 (4.8%)	12 (9.9%)			
Other Country	5 (3.4%)	4 (3.3%)			

Tests for psychopathology on the six symptom dimensions across each time wave (see Table 5.2) proved invariant between the structural sample and excluded cases except for the somatic concern dimension at Time 2, with excluded cases demonstrated higher levels of somatic concern than cases used in the structural sample.

Table 5.2
Psychopathology on the Six Dimensions of Psychosis Compared Between Cases Used in the Structural Model and Excluded Cases

Symptom Dimension	Structural Cases	Excluded Cases	df	*t*	*p*
Depression Time 1	2.16 (± 1.09) (n=145)	2.10 (± 1.13) (n=106)	249	.427	.670
Depression Time 2	1.82 (± .92) (n=145)	1.91 (± 1.06) (n=40)	183	-.548	.584
Depression Time 3	1.77 (± 1.00) (n=145)	1.85 (± 1.12) (n=32)	175	-.374	.709
Phobia Time 1	1.96 (± .92) (n=145)	1.96 (± 1.03) (n=106)	249	-.020	.984
Phobia Time 2	1.65 (± .76) (n=145)	1.74 (± .90) (n=39)	182	-.616	.539
Phobia Time 3	1.62 (± .76) (n=145)	1.76 (± .99) (n=32)	175	-.616	.387
Somatic Concern Time 1	1.51 (± .75) (n=145)	1.62 (± .91) (n=107)	250	-1.042	.298
Somatic Concern Time 2	1.38 (± .64) (n=145)	1.72 (± 1.06) (n=40)	183	-2.48	.014
Somatic Concern Time 3	1.38 (± .53) (n=145)	1.56 (± .73) (n=32)	175	-1.59	.112
Anxiety Time 1	1.71 (± .76) (n=145)	1.75 (± .85) (n=107)	250	-.363	.717
Anxiety Time 2	1.53 (± .79) (n=145)	1.61 (± .84) (n=40)	183	-.584	.560
Anxiety Time 3	1.52 (± .68) (n=145)	1.63 (± .74) (n=32)	175	-.816	.416
Paranoia Time 1	1.92 (± 1.03) (n=145)	1.93 (± .99) (n=106)	249	-.100	.921
Paranoia Time 2	1.71 (± .94) (n=145)	1.89 (± .97) (n=39)	182	-1.06	.289
Paranoia Time 3	1.70 (± .88) (n=145)	1.90 (± 1.12) (n=32)	175	-1.11	.265
Cognition Time 1	2.13 (± .91) (n=145)	1.88 (± .92) (n=105)	248	-.626	.532
Cognition Time 2	1.88 (± .92) (n=145)	2.14 (± .88) (n=39)	182	-1.42	.152
Cognition Time 3	1.90 (± .88) (n=145)	2.01 (± 1.03) (n=32)	175	-.623	.534

Note: BSI scale for each item has been transformed from 0 to 4 to 1 to 5.

Examination of the distributions of duration of prodrome, duration of psychosis prior to treatment, hospitalisation and chlorpromazine dose revealed these variables to be highly skewed (2.649, 5.963, 4.327 and 2.520 respectively). To normalise the data, each variable underwent square-root transformations suggested by Tabachnick and Fiddell (1996). Comparisons between the two samples demonstrated that the duration of prodrome, duration of prior psychosis determined under the Royal Park Multi-Diagnostic Instrument for Psychosis (RPMIP) and chlorpromazine dose

were equivalent (see Table 5.3). Duration of hospitalisation however was statistically higher for those cases selected in the structural model.

Table 5.3
Comparison of Length of Prodromal Features, Psychotic Features Prior to Admission, Hospitalisation in Days, and Maximum Daily Chlorpromazine Equivalent Dose Between Cases Used in the Structural Model and Excluded Cases

	Structural Cases	Excluded Cases	df	t	*p*
Duration of prodrome [a]	375.48 (±451.07) (n = 121)	485.86 (±694.56) (n = 93)			
Duration of prodrome [b]	16.70 (± 9.85) (n = 121)	18.25 (± 12.42) (n = 93)	212	-.1.01	.311
Duration of prior psychosis [a]	157.83 (±332.79) (n = 145)	130.07 (±239.69) (n = 121)			
Duration of prior psychosis [b]	9.61 (±8.11) (n = 145)	8.68 (± 7.42) (n = 121)	264	.968	.334
Duration of Hospitalisation [a]	38.96 (±45.90) (n = 123)	26.42 (±24.46) (n = 117)			
Duration of Hospitalisation [b]	5.29 (±3.32) (n = 123)	4.33 (±2.76) (n = 117)	238	2.41	.017
Chlorpromazine Dose [c]	364.57 (±325.81) (n = 145)	320.68 (±267.79) (n = 117)			
Chlorpromazine Dose [b]	17.50 (±7.65) (n = 145)	16.6167(±6.70) (n = 117)	255	.977	.329

Note: Duration period in days; data missing on some structural cases. [a] Raw scores in days; [b] square root transformation; [c] daily maximum chlorpromazine dose.

Internal consistency of the 6 dimensions within the structural model

Alpha coefficients were calculated to assess the internal reliability of the 6 dimensions at each of the three time points in the structural model. Based upon Nunnally's criteria (Nunnally, 1978) a broad range of internal consistency was observed from poor $\alpha = .511$ to good $\alpha = .838$ (see Table 5.4).

Table 5.4
Internal Reliability of the Symptom Dimensions for Each Time Series Within the Structural Model (n = 145)

Dimensions	α	Dimensions	α
Depression Time 1	.807	Anxiety Time 1	.588
Depression Time 2	.796	Anxiety Time 2	.711
Depression Time 3	.863	Anxiety Time 3	.576
Phobia Time 1	.711	Somatic Concern Time 1	.686
Phobia Time 2	.614	Somatic Concern Time 2	.653
Phobia Time 3	.597	Somatic Concern Time 3	.511
Cognition Time 1	.778	Paranoia Time 1	.804
Cognition Time 2	.838	Paranoia Time 2	.812
Cognition Time 3	.800	Paranoia Time 2	.737

Direct and Mediating Effects

Figure 5.1, demonstrates schematically the saturation model for the test for mediation effects. Demonstration of the mediation effect requires the estimation of the structural model, where all three path parameters are freely estimated; $\gamma_{1,1}$ (path from T1 to T2), $\gamma_{3,1}$ (path from T1 to T3) and $\beta_{3,1}$ (path from T2 to T3). This model is then tested as the baseline model on which to compare the direct and mediation models. In the direct model the parameter $\gamma_{1,1}$ (T1 to T2) is constrained to zero, while the other two are freely calculated. In the mediation model the parameters $\gamma_{2,1}$ (T1 to T3) is constrained to zero with other parameters freely calculated. Difference tests based on the chi-square test with 1 degree of freedom between the mediation model and the structural model would determine whether the constrained path ($\gamma_{2,1}$) is important to the model. A non-statistically significant difference between the two models suggests the presence of a mediation effect. Each of the 6 dimensions was independently examined for their direct and mediating effects. These are displayed in Table 5.5.

Table 5.5
Tests of Mediation Effects for Each Symptom Over Time

Model	χ^2	df	Δ df	Δ χ^2	RCFI	RMSEA (CI)
Anxiety						
Saturated	22.417	18			0.998	0.042 (0.000 - 0.089)
$\gamma_{1,1}$= * ; $\gamma_{2,1}$= * ; $\beta_{2,1}$ = *						
Direct Effect	45.410	19	1	22.993 *	0.925	0.099 (0.062 - 0.135)
$\gamma_{1,1}$= 0 ; $\gamma_{2,1}$= * ; $\beta_{2,1}$ = *						
Mediation Effect	22.497	19	1	0.008 ns	1.000	0.036 (0.000 - 0.084)
$\gamma_{1,1}$= * ; $\gamma_{2,1}$= 0; $\beta_{2,1}$ = *						
Cognition						
Saturated	77.281	43			0.965	0.075 (0.047 - 0.100)
$\gamma_{1,1}$= * ; $\gamma_{2,1}$= * ; $\beta_{2,1}$ = *						
Direct Effect	101.879	44	1	24.598 *	0.920	0.096, (0.071 - 0.119)
$\gamma_{1,1}$= 0 ; $\gamma_{2,1}$= * ; $\beta_{2,1}$ = *						
Mediation Effect	77.759	44	1	0.478 ns	0.964	0.073 (0.045 - 0.099)
$\gamma_{1,1}$= * ; $\gamma_{2,1}$= 0; $\beta_{2,1}$ = *						
Depression						
Saturated	31.721	18			0.965	0.073 (0.026 - 0.113)
$\gamma_{1,1}$= * ; $\gamma_{2,1}$= * ; $\beta_{2,1}$ = *						
Direct Effect	59.635	19	1	27.914 *	0.941	0.122 (0.087 - 0.157)
$\gamma_{1,1}$= 0 ; $\gamma_{2,1}$= * ; $\beta_{2,1}$ = *						
Mediation Effect	36.615	19	1	4.894 *	0.987	0.081 (0.039 - 0.119)
$\gamma_{1,1}$= * ; $\gamma_{2,1}$= 0; $\beta_{2,1}$ = *						
Paranoia						
Saturated	43.746	18			0.956	0.100 (0.062 - 0.137)
$\gamma_{1,1}$= * ; $\gamma_{2,1}$= * ; $\beta_{2,1}$ = *						
Direct Effect	57.449	19	1	13.703 *	0.916	0.119 (0.084 - 0.154)
$\gamma_{1,1}$= 0 ; $\gamma_{2,1}$= * ; $\beta_{2,1}$ = *						
Mediation Effect	46.314	19	1	2.568 ns	0.950	0.100 (0.063 - 0.136)
$\gamma_{1,1}$= * ; $\gamma_{2,1}$= 0; $\beta_{2,1}$ = *						
Phobia						
Saturated	38.750	18			0.962	0.090 (0.050 - 0.128)
$\gamma_{1,1}$= * ; $\gamma_{2,1}$= * ; $\beta_{2,1}$ = *						
Direct Effect	80.992	19	1	42.242 *	0.836	0.151 (0.117 - 0.184)
$\gamma_{1,1}$= 0 ; $\gamma_{2,1}$= * ; $\beta_{2,1}$ = *						
Mediation Effect	38.751	19	1	0.001 ns	0.966	0.085 (0.046 - 0.123)
$\gamma_{1,1}$= * ; $\gamma_{2,1}$= 0; $\beta_{2,1}$ = *						
Somatic Concern						
Saturated	28.431	18			0.991	0.064 (0.000 - 0.105)
$\gamma_{1,1}$= * ; $\gamma_{2,1}$= * ; $\beta_{2,1}$ = *						
Direct Effect	38.990	19	1	10.559 *	0.929	0.086 (0.046 - 0.123)
$\gamma_{1,1}$= 0 ; $\gamma_{2,1}$= * ; $\beta_{2,1}$ = *						
Mediation Effect	32.983	19	1	4.552 *	0.966	0.072 (0.026 - 0.111)
$\gamma_{1,1}$= * ; $\gamma_{2,1}$= 0; $\beta_{2,1}$ = *						

Note: * = free parameter, 0 = parameter fixed to 0; RCFI = Robust Confirmatory Fit Index, CFI =Comparative Fit Index, RMSEA = Root Mean Square Error of Approximation, CI = Confidence Interval; ns = non-significant; * = $p < 0.05$; Critical $\chi^2 = 3.84$.

Examination of Table 5.5 indicates that both depression and somatic concern have significantly contributing gamma paths leading from Time 1 to Time 3 ($\gamma_{2,1}$). These results suggest that both these psychological states have a snowballing effect where the conditions at Time 1 effects the conditions of the states at Time 3. This dynamic relationship occurs where the construct has a "wave that gathers momentum" over time where the preceding condition influences subsequent conditions. These relationships do not suggest a linear "domino effect" where the conditions that precede the immediate condition (i.e., Time 1 influencing Time 3) have no direct impact. The other psychological states provide this type of profile over time, where only the preceding condition has any causal influence on the immediate condition of interest (i.e., Time 1 to Time 2, and Time 2 to Time 3). In other words, the other conditions of anxiety, phobia, paranoia and cognition act within a "domino effect" where only the preceding condition has a direct causal influence on the condition of interest. Allowing for the Time 1 to Time 3 gamma path to be freely calculated, most conditions do not account for a significant increase in the chi-square or better explanation to the data. This means that in most instances, the direct effect of Time 1 on to Time 3 apart from the conditions of depression and somatic concern, act in a causally linear pattern.

In conclusion, hypotheses $H_{5\,6b}$ and $H_{5.9b}$ have been supported where the condition of depression at Time 1 has a direct effect on the condition of depression at Time 3, as well as the condition of somatic concern at Time 1 have a direct effect on the condition of somatic concern at Time 3.

In relation to direct effects, all conditions demonstrated direct causal effects where by observing the standardised path coefficients conditions at Time 1

contributed significantly to conditions at Time 2. This means that hypotheses $H_{5.4a}$, $H_{5.5a}$, $H_{5\,6a}$, $H_{5.7a}$, $H_{5\,8a}$ and $H_{5.9a}$ were all confirmed. Such results provide evidence of a domino effect for anxiety, cognition, paranoia and phobia (depression and somatic concern had mediating as well as direct effects), where only the preceding condition affects the subsequent condition.

Cross-lagged Effects

The next stage of the investigation was to examine the cross-lagged effects of cognition on the other constructs. As explained previously, cognitive deterioration appears to be cardinal to the psychotic experience (Bilder et al., 2000; Chadwick et al., 1997; Frith, 1987; 1992; Heaton et al., 2001; Hemsley, 1993). In order to reduce the inflation of chance interaction effects between all combination of constructs, the focus was to concentrate on the effects of cognition on other dimensions and in turn these dimensions on cognition.

The cross-lagged model specifies the calculation of parameters not only across time with each dimension, but also across dimensions between each time frame (see Figure 3.2). For example the influences of Construct 1 at Time 1 on Construct 2 at Time 2 ($\gamma_{2,1}$). This gamma path is inverted where Construct 2 at Time 1 is examined for its influence of Construct 1 and Time 2 ($\gamma_{1,2}$). This cross influential effect is also examined at the next time wave, where the influence of Construct 1 Time 2 on Construct 2 Time 3 is examined ($\beta_{4,1}$). Once again this relationship is inverted by

examining the influences of Construct 2 at Time 2 on Construct 1 at Time 3 are examined ($\beta_{3,2}$).

Table 5.6 presents the fit indices of each modelled pair of dimensions with cognition. The Robust CFI for each model is within the acceptable range (≥ 0.95 recommended by Hu & Bentler, 1999). The standardised root mean square residual (SRMSR) is also below the .09 criterion (Hu & Benlter, 1999). The RMSEA point estimates (< .08) suggest reasonable fit. Based upon the MacCallum, Browne and Sugawara's (1996) RMSEA confidence interval criterion, the results suggest that neither the hypothesis of "close fit" or "not close fit" could be rejected. In the absence of any clearly defined criteria for comparing RMSEA values all models remained relatively viable.

Table 5.6
Fit indices of structural models with construct pairs

Cross-lagged Model	N	df	χ^2	SRMSR	RCFI	RMSEA (CI)
Anxiety and Cognition	145	162	231.273	0 062	0.995	0.055 (0.037 - 0.070)
Depression and Cognition	145	162	269.994	0.056	0.955	0.068 (0.053 - 0.082)
Paranoia and Cognition	145	162	276.423	0.065	0.949	0.070 (0.055 - 0.084)
Phobia and Cognition	145	162	268.277	0.061	0.963	0.068 (0.053 - 0.081)
Somatic Concern and Cognition	145	162	253.875	0.060	0.969	0.063 (0.047 - 0.077)

Note: SRMR = standardised root mean squared residual, RCFI = Robust Confirmatory Fit Index, CFI =Comparative Fit Index, RMSEA = Root Mean Square Error of Approximation, CI = Confidence.

For evaluating the models based on the RMSEA values, sample size and power calculations are necessary (MacCallum et al., 1996). Power for each of the models was over the .80 threshold (Cohen, 1977; Ray & Vermeulen, 1999) and

calculated at 1-β = .96, while the minimum sample size requirement was set at N = 96.

The next procedure was to observe the standardised coefficients of each cross-lagged path in each model for statistical significance. Standardised coefficients can be compared to determine the relative strength of path associations (Saris, 1989). As was mentioned earlier, it has been argued by Kline (1998), that "standardised path coefficients with absolute values less than 0.10 may indicate a 'small' effect; values around 0.30 a 'medium' one; and 'large' effects may be suggested by coefficients with absolute values of 0.50 or more" (p. 149).

In addition to examining the standardised coefficients, it is also useful to calculate the squared multiple correlations for each endogenous variable. Squared multiple correlations (R^2) provide a measure of the percentage variance explained in each endogenous variable. This is calculated by subtracting the squared standardised disturbance terms from 1 (Tabachnick & Fiddell, 1996). This is expressed in the following formula: $R_j^2 = 1 - D_j^2$ (where D equals the standardised value of the disturbance term).

The following table demonstrates the standardised path coefficients for each time wave including the cross influential effects from Time 1 to Time 2 ($\gamma_{2,1}$; $\gamma_{1,2}$), and from Time 2 to Time 3 ($\beta_{3,2}$; $\beta_{4,1}$). Direct effects were also examined within each dimension from Time 1 to Time 2 ($\gamma_{1,1}$; $\gamma_{2,2}$) and Time 2 to Time 3 ($\beta_{3,1}$; $\beta_{4,2}$) and across time from Time 1 to Time 3 ($\gamma_{3,1}$; $\gamma_{4,2}$).

Not all direct paths within dimensions proved significant (see Table 5.7). Medium effects were found for phobia from Time 1 to Time 2 (.469 $p < .01$), and

cognition with the pairs of phobia, paranoia, anxiety and somatic concern (.430, $p <$ .05; .490, $p < .01$; .407, $p < .05$; .528, $p < .05$, respectively). Cognition from Time 2 to Time 3 was also significant for the phobia and paranoia pairs (.705, $p <.05$; .352, $p < .05$, respectively). Pathways of depression from Time 2 to Time 3 were also significant (1.002, $p < .01$). There was no significant lag from Time 1 to Time 3 for any of the dimensions.

Table 5.7
Standardised path estimates for the full cross lagged model for each dimension paired with paranoia

Parameter	Time 1	Time 2	Standardised Path Estimate	Parameter	Time 2	Time 3	Standardised Path Estimate
	KSI (ξ) Construct	ETA (η) Construct			ETA (η) Construct	ETA (η) Construct	
(γ1,1)	Phobia	Phobia	.699 **	(β3,1)	Phobia	Phobia	.866
(γ2,2)	Cognition	Cognition	.384 *	(β4,2)	Cognition	Cognition	.676 *
(γ3,1)	Phobia					Phobia	-.064
(γ4,2)	Cognition					Cognition	.073
(γ2,1)	Phobia	Cognition	.150	(β4,1)	Phobia	Cognition	-.062
(γ1,2)	Cognition	Phobia	-.055	(β3,2)	Cognition	Phobia	-.120
(γ1,1)	Paranoia	Paranoia	.499 **	(β3,1)	Paranoia	Paranoia	.638 **
(γ2,2)	Cognition	Cognition	.492 **	(β4,2)	Cognition	Cognition	.376 *
(γ3,1)	Paranoia					Paranoia	.138
(γ4,2)	Cognition					Cognition	.136
(γ2,1)	Paranoia	Cognition	-.002	(β4,1)	Paranoia	Cognition	.301 *
(γ1,2)	Cognition	Paranoia	- .138	(β3,2)	Cognition	Paranoia	- .089
(γ1,1)	Anxiety	Anxiety	.552 *	(β3,1)	Anxiety	Anxiety	.399
(γ2,2)	Cognition	Cognition	.399 *	(β4,2)	Cognition	Cognition	.528
(γ3,1)	Anxiety					Anxiety	.004
(γ4,2)	Cognition					Cognition	.043
(γ2,1)	Anxiety	Cognition	.118	(β4,1)	Anxiety	Cognition	.149
(γ1,2)	Cognition	Anxiety	- .081	(β3,2)	Cognition	Anxiety	.098
(γ1,1)	Depression	Depression	.577	(β3,1)	Depression	Depression	.984 **
(γ2,2)	Cognition	Cognition	.375	(β4,2)	Cognition	Cognition	.525
(γ3,1)	Depression					Depression	.185
(γ4,2)	Cognition					Cognition	.033
(γ2,1)	Depression	Cognition	.083	(β4,1)	Depression	Cognition	.142
(γ1,2)	Cognition	Depression	- .084	(β3,2)	Cognition	Depression	- .503
(γ1,1)	Somatics	Somatics	.368	(β3,1)	Somatics	Somatics	.383
(γ2,2)	Cognition	Cognition	.495 **	(β4,2)	Cognition	Cognition	.172
(γ3,1)	Somatics					Somatics	.234
(γ4,2)	Cognition					Cognition	.173
(γ2,1)	Somatics	Cognition	-..003	(β4,1)	Somatics	Cognition	.482
(γ1,2)	Cognition	Somatics	.045	(β3,2)	Cognition	Somatics	.202

Note: Somatics = Somatic Concern; * = $p < .05$ ($t > 1.96$); ** = $p < .01$ ($t > 2.58$), ns = non-significant.

Observation of the cross-lagged paths between dimensions demonstrate that only one path coefficient proved statistically significant at the $p < .05$ level. This coefficient runs from paranoia at time 2 to cognition at Time 3 (see Appendix VI). By using Kline's (1998) guidelines, this path shows a modest or 'medium' effect. This medium effect size suggests that the influences of paranoia may be susceptible to situational influences unexplained by the paranoid state.

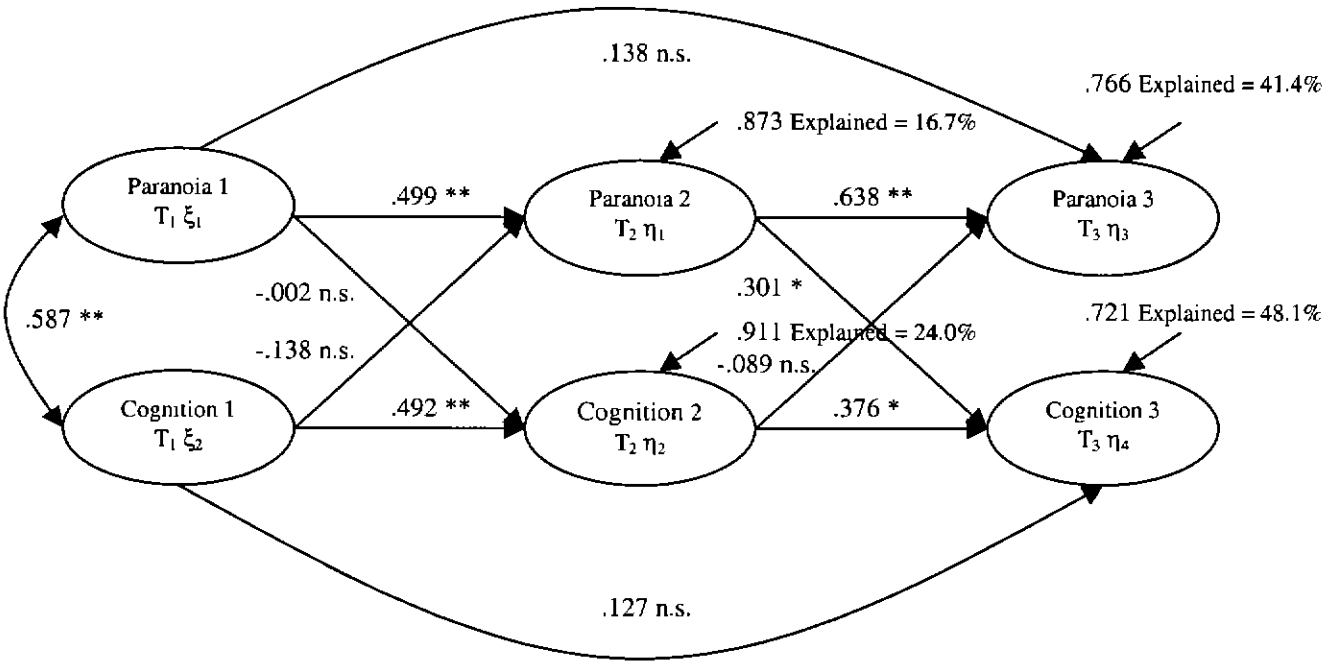

Figure 5.2 Model 1: Full cross-lagged model between Cognition and Paranoia.
Note: measured indicators, error terms, and disturbance terms not shown for ease of interpretation. T1 = Time 1, baseline; T2 = Time 2, 6 months from baseline; T3 = Time 3, 12 months from baseline. * $p < .05$, ** $p < .01$. Residual correlations at T1 = .731, T2 = .756, and T3 = .874.

To further assist in the interpretation of this cross-lagged effect, the standardised path coefficients for cognition and paranoia are represented diagrammatically in Figure 5.1. In addition to the standardised path coefficients in Figure 5.1 is the amount of variance explained (R^2) in each of the endogenous (η) variables at Time 2 (T2) and Time 3 (T3). It can be seen that a significant amount of

explained variance in the endogenous variables was explained by the exogenous variable from Time 1 to Time 2, and more so by the endogenous variables from Time 2 to Time 3. By observing the squared multiple correlations (R^2) for each endogenous variable, the first time wave explained 16.7% of variance for paranoia and 24% of the variance for cognition. By Time 3, the cumulated effects of Time 1 and Time 2, including the cross-lagged paths, accounted for 48.1% of the variance for cognition and 41.4% for paranoia. This almost doubling in the amount of explained variance from one time frame to the next indicates a cumulating effect where the previous time frame causes a linear and progressive increase in influence from one time to the next. In addition, by the second time wave the paranoid state of condition then contributes a medium effect on the cognitive condition at Time 3. In other words, paranoia at Time 2 has a cross-influential effect on cognition at Time 3. This result rejects the null position of hypothesis $H_{5.12b}$, providing a cross-lagged effect in favour of paranoia affecting cognition. All other hypotheses within the cross-lagged series (hypotheses $H_{5.10a}$ to $H_{15\ 14b}$) remain in the null position.

Dimensionality of cognition and paranoia over time

In addition to the cross-lagged effects, further analysis was conducted to examine the correlation between the dimensions of cognition and paranoia over time. To examine this, 2-first order factors, and 1-higher order factor were assessed for each time wave. Observation of the coefficients over time indicate the correlations to be high, for example for Time 1, 2, and 3 these correlations were $\Phi_{1,2} = .743$, $\Phi_{1,2} = .698$, $\Phi_{1,2} = .881$, respectively. Such high correlations between the two dimensions

particularly at Time 3 indicate that the two dimensions appear to merge as the psychotic condition proceeds (see Table 5.8), and may throw some doubt on the discriminability of the two constructs.

Table 5.8
Comparison of a Two-Factor First Order Model With an Equivalent Higher-Order Factor Model

Model	χ^2	df	S-B χ^2	RCFI	SRMR
Time 1					
Null	385.783	21	-	-	-
2-First Order Factors	42.867	13	28.114	.940	.057
1- Higher Order Factor	42.867	13	28.116	.940	.057
Time 2					
Null	472.903	21	-	-	-
2-First Order Factors	48.628	13	27.963	.938	.062
1- Higher Order Factor	48.628	13	27.949	.938	.062
Time 3					
Null	376.805	21	-	-	-
2-First Order Factors	43.542	13	25.942	.929	.061
1- Higher Order Factor	43.542	13	25.944	.929	.061

Note: A Target Coefficient of 1.00 indicates that the first-order factors explain perfectly the second-order factor.

In order to test the hypothesis that the first-order model has discriminant validity, the covariance between the two constructs was fixed at 1 (i.e., perfect correlation between constructs). The χ^2 difference (with 1 degree of freedom) between this model and a model where the covariance was freely estimated was examined. A non-statistically significant result would indicate that the two constructs were uni-dimensional. The results showed the nested modelling comparison procedure produced an increment in chi-square that was statistically significant in two out of three comparisons: This was $\Delta\chi^2$ (1 df) = 33.629, $p < .001$, for Time 1; while

for Time 2 the result was $\Delta\chi^2$ (1 df) = 20.724, $p < .001$. For Time 3, however, no deterioration in model fit occurred that was statistically significant ($\Delta\chi^2$ (1 df) = 3.109, $p > .05$), suggesting that cognition and paranoia could also be presented as a one-dimensional construct.

Discussion

The purpose of the structural model was to build on the previous chapter by examining the cross influences between pairs of dimensions. Due to the predominance in the literature on the neurocognitive and neurodegenerative models of schizophrenia and psychosis (Bullmore et al., 1997; Feinberg, 1997; Keshavan, 1997; Keshavan & Murray, 1997), the intention was to pair the cognitive construct with the other conditions and to examine their cross influential effects. Prior to this investigation a number of preliminary analyses were undertaken.

The first procedure was to compare the patient characteristics between structural cases with those who were excluded from the analysis. Since the structural model requires three time waves of data, those cases who did not complete the SCL-90 (Derogatis, 1993) on each of the three occasions were dropped from the analysis. Comparison made between excluded cases and those included in the structural model on gender, age, age at onset, educational level, diagnosis, and country of birth revealed the two samples to be statistically equivalent. The only differences on patient characteristics revealed that cases used in the analysis tended to be single or never coupled and hence not living with a spouse. The cases used for the structural model provide a description of subjects who are relatively young (21.79 years of age), born in Australia or New Zealand (89.0%) with a diagnosis of schizophrenia (53.4%) and have never married. They could be either male or female (M = 54.4%) and have obtained a trade or technical diploma and more likely to be living with their parents (71.7%).

These samples were also compared for their psychopathology over the six symptom dimensions over the three time periods. There was no difference in psychopathology between those cases used to test the structural model and those cases excluded from the analysis over each symptom and over each time period except somatic concern at Time 2, with excluded cases recording significantly higher scores. Overall, these results indicate that the cases used to test the structural model are relatively equivalent to excluded cases in terms of psychopathology across the six dimensions across time.

In addition to patient characteristics and levels of psychopathology, inpatient service use, duration of prior psychosis and prodromal features as well as daily maximum chlorpromazine dose was also examined. Results indicated that both samples (after square root transformation of the data) were equivalent for duration of prodromal feature and prior psychosis and chlorpromazine dose. Those cases used to test the structural model had a statistically higher duration of hospitalisation (38.96 versus 26.42 days). Although hospitalisation rates are often seen as a proxy measure of pathology (Preston, 2000; Preston & Fazio, 2000), these results are somewhat anomalous to the invariant scores on psychopathology based upon the 6 symptom dimensions. It is possible that the longer hospitalisation rate at the initial stages of the study, may account for these cases being more successfully followed up over the twelve month time period. The fact that each case was assessed at a "stabilisation period" in the formulation of their psychotic illness (Edwards et al., 1999), indicates that it is possible that the psychopathology scores to be the same between those used to test the model and excluded cases. This is because a stable psychopathological state would define their inclusion into the second assessment stage of the study, which is the stabilisation period. In other words, those cases used to test the model had to

wait longer in hospital before their assessment to be made at the "stabilisation period".

Despite the differences in hospitalisation between the two samples, most patient characteristic data including gender, age, diagnosis and psychopathology remained the same between those cases used to test the structural model and those that dropped out of the study. On most accounts, it is appropriate to interpret the samples as being equivalent in patient characteristics. This means that the cases used in the measurement model and those used to test the structural model over time are similar to those who were involved in the original EPPIC study (McGorry et al., 1996; Yung et al., 1999). It is with confidence that the research can assume that those cases used to test the structural model are typical of those cases that are described as patients with an early psychosis condition.

Before the direct and mediating effects of each dimension was explored over time, each dimension was examined for its internal consistency over each time wave in the data. Alpha coefficients ranged from poor $\alpha = .511$ to good $\alpha = 838$. Observation of Table 5.4 indicate deteriorating internal reliability of the scales over time with somatic concern ($\alpha = .511$), phobia ($\alpha = .576$) and anxiety ($\alpha = .597$) displaying internal reliabilities lower than the acceptable threshold of 0.70 (Nunnally, 1978) at the third time wave. Such results may affect the calculation of structural models which include these variables at Time 3, and may underestimate the true relationship between these variables. It is difficult to say whether such poor internal reliability with these variables may have accounted for no cross influential effects with cognition, or whether no true association exists. What is understood however, is that for cognition and paranoia, where a cross influential effect does exist, the internal

reliabilities for each dimension remain stable over time. It is important to keep in mind when interpreting the results of this chapter that the internal reliability of some constructs over time are not optimal and may underestimate true association amongst these variables and cognition.

Prior to examining the cross-lagged effects of cognition on the other dimensions and vice versa, direct and mediating effect within the dimensions over time was explored. As stated before, this was done in order to examine whether symptoms operate within a "domino effect", where the preceding condition only influences the next condition, or a "snowball effect", where conditions that are present before the preceding condition, in addition to the preceding condition influence the next condition. This examination was conducted to take into account the varying effects of symptomatology over time and to examine the dynamical relationship of variables over time. The results indicated that all conditions had a domino effect, where the preceding condition did influence the next condition in a sequence of events. In addition to these findings, both depression and somatic concern had a direct effect where conditions at Time 1 had an influence on Time 3.

Much has been discussed in the literature on the enduring effect of negative symptoms (Andreasen, 1982; Arndt, Andreasen, Flaum, Miller, & Nopoulos, 1995; Dworkin, 1990; Eaton, Thara, Federman, Melton, & Liang, 1995; Edwards et al., 1999; Frith, 1987; Kelley, Gilbertson, Mouton, & van Kammen, 1992; Malla et al., 2002b; Maziade et al., 1996; Penadés et al., 2001; Selten, Wiersma, & van der Bosch, 2000b). Negative symptoms are best aligned to the dimensions of depression and cognition described in the measurement model (see Table 4.9 for item description of measurement model). It is possible that such a state as depression may accumulate

over time, contributing to worsening course or outcome of patients with a psychosis most notably schizophrenia (Arndt et al., 1995; Ayuso-Gutierrez & del Rio Vega, 1997; Malla et al., 2002a; Moritz et al., 2001; Torgalsboen, 1999). What may be described in these mediation models is a snowballing effect of negative symptoms described by the symptom dimension of depression accumulating as the course of the illness expands. Most research has shown that while positive symptoms are reasonably responsive to treatment, most notably with neuroleptic medication (Crow, 1980), negative symptoms are harder to treat. This may tie into the neurodevelopmental hypothesis (Keshavan & Murray, 1997; Murray et al., 1999) where enduring symptomatology leads to poorer treatment outcomes. Such results where the condition of depression at Time 1 directly effect conditions at Time 3 in addition to Time 2, give some evidence of an illness that has an accumulating effect. It appears that in reference to depression and somatic concern, this snowball effect acts within the symptom itself over time and does not "spill over" to influence the condition of cognition. While these symptoms accumulate at pace over time, they do not seem to drive the illness and may be passive to the cardinal symptomatology of depression or paranoia.

The final stage of the analysis examines the cross-lagged effects of cognition with the other symptoms. Carl Schneider as early as 1930 described some cognitive abnormalities amongst people with psychosis, such as *vershmezung* (fusion), *faseln* (muddling); *entgleiten* (snapping off); and *entgleisen* (derailment). Some of the descriptions within the cognitive factor such as *your mind going blank* could describe the experience of *entgleiten*. The cognition factor may best describe elements of formal thought disorder described by Schneider. The focus of this condition was to examine the experience of cognition being unreliable. With recent literature arguing a

neurodevelopmental hypothesis (Keshavan & Murray, 1997; Murray et al., 1999), it was with this research interest in mind that the dimension describing cognitive performance be paired with the other five dimensions to observe their dynamical interaction. It has been known for some time that people who meet the diagnostic criteria for schizophrenia show poorer performance than non-clinical populations on a range of cognitive tasks (see Cutting & Murphy, 1988 for detailed review). There is evidence to show that patients with schizophrenia have on average a 10 point lower premorbid IQ (Saykin et al., 1991), and more specifically have deficits in verbal learning and memory functions (McKenna, Tamlyn, Lund, & Mortimer, 1990). The underlying mechanism of a brain disorder that may underpin psychosis is unknown. The latest theories suggest that disorders in a variety of neuronal circuits may underpin psychosis (Fowler, Garety, & Kuipers, 1996), and that these circuits have extensive connection in the cerebral network which may disrupt many aspects of experience and behaviour (Hoffman & McGlashan, 1993).

In a rudimentary sense, the research is focussed on how constructs interact with cognition to influence each other over time. By pairing the constructs together, and to examine their sequence inter-actively over time, one may elucidate whether cognition has a causal influence on the other dimensions or other dimensions in turn have a causal influence on cognition. This was done within the cross-lagged modelling process. The results indicated that only one pair of constructs demonstrated a cross-lagged effect (see Table 5.7). This path leads from paranoia at Time 2 to cognition at Time 3 ($\beta_{4,1}$). The standardised path estimate of .284 indicates a modest effect using Kline's guidelines (Kline, 1998), suggesting that paranoia has a moderate influence on cognition. In conjunction with the other paths that lead to cognition at Time 3 ($\beta_{4,2}$; and $\gamma_{4,2}$), the amount of explained variance was 48.1%,

suggesting that paranoia plays a substantial role in cognition at Time 3. Interpreting these results suggests that the paranoid state over a prolonged period can detrimentally impact on cognition (this is because the standardised path coefficient is a positive not negative or suppressing association). It is interesting to note that these cross-lagged paths did not prove significant at the first time wave from Time 1 to Time 2, but only through prolonged exposure to paranoid states does cognition start to be affected some 6 to 12 months after the initial assessment. Without the third time series in the analysis, it would be unlikely that these associations would be found.

Contrary to expectation, it was paranoia that demonstrates a cross-lagged effect on cognition and not the other way round. This suggests that the experience of suspiciousness lead to distortions in thinking, that over an extended time period appear to deteriorate. This means that the affective state of suspiciousness may overwhelm the rational cognitive processes of the mind and lead to distortions in thinking. This effect only occurs over prolonged exposure to paranoia and was not evident at the early stages of the assessment period (i.e., between stabilisation and 6 months). Such results support the position that paranoia occurs as a self-serving attempt to limit the discrepancy between the ideal and actual selves (Higgins, 1987). Bentall Kinderman and Kaney (1994), argue that the origins of an exaggerated interpersonal defence lie in early childhood. The paranoid state described by the dimension indicates a feeling or state that is self protective (i.e., *feeling that most people cannot be trusted, feeling that people are unfriendly or dislike you,* and *feeling that people will take advantage of you if you let them*). When a psychotic episode becomes overwhelming it is possible that the paranoid state allows for the self to be protected against its own demise. Other theorists argue that paranoia is more to do with a negative evaluation of others as opposed to a self-protective mechanism. A

study by Chadwick and Trower (1997) found that paranoid people tend to make more negative appraisals of self to others while depressed people make more negative appraisals of self to self (Chadwick et al., 1997). The paranoid person tends to see others as a threat, while the depressed person sees himself or herself not as a threat but in some way deficient. Indeed it is possible that two paranoid states exist one of 'poor me', where a subject feels persecuted by others and the other 'bad me' where the subject feels that punishment is justified due to them deserving some sort of punishment (Chadwick et al., 1997). What makes the results in this study interesting is that the self-protective state exemplified by the paranoid state has some causal influence on thinking. In other words the feeling state of threat (i.e., *feeling that people will take advantage of you if you let them*) and persecution (i.e., *feeling that people are unfriendly or dislike you*) and general mistrust (i.e., *feeling that most people cannot be trusted*) leads to problems in thinking clearly relating directly to cognitive performance. The causal predominance of these results implies that the feeling state (paranoia) influences the thinking state (cognition).

