

## Editorial:

### OXIDATIVE STRESS

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Oxidative stress is one of the most popular topics in medical research (Hengstler and Bolt 2007, 2008). Exposure to a multitude of reactive oxygen species-producing contaminants is inevitable. For example, generation of oxidative stress is a key mechanism of many non-genotoxic carcinogens. Oxidative stress damages all cellular biomacromolecules, particularly lipids, and can result in various diseased states, especially those associated with advancing age. The brain is particularly sensitive to oxidative stress for several reasons, including the presence of a high fraction of oxidizable polyunsaturated fatty acids, high iron content, and relatively low activities of antioxidant enzymes. In Table 1, the key message of recently published studies on oxidative stress has been summarized.

**Table 1:** Recent studies in **oxidative stress** research

Key message	Reference
Statins have antioxidative effects. Besides their cholesterol-dependent effects, they inhibit isoprenoids, which serve as lipid attachments for small Rho GTPases.	Adam and Laufs, 2008
The oxime 3-(phenylhydrazono) butan-2-one showed antioxidant effects in vitro. However, in vivo oxime pre-treatment of mice did not modify basal or induced lipid peroxidation.	Puntel et al., 2008
Methotrexate causes nitrosative stress in the small intestine of rats, which may be a critical mechanism for methotrexate-induced small intestinal damage.	Kolli et al., 2008
Beta-sitosterol shows antioxidative activity and may be further evaluated as a chemopreventive agent.	Paniagua-Pérez et al., 2008
Oxidative stress is involved in the induction of preneoplastic foci in rat liver by the peroxisome proliferator fenofibrate.	Nishimura et al., 2008
Curcumin decreases oxidative stress in the small intestine of rats.	Sivalingam et al., 2008
A comprehensive review on oxidative stress: modification of protein phosphatases and kinases, and transcription factors and their influence on cell proliferation, apoptosis and carcinogenesis.	Mates et al., 2008
Cyclophosphamide induces increased expression of paraoxanase (PON1) in kidney of rats, which can be interpreted as a mechanism to protect against oxidative stress.	Abraham and Sugumar, 2008
n-Hexane toxicity in vitro is caused by oxidative stress.	McDermott et al., 2008

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