Ophthalmologists is more helpful and avoids confusion. This states:

The minimal visual field for safe driving is a field of vision of at least 120 degrees on the horizontal measured by the Goldmann perimeter using the III4e settings (or equivalent perimetry). In addition there should be no significant field defect in the binocular field which encroaches within 20 degrees of fixation either above or below the horizontal meridian. By these means homonymous or bitemporal defects which come close to fixation, whether hemianopic or quadrantanopic, are not accepted as safe for driving. Isolated scotomata represented in the binocular field near to the central fixation area may also be inconsistent with safe driving.²

This definition is incorporated in the forthcoming revised edition of the booklet *Medical Aspects of Fitness to Drive*³ and is thought to be the best interpretation of the standard as it is applied to individual patients.

ANDREW R ELKINGTON
President

Royal College of Ophthalmologists,
London NW1 4QW

1 Potamitis T, Aggarwal RK, Tsaloumas M, Rene C, McLaughlin J, O'Neill E. Driving, glaucoma, and the law. *BMJ* 1994;309:1057-8. (22 October.)

2 Royal College of Ophthalmologists. *Definitions of the minimum field of vision for safe driving for group 1 ordinary drivers*. London: RCP, 1994.

3 Medical Commission on Accident Prevention. *Medical aspects of fitness to drive*. London: MCAP (in press).

Childhood antecedents of schizophrenia

Difference in antecedents between sexes may not be genuine

EDITOR,—In their study of the 1958 birth cohort of the national child development study D John Done and colleagues suggest that there may be significant differences between the sexes in the childhood antecedents of schizophrenia.¹ They base their conclusion on the finding that, despite a similar behavioural profile for both sexes at age 7, by age 11 the girls showed an appreciable tendency towards underreaction (social withdrawal and isolation), which was not seen in the boys.

There seem to be at least three possible ways to interpret this finding. Firstly, as the authors suggest, there may be a genuine difference in antecedent behavioural risk factors between the sexes, with social underreaction being confined mainly to preschizophrenic girls.

Secondly, the difference between the sexes at age 11 may be a transient phenomenon related to the earlier onset of behavioural underreaction in girls compared with boys. For example, Bettles and Walker have shown that negative psychotic symptoms such as social isolation and inappropriate affect are more prevalent in girls than boys at age 11-12.² By age 15-16, however, rate of negative symptoms in boys are equal to those in girls, and by late adolescence the rates have become higher in boys.

Finally, the symptoms of behavioural underreaction at age 11 may represent the first signs of an emerging schizophrenic illness. In this case one would predict that there would be more girls than boys among the subjects with the very earliest onset of schizophrenia in the study. Clearly, this would challenge the general assumption of an earlier onset of schizophrenia in male subjects.

An important message from Done and colleagues' study is that the behavioural antecedents of schizophrenia are not static but change with age. The question of whether the difference in antecedents between the sexes is genuine or transient needs to be resolved by study of the subjects at a third time point—namely, the 1974 follow up at age 16. Knowledge of the temporal relation between the onset of behavioural underreaction and the onset of schizophrenia would also

help to resolve the question of whether behavioural underreaction should be considered to be an antecedent risk factor or a prodrome of schizophrenia.

CHRIS HOLLIS

Medical Research Council training fellow

Department of Child and Adolescent Psychiatry,
Institute of Psychiatry,
London SE5 8AF

1 Done JD, Crow TJ, Johnson EC, Sacker A. Childhood antecedents of schizophrenia and affective illness: social adjustment at ages 7 and 11. *BMJ* 1994;309:699-703. (17 September.)

2 Bettles BA, Walker E. Positive and negative symptoms in psychotic and other psychiatrically disturbed children. *J Child Psychol Psychiatry* 1987;28:555-68.

Environmental influences should not be ignored

EDITOR,—D John Done and colleagues claim that there is no strong evidence from twin, family, and adoption studies of environmental factors having a role in schizophrenia.¹ I am surprised that they are not aware of the many publications of Tienari and colleagues in Finland.^{2,3} Their nationwide studies evaluated the mental health of the families into which children whose biological parents had schizophrenia were adopted. Healthily functioning families had a significant protective effect on the prevalence of serious psychopathology and psychosis in the adopted children. Done and colleagues' dismissal of environmental contributions to schizophrenia may have led to their not considering evaluating the psychological health of the families in which they detected childhood antecedents of schizophrenia and affective illness.