No other cross-lagged paths with other symptom pairs proved to be significant. This suggests that each of the other dimensions appear to operate parallel to cognition, but do not causally cross influence cognition. In some sense, each dimension appears inert of the other, where only the paranoid state having a causal influence on the cognitive condition (see Table 5.7).

Although these results suggest that there is a relationship that exists between paranoia and cognition, the extent of this influence may be influenced by whether the constructs have undergone alpha, beta or gamma change (Milsap & Hartog, 1988; Thomas et al., 1988). It is important to understand whether the relationship between

the constructs of paranoia and cognition are a true stable reflection of their influence or need to be interpreted in light of the symptoms undergoing recalibration or reconceptualisation in the meaning and extent of their construct interpretation. This recalibration of the constructs refers to beta change while reconceptualisation of the construct refers to gamma (Milsap & Hartog, 1988) change. Testing for these effects is the concern of the next chapter.

CHAPTER VI

ALPHA, BETA AND GAMMA CHANGE

In the previous chapter, evidence was provided demonstrating a causal link between paranoia and cognition. It was demonstrated that paranoia at Time 2 had a cross-lagged effect on cognition at Time 3. This beta path of paranoia to cognition ($\beta_{4,1}$, see Figure 3.2) demonstrated a moderate causal influence (standardised path estimate = .284) (Kline, 1998). Being a positive estimate suggests that paranoia contributes to an increase rather than suppression of cognitive deterioration. In other words, the paranoid state at Time 2 leads to worsening cognitive symptomatology at Time 3. The fact that an association between paranoia and cognition was found at the second stage of the time wave (from Time 2 to Time 3) suggests that these cross-lagged effects occur later on in the development of the psychotic experience. Observation of the disturbance terms for each dimension in the sequence from Time 2 (paranoia: ζ_1; cognition: ζ_2) and Time 3 (paranoia: ζ_3; cognition: ζ_4) show a doubling of the amount of explained variance with 16.7% and 24.0% for paranoia and cognition respectively at Time 2 to 41.4% and 48.1% at Time 3. This provides evidence that the conditions have an increasing influence on themselves over time, with paranoia also contributing to cognitive deterioration at Time 3. These results indicate that the affective component that describes the paranoid state influences the thinking symptom of cognition. Contrary to expectation it is the feeling condition of paranoia that operates a causal predominance on the cognitive component of the psychotic condition.

Psychosis, self awareness and self appraisal

Before claims of causality are made however, longitudinal analysis requires consideration of beta and gamma change (Golembiewski et al., 1976; Thomas et al., 1988). The assumption in measuring change over time is that self reported change occurs " ...along relatively stable dimensions of reality" (Schmitt, 1982, p. 343). This issue of psychometric stability is compounded by the condition under investigation. The very nature of psychosis presupposes some distortion in "reality" (Sims, 1997) where self-awareness (insight) and self-analysis (reflection) are compromised. This means that the stability of the constructs is not only threatened by a reappraisal of meaning, but by the very apparatus that makes judgement in the first place, the mind. It is possible that the appraisal of ones psychotic condition over time could come from an improved state of rationality and hence elements of change may have a totally reconceptualised notion of ones condition (referred to as gamma change). On the other hand, insight and self reflection could deteriorate to such an extent that reconceptualisation of one's condition occurs any way. In both instances gamma change has occurred, one for the better, and one for the worse.

It is important to bear in mind that most psychological and psychiatric treatments are in the "end game" of affecting change with patients. In reference to the psychotic condition, the aim is to stabilise or improve cognitive and social functioning to such an extent that sufferers can lead fulfilling and productive lives. To achieve radical reappraisal of ones condition (gamma change) may be a useful goal in therapy. In some instances a change in the extent of a condition (alpha change), may not be as desirable as a reconceptualisation of the person's relationship with the condition

(gamma change). This is particularly evident where the use of substances may exacerbate and compound the psychotic condition (Ayuso-Gutierrez & del Rio Vega, 1997; Harlow, Newcomb, & Bentler, 1986; Stein, Newcomb, & Bentler, 1987). It may not be desirable for patients to simply reduce their substance misuse, but to totally approach their relationship with substance misuse on another level.

In order for the causal association between paranoia and cognition to be examined as a "true" relationship, it is important to understand what type of change occurred over the three time waves between these two states. Since only paranoia and cognition demonstrated a causal relationship between each other, only these two conditions will be examined. In addition to this, the latent means of these two dimensions will be explored over time to examine the extent of how these dimensions change over time. The purpose of this chapter is to explore the type of change, and to interpret the results in light of the change type. In order for this to occur a more thorough explanation of alpha, beta and gamma change and how this is measured is required.

Concepts of alpha, beta and gamma change

The concept of alpha, beta and gamma change has arisen mainly out of educational and organisational psychology research (Byrne et al., 1989; Hoyle & Panter, 1995; Milsap & Hartog, 1988; Schmitt, 1982). In classical test theory, it is assumed that changes in a scale score over time reflect the combination of the change in a true score which is a combination of the true score plus error. This assumes that the change in the scale score is attributed to changes in the extent of what is being

measured which is known as alpha change (e.g., a psychological condition). What is not assumed within the scale however, is whether the strength of the items that make up the scale have been recalibrated in the mind of the rater (beta change), or whether the way the rater conceptualises the construct which is designed to measure the scale has changed fundamentally (gamma change). Golembiewski, Billingsley and Yeager (1976) were the first theorists to label these change conditions as alpha, beta and gamma. Specifically, they describe these three states of change as follows:

> Alpha change involves a variation in the level of some existential state, given a constantly calibrated measuring instrument related to a constant conceptual domain.
>
> Beta change involves a variation in the level of some existential state, complicated by the fact that some intervals of the measurement continuum associated with a constant conceptual domain have been recalibrated.
>
> Gamma change involves a redefinition or reconceptualisation of some domain, a major change in the perspective or frame of reference within which phenomena are perceived and classified, in what is taken to be relevant in some slice of reality (Golembiewski et al., 1976, p. 134-135).

Psychometrically, researchers may hope for alpha change in their time wave data since associations between the variables are uncomplicated. The interpretation of

an alpha condition means that change scores are viewed in terms of " ...an actual or absolute change" (Milsap & Hartog, 1988 p. 574). With beta and gamma change the interpretation of the change is problematic since the extent of the change requires it to be inbedded within the complication of a reconceptualised or recalibrated construct. On the other hand, beta and gamma change may be a desired goal in a change or therapeutic process. For example, Golembiewski et al. (1976) provided an example where an observed change score was in the negative direction implying that the intervention program had no impact. However, closer examination revealed that the observed change resulted in the " ...respondent's subjective metric toward a more 'realistic' outlook and this shift was in fact a positive outcome of the intervention" (Milsap & Hartog, 1988, p. 574). This process of change can be expressly present with people who have a psychotic condition. Particularly with those who have a grandiose delusional disorder where more realistic appraisal of their situation may lead to a depression which may be the first step to leading toward an insightful recovery. If a delusional system is seen as a defence from negative self appraisal (Fowler et al., 1996), then insight could lead to a deterioration in depression, prior to a more realistic appraisal of the self. By maintaining the delusional system, the patients may rate themselves in a similar way over time, with the same conceptualised framework of the self, intact.

Analysis

A number of techniques have been proposed to measure the more problematic beta and gamma change (Terbog, Howard, & Maxwell, 1980; Zmud & Armenakis,

1978). These include using a "then" measure after an intervention has occurred in addition to the pre and post measures. Differences between the "pre" and "then" measures are taken as evidence of a response shift or beta change (Schmitt, 1982). The problem with such a technique is that it necessitates the lengthening of the measuring instrument by a significant amount. Further, such techniques require multiple tests of significance inflating the reporting of chance effects. If however the researcher focuses on the variance accounted for, rather than the significance level, this problem can be reduced as suggested by Terbog et al. (1980).

The procedure most accepted by researchers is where the pattern of factor loadings, the scale metric or units of measurement, and the uniqueness are examined using confirmatory factor analysis (Schmitt, 1982). The advantage of such a technique over the scaling procedure mentioned above is that it provides an overall test of the similarity of the time series variance-covariance matrices, as well as testing for various types of change and degree of variance associated with each time wave (Schmitt, 1982). To do this, the time series data used in the structural model (n = 145) is disaggregated into three data files representing each time wave in the series (Time 1, Time 2 and Time 3). Next using the multi-sample procedure in EQS (Bentler & Wu, 1999), incremental fit indices and chi-square difference tests were used to assess change across time. Procedures for the assessment of gamma and beta change are described in Schaubroeck and Green (1989), and Vandenberg and Self (1993). Because the interest in this study was the causal model involving paranoia and cognition, the measures were analysed simultaneously rather than independently. First, a baseline two-factor model (Model 1) was estimated and evaluated for fit across the three data points. This model was specified with the same pattern of free and fixed parameters for cognition and paranoia across time. Model 2 examined

gamma change by constraining the covariance amongst the two factors to be equal at Time 2 and Time 3, to the values calculated at Time 1. With the same constrained parameters as Model 2, Model 3 examined whether beta change had taken place, by constraining the factor variances to be equal between Time 2 and Time 3 with the values in Time 1. Model 4 assessed beta change at the indicator level, where in addition to the previous constraints, the item loadings for each factor (less the one indicator per factor that was fixed at 1.00 for identification purposes) was held to be equal between Time 2 and Time 3 with the values in Time 1. If at the end of these increasingly restrictive hypotheses, all indices between nested models are within the recommended limits, the measurements are said to remain within an alpha state.

To assess alpha change the interest is in the measurement of latent structures (i.e., unobservable values), as opposed to the testing of statistically significant differences between groups (or within groups) based on observed measures and well-established procedures such as analysis of variance (ANOVA). Once the change state was determined, this procedure was performed to investigate changes in the dimensions of paranoia and cognition over time.

The latent constructs derive their structure indirectly from the indicator or manifest variables, which can be measured. However, in order to access the mean structure of a latent construct, an additional parameter needs to be added to the model that was not required in the conventional measurement model analysis, due to the assumption that indicators were measured as deviations from their zero means (Bentler, 1995).

The additional parameter is introduced in EQS via a dummy variable (V999) that represents the intercept (α), and functions as an additional independent variable

in the measurement model. However, the intercept, although necessary for defining the mean, does not equal the mean of y (μ_y) - the dependent variable. The mean of the latent construct can be expressed in terms of the following equation (Bentler, 1995): $\mu_y = \alpha + \beta\mu_x$ where α is the constant (or intercept), β is the regression coefficient, and μ_x is the mean of the independent variable.

Figure 6.1 represents a model of mean structures (Bentler, 1995) assessed over a three-wave data set. The three-construct model (η_1 to η_3) is defined at Time 1 by manifest variables y_1 to y_3, at Time 2 by manifest variables y_4 to y_6, and at Time 3 by manifest variables y_7 to y_9 Each manifest variable has an arrow pointing to it that represents error residual. The construct has one of its manifest variables fixed to 1 at Time 1, Time 2, and Time 3 for scaling purposes. Also, the construct at Time 1, Time 2, and Time 3 has a disturbance term (zeta ζ) to account for unexplained variance due to influences not contained within the model shown in the figure. Because the covariance among the latent variables (the Phi matrix, or Φ) is not modelled, it is substituted by the covariance between the disturbance terms (the Psi, or Ψ matrix).

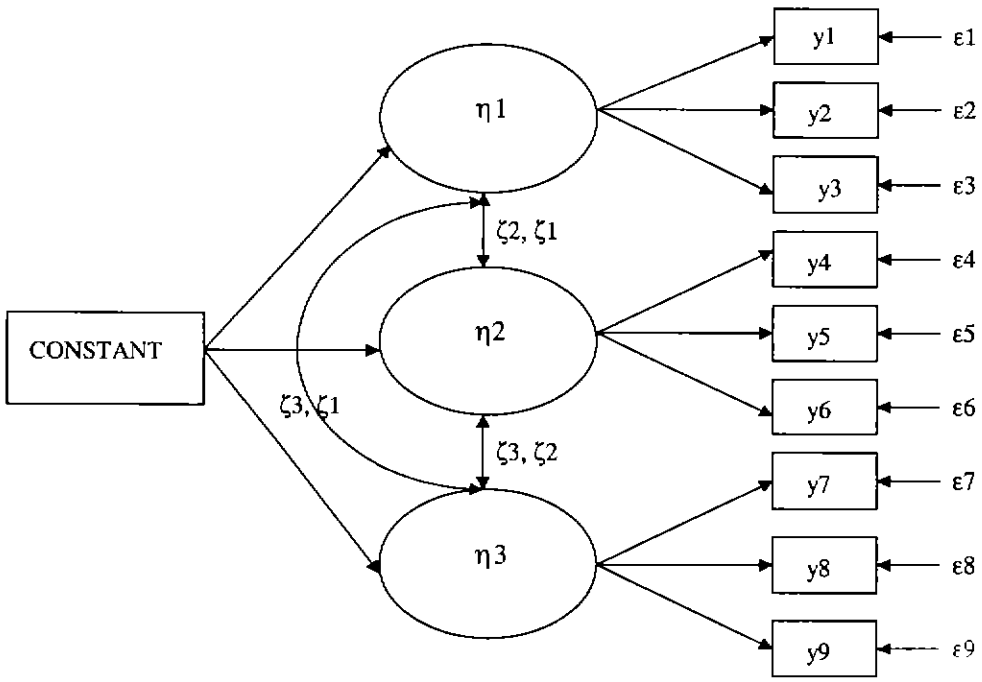

Figure 6.1. Model of latent mean structures assessed over time.

The CONSTANT shown in the figure is necessary in order to transform the model from a covariance structure model into a mean/covariance structure model. The arrows pointing from the variable CONSTANT to each of the three latent variables represent the intercepts in the model. All parameters shown in the model, except indicators y_1, y_4, and y_7 (that are fixed to 1), are freely calculated (Bentler, 1995).

Results

The results of the invariance analysis, aimed at determining whether the meaning of the constructs remained constant for respondents over the three time periods. These results are shown in Table 6.1.

Table 6.1
Assessment of Beta & Gamma Change

Model	χ^2	df	$\Delta\chi^2$	Δ df	CFI	SRMR
Model 1 - Two Factor Baseline Model	135.036	39	--	--	.918	.060
Model 2 - Covariance amongst the factors equal	135.811	41	0.775^{ns}	2	.919	.065
Model 3 - Variances of factors equal	139.933	45	4.122^{ns}	4	.919	.066
Model 4 - Item loadings equal	147.046	55	7.113^{ns}	10	.921	.071

Note: n.s. = not significant; Model 2 = Gamma Change; Models 3 & 4 = Beta Change. CFI = Comparative Fit Index; SRMR = standardized root mean squared residual.

Observation of the differences in chi-square relative to the change in degrees of freedom demonstrates that each constrained model remained invariant between each time wave. Such results suggest that the constructs of paranoia and cognition remained stable over time and did not undergo beta or gamma change. Given the non-robustness conditions due to sample size, the fit indices (CFI $\geq$ 0.918, SRMR $\geq$ 0.060) suggest a reasonable but not good fit to the data within the baseline model (Hu, & Bentler, 1999).

The results show that despite the imposition of highly restrictive conditions, the proposed structural model between paranoia and cognition remains stable over the

study period and generalises adequately between the three waves of data. As a consequence of adequate alpha state conditions over time for cognition and paranoia, the next investigation was to examine the longitudinal effects of the dimensions over time.

The longitudinal modelling of the constructs was carried out separately. For cognition for Time 1, Time 2 and Time 3 the results in latent means were as follows: $\mu_{Y1} = 2.240$, $\mu_{Y2} = 1.935$, $\mu_{Y3} = 2.094$, respectively. All latent means showed statistically significant differences ($p < .001$) when compared to the mean of a previous time period. For the modelling of paranoia over time the results of the latent means were: $\mu_{Y1} = 1.992$, $\mu_{Y2} = 1.784$, $\mu_{Y3} = 1.729$, for Times 1, 2, and 3, respectively. These latent means were also statistically significant when compared to the mean of the previous period ($p < .001$). Comparison of the latent means between Time 1 and Time 3 for both cognition and paranoia were also statistically significant.

Discussion

Overall, the results showed that the dimensions of paranoia and cognition held within the same construct definition over the three time waves. Structural equation modelling, using the multi-sample procedure by treating each time wave as a separate sample, and examining the structural relationships, demonstrated integrity in construct definition over time. As Millsap and Hartog (1988) state, while groups are examined for their structural invariance, measurement instruments are examined over time to demonstrate their assumed "*stationary*" nature. The results show that the meaning of

the instrument in relation to paranoia and cognition remain stationary over the three time waves. The implications are such that the extent of the causal relationship is "alpha" in nature. In other words, the influence of paranoia on cognition from Time 2 to Time 3 are true in nature and do not require some accommodation in interpretation by recalibrating the instrument or contending with reconceptualisation of the constructs of paranoia and cognition over time.

Observation of the latent means for cognition indicate a U shape trend with cognition dipping statistically significantly at Time 2 between the time points of Time 1 and Time 3. Paranoia demonstrated a steady linear reduction from each successive time wave. The magnitude of scores although statistically significant for the dimensions are relatively small and so suggest modest trends.

CHAPTER VII

SUMMARY AND CONCLUSIONS

The following Chapter provides a summary of the major research findings. From the findings conclusions are drawn, implications are discussed and recommendations for future research are considered. In addition some limitations in the study are also highlighted in order to contextualise the results.

It is argued that the current research makes a unique contribution to the literature on psychosis by presenting evidence in support of the construct validity of a model of psychosis validated by self reported techniques. Via the use of confirmatory factor analysis and structural modelling techniques, across two independent samples of subjects with early psychosis, the research was able to demonstrate a rigorous and scientifically sound measure of psychotic experience. In addition, this measure was examined in terms of the dynamic relationship which exists between the variables over time. Some attention will be made on the unique technique of dynamic modelling over three time waves of data as a way of elucidating the relationship amongst dimensions. It is hoped within this concluding chapter that other practitioners and researchers may see an analytical technique that can be used to further extend the knowledge of how psychosis operates as a dynamical system. Further suggestions will be made on how to link this technique with other levels of analysis including the genetic, neurocognitive and social level.

Measurement Model

One of the major contributions of the research centres on the confirmation of a measurement model of psychosis from the perspective of the subject who experiences the illness. Most if not all studies which investigate the dimensionality of psychosis and schizophrenia in particular centre around observational instruments made by expert opinion (Bell et al., 1998; Bergman et al., 2000; Burger et al., 1997; Cardino, Sham, Murray, & McGuffin, 2001; Claridge et al., 1996; Dollfus & Everitt, 1998; Eaton et al., 1995, Ehman et al., 2001; Lançon et al., 1998; 2000; Peralta & Cuesta, 1998; Peralta et al., 1994; Smith et al., 1998). Most of these studies have been able to describe a structure within psychosis that depicts a 3 to 5 factor dimensionality (Kay & Sevy, 1990; Peralta et al., 1992) (see Table 2.5 for description of current factor analytic research). These structures tend to depict models which describe five major dimensions. The first describe "negative" features which are states that display low arousal states such as anhedonia, and blunted affect. The second dimension is usually termed "positive symptoms" that describe active disturbed states such paranoia, hallucinations and delusional systems. The third dimension consists of those states that describe disruptive cognitive processing. Such disruptions include poor concentration, difficulty in making associational and logical connections to events, and poor short term and episodic memory. The fourth dimension describes emotional states such as depression and anxiety. The final dimension concerns excited or agitated states, such as emotional dis-regulation, hyper-vigilance and high arousal. Recent confirmatory factor analytic research has described in decreasing order of importance a structure which describes negative, excitement, depression, positive and cognitive impairment dimensions (Lykouras et al., 2000).

The major criticism that can be aimed at these studies is two-fold. First, they use observational measures that do not meet independence of measurement required in factor analysis, and second, no study has employed a cross-validation sample to test for structural invariance of their models. Each point will be discussed in turn.

One of the major arguments which underpins the research in this thesis is based upon meeting the strict notion of independence of measurement central to effective factor analysis. Independence of measurement requires that each observation in the data set is independent of the next. This means that each response is not filtered through one person observing the phenomena, but instead, each phenomenon of interest is reported from the source itself (the patient). The problem with observational instruments is that they come ladened with preconceived notions of the structure of psychosis. The preconceived ideas exist both in the training of expert raters and in the very way some of the measuring instruments are designed (Kay et al., 1986a). For example in the Positive and Negative Syndrome Scale (PANSS) (Kay et al., 1986a), raters are asked to rate phenomena along a strict structure of positive, then negative and then general symptoms (see Table 2.4). While this may be appropriate for performance tests such as the Weschler Adult Intelligence Test, for instruments measuring symptomatology, it carries a number of problematic issues in psychometrics. Most notably the requirement of any factor analytic research is to reduce the effects of common method variance (Tabachnick & Fiddell, 1996), and response set bias. The very purpose of any factor analysis whether exploratory or confirmatory is to examine the natural covariation of conditions and to provide a pattern matrix which explains some underlying structure (Anderson & Gerbing, 1988). Placing items within a cluster prior to examination of dimensions does not allow for natural covariation to occur amongst the items. In addition, training raters

to observe phenomena a certain way has its own limitations when using factor analytic techniques. Such an approach creates an "observer effect" where phenomena are already filtered through a structure in seeing the world. While this is not problematic for case study research or clinical diagnosis, it is for statistical analytical techniques where structure is concerned. An example of this is when one critic of the World Health Organisation ten country study noticed that Russian medical doctors were observed to be "over diagnosing" schizophrenia. The remedy to this problem was to retrain the doctors on how to measure schizophrenia which resulted in rates and incidents of schizophrenia dropping in line with the other countries involved in the study (Marshall, 1996). While this may be seen as a legitimate approach in measuring the incidence of psychosis around the world (Jablensky et al., 1992; Jablensky, Schwartz, & Tomov, 1980), other commentators may see it differently. For example, it could be seen as a socially construed way of observing phenomena (Marchall, 1995), not free of the social constraints by which it is seen and by whom it is seen by (Gergen, 2001). Ownership of the phenomenon and how it is construed is equally important in a post modern world as the phenomenon itself.

In virtually all confirmatory factor analytic studies that model schizophrenia and psychosis, no paper has used a cross-validation sample. The issue of construct validity needs to be addressed not only in examining the underlying structure of a given phenomenon, but whether this structure is replicable across an independent sample (Byrne, 1994; Byrne et al., 1989). That is, although a model may demonstrate good fit to the data, this fit may be particular to the data set or sample of interest. Where structural modelling has its greatest contribution to psychology and social sciences in general, is whether the theoretical models proposed can be replicated (Anderson & Gerbing, 1988). What researchers must keep in mind is that " ...models

are never confirmed by data, rather, they gain support by failing to be disconfirmed" (Anderson & Gerbing, 1988 p. 421). This is a true Popperian notion of falsification since it withstands being disconfirmed (Chalmers, 1976). A cross-validation sample attempts to do this by demonstrating that the model is difficult to disconfirm by replication on another sample. Split sampling is often suggested by researchers (Anderson & Gerbing, 1988; Byrne, 1994), however it is possible that the same sample may have certain selection bias that may favour the replication of a given sample. A totally independent sample separated in location and protocol may be more preferable since this is more robust if the theoretical model proves invariant between the samples.

The attempt in the present program of research was to cross-validate a self reported measure on two samples separated geographically (i.e., Victoria versus Western Australia), but consisting of the same patient characteristics (i.e., cases being treated for their first episode of psychosis). The contribution of this research is two-fold. First, it attempts to meet the limitations of previous research confounded by nested observational instruments by using a self reported measure of psychopathology (Derogatis, 1982). By doing this, the research meets the assumption of independence of measurement, free of any systematic bias that may underpin observational instruments of psychosis. The second significant contribution to the field of research in psychosis is that the model of psychotic experience was replicable between two independent samples. Such a procedure provides evidence for a stable and replicable measure of psychosis on two samples that are similar in type but separated in location. Replicability of the measurement model suggests stability of the constructs beyond the initial data source.

Construct validation, as a process, needs to be grounded in a sound theoretical base (Byrne, 1994; Hair et al., 1992). For the present research, the development of the measurement model was theoretically grounded in previous models reported in the literature on schizophrenia (Kay & Sevy, 1990; Lançon et al., 1998; 2000; Peralta & Cuesta, 1995; 1998; Peralta et al., 1992; 1994). These studies (and others) formed the basis by which the measurement model was constructed. It was noted that the instrument used (Derogatis, 1982) was not a specific measure of a schizoid type life as others have measured (Bergman et al., 2000; Claridge et al., 1996), but rather a measure of general symptomatology. This has its advantages and disadvantages. The advantages of a general instrument of psychopathology lie in the fact that these instruments are often validated on a wide range of psychiatric populations (Johnson et al., 1996; Royse & Drude, 1984; Wood, 1982; Wood, 1987) and hence need to be consistent and stable. Previous research has been able to demonstrate that the Brief Symptom Inventory (BSI) has good convergent and discriminate validity with recognised observational measures of psychopathology, such as the Brief Psychiatric Rating Scale (Morlan & Tan, 1998) and the Positive and Negative Syndrome Scale (Preston & Harrison, 2003). By identifying the items within the BSI that best describe previous constructs identified in the literature, the measurement model was able to be constructed by informed theoretical considerations and empirical investigation. Most notably, through the use of the maximum likelihood method both in an exploratory and confirmatory factor analytic approach, a measurement model which described *anxiety, cognition, depression, paranoia, phobia* and *somatic concern* was proposed. In addition, this model was cross-validated against an independent sample of similar cases with early psychosis and demonstrated structural invariance at the form, factor loadings and factor convariances levels. Such results of

a cross-validated measure of psychotic experience have never been achieved with a self-reported instrument, nor has a model of psychosis been cross validated on an independent sample. The form of the measurement model can be somewhat overlaid with von Korring and Lindström's five factor pyramidical model of schizophrenia displayed in the following figure

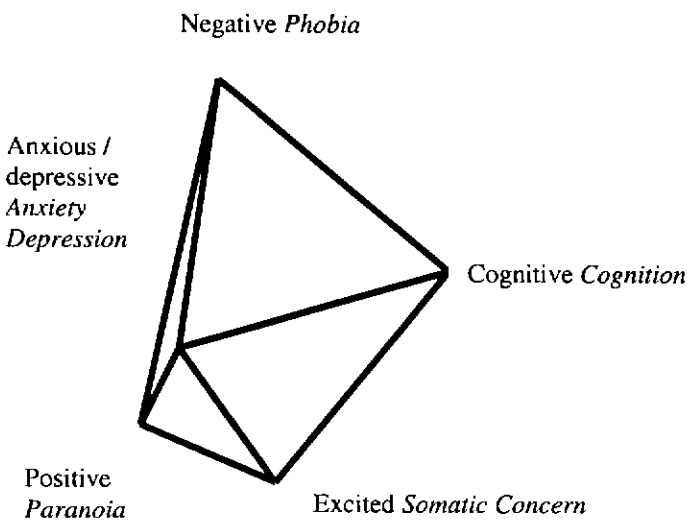

Figure 7.1. Schematic representation of the five factor pyramidical model of schizophrenia proposed by Lindström and von Knorring with measurement model factors overlayed.
Note: measurement model factors in italics.

The results of the measurement model have a number of significant contributions to research in psychosis. First the study was able to demonstrate that it is possible at the early stages (i.e., in the first 2 years) of a psychotic illness, to model a symptomatological structure from self reported data. Not only was this possible, but the same structure proved invariant on an independent but similar population of patients with psychotic conditions. Such results put doubt to the assertion that self reported measures of psychopathology are unreliable and invalid from subjects with a psychotic condition. At the early stages of the illness, it is possible to model a

measure of psychosis that remains stable between similar but independent samples. Indeed it has been argued in the literature that differences in convergent and construct validity between self reported and observational instruments may not lie in the inability of the psychotic patient to report their condition, but in the varying complexity by which constructs are defined and understood by "experts" and by "sufferers" (Preston & Harrison, 2003). In other words, divergence begins where subtlety in the nomenclature begins. When theoretical constructs are divided and separated by ever increasing notions of definition and nuance, the likelihood of measurement error becoming greater is also possible. Where constructs are kept well defined, and the empirical development of the measurement model attempts to identify cardinal symptoms rather than to account for every complex interplay of psychotic phenomena, it is possible to develop a measurement model which is stable and replicable. The process of creating a parsimonious measurement model is even more crucial with conditions that affect self awareness such as psychosis, and in particular schizophrenia, but also to make it simple and stable enough to withstand dynamical changes over time.

Second, the measurement model was able to provide evidence of a shorter version of the BSI that demonstrated good internal consistency, convergent and discriminate validity. It is possible that the measurement model could be used in future research that wishes to employ a more pithy measure of mental health with good and stable psychometric properties. This will allow additional measures to be included in a cohort study that is less likely to over burden the subject. Since psychosis is primarily seen as a disorder related to neurocognition, a shortened measure of symptomatology is a useful way of gaining an accurate measure of a patient's condition. It may also allow for the inclusion of other measures of interest

such as side effect profiles from medication or measures of social and occupational functioning without overwhelming the test battery.

Since previous research suggests the BSI to have a similar construct structure as observational measures of psychopathology (Morlan & Tan, 1998, Preston & Harrison, 2003), the measurement model suggests that the internal experience of psychosis is one that is concurred with what is observed. Since no previous research has been done on modelling psychosis from the sufferers direct report, this current research provides a unique contribution to research in the field. The measurement model was able to demonstrate that the internal state of the psychotic condition holds in similar construct definition as those developed by experts in the area of psychopathology. While previous attempts in modelling psychosis have relied exclusively on observational measures, such a technique of using self reported data as posited within this thesis points the way to using multi-trait multi-method techniques (MTMM) in modelling psychosis. The author has argued elsewhere that modelling of psychosis has been too reliant on measuring from one perspective, namely the observer or expert position (Morlan & Tan, 1998; Preston & Harrison, 2003). While this is not an illegitimate position, it does pose its own limitations as has been previously described. Using self reported measures could allow for the modelling of psychosis from a number of perspectives or methods of gathering data. MTMM operates by examining the covariate structures of each model from the two sources of measurement and tests for their structural validity and invariance in measurement, using confirmatory factor analytic techniques (Byrne, 1994). With relation to psychosis, both observational items and self reported items could be measured with the intent of measuring the same construct but from different perspectives. By this technique it may be possible to examine where constructs converge and diverge

depending on the perspective of measurement. This technique is described in the figure below.

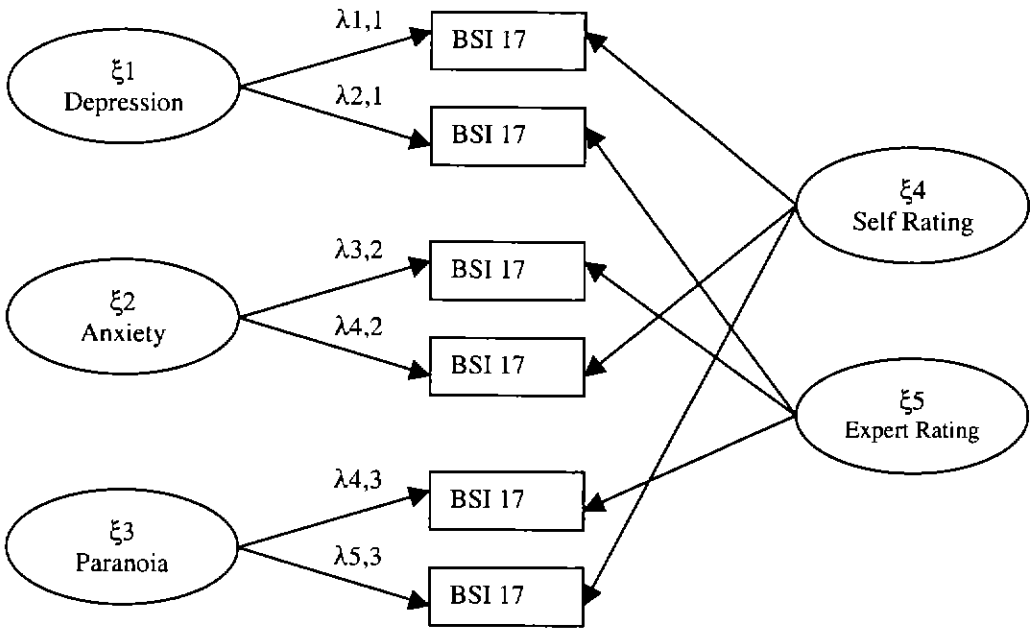

Figure 7.2. Example of a Multitrait-multimethod Model of Psychosis.

Each indicator for each factor in the model has a contribution from both perspectives of rating. In the example of Figure 7.2, an item for each construct is rated from both the subject and the expert. This allows the researcher to test for measurement invariance by observing the factor loading of each item. Observation of the Lagrange multiplier test would indicate whether items were best indicated on another factor thus indicating divergence of construct definition between the two techniques. In addition, observation of the standardised solutions for each item would indicate the strength or contribution each indicator item would have on its specified factor.

The measurement model within this study could assist in a MTMM analysis by using them as the indicator items from the self reported perspective. The measurement model could then be modified into an observational instrument where multi-perspectives can be drawn, which could include, clinicians and carers. As the author has stated elsewhere such a technique could " ...overcome problems of operational definitions, language, and theory construction that have plagued previous research in modelling psychosis" (Preston & Harrison, 2003 p. 225).

It was demonstrated that correlations between the 6-factors for both the validation and cross validation sample were relatively moderate with no evidence of a second order factor driving the symptoms (where $r > 0.80$ is required). The correlation coefficient higher than 0.80 is required in order to show that the second order factor adequately explains the first order factor. The strength of the correlations were similar between the two samples suggesting similar types of associations amongst the factors. Despite these limitations, the factor scale scores were statistically different between the validation and cross-validation sample. This provided evidence of good discriminate validity of the measurement model, because despite both samples being similar in the sense that they were both samples of patients being treated for their first episodes of psychosis, their patients' characteristics were different. The Western Australian sample consisted of older subjects that were assessed at an earlier stage of the illness (within 4 to 6 weeks of being formally accepted into an early episode psychosis program). The Victorian sample were younger and assessed at the "stabilisation period" after their first admission to hospital (Edwards et al., 1999). The difference in patient characteristics was reflected in the scale scores for each factor, with the cross validation sample having statistically significantly higher scores on all factors. The measures therefore, appear capable of

detecting differences in psychopathology, which one would expect if the patient characteristics are different in terms of the time the measure was administered during the course of the illness. Such results confirm that the measurement model not only displayed good construct validity but also demonstrated adequate discriminate validity between samples where one would be expected.

Overall the results of the measurement model provided firm evidence of a measure of psychotic experience that was factorially stable across two independent samples. In addition, the instrument was administered from the perspective of the case themselves and was not reliant on nested observational measures. Such results provide a substantial contribution to the literature on psychosis, since it provided evidence for the first time of a reliable and valid measure of psychosis from the experience and perspective of the sufferers themselves.

Structural Model

The main focus of the program of research was to examine the relationship between the dimensions of psychosis dynamically over time. This investigation was performed through structural equation modelling. The approach was to examine the dynamic interaction between dimensions by observing their cross-lagged effects over time. One way of examining patterns is to observe the inter-relatedness between dimensions. It has been argued throughout this thesis that it is possible to understand more fully the psychotic experience by examining the relationships that are held between the constructs.

The attempt however was not to link each pair of constructs together and to observe what associations exists. This is neither a-priori nor scientific since chance effects are likely to occur with this type of approach (Chalmers, 1976). Instead, the focus was to examine the influence of cognition on the other constructs. This formulation was based upon the emergent neurodevelopmental hypothesis that argues that symptomatological expression occurs due to subtle anomalous brain development (Weinberger, 1987). Such neurocognitive deterioration is thus expressed in poor concentration (Ngan & Liddle, 2000; Penadés et al., 2001), poor memory (McKenna et al., 1990), and thought blocking (Sims, 1997). These conditions are cardinal in describing the cognition factor (ie; *trouble remembering things, difficulty making decisions, your mind going blank* and *trouble concentrating*). This factor laid the groundwork for examining the influence of cognitive deterioration on the remaining five dimensions within the measurement model (ie, anxiety, depression, paranoia, phobia and somatic concern). What is unique to this study, is the notion of examining the interplay between these dimensions, linked to the neurocognitive deficit model primary to the description and expression of schizophrenia and other psychoses (Moritz et al., 2001; Murray et al., 1999). By observing the interaction between the dimensions over time, the research could examine the psychotic experience within a dynamical system where cross-lagged effects may emerge. As such, the approach was to use structural equation modelling techniques adopted from organisational and educational research (Ecob, 1987; Stone-Romero et al., 1995), and to apply these techniques to the field of psychiatry. Both the measurement model and structural model were examined simultaneously within the statistical procedure. Such a complex and comprehensive technique can lay the foundation of a new way of modelling psychosis from a dynamical systems point of view.

Direct and mediating effect of psychological states

One advantage of such a procedure where three time waves are present in the data is to examine direct and mediating effects within constructs over time. This procedure was first examined prior to the cross-lagged models, since it was argued that a thorough understanding of how the conditions behave over time is required prior to examining their inter-relationships. As described in Chapter V, mediation effects occur where the influence of psychological states at Time 1 (the antecedent) on Time 3 (the consequence) is mediated by the extent of the psychological state at Time 2 (the mediator) (James & Brett, 1984). This effect was described as the domino effect, since each condition is only influenced by the preceding condition. Direct effects occur where the antecedent has a direct influence on the consequence, in addition to the effects of some mediated time point in between. This effect was described as the snowball effect, where influence accumulates at pace over time.

Much has been published in the literature on schizophrenia and other psychoses on the deteriorating course of the illness (Arndt et al., 1995; Ayuso-Gutierrez & del Rio Vega, 1997; Goldstein, 1988, Jablensky et al., 1992, Kemppainen et al., 2001). Historically schizophrenia was described as *dementia praecox* where the condition was thought to lead to a worsening condition over time (Barrett, 1998a), leading to progressive mental paralysis (Hirschmüller, 1999). Little is understood whether the psychotic condition operates in a linear progressive fashion, or in an additive snowballing effect. What is understood is that some cases lead to a poor outcome while other cases improve in their condition. Employing techniques that examine mediation and direct effects via structural equation modelling attempts to

examine such effects. To the knowledge of the author, no research has been conducted in psychosis which examines the effects of conditions over time with such a technique. Much interest has been expressed in the literature on the outcome of subjects in the early stages of a psychotic condition (Malla et al., 2002a; Schwartz et al., 2000; Sipos, Harrison, Gunnel, Amin, & Singh, 2001; Torgalsboen, 1999). The attempt with the mediation study was to examine how the dimensions in the measurement model behave within themselves over time with a cohort of subjects experiencing their first episodes of psychosis.

The results indicated that most conditions operated within a linear fashion. These included anxiety, cognition, phobia and paranoia. This means that no direct effect was found between Time 1 and Time 3 suggesting a domino effect in causal influence. At the early stages of the psychotic condition, these psychological states operate in a linear fashion where the extent of the illness is only influenced by the preceding condition. Such an effect was not observed for depression and somatic concern. Both these states demonstrated a direct causal effect between the condition at Time 1 and Time 3, in addition to the condition at Time 2, suggesting an additive effect. Both states may have different reasons for these effects.

Depression (i.e., *feeling blue, feeling hopeless about the future,* and *feelings of worthlessness*) is a state that describes low arousal. This psychological state is closely aligned to (although not directly descriptive of) negative symptoms such as blunted affect, poverty of content of speech, restricted quantity of speech and slow speech (negative symptoms extracted from the Present State Examination) (Wing et al., 1974). Depression is understood as a clinical correlate of negative symptoms (Addington et al., 1993a; 1994; 1996;). It is possible that this snowballing effect of

depression is measuring the correlated effects of negative symptoms which are known in the literature to contribute to poorer outcome over time particularly with schizophrenic conditions (Arndt et al., 1995; Eaton et al., 1995; Edwards et al., 1999; Kelley et al., 1992; Malla et al., 2002b). In other words, the state of depression described by the early psychosis cases demonstrated a direct influential effect between Time 1 and Time 3 that may explain a worsening condition related to negative symptoms.

