Alzheimer's researchers find high protein diet shrinks brain

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One of the many reasons to pick a low-calorie, low-fat diet rich in vegetables, fruits, and fish is that a host of epidemiological studies have suggested that such a diet may delay the onset or slow the progression of Alzheimer's disease (AD). Now a study published in BioMed Central's open access journal *Molecular Neurodegeneration* tests the effects of several diets, head-to-head, for their effects on AD pathology in a mouse model of the disease. Although the researchers were focused on triggers for brain plaque formation, they also found that, unexpectedly, a high protein diet apparently led to a smaller brain.

A research team from the US, Canada, and the UK tested four differing menus on transgenic mouse model of AD, which express a mutant form of the human amyloid precursor protein (APP). APP's role in the brain is not fully understood; however it is of great interest to AD researchers because the body uses it to generate the amyloid plaques typical of Alzheimer's.

These mice were fed either (1) a regular diet, (2) a high fat/low carbohydrate custom diet, (3) a high protein/low carb version or (4) a high carbohydrate/low fat option. The researchers then looked at the brain and body weight of the mice, as well as plaque build up and differences in the structure of several brain regions that are involved in the memory defect underlying AD.

Unexpectedly, mice fed a high protein/low carbohydrate diet had brains five percent lighter that all the others, and regions of their hippocampus

were less developed. This result was a surprise, and, until researchers test this effect on non-transgenic mice, it is unclear whether the loss of brain mass is associated with AD-type plaque. But some studies in the published literature led the authors to put forward a tentative theory that a high protein diet may leave neurones more vulnerable to AD plaque. Mice on a high fat diet had raised levels of plaque proteins, but this had no effect on plaque burden.

Aside from transgenic mice, the pressing question is whether these data have implications for the human brain. "Given the previously reported association of high protein diet with aging-related neurotoxicity, one wonders whether particular diets, if ingested at particular ages, might increase susceptibility to incidence or progression of AD," says lead author, Sam Gandy, a professor at The Mount Sinai School of Medicine in New York City and a neurologist at the James J Peters Veterans Affairs Medical Center in the Bronx NY. The only way to know for sure would require prospective randomised double blind clinical diet trials. According to Gandy, "This would be a challenging undertaking but potentially worthwhile. If there is a real chance that the ravages of AD might be slowed or avoided through healthy eating. Such trials will be required if scientists are ever to make specific recommendations about dietary risks for AD."

More information: Dietary composition modulates brain mass and amyloid beta levels in a mouse model of aggressive Alzheimer's amyloid pathology, Steve Pedrini, Carlos Thomas, Hannah Brautigam, James Schmeidler, Lap Ho, Paul Fraser, David Westaway, Peter Hyslop, Ralph Martins, Joseph Buxbaum, Giulio Pasinetti, Dara Dickstein, Patrick Hof, Michelle Ehrlich and Sam Gandy, *Molecular Neurodegeneration* (in press), www.molecularneurodegeneration.com/

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