SECTION 1

Introduction

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Rationale for and overview of the second edition of *The Recognition and Management of Early Psychosis*

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Introduction and rationale

This is the second edition of our book entitled *The Recognition and Management of Early Psychosis* first published in 1999 (McGorry & Jackson, 1999). Although this book is a second edition, every single chapter is completely new. In fact, we have an almost completely different set of authors for this second edition. The chief reason for this resides in the explosion of literature on early psychosis over the last decade. Different areas of interest have emerged along with another generation of researchers, clinicians and colleagues from a diversity of countries around the globe. Represented in this book are authors from Australia, New Zealand, Canada, the UK, the USA, Spain, the Netherlands, Germany, Denmark and Switzerland.

Using the search terms, 'early psychosis', 'first onset psychosis', 'first episode psychosis', 'first episode schizophrenia', 'at risk for psychosis', 'ultra high risk for psychosis' and 'prodrom* psychosis', we conducted electronic database searches (PsycINFO, MEDLINE, and Web of Science) to locate relevant articles and book chapters from 1988 to 1997. We then did the same for 1998 to 2007. We chose these two 10-year periods because the first edition of our book, although published in 1999, was completed a year before that (1998). The results are shown in Table 1.1, which provides a breakdown according to search term, electronic database and decade.

From Table 1.1, it can be seen that the three databases identify widely different total numbers of papers and different numbers of papers within the seven specific search term categories. Of the three databases, MEDLINE identifies the lowest number of total papers (4263 compared with the other two databases with 7750 and 9828, respectively). But it can be seen that irrespective of the database, there has truly been an explosion in the literature, with MEDLINE identifying a four-fold increase in publications for the 1998–2007 decade compared with the 1988–1997 decade, PsycINFO a 48-fold increase and Web of Science a 6.5-fold increase. Notably, the 'ultra high risk' and 'prodrome' categories have the lowest absolute numbers of publications uncovered in the 1998–2007 period, but very few articles were uncovered for the 1988–1997 decade, so there has been some increase in numbers over time for these two categories. This striking increase in the salience of the 'early psychosis' field reflects the growth of a major new paradigm in the mental health field.

Book overview

The book is organized into eight sections. The first introductory section, comprising this chapter and Ch. 2, provides an overview of the book's content and a staging-model approach to the prevention and intervention of early psychosis. In Ch. 2, McGorry, Allott and Jackson set the scene for a preventatively oriented approach to the recognition and management of early psychosis, and put forward a model for achieving this. The continuum of preventive intervention, namely, universal, selective and indicated prevention, developed by Mrazek and Haggerty (1994), is briefly reviewed. The authors then introduce a clinical staging model – a heuristic framework – which builds upon the

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Table 1.1. Number of citations pertaining to early psychosis in the decades prior to, and following, the first edition of *The Recognition and Management of Early Psychosis*

Search term	PsycINFO		MEDLINE		Web of Science	
	1988–1997	1998–2007	1988–1997	1998–2007	1988–1997	1998–2007
'early psychosis'	17	1071	388	1333	336	1838
'first onset psychosis'	5	133	174	509	144	767
'first episode psychosis'	42	2558	148	907	171	1834
'first episode schizophrenia'	89	3795	275	1210	361	2458
'at risk for psychosis'	6	85	3	2	473	2380
'ultra high risk for psychosis'	0	63	0	50	0	122
'prodrom* psychosis'	1	45	39	252	35	429
Cumulative total[a]	160	7750	1027	4263	1520	9828

[a] Note that there may be duplication of articles across search terms.

preventive intervention model by viewing psychosis as a stage-based illness, whereby each stage requires different treatment strategies and implies different prognoses. The rationale for a clinical staging approach to psychosis is, in part, related to the issues surrounding the validity of the diagnosis of psychotic disorder, particularly when prevention and early intervention is the focus. Issues impacting upon the validity of the diagnosis of psychosis include the fact that symptoms and syndromes are not necessarily concrete and stable across phases of disorder, especially at the earliest stages; the presence of 'non-specific' symptoms in the prodromal and first-episode phases of disorder that do not fall within the psychosis diagnostic category, but clearly require treatment; the phenotypic heterogeneity and continuum of patient presentations, which require clinical judgement as to their level of 'abnormality' or 'psychopathology'; and the relative non-specificity of neurobiological markers of illness. The authors, therefore, argue for a phase-of-disorder and treatment-oriented approach to diagnosis.

