

THE INFLUENCE OF IRON DEFICIENCY ON SELENIUM  
STATUS AND GLUTATHIONE PEROXIDASE ACTIVITY

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Iron deficiency anaemia has been reported to decrease activity of glutathione peroxidase (GSH-Px) in erythrocytes of rabbits (Rodvien et al. 1974) and humans (Cellerino et al. 1976). GSH-Px is a Se-containing enzyme which helps to protect cellular lipids and proteins from potentially damaging peroxides. Since these studies did not measure tissue or dietary Se, it is impossible to establish whether iron deficiency had a direct effect on Se status or a less direct effect on GSH-Px activity. Two experiments investigated the effect of iron deficiency on tissue Se levels and GSH-Px activity in rabbits fed high ( $1 \mu\text{g g}^{-1}$ ) or marginally adequate ( $0.2 \mu\text{g g}^{-1}$ ) Se.

Fifteen 6-week-old rabbits were given the following treatments: Group 1, Fe-deficient diet + phlebotomy; Group 2, Fe-supplemented diet ( $160 \mu\text{g g}^{-1}$ ) + phlebotomy; and Group 3, Fe-supplemented diet. All diets contained  $1 \mu\text{g g}^{-1}$  Se. After 8 weeks, blood was analysed for Hb, Ht, RBC count, MCV, Se GSH-Px, plasma Se, whole blood Se, plasma Fe, TIBC and FEP. Livers were analysed for total and Se-dependent GSH-Px, Se and Fe. Experiment 2 design was the same as experiment 1 except that the diet contained  $0.2 \mu\text{g g}^{-1}$  Se.

Se and Fe concentrations in rabbits fed  $1 \mu\text{g g}^{-1}$  dietary Se (Mean  $\pm$  SD)

Group	Plasma ( $\mu\text{g } 100\text{ml}^{-1}$ )		Liver ( $\mu\text{g g}^{-1}$ )	
	Fe	Se	Fe	Se
1 Fe deficient	$76 \pm 7^a$	$17.0 \pm 3.3$	$14.1 \pm 1.4$	$2.38 \pm 0.29^c$
2 Phlebotomy controls	$244 \pm 20$	$17.4 \pm 4.2$	$76.2 \pm 21.8$	$1.57 \pm 0.72$
3 Controls	$255 \pm 51$	$19.3 \pm 3.0$	$133.5 \pm 36.4^b$	$1.45 \pm 0.38$

a Significantly different from groups 2 and 3 ( $P < 0.001$ ),

b Significantly different from groups 1 ( $P < 0.001$ ) and 2 ( $P < 0.05$ ),

c Significantly different from group 3 ( $P < 0.01$ ).

The severe iron deficiency produced in Group 1 did not adversely affect plasma or liver Se concentrations. Iron status may have an indirect effect on GSH-Px activity. Iron-deficient rabbits showed an apparent increase in erythrocyte GSH-Px activity when expressed per g Hb. This increase was due mainly to the large decrease in Hb in iron-deficient rabbits rather than a real increase in enzyme activity. Iron-deficient rabbits fed  $1 \mu\text{g g}^{-1}$  Se (but not those fed  $0.2 \mu\text{g g}^{-1}$ ) had reduced liver Se GSH-Px activity. This decrease was not due to lack of Se for enzyme synthesis but probably resulted from lower iron-induced oxidative stress.

These results demonstrate that (a) acute iron deficiency does not adversely affect Se status when Se intakes are marginally adequate or high and (b) the use of erythrocyte GSH-Px as an indicator of Se status may be in error in iron-deficient subjects, especially when activity is expressed per g Hb. If erythrocyte GSH-Px is used to assess Se status, then the activity should be expressed per  $10^{10}$  RBC and per ml RBC as well as per g Hb.

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