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Table 1: After treatment of dog glial cell cultures with X/XO and ROS scavengers or metal chelators, oligodendrocytes were classified in three groups according to their morphological appearance: (+). Cells that have lost their peripheral fine branching of processes, (++) cells with major loss of processes and cytoplasmic protrusions, (+++) cell-fragments without processes.

Scavenged Species	Scavenger	Concentration	Damage
O_2^-	SOD	100 U/ml	++
H_2O_2	Catalase	100 U/ml	++
$H_2O_2+O_2^-$	SOD/Catalase	100 U/ml	+
$^{\circ}OH$	Mannitol	50 mM	+
Fe^{++}	Desferal^R	50 µM	+
None			+++

tely provide data for therapeutic intervention of brain damage in encephalitis caused by ROS.

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PATHOLOGICAL FINDINGS IN DIETARY PRODUCED OXIDATIVE STRESS IN GROWING PIGS

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The classical mulberry heart disease (MHD) with microangiopathy (MAP) of pigs, according to Grant (1961), is characterized by vascular lesions principally in the myocardium whilst degenerative changes occur inconstantly. Van Vleet et al. (1977) described an atypical mulberry heart disease involving mainly degenerative changes.

The aetiology of MHD is not yet clearly understood although the present knowledge presume a multifactorial genesis with dietary involvement. According to Korpela (1988), an increased myocardial and hepatic iron concentration in pigs with microangiopathy acts as a risk factor of oxidative damage.

We produced an experimental myodegeneration of skeletal muscles in all 8 pigs with a diet deprived selenium and vitamin E. In order to produce MAP we had provoked the oxidative stress by injecting 3 ml irondextran IM in four pigs.

Macroscopically the condition of nutritional myodegeneration, also known as white muscle disease, was characterized by an overall pale, yellowish colour and a translucence of the skeletal muscles. Further gross pathological changes included ascites (100%), hydrothorax (37%), pericarditis (62%) as well as edema of lymph nodes (50%) and lungs (50%).

The microscopical examination of the skeletal muscle (M. longissimus dorsi) revealed varying degrees of degeneration including swelling of muscle fibers and loosening of the fibrillar pattern. Also the longitudinal and cross striations were no longer visible. The local arrangement of nuclei in chains was considered being a reparative process. The histopathology showed further hepatosis diaetetica and myocardial degeneration as described by Grant (1961) and Van Vleet et al. (1977).

As previous studies show, selenium deficiency is associated with hepatosis diaetetica and nutritional myodegeneration but not with MAP (Lindberg et al., 1972; Moir and Masters, 1979). In our study none of the pigs showed vascular lesions in the myocardium. Further experiments are necessary to study the role of iron supplementation in dietary produced oxidative stress in growing pigs.

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PATHOLOGY OF AGING IN THE CHICKEN

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Although the process of aging in man and laboratory animals has been studied quite extensively during the last couple of decades (Andrew, 1971), not very many data concerning food producing animals are available. This is mainly due to the fact that for economic reasons the life of these animals is considerably shorter than it would be under natural circumstances.

In order to make a contribution to the knowledge of the pathological processes which take place in senescent animals, we carried out a statistical and morphological study on the pathology of aging in the chicken with the belief that these observations could be of some relevance both to veterinary and comparative pathology.

Sixty-seven chickens, 61 females and 6 males, obtained from small farms at different ages were housed in our facilities until natural death occurred. The animals, aged 3 to 11 years, were necropsied immediately after death and the gross lesions were recorded.

The cause of death could be established in 51 animals and the related results are summarized in Table 1.

The data concerning the occurrence of the gross lesions involving the different organ systems are reported in Table 2. The alimentary tract was affected by pathological changes in 93% of the chickens; these mainly consisted of inflammatory, degenerative, necrotic and neoplastic (Fig. 1, 2) lesions affecting the liver parenchyma. Very often ascites and peritonitis could also be observed. 84% of the