Reactive Psychosis in a First-Episode Psychosis Population

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Declaration

"I, Helen Krstev, declare that the Doctor of Psychology (Clinical) thesis entitled *Reactive Psychosis in a First-Episode Psychosis Population* is no more than 40,000 words in length including quotes and exclusive of tables, figures, appendices, bibliography, references and footnotes. This thesis contains no material that has been submitted previously, in whole or in part, for the award of any other academic degree or diploma. Except where otherwise indicated, this thesis is my own work".

I further declare that the ethical principles and procedures specified by the Department of Psychology Ethics Committee of Victoria have been adhered to in the preparation of this thesis.

Signature: Date:

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Abstract

Contemporary western psychiatry is disengaging too readily with the notion that severe mental disorder characterised by psychotic experiences such as hallucinations and delusions can be a primary reaction to overwhelming stress or distress (Ungvari & Mullen, 2000). Reactive psychosis, a term coined over a century ago to differentiate these disorders from other psychotic processes more closely resembling schizophrenia, was investigated in 217 first episode psychosis patients being treated at the Early Psychosis Prevention and Intervention Centre, in Melbourne, Australia. Reactive psychosis criteria were first operationalised using the Reactive Psychosis Rating Form (RPRF). These patients' course and outcome were then compared with their non-reactive psychosis counterparts over a 15-month follow up period. Twenty nine percent of first-episode psychosis patients met criteria for reactive psychosis. The reactive psychosis group had a more rapid initial recovery from their psychosis and social and occupational impairments, compared with their non-reactive psychosis counterparts. Clinical practice at the centre dictated that these patients had discontinued their anti-psychotic medication faster than the non-reactive psychosis group, suggesting the need to reconsider early psychosis treatment guidelines.

Chapter 1: Early Psychosis: The Current State of Play

First-episode psychosis populations have been studied for the last 20 years. This was borne out of a strong desire to revolutionise treatments for early psychosis, with the emphasis on maximising intervention during, what Birchwood coined, 'the critical phase' or the first two to five years after the onset of early signs of psychosis (Birchwood, 2000).

The research impetus in the first decade had been to promote the ethos of early detection, undoubtedly an intuitive endeavour and one familiar to general medicine. Intuitiveness aside, several studies produced in these early years pointed to the deleterious effects of prolonged delay in the treatment of psychosis.

Several studies have found that a longer duration of untreated psychosis (DUP) was significantly associated with a longer time to remission and a poorer level of remission and outcome (Loebel, Lieberman, Alvir, Mayerhoff, Geisler, & Szymanski, 1992). A long DUP has also been linked to an increased risk of relapse (Crow, MacMillan, Johnson, & Johnstone, 1986), more pronounced negative symptoms (Haas, Garratt, & Sweeney, 1998), positive symptoms and general psychopathological symptoms as well as a lower global functioning 15 years after the first psychiatric admission, even after effects of other factors, possibly related to the long-term outcome, were controlled for (Bottlender et al., 2002).

Likewise, a *shorter* DUP has been associated with earlier remission (Loebel et al., 1992), less relapse (Crow et al., 1986) and better outcome (Marshall et al., 2005; Rabiner, Wegner, & Kane, 1986). A study conducted at Orygen Research Centre investigated the relationship between DUP and outcome in a large representative sample of first-episode patients over the medium-to-long term (Harris et al., 2005). At 8 years follow up, a shorter DUP was associated with less severe positive symptoms and enhanced social and occupational functioning and quality of life, independent of psychotic diagnosis. Harris et al. (2005) were able to demonstrate that DUP was directly related to outcome (at least in part) as it was shown to be independent of

potential confounding variables, answering criticism that DUP is merely a proxy for other variables such as premorbid adjustment (Verdoux et al., 1998; 2001). In light of this evidence, it has been suggested that acute psychotic symptoms may reflect an active morbid process, which if not influenced by early treatment, could result in more lasting morbidity (Loebel et al., 1992).

In addition to any assumed biological entrenchment of this disorder, delays in treatment also lead to a number of psychosocial complications, which could further contribute to impaired recovery and future outcomes. Prolonged periods of untreated psychosis may lead to vocational derailment, developmental delay, demoralisation and depression (Edwards et al., 1994). They may contribute to a disruption in protective social networks (Lincoln & McGorry, 1995) and they are associated with more serious family difficulties and increased morbidity amongst family members (Johnstone, Crow, Johnson, & MacMillan, 1986). A French study found that patients with a long delay in treatment were more likely to have lower education levels, poorer global functioning prior to admission and a more severe global clinical state (Verdoux et al., 1998).

Service reform has shown that it is possible to reduce the DUP experienced by those with a first-episode psychosis. The pre-eminent and most comprehensive programme aimed at reducing the DUP is the Norwegian TIPS study (Johannessen et al., 2005). The Norwegian TIPS study compared two regions with an early psychosis detection programme and community awareness campaign to two areas without an intervention. They found they were able to successfully reduce the DUP in the two intervention areas to medians of 5 and 16 weeks (Melle et al., 2004). The early detection group showed an advantage on baseline symptomatic variables, indicating that perhaps the clinical course or picture of early psychosis can be modified by early intervention. Other specialist early psychosis services have demonstrated a significantly lower median duration of untreated psychosis (e.g., Carbone, Harrigan, McGorry, Curry, & Elkins, 1999; Linszen, Lenior, De Haan, Dingemans, & Gersons, 1998) compared to first-episode patients being treated in standard psychiatric services in the early 1990s (e.g. Loebel et al., 1992; Szymanski, Cannon, Gallacher, Erwin, & Gur, 1996).

The duration of untreated psychosis remains one of the most widely researched predictors of outcome. However, it is worth briefly highlighting other factors that may influence outcome. While several studies have shown that duration of untreated psychosis is the most significant predictor of outcome, other factors such as negative symptoms (Emsley, Rabinowitz, & Medori, 2007), age of onset (Emsely et al., 2007), premorbid adjustment (Simonsen, Friis, Haahr, et al., 2007) and higher education level (Simonsen et al., 2007) also contribute to a better symptomatic outcome.

Gender has long been considered a prognostic factor in the course and outcome of schizophrenia. Although males have tended to be overrepresented in older more chronic samples of schizophrenia (Castle & Murray, 1991; Iacono & Beiser, 1992; Kendler & Walsh, 1995) there is little in the way of evidence to suggest that females have a better outcome than males in a first episode psychosis sample. Hafner, Maurer, Loffler and Rossler's (1993) study of 267 first episode patients found that gender differences in the course of psychosis were completely accounted for by their differing age of onset and that the early course of positive and negative symptoms was not related to gender but rather other age dependent variables. Norman, Townsend and Malla's (2001) study of early psychosis showed that male gender was associated with better cognitive performance than their female counterparts, with females having a more significant deterioration.

Similarly, Larsen et al.'s study of first episode psychosis, whilst finding that men had a longer duration of untreated psychosis, found that the clinical correlates of duration of untreated psychosis were not gender specific (Larsen, McGlashan & Moe, 1996). Consistent with other studies looking at the contribution of DUP to outcome, their study found that gender was not a predictor of time to remission or level of positive symptoms (Emsely et al., 2007; Wunderlink, Systema, Nienhuis, & Wiersma, 2009). While females did better than males in social functioning and gender marginally contributed to level of negative symptoms in their regression model, level of negative symptoms at baseline and at 3 months remained the predominant predictors of outcome (Larsen et al., 1996). It appears that whilst gender representations in early psychosis may be askew, outcome measures are generally not related to gender specifically, indicating that this question requires further

consideration. It is apparent even in this brief review of studies focused on predictors of outcome in psychosis that the studies vary considerably in the way outcome is defined and measured.

A recent systematic review of outcome in psychosis has highlighted several limitations in this research field, most notably that there is disagreement about *what* defines 'good outcome' and a lack of consistency in measuring it (Menezes, Arenovich, & Zipursky, 2006). This brings up the difficulty in generalising and comparing research findings highlighting the course of psychotic disorders. Some studies have emphasised the level of remission of positive and negative symptoms or the number of relapses, and others, including consumer groups, have noted the importance of considering social and occupational functioning and quality of life (Bellack, 2006; Menezes, et al., 2006; Resnick, Fontana, Lehman, & Rosenheck, 2005). It is worth noting that all of these aspects of a young person's recovery are important and need to be more consistently measured and defined.

Given the inconsistencies in defining and measuring outcome, it is not surprising that schizophrenia research has long reported varied outcomes, with studies citing between 20-50% of people recovering (Bleuler, 1978; Ciompi, 1980; Harding et al., 1992; Huber et al., 1980; Shepherd et al., 1989; van Os et al., 1996), whilst a majority have multiple episodes (Bleuler, 1978; Ciompi, 1980; Harding et al., 1992; Huber et al., 1980; Shepherd et al., 1989; van Os et al., 1996). A recent meta-analysis of 37 studies including 4100 first episode psychosis patients (schizophrenia spectrum), whilst highlighting the varying domains (symptoms recovery, relapse and social and occupational functioning) and definitions of outcome and its limitations in summarising results, nonetheless reported a 'good' outcome for 42% of the population, an 'intermediate' outcome for 35%, and a 'poor' outcome for 27% (Menezes et al., 2006).

The Remission in Schizophrenia Working Group recognised this need for a consensus definition of remission as applied to schizophrenia and in 2005 published a set of criteria for defining outcome using well established and validated instruments (Andreasen et al., 2005). Specifically, remission was defined as having a score of ≤ 3 on the Brief Psychiatric Rating Scale (Overall, 1974), ≤ 2 on the Scale for the

Assessment of Negative Symptoms (SANS; Andreasen, 1984) and ≤ 3 on the Positive and Negative Symptoms Syndrome Scale (Andreasen & Olsen, 1982) maintained over a 6-month period. Whilst it is a significant improvement in defining symptomatic recovery, it neglects the importance of social and occupational functioning in the recovery process.

In a recent study Henry et al. (2010) utilised Andreasen's criteria in their seven-year follow up of 723 consecutive first-episode psychosis patients. Symptomatic remission at follow-up was observed in 59% of the cohort (using the Brief Psychiatric rating Scale) and 37% (using the combined SANS and BPRS) and social/vocational recovery was observed in 31% of the cohort. Furthermore, over the two years prior to follow up, 46.2% reported never being actively psychotic, 20.8 % reported an episodic course (discrete episodes less than 6 months in duration), 33% reported a continuous course and 2.7% had neither an episodic nor a continuous course. Other studies using Andreasen's criteria have shown that 70% achieved a rating of mild or less on the 8 key PANS remission items, with patients taking an average of 5 months to reduce the severity of their symptomatology (Emsley et al., 2007), whilst some have reported a symptomatic recovery in 50% of the sample at a 2 year follow up (Wunderlink, et al., 2009). Consistent with other authors, Henry et al. (2010) and Andreasen et al. (2005) highlighted the importance of including negative symptoms as an outcome in first episode psychosis as they have been considered a direct manifestation of the basic dysfunctions of schizophrenia (Peralta, Cuesta, Martinez-Larrea, & Serrano, 2000).

Early intervention has made ground in reducing the duration of untreated psychosis. If we are treating people earlier in their phase of psychotic illness, it is likely that at least some of what we are seeing is a less severe or longstanding clinical picture of psychosis and possibly less comorbidity and decline in functioning. This raises the question of what is deemed appropriate treatment for this earlier phase.

The 'new' composition of first-episode psychosis presentations has introduced a new era of research. This is based on some of the seminal work outlined earlier, and the established view that psychotic symptoms occur on a continuum throughout the general population (Johns & van Os, 2001; van Os, Hanssen, Bijl & Ravelli, 2000).

Furthermore, high rates of psychotic-like experiences have been reported in community studies (Laurens et al., 2007; Yung et al., 2007) and these experiences can occur without distress, interference in functioning, therefore questioning the need for treatment or intervention of any kind (Yung et al., 2006).

It leaves open the question of determining when there is a 'need' to intervene. In beginning to answer this question, the clinical staging model of psychiatric disorders could be the next wave of development and research in early intervention. This staging model proposes that earlier treatments could be less invasive and also be more effective than those delivered later in the course of a disorder if, by reducing duration of untreated psychosis, they are able to prevent progression to more severe forms of disorder (McGorry, Hickie, Yung, Pantelis, & Jackson, 2006).

These 'less intrusive' interventions would consist of psychoeducation, psychological and social interventions, considering antipsychotic treatment as second line treatments. It is proposed that antipsychotic treatment would be avoided in the early stages of the disorder and only introduced when less aggressive treatments have had an inadequate impact (McGorry, et al., 2006). The next section briefly outlines the current treatment guidelines for first-episode psychosis and briefly summarises some of the evidence of the medical consequences of considering antipsychotic medication as a first-line treatment.

The guidelines detailing initial treatment for early psychosis are comprehensive, detailed and specific. In order to not lose any of their specificity, they have been reproduced in Table 1 (see page 10) from the International Early Psychosis Association Writing Group, published in the British Journal of Psychiatry 2005 (IEPA Writing Group, 2005). One could argue that the guidelines are quite prescriptive, particularly the focus on antipsychotic medication as first-line treatment.

While antipsychotic medications have been shown to be valuable in the treatment of acute psychosis, there are well-documented and sometimes serious side effects associated with these medications, including significant weight gain and diabetes (Allison & Casey, 2001; Muench & Carey, 2001), possible morphological changes in the brain (Corson, Nopoulos, Miller, Arndt, & Andreasen, 1999), and the

longer-term effects such as tardive dyskinesia (Llorca, Chereau, Bayle, & Lancon, 2002).

A review on metabolic monitoring for patients being treated with antipsychotic medications revealed that patients with serious mental illness had markedly elevated rates of metabolic disturbance, including obesity, diabetes and dyslipidemia, with antipsychotic medication being a contributing factor (Cohn & Sernyak, 2005). In fact, in Canada, recent diabetes treatment guidelines recognise schizophrenia as an independent risk factor for type 2 diabetes (Canadian Diabetes Association, 2003). Furthermore, metabolic disturbance puts individuals at a higher risk of cardiovascular disease.

An Australian consensus statement released by representatives of psychiatry, endocrinology, epidemiology, general practice, mental health nursing, and pharmacy as well as those from community and non-government agencies, contends that the newer, second-generation antipsychotic agents are more likely to produce diabetes and worsening blood sugar control (Lambert & Chapman, on behalf of the consensus working group, 2004). They insist that the "management of psychosis takes priority over concerns about the potential metabolic sequelae of treatment, but the prevalence of the latter requires that all patients taking antipsychotic agents be actively screened and treated" (Lambert & Chapman, on behalf of the consensus working group, 2004 pg. 544,). At the same time they recognise that mortality and medical morbidity is higher in psychotic populations, raising some serious concerns about the position adopted.

These side effects accentuate the necessity for individualised risk benefit analysis, particularly in light of the fact that the introduction of second-generation anti-psychotics has not improved adherence to medication (Kane & Malhotra, 2003). Several studies have reported poor compliance with antipsychotic medication (Oehl, Hummer & Fleischhacker, 2000; Perkins, 1999), one reporting that as many as 60% of first-episode psychosis patients at a specialised treatment service were not compliant or inadequately compliant with anti-psychotic medication (Coldham, Addington, & Addington, 2002).

The early intervention model accepts that early optimal treatment includes the introduction of novel atypical anti-psychotic medication at low doses in order to minimize DUP. Therefore, patients who may be treated with psychological support, without the introduction of anti-psychotics would be considered 'untreated'. In spite of evidence that there is an association between DUP and outcome in first-episode psychosis (Marshall et al., 2005), it has not been established that anti-psychotic medication is the *definitive* treatment for all cases of first-episode psychosis.

In fact, the contrary may be true for those with a more chronic course of psychosis. In a recent meta-analysis conducted, Bola (2006a) found a small, non-significant advantage in outcomes for initially non-medicated groups with established schizophrenia over medicated groups. Bola argued that this finding provided evidence that an initial period of non-medication produces superior outcomes to those who receive medication. Others have reported similar findings (Carpenter, 1997; Johnstone, Owens, Crowe & Davies, 1999), which have directed some to advocate providing psychosocial interventions with short-term withholding of anti-psychotic medication (Bola, 2006b; Carpenter, Appelbaum, & Levine, 2003).

Cognitive behaviour therapy (CBT), behavioural family interventions, in addition to psychoeducation, social and vocational rehabilitation and group activities are recommended approaches for the treatment of psychosis as reflected in contemporary guidelines (RANZCP, 2005). A review of the efficacy of varying types of psychotherapy for psychosis, is beyond the scope of this thesis, but suffice to say that the author advocates for an 'integrated approach' to the treatment of psychotic disorder, considering the importance of psychoanalytically oriented therapy and culturally sensitive practice in addition to contemporary guidelines (Gleeson, Killacky & Krstev, 2008).

Current treatments for psychosis are revolutionary and certainly have a come a long way from the practice of long-term admissions, use of typical medications at high doses causing deleterious side effects such as parkinsonian syndromes, etc. However, the current treatment guidelines for first-episode psychosis are generic and weighted in favour of use of anti-psychotic medications without paying heed to type

of psychotic diagnosis, the stress-vulnerability model (Nuechterlein & Dawson, 1984), aetiological considerations and psychotherapeutic interventions.

The next endeavour in this field of research, in reference to the staging model (McGorry et al., 2006) is identifying which first episode psychosis patients may benefit from treatments with fewer side effects. One place to start is considering those patients with more 'psychogenic' components to their disorder, in particular, examining the role of environmental stress and the acuity of onset. This thesis could be seen at complementing the current research milieu (particularly in light of the pilot stages trial at the Orygen Research Centre, randomising first-episode psychosis patients who are anti-psychotic naive to an initial delay in antipsychotic treatment but with intense psychosocial support), as a secondary outcome from our study will raise the question of whether there is a subgroup of patients that can be treated without antipsychotic medication or for shorter periods of time.

Although not addressing these concerns directly, this study hopes to shed light on the usefulness of applying the reactive psychosis diagnosis in the Australian psychiatric setting, which may have implications for the treatment of first-episode psychosis.

There is a breadth of research examining prognostic variables affecting the onset, course and medium to long term outcome of psychotic disorders, particularly those of a biological or genetic basis and relatively little attention has been paid to the role of major life stressors in the aetiology, onset and course of psychosis, despite its potential clinical implications. Furthermore, creating the generic, all inclusive term early psychosis was an attempt to capture the essence of an early psychosis, its flux and fluid course, however in the process, the discourse regarding psychopathology and diagnosis has perhaps been undervalued.

The following chapter will provide a short review of the role of stressors and trauma in the development of psychotic illnesses within the predominant stress vulnerability paradigm.

Table 1. International clinical practice guidelines for early psychosis (2005)

Clinical guidelines for early psychosis: Initial management
1. Before initiating treatment, it is important to consider physical illnesses that can cause psychosis. 2. Extrapyramidal side-effects from antipsychotic treatment should be avoided in order to encourage future adherence to medication. Although typical antipsychotics may be as efficacious as atypical antipsychotics in reducing positive psychotic symptoms, they are frequently less well tolerated even at low doses. For this reason alone, atypical antipsychotics should be used as first-line therapy, commencing with a low dose and titrating upwards very slowly over a period of several weeks ('start low, go slow'). 3. Examples of appropriate initial target doses for most patients are risperidone 2 mg/day or olanzapine 7.5–10.0 mg/ day. Initial target doses of other medications such as quetiepine, ziprasidone and amisulpride are yet to be established. 4. Half to two-thirds of patients might be expected to achieve a good response in positive psychotic symptoms within 3 weeks at the initial dose, but if necessary the doses can be increased to 4 mg/day risperidone or 20 mg/day olanzapine. 5. The level of clinical response and risk should be assessed frequently, but the dose of the antipsychotic should be increased only at widely spaced intervals (after initial titration, usually 14–21 days) if the response has been inadequate, and then only within the limits of sedation and the emergence of extrapyramidal side effects. However, extrapyramidal side effects should not be tolerated. 6. If the response is not adequate at therapeutic doses by 6–8 weeks, another atypical antipsychotic should be tried. When use of typical antipsychotics is unavoidable, they should be commenced at very low doses (1–2mg haloperidol or equivalent) and titrated very slowly within the limits of extrapyramidal side-effects. 7. Generally, this will be a maximum of 4–6mg haloperidol or equivalent in first-episode psychosis. 8. Low doses of antipsychotic medication will not have a rapid effect on distress, insomnia and behavioural disturbances secondary to psychosis; skilled nursing care, a safe and supportive environment, and regular and liberal doses of benzodiazepines are essential interim components of management in many cases. 9. Although some atypical antipsychotics have initial sedative side-effects, treatment of psychosis should be separated conceptually from the need for tranquillisation. 10. If positive psychotic symptoms persist after a trial of two first-line atypical antipsychotics (around 12 weeks), the reasons for the failure of treatment should be reviewed. Possible contributing factors include adherence problems, family stresses and substance misuse. 11. Slow recovery or early treatment resistance of this kind is of concern and requires more intensive intervention. 12. Clozapine and cognitive–behavioural therapy for persistent symptoms are obvious alternatives to consider. 13. Supportive crisis plans are needed to facilitate recovery and acceptance of treatment. 14. Specific psychosocial strategies should be employed when poor adherence,

family stresses, increased suicide risk and substance misuse occur.
15. Families are usually in crisis at the point of initiation of treatment and require emotional support and practical advice.
16. Families and other members of the person's social network, possibly including friends, teachers and employers, should be progressively informed and educated about the nature of the problem, treatments and the outcomes expected
17. If there are frequent relapses or slow recovery, a more intensive and prolonged psychoeducational and supportive intervention for families may be required. A calm and optimistic approach is vital, especially if the early course is stormy or there are additional family problems or secondary consequences of untreated psychosis.
18. Family therapy may be indicated when there is a high degree of distress in the family.
19. Structured group programmes tailored to the immediate needs of the patient should be available.

NB: This table is reproduced from: International Early Psychosis Association (IEPA) Writing Group. (2005). International clinical practice guidelines for early psychosis. *British Journal of Psychiatry, 187,* s120-s124.

Chapter 2: Trauma and Psychosis

2.1 Incidence of Trauma in Psychosis

There is sufficient literature to suggest that trauma or stressors are associated with psychotic illness. The focus of this thesis will be on negative life events or experiences in general, therefore drawing from the literature on the role of trauma and stressors in the development of psychosis. Several studies report that the incidence of trauma is higher in people who develop psychotic disorders than those in the general population. Shaw's (2002) entire sample of 42 people with severe mental illness had at least one traumatic event in their lives; 36.8% had experienced two and 43.4% had experienced three or more. In Mueser et al.'s (1998) sample of 275 people with serious mental illness, 98% had experienced at least one traumatic event in their lifetime. This trend is not confined to people diagnosed with serious mental illness. An association has also been found between childhood maltreatment and higher levels of unusual perceptions and beliefs in college undergraduates who do not meet criteria for a serious mental illness (Berenbaum, 1999).

High levels of trauma and posttraumatic stress disorder (PTSD) are found in people with psychosis (Resnick, Bond, & Mueser, 2003), and high rates of psychosis have been found in individuals with PTSD, compared with the general population (Kinzie & Boehnlein, 1989). One recent study found that people who had experienced trauma are at a 15 times greater risk of developing psychosis than people who have not experienced trauma (Bebbington et al., 2004). Morrison, Frame and Larkin (2003) suggested that few empirical studies have examined the relationships between PTSD and psychosis since Jeffries' (1977) initial suggestion. Several studies point out that the lifetime prevalence of PTSD is higher in those who later go on to develop severe mental illness than those in the general population (8-9%; Morrison et al., 2003).

High levels of trauma exposure have also been found in younger patients experiencing a first-episode of psychosis. A study of seminal importance was conducted by Neria and others (Neria, Bromet, Sievers, Lavelle & Fochtmann, 2002). They found that the lifetime prevalence of trauma exposure for their first-episode

psychosis sample (N = 426) was 68.5% and 26.5% of those who were exposed to trauma met the criteria for PTSD. This study's strengths were its large-scale nature and that it used a highly reliable interview schedule to ascertain DSM-IV psychotic diagnosis, PTSD diagnosis, and trauma history. The current controversy in the literature on PTSD and psychosis focuses on the directionality of causation. Does PTSD cause psychosis and can psychosis cause PTSD? Morrison attempted to integrate the responses to trauma and proposed that PTSD and psychosis could be a part of a spectrum of responses to a traumatic event (Morrison, Frame & Larkin, 2003).

Interpersonal trauma is also associated with psychosis (Resnick et al., 2003). A large-scale survey of 8580 private households in Britain, found significantly higher rates of victimizing experiences in people with psychosis than in other patient groups and the general population (Bebbington et al., 2004). The odds ratios were in favour of the psychotic group for a history of childhood sexual abuse and violence in the home. Read, a New Zealand academic, has published extensively on this subject. A recent review highlighted consistent findings that patients with psychotic illnesses are more likely to have experienced a history of childhood sexual abuse and that this history is specifically associated with the development of critical or commanding voices in adulthood (Kilcommons & Morrison, 2005; Read, Mosher, & Bentall, 2004; Read, Van Os, Morrison, & Ross, 1995; Ross, Andersen, & Clark, 1994). Psychologists have attempted to explain the trajectory to the development of critical and commanding voices and have observed that it is possible to link extreme negative self evaluations to early interpersonal trauma that predates the onset of psychosis (Garety, Kuipers, Fowler, Freeman, & Bebbington, 2001).

Not only is there an association between childhood trauma and psychosis, there may be a dose response. A large-scale prospective study of the general population in the Netherlands found that early childhood trauma increased the risk of developing positive psychotic symptoms in adulthood in a dose–response manner (Janssen et al., 2004). Their study was well designed in that the relationship between childhood trauma and psychosis remained significant, even after controlling for confounding variables at baseline.

Other studies in support of a dose-response include a long-term follow-up of Pacific Theatre prisoners of World War II, where a marked increase in the diagnosis of schizophrenia in those prisoners who had experienced the most severe traumas was found (Beebe, 1975). Furthermore, Kilcommons and Morrison's (2005) study of people diagnosed with psychosis, found that 94% of participants reported exposure to at least one traumatic event over their lives and that the more severe the trauma, the more severe were the psychotic and PTSD symptoms. The type of psychotic symptom may be specific to certain traumas. Physical assault was only related to positive psychotic symptoms and sexual assault was only related to hallucinations (Kilcommons & Morrison, 2005).

Socio-economic factors have also been associated with higher rates of psychotic disorders. Cantor-Graae and Selten's (2005) recent meta-analysis and review of schizophrenia and migration, concluded that migration was an important risk factor for schizophrenia. Their interest was piqued by an observation of alarmingly high rates of psychosis among people of African Caribbean background, living in the United Kingdom (Sharpley, Hutchinson, McKenzie, Murray 2001). The reviewers' own study in the Netherlands found higher rates of schizophrenia among people from Australia, Africa and Greenland (Cantor-Graae, Pedersen, McNeil, & Mortensen, 2003). In trying to elucidate the stressors more specifically, it appeared that social isolation might be the key in understanding the stress of migration. A recent study found that the incidence of psychotic disorders was highest amongst immigrants living in neighborhoods where their own ethnic group comprised a small proportion of the population (Veiling et al., 2008).

Although the study of the aetiology of psychosis raises more questions than answers, at times emphasising extremely different viewpoints (e.g. biological, psychological or social), it remains clear that there is a noteworthy amount of research indicating an *association* between trauma and psychosis. The several methodological limitations of the studies presented here include small samples or single case studies, and the more robust studies have inconsistent definitions and methods for assessing trauma, some relying on case note data only, particularly for childhood abuses. These difficulties will attest to interpreting the results with caution, rather than dismissing

them altogether. The question left unanswered in the literature is *how* traumatic life events contribute to the development of psychosis.

The following section will briefly outline models for understanding the development and interplay of stressors with other factors in the development of psychosis.

2.2 A Brief Look at Models for Understanding the Development of Psychosis

The predominant model for understanding the aetiology and development of psychosis (and other mental illnesses) is the stress vulnerability model originally developed by Zubin and Spring (1977). Their original model proposed a shift in the emphasis of uni-factorial explanations and suggested the first integrated aetiological account of schizophrenia. Most importantly, it was promoted as a theoretical framework for integrating psychotherapeutic and biological treatments.

The stress vulnerability models posit that psychotic episodes emerge from an interaction between stable or distal factors (e.g., genetics or personality variables) and transient or proximal factors (e.g., life events, interpersonal conflict) via the activation of latent vulnerability (Ciompi, 1989; Nuechterlein et al., 1992; Perris, 1989; Strauss, Hafez, Lieberman, & Harding, 1985; Zubin & Spring, 1977). The renewed clinical hope emerging from these models was that acute psychotic symptoms can be prevented or ameliorated by some combination of the individual's personal resources, the emotional support of close others, and by biological treatments (Gleeson, Killackey & Krstev, 2008).

In stark contrast to the concept of continuous disease process, the model emphasised the episodic course of schizophrenia, suggesting that individual episodes were triggered by endogenous and exogenous *challenging events* that exceeded the patient's vulnerability threshold (Zubin & Spring, 1977). Vulnerability is seen either as a genetically *or* environmentally acquired level of risk for developing the disorder, which could be counterbalanced by personal resources (e.g. coping capability), and by strengths acquired from previous episodes.

