

Aluminium and Alzheimer's disease

A number of environmental factors have been put forward as possible contributory causes of Alzheimer's disease in some people. Among these is aluminium. There is circumstantial evidence linking this metal with Alzheimer's disease but no causal relationship has yet been proved. As evidence for other causes continues to grow, a possible link with aluminium seems increasingly unlikely. This information sheet looks at the circumstantial evidence and current medical and scientific views.

Researchers believe that, in the majority of those affected, Alzheimer's disease results from a combination of different risk factors rather than a single cause. Such factors, which vary from person to person, may include age, genetic predisposition, other diseases or environmental agents.

The chief symptoms of Alzheimer's disease are progressive decline of memory and other higher mental functions. These changes are associated with the loss of brain cells and the development of two kinds of microscopic damage in the brain - the so-called plaques and tangles.

Plaques consist of an abnormal deposit of a particular protein called beta amyloid between the brain cells. Tangles occur within cells and are formed from abnormal thread-like deposits of a protein called tau, which is normally part of the cell's 'skeleton'.

For a discussion of other factors see the Alzheimer's Society information sheets [Am I at risk of developing Alzheimer's disease?](http://alzheimers.mithril.jadu.co.uk/factsheet/450) and [Genetics and Alzheimer's disease](#).

Evidence linking aluminium and Alzheimer's disease

The 'aluminium hypothesis' was first put forward in 1965 when it was shown that the injection of aluminium compounds into rabbits caused tanglelike formations in nerve cells. However, these experimental tangles differ in structure and composition from Alzheimer tangles and the human brain. Since then a number of other circumstantial links between aluminium and Alzheimer's disease have been claimed.

- Aluminium has been shown to be associated with both plaques and tangles in the Alzheimer brain. Some groups have disputed these claims and, in any case, the presence of aluminium does not prove a causal relationship - it is more likely to be a harmless secondary association.
- It has been claimed that the brain content of aluminium is increased in Alzheimer's disease. However, recent studies in which Alzheimer brains were carefully compared with normal brains failed to find any difference in the overall amount of aluminium.
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- Studies of other sources of aluminium such as tea, antacid medications and antiperspirants have also failed to show a positive association with Alzheimer's disease.

- People with kidney failure are unable to excrete aluminium and yet they frequently have to be treated with compounds that contain aluminium. Studies of the brains of such patients have shown that aluminium accumulates in nerve cells that are particularly vulnerable in Alzheimer's disease. However, even after years of high exposure to aluminium, patients with kidney failure do not develop dementia or the hallmark pathological changes of Alzheimer's disease.
- Treatment with desferrioxamine (DFO), a drug which binds aluminium and removes it from body tissues, has been reported to slow down the mental decline in patients with Alzheimer's disease. However, the effect is small, the drug has to be given by injection into muscle and it also has a major effect on iron stores in the body. Since there is evidence that iron is involved in age-related 'oxidative' damage to tissues, the effects of DFO may have nothing to do with aluminium.
- There have been many experimental studies on animals and on isolated cells showing that aluminium has toxic effects on the nervous system, but in almost all cases the doses of aluminium used were much higher than those occurring naturally in tissues.

Sources of aluminium

The main sources of environmental aluminium are:

- Food - many foods contain small amounts of aluminium.
- Packaging - food may come into contact with aluminium through packaging or using aluminium foil or trays for freezing, storing or cooking. However, the amount of aluminium added to food in this way is usually negligible.
- Pans - cooking in uncoated aluminium utensils can increase the amount of aluminium in certain foods such as fruits which are high in acid. Cooking foods in coated, non-stick or hard anodised aluminium pans adds virtually no aluminium to food.
- Medicines - many antacids used for treating indigestion contain large amounts of aluminium compounds but normally little of the aluminium is absorbed.
- Water - aluminium is naturally present in some water and, in addition, aluminium sulphate is widely used in the treatment of public water supplies. However, intake of aluminium from water is very small in comparison with other sources.
- Air - some aluminium from the air may enter the lungs as dust but this form is highly insoluble and hardly any reaches the rest of the body.

Only a minute proportion of the aluminium we ingest from these various sources is absorbed by the body, and even this small fraction is usually excreted in the urine or harmlessly deposited in bone which acts as a 'sink' to remove aluminium. So effective are these mechanisms that it is estimated that the adult human body contains 30-50mg of aluminium - far less than the amount in a single antacid tablet!

The expert view on aluminium

There have been numerous conferences on aluminium and health ever since the idea that the metal might be a risk factor for Alzheimer's disease was first proposed. The medical research community, international and government regulatory agencies and the aluminium industry all review the evidence at frequent intervals. The overwhelming medical and scientific opinion is that the findings outlined above do not convincingly demonstrate a causal relationship between aluminium and Alzheimer's disease, and that no useful medical or public health recommendations can be made, at least at present.

It has proved extremely difficult to devise studies which could resolve this problem one way or another. Alzheimer's is a common disease with multiple causes, while aluminium is widespread in the environment and there are no methods that allow us to measure an individual's 'body burden' or lifetime exposure to this element.

It is possible that suitable 'transgenic' animal models which develop the pathological features of Alzheimer's disease in their brains will enable scientists to determine if such changes are accelerated or exacerbated by aluminium at levels which correspond to normal human exposure.

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