is associated with high standardised mortality ratio may be different correspondingly.

The point really was to progress from the obvious fact that a standardised mortality ratio of 140 for disease A is not necessarily the same as a proportional mortality rate of 140 for disease B in an epidemiological sense. What West was telling us was that the similarity can be tested by examining the relation of diseases A and B with respect to total mortality.

Perhaps it was unfortunate to discuss this idea with examples we understand as well as smoking. However I think it is clearer when it is emphasised that to suggest that smoking does just bring forward someone's death from coronary heart disease by an average of 20 years is not to excuse cigarettes as a cause. Nor does it necessarily imply that such a relation is extenuated by age. They just become a different kind of cause than if the person died from lung cancer because of smoking, but otherwise would have died, years later, of something else. The notion is that without smoking such a person would only rarely die of lung cancer, even in 300 years.

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Low back pain and cancer

SIR,—Mr R P Cole (3 October, p 840) describes two patients in whom the diagnosis of testicular tumour was delayed because the presenting symptom was back pain. This particular diagnostic trap was described by Dr Cantwell and colleagues,¹ who identified para-aortic lymphadenopathy as the usual cause of the pain. We believe, however, that the more important lesson to be learnt is that any tumour can present in this way.

We have recently described 10 patients with cancer who presented with backache.² In our study the syndrome of persistent back pain, which was severe enough to prevent sleep and characteristically relieved by sitting forwards, accompanied malignant retroperitoneal lymphadenopathy. Young patients presenting in this way should be closely questioned for symptoms of weight loss and night sweats. Full examination is required to detect superficial lymphadenopathy and testicular or pelvic abnormalities. Spinal movements and radiographs are typically normal, but a raised erythrocyte sedimentation rate may be mistakenly attributed to spondylitis (as in Mr Cole's second case). Ultrasound scanning usually shows the retroperitoneal lymphadenopathy,3 although computed tomographic scanning is useful in difficult cases.

Potentially curable cancers, including leukaemia,⁴ lymphoma, and cervical and testicular tumours, can present with back pain in young people,² so an increased awareness of this syndrome could save lives.

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Tobacco and end stage diabetic nephropathy

SIR,—Drs Bemd Stegmayr and Folke Lithner (5 September, p 581) report an association between smoking and diabetic nephropathy in a small group of patients. Before a causal relation can be assumed, however, it is important to establish whether or not there were differences in other possible risk factors between the smokers and nonsmokers. In particular, were there any major differences in long term glycaemic control? Smokers may show differences in their attitude to their illness or in socioeconomic state, and these factors could, in turn, influence diabetic control.

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AUTHORS' REPLY,—Unfortunately there were no glycosylated haemoglobin (HbA_{1c}) measurements for some of the uraemic patients, but our impression from blood and urine concentrations of glucose in this group of patients was that there was often unsatisfactory metabolic control.

For the 22 controls, however, we calculated the mean HbA_{1c} value during the past seven years. There was a slight inverse correlation between the HbA_{1c} value and the latest measured creatinine clearance (n=22, r=-0.495, p<0.05, two tail). Student's *t* test for intergroup difference of HbA_{1c} concentration in controls who never smoked compared with those who were smokers or ex-smokers was not significant (*t*=1.165). The intergroup difference was significant (*t*=2.93, p<0.01) when mean HbA_{1c} values for controls with albuminuria of more than 50 mg/day (mean value=11.74, SD=1.60) were compared with those with less albuminuria (mean value=10.0, SD=1.05).

An arguable point is that diabetic patients who have never smoked may also have taken greater care of themselves in other respects, such as through metabolic and blood pressure checks. A previous case-control study of smoking and nonsmoking diabetics showed a higher prevalence of proliferative retinopathy and macroproteinuria in the smoking group, while there were no intergroup differences in hypertension or glycosylated haemoglobin values.¹

> Bernd Stegmayr Folke Lithner

 Mühlhauser I, Sawicki P, Berger M. Cigarette-smoking as a risk factor for macroproteinuria and proliferative retinopathy in type I (insulin-dependent) diabetes. *Diabetologia* 1986;29: 500-2.

Is schizophrenia a neurodevelopmental disorder?

SIR,—Professor Robin M Murray and Dr Shôn W Lewis (19 September, p 681) have proposed an interesting hypothesis for the aetiology of schizophrenia. This neurodevelopmental hypothesis is based on two notions: the high prevalences of a history of perinatal complications and of abnormalities on computed tomography in schizophrenic patients. They fail to mention, however, that neither of these two abnormalities is specific to schizophrenia,¹ and patients with severe affective disorders including young manic depressive and delusional depressive patients show abnormalities on computed tomography.²³ Moreover, the epidemiology of these abnormalities has not been ascertained. It is also uncertain from their article what type of schizophrenia they refer to in view of the enormous

controversy over its definition, with 10 different available definitions and the widely accepted notion that schizophrenia is a heterogenous disorder more recently subdivided into types 1 (acute) and 2 (chronic). It is difficult to reconcile such a hypothesis with the finding of similiar prevalence rates of schizophrenia in various countries, including developing countries reported in the World Health Organisation international pilot study of schizophrenia.

If perinatal complications are of causal importance then a higher prevalence of the illness might be expected in developing countries. More intriguing has been the finding that schizophrenia has a more favourable prognosis in developing countries, which has been attributed to the relative integrity of family and social networks in those communities.

The neurodevelopmental hypothesis is also difficult to reconcile with the genetic basis of this disorder, which the authors have strongly affirmed in their opening remarks. It is conceivable that the neurodevelopmental process might affect brain structures and functions that mediate the effects of genetic factors. The aetiology of temporal lobe epilepsy could serve as a model for an interaction between an early insult causing selective brain damage (febrile convulsion) and a genetic disposition: temporal lobe epilepsy has been associated with the development of schizophrenia-like psychoses, and temporal lobe epilepsy and schizophrenia are both associated with neuropathological changes affecting the temporal lobe.

An alternative hypothesis to explain the association of schizophrenia with perinatal complications and abnormalities on computed tomography would consider the possibility that selective brain damage is caused by perinatal complications and explains the scan abnormalities and cognitive deficits commonly found in chronic schizophrenia (type 2). This brain damage is a non-specific predisposing factor while the genetic component accounts for the positive symptoms of the illness (type 1). This hypothesis suggests that the negative symptoms of the illness are unrelated to schizophrenia itself but substantially contribute to its outcome: these cognitive deficits put the patient at a greater biological disadvantage and compound the degree of associated disability and handicap caused by the positive symptoms of the illness. After all, schizophrenia is "a notional concept in the inner eye of the beholder," construed in a social context, and the dogma of the positive/negative typology has been seriously questioned.

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Alcohol consumption in England and Wales

SIR,—Dr G C Dunbar and Mr D D V Morgan describe a survey using a quota sampling method in which interviews took place predominantly "on the street" and which purports to represent the pattern of alcohol consumption in England and Ξ

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