10. Early Psychosis: Preventive Medicine, Scientific Assault on Mystical Tendencies, or an Extension of Social Control?

Introduction

In recent years psychiatric researchers have extended the definition of schizophrenia to include a pre-psychotic phase. Detection and intervention programmes have been implemented and neuroleptic medication is used as prophylactic treatment in the belief that it can prevent the development of psychosis in people who are thought to be at-risk.[1] The pre-psychotic signs of schizophrenia are usually referred to as 'early psychosis'[2] or as 'prodromal' symptoms of schizophrenia.[3] Australia has become a particularly active site for this type of research and a National Early Psychosis Project has been launched as "a collaborative endeavour between the Commonwealth, State and Territory governments of Australia to develop and promote a national model of best practice for early intervention in psychosis".[4]

Promoters of the concept see pre-psychosis detection and intervention as a form of preventive medicine. Their basic argument is that if the incidence of schizophrenia can be reduced by early identification and treatment, as has been the case with prevention programmes for other diseases, then numerous community benefits will follow in the form of cost savings and in the avoidance of personal trauma and family disruption.[5]

However, people who are already sceptical about bio-medical approaches to schizophrenia are likely to interpret drug-based preventive medicine campaigns differently. Apart from the risks involved in the prophylactic use of neuroleptic drugs, so-called preventive medicine might be variously seen as an unnecessary expansion of social control, a threat to human diversity through the enforcement of hyper-normality, a violation of human rights, a campaign against mystical tendencies in young people, and a marketing ploy for the new generation of atypical neuroleptic drugs.

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[1] Alison R. Yung, Patrick D. McGorry, Colleen A. McFarlane, Henry J. Jackson, George C. Patton and Arun Rakkar, 'Monitoring and Care of Young People at Incipient Risk of Psychosis', Schizophrenia Bulletin, Vol. 22, No. 2, 1996, p. 300.

[2] Early psychosis is currently used in three different ways. It describes psychosis in young people, first-episode psychosis, and a supposed pre-psychotic phase of schizophrenia.

[3] Tor K. Larsen and Stein Opjordsmoen, 'Early identification and treatment of schizophrenia: conceptual and ethical considerations', Psychiatry: Interpersonal and Biological Processes, Vol. 59, No. 4, 1996, pp. 371-381.

[4] National Early Psychosis Project, Australian Commonwealth Government, 1999, Available URL, http://yarra.vicnet.net.au/-eppic/nepp.html

[5] Chris Jackson and Max Birchwood, 'Early intervention in psychosis: Opportunities for secondary prevention', British Journal of Clinical Psychology, Vol. 35, No. 4, November, 1996, pp. 487-502.

Early psychosis identification and treatment is a recent extension of the medical model for schizophrenia and the mystical and myth-of-mental-illness models have not yet been analysed in relation to it. It is proposed in this chapter to see whether a focus on the supposed prodromal symptoms can bring the three aetiological models for schizophrenia into clearer focus.

Early Psychosis as Preventive Medicine

Medical historians have identified an epidemiological transition which occurred around 1940 involving a shift of medical emphasis from the control of infectious diseases to the control of chronic diseases like cancer and heart disease.[6] The medical emphasis that has been applied to chronic diseases in the second part of the twentieth century has largely involved various programmes of health promotion and disease prevention. Where lifestyle has been found to be a major contributor to the development of chronic diseases publicity campaigns have been put into place to guide people away from self-damaging habits and behaviours, to adopt healthy diets, and to take precautions and avoidance measures.[7]

Similarly, where early or prodromal signs and symptoms can be identified, and where early treatment can effectively prevent or cure, or make the management of a chronic disease easier, screening programmes have frequently been inaugurated and publicity campaigns launched to alert the public about dangerous signs.[8] An on-going campaign against skin cancer in Australia, which alerts people to the dangers of exposure to ultraviolet light and encourages them to seek early treatment for suspicious skin lesions, is a typical example of this kind of preventive medicine.

Various preventive campaigns against mental diseases also fit into this pattern:

> Mental health prevention has three primary aims: (1) decreasing the occurrence of new cases, (2) delaying onset, and (3) decreasing the duration of early symptoms or halting the progression of severity. The first aim is known as primary prevention or reducing the incidence of disorder; the second aim, secondary prevention or reducing the prevalence of disorder; the third aim, tertiary prevention or reducing the morbidity of disorder.[9]

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[6] Kenneth R. McLeroy and Carolyn E. Crump, 'Health promotion and disease prevention: a historical perspective', Generations, Vol. 18, No. 1, 1994, pp. 9-18.

[7] Mary T. Shannon, 'Health promotion and illness prevention: a biopsychosocial perspective', Health and Social Work, Vol. 14, No. 1, February, 1989, pp. 32-41.

[8] F. Douglas Scutchfield, Karma T. Hartman, 'Physicians and preventive medicine', JAMA, The Journal of the American Medical Association, Vol. 273, No. 14, April 12, 1995, pp. 1150-1152.

[9] Thomas H. McGlashan, 'Early Detection and Intervention in Schizophrenia: Research', Schizophrenia Bulletin, Vol. 22, No. 2, 1996, p. 328.

Whereas primary prevention often only involves strategies to avoid the risk of disease, secondary and tertiary prevention enlist both preventive strategies and active treatment.

In the United States there is a long history of strategies to prevent mental illness which go back to the turn of the century.[10] A recent large scale project was initiated in 1992 when the Senate Appropriations Committee of the US Congress "mandated the National Institute of Mental Health to enter into an agreement with the Institute of Medicine (IOM) to prepare an integrated report of current research with policy-oriented and detailed long-term recommendations for a prevention research agenda."[11]

The 1994 report that emerged from this investigation divided mental health intervention into a spectrum of stages.[12] The over-arching stages were Prevention, Treatment and Maintenance. Prevention was divided into three sub-groups: Universal, which is aimed at the general public or entire populations; Selective, which involves individuals or groups with above average risk factors; and Indicated, which targets "high risk individuals who are identified as having minimal but detectable signs or symptoms foreshadowing mental illness".[13]

The investigators reviewed the various theories about the aetiology of schizophrenia and the evidence for certain risk factors and indicators. They concluded that there was not sufficient aetiological evidence at this stage to warrant preventive intervention for schizophrenia at either the Universal or Selective stages. However, they did support

> indicated preventive interventions targeted at individuals manifesting precursor signs and symptoms who have not yet met full criteria for diagnosis. The identification of individuals at this early stage, coupled with the introduction of pharmacological and psychosocial interventions, may prevent the development of full-blown disorder.[14]

For the purpose of developing indicated prevention strategies for schizophrenia, researchers have divided the condition into three stages of development: premorbid, prodromal and onset.[15] Early psychosis detection and intervention programmes generally aim at reducing the duration of untreated psychosis (DUP). The DUP is the period preceding first treatment for schizophrenia during which symptoms and signs of an impending psychological crisis are present. It is argued by

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[10] Patricia J. Mrazek and Robert J. Haggerty, Reducing Risks For Mental Disorders: Frontiers For Preventive Intervention Research, National Academy Press, Washington, 1994, pp. 8-11.
[11] Ibid., p. xi.
[12] Ibid., p. 23.
[13] Ibid., p. 25.
[14] Ibid., p. 154.
[15] Tor K. Larsen, Thomas McGlashan, and Lars Conrad Moe, 'First-Episode Schizophrenia: 1. Early Course Parameters', Schizophrenia Bulletin, Vol. 22, No. 2, 1996, p. 241.

bio-medical psychiatrists that the DUP for most people who develop psychosis is much longer than it should be and can often be measured in years.[16] There are on-going debates about the relevance of the length of the DUP to the intensity of the subsequent psychotic experience, to the response of the patient to medication, and to the course of post-psychotic morbidity.[17]

It has been claimed that "the cost of treatment for patients with a DUP greater than 6 months is twice the cost of those with a DUP less than 6 months".[18] Some psychiatric researchers argue that brain damage is occurring during the DUP and that the longer it continues the less chance a person has of ultimate recovery: "most of the neurobiological damage is already accomplished by the time it is possible to make a valid DSM-IV diagnosis"[19] and "applying existing schizophrenia treatment as soon as possible in the course of the disorder may slow or stop deterioration".[20] But apart from the equivocal evidence of unconfirmed pilot studies (see below)[21] there is nothing much of substance to support these bio-medical contentions.

Nevertheless, despite the weak theoretical base, claims have been forcefully made that when a programme of early detection is put into place the incidence of full-blown psychosis in the community can be reduced. This is supposedly achieved by applying combined psycho-social and neuroleptic treatment to people deemed to be at risk and thereby diverting the progression of their developing psychological crisis.

Early Psychosis Programmes

Falloon has reported on a pilot project which commenced in 1984 in Buckingham, UK.[22] The Buckingham Project enlisted the participation of 18 family doctors to screen a population of 35,000 people over a four year period. The screening process involved ten questions which the doctors were required to ask their patients and an eight point checklist of prodromal signs for schizophrenia, which the doctors would look for. The eight prodromal signs were derived from a list of prodromal

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16 Ibid., pp. 243-244.

17 John Cocks, 'The use of very-low-dose antipsychotic medication in the treatment of first-episode psychosis', Early Psychosis News, No. 9, June 1998, p. 5.

18 Thomas H. McGlashan and Jan Olav Johannessen, 'Early Detection and Intervention With Schizophrenia: Rationale', Schizophrenia Bulletin, Vol. 22, No. 2, 1996, p. 212.

19 Ibid., p. 209.

20 Ibid., p. 201.

21 Jay W. Pettegrew, Matcheri S. Keshavan, Kanagasabai Panchalingam, Sandra Strychor, David B. Kaplan, Marjorie G. Tretta, and Maureen Allen, 'Alterations in Brain High-Energy Phosphate and Membrane Phospholid Metabolism in First-Episode, Drug-Naive Schizophrenics: A Pilot Study of the Dorsal Prefrontal Cortex by In Vivo Phosphorus 31 Nuclear Magnetic Resonance Spectroscopy', Archives of General Psychiatry, Vol. 48, June 1991, pp. 563-568.

22 Ian R. H. Falloon, Robert R. Kyd, John H. Coverdale and Tannis M. Laidlaw, 'Early Detection and Intervention for Initial Episodes of Schizophrenia', Schizophrenia Bulletin, Vol. 22, No. 2, 1996, pp. 271-282.

indicators outlined in DSM-III. The doctors were assured that any person they referred would receive specialised psychiatric assessment without delay.

The screening questionnaire and prodromal checklist were as follows:

10-question screening

1. How have you been sleeping in the past week? Any difficulties getting to sleep? Wake early?
2. Have you lost your appetite recently? Weight loss of two or more kilograms?
3. Have you experienced loss of energy or interests recently?
4. Have you been worrying a lot about everyday problems?
5. Have you had difficulty concentrating on reading or watching television? Have you been more forgetful than usual?
6. How do you see the future? Do you feel that life is not worth living Have you ever felt you would like to end it all?
7. Have you any odd habits, like checking or cleaning more than other people?
8. Do you ever have attacks of palpitations, sweating, shaking, or dizziness accompanied by feelings of intense fear?
9. Has anybody commented that your speech has become odd or difficult to understand?
10. Have you ever had the experience of hearing people's voices speaking when nobody seems to be around?

Prodromal signs checklist

Onset of one of the following without explanation:

* Marked peculiar behaviour
* Inappropriate, or loss of, expression of feelings
* Speech that is difficult to follow
* Marked lack of speech and thoughts
* Marked preoccupation with odd ideas
* Ideas of reference — things have special meanings
* Persistent feelings of unreality
* Changes in the way things appear, sound, or smell[23]

People who failed the screening test with the family doctor were referred on for a more formal psychiatric assessment. This involved completing another questionnaire in the company of a

[23] Ibid., p. 274.

relative or household member. This psychiatric assessment was designed to "identify prodromal symptoms, particularly those of a subtle nature, such as interpersonal withdrawal."[24]

> When a person was suspected of experiencing an early phase of schizophrenia, an integrated crisis management program was initiated without delay. Each component of this program, which included education, stress management, and neuroleptic medication, was tailored to individual needs within a clinical management protocol.[25]

It was claimed for the Buckingham Project that the incidence of schizophrenia in the 35,000 person catchment area was lowered during the four years of the pilot scheme from an expected annual rate under normal conditions of 7.4 new cases of schizophrenia per 100,000 population to an annual rate of 0.75 new cases per 100,000 population.[26] But the researchers admit that "during the 4-year period, 15 other cases with symptom patterns suggesting an early phase of a florid schizophrenic episode were observed; however, these cases failed to reach the diagnostic thresholds for functional psychotic disorders".[27] Since all of these 15 people were treated for schizophrenia it seems likely that the interpretation of the "diagnostic threshold" was intended to be flexible enough to provide statistical evidence to support claims of a successful pilot project.

