# Schizophrenia: A review of current research and thinking

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**Introduction**

Schizophrenia is the major mental illness of our time. It was first described by Kraepelin (1896) as “dementia praecox” and later given the name “schizophrenia” by Bleuler in 1911. It is a condition characterised by disturbances of thought, perception and a blunting of affect. These disturbances “involve the most basic functions that give the normal person a feeling of individuality, uniqueness, and self-direction” (WHO 1992). In 1959 a German Psychiatrist identified what he considered to be first rank symptoms of schizophrenia (Schneider 1959). Schneider grouped the collection of symptoms into three main categories, namely, auditory hallucinations, passivity experience and delusional thinking. Schizophrenia sufferers experience hallucinatory “voices” which may either provide a running commentary on one’s movements or instruct the person to carry out certain tasks. Some sufferers experience voices which are derogatory or insulting. Passivity feelings refer to those feelings, thoughts or

behaviours which the individual experiences as being under the influence of a third party. Delusional thinking arises from perceptions which may be distorted. Delusional thinking is often insightless and unamienable to reason. Although these symptoms are no longer used as the sole diagnostic aid Schneider's categorisation of the symptoms gives a glimpse of the level of disturbance those with schizophrenia experience. Schizophrenia however is also associated with a wide range of other symptoms including social withdrawal, incongruent affect and thought disturbances, which contribute to the devastating effects this illness can have on the person.

The cost of schizophrenia in both human terms and in its cost to the nation is immense. As well as the symptoms described above loss of social contacts and career prospects often go hand-in-hand with the illness. Davies and Drummond (1994) estimated the cost of schizophrenia to the country at £397m, or 1.6% of the total health care budget.

This paper will attempt to review current developments in the classification, causes and treatment of schizophrenia and hopefully draw some conclusions from the literature on the future developments in the treatment of this illness.

## Classification and Diagnostic Criteria

The classification of schizophrenia has been described mostly by psychiatrists. While there are numerous diagnostic texts for mental illness many are used solely for the purposes of research and are seldom seen in clinical practice. This paper will therefore concentrate on those used in clinical practice. There are currently two main texts which are used in clinical settings to classify, and describe for diagnostic purposes, mental illnesses including schizophrenia. These are namely the International

Classification of Diseases-10 (ICD-10) and the Diagnostic and Statistical Manual of Mental Disorders -IV (DSM-IV).

The International Classification of Diseases-10 (ICD-10) provides diagnostic categories for mental illnesses including schizophrenia (WHO 1992). Schizophrenia is described as "a syndrome with a variety of causes, and a variety of outcomes, depending on the balance of genetic, physical, social and cultural influences". ICD-10 describes a number of sub-categories of schizophrenia. These are paranoid, hebephrenic, catatonic, undifferentiated, residual, simple schizophrenia and post-schizophrenic depression.

According to ICD-10 paranoid schizophrenia is the most common type of schizophrenia in most parts of the world. It is characterised by "stable, often paranoid, delusions usually accompanied by (auditory) hallucinations and perceptual disturbances" (WHO 1992). Hebephrenic schizophrenia usually commences in late adolescence or early adulthood and is considered to have a poor outcome. It is characterised by affective changes, fleeting delusions and hallucinations, irresponsible or unpredictable behaviour (WHO 1992). Catatonic schizophrenia is rarely seen in industrial countries for reasons which are as yet unknown. It is characterised by disorders of movement and volition. As well as episodes of extreme stupor, sudden violent excitement has been described. Of the other sub-types of this illness residual schizophrenia is probably the most common. It is characterised by prominent negative-type symptoms which follow from an early stage of the illness which included florid symptoms. The person should have had a period of at least a year when the intensity of these florid symptoms have been substantially reduced (WHO 1992). ICD-10 suggests a one month period between onset of symptoms before a diagnosis of

schizophrenia is arrived at. This is to help distinguish schizophrenia from acute psychotic episodes which may resolve without treatment within 2-3 weeks.

