Correspondence

EDITED BY MATTHEW HOTOPF

Advising relatives of risk of Alzheimer's disease

Liddell *et al* (2001) reviewed what knowledge we have on the genetic epidemiology of Alzheimer's disease for the purpose of informing relatives of patients about their own risks. We read their review with interest; however, we disagree on several points.

First, in many cases of late-onset dementia, differentiating between the common causes of Alzheimer's disease and vascular dementia is difficult. In everyday clinical practice even differentiating Alzheimer's disease from Lewy-body disease and frontal–temporal dementia is not always feasible. To what extent these distinctions are relevant to genetic counselling with respect to late-onset dementia is not clear.

Second, the very high prevalence of dementia found in centenarians (Asada *et al*, 1996; Blansjaar *et al*, 2000) is not the only argument against a slowing down of the rate of increase in dementia over 85, 90 or 95 years of age. Meta-analyses, not included in the review, did not find evidence for such a slowing down (Gao *et al*, 1998; Jorm & Jolley, 1998). Therefore, the prevalence of dementia almost certainly increases substantially, exceeding 15% from the age of 85.

Most investigations attributed some three-quarters of late-onset dementia to Alzheimer's disease. We agree that the literature indicates a three- to fourfold risk in first-degree relatives of patients with late-onset dementia (seven- to eightfold with two affected first-degree relatives). We can only conclude that this leads to a risk of one in three, if not higher, for those first-degree relatives who reach the age of 85 years. Obfuscating this information by showing graphs to anxious relatives is, in our opinion, not an appropriate reassurance. We feel that better consolation can be effected by proffering the view that most people do not reach the age of 85, and by explaining the slowly progressive course of most cases of late-onset dementia.

Asada, T., Yamagata, Z., Kilnoshita, T., *et al* (1996) Prevalence of dementia and distribution of ApoE alleles in Japanese centenarians: an almost-complete survey in Yamanashi Prefecture, Japan. *Journal of the American Geriatrics Society*, **44**, 151–155.

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Gao, S., Hendrie, H. C., Hall, K. S., *et al* (1998) The relationships between age, sex, and the incidence of dementia and Alzheimer's disease: a meta-analysis. *Archives of General Psychiatry*, **55**, 809–815.

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Authors' reply: For the most part, the points of disagreement that Drs Blansjaar and van Schaick raise are differences more of emphasis than of substance.

True, the diagnosis of the type of dementia, particularly in late old age, is not always easy. Dementia in later life is probably best described as a syndrome, the emergence of clinical dementia being dependent upon the interplay of two or more pathologies. The 'Nun Study' by Snowdon *et al* (1997) is probably one of the best demonstrations of this. Yet, it is believed that Alzheimer's disease is a major cause of dementia in later life. Even without having seen the patient, one is going to be correct in a diagnosis of Alzheimer's disease, or Alzheimer's disease and cerebrovascular disease, 75% of the time. Rarer diagnoses, such as frontal–temporal dementia and Lewy-body disease, should suggest themselves if they are kept in mind, a careful history taken and the patient followed-up so that departures from the normal symptom progression for Alzheimer's disease are noted. Of course, mistakes in diagnosis will occur, but we think that this will occur insufficiently frequently to compromise the very broad-brush approach to estimating the familial risk of dementia that we have advocated.

As to whether the rate of increase in the incidence and prevalence of dementia begins to slow or goes on increasing exponentially into extreme old age, this is a controversial area, which is, in fact, also highlighted in the two meta-analyses cited by Drs Blansjaar and van Schaick. Jorm & Jolley (1998) suggest that "the incidence rises exponentially up to the age of 90 years". Gao *et al* (1998) suggest that "the acceleration of incidence rates for AD and dementia slows down with the increase in age, although we find no evidence of a rate decline". Faced with such difficulties of interpretation, we can only commend the clarity of Blansjaar *et al*'s own study (2000), which suggests that the increase in dementia prevalence does not slow down in extreme old age.

We agree that the risk of a first-degree relative of a proband with Alzheimer's disease developing the disorder once they reach the age of 85 may be one in three, if not higher. Perhaps this point could have been made more clearly in our review. The main point we tried to make was that the actual likelihood of surviving to age 85 *and* developing Alzheimer's disease is lower. We disagree that showing graphs to anxious relatives is "obfuscating this information", but we accept that Drs Blansjaar and van Schaick and, indeed, other clinicians may think differently.

