alpha-emitting and other radionuclides following environmental exposure is poorly known, with the exception of hot particles in lung tissue arising from the decay products of radon-222.

Analysis of gross tissues or organs is unlikely to be useful if a particular radionuclide is associated with a small area or volume of cells. Possible biochemical pathways of interest are the trapping of rare earths, such as cerium by phosphate ions which are released by phosphatases and an association with the intracellular protein ferritin.

So far there is no indication that localization of heavy radionuclides in human testes occurs; concentrations of other conservative radionuclides present in the systemic circulation are unlikely to be of concern in relation to uniform distributed dose received by the testis. However, an inert element, such as thorium, can be detected in human blood and urine and is enriched in bone for occupational and non-occupational exposure[4].

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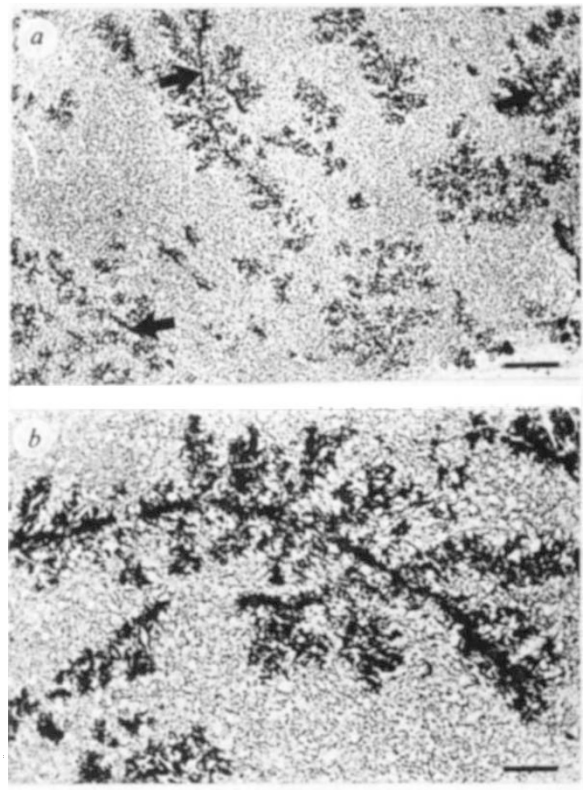
Cortical β-amyloid

SIR—The presence of amyloid β-protein, the essential component of the amyloid substance found in the brains of patients with Alzheimer's disease[1], has been demonstrated by a variety of immunohistochemical techniques[2]. More recently, amyloid β-protein has been found in tissues other than brain (for example, skin, subcutaneous tissue and intestine), in conformity with the hypothesis that there is a circulating form of the β-protein[3].

The improved sensitivity of the immunohistochemical techniques that results from pretreatment of nervous tissue with formic acid, has revealed amyloid deposits that have infiltrated the grey matter of the cortex of patients with Alzheimer's disease[4]. We have achieved a further improvement of sensitivity in the detection of amyloid deposits by a technique involving pretreatment with periodic acid, the effect of which is to remove saccharides, which are known to interact strongly with all amyloid susbstances[5].

Using this technique, we have been able to demonstrate amyloid substance in the immediate vicinity of capillaries in the cortical white matter of patients with Alzheimer's disease (see figure). To our knowledge, this is the first report of amyloid β-protein in cortical white matter. The observation agrees with the hypothesis that amyloid β-protein is vascular in origin.



Amyloid deposits in the white matter of the occipital cortex of a patient with Alzheimer's disease who died at 75 years are revealed by immunolabelling with antibodies against synthetic peptides. Amyloid deposits associated with capillaries are denoted by arrows. Scale bars: *a*, 100 μm; *b*, 50 μm. Tissue was fixed in Carnoy's mixture and paraffin sections were treated with periodic acid before immunolabelling[5]. Similar observations were made with two other patients who had died at 77 years, but no amyloid deposit was detected in the cerebral white matter of three aged controls who died at 80 years.

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Collagenous proteins multiply

SIR—The collagen stalk motif of three amino-acid residues, Gly-X-Y, has been found not only in extracellular matrix collagens but also in other eukaryotic secretory proteins such as complement C1q, pulmonary surfactant apoprotein, asymmetric acetylcholine esterase and serum mannose-binding protein. Now it has also been demonstrated that the motif is used in a non-secretory protein — the macrophage scavenger receptor[1–3], a membrane protein with important implications in atherosclerosis.

We are concentrating on studies of bacterial viruses with a membrane[4]. One of these, the *Escherichia coli* bacteriophage PRD1, has an external icosahedral protein coat, composed of major and minor capsomers, surrounding the viral membrane. We have very recently discovered that the minor capsomer protein (34.3 K) forms an asymmetric homotrimer where amino-acid sequence, deduced from the DNA sequence of the minor capsid protein gene, reveals a carboxy-terminal domain of 199 amino acids including all the three cysteine residues of the protein, and an amino-terminal domain 123 amino acids long.

These two domains are separated by a short collagen-like region with 6 continuous Gly-X-Y triplets. Four out of the 6 Y amino acids are proline or lysine. In addition, microbial collagenase cleaves the protein into the carboxy- and amino-terminal domains[5].

We believe that this trimeric minor capsomer of PRD1 has a structural role and that the short collagen-like motif assures the multimerization needed to satisfy the symmetry requirements. Our finding suggests that this peptide motif is evolutionarily old, and that it may be found in a number of trimeric proteins.

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