The Diagnostic Statistical Manual of Mental Disorders-IV (DSM-IV) categorises schizophrenia for the purposes of diagnosis (American Psychiatric Association 1994). Diagnostic criteria within DSM-IV differs slightly to that of ICD-10. DSM-IV criteria advise that signs and symptoms of schizophrenia must be present for "a significant portion of time during a 1 month period with some signs of the disorder persisting for at least 6 months". This is in contrast to the ICD-10 criteria which requires symptoms to be present for a one month period. DSM-IV descriptions of subtypes of schizophrenia are largely similar to that of ICD-10. It describes 5 main subtypes namely Paranoid, Disorganised, Catatonic, Undifferentiated and Residual types. Descriptions of the subtypes are largely similar in both texts. The similarities are a result of wide international consultation between the two texts and the desire to have similar descriptions to facilitate compatibility for research purposes.

**Epidemiology**

Opit (1994) discusses the methodological problems associated with epidemiological studies and warns about "undeserved causal hypothesising". These problems occur because epidemiology depends almost entirely on inductive reasoning and often, Opit (1994) suggests, the "limits of inference", which are highly dependant on the method employed, are ignored entirely. Jabelensky (1993) has also raised the issue of methodological difficulties particularly in relation to schizophrenia. These difficulties centre around the "still hypothetical nature of the disease concept of schizophrenia

itself and the lack of validated objective disease markers". The following brief review is therefore presented with these points in mind.

## Incidence

The World Health Organisation's ten-country study into the incidence and course of schizophrenia (Jabelensky et al 1992) found that when using a homogenous computerised diagnostic criteria to identify first cases of the illness a range of 0.7 -1.4 per 10,000 population was apparent. This showed that within the 24 month period of this study there was little variation across study sites. The authors concluded that the "rates of manifestation of the schizophrenic syndrome at the population level were similar across cultures". There have been some suggestions in the professional press that the incidence of schizophrenia has been reducing over recent years. Der et al (1990) suggested that schizophrenia might be "disappearing" based on a 50% reduction in the admission rate to hospitals for schizophrenia over a 20 year timescale. There are however obvious problems with such a conclusion. Given the large scale hospital closures and government policy of care in the community it is likely that admissions to hospital for all diagnoses will have shown similar reductions. The issue of classification and diagnosis is also an important influencing factor. If the description and diagnosis of schizophrenia had remained static during the period examined then possibly more could be made of the findings. This of course has not been the case. Classification and diagnosis of schizophrenia for research is beset with problems. Different hospitals use different criteria and the criteria contained within the main classification systems have themselves changed numerous times during the last 20 years. In fact Taylor and Abrams (1978) suggested that prevalence rates have remained stable with "only diagnostic habits showing change". Harrison et al (1991)

found in their study in the Nottingham region "no significant decline in first contact rates" and suggested that these findings may be a result of more assertive case finding which is a fundamental part of the services in the area studied. Hypothesising as to why rates of schizophrenia in Nottingham may be showing increases when subtypes are grouped together Harrison et al (1991) suggest that immigration may be responsible. They report findings from unpublished research conducted by a social anthropologist attached to their teams which showed that 40% of newly identified cases of schizophrenia in their area were either born outside Britain or were the offspring of an immigrant. They conclude with a further theory that biological environmental factors may conceivably be responsible. It would seem that Opit's (1994) warning about "undeserved causal hypothesising" could have usefully been heeded by Harrison and colleagues.

## Age and Sex

The World Health Organisation's study (Jabelensky et al 1993) found there to be an excess of men in the age group 15-24 years, when looking at first contacts in schizophrenia, but that women were over-represented in the age group 15-54 years. This study concluded that the life-time (15-54 years) risk for the illness was similar among both sexes. Haffner et al (1993) explored the influence of age and sex on the onset of schizophrenia in a German sample of 267 people with schizophrenia. The mean age at onset differed by three to four years in this sample with the age distribution showing a steep increase up to 25 years in males and a second peak in women between the ages of 45 to 79 years.

