It is very important which cell types are used in cytogerontological experiments on testing potential geroprotectors—normal or transformed cells of multicellular organisms, unicellular eukaryotic or prokaryotic organisms, *etc.* As noted above, differences in interpreting the results of geroprotector testing that were obtained on normal and transformed human or animal cells can become quite apparent when these results are extrapolated to humans, many of whom die from cancer. In particular, the biologically active compounds that reduce the viability of cultured cancer cells can extend the life span of humans and experimental animals, similarly to the agents that increase the viability of normal cultured cells. The use of unicellular organisms, such as bacteria or yeast, makes it possible to estimate the effect of various agents on the cells that represent independent organisms. However, a bacterium, for example, is so dramatically different from a mammalian cell that the same compound can kill the former but have hardly any effect on the viability of the latter (for example, this refers to antibiotics).

In our opinion, the use of the stationary phase aging model in many cases makes it possible to avoid many of these problems because the key factor that triggers the "aging" of all cells used in experiments is the restriction of cell proliferation with the help of various quite physiological impacts. A classic example is the chronological aging of yeast,[74,80] the results of studies of which are often pretty successfully used for studying the mechanisms of aging of humans and animals. In particular, experiments with the yeast *Saccharomyces cerevisiae* showed that rapamycin, a well-known mTOR inhibitor, in small doses that are sufficient for slowing down the proliferation of yeast cells but do not completely block this process, increases the culture life span in the chronological aging model.[100,101] Later this compound was shown to extend the life span of experimental animals—mice[102,103] and fruit flies.[104] It should be noted that, according to the ideas of some researchers,[101,105] the positive "gerontological" effect of rapamycin may be associated with the activation of autophagy. It also cannot be ruled out that the beneficial effect of rapamycin on the life span of animals may be due to its ability to suppress the emergence and development of malignant tumors.[17,106] As already mentioned above, in this case, it can hardly be considered a geroprotector. In addition, it is interesting to note that animals may develop tolerance to rapamycin over time. For this reason, some authors suggest that this drug should be used in combination with other active compounds, such as resveratrol.[107] Unfortunately, such problems are unlikely to be "caught" in cytogerontological studies.

4.5 Some Words About Biomarkers of Cell Aging/Senescence

It appears that, today, the construction of the survival curves of the test animal/human cohorts is the most reliable way to estimate the efficiency of interventions in the aging process. Unfortunately, this method is inefficient