

BIOMARKERS OF OXIDATIVE STRESS AND CARDIOVASCULAR DISEASE IN HUMANS AND CHIMPANZEES

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Humans and chimpanzees are remarkably similar; but humans live twice as long as chimpanzees and therefore are believed to age at a slower rate. In the oxidative stress hypothesis of aging, aging is the result of cumulative damage by reactive oxygen species. The purpose of this study was to compare biomarkers for cardiovascular disease and oxidative stress between 10 captive male chimpanzees and 10 humans. Compared with men, male chimpanzees were at increased risk for cardiovascular disease because of their significantly higher levels of fibrinogen, IGF1, insulin, lipoprotein a, and large high-density lipoproteins. Chimpanzees showed increased oxidative stress, measured as significantly higher levels of 5-hydroxymethyl-2-deoxyuridine and 8-iso-prostaglandin F2 α , a higher peroxidizability index, and higher levels of the prooxidants ceruloplasmin and copper. Chimpanzees also had decreased levels of antioxidants, including α - and β -carotene, β -cryptoxanthin, lycopene, and tocopherols, as well as decreased levels of the cardiovascular protection factors albumin and bilirubin. In addition, genetic evidence indicates that chimpanzees have a lower expression of peroxiredoxins and elevated expression of thioredoxin interacting protein suggesting that chimpanzees are at increased risk for oxidative stress, compared to humans. As predicted by the oxidative stress hypothesis of aging, male chimpanzees exhibit higher levels of oxidative stress and a much higher risk for cardiovascular disease, particularly cardiomyopathy, compared with men of equivalent age. Given these results, we hypothesize that the longer lifespan of humans is at least in part the result of greater antioxidant capacity and lower risk of cardiovascular disease associated with lower oxidative stress.

Keywords: Pan troglodytes, Homo sapiens, antioxidant levels, aging