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Brief Communication

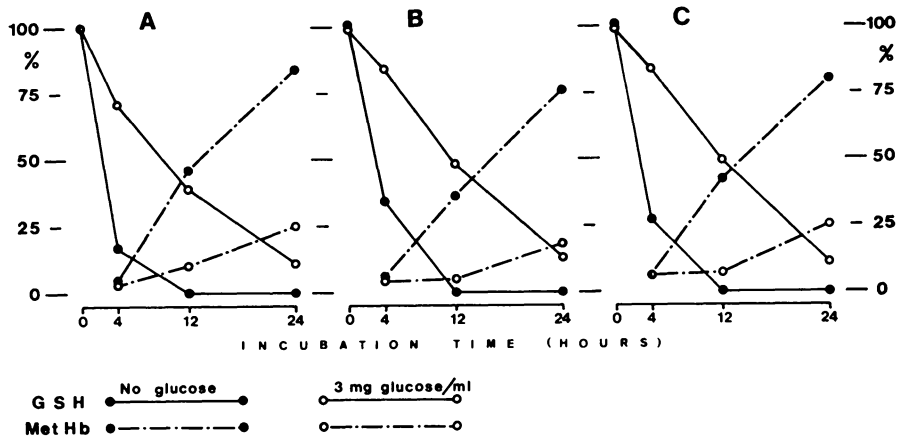
THE EFFECT OF SELENIUM DEFICIENCY ON COPPER-INDUCED OXIDATION IN SHEEP ERYTHROCYTES

In 1972 *Rotruck et al.* reported that dietary selenium protected rat erythrocytes against ascorbic acid-induced methemoglobinenia and hemolysis in vitro, provided that glucose was present in the incubation medium. Dietary vitamin E protected against hemolysis only.

The effects produced by Cu^{++} ions on erythrocytes from sheep and other species in vitro (*Steiner* 1973, *Sivertsen* 1980) are similar to the ascorbic acid-induced changes. From this, one might expect an effect of selenium status on erythrocyte resistance to copper. In the present work Cu^{++}-induced glutathione and hemoglobin oxidation was studied in vitro in erythrocytes from sheep with deficient and adequate selenium status.

Three ewe lambs born in the spring of 1978 were fed low-selenium hay and barley from October 1978 to July 1979. Whole blood selenium concentrations fell from 0.06—0.13 μg/ml in October to 0.02—0.05 μg/ml in April, and plasma tocopherol concentrations from 1.3—1.7 μg/ml to 0.3—0.8 μg/ml in the same period. From May 16 onwards, one of the animals (Ewe No. 1) was given injections of selenium-vit. E (Tokosel® (Agrivet), 5 ml (3.0 mg Se/150 mg tocoph. acet.) initially, thereafter 2 ml every 10th day). At the end of June, she had a blood selenium level of 0.09—0.13 μg/ml and an erythrocyte GSH-Px activity of 700—750 μkat/l, while plasma tocopherol varied from 0.5 to 1.2 μg/ml. Corresponding levels in the uninjected ewes No. 2 and No. 3 were 0.02—0.04 μg Se/ml blood, 100—160 μkat GSH-Px activity/l erythrocytes, and 0.2—0.7 μg tocopherol/ml plasma.

Copper incubation experiments were done with eight blood samples from these animals, taken at March 28, April 4, May 14, June 14 and June 22. The erythrocytes were separated from plasma, washed twice and incubated with Cu^{++} and/or glucose for 24 h. Conditions of incubation and experimental methods were as described in a previous study (*Sivertsen*), except that the flasks were agitated continuously, and the 10 μg Cu^{++}/ml parallels were omitted.



F i g u r e 1. GSH and methemoglobin in sheep erythrocytes, incubated at 37°C in physiological saline solution with 5 μg Cu^{++}/ml; with and without glucose. GSH is given in per cent of zero-time value, methemoglobin in per cent of total hemoglobin.

A. Erythrocytes from Ewe No. 1, March 28, 1979. Blood Se conc. 0.02 μg/ml.

B. Erythrocytes from Ewe No. 1, June 22, 1979. Blood Se conc. 0.11 μg/ml, GSH-Px activity 700 μkat/l.

C. Erythrocytes from Ewe No. 3, June 22, 1979. Blood Se conc. 0.03 μg/ml, GSH-Px activity 140 μkat/l.

Fig. 1 shows GSH and methemoglobin values in the flasks containing Cu^{++}, for three representative incubation experiments. The variations from experiment to experiment were generally small. There was a covariation between GSH oxidation and methemoglobin formation, but significant correlation to the blood selenium level was found only in GSH values after 4 h' incubation (without glucose). The effect of glucose in counteracting hemoglobin oxidation showed practically no variation.

In the flasks lacking Cu^{++}, GSH oxidation was much slower and methemoglobin oxidation was never above 10 %; with glucose present both were negligible. Hemolysis was minimal (< 2 % in 24 h) in all flasks.

In conclusion, the experiments did not show any effect of selenium status similar to that observed by *Rotruck et al.* on ascorbic acid oxidation in rat erythrocytes. It is possible that a more extreme selenium deficiency is needed to reveal an effect of this kind. However, the blood selenium values recorded in the present study cover a practical range from deficient to adequate selenium status (*Lunde & Ødegaard* 1972). The results there-

fore indicate that under field conditions the copper resistance of sheep erythrocytes is not affected by selenium deficiency to any practically significant degree. This is in accordance with the results of a previous study on selenium content in livers of normal and copper-poisoned sheep (*Sivertsen et al.* 1978).

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