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Neosporosis in dairy cattle: An update from an epidemiological perspective

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Abstract

Our understanding of the epidemiology of bovine neosporosis is advancing rapidly with considerable research activity being facilitated by improving methods. The dynamics of the infection in the known definitive hosts, the dog and the coyote, are being described. Improved procedures for production of oocysts enables the horizontal transmission to intermediate hosts and the subsequent more natural infection process to be studied. Details of the sylvatic cycles, potentially involving other animals in the dairy environment, are also emerging.

Keywords: *Neospora caninum*; Neosporosis; Fetal loss; Abortion; Dairy cattle

1. Introduction

The protozoal parasite *Neospora caninum* was first observed as a clinical disease of dogs in 1984 [1] and of calves in 1987 [2,3] and as a cause of fetal loss in cattle in 1989 [4]. First isolated and proposed as a new genus and species in 1988 [5], *N. caninum* is now recognized as causing neosporosis, a major cause of bovine fetal loss worldwide. As the subject of much active research, our understanding of the parasite, the host–parasite relationship and the epidemiology of the infection are rapidly advancing although, as the agent was only recently identified, remains incomplete in important respects. An ISI Web of Science® search on the terms ''neospora'' or ''neosporosis'' returned almost 1200 indexed papers, 618 including the bovine in some fashion (e.g., terms such as cow, calf, cattle, bovine), with126 published in 2004. These include extensive reviews of general and specific aspects of bovine neosporosis [6–14] as well as many primary research papers. With an emphasis on the more recent publications that expand our understanding of neosporosis but not covered in the above reviews, the following are selected from these.

2. Causative organism and life cycle

N. caninum is an Apicomplexa protozoan that is closely related to and in some phases morphologically similar to Toxoplasma, Sarcocystis and Hammondia. However, it is biologically distinct from these other protozoa. The dog [15,16] and the coyote [17] have been shown to be naturally infected definitive hosts, meaning that they may shed oocysts in their feces after consuming tissue cysts from infected intermediate hosts, and the infectivity of such oocysts for the bovine has been demonstrated [18]. The cow and the white-tailed deer [19] are common intermediate hosts; they may be infected by sporulated *N. caninum* oocysts, which in turn produce tissue cysts in the intermediate

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host's body tissues. The infectivity of tissues for definitive hosts has been demonstrated, particularly the placenta from naturally infected intermediate hosts [20]. The development of improved detection techniques is facilitating clarification of transmission cycles of this two-host parasite. For example, a PCR-based method to detect *N. caninum* oocysts in the feces of definitive hosts has been recently developed [21].

3. Fecal shedding

Initial work, particularly by McCallister and coworkers, described the dynamics of fecal oocyst shedding by definitive hosts, although the picture remains incomplete. In a three-period survey of 15 dogs fed beef carcasses, 1 dog shed 378 oocysts per gram on one occasion and was fecal positive but not quantified on another [22]. An experimental study included the feeding of oocysts to calves, later feeding tissues from the infected calves to dogs and sampling their feces daily for 30 d [23]. Eight of the nine dogs fed tissues from infected calves shed oocysts in their feces over a period of 70 d. During this period, oocyst counts in "positive" feces were <1000, 1000–10,000 and >10,000 for 38, 36 and 26% of the time, respectively. On first exposure, dogs fed infective tissues from calves shed greater numbers than those fed infective tissues from mice and young dogs shed greater numbers than adults. In a subsequent study of shedding response to re-exposure, two of these previously shedding dogs did not shed oocysts when fed tissues from the infected calves 8 months after their initial exposure, whereas two of three dogs shed oocysts when exposed a year and a half later [24]. In this study, only the total numbers of oocysts shed, not the daily shedding levels and duration of shedding, were reported. This was unfortunate as point exposures to single bowel movements seem considerably more likely and more difficult to prevent than point exposures to feces accumulated over multiple days. Of note, both of these studies show an inconsistent relationship between the serological and shedding status of dogs, suggesting that serological testing of specific dogs is not a useful prevention tool. In another study, one of four 12-week-old coyotes shed approximately 500 oocysts over 3 d after being fed tissues from infected calves [17]. These findings supported the recommendation that the risk of contamination of cattle feed and water should be minimized. However, considerably more work is indicated on the dynamics of infection in the definitive host (such as the frequency and level of oocyst shedding), and on transmission from definitive to the intermediate hosts.

4. Transmission following oral exposure

Two experiments exposing pregnant cattle orally to oocysts were recently reported. In one, three cows challenged with 600 oocysts at 10 weeks of gestation gave birth to uninfected calves [25]. When euthanized 4 months after calving, the cows were seronegative, although their brains were PCR-positive. In a subsequent experiment, 17 of 19 (89%) cows in various stages of gestation given oral doses ranging from 1500 to 115,000 oocytes, became seropositive [26]. Transplacental infection occurred in 6 of the 17 fetuses and 1 aborted due to neosporosis. The authors concluded that the odds of transplacental infection increase with larger oocyte doses and advancing gestation. It was noteworthy that the lowest oocyst dose resulted in a congenital infection, and a single abortion was associated with a mid-range dose. These findings suggest that the relationship between oral oocyst exposure, as would occur with a herd point exposure from feed contamination, and reproductive consequences, such as an abortion storm, is complex.

5. Venereal transmission

Limited initial evidence suggests that semen from infected bulls may be a potential vector for venereal transmission of bovine neosporosis. Fifteen of 102 (15%) semen samples from 5 of 8 naturally infected bulls were *N caninum* DNA PCR-positive over a 22-week period, with an estimated parasite load of 1 to 10 parasites/mL [27]. None of the PCR-positive semen samples were bioassay positive, although the authors note that this may have been due to the toxic effects of semen and to the low parasite numbers. In another experiment by the same group, all of nine heifers administered 10^7 tachyzoites by intrauterine inoculation of artificially contaminated semen responded serologically and six had *N caninum* DNA PCR-positive tissues when necropsied 36 d post-insemination [28]. Considerably more work remains to be done to determine whether or not natural transmission via the venereal route represents an important risk.

6. Vertical transmission

Although the relative importance of each potential route of infection has not been well established, vertical transmission remains most likely for the individual dairy cow in typical farm situations. In a prospective study of a 240-cow California dairy herd involving monthly sampling of pregnant cows and pre-colostral