

Schizophrenia: orthodoxy and heresies. A review of alternative possibilities

T. M. KEEN MSc RMN RNT

Senior Lecturer, Institute of Health Studies, University of Plymouth, Exeter Campus, Exeter, UK

Correspondence:

T.M. Keen

Senior lecturer

Institute of Health Studies

University of Plymouth

Exeter Campus

UK

KEEN T. M. (1999) *Journal of Psychiatric and Mental Health Nursing* 6, 415–424

Schizophrenia: orthodoxy and heresies. A review of some alternative perspectives

Conflicts about the nature, causes and treatment of schizophrenia have never ceased. Recently, however, a particular set of beliefs ('the Orthodoxy') has become influential, and dominates political and managerial agenda for controlling clinical practice and educational initiatives. Theories and therapeutic strategies that differ from the particular biological, behavioural, cognitive and family management approaches favoured by orthodox clinicians have been given far less academic and clinical air-space. This review surveys some alternative ideas and practices ('the Heresies') and critically refers to some of the orthodox tenets such as the genetic and biological bases of schizophrenia, the disease model, 'family blaming' and drug treatment. Heresies briefly explored include schizophrenia as an evolutionary inevitability, creative sublimation of schizotypal tendencies and systems-theory-based family therapies.

Keywords: anti-psychiatry, family management, family therapy, schizophrenia

Accepted for publication: 23 July 1999

Introduction

In its century of existence, 'schizophrenia' has never been the subject of professional consensus. The diagnosis has been a fertile source of contention since 1898 when Kraepelin expanded Morel's term 'dementia praecox' to subsume the pre-existing concepts 'hebephrenia', 'catatonia' and 'paranoia' (Kringlen 1994, Thomas 1997). Kraepelin postulated the existence of a single disease that he believed likely to have a metabolic cause. A decade later, Bleuler reformulated Kraepelin's proposals and suggested the name 'schizophrenia' for a group of disorders with complex aetiologies, including psychosocial causes.

Kraepelin imputed organic causation, pessimistic prognosis and emphasized the incomprehensibility of his putative illness. Bleuler more optimistically used interactional and social perspectives to stress the comprehensibility and psychodynamic causes of at least some of his proposed group of reactions. As Clare (1976, p.119) has pointed out, this schism has been reflected in divergent, apparently irreconcilable approaches to the conceptualization, identi-

fication and treatment of 'schizophrenia' throughout the century.

Opinion has always been conflicted about the nature and causes of the diagnosis. Does it validly describe a real disease, represent spurious pseudo-medical categorization or provide a convenient shorthand for a group of disorders (Bentall 1993)? Is it, or are they, functional (Arieti 1974, 1979) or organic (Andreasen 1985) in nature? Should emphasis be placed upon genetic, biological, interactional, social or economic causes (Warner 1985, Davey 1996)? How should we respond to distressing experiences and behaviour resulting from the disorder/s? Has the diagnostic process itself failed to conform to sound scientific practice? (Boyle 1996). The supposedly discredited 'antipsychiatric' theories of the 1960s (see Laing 1960, 1961, Szasz 1962, Scheff 1966, Cooper 1967) continue to echo in professional debate (Boyle 1997, Johnstone 1997a, 1999). 'Schizophrenia' has many critics, who find that the diagnosis lacks logical, moral, or clinical integrity (Warner 1985, Johnstone 1989, Bentall 1990, 1992, 1993, Boyle 1990, 1996, Jenner *et al.* 1993, Thomas 1997).

During the 1970s, international failure to agree diagnostic criteria chronically bedevilled research (Buckley *et al.* 1995). Psychiatric texts commonly claim that this problem disappeared when agreed diagnostic systems (Wing *et al.* 1974) were accepted in the late 1970s, but some recent research suggests otherwise. British and French psychiatrists continued to use different diagnostic criteria in the early 1990s (Van Os *et al.* 1993), and a survey of British psychiatrists found that views of schizophrenia remained largely individualistic (Cape *et al.* 1994).

Given the history of argument about the diagnosis, it is not surprising that nurses' views are disparate and contradictory. Where consensus exists, it is likely to be based upon simplistic beliefs couched in loose terminology or vaguely expressed theories (Keen 1996). However, recent material published in nursing journals suggests that academic and clinical opinions about schizophrenia have become largely united. An orthodoxy has emerged which dismisses alternative research findings or theoretical possibilities (see Gournay 1995, 1996, Repper 1995, McCrone 1996, Coffey 1998). This remarkable conformity is founded upon advances in neurological research technology: quantitative methodology, developments in 'expressed emotion' theory, and allied medical-behavioural approaches to case management. These scientific approaches derive from psychiatric medicine and emphasize the 'treatment' role of nursing. Playle (1995) describes a complementary 'caring' role, which focuses on the art of nursing, and is underpinned by a humanistic rather than a purely scientific theory.

Dissenting voices can occasionally be heard amongst the sceptical ranks of mental health nurses (Clarke 1997, 1999, Dawson 1997, 1998), but objections to excessive 'scientism' in nursing are given short shrift (e.g. Rogers 1999). The etiological claims, conceptual certainties, biological facts and treatment imperatives which comprise the emergent scientific orthodoxy are enshrined in influential clinical recommendations (e.g. SIGN 1998). Similar guidelines will probably be endorsed by the new quality assurance machinery assembled by the British Department of Health (DoH 1998) and thenceforward provide the basis for mandatory clinical treatment and nursing care. There are however, several issues about schizophrenia which seem to have been casually disregarded, or treated with injudicious contempt in the campaign to get mental health workers 'on-message' (see CSAG 1995). Some of these possible truths are so at odds with the orthodox assertions that they may be similarly described as heretical beliefs, despite any intrinsic rational or empirical worth.