In a further development on the theory, Zubin and Steinhauser (1984) added the concept of *etiotypes* to the model (i.e., heterogeneous pathways leading to the development of schizophrenic vulnerability with equivalent behavioural and symptom outcomes). It was argued that *aetiological life events* could produce various etiotypes through genetic, biochemical, neurophysiological, developmental, or learning mechanisms. That is, many pathways potentially lead to vulnerability and ultimately psychosis. Gleeson et al. (2008) argued that these theorists foreshadowed the trauma-vulnerability pathway highlighted by Read and colleagues in their traumatogenic model (Read, Perry, Moskowitz, & Connolly, 2001).

Although conceptualised as a step in the right direction in a move away from the disease model of psychosis, namely, schizophrenia, there was some criticism of these early stress vulnerability models. Concerns were raised that the aetiological pathways to vulnerability were not specific to schizophrenia and that 'stress' did not consider one's subjective appraisal of life events (Nicholson & Neufeld, 1992). Recovery between episodes focused on the remission of positive psychotic symptoms and did not consider deterioration in other symptom domains, such as negative symptoms, that are potentially more debilitating (Carpenter, 1981).

Although a multiplicity of models has come forward since these original ideas, with varying fundamental assumptions, there is inadequate space in this report to adequately outline them. Bentall, Fernyhough, Morrison, Lewis, and Corcoran (2007) have recently proposed a broader conceptualisation that has been received with considerable enthusiasm in the UK. This model, using theories of normal psychological development, posits the need to reconsider the aetiology, particularly the role of trauma, in the diagnoses, assessment and therefore treatment of first-episode psychosis (Bentall et al., 2007). "Cognitive biases that play a central role in the symptoms of psychosis can be seen as end-points of trajectories of cognitive development that are influenced by environmental, genetic and neurodevelopmental factors" (Bentall et al., 2007, p. 160). A second area of developmental psychology purported as being useful in broadening the stress vulnerability model, is theory of mind (ToM). ToM deficits have been implicated in some symptoms of psychosis (Bentall et al., 2007). It is suggested that the transmission of psychosis intergenerationally may not entirely be genetic and may be partly mediated by

individual differences in parental mind-mindedness. Finally, a third area of developmental psychology that Bentall and others have cited as being compromised in patients experiencing auditory hallucinations, is source monitoring (Bentall & Slade, 1985; Johns et al., 2001).

2.3 The Nature of the Trauma as Reflected in the Symptoms of Psychosis

In the investigation of the causality of psychotic illness, the discourse of the quality of psychotic symptoms has perhaps been underplayed. In English-speaking western psychiatry there is a small amount of research examining how trauma is reflected in the content of the psychotic symptoms and how they may function as a means of 'escaping' or 'working' the effects of the trauma. Some of the literature is summarised below.

Read and Argyle (1999) found a link between the nature of abusive experiences and the content reflected in psychotic symptoms. They examined the relationship between hallucinations, delusions, and thought disorder and childhood physical and sexual abuse among psychiatric inpatients. Although a small study that relied on case notes, they found that 77% of patients with an abuse history exhibited at least one psychotic symptom and that half of the symptoms (for which content was recorded) appeared to be related to the abuse. Furthermore, Read, Agar, Argyle and Aderhold's (2003) study of 200 outpatients, found that hallucinations were significantly related to sexual abuse and childhood physical abuse. This was particularly the case for commenting voices and command hallucinations. Goff, Brotman, Kindlon, Waites and Amico (1991) have also suggested that such a history of childhood sexual abuse may contribute to the symptomatology and course of a psychotic illness. Several others have found congruence between the nature of traumatic experiences and the form and content of psychotic symptoms (Raune, Kuipers, & Bebbington, 1999; Fowler, 2000).

More specifically, a case study of delusional parasitosis (the belief that one is infested with parasites such as mites, lice, insects, or bacteria, often in or under the skin but sometimes internally or around bodily orifices) was documented following rape and sexual assault (Oruc & Bell, 1995). Perhaps not surprisingly, patients with

histories of childhood incest were more likely to have sexual delusions (Beck & van der Kolk, 1987).

The question remains whether the association between trauma and content of psychotic symptoms is a causal link. An alternative view is that psychotic symptoms are always related to the person's developmental history and that if this contains traumas, this will be used in his or her development of explanations for anomalous experiences.

As a consequence of a psychoanalytic understanding of psychiatric difficulties not 'falling out of favour', non-English speaking psychiatry (in Western Europe) has long held onto the belief that the content of trauma may be reflected in psychotic symptoms. One such conceptualisation is that of reactive psychosis. Whilst recognising the traumatic nature of psychosis itself, this study is focused on the role of stressors prior to the onset of psychosis, whilst acknowledging that the path may be via PTSD, for some patients.

The following chapter outlines the history of the reactive psychosis concept, its current standing in commonly used diagnostic nomenclature and clinicians' and other proponents' attempts at providing construct/external validity by examining outcome studies.

Chapter 3: Historical and Contemporary Perspectives on Reactive Psychosis

Reactive psychosis is a term used by some to describe a subset of psychotic presentations that has been constituted as being separate from schizophrenia and affective disorders. This distinction is based on clinical observations and whilst having some merit, the concept reactive psychosis requires empirical investigation. These clinical observations have noted a relatively fast onset of psychotic symptoms that emerge as a response to a significant life stressor. The psychosis can be associated with perplexity and confusion and the psychotic symptoms are often transient. In presentations considered reactive the meaning and the content of the psychotic symptoms often reflect the stressor and are argued to function as an escape or release from the original trauma. Aside from psychiatric clinical populations, reactive psychosis has been reported in people with a terminal illness (Onishi et al., 2003) and as a reaction to news of war (Omigbodun & Okunade, 2002; Rushing, & Jean-Baptiste, 2003).

In conceptualizations of reactive psychosis the psychosis is seen as a defence against, or a retreat from, otherwise intolerable feelings (Gallwey, 1985) and may contain and defuse primitive responses (Steiner, 1993). A simple illustration of this is as follows: *A man who becomes imprisoned develops a delusional belief that he is a bird and can fly.* Here, the function of the belief that the man is a bird acts to relieve the man of the psychic pain of having his freedom removed. Although it can be argued that most psychotic breaks are functional for the individual, in reactive psychosis it is particularly apparent, as it is suggested that there is a close temporal relationship between the stressor and the psychotic symptoms. Once the stressor is removed or otherwise ameliorated (perhaps via support, psychotherapy, neuroleptic medication), the psychosis is said to resolve. Most importantly, this subcategory of psychosis has been thought to have a good outcome whereby the individual has a rapid return to premorbid levels of functioning (Jaspers, 1963; McCabe, 1975).

Reactive psychoses appear as a direct contradiction to the traditional notion of psychotic disorder as a progressive disease that ends in the deteriorating or destruction of mental faculties. The paradox of this *disorder* lies in the psychotic presentation being no less severe than its schizophrenia spectrum counterparts, but there is a rapid return to premorbid functioning.

Despite the clinical distinction of this class of psychotic disorders, it is becoming less recognised in English-speaking Western psychiatry, while maintaining its popularity in Scandinavian and some developing nations (particularly Africa and India). Some of the dissent can be attributed to the lack of agreement amongst proponents as to what distinguishes this diagnosis from others (at least at the subtle level).

This chapter outlines the conceptual history of reactive psychoses (dating back over a century), cross cultural nuances in the diagnosis of reactive psychosis, the current classification of reactive psychoses in ICD-10 (WHO, 1992) and DSM-IV (APA, 2004), epidemiology, outcome studies and a critique of the reactive psychosis concept leading to the aims of the present study.

3.1 First Introduction of Reactive Psychosis Throughout History

Ungvari and Mullen (2000) have argued that contemporary western psychiatry is disengaging too readily with the notion that severe mental disorder characterised by psychotic experiences such as hallucinations and delusions can be a primary reaction to overwhelming stress or distress. If an understanding of a psychotic disorder (and the same can be said of higher prevalence disorders such as depression and anxiety) is removed from an individual's psychological and social sphere, then we may be left with an over-reliance on biological models to explain and treat psychotic illnesses.

Reactive psychosis is the traditional term designated to describe the idea that there is a class of psychotic processes/syndromes distinct from schizophrenia-like disorders that emerge as a direct result of an environmental stressor. Throughout history this has been referred to as a 'third psychosis' with the proposition that the

third psychosis lies between the affective illnesses and the schizophrenic syndromes (Jauch & Carpenter, 1988).

There was an attempt to challenge the disease notion of psychoses, even in the beginning of its inception. Ludwig Kahlbaum, in his work *Die Gruppirung psychischer Krankheiten* (1863), conceptualised dysphrenia as a group of severe psychotic disorders that had a good prognosis and a return to premorbid functioning. Rather than the typical disease processes underlying these disorders, he argued that an underlying epileptic, sexual or rheumatic process caused them. His work did not become influential; instead Kraepelin's narrow deterministic view of *dementia praecox* or schizophrenia dominated psychiatry in the 20th century.

Kraepelin took a dichotomous position on the diagnosis of psychosis, arguing that dementia praecox had an underlying disease process with a deleterious outcome; the other was manic illness. Although he took a degenerative disease approach to psychosis, case studies of the brief and acute kind can be found in his early textbooks. His early illustrations were of how these disorders developed within 2-3 days, were accompanied by vivid hallucinations, delusions, changes in mood, and lasted several weeks to months. Although gaining initial attention in Kraepelin's diagnostics system under *periodic delirium* (Kraepelin, 1893), they were then completely subsumed under the category of manic illness as delirious mania in subsequent editions and eventually were not distinguished at all (Kraepelin, 1896; 1899; 1904; 1913). Pillman and Marneros (2003) attributed this misclassification as the source of the present day difficulty in accepting reactive psychosis into official diagnostic systems.

Eventually, Kraepelin conceded that there was a certain group of patients who did not fit into either of his dichotomous categories, but rather than championing the cause, he viewed it as an annoyance (Pillman & Marneros, 2003). Nonetheless, interest in the less chronic, less severe, non-endogenous '3rd psychosis' was sparked and maintained. The genesis of this idea has been jointly accredited to Scandinavian psychiatrist August Wimmer (1916) and German psychiatrist Karl Jaspers (1963) but in actuality, reactive psychosis as we know it today was first described by Esquirol in 1845 in his "treatise on insanity" (Jauch & Carpenter, 1988).

Wimmer's (1916) paper on psychogenic psychoses stated:

> As psychogenic psychoses we designate…..the various clinically independent psychoses, the main feature of which is that they – usually on a (definite) predisposed foundation – are caused by mental agents ('mental traumata'), and in such a way these pathemata determine the point in time of the start of the psychosis, fluctuations (remissions, intermissions, exacerbations) in the disease, very often also its cessation. Likewise the psychosis in form and content is more or less directly and completely ('comprehensively') determined by the precipitating mental factors. To these criteria, finally, be added the predominant tendency, of these disorders to recover, and more specifically, that they never end in deterioration (translated by Stromgren, 1986; p. 261).

Jaspers (1963) added that reactive psychosis accounts for personality and life history and an individual's vulnerability to psychological trauma. Furthermore, he stated that it cannot be classified by symptoms alone, as the whole range of symptoms may be present, albeit transient. In his text *General Psychopathology,* he outlined factors he considered essential in determining reactive or psychogenic conditions. (1) The onset of the illness must have a clear temporal relationship to a precipitating stress, (2) the precipitating stress must be adequate, (3) there must be a meaning for the reaction- it should serve as a defense, a wish fulfilment or as an escape (4) the contents of the reaction must reflect the stress in a meaningful way (Jaspers, 1963).

Jaspers' (1965) account of reactive psychosis was most elaborate in his seminal work *Allgemeine Psychopathologie*, where he revised the concept further to state 3 criteria as being necessary for reactive psychosis (1) an adequate precipitating event in close temporal relationship with the reactive state (2) a comprehensive connection between the content of the event and that of the abnormal reaction (3) resolution of the abnormality with the course of time, or especially, with the cessation of the primary cause. Jaspers purported that certain disorders are reactive, whereas others are not, further cementing the argument that these disorders contradict traditional disease entity notions of psychoses (Shorter, 1997).

Since the initial conceptualisation of reactive psychosis, many non-English speaking western and developing countries have developed the nosology, investigated the epidemiology and attempted to clarify the diagnostic conditions of reactive psychosis, adapting the concept to suit the needs of their psychiatric populations. Each nation has acquired a distinct version of what constitutes reactive psychosis, which aspects to emphasise and which to eliminate, often making further observations on the disorder. What follows is an overview of some of the cross-cultural aspects of reactive psychosis.

3.2 Developments in Conceptualisation of Reactive Psychosis and Differences Across Nations

Before undertaking this discussion it is worth considering why different nations have variations on the diagnosis. Pillman and Marneros (2003) shed some light on this and asserted that the common challenges faced by proponents of the reactive psychosis concept are (1) they have to be accommodated in the respective nosological systems (2) diagnostic criteria have to be assigned that delineate them from other psychotic disorders and (3) they demand an aetiological explanation for the coexistence of severe disturbance and good prognosis.

3.2.1 Scandinavia

Scandinavian psychiatry has been the most persistent in pursuing the idea of reactive psychosis. The seminal work of Langfeldt (1939), Strømgren (1974), Faergeman (1945), Retterstol (1966), Noreik (1970) and others made reactive psychosis a popular concept in Denmark and Norway. There appears to have been a delineation of at least four differing constructs of reactive psychosis among Scandinavian nations levelling criticism at the reliability and validity of the original construct (Guldberg, Dahl, Bertelsen, et al., 1996). The four positions are outlined below.

1. Strømgrem (Denmark): According to Strømgrem (1974) reactive psychoses are entirely psychogenic processes that are caused by a traumatising psychological situation influencing the already vulnerable individual in a

catathymic area of their personality. The patient's thoughts would reflect the trauma and most importantly, the psychosis would recede when the psychological conflict resolves. His notion of reactive psychosis most closely resembles Jaspers' (1963).

2. Ødegård (Norway): Reactive psychoses are those defined as not resembling schizophrenia or manic-depression (Ødegård, 1968). What differentiates this Norwegian concept is that the term 'reactive' is viewed as neutral, without any aetiological implications. Thus, reactive psychoses are defined by exclusion, having more in common with DSM-III-R's atypical psychoses (APA, 1987) than the diagnosis formulated by Wimmer or Jaspers.

3. Astrup and Noreik (1966): According to this Norwegian group, reactive psychoses are defined as functional psychoses of a non-schizophrenia nature with good outcomes. The difficulty with this diagnosis is that it cannot be made until the course and outcome of the psychoses are manifest.

4. Retterstøl (1987): The most commonly held view of reactive psychosis in Denmark and Norway is this interactive notion where reactive psychoses are characterised by an interaction of trauma and personal predisposition (vulnerability). Retterstøl (1987) argued that reactive psychoses are triggered by psychogenic or somatic traumatising factors. The trauma strikes the person, who, due to their vulnerable constitution or somatic status at the time, is vulnerable to a point that the only way to react is through a psychotic breakdown.

3.2.2 French constructs – Bouffée Délirante

In 1886 Valentin Magnan (1893) embedded reactive psychosis within France's existing category of bouffée délirante. In contrast to English speaking western psychiatry, it enjoys the status of being one of the most influential and eminent diagnostic concepts in French psychiatry (Appia, 1964; Pichot, 1986a, 1986b), with up to 38% of patients presenting with acute or chronic paranoid symptomatology, receiving a diagnosis of bouffée délirante (Pichot, 1986).

Bouffée délirante shares commonalities with reactive psychosis in that it also recognises that psychosis *may* result from the impact of psychological stress on a vulnerable personality and the psychotic symptoms are relatively short with a return to premorbid adjustment (Allodi, 1982; Hatotani, 1996; Pichot, 1986a, 1986b). The French construct highlights a sudden onset of delusional ideas and the rapidly evolving intense symptoms conferring different and often changing contents such as a polymorphous psychosis (e.g. megalomania, persecution, hypochondrias) that remits after a short time (Pichot, 1986a).

Although there are phenomenological similarities to the psychogenic psychoses described, bouffée délirante does not *necessitate* the presence of a stressor. Fundamentally the concepts are varied as the aetiology of reactive psychosis is embedded in a history of trauma on a vulnerable personality in contrast to bouffée délirante whose aetiology is embedded in an understanding of degeneration. It is interesting how one can fit the idea of a more reactive psychosis within the degeneration theory. Magnan postulated that in these acute psychotic episodes, degeneration was a static concept for the individual, so that the process of degeneration could occur over generations but not necessarily imply progressive deterioration in the individual patient (Appia, 1964).

Magnan's original degeneration theory was heavily critiqued and his work made way for French psychiatry's influence by Kraeplin (Appia, 1964; Pichot 1986 a & b). Bouffée délirante was reintroduced by Henri Ey (1900 – 1977) and soon came to dominate French Psychiatry (Ey, Bernard, & Brisset, 1960). It was included in the first official French diagnostic system (INSERM, 1969) and has been operationalised (Pull, Pull, & Pichot, 1984, 1987).

3.2.3 Hysterical psychosis

Freud and Breuer initially discussed hysterical psychosis at the beginning of the last century, although it lost favour as a diagnostic category until Hollender and Hirsch's (1964) report. Their report used Freud's extension of his work on hysteria as the foundation for their understanding of hysterical psychosis. Freud's (1896) second

paper on neuropsychoses suggested that psychotic symptoms, in this state of hysteria, were due to a failure in repression and in response to a stressor. The psychotic symptoms function to (a) permit the eruption of material out of awareness and (b) modify the ego function concerned with evaluating reality.

Hollender and Hirsch (1964) added that hysterical psychosis was characterised as having a sudden and dramatic onset, temporally related to a stressor, with a duration of less than three weeks (generally), with few residual symptoms, resolving as rapidly as it began but relapses are likely. Furthermore it is more commonly seen in women with a hysterical personality. Symptoms include hallucinations, delusions, depersonalisation, grossly unusual behaviour, volatile affectivity, and only transient circumscribed thought disorder (Modestin & Bachmann, 1992). Importantly, the condition is amenable to psychotherapy. Although their report saw a flurry of resurgent interest in the diagnosis, varied attention has been given to aspects of the diagnosis.

Some have emphasised the variable onset, variable course and its influence by the social environment. Cavenar, Sullivan and Maltbie (1979) distinguished it from acute schizophrenia by stating hysterical psychosis was characterised by emotions that have been engendered by an overt sexual advance or rage and disappointment over a lack of a sexual advance. There has been little conceptual consistency in the construct, some arguing that it is essentially hysteria with psychotic symptoms (Mallett & Gold, 1964).

Despite these differences, the descriptions of hysterical psychosis given by Hollender and Hirsch (1964) most closely resemble the reactive psychosis construct. They share similar features in that both suggest that the clinical picture of psychosis develops in the context of a vulnerable (hysterical) personality, meaning the person has limited coping mechanisms to deal with stressors. The ego's weaknesses are apparent when a person reacts to a crisis by feeling distraught. As anxiety increases, the person becomes overwhelmed and an altered ego state resulting in hallucinations and delusions is experienced. The psychotic symptoms are not functioning as a defense as in those with a psychotic structure (Freud, 1896), but rather seen as a disruption of the ego function or breakdown of the ego boundary (Federn, 1952). The

content of the psychotic symptoms is shaped by the nature of the precipitating situation. Consistent with the proposal that psychotic experiences exist on a continuum with normal experiences, this report argued that a less vulnerable or hysterical personality is needed for a psychotic break when the stressor is more severe (i.e. in combat or biological stress such as exhaustion and malnutrition). To reemphasise one of the key differentials of hysterical or reactive psychosis, it is the ability to quickly reintegrate the ego function.

A case example is as follows:

The patient, a 45-year-old widow, had worked as a housemother in an orphanage so that she could raise and support her daughter. After 20 years, her daughter, who had finished college, left to establish a life of her own. Other housemothers pointed out to the patient that she was still relatively young and attractive and she might get married now that she had fulfilled her responsibilities to her daughter. Reluctantly, she had accepted their advice. Family members found a suitable suitor for her, a widower 10 years her senior. After going out with him twice, she developed an acute psychotic disorder which took the form of fears that men were breaking into her house and of hallucinations in which she was being called a prostitute or something similar. She was admitted to the psychiatric service of a general hospital. Her therapist suggested that the stress of marriage, after being a widow so many years, would be too much for her, and he recommended that she return to a position similar to her former one. The psychotic symptoms cleared up in less than a week and she was discharged from hospital (Hollender & Hirsch, 1964: p. 1069-1070).

3.2.4 Cycloid psychoses

Cycloid psychosis (CP) was recognised by German psychiatrist Wernicke as a distinct psychotic disorder more than 100 years ago. The diagnosis has also seen dissent and evolution, as with other psychogenic/reactive psychoses. Interestingly, Wernicke, as early as 1900, recognised that this group generally had a good prognosis (Wernicke, 1900). The early descriptions were of anxiety psychosis, characterised by

anxious affect leading to psychotic symptoms, often paranoia. His second classification described motility psychosis whereby people displayed motor symptoms including hyperkinetic, akinetic and mixed forms. His main focus was to classify these disorders on pathogenic hypothesises in terms of dysfunction of brain systems and he was not concerned with prognosis or aetiology, thereby not differentiating anxious or motility psychosis from deteriorating psychotic disorders (Pillman et al., 2000).

Cycloid psychosis was further elaborated on by Kleist (1926) and Leonhard (1961) who added a third form, anxiety-elation psychosis (Leonhard, 1957) which added to Wernicke's' anxious-psychosis, an elated mood accompanied by the patient's desire to please others. In their final classification, cycloid psychoses (Leonhard, 1957) consisted of three distinct forms and were characterised by a phasic course, remission without residual symptoms and a bipolar appearance (anxiety-elation psychosis, hyperkinetic-akinetic motility psychosis and excited-retarded confusional psychosis).

Perris and Brockington (1981) combined these phenomenologies to reflect a psychological state distinguished from other reactive psychoses by the presence of psychodelic experiences, dysperceptions, derealisation and depersonalisation. A recent study found that a diagnosis of cycloid psychosis (using Perris' & Brockington's formulation) was associated with perplexity, mood-incongruent delusions, pananxiety, mood swings and concern with death. Importantly there was significant overlap with brief psychotic disorder, bipolar disorder and schizophrenia. Psychiatric prevalence rates have varied from 8-24% (van der Heijden, Tuinier, Kahn, & Verhoeven, 2004).

Traditional cycloid psychosis diagnoses have been lost in the ICD-10 system. Van der Heijden et al., (2004) argued that it is likely they are being picked up elsewhere and noted that there may be an overlap between cycloid psychosis and personality disorders, namely, borderline personality disorder.

3.2.5 Schizophreniform psychoses – Langfeldt

Langfeldt differentiated schizophreniform psychoses from what he termed *process* schizophrenia, in which prognosis was invariably poorer. He designated five types of psychosis under schizophreniform including (1) preponderantly endogenically conditioned (constitutional) forms (2) neurosis-like, psychogenically more comprehensible cases, (3) symptomatic and other predominantly exogenically (infectiogenic, traumatogenic, psychogenic) precipitated psychoses, (4) cases belonging to the manic-depressive group of psychoses, and (5) real atypical schizophrenias (Langfeldt, 1982). In a letter to the British Journal of Psychiatry in 1982, addressing the confusion in his terminology, he was emphatic that his description of psychoses had 'nothing to do' with the DSM definition of schizophrenia and other psychotic classifications.

3.2.6 Atypical psychosis

Atypical psychoses have similar presentations to those described above and have been used as short hand to describe unclassifiable psychotic disorders termed "psychotic disorders not otherwise specified" in DSM-III and DSM-III-R (APA, 1980; 1987). The classification includes conditions such as post-partum psychosis that does not meet its full criteria, disorders that do not meet the duration criteria, and psychotic presentations with more obscure symptoms that do not fit other diagnoses.

Atypical psychoses have commonly been applied to psychotic illnesses in Japan since Hisatoshi Mitsuda's initial Japanese publication in 1941. He described the clinical course of atypical psychosis as kaleidoscopic in appearance, rapid fluctuations with initial mood disturbances and confusional states followed by hallucinations and delusions (Mitsuda, 1965). Prognosis, like reactive states, is encouraging with some exceptions. Mitsuda linked these conditions seen in Japan with epilepsy, likening the disturbance of consciousness, episodic course, and electro-encephalographic disturbances. He also reported higher incidences of epilepsy in the relatives of people with atypical psychotic presentations (Mitsuda, 1965). For Mitsuda, these diagnoses are situated at the border of schizophrenia, manic depression and epilepsy.

3.2.7 Culture bound syndromes

A clinical picture of acute psychoses with good prognosis is common in developing countries (Wig, 1990). In Susser and Wanderling's (1994) WHO 10 nation study the incidence of 'nonaffective acute remitting psychosis' was found to be ten times higher in developing countries than in industrialized countries. There is some evidence that some of these cases may have a different clinical profile. In an Indian study Pandurangi and Kapur (1980) found more cases of histrionic behaviours, excitement and sleep disturbances and less confusion, irritability and depression than the Scandinavian studies.

Clinicians in non-western countries have heralded the usefulness of considering acute brief psychoses for some time, arguing that western classification of psychoses does not accurately capture the clinical picture that emerges in their psychiatric settings (Langness, 1967). These culturally specific syndromes have been referred to as culture-bound syndromes.

Terms such as Yak, latah, koro, whitigo psychosis and amok among others, are used (Langness, 1967). Culture-bound syndromes provide examples of how psychotic illness may appear as socioculturally sanctioned behaviour expressing extreme distress. In Native American tribes, ghost sickness refers to the condition where the sufferer may be obsessed with death or a deceased person whom they believe to be the source of their affliction. As well as presenting with physical symptoms such as weakness and fatigue, diminished appetite, or other digestion problems, psychological symptoms may occur including nightmares, other sleep disturbances, anxiety, or a sense of being in danger, hallucinations or confusion (APA, 1994). In Southeast Asia, terms such as Amok (meaning mad with rage) and Latah (where sufferers may lose control of their behavior, mimic the speech and actions of those around them and sometimes obey any commands given) are commonly used. These culturally sanctioned disorders have increasingly become recognised in western psychiatry and were included in the fourth version of the *Diagnostic and Statistical Manual of Mental Disorders* (APA, 1994) under the term culture-bound syndrome. The term culture-bound syndrome denotes:

> Recurrent, locality-specific patterns of aberrant behavior and troubling experience that may or may not be linked to a particular DSM-IV diagnostic category. Many of these patterns are indigenously considered to be 'illnesses', or at least afflictions, and most have local names. Although presentations conforming to the major DSM-IV categories can be found throughout the world, the particular symptoms, course, and social response are very often influenced by local cultural factors. In contrast, culture-bound syndromes are generally limited to specific societies or culture areas and are localized, folk, diagnostic categories that frame coherent meanings for certain repetitive, patterned, and troubling sets of experiences and observations. (p. 844)

3.3 Reactive Psychoses in ICD and DSM Classification Systems

Reactive psychosis was first included in international classification systems with the 8th edition of the International Classification of Disorders (ICD-8: WHO, 1967), and the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R: APA, 1980). The shift toward removing subjectivity from diagnosis meant that reactive psychosis no longer occupied a separate diagnostic category with each revision of the ICD (WHO, 1978; 1992) and DSM (APA, 1987; 1994). The definition of reactive psychosis became narrower than Jaspers' original concept and reactive psychosis was subsumed under ICD-9 as reactive depression, reactive excitation, reactive confusion and acute paranoid reaction.

In ICD-10, the closest description to the original reactive psychosis diagnosis is subsumed under the category of acute transient psychotic disorders (ATPD: WHO, 1992). This category was devised to encapsulate not only reactive psychosis but also some of the historical diagnoses discussed earlier such as bouffée délirante, cycloid psychosis and schizophreniform. ATPD is described as:

> A heterogeneous group of disorders characterized by the acute onset of psychotic symptoms such as delusions, hallucinations, and perceptual disturbances, and by the severe disruption of ordinary behaviour. Acute onset

> is defined as a crescendo development of a clearly abnormal clinical picture in about two weeks or less. For these disorders there is no evidence of organic causation. Perplexity and puzzlement are often present but disorientation for time, place and person is not persistent or severe enough to justify a diagnosis of organically caused delirium. Complete recovery usually occurs within a few months, often within a few weeks or even days. If the disorder persists, a change in classification will be necessary. The disorder *may or may not* be associated with acute stress, defined as usually stressful events preceding the onset by one to two weeks (WHO, 1992: p. 121).

This reduces the presence of a stressor to a specifier or moderator and requires a complex judgement of the interaction between personality, stressor and psychosis. In essence a clinical judgement is warranted. There are 6 subtypes in the ICD-10 classification:

- Acute polymorphic psychotic disorder without symptoms of schizophrenia
- Acute polymorphic psychotic disorder with symptoms of schizophrenia
- Acute schizophrenia-like psychotic disorder
- Other acute predominantly delusional psychotic disorders
- Other acute and transient psychotic disorders
- Acute and transient psychotic disorder, unspecified (apparently linked to the original reactive psychosis).

