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Editorial

HIV Infection - From Immune Deficiency to Immune Activation

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Editorial

Ever since its definition, HIV infection has become the subject of the most dynamic researches. First of all, they have led to enormous progress in the treatment of the disease itself, but at the same time they have enabled the interpretation of numerous medical dilemmas and previous unknowns. Today, when therapeutic control of immunodeficiency (a basic path genetic mechanism) is achieved, studies are focused on immune activation, which follow HIV infection, and is associated with non-AIDS related co morbidities. These conditions, including cardiovascular, chronic renal, liver and pulmonary diseases, diabetes and malignancies, are now the primary cause of mortality among HIV-infected patients on ART. Together, with more frequent osteoporosis and neurocognitive disorders, they define the aging process, confirming the assumption about its inflammatory origin, a challenging and promising new field of research [1]. Basically, the immune system undergoes a process of senescence accompanied by the increased production of pro inflammatory cytokines, causing a chronic subclinical condition called "inflammaging". According to theory of antagonistic pleiotropy, it is believed that immunosenescence is not a random deteriorating phenomenon, but a reversed evolutionary pattern, and that most of the affected parameters are under genetic control [2,3]. Independently of this complex genetic basis, immune activation during HIV infection can represent a model for a better understanding of immunosenescence4, as a result of chronic antigenic overload (even when viral replication is controlled), especially in terms of price paid to immunological memory which is one of the main features of the latest and most modern type of immunity.

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