Matters arising

Could Parkinson's disease follow intra-uterine influenza? A speculative hypothesis

Sir: We read with interest the speculative hypothesis advanced by Mattock *et al.*,¹ proposing an intrauterine influenza infection as the possible aetiology of Parkinson's disease.

We feel that this hypothesis, although intriguing and obviously requiring further substantiation, appears to be basically flawed by already existing data from the various twin studies in Parkinson's disease.²⁻⁵ These studies designed to examine concordance for Parkinson's disease in mono and dizygotic twins have demonstrated that the concordance rate for monozygotic twins is extremely low. This evidence confirmed by several studies indicates that genetic factors do not play a prominent role in the development of Parkinson's disease.

The data from these studies can also be used as an argument against the intrauterine infection hypothesis of Parkinson's disease since it seems very unlikely that an intrauterine infection would affect only one of two twins sharing the same environment. Although congenital malformations have been reported in one of two monozygotic twins,³ the incidence of a viral infection affecting only a single twin, we reason, must be very low.

We can look, for example, at congenital cytomegalovirus infection in twins. CMV is the commonest cause of congenital infection in man with an incidence ranging from 0.5–2% of all live births.⁶ Of these infected infants, 10% will suffer from mental retardation.⁷ Twins with CMV infection are des-

cribed to usually have concordant clinical or laboratory evidence of disease.⁸ There are no reports of monozygotic twins where only one of two CMV infected twins is symptomatic or where only one twin of the set is actually infected.⁸ The latter situation, although possible, is the least common and these cases reported are either dizygotic twins or situations suggestive of dizygotic twins (two placentas).⁹

In the case of herpes simplex virus, perinatal infection with HSV will also affect both twins in a clear majority of cases.⁹

Toxoplasmosis, a non-viral but also haematogenously spread intrauterine infection, similarly has a distinct difference in clinical patterns of infection between monozygotic and dizygotic twins.¹⁰ In monozygotic twins the clinical pattern of involvement tends to be similar in both twins. In dizygotic twins, discrepancies in clinical findings are more frequent and marked. However, only in a minority of dizygotic twins is one of the twins exclusively infected.

If an intrauterine influenza infection predisposed an individual to develop Parkinson's disease, then it would appear, based on the pattern of viral and nonviral intrauterine infections, that monozygotic twins (and perhaps even dizygotic twins) should have a high rate of concordance for Parkinson's disease. Since the concordance rate for Parkinson's disease is only 2–5% in monozygotic twins,³ the hypothesis of Mattock *et al.*¹ seems to be an error and there may be some other reason for the relationship they described between year of birth and crude mortality data for influenza.

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Book reviews

Neurobiology of Amino Acids, Peptides and Tropic Factors. Edited by James A Ferrendelli, Robert C Collins, Eugene M Johnson. (Pp 272; £44.95.) Dordrecht: Martinus Nijhoff, 1988.

Advances in neurobiology over the past decade have radically altered our concepts of how the nervous system works. Previously undiscovered neurotransmitters or neuromodulators, such as the excitatory amino acids and neuropeptides have been recognised. The plasticity of the nervous system also has been realised as has the potential for trophic factors to prevent

neuronal cell death and to induce regeneration. These are now key areas fundamental to the development of therapy for nervous system disorders in the future.

This book represents the proceedings of a meeting held in the Autumn of 1986. It was the third symposium in a series exploring communication between the CNS and other body systems so as to monitor frontiers of research into neuronal and hormonal regulatory interactions and their application to medicine. The content is a combination of contributions on the roles of excitatory amino acids, trophic factors and neuropeptides.

I found the mixture of topics made it difficult to define a central theme for the volume. Indeed, the chapters themselves in

the various sections vary from good overviews of a selected area to highly specific research contributions. Indeed, with notable exceptions such as the chapters by Olney, Meldrum, Johnson and Bloom, most appeared to miss the basic concept of the meeting. This is not a criticism of any individual contribution since most chapters were themselves excellent in their own way. It is a criticism of the manner in which the volume has been conceived. Most disturbing are the series of short communications at the end of the volume. Here the reader is presented with 13 contributions of two to five pages in length, none of which can possibly convey a clear insight of the topic to the reader. I also dislike the 31 pages given over to the preface, contents, list of participants and dedication.