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Strain differences in sympathetic neurotransmission in spleens of rats subjected to reduced sympathetic tone

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Senescence of innate and adaptive responses and low-grade inflammation(inflammaging) hallmarks normal aging, which increases vulnerability to infectious diseases, autoimmunity and cancer. In normal aging, sympathetic dysregulation contributes to the dysregulation of innate and adaptive immunity and inflammaging. Sympathetic innervation of immune cells in secondary immune organs regulates immune responses. Different profiles of sympathetic signaling during aging may bring about different effects on neurotransmission in immune cells that may lead to immunity variation in senescence. We investigated whether increased sympathetic nerve activity (SNA) in the aging spleen contributes to age- related sympathetic neuropathy and altered neurotransmission in splenic lymphocytes of two strain of rats of strikingly different sympathetic activation and behavior profiles. To answer this question, we injected 15 month-old rats, of either strain, 0, 0.5 or 1.5 μg/kg/day rilmenidine intraperitoneally, for 90 days to lower sympathetic tone. Untreated young and age-matched rats controlled for effects of age. We found that in Fischer 344 (F344) rats, an age-related increase in sympathetic tone and sympathetic dysfunction in beta-adrenergic receptor (AR) signaling of splenic lymphocytes contribute to immune senescence. In the much longer-lived Brown-Norway (BN) rats, we observed that elevated SNA in the aging BN rat spleen does not contribute significantly to sympathetic neuropathy or the aging-induced impairment of canonical β-AR signal transduction. Despite the rilmenidine-induced increase in β-AR (Adrenergic Receptor) expression, splenocyte c-AMP (Cyclic adenosine monophosphate) production was comparable with age-matched controls, thus dampening nerve activity had no effect on receptor coupling to adenylate cyclase. Understanding how aging differentially affects neuroimmune regulation in healthy aging rodent of different strain models can help us formulate strategies to improve health in aging populations that are most vulnerable to immunosenescence and low- grade systemic inflammation.

Biography

Samuel Perez is currently working as an assistant professor in Washington Adventist University, Maryland. He acheived his PH.D in the area of Neurophysiology/Neuroimmunology at Loma Linda University School of Medicine, California. Sam D Perez obtained the degree of master's in Molecular Physiology at Loma Linda University. His scientific research interest includes: Study the effects of neuroprotective micronutrients on learning and memory function in animal models using molecular biology tools and animal models to understand neuroimmune mechanisms of cell protection and many more.

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