Genetic risk factors and variation in European suicide rates

Marušić & Farmer (2001) adduce selective evidence in support of the hypothesis of greater genetic influence on suicide than has previously been considered. In advancing this idea, they pay insufficient regard to national variations in a number of factors potentially relevant to differences in suicide rates. Data on national differences in the prevalence of psychiatric disorders—surely an important potential confounder—are glaringly absent from the paper, and cultural differences not appearing in conventional tables of ‘known’ risk factors, including patterns of alcohol consumption, are also neglected.

At the level of population genetics, Marušić & Farmer make a crucial error in grouping Hungarians genetically with Finns: Finno-Ugrian is a language family, not an ethnic one; and although both Finnish and Hungarian populations contain comparable (although by no means identical) proportions of non-European genes (see, for example, Guglielmino et al., 1990), the Finnish population is highly unusual, enriched as it is with certain rare hereditary disorders (see, for example, Ranta et al., 2001). Cavalli-Sforza (2000) suggests that this is due to abnormal statistical fluctuations that arose in a very small founding population. Unless Marušić & Farmer can suggest genes that might plausibly affect suicidal behaviour, specific polymorphisms which are shared by different Finno-Ugrian-speaking populations but not by less suicide-prone populations, then their grouping together of these geographically separated nations appears, at best, questionable. Accepting that there are true differences in suicide rates between, say, Finnish and Swedish populations, it is sensible to consider potential, unexamined cultural explanations. There are several from the sociological literature, including the proposal that Finland has a more anxious culture than Sweden (reflected in its significantly higher score on a measure termed ‘uncertainty avoidance’; Hofstede, 1991). Another is differential social capital. Interestingly, in this regard, Finnish-speaking Finns have a longer active life than Finnish-speaking Finns (Hyppa & Maki, 2001); the authors interpret this as reflecting differences in social capital, and certainly it illuminates the possibility that such differences might influence propensity to suicide. There are other relevant comparisons. Estonia, for example, is cited by the authors as having the third highest suicide rate in Europe, in contrast to a relatively low rate in Sweden. The fact that Estonia and Finland share a (Lutheran) faith with Sweden does not lead to the conclusion that attitudes toward death and suicide are identical.

The purported ‘black swan’ of Slovenia, matched with Mediterranean neighbours such as Italy on some psychosocial variables but with a higher suicide rate, is an interesting observation meriting further study. But there is another, non-genetic explanation for the difference: a 40-year history of separation. There is ample anecdotal evidence from the former USSR of decreasing confidence, self-esteem and standards of health after the fall of communism.

By emphasising the likely interaction of environmental and genetic influences on suicide, Marušić & Farmer implicitly recognise that suicide is a complex and emergent trait, yet they make too much of the implications for suicide prevention of genetic research into suicide. I would argue that a more realistic target for future genetic research in this field might be the detection of genetic influences on various measures of impulsive behaviour, including some forms of suicide, as a means to guide biological research into impulsivity itself—a mercurial construct that probably has complex associations with clinical outcomes.

Marušić & Farmer (2001) argued that genetic factors may play a role in the variation of suicide rates in European nations. They suggested the role of the Finno-Ugrian ethnic group and the possibility that genetic factors play a role in the alcohol–suicide link. We have conducted two studies that support their argument.

First, we quantified the influence of Finno-Ugrians on European suicide rates by correlating the suicide rate of all 30 European nations with the percentage of Finno-Ugrians in the population (Kondrichin & Lester, 1997). The Pearson correlation coefficient was 0.58 (two-tailed P < 0.01).

Second, Lester (1987) calculated the proportion of people with type O blood in 17 industrialised nations (including 12 Western European nations) and correlated this with the suicide rates. The Pearson correlation was −0.67 (two-tailed P < 0.01). Lester then noted that people in Hungary and Czechoslovakia (both in the Eastern European bloc at the time and not in the original sample) had very low proportions of type O blood and very high suicide rates compared with the original sample.