The tendency to ignore environmental factors in conditions with a genetic component reminds me of the situation in Huntington's disease, known since 1872 to be a genetic condition. For years considerable resources have been directed at trying to identify the mechanism by which the (now located) gene causes its damage. This effort was not, however, balanced by any attempt to pursue the possibility of environmental and family factors having a role in the perpetuation of the disease. One hundred and thirty years after Huntington's discovery only a few professionals have begun to provide simple systematic information and counselling services for those at risk of Huntington's disease.⁴ Some of the results are striking: those counselled about their genetic vulnerability reduced up to fivefold the number of babies born with the gene.

I do not suggest that focusing on the influence of the family on those with a genetic predisposition to schizophrenia should have a similar aim or outcome. I wish, however, to make the point that when scientific evidence about environmental influences on schizophrenia is selectively ignored a similarly tragic situation is perpetuated whereby preventive psychotherapeutic interventions for these environmental factors are not even considered in the present, while we excessively emphasise the hope in the future from genetic and biological studies.

BRIAN MARTINDALE

Consultant psychiatrist in psychotherapy

Parkside Clinic,
London W11 1QG

1 Done DJ, Crow TJ, Johnstone EC, Sacker A. Childhood antecedents of schizophrenia and affective illness: social adjustment at ages 7 and 11. *BMJ* 1994;309:699-703. (17 September.)

2 Tienari P. Interaction between genetic vulnerability and family environment: the Finnish adoption family study of schizophrenia. *Acta Psychiatr Scand* 1991;84:460-5.

3 Tienari P. Interaction between genetic vulnerability and rearing environment. In: Werbart A, Cullberg J, eds. *Psychotherapy of schizophrenia: facilitating and obstructive factors*. Oslo: Scandinavian University Press, 1992:154-79.

4 Martindale BV, Yale R. Neglected opportunities for preventive medicine. *Lancet* 1993;341:634-6.

5 Harper PS, Tyler A, Smith S, Jones P, Newcombe RG, McBroom V. Decline in the predicted incidence of Huntington's chorea associated with systematic genetic counselling and family support. *Lancet* 1981;ii:411-3.

Conclusions not supported by data

EDITOR,—D John Done and colleagues' conclusions—that abnormalities of social adjustment are detectable in childhood in some people who later develop psychotic illness—seem to go well beyond the data presented.¹ It is difficult to see how comparing mean scores of groups at only two ages can reasonably lead to the conclusion that the rate of development of different components of the capacity for social interaction is an important determinant of the risk of psychosis and other psychiatric disorders in adulthood. The authors do not seem to have measured rates of change in individual children or to have attempted to correlate them with the risk of psychiatric disorders.

The authors' generalisation that "even at the age of 7 subjects who will later develop schizophrenic illness differ from schoolmates in the eyes of their teachers" is also questionable, especially as it implies that they are a recognisable group. The number of subjects who were later admitted to hospital with schizophrenia and for whom social adjustment scores were available at age 7 was fairly small (33). Also, although this group's mean scores were higher than those of the control group, little detail of the individual scores of children is provided.

The authors state that half the boys in this group were considered to be as deviant as the most deviant tenth of the school population. Even if all those who later developed schizophrenia had been among the most deviant tenth of 7 year olds, however, the risk of a child in the most deviant tenth developing schizophrenia by the age of 28 would have been only about 1 in 40 (using the authors' figure that 40 out of over 15 000 subjects in the national child development study had been admitted to a psychiatric hospital with schizophrenia by this age).

JOHN TEMPLE

Part time lecturer in general practice

Department of General Practice,
Medical School,
Queen's Medical Centre,
Nottingham NG7 2UH

1 Done DJ, Crow TJ, Johnstone EC, Sacker A. Childhood antecedents of schizophrenia and affective illness: social adjustment at ages 7 and 11. *BMJ* 1994;309:699-703. (17 September.)

Authors' reply

EDITOR,—John Temple suggests that our data do not permit us to discuss rates of development. We agree that extrapolating from two datapoints to derive a statement about rate is unwise. We do, however, have additional information—namely, that psychotic behaviour was documented in adult life. The children did not manifest psychotic symptoms, but these symptoms developed some 15 or more years later, rather than emerging spontaneously. In the development of cognition (for example, problem solving) qualitative changes occur at different ages, and thus the rate of development cannot be explained by a numerical statement. We suggest that the early abnormalities of social interaction that we reported represent a state in the abnormal development of the capacity for social interaction which culminates in psychotic behaviour. To regard the results simply as potential predictors or markers of a later pathological event seems blinkered. Delusions, hallucinations, and the other phenomena of psychosis have remained isolated from any acceptable theory of psychological functioning. As a result they remain unexplained phenomena. We suggest that an alternative approach, based on analysis of the pattern of development of these symptoms, may prove to be more illuminating.

Our other point concerns the practical relevance of our findings. The lifetime risk of schizophrenia is about 8 per 1000, and any behavioural measure