it is this replicative senescence that was subsequently named the "Hayflick phenomenon"). The term "cytogerontology" has then been extrapolated to any studies on the mechanisms of aging in experiments on cell cultures.[34,38–41]

Weismann was the first to emphasize the essential distinction between germ line cells, whose population is basically immortal, and somatic cells, which age and die. Thus, the cornerstone of his concept is that there exist the mortal soma and the immortal "germ plasm" (*Keimplasma*). However, Weismann failed to give a clear definition of what cell aging/senescence is, and this probably accounted for the findings and conclusions made by Alexis Carrel,[42,43] who laid the experimental foundations of cytogerontology in the early 20th century.

Carrel was interested to test whether somatic cells isolated from higher animals would "senesce" and die instead of propagating indefinitely. To this end, he developed a procedure for culturing epithelial or fibroblast-like cells in special flasks, which is still used today with only minor modifications. However, the results of his experiments did not fit the "mortal soma" concept: some cell strains derived from chicken embryos could be maintained in culture almost indefinitely, without showing any signs of degradation. This is why gerontologists in the 20th century for almost 50 years considered somatic cells to be capable of unlimited replication, until the experiments performed in the 1950s and 1960s by Swim and Parker[23] and, subsequently, by Hayflick[24,25,27] showed that the results obtained by Carrel were apparently artifactual. In fact, almost all normal animal cells have proved to have a limited proliferation potential, being capable of no more than 100–120 divisions in culture (about 50 cell population doublings).

Unfortunately, the model based on the Hayflick limit concept (aging *in vitro*) is apparently not directly related to the mechanisms of aging, as has been repeatedly noted previously.[2–6,44–46] In other words, we cannot conclusively explain why we age by relying solely on the phenomenon of limited mitotic potential of normal cells, which is practically never fully utilized *in vivo*. However, owing to Olovnikov's theory of marginotomy,[47–49] we at least know today how this phenomenon is realized in the cells.

It is not excluded that, if the human life span were extended severalfold, some cell populations would eventually exhaust their mitotic potential (thereby reaching the Hayflick limit), which could have resulted in the "second wave" of aging, but this has not occurred so far. It should be noted, however, that some researchers still hold the opinion that the shortening of telomeres in the cells is the key mechanism of aging. In particular, according to the point of view described by Mikhelson,[50,51] a certain "mosaicism" in the proliferative parameters, observed in a highly organized multicellular organism, allows the shortening of telomeres to be considered as an important factor in aging and longevity.

The body of evidence for the gerontological value of the Hayflick phenomenon is based only on a series of *correlations*,[6,40] like reduced mitotic potential of fibroblasts from the patients with progeria, direct relationship of this