In agreement with Ungvari and Mullen (1997) these subcategories are of 'dubious' heuristic value and certainly do not capture most cases otherwise described as reactive. In Castingini's (2007) examination of the Danish Psychiatric Central Register, a diagnosis of reactive psychosis was recorded in 19.2% of patients with functional psychoses in 1992–1993. The overall prevalence of ATPD dropped to only 8.7% of those with non-organic psychotic and affective disorders in 1994–1995. Others reported similar findings, calling for a reform in the operationalism of ICD-10

criteria (Mojtabai, Varma, & Susser, 2000). Although there are only a few studies of this kind, it is concerning that many cases of reactive psychosis may be missed in the revised classification system.

In the latest edition of the DSM (APA, 1994), the diagnosis with closest resemblance to reactive psychosis is brief psychotic disorder with marked stressor. Again, this diagnosis reduced the importance of the presence of a stressor to a specifier or moderator implying that the psychosis was not necessarily reactive, thus negating the core criterion. Furthermore, psychoses must be limited to one month. One advantage of the ICD-10 is that allows acute and transient psychoses to last up to 3 months (WHO, 1992). A further critique is that the international classification systems do not adequately capture Jaspers' original construct as they exclude schizotypal personality disorder and ignore emotional reactions (Jauch & Carpenter, 1988).

Although it may be difficult to recognise and separate a vulnerable personality during a presentation of acute psychosis, there is a significant body of theory and research linking a vulnerable personality to reactive psychoses. The psychoanalysts Stern (1938) and Kernberg, (1986) propagated the idea that borderline personality was indicative of somebody 'bordering on' psychosis and drew attention to the occurrence of brief psychotic episodes.

Personality difficulties such as shyness, oversensitivity and anxiety have more commonly been observed in reactive psychosis patients compared with their non-psychotic siblings (McCabe, 1975) and non-reactive psychoses (Kapur & Pandurangi, 1979). Others have found a greater prevalence of cyclothymic and suspicious traits in the pre-morbid personality of those with reactive psychosis compared with controls (Chavan & Kulhara, 1988).

Furthermore, Koenigsberg, Kaplan, Gilmore and Cooper (1985) found that of patients with Brief Reactive Psychosis (APA, 1980), 18% met the criteria for borderline personality disorder, compared with one per cent of other psychotic patients in their sample. Others have reported an association between Borderline personality and other personality disorders (such as antisocial personality disorder)

and unspecified or non-schizophrenia-like psychoses (Clarke, Hafner & Holme, 1995; Ekselius, Von Knorring, Lindstrom & Persson, 1994; Jorgensen, Bennedsen, Christensen & Hyllested 1996).

3.4 Epidemiology and Outcome Studies

Owing to the varying criteria used to define and operationalise reactive psychosis, there is a scarcity of raw data on the incidence and prevalence of reactive psychosis with the exception of a few studies from the Nordic countries. Scandinavian national statistics estimated that the life-time risk of reactive psychosis was 0.3-1% (Faergeman, 1963; Stromgren, 1974). Their national registries estimated that between 13-30% of all psychiatric admissions are diagnosed with reactive psychosis (Daahl, 1986). In Denmark, a review of first psychiatric admission registries between 1970 and 1988 found that a diagnosis of reactive psychosis was more than five times as likely as a diagnosis of schizophrenia (Jorgensen & Mortensen, 1992).

To date there are no studies examining the outcome for those diagnosed with the Scandinavian concept of reactive psychosis but some have researched the outcomes of the related constructs outlined here. The following is a brief summary of some of these seminal studies.

Pillman, Haring, Balzuweit, Blöink and Marneros' study (2002) looked at differences between DSM-IV brief psychotic disorder and 'positive' schizophrenia and found proportionately more females in the brief psychotic disorder group, indicating that the diagnosis is a different entity. This group had as many relapses as the schizophrenia group, no deterioration in their functioning, and were just as likely to be have been treated with medication. Despite frequent relapse, the brief psychotic disorder group had much better social and occupational outcomes including occupational status, relationships, independent living, functioning in social roles, psychological impairment, and global functioning than the schizophrenia group. Pillman et al. (2002) concluded that they cannot categorically answer the question whether it is more appropriate to regard brief psychotic disorder and schizophrenia as

parts of one psychotic continuum or as different nosological entities. Their study's unique gender ratio seems to separate brief psychotic disorder from schizophrenia and the high level of relapse contradicts the notion that it is a less severe form of psychotic illness.

Further support for separating reactive-type psychoses from schizophrenia comes from the work of Jager who examined the descriptive validity of ICD-10 schizophrenia (Jager, Bottlender, Strauss & Hans-Jurgen, 2003). Their study found that those with schizophrenia had more negative symptoms at assessment and discharge, lower global functioning and more first rank symptoms at discharge compared to those with acute and transient psychotic disorders (Jager et al., 2003). Other studies have found more favourable outcomes for ICD-9 hysterical psychosis or reactive/psychogenic psychosis compared with schizophrenia (Modestin, & Bachmann, 1992).

Another important consideration when establishing the validity of a diagnosis is how stable the diagnosis is. Several follow up studies have indicated varying rates of change of diagnosis during subsequent episodes (Jorgensen, Bennedsen, Christensen, & Hyllested, 1997; McCabe, 1975; Susser et al., 1995). From these studies, Jauch and Carpenter (1988) concluded that at least half of patients with an original diagnosis of reactive psychosis maintain their diagnosis.

3.5 Commonalities Among Reactive Psychosis Constructs and Contrasts with Schizophrenia

The following illustration from Ungvari and Mullen (1997) of a case of reactive psychosis exemplifies the core features of the clinical picture including perplexity, distress and psychotic symptoms whose meaning and content was understandable in terms of the precipitant stressor:

> A 19-year old student presented at the emergency room in a distressed state. She was speaking rapidly and constantly repeating that something dreadful would happen to her. She had a perplexed air and was disoriented in time but not place. She reported auditory hallucinations accusing in nature. She was

admitted and given night sedation. The next day she was less agitated but still expressing fears of being in danger though the source of the threat was ill defined. She was markedly self-referential and clearly puzzled by the meaning of even the most innocent remark or mundane occurrences. A history was obtained from her mother and later amplified by the patient. She was an academically able young woman, though somewhat shy and oversensitive. She had become involved at university in a relationship with a fellow student who though reputedly talented was erratic and known to be abusing a range of drugs. She had become pregnant and, under pressure from the young man and her parents, had an abortion 2 months previously. A week prior to her admission the young man had died as a result of what was presumed to be an accidental overdose of opiates. Prior to the admission, her parents had been astonished at how well she had coped with events. She remained hallucinated and self-referential over the next week but with a gradual decrease in the intensity of the distress and psychotic symptomatology. Ten days after admission, she showed no continuing abnormalities of mental state but was able to express understandable sadness and anger about recent losses (p. 52-53).

In summary, it is apparent from the literature that there is variation in which aspects constitute a reactive psychosis clinical picture, or at the very least, which are being emphasised. This makes it difficult to advocate for its inclusion in classification systems. It is important to elucidate which factors of reactive psychosis are common amongst the varying proponents.

In summary, commonalities in reactive psychosis constructs arising from the aforementioned review are:

- Presence of a trauma
- Background of vulnerable personality
- Acute or rapid onset of psychotic symptoms
- Short duration of psychotic symptoms
- Return to premorbid functioning

- Confusional states

Less commonly emphasised is that the nature of the trauma is reflected in the content of the psychotic symptoms. One explanation could be the subjective nature of its assessment.

Reactive psychosis diagnosis differs from the diagnostic criteria of schizophrenia, which emphasise a minimum of six-month duration of symptoms. The presence of trauma (although common, see review on trauma and psychosis), a background of vulnerable personality and confusional states may occur in individuals with schizophrenia, but they are not required for a diagnosis of schizophrenia.

Cycloid psychosis appears to be the only reactive psychosis related diagnosis highlighting the possibility of an episodic course, which may be important given its implications for the medium to long-term outcome. Although not a part of the diagnostic criteria for brief psychotic disorder, it is worth noting that Pillman et al. (2002) found a pattern of relapses in those diagnosed with brief psychotic disorder.

3.6 A Brief Note on Treatment

A thorough review of treatments for psychotic disorders is beyond the scope of this chapter but it is worth highlighting that the seminal researchers in the field of reactive psychosis advocate that anti-psychotic medication is useful in the early stage of the condition (McGlashan & Krystal, 1995; Modestin & Bachmann, 1992; Stevens, 1987) and high-dose or prolonged medication is not usually required (Jamminga & Carpenter, 1982). Almost all would agree that supportive psychotherapy should also be considered as first line treatment alongside medication (Murphy, 2000).

3.7 Criticisms of the Reactive Psychosis Concept

Much criticism has been levelled at the reactive psychosis concept. The majority of it is concerned with the disagreement amongst clinicians as to how to operationalise the construct, with some clinicians emphasising different criteria than others, and then the limited reliability in diagnosing reactive psychosis (Lewis, 1972).

Some researchers have emphasised the temporal relationship between the onset of the stressor and psychosis (Jorgensen & Jensen, 1988; Munoz, Amado, Hyatt, 1987), however classically this was not considered important (Faergman, 1963; Jaspers, 1963).

There is little agreement as to whether organic factors can be classified as stressors sufficient to induce a psychotic break. Some of the European researchers have found that infections, operations, sleep deprivation, physical exhaustion that induce a psychotic break can be conceptualised within the reactive psychosis construct (Faergeman, 1963; Jaspers, 1963; Stromgren 1974) whereas others have maintained the artificial distinction between organic and non-organic psychoses (Jorgensen & Jensen, 1988; Opjordsmoen, 2001).

3.8 Development of the Reactive Psychosis Rating Form

In an attempt to address the growing dissatisfaction with the reactive psychosis concept, Hansen et al. (1992) prepared 30 case histories of patients that were treated for Reactive Psychosis at the Department Psychiatry, University of Oslo. Each patient was rated according to three systems, the ICD-10, DSM-IV and the Nordic traditional diagnostic system. The interrater reliability for reactive psychosis was adequate at .52-.84. Furthermore, the interrater reliability of reactive psychosis was comparable to schizophrenia and for affective psychosis in ICD-9. In the Nordic systems there were slight differences in Kappa between schizophrenia, affective psychosis and reactive psychosis.

The same group of researchers also attempted to improve the operationalisation of the construct and establish its reliability (Guldberg et al., 1996). This led to the development of the Reactive Psychosis Rating Form (RPPF) that delineated four major factors in the operationalisation of reactive psychosis; Acute onset, a precedent traumatic event, short duration and good outcome. Their study on the same 30 case summaries of reactive psychosis demonstrated that the RPRF has adequate construct and discriminant validity (Guldberg et al., 1996). The variable *favourable outcome* was considered a prognostic factor so was excluded from the factor analysis. Three factors emerged with eigen values greater than 1, accounting

for 68% of the variance. The three factors that emerged were: *Stressor* which incorporated the variables severity of stressor, meaning, content of psychosis, and presence of perplexity, confusion or emotional turmoil and the internal consistency (standardised Cronbach's alpha) was 0.69. The second factor that emerged was called *onset* and encompassed the variables onset of stressor and duration of stressor and the internal consistency was 0.91. The third factor *change* incorporated highest GAF before the onset of psychosis and the development of the psychosis (internal consistency 0.69). The content and meaning of the psychosis, originally included in the definition of reactive psychosis by Jaspers (1963), as well as the severity of the stressor failed to reach adequate interrater reliability. This may be due to the subjective nature of the variables, particularly severity of stressor (Guldberg et al., 1996).

Guldberg et al.'s (1996) study represents the first attempt to reliably establish a diagnostic criterion for reactive psychosis. This is critical in an era where reliability of psychotic diagnosis, particularly schizophrenia is becoming increasingly important, even if some of it is at the sacrifice of validity (P. Dudgeon, personal communication, 2002). However Guldberg's observations were derived from a very small sample size where one may argue the appropriateness of performing a factor analysis. Furthermore, although the case summary technique they employed to establish diagnoses may have been convenient, particularly considering the multi-centre nature of the trial, this was at the sacrifice of the richness of data one may need to establish such a holistic diagnosis. The current study draws on Guldberg's work and examines the relevance and outcome of reactive psychosis within a first-episode psychosis population, using the Reactive Psychosis Rating Form. A more detailed discussion of the rationale and objectives of this study will follow in chapter 4.

Chapter 4: Rationale and Objectives of the Study

In summary, reactive psychosis is a psychiatric diagnostic construct with minimal prominence in western psychiatry due to inconsistency in what constitutes reactive psychosis, lack of empirical evidence and increasing criticism regarding the subjective nature of the construct. Whilst acknowledging these criticisms, it is perhaps important to recognise that failing to identify reactive-type psychoses could have consequences for the treatment of psychotic disorders, particularly in the early phase. The current clinical guidelines for early psychosis recommend maintenance antipsychotic medication for 1 to 2 years after the resolution of psychotic symptoms, irrespective of initial clinical presentation (International Early Psychosis Association Writing Group, 2005). If there is an identifiable subgroup of individuals with reactive psychosis who have an inherently better prognosis, these guidelines may need to be further considered.

Guldberg et al.'s (1996) study represents a first attempt at addressing the criticisms, however what is required are larger studies, using carefully operationalised diagnostic criteria, examining the incidence of this reactive psychosis within psychiatric samples. An early psychosis service that has a strong research impetus and treats patients with a first-episode of psychosis who are neuroleptic naïve, is an ideal location for taking the next step in the study of reactive psychosis. This study aims to identify a group of early psychosis patients who meet the criteria for reactive psychosis and determine whether they differ from a non-reactive psychosis group in ways that are consistent with the reactive psychosis construct.

The first objective of the current study is to establish the prevalence of the reactive psychosis diagnosis in a sample of 217 young people (aged 16-30) experiencing their first episode of psychosis in an Australian psychiatric setting. Criticism levelled at the subjective nature of the reactive psychosis concept led Dahl and Guldberg to attempt to operationalise reactivity. They devised the Reactive Psychosis Rating Form (Guldberg et al., 1996) that included all components central to the Scandinavian concept of reactive psychosis, except for personal predisposition (deemed difficult to assess without separate personality inventory). These include, (a)

severity of stressor, (b) onset of stressor, (c) duration of stressor, (d) development of psychosis, (e) meaning of psychosis, (f) content of psychosis, (g) perplexity, (h) highest pre-GAF, (i) post-GAF, and (j) duration of psychosis. Although they operationalised the criteria, there were no guidelines as to where to establish a cut off score for each item or how many items need to be endorsed to warrant a diagnosis. Although best conceptualised as a dimensional construct (personal communication Guldberg, 1996), the strength of the large sample size in this study would be adequate to determine a set of criteria for a diagnosis of reactive psychosis.

4.1 Aims and Objectives 1

Hypothesis 1: There will be an identifiable group of patients within a first episode population who meet the criteria for reactive psychosis according to the RPRF.

4.2 Aims and Objectives 2

Prior to undertaking an analysis of clinical outcome for the reactive psychosis groups, a description of general sample characteristics and baseline clinical variables will be provided and examined in order to highlight any similarities and differences between the groups in addition to identifying any potential confound variables. Consistent with the literature review, it is expected that there will be no differences in level of psychopathology or social and occupational functioning between the reactive and non reactive groups at baseline. In contrast, it is expected that there will be proportionately more females in the reactive psychosis group and differences in onset variables such as acuity and length of untreated illness as these onset variables form part of the reactive psychosis diagnosis.

Hypothesis 2a: There will be no significant differences between reactive psychosis and non-reactive psychosis groups at baseline on the level of general psychopathology.

Hypothesis 2b: There will be no significant differences between reactive psychosis and non-reactive psychosis on the severity of positive psychotic symptoms at baseline.

Hypothesis 2c: There will be no significant differences between the reactive psychosis and non-reactive psychosis groups on the severity of negative symptoms at baseline.

Hypothesis 2d: There will be no significant differences between the reactive psychosis and non-reactive psychosis groups on level of social and occupational functioning at baseline.

Hypothesis 2e: The reactive psychosis group will have a significantly shorter duration of prodromal symptoms than the non-reactive psychosis group

Hypothesis 2f: The reactive psychosis group will have a shorter duration of untreated psychotic symptoms than the non-reactive psychosis group.

4.3 Aims and Objectives 3

The third objective of this study is to determine the clinical outcome of individuals identified as experiencing a reactive psychosis. There is a range of possible ways to define outcome and, as noted in Chapter 1, there is limited consistency in defining outcome with the exception of some agreement that both symptomatic recovery and functional recovery should be considered, and others arguing for the inclusion of negative symptoms. This section will examine symptomatic recovery, whilst functional recovery will be considered in the following objective. Andreasen et al. (2005) attempted to address inconsistencies in the research field and operationalised recovery in the domains general psychopathology and negative symptoms.

The conceptualisation of reactive psychosis suggests a more favourable clinical outcome than other psychotic disorders. Specifically, it is expected that there will be a shorter duration of symptoms and a return to premorbid functioning, hence the need to examine these variables. While the reactive psychosis construct suggests a return to premorbid function, the literature does not identify a specific timepoint by which this would be expected. There is also a difficulty in establishing premorbid functioning in an adolescent population given that the illness impacts on ongoing development. However it is expected that individuals experiencing reactive psychosis will have a more favourable outcome on clinical dimensions such as duration of psychosis, recovery from positive psychotic symptoms, negative symptoms, and

general psychopathology (which includes other psychotic symptoms such as mannerisms and posturing, motor retardation, and disorientation). Since some historical accounts connected affective states with reactive psychosis, mood symptoms will also be examined both as an outcome and as a potential confound. It is noted however that affective states did not emerge among the commonalities in reactive psychosis constructs identified in Chapter 3.

In addition to examining these clinical dimensions, one of the objectives of this study is to determine whether people with a diagnosis of reactive psychosis use fewer acute and outpatient services over the course of their treatment and lastly, whether they are prescribed lower doses of antipsychotic medication and for shorter periods of time. It is expected that there will be no differences in service use and medication in the first 3 months due to comparable levels of illness acuity. However the reactive psychosis group is expected to use fewer services and medication at 9 and 15 months, consistent with shorter duration of symptoms. These variables are being used as proxy measures of remission, given that a more severely unwell patient will be more likely to use both acute and outpatient services and require more medication.

Given the exploratory nature of this study and it being one of the few studies to attempt to empirically describe the clinical characteristics of reactive psychosis in the early stages of psychosis, identified confound variables will be noted for further discussion and results interpreted with caution. Consistent with other studies, it is likely this study will find differences in distribution of gender between groups but the literature indicates no straightforward relationship between gender and outcome in first episode psychosis. The contribution of gender to outcome in reactive psychosis is beyond the scope and design of this exploratory study and will therefore not be controlled for in analyses.

Duration of untreated psychosis is likely to be different between the groups as it is related to rapid development of psychoses, which forms part of the diagnostic criteria for reactive psychosis and therefore will not be treated as a confounding variable in *this* design. Again any potential contribution to outcome will be noted for discussion.

The various outcome variables described above will be measured at four time points:

(1) Entry to the service

(2) 3 months of treatment (recovery/stabilisation phase)

(3) 9 month follow up

(4) 15 month follow up.

Hypothesis 3a: The reactive psychosis group will have a significantly shorter duration of psychosis and experience higher rates of remission from their psychotic episode at the 3-month follow up point than the non reactive psychosis group.

Hypothesis 3b: The reactive psychosis group will have significantly less severe general psychopathological symptoms than the non reactive psychosis group at 3, 6 and 15 month follow up points.

Hypothesis 3c: The reactive psychosis group will have significantly less severe positive psychotic symptoms than the non reactive psychosis group at 3, 6 and 15 month follow up points.

Hypothesis 3d: The reactive psychosis group will have significantly less severe negative symptoms than the non reactive psychosis group at 3, 6 and 15 month follow up points.

Hypotheses 3e: There will be no differences between the reactive psychosis and non reactive psychosis group on the severity of depressive and manic symptoms at 3, 6 and 15 month follow up points.

Hypothesis 3f: There will be no significant differences between groups in the degree of outpatient service use during the acute phase (first 3 months).

Hypothesis 3g: The reactive psychosis group will use outpatient services significantly less than the non reactive psychosis group at 9 and 15 month follow up.

Hypothesis 3h: There will be no significant differences between groups in the degree of acute service use (YAT and inpatient unit) during the acute phase (first 3 months).

Hypothesis 3i: The reactive psychosis group will use acute services (YAT and inpatient unit) significantly less than the non reactive psychosis group at 9 and 15 month follow up.

Hypothesis 3j: There will be no significant differences between groups in the dose or likelihood of being prescribed an antipsychotic medication during the acute phase (first 3 months).

Hypothesis 3k: The reactive psychosis group will have significantly lower doses and be less likely to be prescribed an antipsychotic medication than the non reactive psychosis group at 9 and 15 month follow up.

4.4 Aims and Objectives 4

This study advocates a more broad definition of outcome highlighting the importance of considering social and occupational functioning and quality of life in the course and recovery of psychosis. One of the criteria in considering a diagnosis of reactive psychosis is the rapid return to premorbid functioning, it is therefore expected that the reactive psychosis groups will be less impaired in this domain.

Hypothesis 4a: The reactive psychosis group will have significantly better social and occupational functioning than the non reactive psychosis group at 3, 6 and 15 month follow up points.

Hypothesis 4b: The reactive psychosis group will have significantly better quality of life than the non reactive psychosis group at 3, 6 and 15 month follow up points.

4.5 Aims and Objectives 5

There are historically varied presentations subsumed under the category or that share similarities with reactive psychosis. The fifth objective of this study is to determine whether reactive psychosis is closely related to other psychotic diagnoses, including traditional German and French psychiatric diagnoses and more contemporary systems such as ICD-10 and DSM-IV. ICD-10 and DSM-IV have narrowed the diagnostic construct of reactive psychoses with each revision and it may be important to consider if any cases of reactive psychosis, as conceptualised in this study, are being lost in the current revisions of these diagnostic systems. The aim here is an exploratory one and there are no specific predictions or hypotheses:

Aim: *To examine the correlations of reactive psychosis diagnosis with other historical and contemporary classifications of psychotic disorders.*

Chapter 5: Method

5.1 Organisational Context

The study was conducted at The Early Psychosis Prevention and Intervention Centre (EPPIC), established in 1992 (McGorry, Edwards, Mihalopoulos, Harrigan, & Jackson, 1996). The program services young people aged between 15 and 30 years of age who reside in the western and north-western regions of Melbourne and are experiencing their first episode of psychosis. First-episode is defined as at least two weeks of active psychotic symptoms with not more than six months of prior treatment with antipsychotic medication. The program has a large research component that has affiliations with The University of Melbourne's Psychiatry and Psychology Departments.

5.2 Methodological Approach and Rationale for the Research Design

The study was a naturalistic, prospective, three, nine and fifteen-month follow up study after initial treatment. Initial treatment is defined as three months of low dose atypical anti-psychotic medication and intensive case management as a minimum standard. Clients are also offered family therapy and access to a group program.

This study design is most appropriate to be able to map the clinical course and outcome of a group of first episode psychotic clients. The data collection had been completed. Therefore this study used archival data previously collected by the current investigator.

The study was subject to ethics approval from the Royal Park Research and Ethics Committee and all participants signed written consent forms. Two hundred and seventeen consecutive-admission clients consented to the study during the recruitment period of 1994 to 1998, with approximately 20% of patients who met criteria refusing participation.

Inclusion criteria required that all patients be aged 16-30, experiencing their first episode of psychosis, have no prior history of treatment for a psychotic illness (defined as no more than six months of previous anti-psychotic medication), and be fluent in English. Clients with an intellectual disability or a clear organic cause for psychosis were excluded from the study, the rationale being that the psychiatric assessment tools utilised in this study have not been validated in these populations. Patients underwent organic screens, including MRI scans, drug screens, and physical examinations to exclude drug-induced psychosis, dementia, and delirium.

5.3 Instruments

5.3.1 Diagnoses

5.3.1.1 Royal Park Multidiagnostic Instrument for Psychosis.

Individuals were diagnosed according to a semi-structured instrument, the Royal Park Multidiagnostic Instrument for Psychosis (RPMIP: McGorry, Copolov, & Singh, 1990; McGorry et al., 1994; see Appendix A) which allows diagnoses of psychotic disorders to be made according to a variety of contemporary nosological systems, including the DSM-III/III-R & IV criteria (APA, 1980; 1987; 1994), ICD-10 (WHO, 1992) and historical diagnoses.

The historic diagnoses that will be used include the following:

- Schneider schizophrenia
- E Bleuler Schizophrenia
- M Bleuler Schizophrenia
- Langfeldt Schizophrenia
- Kraepelin Schizophrenia
- SCAAPS Acute Psychotic Episode
- Cycloid Atypical psychosis
- Dongier Atypical psychosis
- Bouffée délirante atypical psychosis
- Cloninger Schizophrenic symptom scale

- Taylor & Abrams Schizophrenia
- Feigner Schizophrenia
- Feigner schizoaffective disorder
- RDC unspecified functional psychosis.

The RPMIP also derives a past and current diagnosis of major depressive disorder and bipolar disorder (according to DSM-IV: APA, 1994) by mapping the history and course of affective disturbance. Onset and offset dates of affective disorders are assessed and documented using a combination of patient interview, review of case notes and, where possible, an informant interview with a carer. This procedure allows raters to calculate an estimate of the number of days a person experienced affective symptoms (both manic and depressive).

The RPMIP assessment procedure derives its diagnoses based on combining ratings undertaken at both admission and three months. A specific illness duration interview (using an informant interview where possible), part of the RPMIP procedure, provided a reliable estimate of Duration of Untreated Psychosis (DUP).

Furthermore, the RPMIP measures the duration of psychosis (as measured at 3-month follow up) by mapping and dating the course of the disorder in the same manner as mapping affective disorders and duration of untreated psychosis. That is, a combination of semi-structured interview, review of case notes and informant interview where possible. This procedure allows the rater to calculate an estimate of the number of days a person was psychotic up until their second assessment (3-months after entry into the service). The criteria for onset and remission of psychosis are consistent with DSM-IV criteria for a psychotic disorder (APA, 1994) and Andreasen et al.'s (2005) remission criteria, respectively.

Completion of the RPMIP is estimated to take between 6 and 7 hours, including at least 2 hours of patient interview time, 1 hour for each illness duration or informant interview, and at least 2 hours to collect relevant information from casenotes, collation of all information, and application of diagnostic decision rules.

The only interrater reliability study (see McGorry et al., 1990b) for the RPMIP used a first-episode psychosis sample assessed at the Aubrey Lewis Unit, Parkville. The study produced K values > .6 for all diagnostic categories, except for DSM-III's schizoaffective disorder, atypical psychosis and nonpsychotic depression, attributed to low base rates of these diagnoses in the sample (McGorry et al., 1990b). Adequate reliability was demonstrated with a mean K value of .7 calculated for 260 of the individual items (McGorry et al., 1990b). Formal interrater reliability has also been established for the measurement of onset and duration of symptoms in the RPMIP, with a mean K of 0.79 (McGorry et al., 1990b). Further to this, each rater was required to attend weekly group supervision which was facilitated by experienced clinicians and researchers, where ratings were discussed and consensus was being consistently established and developed. See Harrigan, McGorry and Krstev (2003) for more details about the measurement of duration of untreated psychosis.

5.3.1.2 Reactive psychosis.

The Reactive Psychosis Rating Form (RPRF; Guldberg et al., 1996, see appendix B) was used to derive a diagnosis of reactive psychosis. As it is a diagnostic tool, the interview questions pertinent to the RPRF's and subsequent scale items were incorporated and were therefore completed as part of the RPMIP interview. The instrument was reviewed in Chapter 3 of this thesis but a brief summation is as follows. In the original development of their scale, Guldberg et al. (1996) used the RPRF to rate a sample of 30 case summaries of reactive psychosis, previously diagnosed by expert clinicians in the Nordic Multicentre study of reactive psychosis (Hansen et al., 1992). Their study on the same 30 case summaries of reactive psychosis demonstrated that the RPRF has adequate construct and discriminant validity (Guldberg et al., 1996). Despite the limited interrater reliability for some of the variables measured, including content and meaning of psychosis and severity of stressor, we proceeded to use the scale simply because there are no expert clinicians in Australia to produce a 'gold standard' diagnosis. This study did not produce any information specifically about inter-rater reliability for the RPRF. However, as mentioned in the section on the RPMIP, raters were thoroughly trained.

The final 9-item scale was used in this study, measuring the following constructs (see appendix) on a 6-point scale:

1. Severity of stressor
2. Onset of stressor (time between stressor and onset of psychosis)
3. Duration of stressor
4. Development of psychosis (rate of onset of psychotic symptoms)
5. Meaning of psychosis
6. Content of psychosis
7. Perplexity
8. Highest pre-GAF/Post-GAF
9. Duration of psychosis

A brief description of each item is as follows.

1. Severity of stressor: The difficulty in evaluating traumatic life events is well known, given the subjective nature of them (Creed, 1993). For the purposes of this study a stressor is defined as "a change in the external environment which occurs sufficiently rapidly to be dated in a well-defined way (Guldberg et al., 1996; p 115)" and axis IV of the DSM-III-R is used to measure the severity of the event (Zimmerman, Porhl, & Stangl, et al., 1985). The questions were open-ended questions and were framed to elicit what the patient had considered a life stressor, then objectively and consistently rated according to the DSM-IIIR axis IV. The limitations of this are the attempts to rate severity of stressors objectively e.g. change of residence may be more stressful for one person than for another, but for the purposes of consistency, each stressor that was elicited was rated using the anchors on axis IV of the DSM-III-R (see Appendix B for a copy of the DSM-III-R Severity of Psychosocial Stressors Scale).