Sham et al (1994) in their study of 195 people with functional psychosis examined the risk of developing schizophrenia in relatives of sufferers and the relationship to age at onset and gender. They found that the lifetime risk of developing schizophrenia in first degree relatives were 5-10 times higher than population risks based on previous reported research. In particular relatives of women who had schizophrenia and who had developed the illness before 22 years had a higher risk than relatives of men with schizophrenia or those who had a late onset of the illness (Sham et al 1994).

Ethnicity

There has been some interest in the relationship between ethnicity and mental health for some time. Some studies have shown increased rates of mental illness among immigrant groups (Stopes-Roe and Cochrane 1980) while others have demonstrated increased rates of diagnosis of schizophrenia among Afro-Caribbean's (Dean et al 1981). More recently Harrison at al (1988) reported a tenfold increase in first contact rates for Afro-Caribbean's with schizophrenia in the Nottingham area. However these figures were based on a rather small sample (n=42) of Afro-Caribbean's within the study. A more substantial study was conducted by Wessley et al (1991) using a case register for the Camberwell area of London for the years 1965 to 1984. They demonstrated an increased relative risk among Afro-Caribbean's for developing schizophrenia and showed that this risk had been increasing over the years studied. Jabelensky (1993) advises caution when considering findings on rates of schizophrenia in relation to ethnicity. He points out that there is little "validated population incidence data" available on the size and age structure of, for example, the Afro-Caribbean population in Britain. Jabelensky (1993) also notes that comparison with country of origin is required when examining schizophrenia and ethnicity and the

presence of conflicting data makes this difficult to do. The hypothesis that the increased rates of schizophrenia are due to the readiness of psychiatrists to diagnose black patients as schizophrenic on the basis of their behaviour has also been raised. It has been suggested for instance that it is this readiness which is responsible for the increase in rates of schizophrenia among Afro-Caribbean's. Bebbington et al (1994) examined ethnicity and mental illness in their study and were unable to detect such a link.

## Aetiology

In recent years there has been many advances in our knowledge into the aetiology of schizophrenia. While no single causative agent has been identified there remains many new insights into the working of the human brain in general and specifically the brain physiology of those with schizophrenia. It appears now that most authors are in broad agreement that the disorder is developmental in origin (Sharma and Murray 1993). Aetiological factors have been identified in three main areas. These are genetic factors, brain abnormalities and environmental factors.

### Genetic Factors

A large number of studies exploring the causative factors in schizophrenia have examined the influence of genetics on incidence. Some of these studies have used research on twins as one method of exploring the impact of genetics in schizophrenia. In their study comparing single hospital design twin studies with studies using national twin registers, Walker et al (1991) suggested concordance rates of 41% for monozygotic twins and 15 % for dizygotic twins. These rates are lower than previous studies mainly because studies based on single hospital twin registers are, the authors

suggest, vulnerable to bias. Sharma and Murray (1993) cite the findings of a Norwegian twin register study which suggested that "paranoid schizophrenia shows a lower monozygotic-dizygotic concordance ratio than non-paranoid schizophrenia". This raises the question, not yet answered, of whether there are different levels of predisposition in the subtypes of schizophrenia.

Molecular geneticists have used advances in DNA technology to investigate schizophrenia. A number of studies have examined individual chromosomes in those diagnosed with schizophrenia. Hallmayer et al (1992) have excluded the serotonin receptors on chromosome 5 as a causative factor in schizophrenia while Gill et al (1993) has similarly ruled out a portion of chromosome 11. Both serotonin and dopamine receptor genes have been linked with schizophrenia but a number of studies have now excluded some of these (Sharma and Murray 1993). Sharma and Murray (1993) report findings from a large number of studies which are screening whole chromosomes. These have so far excluded chromosome 12 and regions of chromosomes 11, 15, 19 and 22. The authors conclude their review with the view that a genetic susceptibility to schizophrenia may result from "a disadvantageous neurodevelopmental consequence of the normally advantageous response to viral infection". They do not however provide evidence for this theory.