However, this arbitrariness in the definition of psychosis was overlooked and the apparent success of the Buckingham Project was well received by schizophrenia researchers in various parts of the world. A considerable literature is building as early psychosis projects are commenced in a number of countries. Falloon has since moved to Auckland, New Zealand, where he has another early psychosis project operating.[28] In 1995 a symposium was organised in Norway to bring together early psychosis researchers from the United States, Australia, New Zealand and Scandinavia. The symposium stimulated numerous research papers and the following year an edition of the Schizophrenia Bulletin[29] was devoted to papers on the subject.

Some of the early psychosis research projects currently operating in various parts of the world include: the Clarke Institute of Psychiatry in Toronto, Canada; Hillside Hospital in Glen Oaks, New York; the Schizophrenia Research Program at the London Health Sciences Center in London, Ontario, Canada; Nova Scotia Hospital in Dartmouth, Nova Scotia, Canada; Rogaland Psychiatric Services in Stavangar, Norway; the National Early Psychosis Project, University of Melbourne, Royal Park Hospital Department of Psychiatry, Parkville, Victoria, Australia; Mental Health

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[24] Ibid., p. 276.
[25] Ibid., p. 277.
[26] Ibid., p. 278.
[27] Ibid., p. 279.
[28] Thomas H. McGlashan, 'Early Detection and Intervention in Schizophrenia: Editor's Introduction', Schizophrenia Bulletin, Vol. 22, No. 2, 1996, p. 198.
[29] See, Schizophrenia Bulletin, Vol. 22, No. 2, 1996.

Clinical Research Centre, University of North Carolina, NC Neurosciences Hospital, Chapel Hill, North Carolina; University of Pittsburgh Medical Center/Western Psychiatric Institute and Clinic, Psychosis Research Program, Pittsburgh, Pennsylvania; West Birmingham Mental Health Services, The Archer Centre, All Saints Hospital, Birmingham, UK.[30]

Developing a consensus about vulnerability markers for schizophrenia is one of the first priorities: "so many markers have emerged that it seems reasonable to begin thinking about using them in 'normal' populations to identify groups that are at heightened risk for psychoses".[31] It is argued that current treatment for schizophrenia is only applied palliatively but that if an 'at-risk' population could be identified then vulnerability could be treated directly. It is further argued that whereas the incidence of schizophrenia in the general population is about 1-2% it would be much higher in a hypothetical population of people who all carried the vulnerability markers.[32] The assembly of such a hypothetical group would therefore make surveillance of potential schizophrenics much easier.[33]

Researchers believe that the use of vulnerability markers as an initial means of screening the population would reduce the incidence of false positives.[34] But a comprehensive list of vulnerability markers compiled for initial discussion involves many controversial aetiological and diagnostic hypotheses and it is unlikely to win the consensus support necessary for this type of screening to begin.

Vulnerability markers

Clinical

Cluster A personality disorders

Schizotypy in subjects, families

Psychosis proneness

Behavioural

Early neurointegrative deficits in temperament, arousal, development (pandysmaturation)

Premorbid behavioural problems: perceptual-cognitive, emotional, neuromotor, social, scholastic, functional patterns

Environmental

Perinatal factors: winter births, influenza, starvation, RH incompatibility, pregnancy and birth complications

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[30] Neuropsychiatry Branch, National Institute of Mental Health, accessed July 1998, Available URL, http://silk.nih.gov/silk/NPB/treat.html

[31] McGlashan and Johannessen, op.cit., pp. 204-205.

[32] Ibid., p. 205.

[33] Steven Adlard, 'Early Warning: The Early Detection of Psychosis', Early Psychosis News, No. 7, September 1997, pp. 1-2.

[34] McGlashan and Johannessen, op.cit., p. 205.

Psychosocial stress: low socioeconomic status, unstable rearing environment, negative affective climate
Anatomy/neuroanatomy

Minor physical anomalies
Fluctuating anatomical asymmetries
Structural brain abnormalities

Chemistry
HVA in plasma and CSF of SPD
MAO in platelets of SPD

Motor processes
Smooth-pursuit eye movements
Visual scanning/fixation
Grip-induced muscle tension

Perceptual processes
Arousal: psychophysiology
Sustained attention: Continuous Performance Task
Selective attention: Span of Apprehension Task
Discrimination: sensory saltation
Processing: cognitive inhibition, sensory motor gating, startle, prepulse inhibition, backward masking, negative priming, event-related potentials, mismatch negativity, P300 latency
Contextual set: semantic priming, Stroop Test
Hemispheric integration/asymmetry: dichotic listening, covert visual attention
Perceptual-motor speed

Neuropsychology
Intelligence
Abstraction
Mental control/encoding
Verbal, spatial, story memory
Language
Dyslexia [35]

Apart from the vulnerability markers which are listed above under the headings of "Clinical" and "Behavioural", and perhaps some under the heading of "Neuropsychology", most of the others on this list are highly controversial, even within the confines of the medical model. They represent a variety of observations that have been made about some schizophrenics after diagnosis. But mostly these signs are not thought to be reliable enough for use as diagnostic indicators. The authors of this

[35] Ibid., pp. 204-205.

list do not mount any discussion about how many, and in what combinations, these indicators would be useful for identifying at-risk individuals in a screening programme and it is unlikely that the full inventory of vulnerability markers will be adopted by early psychosis researchers in the near future.

In the meantime it is usually proposed to rely mostly on clinical and behavioural markers. In follow-up and follow-back studies it has been found that "social dysfunction and behavioural deviance reported by teachers are reliable predictors of later schizophrenia".[36] School reports have indicated that in childhood and adolescence, before the development of psychosis, schizophrenics tend to be shy, passive and withdrawn, with few friends and with low academic grades.[37] But it is generally believed that the predictive indicators are too uncertain in childhood for diagnosis and it is only with the onset of adolescence that it becomes possible to more positively identify them: "Deviant behaviours tend to become more prominent in adolescence, a time of life that may present more socially challenging situations. Sex differences in social adjustment have also been noted with males showing more antisocial behaviours and females showing more passivity and withdrawal".[38]

It has been proposed that schizophrenic-type people can be identified by a combination of a psychiatric interview and reference to school teachers' reports. A six-point screening device has been suggested:

Composition of the six indicators of schizotypy

Indicator and source	**Item**
Social withdrawal	
Psychiatric interview —	No friends during childhood
	No friends during and after adolescence
	Has never been a member of any club
	Avoids active social contact with peers
Teacher's report —	Rejected by peers
	Appears content with isolation
Social anxiety	
Psychiatric interview —	Tense during interview
	Finds it extremely difficult to make friends
	Uncomfortable in the presence of strangers
Teacher's report —	Anxious and restrained with peers
	Anxious and restrained with teacher
	Appears to be a nervous individual

[36] Su-chin Serene Olin and Sarnoff A. Mednick, 'Risk Factors of Psychosis: Identifying Vulnerable Populations Premorbidly', Schizophrenia Bulletin, Vol. 22, No. 2, 1996, p. 229.
[37] Ibid., pp. 229-230.
[38] Ibid., p. 230.

Passivity	
Teacher's report —	Waits passively for instructions
	Rarely takes part in spontaneous activities
Flat affect	
Psychiatric interview —	Affect flat or inappropriate in interview
	Facial expression flat or inappropriate
	Schizoid
Teacher's report —	Seldom laughs or smiles; serious expression
	Doesn't react when praised or encouraged
Peculiarity	
Psychiatric interview —	Queer, peculiar, eccentric, distrustful, or superstitious
Poor prognosis	
Psychiatric interview —	Prognosis bad or dubious
Teacher's report —	Likely to develop psychiatric or emotional problems

Source: S. S. Olin and S. A. Mednick, 'Risk Factors of Psychosis: Identifying Vulnerable Populations Premorbidly', Schizophrenia Bulletin, Vol. 22, No. 2, 1996, p. 234.

Researchers argue that the frequent correlations that can be found between psychiatric indicators uncovered in clinical interviews, and observations already recorded in existing teachers' reports, confirm that "schizophrenia does not appear suddenly in early adulthood".[39] But school teaching is only one of many possible observations points in the community that could be utilised to filter adolescents and separate those who have the supposed early signs of psychosis. Other observation points that have been suggested include family doctors, parents and other family members, neighbours, youth workers, unemployment case managers, sports coaches, college counsellors, homeless agencies and police.[40]

A programme of detection and intervention operated by researchers from the Rogaland Psychiatric Hospital in Stavenger, Norway has set up a particularly ambitious system of detection. Their Early Treatment and Intervention in Psychosis (TIPS) project,

> features an educational campaign about the early signs of psychosis. This campaign is aimed at health care professionals, treatment centers, teachers, school nurses, and the public, using radio, newspaper, movies, and television advertisements, as well as brochures mailed to every household in Rogaland County. The study also features a

[39] Ibid., p. 233.
[40] Max Birchwood, Pat McGorry and Henry Jackson, 'Early Intervention in Schizophrenia', British Journal of Psychiatry, Vol. 170, No. 1, January 1997, pp. 2-11.

> special early detection team on-call 7 days a week, ready to respond within hours to calls about possible cases of first-episode psychosis or prepsychotic symptoms.[41]

The essential task for the promoters of early psychosis identification and intervention is to reach consensus on three points: (1) an inventory of easily recognisable symptoms; (2) the design of a community-based catchment system that funnels at-risk people into a clinical setting; and (3) an appropriate pre-psychosis treatment programme.

Case Study — The EPPIC Programme

Perhaps the most advanced programme at this stage, and one that is consistently cited as a model, is run by the Early Psychosis Prevention and Intervention Centre (EPPIC) in Melbourne, Victoria, Australia.[42] EPPIC was established by the Victorian government Department of Health and Community Services to provide a statewide specialised service in first episode psychosis.

The researchers at EPPIC use the term 'early psychosis' variously to describe first episode psychosis, psychosis in young people, and the prodromal stage of psychosis. As a way of illustrating what they mean by prodromal symptoms analogies are drawn between measles and schizophrenia.[43] People infected with measles display a prodrome of cough and coryza which usually precedes the measles rash by 3 to 4 days. These early signs are not specific to measles and so it is not until the characteristic rash appears that it is possible to diagnose measles with certainty. The relationship of angina to an increased risk of heart attack is also used to illustrate the concept of early psychosis and further analogies are drawn between indicator signs of latent schizophrenia, like overvalued ideas and delusional mood, with symptoms indicating a developing heart disease.[44]

The EPPIC researchers acknowledge that "the onset and course of psychotic disorders is more complex than in measles, which is an 'all or nothing' phenomenon; that is, either the full disorder develops or it does not. In psychosis, defining the onset of disorder involves a degree of judgment."[45] One of the problems with defining psychosis is deciding whether or not psychotic disorders, of necessity, have to represent a break from normal psychological experience. Prodromal symptoms for schizophrenia are, by definition, indicators that precede, and are less significant than, a break with normal perceptions of reality. This suggests that it may not be correct to refer to a prodromal phase as early 'psychosis'.

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[41] Joan Stephenson, 'Schizophrenia Researchers Striving for Early Detection and Intervention', JAMA, The Journal of the American Medical Association, Vol. 281, No. 20, May 26, 1999, p. 1877.

[42] Patrick D. McGorry, Jane Edwards, Cathrine Mihalopoulos, Susan M. Harrigan, and Henry J. Jackson, 'EPPIC: An Evolving System of Early Detection and Optimal Management', Schizophrenia Bulletin, Vol. 22, No. 2, 1996, pp. 305-326.

[43] Yung et al., 'Monitoring and Care of Young People at Incipient Risk of Psychosis', op.cit., p. 284.

[44] Ibid.

[45] Ibid., p. 286.

The EPPIC researchers sometimes refer to the prodromal phase as being 'putative'.[46] They argue that it is important to reach a consensus about whether schizophrenia can be said to begin in the prodromal phase or whether the actual onset is not until the point is reached of a full-blown psychotic break.