Tenets of the schizophrenia orthodoxy

- Schizophrenia is a biological disease (Gournay 1995), not a psycho-social response or an existential crisis.
- Because schizophrenia is a biological disease, neuroleptic medication is the appropriate first line of treatment (McKenna 1997).
- Lack of insight can lead to noncompliance with prescribed medication regimes, so patients need persuasive education and, if necessary, legal coercion to gain their co-operation (Kissling 1994, Macpherson *et al.* 1997).
- Schizophrenia is of genetic origin. Faulty genes cause neuro-anatomical, neuro-chemical or neuro-developmental defects, which manifest in the disease (Murray & McGuffin 1993, Gottesman 1994).
- Because of its primarily biological or genetic origin, childhood experiences, family life or upbringing cannot cause schizophrenia. Parents and colleagues in close relationships do not induce schizophrenia in vulnerable infants (Barrowclough & Tarrier 1992).
- Family members become stressed by years of attempting to understand, cope with and care for their vulnerable schizophrenic. This stress leads to relatives developing high levels of emotional involvement, detachment, hostility or criticism. These behaviours reciprocally stress their vulnerable relative, and induce relapses. Family management strategies aim to reduce relapses by supportive, problem-solving and psycho-educational interventions (Falloon *et al.* 1984, Falloon & Fadden 1993).
- Previous formulations about schizophrenia were guilty of 'blaming' families for causing the disease, thus hurting already grieving people, and alienating potentially valuable partners in psychiatric treatment (Kuipers *et al.* 1997).

Alternative possibilities – some schizophrenia heresies

- The diagnoses of schizophrenia encompass a number of different conditions. Some are likely to conform to medical definitions of disease, whilst others may better be described as response patterns, or contextual disability. Outcomes vary widely and there is wide diagnostic heterogeneity (Harding & Zahniser 1994, Frangou & Murray 1996).
- It is a myth that patients must continue taking neuroleptics all their lives. Only a small percentage of sufferers probably need indefinite medication (Harding & Zahniser 1994, Reed 1999).
- To establish that schizophrenia (however, construed)

is biological does not imply that it is a disease. All human behaviours, thoughts and feelings, whether conscious or unconscious) are biological. The phenomena called schizophrenia could be construed as behavioural and experiential responses across a range of human difference, rather than as disease, deficit or defect (Claridge 1989, Jenner 1993).

- The genotypes of schizophrenia are probably far more commonly and widely distributed in the general population than revealed by the clinically diagnosed, damaged tip of the schizotypal iceberg (Richter 1984, Crow 1994, Chadwick 1997).
- The easy presumptions of genetic determinism should be doubted. The human genome is better understood as a flexible blueprint, subject to environmental interpretation, adaptation and elaboration, rather than as a rigid, bio-psychological absolute despot (Jones 1996, Clarke 1999, Dawson 1998).
- There is evidence that parental communication and affective styles are significant environmental factors in the initial development of a diagnosis of schizophrenia (Tienari *et al.* 1994, Lehtonen 1994, Chadwick 1997, Dawson 1998).
- Systems-theory-based family therapies which predate the orthodox 'family management' models can be effective in changing both family interaction and schizophrenic pathology (Burbach 1996). Profound long-term changes can be demonstrated which go beyond the more modest or pessimistic claims of family management protagonists (Jones 1987).
- The convoluted logic implicit in the orthodox assertions about schizophrenic family functioning (Johnstone 1993) disguise the political and economic imperatives underlying the official support for orthodox family management and medication strategies.

The increasing acceptance of cognitive treatments for schizophrenic symptoms (Birchwood 1999) is an adjunct to the new orthodoxy that might previously have been considered heretical. Basic psychiatric texts once insisted that schizophrenic 'signs' and 'symptoms' had no personal meaning or significance. The so-called 'utterances' of a schizophrenic were merely the psychiatric equivalent of bad-breath or spots, revealing underlying pathology. Schizophrenic emotion, thoughts, beliefs, perceptions or actions had no metaphorical meaning, no explanatory power or personal-historical reference. However, persevering, dissident clinicians and users have slowly been able to win approval for less dismissive beliefs (Romme & Escher 1993). Cognitive interventions potentially narrow the distance between some orthodox and heretical assertions, and lend empirical validity to earlier dissident

hypotheses (see Kingdon & Turkington 1994, Fowler *et al.* 1995, Haddock & Slade 1996, Nelson 1997, Garety & Hemsley 1997).

A many splendoured thing . . .

Probably the least controversial heresy is the claim that there are many forms of schizophrenia. Both major systems of psychiatric classification (DSM IV & ICD 10) describe several subcategories of the primary diagnosis, yet much of the literature continues to emphasize its singularity, and to discuss the search for single genetic or neurological causes. It is difficult to find any rational counter-argument to the proposition that there are probably many conditions covered by the umbrella-term 'schizophrenia'. Even orthodox protagonists concede when writing for colleagues that there are probably at least two: a genetic and a nongenetic form (Leff 1996). It seems pointless to debate the possible cause; it is likely that there are several. Moreover each kind of 'schizophrenia' will probably have several etiological factors, from genetic through neuro-biological to psychosocial, interactive and economic.

Liddle (1987) determined three different schizophrenic syndromes using the factor analysis. Later research using PET scans confirmed three distinct patterns of cerebral blood flow, each related to the three symptom-clusters and to different neuro-anatomical regional structures (Liddle *et al.* 1992). However, so many subdivisions and atypical responses exist, that one eminent neuro-researcher concludes it is better to investigate each behavioural sign or psychological symptom separately (Frith 1992). This is a conclusion shared by at least one dissident psychologist (Bentall 1993). Idiosyncratic classifications, paradoxical observations and genetic vagaries have so frustrated psychiatrists, that it has not only been suggested that attempts at classifying schizophrenia should be abandoned, but also that psychiatry should return to the concept of a single psychosis, encompassing schizophrenia, the affective disorders and some personality disorders (Kringlen 1994). Rather than eventually being shown to be a family of disorders, the schizophrenias may well turn out to be more like a bus-queue: unrelated, but superficially demonstrating enough common purpose, location or behaviour to be treated by a casual researcher as demographically singular.

You are what you eat . . .