The remainder of the chapter is devoted to describing the early stages of the four-stage clinical staging model of diagnosis and intervention in psychosis, which guides the clinician in selecting the safest and most effective treatments that are most appropriate to the specific stage of illness. The clinical staging model implies that early successful treatment may improve prognosis and prevent progression to more severe stages of disorder. In line with the theme of the book, the early stages of psychosis are unpacked in detail in relation to the staging model: stage 0, increased risk for psychosis; stage 1a, mild non-specific symptoms, mild/moderate fall in functioning; stage 1b, ultra-high risk (UHR) or prodromal phase; stage 2, first-episode psychosis (FEP); stage 3a, incomplete recovery or treatment resistance; and stage 4, sustained disability and treatment resistance. Over time, and with further research, the eventual aim is to move toward a 'clinicopathological' staging model as in other disorders, which incorporates clinical phenomena, functioning and neurobiological variables.

In Section 2 (Chs. 3 to 5), the broad and critical area of risk and vulnerability for psychosis is explored, with a specific focus on the role of genetic, neurobiological and environmental risk factors and their interactions in the expression of psychotic illness. Chapter 3 (Weinberger and Berger) provides a comprehensive overview of current knowledge regarding the complex area of psychosis genetics. The authors point out that despite family, twin and adoption studies revealing a high genetic liability, with a point estimation of 81%, single major-effect genes have not been detected and the precise molecular aetiology of psychosis currently

remains unknown. The authors argue that the low effect size of individual marker loci and the heterogeneous phenotype of schizophrenia make replication of genome-wide linkage studies difficult. Nevertheless, linkage and allelic association studies have identified several candidate genes for susceptibility to psychotic disorder, including the genes for dysbindin (*DTNBP1*), neuregulin (*NRG1*), D-amino acid oxidase activator (*DAOA*; G72), regulator of G-protein signalling-4 (*RGS4*), catechol-*O*-methyltransferase (*COMT*), the 'disrupted in schizophrenia' genes (*DISC1* and *DISC2*) and the gene for brain-derived neurotrophic factor (*BDNF*). The authors also briefly describe the fields of intermediate phenotype (i.e. endophenotype) and epigenetic research, as adjuncts to traditional genetic approaches. It is argued that, to date, the evidence points towards either the involvement of multiple genes, with small effects across diverse populations or a heterogeneous aetio-pathology – or a combination of both. It is suggested that complex inheritance patterns of detrimental and protective genes may define the threshold for expression of psychosis, but only in the presence of certain environmental impacts (e.g. obstetric complications, substance use, stressful life events) and during critical developmental periods (e.g. prenatal, adolescence). The authors argue that, although the research base is still immature, genome-wide linkage studies and the identification of (new) genes in addition to intermediate phenotypes (e.g. cognitive dysfunction, abnormal brain function) is likely to improve the validity of diagnosis and provide inroads into formulating the staging model for psychosis and improving prevention and early intervention of the illness.

