parameters are very often confused in the cytogerontological literature). At the same time, data according to which the aging of an organism is largely determined by its postmitotic or very slowly proliferating cells (neurons, cardiomyocytes, hepatocytes, egg cells, *etc.*), which never have enough time to realize even the "normal" proliferative potential during the lifetime of the "host", have been ignored.[6,99] The majority of human cells do not proliferate or proliferate very slowly because they *should not* do it rather than because they *cannot*. Therefore, the induction of telomerase activity in the normal cells, leading to a significant increase in their mitotic potential (possibly, even making it unlimited), cannot be realized in these cells. And for the cells of the organism that already have telomerase (stem and germ line cells), this induction is even more useless.

If a test compound has a positive effect on the proliferative *activity* of cells (which is manifested, for example, in increasing their CFE), the effects of this drug on the organism can be dual type. On the one hand, for some cells (for example, those involved in the regeneration processes), such stimulation can be useful. On the other hand, this effect can, firstly, stimulate the proliferation of those cells that, as mentioned above, should not divide, and secondly, increase the probability of a rapid propagation of the precancerous (or even cancerous) cells present in the organism. An increase in the incidence of benign tumors also cannot be ruled out. However, it should be emphasized that the evaluation of the CFE of cells is one of the few methods that provide data on the characteristics of *individual* cells rather than the cell population in general.[92] In the latter case, information about possible subpopulations of cells that may differently respond to the test compound is lost due to averaging. For example, under the influence of a test factor, the content of 8-oxo-2′-deoxyguanosine, a popular aging biomarker,[70] in DNA of different cells may increase, decrease, or remain unchanged. As a result, the estimation of the content of 8-oxo-2′-deoxyguanosine *on average* can lead to the conclusion about the absence of changes in this parameter.

Some parameters used to assess cell viability in cytogerontological experiments can be purely "correlative",[40] so that their interpretation becomes even more complicated. For example, this applies to the saturating density of a cell culture. It is known that, for normal diploid cells, this parameter is inversely well correlated with the age of the cell donor (in this case, the cause–effect relationships remain unclear). It was this parameter we used in our cell kinetics model (see Section 4.2) to assess potential geroprotectors. It was assumed that the factors that increase the saturating density of the culture and, thereby, reduce the "biological age" of cells should have a positive effect on the viability and aging of a multicellular organism. However, in this case, we may face the same problems as in interpreting the data of experiments on CFE. It is not obvious that an improved ability of cells to reach a high saturating density in culture will slow down the aging of a multicellular organism in all cases. It cannot be ruled out that it may have no effect on the aging process at all or may even accelerate it.