It appears that among those researching the causes of schizophrenia there remains an optimism that a genetic cause will be identified. Any other conclusion would seem unlikely given that many depend on such conclusions to finance further research.

Brain Abnormalities

Separating out genetics from abnormalities of the brain is an artificial device which is used in this paper for the purposes of clarity. In reality they are not so easily separated.

A theory which has gained prominence suggests that abnormal neuroanatomy is a result of genetically linked vulnerability possibly during gestation (Lewis and Murray 1987). Cannon et al (1993) found that cortical and cerebellar abnormalities in genetically at risk subjects were correlated with degree of genetic risk. That is, the incidence of abnormality in these areas was predictive of increased genetic loading for schizophrenia in the subjects examined.

Other researchers have looked at the area of brain changes in adolescence. It has been shown that what is referred to as "synaptic pruning" occurs normally in adolescence (Huttenlocher 1979). Hoffman and McGlashan (1993) now offer a hypothesis which suggests that in vulnerable subjects this synaptic pruning somehow goes beyond its normal developmental limits. This particular hypothesis remains to be proven.

McGlashan and Johannessen (1996) further suggest that "a major neurobiological alteration" occurs with overt psychosis and that this alteration is "a neurologically deteriorative one". They support this hypothesis by use of indirect links with the pathophysiological deterioration seen in schizophrenia.

Enlarged ventricle size in schizophrenia has been noted by some studies but there remains some variation in the prevalence rates found in these studies. Van Horn and McManus (1992) reviewed 39 studies, using meta-analysis, of ventricular size in schizophrenia which used the ventricle to brain ratio (VBR). Enlarged ventricle size had been taken to suggest that in schizophrenics there is a corresponding decrease in grey matter. Van Horne and McManus appear to concur with this opinion although their analysis showed that their was less difference between controls and those with schizophrenia when variables such as diagnostic criteria and methodological problems

were taken into account. They conclude therefore that the use of ventricle to brain ratio in those with schizophrenia while interesting does not provide a criterion for schizophrenia.

## Environmental Factors

The influence of environmental factors is an area which has received investigation in the pursuit of causative elements in schizophrenia. Agents in the environment as causative factors in schizophrenia are primarily thought to act during gestation. An excess of late winter and early spring births in those with schizophrenia has been demonstrated repeatedly. This has prompted researchers to develop numerous theories as to why this should be the case. Many theories find favour with researchers and the influence of viral agents such as influenza epidemics attracts much interest. Sham et al (1992) studied influenza deaths and births of schizophrenics for each month between the years 1939 and 1960. They found that the greatest risk for developing schizophrenia in later life occurred for those people who were between the third and seventh month of their gestation period. In more recent studies this association has not been shown. Cannon et al (1996) traced a cohort of individuals exposed to the 1957 influenza epidemic during gestation (n=238) and compared them to non-exposed controls (n=287) for risk of schizophrenia in adult life. They found no increased risk for those exposed to influenza during gestation but did find an increased risk of affective disorders. In another recent study Hettema et al (1996) sought to explore the hypothesis that there may be an increased familial risk in relatives of people with schizophrenia born in the seasons earlier identified as high risk. This is based on the theory that relatives of people with schizophrenia may have inherited a susceptibility

to either a seasonal viral or toxic agent which might predict a higher increased risk of developing the illness in later life. Hettama et al (1996) were unable to show such a risk and conclude that season of birth did not, in their sample, identify people with schizophrenia with particularly high, or low, familial vulnerability to the illness. The conflicting results of investigations into the relationship between prenatal influenza and the risk of schizophrenia prompted Sharma and Murray (1993) to suggest that this relationship "remains at the level of epidemiological association without a satisfactory explanatory mechanism". It may be that these conflicting results are due to methodological problems. Studies which are more rigorous in their approach appear to report associations which are statistically nonsignificant. For instance Cannon et al (1996) report no increased risk following exposure to prenatal influenza. This finding was arrived at following examination not only of all available medical records and population data but also by follow-up and blind interviews of mothers of both subjects and controls. They report a large attrition rate (only 54% of the sample n=238 could be traced) and speculate as to whether this may have been caused by those who develop schizophrenia being harder to trace. However since their study interviewed mothers and was supplemented by case register data they conclude that this would have had little effect on their study.