In Ch. 4, van Os and Poulton review the environmental risk factors for psychosis and their interaction with genetics. This approach differs from the linear gene–phenotype approach in that environmental risk factors are seen to play a *causal* role in the expression of psychosis and genes are believed to play an *indirect* role by moderating environmental impacts. Gene–environment relationships may reflect either gene–environment interaction ($G \times E$), which depicts how genetics moderate *sensitivity* to environmental factors to determine outcomes or gene–environment correlation (*rGE*), whereby differences in an individual's genotype may moderate *exposure* to differential environments. In studies aimed at detecting $G \times E$, *rGE* may operate as a confounding factor and needs to be ruled out. The authors review the evidence for $G \times E$ in psychosis based on first- and second-generation studies. Most of the evidence for $G \times E$ in psychotic disorder comes from first-generation studies using non-specific or indirect (proxy) measures of genes and environment, including epidemiological studies; twin, adoption and family studies; studies examining psychosis liability using psychometric measures; and studies of environmental impact upon DNA sequence and DNA methylation. Newer second-generation studies have directly tested for interactions between particular measured genes and environments. Specifically, the interaction between *COMT* genotype and cannabis use has received the most research attention. The results of these studies have shown strong evidence for an increased risk for psychosis in individuals who carry the COMT allele encoding valine (Val) at position 158 and use cannabis during adolescence. Van Os and Poulton highlight some of the methodological challenges associated with $G \times E$ research and suggest future research directions. The authors make the case that, through increasing our understanding of the combination of genetic risk factors (i.e. genetic polymorphisms, endophenotypes) with environmental exposures, it will be possible to make more robust predictions regarding transition to psychosis, thus improving early identification and intervention.

Pantelis and colleagues tackle the exploding field of neurobiology and early psychosis in Ch. 5. They review the relevant research for at-risk-for psychosis populations and first-episode psychotic patients under headings of neuropsychology, psychophysiology, functional imaging and structural imaging. Key neuropsychological findings suggest that prior to psychosis onset there are relatively subtle impairments in self-ordered working memory tasks, certain types of memory requiring rapid and complex organization of material, and in olfactory identification ability.

Regarding psychophysiological markers, there are mixed findings: the authors conclude that mismatched negativity may be a marker of progression rather than an endophenotype in the traditional sense. Similarly, it

is not entirely clear that P300 is a stable trait marker of illness as there is no clear evidence of progressive abnormality as with mismatched negativity, although this may suggest that the P300 may index poorer prognosis. In terms of P50, the authors conclude that this may be a more stable marker for the early recovery phase, although such deficits may be developmentally dependent. After exploring the available functional imaging studies conducted prior to and during the transition to psychosis, the authors review potential genes coding for brain maturation that may prove useful.

Finally, a number of brain structural abnormalities are identified as potential endophenotypes of schizophrenia and psychosis; however, a review of the studies in pre-psychotic individuals at high risk for transition to illness has not provided compelling evidence to support these abnormalities as illness-related markers (although some may prove promising). Rather, it would seem that many of the findings represent state-related abnormalities or changes that occur dynamically over the course of the illness.

The authors conclude that the dynamic brain changes occurring in adolescence and early adulthood may provide a context for interpreting some of the more important findings, where the most promising markers, such as certain executive functions (e.g. measured with self-ordered working memory tasks) and more direct measures of frontal lobe integrity (derived both from psychophysiology and functional and structural imaging), relate to frontal and perhaps temporal cortices; these are the brain regions that are changing dynamically during adolescence and early adulthood.

Nevertheless, Pantelis *et al.* sound a number of salutary warnings. They conclude that (1) the results reviewed by them do not support findings from studies of patients with more chronic psychosis; (2) the variables of interest may represent markers of illness progression and may not represent true endophenotypes; (3) other neurobiological factors may emerge with illness progression, or chronicity, or the same neurobiological factors may worsen; and (4) researchers have failed to take account of maturation as regards abilities and brain structures – the authors argue that brain structures are still developing around the time of the key ages of onset for both males and females. Abnormalities in patients with FEP or UHR for psychosis may represent failure to mature.

