Invited commentaries on: Premorbid adjustment and personality in people with schizophrenia†

HIGH RISK, LOW PREDICTION: IMPLICATIONS FOR EARLY INTERVENTION

The Swedish conscript study, with its sample size of 50 054, is indeed unique in many respects and has already generated at least one important finding in schizophrenia research: the demonstration of a predictive effect for premorbid cannabis use on the risk of developing schizophrenia (Andreasson *et al*, 1987). Malmberg *et al* report on their analysis of self-administered questionnaire data collected at the initial evaluation of the cohort, in relation to the subsequent 195 onsets of schizophrenia and 193 onsets of other psychoses during the 13 years of follow-up (i.e. over a period in which the cohort had passed through 40–50% of the lifetime risk for schizophrenia in males). Their statistical analysis is adequate, using odds ratios with confidence intervals and logistic regression to assess effect independence. There is one result that is remarkable: a composite variable loaded by four items (having fewer than two close friends; preference for small groups; feeling more sensitive than others; and having no steady girlfriend) was associated with an odds ratio of 30.7 (CI 12.9–73.8) for schizophrenia, if all four variables scored positively. This is one of the highest risk ratios for any suspected factor in schizophrenia ever reported, and the prospective nature of the data, in conjunction with the good statistical power due to the large sample size, give it credence. At the same time, its predictive value is moderate to low, because of the high population incidence of each one of the four behavioural traits making up the composite variable. I wish that Malmberg *et al* had reported the positive predictive value and the negative predictive value, as well as the percentage outcome variance (onsets) explained by their composite variable. As regards the interpretation of this result, it is tempting to speculate that the four behavioural traits might represent a proxy for something else which was not directly measured at the initial evaluation of the cohort, and that there might be a single underlying factor, for instance, a neurocognitive peculiarity of a developmental origin. However, an explanation in terms of a stochastic mix of temperamental characteristics is equally plausible, especially in view of the possibility that the effect may be multiplicative, rather than just additive, as suggested by the changes in the odds ratios and in attributable risk which occur as the number of variables scoring positively increases from one to four (Table 4; p. 311)

Andreasson, J., Allbeck, P., Engstrom, A., *et al* (1987) Cannabis and schizophrenia. A longitudinal study of Swedish conscripts. *Lancet*, *ii*, 1483–1486.

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WHO GETS SCHIZOPHRENIA

This historical cohort study by Malmberg *et al* achieves what Ambelas (1992) undertook to do; it adds to the evidence and sharpens the focus of inquiry into premorbid adjustment of personality before psychosis. This line of developmental epidemiology has two related goals. The first is to inform the understanding of causation and mechanisms underlying psychosis. This assumes that some components of the relevant causal constellations are put in place early in life, remote from the disorder itself. The second, even more ambitious goal, is early identification, intervention and prevention.

This niche of research is not straightforward in terms of time or techniques. It can sometimes seem akin to undertaking a physical examination of a subject who has kept their clothes on: the answers are in there, but you never quite get your hands on them. After the hurdle of 'first find your data' is cleared, half the fun is removing layer upon layer of selection bias, information bias and known confounders. Even then, one is left with only one perspective of the truth, to be judged together with other views. The best, unbiased exposure information is always what was thought relevant at the time, not what one would wish to ask now. Even that would be out of kilter with future ideas.

With these general caveats in mind, Malmberg's results are of great interest. The many strengths of the study are well rehearsed in the paper, in particular the large sample size and relevant data on personality at age 18 years. The processing of 50 000 subjects in two years must truly have been a military operation. Restriction to men is a drawback, although it keeps the focus on psychosis in a population at risk, rather than on confusing differences between the genders. Low prevalence of schizophrenia, even with a broad diagnosis, and reliance on admissions for case ascertainment remain factors which may have contributed to bias.

PERSONALITY IN CAUSAL CONSTELLATIONS

So what of these results? The collection of variables in the final analysis sits comfortably with the notion of a schizoid personality. The contribution of such a personality to subsequent schizophrenia is defined further in terms of sociability, which Malmberg *et al* link to theory of mind.

Inevitably, some other attributes of personality are not available, but the evidence against the effect in terms of sociability merely being part of the prodrome is convincing. As the authors note, it complements other work indicating that the first signs of such behaviour and other differences are already present in childhood many years before adulthood and the

†See pp. 308–313, this issue.

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