MECHANISMS OF ANEMIA INDUCED BY *BABESIA GIBSONI* INFECTION WITH SPECIAL REFERENCE TO THE FUNCTION OF MACROPHAGES IN INFECTED DOGS

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To clarify the mechanisms of anemia caused by *Babesia gibsoni* infection in dogs, the erythrophagocytic ability of macrophages in infected dogs was investigated *in vitro*. In the present study, macrophages obtained from the peripheral blood and bone marrow of infected dogs with prolonged anemia (for more than 20–24 months) after splenectomy were mainly examined. The macrophages derived from both peripheral blood and bone marrow of these dogs with chronic anemia showed increased activity of erythrophagocytosis compared to those from normal dogs when the macrophages were incubated with phorbol myriptate acetate (PMA). In particular, bone-marrow-derived macrophages in dogs with chronic anemia exhibited a significant increase in erythrophagocytosis, even when incubated without PMA. In these dogs, phagocytic activities of macrophages against both auto- and iso-erythrocytes of normal dog erythrocytes were at the same level. Administration of an antiprotozoal drug, diminazene diaceturate, resulted in a significant decrease of erythrophagocytic activity of both peripheral blood- and bone-marrow-derived macrophages, followed by an increase of hematocrit values and a decrease of reticulocyte counts in the infected dogs with chronic anemia.

In dogs with acute *B. gibsoni* infection, the erythrophagocytic activity of bone-marrow-derived macrophages was also elevated even when incubated without PMA. This phenomenon, however, was not observed in dogs with acute hemolytic anemia induced by onion administration.

These results suggest that the macrophages of dogs infected with chronic anemia might be activated by an unknown mechanism, and that, as a result, the erythrophagocytic ability of the activated macrophages might be increased, resulting in anemia in infected dogs.