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Some possible answers to questions about schizophrenia that have concerned John Gunn

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Over the past 30 years John Gunn has made an enormous contribution to Forensic Psychiatry both in the UK and abroad. John was appointed Professor of Forensic Psychiatry at the Institute of Psychiatry in 1978 and on his retirement leaves a department renowned worldwide for its exemplary clinical work, teaching and research. More personally, John has been a wonderful role model for the rest of us at the Institute and Maudsley. We have particularly admired his caring mentoring of junior colleagues, and his enduring devotion to the most challenging, neglected and poorly understood patients, individuals that society and indeed most psychiatrists reject. Such characteristics are not measured as part of the Research Assessment Exercises so beloved of university bBurocratsbbureaucrats but their impact is rather more lasting.

Many of John Gunn's patients have suffered from schizophrenia, and here we would like to present new evidence concerning some of the questions about the disorder that John and the senior author of this article have often discussed over the years.

Is there a role for social factors in the aetiology of schizophrenia?

Ideas about the causes of schizophrenia have changed over the century since the concept emerged but the predominant view since 1976, when the first brain-scan study was reported, has been that schizophrenia is a genetically determined organic brain disease. This perspective has led to considerable improvements in our understanding of the biological roots of the disorder. However, the genetic-brain-disease model has been a contentious issue for many patients who find it demoralizing and degrading. John Gunn has also been dissatisfied with the more reductionist variants of this model, and has repeatedly emphasized that we must also consider the role of social factors in the onset and the outcome of the disorder.

This perspective has not been widely shared by schizophrenia researchers over the past 25 years but the orthodox view has begun to change recently.

Models in which schizophrenia was thought to result from the effect of a single major gene have given way to multi-factorial models in which a vulnerability to the illness is inherited rather than the disease per se. Similarly, researchers continue to believe that early neurodevelopmental risk factors for schizophrenia exist but increasingly accept that these are unlikely to be sufficient for expression of the disorder. Consequently, there has been a renewed interest in psychosocial and psychological factors that may precipitate the onset of disease (Murray et al., 2002).

Among the forces that have produced this change have been the reports that psychosis is more common than expected in immigrant populations, in particular in African-Caribbean (AC) and African people living in the UK. For example, one study in South London showed the rate of schizophrenia to be between four and eight times higher in individuals of African-Caribbean origin compared with Caucasians (Castle et al., 1991). This increased prevalence is particularly noticeable among second-generation immigrants (the offspring of the first generation who originally migrated). Hutchinson et al. (1996), for example, found the morbid risk of schizophrenia to be 4.3% and 6.6% in the siblings of white and first-generation schizophrenic probands respectively. However, the risk rose to 26.3% in the siblings of second-generation AC schizophrenic individuals. These marked differences clearly cannot be accounted for by genetic factors alone; some environmental factor prevalent in the UK must be impacting on second-generation African-Caribbeans to increase their risk of the disease. One possibility was an excess of pregnancy and birth complications (PBCs). However, by comparing the frequency of such complications in White and AC psychotic patients, Hutchinson et al. (1997) found the rate of PBCs to be higher in white patients, thus disproving this theory. Indeed, so far the excess of schizophrenia has not been explained by any known biological risk factor, and investigation has turned to the possible role of social environment.

Social isolation and discrimination may be important. Research in the United States has shown an association between the proportion of an ethnic minority living in a particular area and their rates of admission for mental illness. Here in the UK, Boydell et al. (2001) investigated whether the incidence of schizophrenia among people from non-white ethnic minorities is greater in neighbourhoods where they constitute a smaller proportion of the total population. By studying 15 electoral wards in Camberwell, South London, she was able to show an inverse dose–response relation between the proportion of people from a non-white ethnic minority group living in an area and their incidence rate for schizophrenia. In the third of wards where non-white ethnic minorities formed the largest proportion, the incidence rate ratio was 2.28 compared with whites, with this figure rising to 4.4 in the third of wards where they formed the smallest proportion. What seems to be important is the absolute number or concentration of people from non-white groups in the immediate vicinity. Thus a possible mechanism is increased exposure to, and/or

reduced protection against, stress and life events. Specific stresses worth examining may be overt or perceived discrimination, institutionalized racism and perceived alienation and isolation. The more isolated a member of an ethnic minority, the more likely he or she may be to encounter, or be vulnerable to, such stresses.

When do delusions begin?

A major issue in forensic psychiatry is how and when delusions begin. The Dunedin Multidisciplinary Health and Development Study has provided the opportunity to answer the question of whether children who later become schizophrenic are more likely to have strange ideas than their peers. The Dunedin Study is a prospective, longitudinal investigation of the health, behaviour and development of a complete birth cohort born between April 1972 and March 1973, in Dunedin, a city on New Zealand's south island (Silva and Stanton, 1996). Data were collected at birth from study members, who were then invited for assessment when they were aged 3, 5, 7, 9, 11, 13, 18, 21 and 26. An important feature of the Dunedin Study is its very high participation rate: even after 26 years, 96% of the study members are still alive and continue to participate in the study.

