Factsheet: Professor Emeritus David Smith’s Lecture on Dementia

Professor Emeritus David Smith of the University of Oxford - 3rd February 2016, AMRF Lecture Room, University of Auckland.

Who is Professor Emeritus David Smith?

Professor David Smith of the University of Oxford, has worked at the frontiers of neuroscience for more than 50 years. In particular, he has been at the forefront of research into novel treatments and techniques for Alzheimer’s disease and Dementia.

What was the focus of the lecture?

Professor Smith posed one provocative question: Can we prevent Alzheimer's Disease? The answer, presented with supporting evidence, was a resounding “yes we can!” During his lecture, he alternated between calling the problem ‘Alzheimer’s’ and ‘dementia’, because the most common kind of dementia is Alzheimer’s disease.

Facts about dementia

Each year in NZ about 15,000 elderly people develop dementia. That’s 40 every day. Almost twice as many develop mild cognitive impairment, or memory impairment, which is the precursor of Alzheimer’s disease. About half of them will go on to develop dementia.

Dementia increases in prevalence with age

Alzheimer’s is associated with very specific changes in the brain that are not found in everyone. The primary marker for Alzheimer’s is the presence of neurofibrillary tangles, which are aggregates of hyperphosphorylated tau protein.

The tangles are not spread through the whole brain but in certain specific areas, for example the hippocampus, which is involved in memory. Affected regions of the brain shrink as the disease progresses, i.e. atrophy.

The major cause of dementia is non-genetic risk factors. These are factors in the environment (e.g. smoking) and/or associated with other
diseases/abnormalities of the body (e.g. high blood pressure, diabetes, obesity) that lead to an increased risk of dementia (see details below).

Alzheimer’s and dementia are multi-factorial diseases which lead to degeneration of the nerve cells and brain shrinkage. Several factors have to come together to cause the disease.

The non-genetic risk factors for dementia

Many non-genetic risk factors have been postulated, but only a few proven. The best evidence points to these:

- Low education
- Smoking
- Mid-life high blood pressure, high cholesterol and obesity
- Depression
- Low social activity
- Physical inactivity
- Diabetes & high blood sugar
- Low intake of omega-3 fatty acids
- High blood homocysteine (due to low-normal B vitamin status).

“Myth #1: Dementia is an inevitable part of ageing

“No it is not. You can age successfully and still have a healthy brain. We all know people in their 90s who show no signs of cognitive decline. Many of us don’t age successfully, but it is due to a disease process going on in the brain.”

“Myth #2: It’s all in the genes

“The second myth I’d like to dispel is that it’s all in the genes. It’s not. Genes determine approximately 1% of dementia. However there are many genetically-determined risk factors. These are quite common mutations in genes that affect quite a large part of the population and are called susceptibility genes that slightly increase your risk of developing dementia. It’s been estimated that between 20% and 30% of dementia cases might be related to various combinations of genetic risk factors.”

“Most of these are also risk factors for heart disease and stroke. That’s a good sign, because we know in the case of heart disease and stroke we can dramatically reduce the incidence of these diseases by modifying the risk factors. There’s been a decline of about 70% in deaths due to heart disease over the last 50 years in western countries. Of course quite a bit of that is due to medical advances – catheters, echocardiography and drugs – but more than half of the decline in deaths is due to modification of risk factors, like smoking, high blood pressure and high cholesterol. It works for heart disease. Will it work for dementia?

“A very important study was published from Cambridge by Carol Brayne and her colleagues. They compared the prevalence of dementia in an elderly population in 1991 with the prevalence in 2011. Because they knew the prevalence in 1991, they could estimate what it should be in 2011, based on the age structure of the population.”
They were able to ascertain that they should have found 884,000 cases of dementia in England and Wales as a whole in 2011, but they found only 670,000. So there were 214,000 fewer cases of dementia than had been predicted. This was a dramatic result. A 24% reduction in prevalence. As an explanation, they could only suggest that it was modification of risk factors. This finding confirms other studies around the world.” Professor David Smith.

High homocysteine is associated with an increased risk of cognitive impairment and Alzheimer’s disease

Professor Smith talked about a study he was involved with: Preventing Alzheimer’s disease-related grey matter atrophy by B-vitamin treatment. This study was published in 2013.

Professor Smith and his colleagues discovered that those participants in the study with mild cognitive impairment who had raised blood levels of homocysteine suffered a rapid rate of shrinkage of particular regions of the brain. Mild cognitive impairment is when people have problems with their memory but not full dementia. Around a sixth of over-70 year olds are thought to suffer from it.

The main determinant of blood homocysteine is the body’s status of three of the B vitamins - folate, vitamins B6 and B12. Homocysteine levels can be lowered by administering these vitamins. This accelerated brain shrinkage was dramatically slowed down in those given the B vitamins.

MRI scans were done at recruitment. Half the participants were given high doses of the three B vitamins and half took a placebo tablet. After two years, everyone was scanned again. The result is shown below; yellow indicates areas with atrophy after two years.

In the B vitamin group there was a 90% slowing of brain atrophy in the regions highlighted. These are the same regions that are atrophied in Alzheimer’s disease.

The professor and his team were able to draw these conclusions:

- It is possible to markedly slow down the disease process in people with raised blood homocysteine levels.
- About half of the elderly population have such raised levels of homocysteine.
- Screening for blood homocysteine would identify those at risk of increased brain atrophy and eventual dementia.
- They could be offered treatment with high-dose B vitamins, which are considered safe.
Professor Smith’s recommendations for Alzheimer’s prevention

- Stop smoking.
- Do 30 minutes of brisk exercise each day (enough to get you puffing and sweating).
- If you have diabetes and/or high blood pressure, take drugs that effectively treat these disorders.
- Increase the Mediterranean elements of your diet, especially your 5+ a day of fruit and vegetables.
- Eat fish once or more a week.
- Watch your blood glucose.
- Make sure your vitamin D and B12 status are good.

- If you do have memory problems, get your homocysteine checked. If it’s high, take B vitamins in consultation with your GP.
- Keep mentally and socially active.

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