

Brief Communication

GLUTATHIONE PEROXIDASE AND HEALTH IN SWINE

The selenium dependent enzyme glutathione peroxidase (GSH-Px) participates in the mechanisms protecting cells against damage due to peroxides. During the phagocytic action of macrophages and leucocytes considerable amounts of hydrogen peroxide generate in these cells, which normally have high GSH-Px activity (*Serfass & Ganther 1976*). Since GSH-Px activity in many tissues closely reflects the Se-status of an animal, one might expect Se-deficiency to cause decreased resistance to infections. Such an effect was found in rats where macrophages isolated from Se-deficient animals had only 5—10 % of the GSH-Px activity found in cells from control rats, and their microbicidal activity was lower than that of normal macrophages (*Serfass et al. 1974*). Similar results were reported for leucocytes from Se-deficient steers (*Boyne & Arthur 1978*). Se-supplementation of Se-vitamin E deficient pigs has also been found to prolong the incubation period in experimental swine dysentery (*Teige et al. 1978*).

A high incidence of infectious diseases such as pneumonia and diarrhoea is one of the major problems in pig production today. Besides, the Se-status of Danish Landrace pigs varies considerably, which to a certain extent is genetically determined (*Jørgensen et al. 1977, Wegger et al. 1979*). These facts together with the results cited above led us to investigate the incidence of disease in swine in relation to their Se-status.

The study comprised 691 Danish Landrace pigs reared at a Danish progeny testing station from about 22 kg of body weight till slaughter (90 kg body weight). For details of management see *Pedersen (1979)*. The GSH-Px activity in erythrocytes was used as parameter for Se-status (*Sivertsen et al. 1977*). Blood samples were taken when the pigs had been at the station approx. 3 weeks. GSH-Px activity was measured as described by *Jørgensen et al.* Morbidity and diagnosis were recorded for each individual pig at the station and on the slaughter line by the routine meat inspection. The results were grouped in classes according to GSH-Px activity, the class width being 24 u/g of haemoglobin. Within each class the percentage of pigs affected

by disease at any time during the growth period was calculated, and so was the percentage of pigs showing signs of present or past disease on the slaughter line.

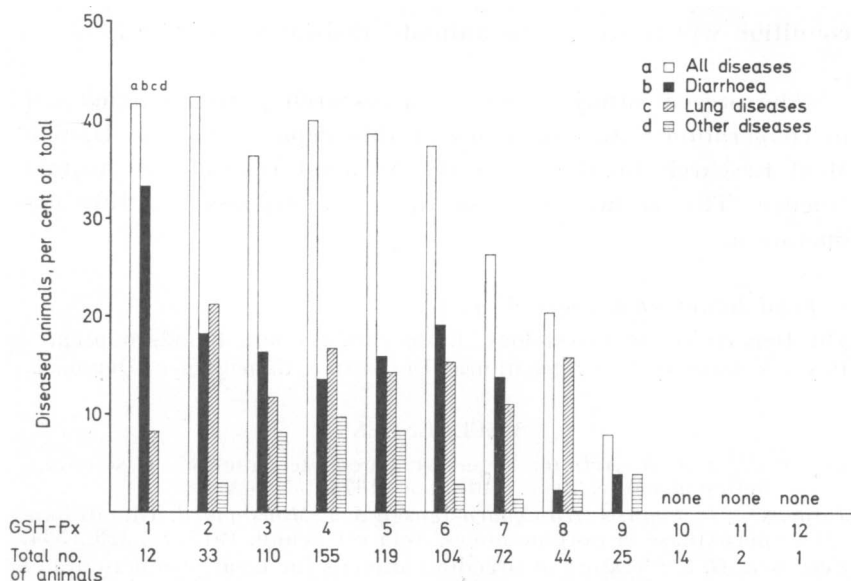


Figure 1. Disease frequency during the growth period of pigs in relation to erythrocyte GSH-Px activity. The enzyme activity increases gradually from Group 1 to Group 12 within the range 7—288 u/g of haemoglobin, the class width being 24 u/g of haemoglobin.

Figure 1 shows the results obtained for the growth period. Diarrhoea and pneumonia were the predominant diagnoses in all groups, but as seen from the figure, pigs with a high level of GSH-Px activity, i.e. a high Se-status, were less susceptible to disease than those with low GSH-Px. The term “Other diseases” comprises a wide variety of disorders each occurring with a low and inconsistent frequency not related to GSH-Px classes. The correlation between GSH-Px activity and total percentage of pigs with disease remarks was highly significant ($P < 0.001$). A similar although less pronounced relationship was found on the slaughter line ($P < 0.05$).

It should be underlined that the animals were fed a Se-vitamin E adequate diet (*Pedersen*), and no clinical signs of Se-deficiency were apparent. Thus the results indicate that individual differences in Se-requirement exist in pigs. Taking into account the standardized management of the animals the most

likely explanation is the existence of genetic variation between pigs in their ability either to absorb selenium, to incorporate this element in GSH-Px or to synthesize the apoenzyme. These differences may result in marginal Se-deficiency in some pigs, a condition which lowers the animals' resistance to infections.

