Alzheimer's Disease

CIHR IRSC

The Canadian Institutes of Health Research (CIHR) is the Government of Canada's agency for health research. Through CIHR, the Government of Canada invested approximately $22.2 million in 2006-07 across Canada in research on Alzheimer's disease (AD).

The Facts

- Caring for people with AD costs about $5.5 billion each year in Canada.
- One in ten Canadians over age 65 – and one in four over age 85 – will develop AD.
- AD is the most common form of dementia, accounting for nearly two-thirds, or 64%, of all dementias.
- Twice as many women as men have dementia.
- By 2031, more than 750,000 Canadians are expected to have AD and related dementias.
- Thirty-two per cent of Canadians know someone with AD and 21% of Canadians have someone in their family with the disease.

Canadian Institutes of Health Research
160 Elgin St., 9th Floor, Ottawa ON K1A 0W9
www.cihr-irsc.gc.ca

About CIHR

The Canadian Institutes of Health Research (CIHR) is the Government of Canada's agency for health research. CIHR's mission is to create new scientific knowledge and to catalyze its translation into improved health, more effective health services and products, and a strengthened Canadian health-care system. Composed of 13 Institutes, CIHR provides leadership and support to more than 11,000 health researchers and trainees across Canada.

Finding Solutions

Researchers identify a new Alzheimer's gene

Dr. Peter St George-Hyslop, a CIHR-funded researcher from the University of Toronto, has helped uncover another important piece of the AD puzzle, information that could eventually lead to better diagnostic tools and treatments for AD. He and his colleagues recently discovered that variations in a gene called SORL1 are associated with the development of late-onset AD. When SORL1 is mutated, the cells of the brain produce higher levels of amyloid-beta, a toxic peptide that kills brain cells and may be involved in the development of AD.

A sweet solution

A team of CIHR-funded researchers from the University of Toronto has identified a drug that halts AD in mice. The drug, a sugar-like substance known as "scyllo-cyclohexanehexol", blocks the accumulation of a toxic peptide called amyloid-beta in the brains of lab mice. Amyloid-beta kills brain cells and triggers the formation of the neuritic plaques that are characteristic of AD. Dr. JoAnne McLaurin and her colleagues have obtained permission from Health Canada to proceed with human trials of this promising new drug.

Inheriting early-onset dementia

Drs. Ian MacKenzie and Howard Feldman at the University of British Columbia have identified a gene that, when mutated, causes an inherited form of early-onset dementia. The disorder, known as frontotemporal dementia (FTD), usually strikes between the ages of 50 and 60 and is inherited in about 50% of cases. FTD gradually impairs a patient's ability to speak and can result in dramatic behaviour changes. The mutations discovered by Drs. MacKenzie and Feldman prevent the progranulin gene from generating enough of the progranulin protein, which is necessary to keep brain cells alive. This CIHR-funded discovery could lead to new screening tests and treatments for FTD.

The Researchers

Dr. Weihong Song – Explaining the link between Alzheimer's disease and Down syndrome

Alzheimer's disease strikes about 10% of people over the age of 65. For people with Down syndrome, the situation is much worse; estimates vary, but researchers believe that most people with Down syndrome who live past middle age will develop AD.

Dr. Weihong Song, a CIHR-funded researcher at the University of British Columbia, has been investigating the connection between AD and Down syndrome. Since the two conditions are so closely related, identifying links could lead to new treatments for AD and improved quality of life for older people with Down syndrome.

"When I started my career, Alzheimer's research was a wide-open field. There were no genes that had been identified that were related to AD," says Dr. Song. "With my background as a clinical psychiatrist and a molecular biologist, I felt I could contribute a lot."

Last year, Dr. Song and his colleagues identified a gene that may partly explain why AD is so common in people with Down syndrome. Activation of this gene, called BACE1, triggers a series of chemical reactions that produces a brain cell-killing substance called amyloid-beta protein. According to Dr. Song's research, people with Down syndrome do not transport or break down the BACE1 protein properly, so it slowly accumulates in their brains, contributing to the accumulation of amyloid-beta protein.

Based on this work, Dr. Song and his team made another important discovery about a related gene called BACE2. "What we found is that BACE2 actually degrades the amyloid-beta protein. So instead of increasing the chance of getting AD, BACE2 prevented it," said Dr. Song.

These discoveries could lead to the development of medications that prevent and treat AD in the elderly and people with Down syndrome.

For more information, go to
www.healthresearchatwork.cihr-irsc.gc.ca
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