**Approaches to treatment and rehabilitation**

There are currently two main approaches to the treatment and care of people with schizophrenia which attract most interest within the field of mental health. These are the psychopharmacological approach and the psychosocial approach. Although they will be considered separately here in reality it is probably true to say that people with schizophrenia often receive a combination of the two with only the emphasis

differing. It has to be acknowledged however that this combination may "yield only modest gains in chronic schizophrenia" (Marks 1992).

It is to be acknowledged that there exists a number of models of care delivery which will not be explored here. These include hospital-based versus community-based care which has been studied by Marks et al (1994), case management models (Bachrach 1993, Thornicroft et al 1993) and the use of cognitive interventions to allay specific symptoms of schizophrenia (Chadwick and Birchwood 1994)

## Psychopharmacology

Recent years have seen the development of newer chemotherapy for treating schizophrenia. Traditionally neuroleptics have been used to treat schizophrenia and since the development of long-acting depot injections these have been the treatment of choice for many prescribers (Dixon et al 1995). In recent times however the re-emergence of Clozapine and the development of Risperidone (Umbricht and Kane 1995) have added a new element to the treatment of schizophrenia.

Clozapine was initially manufactured in the early 1959 but following a number of deaths thought related to its neutropenic effects it was withdrawn from the market. Its' re-emergence is a result of the increasing demand for more effective drug treatments and new safeguards to check regularly the white cell count of those taking the drug. It is seen by some prescribers as the drug of choice in what has been termed treatment-resistant schizophrenia. Some evidence also exists for its efficacy in those with negative symptoms. Fewer extra-pyramidal side effects have been noted with Clozapine and Buchanan (1995) notes that Clozapine has marked anticholinergic effects and causes less extra-pyramidal side effects than conventional anti-psychotics. In his review of studies which examined the efficacy of Clozapine over conventional

anti-psychotic medications in acute episodes of schizophrenic illness Buchanan (1995) reports that most studies reviewed reported "Clozapine to be equally or more effective" than traditional anti-psychotics. Buchanan (1995) however acknowledges the small sample sizes of the studies reviewed and suggests that definitive conclusions are therefore unable to be drawn from the results presented. The difficulty arises in comparing Clozapine to traditional medications. That is, unless patients have experienced adequate trials on other neuroleptics before commencing Clozapine it is difficult to conclude that it is more effective. His statement that Clozapine is "equally or more effective" also bears some examination. If Clozapine is "equally" effective then there may be strong reasons for not prescribing it. Clozapine not only costs much more than conventional anti-psychotic medication but has potential serious and life-threatening side effects. It may be more prudent to invest the substantial cost of prescribing a drug which has only equal benefits to patients in other forms of treatment. It would appear from Buchanans' (1995) review that more rigorous investigation of the effectiveness of this treatment is required.

## Psychosocial Interventions

The use of psychosocial interventions in treating people with schizophrenia is largely based on a stress-vulnerability model. The theory underpining this model is that people with schizophrenia are liable to a relapse of their illness when exposed to increasing levels of stress in their daily lives. Psychosocial intervention is therefore targeted at reducing these stresses through a number of methods including family education about the illness, assessment of needs of each family member, health education, stress management programmes and attempting to increase the social and personal functioning of those within the family. The stresses within families with a

member who has schizophrenia have been studied and have given rise to the term Expressed Emotion (EE). Assessments of EE are derived from key scores in the areas of critical comments, hostility and emotional overinvolvement obtained by frequency count during a standardised interview (Kavanagh 1992). In a useful review of this area Kavanagh (1992) presents data from 26 studies into the predictive quality of high-EE scores on relapse rates in schizophrenia. The median relapse rate for these studies with a total sample size of 1323 was 48% in the high-EE group which was more than double the relapse rate in the low-EE group. Kavanagh (1992) suggests that the phenomenon is "as valuable clinically as medication" as a predictor of relapse. Tarrier et al (1988) used EE to randomly allocate families into groups for treatment using either routine, educational or behavioural methods. They found that relapse rates were higher for patients of families with high-EE who were not receiving effective intervention i.e. high-EE education and routine treatment. They also demonstrated that those patients in high-EE families receiving a behavioural intervention had a significantly lower rate of relapse