In non-Mendelian Alzheimer's disease it is difficult to estimate how much the risk increases as the number of affected first-degree relatives goes up, principally because few studies have addressed this issue. However, the 'conjugal Alzheimer's disease' study of Bird *et al* (1993), which we cited, and the transmission study of Farrer *et al* (1990), which we did not cite, indicate that the risk increases substantially. With such pedigrees showing apparently high genetic loading for Alzheimer's disease, we suggested that a psychiatrist seek the advice of a clinical geneticist.

Finally, we agree that it is often reassuring to point out that the course of dementia in late old age is usually more slowly progressive and more benign than dementia occurring in a younger person.

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Treatment for Alzheimer's disease in people with learning disabilities: NICE guidance

In January 2001 the National Institute for Clinical Excellence (NICE) published *Guidance on the Use of Donepezil, Rivastigmine and Galantamine for the Treatment of Alzheimer's Disease*. The guidance indicates that the drugs should be made available within the National Health Service to people with mild to moderate Alzheimer's disease whose mini-mental state examination (MMSE) score is above 12 points. The Institute's guidance does not mention the use of anti-dementia drugs in people with learning disabilities and Alzheimer's disease. Studies have shown that the prevalence of Alzheimer's disease in those with learning disabilities is higher than in the normal population (Patel *et al*, 1993). This is likely to increase in the future because of the rising life expectancy of people with learning disabilities (Zigman *et al*, 1997). In Down's syndrome, approximately 40% develop dementia of Alzheimer type by the age of 60 (Holland *et al*, 1998).

It is known that clinical evidence for the effectiveness of various psychiatric treatments in the learning disability population is scanty and specialists rely on evidence from the normal population. In this situation, a specialist in the psychiatry of learning disability might consider following the NICE guidance in treating dementia in the people under his or her care. However, there is a major problem, as NICE guidance suggests that treatment should be monitored by MMSE score but the MMSE cannot be used reliably in people with learning disabilities (Deb & Braganza, 1999). This means that NICE guidance on the use of anti-dementia drugs is not applicable to people with learning disabilities. This is likely to discourage specialists from prescribing treatment for some patients with a learning disability and Alzheimer's disease who may benefit from it in future. In its guidance, NICE mentioned limitations on the use of the MMSE in people whose Alzheimer's disease is complicated by dysphasia and whose first language is not English, but failed to identify that the MMSE is not standardised for people with learning disabilities who make up 2% of our population. The fact that this group of people, with a high prevalence of dementia, was completely ignored within the guidance is quite worrying. We appreciate that the guidance from NICE is not prescriptive and does not replace individual judgement; however, complete omission of learning disability could potentially exclude people from receiving beneficial treatment.

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Community care for mental disorders in developing countries: a perspective

Given the limitations of the existing model of community care for mental disorders in developing countries, Jacob (2001) has tried to construct another model and has focused on some of the constituent elements of such a model. Although Jacob insists on a potentially innovative approach to the provision of mental health services in developing countries, the framework within which to take forward the debate regarding community care fails to analyse in depth the sociopolitical and economic contexts in which community care is constructed. Owing to the strong emphasis placed upon discriminatory social and political structures, an analysis of what it is to be mentally ill, and the sociological and psychological implications of this, has largely been ignored.

I agree with Jacob that cooperation between governments and non-governmental organisations (NGOs) in providing community care will help in implementing health care policies. However, by their very nature, NGOs are heterogeneous and vary from large agencies operating in many countries (e.g. Oxfam, Save the Children Fund) to very small organisations operating at village level. Despite the growth of NGO activity in the past decade, there remain questions regarding their effectiveness in achieving their stated objectives (Nyoni, 1987). Evaluation of an NGO's effectiveness can become something of a propaganda exercise, aimed more at impressing donor agencies than at a critical analysis of the NGO's activities. A related issue concerns the mixed accountabilities of NGOs – 'downwards' to their collaborating partners and 'upwards' to their donor agencies. These issues result in difficulties of monitoring and enforcement (Brett, 1993).

We know that the lives of individuals with mental illnesses around the world are usually limited far more by prevailing social, cultural and economic constraints than by their illnesses. If this is the case, then the issues related to community care for people with mental disorders move from those of health to those of human rights. Their lives are hard indeed. Mental health professionals can help to change this state of affairs. Whether the issue is community care in urban London or in rural India, professionals who work on mental

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