It was possible for mainstream psychiatry to discredit radical theories of schizophrenia like Laing's (Laing, 1960) partly because of his apparent contempt for the organic brain, rather than the functional mind. It seems absurd to protest against the profound simplicity of the fact that

everything we think, feel, will or do is biologically directed. This is as true of human love, artistry, science or society as it is of our sadness, badness or madness. Whilst this assertion may spiritually challenge many (and it certainly did in the paradoxically psychedelic 1960s), it cannot be scorned or wished away. Acknowledging mind-brain unity as a scientific fact (despite human vanity and all the unknowns and complexities) may move the debates about schizophrenia into more productive areas (Gournay 1995, 1996). Accepting the fundamentally biological nature of our humanity, however, does not lead inevitably to embracing neuro-psychiatric models of schizophrenia. The key concept is that of *continuity* vs. *discontinuity* (Claridge 1989) or *dimensionality* (Richter 1984).

The discontinuity model of illness presumes that disease states are qualitatively different from normal functioning. One either has or does not have a broken leg, carcinoma or abscess. Such states are abnormal and pathological, subject to medical diagnosis, treatment and cure. The concept of continuity stresses the quantitative range of a condition. One can be more or less intelligent, have a typical blood-pressure at any point of a long continuum, or show more or less of the facets of schizophrenia. The continuity model suggests that schizophrenic signs and symptoms are exaggerated or contextually shifted forms of normal behaviour or mental processes.

The orthodox illness model construes schizophrenia as discontinuous with normal states, whereas the continuity model implies that schizophrenic phenomena may be better understood as disabilities, if any categorization is necessary. I may be disabled by an excessively slow heartbeat, and a doctor will inform me I am suffering from bradycardia. It would be misguided however, to pathologise a highly trained athlete whose slow pulse represents an adaptation that enables success in a specific area of human activity. Furthermore, however fit, we are all prone to influenza, which occurs in many bafflingly mutating forms. We occasionally have so much of the viral beast in our systems that we become symptomatic and say we have 'flu'. (However, whether symptom-free or ill, the term 'influenic' has no common usage!)

A disease or some ways of being human?

The continuity/discontinuity distinction underlies many disagreements about schizophrenic phenomena (Thomas 1997) and begs the question 'What psychological function/s may be analogous with physical signs like blood pressure, etc., and lead to diagnoses of schizophrenia when apparently beyond normal range?' One authoritative suggestion implicates the human capacity for symbolic communication and language development (Crow 1994).

Crow's interpretation of the World Health Organisation 10 Country Study (Jablensky 1993) is that the incidence of schizophrenia is constant both geographically and over time. This is remarkable given the enormous global variation in environmental factors, and the breeding disadvantage conferred by the disease. The fact that people with schizophrenia produce less children than the average population (Nanko & Moridairo 1993), and that the disease appears genetically and socially disabling should lead to its evolutionary extinction. Crow concludes that as this appears not to be the case, there must be some evolutionary advantage associated with the diagnosis:

'One solution to the paradox is that the genes which predispose to psychosis are also those that have contributed to the evolution of ...the capacity for language and complex social interactions.' (Crow 1994, p. 41)

This suggestion which, as Crow states, is of enormous significance, appears even more plausible when set alongside recent findings from neuro-imaging research.

Neuro-imaging studies suggest that much schizophrenic cognition involves significant differences in the use of various frontal and temporal brain centres (Frith 1992). These centres are implicated in concept formation and language production, and correspond closely to the areas implicated in the clinical syndromes proposed by Liddle (1987, Liddle *et al.* 1992). More recent findings in brain imaging provide further corroboration (Vogeley & Falkai 1999). Frith allies his findings with psychological research to propose a cognitive neuro-psychological hypothesis explaining the development of schizophrenic symptoms. In order to develop interactive skills and use the language to a consistent social effect, two underlying cognitive functions must be present: the 'theory of mind' and 'meta-representation'. 'Theory of mind' is the ability to accurately perceive another person's intention, and 'meta-representation' refers to the necessity to constantly remind ourselves of the context in which we are thinking, feeling or communicating. ('*I wish that was my money*' or '*I'm pretending that I am a tree.*') If we fail to maintain awareness of this underlying context, then we are left with only unrealistic representations ('That was my money' or 'I am a tree'). Frith's theory offers fascinating possibilities for developing nursing techniques and strategies based on cognitive correction. It also raises tantalizing echoes of earlier theories of schizophrenic function: the use of paralogical or proto-linguistic metaphor, such as the Von Domarus hypothesis (Maher 1970); the inability to cope easily with conflicting messages (Bateson 1972); and a loose construct formation (Bannister & Fransella 1971).

An individual thus affected may well be disabled in producing contextually appropriate communication behaviours, or develop bafflingly unusual convictions about

the nature of specific experiences, beliefs or perceptions (see Peters *et al.* 1999). There are many not diagnosed as schizophrenic in society who may merit that description, when their behaviour or experiences are measured by conventional yardsticks or against conformist values. In order to explore the implicit possibilities, neuro-imaging research could focus more widely than diagnoses and random controls. It may be instructive to investigate the cerebral functioning of eccentric inventors, artists and writers, religious mystics, channellers and mediums, innovative musicians, political dissidents, experimental film-makers, cult leaders, method actors and colleagues whose interests and aptitudes lie outside of conventional belief systems or communication modes.

Crow's hypothesis was anticipated a decade ago:

'Detrimental genes responsible for a serious illness such as schizophrenia would be gradually eliminated from the population by normal processes of natural selection and breeding disadvantage unless associated with some favourable characteristic which tended to increase the frequency of the genetic alteration' (Richter 1984, pp. 104–5).

In trying to identify just what that 'favourable characteristic' may be, studies of artists, writers, poets, and intellectuals resulted in some significant findings (Richter 1984, p. 105). Crow's analysis suggests that schizophrenia as clinically diagnosed represents only a pathological minority of people who whilst possessing similar neuro-anatomical or neuro-functional changes, remain undiagnosed.

