In recent years, however, cell senescence is understood primarily as the appearance or accumulation in the cells (most often, transformed cells not prone to replicative senescence) of certain "BA" (this time in quotation marks, because the situation is by no means related to real aging) under the impact of various external factors causing DNA damage (oxidative stress, H_2O_2, mitomycin C, doxorubicin, ethanol, ionizing radiation, *etc.*).[30,108–111] This phenomenon is referred to as DNA damage response (DDR). Within this definition, the "senescence" of cells takes place under the impact of DNA-damaging agents rather than on itself. It is also called "stress-induced premature senescence".[112] The aforementioned BA include senescence-associated beta-galactosidase (SA-β-Gal) activity, expression of p53 and p21 proteins as well as of regulators of inflammation such as IL-6 or IL-8, activation of oncogenes, *etc.* Therefore, cell "senescence" in the context of the above definition occurs not by itself but *because of* the impact of DNA-damaging agents. In our opinion, such an approach is very important for defining the strategy of cancer control but, yet again, leads away from the study of actual mechanisms of organismal aging.[31] A similar view was expressed by famous gerontologist Denham Harman in his brief comment published in the journal *Biogerontology*.[113] It should be emphasized that in our "stationary phase aging" model[5,33,59,60] we also observe certain BA in cell cultures, but in this case they appear *due to* restriction of proliferation by contact inhibition, *i.e.*, by a physiological factor that itself causes no damage to the cells. This situation is closely similar to what takes place in a multicellular organism.

The most popular biomarker of cellular senescence is SA-β-Gal (β-galactosidase pH 6.0). The enzyme β-galactosidase, a lysosomal hydrolase, cleaves off the terminal β-galactose from the compounds containing it (lactose, keratin sulfates, sphingolipids, *etc.*). It is involved in some "minor" metabolic reactions and is present in almost all tissues. This enzyme exhibits maximum activity at pH 4.0; however, the difference in this index between the "old" and "young" cells can be better detected by certain biochemical methods at pH 6.0. The feasibility of using SA-β-Gal activity as a BA was first postulated in 1995 by Dimri *et al.*,[114] who demonstrated that the expression of this enzyme increases with aging both *in vitro* and *in vivo*. In subsequent years, this BA was widely used in cytogerontological experiments to assess the "age" of cells and is currently the most common in the studies[29,115] based on the definition of cellular senescence that we do not accept. However, in parallel, several studies were published whose authors emphasized that SA-β-Gal activity in cells is not so good a BA, because, in many cases, it depends not so much on age (both *in vivo* and *in vitro*) as on the method of research and/or the presence of certain pathologies as well as, what is most important, on the proliferative status of the cells.[116–122] It seems that cell proliferation restriction, for whatever reason (differentiation, contact inhibition, DDR, some diseases, *etc.*), is the factor that causes stimulation of SA-β-Gal expression. In other words, SA-β-Gal appears even in the "young" cells if their proliferation is suppressed. Not long ago, we showed[71] that in the stationary phase culture of transformed Chinese hamster cells, the proportion of cells