In keeping with the book's focus on prevention and early intervention, Section 3 deals with the identification and treatment of individuals with an 'at risk mental state' (ARMS) and the prediction of their transition to FEP. Researchers from Australia, Germany and the USA, namely, Yung, Klosterkötter, Cornblatt and Schultze-Lutter, author Ch. 6, which focuses on defining the putative prodromal or UHR population and identifying the factors that predict transition to psychotic disorder in UHR individuals. Retrospective reports show that FEP is generally associated with a prodromal phase. The psychosis prodrome has been reported to include non-specific signs and symptoms (such as depressed mood, anxiety, sleep disturbance and deterioration in role functioning), subtle self-experienced cognitive and affective disturbances known as 'basic symptoms' (such as thought interference, disturbance of receptive language and visual perception disturbances), attenuated or subthreshold psychotic symptoms, neurocognitive deficits, and neurobiological changes measured via magnetic resonance imaging (MRI). Increasing improvements in the methods used to identify those truly at high risk for psychotic disorder has paved the way for early intervention strategies in this population and increased the possibility of minimizing distress and disability and delaying or even preventing the onset of full-blown psychotic disorder.

Yung and colleagues describe three strands of research that have focused on the identification of 'prodromal' or UHR individuals: (1) early intervention studies conducted at the Personal Assessment and Crisis Evaluation (PACE) clinic in Melbourne, Australia, using 'close-in' UHR criteria developed by their research group; (2) schizophrenia 'basic symptoms' research and intervention conducted at the Early Recognition and Intervention Centre for Mental Crisis (FETZ) in Cologne, Bonn, Düsseldorf and Munich, Germany, using early and late initial prodromal state criteria; and (3) genetic high-risk studies investigating the causes of schizophrenia undertaken at the Hillside Recognition and Prevention (RAP) programme in New York, USA. These methods of identification of

people at risk of psychosis or schizophrenia have honed the identification from the general population rate of 1% to a rate of approximately 30%. However, some criticisms of this approach are highlighted, particularly the high false-positive rate that occurs in these selective samples. The second half of the chapter is mainly devoted to describing the predictive validity of a range of psychopathological (i.e. schizotypal features, positive psychotic phenomena, negative symptoms, basic symptoms, depression, anxiety and distress), clinical (i.e. poor functioning, substance use, stress), neurocognitive (i.e. working memory, olfactory identification, sensory gating) and neurobiological variables (i.e. hypothalamic–pituitary–adrenal axis function, brain structure and morphology) in predicting transition to psychosis. The goals of future work in this field are to improve the accuracy of predictive tools and further develop the most appropriate phase-specific interventions, while minimizing false positives and unnecessary iatrogenic harm.

In Ch. 7, Phillips, Addington and Morrison comprehensively review specific interventions for managing the broad range of symptoms and functional difficulties of individuals identified as having ARMS. Ethical considerations associated with the treatment of young people meeting ARMS criteria are flagged by the authors, including possible stigma associated with being labelled as having an ARMS, unnecessary treatment of 'false positives', how to discuss ARMS with individuals and their families, and how long treatment should be provided for. The bulk of the chapter is dedicated to outlining the case for and against specific interventions. Antipsychotic medication is reviewed first. Based on a limited number of studies with small samples, low-dose atypical antipsychotic drugs appear to at least delay, if not prevent, the conversion to fully fledged psychosis and enhance symptomatic and functional recovery, particularly in individuals in the late pre-onset period. However, there are potential risks and disadvantages associated with the use of antipsychotics in individuals with ARMS. These include the potential (serious) side effects associated with all antipsychotic medications (e.g. extrapyramidal side effects, weight gain, diabetes, sexual dysfunction), the possibility of 'feeding into' the commonly held belief that they are going 'mad', the potentially greater prominence of non-psychotic phenomena that may be best treated via other methods, the alleviation of psychotic symptoms that may be rightly or wrongly experienced by the individual as pleasurable or functional, and reduced acceptability of antipsychotic medication (as indicated by poor adherence and higher dropout rates in medication arms of intervention studies with this population).

