in which SA-β-Gal is detected by the method of Dimri *et al.* increases with time, and this is accompanied, on the one hand, by an increase in the level of poly(ADP-ribose) in the cells and, on the other hand, by a decline in their capacity to synthesize poly(ADP-ribose) in response to DNA damage induced by H_2O_2. Such data, in our opinion, provide further evidence of the viability of our concept of aging, which postulates the crucial role of cell proliferation restriction in the accumulation in cells of various macromolecular defects (the most important of which are DNA lesions), which, in turn, lead to deterioration in the functioning of organs and tissues and further increase in the probability of death of macroorganisms.[5,6,22]

It is also interesting to note that, in the experiments designed to compare the effects of "stationary-phase" or "stress-induced" (exposure to 4% ethanol for 2 h per day for 5 days) aging on the transformed Chinese hamster cells, we showed that the percentage of cells stained for SA-β-Gal by the method of Dimri *et al.* in a 14 day-old "stationary-phase-old" culture was much higher than in the "young" (7 day-old) control culture but comparable to that detected in 7 day-old cells incubated with ethanol.[123]

Finally, we would like to mention another study,[124] the authors of which showed that, both in the "stress-induced premature senescence" and in the replicative senescence "according to Hayflick," SA-β-Gal does not accumulate if the expression of the *GLB1* gene, which encodes the lysosomal β-galactosidase, is disrupted.

4.6 Conclusions

(1) We think that any "true" geroprotector should retard the age-related increase in the probability of death of aging organisms causing a rightward shift of the survival curve and increasing both the average and maximum life span.
(2) We do not think that the drugs that are used to combat age-related diseases could be considered geroprotectors, as well as the factors that increase the life span of the non-aging organisms.
(3) At present, there are several cytogerontological models that are used for testing of potential geroprotectors. The most popular among them are the Hayflick model, the stationary phase (chronological) aging model, and the cell kinetics model. In our opinion, the least number of problems associated with interpreting the results of testing potential geroprotectors in cytogerontological experiments arises when such studies are performed using the model of stationary phase aging (which is based on the concept of cell proliferation restriction as the main cause of accumulation of macromolecular lesions in cells of multicellular organisms with age, leading to the deterioration of the functioning of tissues and organs and, as a result, an increase in the probability of death) of normal cells. However, even this approach will not give the *final* answer to the question of whether or not the studied factor is a