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# Biolab Medical Unit

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## Why measure superoxide dismutase?

Superoxide dismutase (SOD) is essential for aerobic life and is one of the antioxidant enzymes that protect cells against damage by reactive oxygen species (ROS) and other free radical intermediates.

Antioxidant enzymes are preventative antioxidants, as opposed to radical-scavenging antioxidants (which directly remove ROS). It is generally found that changes in their activities precede cellular damage due to oxidative stress. This group of enzymes includes:

Superoxide dismutase (which has three isoforms – copper/zinc, manganese and iron),  
Catalase (a haem enzyme),  
Glutathione peroxidase (a selenoenzyme),  
Paraoxonase (which is found in HDL),  
Glutathione reductase (which regenerates GSH from oxidized glutathione inside the cell),  
Glutathione-s-transferase (which detoxifies carbon-centred electrophilic agents, such as many drugs).

Oxidative stress, a disturbance of the pro-oxidant / anti-oxidant balance in favour of the former (Sies, 1985) is a condition in which products of oxidative damage may be detectable in body fluids (for example an increase in malondialdehyde production from lipid peroxidation). Other effects may be evident, for example damage to cell membranes (causing loss of magnesium and membrane essential fatty acid deficiencies) as well as damage to DNA, protein and carbohydrate residues. An excess of pro-oxidants (free radicals) and a relative deficiency of anti-oxidants is a characteristic of many disease states.

Although it is primarily an intracellular enzyme, SOD has a general anti-inflammatory effect, protecting against excess superoxide radicals formed during leukocyte phagocytosis (killing of bacteria by white cells). This is probably why SOD deficiency can be associated with skin lesions in affected subjects.

**Elevated red cell SOD** activity suggests an early response to oxidative stress, in which increased cellular demand induces synthesis of the enzyme.

**Reduced red cell SOD** activity suggests micronutrient deficiencies, either due to poor diet or a later consequence of oxidative stress. These deficiencies are of zinc and copper from the cytosol, or of manganese from the mitochondria. In general ensuring adequate zinc nutrition, along with alleviation of the cause of the oxidative stress, allows these deficiencies to correct. In some cases conservative replacement of copper or manganese may be indicated. Note that copper may be present in excess in the extracellular fluid while deficient intracellularly, having been exported from the cell as part of an inflammatory response.