These others are likely to include all those biologically, genetically predisposed people who do not, when adopted away, develop the diagnosis. It also includes all the monozygotic twins in twin-studies who remain unschizophrenic, despite their genetically identical brother or sister becoming ill (Gottesman 1994). Furthermore, it seems reasonable to suggest that there are others. Ordinary people, neither schizotypal twins nor adopted children of schizophrenics, but born with the same cerebral differences, may grow up escaping or responding differently to whatever stresses led to the expression of diagnosable behaviour in other vulnerable individuals. Presumably such a hidden mass of nondiagnosed schizophrenics would have been able to put their cognitive abnormality, or symbol-crunching difference to sublimated effect. A supposed defect becomes a talent which can be put to good use, perhaps, in media work, linguistics, philosophy, information technology, art and design, or psychology.

Arieti (1976) offers an analysis of the similarities between creativity and madness that helps make schizophrenic excesses seem less alien and more meaningful. Morter (1997) clarifies the familiar role that artistic media can play in bridging the gap between professional self and

schizophrenic other. Chadwick (1997, chapters 2 and 3) gives a reasoned account of positive advantages in the perception and the reasoning conferred by the schizophrenic difference. He lists artists and musicians who have been able to sublimate their experiences creatively, and describes his own struggle to disentangle an intact self from imposed moral and cultural strictures. His experiences will strike chords in many mental health workers whose involvement with patients goes beyond assisting in medical treatment, restraint and observation.

It is important to distinguish the implications of the above heresies with what critics of antipsychiatric polemic have seen as the admiration or idealization of madness. Arieti (1979), whilst acknowledging the links between creative processes and schizophrenia, distinguishes the condition from adolescent day-dreaming, fantasy or cultural experimentation. Schizophrenia represents a more compelled and drastic aversion from conventional functioning than sexual ambivalence, punk nihilism or gothic angst:

'These (schizophrenic) states of loneliness are . . . at least as undesirable as broken bones. (To) romanticise madness . . . is a precarious assumption, especially if it is based on the idea that there is a mystical world to discover; some kind of extra-human reality beyond the social reality of . . . human consensus. We wish to recommend accepting mysteries, but not the mystification of them.' (Jenner *et al.* 1993, p. 16)

We need deeper understanding of the biological similarities between diagnosed schizophrenics and nondiagnosed, sublimated schizotypal people. Such knowledge could be combined with qualitative research into how successful integration is achieved, enabling more effective, less invasive help to be offered to those struggling with the debilitating impact of the condition. Recovered sufferers and politically active survivors offer similar opportunities (Chadwick 1997, Emmons *et al.* 1997).

Family life

The major tenets of orthodox beliefs about schizophrenia include the assertion that there is no evidence of family atmosphere causing initial disturbance or precipitating schizophrenic breakdown (Kuipers *et al.* 1997). This claim seems counter-intuitive when examined alongside other pathological conditions where parental behaviour is deemed to be a causative factor (see Weir & Douglas 1999). The influence of early relationships on adult depression has often been asserted. Examples include Brown & Harris's (1978) research into social causes of depression (Harris & Brown 1996); Bowlby's (Bowlby 1988) work on attachment and care-giving; the transactional-analysis concept of 'life-scripts' (Stewart & Joines 1987); Ryle's

(Ryle 1990) development of therapy based upon reconstructing the narrative of sufferers' life stories; Holmes (1993) clinical applications of attachment theory. These and many others seem to claim, without risking professional excommunication, that childhood experiences significantly affect the likelihood of grown-up misery (Weir & Douglas 1999). It seems relatively uncontroversial to attribute etiological significance for many personality disorders to traumatic childhood abuse, neglect or deprivation (Gallop 1999). Therapeutic systems for such disorders are often organized around long-term models of parataxic re-parenting (Linehan 1993, pp. 56–58, 225). British politicians frequently threaten to introduce parent-targeted initiatives to deal with disturbed or delinquent children. Advocating 'parenting classes' and fines reflects the widespread belief that deviant behaviour can be a consequence of parenting style, or lack of parenting skills. Bullies beget bullies. Christian families typically produce Christian offspring. Tories mould or create infant Tory-boys and girls. Or if not, their children's personalities are built in reactance to parental influence. Either way, common-sense suggests that our personality traits, belief systems, emotional resilience, social confidence, etc. are significantly affected by the interactive climate created within close relationships as we mature (Erikson 1968). Moreover, neuroscience increasingly informs us that children's brains are plastic. Neuronal maps, synaptic connections, etc. are formed and reformed within and in reaction to the social learning climate that prevails as we develop (Greenfield 1997, p. 122, Siegel 1999). However, orthodox psychiatry defines schizophrenia as an illness, not an aspect of personality. If the continuity hypothesis is rejected, and heretics concede that schizophrenia is an illness bolted on to an otherwise intact personality, then it becomes necessary to reject such neuro-developmental science. A genetic predisposition to schizophrenia would somehow dictate a high degree of protection for a pre-existing damaged personality, so that it could not be influenced for good or ill by prevailing interactive climates.

However, common-sense is a nonexistent sense, and has no place in the world of hard science. Consequently, the biological certainty of some professionals (Gournay 1996) leads them to implicitly suggest that if we are vulnerable to schizophrenia, our early experience of communication and the construing of others in no way stresses us sufficiently to induce active manifestation of our latent defect. One commentator expresses his puzzlement thus:

'The great heresy in psychiatry seems to be any suggestion that parents drive children mad... Why parents shouldn't be influential, from a position of dominance in the family, is a mystery.' (Newnes 1996, pp. 3–4)

As well as such rational evidence for disputing the 'no parental cause' claim, there is also sound contradictory research evidence. Rund (1994) reviews the evidence that parental communication deviance drastically affects the development of language, attention and social functioning in offspring, and supports the hypothesis. The long-term longitudinal project in Finland (Tienari *et al.* 1994) concludes that symptomatic illness behaviour in vulnerable children was only manifested when they were adopted into 'a disturbed family environment'. As Lehtonen (1994) states in his commentary on Tienari's report:

'The conclusion reached by Tienari and his group seems well founded: there are not one, but two factors necessary for a schizophrenic outcome in the adoptee – a genetic factor and a disturbance in psychological interaction between the child and his parents.' (Lehtonen 1994, p. 27)

This finding validates the autobiographical accounts of many sufferers (Chadwick 1997), as well as the anecdotal experiences of many mental health nurses. Proctor's (Proctor 1981, 1985, 1986) 'family constructs' hypothesis provides one cognitive-systemic explanation of how familial communication may induce schizophrenic symptoms in vulnerable individuals. Siegel (1999) describes the neurobiological impact of attachment experiences on the physical development of memory, emotion and representation. However painful or confusing for families or politically incorrect for many professionals, such evidence should not be ignored when devising therapeutic strategies or debating clinical issues (Schoenewolf 1996).