2. Onset of stressor was measured (again on a 6 point scale) as the time between the onset of the stressor and prominent psychotic symptoms, therefore not including the onset of prodromal symptoms but rather the onset of full threshold psychotic symptoms that met EPPIC's criteria for a psychotic episode, i.e. 2 weeks of prominent psychotic symptoms. While Guldberg et al. (1996) argued that precipitating causal events can occur several months before the onset of psychosis

(Bebbington, Wilkins, Jones et al., 1993), a shorter period between stressor and symptoms is considered an indicator of greater reactivity.

3. Duration of stressor: When rating the stressor, what is taken into consideration is the actual length of the traumatic event, and not its effects. For example, domestic violence may endure for 12 months or more, but the sudden death of a loved one, although the effects may be long lasting, lasts less than 48 hours, and hence would be scored 6. Unfortunately Guldberg et al. (1996) gave no clear rationale for why considering the duration of the stressor was important to the diagnosis of reactive psychosis. However, a key component of reactive psychosis is the reactivity of onset. Therefore one may consider that a sudden disruption in one's life or environment is more likely to produce the disorganisation of psychosis than a more ongoing stress (eg sudden death of a loved one may be more acutely disorganising than a long illness culminating in death where some anticipation and adaptation my occur over time). It doesn't necessarily mean sudden death is more stressful, just that it may be more acutely disorganising. Severity of stress, time of onset and level of sudden disruption are all relevant – sometimes they might all be aligned (all rate high) but not necessarily.

4. Development of psychosis (rate of onset of psychotic symptoms): This item relates to the time taken for prominent psychotic symptoms to appear. Rate of onset is defined as the time from the first noticeable symptoms or changes in behaviour to the first appearance of prominent psychotic symptoms, registered either by personal observation or by others. The DSM-IV definition of prodromal/residual phase was used and consists of **two** of symptoms of affective flattening; alogia; avolition; loss of interest; odd beliefs; magical thinking; unusual perceptual experiences; peculiar behaviour; digressive, vague, overelaborate or metaphorical speech; marked impairment in personal hygiene or grooming; or social isolation/withdrawal (APA, 1994). For example, a person may have experienced social withdrawal and low-grade hallucinations such as hearing his or her name being called or murmuring once or twice a week for 2 months before experiencing daily auditory hallucinations for at least a week. The onset of psychosis in this example would be considered 2 months. Further information on establishing the onset of full threshold psychosis can be found in Yung, Phillips, Yuen et al., (2003).

5. Meaning of the psychosis: This item requires a complex judgment on the part of the rater in order to determine how the psychotic symptoms can serve a meaningful purpose as an escape from the "psychic pain" triggered by an apparent conflict or stressor. A psychosis can serve as an escape from the emotional distress in a conflict or stressor **without** implying that this should lead to the content of the psychosis reflecting the stressor or conflict. An example of this is when the psychopathological picture of the psychosis is dominated by confusion, perplexity or disorientation, i.e. "confusional states" or "hysterical psychosis". Another illustration of this point is as follows: A woman's husband is unfaithful and she subsequently develops delusions that she is an important doctor. This woman would score 5-6 on this item, as she is escaping the hurt and humiliation (escape from pain) elicited by her husband's infidelity, even though the *content* of her delusion is not clearly related to the stressor.

6. Content of the psychosis: This item also requires a complex judgment as to how psychotic symptoms can reflect the stressor or conflict Guldberg et al., (1993). In the example of the woman whose husband had the affair, instead of developing delusions that she is an important doctor, she may develop delusions that she is unworthy, and that it is her fault the infidelity occurred, that she is an alcoholic, and this is why her husband left her. The delusions of worthlessness and guilt would rate highly on this item also (5-6), as she is reflecting the blame of the incident (symptomatically) upon herself.

7. Perplexity: is rated on a scale of 1 to 6 ranging from very mild (2) to misidentifications (4) and marked spatial and temporal disorientation (6).

8. Highest pre-GAF/Post-GAF compares functioning premorbidly with functioning post psychosis: It is also a scale measuring 1-6, anchor points ranging from moderate to severe reduction in functioning (1) to improved functioning post psychosis (6). Note that patients who continue to experience full threshold psychotic symptoms at the second point of assessment score a zero as being unable to be assessed. The 3-month assessment point is used as it is based on the assumption that

most people will have recovered from their psychotic episodes, despite possibly experiencing residual symptoms.

9. Duration of psychosis: This item relates to the duration of the psychotic symptoms only (not prodrome), according to the criteria set out by Yung et al. (2003), until their first remission with treatment. The criteria for onset and remission of psychosis are consistent with DSM-IV criteria for a psychotic disorder (APA, 1994) and Andreasen et al.'s (2005) remission criteria, respectively. Information for the number of days a patient experienced psychotic symptoms is based on clinical interview, i.e. retrospective patient report, and clinical notes and is derived from the RPMIP illness duration interview.

5.3.2 Symptom measures

5.3.2.1. Scale for the Assessment of Negative Symptoms (SANS).

The Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1982; Andreasen 1984; Andreasen, Larsen & Schultz, 1989; Appendix C) is a 25-item measure of negative symptoms that consists of five subscales; affective flattening or blunting, alogia, avolition-apathy, anhedonia-asociality and attention. Andreasen et al. (1989) demonstrated good internal consistency for the SANS with alpha coefficients not reported lower than 0.63. Similarly, Mueser, Sayers, Schooler, Mance and Haas (1984) reported adequate internal consistency, coefficient alphas for each subscale ranging from 0.51 to 0.93, $p < 0.05$. Discriminative validity for negative symptom distinction has also been established. Andreasen and Olsen (1982) were able to discern individuals suffering from predominantly negative versus positive symptoms in domains such as education levels, age at admission and cognitive performance.

5.3.2.2 Brief Psychiatric Rating Scale.

The expanded 24-item version of the Brief Psychiatric Rating Scale (BPRS version 4.0; Lukoff, Liberman, & Nuechterlein, 1986, see Appendix D) was used in this study. This version is an improvement on the 18-item version (Overall & Gorham, 1962) as it was devised for outpatient settings, had improved anchor points

for the scale and was accompanied by a manual with extensive examples. The 21-item semi-structured interview assesses the degree of psychopathology, both observed (e.g., tension and excitement) and interview-elicited (e.g., unusual thought content). The items pertain to three broad areas. The first assesses the patient's responses in a clinical setting to questions about depression, anxiety, hallucinations and delusions. The second set comprises observational data pertaining to irrelevance of speech, flattened or incongruous affect, poverty of speech and retardation. The third set of items measure the Parkinsonian side effects of anti-psychotic medication.

The BPRS is used for the measurement of change in psychotic symptoms during treatment and also used to determine relapse of psychotic symptoms. A psychotic relapse is defined by a score of ≥ 4 on items eliciting suspiciousness, unusual thought content, hallucinations or conceptual disorganization.

Sound psychometric properties in the original version have been demonstrated in a number of studies. Although traditionally a measure of overall psychotic state, Overall (1974) identified the following 5 factors, through factor analysis; thought disorders, emotional withdrawal, anxiety-depression, aggressiveness and agitation. Excellent interrater reliability coefficients have been established for the expanded version of the BPRS with scores ranging from .93 to .74 for interview elicited items (Ventura Green, Shaner, & Liberman, 1993). Ventura's study demonstrated good median Pearson and intraclass coefficients ranging from .67 to .88 (Ventura et al., 1993). A study using nurse-raters established good reliability for the acute presentation of first-episode psychosis. Weighted Kappas for the interview-elicited items ranged from .90 to .78, and the lowest Kappa for observation-based items was calculated at .63 (McGorry, Goodwin, & Stuart, 1988).

5.3.3 Social and occupational functioning measures

5.3.3.1 Premorbid adjustment.

Premorbid adjustment was measured using the Premorbid Adjustment Scale (PAS; Cannon-Spoor, 1982; see Appendix E). The PAS is a 26-item instrument, with scoring for each item ranging from 0 to 6. Scores are derived for early childhood (0 to

11 years), early adolescence (12 to 15 years), and late adolescence (16 to 18 years) in four domains (a) sociability-isolation; (b) peer relationships; (c) ability to function outside of the nuclear family; and (d) capacity to form intimate sociosexual ties. A more general global estimate is then made based in the highest level of functioning that the person achieved before becoming ill. Only the general score was used in our study. An internal consistency analysis has revealed a Cronbach alpha of .92 in an EPPIC sample (n = 238) (S. Harrigan, personal communication, June 15, 2001).

5.3.3.2 Quality of Life.

Quality of Life was assessed using the Quality of Life Scale (QLS; Heinrich and Carpenter, 1986 see Appendix F). It is a 21-item, semi-structured interview that measures four constructs including intrapsychic functioning, interpersonal functioning, occupational functioning and level of involvement in common place activities. This scale has demonstrated high sensitivity to both change and treatment effect and moderate-to-high correlations with other measures of quality of life (Cramer et al., 2000).

5.3.3.3 Social and Occupational Functioning Assessment Scale (SOFAS).

The Social and Occupational Functioning Assessment Scale (SOFAS: Goldman, Skodol & Lave, 1992; see Appendix G) was used as a global measure of the individual's present level of social and occupational functioning. The rating of overall psychological functioning was first operationalised by Luborsky in the Health-Sickness Rating Scale (Luborksy, 1962) and the combined SOFAS was derived from the Global Assessment Scale (Endicott, Spitzer, Fleiss & Cohen. 1976; Goldman, 1992). Participants are rated from 0 to 100 where a score of 0 to 10 indicates persistent inability to maintain personal hygiene; unable to function without harming self or others or without considerable external support (e.g., nursing care and supervision), a score of 41 to 50 indicates a serious impairment in social, occupational, or school functioning (e.g., no friends, unable to keep a job) and a score of 81 to 90 indicates good functioning in all areas, occupationally and socially effective This scale is included in the DSM-IV and although related to the Global Assessment of Functioning Scale (GAF)

it focuses exclusively on the patient's social and occupational functioning and is not directly influenced by the patient's psychological symptoms (APA, 1994) .

5.3.4 Service use

The Early Psychosis Service Utilisation and Outcome Questionnaire (EPSUOQ; see Appendix H) is a locally derived instrument that aims to track service utilisation throughout the follow up period. It also assesses and tracks relapse of psychosis, mood disorders, anxiety symptoms, drug and alcohol problems and medical problems (Mihalopolous, Carter & McGorry, 1999). It uses both interview elicited material, where patients are asked about the dates of onset and recovery of symptoms throughout each assessment time point, using time anchors and prompts to ascertain dates with as much precision as possible (e.g. using birthdays, anniversaries, seasons, school semester dates etc). This patient elicited information is used in combination with information gleaned from the patient's clinical file. Convergent validity had been calculated by comparing patient responses with patient file data. The intraclass correlation coefficient was calculated at .68 (Mihalopolous et al., 1999). This instrument proved insufficient in being able to accurately assess relapse throughout the course of the follow up period. Relapse is measured according to (a) whether somebody was psychotic at each assessment point and (b) how many days a person experienced psychotic symptoms between assessment time points. It is important to note that the instrument does not adequately distinguish between relapse and non-recovery from index episode nor does it accurately derive an estimate as to a psychotic relapse and potential recovery between follow up periods.

5.4 Procedure

The RPMIP interview was started when patients first presented to the service and completed three months after commencement of treatment at EPPIC, a time referred to as the recovery/stabilisation period, when participants were considered to be more settled (with the diagnoses being based on a composite of admission and discharge ratings). This procedure has been adopted by the research team at the Early Psychosis Research Centre since 1986. This procedure is regarded as more valid than

a cross-sectional assessment of diagnosis as it allows the researcher to capture any marked changes in the individual's clinical picture (McGorry et al., 1990; McGorry et al., 1994).

Several trained research assistants, including the current investigator, with at least four years psychology training also rated patients on a range of psychopathological and psychosocial functioning scales at four time points, including; (i) treatment entry, (ii) recovery/stabilisation (completed within three months of entry), (iii) nine months post admission, and finally (iv) 15-months post admission. Patients were contacted either by telephone, letter or through their case manager at the time of their follow-up and assessments were usually conducted onsite. As an appreciation of their time and effort (as well as to cover transportation costs) all patients received $20 for their participation in the 9 and 15-month follow up. Table 2 outlines the assessment procedure during the study period.

Table 2. *Outline of assessment procedure for course of study*

Measure	Baseline (admission into EPPIC)	Recovery/ stabilisation (3-month follow up)	9- month follow up	15- month follow up
Royal Park Multidiagnostic Instrument for Psychosis	*	* completed		
Reactive Psychosis Rating Form	*	* completed		
Scale for the Assessment of Negative Symptoms	*	*	*	*
Brief Psychiatric Rating Scale	*	*	*	*
Premorbid Adjustment Scale	*	*completed		
Quality of Life Scale		*	*	*
The Social and Occupational Functioning Assessment Scale	*	*	*	*
The Early Psychosis Service Utilisation and Outcome Questionnaire		*	*	*

5.5 Method of Data Analysis

A correlational matrix will be used to assess the reliability of the Reactive Psychosis Rating Form. Regression analyses or time series analyses were considered in the statistical design of this study but given the nature of this exploratory study and there being missing data at each follow up time point, a decision was made not to impute data and a cross sectional statistical design was considered more appropriate. The presence of missing data was the result of some patients being unable to be reached for a particular follow-up assessment or, due to time constraints, information was missing to appropriately assess or score an item on a psychiatric scale. Furthermore, the purpose of this study was to examine the validity of reactive psychosis as a diagnostic entity in itself, rather than examining its symptoms separately or taking a dimensional approach. Therefore, Analysis of Variance (ANOVA) will be used in order to ascertain any differences between the reactive psychosis and non-reactive psychosis groups in the aforementioned baseline and outcome variables. It is important to acknowledge the risks of false positive inferences associated with large numbers of analyses. In light of this, the results will be interpreted with caution and attention paid to the overall pattern and consistency of any findings. Although attention will be given to identifying possible confounds for future study, examining the statistical contribution of reactive psychosis over and above other predictors of psychosis was beyond the scope of this study. Due to the very high positive skewness of many of the clinical variables, it will be necessary to transform these variables prior to analysis. In order to explore the relationship between various diagnostic systems, a correlational matrix will be performed.

Chapter 6: Results

6.1 Reliability of Reactive Psychosis Rating Form

The first step in testing the hypotheses is to assess the reliability of Guldberg et al.'s (1998) Reactive Psychosis Rating Form. In order to establish the reliability of the scale a reliability analysis was performed on the first 7 items of the scale that will be used to establish reactive psychosis groups. Items 8 and 9 (duration of psychosis and pre vs post psychotic functioning) were not included in the reliability analysis as they are considered prognostic outcome variables in themselves that are fundamental to the hypotheses of the thesis. Table 3 details the inter-item correlation matrix. Cronbach's α was considered adequate at .764 indicating satisfactory internal consistency of the reactive psychosis rating form items. Repeating the reliability analysis with all 9 items yielded a similar result ($\alpha = .749$). A closer look at the correlations shows that the item *degree of perplexity* was actually negatively correlated with several items and not correlated with the remaining items. When this item was removed, Cronbach's alpha increased to .812, considered good internal consistency. The item *degree of perplexity* was therefore removed from the scale.

Table 3. *Inter-Item Correlation Matrix for Reactive Psychosis Rating Form*

	Time between stressor and onset of psychosis	Duration of stressor	Severity of stressor	Development of psychosis	Degree of perplexity	Psychological meaning of the psychosis	Psychological content of the symptoms
Time between stressor and onset of psychosis	-						
Duration of stressor	.637	-					
Severity of stressor	.538	.587	-				
Development of psychosis	.269	.072	.028	-			
Degree of perplexity	-.056	-.108	-.027	.082	-		
Psychological meaning of psychosis	.471	.358	.688	.059	.000	-	
Psychological content of symptoms	.549	.435	.722	.071	.034	.902	-

The covariance matrix is calculated and used in the analysis.

6.2 General Sample Characteristics

The general sociodemographic characteristics of the sample are described in this section. A more comprehensive examination of sociodemographic and illness related baseline characteristics will be presented according to reactive psychosis groupings in Section 6.4. The sample of 217 first episode patients ranged from age 16-30, with an average age of 22.7 (SD = 3.35). There were more males in the sample than females (73% males), a trend often seen in first episode psychosis research. Twenty seven percent of the sample had commenced or completed tertiary education, with 54.2% of the sample having not completed secondary school education. Eighty one percent of the sample were single or had never been married and 67% were living with their parents at the time of initial assessment. Only 7% of first episode psychosis patients were living on their own. Nineteen percent of first episode psychosis patients were born overseas and 57% had at least one parent born overseas.

6.3 Establishing Reactive Psychosis Groups

In order to determine a threshold for classification of reactive psychosis, it was necessary to examine the frequency with which each item of the reactive psychosis scale was endorsed. Tables 4 to 12 describe the frequency of endorsement of items in the reactive psychosis rating form. In the sample of 217, 65.5% of first episode psychosis patients had experienced a moderate to extreme stressor prior to the onset of their psychosis, 29% had experienced a stressor within 3 months of the development of psychosis and 50% had a stressor that lasted less than 3 months. Sixty two percent had a rapid onset of psychosis that developed within 3 months of the onset of prodromal symptoms. Although it was omitted from subsequent analyses, it was important to note that only 14.3% had experienced significant perplexity (misidentification to severe spatial disorientation). Thirty four percent of patients' psychosis had an identifiable psychological meaning and for 40.6% of patients the psychological content of their stressor was reflected in their psychosis. Thirty eight percent of patients' psychosis lasted less than 3 months and 20.7% had returned to previous functioning or had improved post psychotic functioning.

Table 4. Severity of stressor

Score	6. Extreme	5. Severe	4. Moderate	3. Mild-moderate	2. Mild	1. Very mild	0. No stressor or impossible to rate	Total
Frequency	20	65	57	21	3	1	50	217
Percent	9.2	30	26.3	9.7	1.4	.5	23	100

Table 5. Time between stressor and onset of psychosis

Score	6. Within 48 hrs	5. 2-7 days	4. 1-4 wks	3. 1-3 mths	2. 4-12 mths	1. >12 mths/ continuous	0. no stressor or impossible to rate	Total
Frequency	0	5	28	30	50	47	57	217
Percent	0	2.3	12.9	13.8	23	21.7	26.3	100

Table 6. Duration of stressor

Score	6. < 48 hrs	5. 2-7 days	4. 1-4 wks	3. 1-3 mths	2. 4-12 mths	1. >12 mths/ continuous	0. No stressor/ Impossible to rate	Total
Frequency	58	17	18	16	20	25	63	217
Percent	26.7	7.8	8.3	7.4	9.2	11.5	29	100

Table 7. Development of psychosis

Score	6. < 48 hrs	5. 2-7 days	4. 1-4 wks	3. 1-3 mths	2. 4-12 mths	1. >12 mths/ continuous	0. uncertain	Total
Frequency	16	39	36	45	29	39	13	217
Percent	7.4	18	16.6	20.7	13.4	18	6	100

Table 8. Degree of perplexity

Score	6. Marked spatial disorientation	5. Spatial temporal disorientation	4. Misidentification	3. Perplexed, simple clouding	2. Very mild	1. Not present	Total
Frequency	9	11	11	51	31	104	217
Percent	4.1	5.1	5.1	23.5	14.3	47.9	100

Table 9. Psychological meaning of psychosis

Score	6. Clearly related	5.	4 Probably related	3	2 Possibly related	1 Not understand able	n/a	total
Frequency	15	21	38	21	49	23	50	217
Percent	6.9	9.7	17.5	9.7	22.6	10.6	23	100

Table 10. Psychological content of psychosis

Score	6. Clearly reflects stressor	5	4. Some aspects reflected	3	2. Not clear if it reflects stressor	1. Clearly not related	n/a	total
Frequency	16	18	54	37	28	14	50	217
Percent	7.4	8.3	24.9	17.1	12.9	6.5	23	100

Table 11. Duration of psychosis

Score	6. Within 48 hrs	5. 2-7 days	4. 1-4 wks	3. 1-3 mths	2. 4-12 mths	1. >12 mths/ continuous	Total
Frequency	0	2	22	59	91	43	217
Percent	0	.9	10.1	27.2	41.9	19.8	100

Table 12. Premorbid vs post psychotic functioning

Score	6. Improved function	5. Mild improve	4. Same functioning post psychotic	3. Mild reduction	2. Moderate reduction	1. Moderate to severe reduction	0. Uncertain	Total
Frequency	6	12	27	30	59	43	40	217
Percent	2.8	5.5	12.4	13.8	27.2	19.8	18.4	100

Given we do not have a 'gold standard' for reactive psychosis in this population, the Norwegians determined the diagnosis via consensus diagnosis by expert clinicians, who were also proponents of the reactive psychosis construct. To our knowledge, no such experts exist within the Australian context, as typically the diagnosis of reactive psychosis is not considered in Australian psychiatric settings. Instead, we have relied on the Reactive Psychosis Rating Form in order to determine a diagnosis of reactive psychosis. Guldberg and colleagues did not define a cut off for what they considered the threshold to be for a reactive psychosis diagnosis. A number of methods were considered in attempting to define a cut-off, including adding up the items in the scale to obtain a whole reactive psychosis score and then determining a cut off point. Although it may be considered a statistically adequate method, this method would not give much face validity for a diagnosis of reactive psychosis. An alternative method would be more in keeping with the DSM-IV-TR (APA, 2000) method of determining a psychiatric diagnosis, in which a certain number of criteria need to be endorsed at a moderate to severe level in order to meet criteria for a diagnosis. This method would provide more face validity for the reactive psychosis construct.

Therefore it was decided that patients needed to satisfy the following criteria to meet a diagnosis of reactive psychosis:

(A) Presence of a stressor: the central tenet of the reactive psychosis diagnosis is that psychosis develops in response to a stressful life event

(B) Patients needed to endorse at least 4 out of the 6 items. Although this may be considered a somewhat arbitrary number, in the absence of a 'gold standard' for diagnosing reactive psychosis in a first episode population and there being no previous attempts to operationalise the criteria, meeting at least two thirds of the reactive psychosis criteria could be deemed adequate to warrant the diagnosis. This approach is consistent with the polythetic system used in the DSM.

(C) In order to meet the criteria for a particular item patients need to score at least 'moderate' on the item (ie. 4 or above). A score of 4 or above on the reactive psychosis items approximates some of the criteria for a brief psychotic disorder (BPD) with marked stressor (DSM-IV; APA, 1994).

a. for example in BPD the duration of psychosis is less than month (score of 4 or higher on reactive psychosis scale),

b. the psychosis develops within 4 weeks if it is a postpartum onset (time of onset between stressor and psychosis is at a maximum of 4 weeks, indicated by a score of 4 on the reactive psychosis scale),

c. and the presence of a marked stressor (although not adequately defined in DSM-IV); a score of 4 on the reactive psychosis scale is considered a moderate stressor.

Each participant's individual items were rated according to whether he or she scored 4 or above on that item. Items 8 and 9 (duration of psychosis and pre vs post psychotic GAF) were not included in any further analyses as they are considered prognostic outcome variables that are fundamental to the hypotheses of the thesis. As stated earlier, degree of perplexity was removed from the reactive psychosis scale as it was not correlated with the other reactive psychosis criteria.

Participants who had a stressor present and who met the criteria on at least 4 of the 6 items on the reactive psychosis scale were grouped into the "Reactive psychosis" group. Those that did not were grouped into the "non-reactive psychosis" group.

The proportion of the sample who met the reactive psychosis criteria is shown in Table 13. Sixty-three first episode patients were identified as having reactive psychosis whilst 154 were in the non- reactive psychosis group. Twenty nine per cent of the first episode sample fell into the reactive psychosis group. Thus in accord with hypothesis 1 there was an identifiable group who met the criteria for reactive psychosis according to the RPRF and the criteria developed for this study.

Table 13. Reactive psychosis diagnostic groups

	Frequency	Percent
Reactive psychosis group	63	29
Non-reactive psychosis group	154	71
Total	217	100.0

6.4 Sociodemographic Characteristics of Reactive Psychosis and Non-Reactive Psychosis Groups

The baseline demographic and illness related variables were examined for both groups so as to identify any confounding variables that may impact upon clinical outcome. The average age of the reactive psychosis and non-reactive psychosis groups were 22.08 (SD 3.43) and 22.34 years (SD 3.32), respectively. Analysis of variance revealed no significant differences between the groups in age (F= .279;df = 1, 215, p =.598).

A chi-square test revealed significant differences between groups in gender representation $\chi^2(2, N=217)=7.61, p=.006$. The adjusted residual values indicated that there were proportionately more females in the reactive psychosis group than in the non reactive psychosis group.

There were no differences between the three groups in other sociodemographic variables such as educational achievement $\chi^2(2, N=216)=1.90$, p=.168, marital status $\chi^2(2, N=217)=.001, p=.971$, living arrangements [living on own,

χ^2(2, N=216)=.145, *p*=.704; living with parents, χ^2(2, N=216)=.444,*p*=.505], likelihood of being born overseas χ^2(2, N=217)=.119, *p*=.730 or parents being born overseas (*p*=.791 & .360 for mother and father being born overseas respectively).

A total of 29 patients (13.4%) had at least one parent with a psychotic illness, 12.3 percent of the non-reactive psychosis group had at least one parent with a psychotic illness compared with 15.9% of the reactive psychosis group. A chi square analysis did not reveal significant differences between the groups [χ^2(1, N=217)=.483, p=.487].

Although females were more likely to be represented in the reactive psychosis group this difference was noted for further discussion but was not controlled for in subsequent analyses due to its undetermined influence on clinical outcome. The next stage of the analysis was to examine if the groups differed on any baseline illness related variables (assessed at entry to the clinical service).

6.5 Illness Related Characteristics and Level of Psychopathology at Admission to the Service for each Reactive Psychosis Group

This section, after reviewing age of onset and premorbid adjustment, will report on analyses examining hypotheses 2(a) to 2(f). Tables 14 to 19 display the data for the selected baseline illness related variables considered to have an impact on clinical outcome. Examination of the central tendency of the data revealed that several variables were skewed but non-transformed data are presented in the tables. One-way ANOVAs revealed no significant differences in age of onset of psychosis between the groups (F=.391; df=1, 215; p=.533).

Table 14. Age of onset for non-reactive and reactive psychosis groups

Groups	Mean	SD	Skewness	Standard error skewness	Kurtosis	Median	min	max
non-reactive psychosis (N=154)	21.57	3.353				21		
reactive psychosis (N=63)	21.89	3.502				21		
Total (N=217)	21.66	3.391	.136	.165	-.634	21	13	29

Table 15 reports the general subscale from the Premorbid Adjustment Scale. The reactive psychosis group had significantly better premorbid adjustment than the non-reactive psychosis group as indicated by ANOVA (F=3.971; df=1, 199; p=.048).

Table 15. Premorbid adjustment for reactive psychosis groups

Reactive psychosis group	M	SD	Skewness	Standard error skewness	Kurtosis	Median	Min	Max
Non-reactive psychosis (N=144)	.41	.19				.39		
Reactive psychosis (N=57)	.35	.14				.33		
Total (N=201)	.391	.18	.37	.17	-.38	.37	.06	.93

The baseline Brief Psychiatric Rating Scale (BPRS) total score had a skewness coefficient of 3.95. A natural log transformation reduced the coefficient to 1.09, therefore the transformed data was used in the ANOVA which, as predicted by hypothesis 2(a), revealed no significant differences in baseline level of psychopathology as measured by the BPRS (F=.093; df=1,212; p=. 761). A Mann-Whitney nonparametric test confirmed this result $\chi^2(2, N=214)=.641$, p=.726.

The BPRS psychotic subscale, which measures only the core psychotic symptoms, satisfied the assumption of normality. As predicted by hypothesis 2(b), there were no significant differences between the two groups on severity of psychotic symptoms at entry into the service (F=1.788; df=1, 212; p=. 183).

Table 16. Brief Psychiatric Rating Scale (BPRS) and BPRS psychotic subscale (BPRSps) scores for reactive psychosis groups at admission to the service

	Group	M	SD	Skewness	Standard error skewness	Kurtosis	Median	Min	Max
BPRS_1	non reactive psychosis (N=152)	30.70	10.26				29.5		
	reactive psychosis (N=62)	31.23	9.94				30		
	Total (N=214)	30.86	10.15	.66	.17	.31	30.00	12	67
BPRSps_1	non reactive psychosis (N=152)	11.42	3.65				11		
	reactive psychosis (N=62)	10.66	3.96				11		
	Total (N=214)	11.20	3.75	.006	.17	-.1.65	11	1	20

Nb: BPRS_1 & BPRSps_1 denotes total BPRS & total BPRSps score at entry to the service, respectively.

