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EDITED BY MATTHEW HOTOPF

Genetic risk factors and variation in European suicide rates

Marušič & 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, Marušič & 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 Marušič & 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, Swedish-speaking Finns have a longer active life than Finnish-speaking Finns (Hyyppa & 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, Marušič & 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.

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Marušič & 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 Marušič & 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 some such factors evens 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, Slovenia.

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 none 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 & Marušič , 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

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