Don't blame me, I'm only human!

The above heresy is of course the kind of belief that family management proponents label 'family blaming' (Barrowclough & TARRIER 1992, Kuipers *et al.* 1997). 'Blaming' implies intent, as if people wilfully set out to do harm to their relatives. It would make no more sense to 'blame' families for their parenting style than it would to blame God. If one construes offspring as being moulded by their parents, who are therefore to 'blame' for their offsprings' behaviour, then those same parents need only plead their innocence by blaming their parents. The blaming process regresses into inaccessible history, back to Adam and Eve's irresponsibly thoughtless neglect of Cain and Abel's personalities.

The accusation of 'blaming' is a response to Laing's work (Laing & Esterson 1964), but trivializes the complexity of systems-based family work. Family therapists have long applied systemic theory to their work in effectively treating families of diagnosed schizophrenics (Hoffman 1981, Burbach 1996). One assumption made by systemic thera-

pists is that behaviours, thoughts and feelings are circularly induced, not linearly caused. Each person in a relationship participates in mutual sequences of cognitive-behavioural transactions, responsible for their own construing and consequent behaviour (Dallos 1991). Systems theory implicitly recognises that each person is responsible for cementing in place the repetitive sequences that characterize the kind of rigid chaos schizophrenic families often display. The complex theories informing systemic therapies of schizophrenic families are not adequately summarized by the simple pejorative description of 'family blaming'.

Supposedly comprehensive reviews of family interventions in schizophrenia (Lam 1991, Roth & Fonagy 1996) omit any reference to the many studies contributed by systemic family therapists. This could be explained by the orthodox assertion that such work is heavily theoretical and descriptive, but poorly researched. Nevertheless, the omission of such evidence as Jones's (Jones 1987) detailed descriptions of successful casework with schizophrenic families is to be regretted. Systemic approaches to schizophrenic families have been routinely and effectively applied in community mental health centres in the UK since 1980 (Proctor & Steves 1984; Proctor & Pieczora 1993). Neutral enquirers can at least offset any unintentional academic or clinical cleansing by reading Carr's (Carr 1991) review of empirical studies of systemic therapy and Burbach's (Burbach 1996) attempt to begin a rapprochement between systemic therapy and family management.

The 'family blaming' attribution is explored by Johnstone (1993, 1994, 1999) in critical explorations of the 'family management' orthodoxy. Proponents of the orthodoxy replied briefly (Leff & Vaughan 1994) in a style that seems unnecessarily perfunctory and dismissive. Johnstone raises several contentious issues, including the possibility that family management approaches reinforce a 'sick role', and may ironically 'blame' the schizophrenic patient for family difficulties. She claims that evidence is used selectively to convince sufferers and families of the need for medication (Johnstone 1993, p. 258–260) and argues that the same evidence could be used to support a different conclusion – that medication is often unnecessary. Harding & Zahniser (1994) agree, claiming that up to 50% of sufferers function well with no medication. Using systemic and psychotherapeutic interventions, Finland's API Project (Lehtinen *et al.* 1996) finds that only 22% of first-episode schizophrenics need medication (Reed 1999).

The Gene Genie

A fundamental tenet of the schizophrenia orthodoxy is that schizophrenia is primarily a genetic disorder. Sporadic excitement occurs whenever new loci are proposed for a

schizophrenia-gene and despite the improbability of a simple genetic mechanism for such complex and disparate conditions (Cloninger 1997), the search continues. Less zealous researchers seem more uncertain of the implications of familial inheritance findings, and counsel caution about over-interpretation of genetic research (Jenner 1993, Stirling 1997, Thomas 1997, Dawson 1998, DeLisi 1999). Most diagnosed people with schizophrenia do not have parents with schizophrenia, and between 50 and 75% of identical twins are not diagnosed after their sibling develops the disorder. To explain these anomalies, orthodox protagonists have suggested there are two forms of the disorder: genetic and nongenetic schizophrenia (Leff 1996). This places orthodox clinicians in the convenient if logically uncomfortable position of upholding the claim that the illness is genetic, except where there is no evidence that it is, in which case it must be a different illness.

Clarke (1997, 1999) warns against the limiting and potentially brutalising effects of mindlessly incorporating genetic and biological determinist theories into nursing practice. Such thinking is too narrow to provide nurses with meaningful bases for therapeutic relationships with people diagnosed with schizophrenia. DeLisi (1999) expresses disappointed puzzlement that little progress has been made in identifying genetic causes with any certainty.

'The field of psychiatric genetics...is in crisis. It is unclear... why no progress has been made towards finding genes or even one gene for this disorder (schizophrenia).' (DeLisi 1999, p. 36).

Genes enable the transmission of potential (Jones 1996), and in the case of schizophrenia, we all inherit an incredibly complex capacity to develop language and elaborate forms of communication (Frith 1992, Crow 1994). The intricate neuronal networks needed to realize this potential develop throughout childhood (Greenfield 1997), in interaction with the emotional climate and cognitive culture we are exposed to as we mature (Tienari 1994, Lehtonen 1994, Siegel 1999). Thus, we are vulnerable in three ways. We may be born with genetic instructions that prejudice our chances of building appropriate cerebral architecture. Or the blueprint may be misinterpreted during the elaborate process of building operational minds from biochemical supplies. Finally, the human adults upon whom we depend for cognitive and affective clarity may be providing poor feedback with which to monitor the integrity of our evolving brain-mind infrastructure.

