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Posttranslational modification of proteins in aging: impact of exercise

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Oxidative stress results in increases in protein carbonylation, which has been shown to associated with impaired cellular function. Regular exercise, on the other hand, decreases the level of oxidative damage on proteins, and enhances physiological function. Silent information regulators are potent NAD+-dependent protein deacetylases, which have been shown to regulate gene silencing, muscle differentiation and DNA damage repair. Acetylation/eacetylation could effect protein stability, since lysine residues are the site of ubiquitination and acetylation as well. The age associated decrease in SIRT1 activity could result in increased level of acetylation, hence decreased ubiqutination, which could result in increased half-life of proteins. Aging is associated with decreased rate of protein turnover, which one of the reason of increased accumulation of free radical induced oxidative damage. Therefore, it appears that the oxidative stress associated impairment of cell function during aging, at least a part, could be due to the SIRT1 mediated decreases in deacetylation. This idea is supported by activating effects of caloric restriction on SIRT1. In addition, caloric restriction is a known mean to shorten the half-life of proteins. Regular exercise attenuates the age-associated decline in the activity of SIRT1, which impacts ubiqutination and protein turnover.