The present study is part of a research project carried out in cooperation with colleagues at this department, the Danish Meat Research Institute and the National Institute of Animal Science. The authors express their gratefulness for this cooperation.

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REFERENCES

- Boyne, R. & J. R. Arthur*: Defective leucocyte function in selenium deficient cattle. *Proc. Nutr. Soc.* 1978, **38**, 14A.
- Jørgensen, P. Fogd, J. Hylgaard-Jensen & J. Moustgaard*: Glutathione peroxidase in porcine blood. *Acta vet. scand.* 1977, **18**, 323—334.
- Pedersen, O. K.*: Testing of breeding animals for meat production and meat quality in Denmark. *Acta agric. scand.* 1979, Suppl. 21, 122—135.
- Serfass, R. E. & H. E. Ganther*: Effects of dietary selenium and tocopherol on glutathione peroxidase and superoxide dismutase activities in rat phagocytes. *Life Sci.* 1976, **19**, 1139—1144.
- Serfass, R. E., R. D. Hinsdill & H. E. Ganther*: Protective effect of dietary selenium on salmonella infection: Relation to glutathione peroxidase and superoxide dismutase activities of phagocytes. *Fed. Proc.* 1974, **33**, 694.
- Sivertsen, T., J. T. Karlsen & A. Frøslie*: The relationship of erythrocyte glutathione peroxidase to blood selenium in swine. *Acta vet. scand.* 1977, **18**, 494—500.
- Teige, J., F. Saxegaard & A. Frøslie*: Influence of diet on experimental swine dysentery. 2. Effects of a vitamin E and selenium deficient diet supplemented with 3 % cod liver oil, vitamin E or selenium. *Acta vet. scand.* 1978, **19**, 133—146.
- Wegger, I., K. Rasmussen & P. Fogd Jørgensen*: Selenomsætningen hos svin. Glutathion peroksydase aktivitet i lever og nyre som indikator for selenstatus. (Selenium metabolism in swine. Glutathione peroxidase activity in liver and kidney as indicator of selenium status). *Kgl. Vet.- og Landbohøjsk., Inst. Sterilitetsforsk., Årsberetn.* 1979, **22**, 127—134. København.

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Figure 1. Scanning electron micrograph of anterior duodenum (d1), calf 2. Blunt, nodular villi with furrows (arrows). Large irregular villus (LV). $\times 150$.

Figure 2. Scanning electron micrograph of an area adjacent to Fig. 1. Bridge-like connections between villi (arrows). $\times 200$.

Figure 3. Scanning electron micrograph of middle duodenum (d2), calf 2. Tongue-shaped villi with prominent transverse furrows (arrows). $\times 150$.

Figure 4. Villi adjacent to Fig. 3 with a moderate mononuclear infiltration of lamina propria, vacuolation (V) of apical enterocytes, subepithelial capillaries (C), dilated lacteals (L), and furrows (F). Crypts of Lieberkühn (Cr) are also visible. HE, $\times 130$.

Figure 5. Scanning electron micrograph of villi in anterior jejunum (aj3), calf 3. Finger-shaped villi and "buttons" at the tips of the villi, probably enterocytes to be shed during normal extrusion (arrows). $\times 150$.

Figure 6. Scanning electron micrograph of middle jejunum (mj), calf 2. Leaf-shaped villi. $\times 100$.

Figure 7. Scanning electron micrograph of ileum, calf 3. Villi are conical and of different sizes. Small "pseudovilli" (P) with bulging of individual cells are seen between the other villi. $\times 150$.

Figure 8. Ileum, calf 2. A "conventional" villus has numerous goblet cells (G) whereas the "pseudovillus" (P) has few goblet cells and a stroma consisting of lymphoid cells. Many polymorphonuclear leukocytes (arrows) are seen in the epithelium of the "pseudovillus". AB-PAS, $\times 130$.

Figure 9. Scanning electron micrograph of the apex of a "pseudovillus", posterior jejunum (pj 2), calf 1. Individual cells are bulging and some have pores (arrows). $\times 1000$.

Figure 10. Apex of a "pseudovillus" in ileum, calf 2. Vacuoles (V) and leukocytes (L) in the epithelium. HE, $\times 510$.

Figure 11. Apex of villus in middle duodenum (d2), calf 2. The enterocytes have prominent vacuoles (V) and sometimes displaced nuclei. C = subepithelial capillaries. HE, $\times 550$.

Figure 12. Area adjacent to Fig. 11. The enterocytes contain large fat droplets (arrows) and crescent-shaped nuclei (N). Oil red 0, $\times 550$.

Figure 13. Scanning electron micrograph of colon, calf 1. Smooth mucosa with narrow clefts (large arrows) and openings of goblet cells (small arrows). $\times 150$.

Figure 14. Colon, calf 3. Crypts are rich in goblet cells (small arrows) and a furrow (large arrow) is seen. AB-PAS, $\times 150$.