Some studies have sought to demonstrate more general gains from psychosocial interventions. In a quasi-experimental study Brooker and colleagues trained CPN's (n=9) to deliver psychosocial interventions to people with a diagnosis of schizophrenia and their families (Brooker et al 1993). The study consisted of an experimental group and a matched control group. This study did not measure EE or seek to reduce it but instead sought "to improve the quality of life for such families and enhance their ability to solve problems". The study was able to demonstrate improved General Health Questionnaire (GHQ) scores for relatives within the experimental group. The experimental group also showed signs of symptom improvement in the areas of anxiety, depression and retardation. There were no other

significant differences between the groups in relation to symptom improvement. Social adjustment scores improved in the experimental group but not in the control group. While the results of this study provide encouraging reading for the concept of providing people with schizophrenia and their families psychosocial interventions, they should be read with caution. The sample size of the study was small, 54 families initially being the target sample but only 30 families completed the study. The experimental group made up just 17 families. The generalisability of the findings are therefore limited. The improvement in symptoms may also be accounted for by a "tendency to reduction" in medication levels within the experimental group. The authors had difficulties in recruiting families for the research and the cost of training CPN's (approx. £600 each in 1990) may also have placed some limitations on the size of the study. The findings suggest that further research into this area would seem a worthwhile if expensive option.

**Course and outcome**

Jabelensky (1993) optimistically concludes from a review of international studies on schizophrenia that "progressive deterioration is not at present the central tendency in the course and outcome of schizophrenia". He offers a number of reasons for this none of which alone offers convincing evidence but taken together suggest that remission from the illness may be a realistic outcome. Jabelensky (1993) suggests an "overall benefit from the transfer to community care" for those with schizophrenia. In a study investigating the use of hospital-based care versus home-based care Marks et al (1994) reported improved symptoms and social adjustment in the seriously mentally ill. The patients in contact with this service were not exclusively those with schizophrenia but they did make up a substantial proportion. Test (1990) had

previously cautioned however that long-term support would have to continue or gains would be lost and despite continued input Marks et al (1994) reported that most gains were lost after 20 months.

Some studies have demonstrated that remission of illness varies across cultures. Leff et al (1992) and Jabelensky et al (1992) provide data which shows that people with schizophrenia from non-Western cultures have earlier remissions which last longer. Jabelensky et al (1992) also show that people in developing countries have a better prognosis for schizophrenia than those in Western countries. The precise reason for this has yet to be demonstrated.

Some studies have shown differences in outcome between the subtypes of schizophrenia. Fenton and McGlashan (1991a) have suggested from their study of 187 patients that those with paranoid schizophrenia experience a remittent course which is associated with less disability while those with hebephrenia tend to have a poor long-term prognosis. There appears now however to be some evidence to suggest that earlier intervention may alter the course of the illness. McGlashan and Johannessen (1996) have hypothesised that both early detection and treatment may be effective in reducing the severity of symptoms and provide better outcomes.

There have been some studies which have suggested that the presence of negative symptoms in schizophrenia are predictive of course and outcome. Fenton and McGlashan (1991b) has reported that negative symptoms are associated with a progressive course and greater disability in patients who experience them. Ring et al (1991) however found that negative symptoms can occur at any time during a schizophrenic illness and that they were not predictive.

**Conclusions**

In stark contrast to how the illness is portrayed by the media and perceived by the public many of those who write about schizophrenia do so from the stand point of optimism. Regardless of the purpose of the research or comments on schizophrenia professionals are largely in agreement that a cause will be found and that improvements in the care and treatment of those with the illness will continue.