> If the prodromal period is considered to be part of the disorder itself, then intervention at this stage would be seen as secondary — albeit early secondary — prevention. If, however, the prodrome is viewed as a separate syndrome conferring heightened but not inevitable risk for psychosis, then intervention would be viewed as primary prevention.[47]

Retrospective studies of schizophrenic people, which have sought to clarify the indications and symptoms of impending psychosis, demonstrate that the most common symptoms are, "in descending order of frequency, reduced concentration and attention, reduced drive and motivation, anergia, depressed mood, sleep disturbance, anxiety, social withdrawal, suspiciousness, deterioration in role functioning, and irritability."[48]

The EPPIC researchers, however, found these to be too nonspecific for clinical work and instead looked to the list of prodromal symptoms for schizophrenia supplied in DSM-III-R. This definition of the prodrome requires:

> at least two of the symptoms listed below:
> (1) marked social isolation or withdrawal
> (2) marked impairment in role functioning as wage-earner, student, or home-maker
> (3) markedly peculiar behaviour (e.g., collecting garbage, talking to self in public, hoarding food)
> (4) marked impairment in personal hygiene and grooming
> (5) blunted or inappropriate affect
> (6) digressive, vague, overelaborate, or circumstantial speech, or poverty of speech, or poverty of content of speech
> (7) odd beliefs or magical thinking, influencing behaviour and inconsistent with cultural norms, e.g., superstitiousness, belief in clairvoyance, telepathy, "sixth sense," "other can feel my feelings," overvalued ideas, ideas of reference

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[46] Alison R. Yung and Patrick D. McGorry, 'The Prodromal Phase of First-Episode Psychosis: Past and Current Conceptualisations', Schizophrenia Bulletin, Vol. 22, No. 2, 1996, pp. 353-370.
[47] Yung et al., 'Monitoring and Care of Young People at Incipient Risk of Psychosis', op.cit., p. 287.
[48] Ibid.

(8) unusual perceptual experiences, e.g. recurrent illusions, sensing the presence of a force or person not actually present
(9) marked lack of initiative, interests, or energy[49]

Although the above prodromal symptoms for schizophrenia were included in the diagnostic criteria of the 1987 DSM-III-R they have been omitted from the more recent DSM-IV in order to simplify the definition of schizophrenia.[50] One of the problems was that the same list was used in DSM-III-R to describe both prodromal symptoms, which precede psychotic onset, and residual symptoms, which follow remission from psychosis. The same list was used presumably because the residual phase of one psychotic break was thought to also be the prodromal phase of the next. This duplication of residual and prodromal symptoms points to a earlier problem researchers had with not being sure whether the prodromal symptoms of first break psychosis were the same as the more familiar prodromal symptoms found in the intervals between recurring bouts of psychosis.

But the EPPIC researchers were mainly concerned with identifying and classifying the symptoms which precede the first psychotic break and while the DSM-III-R list was attractive because it gave a seal of professional consensus to the concept of prodromal symptoms they were uncertain about its accuracy as a screening device for adolescents: "no one knows how common these symptoms are in similarly aged persons with no disorder".[51]

The problem of false positive diagnosis was very apparent to them and they hypothesised that there might be two groups of adolescents who would show the DSM-III-R vulnerability markers, but who would not go on to develop schizophrenia: (1) those who would eventually develop a different mental disorder, who were referred to as true-false positives, and (2) those who would avoid or prevent psychotic onset by learning some kind of adaptation or coping skills, who they called false-false positives. The false-false positives were thought of as people who had somehow made a "recovery before the frank psychosis develops".[52]

However, when the EPPIC researchers conducted a community survey of Australian high school students to determine the prevalence of the nine DSM-III-R prodromal symptoms in the general population of adolescents they found that "nearly half the sample (49.2%) had two or more symptoms and hence met the criteria for DSM-III-R schizophrenia prodrome".[53] This figure was unrealistically high for their purposes so they adjusted the threshold for diagnosis by reducing the

[49] American Psychiatric Association, Diagnostic and Statistical Manual of Mental Disorders, Third Edition — Revised, (DSM-III-R), American Psychiatric Association, Washington, 1987, pp. 194-195.
[50] American Psychiatric Association, Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, (DSM-IV), American Psychiatric Association, Washington, 1994, p. 779.
[51] Yung et al., 'Monitoring and Care of Young People at Incipient Risk of Psychosis', op.cit., p. 288.
[52] Ibid., p. 288.
[53] Ibid., p. 289.

number of symptoms. They further restricted the threshold by requiring that the symptoms needed to have been present for at least a week but less than 5 years. The 5 year limit was specified to exclude features that might have been incorporated as personality traits. When the new restricted criteria were tested on ordinary high school students it was found that only "10 to 15 percent of the sample met the criteria for schizophrenia prodrome."[54]

The usually accepted rate for schizophrenia in the general population is about 1 percent[55] and the discrepancy between that figure and the 10-15 percent for the prodrome was explained as being due to the inclusion of large fractions of both false-false positives — i.e. "students [who] were undergoing 'outpost syndromes', that is, syndromes that resemble schizophrenia prodromes but that resolve spontaneously"[56] — and true-false positives — i.e. students who will go on to develop a mental disorder other than schizophrenia.

Satisfied that the modified DSM-III-R prodromal criteria could be used to correctly identify their target group the EPPIC researchers went on to implement "a specialised outpatient service to monitor and care for young people thought to be at high risk for psychosis".[57] Demonstrating a skill for coining catchy acronyms they called their new clinic PACE — Personal Assistance and Crisis Evaluation. Although PACE was to be a subsidiary programme of EPPIC it was decided it should not be located with EPPIC. The reason given was to protect the at-risk group of clients from the stigma that might attach to them if they were known to be visiting a clinic which was clearly dedicated to the treatment of serious mental illness. To further avoid this association the PACE clinic was located at a generalised outpatient service and health promotion centre called the Centre for Adolescent Health.[58]

This deception was motivated by more than a simple concern for protecting the clients from stigma. The EPPIC researchers did not want anything to impede the flow of clients into their clinic. It was thought that if a frank association with mental illness were declared up front it might "affect referrals, as primary caregivers may be afraid of the perception that they are labelling young people detrimentally. Stigma can also lead to attendance problems".[59]

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[54] Ibid.
[55] Eadbhard O'Callaghan, Tessa Gibson, Hubert A. Colohan, Peter Buckley, David G. Walshe, Conall Larkin and John L. Waddington, 'Risk of schizophrenia in adults born after obstetric complications and their association with early onset of illness: a controlled study', British Medical Journal, Vol. 305, No. 6864, 21 November, 1992, pp. 1256-1260.
[56] Yung et al., 'Monitoring and Care of Young People at Incipient Risk of Psychosis', op.cit., p. 289.
[57] Ibid.
[58] Ibid., p. 291.
[59] Ibid.

On top of the deception in the naming and location of PACE it also appears as if the clients themselves were not properly informed about the real purpose of the programme into which they were inducted. Instead of informing the clients that they had been diagnosed for being at-risk of developing psychosis, and were therefore being treated for early psychosis, "the need for intervention was explained in relation to the patient's presenting problems. For example, the focus might be on helping a young person with social skills and coping at school."[60] In other words, the clients were led to believe that their self-evident symptoms were all that was wrong with them and they were not told that these minor deviations were thought to be early indicators of serious mental disease.

The initial programme of the PACE clinic targeted young people between 16 and 30 years of age. These people were divided into three groups by categorising their symptoms. Group 1 were people who met the DSM-III-R criteria for schizophrenia prodrome and who also had a first or second degree relative with a history of a DSM-III-R psychotic disorder or schizotypal personality disorder. Group 2 were people who had one or more of the DSM-III-R positive-only criteria for schizophrenia prodrome — i.e. (1) markedly peculiar behaviour; (2) digressive, vague, overelaborate, or metaphorical speech; (3) odd or bizarre ideation or magical thinking; (4) unusual perceptual experiences. Group 3 were "young people with a history of fleeting psychotic experiences that spontaneously resolved (called brief limited intermittent psychotic symptoms, or BLIPS) within 1 week".[61]

To detect these types of people in the community, and channel them into the PACE clinic, a public education campaign was launched aimed particularly at general practitioners and other specialised professionals who are frequently in contact with young people — like school counsellors, teachers and youth workers. The Early Psychosis Prevention and Intervention Centre itself, which specialises in assessing and managing young people who are deemed to have already crossed over into psychosis, proved useful in channelling a number of people who had failed to meet the threshold criteria for actual psychosis, but who appeared to be on the way.

EPPIC's "mobile Early Psychosis Assessment Team, which, through extensive community networking, comes into contact with not only young people experiencing psychosis but also some 'doubtful' cases who may be in the prodromal phase",[62] was also a useful source of referrals.

The initial PACE programme involved 52 patients; 22 were students, 6 were in employment and 24 were unemployed. Their "most frequently occurring DSM-III-R prodromal symptoms were magical thinking, perceptual disturbance, and impaired role function, present in 67.7, 54.8, and 54.8 percent

[60] Ibid., p. 292.
[61] Ibid.
[62] Ibid., p. 290.

of the subjects, respectively".[63] The researchers were aware that many of these patients could have been false positives and so they "carefully weighed the benefits of receiving treatment during the at-risk mental state versus the risks involved, if such treatment was unnecessary."[64]

Treatment involved either psychosocial talking therapy or neuroleptic medication — and sometimes a combination of the two. It was thought that:

> psychosocial interventions may be justified when nonspecific symptoms only are present. But prescribing neuroleptic medication may not be justified, because of the risk of side effects including tardive dyskinesia, until more specific signs occur. Using neuroleptic medication at this early stage may be highly effective, however; hence, the duration of neuroleptic treatment may only need to be brief, thereby reducing the likelihood of short- and long-term side effects.[65]

By 1996 the EPPIC researchers were ready to claim success for the initial PACE programme which, they say, proved "that it is possible to identify and follow possibly prodromal individuals in the community".[66] But they were concerned with the fact that many of the patients monitored during the course of the programme did not make the transition to full psychosis. The transition rate to psychosis of people who have been identified with prodromal symptoms presents an interesting problem of interpretation. On the one hand, if most of the people treated for prodromal symptoms fail to cross the threshold into psychosis it can be claimed that the preventive treatment was successful. But on the other hand, it might also indicate that the prodromal indicators were not accurate and that a substantial fraction of false positives were included amongst the patients.

One way to resolve the interpretative problem would have been to follow the lead of the Buckingham Project and attempt to calculate whether the overall incidence of psychosis was reduced in the catchment area. But the PACE sample was apparently too small, and the catchment area too large, to make this approach practical. Instead the researchers decided to interpret the low transition rate of their patients as indicating that a substantial fraction were actually false/positives.[67]

Notwithstanding this lack of confidence in their diagnostic and treatment procedures the researchers began a new prospective study of at-risk individuals using up-dated diagnostic criteria. The same 16 to 30 years age group was targeted and there was the same division of the clients into three study

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[63] Ibid., p. 293.
[64] Ibid., p. 291.
[65] Ibid.
[66] Ibid., p. 299.
[67] Ibid.

groups. But this time Group 1 was defined by a combination of having a first degree relative with a history of DSM-IV psychotic disorder or schizotypal personality disorder together with "any change in mental state or functioning resulting in a loss of 30 points or more on the Global Assessment of Functioning (GAF; American Psychiatric Association 1987) scale, including nonspecific 'neurotic'-type presentations such as anxiety and depressive syndromes".[68]

The GAF scale assumes that the level of an individual's psychological, social and occupational functioning can be plotted on a continuum which extends from prime mental health to serious mental illness. The continuum consists of nine paragraphs, each representing 10 points on the scale, which describe levels of functioning which progress from very high functioning to almost complete dysfunctionality. The task of measuring a person on the GAF involves choosing the paragraph that best describes the subject's level of functioning at the time of an interview and allotting a score between 1 and 90 according to the relative position of the chosen paragraph on the continuum.[69]

The revised Group 2 required the presence of one DSM-IV symptom for schizotypal personality disorder. Schizotypal personality disorder is a non-psychotic disorder of personality that has a number of schizophrenia-like symptoms: "The essential feature of Schizotypal Personality Disorder is a pervasive pattern of social and interpersonal deficits marked by acute discomfit with, and reduced capacity for, close relationships as well as by cognitive or perceptual distortions and eccentricities of behaviour."[70] There are nine symptoms listed for this condition and a diagnosis usually requires the presence of five or more.