Further open-minded research will help clarify all three possibilities, but psychiatric orthodoxy has been accused of aggressively rejecting heretical ideas (Johnstone 1997b). Suggesting alternative possibilities to orthodox psychiatric tenets raises strong feelings, as though one was courting academic scorn or clinical censure (see Leff & Vaughan

1994, Cannon *et al.* 1999). The schizophrenia orthodoxy has undoubted scientific and heuristic integrity, as do the alternative, heretical ideas discussed, yet the latter receive reactions ranging from short shrift to contempt.

Economic and political realities are important. Community care requires a caring workforce of blame-free families. The public's tabloid-fed anxieties need assuaging. Voracious pharmaceutical research budgets demand commercial success. A simple, coherent doctrine based upon familiar scientific principles like biological illness and behavioural control is much more politically attractive than continued uncertainty. Measured outcomes and tight definitions are comfortable, cost-able commodities for a modern audited health service. But should pragmatic expediency restrict the quest for understanding? The risks of discouraging intelligent questioning of orthodox assertions about schizophrenia include blurring the boundaries between truth and pragmatism; damaging nursing relationships by stigmatizing or alienating sufferers, and creating a workforce of distanced, potentially oppressive nurses.

References

- Andreasen N. (1985) *The Broken Brain*. Harper & Row, New York.
- Arieti S. (1974) *The Interpretation of Schizophrenia*. Basic Books, New York.
- Arieti S. (1976) *Creativity: the Magic Synthesis*. Basic Books, New York.
- Arieti S. (1979) *Understanding and Helping the Schizophrenic*. Pelican, Harmondsworth.
- Bannister D. & Fransella F. (1971) *Inquiring Man: the Theory of Personal Constructs*. Penguin, Harmondsworth.
- Barrowclough C. & Tarrrier N. (1992) *Families of Schizophrenic Patients*. Chapman & Hall, London.
- Bateson G. (1972) *Steps to an Ecology of Mind*. Paladin, London.
- Bentall R.P. (1992) The classification of schizophrenia. In: *Schizophrenia: an Overview and Practical Handbook* (ed. Kavanagh, D.J.), pp. 23–44. Chapman & Hall, London.
- Bentall R.P. (1993) Deconstructing the concept of 'schizophrenia'. *Journal of Mental Health* 2, 223–228.
- Bentall R.P. (1990) *Reconstructing Schizophrenia*. Routledge & Kegan Paul, London.
- Birchwood M. (1999) Psychological and social treatments: course and outcome. *Current Opinion in Psychiatry* 12, 61–66.
- Bowlby J. (1988) *A Secure Base: Clinical Applications of Attachment Theory*. Routledge, London.
- Boyle M. (1990) *Schizophrenia – a Scientific Delusion?* Routledge, London.
- Boyle M. (1996) Schizophrenia: the fallacy of diagnosis. *Changes* 14, 5–13.
- Boyle M. (1997) Minimum contents. *Openmind* 87, 12–13.
- Brown G.W. & Harris T. (1978) *The Social Origins of Depression: a Study of Psychiatric Disorder in Women*. Tavistock, London.
- Buckley P., Bird J. & Harrison G. (1995) *Examination Notes in Psychiatry*, pp 26–27. Butterworth-Heinemann, London.
- Burbach F. (1996) Family-based interventions in psychosis: an overview of, and comparison between, family therapy and family management approaches. *Journal of Mental Health* 5, 111–134.
- Cannon B., Coulter E., Gamble C. *et al.* (1999) Personality Bashing, Letter. *Mental Health Care* 2, 319.
- Cape G., Antebi D., Standon P. & Glazebrook C. (1994) Schizophrenia: the views of a sample of psychiatrists. *Journal of Mental Health* 3, 105–113.
- Carr A. (1991) Milan systemic family therapy: a review of ten empirical investigations. *Journal of Family Therapy* 13, 237–263.
- Chadwick P. (1997) *Schizophrenia: the Positive Perspective*. Routledge, London.
- Clare A. (1976) *Psychiatry in Dissent*. Tavistock, London.
- Claridge G. (1989) Schizophrenia and Human Individuality. In: *Mindwaves* (eds. Blakemore C. & Greenfield S.), pp. 29–42. Basil Blackwell, Oxford.
- Clarke L. (1997) Schizophrenic patients fare badly in the company of scientists. *Journal of Advanced Nursing* 25, 429–430.
- Clarke L. (1999) Nursing in search of a science: the rise and rise of the new nurse brutalism. *Mental Health Care* 2, 270–272.
- Cloninger C.R. (1997) Multi-locus genetics of schizophrenia. *Current Opinion in Psychiatry* 10, 5–10.
- Coffey J. (1998) Schizophrenia: a review of current research and thinking. *Journal of Clinical Nursing* 7, 489–498.
- Cooper D. (1967) *Psychiatry and Anti-Psychiatry*. Tavistock, London.
- Crow T. (1994) Aetiology of schizophrenia. *Current Opinion in Psychiatry* 7, 39–42.
- CSAG (Clinical Standards Advisory Group) (1995) *Schizophrenia*. Vols 1–2. HMSO, London.
- Dallos R. (1991) *Family Belief Systems, Therapy and Change*. Open University Press, Milton Keynes.
- Davey B. (1996) Upbringing and psychosis: an afterword. *Changes* 14, 50–61.
- Dawson P.J. (1997) A reply to Kevin Gournay's 'Schizophrenia: a review of the contemporary literature and implications for mental health nursing theory, practice and education'. *Journal of Psychiatric and Mental Health Nursing* 4, 1–7.
- Dawson P.J. (1998) Schizophrenia and genetics: a review and critique for the psychiatric nurse. *Journal of Psychiatric and Mental Health Nursing* 5, 299–307.
- DeLisi L.E. (1999) A critical overview of recent investigations into the genetics of schizophrenia. *Current Opinion in Psychiatry* 12, 29–39.
- DoH (1998) *A First Class Service: Quality in the New NHS*. Department of Health, London.
- Emmons S., Geiser C., Kaplan K.J. & Harrow M. (1997) *Accelerated Development*. Taylor & Francis, Washington, D.C.
- Erikson E. (1968) *Identity: Youth and Crisis*. Faber, London.
- Falloon I. & Fadden G. (1993) *Integrated Mental Health Care*. Cambridge University Press, Cambridge, UK.
- Falloon I., McGill C. & Boyd J. (1984) *Family Care of Schizophrenia*. Guilford Press, New York.
- Fowler D., Garety P. & Kuipers E. (1995) *Cognitive Behaviour Therapy for Psychosis: Theory and Practice*. John Wiley & Sons, Chichester.

