parameter to the species lifespan, or its inverse relationship to the age of cell donor. When demonstrating that Hayflick's model is appropriate for studying aging mechanisms, it is usually emphasized that various changes, similar to those in the cells of an aging organism, take place in normal cultured cells as the number of cell population doublings increases.[24–26,33,52] In other words, cells either accumulate or lose something during aging *in vitro* in the same way as during aging *in vivo*. Therefore, it is again the case of *correlation*; this time it is correlation of the changes of certain biomarkers of aging (BA).

Despite its "correlativity," Hayflick's model has been widely used. Based on this model, a large body of data was obtained, which explained many problematic aspects in the life activity of organisms. In particular, it concerned the mechanisms of development and malignant transformation. However, the study of aging *in vitro*, in our opinion, practically did not help gerontologists to understand the fundamental mechanisms of aging and longevity.

Keeping in mind the main topic of the review, we should note that, when testing geroprotectors on the model system, researchers have followed either: (1) the proliferative potential of the cells studied, or (2) the process of the accumulation of various BA.

We developed another "correlative" model for testing of geroprotectors and geropromoters—the "cell kinetics model".[39,53,54] It is based on the well-known inverse correlation between the "age" of cultured cells (*i.e.*, age of their donor) and their saturation density.[55] This term is used for the maximum density (number of cells per square unit) of a cell culture in the stationary phase of growth when the cells stop propagating due to contact inhibition. It was assumed that the higher the saturation density is, the "younger" the cells studied are. The model allowed us to perform preliminary testing[33,39,56,57] of a lot of different compounds and factors (gamma-irradiation, DNA-alkylating agent thiophosphamide, low frequency electromagnetic field, antioxidants 2-ethyl-6-methyl-3-hydroxypyridine chlorohydrate and butylated hydroxytoluene, *etc.*) that are interesting from a gerontological point of view, but, unfortunately, it also revealed no information about the real mechanisms of aging or its modulation.

Unlike the Hayflick model and the cell kinetics model, which are based on a series of correlations, our model of "stationary phase aging" (accumulation of "age-dependent" injuries in cultured cells whose proliferation is restricted in a certain way, preferably by contact inhibition) is a "gist" model based on the assumption that processes taking place in this model system are essentially similar to those in an aging multicellular organism.[33,40,41,58–62] In fact, this assumption directly issues from our concept that the restriction of cell proliferation is the main mechanism providing for the accumulation of macromolecular defects in cells of aging multicellular organisms.[2,5,6,33]

Our numerous experiments provide evidence that changes in the cells occurring in our model system are indeed similar to those in the cells of aging multicellular organisms. They include accumulation of DNA single-strand breaks and DNA–protein crosslinks, DNA demethylation, changes in the level of spontaneous sister chromatid exchanges, structural defects in the cell nucleus, alterations in the plasma membrane, retardation of