(1) ideas of reference (excluding delusions of reference)
(2) odd beliefs or magical thinking that influence behaviour and is inconsistent with subcultural norms (e.g., suspiciousness, belief in clairvoyance, telepathy, or "sixth sense", in children and adolescents, bizarre fantasies or preoccupations)
(3) unusual perceptual experiences, including bodily illusions
(4) odd thinking and speech (e.g. vague, circumstantial, metaphorical, overelaborate, or stereotyped)
(5) suspiciousness or paranoid ideation
(6) inappropriate or constricted affect
(7) behaviour or appearance that is odd, eccentric or peculiar
(8) lack of close friends or confidants other than first-degree relatives

[68] Ibid.
[69] American Psychiatric Association, Diagnostic and Statistical Manual of Mental Disorders, Third Edition Revised, (DSM-III-R), op.cit., p. 12.
[70] American Psychiatric Association, Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, (DSM-IV), op.cit., p. 641.

(9) excessive social anxiety that does not diminish with familiarity and tends to be associated with paranoid fears rather than negative judgments about self [71]

To be included in Group 2 of the new PACE programme the person's schizotypal symptom needed to occur several times a week and have been present for at least one week. The symptom should also "deviate significantly from normal as defined by a score of 2 or more on the unusual thought content scale of the BPRS or be held with a reasonable degree of conviction as defined by a score of 2 on the CASH rating scale for delusions".[72]

(The BPRS is a system of psychiatric assessment devised in the early 1960s called the Brief Psychiatric Rating Scale. The CASH is a more recently invented assessment tool and stands for the Comprehensive Assessment of Symptoms and History. These rating systems will be discussed a little further on.)

The revised criteria for Group 3 required that a person have a history of transient psychotic symptoms like delusions, hallucinations or unusual thoughts.

This revised PACE study was also aimed at testing the "predictive power of a number of putative trait markers of schizophrenia".[73] As well as the various "attenuated psychotic symptoms" listed above, the putative trait markers being examined included "neurobiological markers such as increased ventricular brain ratio and ventricular enlargement, neurochemical markers such as reduced dopamine uptake by platelets, and neuropsychological markers such as information processing deficits".[74]

The project was divided into two stages. In the first stage the young people thought to be at-risk of developing schizophrenia were to be divided into two streams: (1) those not receiving any treatment at all, and (2) those receiving a combination of psychosocial therapy and neuroleptic medication. The object of the first stage was to compare the transition rates to full psychosis for these two streams. The second stage divided another intake of patients into a further two comparative streams: (1) those who only receive psychosocial therapy, and (2) those who only receive neuroleptic medication. The purpose of this second stage was to similarly observe the comparative rates of progression on to full psychosis.[75]

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[71] Ibid., p. 645.
[72] Yung et al., 'Monitoring and Care of Young People at Incipient Risk of Psychosis', op.cit., p. 299.
[73] Ibid.
[74] Ibid.
[75] Ibid., p. 300.

In a recent publication the EPPIC researchers discuss progress they have made in their project and they disclose some of their findings.[76] The critical issues which they set out to explore were whether the revised prodromal symptoms could accurately identify a group of people who were at-risk of psychosis and whether some kind of prophylactic treatment might help to protect them from psychosis. However, the researchers apparently encountered a major obstacle with these lines of inquiry.

Their problem lay in the imprecision of the existing psychiatric understanding of psychosis. All the individuals in the study had been chosen because they had psychotic-like tendencies. But at the time of selection they were deemed to be not yet psychotic. The purpose of the research was to make precise observations of their individual progression in relation to the threshold of psychosis. What the researchers discovered was that this threshold had no precise definition: "The main outcome measure in this study was the development of psychosis. The point of onset is difficult to define prospectively and has to be defined arbitrarily."[77]

When the point of psychotic onset is determined retrospectively, which is the usual way of making a determination, it can be simply pegged to the point at which a person was first thought to be in need of psychiatric care, treatment and control. But psychiatric treatment and care had been already given to the people in the PACE study while they were still in an acknowledged pre-psychotic state, so their situation raised the issue of psychotic onset as something that required more precise definition.

This was not a new problem for some members of the EPPIC team. In earlier research, as part of a satellite study to the DSM-IV field trial for schizophrenia, the National Health and Medical Research Council Schizophrenia Research Unit's Early Psychosis Prevention and Intervention Centre had conducted a programme which assessed people with first-episode psychosis. Patrick McGorry, the Director of EPPIC, had participated in this work. In the research independent raters used four different procedures to determine a diagnosis for psychosis. They found there was a high level of misclassification which arose from variations in the methods of assigning criteria which determine the onset of psychosis. They warned that this lack of consensus would impede future research in the area of early psychosis.[78]

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[76] Alison R. Yung, Lisa J. Phillips, Patrick D. McGorry, Mats A Halgren, Colleen A. McFarlane, Henry J. Jackson, Shona Francey and George C. Patton, 'Can we predict the onset of first-episode psychosis in a high-risk group?', International Clinical Psychopharmacology, Vol. 13 (suppl 1), 1998, pp. s23-s30.

[77] Ibid., p. s26.

[78] Patrick D. McGorry, Cathy Mihalopoulos, Lisa Henry, Jenepher Dakis, Henry J. Jackson, Michael Flaum, Susan Harrigan, Dean McKenzie, Jayashri Kulkarni and Robert Karoly, 'Spurious precision: procedural validity of diagnostic assessment in psychotic disorders', American Journal of Psychiatry, Vol. 152, No. 2, February 1995, pp. 220-224.

The 'arbitrary' method of definition that was settled upon for the PACE study involved the specification of supposedly precise levels of a variety of psychotic indicators — like hallucinations, delusions, thought disorder and suspiciousness — on the BPRS and CASH rating scales. The EPPIC researchers reassured themselves that this definition of onset is similar to one used by other researchers in the field "and is in line with common clinical practice for instigation of neuroleptic treatment".[79]

Accordingly they found that 48% of the people inducted into the research programme became psychotic within the first 12 months. The transition rate at 6 months was 40%.[80] Satisfied that they were on the right track with the revised symptomatology the researchers pointed to their next hurdle: "The ultimate question is, having identified precursor features with good positive predictive power, can the onset of psychosis be prevented by early intervention?"[81]

This question points to a serious gap in the material published about the PACE programme. The research programme using the schizotypal symptoms, as it was outlined in the Schizophrenia Bulletin[82] in 1996, had specifically intended to compare the transition to psychosis of patients who were given no treatment at all against those who received a combination of psychosocial therapy and neuroleptic treatment. It had also intended to compare the efficacy of psychosocial therapy against neuroleptic treatment.[83] Unfortunately the results of this research have not been published yet. What is required are precise details about whether a particular kind of prophylactic treatment might help to prevent psychosis. Or whether, on the other hand, another kind of treatment might actually help to induce psychosis. In a recent interview with a journalist McGorry claimed that:

> Some preliminary results of a study comparing a small group of high-risk patients who received low doses of one of the newer antipsychotic medications (risperidone) with a control group of patients who were offered supportive treatment and monitoring found that in the 6-month treatment phase of the study, only 9.5% of the patients receiving drug therapy progressed to psychosis vs 36% of the control group.[84]

The problem with this anedote is that the control group is not identified so it is not clear whether the two groups McGorry describes involve the drugs plus psychosocial treatment vs no treatment comparisons or the drug treatment vs psychosocial treatment comparisons. Whichever is the case it

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[79] Yung et al., 'Can we predict the onset of first-episode psychosis in a high-risk group?', op.cit., p. s26.
[80] Ibid., p. 28.
[81] Ibid., p. s29.
[82] Yung et al, 'Monitoring and Care of Young People at Incipient Risk of Psychosis', op. cit., pp. 283-303.
[83] Ibid., p. 300.
[84] Stephenson, op.cit.

seems apparent that McGorry is preparing the ground for the promotion of drug-based treatment for prepsychotic symptoms.

Perhaps a reason for the delay in publishing full details of this research stems from an unresolved ethical dilemma concerning the treatment of non-psychotic people with neuroleptic medication. If only about half of people with prodromal symptoms progressed on to psychosis it could mean that the remaining half are false/positives: "because over 50% of cases do not develop psychosis within twelve months routine treatment of this group would result in many young people being subject to unnecessary treatment and labelling."[85]

But unnecessary treatment of false/positives is not the only ethical question exercising the minds of the PACE researchers. In 1997 they published an article that gave some insight into other types of ethical matters that also worried them. Under the heading of "Ethical Issues" they rhetorically asked: "is our belief that someone is at high likelihood of imminent deterioration into psychosis enough to invoke involuntary status under the Mental Health Act?"[86] Involuntary treatment is a key issue with these psychiatrists since two-thirds of their patients at EPPIC's first-episode psychosis clinic are involuntary patients.[87] In the same article they also went on to discuss the cost/benefit ratios of psychosocial and neuroleptic treatments and foreshadowed that "with the advent of newer antipsychotic medications with fewer side-effects especially at low doses, one could argue that a shift downwards in the cost/benefit ratio is occurring."[88]

Despite the apparently unresolved ethical dilemmas, and the delayed publication of treatment results, the EPPIC researchers are clearly using their PACE research programmes to establish a degree of hegemony in the theory of diagnosing and treating early psychosis. In conjunction with the University of Melbourne's Department of Psychiatry they have recently initiated a Graduate Diploma in Mental Health Sciences: "EPPIC Statewide has become sensitised to a growing demand amongst clinicians for a program of study with a focus on maximising the preventive opportunities during the onset phase of serious mental illness in young people".[89] The course is offered via distance education and is designed for health care professionals who are already qualified in the

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[85] Yung et al., 'Can we predict the onset of first-episode psychosis in a high-risk group?', op.cit., p. s28.

[86] Alison R. Yung and Patrick D. McGorry, 'Is pre-psychotic intervention realistic in schizophrenia and related disorder?', Australian and New Zealand Journal of Psychiatry, No. 31, 1997, p. 802.

[87] Paddy Power, Early Psychosis Prevention and Intervention Centre, in answer to a question after presentation of paper entitled, An Analysis of the Initial Treatment Phase and Follow-Up of First Episode Psychosis Patients, Second National Conference on Early Psychosis, Hobart Tasmania, 4-5 September, 1998.

[88] Yung and McGorry, 'Is pre-psychotic intervention realistic in schizophrenia and related disorder?', op. cit., p. 803.

[89] Early Psychosis Prevention and Intervention Centre, Graduate Diploma in Mental Health Sciences (Young People's Mental Health) 1998 Course Handbook, University of Melbourne, 1998, p. 3.

areas of psychiatry, medicine, psychology, nursing, occupational therapy, social work and other related disciplines.[90]

The EPPIC researchers have also been active in developing national standards of best practice for early psychosis intervention and treatment in Australia. In early 1996 EPPIC won a tender to undertake the National Early Psychosis Project (NEPP).[91] The EPPIC clinic in Melbourne was made the location of the National Manager of NEPP. NEPP was jointly funded by the commonwealth and state governments of Australia to fulfil a number of aims:

* To facilitate the development and promotion of best practice in the identification and optimal early intervention in psychosis
* To progress mental health policy to ensure that services adopt and incorporate best practice principles in early psychosis delivery.
* To enhance the capacity of mental health professionals around Australia to meet the needs of young people with emerging psychosis.
* To develop a network through which mental health professionals, consumers, other key stake holders can share information and ideas about early psychosis.
* The emphasis of the project is on the development of collaborative and cooperative endeavours which can foster national agreement on best practice in this area of work whilst achieving a sustainable legacy of enhancements within the field of early psychosis.[92]

To facilitate this project a national office of NEPP was located with EPPIC in Melbourne, as well as the Victorian office of NEPP, while other state offices were located in the capital cities of each Australian state. NEPP was conceived as an 18 month project and was set-up to run until January 1998. Throughout the life of the project EPPIC was clearly its major driving force and its principal centre for research and policy development. It can therefore be assumed that PACE research projects have been influential in devising the national best practice model that has emerged from NEPP.

This best practice model is embodied in the recently published Australian Clinical Guidelines for Early Psychosis.[93] These guidelines have been developed to guide best practice in the diagnosis and

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[90] Ibid.

[91] Early Psychosis Prevention and Intervention Centre, Newsletter of the Early Psychosis Prevention and Intervention Centre, Number 4, April, 1996, p. 8.

