SPECIFIC IMMUNITY IN FARM ANIMALS TO HEARTWATER

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ABSTRACT

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The duration of immunity to heartwater normally varies from 6 months to 4 years. In Angora goats it may be difficult to produce a specific immune response with vaccination. Humoral immunity does not appear to play a role in the development of immunity. It has been suggested that cellular immunity may play a role, but the actual mechanism involved remains obscure. The specific immune response developing in farm animals following infection with *Cowdria ruminantium* is reviewed.

INTRODUCTION

There is ample evidence from both field observations and laboratory results that farm animals develop a specific immune response after recovery from infection with Cowdria ruminantium (Alexander, 1931). The severity of the heartwater reaction does not have any influence upon the degree of immunity, providing a reaction is produced (Alexander, 1931). However, this may not apply to Angora goats (Du Plessis, Jansen & Prozesky, 1983).

IMMUNITY IN VARIOUS SPECIES

Sheep: There are widely differing opinions on the duration of immunity in sheep as determined by challenge after recovery from heartwater. The immunity may wane after 6 months (Du Plessis, 1981). Out of 9 sheep inoculated with the Ball 3 strain, 4 developed febrile reactions to homologous challenge 6 months later. Neitz (1939), on the other hand, found all sheep to be solidly immune to challenge within 6 months of recovery, and only 10 out 121 developed a febrile reaction when challenged 7–34 months after recovery. Neitz, Alexander & Adelaar (1947), found a progressive decrease in immunity. Animals challenged 2 months after recovery were solidly immune, and subsequently there was a gradual decrease in immunity that remained sufficient to protect animals against a fatal outcome for at least 4 years.

Goats: The duration of immunity in goats is poorly documented. Ilemobade (1976, cited by Uilenberg, 1983) showed that immunity could last for up to 8 weeks after recovery. Uilenberg (1983) challenged 12 goats 1–2 months after the initial infection and 9 animals were solidly immune.

The development of a specific immune response to heartwater is more difficult to achieve in Angora goats than in other farm animals, and, in fact, immunization of this breed may be difficult and hazardous (Du Plessis et al., 1983). Treatment of Angora goats on the 2nd and 3rd days of the febrile reaction after immunization failed to prevent high mortality, whereas those treated on the 1st day of the reaction survived. Immunity to subsequent challenge, however, was poor. Out of the 4 goats that survived after treatment on the 2nd and 3rd days of the reaction, 3 were shown to be immune to challenge 107–205 days later, showing that Angora goats can nevertheless develop a specific immune response.

Cattle: In cattle, the duration of immunity appears to vary from 6 months—3 years. Neitz & Alexander (1945) reported that out of 1 374 Aberdeen Angus calves immunized at pasture in an endemic area, 8,8 % con-

tracted heartwater in the subsequent 3 years as against 24,6 % of 195 unvaccinated controls.

Du Plessis, Bezuidenhout & Lüdemann (1984) vaccinated 9 calves with a tick stabilate. When challenged 6 months later, 3 developed severe reactions. Henning & Haig (1953) cited by (Henning, 1956) found that many immunized cattle could withstand a challenge dose of heartwater from 12–18 months after the initial infection.

MECHANISM OF IMMUNITY

The mechanism by which the immune response develops is largely unknown. Serum or large quantities of gamma globulin from immune animals, whether given at the time of infection or during the incubation period, does not influence the outcome of infection (Alexander, 1931; Du Plessis, 1970). There is no correlation between antibody titres found in the indirect fluorescent antibody test and the immune status of the animal (Du Plessis *et al.*, 1984).

The importance of the persistence of *C. ruminantium* in the host in the development and maintenance of protective immunity is not known. Neitz (1939) was able to recover the organism from the circulation, endothelial scraping or organ suspension for up to 60 days after infection, but not after that period. This resulted in a coinfectious immunity, followed by a sterile immunity, which declines at a widely varying rate in different sheep. Re-infection results in a repetition of this cycle of co-infectious immunity, followed by a sterile immunity. Ilemobade (1978) showed that blood could be infective up to 50 days following homologous challenge of immune animals.

Alexander (1931) stated, however, that immunity was not demonstrated in any farm animal that had not passed through an attack of the disease. Neitz & Alexander (1941), however, described a number of calves under 22 days of age which showed no reaction after infection but were shown to be immune when challenged between 28 and 300 days later. Thomas & Mansvelt (1957) described 5 goats under 4 weeks of age that did not develop a fever reaction after infection but were immune when challenged 52 days later. Mare (1972) was able to suppress the clinical manifestations of clinical disease by feeding oxytetracycline in the feed which did not interfere with the subsequent development of immunity. Although it is not clear from the results, there were at least 7 and probably more adult sheep that had no temperature reaction after inoculation but were immune to challenge 38 days later. Poole (1962), by the so-called "block method" of immunization, treated 14 goats on the 6th day post-infection. This resulted in a reduction in the number of animals that showed a temperature reaction from 90 % to 40 %. When these goats were subsequently exposed to a field outbreak of heartwater, only I death occurred. No direct attempt was made, however, to determine whether the animals that did not show a temperature reaction were immune. These results, taken together, suggest that it is possible to develop a specific immunity without developing a reaction.

Alexander (1931) suggested that immunity was probably cellular and not humoral, in spite of the fact that the parasitized cell undergoes no reaction in the presence of *C. ruminantium*. Pienaar, Basson & Van der Merwe (1966) state that the perivascular infiltration is mild and offer no explanation for the associated nervous signs. Prozesky & Du Plessis (1985) found that there was no correlation between the severity of the lesions and the concentration of the organisms.

These findings suggest that classical, delayed type hypersensitivity reactions with infiltration of macrophages are not important in the immune response to heartwater.

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