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Introduction

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Psychiatry has recently rediscovered its roots. It seemed as if its long history of interest in the impact of society on the rates and course of serious mental illness had been forgotten, overtaken by the inexorable advance of neuroscience and genetics. However, as our knowledge of the physiological and genetic processes linked to psychosis has advanced, it has become increasingly clear that social conditions and experiences over the life course are important in the aetiology of psychosis. Old dichotomies and controversies are giving way to genuinely integrated models, in which social, psychological and biological factors are seen to interact over time, culminating in the onset of psychosis. The influence of society extends beyond onset to shape course and outcome, with important implications for public policy and service delivery. In this context, it is useful to take stock of what is currently known about the links between society and psychosis, limitations to this knowledge, unanswered questions and future research priorities. *Society and Psychosis* aims to do this.

Categories and continua

There have been many attempts to define psychosis. Wing (1978), for example, gave a relatively narrow description: 'A 'psychotic' state is one characterised by delusions or hallucinations, in which the individual is unable to differentiate his grossly abnormal thought processes from external reality and remains unaware of his deficiency.' (pp. 44–5.) Less restrictive definitions include hallucinatory experiences that the sufferer realises are abnormal and, more broadly still, others include disorganised speech and grossly disorganised behaviour (APA, 1994). Psychotic symptoms can occur in a range of disorders identified in the *Diagnostic and Statistical Manual* (APA, 1994) and the *International Classification of Diseases* (WHO, 1992), including schizophrenia spectrum disorders, affective disorders, a range of brief psychotic disorders and grief reactions.

The purposes of classification and diagnosis in psychiatry are the same as in the rest of medicine. That is, diagnosis is intended to communicate information about

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symptoms, aetiology, prognosis and optimal treatment. In relation to psychotic mental disorders, there have been recurrent questions about whether specific diagnoses, particularly schizophrenia, provide such information reliably. For example, it has long been acknowledged that the outcome of schizophrenia is variable. While the textbook account – that approximately a third recover, a third have an episodic course and a third have a continuous course – may need to be revised as new research emerges, there is, nevertheless, clear heterogeneity in outcome for those diagnosed with schizophrenia (and those with other psychotic disorders) (Menezes *et al.*, 2006). Likewise, responsiveness to antipsychotic medication is not uniform, and there is a sizeable minority of subjects who remain resistant to most common forms of treatment. Furthermore, an increasing body of recent research suggests that large numbers of people in the general population experience psychotic (or psychotic-like) symptoms: 10–15% in some studies (Verdoux and van Os, 2002). As a consequence, the debate has resurfaced on whether psychotic disorders are discrete entities, marked by a clear disjunction from normal experience, or whether they lie on a continuum with normality (van Os *et al.*, 2000). This debate is fuelled by research in cognitive psychology focusing on specific psychotic symptoms, such as hallucinations and delusions, rather than on diagnostic categories (see Chapter 14). The lack of diagnostic specificity of such positive psychotic symptoms is one observation that has led some to argue that it is negative symptoms (e.g., blunted affect, asociality, anhedonia, poor self-care, etc.) that are at the core of schizophrenia. This is also contributing to the renewed debate about the validity and utility of schizophrenia as a diagnostic entity (Bentall, 2003; Lieberman and First, 2007).

This book is concerned with psychosis in a broad sense, and the tension between whether the focus should be on psychotic symptoms, conceived as lying on a continuum with normality, or on discrete diagnosable psychotic disorders will be evident throughout these pages. As this issue remains unresolved, this tension is welcome; research from both perspectives promises to increase understanding and in time will, hopefully, contribute to resolving this debate. This is not simply an academic point. Efforts to understand and treat psychosis will depend to a large degree on accurate conceptualisations, and it may be that our current efforts are hampered by lack of clarity over what the unit of investigation should be: symptoms, such as delusions and hallucinations, or categories, such as schizophrenia and bipolar disorder. This is one of the central issues in psychosis research.

A final point on this is necessary. While this book is concerned with psychosis in a broad sense, as much of the existing research focuses on schizophrenia, this will frequently be used as an example, on the basis that understanding schizophrenia in particular may give us insights into psychosis in general.

Changing views of the epidemiology of schizophrenia

One of the basic tenets of the epidemiology of schizophrenia has been that the incidence is more or less uniform around the world (Crow, 2000). The WHO multi-country studies of the 1970s and 1980s contributed much to establishing this orthodoxy, particularly the finding from the Determinants of Outcomes of Severe Mental Disorders (DOSMeD) study that there were no statistically significant differences between the 12 centres studied in the incidence of narrowly defined schizophrenia (Jablensky *et al.*, 1992). The apparent invariance of schizophrenia has been taken as evidence that the disorder is primarily genetic; the usual variability that would be expected if the occurrence of schizophrenia was influenced by local social environments was simply not evident (Crow, 2000).