[92] National Early Psychosis Project, The Development and Promotion of a National Best Practice Model in Early Intervention in Psychosis, National Early Psychosis Project, accessed September 1997, Available URL, http://yarra.vicnet.net.au/-eppic/nepp.html

[93] National Early Psychosis Project, Australian Clinical Guidelines for Early Psychosis, National Early Psychosis Project, Melbourne, 1998.

treatment of early psychosis in Australia. The guidelines extend the definition of early psychosis to include "the period described as the prodrome and .... also .... to include the critical period up to five years from entry into treatment for the first episode".[94] Intervention during the prodromal phase is considered to be prevention and a chart supplied in the guidelines divides preventive strategies into the three levels of application described earlier: universal, selective and indicated.[95]

Critical Analysis of Early Psychosis

To those who are sceptical about the medical model for schizophrenia, and who prefer one of the alternative models, the extension of the pathological definition of schizophrenia to include a prodromal phase should be disturbing. Without a prodromal phase a person is safe from unwanted psychiatric attention so long as a threshold of eccentricity is not crossed. This boundary of social tolerance, which psychiatrists refer to as a psychotic break, allows supporters of the mystical and myth-of-mental-illness (M-M-I) models to console themselves with the understanding that it is only people who can not control overt displays of mental deviance that are at risk of coercive psychiatric intervention in their lives.

In these circumstances supporters of the mystical model can argue that it is only unlucky or incompetent mystics who get labelled as schizophrenics. Similarly, advocates of the M-M-I model can believe that it is only careless or indifferent social deviants that allow themselves to become vulnerable to psychiatric scape-goating and out-casting. The existence of a commonly understood threshold which separates normal people and normal behaviour from mad people and mad behaviour gives a measure of reassurance to non-conformists and their supporters that psychiatric detection and intervention can be avoided so long as care is taken.

But the concept of early psychosis classifies mere tendencies towards mental deviance as indications of serious mental illness. The identification of a prodrome means that it is no longer required of a person to dive into the mystical waters to be branded as mentally disordered. Where there is a community screening programme for early psychosis it likely that a young person will be diagnosed with mental illness merely for showing and interest in the idea of mysticism.

Similarly, from the M-M-I perspective, it becomes no longer necessary to be recognisable as a social outcast, or to be used as a scapegoat, before the schizophrenia label is applied — now all that is necessary is to have a shortage of friends or to admit to minor difficulties in social functioning. A screening programme which purports to prevent schizophrenia, from the perspectives of these other models, can also be seen as preventing interest in the mystical experience and/or preventing individual difference.

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[94] Ibid., p. 11.
[95] Ibid., p. 15.

At this stage of development the medical model's definition of a prodrome for schizophrenia seems highly vulnerable to accusations of arbitrariness. The EPPIC research, which experimented with various lists of prodromal symptoms, making adjustments when the fraction of school children caught in the net was too high, clearly demonstrates arbitrariness. A similar level of arbitrariness in relation to the definition of the point of entry into psychosis has been admitted by the researchers. This arbitrariness seems so transparent and pervasive that it raises questions whether the extension of the concept of schizophrenia into a prodromal phase could possibly threaten the plausibility of the whole medical model of schizophrenia.

It does not require much scepticism, for instance, to find the early psychosis screening system used in the Buckingham Project somewhat doubtful. Doctors were required to question their patients about such matters as sleeping patterns, appetite, level of interest in things, everyday worries, ability to concentrate on television, level of optimism, odd habits, panic, speech difficulties and hearing voices. A screening device like this is so broad-ranging that almost everybody would be able to give a positive answer to at least one of the symptoms at any given time.

What does it mean, for instance, when a person admits to a raft of everyday money and family worries if there is an economic recession in progress and the source of the problem is anxiety about losing employment? Or what does it mean if a person's sleeping patterns are disrupted when a spouse is doing shift work? There are so many different reasons why a person might manifest some of those symptoms, and they are such common experiences, (except for hearing voices which is normally specified as a symptom of psychosis, not the supposed prodrome), that it is hard to escape the conclusion that the Buckingham Project's criteria make the experience of minor hardships in life a sign of pathology.

In the above account of the PACE research programmes it is interesting to note that in the initial programme the most frequently occurring symptom, for which nearly 70% of the young people were treated, was magical thinking. But the standard psychiatric definition of magical thinking does not describe a particularly debilitating symptom. Many non-scientific approaches to dealing with 'problems of living', like prayer for instance, can easily fit the definition. Indeed, even some established phenomena of medical practice, like the placebo effect, could also fit. The Glossary of Technical Terms in DSM-IV defines it as:

> **magical thinking** The erroneous belief that one's thoughts, words, or actions will cause or prevent a specific outcome in some way that defies commonly understood laws of cause and effect. Magical thinking may be part of normal child development.[96]

It is not immediately apparent whether the programme by early psychosis researchers to eliminate magical thinking in young people supplies more evidence in support of the mystical model or the M-M-I model. But it does not supply good evidence to support the medical model. If magical thinking is indeed a normal part of child development, as DSM-IV suggests it might be, what conclusions are to be drawn about adolescents who still experience it? Are they deliberately contravening social conventions by persisting in patterns of thought that defy "commonly understood laws of cause and effect"? If so they would be better understood from the angle of the M-M-I model as social deviants. On the other hand they might be resisting conformity of thought in a way that will later lead them into the unusual psychological phenomena of mystical experience. Should we then consider magical thinking as a part of a prodrome for mystical experience?

In fact there is something quite extraordinary in the fact that the EPPIC researchers have been psychiatrically treating people for magical thinking. Patrick McGorry, the Director of EPPIC, is the one of the principal authors of a 1997 publication called the Early Psychosis Training Pack. In this document earlier research of McGorry's is cited which found that 51% of Australian 16-year-olds experience magical ideation.[97] If an outright majority of young people are known to have magical ideas should this type of mental activity be considered normal, and therefore not an indication of impending psychosis? Or, alternatively, is it an indication of the deplorable mental health of young Australians since a clear majority are shown to have a symptom of the schizophrenia prodrome? The EPPIC researchers do not mount any discussion about these choices of interpretation and their use of magical thinking as a prodromal symptom would appear to require some explanation.

The PACE research programmes also appear to raise some serious problems in regard to informed consent that might make them somewhat doubtful models for the development of national best practice standards for Australia. In their Schizophrenia Bulletin[98] article the EPPIC researchers Yung et al were surprisingly candid about the deliberate deception in the choice of the name and location of PACE. They even suggest they might have deliberately misled general practitioners about the real purpose of PACE so as not to risk losing referrals. The patients themselves were not informed about the true nature of their clinical diagnoses and a pretence was maintained that

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[96] American Psychiatric Association, Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, (DSM-IV), op.cit., p. 768.

[97] Patrick D. McGorry and Jane Edwards, Early Psychosis Training Pack, Module 1, Gardiner-Caldwell Communications, Macclesfield, Cheshire, 1997, p. 17, cited in table adapted from, McGorry et al., 'The prevalence of prodromal features of schizophrenia in adolescence: a preliminary survey', Acta Psychiatrica Scandanavica, 92, 1995, pp. 241-249.

[98] Yung et al., 'Monitoring and Care of Young People at Incipient Risk of Psychosis', op.cit., pp. 283-303.

treatment was being given to correct minor social problems. These deliberate deceptions on the part of the EPPIC researchers suggest that even the researchers themselves might lack confidence in the validity of a medical model for the prodrome of schizophrenia.

Although it is not clearly stated, this lack of an appropriate level of informed consent appears to also extend to the more recent PACE prospective research project.[99] In the first stage of this project, for which the subjects were divided into two groups, it seems likely that at least the people in the untreated group were not properly informed about the true nature of the research. There would seem to be little reason for people to remain in the programme, after being told they had the early symptoms of serious mental illness, if they had been properly informed that they had been assigned to a group for observation and would not receive any treatment.

The revised criteria for inclusion in this prospective study also require some comment in light of the alternative models for schizophrenia. The criteria for Group 1 suggest that if a person has a first degree relative with a history of psychosis or schizotypal personality disorder then any kind of temporary set-back in personal relationships or social functioning is psychiatric evidence of impending psychosis.

The GAF scale on which a set back in social functioning is to be measured does not provide any opportunity for evaluating the reason why such a set back might occur. Although the scale provides an instruction to exclude "impairment in functioning due to physical (or environmental) limitations"[100] there is no provision to take into account other common factors like loss of employment, exam failure, residential disruption or disruption in personal relationships.

The significance of a 30 point difference on the GAF scale can be illustrated by comparing two descriptions from the scale published in DSM-IV. At the top of the scale on 100 points a person can be described as having:

> Superior functioning in a wide range of activities, life's problems never seem to get out of hand, is sought out by others because of his or her many positive qualities. No symptoms.[101]

While 30 points down a person might have:

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[99] Ibid., pp. 299-300.
[100] American Psychiatric Association, Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, (DSM-IV), op.cit., p. 32.
[101] Ibid.

> Some mild symptoms (e.g. depressed mood and mild insomnia) or some difficulty in social, occupational, or school functioning (e.g., occasional truancy, or theft within the household), but generally functioning pretty well, has some meaningful interpersonal relationships.[102]

An underlying assumption of the PACE researchers apparently is that when a person has a first degree relative with a history of mental illness, although the person might only have a "few mild symptoms" and be "generally functioning pretty well", if this level of functioning is still considerably lower than a previous level, then this set back is to be read as part of a slide towards psychosis. Given this criteria it would seem wise for any adolescent in the EPPIC catchment area of Melbourne, who has a mentally ill relative, to always put on a cheerful face in the company of medical practitioners.

Group 2 patients for this prospective study were identified by the presence of any single symptom on the DSM-IV diagnostic criteria for schizotypal personality disorder. It was required that the degree of abnormality of this symptom should be sufficient to meet a specified level of deviation on two independent rating scales — the BPRS (Brief Psychiatric Rating Scale) and the CASH (Comprehensive Assessment of Symptoms and History). Any of the schizotypal personality disorder symptoms used to identify a psychotic prodrome had to be scored at two or more on the BPRS or two or more on the CASH rating scale for delusions.

Both the BPRS and the CASH are similar to the GAF in that an interviewer is required to subjectively estimate a person's level of mental function by observing specified characteristics and choosing an appropriate level of dysfunction according to a scale. In the case of the BPRS there are 16 specified symptoms of dysfunction that have to each be rated for intensity on a 7 level scale — i.e. not present, very mild, mild, moderate, moderate severe, severe, extremely severe.[103]

If level 1 is a determination that the symptom is "not present" and level 2 only describes a symptom as being "very mild" then this means that a specification that a symptom rate at 2 or more on the BPRS actually means that only the slightest trace of the symptom needs to be found. Similarly, the CASH rating scale for delusions is a five level scale — questionable, mild, moderate, marked, severe — and a rating of 2 (mild) therefore only requires a degree more certainty than that of "questionable".[104]

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[102] Ibid.

[103] John E. Overall and Donald R. Gorham, 'The Brief Psychiatric Rating Scale', Psychological Reports, Vol. 10, 1962, p. 803.

[104] Nancy C. Andreasen, Michael Flaum and Stephan Arndt, 'The Comprehensive Assessment of Symptoms and History (CASH)', Archives of General Psychiatry, Vol. 49, August 1992, p. 618.

The purpose of specifying a minimum rating for schizotypal symptoms is apparently to ensure that the diagnostic threshold is set high enough to exclude people who only have mild symptoms. However, the above analysis clearly demonstrates that a threshold of 2 on the BPRS and CASH scales still allows a person with the slightest trace of a symptom to be included. This begs the question: why specify a diagnostic threshold if the setting is too low to be exclusive?

There is also another curious aspect about these threshold specifications. Ostensibly the idea is that a diagnostician should measure the severity of a schizotypal symptom by matching it to scaled severity descriptions on the BPRS and CASH scales. To do this there needs to be a close correlation between the description of schizotypal symptoms and the symptoms that are rated for severity on the BPRS and CASH — otherwise a match cannot be made. But when these comparisons are made it is evident there is only a partial and fragmentary correlation between the 9 schizotypal symptoms and the 16 BPRS symptoms. On top of this there is no correlation at all between the CASH rating symptom for delusions and the schizotypal symptoms because delusions have been specifically excluded from the schizotypal diagnostic criteria.[105]

Added together these anomalies concerning the BPRS and CASH rating specifications suggest that they actually have no significance at all for distinguishing people who have the psychotic prodrome. It is difficult to escape the conclusion that these rating specifications have only been introduced to give a superficial semblance of scientific precision to the research.

Perhaps there was a reason why this was thought to be necessary. The PACE prospective study, by utilising the symptoms of schizotypal personality disorder, appropriates, without explanation, diagnostic criteria for a mental disorder that is not normally considered to be a prodrome for psychosis. In fact the DSM-IV description of the condition specifically states that, "Schizotypal Personality Disorder has a relatively stable course, with only a small proportion of individuals going on to develop Schizophrenia or another Psychotic Disorder".[106] If this is true about people who meet the normal diagnostic criteria for the disorder, by having five or more of the symptoms, what is to be assumed about the real risk of psychosis for people who meet the PACE diagnostic criteria for 'putative' psychotic prodrome by only having one symptom, and that single symptom perhaps only in very mild form?

