Exploring the links between Alzheimer´s Disease, Diabetes and Obesity – Is there Genetic Evidence of Inflammation and Insulin Resistance the Human Brain?

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Country

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Exploring the links between Alzheimer´s Disease, Diabetes and Obesity - Is there Genetic Evidence of Inflammation and Insulin Resistance the Human Brain?

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5

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Research Abstract
Obesity is a risk factor for cardiovascular disease, cancer and diabetes and may cause insulin resistance (IR). IR and diabetes are major risk factors in Alzheimers disease (AD). Studies show that obesity in itself might also be related to dementia. A constant low-grade inflammation plays

a key role linking obesity and diabetes to cardiovascular disease and cancer but the role of inflammation in neurodegenerative disease remains obscure. As 36 million people suffer from AD and estimates show this number will be doubled in 20 years there is an urgent need to understand the underlying mechanisms. In AD, accumulation of Abeta Amyloid interferes with insulin receptor signalling and glucose utilization is comprimised in the brain of AD patients suggesting that AD is a “type 3 diabetes” Here we investigate the extent of inflammation and altered insulin signaling in the brain of AD patients using state of the art bioinfomatic tools on microarray, RNA sequencing and methylation data avalible to our thourgh our collaboration with the Lieber Institute for Brain Development. We hypothosize that alterations in insulin signaling and inflammatory levels are an early event in AD neurodegeneration which can be induced by the presence of obesity-related inflammation alone or in the presence of diabetes. We will test this hypotheis by comparing our AD data to similar data from diabetic patients and neurologically sound individuals of various body weight. Our study includes new microarray data on the hypothalamus generated especially for this project. The hypothalamushas the overall metabolic control of the body and we thus aim to see if inflammation or altered insulin signalling play a role in linking systemic and brain metabolism to neurodegeneration. We hope this project will improve our understating of neurodegenerative processes and the impact of metabolic disorders on brain function hereby revealing potential targets for neuro-protectiive pharmacological interventions.

Further information available at:

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