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AUTHORS' REPLY: Wright & Taylor question our use of the term 'tardive dyskinesia' on the grounds that a small minority of cases were not receiving antipsychotic medication at the time of assessment. In fact, all patients had received antipsychotic medication at some time. In the text of the paper we referred explicitly to either orofacial dyskinesia or to trunk and limb dyskinesia to minimise ambiguity, but nonetheless consider that our use of 'tardive dyskinesia' in the title is reasonable. Wright & Taylor also suggest that our conclusion is unwarranted because our patients were atypical. As we reported, our patients were either undergoing rehabilitation or were long-stay patients, and hence represent a seriously disabled group. The negative symptoms of our patients might reasonably be described as symptoms of the defect state. Our study supports the hypothesis that these persistent negative symptoms are associated with earlier onset of orofacial dyskinesia. The study was not designed to determine whether other negative symptoms, such as the transient negative symptoms that sometimes accompany acute exacerbations of illness, are associated with vulnerability to dyskinesia.

With regard to the issue of institutionalisation, there was no significant difference in the duration of the current hospital admission between those with and those without orofacial dyskinesia, within each age band. With regard to sex differences, orofacial dyskinesia increased with age within both sexes.

Wright & Taylor imply that had we recruited a sample more representative of young schizophrenic patients in general, we might have concluded that trunk and limb dyskinesia increases with age. If we had done so, we would have been in danger of drawing a spurious conclusion, because it is virtually inevitable that elderly patients, with whom the young are compared, will have suffered sustained illness. For the purpose of our study, it was desirable to recruit in a setting that minimised the risk that young patients would represent a less severely ill group. Our finding that the prevalence of trunk and limb dyskinesia was independent of age is not only consistent with the other studies we reviewed in our paper, but also increases our confidence that the observed increase in prevalence of orofacial dyskinesia with age cannot simply be attributed to a selection bias, such that the younger patients had intrinsically less persistent illness associated with a lesser amount of non-specific neurological dysfunction. Clearly, it would be preferable to study patients longitudinally, but bias due to loss of contact with recovered cases during a five-decade study might still be a problem.

Simon Taylor raises the question of whether or not drug-induced Parkinsonism might have influenced our results. In a subsample of 105 cases in whom Parkinsonism was assessed, tremor and limb tone were not significantly worse in patients with orofacial dyskinesia than in those without, and hence it is unlikely that a global Parkinsonian syndrome affecting limb and facial muscles confounded our results. However, it is not possible to exclude influence from drug-induced diminution of facial expression. It is possible that the diminution of facial expression due to drugs and that intrinsic to the illness both reflect dopaminergic underactivity. We agree that it is also possible that dopamine-blocking drugs might contribute to negative symptoms in schizophrenic patients, although the evidence for the existence of negative symptoms before the development of antipsychotic drugs implies that factors intrinsic to schizophrenia play a substantial role.

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Low serum cholesterol and suicide attempts

SIR: We read with interest the article (*BJP*, June 1993, **162**, 818–825) commenting on the association between low serum cholesterol and suicide attempts among psychiatric patients. Although findings have been inconsistent (Pekkanen *et al*, 1989; Davey Smith *et al*, 1990), a meta-analysis (Muldoon *et al*, 1990) and recent cohort studies (Lindberg *et al*, 1992; Schuit *et al*, 1993) have found an increase in deaths from external causes, including suicide, to be associated with low serum cholesterol. However, Goble & Worcester (1992) have argued that this observation could be due to a confounder – depressed patients (at risk of suicide) with decreased

appetite and weight loss causing the lowering of blood cholesterol levels. We set out to clarify this in a case-control study.

Of 139 patients consecutively admitted to the emergency unit at the Teikyo University Hospital following a suicide attempt, 121 were given a DSM–III–R diagnosis (American Psychiatric Association, 1987) of a functional psychosis, or personality or 'neurotic' disorder and were discharged alive. For comparison, we used two control groups: 96 patients consecutively admitted to the psychiatric ward in the same university hospital during the same period, who suffered a similar psychiatric condition but had no history of suicide attempt ('psychiatric controls'); and 54 randomly selected patients admitted to the emergency unit ('normal controls') suffering trauma, toxicosis, or burns. All normal controls were also alive at discharge. None were found to be on cholesterol-lowering drugs or to have a cancer.

Complete data were available for more than 87% of subjects. The data from blood samples obtained within 24 hours of admission were used for analyses. Analysis of covariance (ANCOVA) and logistic regression showed that 105 'cases' had a significantly ($F=8.08$, d.f.$=1$, $P=0.005$) reduced serum cholesterol compared with 88 'psychiatric controls' when sex, age (5-year bands), and diagnosis (schizophrenic spectrum group, bipolar disorders, depressive disorders, or personality and miscellaneous 'neurotic' disorders combined) were covariates and duration of illness, red blood cell counts, total protein, and triglyceride were also covariates. Thus, account was taken of the effects of treatment and nutritional status.

The adjusted mean cholesterol was 161.1 and 178.0 mg/dl for cases and psychiatric controls, respectively. A marginally significant ($P=0.046$) difference in cholesterol across the four diagnostic groups was found, but no significant interacting effect of case–control status and diagnosis on cholesterol was present. Among the cases, depressed patients had the relatively higher adjusted mean cholesterol level (164.5 mg/dl).

Subsequent logistic regression analysis, in which the same variables as the above ANCOVA were controlled for, showed that an increase in 10 mg/dl cholesterol reduced, by 20% (95% CI 8.9% to 29.7%), the likelihood of being a case compared with a psychiatric control. Even when only first-admission subjects were used, this effect (19% reduction; 95% CI 4.1% to 30.8%) remained significant. ANCOVA also revealed a significant difference ($P=0.041$) in cholesterol levels between groups for the first-admission series, while the diagnostic difference became less marked ($P=0.453$).

The ANCOVA allowing for sex, age, and physical condition at admission and red blood cell counts showed that 84 cases had a significantly ($P=0.013$) lower cholesterol level than the 53 normal controls. The logistic regression analysis, in which the same possible confounders as the above were controlled for, showed that an increase of 10 mg/dl cholesterol was associated with an 18% reduction (95% CI 4.6% to 30.0%) in the likelihood of being a case than a normal control.

Despite the fact that measuring serum cholesterol immediately after admission is a proxy to a built-in baseline, these results suggest that low cholesterol levels are associated with the risk of suicide attempt among psychiatric patients. This effect was not specific to depressive disorders, and it remained even when nutritional indicators were controlled for in the analyses.

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