Andrei Vlassenko - selected references

1 Vlassenko, A. G., Benzinger, T. L., & Morris, J. C. (2012). PET amyloid-beta imaging in preclinical Alzheimer's disease. *Biochim.Biophys.Acta, 1822,* 370-379.
Notes: Alzheimer's disease (AD) is the leading cause of dementia, accounting for 60-70% of all cases [Hebert et al., 2003, 1]. The need for effective therapies for AD is great. Current approaches, including cholinesterase inhibitors and N-methyl-d-aspartate (NMDA) receptor antagonists, are symptomatic treatments for AD but do not prevent disease progression. Many diagnostic and therapeutic approaches to AD are currently changing due to the knowledge that underlying pathology starts 10 to 20 years before clinical signs of dementia appear [Holtzman et al., 2011, 2]. New therapies which focus on prevention or delay of the onset or cognitive symptoms are needed. Recent advances in the identification of AD biomarkers now make it possible to detect AD pathology in the preclinical stage of the disease, in cognitively normal (CN) individuals; this biomarker data should be used in the selection of high-risk populations for clinical trials. In vivo visualization of AD neuropathology and biological, biochemical or physiological confirmation of the effects of treatment likely will substantially improve development of novel pharmaceuticals. Positron emission tomography (PET) is the leading neuroimaging tool to detect and provide quantitative measures of AD amyloid pathology in vivo at the early stages and follow its course longitudinally. This article is part of a Special Issue entitled: Imaging Brain Aging and Neurodegenerative disease

2 Vlassenko, A. G., Mintun, M. A., Xiong, C., Sheline, Y. I., Goate, A. M., Benzinger, T. L. et al. (2011). Amyloid-beta plaque growth in cognitively normal adults: longitudinal [11C]Pittsburgh compound B data. *Annals of Neurology, 70,* 857-861.
Notes: Amyloid-beta (Abeta) accumulation was evaluated with 2 [(11)C]Pittsburgh compound B (PiB) positron emission tomography scans about 2.5 years apart in 146 cognitively normal adults. Seventeen of 21 participants with initially elevated Abeta deposition demonstrated subsequent Abeta plaque growth (approximately 8.0% per year), and none reverted to a state of no Abeta deposits. Ten individuals converted from negative to positive PiB status, based on a threshold of the mean cortical binding potential, representing a conversion rate of 3.1% per year. Individuals with an epsilon4 allele of apolipoprotein E demonstrated increased incidence of conversion (7.0% per year). Our findings suggest that the major growth in Abeta burden occurs during a preclinical stage of Alzheimer disease (AD), prior to the onset of AD-related symptoms

3 Vlassenko, A. G., Vaishnavi, S. N., Couture, L., Sacco, D., Shannon, B. J., Mach, R. H. et al. (2010). Spatial correlation between brain aerobic glycolysis and amyloid-beta (Abeta) deposition. *Proceedings of the National Academy of Sciences of the United States of America, 107,* 17763-17767.
Notes: Amyloid-beta (Abeta) plaque deposition can precede the clinical manifestations of dementia of the Alzheimer type (DAT) by many years and can be associated with changes in brain metabolism. Both the Abeta plaque deposition and the changes in metabolism appear to be concentrated in the brain's default-mode network. In contrast to prior studies of brain metabolism which viewed brain metabolism from a unitary perspective that equated glucose utilization with oxygen consumption, we here report on regional glucose use apart from that entering oxidative phosphorylation (so-called "aerobic glycolysis"). Using PET, we found that the spatial distribution of aerobic glycolysis in normal young adults correlates spatially with Abeta deposition in individuals with DAT and cognitively normal participants with elevated Abeta, suggesting a possible link between regional aerobic glycolysis in young adulthood and later development of Alzheimer pathology