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CORRESPONDENCE

To the Editor:

We read with interest the article, 'Morphometry in schizophrenia revisited: height and its relationship to pre-morbid function' (Nopoulos *et al*. **28**, 655–663). The authors have shown that the mean height of schizophrenic patients is significantly shorter than that of the controls by about 2 cm. Schizophrenic patients allocated to the lower quartile with regard to height had a significantly poorer pre-morbid function, as measured by psychosocial adjustment and cognitive function, than those in the higher quartile.

The authors point out that height is determined by some combination of genes and environmental factors. Since previous studies have shown the association of various factors of early insults, such as viral infection, maternal malnutrition, rhesus factor incompatibility and birth injury, with the risk of later development of schizophrenia, the authors first considered whether these could account for their findings of short stature in individuals with schizophrenia. Socio-economic status was considered to be a major determinant of the differential environmental effects during childhood. On the grounds that there was no significant difference in parental socio-economic status between their samples of schizophrenic patients and controls, the authors dismiss the role of these environmental insults as implausible. However, in order to test for a group difference in socio-economic status, the *t* test was used in the study. This cannot be appropriate, as this factor is a nominal variable. As the authors note, the relationship between global growth deficits and family history should be examined to differentiate directly between environmental and genetic effects.

Although the claim that schizophrenics tend to be shorter in stature is of interest, the finding could be an artefact. The total lifetime dose of neuroleptics, which may affect mineral density (Halbreich *et al*. 1995; Halbreich & Palter, 1996; al-Adwani, 1997) and hence is a potential confounding factor in assessing height, ought to be taken into account. It is well known that patients with schizophrenia frequently suffer from osteoporosis. Baastrup *et al*. (1980) have found that a group of schizophrenic patients receiving neuroleptic drugs exhibited a low bone mineral content averaging 86% of that for normal controls. Hunt (1996) reported on the relationship between decrement in height and osteoporosis in a sample of 76 subjects in a bone health clinic. The study has shown that excessive height loss was related to bone mass, and also found that 40% of the subjects with excessive height loss had suffered compression fractures. These findings are consistent with a study by Huston & Bloom (1975) who have shown that males with chronic schizophrenia are shorter in stature compared with male controls, but that there is no difference in height between acute schizophrenics and controls. Indeed, Huston & Bloom (1975) have attributed their observations to the differential effects of antipsychotic drugs taken by acute and chronic patients with the disorder. Furthermore, anteroflexed posturing due to side effects of antipsychotic treatment (i.e. neuroleptic-induced parkinsonism) is commonly observed in schizophrenic patients. Therefore, one may raise the question of the precision in measuring height, in particular for the patient population.

The authors have demonstrated that schizophrenic patients in the lower quartile with regard to height have a poorer pre-morbid function than those in the higher quartile. Patients with poorer pre-morbid function are thought to have a severe form of illness, and thus are likely to require a higher dose of neuroleptics, which in turn may lead to osteoporosis and drug-induced parkinsonism. Therefore, one may detect the spurious relationship between small stature and poor pre-morbid function in schizophrenia.

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The Author replies:

In their letter, Drs Kawai, Takei and Mori suggest that the findings of decreased height in the sample of patients with schizophrenia compared to healthy controls (Nopoulos *et al*. 1998) may have been related to exposure to antipsychotic medication, which may result in osteoporosis and therefore reduction in height. Although this phenomenon may be true, it is no doubt a process that occurs after *long-term* exposure to neuroleptics. The authors quote an article by Hunt (1996), which reported the relationship between height loss and osteoporosis in a sample of 76 subjects. The average age of that sample was 60·4 years. The average age of our sample was considerably younger at 33 years. It seems quite unlikely that men at this age would have significant height loss due to severe osteoporosis.

Furthermore, our data set was reanalysed looking at the relationship between neuroleptic exposure and height. We have two variables that indicate neuroleptic exposure: months of neuroleptic exposure; and dose-years, which is an estimate of neuroleptic exposure taking into account not only length of exposure, but dose of drug as well (Miller *et al*. 1995). The mean length of neuroleptic exposure of the sample ($N = 226$) was 88 months, then mean dose-years was 55. There was no relationship between dose-years and height (Pearson's $r = -0{\cdot}06$, $P = 0{\cdot}32$), and there was no relationship between length of neuroleptic exposure and height (Pearson's $r = 0{\cdot}000$, $P = 0{\cdot}99$).

These findings support our original interpretation of reduced stature in schizophrenia as a manifestation of a global deficit in growth and function and do not support the theory that the reduction in height is due to severe osteoporosis from chronic neuroleptic exposure.

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