Dianne Lorton et al., J Clin Cell Immunol 2014, 5:5
http://dx.doi.org/10.4172/2155-9899.S1.017

OMICS Group Conferences
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3rd International Conference and Exhibition on
Clinical & Cellular Immunology
September 29-October 01, 2014 DoubleTree by Hilton Baltimore-BWI Airport, USA

The sympathetic nervous system: A not so "sympathetic" regulator of immune function in autoimmune disease- Rheumatoid arthritis as an example

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Mechanisms that cause autoimmune disease are complex and include interactions with genetic, environmental, immunologic, and neural-endocrine factors. How tolerance is broken and disease onset is initiated remain enigmatic. These events are often separated by many years, suggesting that disease onset requires a triggering event that if understood, could be targeted therapeutically. Physical and psychological stressors are implicated in the development of autoimmune disease, based on several observations. First, severe life stressors are strongly associated with disease onset in up to 80% of patients. Second, the major stress pathways, the sympathetic nervous system (SNS) and hypothalamic -pituitary adrenal (HPA)-axis function to restore immune system homeostasis after immune challenges and regulate immune responses that become pathological in autoimmune diseases. Finally, there is a common "trifecta" of dysregulated immune functions, elevated SNS activity and low HPA-axis responsiveness across most autoimmune diseases. Understanding the immune-to-nervous system and nervous -to immune system cross-talk that leads to this 'trifecta" is key to understanding autoimmune disease mechanisms. Here, we focus on findings indicating elevated SNS tone and altered nerve to β_2-adrenergic receptor signaling to the immune system are pathological events required for triggering autoimmunity using in an animal model of rheumatoid arthritis as an example.

Biography

Dianne Lorton completed her PhD in Neurosciences at Indiana State University in affiliation with Indiana University School of Medicine. She completed Post-doctoral training in Pharmacology at Duke University and in Neuroimmunology at the University of Rochester School of Medicine. She is currently an Assistant Professor at Kent State University in the College of Arts and Sciences. She has published over 70 papers (manuscripts, reviews, and book chapters) on neuroimmunology focusing on sympathetic nervous system regulation of immunity.

Denise Bellinger completed her PhD at Indiana State University with research training at Indiana University School of Medicine's onsite campus, and postdoctoral training in Psychoneuroimmunology from the University of Rochester School of Medicine. She is an Associate Research Professor in the Department of Pathology and Human Anatomy at Loma Linda University School of Medicine. She has published over 80 peer reviewed papers, review articles, and book chapters on various aspects of neural-immune interactions in aging and age-related diseases.

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