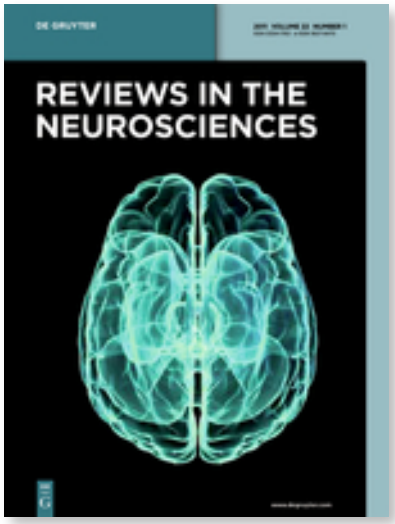

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The Neuroanatomy of Alzheimer's Disease

R.C.A. Pearson, / T.P.S. Powell,

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The Neuroanatomy of Alzheimer's Disease

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ABSTRACT

It is proposed that Alzheimer's disease is initiated by entry of some environmental factor into the brain via the olfactory pathway and that this factor spreads through the brain from neuron to neuron along identified axonal connections. The proposed agent may be a virus or virus-like particle. The characteristic pathological changes, neurofibrillary tangles and neuritic plaques, are not confined to the cerebral cortex, nor are they evenly distributed within the cortex. Their distribution within subcortical nuclei suggests that neurons which project to affected regions of cortex often contain tangles, those which receive fibres from such regions usually contain plaques, and nuclei having reciprocal connections with the cortex commonly contain both plaques and tangles. Nuclei with no cortical connections rarely show significant pathological change. Within the cortex, the major pathological changes centre on regions in the medial temporal lobe, including the hippocampus, amygdala, entorhinal cortex and parahippocampal gyrus. Areas of association cortex in the parietotemporal and frontal lobes which have fibre connections with these heavily affected areas are involved to an intermediate degree, and areas without corticocortical connections with the medial temporal cortex are much less affected by the disease process. Unlike the other sensory pathways, the olfactory system is severely affected in Alzheimer's disease, and pathology in parts of the brain related to olfactory pathways may be amongst the earliest changes in the disease. The etiology of Alzheimer's disease has a genetic component, which is accentuated in aging and in Down's syndrome. Such a genetic predisposition might be a defect in immune-competence, permitting entry of the proposed agent into the brain from the nose. It is also suggested that the presence of this agent within neurons causes a failure of the normal protein synthetic machinery of the cell with consequent abnormal post-translational processing of the amyloid precursor protein, leading to deposition of the amyloid peptide and the formation of plaques and tangles.

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INTRODUCTION

Dementia is "an acquired global impairment of intellect, reason and personality but without impairment of consciousness" /1/. The clinical symptomatology is considered to be "the result of a more or less extensive destruction or disorganization of the cerebral cortex" /1/. The most common cause in adults of any age is the Alzheimer's type of degeneration, accounting for over 50% of all dementia in the Western world /1-3/. As the average life expectancy increases, the number of people suffering from this disease is increasing, affecting between 5 and 10% of people over 65 /2,3/. Such a prevalence,

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