corticosteroids along with the decrease of thyrotropin and L-3,3′,5-triiodothyronine in blood plasma of rats after the BHT administration.

Hence, BHT induces the substantial shift in the activity of adenohypophysis gland, which is the source of corticotropin and thyrotropin hormones, and this is accompanied by the relevant shifts in the activity of peripheral endocrine glands, the adrenal cortex (the source of corticosteroids) and the thyroid gland (the source of triiodothyronine).[56,57] It is common knowledge that the release of corticotropin into blood followed by an increase in the synthesis of corticosteroids and a decrease in the synthesis of thyroid hormones is a significant phase of the system's adaptation to stress. It seems that, with regular introduction into animals' food, BHT as a mild stress factor 'trains' the neuro-hormonal system and, thus, increases the systems reliability, *i.e.* adaptive capabilities of the organism.[56–59] Hence, BHT is actually able to decrease the level of active oxygen species in myocardial cells and, probably, other cells too. However, the beneficial effect of this antioxidant is manifested not through direct radical elimination (scavenging), but in a preventive manner, *i.e.*, upon a decrease in the probability of their generation. Besides, it was shown by the spin probe technique that serum albumin,[54] a blood protein among the functions of which is transport of the hormone aldosterone, sorbs the hydrophobic BHT molecules. The hormone transport proteins can presumably serve as the molecular targets of the antioxidant. In addition, the BHT injections gave rise to the ESR signal from the nitrosyl complex of hemoglobin (NO-Hb) in the animal's blood.[58] It is generally known that nitric oxide serves as the signal molecule that causes, in particular, the relaxation of arterial smooth muscles that enhances the oxygen supply in myocardium.[60] Thus, there are the reasons to believe that BHT performs preventive maintenance against $O_2^{\bullet-}$ and its reactive products *via* hormonal/NO regulation.

The so-called mitochondria-targeted antioxidants can also act in a preventive manner. As the phenolic compounds, MitoVit-E and SkQ[41,42] have weakly acidic properties and, as such, they can serve as protonophore uncouplers, like, for example, 2,4-dinitrophenol, uncoupling electron transport and ATP synthesis in mitochondria.[24] In addition, hydrophobic cations can transfer counter-ions (anions) through a mitochondrial lipid membrane, thereby decreasing the transmembrane potential. Again, it should produce the uncoupling effect, according to the Mitchell theory of oxidative phosphorylation.[24] Indeed, molecules combining a hydrophobic part and a cationic group, in particular, a triphenylphosphonium group, serve as efficient uncouplers of oxidative phosphorylation in mitochondria. Actually, they were synthesized for this purpose, as uncouplers, about 40 years ago.[61] It is also known that the electron transport in mitochondria experiences a "back pressure" from the transmembrane potential.[24] Therefore, oxidative phosphorylation uncouplers, in particular, transmembrane transfer agents of protons and anions, decrease the transmembrane potential and thus decrease the generation of $O_2^{\bullet-}$ and other ROS in mitochondria. This provides grounds for believing that mitochondria-targeted antioxidants not so much scavenge directly the