Indeed, the Australian Clinical Guidelines for Early Psychosis do not recommend the use of the schizotypal symptoms. Instead a 16 item list of "Prodromal Symptoms and Signs" of psychosis is provided:

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[105] American Psychiatric Association, Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, (DSM-IV), op.cit., p. 641.

[106] Ibid., p. 643.

> Suspiciousness; Depression; Anxiety; Tension; Irritability; Mood swings; Anger; Sleep disturbances.
> Appetite changes; Loss of energy or motivation; Memory or concentration difficulties; Perception that things around them have changed; Belief that thoughts have speeded up or slowed down; Deterioration in work or study; Withdrawal and loss of interest in socialising; Emerging unusual beliefs.[107]

But even these officially endorsed indicators of the prodrome are supplied with a qualification. In order to cover themselves the authors of the Clinical Guidelines acknowledge that "these signs and symptoms are not usually indicative of a developing psychosis."[108] This is apparently said to prevent over-zealous usage and to minimise the alarm that might arise in normal people who encounter this list and reflect on themselves. The guidelines advise that these signs are only meant to be used as symptoms of impending psychosis when "they occur in individuals who have been identified as *'at-risk'* of psychosis".[109]

There is a separate table with a list that can be used to identify the people who are *'at-risk'*. The idea is to first narrow down the field before using the prodromal symptoms and signs. The narrowed field focuses on adolescents and young people. There is a fairly extraordinary mixture of further risk factors that range from the quite specific "Family history of psychotic disorder" to the equivocal "Season of birth" to the thoroughly non-specific "Life events" and "Subjective/functional change in the person".[110] Essentially the risk factors seem to be tailored to fit almost any young person who is a bit worrisome to parents, school teachers or other authority figures.

Apart from supplying its own lists of symptoms and risk factors the Clinical Guidelines also advise that "[i]nformation currently available to promote awareness and identification of symptoms is captured in the pamphlet 'Something is Not Quite Right' (SANE Australia)".[111] SANE Australia is a business name of Schizophrenia Australia Foundation which generally purports to represent the interests of relatives of schizophrenic people. The pamphlet is distributed on request with a note on letter-head which announces that the pharmaceutical company Pfizer is one of the organisation's sponsors. (Pfizer make a new atypical neuroleptic called ziprasidone).

The SANE pamphlet is directed at parents, teachers, employers and workmates of "difficult" people. Two checklists of symptoms are supplied to assist in recognising the severity of the

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[107] National Early Psychosis Project, Australian Clinical Guidelines for Early Psychosis, op.cit., p. 13.
[108] Ibid.
[109] Ibid.
[110] Ibid., p. 12.
[111] Ibid., p. 19.

underlying mental illness that might be giving rise to the difficulties. Boxes are provided beside each symptom so that observers can tick off a person's faults. Checklist 1 is:

> Behaviour which is considered **normal** although difficult. *Difficult behaviour at home, school or in the workplace*. People may be —
>
> | | | |
> |---|---|---|
> | rude | irritable | over-sensitive |
> | lazy | rebellious | weepy |
> | argumentative | over-emotional | withdrawn |
> | thoughtless | shy [112] | |

Observers are warned that these behaviours may not be cause for alarm but if they persist or are too disruptive then advice should be sought from a GP, school or workplace counsellor, Citizens Advice Bureau or Mental Health Centre.

Checklist 2 is a list of 18 behaviours which are said to be definitely abnormal and which require medical assessment as soon as possible.

* withdraw completely from family, friends and workmates.
* be afraid to leave the house (particularly in daylight hours).
* sleep or eat poorly. Sleep by day and stay awake at night, often pacing around.
* be extremely preoccupied with a particular theme, for example, death, politics, or religion.
* uncharacteristically neglect household or personal or parental responsibilities, or personal hygiene or appearance.
* deteriorate in performance at school or work, or leave jobs.
* have difficulty concentrating, following conversation or remembering things.
* talk about or write things which do not really make sense.
* panic, be extremely anxious or markedly depressed, or suicidal.
* lose variation in mood, be *flat*. Lack emotional expression, for example, humour, friendliness.
* have marked changes in mood, for example from quiet to excited or agitated.
* have inappropriate emotional responses, for example, giggling on hearing sad news.
* hear voices that no-one else can hear.
* believe, without reason, that others are plotting against, spying on, or following them and have extreme fear of, or anger at, those people.

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[112] SANE Australia, Something is not quite right, pamphlet produced by SANE Australia, Melbourne, 1998.

* believe they are being harmed, or influenced to do things against their will — by television, radio, aliens or the devil, for example.
* believe they have special powers, for example — that they are important religious leaders, politicians or scientists when this is not the case.
* believe their thoughts are being interfered with or that they can influence the thoughts of others.
* spend extravagant and unrealistic sums of money.[113]

The SANE pamphlet advises that if the person demonstrates "outright resistance to the idea of visiting the doctor, consult with the doctor yourself to work out a plan over time. It may be possible and appropriate for the doctor to assess the person at home".[114] When a person is reluctant to submit to a medical assessment it is likely that the doctor will see the friends or relatives as his/her client rather than the person to be assessed. This introduces a great deal of scope for bias in the assessment particularly since the symptomatology is largely a matter of opinion. Summary detention in a mental hospital or coercion to participate in a pre-psychosis treatment programme are likely outcomes.

The SANE checklists of symptoms have been reproduced in full because their official endorsement by the Clinical Guidelines has given them special significance. They are not simply the opinions of an interest group but have been integrated into the official definition of pre-psychotic schizophrenia. This special significance should be considered in the context of a discussion about competing explanatory models for schizophrenia. The SANE programme of using non-medical people as front-line diagnosticians and encouraging them to identify and report people who are irritating/offensive/disturbing must give some credence to the myth-of-mental-illness model. The potential of using psychiatric coercion for social control is particularly evident in the fourth symptom of the above list: "be extremely preoccupied with a particular theme, for example, death, politics, or religion".

Drug Company Influence

Some of the risk factors for psychosis specified in the Clinical Guidelines are based on hypotheses of aetiology for schizophrenia that remain unconfirmed. "Season of birth", for instance, is a hypothesis of doubtful merit. Research undertaken in Scotland found that 13,661 schizophrenics born between 1914 and 1960 had fluctuations in the numbers born in the months of February, March, April and May. The fluctuations appeared to be tied to the temperature pattern six months earlier — the colder the autumn, the higher the incidence of schizophrenic births the following

[113] Ibid.
[114] Ibid.

spring.[115] However, these findings are not confirmed by research in other countries. Korean researchers, for instance, who attempted to replicate the Scottish findings in their own country found no statistical link to season of birth at all.[116]

It has been claimed that statistical research undertaken in Queensland, Australia confirms the season of birth hypothesis by indicating that schizophrenics born in the Southern Hemisphere have a seasonal pattern of birth which mirrors those born in the Northern Hemisphere.[117] But any bias towards season of birth in either hemisphere, if it exists at all, is obviously only a minor factor. Even the proponents of the season of birth hypothesis only claim that there is rise in the number of schizophrenic births at certain times of the year, not that all, or even most, schizophrenic births are tied to a seasonal calendar. The hypothesis might be a useful adjunct to aetiological research but to use season of birth as a diagnostic indicator, as the Clinical Guidelines do, is quite ludicrous. The majority of people who develop schizophrenic symptoms are still born outside the 'risk season' while the vast majority of people who are born within the 'risk season' are obviously not at any risk of developing schizophrenia.

So why does season of birth appear as a 'risk factor' in the Clinical Guidelines? Perhaps it is because the list of 'risk factors' and the list of 'symptoms and signs' were not originally devised for the Clinical Guidelines but were adopted without comment from a publication called the Early Psychosis Training Pack.[118] Although the principal authors of this Training Pack are the Director and Assistant Director of EPPIC the document was produced by a British public relations company which specialises in pharmaceutical marketing called Gardiner-Caldwell Communications.[119] The Training Pack was funded by an 'educational grant' from the pharmaceutical company Janssen-Cilag.[120] Janssen-Cilag manufacture a new atypical neuroleptic used for treating schizophrenia called Risperdal (risperidone).

It is perhaps worth noting that Gardiner-Caldwell also publishes a web-based journal entitled Influenza Bulletin[121] for an organisation called the European Scientific Working-Group on Influenza (ESWI). ESWI receives funding from a number of pharmaceutical companies — two of which are Solvay Pharma and SmithKline Beecham. Both Solvay and SmithKline Beecham

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[115] R. E. Kendell, and W. Adams, 'Unexplained Fluctuations in the Risk for Schizophrenia By Month and Year of Birth', British Journal of Psychiatry, Vol. 158, 1991, pp. 758-763.

[116] C. E. Kim, Y. S. Lee, Y. H. Lim, I. Y. Noh, and S. H. Park, 'Month of Birth and Schizophrenia in Korea. Sex, Family History and Handedness', British Journal of Psychiatry, Vol. 164, No. 6, 1994, pp. 829-831.

[117] E. Fuller Torrey, 'Theories of Causation of Schizophrenia', The Jim Brownlie Memorial Lecture, 20th Anniversary Conference of the Schizophrenia Fellowship of New Zealand, 6-7 September, 1997.

[118] McGorry and Edwards, Early Psychosis Training Pack, op.cit., 1997.

[119] Ibid., Module 1, p. 30.

[120] Ibid., p. 31.

[121] Gardiner-Caldwell Communications, Influenza Bulletin, accessed July 1998, Available URL, http://www.eswi.com/bull/5/home.htm

manufacture influenza vaccines. It seems Gardiner-Caldwell have developed a public relations speciality whereby they provide promotional assistance for medical researchers which, at the same time, helps to expand the potential markets for their pharmaceutical sponsors. The Early Psychosis Training Pack should be considered in this light. But in making this consideration questions arise as to why the clinical guidelines for early psychosis 'best practice' in Australia have utilised vital material from a public relations Training Pack without explanation.

A popular hypothesis amongst schizophrenia researchers about the season of birth postulates a link between influenza infection of mothers in the second trimester of pregnancy and schizophrenia in offspring. Although this theory is quite widespread a review of the evidence shows it to be doubtful.[122] Despite the flimsy evidence some psychiatric researchers have called for an influenza vaccination programme for all women of child-bearing age as a preventive measure against mental illness.[123]

It is an interesting speculation to consider whether Gardiner-Caldwell's public relations work to expand the market for influenza vaccines might have some linkage to the specification in the Training Pack and the Clinical Guidelines of season of birth as a risk factor for early psychosis. Is it possible Gardiner-Caldwell might be using their influence with early psychosis researchers to position the season of birth hypothesis so that a perceived need for influenza vaccination of child-bearing age women can be made a part of future 'best practice' in preventive medicine for schizophrenia?

This type of public relations activity on behalf of drug companies does indeed seem to play a role in other early psychosis research. Most often, though, the public relations work is on behalf of companies that manufacture new schizophrenia drugs.

A community education programme supporting a new two-step program for early intervention in first episode psychosis at the London Health Sciences Centre, London, Ontario, is being sponsored by Zeneca.[124] The community education involves teaching doctors, parents, school teachers, college teachers and guidance counsellors how to identify the signs and symptoms of early psychosis in young people and where to direct young people for psychiatric intervention. Zeneca manufacture a new atypical neuroleptic called Seroquel (quetiapine).

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[122] Daniel R. Weinberger, 'From neuropathology to neurodevelopment', The Lancet, Vol. 346, No. 8974, 26 August, 1995, pp. 552-558.

[123] R. Livingston, B. S. Adams and H. S. Bracha, 'Season of birth and neurodevelopmental disorders: summer birth is associated with dyslexia', Journal of the American Academy of Child and Adolescent Psychiatry, Vol. 32, No. 3, May, 1993, pp. 612-616.

