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Reduced level of serum thiols in patients with a diagnosis of active disease.

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Source

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Abstract

Oxidative stress, or the production of oxygen-centered free radicals, has been hypothesized as the major source of DNA damage that in turn can lead to altered genetic expression, disease, and aging of humans. Serum protein thiol levels in blood are a direct measure of the in vivo reduction/oxidation (redox) status in humans, because thiols react readily with oxygen-containing free radicals to form disulfides. Moreover, serum thiols also reflect DNA repair capacity and the possible eventual accumulation of genetic damage, since a key DNA repair enzyme, poly ADP-ribose polymerase (PARP), is thiol/disulfide redox regulated. This study tests the hypothesis that serum protein thiols can be used to estimate individual aging status by comparing the levels of apparently healthy subjects (n = 90) to those of individuals (n = 306) with an active disease diagnosis. Nine categories of human disorders all showed highly significant reductions in serum protein thiols from 46 to 91 nM cysteine/200 microL serum (mean +/- S.D. = 59 +/- 40) compared to a control mean of 128 +/- 39 nM cysteine/200 microL serum (p <.001). These data strongly confirm an important role of oxidative stress in human disease development, and identify serum thiol status as a potential biochemical endpoint useful in the assessment of aging.

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