Table 17 displays the descriptive statistics for the Scale for Assessment of Negative Symptoms (SANS) total score as measured at baseline (admission to the service) that had a skewness coefficient of 4.4. A square root transformation reduced the coefficient to 0.12, therefore the transformed data was used in the ANOVA. As predicted by hypothesis 2(c), there were no significant differences between the reactive and non-reactive psychosis groups in their baseline level of negative symptoms as measured by the SANS (F=.397; df=1,213 ; p=.529). A Mann Whitney nonparametric test confirmed this result $\chi^2(1, N=215)=.509$, p=.476.

Table 17. Scale for the Assessment of Negative Symptoms (SANS) scores for reactive psychosis groups at admission to the service (baseline)

	Reactive psychosis group	M	SD	Skewness	Standard error skewness	Kurtosis	Median	Min	Max
SANS_1	Non reactive psychosis (N=152)	23.70	14.24				22		
	reactive psychosis (N=63)	21.88	12.21				21		
	Total (N=215)	23.16	13.67	.73	.17	.10	22.00	1.00	64.00

Nb: SANS_1 denotes total SANS score at admission to the service

Table 18 displays the descriptive statistics for the Social and Occupational Functioning Scale (SOFAS) score as measured at baseline (admission to the service) which satisfied the assumption of normality. As predicted by hypothesis 2(d), there were no significant differences between the reactive and non-reactive groups on this measure of social and occupational functioning at entry into the service (F= 1.606; df=1, 212; p=.206).

Table 18. Social and Occupational Functioning Scale (SOFAS) score for reactive psychosis groups at admission to the service (baseline)

	Reactive psychosis group	M	SD	Skewness	Standard error skewness	Kurtosis	Median	Min	Max
SOFAS_1	Non reactive psychosis (N=152)	45.22	12.36				45		
	reactive psychosis (N=62)	42.85	12.38				40		
	Total (N=214)	44.53	12.39	.44	.17	.25	41	15	90

Table 19 displays the descriptive statistics for the duration of prodromal symptoms and duration of untreated psychotic symptoms in days. Duration of

prodromal symptoms and duration of untreated psychosis are extremely positively skewed variables displaying skewness coefficients of 10.89 and 19.88, respectively. Neither log transformation nor square root transformation brought the skewness coefficient of duration of prodromal symptoms down to a satisfactory level of less than < 3.0. Given that the assumption of normality could not be satisfied, the Mann-Whitney non-parametric test was used which revealed that the reactive psychosis group had a significantly shorter prodromal period than the non-reactive psychosis group $\chi^2(1, N=217)=12.160$, p=.001, supporting hypothesis 2(e).

A log transformation reduced the duration of psychotic symptoms skewness coefficient to -1.04. The log transformed data were used in the ANOVA which revealed significant differences between groups in duration of untreated psychosis (F=22.387; df=1,215; p=.000) with the reactive psychosis group having a shorter duration as predicted by hypothesis 2(f). A Mann-Whitney nonparametric test confirmed this result $\chi^2(1, N=217)=21.812$, p=.000.

Table 19. Duration of prodromal symptoms and untreated psychotic symptoms (no. of days) for reactive psychosis groups

	Reactive psychosis group	M	SD	Skewness	Standard error skewness	Kurtosis	Median	Min	Max
Duration of Prodrome (days)	Non-reactive psychosis (N=154)	584.51	712.39				305.5		
	reactive psychosis (N=63)	315.14	587.06				78		
	Total (N=217)	506.30	688.05	1.80	.17	2.86	212	0	3257
Duration of untreated psychotic symptoms (days)	Non-reactive psychosis (N=154)	249.63	403.80				99.5		
	reactive psychosis (N=63)	69.02	111.89				21		
	Total (N=217)	197.19	354.74	3.28	.17	11.99	62.00	0	2192

6.6 Length of Psychosis, Rate of Remission, Severity of Psychopathology and Service Use Measured at 3 Months, 9 Months and 15 Months for the Reactive and Non-Reactive Psychosis Groups

There were no differences between the groups on level of psychopathology and global functioning at admission to the service, although as expected, the non-reactive psychosis group were unwell for longer periods of time before being treated. The following section outlines the analyses for hypotheses 3(a) to 3(k). Tables 20 to 24 display the descriptive variables for the measures of psychopathology as measured at 3, 9 and 15 months after admission to the service.

6.6.1 Length of psychosis and rate of remission

The length of psychosis was measured by calculating the total number of days a person was psychotic from onset of psychosis until the 3-month assessment point. Given the tendency of first episode patients to recover in the first few months of treatment, the data were skewed, displaying a skewness coefficient of 18.18. A log transformation brought the skewness coefficient down to 0.14, therefore the transformed data were used in subsequent analyses (see Table 20).

Hypothesis 3(a) was supported as the reactive psychosis group had a significantly shorter length of psychosis at 3 months after entry to the service [ANOVA ($F=31.62$; $df=1,215$; $p<0.001$)], confirmed by a Mann-Whitney non-parametric test $\chi^2(1, N=217)=30.98$, $p<0.001$. There was further support for hypothesis 3(a) as for 85.7% of the reactive psychosis group, the psychosis had remitted by 3-month follow up compared with 71.4% of the non-reactive psychosis group. A chi-square test revealed significant differences between groups $\chi^2(1, N=217)=4.94$, $p=.026$.

Table 20. Length of psychosis for reactive psychosis groups

	Reactive psychosis group	M	SD	Skewness	Standard error skewness	Kurtosis	Median	Min	Max
No of days psychotic_3	Non-reactive psychosis (N=154)	313.90	412.56				156.5		
	Reactive psychosis (N=63)	110.02	129.94				68		
	Total (N=217)	254.71	366.08	3.09	.17	10.77	122	7	2237

NB: Means and SDs for the data for the 3-month follow up are based on the **total** number of days people experienced various symptoms from onset to 3-month follow up.

6.6.2 Severity of general psychopathology and positive symptoms

The data for the Brief Psychiatric Rating Scale total score as measured at 3 and 9 months post admission were skewed, displaying skewness coefficients of 5.46

and 4.12 respectively. A square root transformation brought the skewness coefficients down to 0.82 and 0.78, respectively; therefore the transformed data were used in subsequent analyses and are displayed in Table 21. The Brief Psychiatric Rating Scale total score as measured at 15 months post admission to the service satisfied the assumption of normality.

Hypothesis 3(b) was not supported but there was a trend for the reactive psychosis group to have less severe general psychopathological symptoms than the non-reactive psychosis group at 3 months post admission to the service as indicated by ANOVA (F=3.370; df=1,215; p=.068). This trend was not maintained at the 9 month follow up point (F= .921; df=1, 175; p=.339) or the 15 month follow up point (F=.027; df=1,165; p=.869). The low mean scores indicate that both groups made a good recovery by 9 months that was maintained at 15 months post admission to the service.

Table 21. Brief Psychiatric Rating Scale total scores as measured at 3, 9 and 15 months post entry into the service

	Reactive psychosis group	M	SD	Skewness	Standard error skewness	Kurtosis	Median	Min	Max
BPRS_3	Non-reactive psychosis (N= 154)	17.26	9.24				16		
	Reactive psychosis (N= 63)	15.02	9.48				14		
	Total (N=217)	16.61	9.34	.90	.17	.90	16	1	51
BPRS_9	Non-reactive psychosis (N= 124)	14.10	9.86				10		
	Reactive psychosis (N= 53)	12.31	8.20				9		
	Total (N=177)	13.57	9.41	.75	.18	.48	12	0	47
BPRS_15	Non-reactive psychosis (N= 119)	13.46	8.71						
	Reactive psychosis (N= 48)	13.20	9.80						
	Total (N=167)	13.39	9.00	.47	.19	-.24	13	0	43

Table 22 displays the descriptive variables for the Brief Psychiatric Rating Scale psychotic subscale as measured at 3, 9 and 15 months after admission to the service.

The data for the Brief Psychiatric Rating Scale psychotic subscale as measured at 3, 9 and 15 months post admission were skewed, displaying skewness coefficients of 6.84, 6.14 and 5.10 respectively. A square root transformation brought the

skewness coefficients down to -.96, 1.20, and -0.27 respectively, therefore the transformed data were used in subsequent analyses.

Hypothesis 3(c) was partially supported in that the reactive psychosis group had significantly less severe positive psychotic symptoms than the non-reactive psychosis group at 3 months post admission to the service as indicated by ANOVA (F=5.661; df=1,215; p=.018). A Mann Whitney nonparametric test confirmed this result $\chi^2(1, N=217)=5.799$, p=.016. However, there was only a trend for the reactive psychosis group to maintain a better recovery from psychotic symptoms at the 9 month follow up point (F=3.288; df=1, 174; p=.072). A Mann Whitney nonparametric test confirmed this trend $\chi^2(1, N=176)=3.186$, p=.074. These differences were not maintained at the 15 month follow up point (F=.285; df=1,166; p=.594).

Table 22. Brief Psychiatric Rating Scale Psychotic Subscale total score as measured at 3, 9 and 15 months post entry into the service

	Reactive psychosis group	M	SD	Skewness	Standard error skewness	Kurtosis	Median	Min	Max
BPRSps_3	Non-reactive psychosis (N= 154)	4.58	3.80				4		
	Reactive psychosis (N= 63)	3.48	3.86				2		
	Total (N=217)	4.26	3.84	1.13	.17	1.08	3.00	0	19
BPRSps_9	Non-reactive psychosis (N= 123)	3.94	4.28				2		
	Reactive psychosis (N= 53)	2.77	3.63				0		
	Total (N=176)	3.59	4.12	1.12	.18	.37	2.00	0	16.00
BPRSps_15	Non-reactive psychosis (N= 120)	3.95	3.82				0		
	Reactive psychosis (N= 48)	3.65	3.93				1		
	Total (N=168)	3.86	3.84	.95	.19	.39	3.00	0	17.0

6.6.3 Severity of negative symptoms

The data for the Scale of the Assessment of Negative Symptoms total score as measured at 3, 9 and 15 months post admission were skewed, displaying skewness coefficients of 4.29, 4.82, and 4.47 respectively (see Table 23). A square root

transformation brought the skewness coefficients down to –1.41, -1.24, and -1.42 respectively, therefore the transformed data were used in subsequent analyses.

Support for hypothesis 3(d) was limited but there was a trend for the reactive psychosis group to have significantly less severe negative symptoms than the non-reactive psychosis group at 3 months post admission to the service as indicated by ANOVA (F=2.812; df=1,215; p=.095). A Mann Whitney nonparametric test confirmed this trend $\chi^2(1, N=217)=3.122$, p=.077. This trend was not maintained at the 9 month follow up point (F= .404; df=1, 171; p=.526) or the 15 month follow up point (F=2.287; df=1,163; p=.132).

Table 23. Scale for the Assessment of Negative Symptoms (SANS) total score as measured at 3, 9 and 15 months post entry into the service

	Reactive psychosis group	M	SD	Skewness	Standard error skewness	Kurtosis	Median	Min	Max
SANS_3	Non-reactive psychosis (N= 154)	21.79	14.77				14		
	Reactive psychosis (N= 63)	17.92	13.22				17		
	Total (n=217)	20.66	14.47	.73	.17	-.06	18.00	0	63
SANS_9	Non-reactive psychosis (N=121)	18.40	12.83				16		
	Reactive psychosis (N=52)	17.52	13.12				11		
	Total (N=173)	18.14	12.88	.92	.19	1.12	16.00	0	72
SANS_15	Non-reactive psychosis (N=117)	18.12	13.81				14		
	Reactive psychosis (N=48)	14.66	12.52				7		
	Total (N=165)	17.12	13.50	.85	.19	.38	14.00	0	60

6.6.4 Degree of comorbid affective symptoms over the course of the follow up period

The degree of comorbid affective illness over the course of follow up was measured by calculating the total number of days a person was depressed or had manic symptoms between follow up points (except for the first follow up period

where onset of symptoms prior to entry to the service was included). The untransformed data are displayed in Table 24. Again, the data over the follow up period were highly skewed, displaying skewness coefficients over the 3, 9 and 15 month follow up period of 28, 33.2, and 17.35 respectively for depression and 18.93, 42.89, and 79.92 respectively for mania. A log transformation brought the skewness coefficients down to 4.23, 7.43 and 7.75, respectively for depression. The transformed data were used in subsequent analyses and reported here, but the limitations in approximating the assumptions for use in parametric tests, need to be acknowledged. Mann-Whitney non-parametric tests were also performed and results reported.

Hypothesis 3(e) was supported as there were no significant differences between the reactive psychosis groups in degree of comorbid affective illness as measured by total number of days depressed at 3 months [ANOVA (F=1.185; df=1,215; p=.278); Mann-Whitney χ^2(1, N=217)=1.15, p=.285], 9 months post admission [ANOVA (F=.280; df=1,174; p=.598); Mann-Whitney χ^2(1, N=176)=.450, p=.502] or 15-month follow up [ANOVA (F=.011; df=1,151; p=.915); Mann-Whitney χ^2(1, N=153)=.027, p=.870]. Fifty-five people presented with a history of manic symptoms at 3 month follow up, with length of manic symptoms ranging from one week to 168 days in total. Non-parametric tests were used for all time points, as transforming the data did not meet the assumption of normality. A Mann-Whitney non-parametric test revealed a trend at 3 months for the reactive psychosis group to have longer comorbid manic symptoms than the non- reactive psychosis group [χ^2(1, N=217)=3.11, p=.078]. This trend was not maintained for 9 months post admission [χ^2(1, N=176)=.425, p=.515] or 15-months post admission [χ^2(1, N=154)=1.00, p=.317]. Overall there were no statistically significant differences between groups in the number of days they had experienced affective symptoms, suggesting affective symptoms not be considered a confound variable.

Table 24. Degree of affective symptoms over the course of the follow up period for the reactive psychosis groups

	Reactive psychosis group	M	SD	Skewness	Standard error skewness	Kurtosis	Median	Min	Max
No of days depressed_3	Non-reactive psychosis (N= 154)	115.34	287.32				0		
	Reactive psychosis (N= 63)	122.67	372.80				0		
	Total (N=217)	117.47	313.65	4.62	.165	26.85	0	0	2720
No of days depressed_9	Non-reactive psychosis (N=122)	19.20	44.63				0		
	Reactive psychosis (N=54)	22.0	56.69				0		
	Total (N=176)	20.06	48.50	2.96	.18	8.48	0	0	239
No of days depressed_15	Non-reactive psychosis (N=111)	17.65	45.38				0		
	Reactive psychosis (N=42)	18.67	52.66				0		
	Total (N=153)	17.93	47.32	3.47	.20	13.14	0	0	281
No. of days manic_3	Non-reactive psychosis (N=154)	8.71	20.08				0		
	Reactive psychosis (N=63)	15.57	31.24				0		
	Total (N=217)	10.70	23.99	3.12	.17	12.34	0	0	168
No. of days manic_9	Non-reactive psychosis (N=122)	2.08	15.02				0		
	Reactive psychosis (N= 54)	3.91	18.15				0		
	Total (N=176)	2.64	16.01	7.85	.18	66.69	0	0	161
No. of days manic_15	Non-reactive psychosis (N=112)	.95	7.59				0		
	Reactive psychosis (N= 42)	.62	3.01				0		
	Total (N=154)	.86	6.65	9.59	.12	99.73	0	0	74

6.6.5 Service use measured at 3 months, 9 months and 15 months for reactive and non-reactive psychosis groups

The degree to which patients used the service was measured by calculating the number of outpatient appointments attended (to case manager), the number of visits by the Youth Assessment Team (YAT; acute home based treatment team) and the number of days in hospital over the follow up period. In this study, degree of service use was used as a proxy variable for recovery. The data are displayed in Table 25.

The data for the number of outpatient appointments to a case manager as measured at 3 and 9 and 12 months post admission were skewed, displaying skewness coefficients of 9.7, 9.7 and 12.97 respectively. A log transformation brought the skewness coefficient for 3 and 15 month data down to -2.14 and -.10, respectively. A square root transformation brought the skewness coefficient for 9 month data down to 1.55. The transformed data were used in subsequent analyses. There were no significant differences in service use as measured by number of outpatient appointments between the reactive and non-reactive psychosis groups at 3 months post admission [ANOVA (F=.862; df=1,215; p=.431)], supporting hypothesis 3(f). This result was confirmed by a Mann-Whitney non-parametric test [χ^2(1, N=217)=.569, p=.451]. The reactive psychosis group had significantly fewer outpatient appointments at 9-month follow up (F=4.10; df=1,173; p=.044), confirmed by a Mann-Whitney non-parametric test [χ^2(1, N=175) =4.12, p=.042], partially supporting hypothesis 3(g). However, this difference was not maintained at 15-month follow up [(F=.016; df=1,152; p=.899); χ^2(1, N=154) =.006, p=.940].

Table 25. Service use for reactive psychosis groups at 3, 9 and 15 months post entry into the service

	Reactive psychosis group	M	SD	Skewness	Standard error skewness	Kurtosis	Median	Min	Max
No of OCM visits_3 (days)	Non-reactive psychosis (N=154)	8.36	8.24				6.00		
	Reactive psychosis (N=63)	8.40	10.60				6.00		
	Total (N=217)	8.37	8.97	1.66	.17	3.88	6.00	0	48
No of OCM visits_9 (days)	Non-reactive psychosis (N=121)	14.76	11.01				12		
	Reactive psychosis (N=54)	12.56	12.91				11		
	Total (N=175)	13.99	11.65	1.74	.18	4.56	12	0	75
No of OCM visits_15 (days)	Non-reactive psychosis (N=112)	8.89	11.27				5.5		
	Reactive psychosis (N=42)	9.57	12.14				5		
	Total (N=154)	9.08	11.48	2.53	.20	6.07	5	0	48
No of YAT visits_3 (days)	Non-reactive psychosis (N=71)	10.83	18.31				4		
	Reactive psychosis (N=27)	15.74	23.00				3		
	Total (N=98)	12.18	19.71	2.37	.24	5.80	4	0	97
No of YAT visits_9 (days)	Non-reactive psychosis (N=122)	2.94	6.76				0		
	Reactive psychosis (N=54)	3.94	7.87				0		
	Total (N=176)	3.25	7.11	2.30	.18	3.93	0	0	24
No of YAT visits_15 (days)	Non-reactive psychosis (N=111)	4.16	11.52				0		
	Reactive psychosis (N=42)	5.07	14.18				0		
	Total (N=153)	4.41	12.26	3.66	.20	14.41	0	0	72
Total no. of hospital	Non-								

days_3	reactive psychosis (N=154)	17.97	22.67				12		
	Reactive psychosis (N=63)	17.76	18.89				13		
	Total (N=217)	17.91	21.6	1.85	.165	3.85	12	0	108
Total no. of hospital days_9	Non-reactive psychosis (N=123)	5.50	18.68				0		
	Reactive psychosis (N=54)	2.83	8.75				0		
	Total (N=177)	4.69	16.32	5.76	1.83	39.85	0	0	146
Total no. of hospital days_15	Non-reactive psychosis (N=112)	6.12	20.95				0		
	Reactive psychosis (N=43)	2.44	9.97				0		
	Total (N=155)	5.10	18.60	6.06	.20	42.03	0	0	159

The number of visits by YAT (the local acute care Youth Assessment Team) was measured at 3 and 9 and 15 months post admission. The data were skewed, displaying skewness coefficients of 9.88, 12.78 and 18.3, respectively. Log transformation brought the skewness coefficients down to 1.29, 8.02 and 9.26, respectively. Only non-parametric tests were reported if the data were unable to be transformed satisfactorily. There were no significant differences between the reactive psychosis groups in use of acute services as measured by number of visits by YAT at 3 months [(F=.366; df=1,96; p=.547); χ^2(1, N=98)=.625, p=.429], supporting hypothesis 3(h). However unexpectedly, and contrary to hypothesis 3 (i), there were no differences in use of acute services at 9 months post admission [χ^2(1, N=176)=.352, p=.553] or 15 month follow up point [χ^2(1, N=153)=.116, p=.733] .

The total number of days patients spent in hospital was measured at 3 and 9 and 15 months post admission. One hundred and forty two patients had an initial hospital admission during their 'acute phase' or first 3 months of treatment. The average length of hospital stay during the first 3 months was 17.91 days.

The data for length of hospital stay at 3 and 15 month follow up were skewed, displaying skewness coefficients of 11.21 and 30.30, respectively. A log

transformation brought the skewness coefficients down to -1.92 and 10.15, respectively; therefore the transformed data were used in subsequent analyses. There were no significant differences between the reactive psychosis groups in use of acute services as measured by total number of hospital days at 3 months post admission, supporting hypothesis 3(h) [ANOVA (F=1.56; df=1,215; p=.213); χ^2(1, N=217)=.943, p=.332]. There was only partial support for hypothesis 3(i) as there were no differences between groups in the total number of days in hospital at 9 months post admission [ANOVA (F=1.01; df=1,175; p=.318); χ^2(1, N=177)=1.75, p=.185] but the non reactive psychosis group had significantly longer hospital admissions at 15 month follow up [ANOVA (F=3.71; df=1,153; p=.056] as confirmed by a Mann-Whitney non-parametric test [χ^2(1, N=155)=4.62, p=.032].

6.6.6 Dosage of antipsychotic medication and number of days on antipsychotic medication measured at 3 months, 9 months and 15 months for reactive and non-reactive psychosis groups.

The average dose of antipsychotic medication was measured at 3, 9 and 15 months follow up (see Table 26). Several different types of antipsychotic medication had been prescribed, ranging from typical to the newer atypical medications. Therefore each antipsychotic medication was reported in chlorpromazine equivalents. Recording the number of days on antipsychotic medication was included as a variation, after the study had commenced, and therefore was calculated for a subsample of people (N=94).

The data for average dose of medication were highly skewed, displaying skewness coefficients of 15.82, 11.24 and 7, for 3 and 9 and 15 months post admission respectively. A square root transformation brought the skewness coefficients down to 1.48, -0.41 and 0.59 respectively; therefore the transformed data were used in subsequent analyses.

Overall, 97% of patients were prescribed antipsychotic medication at 3-month follow up. Hypothesis 3(j) was supported as there were no differences between groups in likelihood of being prescribed anti-psychotic medication at 3 month follow up (chi-

square analysis revealed that 97% of the non-reactive psychosis and 95% of the reactive psychosis group, were prescribed an antipsychotic medication, p=.408). The average dose of antipsychotic medication (at 3 months) in chlorpromazine equivalents was 185.65 mg. In further support of hypothesis 3(j), there were no significant differences in the dose of antipsychotic medication prescribed between the reactive psychosis groups at 3 months post admission [ANOVA (F=1.66; df=1,202; p=.199)]. This result was confirmed by a Mann-Whitney non-parametric test [χ^2(1, N=204) =.451, p=.451].

The average dose of antipsychotic medication at 9 month follow up was 141.45 mg and, contrary to hypothesis 3(k), there were no significant differences in dosage of antipsychotic medication between groups [ANOVA (F=.766; df=1,170; p=.383)], confirmed by a Mann-Whitney non-parametric test [χ^2(1, N=172) =.537, p=.464]. Interestingly, chi-square analysis showed that 84% of the non-reactive psychosis group and fewer in the reactive psychosis group (75.5%) were prescribed antipsychotic medication at 9 month follow up (p=.183).

The average dose of antipsychotic medication at 15 month follow up was 138.32 mg and there was a trend for the reactive psychosis group to have a lower dosage of antipsychotic medication than the non-reactive psychosis group [ANOVA (F=3.46; df=1,150; p=.065)], confirmed by a Mann-Whitney non-parametric test [χ^2(1, N=152) =3.32, p=.068]. In partial support of hypothesis 3(k), the reactive psychosis group were less likely to be prescribed an antipsychotic medication at 15 month follow up (58.1% compared with 76.6% of the non-reactive psychosis group); [χ^2(1, N=152) =4.32, p=.038].

Table 26. Dosage and number of days on antipsychotic medication for reactive psychosis groups at 3, 9 and 15 months post entry into the service

	Reactive psychosis group	M	SD	Skewness	Standard error skewness	Kurtosis	Median	Min	Max
Average dose of anti-psychotic medication _3 (CPZ equivalence)									
	Non-reactive psychosis (N=145)	193.19	142.05				150.0		
	Reactive psychosis (N=59)	167.13	99.92				150.0		
	Total (n=204)	185.65	131.55	2.69	.17	13.16	150.0	0	1000
No. days on anti-psychotic medication_3									
	Non-reactive psychosis (N=68)	86.13	34.23				87		
	Reactive psychosis (N=26)	78.15	36.19				78.5		
	Total (N=94)	83.93	34.77	.58	.25	1.40	84.5	14	216
Average dose of anti-psychotic medication _9 (CPZ equivalence)									
	Non-reactive psychosis (N=119)	143.91	131.35				100		
	Reactive psychosis (N=53)	135.93	143.12				100		
	Total (n=172)	141.45	134.72	2.08	.185	6.94	100	0	850
No. days on anti-psychotic									

medication_9									
	Non-reactive psychosis (N=118)	147.20	98.92				160.5		
	Reactive psychosis (N=52)	133.33	94.89				157.5		
	Total (N=152)	142.95	97.63	.64	.17	3.1	160.50	0	653
Average dose anti-psychotic medication _15 (CPZ equivalence)									
	Non-reactive psychosis (N=109)	149.04	14.61				105.56		
	Reactive psychosis (N=43)	111.16	21.88				50		
	Total (N=152)	138.33	150.55	1.40	.20	1.72	100	0	617.95
No. days on anti-psychotic medication_15									
	Non-reactive psychosis (N=100)	164.33	124.06				163		
	Reactive psychosis (N=34)	127.03	121.04				122.5		
	Total (N=134)	154.87	123.92	.74	.21	.46	157.50	0	560
No. (%) patients on antipsychotic medication_15									
	Non-reactive psychosis (N=109)	82 (75.2%)							
	Reactive psychosis (N=43)	25 (58.1%)							
	Total 152 (N=152)	107 (70.4%)							

6.7 Functional recovery and quality of life

6.7.1 Social and occupational functioning

The following section outlines the analyses pertaining to hypotheses 4(a) and 4(b). Table 27 displays the descriptive statistics for the Social and Occupational Functioning Scale (SOFAS) score measured across the three follow up time points, which satisfied the assumption of normality. Although both the reactive psychosis and non-reactive psychosis groups started with the same level of social and occupational functioning at baseline, ANOVA showed that the reactive psychosis group had significantly better social and occupational functioning than the non-reactive psychosis group, three months after admission ($F=6.477$; $df=1,215$; $p=.012$), partially supporting hypothesis 4(a). However, these differences were not maintained at the 9 month ($F=1.571$; $df=1,176$; $p=.212$) nor the 15 month follow up point ($F=.883$; $df=1,168$; $p=.349$).

Table 27. Social and Occupational Functioning Scale (SOFAS) score for reactive psychosis groups at 3, 9 and 15 months post entry into the service

	Reactive psychosis group	M	SD	Skewness	Standard error skewness	Kurtosis	Median	Min	Max
SOFAS_3	Non-reactive psychosis (N=154)	52.69	11.74				55		
	Reactive psychosis (N=63)	57.43	14.01				60		
	Total (N=217)	54.07	12.59	.34	.16	.241	55.0	30	90
SOFAS_9	Non-reactive psychosis (N=125)	57.33	16.28				60		
	Reactive psychosis (N=53)	60.60	15.10				55		
	Total (N=178)	58.30	15.97	.295	.182	-.809	56.0	30	90
SOFAS_15	Non-reactive psychosis (N=122)	58.47	17.32				65		
	Reactive psychosis (N=48)	61.23	17.05				70		
	Total (N=170)	59.25	17.24	.11	.19	.99	60.00	25	90

6.7.2 Quality of Life Scale

The quality of life scale was only measured for a subsample of 98 patients at the 3 month post admission point. Data for quality of life at all three measured time points satisfied the assumption of normality. Although the reactive psychosis group scored on average 7 points higher on the quality of life scale than the non-reactive psychosis group at 3 months post admission, contrary to hypothesis 4(b), ANOVA

determined that the difference was not significant (F=2.216; df=1,96; p=.140). Quality of life was measured at 9 and 15 months post admission for the whole sample that was followed up. There were no significant differences in quality of life between the groups as measured at 9 (F=2.426; df=1,175; p=.121) and 15 months post admission (F=.953; df=1,163; p=.330).

Table 28. Quality of Life for reactive psychosis (RP) and non-reactive psychosis (Non-RP) groups at 3, 9 and 15 months post entry into the service

	Reactive psychosis group	M	SD	Skewness	Standard error skewness	Kurtosis	Median	Min	Max
Quality of Life_3	Non-RP (N=71)	68.10	20.67				70		
	RP (N=27)	75.00	20.01				69		
	Total (N=98)	70.00	20.62	.31	.24	-.34	68.50	31	118
Quality of Life_9	Non-RP (N=124)	72.71	23.86				74		
	RP (N=53)	78.68	22.22				73		
	Total (N=177)	74.50	23.48	.04	.18	-.64	75.00	19	125
Quality of Life_15	Non-RP (N=118)	78.11	25.01				82		
	RP (N=47)	82.24	23.33				84		
	Total (N=165)	79.29	24.54	-.05	.19	-.77	80	24	126

6.8 Examining the Degree of Overlap of Reactive Psychosis with DSM, ICD-10 and Other Historical Psychotic Diagnoses

The fifth objective of this study was to examine the amount of overlap between reactive psychosis and other diagnoses. The proportion of reactive psychosis diagnoses that overlapped with psychotic diagnoses within conventional diagnostic systems (DSM and ICD-10) and with more historical psychotic diagnoses was examined using chi-square analyses.