The snow ball effect of somatic concern may have different etiological causes that may not be attributed to the psychotic condition but to the monitoring of symptoms. Somatic concern is described in the measurement model as that factor where subjects describe bodily sensations (i.e., *trouble getting your breath, hot or cold spells,* and *numbness and tingling in parts of your body*). Nearly all cases in the structural model were prescribed anti-psychotic medication (n = 140, 96.5%) which is known to have significant side effects (Singh & Kay, 1975; Voruganti, Heslegrave, Awad, & Seeman, 1998). Part of the national guidelines for early psychosis is to inform patients to monitor their symptoms that include the side effects of medication (Whiteford, 1996). Such neuroleptic side effects can produce thermic disregulation (hot or cold spells) and other effects such as numbness or tingling in hands and feet. By encouraging patients to monitor side effects of medication can also paradoxically, make patients "somatisisers" where every body sensation real or imagined may be considered. Coupled with the fact that somatic concern has been identified as symptomatic of psychotic conditions, such as the Schneider's notion of somatic passivity (Sims, 1997), it is difficult to partial the effects of medication and the psychotic condition on the dimension of somatic concern. It is likely that both may

contribute to cases reporting a direct effect of somatic concern at Time 1 contributing to the effect of somatic concern at Time 3.

Relationship between paranoia and cognition: affect influencing thinking

Having established how psychological states "behave" over time by examining their direct and mediating effects, the thesis was then able to focus on how these states interact with cognition. As mentioned throughout, the neurodevelopmental hypothesis (Keshavan & Murray, 1997; Murray et al., 1999) has underpinned much research into the aetiology and course of schizophrenia and other psychoses. Such a theory posits that changes in neurological functioning, often expressed in early to late adolescence, leads to symptomatological expression characteristic of schizophrenia (Weinberger, 1987). Cardinal to this expression is the deterioration in cognitive functioning. The focus of the research was to measure this influence from the subjective experience from the subjects themselves. By modelling the interplay between cognition and the other states, the research attempted to elucidate a "causal hypothesis" suggesting that cognition would play a causal influence on the other states. Such a hypothesis suggests that the cognitive experience of psychosis drives or leads the other states into their expression over time. The driving force stipulates that the thinking condition concerned with cognition predominates over the feeling condition concerned with affect. This means that thought precedes feeling in relation to the psychotic experience and as such is causally predominate. The uniqueness of the research lies in linking the psychological states within a dynamical system, and to observe their influence from a

subjective or experiential point of view. After establishing a stable and reliable measurement model, and understanding that most conditions apart from depression and somatic concern operate within a linear causal mechanism, the main focus was to investigate the causal hypothesis.

Since there appears to the knowledge of the author no previous modelling of the symptomatological structure of psychosis and its causal influence over time, the research was as much exploratory as it was confirmatory. In other words, the originality of the methodological approach in the research meant that the research could not be guided by previous modelling procedures within the literature. As such, the thesis is the first attempt to link the influence of symptoms over time within a causal pattern. As stated by Riecher-Rössler and Rössler (1998), most studies in schizophrenia research examining course are often selective, rarely prospective in design and almost never continuous in the collection of data. For a condition that is episodic in nature and highly unstable it seems that longitudinal studies should be central to the focus of research in schizophrenia and other psychoses. The advantage of the current data set in this thesis is that it is prospective in design and attempts to examine the influence of psychological states at the early stages of the expression of a psychotic condition.

The structural model paired each of the five constructs within the measurement model with cognition. Each time wave was examined for its cross-lagged effect. For example, cognition affecting depression from Time 1 to Time 2 is examined, and in turn this relationship inverted in the next time wave with depression at Time 2 affecting cognition at Time 3. Such an approach followed the lead of Ecob (1987), who used this technique to examine the cross-lagged effects of learning difficulties and reading over three continuous years of education. Within a cross-

lagged modelling hypothesis, four hypotheses are examined. This is demonstrated in the following figure with the unbroken line representing the path within the structural model that tests the hypothesis.

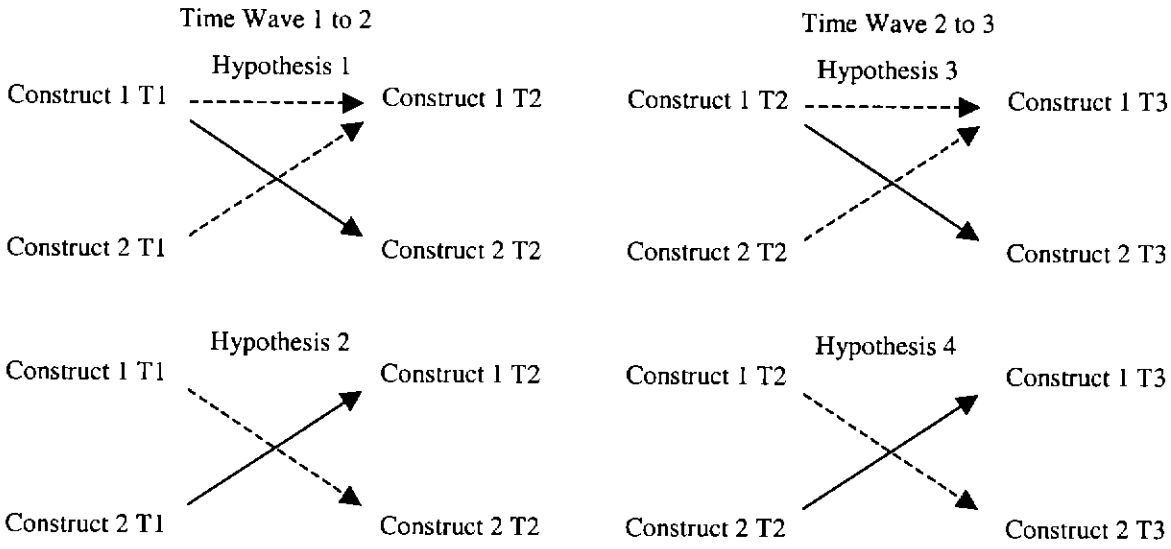

Figure 7.3. Structural models examining cross-lagged hypotheses.
Note: Unbroken line represents the path coefficient tested in the hypothesis. Figure adopted from R. Ecob, Applications of structural equation modelling on longitudinal educational data, 1987, Structural modelling by example: *Applications in educational, sociological, and behavioural research.* Cambridge University Press p. 143.

The results indicated that one cross-lagged path proved statistically significant with paranoia at Time 2 influencing cognition at Time 3 (β4,1, standardised path coefficient = .284 $p < .05$). This result was contrary to expectation, where cognition was expected to causally influence the other states including paranoia. With this result, it is the affective state of paranoia (i.e., *feeling that most people cannot be trusted, feeling that people are unfriendly or dislike you,* and *feeling that people will take advantage of you if you let them*) influencing cognition (ie; *trouble remembering things difficulty making decisions, your mind going blank* and *trouble concentrating*). Observation of the amount of explained variance for the endogenous variables at Time 2 (paranoia = 24.0% and cognition = 16.7%) almost doubled at Time 3

(paranoia = 41.2% and cognition = 47.6%) suggesting that the effects of the preceding condition increase substantially over time. It is possible that this increasing condition from the 6 month time period to the 12 month time period for each condition, leads to the cross-lagged effect by 12 months. This may indicate that paranoia grows at pace to finally have some causal influence on cognition. In reference to the causal predominance hypothesis, it is contrary to what was expected since there was some *a priori* expectation that cognition would have a significant influence on the other symptoms. The fact that cognition did not influence any of the other conditions and paranoia had some moderate cross influential effect on cognition was unexpected. In light of these results, some explanation is needed.

It appears that the affective state that denotes arousal of threat and mistrust, influences the thinking condition. It is well known in the literature that the paranoid state may lead to over valued or exaggerated attributional styles of thinking (Fowler et al., 1996). Earlier theorists such as Freud (1915/1956) and Laing (1960) argued that psychotic states arise as a consequence of how people may adapt to emotional threats. More recently other theorists such as Ciompi (1988) and Perris (1989) believe that there are interactions between biological factors (acquired or inherited neurobiological dysfunction) and dysfunctional cognitive structures or emotional schemata which may arise from adaptation to adverse life predicaments. They argue that poor socio-emotional learning may lead to psychotic disorder when faced with significant life events. Fowler, Garety and Kuipers (1996) argue that there are two current views on emotion and psychosis. The first concerns an emotional reactivity to the psychotic experience such as depression and anxiety may arise as a consequence of appraising the experience of psychotic illness. Other researchers support this claim that anxiety, depression and panic attacks can lead to a decreasing quality of life and

increase in suicidal behaviour (Goodwin & Davidson, 2002; Huppert et al., 2001). The second view concerns whether certain types of psychological states that include paranoia may arise partially as the consequence of emotional disturbance (Fowler et al., 1996). This second view argues that with increasing emotional disturbance the sufferer is unable to attribute events in life in a balanced way both in terms of emotional response and cognitive attributions. Persecutory delusions and paranoid states may arise from abnormal attention to threat-related stimuli (Bentall et al., 1994), with biases in the recall of threat-related and negative propositions (Kaney & Bentall, 1989). This biased recall may also lead to deterioration in recall in general, which is part of the item descriptors of cognition (i.e., *trouble remembering things*). In other words, the emotional state caused by paranoid delusions may swamp or overwhelm the cognitive processes leading to further deterioration in thought processes.

Heightened states of mistrust not only harbour ineffective emotional reaction to events, but appear to influence the way thoughts are processed and executed. The basis of this relationship may be variants of psychoanalytic ideas, which suggest that paranoia " ...may arise as a result of psychological defences" (Fowler et al., 1996, p. 65). Research in the past decade suggests that cognitive styles where an explanatory bias towards attributing negative outcomes to external causes, is characteristic of patients with psychosis (Bentall, Kaney, & Dewey, 1991; Kaney & Bentall, 1992; Kinderman, 1994). Bentall, Kinderman and Kaney (1994) argue that these external attributions to internal threat are defences against threats to self-image. In order for the subject to maintain a certain self-image, threat is externalised as "out there" rather than as an internal state. Such a realisation of an internal state would require the person to alter their self-image. Coupled with the trauma of a psychotic experience

this may be too difficult to consider and so the self continues to externalise the threat. Although emotional instability may be a contributory factor to the development of psychotic disorder, it is unlikely to be the primary cause (Fowler et al., 1996). Other factors mentioned before such as the neurodevelopmental hypothesis and psychosocial factors need to be considered but are beyond the data herein.

For example, Frith proposes that a precursor to delusion formation may be an inability to understand accurately the intentions of other people (Frith, 1992). Frith argues that schizophrenia arises where " if we found ourselves thinking without any awareness of the sense of effort, that reflects central monitoring, we might well experience these thoughts as alien and thus being inserted into our minds" (Frith, 1992, p. 81). Frith argues that this is a deficit in self-monitoring where the person is unable to monitor effectively their intentions or whether their intentions are emanating from themselves. Such a deficit in cognitive processes could lead an individual to make incorrect inferences or become highly confused about the intentions of others. Such a disorder in social understanding would lead then to an experience of the social world as different and unusual. Such odd social experience may lead to paranoid delusions as the person attempts to understand others without knowing that he or she has lost the ability to develop accurate inferences about other people (Fowler et al., 1996).

Since the current research did not measure attributional style, it is difficult to investigate whether changes in attributional style would lead to a deficit in cognitive functioning or an increase in subsequent paranoid delusional systems. Frith's theory proposes that the cognitive processes related to attributional style lead to paranoid threat. Such a theory is possible, however, in the current thesis this was not

measured. What was discovered was that the paranoid state lead to further deterioration in cognition. The cognition factor within the measurement model did not measure style but function. However the paranoid factor, although measuring an affective state of feelings (i.e., *feeling that most people cannot be trusted, feeling that people are unfriendly or dislike you,* and *feeling that people will take advantage of you if you let them*), also could be interpreted as measuring a style of thinking. In other words, perceived emotional threat can originate from a style of thinking that falls under Frith's self-monitoring deficit model (Frith, 1992; Frith & Done, 1989). This style of thinking, expressed by delusional feelings of threat, lead to deterioration in cognitive processing. In short, the style of thinking leads to distortions in feeling which lead to deterioration in thinking capacity. This is expressed in the following causal hypothesis.

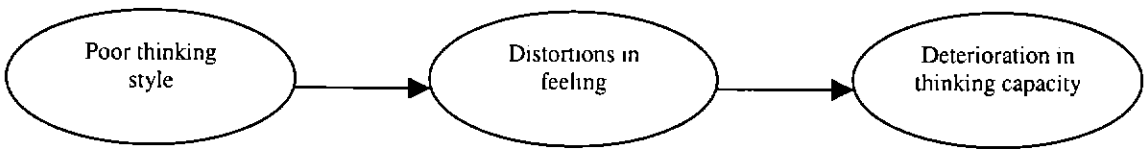

Figure 7.4. Causal predominance hypothesis.

The relationship between paranoia and cognition only became apparent between Time 2 and Time 3 within the time series. As mentioned before, the paranoid state needed to gain momentum in order for its influence on cognition to be detected. At the initial stages of the illness (between Time 1 and Time 2), no relationship was found between any of the constructs, suggesting that these do not have cross-influential effects on each other. As the time series progressed, the emergence of paranoia into cognition became apparent as the amount of explained variance increased. It is possible with further time series measurement (Time 4 etc.)

that an inverse relationship between cognition and paranoia exists. That is, the cognitive state may then have a causal influence on paranoia, leading to an ever-increasing deteriorating state within the psychotic condition. Further deteriorating cognitive performance may not only lead to an increase in paranoia but may then expand into influencing other psychological states over time. This could lead to a bifurcating event (Gleick, 1987) where the influence of cognition becomes predominant towards other conditions over time. This theory is diagrammatically represented in the following figure, where cognition at Time 3 influence anxiety, depression, phobia and somatic concern at Time 4. In order for this effect to occur it may require the psychotic experience to deteriorate at a significant level. It is also possible that not all dimensions are influenced by deterioration in cognition, nor in the same degree. Other aetiological effects may be present that influences the expression of other psychological states. For example, the "snow ball" effect of somatic concern previously stated may be due to an increase in self monitoring, as response to the introduction of neuroleptic medication as part of treatment.

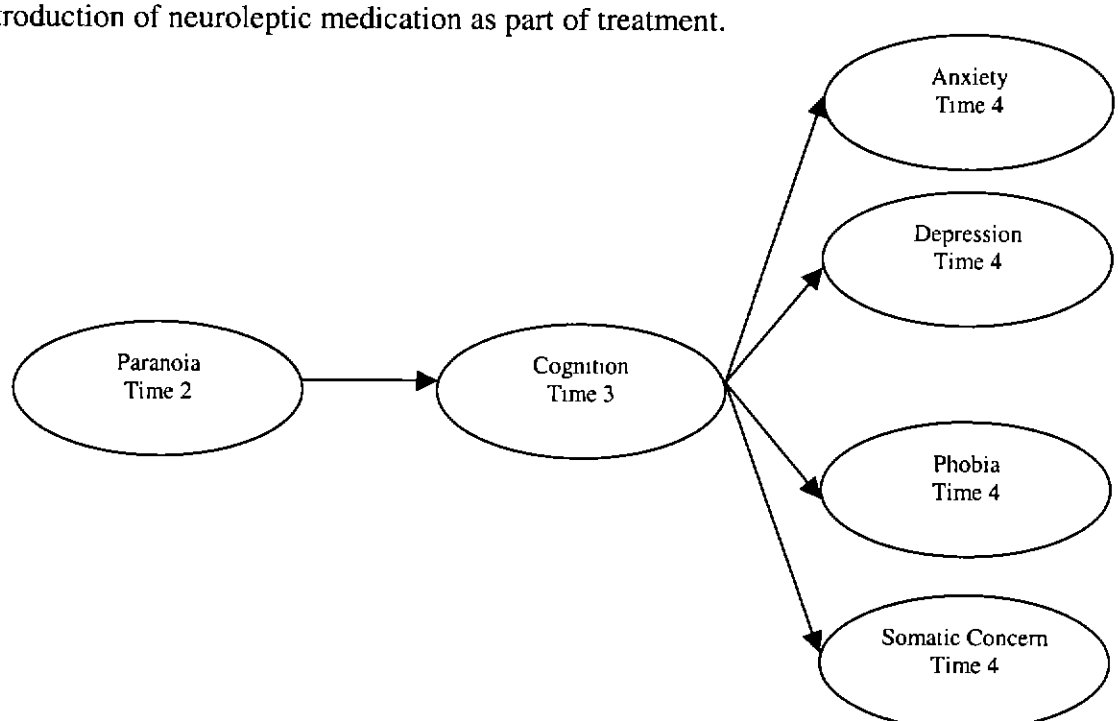

Figure 7.5. Bifurcation of causal influence of cognition on other psychological states within an extended time series.

Some discussion is also required on the other structural models that demonstrate no cross-influential effect between cognition and the other constructs. By turning to the literature the term symptoms will be used to denote conditions expressed by psychotic patients, which are determined by expert opinion and observation and not to internal state. Recent literature suggests that symptom clusters may act independently over time. A study conducted by Arndt et al. (1995) followed up neuroleptic naïve patients suffering from schizophrenia over a two -year period. Subjects were administered a structured interview using the SAPS (Andreasen, 1984a) and SANS (Andreasen, 1984b). A 4-factor model was examined looking at positive symptoms, negative symptoms, psychoticism and disorganisation. By performing a longitudinal factor analysis over the two-year period, negative symptoms were found to be stable and prominent while positive symptoms, psychoticism and disorganisation were less prominent and tended to be unstable over time. Ardent found the patterns of exacerbation and remission during the course of schizophrenia to be independent in each of the symptoms. Ardent argues that the symptoms in fact have an "independent pattern of evolution" (Arndt et al., 1995 p. 352). A study by Eaton et al. (1995) followed up 90 patients with schizophrenia, where symptoms were rated every month for 10 years following first hospitalisation. Bivariate dichotomous time series and dichotomous factor analysis were performed to examine the relationship between the symptoms. Eaton found that there was a tendency for positive and negative factors to merge over time. Both positive and negative symptoms in the preceding month were highly predictive of the following month. Neither symptoms however, were strongly associated with the other type in the following month when both symptoms were included in the model. Eaton

concludes that both the negative and positive symptom clusters are independent both cross sectionally and longitudinally. Such results are similar to those found in this thesis, where most psychological states did not show a cross-lagged effect suggesting that they act independently of each other. What is interesting with the Eaton study is the suggestion that the negative and positive symptoms merge over time. Paranoia is classified as a positive symptom while the item descriptors in the cognitive factor can be classified under negative symptoms. The current series of research may lend support to Eaton's suggestion that these two dimensions merge over time where both the paranoid state and cognitive functioning become less separate entities and more convergent over time.

Long and Brekke (1999) examined the longitudinal factor structure of the Brief Psychiatric Rating Scale (BPRS) (Overall & Gorham, 1962) over a three year period. A 4-factor structure describing thought disturbance, anergia, affect and disorganisation were found to be relatively stable over time. However this stability was found only at the factor level known as configural invariance and not at the metric or phi invariance level (Horn & McArdle, 1992). Although the researchers found the factor structure to endure over the three year time period, the strength of their interrelationships varied (Long & Brekke, 1999 p.505).

Such evidence as the studies mentioned above suggests that the interrelationship between symptoms in psychosis may be tenuous at best and possibly unrelated. The current series of research in relation to cognition supports this assertion with the exception of paranoia. It is possible that each symptom behaves without any cross-influential effect, and that no such causal predominance between symptoms exist. The suggestion of the bifurcating event where cognitive

deterioration sets off a causal sequence that influences other symptoms demonstrated in Figure 7.5 may not be plausible. Some suggestions on further research to address these concerns will be discussed in detail later.

The course of paranoia and cognition over time

After establishing the structural relationships between paranoia and cognition, a supplementary analysis was conducted to examine the direction of psychopathology for each symptom independently over time. By using latent mean structures with the dimensions of cognition and paranoia each time wave was examined over time in terms of trends. The trends demonstrated a mild U shape curve for cognition and a mild linear reduction in paranoia over time. This suggests that the reduction in psychopathology was evident but in mild terms. The real interpretative quality lies not in the trends of each symptom over time but the structural influences they play when interacting with each other. With paranoia having a cross-lagged effect on cognition, the results suggest that there is a deteriorating influence emerging between paranoia and cognition. When one additionally takes into account the standardised path coefficient leading from paranoia to cognition that shows an influential affect (Kline, 1998), the emergent picture is one of a deteriorating course in the illness. This can only be interpreted in relation to these pair of constructs and their inter-relational nature, but it is possible to assume that the influence of paranoia on cognition over time would have a harmful effect. This effect only examines the course of the illness at the early stages of the illness and provides a simplistic causal picture of outcome. Many studies in the area cite the influence of strong situational factors such as drug

use (Linszen et al., 1994), and duration of untreated psychosis as predictors of relapse (Carbone et al., 1999; Edwards et al., 1999; Ho, Andreasen, Flaum, Nopoulos, & Pharm, 2000; McGorry et al., 2001). Added to this are concerns that schizophrenia in reality is a heterogenous condition with varying outcome. This is mainly because of the methodological flaws that plague most longitudinal studies in the area. The greatest concern is that often studies do not account for differences between those who stay in the study and those who drop out. Often longitudinal studies ignore cases with very good outcome on one hand, and those with very poor outcome on the other (Riecher-Rössler & Rössler, 1998). With these criticisms in mind it is difficult to predict with those cases used in the structural model, what the likely outcome or course of their condition is. What is generally accepted however, is that the steady degenerative course of the illness describe by Kraepelinian notions of dementia praecox is a too simplistic description of the course and outcome of all cases with schizophrenia and other psychoses. More complex situational and biological factors are at play that determines the history of the illness for any given person.

Beta Gamma Change

After identifying the cross-lagged effects between cognition and the other constructs, the research analysed whether these constructs remain in the same condition at each time period. As noted in the Long and Brekke's study (1999), concern in the literature has been mounting over the stability of symptom structures in longitudinal schizophrenia research. This is particularly important in studies that investigate the impact of antipsychotic drugs and various treatment modalities. Like

all longitudinal research in schizophrenia, the concern has been with changes in the mean scores of constructs over time, without much consideration whether the meaning of the constructs have in fact changed over time. As Long and Brekke (1999) have pointed out, "researchers need an invariant symptom structure to unambiguously interpret changes in symptom level" (p. 498). The investigation of alpha, beta and gamma change is the statistical technique that investigates these changes.

To recapitulate, alpha change is understood as change in the extent of a given measure or scale. Beta change occurs where the measuring instrument has undergone some recalibration. Gamma change is where some domain measured is fundamentally redefined or reconceptualised (Golembiewski et al., 1976). Conceptually, researchers in psychological medicine may hope for alpha change since any variation in scores do not have to take into account reconceptualisation or recalibration of the meaning of the instrument. Alpha change is considered as an "absolute change" (Milsap & Hartog, 1988 p. 574) since the underlying construct is understood to be the same but the extent of the change varies. It was noted that the issue of psychometric stability is compounded by the very condition under investigation. The very nature of psychosis pre-supposes some distortion in reality (Sims, 1997) where self-awareness (insight) and self-analysis (reflection) are compromised. This means that stability of construct definition is threatened by the stability of the mind, let alone how one conceptualises a given construct with a sound mind. With psychosis, gamma change may occur from two positions. One is where the person has improved to such an extent that they appraise the world inherently differently, than when they were in an acute stage of the illness. The other position is equally likely, where the person has so significantly deteriorated that they appraise

themselves equally from a very different conceptual framework. Each position paradoxically demonstrates gamma change; one for the better and one for the worst.

Only the structural model between paranoia and cognition was examined for alpha, beta, gamma change, since it was these constructs where a cross-influential effect was found. Although a cross-lagged effect occurred between paranoia and cognition, it is not sufficient to assume that these relationships operate at the same stages of change over the time periods within the study. The purpose of investigating alpha, beta and gamma change is to qualify the relationships with reference to whether the constructs have undergone a change in the extent of the construct (alpha change), conceptual understanding of the items (beta change), or conceptual understanding of the construct itself (gamma). Evidence for beta or gamma change would require the relationship between paranoia and cognition to be placed within the context of the change process evident with the constructs over time. Investigation of alpha, beta and gamma change proceeded by examining the baseline model of the two-factor theory of paranoia and cognition. Invariance in form between the three time waves assumes alpha change. The second procedure examined gamma change by constraining the covariances amongst the factors to be equal between each time frame. The third model tested the first stage of beta change, where the variances within the factors were constrained to be equal across each time wave. Finally, model four, in addition to all the other constraints, the item loadings for each factor are held to be equal between each time wave. If at the end of all of these increasingly restrictive hypothesis the null position is accepted, the measurements are said to remain within an alpha condition (Schmitt, 1982).

The analysis demonstrated that the structural model between paranoia and cognition met each restrictive hypothesis within the null position, confirming that both constructs did not move from an alpha change position. Such a result allows for the associations interpreted between paranoia and cognition to operate within an "absolute change" framework (Milsap & Hartog, 1988 p.574). This demonstrates that the research underwent the strictest tests concerning change, and found the associations within the structural model not be threatened by beta or gamma change conditions.

Limitations of the research

As in all research, some limitations became evident in the thesis that need to be addressed. Some of the following limitations are caveats that need to be considered when interpreting the results reported and others suggest directions for further research.

The first concern lies with the measurement instrument itself. As described in Chapter IV, the Brief Symptom Inventory (BSI) (Derogatis, 1982) is not an instrument designed to measure specifically psychosis or personality style suggestive of persons vulnerable to psychosis (Cuesta et al., 2000a). The instrument is designed as a broad measure of psychopathology typical of patients receiving psychiatric care. In this instance it could be argued that the BSI lacks specificity in measuring psychosis itself. Although the measurement model describes a model of psychosis, the factors used (apart from paranoia) are not necessarily descriptive of psychosis. Other factors in the measurement model could exist in any number of other

psychiatric conditions including depressive and anxiety disorders. The threat to construct validity in this instance lies in the choice of instrument. Recent studies have attempted to address this limitation by examining schizotypal personality types and their risk in expressing psychosis (Mc Miller et al., 2002; Miller et al., 2002). However, even with instruments with higher specificity of measurement of psychotic phenomenon, they demonstrate poor predictive validity in determining persons who may become psychotic or develop schizophrenia. For example in Miller et al. (2002) study of 78 subjects with high schizotypal personality type, only 7 cases developed schizophrenia within 39 months of administering the questionnaire. Such results suggest that although other instruments may be more specific in the measurement of psychotic experience, there is no guarantee that such an instrument may be more predictive in determining whether a case will express schizophrenia.

The uniqueness of the present study does not lie in the choice of measuring instrument but on the population of interest and the fact that the source of the data came from self-reported measurement techniques. The measurement model was not in relation to the measuring instrument itself but in the population of interest. Both the validation and cross validation samples used DSM-III-R (APA, 1987) definitions of psychosis and each case was treated under the national clinical guidelines for treating patients with their first episodes of psychosis (Whiteford, 1996). Despite identifying differences in age and diagnostic differences (this is due to differences in the time when the instrument was administered and the age group in each treatment population), a common measurement model was found under strict confirmatory factor analytic techniques. In addition, self reported measures that measure schizotypal personality are relatively under developed in psychiatric research. Often there has been an over reliance on observational measures as the main investigative

technique when measuring psychotic experience (see Table 2.5). Only recently has research seriously considered self reported measures of schizotypal personality as a predictive measure of the expression of psychosis (Bergman et al., 2000; Claridge et al., 1996). To gather this type of data over a one year time period in a prospective cohort study as the one described in this thesis would be beyond the scope and resources of the author.

Another limitation of the study concerns the heterogeneity of the cohort used in the structural model. Although the majority of cases in the structural model were diagnosed with schizophrenia (53.4%), not all cases had the same condition. The concern with the research was on psychotic experience per se as opposed to "schizophrenic" experience. Improved specificity of measurement does not lie only within the measuring instrument but also within the cohort itself. The more homogenous the cohort, the greater the specificity of what phenomenon one is describing. Although ideally it is better to investigate a cohort that is homogenous in nature it is difficult in practicality.

In reference to schizophrenia, a number of problems are faced with a researcher using longitudinal research designs. First, schizophrenia as a specific condition is a low prevalence disorder. Recent research within Australia placed persons at risk of developing schizophrenia but who are currently asymptomatic at 20 per 100 000 in the general population (Jablensky, 2000). This classes schizophrenia as a low prevalence disorder. Studies that use schizophrenia as a longitudinal cohort are plagued by the difficulty in finding genuine cases of schizophrenia and retaining them within the study. Due to the nature of the illness, persons with schizophrenia are often transient, difficult to engage in treatment and research, and are often too

disorganised to participate in systematic studies. This leads to problems of high drop out rates where those who stay within the study are likely to be of relatively high functioning. The lowest functioning cases find it difficult to participate, and the highest functioning patients no longer desire to participate in a study that reminds them of times of illness or distress (Riecher-Rössler & Rössler, 1998). All studies that involve schizophrenia are faced with the same limitations. The present study attempted to address the issue of drop out, by comparing scores on the dimensions in each time wave compared to those who remained in the study (see Table 5.2). Of the 18 comparisons only one dimension (somatic concern at Time 2) proved statistically higher for excluded as opposed to cases within the study. This suggests that the drop out cases in this study were not significantly different from those who remained. This was also the case for patient characteristics of age, age at onset, educational level, diagnosis, gender and maximum chlorpromazine equivalent dose. Some difference did exist however between the two samples. Cases used for the structural model had a longer duration of hospitalisation and were less likely to have ever been in a relationship.

In reference to diagnosis, at the early stages of a psychotic disorder, it is very difficult to assume stability (McGorry et al., 1995b; Schwartz et al., 2000). Added to this concern is that even within diagnosis, the experience of psychosis is variable and transient in nature (Riecher-Rössler & Rössler, 1998). So not only is the diagnosis unstable in the first one to two years of a psychotic illness, but also the expression of the illness over time is dependent upon a number of factors that are extraneous and unpredictable (i.e., drug use or family dynamics). This makes modelling a sequence of events within psychosis rather problematic when the diagnosis is unstable and the expression of the illness is also variable. The concern with the current research was to

explore linkages between dimensions during the first stages of a psychotic illness. There was no real attempt to claim that the cases in the structural model are part of a particular nosological structure within psychotic disorders. To do so would be making claims beyond what the literature understands psychosis to be. That is a highly unstable phenomenon at the early stages of the illness. To this end, the modelling procedure was on psychotic experience and not 'psychotic experience of persons with schizophrenia' (although the majority of the cases in the structural model had a diagnosis of schizophrenia). Although previous studies have found schizophrenia to be a relatively stable diagnosis over time (90%) (Fennig et al., 1994), less specific DSM-III-R disorders such as schizoaffective disorder (36%) and psychoses not otherwise specified (44%) are below chance and hence highly unstable. To be cautious it was prudent not to assume in the research that the modelling procedures examined the causal predominance of schizophrenia but of psychosis in general.

Another limitation to the research is the assumption that all cases within the structural model had the same course or outcome. As been stated before, there are many limitations in longitudinal psychoses research due to the variability of the illness over time (Riecher-Rössler & Rössler, 1998). Often cases within a time series are assumed to follow the same general pattern. This is often not the case, as between 30% to 60% of early psychosis cases demonstrate good recovery some three years after initial treatment (Singh et al., 2000). This wide variability makes it difficult for researchers to make accurate causal inferences with their data. With respect to this thesis, to identify and divide cases with good, stable or poor outcomes and analyse the different effects would run the risk of making the study so statistically underpowered that it would make it impossible to proceed. Coupled with the fact that psychosis is a

low prevalence disorder, to gather enough data for one single doctoral thesis, modelling three separate outcomes would be beyond the scope of the researcher's resources. In addition, the time frame needed to gather these outcomes in equal proportions for adequate modelling to proceed would be possibly longer than the expected time to complete a doctoral thesis.

Another concern of the data was the low internal reliability of some of the factors over the time wave (see Table 5.4). By the third time wave, the constructs of anxiety ($\alpha = .597$), phobia ($\alpha = .576$), and somatic concern ($\alpha = .511$) displayed internal consistency lower than the acceptable threshold of .70 (Nunnally, 1978). Such results may have effected the true underlying association between the variables within the cross-lagged models. It is quite possible that with better internal consistency, more reliable associations between the constructs over time could have been examined.

The final concern is that the current research was unable to link biological and situational factors in the modelling process. In many studies in psychosis, there is recognition that both biological factors such as neuropathological effects (Kotrla et al., 1997), and situational effects such as drug use (Ayuso-Gutierrez & del Rio Vega, 1997; Newcomb & Bentler, 1989; Newcomb, Maddahian, Skager, & Bentler, 1987; Stein et al., 1987), can effect the expression and course of psychosis. It would be of major interest to observe these endogenous effects on the exogenous variables. Such an analysis would have looked at causative variables outside of the constructs themselves. The current study examines an 'internal state', where a particular psychological state or set of states effects each other in turn. It was not possible at the stage of data collection to examine the causal influence of variables that lie outside

the expression of these states. Since the current research could be seen as a 'first stage' study (since no other studies have used structural equation modelling to examine the causal influence of constructs) to include these variables (if they were at all possible to collect), may have been a little premature. With larger data sets a more complete model could be proposed by examining all cross-lag influences between symptoms simultaneously. The contribution of this thesis is that it paves the way for more complex structural models to be examined in light of the procedures set down within research. With this in mind attention will now turn to directions for further research.

Further research in modelling psychosis

Despite the limitations, there are many positive elements to the research that points a way to new areas of investigation. The uniqueness in the research may not lie necessarily in what was found, but the technique by which it was explored. The research provides a unique contribution to the literature through the use of confirmatory factor analysis, and structural equation modelling. First, the research was able to demonstrate a measurement model of the psychotic experience that was confirmed on two independent samples of early psychosis subjects. To the knowledge of the author this is the first time in psychosis research that (a) a self reported measure was used as an underlying measure of psychosis and (b) that the measurement model proposed was cross validated on an independent sample. Although many studies in psychosis have used confirmatory factor analysis to model the illness (see Table 2.5), none of these studies used a cross validation sample.

Second, once the measurement model was confirmed the research examined the influence of these constructs on each other causally over time. The major focus was to examine the influence of cognition on the other psychological states. This is the first time that structural equation modelling has been used on an early psychosis cohort, to examine the influences of psychological states causally over time. The following suggestions for further research will explore this framework of examining psychosis as a dynamical system within the methodologies employed in this thesis. To this end it is anticipated that other researchers may develop the techniques used herein as a basis of further research into psychosis.

Aetiology, pathophysiology and symptom expression of psychosis

Although there appears some general consensus as to the structure of psychotic experience, little is known of the causal connection between their expression and antecedent variables. What is understood however, is that schizophrenia and related psychotic illnesses do not have a single aetiological cause nor is it a syndrome where it consists of many independent causes like pneumonia (Feinberg, 1997). It is more likely that the emergence of schizophrenia is due to multimodal and interactional causes. Many studies have been conducted in brain pathology, familial genetic exposure, social predictors, neuropsychological, pre natal and birth complications and post-natal markers (Jablensky, 1997). Although all of these studies have shown some significant contribution of each of these factors towards the emergence of schizophrenic type symptoms, very few studies if any, have been able to integrate the interactional nature of the antecedent variables that

contribute to schizophrenia. Only recently has there emerged discussion towards an integrated model of psychotic experience particularly within schizophrenia (Keshavan & Murray, 1997). Such discussion has centred around developing a unifying theory which examines the antecedent factors contributing to the course and outcome of schizophrenic expression.

Theorists such as Keshavan (1997) have proposed that the:

> schizophrenic syndrome results from a cascade effect of possibly genetically mediated derailment in early and late maturational processes of brain development interacting with adverse humoral and psychosocial factors which occurs later during adolescence and early adulthood (p.267).

Two views have emerged as to the aetiology of schizophrenia. The first view identifies pre and perinatal markers where abnormal brain development occurs perhaps during the second half of gestation. It is argued that a fixed legion somewhere early in life interacts with normal neurodevelopment (Murray & Lewis, 1987; Weinberger, 1987). The second view supports a later stage in neurodevelopment where the pathology has its onset in post-natal stages of development (Keshavan, 1997). These theorists suggest that schizophrenia may result from an abnormality in periadolescent synaptic pruning (Feinberg, 1997). The later view is supported by the fact that the onset of schizophrenia and other psychoses usually occurs in late adolescence to early adulthood (Bell et al., 1998; McGorry et al., 1995a; Yung et al., 1999). Whether the insult occurs during pre and perinatal periods, or sometime later in early adolescent, it is possible that these different

markers lead to the expression of brain pathophysiology. The brain pathophysiology, in turn, lead to symptom clusters (factors) which are manifested in external symptomatology. Such a causal pathway is expressed in the following figure.

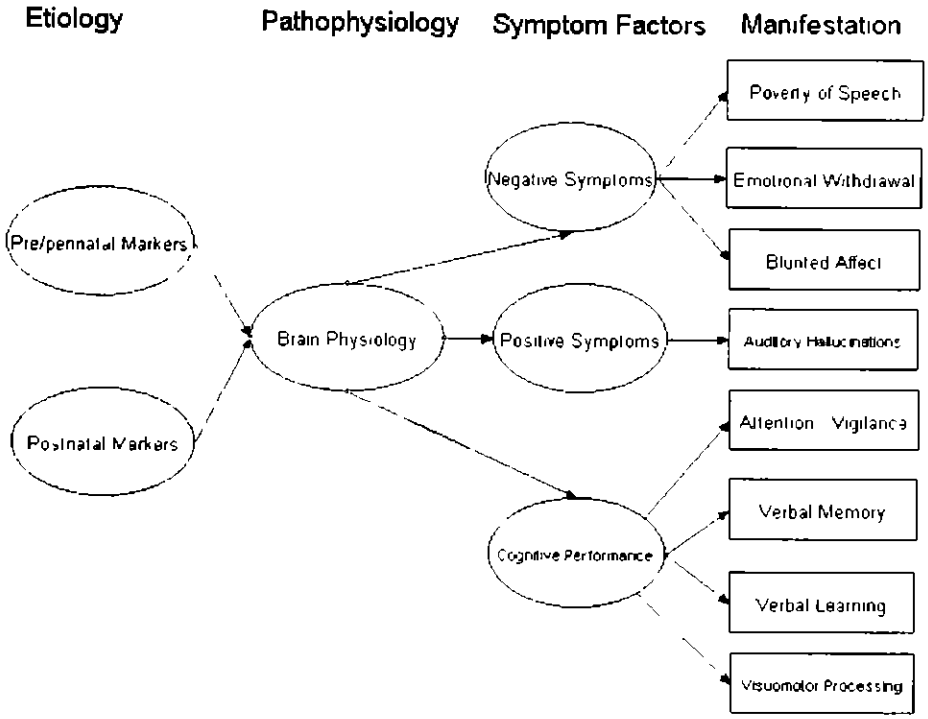

Figure 7.6. Schematic representation of causal pathways leading to the expression of psychotic symptoms.

