from 35 to 90 years), other mammals, flies, and mollusks.[1,2] Moreover, it was shown that aging of prokaryote cells, *Acholeplasma laidlawii*, in the stationary phase of growth—namely loss of viability measured as their ability to form macro-colonies—follows the same kinetic pattern.[21] It is noteworthy that the cited work[21] was the first one where it was demonstrated that the cell viability in cell cultures declines accordingly to the Gompertz law. Thus, the universal feature of aging, the Gompertz law of mortality, gets its explanation in the context of the reliability-theory approach stated above.

The limit life-span T in eqn (8.1) has appeared as the direct result of existence of the limit dysfunction, m_c, for LAS. This limit T is the life-span of an "ideal" organism with no initial flaws at $t = 0$. If we take from the review[1] that the maximum life-span for human populations, on average, is about 95 years, the magnitude of γ varies from 0.0612 to 0.119 years^{-1} and the magnitude of h_0 varies from 0.820×10^{-3} to 0.022×10^{-3} years^{-1}, then, using the expression for h_0, we find that $N \approx 5–15$. The values of N for dogs, mice and mares calculated by the relevant values of parameters (T, h_0, γ) fall in the same order of magnitude too.

An analytical transition from the abstract "longevity-assurance structures" to real biomolecular structures seems to be not easier than similar transitions from the "generalized co-ordinates" in theoretical physics. It is worthy, however, to note that this estimation corresponds, by the order of magnitude, to the number of the so-called "longevity-assurance genes" which have been recently discovered in nematodes, yeasts, drosophilae, mice, and other organisms (see in ref. 2 and 22). In humans and animals, these "longevity-assurance genes" are believed to be located in the special neurons of the suprachiasmatic nucleus of the hypothalamus.

8.3 Free-Radical Failures

8.3.1 Free-Radical Malfunctions of Electron-Transport Nanoreactors

The oxygen radical anion ($O_2^{\bullet-}$), the most important source of chemically reactive "toxic" oxygen species, is produced in cells and tissues of all aerobic organisms.[23–27] The main bulk of $O_2^{\bullet-}$ is formed as by-product of electron transport in cell mitochondria, the organelles that use up to 99% of all oxygen consumed by cells for ATP synthesis. Normal functioning of electron-transport nanoreactors (ETN) of mitochondria lies in the transport of electrons from the oxidation substrates, NADH and succinate, to cytochrome oxidase and then to oxygen with reduction of oxygen molecules to water and synthesis of ATP (see ref. 24). However, the reliability characteristics of mitochondrial nanoreactors are not perfect. As a result, normal elementary acts of electron transfers alternate with accidental malfunctions, which result in the formation of $O_2^{\bullet-}$. From the reliability point of view, the fact that this radical appears is to be considered as the random malfunction of ETN, similarly to "recurrent failures" in engineering.[10–19] Among other possible generators of