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Antioxidative mechanisms and plasma growth hormone levels: potential relationship in the aging process.

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Factors affecting longevity are complex and poorly understood. We have recently found that Ames dwarf mice (df/df), which are deficient in growth hormone (GH), prolactin, and thyroid-stimulating hormone, live significantly longer than their normal siblings whereas transgenic mice that overexpress GH exhibit reduced life-spans and various indices of premature aging. The production of reactive oxygen species increases with aging and is associated with DNA damage to the tissues. However, several cellular oxygen scavenging/detoxifying systems exist that improve the antioxidative defense capacity of cells. We evaluated the activity of enzymes involved in this defense system in liver, kidney, and heart tissue from dwarf, phosphoenol-pyruvate carboxykinase-bovine GH transgenic, and corresponding groups of normal mice. Liver glutathione and ascorbate levels were lower ($p < 0.0025$) in dwarf animals compared to normal and GH transgenic mice. By contrast, the level of catalase activity, which detoxifies hydrogen peroxide, in dwarf liver and kidney was significantly higher when compared to the other groups. Animals deficient in GH (dwarf) live longer and exhibit enzyme activities and levels that may combat oxidative stress more efficiently than normal mice and those overexpressing GH.

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