[124] Anonymous, 'Young Adults Experiencing Psychosis Remain Undiagnosed', Mental Health Net, accessed August, 1996, Available URL, http:// www. cmhc.com/articles/young.htm

The Western Psychiatric Institute and Clinic (WPIC) in Pittsburg, Pennsylvania is running a Program for Assessment and Care in Early Schizophrenia (PACES).[125] To facilitate this research WPIC educates primary health care suppliers and educational professionals in their catchment area about the early signs of psychosis. A part of the research in conjunction with this programme is to test the efficacy of three new atypical neuroleptics. One is a study of the long-term effects of Janssen-Cilag's drug Risperdal (risperidone). This study is funded by Janssen-Cilag. Another looks at the therapeutic efficacy and safety of Eli Lilly's new drug Ziprexa (olanzapine). This study is funded by Eli Lilly. A third study examines the outcome of switching schizophrenia treatment from conventional neuroleptics to Pfizer's new atypical ziprasidone. This study is funded by Pfizer.[126]

A recently established programme in the United States called SOS aims to increase the awareness of schizophrenia by emphasising the importance of early intervention and detection. "The SOS programme — known in full as 'SOS - Signs of Schizophrenia: What To Look For, What To Do' — was set up by the National Mental Health Association in conjunction with Janssen Pharmaceutica in the USA."[127]

EPPIC's preventive treatment centre for young people, PACE, also receives drug company funding from Janssen-Cilag.[128] This may well have paid off handsomely for the company. The EPPIC researchers have established a leadership role in early psychosis research and treatment in Australia and this was apparent in the organisation of the National Early Psychosis Project and the Clinical Guidelines that emerged from the Project. It may not be coincidental that a half page of the Clinical Guidelines is dedicated to dosage recommendations for using risperidone in first-episode psychosis.[129] The Clinical Guidelines do not extend these dosage recommendations to include other schizophrenia drugs and the recommendations for risperidone give the appearance of an official endorsement of the drug.

A further indication of the influence gained by Janssen-Cilag through sponsoring EPPIC initiatives can be found in a Resource Kit for General Practitioners which has been assembled to assist doctors "in dealing with young people at risk of Serious Mental Illness".[130] The Resource Kit outlines a plan for integrating general practitioners into the ongoing out-patient services for young people who have been given psychiatric treatment for early psychosis. The plan is modelled on the existing

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[125] Western Psychiatric Institute and Clinic (WPIC), accessed July 1998, Available URL, http://brains2.wpic.pitt.edu/paces.html

[126] Ibid.

[127] Anonymous, 'Early Detection and Intervention in Schizophrenia: The Patient's Perspective', Schizophrenia Review, Vol. 6, No. 1, January 1998, p. 4.

[128] Early Psychosis Prevention and Intervention Centre, PACE Background, Early Psychosis Prevention and Intervention Centre, accessed July 1998, Available URL, http://brains2.wpic.pitt.edu/paces.html

[129] National Early Psychosis Project, Australian Clinical Guidelines for Early Psychosis, op.cit., p. 28.

[130] Early Psychosis Prevention and Intervention Centre, Resource Kit for General Practitioners, Clyde Consulting, Yarraville, Victoria, 1998, cover page.

practice at EPPIC and involves an 18 month schedule during which responsibility for the patient's supervision is progressively transferred from psychiatric specialists to a local GP. The Resource Kit is designed to be kept as a reference book by GPs. To ensure GPs are encouraged to prescribe the sponsor's products a prominently displayed banner across the cover bears the Janssen-Cilag name and logo.

As well as this, the Second National Conference on Early Psychosis — "Realising the Potential" — organised and hosted by EPPIC at Hobart Tasmania in early September 1998, was principally sponsored by Pfizer, with additional sponsorship coming from Janssen-Cilag, Eli Lilly and Novartis (manufacturer of clozapine).[131] The conference was attended by psychiatrists and mental health workers, most of whom were either involved in already-operating early psychosis programmes, or were in the process of setting one up.

The conference was held at the Hobart casino and the foyer of the conference venue was given a carnival atmosphere by the presence of stalls set up by the four drug companies. During intervals between conference sessions barkers from the drug company stalls competed with one another for the attention of conference delegates, with public relations teams distributing literature, coffee, and numerous gifts including pens, tea-towels, writing pads and rubber balls all prominently stamped with company and product logos. A popular gift was a soft, sponge rubber brain replica from Eli Lilly. The brain is designed to be held in the hand so the ridges and crevices can be contemplatively probed and squeezed by psychiatric therapists. Eli Lilly's new atypical neuroleptic brand name, Ziprexa, is prominently stamped on each side of the brain as a reminder of the preferred form of therapy.

In the final plenary session of this conference I managed to ask a question of the assembled delegates: "Why are early psychosis programmes taking off now — and why is it happening in Australia — when there does not seem to have been a breakthrough in knowledge about the aetiology of schizophrenia and Australia does not normally lead the world in mental health initiatives?"[132]

The reaction to this question was very interesting. The delegates became animated as they questioned one another for the answer. But strangely, nobody seemed to have one. In the end my working hypothesis was left intact: i.e. early psychosis research and intervention programmes were being driven by funding and lobbying from the pharmaceutical companies that have recently launched new atypical neuroleptics onto the market.

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[131] Early Psychosis Prevention and Intervention Centre, Registration Brochure, Second National Conference on Early Psychosis, Early Psychosis Prevention and Intervention Centre, 1998.

[132] Richard Gosden, question from the floor, final Plenary Session, Second National Conference on Early Psychosis, Hobart Tasmania, 4-5 September, 1998.

The objective of these pharmaceutical companies is to expand the market for the new drugs. The size of the market for palliative treatment of the psychotic and post-psychotic stages of schizophrenia is limited by the diagnostic conventions that have been established for schizophrenia. This is the market they are entering with their new drugs. But the size of the market for prophylactic treatment of pre-psychotic schizophrenia is potentially much larger. This is the expanded market they are seeking to create. Australia is figuring prominently in this strategy because it is being used as a proving ground for the idea of preventive medicine for schizophrenia. This is in preparation for the introduction of full-scale preventive medicine campaigns in the much larger drug markets of North America and Europe.

But prophylactic treatment with neuroleptic medication, of people who have not manifested a psychological crisis, and who are currently coping, carries an enormous burden of ethical responsibility. This is because of the severe risks of drug induced diseases that are incurred by taking the new drugs. The manufacturers of atypicals are currently warning prescribing psychiatrists about these risks by including long lists of adverse drug reactions in advertisements published in psychiatric journals. There is an extraordinary range of these drug-induced diseases and sometimes the warnings are so extensive they run to two pages of extremely small type.

The more serious adverse reactions identified in the warnings, like agranulocytosis[133] and neuroleptic malignant syndrome,[134] cause sudden death. The advertisements also warn about laboratory evidence which indicates the new drugs are carcinogens[135] and mutagens.[136] Despite the claims from some quarters that tardive dyskinesia is not a problem with atypicals most of the advertisements warn that these drugs do cause the disease. An advertisement for Risperdal (risperidone) spells it out clearly under the heading of WARNINGS: "**Tardive Dyskinesia**. A syndrome of potentially irreversible, involuntary, dyskinetic movements may develop in patients treated with antipsychotic drugs. Whether antipsychotic drug products differ in their potential to cause tardive dyskinesia is unknown."[137]

Paradoxically, the manufacturers also warn about the possibility of adverse mental and behavioural reactions. Many of these psychiatric reactions are the very disorders that prophylactic treatment

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[133] Novartis (Sandoz Pharmaceuticals Corporation), Clozaril (clozapine) advertisement, Archives of General Psychiatry, Vol. 55, No. 1, January, 1998, p. 8.

[134] Janssen Pharmaceutica, Risperdal (risperidone) advertisement, Psychiatric Services, Vol. 49, No. 9, September, 1998, p. 1124.

[135] Eli Lilly and Company, Zyprexa (olanzapine) advertisement, Psychiatric Services, Vol 49, No. 3, March, 1998, p. 310.

[136] Zeneca Pharmaceuticals, Seroquel (quetiapine) advertisement, Psychiatric Services, Vol. 49, No. 3, March, 1998, p. 284.

[137] Risperdal (risperidone) advertisement, op.cit.

with the drugs is intended to prevent. In other words, instead of preventing psychosis the advertisements are warning that the new atypicals are likely to induce the condition. An advertisement published by Zeneca Pharmaceuticals, for instance, after warning about an extraordinary variety of ways their new atypical can induce ill-health, identifies "Other Adverse Events Observed During the Pre-Marketing Evaluation of SEROQUEL".[138] These include:

> abnormal dreams, dyskinesia, thinking abnormal, tardive dyskinesia, vertigo, involuntary movements, confusion, amnesia, psychosis, hallucinations, hyperkinesia, libido increased, urinary retention, incoordination, paranoid reaction, abnormal gait, myoclonus, delusions, manic reaction, apathy, ataxia, depersonalisation, stupor, bruxism, catatonic reaction, hemiplegia.[139]

A Clozaril (clozapine) advertisement also warns about the risk of a variety of drug-induced negative and positive symptoms like loss of speech, amentia, delusions/hallucinations and paranoia.[140] If treatment with atypical neuroleptics can induce psychosis, hallucinations and delusions, as is frankly being admitted in advertisements for the drugs, questions most definitely arise about the application of these drugs as prophylactics against psychosis. In the long term, will prophylactic treatment actually increase the incidence of psychosis rather than reduce it? This is a question that does not seem to have been considered in the literature.

Another obvious question to be addressed concerns how to interpret the significance of transition to psychosis by a person who has been receiving prophylactic treatment. Does such an event indicate accuracy in the diagnosis of prodromal symptoms, and ineffectiveness in the prophylactic treatment to prevent the psychosis? If so then perhaps it might encourage the prescription of increased doses of prophylactic drug treatment for other patients.

But on the other hand such an event could simply indicate an adverse drug reaction by a person with a false/positive diagnosis. If this were the correct interpretation then it would be better to take other patients off their prophylactic medication altogether, rather than increase the dosage. Once again these lines of discussion are not arising in the literature.

Perhaps the most insidious of the ethical burdens for the promoters of the prophylactic use of atypicals comes from the growing body of evidence that withdrawal from some of these drugs can sometimes cause a psychotic reaction. It seems that the brain chemistry of some people treated with atypicals is changed in a way that makes them dependent on continued treatment. When atypical

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[138] Seroquel (quetiapine) advertisement, op.cit.
[139] Ibid.
[140] Clozaril (clozapine) advertisement, op.cit.

neuroleptic treatment is withdrawn from them they experience an immediate psychotic reaction that can only be rectified by recommencement of treatment.[141]

The ethical burden for psychiatrists treating the supposed prodrome of schizophrenia will include resisting the temptation to interpret psychosis induced by atypical neuroleptic withdrawal as merely being evidence that the person was correctly diagnosed in the first place. Unethical psychiatrists may be tempted to argue that it was the prophylactic treatment which, up to the point of withdrawal, prevented the person from entering psychosis. In this way the original diagnosis and prophylactic treatment could easily be vindicated, when in fact they might both be at fault.

Conclusion

There are at least two methods by which advocates of the medical model could convincingly demonstrate the existence of an identifiable pre-psychotic phase of schizophrenia. The first would be to undertake diagnostic interviews with a large sample of adolescents and predict who would later become psychotic, and who would not. This could be done without informing the people involved of the particular prediction that had been made for them and without offering treatment to those people who were thought to be heading for psychosis. Using such facilities as electoral rolls and admission records to mental hospitals and psychiatric units the predictions could then be tested over extended time periods. But there is no indication that this relatively simple and obvious method of testing theories about pre-psychotic symptoms has been utilised anywhere in the world.

The second approach would be to detect and treat all the people in a given catchment area who manifested the pre-psychotic signs. This approach would seek to demonstrate, through early treatment, a significant reduction in the number of people who become psychotic in that area. Falloon's Buckingham Project claimed to have done this but the flexibility of his definition of psychosis cast doubt on the outcome.

As things stand the early psychosis projects that have been put into operation are largely of the same type as the Buckingham Project. They involve both detection and treatment and thereby attempt to reduce the incidence of psychosis in the catchment areas in which they operate. However, there is no certain evidence that this objective has been achieved anywhere, or indeed, is achievable. The efficacy of detection and treatment methods for pre-psychosis remains largely hypothetical at this stage. This means that the various programmes that have been put into effect are largely experimental.

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[141] J. K. Stanilla, J. de-Leon and G. M. Simpson, 'Clozapine withdrawal resulting in delirium with psychosis: a report of three cases', Journal of Clinical Psychiatry, Vol. 58, No. 6, June 1997, pp. 252-255.

This raises concerns about human rights, specifically in relation to informed consent. Coercion is readily apparent in the area of early psychosis intervention and two-thirds of the patients being treated by EPPIC's first-episode psychosis clinic are involuntary patients.[142] Although at this time it is not apparent that people are being treated involuntarily for prodromal symptoms, the possibility is already being discussed in the literature[143] and it is likely to happen in selected cases in the future.[144]

Problems with symptomatology and treatment make it unlikely that pre-psychosis detection and intervention programmes will ever deliver the kind of unequivocal social and community health advantages that are generally expected from preventive medicine campaigns. This means that the extension of the definition of schizophrenia into a prodromal phase is unlikely to provide further support for the medical model of schizophrenia.

