



Halting Alzheimer's

Stopping mental degradation from Alzheimer's makes quality ageing more possible by improving cognition and slowing the rate of decline.

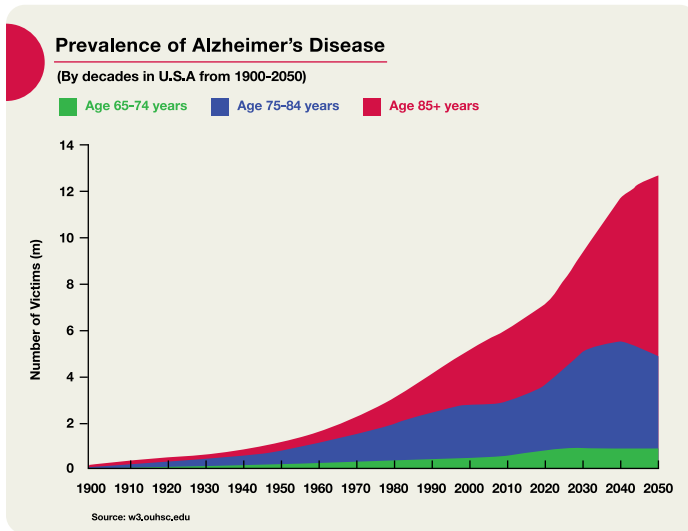
In our discussions, one of the big bets for the future in the health arena that many are focused on is being able to halt the rise of Alzheimer's disease. This disease is a physical condition affecting the brain and is the most common cause of dementia. Today, worldwide, 35 million people have Alzheimer's and, as the ageing demographic shift has greater impact, this is projected to rise to 115 million by 2050.

There is currently no cure for Alzheimer's disease. However, drug treatments are available that can alleviate the symptoms and even slow down the disease's progression in some patients. Although the exact causes of the disease are still being investigated, people with Alzheimer's are known to have a shortage of the chemical acetylcholine in their brains. Drugs available to people in the moderate stages of dementia, such as Aricept, Exelon and Reminyl, work by maintaining existing supplies of acetylcholine, the chemical compound which activates muscles. Another drug, the only one that is suitable for use in people in the middle to later stages of dementia, is called Ebixa and works in a different way – it prevents the excess entry of calcium ions into brain cells. Excess calcium in the brain cells damages them and prevents them from receiving messages from other brain cells. These drugs are not a cure for Alzheimer's, as they have no effect on the underlying degenerative process of the disease, but they can stabilise some of the symptoms for a limited period of time.

Finding a way forward for Alzheimer's is a burning issue, especially in the US where there are currently 5.5 million patients and the direct and indirect costs of the disease amount to over \$100 billion annually. In addition, the disease is well recognised as placing heavy economic and social burdens on caregivers. By 2050, with more people living longer and so a greater percentage of the population susceptible to the disease, the US is forecast to have 14 million people with Alzheimer's and the burden on the healthcare system could be as high as \$500 billion.

While finding a cure for Alzheimer's may take longer than our 2020 horizon, a capability of stopping degradation is highly probable. In fact, over the past few years, there have been some major developments which give hope. Key to many of these is the use of adult stem cells as a base for developing

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new healthy brain cells. As was highlighted during the Future Agenda programme, 'several treatments in development are designed either to improve cognition or to slow the rate of decline' and some of the most promising avenues of research being undertaken at universities including Berkeley focus on reducing 'amyloid beta' levels.

An increasingly accepted hypothesis about Alzheimer's is that it involves the accumulation of a particular protein known as amyloid beta, which is a peptide of around forty amino acids. In Alzheimer's patients, two enzymes, beta secretase and gamma secretase, effectively cut amyloid beta segments out

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of larger, normal parent molecules. These segments damage neurons and synapses and, as they build up, either in soluble form or clumped into plaques, cause the early onset of Alzheimer's. Some of the processes under development are focused specifically on preventing amyloid beta from being formed in the first place and also try to stop the molecules from attaching to one another if they do form. Once the symptoms of Alzheimer's, such as memory loss, are evident, a great deal of neurological damage has already been done. Therefore, by monitoring and measuring amyloid beta levels in healthy people before any degradation, the onset of the disease can be detected in advance and remedies applied. Others suggest that 'a broader strategy is needed, one that incorporates the role of newly identified genes and even the chemical processes responsible for killing nerve cells.'

By 2020, these and other new developments in genetics and biotechnology are expected to 'slow the progression of the disease and further alleviate its behavioural and psychological effects'. As a consequence, those diagnosed and treated in the early stages of Alzheimer's will probably enjoy far greater self-sufficiency than is possible today.

In a similar vein, it is increasingly probable that by 2020 we will also be able to halt the decline of patients with Parkinson's disease, the degenerative disorder of the central nervous system that impairs the sufferer's motor skills, speech and other functions. Nearly 1 million people in the US are living with Parkinson's disease in 2010, again with no known cure but medication or surgery providing relief from the symptoms. By 2030, health forecasts predict up to 9 million patients globally, with around half of these in China. Following recent campaigns, including

especially the one led by the actor Michael J. Fox, there has been increased investment and focus on this disease. The big hope here is that stem cell-based techniques will play a big part. One approach being pursued in Sweden involves the removal and cloning of adult neural cells and coaxing them to become dopamine-producing neurons for subsequent re-implantation into patients. Another study under way at Oxford University is using induced pluripotent stem cells taken from a patient's skin to grow new brain neurons. These and similar studies could well be the next step in cell replacement therapy for Parkinson's disease.

In the areas of both Alzheimer's and Parkinson's, the new developments on the horizon open the door for even longer and more productive lives. In bringing mental degradation under the same control as physical, this could provide an end to the increasingly common situation of the mind degrading faster than the body – and certainly delay the onset of decline.



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