Table 29 displays the percentages and chi-square statistics of the degree of overlap of reactive psychosis with the DSM and ICD-10 diagnoses of psychosis. Where there were too few cases to report chi-square statistics (using the criteria of less than 25% of cases having an expected count less than five), exact statistics were reported.

Unexpectedly, there were no cases within the first episode psychosis group that were diagnosed with DSM-III and III-R brief reactive psychosis and only two cases of DSM-IV brief psychotic disorder. Although both cases fell into the reactive psychosis group, the numbers were small so an exact score of .083 has limited interpretability. Similarly, there was only one case of ICD-10 acute and transient psychotic disorder and that case fell into the non-reactive psychosis group. A significantly larger proportion (27%) of reactive psychosis cases fell into ICD-10 other nonorganic psychotic disorder compared with non-reactive psychosis cases (14.3%).

As expected, there were significantly fewer cases of reactive psychosis that were also diagnosed within the DSM-IV schizophrenia spectrum disorders. An examination of adjusted residuals within the chi-square analysis revealed that there was proportionately more overlap between non-reactive psychosis and schizophrenia (adjusted residual of 5.0) but no differences within the schizophreniform group (with or without a good prognosis). There were also significantly more cases of schizophrenia in the non-reactive psychosis group (43%) using the ICD-10 system.

An examination of reactive psychosis with DSM-IV affective diagnoses indicated that there was significantly more affective disturbance within the reactive psychosis group. 23.8% of the reactive psychosis group also met DSM-IV criteria for bipolar disorder with psychotic features versus 13% in the non-reactive psychosis group.

Furthermore, 20.6% of the reactive psychosis group met DSM-IV criteria for major depressive disorder with psychotic features versus 8.4% in the non-reactive psychosis group. There were no differences in degree of overlap between reactive psychosis groups and ICD-10 bipolar affective disorder, manic episode with psychotic symptoms or severe depression with psychotic symptoms.

Although there were no differences between the reactive psychosis groups in the degree of overlap with DSM-IV schizoaffective disorders there were significantly more cases of reactive psychosis that also met criteria for ICD-10 schizoaffective disorder. Eleven per cent of reactive psychosis cases also met criteria for ICD-10 schizoaffective disorder bipolar type (adjusted residual 2.3) and 18% of the reactive psychosis group met criteria for schizoaffective disorder depressed type (adjusted residual 2.3).

Table 29. Frequency of overlap of Reactive psychosis with DSM and ICD-10 psychotic diagnoses

	Frequency of reactive psychosis diagnosis within diagnostic category		Chi-quare statistic	P value
	Reactive psychosis group (N=63)	Non-reactive psychosis group (N=154)		
DSM-III				
Brief reactive psychosis	0	0	n/a	-
Acute paranoid disorder	1 (0.5 %)	0	$\chi^2(1, N=217)=2.45$	.290 (exact)
Atypical psychosis – type 2	2 (3.2%)	2 (1.3%)	$\chi^2(1, N=217)=.87$	.582 (exact)
DSM-III-R				
Brief reactive psychosis	0	0	-	-
DSM-IV				
Schizophrenia	9 (14.3%)	79 (51.3%)	$\chi^2(3,N=217)=26.31$	.000 (exact)
Schizophreniform	1 (1.6%)	3 (1.9%)		
Schizophreniform with good prognosis	7 (11.1%)	12 (7.8%)		
Brief psychotic disorder	2 (3.2%)	0	$\chi^2(1, N=217)=4.93$	.083 (exact)
Delusional disorder	1 (1.6%)	2 (1.2%)	$\chi^2(1, N=217)=.027$	1.00 (exact)
Schizoaffective disorder				
Bipolar type	3 (4.8 %)	5 (3.2%)	$\chi^2(2, N=217)=.297$	.862
Depressive type	5 (7.9 %)	13 (8.4%)		
Psychotic disorder NOS	6 (9.5 %)	7 (4.5%)	$\chi^2(1, N=217)=1.97$	.206 (exact)
Substance induced psychotic disorder	1 (1.6%)	2 (1.3%)	$\chi^2(1, N=217)=.027$	1.00 (exact)
Bipolar disorder with psychotic features	15 (23.8%)	20 (13%)	$\chi^2(1, N=217)= 3.87$	.049
Bipolar disorder II	1 (1.6%)	0	$\chi^2(1, N=217)=2.46$	.290 (exact)
Major depressive disorder with psychotic features	13 (20.6%)	13 (8.4%)	$\chi^2(1, N=217)=6.3$	.012
ICD-10				
Schizophrenia	15 (23.8%)	66 (42.9%)	$\chi^2(1, N=217)=6.93$	.008
Delusional Disorder	1 (1.6%)	4 (2.6%)	$\chi^2(1, N=217)=.203$	1.00 (exact)

Acute and transient psychotic disorder	0	1 (0.6%)	$\chi^2(1, N=217)=.411$	1.00 (exact)
Other persistent delusional disorders	6 (9.5%)	39 (25.3%)	$\chi^2(1, N=217)=6.79$	.009
Schizoaffective disorder				
Bipolar	7 (11.1%)	5 (3.2%)	$\chi^2(3, N=217)=11.56$	.007 (exact)
Depressed	11 (17.5%)	11(7.1%)		
Mixed	1 (1.6%)	2 (1.3%)		
Manic episode with psychotic symptoms	2 (3.2%)	2 (1.2%)	$\chi^2(1, N=217)=.87$	.582 (exact)
Bipolar affective disorder	2 (3.2%)	1 (0.6%)	$\chi^2(1, N=217)=2.09$	.203 (exact)
Severe depression with psychotic symptoms	1 (1.6%)	1 (0.6%)	$\chi^2(1, N=217)=0.43$	.497 (exact)
Psychotic disorder with substance use	28 (44.4%)	50 (32.5%)	$\chi^2(1, N=217)=2.79$	.095
Other nonorganic psychotic disorder	17 (27%)	22 (14.3%)	$\chi^2(1, N=217)=4.89$	.027

NB: exact in parentheses indicates that the exact significance has been reported as more than 20% of case have an expected count less than 5.

The level of overlap between reactive psychosis and other historical psychoses dating back to Kraepelin and Bleuler psychotic diagnoses was explored. Chi-square analyses were used to examine group differences with results displayed in Table 30. Again, where there were too few cases to report chi-square statistics, exact statistics were reported.

The non-reactive psychosis group was not more likely to overlap with some historical diagnoses closely related to modern day schizophrenia such as Kraepelin, Langfeldt, E Bleuler and M Bleuler. However there were significantly more non-reactive psychosis cases in the Taylor and Abrams schizophrenia construct (21.4%), Feigner schizophrenia (definite diagnosis 18.2%) and a trend for more non-reactive psychosis cases to overlap with Schneider's construct of schizophrenia.

Nineteen per cent of cases in the reactive psychosis group also met criteria for SCAAPS acute psychotic episode, significantly more than within the non-reactive psychosis group. Interestingly, of the 6 cases of first episode psychosis that also met criteria for Bouffée délirante, 5 fell within the reactive psychosis group, revealing significant differences between the reactive psychosis groups.

As expected, reactive psychosis was more likely to overlap with atypical psychotic diagnoses such as cycloid atypical psychosis, with 36 % of reactive psychosis cases meeting criteria for a probable or definite diagnosis. Eleven per cent of reactive psychosis cases also met criteria for Dongier atypical psychosis.

Table 30. Frequency of overlap of Reactive psychosis with historical psychotic diagnoses

	Frequency of reactive psychosis diagnosis within diagnostic category		Chi-quare statistic	P value
	Reactive psychosis group (N=63)	Non-reactive psychosis group (N=154)		
Schneider schizophrenia	45 (71.4%)	128 (83.1%)	$\chi^2(1, N=217)= 3.78$	.052
E Bleuler Schizophrenia				
Probable	10 (15.9%)	35 (22.7%)	$\chi^2(1, N=217)= 0.008$	.930
Definite (groups collapsed for analyses)	26 (41.3%)	52 (33.8%)		
M Bleuler Schizophrenia	28 (44.4%)	63 (40.9%)	$\chi^2(1, N=217)=.23$	.632
Langfeldt Schizophrenia	3 (4.8%)	2 (1.3%)	$\chi^2(1, N=217)= 2.38$	.123
Kraepelin Schizophrenia				
Probable	29(46.0%)	80 (51.9%)	$\chi^2(2, N=217)= 2.87$	.238
Definite	6 (9.5%)	23 (14.9%)		
SCAAPS Acute Psychotic Episode	12 (19%)	13 (8.4%)	$\chi^2(1, N=217)= 4.93$	.026
Cycloid Atypical Psychosis				
Probable	12 (19%)	15 (9.7%)	$\chi^2(1, N=217)= 7.67$	.006
Definite (groups collapsed)	11 (17.5%)	14 (9.1%)		
Dongier Atypical psychosis	7 (11.1%)	6 (3.9%)	$\chi^2(1, N=217)= 4.13$	.042
Bouffée délirante atypical psychosis	5 (7.9 %)	1 (0.6)	$\chi^2(1, N=217)= 8.83$	.003
Cloninger Schizophrenic symptom scale	51 (81 %)	132 (85.7%)	$\chi^2(1, N=217)=.767$	.381
Taylor & Abrams Schizophrenia	6 (9.5%)	33 (21.4%)	$\chi^2(1, N=217)= 4.30$	.038
Feigner Schizophrenia				
Probable	3 (4.8%)	9 (5.8 %)	$\chi^2(1, N=217)=9.11$	.003
Definite (groups collapsed)	1 (1.6%)	28 (18.2%)		
Feigner schizoaffective disorder-depressed	27 (42.9%)	41 (26.6%)	$\chi^2(1, N=217)= 5.48$	.019
Feigner schizoaffective disorder-manic type	20 (31.7%)	31 (20.1%)	$\chi^2(1, N=217)= 3.36$	.067
RDC unspecified functional psychosis	9 (14.3%)	11 (7.1%)	$\chi^2(1, N=217)= 2.73$	.099

NB: exact in parentheses indicates that the exact significance has been reported as more than 20% of case have an expected count less than 5.

Chapter 7: Discussion

This is the first study, in an English speaking western country, to examine the prevalence, validity and short to medium term outcome of reactive psychosis within a first-episode psychosis population. Although preliminary, findings from this study indicate the foundation of both descriptive and external validity for the reactive psychosis diagnosis.

It is apparent that there are a number of young patients who have a moderate to severe stressor that temporally precedes the onset of their psychotic episode, who present as acutely unwell, and have compromised social and occupational functioning to the point of requiring the assistance of acute psychiatric services. Reactive psychosis patients present with the same level of acuity as their non-reactive counterparts and are also struggling with their role functioning to the same degree. Consistent with historical (Jaspers, 1965) and contemporary views (Daahl, 1986) of reactive psychosis, reactive psychosis patients in this study recovered more quickly from their index episode. However there are fewer than expected discernable differences throughout the course of follow up, leaving open the question as to whether this is indeed a distinct syndrome or a less severe form of psychosis.

7.1 Reliability of the Reactive Psychosis Rating Form

The Reactive Psychosis Rating Form (Guldberg et al., 1996) used in this study was slightly modified from the original by removing the two items that were considered outcome variables (duration of psychosis and pre and post psychotic functioning). Duration of psychosis is well argued as a prognostic factor in the outcome of psychosis (Harris et al., 2005). Social and occupational functioning are variables of interest in prognosis and arguably as, if not more important, than symptom outcome. We wanted to study these variables as prognostic factors and secondly, we hoped to establish whether the illness characteristics, collectively described as reactive psychosis, have a better prognostic outcome than other non-reactive psychotic diagnoses.

An inter-item reliability analysis revealed a good internal consistency (alpha = .812) indicating that reactive psychosis represents a unified construct. A good internal consistency is considered an essential criterion for establishing a diagnosis. Guldberg et al. (1996) did not report an overall Cronbach's alpha in their study, but the three factors they extracted showed a similar moderate to good internal consistency. Unlike their study, degree of perplexity was not well correlated with the other reactive psychosis items in our study. Only 14 % of patients presented with severe perplexity to the point of misidentification to severe spatial disorientation. It is possible that we do not commonly see first episode psychosis with such marked spatial and temporal disorientation, perhaps more typically seen in more chronic or acutely psychotic groups. Alternatively, it may be attributed to a rater bias, as this symptom may be more difficult to assess.

Not surprisingly, the duration of stressor, time between stressor and onset of psychosis and severity of stressor were moderately-to-highly correlated. It appeared that the more severe the stressor, the more likely it would be reflected in the content and meaning of the psychotic symptoms. Furthermore, the closer the onset of psychotic symptoms to the trauma, the more likely it would be reflected in the content and meaning of the psychotic symptoms. These correlations give some weight to the premise that psychotic symptoms are indeed a way of 'working through' or managing the psychic pain associated with trauma (Federn, 1952; Freud, 1896; Gallwey, 1985).

The insidiousness of the onset of psychosis was not associated with any other factors (with the exception of time between stressor and onset of psychosis) despite 62.7 % of the sample having a rapid onset that developed within 3 months of the onset of prodromal symptoms. This challenges one of the central assumptions of reactive psychosis that these individuals have a more rapid onset of psychosis than those with a non-reactive psychosis. It is likely to be a difficult assumption to uphold in a first-episode psychosis population were the focus is on treating people earlier in the course of their illness. The EPPIC service's heavy focus on prevention and early detection, has likely shifted or changed the usual presentation of first-episode psychosis (Krstev et al., 2004) as has been observed in the TIPS campaign in Scandinavia where they reduced the average duration of untreated psychosis significantly (Melle et al., 2004).

A significant limitation in the use of this scale in this study is that it did not produce any information specifically about inter-rater reliability for the RPRF, although raters did meet weekly in order to establish consensus amongst ratings. This may be particularly important for items that have a more subjective nature such as meaning and content of stressor or severity of stressor (although there were some clear anchor points for this item) and is something to consider for future use of the RPRF. Furthermore, providing 'expert' clinician consensus rating alongside the use of the scale could also be considered also helpful.

7.2 Prevalence of Reactive Psychosis

Hypothesis 1 was supported, as it was possible to identify a group of patients within a first episode psychosis population who met criteria for reactive psychosis. Furthermore, trauma commonly precedes the onset of psychosis, even in young patients. The prevalence of a moderate to severe stressor preceding the onset of psychotic illness in this sample of first-episode psychosis was 65.5 %. These findings are consistent with the large-scale first-episode psychosis study of Neria et al.'s (2002) who found that 68.5 % had a lifetime prevalence of trauma exposure. Much higher levels of trauma have been reported in more chronic populations (Mueser et al., 1998).

Of most interest is that, of these patients, almost a third had experienced their stressor within 3 months of the development of their psychotic illness, suggesting that the stressor is likely implicated in the development of psychosis for a significant proportion of first-episode psychosis patients. Additionally, for at least a third of the patients, the psychotic symptoms' content and meaning was related to their stressor, giving further weight to the assertion that psychotic symptoms function as a way to 'work through' or perhaps as a means of escape from the psychic pain associated with the trauma.

This summation of the prevalence of two key criteria of reactive psychosis (i.e. presence of a stressor and present temporally with the onset of psychosis) provides some face validity to the construct of reactive psychosis as separate and distinct entity within the realm of psychotic diagnoses.

One of the aims of our study was to ascertain a threshold or 'cut-off' point for reactive psychosis, using the Reactive Psychosis Rating Form (Guldberg et al., 1996; personal communication Guldberg, 1996). This is the first attempt to systematically operationalise and diagnose reactive psychoses within an Australian psychiatric setting. A method consistent with the DSM-IV (APA, 1994) diagnostic system was used, in which a certain number of items needed to be endorsed at a moderate to severe level in order to meet criteria for a diagnosis. Using the procedure, outlined in the method section, participants who had met criterion A (presence of a stressor) and who also met the criteria on at least 4 of the 6 items on the reactive psychosis scale were grouped into the "Reactive psychosis" group.

In this sample of first episode psychosis patients, 29 % (N=63) met criteria for reactive psychosis. These results are consistent with Scandinavian studies where up to a third of psychiatric admissions were considered to meet criteria for reactive psychosis. Their national registries estimated that between 13-30 % of all psychiatric admissions were reactive psychosis (Daahl, 1986). In Denmark, a review of first psychiatric admission registries between 1970 and 1988 found that a diagnosis of reactive psychosis was five times more likely than a diagnosis of schizophrenia (Jorgensen & Mortensen, 1992). However, in their follow up, they found that 30 to 50% of those initially diagnosed as reactive psychosis were later diagnosed as having schizophrenia or affective illness. It would have been useful to compare these rates to our sample but a limitation in our study is that patients did not receive a follow-up diagnosis. The lengthy diagnostic interview was not repeated at 3, 9 and 15 months in order to keep the follow-up commitment manageable for patients. One way around this would have been to include an updated diagnosis from the clinical file as part of the treatment questionnaire. However this may have been a redundant gesture, given that clinicians do not generally assign reactive psychosis as a diagnosis.

The prevalence rates of reactive psychosis in this study are still higher than in Castagnini et al.'s (2007) study where 19.2 % of patients were classified as reactive psychosis, using ICD-8 classification of other functional psychoses. This attests to some of the criticism that ICD's classification of reactive psychoses is too restrictive (Ungvari & Mullen, 2000).

7.3 Description of Reactive and Non-reactive Samples at Baseline

Hypotheses 2(a) to 2(f) were supported. As expected there were no differences between the reactive and non-reactive groups in the severity of baseline general psychopathology (including psychotic symptoms, suicidality, depressive symptoms, hostility), severity of negative symptoms and social and occupational functioning. The average SOFAS score was 44 (out of a possible 100), indicating a major impairment of social and occupational functioning in several areas for the entire sample (Goldman et al., 1992).

As expected, the reactive psychosis group had a less insidious onset as measured by their significantly shorter duration of prodromal symptoms, and a shorter duration of psychosis before entry to the service. The average length of untreated psychosis for the reactive psychosis group was 69 days, compared with 249 days in the non-reactive psychosis group.

The presenting picture of the reactive psychosis group so far is a group with a preponderance of females, whose presenting psychotic symptoms were as severe as the non-reactive psychosis groups, and who had poor functioning and significant comorbid psychopathology. The next section will examine the differences in the course of illness between the reactive and non-reactive psychosis groups by describing their clinical outcome using measures of severity of symptoms as well as proxy variables such as service use and prescription of antipsychotic medication.

7.4 Clinical Outcome of Reactive and Non-reactive Psychosis

7.4.1 Duration of psychosis, rate of remission and severity of psychopathology across the course of follow up

Hypothesis 3(a) was supported as reactive psychosis patients recovered more quickly from their psychotic symptoms in their index episode and had higher rates of remission from their index episode. Hypothesis 3(b) was not supported and there was partial support for hypothesis 3(c). Unexpectedly, there was only a trend for the

reactive psychosis group to make a better recovery from general psychopathology symptoms. The reactive psychosis group had significantly less severe positive psychotic symptoms than the non-reactive group at 3 months post admission, but there was only a trend toward maintaining a better recovery at 9 months.

In order to establish the predictive validity (as a type of external validity) of reactive psychosis as a separate and distinct psychotic diagnosis, we need to demonstrate that it has a distinct course. One central component in establishing the validity of the reactive psychosis diagnosis is the recovery from psychotic symptoms. As expected, the reactive psychosis group, although *presenting* with the same severity of psychotic symptoms (as measured by the presence and severity of hallucinations, delusions, disorganised behaviour and formal thought disorder), by 3-month follow up they demonstrated shorter duration of psychosis, and a higher rate of remission. At the 3-month time point they had significantly less severe positive psychotic symptoms. There was a trend for them to maintain this advantage at 9 months, but by 15 months the non-reactive psychosis group had caught up to the reactive psychosis group. This is consistent with several early psychosis studies that show patients make a good level of recovery from their psychotic symptoms (Carbone et al., 1999; Harris et al., 2005).

Another important consideration in establishing the course of psychiatric illness is recovery from general psychopathology. The most intense treatment for psychosis (i.e. input from acute, medical staff and outpatient services) is during the first 3 months after entering the service (Bertolote & McGorry, 2005), so not surprisingly both groups showed improvement in general psychopathology, which included depression, anxiety, as well as psychotic symptoms. There was a trend for the reactive psychosis group to have less severe psychopathology than the non-reactive psychosis group after three months of treatment. Perhaps as a consequence of the 18–month mandated period of care, it appears that the non-reactive psychosis group ‘caught up’ to the reactive psychosis group by 9 and 15-month follow up. Nonetheless, the results tentatively indicate that although the reactive psychosis group may present with the same severity of general psychopathology, they have made more improvement than the non-reactive psychosis group in the initial phase of treatment.

Unexpectedly, there was limited support for Hypothesis 3(d), although there was a trend for the reactive psychosis group to have less severe negative symptoms than the non-reactive psychosis group at the 3-month time point. Negative symptoms are defined as a decrease in or loss of normal functions and are typically associated with a diagnosis of schizophrenia (APA, 1994), therefore separating a schizophrenia-like process from other psychotic processes. It can be difficult to assess the primacy of negative symptoms (Andreasen, 1982) as they have similar features to affective disorders, and can be confused with the side effects of medication. Our study only examined the negative symptom total scores and not each individual domain as Andreasen (1984) first devised. There were no differences between groups in severity of presenting negative symptoms, perhaps owing to this being a first-episode group where negative symptoms may be more typically seen later in the course, or in more established psychosis with an episodic course. However, the trend indicates that the reactive psychosis group had less severe negative symptoms than the non-reactive psychosis group at the three-month follow up point with the non-reactive psychosis groups 'catching up' throughout the course of their treatment. Although these findings need to be interpreted with caution, they are comparable with Jager et al.'s (2003) study that compares ICD-10 acute and transient psychotic disorders to other psychotic disorders and found that the acute and transient psychosis groups had fewer negative symptoms and better global functioning. Furthermore, given that negative symptoms are more likely associated with more chronic samples of psychosis such as schizophrenia, they may have a limited capacity to discriminate between reactive and non-reactive psychosis in a first episode sample.

7.4.2. Affective symptoms over the course of follow up

This study examined the course of affective symptoms over the follow up period as affective disorders have sometimes been associated with reactive psychosis diagnoses, although there is very little empirical support. In support of hypothesis 3(e), there were no discernable differences in presentation of comorbid depressive symptoms, either in the clinical picture preceding treatment or throughout treatment. However, the reactive psychosis group had a trend towards more manic symptoms than the non-reactive psychosis group in period up to 3-months post admission,

although this trend was not sustained at follow up. One explanation is that our reactive psychosis group could more closely resemble historical notions of reactive psychosis. In Kraepelin's (1893) original notion of reactive psychosis, he often joined reactive psychosis and manic type presentations, seeing an overlap in his patients. Several other constructs related to reactive psychosis had emphasised the affective quality to this presentation, most notably Wernicke's cycloid psychosis (1900). Given that there were no *statistically* significant differences between groups in the presentation of affective symptoms, they could not be considered as a confound variable and therefore not controlled for in analyses. It is worth noting that there has been little empirical support for the notion that reactive psychoses are associated with affective symptoms/syndromes but there appears to be some merit in teasing out this relationship further.

7.4.3 Service use

Consistent with the hypothesis that a reactive psychosis diagnosis is associated with a better course and outcome than other psychotic diagnoses, it may be expected that patients with a reactive psychosis diagnosis use fewer clinical resources than the non-reactive psychosis diagnoses. The EPPIC clinical service, with its strong links with the research program, had only recently begun to collect data on clinical service use in an effort to 'weigh up the costs' of an early intervention psychosis service in an effort to answer criticism of this (at the time) major service reform (Mihalopoulos et al., 1999). The data used in the current study represented the centre's first attempt at quantifying service use in a more useful, consistent and specific way, than was required for purposes of reporting to government. It was difficult to establish from the outset what may be useful to collect, and in retrospect collating simply the number of visits by the acute response service (YAT) and number of outpatient appointments, may seem rather crude. Nonetheless, it provides an estimate of service use, which has been used as a proxy variable of clinical outcome as it approximates the level of acuity and need for care. This did not take into account those patients who disengaged from the service, either because they were acutely unwell or 'recovered'.

Consistent with hypothesis 3(f), there were no differences between the reactive and non-reactive psychosis groups in the number of case management visits

in the first three months of treatment, indicating that the reactive psychosis group still require the same level of intensive treatment during the initial phase as the non reactive psychosis group, despite them recovering more quickly. It is worth noting that the early psychosis guidelines (see Table 1 in Chapter 1) suggest at least weekly contact by the outpatient service, irrespective of acuity of the patient's psychotic presentation (and irrespective of psychotic diagnoses) during the first three months of treatment.

A significant proportion of patients required hospitalisation during their acute phase, with sixty five percent requiring an admission to manage their psychosis. The average length of hospitalisation for the acute phase was eighteen days. There was support for hypothesis 3(h) in that, consistent with the level of outpatient service use, the reactive psychosis patients required similar lengths of hospitalisation as non-reactive psychosis patients.

The number of visits by the acute home-based treatment team (YAT) was considered an accurate depiction of need for their service. Although the reactive psychosis group made a more rapid recovery from their psychosis, they did not differ in their use of acute response services. These results do not take into account the number of telephone calls that YAT made which is a core item of business for the team.

The reactive psychosis group used less outpatient services during their 'recovery phase' or between three and nine months of treatment suggesting that the non-reactive psychosis group required more intensive treatment, at least for a longer period of time, to 'catch up' during this phase (in partial support of hypothesis 3(g)). The emphasis during this phase is on outpatient treatment. Interestingly the reactive psychosis group were not less likely to use acute services during this phase, with the average number of visits for the patients reducing from twelve to three. The large standard deviation suggests that there were a small number of patients who required intensive treatment.

Interestingly, the non-reactive psychosis group, despite recovering initially, had significantly longer hospital admissions in their final phase of treatment. It is

unclear whether this was due to their experiencing more psychotic relapses, requiring hospitalisation or alternately, they could have been hospitalised for risk to self and/or others. As indicted in the method section, we did not have an accurate measure of relapse therefore the data were not presented. The issue of relapse is an important one in any discussion of the prognosis of illness and particularly interesting in the field of reactive psychosis where the conceptual theory of reactive psychosis would suggest that reactive psychoses have fewer relapses than other psychotic diagnoses but it is not consistent with Pillman et al.'s (2002) empirical study that found higher relapse rates amongst their brief psychotic disorder group compared with other acute psychotic diagnoses. Further studies could better explore the question of rate of relapse by using less crude measures of relapse than the EPSUOQ and measures specifically designed to more accurately define and quantify relapse.

In summary, the difference in level of recovery at the 3 month time point was not evident in the proxy measures of recovery (i.e. in service use & also medication as the following section will indicate), but there were significant differences between the groups at 9 month follow up (outpatient appointments) and at 15 months (inpatient days and medication use). One interpretation is that this may due to a lag in service response to symptom differences at 3 months and/or these proxy measures may be picking up a difference in presentation at this later stage that the symptom and social and occupational measures are not picking up. Although difficult to conclude at this stage that the reactive psychosis group has a definite and distinct course, the findings indicate some expected differences, warranting further investigation.

7.4.4 Use of anti-psychotic medication

Consistent with guidelines that antipsychotic medication is the first line treatment for psychosis (International Early Psychosis Association Writing Group, 2005), ninety seven percent of patients in this sample were prescribed antipsychotic medication during the acute phase, with an average (Chlorpromazine equivalent) dose of 186.65 mg. As expected, there were no differences in dosage or likely use of medication between reactive and non-reactive psychosis groups in the initial treatment phase, supporting hypothesis 3(j). This is consistent with reform guidelines advocating for the prescription of low doses of antipsychotic medication, at least in

the first instance (Bertolote & McGorry, 2005). Low dose practices have been recommended due to young patients being neuroleptic naïve, therefore requiring smaller doses for effective resolution of psychotic symptoms (Bertolote & McGorry, 2005). There will always remain a subgroup of patients that are considered 'treatment resistant' and may require several changes in antipsychotic medication at varying doses. The initial results of this study adhere to first-episode psychosis practice guidelines. It is encouraging that the average dose of antipsychotic medication was kept within the guidelines of low doses throughout the course of follow up, and the non-reactive psychosis group were not on higher doses of antipsychotic medication than the reactive psychosis group, although this was unexpected.

As predicted, and in partial support of hypothesis 3(k), the reactive psychosis group had discontinued their medication sooner than the non-reactive psychosis group. In fact, 25% of those diagnosed as reactive psychoses were no longer on an antipsychotic medication by nine-month follow up. By fifteen-month follow up, just over half of the reactive psychosis group were on an antipsychotic medication, significantly less than the non-reactive psychosis group where 77% were still on a maintenance dose of antipsychotic medication.

These results elucidating actual clinical practice have important implications for the treatment guidelines of those presenting with reactive psychosis. If it can be established that there is enough merit to consider reactive psychosis a separate entity, we need to consider whether the clinical guidelines for treatment of early psychosis accurately represent this group. The guidelines are generic in that they represent all patients presenting with psychotic symptoms, considered at threshold for psychosis but one could consider including a caveat for those presenting with reactive psychosis. One way of approaching this issue is to examine current clinical practices for treating these patients. Despite evidence based guidelines, patients and clinicians invariably work together in individualising treatment and jointly make decisions, evaluating the efficacy along the way, deciding which antipsychotic medication to prescribe, their dose, all the while considering their side-effect profile.