In recent years, new research and meta-analyses have challenged the interpretation that schizophrenia, even narrowly defined, has a uniform incidence (Cantor-Graae and Selten, 2005; McGrath *et al.*, 2004). A comprehensive meta-analysis of 100 incidence studies by John McGrath and his colleagues (2004) at the University of Queensland found marked variations in the incidence of psychosis by place and persons. For example, the variation in incidence rates between sites covered in the studies reviewed was more than fivefold. The review further confirmed higher rates in urban centres and in migrant groups, this latter finding being replicated in a more specific review (Cantor-Graae and Selten, 2005). In fact, from the beginning, the interpretation of a uniform incidence did not go unchallenged. A number of commentators pointed out that, although statistically non-significant, there was a twofold difference between the highest and lowest reported incidence rates for narrow schizophrenia in the DOSMeD study, and, for broadly defined schizophrenia, there were marked differences between the various centres (Kleinman, 1991). As McGrath (2007) has commented, it seems that the contours of the epidemiology of schizophrenia are not flat after all.

An uneven epidemiological terrain does not, in itself, point towards a particular aetiology, but it does open the door for investigating causes through the lens of differences in incidence between populations and places.

The aetiology of psychosis

The causes of schizophrenia and other psychoses have been the subject of intense research efforts and frequently acrimonious debates. In the crudest terms, these debates have centred on the question of whether the causes reside in individual biology, intrapsychic conflict or socioenvironmental stress. At various points there have been attempts to bridge these positions within biopsychosocial frameworks (e.g., Engel, 1980). However, it is arguable that, for all the lip service paid to some

kind of vague biopsychosocial model of aetiology, at various points one side or other has dominated. In the past 20 years, for example, the dominant view has been that schizophrenia (psychosis) is a genetic brain disease, the onset of which is the product of a neurodevelopmental process (Andreasen, 2000). Social factors, if they have been assigned a role at all, have been relegated to the status of triggers, serving merely to hasten the onset of a largely biologically determined disease. This view, however, is changing.

The proposition that socioenvironmental factors are aetiologically important in psychosis has, in the past, been undermined by two particular problems. First, as schizophrenia and other psychoses are often preceded by a period of functional decline, leading to problems in maintaining social relationships and employment, it is extremely difficult to determine the causal direction of any association between markers of socioeconomic adversity and schizophrenia. Second, the mechanisms by which society impacts on individuals to increase risk of schizophrenia and other psychoses have been poorly specified. The numbers of people who are exposed to adverse social conditions, traumatic life events, and so on, far outstrip the numbers who ever experience serious mental illness. The types of adverse social conditions associated with psychosis are not specific (they are also associated with a range of other disorders), and most people who are exposed do not develop a serious mental illness. If such experiences are relevant to the onset of psychosis, how is it that such a relatively small proportion develops schizophrenia? The chapters in Part II of this book address these questions directly.

There are at least three developments that are contributing to the renewed interest in the role of the social environment in the aetiology of psychosis. First, as already discussed, it is becoming clear that there are notable variations in the incidence of psychosis both between and within countries. The higher incidences in urban centres and in migrant and ethnic minority groups, in the absence of concrete evidence one way or the other, at the very least suggests that there are social factors that occur more commonly in these settings and groups and that merit further study. Second, there has been a series of recent studies that have overcome the problem of direction of causation by using data from large population-based registers and prospective cohorts (Janssen *et al.*, 2004; Pedersen and Mortensen, 2001). These have continued to produce findings that link exposure to negative social experiences and circumstances prior to the development of psychosis and subsequent onset (e.g., Spauwen *et al.*, 2006). Where the extent of exposure, either in terms of frequency or severity, has been measured, some of these studies have found evidence of dose–response relationships, such that the greater the exposure to, say, sexual abuse, the greater the risk of psychosis (e.g., Janssen *et al.*, 2004). Finally, and perhaps most importantly, one consequence of the recent rapid advances in neuroscience and genetics is that we are beginning

to understand how social experience along the life course interacts with genotype, and impacts on biological development, to shape adult outcomes. These insights are now being used to produce biological models linking adverse social experiences, including childhood trauma, and adult psychosis (e.g., Spauwen *et al.*, 2006; Teicher *et al.*, 2003). All of the chapters in this book that address aetiology reflect this development; they all propose candidate mechanisms that, at least in theory, could account for the observed associations between the various social exposures and psychosis. Vague notions of susceptibility or diathesis, proposed in the past, are being replaced by concrete evidence-based biological mechanisms linking social experience with brain development and psychosis (Teicher *et al.*, 2003).

