in terms of labor, time, and finance expenditures. Because of this, overeager gerontologists currently rely mainly on so-called BA. Space limitations do not allow us to dwell on the essence of this term, but this is not necessary, since the relevant literature is available to any reader. It should only be noted that the researchers who use this term usually have in mind not so much the markers of aging itself as the markers of biological age. In other words, the markers (parameters) are well correlated with the chronological age of the test organisms but not with aging, *i.e.*, the time-dependent increase in the probability of death.

An illustrative example to this issue is the situation with human hair turning gray: the relative amount of gray hairs is well correlated with age but shows practically no correlation with mortality. Thus, relevant parameters in gerontology are those related to the basic mechanisms of aging, preferably in a cause-and-effect mode, and the majority of gerontologists consider that these are cellular or molecular mechanisms. The batteries of tests for determining the biological age (in other words, the degree of senescence) based on evaluation of various physiological parameters, which have been used on a wide scale, gradually recede in the past, giving way to studies with emphasis on "fundamental" BA—that is, on certain cellular or molecular characteristics. Moreover, these parameters are currently usually tied in with the phenomenon named cell/cellular senescence, which is central in cytogerontology.

It was initially considered that cell senescence takes place "by itself", *i.e.*, it is driven by an intrinsic mechanism, and all subsequent changes in the cells are mere *consequences* of this process. In fact, this fully applies to the mechanism of telomere shortening with every cell division, discovered by Alexey M. Olovnikov.[47] In the 1980s, one of us formulated the concept of aging,[33] according to which the restriction of cell proliferation imposed during development (due to the formation of populations of highly differentiated postmitotic or very slowly dividing cells) is the main cause of age-dependent accumulation of various macromolecular defects (mainly DNA damage) in the cells. This concept provides a simple explanation to "age-dependent" changes in senescent cell cultures: as cell proliferation at later passages is retarded, spontaneous DNA injuries are no longer "diluted" among newly emerging cells and their frequency in the population as a whole increases. The population aspect is very important since some cells fully retain the ability to divide, but their proportion decreases with passaging, so that cell senescence is manifested at the level of whole *cell population*. In essence, our "stationary phase aging" model[5,33,58–61] was based on 100% suppression of cell proliferation in culture by contact inhibition or some other physiological factor, with consequent accumulation of "age-dependent" defects in the cell population. In this case as well, we first made the cells "senesce" and only then analyzed them for certain biomarkers of *in vivo* aging (*e.g.*, DNA breaks). Thus, in the "classic" approach it was assumed that cell senescence is driven by a certain intrinsic mechanism, which leads to the emergence of various macromolecular defects (first of all, DNA damage) in the cells.