Current practice at EPPIC, as highlighted in our study, suggests that the reactive psychosis group do not need to be on medication for as long as their non-

reactive counterparts, inconsistent with guidelines that advocate for a maintenance dose of antipsychotic medication for 1 to 2 years after recovery. The reactive psychosis group still require psychosocial interventions, but perhaps there could be less emphasis on medication, or at least its necessity be regularly evaluated. These findings are consistent with the views of the original proponents of the reactive psychosis diagnosis who have long maintained that this group is separate, would benefit from antipsychotic medication in the initial phase (McGlashan & Krystal, 1995; Modestin & Bachmann, 1992; Stevens, 1987) while high-dose or prolonged medication is not usually required (Jamminga & Carpenter, 1982). Furthermore, this group would benefit from psychotherapy as a first line treatment, rather than antipsychotic medication (Murphy, 2000). Treatment recommendations will be further fleshed out later in this discussion.

7.5. Social and Occupational Functioning and Quality of Life

In establishing, at least in part, the potential predictive validity of reactive psychosis, it would be useful to next consider clinical domains such as social and occupational functioning and quality of life that are equally important prognostic indicators. The current study is not naturalistic and outcome is influenced by treatment in a dose-response manner. The first episode program has a strong emphasis on social and occupational intervention with an on-site group program that focuses on social rehabilitation and where staff work hard at reestablishing an age-appropriate level of role functioning (Edwards et al., 1994).

Consistent with results so far, the reactive psychosis group, although initially equally functionally impaired, had significantly better social and occupational functioning at the three-month follow up point, in partial support of hypothesis 4(a), with the non-reactive psychosis group ‘catching up’ throughout the course of their treatment. These results are consistent with a study by Pillman et al. (2002) who found that their brief psychotic disorder sample did better on several domains including occupational status, relationships, independent living, functioning in social roles, psychological impairment, and global functioning. It is difficult to explain why the reactive psychosis group in this study did not differ from the non-reactive group in their course of recovery in interpersonal and intrapsychic (quality of life) domains.

Given there was no baseline measure of this instrument, interpretation of these results needs to be cautious.

7.6 Overlap of Reactive Psychosis with Other Psychotic Diagnoses

How does the traditional Scandinavian concept of reactive psychosis, as operationalised in this study, overlap with conventional western classification systems including the DSM-IV (APA, 1994) and ICD-10 (WHO, 1992)? There is just criticism that the DSM-IV classification of reactive psychosis is too restrictive, as only two cases of brief psychotic disorder were found in our sample, despite the high prevalence of reactive psychosis. This is likely due to DSM-IV's criteria maintaining that the psychosis must remit within one month. In our sample of reactive psychosis, the average number of days taken to remit was significantly longer. Similarly, there was only one case of ICD-10 acute and transient psychotic disorders, which paradoxically fell into the non-reactive psychosis group. This is likely due to the newer classification of ICD-10 not specifying a stressor preceding the onset of the acute and transient psychotic disorders. We concur with Castagnini et al. (2007), who argued the newer criteria were too restrictive after they found that 19% of all non-organic psychosis admissions were classified by ICD-8's either reactive depressive psychosis, acute paranoid reaction or psychosis reactiva but significantly fewer cases met criteria for ICD-10 acute and transient psychotic disorders.

Similarly, Mojtabai et al. (2000) found in their sample of reactive psychosis, very few cases that also met criteria for acute and transient psychotic disorders. They called for a reform in the operationalism of ICD-10 criteria, firstly by expanding the criteria to six months, and eliminating all of the subcategories, as there is little empirical evidence to support them.

In our sample, the reactive psychosis cases were more likely associated with ICD-10's other nonorganic psychotic disorders, a default category for those not meeting any other psychotic diagnosis (WHO, 1992). Given the evidence that this group has distinct clinical features, it appears that the ICD-10 has missed the mark in reducing them to an 'other' category, which underscores little of their distinctness.

In essence, qualifying the number of days the initial episode lasts is perhaps too restrictive as it is currently conceptualised in DSM-IV (APA, 1994). The length of an initial episode is contingent upon many factors including the resolution of the stressor, adequate psychosocial support and treatment.

Further evidence that reactive psychosis is a clinically distinct category from schizophrenia emerged from there being significantly more cases of non-reactive psychosis classified as schizophrenia, using both ICD-10 and DSM-IV. Only 9 cases diagnosed as reactive psychosis also met criteria for DSM-IV schizophrenia. Importantly, reactive psychosis made up a significant proportion of the sample, comparable to schizophrenia (40%). This is inconsistent with results from a Denmark review of first psychiatric admission registries between 1970 and 1988 that found a diagnosis of reactive psychosis was five times more likely than a diagnosis of schizophrenia (Jorgensen & Mortensen, 1992). It is difficult to discern why only 12% of our sample met criteria for depression with psychotic features and only 10% met criteria for schizophreniform disorder.

Early advocates of the reactive psychosis construct have described significant affective disturbance in their cases (Schneider, 1927; Kraepelin, 1893). This was evident in our sample, where 24% of those with reactive psychosis also met criteria for DSM-IV bipolar disorder and 21% met criteria for depression with psychotic features. Perris and Brockington (1981) also found that mood swings were commonly associated with their conception of cycloid psychoses.

First rank psychotic symptoms are traditionally used to differentiate schizophrenia from other psychotic diagnoses. In our sample there were fewer cases of reactive psychosis that fell into Taylor and Abram's and Feigner's traditional notion of schizophrenia. These nosologies emphasise that schizophrenia has a chronic course, the absence of affective disorder and perplexity and appear to closely resemble the differentiation of reactive psychosis from other psychotic diagnoses.

Bouffée délirante was only diagnosed in six patients in this entire first-episode sample, but five of these fell within the reactive psychosis group, despite that a

diagnosis of bouffée délirante does not necessitate a stressor. This is likely due to operationalisation of bouffée délirante that specifies perplexity, as revealed earlier, uncommon in this sample and an age of onset between twenty and forty years, missing a significant proportion of our sample where the lower age limit is sixteen years. Our results indicate that the otherwise, commonly diagnosed French bouffée délirante (Pichot, 1986) is not prevalent in a young first episode-psychosis sample.

Cycloid atypical psychosis was more likely associated with reactive psychosis than non-reactive psychosis, in that thirty six percent of the reactive psychosis group also met criteria for cycloid psychosis. Interestingly, there was a relatively higher prevalence of cycloid psychosis (23%) in this sample than others (van der Heijden, Tuinier, Kahn, & Verhoeven, 2004).

7.7 Summary of Diagnosis and Clinical Course of Reactive Psychosis

At this point it is worth briefly summarising what is known about the diagnosis and clinical course of reactive psychosis in this sample. There are a significant number of patients in this first-episode psychosis sample that have features resembling the traditional notions of reactive psychosis. There is a substantial proportion of patients who present with a stressor preceding their onset of psychosis, have a rapid onset, recover more quickly from both psychotic symptoms and social and occupational deterioration, have a trend to recover more quickly from negative symptoms, are possibly less likely to relapse (in this study only suggested by fewer hospital days in the 9 to 15 month period) during the first 18 months of treatment and have symptoms of associated affective disorders. Consistent with traditional notions of reactive psychosis, there are a disproportionate number of females in this group. It is also apparent that the reactive psychosis group uses fewer clinical resources in the later course of their treatment, remembering that there was a mandate to treat all first episode patients for eighteen months (currently revised to 2 years). They are also less likely to require longer periods of maintenance antipsychotic medication. However, although there are some preliminary findings to suggest our early psychosis reactive psychosis group had some similarities with traditional notions of reactive psychosis and could be distinguished from a non-reactive psychosis group, there were fewer than expected differences in the course of follow up indicating that the predictive

validity of the reactive psychosis construct in early psychosis is limited. It is also possible that that the RPRF's validity needs to be better established as distinguishing a clinical picture of reactive psychosis in early psychosis may be different from that in older patients with a more established course. Most notably, the reactive psychosis group, although making a quicker recovery from their index episode of psychosis than the non-reactive psychosis group, were not able to maintain this advantage later in the course of their illness.

Overall the findings of this study provide some support for the validity of reactive psychosis as a distinct diagnosis at least in its prevalence, and for the proposition that reactive psychosis has a more favourable initial recovery (both symptomatically and functionally), than non-reactive psychosis. The findings suggest the following criteria form a basis for the diagnosis of reactive psychosis:

(A) Presence of psychotic symptoms
(B) Presence of a stressor prior to the onset of psychosis
(C) Four of the following six features are present:
(i) The stressor is of at least moderate severity
(ii) The stressor occurs within the 4 weeks preceding onset of psychosis
(iii) Duration of the stressor is 4 weeks or less
(iv) The symptoms of psychosis develop relatively rapidly, specifically over a period of 4 weeks or less
(v) The psychosis has an identifiable psychological meaning clearly or probably related to the stressor
(vi) The psychological content of the psychotic symptoms clearly reflects the stressor, at least in some aspects.

This study has demonstrated that the Reactive Psychosis Rating Form has some utility as a screening instrument but we would advocate further research into its reliability and validity. The following section outlines the clinical implications of the findings, making way for potential modified first episode treatment recommendations for reactive psychosis.

7.8 Treatment Recommendations

Although we cannot positively answer the question as to whether this group, presenting as having a reactive psychosis, is a clinically distinct entity or a less severe form of psychosis to be placed on a continuum with other psychotic disorders, there is enough evidence suggesting their distinction, to begin to consider the clinical implications for this group of patients. The current guidelines for treating early psychosis (see Table 1 in Chapter 1) suggest a comprehensive approach focusing on low doses of antipsychotic medication as well as psychosocial treatments. However, there is an absence of clear guidelines for discontinuing medication (International Early Psychosis Association Writing Group, 2005) with current recommendations varying from at least a year after clear resolution of symptoms to 'indefinite'. Given the known risks of weight gain and diabetes in the long-term use of antipsychotic medication (Allison & Casey, 2001; Muench & Carey, 2001) and tardive dyskinesia (Llorca et al., 2002), it is important to reconsider these guidelines, at least for a subset of patients. A caveat could be added to guidelines declaring that those patients with reactive psychosis may benefit from discontinuing antipsychotic medication sooner than other diagnostic groups. This is line with the aforementioned 'new wave' of research using a clinical staging model as outlined in Chapter 1. If the reactive psychosis diagnosis is to become part of the clinical staging approach, clinicians will need to be trained to recognise, assess and diagnose reactive psychosis.

Furthermore, this group, although not presenting as any less acute, may benefit from considering more benign psychosocial treatments as first line treatment with close monitoring to ensure not only recovery, but also the absence of further deterioration. The trauma is clearly played out in the content of psychotic symptoms for a significant proportion of people providing more evidence that psychotherapy is indicated. Give the high proportion of patients who present with a history of trauma, psychological interventions may need to be informed by the work done in the field of trauma, all the while being mindful of the potential for further deterioration of an already fragile psyche. These findings warrant a more systematic evaluation of the role of psychosocial treatments for psychosis.

There is a distinct lack of focus on psychological interventions for psychosis, often seen as the 'poor cousin' to pharmacotherapy. Contributors to a recent book (Gleeson et al., 2008), inspired by enthusiasm from clinicians working in the field of psychosis, lament the gap between the 'new' enthusiasm for an integrated approach to the treatment of psychosis in the 1970s and current practice. With this publication, they call for a renewed global enthusiasm for a 'truly' integrated perspective on treatment of psychosis, one not held back by Big Pharma's (a term coined by John Read) political and economic interests which have diminished the role of the psychological and social aspects of the stress vulnerability model (Gleeson et al., 2008). This is but one obstacle, as many clinicians working in public mental health services may lack counselling skills, let alone skills in family work or psychotherapy specific for psychosis (Fadden, 2006). Furthermore, frequently large caseloads in public community health settings are often not conducive to psychotherapeutic interventions, and funding systems are overly focused on expediting throughput via early discharge to primary health system.

7.9 Limitations and Future Directions

The strengths of this study are that it is the first early psychosis study to examine the prevalence and outcome of reactive psychosis in a clinically representative or 'real world' setting, the good sample size and good follow up rates over a fifteen-month period. The study was conducted in a 'real world' psychiatric setting, which has its strengths in that it more accurately represents a first episode psychosis sample. However, establishing the validity of a diagnostic construct such as reactive psychosis as distinct from others may be constrained in a psychiatric setting where clinical and functional outcomes are influenced by treatment in a dose response manner and we are not observing the natural course of the disorder. One possible explanation for the findings that the reactive psychosis group make a better recovery is that they respond more quickly to treatment as there would be less delay in commencing treatment. Although EPPIC is a first episode psychosis service, patients who have had up to 6 months of prior treatment (i.e. defined as duration of antipsychotic medication) are still eligible for the service. This brings to bear one the inherent difficulties in conducting research in 'real world' settings. Unfortunately, data on how much treatment patients have had *prior* to entry to the service has not

been available for research purposes (nor is it clearly documented in a meaningful way), leaving the question open for future research endeavours (as discussed below). A further obvious limitation to the study is that the reactive psychosis rating form was being operationalised for the first time, requiring a 'cut-off' to be ascertained without the aid of a gold standard diagnosis or 'expert' clinician to validate each diagnosis, therefore all results need to be treated with caution.

Further limitations are in the way of potential confounding variables. In order to interpret preliminary findings that reactive psychoses have a better initial prognosis than non-reactive psychoses, it was crucial to ascertain any confounding variables that may have an impact on the outcome of psychosis. Aside from proportionally more females in the reactive psychosis group, it appeared that the two groups were comparable in terms of demographics, with an average age of 22 years. Their educational achievement, cultural background, and level of family support (as measured by likelihood of living with parents) were also comparable. Pillman et al. (2002) also observed a preponderance of females in their brief psychotic disorder sample as did Castagnini et al. (2007) who observed that 62% of their reactive psychosis sample was female. Although gender has traditionally been seen as a potential confounding variable in more established psychotic populations (Castle & Murray, 1991; Iacono & Beiser, 1992; Kendler & Walsh, 1995), its relationship in early psychosis is complex and there is little in the way of evidence to suggest that females have a better outcome than males (Hafner, et al., 1993; Larsen, et al., 1996). It is difficult to understand why there may be more females than males with reactive psychosis but it indicates that we could be looking at a distinct clinical syndrome.

It may be argued that those with a less reactive psychosis, or who present with a more typical psychotic presentation, would be associated with a greater genetic loading for psychosis in that it is less likely to be influenced by environmental factors alone. Unexpectedly, those with a non-reactive psychosis were not more likely to have a first degree relative with psychosis. Almost 16% of the reactive psychosis sample had a first degree relative with psychosis, compared with 12% of the non reactive group (although not a statistically significant difference). It is possible that this result is confounded by the possibility that the non-reactive group had been influenced by other environmental factors such as substance abuse, a significant

problem in this first episode psychosis group. Comorbid substance use was certainly not excluded from this study of a 'real world' sample. It is worth noting that 36% of the sample met ICD-10 criteria for psychotic disorder with substance use and although it was high in both groups it is difficult to tease out the effects it may have had on the development of psychosis. Examining the potential genetic influences and on reactive and non-reactive psychosis is well beyond the scope of this work, but such influences need to be kept in mind.

A criticism of the reactive psychosis construct contending to have a better prognosis than non-reactive psychosis is that one may presume that the reactive groups generally have better outcome because they have a better premorbid level of functioning, are unwell for shorter periods of time and therefore have a better prognosis. This study found that the reactive psychosis group had the same age of onset (21 years) but did have a better premorbid adjustment than the non-reactive psychosis group. Although few studies have measured premorbid adjustment, making comparison difficult, at least one study found that those diagnosed with brief psychotic disorder had a better premorbid adjustment (Pillman et al., 2002), in that they had achieved more educationally. Our measure of pre-morbid adjustment was broader than just disruption to schooling, which is almost inevitable in this comparatively young sample (Edwards et al., 1994). It is difficult to tease out the influence of premorbid adjustment on outcome in this younger population, as many had likely not completed schooling at the onset of psychosis. The relationship between premorbid adjustment and outcome has been minimised by the influence of DUP (see chapter 1 for a review), therefore any further inquiry in reactive psychosis may have to consider the potential contribution of both variables. As expected, there were significant differences in DUP between groups. Given the exploratory nature of the study, DUP was not controlled for in this study but may be considered a confound variable that could explain some of the differences in outcome in this study. An important consideration in any further work distinguishing reactive psychosis in an early psychosis group is the contribution of the reactive psychosis diagnosis to short and medium term outcome over and above the contribution of DUP.

Interestingly, there were no differences between reactive and non-reactive groups in presence or history of depressive symptoms (as measured by the number of

days depressed at 3 month time point) indicating that depressive symptoms did not act as a confound variable in relation to predicting outcome. This is inconsistent with the reported findings that the reactive psychosis group were more likely to meet the DSM-IV criteria for depression with psychotic features and needs further investigation to be better understood. One explanation could be that despite there being no differences in the frequency of depressive symptoms between the groups, reactive psychosis patients' depressive symptoms dominated their clinical picture, warranting a DSM-IV diagnosis of affective disturbance with psychotic symptoms over a schizophreniform/schizophrenia diagnosis. Consistent with results that reactive psychosis patients were more likely to meet criteria for Bipolar disorder, there was a trend for the reactive psychosis group to have more days with manic symptoms (as measured at 3 month time point) so this could be a potential confound, but its impact on the course of recovery in early psychosis is not well understood. It was beyond the scope of this study, but further research could consider using statistical modelling factoring in gender, premorbid adjustment, affective symptoms and DUP.

Other studies investigating trauma and psychosis have elucidated the content of the traumas and linked them to types of psychotic symptoms (Read et al., 2005) an obvious oversight in this study. It would have useful to explore the content of each stressor, its relationship to the type of psychotic symptoms and to examine cumulative stressors with the aim of deciphering if there is indeed a 'dose response' on the impact on psychosis. The RPRF's *objective* rating of the severity of stressor is also problematic as it needs to be considered that what may be considered a severe stressor for one person may not be for another (e.g. change of schools or residence). The scale could be revised to incorporate a subjective rating of the severity of stressor on the behalf of the patients. In addition, the instrument could be revised in order to develop a better understanding of the relationship between the stressor and the onset of psychosis, e.g., if a psychotic episode has a slow onset it may more difficult to attribute the onset to a specific stressor.

Furthermore, some illustrative case examples of reactive psychosis drawn from our sample would have improved our understanding of the clinical picture. However we did not seek ethics approval and consent for this at the inception of the study.

Psychogenic psychoses have in common that a vulnerable personality plays a role in the onset of this class of reactive psychoses (Clarke et al., 1995; Ekselius et al., 1994; Jaspers, 1963; Jorgensen et al., 1996; Strømgrem, 1974). A research endeavour could consider the role of personality disorders as a vulnerability to reactive psychosis in a first-episode psychosis population. A major oversight in this study is not assessing for axis II disorders (APA, 1994). This proposed direction would fit nicely into an anticipated, although controversial, new intervention within the EPPIC program, called "the Mauve Zone" which was due to commence implementation in 2009. The mauve zone refers to a complex group of young people who are being treated within the first-episode psychosis program who were presenting with psychotic symptoms, which were either complicated by additional co-occurring personality difficulties (cluster B) or different to the psychotic symptoms of those who were thought to have a more typical schizophreniform psychosis. It is estimated that 25 to 30% of first-episode patients being treated within the service meet full threshold criteria for borderline personality or anti-social personality disorder. Despite at least a century of clinical wisdom pointing out the overlap between the two, as C. Mulder (a senior clinician within the EPPIC service) states in her report "While we await further research into the area, we need to avoid dichotomous thinking, where patients are thought of as having either one disorder or the other, and remain open to the possibility that some patients may have both first-episode psychosis and personality dysfunction" (Mulder, 2009, p. 1).

In conclusion, distinguishing reactive psychosis from other psychotic disorders has some prognostic impact but the nature of this distinction needs to be further clarified. Pillman et al. (2002) argued that the relationship of reactive psychoses and schizophrenia is more complex than just a difference in severity and although it is possible to interpret the findings of this thesis as providing some preliminary support for Pillman's claim, we would advocate that further research is needed to clarify some of the questions raised in this thesis, before we can consider reactive psychosis as a separate diagnostic entity.

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Appendices

Appendix A: Royal Park Multidiagnostic Instrument for Psychosis

DIAGNOSTIC RESULTS SUMMARY – Royal Park Multidiagnostic Instrument for Psychosis

PATIENT'S NAME.....

UR NUMBER.....

DATE COMPLETED ..../...../.....

COMPLETED BY.....

KEY FOR SYMBOLS USED

D	Definite
P	Probable
+	Emphasizes positive symptoms
-	Emphasizes negative symptoms
+/-	Equal weighting of +/-
MC	Mood congruent
MI	Mood incongruent
*	Affective features considered by diagnostic system

A. SCHIZOPHRENIC DISORDERS

COURSE CONSIDERED

	D	P
Kraepelin (+/-)	❑	❑
Langfeldt (+/-)	❑	❑
*Feighner (+)	❑	❑
*DSM III		
Pos (+)	❑	
Neg (-)	❑	
Mixed (+/-)	❑	
Residual (-)	❑	
with Atyp Dep T1 (-)	❑	
with Atyp Dep T2	❑	
*DSM III R		
Pos (+)	❑	
Neg (-)	❑	
Mixed (+/-)	❑	
Residual (-)	❑	
with MDE NOS T1 (-)	❑	
with BAD NOS T2 (+/-)	❑	
*DSM IV		
Pos (+)	❑	
Neg (-)	❑	
Mixed (+/-)	❑	
Residual (-)	❑	
with MDE NOS T1	❑	
with BAD NOS T2	❑	
*ICD 10	❑	
CROSS SECTIONAL		
Schneider (+)	❑	
E. Bleuler (-)	❑	❑
*Cloninger	❑	
M. Bleuler (+/-)	❑	
*WHO (+/-)	❑	

B. SCHIZOPHRENIFORM DISORDERS

	D	P
Langfeldt (+)	❑	
*DSM III		
Pos (+)	❑	
Neg (-)	❑	
Mixed (+/-)	❑	
with Atyp Dep (+/-)	❑	
*DSM III R		
Pos (+)	❑	
Neg (-)	❑	
Mixed (+/-)	❑	
with Dep NOS (+/-)	❑	
*DSM IV		
Pos (+)	❑	
Neg (-)	❑	
Mixed (+/-)	❑	
with MDE NOS (+/-)	❑	
with BAD NOS (+/-)	❑	

C. SCHIZOAFFECTIVE DISORDERS

*Kasanin		❑
*Welner		❑
*RDC	SAM	❑
	SAD	❑
*Feighner	SAM	❑
	SAD	❑
*DSM III		❑

*Taylor and Abrams (+/-)	❑	
*RDC (+)	❑	❑
*RDC Residual and Depressive Disorder (-)	❑	

*DSM III R		❑
*DSM IV	SAB	❑
	SAD	❑
*ICD 10	SAM	❑
	SAD	❑
	SABM	❑
	NOS	❑

D. AFFECTIVE DISORDERS

BIPOLAR DISORDER (MANIC)		**D**
*RDC		
non-psychotic		❑
psychotic		❑
*DSM III		
non-psychotic		❑
psychotic	MC	❑
	MI	❑
	MC & MI	❑
*DSM III R		
non-psychotic		❑
psychotic	MC	❑
	MI	❑
	MC & MI	❑
BIPOLAR I DISORDER (MANIC)		
*DSM IV		
single episode with psychosis		❑
*ICD 10		
mania with psychotic features		❑
BIPOLAR DISORDER (MIXED)		
*DSM III		
non-psychotic T1		❑
non-psychotic T2		❑
psychotic	MC - T1	❑
	MC - T2	❑
	MI - T1	❑
	MI - T2	❑
	MC & MI - T1	❑
	MC & MI - T2	❑
*DSM III R		
non-psychotic T1		❑
non-psychotic T2		❑
psychotic	MC - T1	❑
	MC - T2	❑
	MI - T1	❑
	MI - T2	❑
	MC & MI - T1	❑

BIPOLAR AFFECTIVE DISORDER		D
*ICD 10		
-current episode manic with psychotic features		❑
-current episode severe depression with psychotic symptoms		❑
MAJOR DEPRESSIVE DISORDER		
*RDC		
non-psychotic		❑
psychotic		❑
*DSM III		
nonpsychotic		❑
psychotic	MC	❑
	MI	❑
	MC & MI	❑
*DSM III R		
non-psychotic		❑
psychotic	MC	❑
	MI	❑
	MC & MI	❑
*DSM IV		
-single episode, psychotic		❑
-recurrent episode, psychotic		❑
*ICD 10		
-Severe depression with psychotic symptoms		❑
-Recurrent depressive disorder current episode severe with psychotic symptoms		❑
BIPOLAR II DISORDER		
-depressed with psychosis specifier		❑

MC & MI - T2	❑
BIPOLAR I DISORDER (MIXED)	
*DSM IV	
most recent episode	
-depressed with psychosis	❑
-manic with psychosis	❑
-mixed with psychosis	❑

E. ATYPICAL PSYCHOSES	**D**	**P**
*SCAAPS Acute Psychotic State	❑	
*Cycloid	❑	❑
*Dongier	❑	
*Bouffee Delirante	❑	
*RDC UFP	❑	
*DSM III Brief Reactive Psychosis	❑	
*DSM III R Brief Reactive Psychosis	❑	
*DSM III Atypical Psychosis T1	❑	
*DSM III Atypical Psychosis T2	❑	
*DSM III R Psychotic Disorder NOS	❑	
*DSM III R Psychotic Disorder NOS and Depressive Disorder	❑	
*DSM IV Brief Psychotic Disorder	❑	
*DSM IV Psychotic Disorder NOS	❑	
*DSM IV Psychotic Disorder NOS with depression NOS	❑	
*ICD 10 Acute Transient Psychotic Disorder	❑	
*ICD 10 Other non organic psychotic disorder	❑	
*ICD 10 Other persistent delusional disorders	❑	

F. OTHER DIAGNOSES	**D**	**P**
*DSM III		
Acute Paranoid Disorder	❑	
Acute Paranoid Disorder with Atypical Depression	❑	
Paranoia	❑	
Paranoia with Atypical Depression	❑	
Shared Paranoid Disorder	❑	
*DSM III R		
Delusional Disorder	❑	
Delusional Disorder with Depressive Disorder NOS	❑	
Type (specify)	❑	
_____		
Induced Psychotic Disorder	❑	
DSM III		
Schizoid PD	❑	
Schizotypal PD	❑	
Other PD	❑	
DSM III R		
Schizoid PD	❑	
Schizotypal PD	❑	
Other PD	❑	
Alcohol related/induced psychosis	❑	❑
Drug related/induced psychosis	❑	❑
DSM IV		
Delusional Disorder	❑	
Delusional Disorder with Depression NOS	❑	
Schizoid PD	❑	
Schizotypal PD	❑	
Other PD	❑	
Shared Psychotic Disorder	❑	
Substance Induced Psychotic	❑	

G. NO PSYCHOTIC Dx FOR CURRENT EPISODE	❑

Disorder

ICD 10

Delusional Disorder	❑
Induced Delusional Disorder	❑
Schizotypal Disorder	❑
Substance use disorder	❑
Late onset substance use disorder	❑
Schizoid PD	❑
Other PD	❑

Appendix B: The Reactive Psychosis Rating Form

The Reactive Psychosis Rating Form (RPRF; Guldberg et al., 1996)

(1) Psychosocial stressor occurred (onset of psychosocial stressor) -specify time before onset of <u>active</u> psychotic symptoms	6	Within 48 hours
	5	Within 2-7 days
	4	Within 1-4 weeks
	3	Within 1-3 months
	2	Within 4-12 months
	1	more than 12 mths/continuous
	0	Impossible to rate
(2) Duration of stressor	6	less than 48 hours
	5	2-7 days
	4	1-4 weeks
	3	1-3 months
	2	4-12 months
	1	more than 12 mths/continuous
	0	Impossible to rate
(3) Severity of stressor (DSM-111R)*	6	Extreme (eg death of parent)
	5	Severe
	4	Moderate (eg change of residence, retirement, etc)
	3	Mild-moderate
	2	Mild (eg change of school)
	1	Very Mild
	0	Unknown/unable to be assessed
(4) Duration of psychosis	6	less than 48 hours
	5	2-7 days
	4	1-4 weeks
	3	1-3 months
	2	4-12 months
	1	more than 12 mths/continuous
	0	impossible to rate
(5) Development of psychosis	6	less than 48 hours
	5	2-7 days
	4	1-4 weeks
	3	1-3 months
	2	4-12 months
	1	more than 12 mths/continuous
	0	impossible to rate
(6) Comparison of functioning premorbidly with functioning post psychosis	6	Improved functioning post psychosis
	5	Mild improvement in functioning
	4	Same functioning post psychosis
	3	Mild reduction in functioning post psychosis
	2	Moderate reduction in functioning
	1	Moderate to severe reduction
	0	Unable to be assessed