Course and outcome of psychosis

In contrast to the controversy that surrounds the possible role of socioenvironmental factors in the aetiology of psychosis, it is generally accepted that the social environment can influence the course and outcome of psychosis. Over 30 years ago, Wing and Brown (1970) showed how living in long-stay institutions contributed to the development of behaviours and symptoms that had been assumed to be intrinsic features of schizophrenia. There is now a considerable body of research showing that critical and hostile (i.e., high expressed emotion) home environments can increase the risk of relapse, particularly in the absence of antipsychotic medication (Kavanagh, 1992). Further, negative social attitudes and responses towards those with psychosis exclude many from opportunities for employment and productive social relationships, opportunities that have been shown to promote recovery (Warner, 2000). The finding from the WHO DOSMeD study, that outcomes are better in developing than in developed countries, is usually interpreted in these terms (Jablensky *et al.*, 1992), i.e., as reflecting the fact that responses to psychosis in the developing world are less stigmatising and sufferers are more readily reintegrated back into family and social groups. This interpretation, however, has never been fully tested and new analyses are beginning to question whether the course and outcome really is more benign in the developing world (Patel *et al.*, 2006).

Research further shows that interventions designed to modify social environments and promote social reintegration can improve course and outcome (Leff and Warner, 2006). The classic example is family intervention to reduce levels of expressed emotion (Kuipers *et al.*, 2002). However, the use of specific targeted social interventions in routine mental health care is sporadic at best, and research on social interventions is swamped by that on psychopharmacology. To a degree, the introduction of novel antipsychotic medication has provided further impetus to psychopharmacological research; whether these deliver the advertised benefits over and above first-generation neuroleptics is questionable (Jones *et al.*, 2006; Lieberman

et al., 2005). In contrast, research on psychosocial interventions is slight; again, however, there are signs of change, particularly with an increasing number of studies of cognitive interventions for psychosis (e.g., Kuipers *et al.*, 2006).

Society and psychosis

The primary purpose of this book is to reflect these current trends in the study of society and psychosis, and to contribute to developing an agenda for future research. There have been many swings and trends in psychosis research, as noted above. In Chapter 2, Julian Leff sets the scene by surveying the shifting fashions of psychiatric research. By reflecting on his own involvement in research over the past 30 years, and analysing trends in the publication of psychosocial and biological papers in the *British Journal of Psychiatry* and the *American Journal of Psychiatry*, Leff argues that the wider social, economic and political context often determines what research is funded and published. It is for future analyses to assess the external pressures that are shaping current shifts towards more fully integrated biopsychosocial models of psychosis. The hope is that, with each shift, we move closer to a fuller understanding that allows for more effective interventions.

Theoretical and conceptual foundations

The first part of the book provides a series of orientating chapters. In attempting to understand the relationship between society and psychosis, there is much that can be learned from the social sciences. The historical relationship between psychiatry and the social sciences, however, has been fraught, and scepticism concerning the role of the social environment in the aetiology of psychosis is reflected in continuing scepticism about the value of the social sciences. In Chapter 3, Craig Morgan provides an overview of this often acrimonious relationship and outlines a number of areas in which the social sciences can provide important contributions to current efforts at investigating links between society and psychosis. In Chapter 4, Dana March and her colleagues provide an introduction to conceptualising the social world. To understand how social conditions and experiences impact on individuals, we need conceptual tools that allow us to define and measure what are continual social processes. As research now shows broad associations between relatively crude variables (e.g., urbanicity, migration) and risk of psychosis, there is a need to move on to investigating directly the social processes that potentially underpin these relationships. In this, basic conceptual and theoretical work will be essential.

Perhaps the one area with the greatest potential for clarifying the nature of the relationship between the social environment and risk of psychosis is that of gene–environment interaction. As more research emerges, showing that the impact of a specific environmental factor, such as life events or cannabis

consumption, on the risk of psychosis is influenced by genotype, this will become an increasingly important area of study. In Chapter 5, Jennifer Barnett and Peter Jones provide a detailed conceptual and methodological overview of gene–environment interplay in psychosis. The ideas introduced here are picked up and illustrated with specific examples in many of the chapters in the second part of the book. The prominence given to gene–environment interactions in these chapters further emphasises the extent to which the social and biological are being combined in current psychosis research.

Social factors and the onset of psychosis

The social environment can be considered at different stages and at different levels: for example, at the level of the individual, the family or society. The chapters in the second part of the book review specific areas of research, setting out what is currently known, the limitations to what is known and, as appropriate, methodological issues and challenges for future research.