With the pre and perinatal markers a number of risk factors have been studied which indicate exposure to the risk of brain adnormalities. For example, genetic studies with twin discordant for schizophrenia shows a large heritability (Cardino et al., 2001; Sharma, Davies, Ribchester, Chitnis, & Murray, 2000). Other studies have examined whether exposure to infectious disease during the prenatal period may contribute to an exposed risk of developing schizophrenia (Clarke et al., 2000). Other prenatal exposures include famine such as the Dutch hunger winter at the end of World War II have also been investigated for increased risk of psychosis in Holland (Hoek, Brown, Neugebauer, & Susser, 2000). Other studies have investigated the

influence of obstetric complications as a contributing factor towards the development of schizophrenia (van Oel, Sitskoorn, Geurtsen, & Kahn, 2000). Such studies as these indicate that researchers identify a multifactorial aetiological structure in reference to pre and peri natal markers that may increase the risk of developing schizophrenia.

Post-natal markers largely concern those around the area of early adolescence. These could include the late development of gene expression (Keshavan, 1997), or hormonal differences between males and females. Psychosocial stressors such family dynamics and drug use (Ayuso-Gutierrez & del Rio Vega, 1997), also play a part in the expression of schizophrenia and other psychoses. These markers for both pre and perinatal and post natal periods are expressed in the following figure.

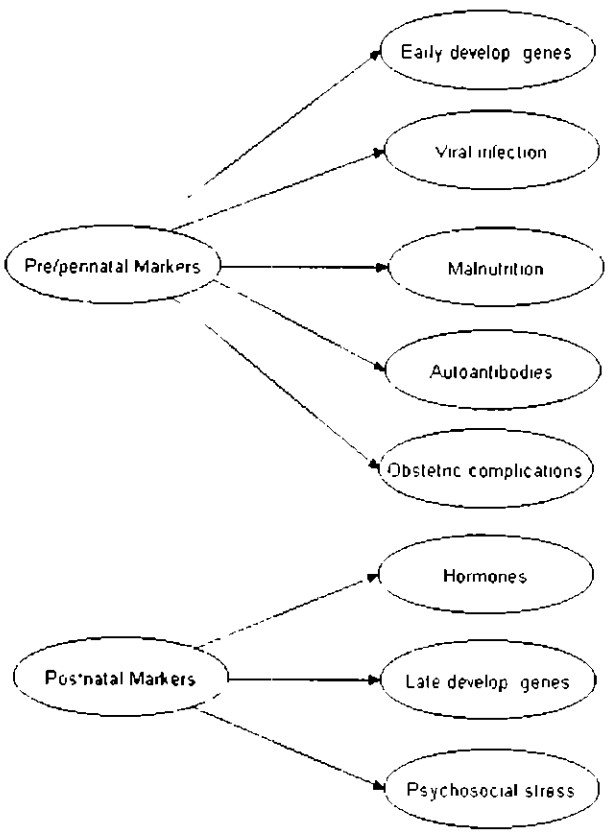

Figure 7.7. Variables which express pre/perinatal and postnatal markers of schizophrenia within a neurodevelopmental hypothesis.

Such markers are argued to contribute to brain pathophysiology which in turn lead to the expression of the clustering of symptom factors demonstrated in Figure 7.8. A great deal of study has emerged in recent years through the advent of Magnetic Resonant Imaging (MRI) and similar imaging techniques to identify structural abnormalities indicative of people who experience schizophrenia (Bullmore et al., 1997). Such brain pathophysiology is expressed in the following causal diagram.

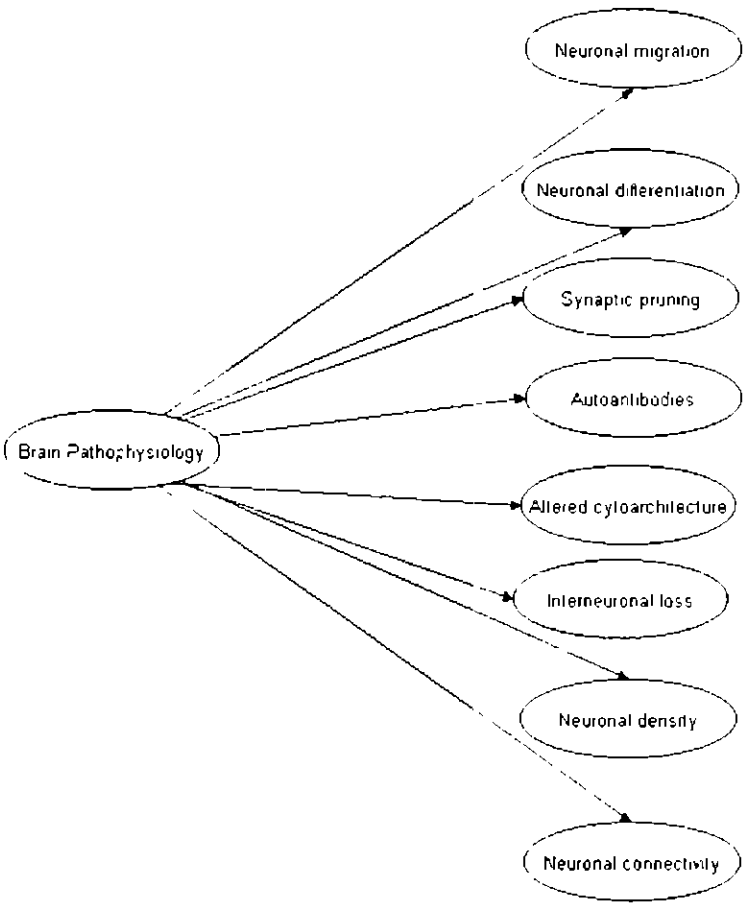

Figure 7.8. Variables which express brain pathophysiology of schizophrenia within a neurodevelopmental hypothesis.

It is not the scope of this thesis to review in detail the studies within brain pathophysiology that relate to schizophrenia, however it is suffice to say that dominant paradigmatic arguments have emerged from such research. Increasingly the link between symptom expression and brain pathophysiology has been recognised. Positive and negative symptoms and cognitive performance deficits are said to arise from these brain abnormalities highlighted in Figure 7.6. Most notably, negative symptoms have been linked latently to abnormal neural network integrity, while positive symptoms are most notably linked towards areas in the brain which control speech and auditory hallucinations appear to be related to dysfunction in language systems (Keshavan & Murray, 1997). With regard to cognitive performance testing, those who suffer from psychotic illness tend to have marked difficulties in verbal memory, verbal learning and visuomotor processes (Bullmore et al., 1997). The manifestation of symptoms highlighted in Figure 7.8 can quite plausibly be latent to marked changes in brain physiology highlighted earlier. In other words, symptomatological models such as those in this thesis may latently describe subtle, but significant neurological changes anteceded by genetic and pre and post-natal markers.

It is important to note that the causal models illustrated in the preceding diagrams do not have causal pathways that lead back to other antecedent variables. This has been deliberately done to simplify the models because very little is known of the interactional nature of the antecedent variables with each other and the expressed symptoms which are said to give rise from these variables. To date there has been no research that looks at the causal mechanisms that look at all causal variables simultaneously.

The contribution of structural equation modelling as demonstrated by the research conducted within the thesis could be used to link the different levels of research with psychoses into an integrative model suggested in Figure 7.6. Indeed, such a technique would allow for each exogenous and endogenous variable within the model to be examined causally and cross influentially. The present research identifies how this could be done when one looks at the interactive nature of psychological states. Further research is required to link the aetiological, pathophysiological and manifest variables into an integrative framework (see Figure 7.6). Since psychosis is a dynamic and highly elusive phenomenon, it requires researchers to be more coordinated between the different levels of research. Only through cross-disciplinary studies that examine relations between aetiological factors (e.g., genetic factors), neuropathology (e.g., reduced synapse density) and phenomenology (e.g., positive and negative symptoms) (Keshavan, 1997) will an integrative framework arise. Otherwise it is likely that the phenomenon will remain mysterious as each discipline continues to narrowly focus on their restricted part of the "jigsaw". It is only by assembling the pieces that a framework of understanding can be achieved. To date, what can be concluded out of an integrative causal framework can be summed up by Keshavan (1997) where he argues that:

> It is reasonable, however, to conclude that disordered brain development mediates the pathogenesis of at least a subgroup of patients with the schizophrenia syndrome. The emergence of psychopathology in adolescence is best explained by an interaction between early neurodevelopmental lesions and a disruption in late postnatal brain maturational processes; genetic factors and environment variables, both biological and psychosocial, may be

involved in mediating the emergent developmental neuropathology (p. 275).

The future of research in schizophrenia and other psychoses lies in integrating these levels of research into a causal framework. By examining different pathways both with fixed and modifiable antecedent variables (e.g. peri and post-natal markers), researcher will be able to cautiously proceed with an integrative framework. In order to understand some of these frameworks some discussion is require on the notion of open and closed causal systems.

Open independent versus closed dependent causal systems

In science, it is important to be reminded that " ...independence is a political, not a scientific term" (Margulis & Sagan, 1995 p 26) Advances in ecology, biology and physics have led scientists further into systemic inter-relational models of reality (Capra, 1982; 1988; 1997). In order for models of mental illness to be both accurate and eloquent, this recognition of interrelatedness must emerge. Systems can only be observed in a dynamical sense within time and can only emerge when time is treated as part of the patterning process. The difficulty in modelling psychotic experience is whether the antecedent variables operate within an open independent system of cause, or a closed dependent system of cause.

The open independent system of cause postulates that antecedent or causal variables drive the expression of the psychotic experience (in this instance the

presence of symptoms) and are not in turn influenced over time, by the symptoms themselves. This is demonstrated in the following figure.

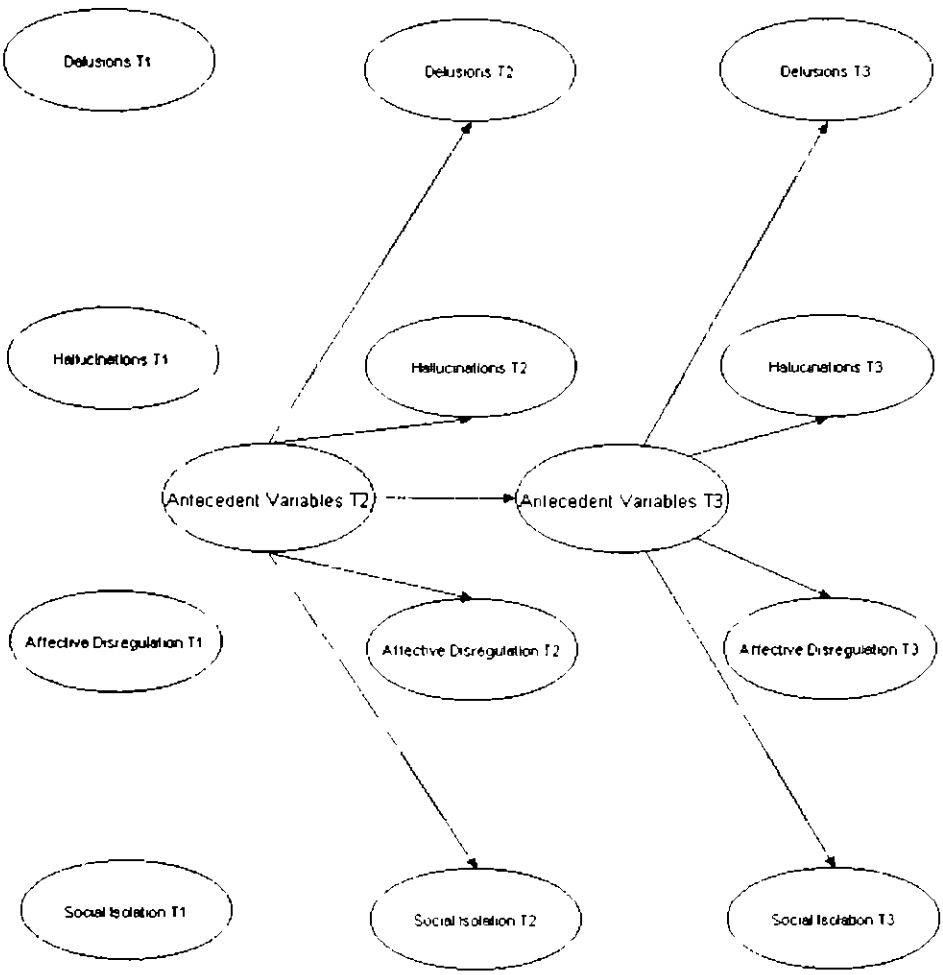

Figure 7.9. Example of an open independent system of cause of psychotic experience.

In Figure 7.9 pathways do not lead back from the symptoms in Time 2 (T2), to contribute to the antecedent variables in Time 3 (T3) (ignore Time 1 in this illustration) which in turn affect the expression of symptoms in Time 3. Such a system equates with the notion that aetiological factors operate independently of symptoms, and are the sole drivers of the expression of symptoms. In such a system,

the model of psychotic experience is a cascading expression of symptoms but the experience does not in turn affect the antecedent variables. This model ignores a psycho-toxic effect where the psychotic experience itself is further damaging to the brain physiology which can lead to a deteriorating course of the illness.

The second system of cause argues that the symptoms themselves can contribute to the antecedent variables and as such, the system is closed and dependent on all of the variables antecedent and expresses an effect on each other. This system is illustrated in the following figure.

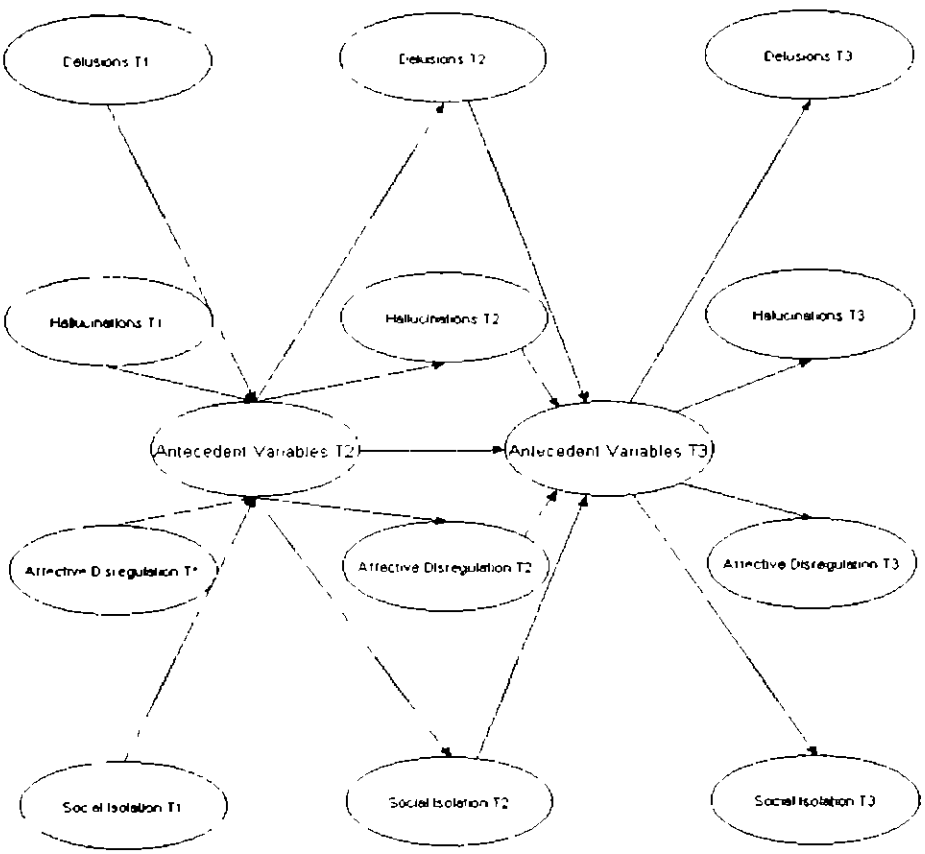

Figure 7.10. Closed dependent system of cause of psychotic experience.

The system illustrated in Figure 7.10 demonstrates causal pathways which lead back to the antecedent variables from the preceding symptoms. Once again, to assist with clarity, pathways leading from symptoms to each other are not presented in this model. Such a closed dependent system argues a psycho-toxic hypothesis as the strength of the psychotic symptoms can in turn affect the antecedent variables. The model can easily be increased in complexity when one considers fixed and modifiable antecedent variables. Such a model is only related to modifiable antecedent variables that can naturally change over time. These could include brain physiological variables. Fixed antecedent variables are those causal variables that only occur once in a given system or cannot be influenced by other variables, these could include birth complication and genetic marker variables.

Problems with modelling psychotic experience

Modelling an illness as complicated as psychosis and in particular schizophrenia is problematic. Currently schizophrenia is largely understood as an illness that has many endogenous and exogenous causal factors that express symptoms over a long period of time. Mapping these variables requires longitudinal data which is time consuming and expensive to execute. To date, no research has been able to track a majority of antecedent variables over time, and to investigate which variables predominate in expressing symptoms over time. Coupled with this, is the requirement of obtaining large sample sizes that meet the statistical assumptions of statistical modelling techniques such as Structural Equation Modelling (SEM). Such techniques are "hungry for statistical power" in order for all parameters to be

effectively calculated. Coupled with the fact that epidemiologically schizophrenia is a low prevalence disorder of approximately 1 in 100 people (Jablensky et al., 1992), large prospective studies are rare and difficult to conduct. Given these limitations, it is only currently possible to investigate one area of the illness and attempt to understand it more comprehensively. However in order to do this effectively it is important for research to be contextualised into a unifying theory of how such complex phenomenology may arise in combination with other factors. Further research into psychosis and schizophrenia needs to be better coordinated at all levels of the phenomenon. Since it is well understood that psychotic experience is a complex phenomenon, the research that is conducted in the area requires the same sophistication to meet the challenges of the condition.

It has been argued in this chapter that structural equation modelling needs to proceed in psychosis research by linking the aetiological factors, neuropathology and phenomenological expression together in a causative framework. In addition, both open independent and close dependent systems of cause have been postulated. These theories examine whether the psychotic experience has a psycho-toxic effect on the brain (closed dependent system) or rather that symptoms are simply an expression of brain pathophysiology (open independent system). A number of diagrams (Figure 7.6, 7.7 and 7.8) demonstrate how these models could be examined if and when research is coordinated at the three levels suggested by Keshavan (1997). To meet these challenges, cross-disciplinary research is required where experts at all levels of analysis are required to link their different levels together. Structural equation modelling (among other techniques) could assist in examining the frameworks of a unified theory posited by the models demonstrated within this chapter that fall under the current neurodevelopmental hypothesis. It is argued that without this unified

approach at all levels of research the psychotic experience will remain elusive to researcher, clinicians and sufferers alike. Without such an approach in psychosis research the following quote by Platt (1964) may remain true in years to come:

> We praise the 'lifetime of study', but in dozens of cases, in every field, what was needed was not a lifetime but rather a few short months or weeks of analytic inductive inference ...We speak piously of taking measurements and making small studies that will 'add another brick to the temple of science'. Most such bricks just lie around the brickyard (p. 351).

It is hoped that the models presented as suggestions for further research attempt not to build bricks but to suggest a building (i.e., a framework of understanding) by which the bricks can be laid.

Conclusions

In conclusion, the present study investigated the causal predominance of cognition on anxiety, depression, paranoia, phobia and somatic concern over three time waves of self reported data measured every six months over one year, with cases experiencing their first episodes of psychosis. In turn, anxiety, depression, paranoia, phobia and somatic concern were examined for their cross-lagged effects on cognition. Cognition was examined under a causal predominance hypothesis as the lead psychological state because of its influence recognised in the literature under the

neurodevelopmental hypothesis (Keshavan & Murray, 1997; Murray & Lewis, 1987). These effects were examined using structural equation modelling. Prior to this investigation, the research was able to demonstrated a stable 6-factor measurement model with these constructs between two independent samples of early psychosis cases, who meet treatment under the Australian National Early Psychosis Treatment Guidelines (Whiteford, 1996). This measurement model demonstrated good internal reliability and construct validity. Most constructs over each time wave had a "domino effect" where the construct prior to the next wave of assessment had an influence on subsequent constructs. This is known as a mediation effect. Somatic concern and depression demonstrated a "snow ball" or direct effect where the extent of the condition at Time 1, influenced directly the condition at Time 3. Structural models, which examined the cross-lagged effect between cognition and the other psychological states, demonstrated an effect between paranoia and cognition. This effect demonstrated that paranoia at Time 2 (ie, 6 months after stabilisation of constructs), had a cross-lagged effect on cognition at Time 3 (ie, 12 months after stabilisation of conditions). It was argued that poor thinking styles, which lead to distortion in feelings of mistrust evident in paranoia in turn, led to deterioration in cognitive or thinking capacity measured in the cognition factor. Other dimensions did not demonstrate a cross influential effect. The independence of the other constructs is supported by previous research, suggesting that these act independently of each other over time (Arndt et al., 2000; Eaton et al., 1995). Further research was suggested by linking the different levels of psychosis research of the aetiological factors (e.g. genetic factors), neuropathology (e.g., reduced synapse density) and phenomenology (e.g., positive and negative symptoms) (Keshavan, 1997) into an integrative framework. It was suggested that structural equation modelling, as exemplified in the

thesis could be used as a technique to examine how these differing levels could be investigated under a unified theory of psychosis based upon the neurodevelopmental hypothesis. The current research provides a unique contribution to the literature on psychosis on how such research could be conducted under a unified framework.

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APPENDIX I

Release letter authorising use of EPPIC data and Consent Form

RELEASE LETTER AUTHORISING THE USE OF EPPIC DATA

Dear Neil I fully endorse Susy's email to you as below Good luck Pat

Dear Pat

Some time ago you and I agreed to allow Neil Preston to use our SCL-90/BDI ratings and selected MIP data from our Cope pilot and Cope proper studies for his PhD.

Neil has now written to me to ask if it would be possible for us to put this consent in writing so that he can demonstrate that we have agreed to the use of this data. All we need to do is to write a simple email indicating that Neil has your consent.

I enclose a draft email for you to use, or you may prefer to draft your own.
Could you please cc me in your response to Neil?

Thanks very much, Pat.

Kind regards and best wishes,
Susy

Dear Neil

I hereby give my formal consent for you to use data from our COPE pilot and COPE proper studies conducted at the Early Psychosis Prevention and Intervention Centre for the purposes of your PhD research. I understand that the data includes SCL-90-R ratings and data from the Royal Park Multidiagnostic Instrument for Psychosis (RP-MIP) database for approximately 270 subjects in these two cohorts.

I would like to make it clear that you have my full support to use this dataset to write your PhD thesis.

Yours sincerely

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CONSENT/REQUEST TO PARTICIPATE IN A RESEARCH PROJECT

TITLE OF RESEARCH PROJECT:. Preventive Strategies in Early Psychosis .....
.....

CONSENT OF PATIENT OR GUARDIAN

The purpose of the above project has been fully explained to me and I have read and signed the attached PLAIN ENGLISH STATEMENT. I understand the aims and procedures of the experiment and any risks to myself which are involved and I request to participate on condition that I can withdraw my consent at any time.

I..... understand as part of the study I will undergo the following:-

1. Diagnostic interview at beginning and end of hospital stay, or during the time of contact with outpatients ..... YES/NO.
2. Completion of self-rating forms ..... YES/NO.
3. Contact six, twelve and twenty four months after leaving hospital, or if seen in outpatients, six, twelve and twenty four months after diagnostic interview ..... YES/NO.
4. Contact member or members of my family for interview ..... YES/NO.
5. Attend COPE therapy for approximately six months ..... YES/NO.

SIGNED: ..... DATE: .....

RESEARCHER

I ..... certify that I have fully explained the aims, risks and procedures of the experiment to the patient named herein (or to the lawful guardian of such patient) and have handed to the patient (or guardian) a copy of this consent together with a plain english statement of the aims and procedures of the experiment and any risks to the patient.
In my opinion the patient (or lawful guardian thereof) appears to understand and wishes to participate.
I undertake to the patient (or lawful guardian thereof) that the confidentiality and anonymity of the patient and his or her records will be preserved at all times.
In the case of a subject under section 12, I undertake to inform the relative named in accordance with the express wish of the subject as set out below.

SIGNED: ..... DATE: .....

GUARDIAN appointed under Guardianship and Administration Board Act to sign.

SIGNED: ..... DATE: .....

FOR PATIENTS UNDER SECTION 12

I, ... DO/DO NOT wish for my relative
..... to be informed of my participation in this research.

WITNESS OF PATIENT'S SIGNATURE

I, ... of .....
..... as an independent witness confirm that the aims and procedures of the experiment and any risks to the patient has been adequately explained to the patient whose signature I witness. In my opinion he/she appears to understand and wishes to participate.

SIGNED: ..... **DATE:** .....

PLAIN ENGLISH STATEMENT

The study we are asking you to take part in involves looking at how the length of time that you are unwell affects your recovery. We are also interested in what the cost is, of being unwell, to you and your family. In addition, we want to look at the way two different services treat people, and to see how this affects your recovery.

We ask you to take part in a number of interviews: two around the time you first come into the service, and one at each of the follow up times - at six and twelve months afterwards. We ask you about yourself, your having been unwell, and how you are feeling. We also ask you to fill out a few questionnaires. With your permission, we would also like to ask someone in your family some questions about how you are going.

All the information you give us will be kept safe and will remain confidential.

You can withdraw your permission at any time, should you decide to. Participating in this study will not affect the treatment you receive.

APPENDIX II

EPOES Client Assessment and Consent Form

EARLY PSYCHOSIS OUTCOME EVALUATION SYSTEM (EPOES) ©
CASE MANAGER ASSESSMENT

TO BE FILLED BY THE CASE MANAGER / CARE COORDINATOR ONLY

CLIENT DEMOGRAPHIC DETAILS / ENROL CLIENT

Unique Medical Record Number	
Client First Name(s)	
Client Last Name	
Date of Birth & Client's Age	____/____/____ D.O.B. _____ years
Residential Post Code of Client	_____
Client's Gender	1 male 2 female

EMERGENT SYMPTOMS

Date of onset of prodromal symptoms	____/____/____	**(Do not include positive symptoms, in your determination)**
Date of onset of psychosis **(Date 1)**	____/____/____	**(The commencement of positive symptoms, do *not* include the period of prodrome)**

PATHWAYS TO CARE

1. Help from non medical service	If client sought help directly from a GP worker go to Q 2			
Date client received help for emergent symptoms	____/____/____			
Who did they seek help from?	1	Teacher	2	Counsellor
	3	Youth worker	4	Alternative Therapist
	5	Religious / Clergy	6	Other
Type of treatment received?	1	Support	2	Referral
	3	Other (specify)		
2. Seeking help from general medical service	If client sought help directly from a mental health service go to Q 3			
Date client received help for emergent symptoms	____/____/____			
Who did they seek help from?	1	GP Medical Practitioner		
	2	Other: who?_____		
Type of treatment received?	1	Antipsychotic Medication	2	Other Medication specify: _____
	3	Other treatment (specify) _____		_____
3. Seeking help from specialist mental health services				
Date treatment first commenced in *any* recognised mental health service (Date 2)	____/____/____			

Estimated duration of untreated psychosis (Date 2 – Date 1)	Duration _____ months		(From the commencement of positive symptoms to treatment from a mental health service)
Was the patient at commencement in *any* recognised mental health service receiving:	Inpatient Services 1	Outpatient Services 2	Community Services 3
Date treatment commenced in your early psychosis program		____/____/_____	

EARLY PSYCHOSIS OUTCOME EVALUATION SYSTEM

INFORMATION AND CONSENT FORM

As mental health professional we are asked to demonstrate the effectiveness of services we provide to our clients. We would like you to assist us in evaluating the service we provide to you by filling out a questionnaire and participate in an interview. The questionnaire and interview will be conducted periodically to track your progress over time. This information will help both evaluate the program as well as give us useful information on how to better manage your treatment.

All information is kept strictly confidential and will be used for purposes of evaluating and researching the effectiveness of services we provide to you. Your participation is totally voluntary and you can terminate your participation in the evaluation system at any time. Not wanting to participate in the evaluation procedure will not in any way effect your right to receive treatment.

If you have any queries about the evaluation please do not hesitate to ask you Case Manager.

I _____ have read this information form and are happy to consent to participating in the evaluation system. _____/_____/_____

APPENDIX III

The Brief Symptom Inventory (BSI)

BSI
Brief Symptom Inventory

The following document is a questionnaire which asks how much you have been feeling about certain things over the past week. As part of an evaluation of the Psychiatric Rehabilitation Services, we would like to know how you have been feeling. This information will assist us in knowing whether we are being effective in the service we provide to you.

There are no trick questions.

You do not have to participate in the survey if you don't want to. All data will be treated in the strictest confidence and is only used for research purposes only.

How to fill out the inventory

On the next page is a list of problems people sometimes have. Please read each one carefully and circle the number which best describes HOW MUCH THAT PROBLEM HAS DISTRESSED OR BOTHERED YOU DURING THE PAST 7 DAYS INCLUDING TODAY. In the example below bodyaches have been ***quite a bit*** distressing for this participant so they circled number ***three*** which corresponds to their response.

	Not at all	A Little Bit	Moderately	Quite a Bit	Extremely	
						EXAMPLE
						HOW MUCH WERE YOU DISTRESSED BY:
	0	1	2	(3)	4	Bodyaches

INSTRUCTIONS

Below is a list of problems people sometimes have. Please read each one carefully, and circle that number which best describes **how much that problem has distressed or bothered you during the past 7 days including today**. Circle one number for each problem and don't skip any items. If you change your mind cross your first number. Read the example before beginning, and if you have any questions please ask about them.

	Not at all	A Little Bit	Moderately	Quite a Bit	Extremely	**HOW MUCH WERE YOU DISTRESSED BY:**
1	0	1	2	3	4	Nervousness or shakiness inside
2	0	1	2	3	4	Faintness or dizziness
3	0	1	2	3	4	The idea that someone else can control your thoughts
4	0	1	2	3	4	Feeling others are to blame for most of your troubles
5	0	1	2	3	4	Trouble remembering things
6	0	1	2	3	4	Feeling easily annoyed or irritated
7	0	1	2	3	4	Pains in heart or chest
8	0	1	2	3	4	Feeling afraid in open spaces or on the streets
9	0	1	2	3	4	Thoughts of ending your life
10	0	1	2	3	4	Feeling that most people cannot be trusted
11	0	1	2	3	4	Poor appetite
12	0	1	2	3	4	Suddenly scared for no reason
13	0	1	2	3	4	Temper outbursts that you could not control
14	0	1	2	3	4	Feeling lonely even when you are with people
15	0	1	2	3	4	Feeling blocked in getting things done

	Not at all	A Little Bit	Moderately	Quite a Bit	Extremely	HOW MUCH WERE YOU DISTRESSED BY:
16	0	1	2	3	4	Feeling lonely
17	0	1	2	3	4	Feeling blue
18	0	1	2	3	4	Feeling no interest in things
19	0	1	2	3	4	Feeling fearful
20	0	1	2	3	4	Your feelings being easily hurt
21	0	1	2	3	4	Feeling that people are unfriendly or dislike you
22	0	1	2	3	4	Feeling inferior to others
23	0	1	2	3	4	Nausea or upset stomach
24	0	1	2	3	4	Feeling that you are watched or talked about by others
25	0	1	2	3	4	Trouble falling asleep
26	0	1	2	3	4	Having to check and double-check what you do
27	0	1	2	3	4	Difficulty making decisions
28	0	1	2	3	4	Feeling afraid to travel on busses, subways or trains
29	0	1	2	3	4	Trouble getting your breath
30	0	1	2	3	4	Hot or cold spells
31	0	1	2	3	4	Having to avoid certain things, places or activities because they frighten you
32	0	1	2	3	4	Your mind going blank
33	0	1	2	3	4	Numbness or tingling in parts of your body
34	0	1	2	3	4	The idea that you should be punished for your sins
35	0	1	2	3	4	Feeling hopeless about the future
36	0	1	2	3	4	Trouble concentrating
37	0	1	2	3	4	Feeling weak in parts of your body
38	0	1	2	3	4	Feeling tense or keyed up
39	0	1	2	3	4	Thoughts of death or dying
40	0	1	2	3	4	Having urges to beat, injure or harm someone
41	0	1	2	3	4	Having urges to break or smash things
42	0	1	2	3	4	Feeling very self-conscious with others
43	0	1	2	3	4	Feeling uneasy in crowds, such as shopping or at a movie
44	0	1	2	3	4	Never feeling close to another person
45	0	1	2	3	4	Spells of terror or panic
46	0	1	2	3	4	Getting into frequent arguments
47	0	1	2	3	4	Feeling nervous when you are left alone
48	0	1	2	3	4	Others not giving you proper credit for your achievements
49	0	1	2	3	4	Feeling so restless you couldn't sit still
50	0	1	2	3	4	Feelings of worthlessness
51	0	1	2	3	4	Feeling that people will take advantage of you if you let them
52	0	1	2	3	4	Feelings of guilt
53	0	1	2	3	4	The idea that something is wrong with your mind

Last Name First

Age Gender __/__/__ Test Date

APPENDIX IV

Early Psychosis Outcome Evaluation System (EPOES) Study

Introducing a statewide evaluation system for early psychosis: Early Psychosis Outcome Evaluation System

Neil J. Preston, Maree L. Stirling, Kanthi Perera, Richard J. Bell, Tracey J. Harrison, Lisa Whitworth, David J. Castle

Objective: This paper describes a system of outcome evaluation for early psychosis programmes and presents the preliminary data generated by the system. The Early Psychosis Outcome Evaluation System (EPOES) was designed for use in a naturalistic, prospective study of a cohort of early-episode psychosis patients. This study describes early psychosis patients in terms of symptoms, substance use, social functioning and family burden, and examines the effectiveness of early psychosis programmes.

Method: Four sites in Perth, Western Australia, participated. Outcome was evaluated from three sources: case manager (CM), patient (P) and family member (FM). Seven clinical outcome measures were used: the Brief Psychiatric Rating Scale (CM), Brief Symptom Inventory (P), Substance Use (CM); Social Functioning Scale (P); Global Assessment Scale (CM); Burden Assessment Scale (FM), and the General Health Questionnaire-12 (FM) Measures were collected at intake (baseline) into a specialist early psychosis service and thereafter every 6 months until discharge from the service.

Results: After the first year of data capture, 84 baseline assessments have been completed, and 23 patients have been followed up at 6 months. Clinicians and patients reported significantly less psychopathology at 6 months compared to baseline Sixty per cent of patients reported marijuana use within 3 months of their baseline assessment, and 30% amphetamine, ecstasy or cocaine use. Significantly increased levels of psychopathology were recorded for substance-using patients Family members (59%) reported significant psychological distress at baseline; this was significantly reduced at 6 months. Patient social functioning and family burden did not improve measurably from baseline to 6 months.

Conclusions: The EPOES is an effective system that provides immediate feedback on the clinical status of early-episode psychosis patients Both observed and self-rated psychopathology and family member psychological distress, is significantly improved after 6 months of intervention. Family burden and patient social functioning did not demonstrate improvement within this time period Patient social functioning is revealed as an important area for treatment focus Substance use is associated with poorer psychopathology. Most notably, EPOES provides a useful and feasible system of collecting routine outcome assessment in early psychosis intervention

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Received 29 August 2002, revised 3 December 2002; accepted 10 January 2003.

Key words: early-episode psychosis, family burden, outcome evaluation.

Australian and New Zealand Journal of Psychiatry 2003; 37:000–000

Mental health services are increasingly required to measure the effectiveness of outcomes in clinical delivery [1]. The Second National Mental Health Plan [2] has set forth priorities for the development of mental health services in Australia. One of these priorities is the integration of consumer outcome measures into the daily practices of clinical services. Through the late 1980s [3] and 1990s [4], the Commonwealth Government sought a nationwide approach to measuring the effectiveness and outcomes of mental health services. As part of this initiative, early-psychosis programmes around Australia are required to demonstrate their effectiveness in providing sound clinical outcomes.

Early intervention with psychosis patients has been found to be effective [5,6], however, ongoing research and evaluation of services for this population are central to the success of new initiatives [7]. McGorry *et al.* [7] note that evaluation allows clinicians to judge the value of an intervention and that outcome data can influence policy makers. Good quality evaluation can help protect innovative services in an environment of scarce resources. The current research works to this end.

The Early Psychosis Outcome Evaluation System (EPOES) was developed by a working party of clinicians working in early-episode psychosis services, and a research psychologist (NP). An essential requirement of the system was to provide outcome evaluation that is relevant to and feeds directly back to the clinician and patient, the service and the system. The EPOES was developed upon the matrix model [8] where outcome data is fed back at all levels including the individual case, clinic and systems levels. It was designed to measure serially over time the effectiveness of early psychosis programmes, where clinicians can produce instant reports on the clinical status of patients in their management. EPOES is a Microsoft Access Database platform, which produces individual and service-wide data. It captures demographic and clinical outcome data, produces individual graphical case reports of patient progress on clinical outcome, and exports data for analysis. Codified forms are coupled with the electronic system making the evaluation of the system automatically established and part of the clinical work practices of staff. EPOES aims are to serve each level of outcome measurement, by allowing the clinicians to be directly involved in outcome research. Often outcome measurement can be slow, esoteric and irrelevant to clinical practices. EPOES tries to overcome these deficiencies by inverting the process, where individual clinical data is considered first, then pooled data at the clinic level, then finally systems level data for research. It is argued that such a system can effectively contribute to the literature on early psychosis programme development by first meeting the needs of clinicians, then turning to the broader needs of the research community in the area of early psychosis.

Method and sample

The study is a naturalistic prospective study of a cohort of early-episode psychosis patients. It employs a repeated measures design that tracks outcomes over time. All patients referred to any one of the four participating specialist early-episode psychosis programmes in Perth, Western Australia, were invited to participate in the study. Ethics approval was granted from each participating programme and written informed consent obtained from subjects. The four current participating programmes are: Early Intervention Psychosis Program (EIPP) Alma Street Centre, Fremantle; Early Episode Psychosis – A System of Care (EPP), Rockingham Kwinana Mental Health Service; First Psychosis Liaison Unit (FPLU), Mill Street Centre, Bentley; and First Episode Psychosis Program (FEPP), Joondalup Community Mental Health Services, Joondalup.

Entry criteria included residing within the catchment areas of the programmes and the person presenting with a first psychotic illness (or within 12 months of onset). Variation in age ranges of the programmes exist: Fremantle aged between 17 and 40; Rockingham-Kwinana, aged 18–40; Bentley aged 16–35; and Joondalup, aged 16–30 years.

The average participation rate across the four services was 35.6%. Since the ethics approval did not allow the research to perform case note reviews of non participants, the study is not able to determine diagnostic and demographic differences between the participating and non participating populations.

Procedure

Data collection is coordinated and entered into the EPOES database by the case manager of the patient. All case managers received 1 day intensive training on how to conduct a Brief Psychiatric Rating Scale (BPRS) interview and use, interpret and administer the other instruments. To be eligible to use EPOES, each case manager had to receive adequate interrater agreement across two case scenarios during the training. Measures were collated by the case manager from three sources: case manager, patient and family member of the patient. The initial assessment is conducted within 4–6 weeks of acceptance into an early psychosis programme. Subjects were approached to participate in the study after sufficient rapport was gained by the case manager. The assessment procedure was repeated every 6 months and also at discharge from the service.

Measures

The measures used in EPOES are gathered from three perspectives. The first perspective is from case manager assessment of patients service use, psychiatric functioning and substance use; the second perspective is from the patients self-reported experience of psychopathology and social functioning; and the third perspective asks family members to report their perceived level of family burden and general health. Each of the perspectives are linked in EPOES through a unique identifier. This allows for cross-referencing of data between each of the outcomes measured by the different informants.

Case manager assessment

1 Demographics: gender, age, onset of psychosis are collected during the case manager assessments. Age of onset is determined when positive symptoms become evident (not including prodromal signs and symptoms). This is estimated using information from the patient, family or significant others and any other collateral history available.