However, whereas programmes of early detection and intervention might not give further support to the medical model for schizophrenia, at the same time, it is entirely possible that over time they might actually enhance the plausibility of the two competing models — perhaps the myth-of-mental-illness model more than the mystical model. When psychiatrists openly refer to pre-psychotic indicators as being 'putative', and then proceed to intervene in the lives of people who are thought to manifest them, treating them with the same potent neuroleptics that are used on supposedly full-blown schizophrenia, then the argument that schizophrenia is just a psychiatric myth seems more persuasive.

This point is emphasised by considering the commercial advantages that are likely to accrue to pharmaceutical companies from an expansion of the definition of schizophrenia to include a pre-psychotic phase. A preventive medicine campaign based on the type of prodromal symptoms and risk factors specified in the Australian Clinical Guidelines for Early Psychosis potentially defines the whole generation of young people as being at-risk and in need of treatment. If proponents of the M-M-I model can demonstrate that pharmaceutical marketing strategies are indeed the primary motivating force behind the campaign for prevention of schizophrenia then it will make it easier to argue that the whole medical model for schizophrenia is a myth that has been invented to serve special interests.

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[142] Paddy Power, Early Psychosis Prevention and Intervention Centre, in answer to a question after presentation of paper entitled, An Analysis of the Initial Treatment Phase and Follow-Up of First Episode Psychosis Patients, Second National Conference on Early Psychosis, Hobart Tasmania, 4-5 September, 1998.

[143] Yung and McGorry, 'Is pre-psychotic intervention realistic in schizophrenia and related disorder?', op.cit., p. 802.

[144] Patrick McGorry, Director of the Early Psychosis Prevention and Intervention Centre, Personal Communication, at the Second National Conference on Early Psychosis, Hobart Tasmania, 4-5 September, 1998.

Similarly, when magical thinking is found to be the most common prodromal symptom that leading researchers in the field are treating then the link between schizophrenia and mystical inclinations would also appear to strengthen. But the use of magical thinking as the pre-eminent symptom can also lend support to the myth-of-mental-illness model. This is because the EPPIC psychiatrists who are treating patients for this symptom have prior knowledge from their surveys that magical thinking is also experienced by the majority of normal young people. They seem to be ignoring evidence they have themselves collected and published that magical thinking is not necessarily a sign of mental pathology at all.

Indeed, the pseudo-authoritativeness characterising much of the literature regularly demonstrates a lack of reflection on the part of early psychosis researchers about the superficial nature of their claims. A good example of this can be found in the Early Psychosis Training Pack. Under the heading of "How to achieve early recognition — Triggers for considering psychosis or pre-psychosis"[145] the Training Pack advises doctors dealing with adolescents and young people to be sure of "[m]aintaining a high index of suspicion - signs to look out for."[146] This advice is followed by the 16 item list of "Signs and Symptoms", the first of which is "suspiciousness". This juxtapositioning of the idea of suspicion, first as an efficiency measure for diagnosticians and then as a sign of pathology in patients, begs the question: Is it credible for psychiatrists to claim that "suspiciousness" in young people is a sign of serious mental illness when the same psychiatrists argue that clinicians should cultivate an attitude of suspicion in themselves as an efficiency measure?

There is a certain degree of irony here, where suspicion is encouraged to combat suspicion, which apparently escapes the authors of the Training Pack. But the contrariness raises an important question as to whether suspiciousness and the other putative signs and symptoms are correctly judged to be indicators of mental pathology. From the M-M-I perspective the duplicitous use of suspicion could easily be interpreted as evidence of using psychiatry for social control. That is, suspicion is a worthy quality when it is used as a tool of efficiency by a person with authority but it becomes a sign of pathology when it is found in a person of low status or in a person who is challenging authority.

The EPPIC researchers have cited a 1938 article by D. Ewen Cameron as their original authority for believing that "suspiciousness may predict subsequent psychosis".[147] This is itself a rather suspicious source. Cameron is the Canadian psychiatrist who gained notoriety in the 1980s after it

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[145] McGorry and Edwards, Early Psychosis Training Pack, op.cit., P.9.
[146] Ibid.
[147] Yung et al., 'Monitoring and Care of Young People at Incipient Risk of Psychosis', op.cit., p. 286.

was revealed he had undertaken cruel and unethical experiments on his patients during the 1950s and 1960s with funding from the CIA.[148]

Using a deep sleep technique combined with multiple daily assaults of ECT Cameron attempted to cure schizophrenia by erasing all memory of self from his patients' minds. The CIA was apparently interested in utilising these techniques in espionage work. In 1988 the CIA acknowledged complicity in Cameron's work when they arranged to pay $750,000 in compensation to some of the victims.[149] Cameron's exploits were the subject of a 1979 book by John Marks entitled The Search for the Manchurian Candidate.[150]

Cameron is perhaps the most widely discredited psychiatrist of all time and contemporary psychiatric researchers who cite him as a source of authority for their own ideas demonstrate, at the very least, a deficiency of judgement. Nevertheless, the Cameron-inspired symptomatology has been incorporated into the Australian Clinical Guidelines for Early Psychosis where suspiciousness is given as the leading symptom of pre-psychotic schizophrenia.[151]

The deficiency of judgement regarding Cameron extends beyond merely adopting his suggestion about the use of suspiciousness as a symptom. Proponents of early psychosis repeatedly cite Cameron as the originator of the whole concept for early detection and intervention programmes for schizophrenia.[152] Patrick McGorry, the Director of EPPIC, even quoted Cameron to lead his introductory essay to a June 1998 early psychosis supplement he edited of the British Journal of Psychiatry:

> "Very early schizophrenia still constitutes a relatively unexplored territory. Entry into this territory calls for new ideas on the social problems involved in bringing the early schizophrenic promptly under treatment, or where the treatment should be carried out and in what it should consist." D. Ewen Cameron (1938)[153]

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148 Report of the Royal Commission Into Deep Sleep Therapy, Justice J. P. Slattery, Royal Commissioner, New South Wales Government, Sydney, Volume 2, 1990, pp. 48-58.

149 Peter Breggin, Toxic Psychiatry, Fontana, London, 1993, p. 250.

150 John Marks, The Search for the Manchurian Candidate, W. W. Norton and Company, New York, 1979.

151 National Early Psychosis Project, Australian Clinical Guidelines for Early Psychosis, op.cit., p. 13.

152 See for example, Alison R. Yung et al., 'Prediction of psychosis: A step towards indicated prevention of schizophrenia', British Journal of Psychiatry, Vol. 172, Supplement, June 1998, pp. 14-20. and Richard Jed Wyatt et al., 'First-episode schizophrenia: Early intervention and medication discontinuation in the context of course and treatment', British Journal of Psychiatry, Vol. 172, Supplement, June 1998, pp. 77-83.

153 D. E. Cameron, 'Early schizophrenia', American Journal of Psychiatry, 95, 1938, pp. 567-578. Quoted in Patrick McGorry, 'Preventive strategies in early psychosis', British Journal of Psychiatry, Vol. 172, Supplement, June 1998, pp. 1-2.

Referring to the origins of the idea of pre-psychotic treatment McGorry went on to say that "[t]his form of preventive intervention, [was] originally fore-shadowed by Cameron."[154] In the 1938 article Cameron writes enthusiastically about the effectiveness of "the newer therapeutic techniques used in schizophrenia". Cameron's 1938 article is followed by a commentary from the leading authority on schizophrenia at the time, Harry Stack Sullivan. Unlike McGorry, and without foreknowledge of Cameron's future notoriety, Sullivan demonstrated disgust with Cameron's proposal and issued a strong rebuttal.

> I would be very deeply disturbed if, as is implied by the last speaker [Cameron], people who show signs of personality disorders, early mental disorder of an indeterminate kind, were to be rushed through treatment with insulin, metrazol and camphor on the chance that they might otherwise have developed schizophrenia. I privately have a suspicion that might have a distinctly unfavourable effect on the general intelligence level and so on of the community.
>
> What does it mean that a person will have schizophrenia which can be detected by the intelligent layman months to years before the schizophrenia appears? In seven and half years of exclusive preoccupation with the schizophrenia problem I was unable to put my finger on anything sufficiently simple and obvious to service this purpose.[155]

Although in the text of his article Cameron did not specify the treatments he had in mind for early psychosis some of the references he gave clearly confirm Sullivan's suspicion that insulin shock was intended. In closing the Discussion of his article, following Sullivan's rebuttal of his ideas, Cameron buckled and lamely reversed his former position: "I don't think that it is any way feasible to consider at the present time treatment of persons suffering from early non-specific symptoms by means of pharmacological shock treatment."[156]

This exchange between Sullivan and Cameron indicates that it was Sullivan's strong rebuttal that made pharmacological shock unfeasible as treatment in the late 1930s. If it is now feasible to use the latest form of pharmacological shock — atypical neuroleptics — on the same symptoms, then perhaps it is only because there is no figure in the psychiatric profession, like Sullivan, with sufficient stature and a commensurate level of social conscience, to mount the necessary protest.

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[154] McGorry, Ibid.

[155] Harry Stack Sullivan, 'Discussion', in D. Ewen Cameron, 'Early schizophrenia', American Journal of Psychiatry, 95, 1938, p. 579.

[156] D. Ewen Cameron, 'Discussion', in 'Early schizophrenia', American Journal of Psychiatry, 95, 1938, p. 581-582.

It is not clear whether McGorry and other early psychosis proponents use Cameron as an authority out of ignorance, indifference or admiration of his unsavoury reputation.[157] But it is certain that their source of inspiration, and their judgement in revealing it, are both unsound. The linking of Cameron's name with a government sponsored preventive medicine campaign for schizophrenia can only strengthen arguments for the myth-of-mental-illness model in the long run.

[157] The question of whether they are ignorant, indifferent or in admiration of Cameron warrants further investigation as it provides a simple method of evaluating the professional integrity of the psychiatrists who are promoting preventive medicine strategies for schizophrenia. When McGorry quoted Cameron to lead his British Journal of Psychiatry article he cited D. Ewen Cameron beneath the quotation, as I have reproduced it above. The end-note reference, however, only identified D. E. Cameron, as his name appeared on his original 1938 article. D. Ewen Cameron is the distinctive form of his name by which Cameron's post World War II career was identified, first as an eminent psychiatrist and then as an unethical CIA-sponsored experimenter. McGorry's unnecessary use of the distinctive D. Ewen indicates that he knew about the post World War II prominence of the author and wanted to associate his own ideas with a psychiatric predecessor who had a sufficient reputation to lend them authority. Before his fall from grace Cameron was one of the most eminent psychiatrists of his time having served as President of the American Psychiatric Association and as first President of the World Psychiatric Association. But it is not clear to which aspect of Cameron's prominence McGorry wished to associate himself. In Toxic Psychiatry (p. 251) Breggin observed that the adverse publicity about Cameron "focussed mostly on the CIA funding rather than on the most scandalous fact of all - that Cameron and his brutalities, although well known throughout the profession, were never criticised by mainstream psychiatry". A similar criticism of the psychiatric profession for acquiescing to Cameron's work was made by Canadian QC George Cooper (quoted in Royal Commission Report, Op. Cit., p. 56.). The NSW Royal Commission into Deep Sleep Therapy devoted ten pages to examining Cameron's work and his link with the CIA (pp. 48-58, Vol. 2). This was done in order to determine whether Dr. Harry Bailey, the main subject of the Royal Commission Inquiry, was also linked to the CIA through association with Cameron. No significant connection was found but it is interesting to note that references to Cameron persistently misidentified him by the name, Dr. Ewan Cameron, throughout the entire twelve volume report. This is not consistent with any of the names by which he was normally known: i.e. D. E. Cameron; D. Ewen Cameron; or Donald Ewen Cameron. The misidentification of Cameron could simply be a typographical error in the final report. Even so, it seems to have led to confusion in the historical record whereby the CIA-sponsored, human rights abusing Cameron now appears to be a different Cameron to the former eminent leader of international psychiatric organisations. This confusion was perpetuated in a 1991 book, (Brian Bromberger and Janet Fife-Yeomans, Deep Sleep: Harry Bailey and the Scandal of Chelmsford, Simon and Schuster, Sydney, 1991, pp. 10-11.) which drew heavily on the Royal Commission report as a source and repeated the misidentification of Cameron as Dr. Ewan Cameron.