- Frangou S. & Murray R. (1996) *Schizophrenia*. Martin Dunitz, London.
- Frith C. (1992) *The Cognitive Neuropsychology of Schizophrenia*. Lawrence Erlbaum, Hove.
- Gallop R. (1999) Personality Disorder: Finding a Way. In: *Advanced Practice in Mental Health Nursing* (eds. Clinton M. & Nelson S.), pp. 194–218. Blackwell Science, Oxford.
- Garety P. & Hemsley D.R. (1997) *Delusions: Investigations Into the Psychology of Delusional Reasoning*. Psychology Press, Hove.
- Gottesman I.I. (1994) Schizophrenia epigenesis: past, present and future. *Acta Psychiatrica Scandinavica* 90 (Suppl. 384), 26–33.
- Gournay K. (1995) New facts on schizophrenia. *Nursing Times* 91, 32–33.
- Gournay K. (1996) Schizophrenia: a review of the contemporary literature and implications for mental health nursing theory, practice and education. *Journal of Psychiatric and Mental Health Nursing* 3, 7–12.
- Greenfield S. (1997) *The Human Brain*. Basic Books, New York.
- Haddock G. & Slade P.D. (1996) *Cognitive-Behavioural Interventions with Psychotic Disorders*. Routledge, London.
- Harding C.M. & Zahniser J.H. (1994) Empirical Correction of Seven Myths about Schizophrenia. *Acta Psychiatrica Scandinavica* 90, 140–146.
- Harris T.O. & Brown G.W. (1996) Social causes of depression. *Current Opinion in Psychiatry* 9, 3–10.
- Hoffman L. (1981) *Foundations of Family Therapy*. Basic Books, New York.
- Holmes J. (1993) *John Bowlby and Attachment Theory*. Routledge, London.
- Jablensky A. (1993) The epidemiology of schizophrenia. *Current Opinion in Psychiatry* 6, 43–52.
- Jenner F.A., Monteiro A.C.D., Zagalo-Cardoso J.A. & Cunha-Oliviera J.A. (1993) *Schizophrenia: a Disease or Some Ways of Being Human*. Sheffield Academic Press, Sheffield.
- Johnstone L. (1989) *Users and Abusers of Psychiatry*. Routledge, London.
- Johnstone L. (1993) Family management in 'schizophrenia': Its assumptions and contradictions. *Journal of Mental Health* 2, 255–269.
- Johnstone L. (1994) Reply to Leff and Vaughan. *Journal of Mental Health* 3, 117–118.
- Johnstone L. (1997a) Talking Taboos. *Openmind* 87, 8–10.
- Johnstone L. (1997b) Psychiatry: are we allowed to disagree? In: *Mental Health and Distress: Perspectives and Practice*. (OU Course K257) Offprints Book. Open University Press, Milton Keynes.
- Johnstone L. (1999) Do families cause 'schizophrenia'? Revisiting a taboo subject. *Changes* 17, 77–90.
- Jones E. (1987) Brief systemic work in psychiatric settings where a family member has been diagnosed as schizophrenic. *Journal of Family Therapy* 9, 3–25.
- Jones S. (1996) *In the Blood: God, Genes and Destiny*. Harper-Collins, London.
- Kingdon D.G. & Turkington D. (1994) *Cognitive-Behavioural Therapy of Schizophrenia*. Psychology Press, Hove.
- Kissling W. (1994) Compliance, quality assurance and standards for relapse prevention in schizophrenia. *Acta Psychiatrica Scandinavica* 89 (Suppl. 382), 16–24.
- Kringlen E. (1994) Is the concept of schizophrenia useful from an aetiological point of view? *Acta Psychiatrica Scandinavica* 90 (Suppl. 384), 17–25.
- Kuipers L., Leff J. & Lam D. (1997) *Family Work for Schizophrenia: a Practical Guide*. Gaskell/Royal College of Psychiatrists, London.
- Laing R.D. (1960) *The Divided Self*. Tavistock, London.
- Laing R.D. (1961) *The Self and Others*. Tavistock, London.
- Laing R.D. & Esterson A. (1964) *Sanity, Madness and the Family*. Tavistock, London.
- Lam D. (1991) Psychosocial family interventions in schizophrenia: a review of empirical studies. *Psychological Medicine* 21, 423–441.
- Leff J. (1996) Schizophrenia: Aetiology, Prognosis and Course. In: *The Primary Care of Schizophrenia* (eds. Jenkins R. & Field V.), pp. 5–16. HMSO, London.
- Leff J. & Vaughan C. (1994) Critics of family management in schizophrenia: Their assumptions and contradictions. *Journal of Mental Health* 3, 115–116.
- Lehtinen V., Aaltonen J., Koffert T., Rakkolainen V., Syvalahti S. & Vuorio K. (1996) Integrated treatment model for first-contact patients with a schizophrenia-type psychosis: the Finnish API Project. *Nordic Journal of Psychiatry* 50, 281–287.
- Lehtonen J. (1994) From Dualism to Psychobiological Interaction: a Comment on the Study by Tienari and his Co-workers. *British Journal of Psychiatry* 164 (Suppl.23), 27–28.
- Liddle P.F. (1987) The symptoms of chronic schizophrenia. *British Journal of Psychiatry* 151, 145–151.
- Liddle P.F., Friston K.J., Frith C.D. *et al.* (1992) Patterns of cerebral blood flow in schizophrenia. *British Journal of Psychiatry* 160, 179–186.
- Linehan M. (1993) *Cognitive-Behavioural Treatment of Borderline Personality Disorder*. Guilford Press, New York.
- Macpherson R., Jerrom B. & Hughes A. (1997) Drug refusal among schizophrenic patients treated in the community. *Journal of Mental Health* 6, 141–147.
- Maher B.A. (1970) *Principles of Psychopathology*, pp. 394–441. McGraw-Hill, New York.
- McCrone S.H. (1996) The impact of the evolution of biological psychiatry on psychiatric nursing. *Journal of Psychosocial Nursing and Mental Health Services* 34, 38–46.
- McKenna P.J. (1997) *Schizophrenia and Related Syndromes*, p. 196. Psychology Press, Hove.
- Mortier S. (1997) Where words fail: a meeting place. In: *Art, Psychotherapy and Psychosis* (eds Killick, K. & Schaverian, J.) Routledge, London.
- Murray R. & McGuffin P. (1993) Genetic aspects of psychiatric disorders. In: *Companion to Psychiatric Studies* (eds. Kendall, R.E. & Zealley, A.K.), pp. 227–261. Churchill Livingstone, Edinburgh.
- Nanko S. & Moridairo J. (1993) Reproductive rates in schizophrenia. *Acta Psychiatrica Scandinavica* 87, 400–404.
- Nelson H. (1997) *Cognitive Behavioural Therapy with Schizophrenia*. Stanley Thornes, Cheltenham.
- Newnes C. (1996) Editorial. *Changes* 14, 3–4.
- Peters E., Day S., McKenna J. & Orbach G. (1999) Delusional ideation in religious and psychotic populations. *British Journal of Clinical Psychology* 38, 83–96.
- Playle J.F. (1995) Humanism and positivism in nursing: Contradictions conflicts. *Journal of Advanced Nursing* 22, 979–984.

