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Editorial

Immunosenescence: A Stagnant Promising Area or a Stagnant but Promising Area?

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Editorial

The gradual increase in age-related diseases is related to increase life expectancy of the population. In this context, the improvement in the life expectancy results in increased costs related to hospitalizations and medical treatments, because of the public health problem both in developed and in developing countries. Aging is associated with a restructuring of the immune system functions, both humoral immunity as well as in cell mediated immunity resulting in increased susceptibility to infection and cancer.

Within the human aging field of study there is one area in particular that catches the interest of some researchers around the world: the immunosenescence. The immune system by itself is fascinating, with all its variables and modulations, but when we looked under the scope of human aging we can see how deep are the effects of the immune system on each amendment, physiological or not, that occurs in the body old guy. Over the past 20 years have seen many discoveries in the field of immunosenescence, we realize the importance of infection with Cytomegalovirus (CMV), we saw the relationship between the cognitive changes of elderly patients with immunological markers as well as their relationship to the physical fragility of the elderly, and finally we saw the rise of the Immune Risk Profile hypothesis. However, we still follow this line. We seek to further understand the immunological changes that occur in the elderly, without understanding objectively as CMV affects the lymphocytes.

Immunosenescence

The aging process is characterized by physiological changes that may be permanent or reversible. The concept of "Inflammaging" is intrinsically linked to aging; this is the term by which we call the inflammatory profile the individual gradually begins to show over the years with higher plasma levels of cytokines and inflammatory mediators. It is curious that despite this, some activities related to cellular functions of innate immunity are reduced over the years, paradoxically compared to inflammaging process. For this reason, most studies have focused primarily on immunosenescence related to aging functions of T cells [1].

The T cells undergo significant and drastic change during aging. Several studies have contributed to the discovery of risk factors associated with increased morbidity and mortality in aging. In this sense, the Immune Risk Profile (IRP) seems to be a good marker to distinguish healthy aging disease. The IRP was set from longitudinal studies of Swedish elderly (octa and nonagenarian) with a CD4:CD8<1 ratio, low proliferation T unspecific and high levels of antibody for cytomegalovirus. The elderly selected with this IRP showed a high mortality over four years of the study, regardless of health status [2-5]. The risk profile was associated with increased inflammatory activity, increase of CD8 effectors / memory expansion CD8 + cells against Cytomegalovirus (CMV) and increased production of specific antibodies against CMV. The differentiation of CD8 + T cells against persistent viral infections (especially CMV) appears to be a central point of immunosenescence.

Cytomegalovirus

In recent years, new studies indicate that the major difference between elderly subjects was the CD8 + cell accumulation with late differentiation. Other recent studies indicate a relationship between CMV infection and worse functional status in elderly [6,7]. CMV is highly prevalent worldwide and their serum conversion can occur at any age, however, the infection rate may increase by 70% after 65 years of age. After the primary infection, the virus enters a state of apparent latency in the host, however, the virus remains interacting with the host immune system through complex mechanisms that develop a balance between viral infection and the missing immune clinical signs. Balance this undoable if the immune system becomes compromised. There is an association between the IRP and CMV infection, which can be summarized by the presence of the expanding CD8 + CD28- cells, and the inversion of the CD4:CD8 ratio.

Regarding the functional integrity of anti-CMV CD8 cells expanded with age, it is known that the number of functional cells is similar in elderly and young individuals. However, the numbers of specific cells which are not highly functional are higher in the elderly. These cells then proliferate at the expense of functional cells, contributing to the inflammatory profile generally described in the elderly, resulting in the individual's inability to mount appropriate responses as you get older.

Other key factors

The relationship between oxidative stress and immunological risk factors are largely unknown. There is the possibility, raised in a recent article from our group [8], that oxidative stress can have an important impact on the immunosenescence, including the reversal of the CD4:CD8 ratio and increased CMV serology, since we already know that there is an association between oxidative stress with the inflammatory state as well as the reactivation of CMV.

Another promising line of study that deserves more attention is the relationship between physical activities (practiced only as an elderly or exercised throughout the life of the individual) as a key part of the process of immunosenescence. In a recent article James E. Turner [9] discusses this hypothesis, and I have no doubt that this

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issue must to be investigated more deeply.

It has also been discussed the importance of intestinal microbiota in this process, including the possibility of modulation by the intake of probiotics. The intestinal microbiota is proving a rich source of information on the maturation of the immune system, then nothing more logical than to assume that it has very rich information about the aging of the immune system as well.

Immunosenescence: stagnant promising area or stagnant but promising area?

Based on everything reported above, I believe that it is clear that the study of immunosenescence, the Inflammaging, Immune Risk Factor (IRP), the influence of Cytomegalovirus, finally, the study of this whole universe is something exciting and highly thought-provoking. I invite all researchers in the field of gerontology to look fondly at the immunological changes suffered by the elderly (who also may be accelerated by external factors appearing in young adults). However, I believe that this field of research is currently at a crossroads; I say this because the great discoveries occurred in the nineties and the first decade of this century, but in the last 10 years we have been discussing basically the same models related to the items mentioned above.

There are new recent articles trying to understand the "microcosm" of CMV activity in its modulation on B lymphocytes and T lymphocytes CD8, which in my view indicates the responsibility of action of CD4 T lymphocytes in this context. So again, more study is needed, more systematic, seeking to understand the immunosenescence. It is necessary to focus more on understanding the mechanisms, interconnect more deeply the immunology knowledge in endocrinology and neurology, passing by nutrology, genetics, biochemistry, physiology of exercise to finally reach in the context of gerontology itself.

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