These two studies support the suggestion of Marušić & Farmer that genetic factors may play a role in the variation in European suicide rates.


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Authors’ reply: We are grateful to Lester & Kondrichin for pointing out the high correlations between the suicide rates in 30 countries with the percentage of Finno-Ugrians in the population, and of the negative correlations of suicide rates with the proportions of people with blood type O in different countries. This certainly provides additional evidence in support of our hypothesis.

Tunstall is more critical and his important comments require a considered response. He states that we have not addressed the issue of other sociocultural factors that may be relevant to the differences in European suicide rates. It has been pointed out elsewhere (e.g. Diekstra, 1993) that rate variation due to such factors even out when considered across countries, and can probably be ignored. Also, as we have pointed out, sociocultural explanations alone cannot explain the rate found in our ‘black swan’ example, Sweden.

For brevity in a short editorial, we did not explore the ‘potential confounder’ of the prevalence of psychiatric disorder in different countries in our paper, although contrary to Tunstall’s assertion, we have not neglected to discuss the complex relationship between alcohol consumption and suicide. Not only do we consider in some detail the possible malignant interaction between alcohol exposure and genetic constitution in Slovenia, but we also point out the complex association between alcohol consumption and suicide rates elsewhere in Europe, citing Sweden and France as examples.

Tunstall suggests that Finland is a more ‘anxious culture’ than Sweden. While this may be true, we note the less contend that such cultural anxiety would also have genetic underpinnings. Measures of trait constructs such as neuroticism have been shown to be, in part, genetically determined, and many of the risk factors previously believed to be entirely psychosocial have also been shown to be at least partly under genetic influence, including religious and political beliefs, marital difficulties and divorce (e.g. Kendler & Karkowski-Shuman, 1997).

Tunstall challenges us to ‘suggest genes that might plausibly affect suicidal behaviour . . . which are shared by different Finno-Ugrian-speaking populations but not by less suicide-prone populations’. In a recent genetic study of Slovenian suicides, we have replicated the tryptophan hydroxylase polymorphism previously reported in the Finnish population (further details available from the authors upon request). The same polymorphism has not been replicated in a UK study (Evans et al., 2000), a country less suicide-prone than either Slovenia or Finland.

Tunstall also suggests that the differences between Slovenia and its neighbours result from ‘40 years of separation’, using the analogy of the USSR. Communism under Tito was far less restrictive and deprived, in terms of lifestyle and travel opportunities, than in the former Soviet Republics. In addition, why does the neighbouring country of Croatia, which experienced the same ‘40 years of separation’, consistently report lower suicide rates (Pavlović & Marušić, 2001) if this were the only explanation of the high Slovenian rates?

Finally, Tunstall suggests that a more realistic target for future research would be the detection of genetic influences on impulsivity. This is exactly what we are planning to do.


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**Aggression in schizophrenia: assessment and prevalence**

In a recent paper Jones et al. (2001) reported an association of aggressive behaviour in schizophrenia with catechol-O-methyltransferase genotype. The authors studied a sample of 136 males and 44 females with schizophrenia. Aggression in patients was clinically assessed by means of the Overt Aggression Scale (OAS; Yudofski et al., 1986). The patients in this sample showed a surprisingly high level of aggression: 52% verbal aggression in male patients (46% in females), 39% aggression against objects (25% in females), 23% against self (9% females) and 39% against other people (34% females) as measured by the OAS. Data on prevalence of aggression and violence in people with schizophrenia differ widely depending on definition and assessment period but most clinical studies in this field have shown lower rates, at least of physical aggression (for review see Schanda & Taylor, 2001; see also Monahan et al., 2000).

In a recent retrospective study we evaluated the patient files of all patients with ICD-9 schizophrenia admitted to the psychiatric hospital of the University of Munich between 1990 and 1995 (n = 2093). Relevant socio-demographic, clinical and psychopathological data were evaluated. Fourteen per cent of patients (n = 292) met the criteria for aggression (verbal and physical) on admission (Soyka & Ufer, 2002).

