third principle states that preventive maintenance replacement of functional elements in cells and tissues is the main line of assuring the high systems reliability. Following the preset genome pattern, unreliable elements should be timely replaced for novel ones before the phase of their wear-out begins. It is the so-called metabolic turnover. The fourth principle states that there is a finite number of critical elements that perform the supervisory functions over the organism's repair and renewal processes, *i.e.* over the metabolic turnover. Since these critical elements of the highest hierarchic level exert the control over the systems reliability, they can be called "longevity-assurance structures" (LAS). Inasmuch as all reliability facilities—among them preventive maintenance, repair, and redundancy of functional elements—are genetically limited, stochastic damages in LAS accumulate up to the preset threshold dysfunction levels. As a result, each organism has a limited life-span.[18,19] Indeed, it is common knowledge that there are neither mice nor rats exceeding 3–4 years of age, and that a human life-span does not exceed ≈120 years provided we take reliable data into account, not sensational press reports or legends. The limited lifetime of diploid cell strains is also a well-known phenomenon. For example, human fibroblasts *in vitro* die or mutate into cancer cells after performing about 50 doublings. American biologist Hayflick discovered this effect in 1961 (see ref. 1 and 2). Russian biologist Olovnikov explained Hayflick's limit suggesting the mechanism of the incomplete copying of telomere ends of DNA. According to Olovnikov's theory of marginotomy, every cell division is accompanied by the reduction of the telomere ends of cell chromosomes.[20] In essence, it means that the cell division stops as soon as the telomere circumcision runs up to the limit fatal level.

Following the reliability-theory approach, the simple mathematical model of aging was suggested first in our papers.[10–12] It was taken that LAS accumulate stochastic flaws resulting in disarray of their functions. Account was also taken of another widespread peculiarity of living systems, *i.e.* the existence of threshold values for the most important functional parameters. The organism has been assumed to perish the moment that any of LAS develops a threshold dysfunction (a limit, m_c). As a matter of fact, the life-span of the organism is determined by the threshold dysfunction of the worst LAS, *i.e.* by the weakest link's longevity. If N is the number of LAS, then the survival function is given by the smallest value of the random sample of size N with the following approximation for mortality rate:

$$h(t) = \Delta n(t)/n(t)\Delta t = h_0 \exp(\gamma t),\ h_0 = \gamma N/[\exp(\gamma T) - 1] \qquad (8.1)$$

where $n(t)$ is the number of live persons of the age t, Δn is the number of those who died during the time interval Δt, parameters h_0 and γ are independent of time.[10–19]

The exponential growth of mortality rate with time has long been known in quantitative gerontology and demography as the so-called "Gompertz law" of mortality. It has been confirmed for people (of age approximately