Abstract

Reactive oxygen species (ROS) are inevitable byproducts during the course of normal aerobic metabolism. Biological cells have developed antioxidant systems to regulate the endogenous level of ROS, which damage the cells at higher concentration and can function as a cellular signaling molecule at lower concentration [1,2]. Peroxiredoxins (prx), a class of thiol-specific antioxidant proteins, together with peroxiredoxin reductases (prxR) are the predominant cellular defenses against oxidative stress and are also involved in cellular signaling pathways. The coordinated interplay between prx and prxR is essential for enhanced peroxidative catalytic rates. Many bacteria possess an alkyl hydroperoxide reductase (AhpR) system, composed of the two most abundant enzymes called AhpF (prxR) and AhpC (prx), which together catalyze the NADH-dependent reduction of H₂O₂ [3]. AhpC undergoes oligomeric states, which are redox-sensitive. Reduced AhpC preferentially assembles five catalytic dimers into a doughnut-like decamer, whereby the oxidized form of the protein leads to a lower order of oligomers between decamer and dimer [4].

In *E. coli*, like in most bacteria, the bipartite alkyl-hydroperoxide-reductase (AhpR), which is being among the ten most abundant *E. coli* proteins and therefore an ideal candidate to obtain a profound and detailed model, is an essential Prx representative. The *E. coli* AhpR system consists of two proteins, the 57 kDa large flavoprotein AhpF and the small subunit AhpC (Mr 21 kDa), which is devoid of chromophoric cofactors. The reduction of peroxides by the dimeric AhpC component uses a redox-active disulphide centre, while AhpF restores the reduced state of AhpC by transferring electrons from NADH onto the catalytic AhpC dimer. Structural and mechanistic insights of the *E. coli* AhpC-AhpF complex will be presented [4-6]. Furthermore, the related Peroxiredoxin complexes of the pathogenic *Enterococcus faecalis* and *Mycobacterium tuberculosis* will be shown as examples of niche adaptation.

Selected References