Jpn. J. Vet. Res. **43**(1): 29–71 (1995)

INFORMATION

Hokkaido University granted the degree of the Doctor of Veterinary Medicine to the following 37 graduates of the Faculty of Veterinary Medicine on 24 March, 1995.

The authors' summaries of their theses are as follows:

OXIDATIVE EFFECTS OF SODIUM *n*-PROPYLTHIOSULFATE, THE CAUSATIVE AGENT OF ONION-INDUCED HEMOLYTIC ANEMIA IN DOGS

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Onions (*Allium cepa*) are known to cause Heinz body hemolytic anemia, called "onion poisoning", in many kinds of domestic animals. Recently, three sulfur-containing compounds responsible for onion poisoning were isolated from boiled onions. In this study, sodium *n*-propylthiosulfate, one of those compounds, was identified as the causative agent of onion poisoning, and the oxidative effects of the compound on canine erythrocytes were examined.

The hemolytic effect of sodium *n*-propylthiosulfate in dogs having erythrocytes characterized by hereditary high reduced glutathione (GSH) and potassium concentrations (HK dogs) was compared with that in normal dogs (LK dogs). The oral administration of sodium *n*-propylthiosulfate showed that HK dogs were more susceptible to the anemia induced by the compound than normal dogs. The erythrocyte oxidized glutathione (GSSG) concentration in HK dogs increased to a higher level than that in LK dogs after administration. Formation of Heinz bodies in erythrocytes was also more prominent in HK dogs than in LK dogs. These results were similar to the hematological changes after oral administration of boiled onions, which have been reported previously.

When erythrocytes in HK and LK dogs were incubated with sodium *n*-propylthiosulfate, the concentration of GSH immediately fell to the lowest level. HK erythrocytes were oxidatively damaged more than LK erythrocytes. The oxidative damage to erythrocytes characterized by hereditary high potassium and normal GSH concentrations (HK/LG erythrocytes) is similar to that in LK erythrocytes. The rate of production of superoxide radicals from erythrocytes depended on GSH consumption during incubation. Furthermore, the oxidative damage caused by sodium *n*-

propylthiosulfate remarkably declined in erythrocytes treated with 1-chloro-2, 4-dinitrobenzene to deplete GSH. These results indicated that oxidative damage due to sodium *n*-propylthiosulfate was dependent on GSH consumption.

When sodium *n*-propylthiosulfate and GSH were incubated in a hemolysate, equimolar quantities of those compounds reacted. The addition of oxidized glutathione to this system promoted hemoglobin (Hb) degeneration. Furthermore, the reaction between sodium *n*-propylthiosulfate and GSH was remarkably catalyzed by purified canine Hb A, but not by glutathione peroxidase, glutathione S-transferase or iron.

Based on these examinations, it was proved that sodium *n*-propylthiosulfate is one of the causative agents of onion poisoning. It was suggested that sodium *n*-propylthiosulfate interacts with GSH to induce oxidative damage catalyzed by Hb.