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Gene expression changes triggered by amyloid beta toxicity

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Multiple genes have been linked to Alzheimer's disease (AD) implicating multiple metabolic pathways in its pathogenesis. It is not clear, however which of these genes are involved in general stress response and which are linked to AD-specific pathologies. To make an attempt to answer this question, we investigated changes in gene expression induced by human β-amyloid peptide (Aβ) in a transgenic *C. elegans* Alzheimer's disease model. Aβ-induced gene expression changes were compared to those caused by a synthetic aggregating protein to identify Aβ-specific genes. Among the Aβ-specific genes identified in this study were genes involved in aging, lifespan regulation, proteasome structure and function and mitochondrial function. Interestingly, a significant overlap between Aβ- and Cry5B (a bacterial pore-forming toxin) induced gene expression alterations was observed. Further testing suggested the involvement of membrane damage in Aβ pathogenesis. In this study, we segregated Aβ-specific gene expression changes from general stress response in a simple metazoan animal and provide evidence implicating membrane damage in Aβ toxicity. We hope this work will eventually help identify novel therapeutic targets and further our understanding of AD pathogenesis.

Biography

Wail M Hassan has completed his Bachelor's degree in Microbiology and a Graduate Diploma in Public Health from Alexandria University in 1990 and 1993 respectively. He has completed his PhD in Microbiology from the University of Southern Mississippi in 2004. He has occupied several Post-doctoral positions between 2004 and 2010 at University of Colorado at Boulder, the National Naval Medical Center and University of Nebraska Medical Center. In 2011, he joined the University of Wisconsin-Milwaukee as an Assistant Professor where he continues his research focusing on the molecular mechanisms of amyloid beta toxicity.

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