(7) Perplexity/confusion/disorientation	6 marked spatial and temporal disorientation 5 spatial or temporal disorientation 4 misidentifications 3 perplexed, simple clouding 2 very mild 1 not present
(8) Psychological meaning of the Psychosis	6 clearly related to stressor 5 4 probably related to stressor 3 2 possibly related to stressor 1 not understandable 0 not applicable (no stressor)
(9) Psychological Content of the Symptoms	6 clearly reflects stressor 5 4 some aspects of stressor are symbolically reflected by psychotic symptoms 3 2 not clear whether psychotic 1 symptoms reflect stressor 0 clearly not related not applicable

DSM-III-R (APA, 1987, p 11) Severity of stressor rating scale: Adults & Children and Adolescents used to rate item 3 of the RPRF

Adults			
Code	**Term**	**Examples of stressors**	
		Acute events	*Enduring circumstances*
1	**None**	no acute events that may be relevant to the disorder	no enduring circumstances that may be relevant to the disorder
2	**Mild**	Broke up with boyfriend or girlfriend, started or graduated from school, child left home,	family arguments, job dissatisfaction, residence in high-crime neighbourhood
3	**Moderate**	marriage, marital separation, loss of job, retirement, or miscarriage	marital discord, serious financial problems, trouble with boss, or being a single parent
4	**Severe**	divorce, birth of first child	unemployment, poverty
5	**Extreme**	death of spouse, serious physical illness diagnosed, or being a victim of rape	serious chronic illness in self or child and ongoing physical or sexual abuse
6	**Catastrophic**	death of child, suicide of spouse, devastating natural disaster	captivity as hostage, concentration camp experience
0	**Inadequate information, or no change in condition**		
Children and Adolescents			
Code	**Term**	**Examples of Stressors**	
		Acute events	*Enduring circumstances*
1	**None**	no acute events that may be relevant to the disorder	No enduring circumstances that may be relevant to the disorder
2	**Mild**	Broke up with boyfriend or girlfriend, change of school	Overcrowded living quarters; family arguments
3	**Moderate**	Expelled from school; birth of sibling	Chronic disabling illness in parents; chronic parental discord
4	**Severe**	Divorce of parents; unwanted pregnancy; arrest	Harsh or rejecting parents; chronic life threatening illness in parent; multiple foster home placements
5	**Extreme**	Sexual or physical abuse; death of a parent	Recurrent sexual or physical abuse
6	**Catastrophic**	Death of both parents	Chronic life-threatening illness
0	**Inadequate information, or no change in condition**		

NB: Further information on the use of the scale is given in the DSM-III-R (APA, 1987; p 18)

Appendix C: Scale for the Assessment of Negative Symptoms

SCALE FOR THE ASSESSMENT OF NEGATIVE SYMPTOMS (SANS)

Name: _____ UR: _____ Date: ____ / ____ / ____ Rater: _____

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0 = None; 1= Questionable; 2 = Mild; 3 = Moderate; 4 = Marked; 5 = Severe

AFFECTIVE FLATTENING OR BLUNTING

1.	Unchanging Facial Expression The patient's face appears wooden, changes less than expected as emotional content of discourse changes.	0	1	2	3	4	5
2.	Decreased Spontaneous Movements The patient shows few or no spontaneous movements, does not shift position, move extremities, etc.	0	1	2	3	4	5
3.	Paucity of Expressive Gestures The patient does not use hand gestures, body position etc, as an aid in expressing his ideas.	0	1	2	3	4	5
4.	Poor Eye Contact The patient avoids eye contact or "stares through" interviewer even when speaking.	0	1	2	3	4	5
5.	Affective Nonresponsivity The patient fails to smile or to laugh when prompted.	0	1	2	3	4	5
6.	Inappropriate Affect The patient's affect is inappropriate or incongruous, not simply flat or blunted.	0	1	2	3	4	5
7.	Lack of Vocal Inflections The patient fails to show normal vocal emphasis patterns, is often monotonic.	0	1	2	3	4	5
8.	**Global Rating of Affective Flattening** This rating should focus on overall severity of symptoms, especially unresponsiveness, eye contact, facial expression, and vocal inflections.	**0**	**1**	**2**	**3**	**4**	**5**

ALOGIA

9.	Poverty of Speech The patient's replies to questions are restricted in amount, tend to be brief, concrete and unelaborated.	0	1	2	3	4	5
10.	Poverty of Content of Speech The patient's replies are adequate in amount but tend to be vague, overconcrete, or overgeneralised, and convey little information.	0	1	2	3	4	5
11.	Blocking The patient indicates, either spontaneously or with prompting, that his train of thought was interrupted.	0	1	2	3	4	5
12.	Increased Latency of Response The patient takes a long time to reply to questions; prompting indicates the patient is aware of the question.	0	1	2	3	4	5

13.	**Global Rating of Alogia** The core features of alogia are poverty of speech and poverty of content.	**0**	**1**	**2**	**3**	**4**	**5**

AVOLITION - APATHY

14.	Grooming and Hygiene The patient's clothes may be sloppy or soiled, and he may have greasy hair, body odour etc.	0	1	2	3	4	5
15.	Impersistence at Work or School The patient has difficulty seeking of maintaining employment, completing school work, keeping house etc. If an inpatient, cannot persist at ward activities, such as OT, playing cards etc.	0	1	2	3	4	5
16.	Physical Anergia The patient tends to be physically inert. He may sit for hours and not initiate spontaneous activity.	0	1	2	3	4	5
17.	**Global Rating of Avolition-Apathy** Strong weight may be given to one or two prominent symptoms if particularly striking.	**0**	**1**	**2**	**3**	**4**	**5**

ANHEDONIA - ASOCIALITY

18.	Recreational Interests and Activities The patient may have few or no interests. Both the quality and quantity of interests should be taken into account.	0	1	2	3	4	5
19.	Sexual Activity The patient may show a decrease in sexual interest and activity, or enjoyment when active.	0	1	2	3	4	5
20.	Ability to Feel Intimacy and Closeness The patient may display an inability to from close or intimate relationships, espec. with the opposite sex.	0	1	2	3	4	5
21.	Relationships With Friends and Peers The patient may have few or no friends and may prefer to spend all his time isolated.	0	1	2	3	4	5
22.	**Global Rating of Anhedonia-Asociality** This rating should reflect overall severity, taking into account the patient's age, family status, etc.	**0**	**1**	**2**	**3**	**4**	**5**

ATTENTION

23	Social Inattentiveness The patient appears uninvolved or unengaged. He may seem "spacey".	0	1	2	3	4	5
24.	Inattentiveness During Mental Status Testing Tests of "serial 7s" (at least five subtractions) and spelling the word "world" backwards: Score 2 = 1 error, score 3 = 2 errors, score 4 = 3 errors.	0	1	2	3	4	5
25.	**Global Rating of Attention** This rating should assess the patient's overall concentration, clinically and on tests.	**0**	**1**	**2**	**3**	**4**	**5**

Appendix D: Brief Psychiatric Rating Scale

BRIEF PSYCHIATRIC RATING SCALE (Version 4.0)

N/A	1	2	3	4	5	6	7
Not assessed	Not Present	Very Mild	Mild	Moderate	Moderately severe	Severe	Extremely severe

Rate items 1-14 on the basis of patient's self report during interview. Mark "N/A" for symptoms not assessed. Note items 7, 12, and 13 are also rated on observed behaviour during the interview. PROVIDE EXAMPLES.

1.	Somatic concern	N/A	1	2	3	4	5	6	7
2.	Anxiety	N/A	1	2	3	4	5	6	7
3.	Depression	N/A	1	2	3	4	5	6	7
4.	Suicidality	N/A	1	2	3	4	5	6	7
5.	Guilt	N/A	1	2	3	4	5	6	7
6.	Hostility	N/A	1	2	3	4	5	6	7
7.	Elevated mood	N/A	1	2	3	4	5	6	7
8.	Grandiosity	N/A	1	2	3	4	5	6	7
9.	Suspiciousness	N/A	1	2	3	4	5	6	7
10.	Hallucinations	N/A	1	2	3	4	5	6	7
11.	Unusual Thought Content	N/A	1	2	3	4	5	6	7
12.	Bizarre Behaviour	N/A	1	2	3	4	5	6	7
13.	Self-neglect	N/A	1	2	3	4	5	6	7
14.	Disorientation	N/A	1	2	3	4	5	6	7

Rate items 15-24 on the basis of observed behaviour or speech of the patient during the interview.

15.	Conceptual Disorganization	N/A	1	2	3	4	5	6	7
16.	Blunted affect	N/A	1	2	3	4	5	6	7
17.	Emotional withdrawal	N/A	1	2	3	4	5	6	7
18.	Motor Retardation	N/A	1	2	3	4	5	6	7
19.	Tension	N/A	1	2	3	4	5	6	7
20.	Unco-operativeness	N/A	1	2	3	4	5	6	7
21.	Excitement	N/A	1	2	3	4	5	6	7
22.	Distractibility	N/A	1	2	3	4	5	6	7
23.	Motor Hyperactivity	N/A	1	2	3	4	5	6	7
24.	Mannerisms and Posturing	N/A	1	2	3	4	5	6	7

25. Confidence in assessment:

_____

(1=not at all, 5 = very confident)

26. Assessment questionable due to:

1 Symptoms possibly drug induced
2 Underreported due to lack of rapport
3 Underreported due to negative symptoms
4 Patient unco-operative
5 Difficult to assess due to formal thought disorder
6 Other

27. Sources of information:

1 = Client, 2 = Parent/relative/friend, 3 = Mental health professional, 4 = File

Appendix E: Premorbid Adjustment Scale

PREMORBID ADJUSTMENT SCALE

CHILDHOOD (UP THROUGH AGE 11)

1. Sociability and Withdrawal

0 Not withdrawn, actively and frequently seeks out social contacts.
1
2 Mild withdrawal, enjoys socialisation when involved, occasionally seeks opportunities to socialise.
3
4 Moderately withdrawn, given to daydreaming and excessive fantasy, may passively allow self to be drawn into contact with others but does not seek it.
5
6 Unrelated to others, withdrawn and isolated. Avoids contacts.

2. Peer Relationships

0 Many friends, close relationships with several.
1
2 Close relationships with a few friends (one or two), casual friendships with others.
3
4 Deviant friendship patterns: friendly with children younger or older only, or relatives only, or casual relationships only.
5
6 Social isolate, no friends, not even superficial relationships.

3. Scholastic Performance

0 Excellent student.
1
2 Good student.
3
4 Fair student.
5
6 Failing all classes.

4. Adaptation to School

0 Good adaptation, enjoys school, no or rare discipline problems, has friends at school, likes most teachers.
1
2 Fair adaptation, occasional discipline problems, not very interested in school, but no truancy, or rare. Has friends in school, but does not often take part in extracurricular activities.
3
4 Poor adaptation, dislikes school, frequent truancy, frequent discipline problems.
5

6 Refuses to have anything to do with school - delinquency or vandalism directed against school.

ADOLESCENCE (EARLY, AGES 12 - 15)

1. Sociability and Withdrawal

0 Not withdrawn.
1
2 Mild withdrawal, enjoys socialisation when involved, occasionally seeks opportunities to socialise.
3
4 Moderately withdrawn, given to daydreaming and excessive fantasy, may passively allow self to be drawn into contact with others but does not seek it.
5
6 Unrelated to others, withdrawn and isolated. Avoids contacts.

2. Peer Relationships

0 Many friends, close relationships with several.
1
2 Close relationships with a few friends (one or two), casual friendships with others.
3
4 Deviant friendship patterns: friendly with children younger or older only, or relatives only, or casual relationships only.
5
6 Social isolate, no friends, not even superficial relationships.

3. Scholastic Performance

0 Excellent student.
1
2 Good student.
3
4 Fair student.
5
6 Failing all classes.

4. Adaptation to School

0 Good adaptation, enjoys school, no or rare discipline problems, has friends at school, likes most teachers.
1
2 Fair adaptation, occasional discipline problems, not very interested in school, but no truancy, or rare. Has friends in school, but does not often take part in extracurricular activities.
3
4 Poor adaptation, dislikes school, frequent truancy, frequent discipline problems.
5
6 Refuses to have anything to do with school - delinquency or vandalism directed against school.

5. Social-sexual aspects of life during early adolescence

0 Started dating, showed a "healthy interest" in the opposite sex, may have gone "steady", may include some sexual activity.
1 Attachment and interest in others, may be same-sex attachments, may be a member of a group, interested in the opposite sex, although may not have close, emotional relationship with someone of the opposite sex, "crushes" and flirtations.
2 Consistent deep interest in same-sex attachments with restricted or no interest in the opposite sex.
3 Casual same-sex attachments, with inadequate attempts at relationships with the opposite sex. Casual contacts with both sexes.
4 Casual contacts with the same sex, no interest in the opposite sex.
5 A loner, no or rare contacts with either girls or boys.
6 Antisocial, avoids and avoided by peers (Differs from above in that an active avoidance of others rather than passive withdrawal is implied).

ADOLESCENCE (LATE, AGES 16 - 18)

1. Sociability and Withdrawal

0 Not withdrawn.
1
2 Mild withdrawal, enjoys socialisation when involved, occasionally seeks opportunities to socialise.
3
4 Moderately withdrawn, given to daydreaming and excessive fantasy, may passively allow self to be drawn into contact with others but does not seek it.
5
6 Unrelated to others, withdrawn and isolated. Avoids contacts.

2. Peer Relationships

0 Many friends, close relationships with several.
1
2 Close relationships with a few friends (one or two), casual friendships with others.
3
4 Deviant friendship patterns: friendly with children younger or older only, or relatives only, or casual relationships only.
5
6 Social isolate, no friends, not even superficial relationships.

3. Scholastic Performance

0 Excellent student.
1
2 Good student.
3
4 Fair student.
5
6 Failing all classes.

4. Adaptation to School

0 Good adaptation, enjoys school, no or rare discipline problems, has friends at school, likes most teachers.
1
2 Fair adaptation, occasional discipline problems, not very interested in school, but no truancy, or rare. Has friends in school, but does not often take part in extracurricular activities.
3
4 Poor adaptation, dislikes school, frequent truancy, frequent discipline problems.
5
6 Refuses to have anything to do with school - delinquency or vandalism directed against school.

5. Social aspects of sexual life during adolescence and immediately beyond

0 Always showed a "healthy interest" in the opposite sex, dating, has gone "steady", engaged in some sexual activity (not necessarily intercourse).
1 Dated regularly. Had only one friend of the opposite sex with whom the patient went "steady" with for a long time. (Includes sexual aspects of a relationship, although not necessarily intercourse; implies a twosome, pairing off into couples, as distinguished from below).
2 Always mixed closely with boys and girls. (Involves membership in a crowd, interest in and attachment to others, no couples).
3 Consistent deep interest in same-sex attachments with restricted or no interest in the opposite sex.
4 Casual same-sex attachments, with inadequate attempts at adjustment to going out with theopposite sex. Casual contacts with both sexes.
5 Casual contacts with same sex with lack of interest in opposite sex. Occasional contacts with the opposite sex.
6 No desire to be with boys and girls, never went out with the opposite sex.

ADULTHOOD (AGE 19 AND ABOVE)

1. Sociability and Withdrawal

0 Not withdrawn, actively and frequently seeks out social contact.
1
2 Mild withdrawal, enjoys socialisation when involved, occasionally seeks opportunities to socialise.
3
4 Moderately withdrawn, given to daydreaming and excessive fantasy, may passively allow self to be drawn into contact with others but does not seek it.
5
6 Unrelated to others, withdrawn and isolated. Avoids contacts.

2. Peer Relationships

0 Many friends, close relationships with several.
1
2 Close relationships with a few friends (one or two), casual friendships with others.

3

4 Deviant friendship patterns: friendly with children younger or older only, or relatives only, or casual relationships only.

5

6 Social isolate, no friends, not even superficial relationships.

3. <u>Aspects of adult social-sexual life</u>

a. Married, presently or formerly:

0 Married, only one marriage (or remarried as a result of death of spouse), living as a unit, adequate sexual relations.

1 Currently married with history of low sexual drive, periods of difficult sexual relations, or extramarital affair.

1 Married, more than one time, currently remarried. Adequate sexual relations during at least one marriage.

2 Married, or divorced and remarried, with chronically inadequate sex life.

2 Married, and apparently permanently separated or divorced without remarriage, but maintained a home in one marriage for at least 3 years.

3 Same as above, but: divorce occurred over 3 years ago, and while married, maintained a home for less than 3 years.

b. Never married, over 30:

2 Have been engaged one or more times or has had a long-term relationship (at least 2 years) involving heterosexual or homosexual relations, or apparent evidence of a love affair with one person, but unable to achieve commitment such as marriage.

3 Long-term heterosexual or homosexual relationship lasting over 6 months but less than 2 years. (If stable, long-lasting homosexual relationship over 2 years, score as "3").

4 Brief, or short-term dating experiences (heterosexual or homosexual) with one or more partners, but no long-lasting sexual experience with a single partner.

5 Sexual and/or social relationships rare or infrequent.

6 Minimal sexual or social interest in either men or women, isolated.

c. Never married, age 20 - 29:

0 Has had at least one long-term love affair (minimum of 6 months) or engagement, even though religious or other prohibitions or inhibitions may have prevented actual sexual union. May have lived together.

1 Has dated actively, had several boyfriends or girlfriends, some relationships have lasted a few months, but no long-term relationships. Relationships may have been "serious", but a long-term commitment such as marriage was not understood to be an eventuality.

3 Brief, short-term dating experiences or "affairs" with one or more partners, but no long-lasting sexual experiences with a single partner.

4 Casual sexual or social relationships with persons of either sex with no deep emotional bonds.

5 Sexual and/or social relationships rare or infrequent.

6 Minimal sexual or social interest in either men or women, isolated.

GENERAL

1. <u>Education</u>

0 Completed college and/or graduate school, or professional school (Law, for example).
1 Completed High School and some college or vocational training school or business school (such as secretarial or computer programming schools).
2 Completed High School.
3
4 Completed eighth grade.
5
6 Did not get beyond fifth grade.

2. <u>During a period of 3 years up to 6 months before first hospitalisation or onset of first episode, patient was employed for pay or functioning in school.</u>

0 All the time.
1
2 Half the time.
3
4 Briefly, about 25 percent of the time.
5
6 Never.

3. <u>Within a period of a year up to 6 months before first hospitalisation or onset of first episode change in work or school performance occurred</u>

0 Abruptly.
1
2 Within 3 months.
3
4 Within 6 months.
5
6 Imperceptibly, difficult or not possible to determine onset of deterioration.

4. <u>During a period of 3 years up to 6 months before first hospitalisation or onset of first episode, frequency of job change. If working, or interruption of school attendance</u>

0 Same job held, or remained in school.
1
2 Job change or school interruption occurred two or three times.
3
4 Kept the same job more than 8 months but less than a year, or remained continuously in school for the same period.
5
6 Less than 2 weeks at a job or in school.

5. <u>Establishment of Independence</u>

0 Successfully established residence away from family home, financially independent of parents.

1
2 Made unsuccessful attempts to establish independent residence, lives in parent's home, but pays parents room and board, otherwise financially independent.
3
4 Lives in parent's home, receiving allowance from parents which patient budgets to pay for entertainment, clothes, etc.
5
6 Made no attempt to leave home or be financially independent.

6. <u>Global assessment of highest level of functioning achieved in patient's life</u>

0 Fully able to function successfully in and take pleasure from (1) school or job; (2) friends; (3) intimate sexual relationships; (4) church, hobbies etc. Enjoy life and copes with it well.
1
2 Able to function well in and enjoys some spheres of life, but has a definite lack of success in at least one area.
3
4 Minimum success and pleasure in three areas of life.
5
6 Unable to function in or enjoy any aspect of life.

7. <u>Social-personal Adjustment</u>

0 A leader or officer in formally designated groups, clubs, organisations, or athletic teams in senior high school, vocational school, college or young adulthood. Involved in intimate, close relationship with others.
1 An active and interested participant, but did not play a leading role in groups of friends, clubs, organisations, or athletic teams, but was involved in close relationships with others also.
2 A nominal member, but had no commitment to, groups of friends, clubs, organisations, etc. Had close relationships with a few friends.
3 From adolescence through early adulthood had a few casual friends.
4 From adolescence through early adulthood had no real friends, only superficial relationships.
5 From adolescence through early adulthood was quiet, seclusive, preferred to be by self, minimal efforts to maintain any contact at all with others.
6 No desire to be with peers or others. Either asocial or antisocial.

8. <u>Degree of Interest in Life</u>

0 Keen, ambitious interest in some of the following: home, family, friends, work, sports, art, pets, gardening, social activities, music, and drama.
1
2 Moderate degree of interest in several activities including social gatherings, sports, music, and opposite sex.
3
4 Mild interest in a few things such as job, family, quiet social gatherings. The interest is barely sustaining.
5
6 Withdrawn and indifferent toward life interests of average individual. No deep interests of any sort.

9. Energy Level

0 Strong drive, keen, active, alert interest in life. Liked life and had enough energy to enjoy it. Outgoing and adequate in meeting life.

1

2 Moderately adequate drive, energy, interest, as described above.

3

4 Moderately inadequate energy level. Tended toward submissive, passive reactions. showed some potential to face life's problems, but would rather avoid them than expend the necessary energy.

5

6 Submissive, inadequate, passive reactions. Weak grasp on life, does not go out to meet life's problems, does not participate actively, but passively accepts his lot without having the energy to help self.

Appendix F: Quality of Life Scale

Quality of Life Scale

Interpersonal relations

1. Household
2. Friends
3. Acquaintances
4. Social activity
5. Social network
6. Social initiative
7. Withdrawal
8. Sociosexual

Instrumental role

9. Occupational role
10. Work functioning
11. Work level
12. Work satisfaction

Intrapsychic foundations

13. Sense of purpose
14. Motivation
15. Curiosity
16. Anhedonia
17. Aimless activity
18. Empathy
19. Emotional interaction

Common objects and activities

20. Commonplace objects
21. Commonplace activities

Appendix G: Social and Occupational Functioning Scale

Social and Occupational Functioning Assessment Scale (SOFAS)

Code	**Use intermediate codes when appropriate, e.g., 45, 68, 72**
100	Superior functioning in a wide range of activities
91	
90	Good functioning in all areas, occupationally or socially effective
81	
80	No more than a slight impairment in social, occupational, or school functioning (e.g. infrequent interpersonal conflict, temporarily falling behind in schoolwork)
71	
70	Some difficulty in social, occupational, or school functioning but generally functioning well, has some meaningful interpersonal relationships
61	
60	Moderate difficulty in social, occupational, or school functioning (e.g. few friends, conflicts with peers or co-workers)
51	
50	Serious impairment in social, occupational, or school functioning (e.g. no friends, unable to keep a job)
41	
40	Major impairment in several areas, such as work or school, family relations (e.g. depressed man avoids friends, neglects family, and is unable to work; child frequently beats up younger children, is defiant at home, and is failing at school)
31	
30	Inability to function in almost all areas (e.g., stays in bed all day; no job, home, or friends)
21	
20	Occasionally fails to maintain minimal personal hygiene; unable to function independently.
11	
10	Persistent inability to maintain minimal personal hygiene. Unable to function without harming self or others or without considerable external support (e.g., nursing care and supervision)
1	
0	Inadequate information

Appendix H: EPPIC Service Utilisation and Outcome Questionnaire

EPPIC Service Utilisation and Outcome Questionnaire

Name: **6/12**

U.R.: **12/12**

Commencement date: **24/12**

Date of questionnaire: **Rater:**

Clinical file available to score FUTQ: **Yes** **No**

Case manager available for comment: **Yes** **No**

1. General Psychiatric Services Follow-up

Type of follow-up	Type of professional	Number of visits
A.		
B.		
C.		
D.		

2. Eppic Follow-up

Type of follow-up	Type of professional	Number of visits	Telephone calls
A.			
B.			
C.			
D.			
E.			
F.			
G.			

3. EPACT Home Treatment.

Date of admission	Date of discharge	Number of visits	Telephone calls
A.			
B.			
C.			
D.			

4. Medication.

Type of Mx	Dosage	Cpz equival.	Date started	Date stopped	Side effects	Compliance
A.						
B.						

5. Other treatment/therapy

Type of therapy	Number of visits
A.	
B.	
C.	
D.	

6. Accommodation

Accommodation	Number of days
A. Kent St	
B. Park St	
C. Residence 27	
D. Evans St	
E. Other	

7. Date of departure from EPPIC programme.

8. Reason.

0 Still involved with service
1 Discharged out of area
2 Wishes no further involvement with service
3 Suicide
4 Death by other means, probably unrelated to psychosis
5 Death related to current psychotic state
6 Well: archived/inactive
7 Completed 2 years with EPPIC
8 Being treated by other service: GP/Mental Health Clinic, etc

9. Rehospitalisation

	First admission	Second admission	Third admission
A.Date of admission			
B. Date of discharge			
C. Number of days; EPPIC Inpatients			
D.Number of days; other			

inpatients			
E. Psychiatric Services Code			
F. High dependency			
G. Psychotic relapse			

10. Relapses

	Time 1	Time 2	Time 3	Confidence
A. Psychotic relapse: onset				
B. Psychotic relapse: offset				
C. Depressive episode: onset				
D. Depressive episode: offset				
E. Manic episode: onset				
F. Manic episode: offset.				
G. Negative symptoms: onset				
H. Negative symptoms: offset				

11. Other non-psychotic relapses.

	Date of onset	Confidence	Date of remission	Confidence	Comments
A.Anxiety disorder					
B.Substance abuse					
C.Medical Condition					
D. Other					

12. Risk taking behaviour

	Time 1	Time 2	Time 3
A. Present/absent			
B. Date			

13. Attempted suicide

	Time 1	Time 2	Time 3
A. Present/absent			
B. Date			

14. Self mutilating behaviour

	Time 1	Time 2	Time 3
A. Type			
B. Date			

15.Aggressive/threatening behaviour

	Time 1	Time 2	Time 3
A. Present/absent			
B. Date			

16. Forensic

	Time 1	Time 2	Time 3
A. Present/absent			
B. Date			
C. Consequence			

Appendix I: Consent Form

Sample consent form
CONSENT/REQUEST TO PARTICIPATE IN A RESEARCH PROJECT

TITLE OF RESEARCH PROJECT: Community Education and Reducing Delay in Early Psychosis

Consent of Client 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 study and any risks to myself which are involved and I request to participate on the condition that I can withdraw my consent at any time.

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

1. Diagnostic interview at the beginning and end of my hospital stay, or during the time of contact with out-patients

2. Completion of self-rating forms

3. Contact six and twelve months after leaving hospital, or if seen in out-patients, six and twelve months after diagnostic interview

Signed: _____

Date: _____

Researcher:

I, ..., certify that I have fully explained the aims, risks and procedures of the study to the client named herein (or to the lawful guardian of such a client), and have handed to the client (or guardian) a copy of this consent together with a Plain English statement of the aims and procedures of the study and any risks to the client.
In my opinion, the client (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 client and his/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 Client's Signature

I, ... of

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

Signed:

_____
__

Date: _____

Appendix J: Ethics approval

ROYAL PARK HOSPITAL

ACCREDITED BY THE AUSTRALIAN COUNCIL ON HOSPITALS STANDARDS

ADDRESS ALL MAIL TO:
PRIVATE BAG No 3 P.O.
PARKVILLE, VICTORIA 3052

PARK STREET,
PARKVILLE, VICTORIA 3052
TELEPHONE (03) 389 2222
FAX: (03) 388 0382

ROYAL PARK RESEARCH & ETHICS COMMITTEES

SERVING HEALTH & COMMUNITY SERVICES
FACILITIES IN HEALTH REGIONS 1, 2 & 6

CONTRACT WITH RESEARCHERS
RESEARCH & ETHICS CONTRACT FOR APPROVAL
RESEARCH PROJECT

REFERENCE No ..

I, Stephen R. Chisholme .....acknowledge that I have Research & Ethics Committee approval for the project

An evaluation of early detection and optimal treatment in first-episode psychosis.

I agree to observe the following conditions:-

(1) To obtain approval of the hospital Superintendent/Director of Clinical Services where appropriate.
(2) To comply with the conditions as outlined in the final and Committee approved copy of the research protocol.
(3) I agree that any variation to the approved protocol or Plain Language Statement requires Research & Ethics Committee review, rewrite and consent.
(4) To provide a report by the 31st December each year or upon request by the Research and Ethics Committees. Failure to do this means cessation of the research protocol and re-application to the Committee must occur before recommencing.
(5) To provide a report on completion of the project and a copy of the published material where applicable.
(6) To ensure that the confidentiality and anonymity of all subjects and their records will be preserved at all times in the manner set out in the protocol.
(7) To provide all researchers and research participants with the Ethics Committee statement on monitoring research projects and a signed copy of the consent form when in use.

Signed: .....(Researcher) Dated: 18/3/96

Date of Final Approval Given by Research Committee: 23/2/96

Date of Final Approval Given by Ethics Committee: 27/3/96

Signed (Ethics Committee Representative): .....

PLEASE NOTE: FAILURE TO COMPLY WITH THIS CONTRACT WILL RESULT IN WITHDRAWAL OF ETHICS COMMITTEE APPROVAL TO CONTINUE THE PROJECT