2 ICD-10 Diagnosis was made by the consultant psychiatrist on the treating community team via clinical interview, observation and collateral information

3 Current medication.

4 Brief Psychiatric Rating Scale [9].

5 Global Assessment Scale (GAS) [10].

6 Recent substance use history [Kavanagh D: personal communication] asks patients about their use of substances (e.g. coffee, cigarettes, amphetamines, cannabis); frequency, type and quantity over the previous 3 months

Patient self assessment

1 Demographics: gender, age, service provider.

2 The Social Functioning Scale (SFS) [11] assesses social functioning relevant to the needs and impairment of individuals with schizophrenia and related disorders. It is a 62-item scale consisting of six subscales. Withdrawal, Interpersonal Communication, Recreation Pro-social Activities, Independence Competence and Independent Performance.

3 The Brief Symptom Inventory (BSI) [12] a 53 item scale measuring psychological functioning; scores from the Positive Symptom Total (PST), Global Severity Index (GSI) and four of the nine symptom dimensions: Depression, Anxiety, Paranoia and Psychoticism are used in the study.

Family/carer assessment

1 Demographics: gender, age, relation to patient, living status.

2 The Burden Assessment Scale (BAS) [13], a 19-item scale measuring the level of burden experienced from caring for someone with a mental illness. It produces five subscales: Disruptive Activities, Personal Distress, Time Perspective, Guilt and Social Functioning

3 The General Health Questionnaire-12 (GHQ-12) [14] assesses the overall general wellbeing of care-givers, it produces a total score and a positive response score, where four or more positively scored items reflect caseness of psychological distress [15].

Analysis

Demographic descriptors of the sample such as age, gender, diagnosis, medication and substance use were collated. χ^2 tests of significance were used to assess differences in categorical measures such as gender and diagnosis. Analysis of variance (ANOVA) and paired-wise t-tests were used to analyse continuous data. Bonferoni adjustments for experiment-wise error were made for subscale analysis for each outcome measure. Pearson's product moment correlation coefficients were used to examine the relationships between continuous variables. To examine diagnostic differences, patient diagnoses were grouped into three categories: (1) Schizophrenia and schzophreniform diagnosis; (2) Substance Induced Psychosis (SIP only); and (3) Other (all other diagnoses). Schizophreniform subjects were placed with schizophrenia subjects from recent evidence suggesting their longitudinal diagnosis to be schizophrenia [16]

Duration of untreated psychosis represents positive psychotic features displayed before seeking formalized treatment. This period is estimated in months by the patients and significant others.

A poly drug-use score was calculated by the addition of a positive response to use of cannabis, amphetamines, sedatives, hallucinogenics, or opiates. Use of tea/coffee, alcohol or cigarettes was excluded from the analysis to focus on illicit substances. To examine the relationship between poly drug use and psychopathology, the poly drug use score was correlated with the total and subscale scores of the BPRS and BSI

Results

Patient characteristics

The baseline sample consisted of 66 males with a mean age of 25.6 years (SD = 5.47) and 18 females with a mean age of 26.22 years (SD = 6.82). There was no statistically significant age difference between males and females ($F = 0.157$, df = 1,82, p = 0.69)

Diagnosis at baseline

Table 1 presents the distribution of diagnoses of patients at baseline. The largest category (one-third), are substance induced psychotic (SIP) disorder followed by schizophreniform disorder

Using diagnostic groups of schizophrenia (n = 26), substance induced psychosis (n = 26) and all other diagnoses (n = 31), χ^2 analysis revealed gender did not contribute to differences in diagnosis ($\chi^2 = 2.30$, df = 2, p = 0.316). The frequencies in each diagnostic group were proportionally equivalent in males and females. Age did not differ between diagnostic categories of schizophrenia (mean = 26.1, SD = 7.0, n = 26), substance induced psychosis (mean = 24.4, SD = 4.7, n = 26) and all other diagnoses (mean = 26.7, SD = 5.6, n = 31) ($F = 1.24$, df = 2, 80, p = 0.29).

Duration of untreated psychosis (DUP)

The average DUP was 7.1 months. There was no statistically significant differences in DUP between males (DUP = 7.30 months, SD = 11.63, n = 62) and females (DUP = 6.52 months, SD = 11.50, n = 17) ($F = 0.060$, df = 1, 77, p = 0.80); (one male case was excluded as an outlier). Diagnoses when grouped into schizophrenia (DUP mean

= 11 6 months, SD = 17.4, n = 25), substance induced psychosis (DUP mean = 4 3 months, SD = 5.1, n = 25) and all other diagnoses (DUP mean = 5.7 months, SD = 7 8, n = 29) were found not to differ on DUP (F = 2.96, df = 2,76, p = 0.058). Observation of the means however, indicates that schizophrenia subjects had twice the length of DUP

Medication

All patients were prescribed medication at baseline The most common medications prescribed were Olanzapine (46, 31.5%), Risperidone (34, 23 3%), and Sodium Valproate (11, 7.5%). Olanzapine and Risperidone accounted for 54 8% of medications. All typical antipsychotic medication accounted for 4 1% of medication prescribed Average daily Chlorpromazine equivalent dose at intake was 362.17 mg with a range from 100 to 1200 mg. Of the two most frequently prescribed atypical antipsychotics, Olanzapine recorded higher levels (mean = 444.4 mg, SD = 193 80) of Chlorpromazine equivalent dose than Respiridone (mean = 278.78 mg, SD = 208.80) (t = 3.60, df = 1,76, p = 0.001)

Differences in psychopathology between gender and diagnosis

There were no statistically significant gender differences in psychopathology as measured by the BPRS total score and subscales: Thinking Disorder, Withdrawal, Anxiety Depression and Activation However, males recorded significantly higher levels of Hostility and Suspicion than females (Male mean = 2 4, SD = 1.1, n = 66; Female mean = 1.6, SD = 0.8, n = 18) (F = 9.76, df = 1,82, p = 0 002). There were no statistically significant gender differences on BSI subscale scores.

Patients with a diagnosis of SIP recorded significantly higher levels of BPRS Activation (mean = 1.83, SD = 0 9, n = 26) than those with schizophrenia (mean = 1 09, SD = 0.2, n = 11) or other diagnoses (mean = 1.58, SD = 0.7, n = 46) (F = 4.17, df = 2,80, p = 0 02). Post hoc analysis revealed a significant difference between SIP and schizophrenia (Tukey HSD mean difference = 0 74, p = 0.02) but not with other diagnoses. Substance induced psychosis also recorded higher levels of Thought Disorder (mean = 2 76, SD = 1.1 n = 26) compared with schizophrenia (mean = 1.98, SD = 1 0, n = 11) or other diagnoses (mean = 2.20, SD = 1 1, n = 46) though it narrowly missed statistical significance (F = 3.01, df = 2, 80, p = 0.055) The subscales of the BSI revealed no statistically significant differences between the diagnostic groups.

Patient social functioning

Examination of gender differences in patient social functioning revealed that males recorded more withdrawal than females (increase denotes improvement) (Male mean = 9.54, SD = 2 4, n = 59; Female mean = 11 2, SD = 2 9, n = 17) (F = 5.54, df = 1,74, p = 0.02), and lower levels of independently performed activities (Male mean = 26 5, SD = 8 5, n = 59, Female mean = 31.9, SD = 4.8, n = 17) (F = 6.43, df = 1, 74, p = 0.013). Gender did not significantly differ on any other social functioning scale subscales or the total score.

Diagnosis did not contribute to differences in social functioning, except with schizophrenia patients showing poorer scores on social withdrawal than the other psychosis group (F = 3.17, df = 2,75, p = 0.047).

Substance use

Sixty per cent of patients (n = 47) reported using cannabis within 3 months of their baseline assessment Thirty per cent (n = 24) reported using amphetamines/ecstasy/cocaine within 3 months of their baseline assessment. Almost 26% of patients (n = 20) reported sedative use 3 months prior to baseline assessment

Table 2 indicates cannabis use was associated with higher levels of psychopathology on BPRS subscales notably thought disorder (Thinking Disorder), paranoid ideation (Hostility and Suspicion) anxiety/depression and agitation (Activation)

There was no significant difference between gender and cannabis use (χ^2 = 0.001, df = 1, p = 0.982) but cannabis users tended to be younger (mean = 24 6, SD = 5 2) than non-users (mean = 27 3, SD = 6 2) (F = 3 92, df = 1 76, p = 0 051)

Amphetamine/ecstasy/cocaine use was significantly associated with increased levels of Anxiety/Depression (User mean = 3.2, SD = 0.93, n = 23, Non user mean = 2.5, SD = 1 1, n = 54) (F = 6.92, df = 1,76, p = 0.01) and Hostility/Suspicion (User mean = 2.8, SD = 1.1, n = 23; Non user mean = 2.1, SD = 1.1, n = 54) (F = 7 89, df = 1,76,

Table 1 Diagnosis of patient at baseline

Diagnosis	Frequency	Percentage
Schizophreniform disorder	15	17.9
Schizophrenia	11	13.1
Schizoaffective disorder	2	2.4
Delusional disorder	3	3.5
Substance induced psychotic disorder	26	31 0
Major depressive disorder with psychotic features	3	3.6
Bipolar affective disorder with psychotic features	5	6.0
Psychotic disorder not otherwise specified	15	17.8
Other	4	4.7
Total	84	100.0

Table 2. One way ANOVAS on Brief Psychiatric Rating Scale (BPRS) total score and subscales with marijuana use and non-use at baseline

Dependent variable	n	Cannabis	Mean	SD	F	p
BPRS total Score	31	No	33.29	11.21	17.52	0.001
	47	Yes	43.96	10.89		
Thinking disorder	31	No	1.911	1.018	9.84	0.002
	47	Yes	2.654	1.028		
Withdrawal	31	No	1.847	1.014	0.672	0.415
	47	Yes	2.021	0.853		
Anxiety depression	31	No	2.298	0.999	6.943	0.010
	47	Yes	2.926	1.048		
Hostility suspicion	31	No	1.753	0.793	15.38	0.001
	47	Yes	2.674	1.137		
Activation	31	No	1.269	0.389	12.34	0.001
	47	Yes	1.844	0.854		

p = 0.006). The BPRS total score also significantly differed between amphetamine users and non-users (User mean = 45.2, SD = 9.2, n = 23; Non-user mean = 37.7, SD = 12.5, n = 54) (F = 6.642, df = 1,76, p = 0.012). Other subscales did not differ significantly between users and-non-users. There was no significant age difference between amphetamine users (mean = 24.6, SD = 4.6, n = 23) and non-users (mean = 25.9, SD = 6.2, n = 54) (F = 0.49, df = 1,75, p = 0.49)

The poly drug-use score showed mild positive correlations with the total and subscale scores of the BPRS and BSI (Table 3).

Family member characteristics

Data from 66 family members had been collected at baseline. The majority of family members were mothers (n = 33, 50%). Fathers were the next largest group (n = 17, 25.8%) followed by spouse (n = 9, 13.6%) and sister (n = 3, 4.5%). At 6 months, data were available from 21 family members; 16 (76%) were mothers. The majority of respondents lived with their relative at the baseline assessment (n = 55, 83.3%). At 6 months, 18 of the 21 lived with their relative (86%).

General Health Questionnaire-12

Psychological distress at baseline was reported by 59% (n = 39) of family members. There were no statistically significant differences on GHQ-12 positive scores between mothers (positive score = 4.9, SD = 3.4) and fathers (positive score = 6.6, SD = 4.5) at baseline (GHQ-12 positive score F = 2.48, df = 1,48, p = 0.12). The sample was also grouped into parents and other family members. The GHQ-12 positive score differentiated parents (mean = 5.4, SD = 3.8, n = 50) from other family members (mean = 3.3, SD = 3.4, n = 16) with parents reporting significantly more psychological distress than other family members (F = 4.05, df = 1,64, p = 0.048).

Burden Assessment Scale

The baseline family member sample was grouped according to mothers and fathers, then parents and other carers. Comparison of the BAS total score and 5 subscales (Disruption, Personal Distress, Time Perspective, Guilt and Social Functioning) revealed no statistically significant differences in reported family burden between these groups. Equivalent levels of family burden are reported across all carers.

Outcome at 6 months of treatment

Twenty-three patients had been successfully followed up at 6 months. The following analyses are based on these cases using paired-wise t-tests to assess within subject effects over time. The BPRS total score and subscale scores statistically significantly reduced (excluding withdrawal after Bonferroni adjustment) at 6 months compared to baseline (Table 4). Patients also reported improvement in psychiatric functioning with all subscales of the BSI showing a reduction from baseline to 6 months (excluding depression after Bonferoni adjustment) (Table 4).

The GAS significantly increased from baseline to 6 months (Table 4) indicating an overall functional improvement in patient outcome. Analysis of subscale scores of social functioning from baseline to 6 months revealed no changes after Bonferoni adjustment. The results indicate no improvement in social functioning within 6 months.

Overall family burden did not significantly reduce from baseline to 6 months (Table 4), with the exception of the subscale Time Perspective that measures concern about the future wellbeing of their relative. Family members reported significantly less concern for their relative's future.

The positive response score of the GHQ-12 significantly reduced from baseline to 6 months (Table 4), indicating family members returned within normal population ranges of psychological wellbeing 6 months after treatment for their family member. At 6 months only 15% of family members reported psychological distress compared to 55% at intake.

Discussion

Early Psychosis Outcome Evaluation System is an effective system for the collation of descriptive clinical

Table 3. Correlations of poly drug use with total and subscale scores on the Brief Psychiatric Rating Scale (BPRS) and Brief Symptom Inventory (BSI) at baseline (n = 77)

	BPRS total	Thinking disorder	Withdrawal	Anxiety depression	Hos Sus	Activation	GSI BSI	Depression	Anxiety	Paranoia	Psychoticism	PST
Poly drug use	0 357**	0 259*	– 0.015	0.330*	0.336*	0.299**	0.362**	0.320**	0.419**	0.338**	0 357**	0.386**

*Correlation is significant at the 0 05 level (2 tailed).
**Correlation is significant at the 0 01 level (2 tailed).
Hos Sus, Hostility Suspicion GSI Global Severity Index; PST, Positive Symptom Total.

outcome data. The system is innovative in that clinicians participating in EPOES now conduct research and evaluation as part of their everyday work duties. Acceptability of EPOES centres around the functional capacity of the system to provide immediate feedback to clinicians on the status of patient's clinical outcome. The success of EPOES lies in the fact that the system was generated by clinicians specializing in early psychosis who were interested in research and outcome evaluation rather than a research group interested in clinician outcome in service delivery. Ownership of EPOES occurs due to the immediate and direct involvement of clinicians to generate the design, implementation and analysis of data that is important in the day-to-day delivery of their clinical services.

The system works on the matrix model [8], where information is fed back at the individual clinical, service and statewide systems levels. In the case of EPOES the system is inverted where the priority lies in providing clinical outcome information to clinicians first, then to the service then finally to the overall system of care. Unlike other clinical outcome evaluation systems implemented in psychiatric services, EPOES was initiated designed and implemented by clinicians. High participation rates among the service sites over the past 4 years is mainly attributed to the system focusing primarily on providing clinical information relevant to the treating clinician. Outcome reports were first constructed at the individual case level. It was anticipated that if the data collected were relevant at this level then systems analysis (i.e. evaluating a service model) would be easier and more realistic. With the focus of the 1998 Second National Mental Health Plan [2] on outcome assessment, EPOES has proven a precursor to how successful outcome evaluation systems can survive beyond their initial implementation. This was done by first considering informatics central to clinical service delivery as opposed to meeting the needs of researchers, or service managers and administrators. This strategy has proven successful with clinicians actively using EPOES as part of their clinical service delivery and not some abstract data collection system used for someone else's benefit or research interests.

Preliminary results identify a significant reduction in psychopathology, both observed and self-reported at 6 months from baseline. Global Assessment Scale scores also significantly improved over 6 months of treatment. Gains were not observed in the depression subscale of the BSI and the withdrawal subscale in the BPRS. This is indicative of the difficulty in treating the negative spectrum of psychotic disorders. Family member general health also significantly improved, although family burden remained unchanged in the strictest statistical

Table 4. Paired t-tests on the BPRS, GAS, BSI, SFS, BAS and GHQ-12 total scores and subscales at baseline and six months

Dependent variables	Baseline mean	6 months mean	Paired diff. mean	t	df	p	95%CI
Brief Psychiatric Rating Scale (BPRS)							
BPRS Total Score	41.57	27.30	14.26	5.05	22	0 000	8.40–20.10
Thinking Disorder	2.70	1.45	1.25	5.23	22	0.000	0.75–1.75
Withdrawal	1.92	1.49	0.43	2.45	22	0.023*	0.07–0.80
Anxiety Depression	2.87	2.00	0.87	2.97	22	0.007	0.26–1.50
Hostility Suspicion	2.17	1.33	0.84	4.21	22	0.000	0.43–1.30
Activation	1.70	1.19	0.51	3.93	22	0.001	0.24–0.78
Global Assessment Scale (GAS) Score	50.4	70.3	– 19.9	– 5.5	21	0.001	– 27.40–12.40
Brief Symptom Inventory (BSI)							
Global Severity Index	1.471	0.863	0.61	4.382	22	0.000	0.32–0.90
Depression	1.587	0.985	0.60	2.629	22	0 015*	0.13–1.08
Anxiety	1.673	0.876	0 80	3.639	22	0.001	0.34–1.25
Paranoia	1.478	0.865	0.58	3.023	22	0.006	0.18–0.98
Positive Symptom Total	35.26	27 52	7.74	1.996	22	0.058	– 0.30–15.78
Social Functioning Scale (SFS) Total	113 30	121.74	– 8.43	– 1.27	22	0.219	– 22 30–5.38
Burden Assessment Scale (BAS)							
BAS Total	40	33.3	6.7	2.09	19	0.051	– 0.02–13.40
GHQ-12 Positive Score	4.35	1.75	2.6	2.65	19	0.020	0.54–4.70

*p-value not significant after Bonferoni adjustment for experiment-wise error.

sense (see Table 4). In terms of patient social functioning, prolonged role functioning difficulties are identified in that there was no social functioning improvement at 6 months. These results suggest social functioning deficits associated with psychotic disorders [17]. It is important however, to qualify these results. These results are on a small sample (n = 23) and hence are only preliminary. For example, family burden and family member general health were slightly under the statistical threshold, indicating the results to be at the lower bounds of power In addition to these concerns, the research has been unable to determine interrater reliability within clinicians and between service sites over the course of the study although this was determined during training It is the intention with further research that these issues of internal consistency of assessment between service sites are addressed

The frequent use of substances by early psychosis patients confirms previous research [18–22] Substance misuse has been associated with a more severe course of schizophrenia [18] earlier and more psychotic relapses in young cannabis-using patients diagnosed with a first schizophrenic episode [20] and increased rates of hospitalization [21] The need for services to address effectively both substance use and psychosis is evident.

Although EPOES is not a classical randomised control trial it can allow for such a design to occur by employing EPOES into regional areas where the minimum standards of operating an early psychosis intervention service are not present. We aim to address this limitation in a controlled experimental design by inviting non specialist service providers of early psychosis patients to participate in using EPOES Since EPOES uses a naturalist design in 'real life clinical settings it is difficult to get control sites to participate where one may expect that their service is inferior or inadequate. By providing control sites with a system that helps them immediately inform their clinical practice (not after the experiment is completed 2 years after implementation) then the evaluation system is likely to find willing candidates. By employing research designs that compare real life clinical practices as opposed to highly funded research projects, then generalizability of the results tends to be higher and more accurate. In addition to this, it is the intention of the researchers to examine treatment fidelity and its contribution to service utilization such as bed days and admission rates.

Acknowledgements

Thanks to Craig Headford for developing the EPOES programme. The Early Psychosis Outcome Evaluation System was funded by Quality Improvement Project: Mental Health Division Department of Health Western Australia.

Written on behalf of the Early Psychosis Group Western Australia.

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APPENDIX V

EQS output of Validation Sample measurement model

```
1
 EQS, A STRUCTURAL EQUATION PROGRAM          MULTIVARIATE SOFTWARE, INC.
 COPYRIGHT BY P.M. BENTLER                   VERSION 5.7b (C) 1985 - 1998.

  PROGRAM CONTROL INFORMATION

   1 /TITLE
   2  GROUP 1 = VICTORIAN SAMPLE N=244
   3  6 FACTOR VICTORIAN BASELINE MODEL
   4  DROPPING HOSTILITY F2
   5 /SPECIFICATIONS
   6  DATA='C:\EQS\PHDRES~1\PRELIM~1\MEASUR~1\VICBSI3.ESS';
   7  VARIABLES= 55; CASES=  244;
   8  METHODS=ML, ROBUST;
   9  MATRIX=RAW;
  10 /LABELS
  11  V1=GROUP; V2=URN; V3=BSI1; V4=BSI2; V5=BSI3;
  12  V6=BSI4; V7=BSI5; V8=BSI6; V9=BSI7; V10=BSI8;
  13  V11=BSI9; V12=BSI10; V13=BSI11; V14=BSI12; V15=BSI13;
  14  V16=BSI14; V17=BSI15; V18=BSI16; V19=BSI17; V20=BSI18;
  15  V21=BSI19; V22=BSI20; V23=BSI21; V24=BSI22; V25=BSI23;
  16  V26=BSI24; V27=BSI25; V28=BSI26; V29=BSI27; V30=BSI28;
  17  V31=BSI29; V32=BSI30; V33=BSI31; V34=BSI32; V35=BSI33;
  18  V36=BSI34; V37=BSI35; V38=BSI36; V39=BSI37; V40=BSI38;
  19  V41=BSI39; V42=BSI40; V43=BSI41; V44=BSI42; V45=BSI43;
  20  V46=BSI44; V47=BSI45; V48=BSI46; V49=BSI47; V50=BSI48;
  21  V51=BSI49; V52=BSI50; V53=BSI51; V54=BSI52; V55=BSI53;
  22  V51=BSI49; V52=BSI50; V53=BSI51; V54=BSI52; V55=BSI53;
  23  F1=DEP; F2=PHO; F3=SOM; F4=COG; F5=ANX; F6=PAR;
  24 /EQUATIONS
  25  V3 =  + *F5  + 1E3;
  26  V7 =  + *F4  + 1E7;
  27  V10 =  + *F2  + 1E10;
  28  V12 =  + *F6  + 1E12;
  29  V19 =  + *F1  + 1E19;
  30  V21 =  + *F5  + 1E21;
  31  V23 =  + *F6  + 1E23;
  32  V29 =  + *F4  + 1E29;
  33  V30 =  + *F2  + 1E30;
  34  V31 =  + *F3  + 1E31;
  35  V32 =  + *F3  + 1E32;
  36  V34 =  + *F4  + 1E34;
  37  V35 =  + *F3  + 1E35;
  38  V37 =  + *F1  + 1E37;
  39  V38 =  + *F4  + 1E38;
  40  V45 =  + *F2  + 1E45;
  41  V47 =  + *F5  + 1E47;
  42  V52 =  + *F1  + 1E52;
  43  V53 =  + *F6  + 1E53;
  44 /VARIANCES
  45  F1 = 1;
  46  F2 = 1;
  47  F3 = 1;
  48  F4 = 1;
  49  F5 = 1;
  50  F6 = 1;
  51  E3 = *;
  52  E7 = *;
```