Only one published study has specifically examined the efficacy of psychological interventions in ARMS individuals, but the results were very positive, with individuals who received cognitive therapy being less likely to progress to psychosis or be prescribed antipsychotic drugs than individuals who were simply monitored. The authors argue that psychological interventions may be most effective and more acceptable to patients during the earlier stages of the putative prodrome, when presenting symptoms are less severe and less specific. Some of the disadvantages associated with providing psychological treatment include the fear and stigma that may be associated with being labelled as having an ARMS and being in 'therapy' and the possible development of a sense of helplessness, although these disadvantages may be addressed by the psychological intervention. Phillips and colleagues briefly review other potential approaches, including social interventions, monitoring and no intervention, of which the last is believed to be the most common, but least optimal scenario. Research into the type and length of intervention that is most effective in ARMS individuals is still in its early stages; however, a number of services worldwide are dedicated to treatment and ongoing research with this population. The authors describe four examples of this; namely, the Prevention Risk Identification, Management and Education (PRIME) clinic in Toronto, Canada; the Personal Assessment and Crisis Evaluation (PACE) clinic in Melbourne, Australia; the Early Detection and Intervention Team (EDIT) in Salford, UK; and the FETZ in Cologne, Germany. The chapter concludes with a case study and general recommendations.

Chapters 8 to 10 discuss the improvement of identification of psychosis and access to services and the relationship between duration of untreated psychosis

(DUP) and outcome. The definition, measurement and associated outcomes of DUP are first tackled by Marshall, Harrigan and Lewis in Ch. 8. Prior to conducting a systematic evaluation of the evidence for the strength and specificity of the relationship between DUP and outcome, the authors thoroughly review the difficulties in defining and measuring DUP, believed to account for the large variability in estimates of DUP across studies. They report the most likely sources of measurement error to be (1) difficulties and inconsistency in defining the onset and offset of DUP; (2) reduced reliability associated with retrospective assessment and illness status of the individual when they are recalling illness onset; (3) discrepancies between the reports of patients and carers (e.g. reporting of subjective and objective phenomena); (4) sample bias (e.g. including only patients who are hospitalized or have non-affective psychosis); and (5) failure to use standardized assessment instruments. Bearing these methodological issues in mind, Marshall and colleagues systematically review follow-up studies of patients with FEP that have examined the association between DUP and outcome and they attempt to determine the degree to which premorbid adjustment may confound any observed association. Primary outcome variables that were included were measures of symptoms, overall functioning and symptom remission. Secondary outcome variables were quality of life, social functioning and measures of relapse. Twenty-six cohorts, with a mean DUP of 124 weeks (103 weeks with exclusion of one outlier), were included in the meta-analysis. Results showed that by 6–12 months following first presentation there was a significant positive correlation between DUP and a range of primary and secondary outcomes (i.e. the longer the DUP, the worse the outcome). Sixteen multiple regression analyses (from nine studies) examined the relationship between DUP and outcome while controlling for premorbid adjustment. After controlling for premorbid adjustment, the association between DUP and outcome remained significant in 12/16 analyses; this association was particularly robust between DUP and positive symptoms. These data provide a clear rationale for reducing DUP through early detection and intervention.

In Ch. 9, Jorm and Wright examine the role of community mental health literacy as a means of facilitating early intervention in psychosis. Their rationale is to reduce DUP by facilitating better and earlier recognition of psychotic (and prodromal) symptoms and help-seeking from appropriate professionals by the person who is affected and/or those close to them. Jorm and Wright review public knowledge about psychotic disorders, specifically focusing on the recognition of psychosis, mental health 'first aid' skills and beliefs about mental health professionals and treatments. One approach to assessing public mental health literacy is to present people with a case vignette of a person with schizophrenia or psychosis and ask the respondent what they believe is wrong with this person. Research shows that although many people recognize a mental health problem of some kind only a minority correctly label it as psychosis. Another approach is to assess the public's beliefs about the helpfulness of particular professionals and also the helpfulness of treatments. Interestingly, as regards the latter, the public tend to favour psychological treatments and be negative about medication and admission to hospital. The authors also examined studies comparing the beliefs of the public and professionals and found some consensus, but also some discrepancies; they also found some improvements over time in the public's mental health literacy for psychotic disorders.