Jones et al. feel that aggression may even be underestimated in their sample. Recent data suggest that the risk of violence is indeed overlooked in psychiatric patients. Sanders et al. (2000) pointed out that while psychiatric patients are asked about suicidal ideas, aggression and the risk of violence are frequently neglected even in patients with clearly violent thoughts.

I have some concerns over whether a single rating scale can be valid and reliable enough to assess aggression adequately in schizophrenia, especially for genetic studies. Aggression and violence in schizophrenia are difficult to predict (Steadman et al. 1998; Wallace et al. 1998; Monahan et al., 2000) and can be both trait or state phenomena in schizophrenia. The OAS is a sensible instrument in this field but aggression is a multi-dimensional phenomenon. In most studies on that issue data from different sources are utilised (Swanson et al., 2000). Steadman et al. (2000) have proposed an actuarial tool for assessing the risk of violence which has been evaluated in civil psychiatric patients (Monahan et al., 2000). Beside clinical interviews and specific psychopathological scales a broad number of other diagnostic instruments can be used to assess aggression and the risk for violence.
in psychiatric patients. A few of them may be mentioned: the Buss-Durkee Hostility Inventory with its sub-scales including ‘physical aggression’ (Buss & Durkee, 1957) or the Brown–Goodwin assessment for lifetime history of aggression (Brown et al, 1979). Assessment and classification of such complex phenomena as aggression should not be based on only one scale, especially in genetic studies with their substantial clinical and possibly forensic/legal implications. Nevertheless, we hope that this research may provide more insight into the biological mechanisms underlying aggression.


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Post-traumatic stress disorder and management of stillbirth

Lovett (2001), commenting on our article ‘Incidence, correlates and predictors of post-traumatic stress disorder in the pregnancy after stillbirth’ (Turton et al, 2001) took issue with our reporting a possible association between post-traumatic stress disorder and holding the dead infant which did not reach statistical significance.

We should like to make two points. First, we were at pains to make it clear that numbers were small and not significant statistically. Second, although your correspondent could not know this at the time of writing, we have subsequently published a paper reporting a significant relationship between seeing the dead infant and disorganisation of mother–infant attachment in the next-born child at the age of 12 months (Hughes et al, 2001). This was an unexpected finding for us.

Our main concern is that a profound change in clinical practice (seeing and holding the dead infant) was introduced with great enthusiasm in maternity units in the UK and elsewhere on the basis of limited and non-systematic clinical observation. Our published findings to date do not offer any evidence in support of this practice. We concur with Dr Lovett that this is an area which demands further investigation and rigorous evaluation.


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Psychiatric morbidity and elderly offenders

While I agree entirely with Fazel et al (2001) that there is an unmet need for psychiatric care for elderly offenders, I wonder whether this need is even greater than is implied by their paper. It is important not to forget those elderly people who do not actually end up in prison but have committed crimes. Yorston (1999) notes that the elderly are less likely than younger offenders to have custodial sentences or fines imposed and are more likely to receive probation orders. Lynch (1988) postulated that the public’s sympathy for the perceived frailty of the elderly is likely to lead to this group being treated more leniently. Bergman & Amir (1973) have also noted a tendency for families to hide deviance, which may lead to offending behaviour in this group being underreported.

One revelation to me which emerges from Fazel et al’s paper was the relatively high number of offenders imprisoned for drug offences (29/203). Older studies (e.g. Taylor & Parrott, 1988) suggested that drug-related crime was of a much lower incidence: indeed, in their study of elderly custodial remand prisoners none aged 55 and over had been charged with a drug-related offence, although they noted that misuse of alcohol appeared to rise steadily with age. I wonder whether Fazel et al are showing us that the victims of the drugs culture, traditionally thought to have been established in the UK in the 1960s, are now starting to feature among the elderly?


