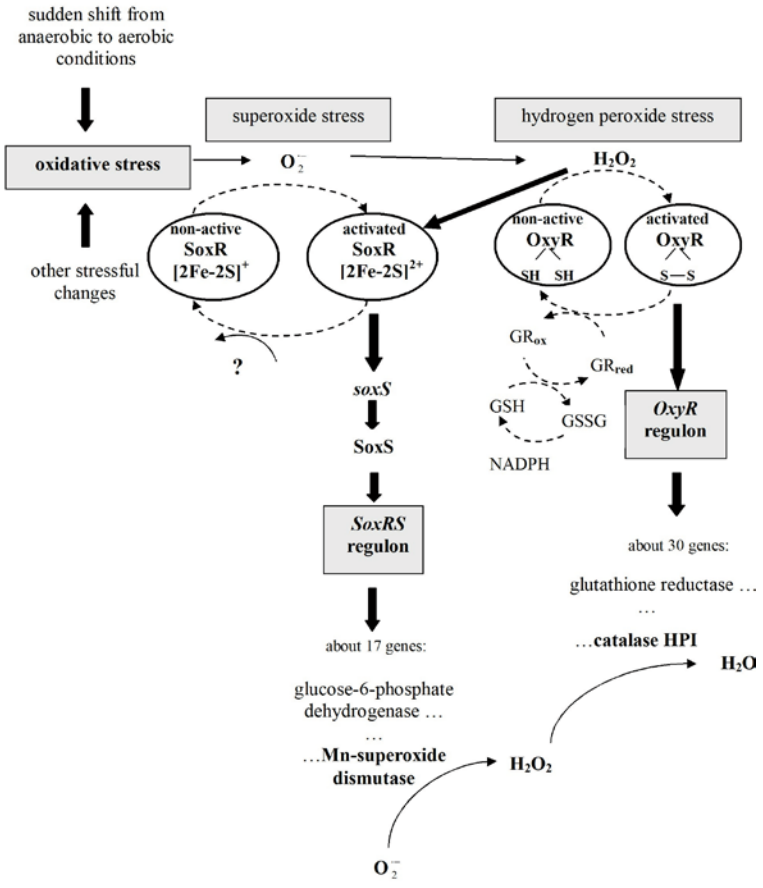

Figure 1. Transcriptional regulation of adaptive response to hydrogen peroxide in the bacteria *Escherichia coli*. Designations: SoxR, transcriptional factor of *SoxRS* regulon; OxyR, transcriptional regulator of *OxyR* regulon; GR_{red} and GR_{ox}, reduced and oxidized glutaredoxin; GSH and GSSG, reduced and oxidized glutathione.

tightly maintained at very low levels [5,6]. Therefore, facultative anaerobes posses a highly specialized antioxidant system that provides for their survival after a sudden shift in environmental conditions: from anaerobic to aerobic. Coordinated reorganization of gene expression is one of the most important aspects of cellular adaptation. Expression of the respective genes is associated with their relative importance for cell survival during oxidative stress. A set of genes encoding antioxidant enzymes is regulated by specific proteins, which can sense relatively small changes in cellular oxidant status [7]. Such a global response makes it possible to induce an adaptive metabolism including ROS elimination, the bypass of injured pathways, reparation of oxidative damages and maintenance of reducing power [8,9].

In the bacteria *E. coli*, most genes induced by oxidative stress are grouped into two regulons *SoxRS* and *OxyR* (Figure 1). The first one is under two-stage control of the transcriptional factor SoxR. Superoxide-generating compounds activate SoxR regulator by the one-electron oxidation of the 2Fe-2S clusters [10]. Oxidized SoxR then induces the expression of SoxS protein, which in turn activates the transcription of structural genes of the *SoxRS* regulon: *sodA* (Mn-superoxide dismutase (SOD)), *zwf* (glucose-6-phosphate dehydrogenase (G6PDH)), *acnA* (aconitase A), *nfsA* (nitrate reductase A), *fumC* (fumarase C), *nfo* (endonuclease IV), *etc.* [1,2,7,11,12]. Important antioxidant enzymes such as G6PDH provide the cell with reducing power NADPH and stress-inducible Mn-SOD are among *SoxRS* regulon members. Another form of SOD in *E. coli* is Fe-containing protein which does not respond to oxidative stress [1,2]. It has been estimated that SoxR is >90% reduced under normal aerobic conditions [13]. Being reduced, it cannot activate the *SoxRS* regulon. Several hypotheses were checked, but the question of how the oxidized SoxR is reduced after removing the oxidative challenge is still open [8,14].

For a long time it was widely believed that the *SoxRS* regulon cannot be induced by hydrogen peroxide [2,15,16]. At present, there are several reports on weak