TITLE: GROUP 1 = VICTORIAN SAMPLE N=244 07/06/01 PAGE : 2
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```
 53  E10 = *;
 54  E12 = *;
 55  E19 = *;
 56  E21 = *;
 57  E23 = *;
 58  E29 = *;
 59  E30 = *;
 60  E31 = *;
 61  E32 = *;
 62  E34 = *;
 63  E35 = *;
 64  E37 = *;
 65  E38 = *;
 66  E45 = *;
 67  E47 = *;
 68  E52 = *;
 69  E53 = *;
 70  /COVARIANCES
 71  F2 , F1 = *;
 72  F3 , F1 = *;
 73  F4 , F1 = *;
 74  F5 , F1 = *;
 75  F6 , F1 = *;
 76  F3 , F2 = *;
 77  F4 , F2 = *;
 78  F5 , F2 = *;
 79  F6 , F2 = *;
 80  F4 , F3 = *;
 81  F5 , F3 = *;
 82  F6 , F3 = *;
 83  F5 , F4 = *;
 84  F6 , F4 = *;
 85  F6 , F5 = *;
 86  /LMTEST
 87    SET=PEE, GVF;
 88  /PRINT
 89   FIT = ALL;
 90  /END
```

90 RECORDS OF INPUT MODEL FILE WERE READ

DATA IS READ FROM C:\EQS\PHDRES~1\PRELIM~1\MEASUR~1\VICBSI3.ESS
THERE ARE 55 VARIABLES AND 244 CASES
IT IS A RAW DATA ESS FILE

TITLE: GROUP 1 = VICTORIAN SAMPLE N=244 07/06/01 PAGE : 3
EQS/EM386 Licensee: Neil Preston

SAMPLE STATISTICS BASED ON COMPLETE CASES

UNIVARIATE STATISTICS

VARIABLE	BSI1	BSI5	BSI8	BSI10	BSI17
MEAN	1.1516	1.2910	0.6230	0.9549	1.2705
SKEWNESS (G1)	0.7618	0.6932	1.6180	1.0780	0.7293
KURTOSIS (G2)	-0.4039	-0.6289	1.7032	-0.0808	-0.6089
STANDARD DEV.	1.1429	1.2674	1.0210	1.2481	1.2736

VARIABLE	BSI19	BSI21	BSI27	BSI28	BSI29
MEAN	0.8730	0.8279	1.1680	0.5492	0.5000
SKEWNESS (G1)	1.1950	1.2235	0.8397	1.9251	2.0927
KURTOSIS (G2)	0.3230	0.6133	-0.3459	2.9597	3.5794
STANDARD DEV.	1.1742	1.1125	1.2474	1.0029	0.9917

VARIABLE	BSI30	BSI32	BSI33	BSI35	BSI36
MEAN	0.4918	0.9180	0.6516	1.1516	1.2500
SKEWNESS (G1)	2.0440	1.2782	1.6500	0.9910	0.7535
KURTOSIS (G2)	3.5865	0.7001	1.7931	-0.2189	-0.4185
STANDARD DEV.	0.9362	1.1835	1.0723	1.3231	1.2201

VARIABLE	BSI43	BSI45	BSI50	BSI51
MEAN	1.0164	0.5697	0.9057	0.9262
SKEWNESS (G1)	1.1200	1.7419	1.1985	1.1772
KURTOSIS (G2)	0.0890	2.0125	0.2577	0.2456
STANDARD DEV.	1.2732	1.0058	1.2320	1.2250

MULTIVARIATE KURTOSIS

MARDIA'S COEFFICIENT (G2,P) = 183.9885
NORMALIZED ESTIMATE = 50.8691

ELLIPTICAL THEORY KURTOSIS ESTIMATES

MARDIA-BASED KAPPA = 0.4611 MEAN SCALED UNIVARIATE KURTOSIS = 0.2659

MARDIA-BASED KAPPA IS USED IN COMPUTATION. KAPPA= 0.4611

CASE NUMBERS WITH LARGEST CONTRIBUTION TO NORMALIZED MULTIVARIATE KURTOSIS:

CASE NUMBER	4	12	198	208	218
ESTIMATE	740.8705	1023.0433	781.2831	1748.4242	842.0760

TITLE: GROUP 1 = VICTORIAN SAMPLE N=244 07/06/01 PAGE : 4
EQS/EM386 Licensee: Neil Preston
COVARIANCE MATRIX TO BE ANALYZED: 19 VARIABLES (SELECTED FROM 55 VARIABLES)
BASED ON 244 CASES.

		BSI1 V 3	BSI5 V 7	BSI8 V 10	BSI10 V 12	BSI17 V 19
BSI1	V 3	1.306				
BSI5	V 7	0.581	1.606			
BSI8	V 10	0.411	0.406	1.042		
BSI10	V 12	0.423	0.503	0.464	1.558	
BSI17	V 19	0.663	0.584	0.460	0.498	1.622
BSI19	V 21	0.694	0.626	0.549	0.550	0.796
BSI21	V 23	0.430	0.663	0.462	0.745	0.487
BSI27	V 29	0.563	0.844	0.512	0.621	0.823
BSI28	V 30	0.336	0.391	0.574	0.317	0.394
BSI29	V 31	0.364	0.529	0.235	0.471	0.342
BSI30	V 32	0.407	0.346	0 297	0.372	0.484
BSI32	V 34	0.515	0.892	0 422	0.350	0.730
BSI33	V 35	0.432	0.493	0 263	0.408	0.379
BSI35	V 37	0.520	0.705	0 494	0.558	0.942
BSI36	V 38	0.427	0.882	0.420	0.398	0.685
BSI43	V 45	0.619	0.547	0 846	0.585	0.810
BSI45	V 47	0.601	0.438	0 516	0.400	0.574
BSI50	V 52	0.492	0.637	0 429	0.469	1.001
BSI51	V 53	0.674	0.684	0 495	0.820	0.637

		BSI19 V 21	BSI21 V 23	BSI27 V 29	BSI28 V 30	BSI29 V 31
BSI19	V 21	1.379				
BSI21	V 23	0.636	1.238			
BSI27	V 29	0.593	0.700	1.556		
BSI28	V 30	0.428	0.428	0.562	1.006	
BSI29	V 31	0.405	0.444	0.438	0.230	0.984
BSI30	V 32	0.408	0.406	0.386	0.235	0.416
BSI32	V 34	0.689	0.657	0.792	0.432	0.469
BSI33	V 35	0.466	0.368	0.421	0.295	0.508
BSI35	V 37	0.690	0.705	0.892	0.505	0.323
BSI36	V 38	0.629	0.586	0.941	0.422	0.414
BSI43	V 45	0.718	0.632	0.652	0.736	0.453
BSI45	V 47	0.739	0.485	0.554	0.509	0.368
BSI50	V 52	0.720	0.654	0.925	0.389	0.393
BSI51	V 53	0.789	0.740	0.687	0.403	0.481

		BSI30 V 32	BSI32 V 34	BSI33 V 35	BSI35 V 37	BSI36 V 38
BSI30	V 32	0.876				
BSI32	V 34	0.411	1.401			
BSI33	V 35	0.497	0.490	1.150		
BSI35	V 37	0.271	0.720	0.382	1.751	
BSI36	V 38	0.333	0.757	0.470	0.863	1.489
BSI43	V 45	0.391	0.623	0.438	0.742	0.658
BSI45	V 47	0.348	0.545	0.352	0.461	0.458
BSI50	V 52	0.380	0.778	0.362	1.101	0.777
BSI51	V 53	0.456	0.541	0.522	0.554	0.500

		BSI43 V 45	BSI45 V 47	BSI50 V 52	BSI51 V 53
BSI43	V 45	1.621			
BSI45	V 47	0.785	1.012		
BSI50	V 52	0.639	0.577	1.518	
BSI51	V 53	0.590	0.618	0.676	1.501

BENTLER-WEEKS STRUCTURAL REPRESENTATION:

NUMBER OF DEPENDENT VARIABLES = 19
DEPENDENT V'S : 3 7 10 12 19 21 23 29 30 31
DEPENDENT V'S : 32 34 35 37 38 45 47 52 53

NUMBER OF INDEPENDENT VARIABLES = 25
INDEPENDENT F'S : 1 2 3 4 5 6
INDEPENDENT E'S : 3 7 10 12 19 21 23 29 30 31
INDEPENDENT E'S : 32 34 35 37 38 45 47 52 53

NUMBER OF FREE PARAMETERS = 53
NUMBER OF FIXED NONZERO PARAMETERS = 25

3RD STAGE OF COMPUTATION REQUIRED 72906 WORDS OF MEMORY.
PROGRAM ALLOCATED 10000000 WORDS

DETERMINANT OF INPUT MATRIX IS 0.80586E-02

TITLE: GROUP 1 = VICTORIAN SAMPLE N=244 07/06/01 PAGE : 5
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CASE CONTRIBUTION TO PARAMETER VARIANCES (IN DESCENDING ORDER)

CASE 94=	0.052	3.38%	CASE 22=	0.045	2.94%
CASE 208=	0.041	2.70%	CASE 198=	0.040	2.59%
CASE 12=	0.037	2.39%	CASE 133=	0.035	2.32%
CASE 199=	0.031	2.00%	CASE 153=	0.030	1.98%
CASE 154=	0.028	1.83%	CASE 26=	0.027	1.74%
CASE 218=	0.026	1.71%	CASE 14=	0.026	1.70%
CASE 4=	0.024	1.59%	CASE 195=	0.024	1.56%
CASE 1=	0.023	1.52%	CASE 172=	0.023	1.48%
CASE 192=	0.022	1.45%	CASE 140=	0.020	1.32%
CASE 148=	0.020	1.31%	CASE 200=	0.019	1.22%
CASE 27=	0.018	1.17%	CASE 232=	0.018	1.17%
CASE 55=	0.018	1.17%	CASE 99=	0.018	1.15%
CASE 43=	0.016	1.07%	CASE 120=	0.016	1.06%
CASE 46=	0.016	1.05%	CASE 231=	0.016	1.05%
CASE 127=	0.016	1.02%	CASE 182=	0.015	0.98%
CASE 156=	0.015	0.98%	CASE 160=	0.015	0.96%
CASE 6=	0.015	0.95%	CASE 237=	0.014	0.93%
CASE 66=	0.014	0.93%	CASE 11=	0.014	0.92%
CASE 184=	0.014	0.92%	CASE 169=	0.014	0.89%
CASE 162=	0.013	0.87%	CASE 72=	0.013	0.85%
CASE 28=	0.013	0.85%	CASE 69=	0.013	0.84%
CASE 96=	0.013	0.82%	CASE 105=	0.013	0.82%
CASE 53=	0.012	0.76%	CASE 51=	0.011	0.75%
CASE 173=	0.011	0.74%	CASE 5=	0.011	0.74%
CASE 150=	0.011	0.73%	CASE 178=	0.011	0.72%
CASE 9=	0.011	0.72%	CASE 213=	0.011	0.69%
CASE 93=	0.010	0.68%	CASE 207=	0.010	0.65%
CASE 77=	0.009	0.61%	CASE 206=	0.009	0.60%
CASE 40=	0.009	0.58%	CASE 166=	0.009	0.57%
CASE 65=	0.009	0.56%	CASE 194=	0.008	0.55%
CASE 241=	0.008	0.55%	CASE 219=	0.008	0.54%
CASE 17=	0.008	0.53%	CASE 186=	0.008	0.53%
CASE 82=	0.008	0.52%	CASE 121=	0.008	0.52%
CASE 119=	0.008	0.50%	CASE 111=	0.008	0.49%
CASE 115=	0.008	0.49%	CASE 191=	0.007	0.46%
CASE 224=	0.007	0.46%	CASE 58=	0.007	0.45%
CASE 209=	0.007	0.45%	CASE 64=	0.007	0.45%
CASE 110=	0.006	0.41%	CASE 239=	0.006	0.40%
CASE 155=	0.006	0.40%	CASE 141=	0.006	0.39%
CASE 225=	0.006	0.39%	CASE 57=	0.006	0.38%
CASE 75=	0.006	0.37%	CASE 242=	0.006	0.36%
CASE 52=	0.005	0.36%	CASE 59=	0.005	0.35%
CASE 137=	0.005	0.35%	CASE 118=	0.005	0.34%
CASE 38=	0.005	0.33%	CASE 32=	0.005	0.32%
CASE 84=	0.005	0.31%	CASE 24=	0.005	0.30%
CASE 158=	0.004	0.28%	CASE 47=	0.004	0.27%
CASE 228=	0.004	0.26%	CASE 79=	0.004	0.26%
CASE 18=	0.004	0.25%	CASE 174=	0.004	0.25%
CASE 229=	0.004	0.25%	CASE 168=	0.004	0.25%
CASE 180=	0.004	0.24%	CASE 220=	0.004	0.24%
CASE 181=	0.004	0.24%	CASE 177=	0.003	0.23%
CASE 159=	0.003	0.22%	CASE 97=	0.003	0.22%
CASE 188=	0.003	0.21%	CASE 167=	0.003	0.21%
CASE 193=	0.003	0.20%	CASE 100=	0.003	0.20%
CASE 107=	0.003	0.19%	CASE 95=	0.003	0.19%
CASE 114=	0.003	0.19%	CASE 175=	0.003	0.19%
CASE 163=	0.003	0.18%	CASE 85=	0.003	0.18%
CASE 179=	0.003	0.18%	CASE 117=	0.003	0.17%
CASE 221=	0.003	0.17%	CASE 165=	0.003	0.17%
CASE 80=	0.003	0.17%	CASE 123=	0.003	0.16%

TITLE: GROUP 1 = VICTORIAN SAMPLE N=244 07/06/01 PAGE : 6
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MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

PARAMETER ESTIMATES APPEAR IN ORDER,
NO SPECIAL PROBLEMS WERE ENCOUNTERED DURING OPTIMIZATION.

RESIDUAL COVARIANCE MATRIX (S-SIGMA) :

		BSI1 V 3	BSI5 V 7	BSI8 V 10	BSI10 V 12	BSI17 V 19
BSI1	V 3	0.000				
BSI5	V 7	0.097	0.000			
BSI8	V 10	-0.053	-0.050	0.000		
BSI10	V 12	-0.060	-0.045	0.037	0.000	
BSI17	V 19	0.152	-0.128	-0.001	-0.031	0.000
BSI19	V 21	0.005	0.026	-0.026	-0.047	0.165
BSI21	V 23	-0.065	0.102	0.023	0.046	-0.055
BSI27	V 29	0.037	-0.045	0.017	0.026	0.051
BSI28	V 30	-0.076	-0.013	0.023	-0.062	-0.015
BSI29	V 31	0.015	0.106	-0.059	0.057	-0.004
BSI30	V 32	0.084	-0.044	0.026	-0.010	0.164
BSI32	V 34	0.045	0.098	-0.021	-0.181	0.041
BSI33	V 35	0.055	0.037	-0.053	-0.038	0.006
BSI35	V 37	-0.032	-0.065	-0.006	-0.014	-0.041
BSI36	V 38	-0.064	0.052	-0.043	-0.157	-0.035
BSI43	V 45	-0.014	-0.075	-0.002	0.002	0.182
BSI45	V 47	0.000	-0.083	0.016	-0.119	0.024
BSI50	V 52	-0.069	-0.145	-0.078	-0.112	0.002
BSI51	V 53	0.126	0.061	0.009	0.045	0.036

		BSI19 V 21	BSI21 V 23	BSI27 V 29	BSI28 V 30	BSI29 V 31
BSI19	V 21	0.000				
BSI21	V 23	0.025	0.000			
BSI27	V 29	-0.057	0.090	0.000		
BSI28	V 30	-0.081	0.040	0.123	0.000	
BSI29	V 31	-0.026	0.020	-0.020	-0.030	0.000
BSI30	V 32	0.010	0.014	-0.037	-0.005	-0.022
BSI32	V 34	0.108	0.112	-0.070	0.040	0.060
BSI33	V 35	0.001	-0.090	-0.073	0.015	-0.002
BSI35	V 37	0.008	0.118	0.057	0.063	-0.051
BSI36	V 38	0.022	0.017	0.041	0.012	-0.014
BSI43	V 45	-0.065	0.035	-0.023	-0.015	0.052
BSI45	V 47	-0.003	-0.047	-0.012	0.066	-0.008
BSI50	V 52	0.028	0.059	0.077	-0.060	0.013
BSI51	V 53	0.111	-0.054	0.012	-0.028	0.011

		BSI30 V 32	BSI32 V 34	BSI33 V 35	BSI35 V 37	BSI36 V 38
BSI30	V 32	0.000				
BSI32	V 34	0.033	0.000			
BSI33	V 35	0.026	0.049	0.000		
BSI35	V 37	-0.075	-0.026	-0.021	0.000	
BSI36	V 38	-0.062	-0.047	0.009	0.083	0.000
BSI43	V 45	0.021	0.020	0.006	0.062	0.028
BSI45	V 47	0.001	0.039	-0.054	-0.134	-0.071
BSI50	V 52	0.029	0.021	-0.047	0.020	-0.015
BSI51	V 53	0.022	-0.062	0.014	-0.096	-0.131

		BSI43 V 45	BSI45 V 47	BSI50 V 52	BSI51 V 53
BSI43	V 45	0.000			
BSI45	V 47	0.103	0.000		
BSI50	V 52	-0.051	-0.027	0.000	
BSI51	V 53	-0.072	0.028	0.016	0.000

AVERAGE ABSOLUTE COVARIANCE RESIDUALS = 0.0443
AVERAGE OFF-DIAGONAL ABSOLUTE COVARIANCE RESIDUALS = 0.0492

TITLE: GROUP 1 = VICTORIAN SAMPLE N=244 07/06/01 PAGE : 7
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MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

STANDARDIZED RESIDUAL MATRIX:

		BSI1 V 3	BSI5 V 7	BSI8 V 10	BSI10 V 12	BSI17 V 19
BSI1	V 3	0.000				
BSI5	V 7	0.067	0.000			
BSI8	V 10	-0.046	-0.039	0.000		
BSI10	V 12	-0.042	-0.029	0.029	0.000	
BSI17	V 19	0.105	-0.079	-0.001	-0.020	0.000
BSI19	V 21	0.004	0.018	-0.022	-0.032	0.110
BSI21	V 23	-0.051	0.072	0.020	0.033	-0.039
BSI27	V 29	0.026	-0.028	0.013	0.017	0.032
BSI28	V 30	-0.066	-0.010	0.022	-0.049	-0.011
BSI29	V 31	0.013	0.085	-0.058	0.046	-0.003
BSI30	V 32	0.079	-0.037	0.027	-0.009	0.138
BSI32	V 34	0.033	0.066	-0.017	-0.123	0.027
BSI33	V 35	0.045	0.028	-0.049	-0.029	0.004
BSI35	V 37	-0.021	-0.039	-0.004	-0.009	-0.024
BSI36	V 38	-0.046	0.034	-0.034	-0.103	-0.023
BSI43	V 45	-0.010	-0.047	-0.002	0.002	0.112
BSI45	V 47	0.000	-0.065	0.015	-0.095	0.019
BSI50	V 52	-0.049	-0.093	-0.062	-0.073	0.002
BSI51	V 53	0.090	0.039	0.007	0.029	0.023

		BSI19 V 21	BSI21 V 23	BSI27 V 29	BSI28 V 30	BSI29 V 31
BSI19	V 21	0.000				
BSI21	V 23	0.019	0.000			
BSI27	V 29	-0.039	0.065	0.000		
BSI28	V 30	-0.069	0.036	0.098	0.000	
BSI29	V 31	-0.023	0.018	-0.016	-0.030	0.000
BSI30	V 32	0.009	0.013	-0.032	-0.006	-0.023
BSI32	V 34	0.078	0.085	-0.047	0.034	0.051
BSI33	V 35	0.001	-0.075	-0.055	0.014	-0.002
BSI35	V 37	0.005	0.080	0.034	0.047	-0.039
BSI36	V 38	0.015	0.013	0.027	0.010	-0.012
BSI43	V 45	-0.043	0.025	-0.015	-0.012	0.042
BSI45	V 47	-0.002	-0.042	-0.010	0.065	-0.008
BSI50	V 52	0.019	0.043	0.050	-0.048	0.011
BSI51	V 53	0.077	-0.040	0.008	-0.022	0.009

		BSI30 V 32	BSI32 V 34	BSI33 V 35	BSI35 V 37	BSI36 V 38
BSI30	V 32	0.000				
BSI32	V 34	0.029	0.000			
BSI33	V 35	0.026	0.038	0.000		
BSI35	V 37	-0.060	-0.017	-0.015	0.000	
BSI36	V 38	-0.054	-0.033	0.007	0.052	0.000
BSI43	V 45	0.018	0.013	0.005	0.037	0.018
BSI45	V 47	0.001	0.033	-0.050	-0.101	-0.058
BSI50	V 52	0.025	0.014	-0.036	0.012	-0.010
BSI51	V 53	0.019	-0.043	0.011	-0.059	-0.088

		BSI43 V 45	BSI45 V 47	BSI50 V 52	BSI51 V 53
BSI43	V 45	0.000			

BSI45	V 47	0.080	0.000		
BSI50	V 52	-0.033	-0.022	0.000	
BSI51	V 53	-0.046	0.023	0.010	0.000

AVERAGE ABSOLUTE STANDARDIZED RESIDUALS = 0.0328
AVERAGE OFF-DIAGONAL ABSOLUTE STANDARDIZED RESIDUALS = 0.0364

LARGEST STANDARDIZED RESIDUALS:

V 32,V 19	V 34,V 12	V 45,V 19	V 21,V 19	V 19,V 3
0.138	-0.123	0.112	0.110	0.105

V 38,V 12	V 47,V 37	V 30,V 29	V 47,V 12	V 52,V 7
-0.103	-0.101	0.098	-0.095	-0.093

V 53,V 3	V 53,V 38	V 34,V 23	V 31,V 7	V 47,V 45
0.090	-0.088	0.085	0.085	0.080

V 37,V 23	V 19,V 7	V 32,V 3	V 34,V 21	V 53,V 21
0.080	-0.079	0.079	0.078	0.077

TITLE: GROUP 1 = VICTORIAN SAMPLE N=244 07/06/01 PAGE : 8
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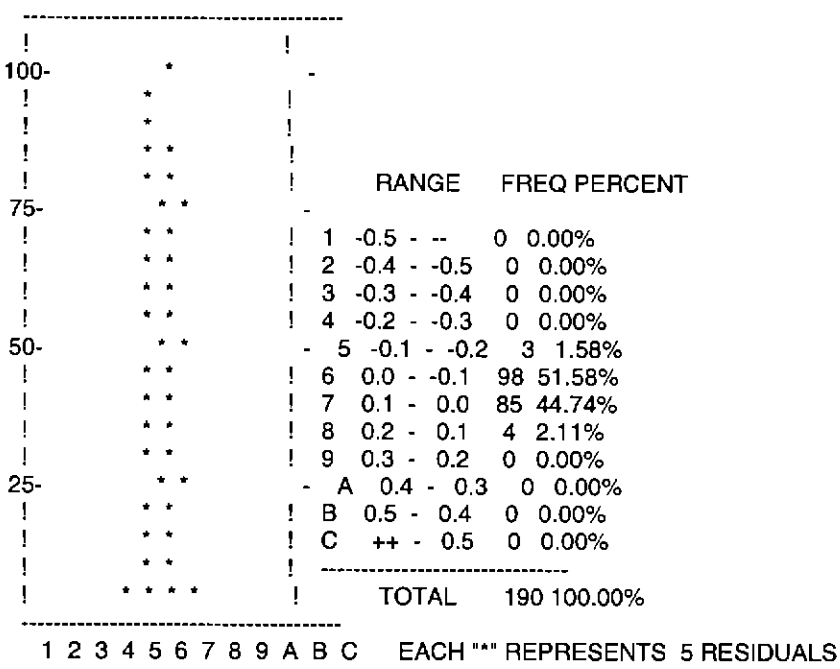
MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

DISTRIBUTION OF STANDARDIZED RESIDUALS



	RANGE	FREQ	PERCENT
1	-0.5 - --	0	0.00%
2	-0.4 - -0.5	0	0.00%
3	-0.3 - -0.4	0	0.00%
4	-0.2 - -0.3	0	0.00%
5	-0.1 - -0.2	3	1.58%
6	0.0 - -0.1	98	51.58%
7	0.1 - 0.0	85	44.74%
8	0.2 - 0.1	4	2.11%
9	0.3 - 0.2	0	0.00%
A	0.4 - 0.3	0	0.00%
B	0.5 - 0.4	0	0.00%
C	++ - 0.5	0	0.00%
	TOTAL	190	100.00%

TITLE: GROUP 1 = VICTORIAN SAMPLE N=244 07/06/01 PAGE : 9
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MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

GOODNESS OF FIT SUMMARY

INDEPENDENCE MODEL CHI-SQUARE = 2458.339 ON 171 DEGREES OF FREEDOM

INDEPENDENCE AIC = 2116.33934 INDEPENDENCE CAIC = 1347.32357
MODEL AIC = 1.03286 MODEL CAIC = -615.07919

CHI-SQUARE = 275.033 BASED ON 137 DEGREES OF FREEDOM
PROBABILITY VALUE FOR THE CHI-SQUARE STATISTIC IS LESS THAN 0.001
THE NORMAL THEORY RLS CHI-SQUARE FOR THIS ML SOLUTION IS 258.358.

SATORRA-BENTLER SCALED CHI-SQUARE = 181.2225
PROBABILITY VALUE FOR THE CHI-SQUARE STATISTIC IS 0.00680

BENTLER-BONETT NORMED	FIT INDEX=	0.888
BENTLER-BONETT NONNORMED	FIT INDEX=	0.925
COMPARATIVE FIT INDEX (CFI)	=	0.940
ROBUST COMPARATIVE	FIT INDEX=	0.970
BOLLEN (IFI)	FIT INDEX=	0.941
McDonald (MFI)	FIT INDEX=	0.754
LISREL GFI	FIT INDEX=	0.899
LISREL AGFI	FIT INDEX=	0.860
ROOT MEAN SQUARED RESIDUAL (RMR)	=	0.060
STANDARDIZED RMR	=	0.044
ROOT MEAN SQ. ERROR OF APP.(RMSEA)	=	0.065
90% CONFIDENCE INTERVAL OF RMSEA (	0.053,	0.075)

ITERATIVE SUMMARY

ITERATION	PARAMETER ABS CHANGE	ALPHA	FUNCTION
1	0.460850	1.00000	1.49931
2	0.080113	1.00000	1.14018
3	0.010316	1.00000	1.13228
4	0.002338	1.00000	1.13186
5	0.000618	1.00000	1.13182

TITLE: GROUP 1 = VICTORIAN SAMPLE N=244 07/06/01 PAGE : 10
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MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

MEASUREMENT EQUATIONS WITH STANDARD ERRORS AND TEST STATISTICS
(ROBUST STATISTICS IN PARENTHESES)

BSI1 =V3 = .747*F5 + 1.000 E3
.069
10.781
(.065)
(11.424)

BSI5 =V7 = .905*F4 + 1.000 E7
.074
12.229
(.067)
(13.414)

BSI8 =V10 = .789*F2 + 1.000 E10
.059
13.367
(.073)
(10.833)

BSI10 =V12 = .826*F6 + 1.000 E12
.077
10.783
(.074)
(11.191)

BSI17 =V19 = .953*F1 + 1.000 E19
.073
12.994
(.068)
(14.012)

BSI19 =V21 = .923*F5 + 1.000 E21
.067
13.806
(.072)
(12.896)

BSI21 =V23 = .847*F6 + 1.000 E23
.065
12.932
(.063)
(13.383)

BSI27 =V29 = .982*F4 + 1.000 E29
.070
14.009
(.065)
(15.185)

BSI28 =V30 = .699*F2 + 1.000 E30
.060
11.642
(.077)
(9.029)

BSI29 =V31 = .688*F3 + 1.000 E31
.063

10.932
(.081)
(8.488)

BSI30 =V32 = .636*F3 + 1.000 E32
.060
10.646
(.088)
(7.252)

BSI32 =V34 = .877*F4 + 1.000 E34
.068
12.871
(.078)
(11.257)

MEASUREMENT EQUATIONS WITH STANDARD ERRORS AND TEST STATISTICS (CONTINUED)

TITLE: GROUP 1 = VICTORIAN SAMPLE N=244 07/06/01 PAGE : 11
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MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)
(ROBUST STATISTICS IN PARENTHESES)

BSI33 =V35 = .742*F3 + 1.000 E35
.068
10.889
(.089)
(8.353)

BSI35 =V37 = 1.032*F1 + 1.000 E37
.075
13.761
(.075)
(13.802)

BSI36 =V38 = .917*F4 + 1.000 E38
.070
13.114
(.073)
(12.480)

BSI43 =V45 = 1.075*F2 + 1.000 E45
.071
15.088
(.077)
(13.926)

BSI45 =V47 = .804*F5 + 1.000 E47
.057
14.127
(.071)
(11.372)

BSI50 =V52 = 1.047*F1 + 1.000 E52
.067
15.575
(.074)
(14.124)

BSI51 =V53 = .938*F6 + 1.000 E53
.072
13.047
(.075)
(12.457)

TITLE: GROUP 1 = VICTORIAN SAMPLE N=244 07/06/01 PAGE : 12
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MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

VARIANCES OF INDEPENDENT VARIABLES

V	F	
	F1 - DEP	1.000
	F2 - PHO	1.000
	F3 - SOM	1.000
	F4 - COG	1.000
	F5 - ANX	1.000
	F6 - PAR	1.000

TITLE: GROUP 1 = VICTORIAN SAMPLE N=244 07/06/01 PAGE : 13
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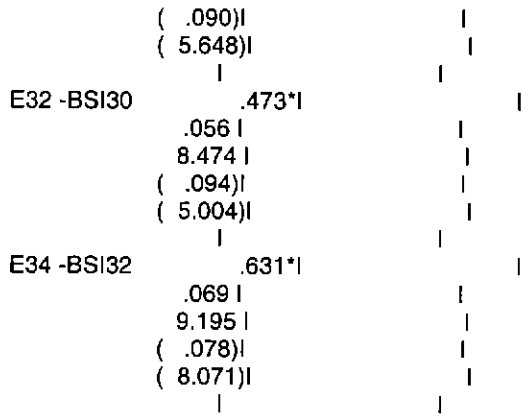
MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

VARIANCES OF INDEPENDENT VARIABLES

	E	D
E3 - BSI1	.749*	
	.077	
	9.765	
	(.088)	
	(8.534)	
E7 - BSI5	.787*	
	.083	
	9.470	
	(.093)	
	(8.451)	
E10 - BSI8	.420*	
	.051	
	8.184	
	(.067)	
	(6.247)	
E12 -BSI10	.875*	
	.093	
	9.406	
	(.119)	
	(7.358)	
E19 -BSI17	.713*	
	.079	
	9.000	
	(.104)	
	(6.859)	
E21 -BSI19	.527*	
	.064	
	8.179	
	(.101)	
	(5.198)	
E23 -BSI21	.521*	
	.064	
	8.093	
	(.093)	
	(5.593)	
E29 -BSI27	.592*	
	.069	
	8.561	
	(.082)	
	(7.182)	
E30 -BSI28	.518*	
	056	
	9 268	
	(098)	
	(5 287)	
E31 -BSI29	.510*	
	.062	
	8.244	


(.090)
(5.648)
E32 -BSI30
.473*
.056
8.474
(.094)
(5.004)
E34 -BSI32
.631*
.069
9.195
(.078)
(8.071)

TITLE: GROUP 1 = VICTORIAN SAMPLE N=244 07/06/01 PAGE : 14
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MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

VARIANCES OF INDEPENDENT VARIABLES (CONTINUED)

E35 -BSI33 .600*
.072
8.280
(.095)
(6.306)

E37 -BSI35 .686*
.080
8.534
(.125)
(5.472)

E38 -BSI36 .648*
.071
9.077
(.098)
(6.609)

E45 -BSI43 .466*
.073
6.337
(.087)
(5.351)

E47 -BSI45 .365*
.046
7.891
(.063)
(5.845)

E52 -BSI50 .421*
.062
6.822
(.079)
(5.340)

E53 -BSI51 .620*
.078
7.992
(.090)
(6.879)

TITLE: GROUP 1 = VICTORIAN SAMPLE N=244 07/06/01 PAGE : 15
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MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

COVARIANCES AMONG INDEPENDENT VARIABLES

V	F	
F2 - PHO		.614*
F1 - DEP		.053
		11.489
		(.066)
		(9.339)
F3 - SOM		.527*
F1 - DEP		.065
		8.107
		(.084)
		(6.272)
F4 - COG		.825*
F1 - DEP		.035
		23.657
		(.040)
		(20.531)
F5 - ANX		.717*
F1 - DEP		.047
		15.386
		(.055)
		(13.057)
F6 - PAR		.672*
F1 - DEP		052
		13 016
		(063)
		(10 656)
F3 - SOM		.541*
F2 - PHO		.065
		8.340
		(.080)
		(6.732)
F4 - COG		.639*
F2 - PHO		.052
		12.369
		(.065)
		(9.779)
F5 - ANX		.789*
F2 - PHO		.041
		19.143
		(.050)
		(15.853)
F6 - PAR		.656*
F2 - PHO		.054
		12.235
		(.069)
		(9.495)
F4 - COG		.678*
F3 - SOM		.055
		12.410

		(.076)|
		(8.913)|
		|
| F5	- ANX	.680*|
| F3	- SOM	.057 |
		12.006 |
		(.071)|
		(9.533)|
		|
| F6	- PAR	.729*|
| F3	- SOM	.054 |
		13.429 |
		(.080)|
		(9.103)|
		|

TITLE: GROUP 1 = VICTORIAN SAMPLE N=244 07/06/01 PAGE : 16
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MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

COVARIANCES AMONG INDEPENDENT VARIABLES (CONTINUED)

F5 - ANX	.717*
F4 - COG	.047
	15.342
	(.053)
	(13.442)
F6 - PAR	.733*
F4 - COG	.047
	15.662
	(.054)
	(13.549)
F6 - PAR	.782*
F5 - ANX	.045
	17.473
	(.062)
	(12.648)

TITLE: GROUP 1 = VICTORIAN SAMPLE N=244 07/06/01 PAGE : 17
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MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

STANDARDIZED SOLUTION: R-SQUARED

BSI1	=V3 = .653*F5 + .757 E3	.427
BSI5	=V7 = .714*F4 + .700 E7	.510
BSI8	=V10 = .773*F2 + .635 E10	.597
BSI10	=V12 = .662*F6 + .750 E12	.438
BSI17	=V19 = .748*F1 + .663 E19	.560
BSI19	=V21 = .786*F5 + .618 E21	.618
BSI21	=V23 = .761*F6 + .649 E23	.579
BSI27	=V29 = .787*F4 + .617 E29	.620
BSI28	=V30 = .697*F2 + .717 E30	.485
BSI29	=V31 = .694*F3 + .720 E31	.482
BSI30	=V32 = .679*F3 + .734 E32	.461
BSI32	=V34 = .741*F4 + .671 E34	.549
BSI33	=V35 = .692*F3 + .722 E35	.478
BSI35	=V37 = .780*F1 + .626 E37	.608
BSI36	=V38 = .751*F4 + .660 E38	.564
BSI43	=V45 = .844*F2 + .536 E45	.713
BSI45	=V47 = .799*F5 + .601 E47	.639
BSI50	=V52 = .850*F1 + .526 E52	.723
BSI51	=V53 = .766*F6 + .643 E53	.587

TITLE: GROUP 1 = VICTORIAN SAMPLE N=244 07/06/01 PAGE : 18
EQS/EM386 Licensee: Neil Preston
MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

CORRELATIONS AMONG INDEPENDENT VARIABLES

V		F	
	F2 - PHO		.614*
	F1 - DEP		
	F3 - SOM		.527*
	F1 - DEP		
	F4 - COG		.825*
	F1 - DEP		
	F5 - ANX		.717*
	F1 - DEP		
	F6 - PAR		.672*
	F1 - DEP		
	F3 - SOM		.541*
	F2 - PHO		
	F4 - COG		.639*
	F2 - PHO		
	F5 - ANX		.789*
	F2 - PHO		
	F6 - PAR		.656*
	F2 - PHO		
	F4 - COG		.678*
	F3 - SOM		
	F5 - ANX		.680*
	F3 - SOM		
	F6 - PAR		.729*
	F3 - SOM		
	F5 - ANX		.717*
	F4 - COG		
	F6 - PAR		.733*
	F4 - COG		
	F6 - PAR		.782*
	F5 - ANX		

END OF METHOD

TITLE: GROUP 1 = VICTORIAN SAMPLE N=244 07/06/01 PAGE : 19
EQS/EM386 Licensee: Neil Preston
MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

LAGRANGIAN MULTIPLIER TEST REQUIRES 126614 WORDS OF MEMORY.
PROGRAM ALLOCATES 10000000 WORDS.

LAGRANGE MULTIPLIER TEST (FOR ADDING PARAMETERS)

ORDERED UNIVARIATE TEST STATISTICS:

NO	CODE	PARAMETER	CHI-SQUARE	PROBABILITY	PARAMETER CHANGE
1	2 12	V47,F2	14.477	0.000	0.486
2	2 12	V7,F1	13.639	0.000	-0.606
3	2 6	E32,E19	12.902	0.000	0.162
4	2 12	V23,F4	12.536	0.000	0.457
5	2 6	E23,E19	11.993	0.001	-0.168
6	2 12	V19,F5	10.101	0.001	0.375
7	2 6	E45,E19	9.423	0.002	0.154
8	2 6	E34,E12	9.171	0.002	-0.167
9	2 12	V53,F5	8.211	0.004	0.508
10	2 6	E53,E23	8.056	0.005	-0.196
11	2 6	E47,E37	8.035	0.005	-0.118
12	2 6	E29,E21	7.880	0.005	-0.128
13	2 6	E30,E29	7.864	0.005	0.119
14	2 12	V52,F2	7.476	0.006	-0.257
15	2 6	E34,E7	7.433	0.006	0.154
16	2 12	V29,F1	7.248	0.007	0.427
17	2 12	V21,F2	6.998	0.008	-0.391
18	2 12	V12,F5	6.988	0.008	-0.444
19	2 6	E37,E23	6.837	0.009	0.127
20	2 12	V12,F4	6.651	0.010	-0.359
21	2 12	V38,F6	6.471	0.011	-0.310
22	2 6	E53,E37	6.157	0.013	-0.132
23	2 6	E34,E29	6.067	0.014	-0.133
24	2 6	E47,E45	5.590	0.018	0.094
25	2 12	V47,F6	5.539	0.019	-0.315
26	2 12	V19,F2	5.434	0.020	0.227
27	2 6	E53,E3	5.363	0.021	0.122
28	2 6	E53,E45	5.315	0.021	-0.115
29	2 6	E37,E32	5.219	0.022	-0.103
30	2 6	E52,E7	5.195	0.023	-0.112
31	2 6	E19,E3	5.185	0.023	0.123
32	2 12	V23,F1	4.870	0.027	0.238
33	2 6	E35,E23	4.831	0.028	-0.102
34	2 6	E38,E37	4.727	0.030	0.114
35	2 12	V47,F1	4.640	0.031	-0.214
36	2 12	V21,F1	4.620	0.032	0.247
37	2 6	E23,E3	4.490	0.034	-0.102
38	2 6	E7,E3	4.199	0.040	0.114
39	2 12	V12,F1	4.168	0.041	-0.243
40	2 12	V34,F5	3.973	0.046	0.223
41	2 6	E45,E29	3.833	0.050	-0.092
42	2 6	E34,E23	3.802	0.051	0.089
43	2 6	E52,E3	3.775	0.052	-0.090
44	2 6	E45,E31	3.705	0.054	0.085
45	2 12	V47,F4	3.652	0.056	-0.199
46	2 6	E47,E30	3.623	0.057	0.066
47	2 12	V21,F6	3.583	0.058	0.293
48	2 6	E31,E7	3.571	0.059	0.092
49	2 6	E29,E12	3.512	0.061	0.104
50	2 6	E30,E19	3.456	0.063	-0.085

51	2	6	E32,E7	3.375	0.066	-0.085
52	2	6	E53,E38	3.283	0.070	-0.092
53	2	6	E45,E21	3.169	0.075	-0.083
54	2	12	V19,F3	3.106	0.078	0.165
55	2	6	E47,E12	3.051	0.081	-0.079
56	2	6	E52,E29	3.028	0.082	0.078
57	2	6	E53,E21	3.005	0.083	0.084
58	2	6	E31,E10	2.950	0.086	-0.064
59	2	12	V3,F3	2.946	0.086	0.197
60	2	6	E21,E19	2.877	0.090	0.083
61	2	12	V34,F3	2.785	0.095	0.179
62	2	6	E47,E7	2.740	0.098	-0.070
63	2	12	V35,F6	2.687	0.101	-0.242
64	2	6	E30,E21	2.672	0.102	-0.067
65	2	12	V37,F5	2.501	0.114	-0.193
66	2	6	E37,E30	2.453	0.117	0.072
67	2	6	E23,E12	2.360	0.124	0.095
68	2	12	V45,F5	2.357	0.125	0.285
69	2	12	V29,F3	2.300	0.129	-0.167
70	2	6	E34,E21	2.295	0.130	0.069
71	2	6	E12,E10	2.244	0.134	0.070
72	2	6	E38,E34	2.228	0.136	-0.080
73	2	12	V47,F3	2.216	0.137	-0.148
74	2	6	E19,E7	2.216	0.137	-0.084
75	2	12	V37,F3	2.182	0.140	-0.142
76	2	6	E38,E3	2.180	0.140	-0.076
77	2	6	E31,E12	2.171	0.141	0.076
78	2	6	E52,E12	2.153	0.142	-0.075
79	2	6	E38,E29	2.121	0.145	0.081
80	2	6	E53,E34	2.086	0.149	-0.072
81	2	6	E38,E7	2.083	0.149	0.084
82	2	12	V7,F3	2.071	0.150	0.168
83	2	6	E53,E7	2.066	0.151	0.079
84	2	12	V38,F5	2.043	0.153	-0.165
85	2	12	V3,F2	2.022	0.155	-0.203
86	2	12	V52,F5	1.884	0.170	-0.160
87	2	6	E52,E47	1.880	0.170	0.049
88	2	6	E53,E12	1.863	0.172	0.094
89	2	6	E29,E7	1.861	0.172	-0.080
90	2	6	E45,E38	1.858	0.173	0.065
91	2	6	E35,E29	1.850	0.174	-0.065
92	2	12	V38,F3	1.850	0.174	-0.149
93	2	6	E37,E12	1.782	0.182	0.079
94	2	6	E37,E19	1.768	0.184	-0.086
95	2	6	E30,E3	1.759	0.185	-0.060
96	2	12	V10,F1	1.696	0.193	-0.106
97	2	6	E31,E19	1.689	0.194	-0.061
98	2	12	V45,F1	1.685	0.194	0.136
99	2	12	V21,F4	1.660	0.198	0.156
100	2	12	V7,F2	1.634	0.201	-0.130
101	2	12	V23,F2	1.604	0.205	0.134
102	2	6	E32,E3	1.532	0.216	0.055
103	2	12	V29,F6	1.507	0.220	0.151
104	2	6	E38,E35	1.504	0.220	0.060
105	2	6	E35,E32	1.487	0.223	0.067
106	2	6	E37,E34	1.481	0.224	-0.063
107	2	6	E32,E10	1.481	0.224	0.043
108	2	6	E53,E52	1.454	0.228	0.056
109	2	6	E52,E45	1.448	0.229	-0.053
110	2	12	V53,F4	1.436	0.231	-0.171
111	2	6	E30,E12	1.423	0.233	-0.059
112	2	6	E38,E12	1.406	0.236	-0.067
113	2	12	V7,F6	1.396	0.237	0.152
114	2	6	E52,E37	1.396	0.237	0.083
115	2	6	E45,E7	1.329	0.249	-0.060
116	2	6	E30,E10	1.275	0.259	0.050
117	2	6	E32,E31	1.243	0.265	-0.057

118	2	6	E38,E32	1.165	0.281	-0.046
119	2	6	E38,E19	1.159	0.282	-0.056
120	2	12	V10,F4	1.138	0.286	-0.090
121	2	6	E47,E34	1.133	0.287	0.041
122	2	12	V32,F5	1.127	0.288	0.111
123	2	12	V45,F3	1.099	0.295	0.106
124	2	12	V10,F3	1.095	0.295	-0.083
125	2	6	E19,E10	1.091	0.296	-0.045
126	2	6	E47,E35	1.075	0.300	-0.041
127	2	12	V30,F4	1.073	0.300	0.086
128	2	6	E38,E21	1.066	0.302	0.048
129	2	6	E35,E10	1.014	0.314	-0.041
130	2	6	E52,E35	0.999	0.318	-0.044
131	2	6	E52,E30	0.956	0.328	-0.038
132	2	12	V31,F6	0.941	0.332	0.133
133	2	6	E37,E35	0.938	0.333	0.050
134	2	6	E30,E23	0.931	0.335	0.039
135	2	6	E53,E35	0.908	0.341	0.048
136	2	6	E47,E23	0.904	0.342	-0.036
137	2	12	V10,F5	0.881	0.348	-0.127
138	2	6	E31,E30	0.878	0.349	-0.037
139	2	6	E35,E3	0.841	0.359	0.047
140	2	6	E45,E30	0.837	0.360	-0.054
141	2	6	E31,E21	0.807	0.369	-0.038
142	2	6	E29,E10	0.798	0.372	0.036
143	2	6	E53,E19	0.773	0.379	0.047
144	2	12	V53,F3	0.771	0.380	0.133
145	2	6	E53,E47	0.757	0.384	0.036
146	2	6	E35,E30	0.731	0.392	0.037
147	2	12	V31,F4	0.712	0.399	0.091
148	2	6	E34,E19	0.638	0.424	0.041
149	2	12	V32,F1	0.637	0.425	0.063
150	2	12	V35,F5	0.628	0.428	-0.096
151	2	12	V29,F2	0.627	0.429	0.076
152	2	6	E52,E31	0.588	0.443	0.031
153	2	6	E52,E23	0.586	0.444	0.032
154	2	12	V3,F4	0.582	0.446	0.089
155	2	6	E35,E34	0.579	0.447	0.037
156	2	12	V35,F1	0.561	0.454	-0.068
157	2	6	E47,E38	0.558	0.455	-0.029
158	2	12	V53,F2	0.558	0.455	-0.088
159	2	12	V37,F2	0.540	0.462	0.074
160	2	6	E45,E12	0.533	0.465	0.040
161	2	6	E53,E10	0.503	0.478	0.030
162	2	6	E23,E7	0.489	0 484	0.035
163	2	6	E10,E3	0.463	0 496	-0.029
164	2	12	V32,F2	0.457	0 499	0.055
165	2	6	E37,E31	0.454	0 500	-0.032
166	2	12	V32,F6	0.451	0.502	0.085
167	2	6	E47,E29	0.444	0.505	0.026
168	2	6	E21,E10	0.442	0.506	0.026
169	2	12	V34,F2	0.428	0.513	0.061
170	2	6	E52,E34	0.402	0.526	0.028
171	2	12	V30,F5	0.373	0.542	-0.076
172	2	6	E34,E10	0.346	0.556	-0.024
173	2	6	E38,E10	0.342	0.558	-0.024
174	2	12	V12,F2	0.342	0.559	-0.068
175	2	6	E12,E3	0.340	0.560	-0.034
176	2	12	V52,F4	0.339	0.560	-0.104
177	2	6	E34,E31	0.337	0.562	0.026
178	2	6	E45,E37	0.333	0.564	0.029
179	2	6	E32,E30	0.332	0.565	-0.022
180	2	6	E21,E7	0.320	0.572	0.028
181	2	12	V3,F6	0.319	0.572	0.083
182	2	12	V34,F1	0.318	0.573	0.086
183	2	12	V19,F6	0.317	0.573	0.063
184	2	6	E47,E31	0.309	0.578	0.020

185	2	12	V7,F5	0.305	0.581	-0.067
186	2	6	E34,E32	0.292	0.589	0.023
187	2	6	E47,E10	0.287	0.592	-0.018
188	2	12	V32,F4	0.267	0.605	-0.052
189	2	6	E52,E32	0.265	0.607	0.020
190	2	12	V35,F2	0.259	0.611	-0.047
191	2	6	E35,E21	0.251	0.616	0.023
192	2	6	E53,E31	0.245	0.621	-0.023
193	2	6	E31,E23	0.236	0.627	0.021
194	2	6	E29,E3	0.236	0.627	0.024
195	2	12	V23,F3	0.230	0.632	-0.066
196	2	6	E31,E29	0.223	0.637	-0.021
197	2	12	V12,F3	0.204	0.651	-0.068
198	2	6	E35,E19	0.204	0.651	-0.023
199	2	6	E52,E38	0.199	0.655	-0.020
200	2	6	E52,E10	0.197	0.657	-0.017
201	2	6	E29,E23	0.195	0.659	0.020
202	2	6	E21,E12	0.192	0.662	-0.023
203	2	12	V37,F4	0.186	0.666	0.077
204	2	12	V23,F5	0.170	0.680	-0.066
205	2	6	E32,E23	0.163	0.687	0.016
206	2	6	E45,E32	0.160	0.689	-0.017
207	2	6	E53,E30	0.159	0.690	-0.018
208	2	12	V53,F1	0.155	0.693	-0.047
209	2	6	E31,E3	0.150	0.699	-0.018
210	2	6	E35,E7	0.149	0.700	0.020
211	2	6	E32,E21	0.145	0.704	-0.015
212	2	6	E19,E12	0.137	0.712	0.022
213	2	6	E38,E23	0.133	0.715	0.017
214	2	6	E45,E23	0.132	0.716	0.016
215	2	6	E32,E12	0.116	0.733	-0.017
216	2	12	V35,F4	0.111	0.739	-0.039
217	2	6	E53,E29	0.103	0.749	0.016
218	2	6	E23,E21	0.099	0.753	0.014
219	2	6	E47,E19	0.089	0.765	-0.012
220	2	12	V38,F2	0.087	0.768	-0.028
221	2	12	V37,F6	0.076	0.782	-0.032
222	2	12	V31,F5	0.068	0.795	-0.029
223	2	6	E45,E35	0.066	0.797	0.012
224	2	6	E29,E19	0.065	0.798	0.013
225	2	6	E30,E7	0.061	0.805	-0.012
226	2	12	V52,F6	0.060	0.806	-0.027
227	2	6	E37,E10	0.058	0.810	0.010
228	2	6	E37,E29	0.051	0.822	0.012
229	2	6	E45,E3	0.050	0.823	0.011
230	2	6	E32,E29	0.049	0.824	-0.009
231	2	6	E38,E31	0.048	0.827	-0.010
232	2	12	V30,F6	0.042	0.838	-0.018
233	2	12	V10,F6	0.041	0.840	0.019
234	2	6	E45,E10	0.040	0.841	-0.014
235	2	6	E52,E21	0.039	0.843	0.008
236	2	6	E38,E30	0.038	0.846	-0.008
237	2	6	E35,E12	0.036	0.849	-0.011
238	2	6	E34,E3	0.035	0.851	-0.009
239	2	6	E12,E7	0.035	0.852	-0.011
240	2	6	E47,E21	0.033	0.855	-0.010
241	2	12	V52,F3	0.033	0.856	-0.016
242	2	6	E21,E3	0.029	0.864	0.009
243	2	12	V19,F4	0.027	0.869	0.028
244	2	12	V31,F2	0.025	0.875	-0.014
245	2	12	V34,F6	0.020	0.887	0.017
246	2	6	E37,E3	0.019	0.890	-0.007
247	2	6	E34,E30	0.018	0.894	0.006
248	2	6	E52,E19	0.017	0.897	0.008
249	2	12	V45,F4	0.016	0.899	0.014
250	2	12	V21,F3	0.011	0.915	0.012
251	2	6	E35,E31	0.011	0.917	-0.006

252	2	6	E37,E7	0.009	0.924	0.005
253	2	6	E53,E32	0.007	0.934	0.004
254	2	12	V38,F1	0.006	0.938	0.012
255	2	12	V30,F3	0.005	0.941	-0.006
256	2	12	V30,F1	0.005	0.943	-0.006
257	2	6	E10,E7	0.004	0.947	0.003
258	2	6	E45,E34	0.004	0.948	-0.003
259	2	12	V31,F1	0.002	0.969	-0.003
260	2	12	V3,F1	0.001	0.974	0.004
261	2	6	E47,E3	0.000	0.984	0.001
262	2	12	V45,F6	0.000	0.987	-0.002
263	2	6	E47,E32	0.000	0.989	0.000
264	2	12	V29,F5	0.000	0.990	-0.001
265	2	6	E37,E21	0.000	0.991	-0.001
266	2	6	E23,E10	0.000	0.996	0.000
267	2	0	F2,F2	0.000	1.000	0.000
268	2	0	F3,F3	0.000	1.000	0.000
269	2	0	F4,F4	0.000	1.000	0.000
270	2	0	F5,F5	0.000	1.000	0.000
271	2	0	F6,F6	0.000	1.000	0.000
272	2	0	F1,F1	0.000	1.000	0.000

TITLE: GROUP 1 = VICTORIAN SAMPLE N=244 07/06/01 PAGE : 20
EQS/EM386 Licensee: Neil Preston
MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

MULTIVARIATE LAGRANGE MULTIPLIER TEST BY SIMULTANEOUS PROCESS IN STAGE 1

PARAMETER SETS (SUBMATRICES) ACTIVE AT THIS STAGE ARE:

PEE GVF

		CUMULATIVE MULTIVARIATE STATISTICS			UNIVARIATE INCREMENT	
STEP	PARAMETER	CHI-SQUARE	D.F.	PROBABILITY	CHI-SQUARE	PROBABILITY
1	V47,F2	14.477	1	0.000	14.477	0.000
2	V7,F1	28.115	2	0.000	13.639	0.000
3	E32,E19	41.018	3	0.000	12.902	0.000
4	V23,F4	53.553	4	0.000	12.536	0.000
5	E23,E19	65.439	5	0.000	11.886	0.001
6	V19,F5	75.110	6	0.000	9.671	0.002
7	E29,E21	83.316	7	0.000	8.206	0.004
8	V53,F5	90.795	8	0.000	7.479	0.006
9	E45,E19	98.236	9	0.000	7.441	0.006
10	E30,E29	105.188	10	0.000	6.952	0.008
11	E34,E12	112.120	11	0.000	6.932	0.008
12	V38,F6	120.182	12	0.000	8.062	0.005
13	E53,E45	126.683	13	0.000	6.502	0.011
14	E34,E7	132.152	14	0.000	5.469	0.019
15	E53,E37	137.426	15	0.000	5.274	0.022
16	E47,E37	143.250	16	0.000	5.825	0.016
17	E7,E3	147.958	17	0.000	4.708	0.030
18	E35,E23	152.597	18	0.000	4.639	0.031
19	E19,E3	157.165	19	0.000	4.568	0.033

1

Execution begins at 10:41:26.07
Execution ends at 10:41:29.80
Elapsed time = 3.73 seconds

APPENDIX VI

EQS output of structural model

between Cognition and Paranoia

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1
 EQS, A STRUCTURAL EQUATION PROGRAM          MULTIVARIATE SOFTWARE, INC.
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  PROGRAM CONTROL INFORMATION

   1 /TITLE
   2 PARCOG1 FULL CROSS INFLUENTIAL MODEL
   3 /SPECIFICATIONS
   4  DATA='C:\EQS\STRUCT3.ESS';
   5  VARIABLES= 67; CASES=  145;
   6  METHODS=ML, ROBUST;
   7  MATRIX=RAW;
   8 /LABELS
   9 V1=UR; V2=ANX1_1; V3=ANX2_1; V4=ANX3_1; V5=COG1_1;
  10 V6=COG2_1; V7=COG3_1; V8=COG4_1; V9=PHO1_1; V10=PHO2_1;
  11 V11=PHO3_1; V12=PAR1_1; V13=PAR2_1; V14=PAR3_1; V15=DEP1_1;
  12 V16=DEP2_1; V17=DEP3_1; V18=SOM1_1; V19=SOM2_1; V20=SOM3_1;
  13 V21=HOS1_1; V22=HOS2_1; V23=HOS3_1; V24=ANX1_2; V25=ANX2_2;
  14 V26=ANX3_2; V27=COG1_2; V28=COG2_2; V29=COG3_2; V30=COG4_2;
  15 V31=PHO1_2; V32=PHO2_2; V33=PHO3_2; V34=PAR1_2; V35=PAR2_2;
  16 V36=PAR3_2; V37=DEP1_2; V38=DEP2_2; V39=DEP3_2; V40=SOM1_2;
  17 V41=SOM2_2; V42=SOM3_2; V43=HOS1_2; V44=HOS2_2; V45=HOS3_2;
  18 V46=ANX1_3; V47=ANX2_3; V48=ANX3_3; V49=COG1_3; V50=COG2_3;
  19 V51=COG3_3; V52=COG4_3; V53=PHO1_3; V54=PHO2_3; V55=PHO3_3;
  20 V56=PAR1_3; V57=PAR2_3; V58=PAR3_3; V59=DEP1_3; V60=DEP2_3;
  21 V61=DEP3_3; V62=SOM1_3; V63=SOM2_3; V64=SOM3_3; V65=HOS1_3;
  22 V66=HOS2_3; V67=HOS3_3;
  23 /EQUATIONS
  24 V5 =  + 1F4  + 1E5;
  25 V6 =  + *F4  + 1E6;
  26 V7 =  + *F4  + 1E7;
  27 V8 =  + *F4  + 1E8;
  28 V12 =  + 1F1  + 1E12;
  29 V13 =  + *F1  + 1E13;
  30 V14 =  + *F1  + 1E14;
  31 V27 =  + 1F5  + 1E27;
  32 V28 =  + *F5  + 1E28;
  33 V29 =  + *F5  + 1E29;
  34 V30 =  + *F5  + 1E30;
  35 V34 =  + 1F2  + 1E34;
  36 V35 =  + *F2  + 1E35;
  37 V36 =  + *F2  + 1E36;
  38 V49 =  + 1F6  + 1E49;
  39 V50 =  + *F6  + 1E50;
  40 V51 =  + *F6  + 1E51;
  41 V52 =  + *F6  + 1E52;
  42 V56 =  + 1F3  + 1E56;
  43 V57 =  + *F3  + 1E57;
  44 V58 =  + *F3  + 1E58;
  45 F2 =  + *F1  + *F4  + 1D2;
  46 F3 =  + *F1  + *F2  + *F5  + 1D3;
  47 F5 =  + *F1  + *F4  + 1D5;
  48 F6 =  + *F2  + *F4  + *F5  + 1D6;
  49 /VARIANCES
  50 F1 = *;
  51 F4 = *;
  52 E5 = *;
```

