# Correspondence

## ALZHEIMER'S AS A COMMON COMPLEX DISEASE.[1]

*To the Editor:*

Certain common diseases have a genetic predisposition in addition to established environmental factors. This may be seen as a slight but worrying increase in the family of a patient which does not, however, follow traditional Mendelian laws. Examples would be insulin-dependent diabetes, coronary artery disease and hyperlipidaemia and some cancers.[1]

With recent advances in clinical and molecular genetics, it is apparent there is a similarity in the genetic background of these conditions. They have been grouped together as the "common complex diseases" with the adjective "complex" referring to the genetic factors.[1]

There is the previously mentioned clustering in the relatives of a case but some few families can also be located in which the disease is more definitely heritable. In them, Mendel's laws do apply and they are amenable to molecular genetic studies. Other similarities are population studies associating the disease with a known genetic marker or linking it to a specific allele.

Consider Alzheimer's disease. It has all of these characteristics. There is a familial form in which 3 gene loci have been identified (heterogeneity).[2] Clustering can be seen in relatives. An association with other chromosomal illnesses has been shown (Down's syndrome).[3] There are links to the human leucocyte antigen (HLA) and the major histocompatibility complex (MHC).[4,5] Perhaps most significantly, amyloid has been reproduced in the brains of transgenic mice.[6]

Why consider Alzheimer's as a common complex disease? Advances in molecular genetics are spectacular and what is discovered for the one might well have application for all. These diseases likely have a spectrum of causes totally hereditary on one end and totally environmental on the other. The worrisome familial clustering is somewhere in the middle and likely draws from both sides in its etiology.

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## THE SHORT HISTORY OF FOCAL BRAIN DEGENERATION

*To the Editor:*

Attig and colleagues have contributed an interesting case of posterior cortical atrophy to the growing literature on focal brain degeneration (Can J Neurol Sci 1992; 20: 154-157). They stated that primary progressive aphasia has been described originally by Mesulam in 1982 and suggest the term "Mesulam syndrome".

Arnold Pick has described several patients with prominent aphasia in the context of degenerative brain diseases, for example Anna Jirinec who had an accentuated atrophy of the left temporal lobe. In 1988 Poeck and Luzzatti[1] listed 6 descriptions of slowly progressive language disturbance published until 1940. In 1926 Onari and Spatz[2] reviewed 9 cases with temporal lobe atrophy.[2] In 1913 Mingazzini[3] discussed patients with focal brain degeneration described by Alzheimer, Ascher, Bischoff, Dejerine and Serieux, Franceschi, Liepmann, Marie and Leri, Mills, Pick, Rosenfeld, Shaw, and Stransky. Paradoxically the history of focal brain degeneration appears to get shorter with time instead of longer.

There is little scientific merit in smart but pedantic remarks on the "earliest description of ...". On the other hand, the struggle against unnecessary eponyms is one of the noblest goals in medicine. Therefore I hope that Attig et al. will tolerate this historic note.

By the way, posterior cerebral atrophy (the "De Renzi - Benson syndrome") has originally been described by Rosenfeld in 1909[4] – I think.

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