Abstract

CO_2, known for its role in acid-base regulations, is a mediator of peroxynitrite-induced oxidative damage and it also enhances Cu,Zn-SOD's antioxidant activity. These two means, by which CO_2 affects free-radical damage, are well-explored, but there are many other mechanisms. When CO_2 joins in free radical reactions, carbonate radical is often produced. The carbonate radical specifically damages substrates, but it is not possible to generalize, if the effect is pro-oxidant or antioxidant. A protective role of CO_2 has been observed during lipid peroxidation and during peroxynitrite-induced oxidation of DNA, when the carbonate radical caused injury to specific bases, but in the same time it prevented DNA strand breaks. Similarly, CO_2 prevented peroxynitrite-induced protein fragmentation as well as it caused injury to specific aminoacids. These observations are mostly based on experiments in a chemical system, which means under simplified conditions. In vivo, CO_2 exerts much more mechanisms to affect free radical reactions. Under more complex conditions, as cell culture is, there was an increase of oxidative stress after CO_2 exposure. Increased concentration of CO_2 causes a change in erythrocyte's function and an increase of oxidative stress on the organism's level.