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```
 53  E6 = *;
 54  E7 = *;
 55  E8 = *;
 56  E12 = *;
 57  E13 = *;
 58  E14 = *;
 59  E27 = *;
 60  E28 = *;
 61  E29 = *;
 62  E30 = *;
 63  E34 = *;
 64  E35 = *;
 65  E36 = *;
 66  E49 = *;
 67  E50 = *;
 68  E51 = *;
 69  E52 = *;
 70  E56 = *;
 71  E57 = *;
 72  E58 = *;
 73  D2 = *;
 74  D3 = *;
 75  D5 = *;
 76  D6 = *;
 77  /COVARIANCES
 78  F4 , F1 = *;
 79  E27 , E5 = *;
 80  E28 , E6 = *;
 81  E29 , E7 = *;
 82  E30 , E8 = *;
 83  E34 , E12 = *;
 84  E35 , E13 = *;
 85  E36 , E14 = *;
 86  E49 , E27 = *;
 87  E50 , E28 = *;
 88  E51 , E29 = *;
 89  E52 , E30 = *;
 90  E56 , E34 = *;
 91  E57 , E35 = *;
 92  E58 , E36 = *;
 93  D5 , D2 = *;
 94  D6 , D3 = *;
 95  /LMTEST
 96   SET=PEE, GVF;
 97  /PRINT
 98   FIT = ALL;
 99  /END