Jorm and Wright then review interventions to improve mental health literacy for psychotic disorders, including four community campaigns conducted in four different countries, school-based programmes, and individual training programmes. They conclude that community campaigns can enhance awareness in the community and with referral sources can increase help-seeking and reduce DUP, particularly where the median DUP is long to start with. School-based programmes and individual training programmes are described as promising but are still in their infancy. The need for more research into the nature, specificity and strength of the relationship between mental health literacy and DUP is highlighted by the authors.

In Ch. 10, Norman and Malla explore pathways and barriers to receiving care and methods of reducing delay into treatment for early psychosis. Their rationale

for reducing DUP through early intervention is to improve outcomes, reduce unnecessary suffering and limit disruptions to social and role functioning commonly associated with psychotic illness. The authors first review what is known about the nature and determinants of help-seeking in early psychosis. Eight key observations regarding help-seeking are discussed: (1) help is often sought *before* the explicit onset of psychosis; (2) help-seeking is often initiated for reasons other than psychotic symptoms (e.g. dysphoria, anxiety, somatic concerns, deterioration in functioning); (3) help-seeking is often prompted by the actions of the sufferer's family or social network; (4) primary healthcare providers (e.g. primary care physicians) are often the first points of contact in accessing 'professional' treatment; (5) some barriers to help-seeking are potentially modifiable (e.g. knowledge of early warning signs and effectiveness of treatment); (6) once help is sought, there is wide variability in how readily it is provided (i.e. multiple contacts with helping professionals is common prior to the commencement of appropriate treatment); (7) there is considerable variation in who facilitates the final referral to appropriate services; and (8) accessing the appropriate service and receiving the correct diagnosis does not automatically denote prompt treatment.

In the next section, Norman and Malla explore the factors that predict treatment delay. There have been mixed findings regarding demographic and personal predictors of DUP; however, having fewer social contacts around the time of illness onset appears to be one factor that is relatively consistently associated with longer treatment delay. Some aspects of illness onset are more robust predictors of longer treatment delay, specifically poorer premorbid functioning, more gradual onset of illness and the presence of specific negative symptoms (i.e. apathy, social anhedonia). The authors conclude the chapter by describing approaches for reducing treatment delay for psychosis, including public education, training and education (i.e. 'up-skilling') of primary healthcare and social service providers, and the implementation of specific early detection programmes.

In Section 5, the chapters take on a practical clinical focus and are concerned with providing thorough and comprehensive assessment and treatment of the client experiencing a FEP or mania. In Ch. 11, Martin Lambert takes the reader through the various components of initial assessment and commencement of treatment – mostly pharmacotherapeutic in nature – of patients presenting with FEP. Key principles that underscore successful acute treatment (e.g. within the first 3 months) and provide a stable foundation for later treatment and maximum recovery are outlined first, including: (1) engagement and development of therapeutic alliance; (2) recognition of psychosis and understanding its personal context; (3) prompt non-traumatizing treatment of behavioural disturbances (e.g. agitation, pathological excitement, suicidal ideation); (4) achievement of symptomatic remission, functional recovery and quality of life; and (5) formulation of an individualized integrated treatment plan. Lambert then reviews the major elements of a comprehensive psychobiological assessment. These include detailed assessment of the individual's current and past psychiatric and personal history (with collateral information obtained from significant others); serial mental status examinations (MSEs); assessment of current and past comorbid axis I and II disorders and medical conditions, serial risk assessment, full biomedical evaluation, neuropsychological assessment and longitudinally based diagnostic evaluation. The remainder of the chapter is devoted to outlining best practice guidelines for pharmacological intervention in FEP, both non-affective and affective, including the management of psychiatric emergencies and the management of adverse events or side effects associated with pharmacological treatment (e.g. extrapyramidal motor symptoms, weight gain, metabolic syndrome, endocrine and sexual side effects). These guidelines are underpinned by a number of important principles: (1) the reduction of treatment delay improves antipsychotic response; (2) integrated treatment is a prerequisite for antipsychotic response (e.g. adjunctive psychosocial intervention); (3) separate approaches to initial pharmacotherapy are applied to non-affective and affective psychoses; (4) patients and relatives should be involved in treatment planning; (5) initial low-dose atypical antipsychotic treatment is recommended; (6) medication side effects should be avoided or treated early to promote response and