- Proctor H.G. (1981) Family construct psychology. An Approach to Understanding and Treating Families. In: *Developments in Family Therapy* (Ed. Walrond-Skinner, S.) Routledge, London.
- Proctor H. & Stephens T. (1984) Using family therapy in the Day Hospital. In: *Using Family Therapy* (Eds. Treacher, A. & Carpenter, J.). Basil Blackwell, Oxford.
- Proctor H.G. (1985) A construct approach to family therapy and systems intervention. In: *Personal Construct Theory and Mental Health* (Ed. Button, E.). Croom Helm, Beckenham.
- Proctor H.G. (1986) Change in the family construct system: therapy of a mute and withdrawn schizophrenic patient. In: *Casebook in Personal Construct Psychology* (Ed. Neimeyer, R. & G.). Springer, New York.
- Proctor H. & Pieczora R. (1993) A Family Oriented Community Mental Health Centre. In: *Using Family Therapy in the 90s* (eds. Carpenter, J. & Treacher, A.), pp. 131–144. Basil Blackwell, Oxford.
- Repper J. (1995) *The NT Guide to Schizophrenia*. Nursing Times Publishers, London.
- Richter D. (1984) *Research in Mental Illness*. Heinemann, London.
- Rogers P. (1999) 'Persecution Complex'. Letter. *Mental Health Care* 2, 319.
- Romme M. & Esoher S. (1993) *Accepting voices*. MIND Books, London.
- Roth A. & Fonagy P. (1996) *What Works for Whom?* Guilford Press, New York.
- Rund B.R. (1994) Cognitive dysfunctions and psychosocial treatment of schizophrenics: research of the past and perspectives on the future. *Acta Psychiatrica Scandinavica* 90 (Suppl. 384), 9–16.
- Ryle A. (1990) *Cognitive Analytic Therapy: Active Participation in Change*. John Wiley, Chichester.
- Scheff T. (1966) *Being Mentally Ill*. Aldine, New York.
- Schoenewolf G. (1996) Schizophrenia in a dysfunctional family. *Changes* 14, 31–42.
- Siegel D.J. (1999) *The Developing Mind: Toward a Neurobiology of Interpersonal Experience*. Guilford Press, New York.
- SIGN (1998) *Psychosocial Interventions in the Management of Schizophrenia*. Scottish Intercollegiate Guidelines Network. Royal College of Physicians, Edinburgh.
- Stewart I. & Joines V. (1987) *T.A. Today*. Lifespan Publishing, Nottingham.
- Stirling J. (1997) Progress in research into the neurobiological bases of schizophrenia: a review and commentary. *Nursing Times Research* 2, 434–440.
- Szasz T. (1962) *The Myth of Mental Illness*. Secker & Warburg, London.
- Thomas P. (1997) *The Dialectics of Schizophrenia*. Free Association Books, London.
- Tienari P., Wynne L.C., Moring J. *et al.* (1994) The Finnish Adoptive Study of Schizophrenia: Implications for Family Research. *British Journal of Psychiatry* 164 (Suppl.23), 20–26.
- Van Os J., Galdos P., Lewis G., Bourgeois M. & Mann A. (1993) Schizophrenia sans frontières: concepts of schizophrenia among French and British psychiatrists. *British Medical Journal* 307, 489–492.
- Vogeley K. & Falkai P. (1999) Brain imaging in schizophrenia. *Current Opinion in Psychiatry* 12, 41–46.
- Warner R. (1985) *Recovery from Schizophrenia: Psychiatry and Political Economy*. Routledge, London.
- Weir A. & Douglas A. (1999) *Child Protection and Adult Mental Health: Conflict of Interest?* Butterworth-Heinemann, Oxford.
- Wing J.K., Cooper J.E. & Sartorius N. (1974) *The Description and Classification of Psychiatric Symptoms*. Cambridge University Press, London.