



Ageing and Its Effects on Stem Cells: The Typical Example of Hematopoietic Ageing and Its Consequences

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Editorial

The continuous increase in life expectancy, the resulting ageing of populations and the related diseases, have caused a great impact in our society [1]. This trend will achieve very high percentages by 2030, when namely more than 20% of European and American populations will be represented by individuals older than 60 years [1]. This will determine a growing increase in the incidence and prevalence of Age-Related Diseases (ARDs). This will represent a very challenge for clinical specialists, epidemiologists and researchers, whose actual efforts, in seeking real solutions (i.e. preventive measures), appear vain or very limited both in the creation and application [2]. In this context, it appears very crucial the development of new strategies and interventions to reduce the clinical conditions related to hematopoietic ageing, ranking from immunosenescence, hematologic malignancies, and anaemia to endothelium dysfunction and onset of ARDs [3]. In order to achieve this goal, it is the time of observing with new eyes the research on Hematopoietic Stem Cells (HSCs) ageing for focussing the interest on objects aiming to identify all mechanisms involved. Our hypothesis is that haematopoiesis' ageing is the result of a very complex interplay of mechanisms, where the senescence of Endothelial Cells (ECs), and particularly the Endothelial Progenitor Cell (EPC) ageing might have the crucial role of hub [4,5]. This central core could trigger a complex network responsible of both Bone Marrow (BM) homeostasis loss and the alterations in composition of HSC niches. This would seem to be evocated by age-related changes in expression and activation of Toll-Like Receptors (TLR) and micro RNAs, and in release of a typical age-related Senescence Associated Secretory Phenotype (SASP), prevalently represented by pro-inflammatory mediators [6,7]. In turn, pro-inflammatory cytokines would create a microenvironment which could facilitate senescence of HSCs/Hematopoietic Progenitor Cells (HPCs) [6]. On the other hand, it has been recently demonstrated that increased IL-6 secretion can disrupt HSCs and HPC homeostasis [5,6]. This would improve immunosenescence and dominance of myeloid cells, which favour and increase inflammatory signaling ways [6]. In the complex context, this would result in raising both deterioration of haematopoietic system and inflammatory state [5]. Consecutively, this would facilitate instauration of a vicious circle that could improve systemic chronic inflammation and clinical conditions, which in turn would contribute to onset of ARDs [5].

Certainly, a better knowledge of the complex interactions between EC/EPC ageing, chronic inflammation, altered BM homeostasis and HSC ageing is necessary and might become object of intense clinical interest. It might derive from the results of more sophisticated studies, including studies integrating HSC ageing with inflammatory and endothelium specific transcriptome, epigenome, proteome, and secretome. In addition, we have recently outlined our views and suggest how foetal programming can contribute to HSC/EPC ageing and diseases in adult life [8]. Consequently, healthy interventions might be appropriate to limit this [9]. Accordingly, our hope is to emphasize that living a healthier life may be the key for both, the health of future generations and trying to delay the continuous increase in ARDs in human populations [9].

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