future adherence; (7) comorbid psychiatric disorder(s) can reduce treatment response and, therefore, should also be treated early; (8) medication adherence should be regularly monitored; (9) pharmacotherapy should be adapted according to diagnostic shift; (10) early identification of patients with an unfavourable outcome is crucial; and (11) some patients require a longer period (e.g. over 8 weeks) to achieve treatment response and remission.

The initial assessment and treatment phase is commonly termed the 'early recovery phase', which may be characterized by either complete or, unfortunately in some cases (ranging from 9% to 30% in the first 1–2 years), incomplete recovery. Chapter 12 by Canadian and Australian researchers and clinicians, Jean Addington, Tim Lambert and Peter Burnett, is concerned with exploring potential reasons for incomplete recovery and providing guidelines for pharmacological, psychological and social treatments during the early recovery phase and beyond. The authors differentiate 'remission' from 'recovery'; in addition to *symptomatic* (*positive and negative symptoms*) *remission*, recovery represents the ability to effectively *function* in social, vocational and community domains. Achievement of complete recovery, therefore, generally represents a longer-term process. The first part of the chapter focuses on the initial 3 months of treatment (i.e. early recovery phase). Reasons and recommendations regarding an inadequate response to initial pharmacological treatment are addressed, including poor efficacy, poor tolerability and medication non-adherence. A major focus is devoted to the rationale for and provision of psychosocial treatments during the early recovery phase, particularly in relation to addressing functional recovery and adaptation to psychosis. Several psychosocial interventions are described, including psychoeducation, individual cognitive – behaviour therapy (CBT), phase-specific group treatment, vocational rehabilitation and family work.

The second part of Ch. 12 centres on incomplete recovery following the initial 3 months of treatment. It is imperative that incomplete recovery is identified as early as possible. Incomplete recovery may be characterized by ongoing positive symptoms; the presence of negative symptoms, depression and anxiety; deficits in social and vocational functioning; poor quality of life; and/or cognitive deficits. When incomplete recovery is identified, the authors recommend a three-stage assessment and treatment approach. In stage 1, unmodifiable (e.g. long DUP, insidious illness onset, intellectual disability, neuropathology) and modifiable (e.g. comorbidity, inadequate psychosocial intervention, poor psychological adjustment, medication adherence) confounders of recovery must be identified. When there is a clear issue with medication adherence, stage 2 involves dealing with this via some form of adherence therapy or depot medication. In the situation where adherence has been effectively dealt with, but recovery remains incomplete, the clinician proceeds to stage 3, which involves determining whether incomplete recovery is a result of treatment resistance (estimated to affect at least 10% of patients with FEP). If treatment resistance is identified, the first line of treatment is clozapine. The authors conclude the chapter by describing treatment approaches for incomplete recovery, including medication strategies and individual, group-based and service-wide psychosocial treatments.

Although we ourselves remain skeptical around clarity of diagnosis in patients with first-episode bipolar disorder, we also acknowledge the burgeoning interest in this area. In Ch. 13, Conus and colleagues argue that Kraepelin's view of outcome for bipolar disorder was excessively optimistic. They conclude that, despite symptom remission, especially in the manic phase, assessment at follow-up shows poor functional or social recovery and high levels of comorbidity, including substance misuse. In line with principles of early intervention in psychosis, the authors make a case for early detection and early intervention in bipolar disorder, describing various factors responsible for the delay in diagnosing bipolar disorders and the unfavourable consequences associated with delayed diagnosis and treatment. The authors review extant treatment guidelines for bipolar disorders, concluding they are invariably based on patients with chronic disorders. They make the case for better definition of the disorder from vulnerability to initial onset to full-blown disorder, and the difficulties, but also the benefits, of doing so. They argue for treatments tailored to patients in the early phase of bipolar disorder and emphasize, in

