J Neurol Disord 2015, 3:3
http://dx.doi.org/10.4172/2329-6895.S1.003

OMICS International
conferenceseries.com

International Congress on Neuroimmunology and Therapeutics

DoubleTree by Hilton Hotel San Francisco Airport, San Francisco, CA, USA

Vitamin D synergies with disease modifying therapies for multiple sclerosis

Bijal Mehta
University of California, USA

Disease Modifying Therapies (DMT) for Multiple Sclerosis have seen a relative explosion from a few treatments a decade ago to over a dozen currently. Vitamin D and an association with Multiple Sclerosis (MS) continue to be demonstrated in the research literature. However, as correction of patient's serum vitamin D levels is occurring in MS clinics world-wide, little data has emerged as to how normal vitamin D levels effects patients on various disease modifying therapies. This presentation will discuss the mechanisms of action of vitamin D on the neuro-immune system, along with the effects of current and future disease modifying therapies. Potential and proven synergies will be discussed from the literature. A discussion of the potential variances between the animal models used to test vitamin D and DMTs with human clinical trials will be discussed. Lastly, a list of other potential synergistic compounds that have yet to be tested in clinical trials will be reviewed.

bijal@yahoo.com

Neuronal interleukin 13 receptor and parkinson's disease

Bruno Conti
The Scripps Research Institute, USA

Neuroinflammation is believed to contribute to the onset and/or the progression of Parkinson's Disease (PD). However, inflammatory mediators contribution to the preferential loss of dopaminergic (DA) neurons remains poorly understood. In order to investigate the pathophysiological role of inflammation in PD, we analyzed the existance of a possible linkage between mediators of infammation and genetic loci associated with PD. We found that the gene encoding for interleukin 13 receptor alpha 1 (IL-13Ra1) lies on the human X chromosome at Xq24 within the Xq21-q25 region found to contain the PARK12 locus of PD susceptibility. IL-13Ra1 is the subunit of a heterodimeric receptor that is responsible for mediating the action of the Th2 cytokines; IL-13 and IL-4. These cytokines are known as important peripheral modulators of allergic reactions, of anti-helmintic response and also for their anti-inflammatory action. Investigation into the distribution of IL-13Ra1 in the mouse brain reveals that it is expressed in the dopaminergic neurons of the substantia nigra pars compacta (SNpC) and the ventral tegmental area (VTA). Thus we hypothesized that neuronal IL-13Ra1 may influence dopaminergic cells survival and functions and tested our hypothesis using mice null for IL-13Ra1 (*Il13ra1*$^{-/Y}$). *Il13ra1*$^{-/Y}$ and *Il13ra1*$^{+/Y}$ had similar number of dopaminergic neurons in the SNc and the VTA and had comparable levels of dopamine. However, *Il13ra1*$^{-/Y}$ animals were protected from loss of DA neurons in a neurotoxic insult as well as in a chronic peripheral inflammation mouse model of PD. *In vitro* studies showed that while IL-13 and IL-4 did not have any intrinsic toxic effect when administered alone, they greatly potentiated the cytotoxicity of reactive oxygen species. Our finidng indicates that neuronal IL-13Ra1 can contribute to the preferential loss of DA neurons during neuroinflammation and may be involved in the pathogenesis and/or the progression of PD.

bconti@scripps.edu