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Elderly mentally disordered offenders are underresearched and poorly understood, but Fazel et al (2001a) demonstrated high levels of ‘hidden’ psychiatric morbidity in a sample of male prisoners over 60 years of age.

The cases of the former Chilean dictator Augusto Pinochet and of Ernest Saunder, involved in the Guinness financial affair, illustrated the inherent difficulties of the older person in the forensic setting.
This was demonstrated when Saunders became the first case to make a full recovery from a diagnosis of Alzheimer’s dementia.

In Fazel et al’s two related papers (2001a,b), significant levels of both psychiatric and physical morbidity are clearly evident that will surely have future service implications as the elderly prisoner population continues its inevitable rise. Prison services for elderly inmates have been slow to develop despite the Reed report (Department of Health & Home Office, 1992) that acknowledged the complex nature of elderly prisoners and demanded a ‘holistic’ approach in their management.

Yorston (1999) contemplates the future of old age forensic psychiatry as a sub-specialty akin to those of learning disabilities and child and adolescent psychiatry. As the number of elderly mentally disordered offenders presenting currently is small, but increasing, he suggests that a regional tertiary referral service for the most difficult or serious cases, with close links between the relevant forensic and old age services, might be preferable at this time.

An integrated approach between old age and forensic services using their different areas of expertise will make assessment and management of elderly offenders more comprehensive, as opposed to management by one team alone. This is in keeping with standards 2 (person-centred care) and 7 (mental health) of the National Service Framework for Older People (Department of Health, 2001), which emphasise the importance of an integrated approach to assessment and care-planning through liaison with specialist services for older people. Standard 2 also suggests that the National Health Service and local councils should ensure a flexible and integrated approach to service provision, regardless of professional or organisational boundaries.

To date, elderly offender research has almost exclusively been retrospective but the studies by Fazel and colleagues suggest that a substantial prospective study of elderly offenders would not only be feasible but also desirable to improve our poor knowledge of this important group.

**The evolutionary psychology debate**

I am very much honoured that a prominent scientific writer like Rose (2001) treats me with the same method as he and his circle have treated E. O. Wilson in their recent collection of essays (Rose & Rose, 2000). Being a mere practising psychiatrist, it puzzles me why it has become acceptable for the anti-sociobiology/evolutionary psychology movement to misquote their opposition, in either a patronising or an openly hostile way, attributing hidden agendas to those who dare to think about human behaviour and psychological functioning in an evolutionary context. Clearly, they feel that the end justifies the means, and that their version of the truth has to be defended at any cost.

Segerstråle (2000), in a detailed analysis of the sociobiology debate, compared the two camps of scientists to gardeners: one side representing the planters, and the other the weeder. It seems to me that both tasks are important in the development of the perfect garden of science. Rose appears to be an overzealous weeder, who is afraid that the dangerous weed of evolutionary psychology will destroy his garden and tries to kill it at every opportunity. The effort is unlikely to succeed. However, I need to point out that in my previous letters concerning the evolutionary psychology debate (Aytton, 2000, 2001) there was nothing to imply ‘some sort of conspiracy in psychiatry to ignore biology’ (Rose, 2001). About 30–40% of all psychiatric references on the Medline database are biological studies, so there is no lack of biological studies and theories. However, what is lacking is a coherent theoretical framework; and evolutionary theory is largely ignored by psychiatric training or academia. It is untenable to state that only proximal causation is relevant to mental states or human behaviour. This was recognised by Darwin and beautifully demonstrated by Bowlby. Despite initial strong criticism, Bowlby’s contribution to the understanding of the mother–infant relationship has become fundamental, and has wiped out earlier explanations.

If ‘nothing in biology makes sense except in the light of evolution’ (Rose, 2001), then surely, human beings and their behaviour cannot be excluded on scientific grounds.

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