  99 RECORDS OF INPUT MODEL FILE WERE READ

DATA IS READ FROM C:\EQS\STRUCT3.ESS
THERE ARE  67 VARIABLES AND  145 CASES
IT IS A RAW DATA ESS FILE
```

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SAMPLE STATISTICS BASED ON COMPLETE CASES

UNIVARIATE STATISTICS

VARIABLE	COG1_1	COG2_1	COG3_1	COG4_1	PAR1_1
MEAN	2.2483	2.1931	1.8755	2.2552	1.9862
SKEWNESS (G1)	0.6934	0.7589	1.4437	0.7839	0.9870
KURTOSIS (G2)	-0.5326	-0.3480	1.1974	-0.2536	-0.3601
STANDARD DEV.	1.2107	1.1920	1.1781	1.1831	1.2692

VARIABLE	PAR2_1	PAR3_1	COG1_2	COG2_2	COG3_2
MEAN	1.8414	1.9448	1.9380	1.9172	1.6483
SKEWNESS (G1)	1.2031	1.1426	1.1028	1.2592	1.6184
KURTOSIS (G2)	0.6689	0.0670	0.3662	0.5649	1.9482
STANDARD DEV.	1.1098	1.2626	1.1317	1.2048	1.0174

VARIABLE	COG4_2	PAR1_2	PAR2_2	PAR3_2	COG1_3
MEAN	2.0207	1.7793	1.5931	1.7655	2.0832
SKEWNESS (G1)	0.9641	1.4856	1.9214	1.5246	1.1204
KURTOSIS (G2)	-0.0986	1.2712	2.7927	1.5805	0.3048
STANDARD DEV.	1.1754	1.1394	1.0704	1.1056	1.2158

VARIABLE	COG2_3	COG3_3	COG4_3	PAR1_3	PAR2_3
MEAN	1.8621	1.8194	1.9034	1.7379	1.5724
SKEWNESS (G1)	1.0886	1.3872	1.2877	1.5568	2.0034
KURTOSIS (G2)	0.3221	1.6374	0.5432	1.8036	3.4317
STANDARD DEV.	1.0582	1.0330	1.2209	1.0476	1.0052

VARIABLE	PAR3_3
MEAN	1.8000
SKEWNESS (G1)	1.4393
KURTOSIS (G2)	0.9689
STANDARD DEV.	1.2053

MULTIVARIATE KURTOSIS

PROGRAM ALLOCATES 10000000 WORDS.

LAGRANGE MULTIPLIER TEST (FOR ADDING PARAMETERS)

ORDERED UNIVARIATE TEST STATISTICS:

NO	CODE		PARAMETER	CHI-SQUARE	PROBABILITY	PARAMETER CHANGE
1	2	6	E35,E34	26.019	0.000	0.350
2	2	6	E8,E6	13.090	0.000	0.304
3	2	6	E36,E28	11.727	0.001	0.185
4	2	6	E29,E27	10.647	0.001	0.180
5	2	12	V13,F4	9.395	0.002	0.564
6	2	12	V36,F1	9.342	0.002	0.289
7	2	6	E34,E28	8.183	0.004	-0.166
8	2	6	E36,E35	7.769	0.005	-0.189
9	2	6	E6,E5	7.666	0.006	-0.238
10	2	12	V36,F4	7.192	0.007	0.252
11	2	6	E52,E49	7.107	0.008	0.214
12	2	6	E58,E14	7.032	0.008	0.208
13	2	6	E58,E13	6.621	0.010	-0.157
14	2	12	V35,F1	6.590	0.010	-0.249
15	2	6	E51,E49	6.516	0.011	0.176
16	2	6	E36,E12	6.367	0.012	0.166
17	2	6	E14,E8	6.366	0.012	-0.195
18	2	6	E57,E28	6.163	0.013	0.132
19	2	6	E57,E34	6.022	0.014	-0.140
20	2	6	E30,E28	5.908	0.015	0.156
21	2	6	E12,E7	5.465	0.019	-0.170
22	2	6	E52,E34	5.407	0.020	-0.152
23	2	12	V57,F4	5.377	0.020	0.221
24	2	6	E56,E49	5.335	0.021	-0.157
25	2	6	E58,E56	5.220	0.022	0.168
26	2	6	E13,E7	5.093	0.024	0.136
27	2	6	E57,E6	4.661	0.031	0.129
28	2	6	E35,E28	4.584	0.032	-0.114
29	2	6	E30,E29	4.552	0.033	-0.124
30	2	6	E57,E52	4.528	0.033	0.131
31	2	6	E35,E12	4.491	0.034	-0.142
32	2	6	E36,E34	4.387	0.036	-0.149
33	2	6	E7,E5	4.211	0.040	0.177
34	2	6	E50,E6	4.206	0.040	0.127
35	2	6	E36,E5	3.832	0.050	0.117
36	2	6	E58,E34	3.832	0.050	0.138
37	2	12	V12,F4	3.655	0.056	-0.381
38	2	6	E27,E8	3.584	0.058	0.124
39	2	6	E8,E7	3.557	0.059	-0.154
40	2	12	V56,F4	3.442	0.064	-0.186
41	2	6	E30,E27	3.304	0.069	-0.114
42	2	6	E50,E36	3.295	0.069	-0.084
43	2	6	E56,E35	3.294	0.070	-0.103
44	2	6	E56,E50	2.988	0.084	0.090
45	2	6	E51,E28	2.956	0.086	-0.101
46	2	6	E49,E36	2.729	0.099	0.102
47	2	6	E14,E13	2.659	0.103	-0.161
48	2	6	E14,E5	2.590	0.108	0.121
49	2	6	E57,E36	2.541	0.111	0.086
50	2	12	V57,F1	2.538	0.111	0.151
51	2	12	V8,F1	2.508	0.113	-0.281
52	2	6	E50,E49	2.503	0.114	-0.102
53	2	6	E52,E50	2.442	0.118	-0.100
54	2	6	E50,E35	2.344	0.126	0.069
55	2	6	E12,E6	2.320	0.128	0.118
56	2	6	E14,E12	2.308	0.129	0.149
57	2	6	E29,E28	2.205	0.138	-0.083

58	2	6	E50,E5	2.156	0.142	-0.081
59	2	6	E34,E29	2.082	0.149	0.072
60	2	6	E51,E7	1.995	0.158	0.087
61	2	12	V5,F1	1.994	0.158	0.257
62	2	6	E52,E28	1.982	0.159	0.096
63	2	6	E51,E5	1.970	0.160	0.088
64	2	6	E13,E8	1.924	0.165	0.088
65	2	6	E57,E27	1.895	0.169	-0.068
66	2	6	E49,E5	1.874	0.171	0.106
67	2	6	E58,E49	1.829	0.176	-0.105
68	2	12	V35,F4	1.809	0.179	-0.126
69	2	6	E13,E6	1.778	0.182	-0.085
70	2	6	E49,E6	1.761	0.184	-0.103
71	2	12	V14,F4	1.761	0.185	-0.269
72	2	6	E51,E14	1.738	0.187	-0.084
73	2	6	E30,E13	1.700	0.192	-0.069
74	2	6	E27,E14	1.691	0.193	-0.080
75	2	6	E35,E6	1.688	0.194	0.079
76	2	6	E50,E30	1.653	0.199	0.066
77	2	6	E57,E49	1.628	0.202	-0.080
78	2	6	E51,E6	1.598	0.206	-0.082
79	2	6	E27,E6	1.569	0.210	-0.082
80	2	6	E12,E5	1.569	0.210	-0.094
81	2	12	V34,F4	1.490	0.222	-0.123
82	2	6	E58,E52	1.474	0.225	-0.093
83	2	6	E58,E27	1.469	0.226	-0.075
84	2	6	E57,E56	1.450	0.228	-0.084
85	2	6	E35,E30	1.432	0.231	0.063
86	2	6	E56,E14	1.367	0.242	0.076
87	2	6	E35,E5	1.323	0.250	-0.068
88	2	6	E50,E27	1.320	0.251	-0.056
89	2	6	E52,E51	1.314	0.252	-0.078
90	2	6	E51,E27	1.307	0.253	0.062
91	2	6	E35,E8	1.279	0.258	0.069
92	2	12	V50,F4	1.236	0.266	-0.107
93	2	6	E36,E8	1.235	0.266	-0.069
94	2	12	V58,F4	1.224	0.269	-0.125
95	2	6	E52,E13	1.203	0.273	0.065
96	2	6	E57,E29	1.192	0.275	-0.049
97	2	6	E56,E27	1.176	0.278	0.058
98	2	6	E30,E14	1.158	0.282	0.071
99	2	12	V28,F4	1.153	0.283	-0.124
100	2	6	E51,E34	1.015	0.314	0.057
101	2	6	E49,E30	1.008	0.315	-0.069
102	2	6	E58,E57	0.972	0.324	-0.071
103	2	6	E56,E5	0.953	0.329	-0.062
104	2	6	E29,E5	0.949	0.330	-0.058
105	2	12	V58,F1	0 942	0.332	-0.107
106	2	12	V52,F1	0 926	0.336	0.102
107	2	6	E36,E6	0 917	0.338	-0.059
108	2	6	E52,E29	0 901	0.343	-0.055
109	2	6	E52,E6	0.898	0.343	-0.071
110	2	12	V27,F4	0.878	0.349	0.102
111	2	6	E27,E13	0.844	0.358	0.046
112	2	6	E35,E14	0.793	0.373	-0.061
113	2	6	E58,E30	0.761	0.383	0.057
114	2	6	E34,E7	0.729	0.393	0.053
115	2	6	E56,E28	0.703	0.402	-0.049
116	2	6	E57,E50	0.692	0.406	0.040
117	2	6	E30,E12	0.686	0.408	-0.054
118	2	6	E28,E7	0.683	0.409	-0.054
119	2	6	E29,E13	0.674	0.412	0.038
120	2	6	E50,E29	0.637	0.425	0.035
121	2	6	E56,E8	0.632	0.427	-0.052
122	2	12	V56,F1	0.627	0.429	-0.078
123	2	6	E49,E8	0.615	0.433	-0.061
124	2	6	E57,E7	0.614	0.433	-0.044

125	2	6	E14,E7	0.612	0.434	-0.057
126	2	6	E57,E12	0.611	0.434	0.046
127	2	6	E58,E51	0.591	0.442	-0.050
128	2	12	V29,F1	0.565	0.452	0.061
129	2	6	E8,E5	0.553	0.457	-0.062
130	2	6	E34,E8	0.547	0.459	-0.050
131	2	6	E28,E8	0.540	0.462	-0.051
132	2	12	V6,F1	0.534	0.465	0.131
133	2	6	E52,E14	0.534	0.465	0.054
134	2	6	E56,E6	0.520	0.471	-0.047
135	2	6	E36,E29	0.498	0.480	-0.033
136	2	6	E58,E28	0.484	0.487	0.046
137	2	6	E56,E36	0.453	0.501	0.038
138	2	12	V7,F1	0.430	0.512	-0.116
139	2	6	E36,E30	0.430	0.512	0.035
140	2	6	E36,E7	0.425	0.514	-0.038
141	2	12	V50,F1	0.399	0.528	-0.053
142	2	6	E52,E12	0.394	0.530	-0.046
143	2	6	E56,E51	0.382	0.537	-0.035
144	2	6	E57,E5	0.371	0.542	0.035
145	2	6	E28,E13	0.362	0.547	-0.032
146	2	6	E56,E52	0.354	0.552	0.040
147	2	12	V34,F1	0.340	0.560	-0.059
148	2	6	E28,E12	0.314	0.575	0.037
149	2	12	V52,F4	0.307	0.580	0.066
150	2	6	E58,E12	0.295	0.587	-0.041
151	2	6	E58,E29	0.288	0.591	0.030
152	2	6	E49,E12	0.283	0.595	0.040
153	2	12	V51,F4	0.268	0.605	0.053
154	2	6	E58,E50	0.238	0.626	0.029
155	2	6	E50,E8	0.225	0.635	-0.027
156	2	6	E56,E13	0.213	0.645	0.024
157	2	6	E49,E13	0.211	0.646	-0.028
158	2	6	E52,E27	0.200	0.655	-0.028
159	2	6	E30,E6	0.198	0.657	0.030
160	2	6	E13,E5	0.196	0.658	0.028
161	2	6	E34,E30	0.196	0.658	-0.026
162	2	12	V28,F1	0.190	0.663	-0.041
163	2	6	E51,E30	0.190	0.663	-0.025
164	2	6	E56,E12	0.189	0.664	-0.029
165	2	12	V51,F1	0.173	0.677	-0.038
166	2	6	E50,E34	0.171	0.679	0.021
167	2	6	E29,E8	0.165	0.685	0.024
168	2	6	E49,E14	0.163	0.686	0.031
169	2	6	E52,E5	0.162	0.688	0.029
170	2	6	E57,E13	0.159	0.690	-0.020
171	2	12	V29,F4	0.158	0.691	0.040
172	2	6	E27,E12	0.157	0.692	0.024
173	2	6	E36,E27	0.153	0.696	-0.020
174	2	6	E30,E5	0.150	0.699	0.026
175	2	12	V30,F1	0.139	0.709	-0.035
176	2	6	E49,E29	0.136	0.712	0.022
177	2	6	E57,E51	0.136	0.712	0.019
178	2	6	E34,E13	0.133	0.716	-0.022
179	2	6	E28,E14	0.132	0.716	0.024
180	2	6	E58,E8	0.131	0.717	-0.027
181	2	6	E51,E36	0.131	0.718	-0.019
182	2	6	E34,E6	0.129	0.720	-0.024
183	2	6	E50,E14	0.127	0.722	-0.020
184	2	6	E52,E7	0.126	0.722	0.025
185	2	12	V30,F4	0.125	0.723	-0.041
186	2	6	E49,E28	0.119	0.730	0.024
187	2	6	E12,E8	0.112	0.738	0.026
188	2	6	E49,E35	0.111	0.739	0.020
189	2	6	E35,E29	0.096	0.757	-0.014
190	2	6	E50,E13	0.081	0.776	-0.013
191	2	6	E56,E7	0.076	0.783	-0.017

192	2	6	E52,E8	0.074	0.786	-0.021
193	2	6	E36,E13	0.072	0.789	0.015
194	2	6	E29,E6	0.069	0.793	0.016
195	2	6	E56,E29	0.062	0.803	-0.012
196	2	6	E27,E7	0.061	0.806	0.015
197	2	6	E57,E8	0.060	0.806	-0.015
198	2	6	E14,E6	0.058	0.809	0.019
199	2	6	E34,E27	0.057	0.811	-0.013
200	2	6	E57,E30	0.056	0.813	-0.012
201	2	12	V49,F4	0.055	0.814	0.028
202	2	6	E34,E14	0.045	0.832	0.015
203	2	6	E58,E35	0.045	0.833	-0.014
204	2	6	E51,E12	0.044	0.834	0.013
205	2	6	E49,E34	0.044	0.834	-0.014
206	2	6	E30,E7	0.043	0.835	0.014
207	2	6	E29,E14	0.043	0.837	0.012
208	2	6	E51,E13	0.040	0.841	-0.010
209	2	6	E50,E7	0.039	0.844	-0.010
210	2	6	E29,E12	0.037	0.848	-0.011
211	2	6	E51,E50	0.035	0 853	-0.010
212	2	6	E34,E5	0.033	0 855	0.012
213	2	6	E28,E27	0.029	0 864	-0.010
214	2	6	E58,E6	0.027	0 870	0.012
215	2	6	E56,E30	0.024	0.876	0.009
216	2	6	E51,E35	0.023	0.879	-0.008
217	2	6	E49,E7	0.022	0 882	-0.011
218	2	6	E58,E5	0.021	0 884	-0.011
219	2	6	E35,E27	0.017	0 896	-0.007
220	2	6	E7,E6	0.013	0 910	-0.009
221	2	6	E35,E7	0.010	0.921	-0.006
222	2	6	E50,E12	0.007	0.935	0.005
223	2	6	E57,E14	0.006	0.939	0.005
224	2	6	E28,E5	0.005	0.942	-0.005
225	2	6	E58,E7	0.005	0.942	0.005
226	2	6	E52,E36	0.005	0.942	0.004
227	2	12	V27,F1	0.005	0.944	0.006
228	2	12	V49,F1	0.004	0.953	0.006
229	2	6	E51,E8	0.002	0.963	-0.003
230	2	6	E13,E12	0.000	0.983	-0.002
231	2	6	E52,E35	0.000	0.998	0.000
232	2	0	V27,F5	0.000	1.000	0.000
233	2	0	V49,F6	0.000	1.000	0.000
234	2	0	V34,F2	0.000	1.000	0.000
235	2	0	F6,D6	0.000	1.000	0.000
236	2	0	F5,D5	0.000	1.000	0.000
237	2	0	F3,D3	0.000	1.000	0.000
238	2	0	F2,D2	0.000	1.000	0.000
239	2	0	V12,F1	0.000	1.000	0.000
240	2	0	V5,F4	0.000	1.000	0.000
241	2	0	V56,F3	0.000	1.000	0.000

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MULTIVARIATE LAGRANGE MULTIPLIER TEST BY SIMULTANEOUS PROCESS IN STAGE 1

PARAMETER SETS (SUBMATRICES) ACTIVE AT THIS STAGE ARE:

PEE GVF

		CUMULATIVE MULTIVARIATE STATISTICS			UNIVARIATE INCREMENT	
STEP	PARAMETER	CHI-SQUARE	D.F.	PROBABILITY	CHI-SQUARE	PROBABILITY
1	E35,E34	26.019	1	0.000	26.019	0.000
2	E8,E6	39.091	2	0.000	13.072	0.000
3	E29,E27	49.744	3	0.000	10.653	0.001
4	V13,F4	59.298	4	0.000	9.554	0.002
5	E34,E28	66.582	5	0.000	7.284	0.007
6	E35,E28	75.494	6	0.000	8.912	0.003
7	E52,E49	82.602	7	0.000	7.108	0.008
8	E51,E49	92.315	8	0.000	9.714	0.002
9	E58,E13	98.216	9	0.000	5.901	0.015
10	E57,E6	103.736	10	0.000	5.520	0.019
11	E57,E28	109.856	11	0.000	6.120	0.013
12	E58,E56	116.587	12	0.000	6.731	0.009
13	E50,E6	121.997	13	0.000	5.409	0.020
14	E12,E7	126.875	14	0.000	4.878	0.027
15	E52,E34	131.648	15	0.000	4.773	0.029
16	V35,F1	135.898	16	0.000	4.250	0.039
17	E14,E8	139.797	17	0.000	3.899	0.048

1
Execution begins at 17:41:03.15
Execution ends at 17:41:07.48
Elapsed time = 4.33 seconds

V 49		0.013	-0.037	0.022	0.099	-0.019
COG2_3	V 50	0.067	-0.047	0.051	0.059	-0.054
COG3_3	V 51	-0.021	-0.029	-0.023	0.003	0.100
COG4_3	V 52	-0.035	-0.173	-0.021	-0.012	0.087
PAR1_3	V 56	-0.036	-0.054	-0.092	0.035	-0.154
PAR2_3	V 57	0.044	-0.087	-0.036	0.039	-0.082
PAR3_3	V 58	0.080	0.087	-0.010	0.069	-0.095

		COG2_3 V 50	COG3_3 V 51	COG4_3 V 52	PAR1_3 V 56	PAR2_3 V 57
COG2_3	V 50	0.031				
COG3_3	V 51	-0.016	-0.002			
COG4_3	V 52	-0.002	-0.045	0.005		
PAR1_3	V 56	0.056	-0.056	0.005	-0.007	
PAR2_3	V 57	0.127	0.017	0.110	-0.052	-0.006
PAR3_3	V 58	0.061	-0.047	-0.030	0.103	-0.014

		PAR3_3 V 58
PAR3_3	V 58	0.019

AVERAGE ABSOLUTE STANDARDIZED RESIDUALS = 0.0503
AVERAGE OFF-DIAGONAL ABSOLUTE STANDARDIZED RESIDUALS = 0.0543

LARGEST STANDARDIZED RESIDUALS:

V 56,V 8	V 57,V 28	V 57,V 6	V 52,V 34	V 58,V 28
-0.190	0.187	0.180	-0.173	0.166

V 36,V 28	V 36,V 5	V 58,V 14	V 56,V 49	V 36,V 12
0.163	0.159	0.154	-0.154	0.150

V 14,V 8	V 8,V 6	V 58,V 13	V 50,V 8	V 36,V 14
-0.147	0.146	-0.134	-0.134	0.131

V 57,V 50	V 34,V 8	V 58,V 8	V 34,V 28	V 12,V 7
0.127	-0.125	-0.125	-0.122	-0.116

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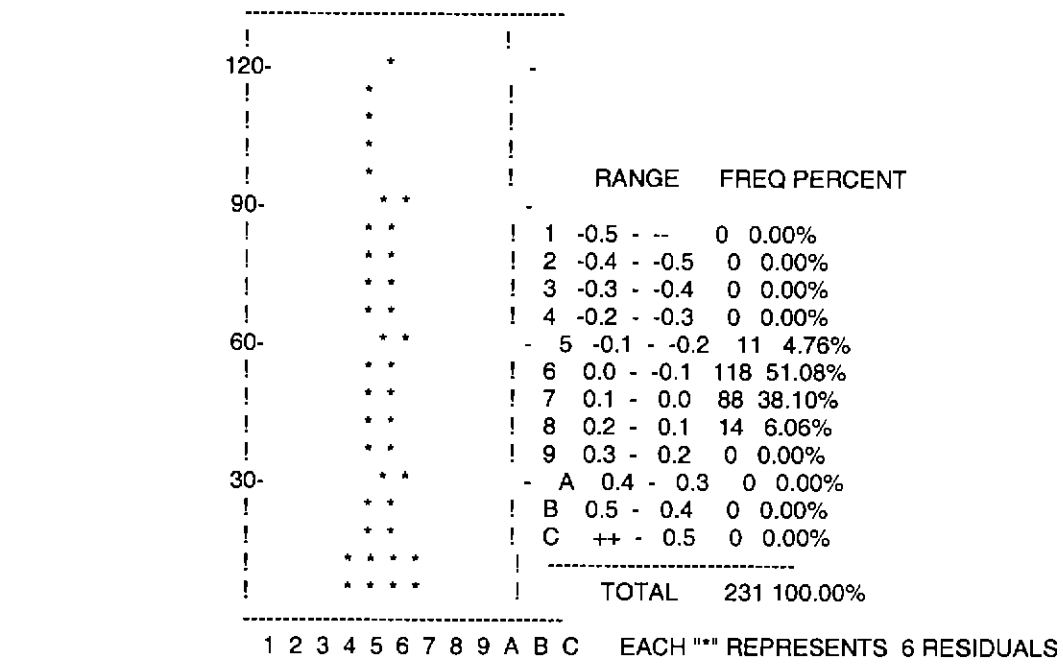
DISTRIBUTION OF STANDARDIZED RESIDUALS



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GOODNESS OF FIT SUMMARY

INDEPENDENCE MODEL CHI-SQUARE = 1560.622 ON 210 DEGREES OF FREEDOM

INDEPENDENCE AIC = 1140.62227 INDEPENDENCE CAIC = 305.50818
MODEL AIC = -47.57669 MODEL CAIC = -691.80756

CHI-SQUARE = 276.423 BASED ON 162 DEGREES OF FREEDOM
PROBABILITY VALUE FOR THE CHI-SQUARE STATISTIC IS LESS THAN 0.001
THE NORMAL THEORY RLS CHI-SQUARE FOR THIS ML SOLUTION IS 272.185.

SATORRA-BENTLER SCALED CHI-SQUARE = 206.4336
PROBABILITY VALUE FOR THE CHI-SQUARE STATISTIC IS 0.01046

BENTLER-BONETT NORMED FIT INDEX= 0.823
BENTLER-BONETT NONNORMED FIT INDEX= 0.890
COMPARATIVE FIT INDEX (CFI) = 0.915
ROBUST COMPARATIVE FIT INDEX = 0.949
BOLLEN (IFI) FIT INDEX= 0.918
McDonald (MFI) FIT INDEX= 0.674
LISREL GFI FIT INDEX= 0.847
LISREL AGFI FIT INDEX= 0.782
ROOT MEAN SQUARED RESIDUAL (RMR) = 0.086
STANDARDIZED RMR = 0.065
ROOT MEAN SQ. ERROR OF APP.(RMSEA)= 0.070
90% CONFIDENCE INTERVAL OF RMSEA (0.055, 0.084)

ITERATIVE SUMMARY

ITERATION	PARAMETER ABS CHANGE	ALPHA	FUNCTION
1	0.474802	1.00000	4.82458
2	0.202701	1.00000	2.93149
3	0.078345	1.00000	2.12832
4	0.032608	1.00000	1.93091
5	0.008648	1.00000	1.92321
6	0.004802	1.00000	1.92116
7	0.002633	1.00000	1.92030
8	0.001824	1.00000	1.91990
9	0.001098	1.00000	1.91970
10	0.000785	1.00000	1.91961

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MEASUREMENT EQUATIONS WITH STANDARD ERRORS AND TEST STATISTICS
(ROBUST STATISTICS IN PARENTHESES)

COG1_1 =V5 = 1.000 F4 +1.000 E5

COG2_1 =V6 = .912*F4 +1.000 E6
.133
6.867
(.135)
(6.768)

COG3_1 =V7 = .991*F4 +1.000 E7
.133
7.458
(.116)
(8.573)

COG4_1 =V8 = .855*F4 +1.000 E8
.130
6.575
(.127)
(6.711)

PAR1_1 =V12 = 1.000 F1 +1.000 E12

PAR2_1 =V13 = .996*F1 +1.000 E13
.114
8.763
(.138)
(7.208)

PAR3_1 =V14 = 1.006*F1 +1.000 E14
.125
8.031
(.158)
(6.360)

COG1_2 =V27 = 1.000 F5 +1.000 E27

COG2_2 =V28 = 1.006*F5 +1.000 E28
.117
8.580
(.160)
(6.299)

COG3_2 =V29 = .914*F5 +1.000 E29
.101

9.056
(.117)
(7.814)

COG4_2 =V30 = 1.044*F5 +1.000 E30
116
8 960
(172)
(6.074)

PAR1_2 =V34 = 1.000 F2 +1.000 E34

MEASUREMENT EQUATIONS WITH STANDARD ERRORS AND TEST STATISTICS (CONTINUED)

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(ROBUST STATISTICS IN PARENTHESES)

PAR2_2 =V35 = .978*F2 +1.000 E35
.114
8.600
(.115)
(8.468)

PAR3_2 =V36 = .993*F2 +1.000 E36
.116
8.552
(.155)
(6.395)

COG1_3 =V49 = 1.000 F6 +1.000 E49

COG2_3 =V50 = .976*F6 +1.000 E50
.129
7.547
(.152)
(6.415)

COG3_3 =V51 = .880*F6 +1.000 E51
.128
6.895
(.138)
(6.360)

COG4_3 =V52 = 1.040*F6 +1.000 E52
.150
6.936
(.156)
(6.653)

PAR1_3 =V56 = 1.000 F3 +1.000 E56

PAR2_3 =V57 = 1.016*F3 +1.000 E57
.136
7.449
(.170)
(5.987)

PAR3_3 =V58 = 1.029*F3 +1.000 E58
.156
6.593
(.232)
(4.435)

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CONSTRUCT EQUATIONS WITH STANDARD ERRORS AND TEST STATISTICS
(ROBUST STATISTICS IN PARENTHESES)

F2 =F2 = .457*F1 - .136*F4 +1.000 D2
.167 .177
2.727 -.773
(.144) (.150)
(3.164) (-.912)

F3 =F3 = .548*F2 - .076*F5 + .108*F1 +1.000 D3
.145 .129 .076
3.771 -.595 1.419
(.198) (.166) (.082)
(2.772) (-.461) (1.321)

F5 =F5 = -.002*F1 + .490*F4 +1.000 D5
.151 .175
-.014 2.795
(.151) (.171)
(-.014) (2.865)

F6 =F6 = .284*F2 + .352*F5 + .127*F4 +1.000 D6
.136 .151 .091
2.080 2.330 1.398
(.134) (.167) (.084)
(2.112) (2.114) (1.522)

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VARIANCES OF INDEPENDENT VARIABLES

V	F	
F1	F1	.869*
		.183
		4.752
		(.201)
		(4.319)
F4	F4	.741*
		.165
		4.500
		(.151)
		(4.897)

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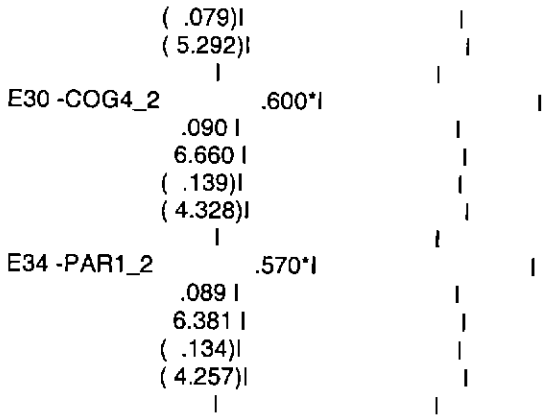
VARIANCES OF INDEPENDENT VARIABLES

	E		D	
	---		---	
E5 -COG1_1	.716*	D2 - F2	.606*	
	.110		.129	
	6.527		4.702	
	(.127)		(.167)	
	(5.640)		(3.634)	
E6 -COG2_1	.815*	D3 - F3	.315*	
	.115		.083	
	7.069		3.811	
	(.132)		(.116)	
	(6.186)		(2.714)	
E7 -COG3_1	.657*	D5 - F5	.559*	
	.103		.114	
	6.390		4.890	
	(.112)		(.139)	
	(5.839)		(4.025)	
E8 -COG4_1	.864*	D6 - F6	.335*	
	.118		.088	
	7.319		3.798	
	(.141)		(.109)	
	(6.107)		(3.075)	
E12 -PAR1_1	.748*			
	.114			
	6.589			
	(.155)			
	(4.814)			
E13 -PAR2_1	.396*			
	.080			
	4.944			
	(.092)			
	(4.312)			
E14 -PAR3_1	.732*			
	.113			
	6.476			
	(.122)			
	(5.981)			
E27 -COG1_2	.519*			
	.079			
	6.538			
	(.096)			
	(5.405)			
E28 -COG2_2	.701*			
	.099			
	7.064			
	(.129)			
	(5.439)			
E29 -COG3_2	.420*			
	.065			
	6.449			


(.079)
(5.292)
E30 -COG4_2 .600*
.090
6.660
(.139)
(4.328)
E34 -PAR1_2 .570*
.089
6.381
(.134)
(4.257)

TITLE: PARCOG1 FULL CROSS INFLUENTIAL MODEL 01/25/03 PAGE : 15
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VARIANCES OF INDEPENDENT VARIABLES (CONTINUED)

E35 -PAR2_2	.472*	
	.078	
	6.077	
	(.104)	
	(4.535)	
E36 -PAR3_2	.485*	
	.080	
	6.026	
	(.107)	
	(4.531)	
E49 -COG1_3	.861*	
	.117	
	7.372	
	(.134)	
	(6.430)	
E50 -COG2_3	.470*	
	.072	
	6.527	
	(.117)	
	(4.020)	
E51 -COG3_3	.569*	
	.080	
	7.157	
	(.114)	
	(4.991)	
E52 -COG4_3	.785*	
	.110	
	7.155	
	(.134)	
	(5.879)	
E56 -PAR1_3	.567*	
	.085	
	6.641	
	(.140)	
	(4.048)	
E57 -PAR2_3	.461*	
	.075	
	6.128	
	(.096)	
	(4.777)	
E58 -PAR3_3	.856*	
	.118	
	7.233	
	(.184)	
	(4.650)	

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COVARIANCES AMONG INDEPENDENT VARIABLES

V		F	
	F4 - F4		.587*
	F1 - F1		.117
			5.016
			(.110)
			(5.357)

TITLE: PARCOG1 FULL CROSS INFLUENTIAL MODEL 01/25/03 PAGE : 17
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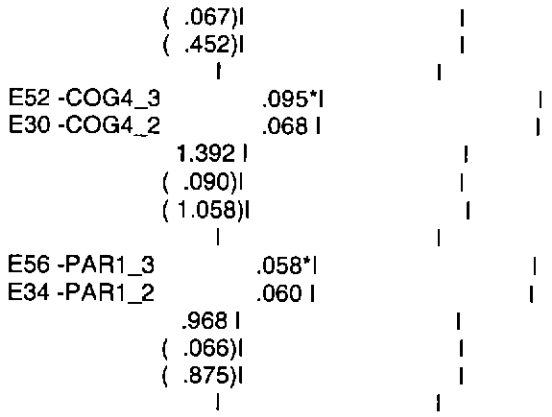
COVARIANCES AMONG INDEPENDENT VARIABLES

	E		D	
	---		---	
E27 -COG1_2	.067*I	D5 - F5		.440*I
E5 -COG1_1	.063 I	D2 - F2		.085 I
	1.074 I		5.195 I	
	(.068)I		(.094)I	
	(.987)I		(4.703)I	
	I		I	
E28 -COG2_2	.091*I	D6 - F6		.284*I
E6 -COG2_1	.069 I	D3 - F3		.062 I
	1.313 I		4.610 I	
	(.075)I		(.087)I	
	(1.209)I		(3.265)I	
	I		I	
E29 -COG3_2	.070*I			I
E7 -COG3_1	.056 I			I
	1.259 I		I	
	(.062)I		I	
	(1.129)I		I	
	I		I	
E30 -COG4_2	.149*I			I
E8 -COG4_1	.071 I			I
	2.088 I		I	
	(.071)I		I	
	(2.111)I		I	
	I		I	
E34 -PAR1_2	.081*I			I
E12 -PAR1_1	.068 I			I
	1.198 I		I	
	(.069)I		I	
	(1.185)I		I	
	I		I	
E35 -PAR2_2	.074*I			I
E13 -PAR2_1	.051 I			I
	1.443 I		I	
	(.062)I		I	
	(1.177)I		I	
	I		I	
E36 -PAR3_2	-.040*I			I
E14 -PAR3_1	.061 I			I
	-.657 I		I	
	(.062)I		I	
	(-.653)I		I	
	I		I	
E49 -COG1_3	.129*I			I
E27 -COG1_2	.067 I			I
	1.918 I		I	
	(.071)I		I	
	(1.814)I		I	
	I		I	
E50 -COG2_3	.224*I			I
E28 -COG2_2	.062 I			I
	3.628 I		I	
	(.090)I		I	
	(2.484)I		I	
	I		I	
E51 -COG3_3	.030*I			I
E29 -COG3_2	.049 I			I
	.618 I		I	


(.067)
(.452)
E52 -COG4_3 .095*
E30 -COG4_2 .068
1.392
(.090)
(1.058)
E56 -PAR1_3 .058*
E34 -PAR1_2 .060
.968
(.066)
(.875)

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COVARIANCES AMONG INDEPENDENT VARIABLES (CONTINUED)

E57 -PAR2_3		.097*			
E35 -PAR2_2		.052			
	1.863				
	(.075)				
	(1.294)				
E58 -PAR3_3		.180*			
E36 -PAR3_2		.069			
	2.608				
	(.080)				
	(2.255)				

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STANDARDIZED SOLUTION: R-SQUARED

COG1_1 =V5 =	.713 F4 + .701 E5	.509
COG2_1 =V6 =	.656*F4 + .755 E6	.430
COG3_1 =V7 =	.725*F4 + .689 E7	.526
COG4_1 =V8 =	.621*F4 + .784 E8	.385
PAR1_1 =V12 =	.733 F1 + .680 E12	.537
PAR2_1 =V13 =	.828*F1 + .561 E13	.685
PAR3_1 =V14 =	.739*F1 + .674 E14	.546
COG1_2 =V27 =	.766 F5 + .643 E27	.586
COG2_2 =V28 =	.718*F5 + .696 E28	.515
COG3_2 =V29 =	.770*F5 + .637 E29	.594
COG4_2 =V30 =	.756*F5 + .654 E30	.572
PAR1_2 =V34 =	.749 F2 + .663 E34	.561
PAR2_2 =V35 =	.772*F2 + .636 E35	.596
PAR3_2 =V36 =	.772*F2 + .635 E36	.597
COG1_3 =V49 =	.655 F6 + .756 E49	.429
COG2_3 =V50 =	.753*F6 + .658 E50	.567
COG3_3 =V51 =	.684*F6 + .730 E51	.468
COG4_3 =V52 =	.686*F6 + .728 E52	.471
PAR1_3 =V56 =	.698 F3 + .717 E56	.487
PAR2_3 =V57 =	.739*F3 + .673 E57	.547
PAR3_3 =V58 =	.632*F3 + .775 E58	.400
F2 =F2 =	.499*F1 - .138*F4 + .912 D2	.167
F3 =F3 =	.638*F2 - .089*F5 + .138*F1 + .766 D3	.414
F5 =F5 =	-.002*F1 + .492*F4 + .872 D5	.240
F6 =F6 =	.301*F2 + .376*F5 + .136*F4 + .721 D6	.481

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CORRELATIONS AMONG INDEPENDENT VARIABLES

V		F	
	F4	F4	.731*I
	F1	F1	I
	I		I

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CORRELATIONS AMONG INDEPENDENT VARIABLES

E		D	
E27 -COG1_2	.110*	D5 - F5	.756*
E5 -COG1_1		D2 - F2	
E28 -COG2_2	.120*	D6 - F6	.874*
E6 -COG2_1		D3 - F3	
E29 -COG3_2	.134*		
E7 -COG3_1			
E30 -COG4_2	.207*		
E8 -COG4_1			
E34 -PAR1_2	.124*		
E12 -PAR1_1			
E35 -PAR2_2	.170*		
E13 -PAR2_1			
E36 -PAR3_2	-.068*		
E14 -PAR3_1			
E49 -COG1_3	.193*		
E27 -COG1_2			
E50 -COG2_3	.389*		
E28 -COG2_2			
E51 -COG3_3	.062*		
E29 -COG3_2			
E52 -COG4_3	.138*		
E30 -COG4_2			
E56 -PAR1_3	.101*		
E34 -PAR1_2			
E57 -PAR2_3	.208*		
E35 -PAR2_2			
E58 -PAR3_3	.280*		
E36 -PAR3_2			

END OF METHOD

TITLE: PARCOG1 FULL CROSS INFLUENTIAL MODEL 01/25/03 PAGE : 22
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LAGRANGIAN MULTIPLIER TEST REQUIRES 122948 WORDS OF MEMORY. 0.000 8.912 0.003

7	E52,E49	82.602	7	0.000	7.108	0.008
8	E51,E49	92.315	8	0.000	9.714	0.002
9	E58,E13	98.216	9	0.000	5.901	0.015
10	E57,E6	103.736	10	0.000	5.520	0.019
11	E57,E28	109.856	11	0.000	6.120	0.013
12	E58,E56	116.587	12	0.000	6.731	0.009
13	E50,E6	121.997	13	0.000	5.409	0.020
14	E12,E7	126.875	14	0.000	4.878	0.027
15	E52,E34	131.648	15	0.000	4.773	0.029
16	V35,F1	135.898	16	0.000	4.250	0.039
17	E14,E8	139.797	17	0.000	3.899	0.048

1
Execution begins at 17:41:03.15
Execution ends at 17:41:07.48
Elapsed time = 4.33 seconds V 49 0.013 -0.037 0.022 0.099 -0.019

COG2_3	V 50	0.067	-0.047	0.051	0.059	-0.054
COG3_3	V 51	-0.021	-0.029	-0.023	0.003	0.100
COG4_3	V 52	-0.035	-0.173	-0.021	-0.012	0.087
PAR1_3	V 56	-0.036	-0.054	-0.092	0.035	-0.154
PAR2_3	V 57	0.044	-0.087	-0.036	0.039	-0.082
PAR3_3	V 58	0.080	0.087	-0.010	0.069	-0.095

		COG2_3 V 50	COG3_3 V 51	COG4_3 V 52	PAR1_3 V 56	PAR2_3 V 57
COG2_3	V 50	0.031				
COG3_3	V 51	-0.016	-0.002			
COG4_3	V 52	-0.002	-0.045	0.005		
PAR1_3	V 56	0.056	-0.056	0.005	-0.007	
PAR2_3	V 57	0.127	0.017	0.110	-0.052	-0.006
PAR3_3	V 58	0.061	-0.047	-0.030	0.103	-0.014

		PAR3_3 V 58
PAR3_3	V 58	0.019

AVERAGE ABSOLUTE STANDARDIZED RESIDUALS = 0.0503
AVERAGE OFF-DIAGONAL ABSOLUTE STANDARDIZED RESIDUALS = 0.0543

LARGEST STANDARDIZED RESIDUALS:

V 56,V 8	V 57,V 28	V 57,V 6	V 52,V 34	V 58,V 28
-0.190	0.187	0.180	-0.173	0.166

V 36,V 28	V 36,V 5	V 58,V 14	V 56,V 49	V 36,V 12
0.163	0.159	0.154	-0.154	0.150

V 14,V 8	V 8,V 6	V 58,V 13	V 50,V 8	V 36,V 14
-0.147	0.146	-0.134	-0.134	0.131

V 57,V 50	V 34,V 8	V 58,V 8	V 34,V 28	V 12,V 7
0.127	-0.125	-0.125	-0.122	-0.116

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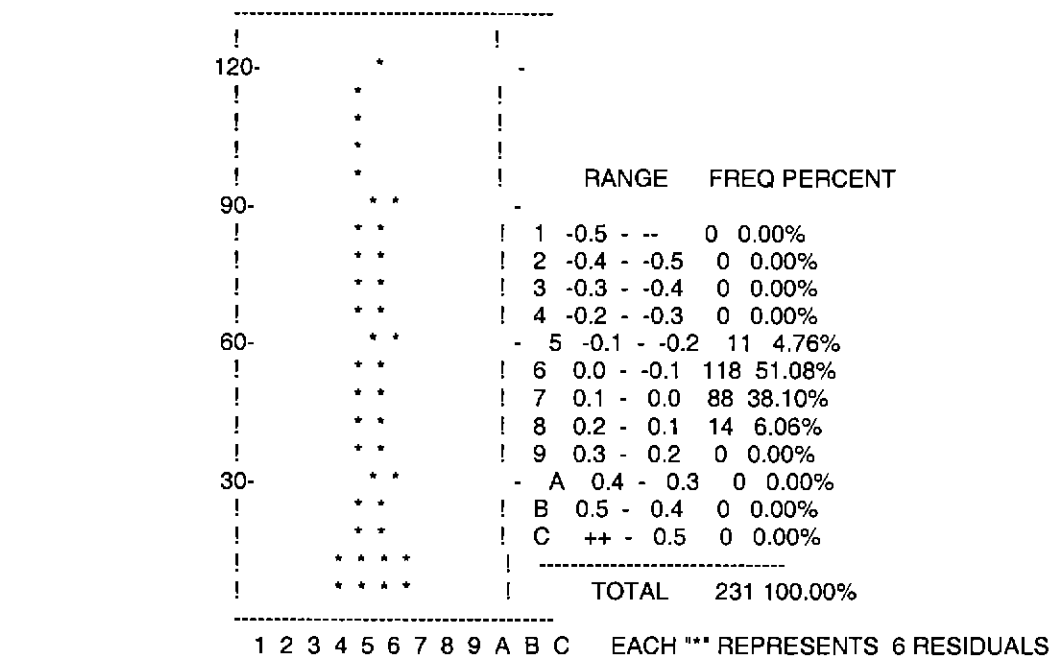
DISTRIBUTION OF STANDARDIZED RESIDUALS



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GOODNESS OF FIT SUMMARY

INDEPENDENCE MODEL CHI-SQUARE = 1560.622 ON 210 DEGREES OF FREEDOM

INDEPENDENCE AIC = 1140.62227 INDEPENDENCE CAIC = 305.50818
MODEL AIC = -47.57669 MODEL CAIC = -691.80756

CHI-SQUARE = 276.423 BASED ON 162 DEGREES OF FREEDOM
PROBABILITY VALUE FOR THE CHI-SQUARE STATISTIC IS LESS THAN 0.001
THE NORMAL THEORY RLS CHI-SQUARE FOR THIS ML SOLUTION IS 272.185.

SATORRA-BENTLER SCALED CHI-SQUARE = 206.4336
PROBABILITY VALUE FOR THE CHI-SQUARE STATISTIC IS 0.01046

BENTLER-BONETT NORMED	FIT INDEX=	0.823
BENTLER-BONETT NONNORMED	FIT INDEX=	0.890
COMPARATIVE FIT INDEX (CFI)	=	0.915
ROBUST COMPARATIVE FIT INDEX	=	0.949
BOLLEN (IFI)	FIT INDEX=	0.918
McDonald (MFI)	FIT INDEX=	0.674
LISREL GFI	FIT INDEX=	0.847
LISREL AGFI	FIT INDEX=	0.782
ROOT MEAN SQUARED RESIDUAL (RMR)	=	0.086
STANDARDIZED RMR	=	0.065
ROOT MEAN SQ. ERROR OF APP.(RMSEA)	=	0.070
90% CONFIDENCE INTERVAL OF RMSEA (	0.055,	0.084)

ITERATIVE SUMMARY

ITERATION	PARAMETER ABS CHANGE	ALPHA	FUNCTION
1	0.474802	1.00000	4.82458
2	0.202701	1.00000	2.93149
3	0.078345	1.00000	2.12832
4	0.032608	1.00000	1.93091
5	0.008648	1.00000	1.92321
6	0.004802	1.00000	1.92116
7	0.002633	1.00000	1.92030
8	0.001824	1.00000	1.91990
9	0.001098	1.00000	1.91970
10	0.000785	1.00000	1.91961

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MEASUREMENT EQUATIONS WITH STANDARD ERRORS AND TEST STATISTICS
(ROBUST STATISTICS IN PARENTHESES)

COG1_1 =V5 = 1.000 F4 +1.000 E5

COG2_1 =V6 = .912*F4 +1.000 E6
.133
6.867
(.135)
(6.768)

COG3_1 =V7 = .991*F4 +1.000 E7
.133
7.458
(.116)
(8.573)

COG4_1 =V8 = .855*F4 +1.000 E8
.130
6.575
(.127)
(6.711)

PAR1_1 =V12 = 1.000 F1 +1.000 E12

PAR2_1 =V13 = .996*F1 +1.000 E13
.114
8.763
(.138)
(7.208)

PAR3_1 =V14 = 1.006*F1 +1.000 E14
.125
8.031
(.158)
(6.360)

COG1_2 =V27 = 1.000 F5 +1.000 E27

COG2_2 =V28 = 1.006*F5 +1.000 E28
.117
8.580
(.160)
(6.299)

COG3_2 =V29 = .914*F5 +1.000 E29
.101

9.056
(.117)
(7.814)

COG4_2 =V30 = 1.044*F5 +1.000 E30
.116
8.960
(.172)
(6.074)

PAR1_2 =V34 = 1.000 F2 +1.000 E34

MEASUREMENT EQUATIONS WITH STANDARD ERRORS AND TEST STATISTICS (CONTINUED)

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MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)
(ROBUST STATISTICS IN PARENTHESES)

PAR2_2 =V35 = .978*F2 +1.000 E35
.114
8.600
(.115)
(8.468)

PAR3_2 =V36 = .993*F2 +1.000 E36
.116
8.552
(.155)
(6.395)

COG1_3 =V49 = 1.000 F6 +1.000 E49

COG2_3 =V50 = .976*F6 +1.000 E50
.129
7.547
(.152)
(6.415)

COG3_3 =V51 = .880*F6 +1.000 E51
.128
6.895
(.138)
(6.360)

COG4_3 =V52 = 1.040*F6 +1.000 E52
.150
6.936
(.156)
(6.653)

PAR1_3 =V56 = 1.000 F3 +1.000 E56

PAR2_3 =V57 = 1.016*F3 +1.000 E57
.136
7.449
(.170)
(5.987)

PAR3_3 =V58 = 1.029*F3 +1.000 E58
.156
6.593
(.232)
(4.435)

TITLE: PARCOG1 FULL CROSS INFLUENTIAL MODEL 01/25/03 PAGE : 12
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MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

CONSTRUCT EQUATIONS WITH STANDARD ERRORS AND TEST STATISTICS
(ROBUST STATISTICS IN PARENTHESES)

F2 =F2 = .457*F1 - .136*F4 +1.000 D2
.167 .177
2.727 -.773
(.144) (.150)
(3.164) (-.912)

F3 =F3 = .548*F2 - .076*F5 + .108*F1 +1.000 D3
.145 .129 .076
3.771 -.595 1.419
(.198) (.166) (.082)
(2.772) (-.461) (1.321)

F5 =F5 = -.002*F1 + .490*F4 +1.000 D5
.151 .175
-.014 2.795
(.151) (.171)
(-.014) (2.865)

F6 =F6 = .284*F2 + .352*F5 + .127*F4 +1.000 D6
.136 .151 .091
2.080 2.330 1.398
(.134) (.167) (.084)
(2.112) (2.114) (1.522)

TITLE: PARCOG1 FULL CROSS INFLUENTIAL MODEL 01/25/03 PAGE : 13
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MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

VARIANCES OF INDEPENDENT VARIABLES

V	F	
F1 - F1		.869*
		.183
		4.752
		(.201)
		(4.319)
F4 - F4		.741*
		.165
		4.500
		(.151)
		(4.897)

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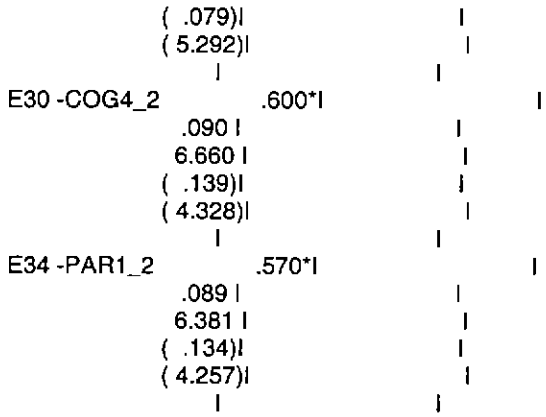
VARIANCES OF INDEPENDENT VARIABLES

E		D	
E5 -COG1_1	.716*I	D2 - F2	.606*I
	.110 I		.129 I
	6.527 I		4.702 I
	(.127)I		(.167)I
	(5.640)I		(3.634)I
E6 -COG2_1	.815*I	D3 - F3	.315*I
	.115 I		.083 I
	7.069 I		3.811 I
	(.132)I		(.116)I
	(6.186)I		(2.714)I
E7 -COG3_1	.657*I	D5 - F5	.559*I
	.103 I		.114 I
	6.390 I		4.890 I
	(.112)I		(.139)I
	(5.839)I		(4.025)I
E8 -COG4_1	.864*I	D6 - F6	.335*I
	.118 I		088 I
	7.319 I		3 798 I
	(.141)I		(109)I
	(6.107)I		(3 075)I
E12 -PAR1_1	.748*I		I
	.114 I		I
	6.589 I		I
	(.155)I		I
	(4.814)I		I
E13 -PAR2_1	.396*I		I
	.080 I		I
	4.944 I		I
	(.092)I		I
	(4.312)I		I
E14 -PAR3_1	.732*I		I
	.113 I		I
	6.476 I		I
	(.122)I		I
	(5.981)I		I
E27 -COG1_2	.519*I		I
	.079 I		I
	6.538 I		I
	(.096)I		I
	(5.405)I		I
E28 -COG2_2	.701*I		I
	.099 I		I
	7.064 I		I
	(.129)I		I
	(5.439)I		I
E29 -COG3_2	.420*I		I
	.065 I		I
	6.449 I		I


(.079)
(5.292)
E30 -COG4_2
.600*
.090
6.660
(.139)
(4.328)
E34 -PAR1_2
.570*
.089
6.381
(.134)
(4.257)

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VARIANCES OF INDEPENDENT VARIABLES (CONTINUED)

E35 -PAR2_2	.472*	
	.078	
	6.077	
	(.104)	
	(4.535)	
E36 -PAR3_2	.485*	
	.080	
	6.026	
	(.107)	
	(4.531)	
E49 -COG1_3	.861*	
	.117	
	7.372	
	(.134)	
	(6.430)	
E50 -COG2_3	.470*	
	.072	
	6.527	
	(.117)	
	(4.020)	
E51 -COG3_3	.569*	
	.080	
	7.157	
	(.114)	
	(4.991)	
E52 -COG4_3	.785*	
	.110	
	7.155	
	(.134)	
	(5.879)	
E56 -PAR1_3	.567*	
	.085	
	6.641	
	(.140)	
	(4.048)	
E57 -PAR2_3	.461*	
	.075	
	6.128	
	(.096)	
	(4.777)	
E58 -PAR3_3	.856*	
	.118	
	7.233	
	(.184)	
	(4.650)	

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COVARIANCES AMONG INDEPENDENT VARIABLES

V		F	
	F4	F4	.587*
			5.016
	F1	F1	.117
			(.110)
			(5.357)

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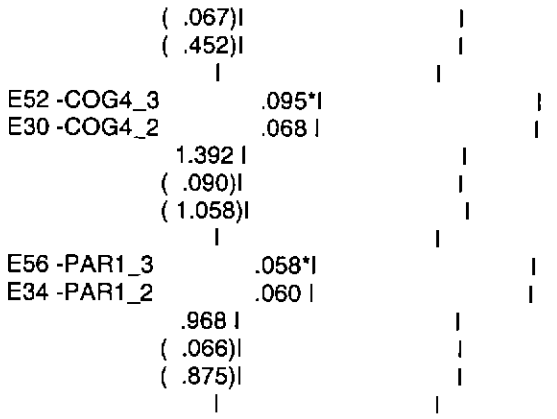
MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

COVARIANCES AMONG INDEPENDENT VARIABLES

	E		D	
	---		---	
E27 -COG1_2	.067*	D5 - F5		.440*
E5 -COG1_1	.063	D2 - F2		.085
	1.074		5.195	
	(.068)		(.094)	
	(.987)		(4.703)	
E28 -COG2_2	.091*	D6 - F6		.284*
E6 -COG2_1	.069	D3 - F3		.062
	1.313		4.610	
	(.075)		(.087)	
	(1.209)		(3.265)	
E29 -COG3_2	.070*			
E7 -COG3_1	.056			
	1.259			
	(.062)			
	(1.129)			
E30 -COG4_2	.149*			
E8 -COG4_1	.071			
	2.088			
	(.071)			
	(2.111)			
E34 -PAR1_2	.081*			
E12 -PAR1_1	.068			
	1.198			
	(.069)			
	(1.185)			
E35 -PAR2_2	.074*			
E13 -PAR2_1	.051			
	1.443			
	(.062)			
	(1.177)			
E36 -PAR3_2	-.040*			
E14 -PAR3_1	.061			
	-.657			
	(.062)			
	(-.653)			
E49 -COG1_3	.129*			
E27 -COG1_2	.067			
	1.918			
	(.071)			
	(1.814)			
E50 -COG2_3	.224*			
E28 -COG2_2	.062			
	3.628			
	(.090)			
	(2.484)			
E51 -COG3_3	.030*			
E29 -COG3_2	.049			
	.618			


(.067)
(.452)
E52 -COG4_3 .095*
E30 -COG4_2 .068
1.392
(.090)
(1.058)
E56 -PAR1_3 .058*
E34 -PAR1_2 .060
.968
(.066)
(.875)

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COVARIANCES AMONG INDEPENDENT VARIABLES (CONTINUED)

E57 -PAR2_3	.097*			
E35 -PAR2_2	.052			
	1.863			
	(.075)			
	(1.294)			
E58 -PAR3_3	.180*			
E36 -PAR2_2	.069			
	2.608			
	(.080)			
	(2.255)			

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MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

STANDARDIZED SOLUTION: R-SQUARED

COG1_1	=V5 =	.713 F4	+ .701 E5			.509	
COG2_1	=V6 =	.656*F4	+ .755 E6			.430	
COG3_1	=V7 =	.725*F4	+ .689 E7			.526	
COG4_1	=V8 =	.621*F4	+ .784 E8			.385	
PAR1_1	=V12 =	.733 F1	+ .680 E12			.537	
PAR2_1	=V13 =	.828*F1	+ .561 E13				.685
PAR3_1	=V14 =	.739*F1	+ .674 E14				.546
COG1_2	=V27 =	.766 F5	+ .643 E27				.586
COG2_2	=V28 =	.718*F5	+ .696 E28				.515
COG3_2	=V29 =	.770*F5	+ .637 E29				.594
COG4_2	=V30 =	.756*F5	+ .654 E30				.572
PAR1_2	=V34 =	.749 F2	+ .663 E34			.561	
PAR2_2	=V35 =	.772*F2	+ .636 E35			.	596
PAR3_2	=V36 =	.772*F2	+ .635 E36				.597
COG1_3	=V49 =	.655 F6	+ .756 E49				.429
COG2_3	=V50 =	.753*F6	+ .658 E50				.567
COG3_3	=V51 =	.684*F6	+ .730 E51				.468
COG4_3	=V52 =	.686*F6	+ .728 E52				.471
PAR1_3	=V56 =	.698 F3	+ .717 E56			.487	
PAR2_3	=V57 =	.739*F3	+ .673 E57				.547
PAR3_3	=V58 =	.632*F3	+ .775 E58				.400
F2	=F2 =	.499*F1	- .138*F4	+ .912 D2		.167	
F3	=F3 =	.638*F2	- .089*F5	+ .138*F1	+ .766 D3		.414
F5	=F5 =	-.002*F1	+ .492*F4	+ .872 D5			.240
F6	=F6 =	.301*F2	+ .376*F5	+ .136*F4	+ .721 D6		.481

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MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

CORRELATIONS AMONG INDEPENDENT VARIABLES

V		F	
	F4	F4	.731*I
	F1	F1	I
	I		I

TITLE: PARCOG1 FULL CROSS INFLUENTIAL MODEL 01/25/03 PAGE : 21
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MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

CORRELATIONS AMONG INDEPENDENT VARIABLES

E		D	
E27 -COG1_2	.110*	D5 - F5	.756*
E5 -COG1_1		D2 - F2	
E28 -COG2_2	.120*	D6 - F6	.874*
E6 -COG2_1		D3 - F3	
E29 -COG3_2	.134*		
E7 -COG3_1			
E30 -COG4_2	.207*		
E8 -COG4_1			
E34 -PAR1_2	.124*		
E12 -PAR1_1			
E35 -PAR2_2	.170*		
E13 -PAR2_1			
E36 -PAR3_2	-.068*		
E14 -PAR3_1			
E49 -COG1_3	.193*		
E27 -COG1_2			
E50 -COG2_3	.389*		
E28 -COG2_2			
E51 -COG3_3	.062*		
E29 -COG3_2			
E52 -COG4_3	.138*		
E30 -COG4_2			
E56 -PAR1_3	.101*		
E34 -PAR1_2			
E57 -PAR2_3	.208*		
E35 -PAR2_2			
E58 -PAR3_3	.280*		
E36 -PAR3_2			

END OF METHOD

TITLE: PARCOG1 FULL CROSS INFLUENTIAL MODEL 01/25/03 PAGE : 22
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MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

LAGRANGIAN MULTIPLIER TEST REQUIRES 122948 WORDS OF MEMORY.
PROGRAM ALLOCATES 10000000 WORDS.

LAGRANGE MULTIPLIER TEST (FOR ADDING PARAMETERS)

ORDERED UNIVARIATE TEST STATISTICS:

NO	CODE		PARAMETER	CHI-SQUARE	PROBABILITY	PARAMETER CHANGE
1	2	6	E35,E34	26.019	0.000	0.350
2	2	6	E8,E6	13.090	0.000	0.304
3	2	6	E36,E28	11.727	0.001	0.185
4	2	6	E29,E27	10.647	0.001	0.180
5	2	12	V13,F4	9.395	0.002	0.564
6	2	12	V36,F1	9.342	0.002	0.289
7	2	6	E34,E28	8.183	0.004	-0.166
8	2	6	E36,E35	7.769	0.005	-0.189
9	2	6	E6,E5	7.666	0.006	-0.238
10	2	12	V36,F4	7.192	0.007	0.252
11	2	6	E52,E49	7.107	0.008	0.214
12	2	6	E58,E14	7.032	0.008	0.208
13	2	6	E58,E13	6.621	0.010	-0.157
14	2	12	V35,F1	6.590	0.010	-0.249
15	2	6	E51,E49	6.516	0.011	0.176
16	2	6	E36,E12	6.367	0.012	0.166
17	2	6	E14,E8	6.366	0.012	-0.195
18	2	6	E57,E28	6.163	0.013	0.132
19	2	6	E57,E34	6.022	0.014	-0.140
20	2	6	E30,E28	5.908	0.015	0.156
21	2	6	E12,E7	5.465	0.019	-0.170
22	2	6	E52,E34	5.407	0.020	-0.152
23	2	12	V57,F4	5.377	0.020	0.221
24	2	6	E56,E49	5.335	0.021	-0.157
25	2	6	E58,E56	5.220	0.022	0.168
26	2	6	E13,E7	5.093	0.024	0.136
27	2	6	E57,E6	4.661	0.031	0.129
28	2	6	E35,E28	4.584	0.032	-0.114
29	2	6	E30,E29	4.552	0.033	-0.124
30	2	6	E57,E52	4.528	0.033	0.131
31	2	6	E35,E12	4.491	0.034	-0.142
32	2	6	E36,E34	4.387	0.036	-0.149
33	2	6	E7,E5	4.211	0.040	0.177
34	2	6	E50,E6	4.206	0.040	0.127
35	2	6	E36,E5	3.832	0.050	0.117
36	2	6	E58,E34	3.832	0.050	0.138
37	2	12	V12,F4	3.655	0.056	-0.381
38	2	6	E27,E8	3.584	0.058	0.124
39	2	6	E8,E7	3.557	0.059	-0.154
40	2	12	V56,F4	3.442	0.064	-0.186
41	2	6	E30,E27	3.304	0.069	-0.114
42	2	6	E50,E36	3.295	0.069	-0.084
43	2	6	E56,E35	3.294	0.070	-0.103
44	2	6	E56,E50	2.988	0.084	0.090
45	2	6	E51,E28	2.956	0.086	-0.101
46	2	6	E49,E36	2.729	0.099	0.102
47	2	6	E14,E13	2.659	0.103	-0.161
48	2	6	E14,E5	2.590	0.108	0.121
49	2	6	E57,E36	2.541	0.111	0.086
50	2	12	V57,F1	2.538	0.111	0.151

51	2	12	V8,F1	2.508	0.113	-0.281
52	2	6	E50,E49	2.503	0.114	-0.102
53	2	6	E52,E50	2.442	0.118	-0.100
54	2	6	E50,E35	2.344	0.126	0.069
55	2	6	E12,E6	2.320	0.128	0.118
56	2	6	E14,E12	2.308	0.129	0.149
57	2	6	E29,E28	2.205	0.138	-0.083
58	2	6	E50,E5	2.156	0.142	-0.081
59	2	6	E34,E29	2.082	0.149	0.072
60	2	6	E51,E7	1.995	0.158	0.087
61	2	12	V5,F1	1.994	0.158	0.257
62	2	6	E52,E28	1.982	0.159	0.096
63	2	6	E51,E5	1.970	0.160	0.088
64	2	6	E13,E8	1.924	0.165	0.088
65	2	6	E57,E27	1.895	0.169	-0.068
66	2	6	E49,E5	1.874	0.171	0.106
67	2	6	E58,E49	1.829	0.176	-0.105
68	2	12	V35,F4	1.809	0.179	-0.126
69	2	6	E13,E6	1.778	0.182	-0.085
70	2	6	E49,E6	1.761	0.184	-0.103
71	2	12	V14,F4	1.761	0.185	-0.269
72	2	6	E51,E14	1.738	0.187	-0.084
73	2	6	E30,E13	1.700	0.192	-0.069
74	2	6	E27,E14	1.691	0.193	-0.080
75	2	6	E35,E6	1.688	0.194	0.079
76	2	6	E50,E30	1.653	0.199	0.066
77	2	6	E57,E49	1.628	0.202	-0.080
78	2	6	E51,E6	1.598	0.206	-0.082
79	2	6	E27,E6	1.569	0.210	-0.082
80	2	6	E12,E5	1.569	0.210	-0.094
81	2	12	V34,F4	1.490	0.222	-0.123
82	2	6	E58,E52	1.474	0.225	-0.093
83	2	6	E58,E27	1.469	0.226	-0.075
84	2	6	E57,E56	1.450	0.228	-0.084
85	2	6	E35,E30	1.432	0.231	0.063
86	2	6	E56,E14	1.367	0.242	0.076
87	2	6	E35,E5	1.323	0.250	-0.068
88	2	6	E50,E27	1.320	0.251	-0.056
89	2	6	E52,E51	1.314	0.252	-0.078
90	2	6	E51,E27	1.307	0.253	0.062
91	2	6	E35,E8	1.279	0.258	0.069
92	2	12	V50,F4	1.236	0.266	-0.107
93	2	6	E36,E8	1.235	0.266	-0.069
94	2	12	V58,F4	1.224	0.269	-0.125
95	2	6	E52,E13	1.203	0.273	0.065
96	2	6	E57,E29	1.192	0.275	-0.049
97	2	6	E56,E27	1.176	0.278	0.058
98	2	6	E30,E14	1.158	0.282	0.071
99	2	12	V28,F4	1.153	0.283	-0.124
100	2	6	E51,E34	1.015	0.314	0.057
101	2	6	E49,E30	1.008	0.315	-0.069
102	2	6	E58,E57	0.972	0.324	-0.071
103	2	6	E56,E5	0.953	0.329	-0.062
104	2	6	E29,E5	0.949	0.330	-0.058
105	2	12	V58,F1	0.942	0.332	-0.107
106	2	12	V52,F1	0.926	0.336	0.102
107	2	6	E36,E6	0.917	0.338	-0.059
108	2	6	E52,E29	0.901	0.343	-0.055
109	2	6	E52,E6	0.898	0.343	-0.071
110	2	12	V27,F4	0.878	0.349	0.102
111	2	6	E27,E13	0.844	0.358	0.046
112	2	6	E35,E14	0.793	0.373	-0.061
113	2	6	E58,E30	0.761	0.383	0.057
114	2	6	E34,E7	0.729	0.393	0.053
115	2	6	E56,E28	0.703	0.402	-0.049
116	2	6	E57,E50	0.692	0.406	0.040
117	2	6	E30,E12	0.686	0.408	-0.054

118	2	6	E28,E7	0.683	0.409	-0.054
119	2	6	E29,E13	0.674	0.412	0.038
120	2	6	E50,E29	0.637	0.425	0.035
121	2	6	E56,E8	0.632	0.427	-0.052
122	2	12	V56,F1	0.627	0.429	-0.078
123	2	6	E49,E8	0.615	0.433	-0.061
124	2	6	E57,E7	0.614	0.433	-0.044
125	2	6	E14,E7	0.612	0.434	-0.057
126	2	6	E57,E12	0.611	0.434	0.046
127	2	6	E58,E51	0.591	0.442	-0.050
128	2	12	V29,F1	0.565	0.452	0.061
129	2	6	E8,E5	0.553	0.457	-0.062
130	2	6	E34,E8	0.547	0.459	-0.050
131	2	6	E28,E8	0.540	0.462	-0.051
132	2	12	V6,F1	0.534	0.465	0.131
133	2	6	E52,E14	0.534	0.465	0.054
134	2	6	E56,E6	0.520	0.471	-0.047
135	2	6	E36,E29	0.498	0.480	-0.033
136	2	6	E58,E28	0.484	0.487	0.046
137	2	6	E56,E36	0.453	0.501	0.038
138	2	12	V7,F1	0.430	0.512	-0.116
139	2	6	E36,E30	0.430	0.512	0.035
140	2	6	E36,E7	0.425	0.514	-0.038
141	2	12	V50,F1	0.399	0.528	-0.053
142	2	6	E52,E12	0.394	0.530	-0.046
143	2	6	E56,E51	0.382	0.537	-0.035
144	2	6	E57,E5	0.371	0.542	0.035
145	2	6	E28,E13	0.362	0.547	-0.032
146	2	6	E56,E52	0.354	0.552	0.040
147	2	12	V34,F1	0.340	0.560	-0.059
148	2	6	E28,E12	0.314	0.575	0.037
149	2	12	V52,F4	0.307	0.580	0.066
150	2	6	E58,E12	0.295	0.587	-0.041
151	2	6	E58,E29	0.288	0.591	0.030
152	2	6	E49,E12	0.283	0.595	0.040
153	2	12	V51,F4	0.268	0.605	0.053
154	2	6	E58,E50	0.238	0.626	0.029
155	2	6	E50,E8	0.225	0.635	-0.027
156	2	6	E56,E13	0.213	0.645	0.024
157	2	6	E49,E13	0.211	0.646	-0.028
158	2	6	E52,E27	0.200	0.655	-0.028
159	2	6	E30,E6	0.198	0.657	0.030
160	2	6	E13,E5	0.196	0.658	0.028
161	2	6	E34,E30	0.196	0.658	-0.026
162	2	12	V28,F1	0.190	0.663	-0.041
163	2	6	E51,E30	0.190	0.663	-0.025
164	2	6	E56,E12	0.189	0.664	-0.029
165	2	12	V51,F1	0.173	0.677	-0.038
166	2	6	E50,E34	0.171	0.679	0.021
167	2	6	E29,E8	0.165	0 685	0.024
168	2	6	E49,E14	0.163	0 686	0.031
169	2	6	E52,E5	0.162	0 688	0.029
170	2	6	E57,E13	0.159	0 690	-0.020
171	2	12	V29,F4	0.158	0.691	0.040
172	2	6	E27,E12	0.157	0 692	0.024
173	2	6	E36,E27	0.153	0 696	-0.020
174	2	6	E30,E5	0.150	0 699	0.026
175	2	12	V30,F1	0.139	0 709	-0.035
176	2	6	E49,E29	0.136	0.712	0.022
177	2	6	E57,E51	0.136	0.712	0.019
178	2	6	E34,E13	0.133	0.716	-0.022
179	2	6	E28,E14	0.132	0.716	0.024
180	2	6	E58,E8	0.131	0.717	-0.027
181	2	6	E51,E36	0.131	0.718	-0.019
182	2	6	E34,E6	0.129	0.720	-0.024
183	2	6	E50,E14	0.127	0.722	-0.020
184	2	6	E52,E7	0.126	0.722	0.025

185	2	12	V30,F4	0.125	0.723	-0.041
186	2	6	E49,E28	0.119	0.730	0.024
187	2	6	E12,E8	0.112	0.738	0.026
188	2	6	E49,E35	0.111	0.739	0.020
189	2	6	E35,E29	0.096	0.757	-0.014
190	2	6	E50,E13	0.081	0.776	-0.013
191	2	6	E56,E7	0.076	0.783	-0.017
192	2	6	E52,E8	0.074	0.786	-0.021
193	2	6	E36,E13	0.072	0.789	0.015
194	2	6	E29,E6	0.069	0.793	0.016
195	2	6	E56,E29	0.062	0.803	-0.012
196	2	6	E27,E7	0.061	0.806	0.015
197	2	6	E57,E8	0.060	0.806	-0.015
198	2	6	E14,E6	0.058	0.809	0.019
199	2	6	E34,E27	0.057	0.811	-0.013
200	2	6	E57,E30	0.056	0.813	-0.012
201	2	12	V49,F4	0.055	0.814	0.028
202	2	6	E34,E14	0.045	0.832	0.015
203	2	6	E58,E35	0.045	0.833	-0.014
204	2	6	E51,E12	0.044	0.834	0.013
205	2	6	E49,E34	0.044	0.834	-0.014
206	2	6	E30,E7	0.043	0.835	0.014
207	2	6	E29,E14	0.043	0.837	0.012
208	2	6	E51,E13	0.040	0.841	-0.010
209	2	6	E50,E7	0.039	0.844	-0.010
210	2	6	E29,E12	0.037	0.848	-0.011
211	2	6	E51,E50	0.035	0.853	-0.010
212	2	6	E34,E5	0.033	0.855	0.012
213	2	6	E28,E27	0.029	0.864	-0.010
214	2	6	E58,E6	0.027	0.870	0.012
215	2	6	E56,E30	0.024	0.876	0.009
216	2	6	E51,E35	0.023	0.879	-0.008
217	2	6	E49,E7	0.022	0.882	-0.011
218	2	6	E58,E5	0.021	0.884	-0.011
219	2	6	E35,E27	0.017	0.896	-0.007
220	2	6	E7,E6	0.013	0.910	-0.009
221	2	6	E35,E7	0.010	0.921	-0.006
222	2	6	E50,E12	0.007	0.935	0.005
223	2	6	E57,E14	0.006	0.939	0.005
224	2	6	E28,E5	0.005	0.942	-0.005
225	2	6	E58,E7	0.005	0.942	0.005
226	2	6	E52,E36	0.005	0.942	0.004
227	2	12	V27,F1	0.005	0.944	0.006
228	2	12	V49,F1	0.004	0.953	0.006
229	2	6	E51,E8	0.002	0.963	-0.003
230	2	6	E13,E12	0.000	0.983	-0.002
231	2	6	E52,E35	0.000	0.998	0.000
232	2	0	V27,F5	0.000	1.000	0.000
233	2	0	V49,F6	0.000	1.000	0.000
234	2	0	V34,F2	0.000	1.000	0.000
235	2	0	F6,D6	0.000	1.000	0.000
236	2	0	F5,D5	0.000	1.000	0.000
237	2	0	F3,D3	0.000	1.000	0.000
238	2	0	F2,D2	0.000	1.000	0.000
239	2	0	V12,F1	0.000	1.000	0.000
240	2	0	V5,F4	0.000	1.000	0.000
241	2	0	V56,F3	0.000	1.000	0.000

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MAXIMUM LIKELIHOOD SOLUTION (NORMAL DISTRIBUTION THEORY)

MULTIVARIATE LAGRANGE MULTIPLIER TEST BY SIMULTANEOUS PROCESS IN STAGE 1

PARAMETER SETS (SUBMATRICES) ACTIVE AT THIS STAGE ARE:

PEE GVF

		CUMULATIVE MULTIVARIATE STATISTICS			UNIVARIATE INCREMENT	
STEP	PARAMETER	CHI-SQUARE	D.F.	PROBABILITY	CHI-SQUARE	PROBABILITY
1	E35,E34	26.019	1	0.000	26.019	0.000
2	E8,E6	39.091	2	0.000	13.072	0.000
3	E29,E27	49.744	3	0.000	10.653	0.001
4	V13,F4	59.298	4	0.000	9.554	0.002
5	E34,E28	66.582	5	0.000	7.284	0.007
6	E35,E28	75.494	6	0.000	8.912	0.003
7	E52,E49	82.602	7	0.000	7.108	0.008
8	E51,E49	92.315	8	0.000	9.714	0.002
9	E58,E13	98.216	9	0.000	5.901	0.015
10	E57,E6	103.736	10	0.000	5.520	0.019
11	E57,E28	109.856	11	0.000	6.120	0.013
12	E58,E56	116.587	12	0.000	6.731	0.009
13	E50,E6	121.997	13	0.000	5.409	0.020
14	E12,E7	126.875	14	0.000	4.878	0.027
15	E52,E34	131.648	15	0.000	4.773	0.029
16	V35,F1	135.898	16	0.000	4.250	0.039
17	E14,E8	139.797	17	0.000	3.899	0.048

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Execution begins at 17:41:03.15
Execution ends at 17:41:07.48
Elapsed time = 4.33 seconds