Natalie Rasgon, J Alzheimers Dis Parkinsonism 2014, 4:5
http://dx.doi.org/10.4172/2161-0460.S1.006

OMICS Group Conferences
Accelerating Scientific Discovery

2nd International Conference on
Alzheimer's Disease and Dementia
September 23-25, 2014 Valencia Convention Centre, Spain

Insulin resistance: Risk factor of dementia

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Insulin resistance (IR) is a putative risk factor for cognitive decline and dementia, and has been shown to impede neuronal glucose metabolism in animal models. This post-hoc study focused on metabolic changes in the medial prefrontal region, a brain region exhibiting decline years before documented cognitive changes, relative to high or low IR status in a cohort of postmenopausal women at risk for dementia who were randomized to continue or discontinue existing stable hormone therapy (HT) for 2 years. Subjects were dichotomized into high and low IR groups based on the homeostatic model assessment of insulin resistance, which was within clinically normal limits for the group as a whole at both baseline and 2-year follow-up. Results showed that high and low IR groups showed significant differences in metabolic decline of the medial prefrontal gyrus, regardless of HT randomization group. However, HT randomization was predictive of metabolic decline only in women with low HOMA. Performance in working memory was consistent with observed metabolic changes. These results suggest IR may be an independent moderator of regional metabolic changes, while protective metabolic effects of HT are most apparent in those at low-end range of IR. If replicated in future studies, these findings will help to better understand the interaction between putative risk and protective factors, and further delineate cohort postmenopausal women who may benefit from HT.

Biography

Natalie Rasgon received her MD and PhD in Obstetrics and Gynecology and Pathological Physiology in the USSR, she began her distinguished career at UCLA School of Medicine, and in 2002, she established the Center for Neuroscience in Women's Health at Stanford. She is the author of more than 130 peer-reviewed publications, 25 book chapters, and is a reviewer for 30 professional journals. Her predominant research focus has been on the treatment of bipolar disorder in women, the use of hormonal interventions during menopause and the effects on mood and cognitive function, and the interplay between endocrine function and aging.

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