In the first of these, Chapter 6, Jane Boydell and Kwame McKenzie examine ecological-level research, an area gaining increasing attention, partly because of the repeated finding that rates of psychosis are higher in urban centres (van Os, 2004), and partly because of increasing interest in social capital and mental illness (e.g., McKenzie and Harpham, 2006). In Chapters 7 and 8, research on early childhood adversity and intrafamilial factors is reviewed. These are contentious areas. In Chapter 7, Helen Fisher and Tom Craig consider the evidence for a link between forms of childhood trauma, including sexual and physical abuse, and the risk of psychosis. Their review reaches a more tentative conclusion than other recent commentators in this area (Read *et al.*, 2005), pointing to important methodological issues for future research. Fisher and Craig present a preliminary theoretical framework as a guide for subsequent research. In Chapter 8, Pekka Tienari and Karl-Erik Wahlberg examine research on families and psychosis. This is a particularly sensitive topic given the unfortunate history of families, particularly mothers, being blamed for causing schizophrenia. As Tienari and Wahlberg explain, families do not cause psychosis. It may, nonetheless, be that certain forms of communication within families impact on child development in such a way as to increase vulnerability to later emotional and mental disorder, Where there is also a genetic susceptibility, the two may interact to increase risk of psychosis. However, these are not predestined pathways, and individual resources and subsequent positive experiences may be protective. The potential links between early adversity and later adversity is one of the themes of Chapter 9, in which Inez Myin-Germeys and Jim van Os consider research on adult adversity. While reviewing the field in general, Myin-Germeys and van Os also present data from a series of innovative studies assessing the impact of daily hassles on the development and exacerbation of psychotic symptoms. It is apparent

from this work that a range of different factors operate over the life course to increase susceptibility to psychosis. The development, or exacerbation, of psychotic symptoms in the vulnerable may be provoked by specific life events or regular daily stresses.

In the final chapter in this part, Chapter 10, Kwame McKenzie and his colleagues focus on migration, ethnicity and psychosis. Within a broad review of this field, they focus in detail on the evidence that the African-Caribbean population in the UK is at greatly increased risk of psychosis and, from this, propose a preliminary sociodevelopmental model of psychosis.

Social factors and outcomes

The third part of the book contains three chapters focusing, broadly, on social responses to psychosis and their effects. In the first, Chapter 11, Richard Warner shows that social interventions can impact positively on the course of psychosis and sufferers' quality of life. In Chapter 12, Graham Thornicroft and his colleagues provide a detailed and wide-ranging review of literature on stigma and psychosis. Schizophrenia remains heavily stigmatised, and sufferers frequently experience discrimination and social exclusion. Such adverse societal responses may worsen outcomes and quality of life for those with schizophrenia. What Chapter 12 makes clear is the need for urgent strategies to tackle stigma and promote social reintegration. In Chapter 13, Kim Hopper reviews the intriguing finding that the outcomes of schizophrenia may be better in developing than developed countries; a finding that, as noted above, has long been considered as evidence that social and cultural contexts are major determinants of course and outcome.

Models and conclusions

In parallel with a resurgence of interest in social factors and psychosis, there has been a rapid development of research from a cognitive psychology perspective, focusing on specific symptoms and examining the role of variables, such as attributions and emotion, in the aetiology of psychosis (e.g., Bentall, 2003). In much of the book, the focus is very much on how social experience interacts with biology to increase the risk of psychosis. A further framework for linking these is a cognitive model of psychosis. In Chapter 14, Paul Bebbington and his colleagues review this expanding field and explain how a cognitive model can provide a further explanatory link between social adversity and psychosis; a framework, moreover, that retains the important role of biology and, arguably, begins to resemble a genuinely biopsychosocial model of psychosis.

In the final chapter, we present a formulation of the state of the art of research into the impact of society on psychosis, and offer thoughts on an agenda for future research. However, distinguishing social from biological research, particularly in relation to aetiology, is increasingly artificial. Studies on the impact of social

factors will need to take account of the potential mediating role of a number of biological variables, including genotype and biochemistry. There appears to be an emerging consensus that new research needs to be undertaken with, rather than in isolation from, specialists in the biological and psychological sciences. Integration of different fields and different types of knowledge is the way forward for research into psychosis and is reflected throughout the chapters of *Society and Psychosis*.

Despite the clear importance of investigating social aspects of psychosis and all the work that has been done to date, there is still much more that needs to be done. Scientists always seem to conclude with a call for more research. We argue for a different type of research, using new methodologies and conceptualisations, which will help us to link knowledge of the social world with knowledge of genetics, biology and psychology to increase our understanding of psychosis.

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