addition to pharmacotherapy, the imperative of providing psychological treatments to such patients, providing some guidelines to best practice in this regard. It is clear from this chapter that much more research is needed into the early identification and treatment of first-episode bipolar disorder and several suggestions as to future research directions are offered by the authors.

The 'critical period' is tackled by the next two sections of the book. First, specific topics pertaining to embedded, comorbid and/or secondary psychopathology in early psychosis are dealt with in Section 6. Substance use or misuse (i.e. abuse or dependence) is ubiquitous, is a problem in itself, is frequently comorbid with psychosis, and is a risk factor for other disorders and for relapse or retarded recovery from a psychotic break. In Ch. 14, Wade and colleagues report that individuals with psychotic disorders are at an increased risk for substance misuse and regular tobacco use compared with individuals with other mental disorders or the general population. In FEP, the lifetime rate of substance misuse is at least 40%, with the most frequently misused substances being cannabis and alcohol. For most individuals, the onset of substance misuse precedes the onset of positive psychotic symptoms, and many continue misusing substances despite involvement with treatment services. Wade and others outline the correlates and consequences of substance misuse in FEP, highlighting the poorer outcomes associated with ongoing substance misuse following entry into treatment. The authors describe three hypotheses that have been proposed to explain the high rate of substance use among individuals with psychosis: (1) that psychosis increases the risk of substance misuse (i.e. self-medication hypothesis); (2) that substance misuse increases the risk for psychosis; and (3) that there are common risk factors for both psychosis and substance misuse. To date, the second hypothesis has received the most empirical research support.

Wade and colleagues then review the evidence for the efficacy of psychological interventions targeting substance misuse in psychosis. Of the relatively few randomized controlled trials conducted to date, findings are mixed, but they do provide some support for psychoeducation, motivational interviewing, CBT and nicotine-replacement therapy in reducing substance misuse and improving secondary outcomes such as mood, other psychopathology and antipsychotic medication dosage. The authors conclude the chapter by providing guidelines for the implementation of interventions for substance misuse with individuals with FEP. These interventions need to be administered within the one treatment setting, via an integrated and comprehensive approach, and in a 'stepped-care' fashion. They include engagement, initial and ongoing assessment and formulation, assessment of motivation to address substance misuse, the provision of assessment feedback and psychoeducation, harm-minimization strategies, motivational interviewing and CBT.

Chapter 15, by Power and Robinson, focuses on the serious issue of suicide prevention and early intervention in FEP. Suicide is a perennial problem and has an elevated risk in psychosis, affecting up to 15% of individuals with psychotic disorders and representing the leading cause of unnatural death during the first 10 years of illness. Power and Robinson provide the legal definition of death by suicide and describe the process and manifestation of suicide during non-psychotic compared with psychotic phases, highlighting the diverse range of mental states and their potential effects on suicidal ideation and behaviour. The authors identify the first years after diagnosis of psychosis as critical, as suicide is more likely to occur during this stage of the illness, particularly during the early recovery phase (e.g. the months following discharge from hospital) – a period that may be characterized by the emergence of insight and feelings of hopelessness, depression and loss. Suicide risk assessment and formulation is, therefore, vital in early psychosis. The authors provide guidelines as to how to conduct comprehensive clinical suicide risk assessments and when hospitalization may be indicated. They emphasize the importance of a collaborative approach (involving the patient, carers and other services) to the initial risk formulation and risk management plan and careful documentation of the same. Power and Robinson provide a comprehensive review of the factors to consider when conducting suicide risk assessments. These include (1) biological risk factors, such as genetics/family history, neurochemical