אוניברסיטת בן-גוריון בנגב

Ben-Gurion University of the
Avram and Stella Goldstein-Goren Department of Biotechnology Engineering
SEMINAR

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How pregnancy with a Down syndrome fetus promotes maternal cognitive decline.

Down Syndrome (DS), caused by trisomy in chromosome 21, is the most common cause of intellectual disability (ID), affecting 1 in 850-1000 live births. Interestingly, Alzheimer's disease (AD) is linked to DS, as the amyloid precursor protein (APP) gene is over-expressed in individuals with DS. The accumulation of APP-derived extracellular amyloid-β (Aβ) deposits in the form of neurotoxic Aβ oligomers, extracellular plaques, and cerebral amyloid angiopathy, together with intraneuronal accumulation of hyperphosphorylated tau protein in the form of tangles characterize late-onset AD (LOAD), a progressive neurodegenerative disease that affects individuals aged 65 and above and is the most prevalent form of AD. These neuropathological features promote neuronal loss, brain atrophy and severe cognitive impairments. Women exhibit an almost doubled prevalence of LOAD, suggesting that in addition to age, gender is a significant risk factor in LOAD. The biological properties responsible for this gender-based prevalence are not yet clear. Moreover, pregnancy with a DS-affected fetus is associated with an almost 5-fold increased risk of developing LOAD later in life, relative to pregnancies with a fetus afflicted with other IDs. Here too, the mechanisms responsible are unknown. The increase in maternal age worldwide, which is a known risk factor for a DS pregnancy, along with persistent prevalence of DS in the world population, stresses the need to elucidate any potential mechanistic link between pregnancy with a DS-affected fetus and maternal LOAD, and ultimately develop a therapeutic approach to reduce the risk of maternal LOAD. In this talk I will present our findings on the potential mechanisms underlying maternal cognitive decline following pregnancy with a Down syndrome fetus.

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