There is consistent evidence of delays in attaining developmental milestones, cognitive and language deficits and abnormalities in social functioning in childhood among individuals who later go on to develop schizophrenia or schizophreniform disorder. By studying the Dunedin cohort, Poulton et al. (2000) have shown that that in those diagnosed with psychosis in their twenties, psychotic symptoms (albeit in a less obvious form) may have been present for many years. When study members were aged 11, they were administered the Diagnostic Interview Schedule for Children and asked the following four questions about possible psychotic symptoms:

- Have other people read your mind?
- Have you ever had messages sent to you through television or radio?
- Have you ever thought that people are following you or spying on you?
- Have you heard voices other people cannot hear?

Among individuals who self-reported strong psychotic symptoms at age 11 ('yes, likely' to two symptoms or 'yes, definitely' to one symptom), 25% met diagnostic criteria for schizophreniform disorder at 26 years, compared with 2% among those who did not report any psychotic symptoms (Poulton et al., 2000).

Does drug abuse increase the risk of schizophrenia?

It is well known that people with schizophrenia are more likely to take illicit drugs than the general population; cannabis is the drug most frequently taken

(Maden et al., 1992). But can drug abuse cause schizophrenia? Whether cannabis use causes schizophrenia or merely heralds the onset of the pre-existing illness has been much debated recently. The strongest evidence that it may be a risk factor comes from a Swedish cohort study, which found heavy cannabis use at the age of 18 increased the later risk of schizophrenia sixfold (Andréasson et al., 1987). However, this study failed to establish whether adolescent cannabis use was a consequence of pre-existing psychotic symptoms rather than a truly independent risk factor. To test whether cannabis is a true causal factor for later psychosis, we need to rule out the possibility that cannabis is a consequence of emerging schizophrenia rather than a cause of it. Arseneault et al. (2002), studying the Dunedin cohort, found cannabis use to be associated with an increased risk of experiencing psychotic symptoms, even after psychotic symptoms present at age 11 years (i.e. preceding the onset of cannabis use) were controlled for. This indicated that cannabis use is not secondary to pre-existing psychotic symptoms. They also found that early cannabis use (by age 15) confers a greater risk for schizophrenia than later use (by age 18). No other illegal drug use was found to increase later risk of schizophrenia.

Does assertive community care decrease risk of violence?

The quest continues to ascertain the factors that determine the aetiology and development of schizophrenia. What primarily concerns forensic psychiatrists, however, is the link between violent behaviour and psychosis in schizophrenia (Taylor and Gunn, 1984). Until the mid-1980s, the consensus was that those with mental disorder were no more likely to be violent than the general population. Since that time, however, robust epidemiological evidence has emerged that has radically challenged that view. Three different approaches have been used to ascertain whether such a link exists. These include studies examining the prevalence of violence in those with schizophrenia, the prevalence of schizophrenia among those who have been violent and large-scale epidemiological studies conducted in the community. The overall consensus is that there is an increased risk of violence in schizophrenia; this risk is, however, small and limited to very few. Additionally, as John Gunn and Pamela Taylor have shown, in the UK the contribution to homicide statistics of those with serious mental illness is falling (Taylor and Gunn, 1999), and there is no convincing evidence that de-institutionalization has led to an increased proportion of violence in society being attributable to those with schizophrenia (Mullen et al., 2001).

While the current research evidence points to a small risk, when a tragedy occurs involving serious violence by a mentally ill person it is often sensationalized in the media and is devastating for those involved. It is the duty of psychiatrists, both general and forensic, to manage this small risk in collaboration with other agencies. Much emphasis is currently placed on risk-prediction

instruments with less attention being paid to practical ways of managing risk among those psychotic patients residing in the community. As part of the UK 700 project, 708 patients with psychosis (registered with general adult psychiatric services) were randomized to receive either intensive case management (ICM) or standard care. The only difference between the two conditions was that those patients randomized to ICM were assigned a case manager who had 10–15 patients on his/her caseload compared with 30+ in the standard group. Over the next two years, physical assault was measured from three data sources: patient interviews, case manager interviews and case note review. Overall, 23% (148) of subjects assaulted another person during the study but no difference was found in the prevalence of assault between the intensive (23%) and standard groups (Walsh et al., 2001). It appears from these findings that increasing the intensity of contact is alone insufficient to reduce assault. The content of contacts is probably more important with strategies that target risk factors for violence such as substance misuse and medication non-compliance needing evaluation in further research.

As John Gunn has often pointed out, violence is not a one-way street with victimization and violence being closely linked. Social drift and the vulnerabilities of the severely mentally ill make them more likely to live in crime-ridden neighbourhoods and be subjected to violence. Some 16% of the UK 700 patients experienced violent victimization over two years, representing a prevalence more than three times greater than suffered by members of the general population dwelling in inner cities in the UK (Walsh et al., 2003). Although victimization was associated with illegal drug use, in the presence of a personality disorder and a history of recent assault, which are also linked to violent behaviour, it was also associated with more severe psychotic symptoms and homelessness, suggesting that the most severely ill may be at particular risk. It is appropriate that at the time of Professor Gunn's retirement there is a newly emerging literature in the area of victimization of the mentally ill. If the public could better understand the difficulties society presents to the mentally ill rather than the difficulties posed by them, this might serve to reduce the disabling stigma attached to mental illness, a cause close to John Gunn's heart (Gunn, 2000).

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