Since the days of Kraepelin and Bleuler the description, classification and diagnosis of the illness has advanced considerably. Shepherd (1994) conceptualises the aim of these advances as a "psychiatric Esperanto" meaning that homogenous description of the illness will lead to better identification and understanding among those who work with people with schizophrenia. The rigorous application of such descriptions may contribute to the development of treatments by ensuring that those who are offered treatment for schizophrenia are actually suffering from the illness. Schizophrenia, some authors suggest, may not be one illness with a few subtypes but many illnesses with dozens of subtypes. Some authors have suggested for instance that illnesses which are characterised by primarily positive or negative symptoms may be new subtypes of the illness (McGlashan and Johannessen 1996). Further advances in our understanding will hopefully produce real gains for those who have schizophrenia.

Often people with schizophrenia can receive treatment based on the current interests of those providing the service. Therefore in some areas they will receive assertive outreach from community mental health teams who provide psychosocial interventions as well as up-to-date pharmacological treatments. In other areas they may be discharged from hospital without support networks with only an outpatients appointment to keep them company. Providing treatment in a consistent manner has

now become the concern of government. The result is a set of clinical standards of care which is aimed at improving the consistency of treatments for those with schizophrenia (Clinical Standards Advisory Group 1995). There remains good evidence that the use of psychosocial interventions with people with schizophrenia has achieved some real gains for those with the illness (Tarrier 1991) However it also appears to be the case that these gains can be lost over time (Marks 1992) and that removal of continuing professional support can eventually lead to relapse of the illness (Marks et al 1994). It therefore appears that people with schizophrenia will need long term support and treatment. This may appear an expensive option but set against the cost of long term hospitalisation and the improvements in social functioning which can accrue if properly supported living in the community is offered the investment would seem worthwhile.

There are now numerous studies investigating the genetic causes of schizophrenia. Despite inconclusive findings Gournay (1990) suggests that a genetic cause for schizophrenia will be found. It seems that each study advances our knowledge another small step and eventually a picture will form of this most complex of puzzles. Gournay (1990) argues that the advances in genetics will radically change the roles of many professionals working with people with schizophrenia as well as changing public perceptions of the mentally ill. Should this be the case then one can only hope that this change in public perception is for the better. The concern is however that if geneticists become able to identify those who are likely to develop schizophrenia before birth will the public demand be that these pregnancies be terminated. As with all developments in molecular genetic research in humans the concept of eugenics raises its ugly head. Productive debate on the morality and ethics of such arguments

may be useful. Professionals can at least be aware of the possibilities and prepare for an informed debate.

While it now seems clear that the incidence of schizophrenia is similar across cultures there remains many questions as there are answers in the findings which suggest that immigrants (Stopes-Roe and Cochrane 1980) and latterly their offspring (Eagles 1991) have a higher risk of developing mental illnesses including schizophrenia than the host population. Some authors suggest that the presence of endemic toxic environmental factors can cause schizophrenia in those with genetic susceptibility to the illness (Sharma and Murray 1993). These potential pathological agents have as yet to be identified. There has however been much interest, among those searching for the cause of schizophrenia, in the effects of viral agents. Inconclusive evidence exists on the role of influenza epidemics (Cannon et al 1996) but as our knowledge increases other environmental agents may be identified which have a causative role. It remains to be seen however whether most of this research will further our knowledge or just lead us up so many blind alleys in our search for the cause of this illness.

One of the difficulties in embarking on a review such as this is the tendency to adhere to the orthodoxy. The papers reviewed here are without exception written by professionals educated in a system which essentially perpetuates itself. There may be debate among those professionals but fundamentally they approach the subject from a medico-social stand point. This means therefore that other opinions are seldom heard or even acknowledged. The effects of this can be that research which does not conform to the orthodoxy will not be funded. The truth may be out there the question maybe though is there anyone prepared to take the risk to find it?

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