Regular exercise training does not elevate oxidative stress or deplete antioxidant defences

TA Watson, LK MacDonald-Wicks, ML Garg

Nutrition & Dietetics, School of Health Sciences, University of Newcastle, NSW, 2308

Prolonged, strenuous and/or exhaustive exercise has been shown to generate reactive oxygen species (ROS), which may exceed the capacity of antioxidant defence mechanisms and lead to oxidative stress. Oxidative stress has been linked to the pathogenesis of many chronic diseases and has been shown to influence factors that adversely affect exercise performance. In a case-control study, F_2 -isoprostane (8-iso-PGF2 α) total antioxidant capacity (TAOC), vitamins A, C and E, β -carotene, uric acid, erythrocyte glutathione peroxidase (GSHPxase) and superoxide dismutase (SOD) activity, intake of dietary antioxidants and physical activity levels of 20 exercise trained subjects and 20 age- and sex-matched sedentary controls were examined to establish relationships between exercise, intake of dietary antioxidants and oxidative stress. Energy intakes of trained volunteers were significantly higher but no significant differences in antioxidant intake (vitamin C and β -carotene), compared to sedentary controls were observed. Energy requirements derived from the International Physical Activity Questionnaire were similar between the two cohorts, however expected differences were observed in percentages of contribution to energy expenditure for the high intensity, low intensity and sitting down activities. Trained subjects had higher plasma levels of vitamins A, C, E and β -carotene when compared to sedentary controls. No significant difference was observed between the groups for plasma F_{2} -isoprostanes, TAOC and uric acid, erythrocyte GSHPxase and SOD activity, or dietary intake of vitamins. Participation in regular strenuous exercise did not induce oxidative stress or reduce antioxidant defences at rest in those who consumed a diet meeting or exceeding recommended levels of antioxidants. It appears that there would be no apparent benefit of antioxidant supplementation in athletes provided their diet provides adequate amounts of antioxidants.

	Control ^{1,2}	Training ^{1,2}	
$\overline{F_{2}}$ -Isoprotane (pg/ml)	46.5 ± 5.2	45.5 ± 3.2	
β -Carotene (µmol/L)	0.15 (0.1-0.2)	0.40 (0.2–0.6)*	
Vitamin A (µmol/L)	1.655 ± 0.13	$2.093 \pm 0.11*$	
Vitamin E (µmol/L)	16.8 ± 1.1	$20.1 \pm 0.9^*$	
Vitamin C (µmol/L)	38.5 ± 5.1	$51.4 \pm 3.3^*$	
Uric acid (mmol/L)	0.29 ± 0.01	0.29 ± 0.01	
GSHPx (U/mgHb)	21.79 ± 1.1	20.11 ± 1.2	
SOD (U/mgHb)	3.14 ± 0.09	3.11 ± 0.08	
Antioxidant Capacity (µmol/L)	287 (247–336)	275 (257–309)	

¹ normally distributed data expressed as mean \pm SEM. ² non-parametric data expressed as median (25th-75th percentile). *P < 0.05.

References

- 1. Davies KJ, Quintanilha AT, Brooks GA, Packer L. Free radicals and tissue damage produced by exercise. Biochem Biophys Res Commun 1982; 107: 1198–1205.
- 2. Bejma J, Ramires P, Ji LL. Free radical generation and oxidative stress with ageing and exercise: differential effects in the myocardium and liver. Acta Physiol Scand 2000; 169: 343–351.
- Ashton T, Young IS, Peters JR, Jones E, Jackson SK, Davies B, Rowlands CC. Electron spin resonance spectroscopy, exercise, and oxidative stress: an ascorbic acid intervention study. J Appl Physiol 1999; 87: 2032–2036.

Key words: exercise, oxidative stress, anti-oxidants