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Involvement of Th17/Treg cells and cytokines in Obstructive Sleep Apnea

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Abstract

Obstructive sleep apnea (OSA) has pathophysiologic characteristics that are known to negatively impact immune function. The repetitive cycles of hypoxia/reoxygenation and connected arousals activate inflammatory pathways, and the cardiovascular dysfunction.

Consequence of hypoxia/reoxygenation phenomena is the activation of NF-kB and increased production of inflammatory cytokines, and the differentiation of Th17 cells. The balance between Foxp3+ Treg and Th17 cells was determined by IL-6 that modulates the TGFβ-induced generation of Foxp3+ Treg and drive Th17 cell differentiation. The balance between TGFβ, an immunosuppressive cytokine, and IL-6, a pro-inflammatory cytokine, might influence the final outcome in the differentiation process of different effector T cell subsets.
Recently, several genes underlying inflammatory pathways has been demonstrated to be differently expressed in peripheral blood leukocytes of patients with OSA, suggesting that OSA activates widespread pro-inflammatory networks increasing the levels of inflammatory mediators that promote development of medical comorbidities.

Our results suggested that in the micro-environment of OSA patients, the increase of pro- inflammatory IL-6 and the reduction of TGFβ potentially promote the Th17/Treg imbalance. The shift of IL-17/TGFβ balance toward IL-17 might enhance the accumulation of inflammatory mediators, and finally generate a proinflammatory loop to amplify proinflammatory environment that in moderate-severe OSA patients shift the balance between Th1/Treg cells toward Th1 cells. Although IL-17 has no significant higher circulating levels in our OSA patients, taking into account its significantly high expression levels, we cannot exclude a role for IL-17 in OSA-related inflammation. Thus, IL-17 in conjunction with reduced TGFβ, modulate the development of Treg cells, causing a Th17/Treg imbalance.

An intriguing hypothesis is that in OSA the balance between Th1 cells and Treg cells as well balance between M1 e M2 could be shifted respectively by up- regulation of IL-6 and down-regulation of TGFβ toward an inflammatory Th1 and M1 cell phenotype.

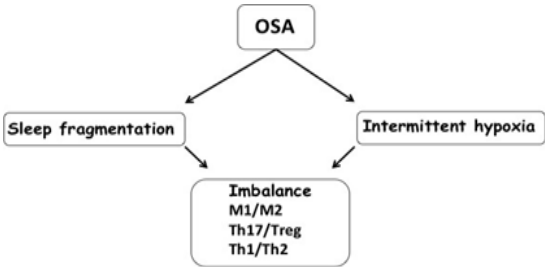

Fig.1Immunological imbalance in OSA

Biography

Dr.Marcella Reale is author of 160 peer-reviewed pubblication and 3 books chapter in neuroimmunology, inflammatory diseases and cytokines (Orcid n. 0000-0002-4164-8781).Dr.ssa Reale's goals are to identify the pathogenic role of inflammatory cytokines and, to follow disease activity and to monitor therapeutic responses. Her research deals with the inflammatory responses and neurodegeneration, in particular the characterization of functional and immunological activation of peripheral cells in inflammatory disease in humans, the cytokines/chemokines network, non-neuronal cholinergic system and their activation pathways, mechanisms for a7nAChR–based pathways in establishing production of inflammatory cytokines as possible strategies for therapeutical intervention by new drugs in autoimmune diseases and neurodegeneration. Dr.ssa Reale is referee of 35 National and International Journals, member of 7 International Editorial Boards and editor in Chief of AIMS Allergy and Immunology. She has many years of experience in research laboratory management and leadership. She enjoy teaching General Pathology and Immunology to undergraduate and graduate medicine students at School of Medicine,of "G.d'Annunzio"University.