A Deterministic Mathematical Model of *Mycobacterium avium* subsp *paratuberculosis* (MAP) Transmission on Commercial US Dairy Farms

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Introduction

Prevalence of Johne's on US dairy farms is estimated at one-fifth of all herds, and is higher for the population of large herds. Of the animals which test positive for MAP, high levels of bacterial shedding are noted in only a small proportion. Despite this low prevalence of high-shedding animals, elimination of MAP from herds has proven exceptionally challenging, with few published reports of successful eradication of MAP from infected farms. Mathematical modeling may aid us in the understanding of this apparent contradiction.

Materials and Methods

In this study we developed a deterministic mathematical model of MAP transmission on commercial US dairies which builds upon and modifies the assumptions in previous work to best reflect the pathobiology of the disease. Transmission was modeled using ordinary differential equations. Calculation of transmission parameters in these models is necessarily non-linear. Previous models of disease have only utilized linear dynamics and therefore lack sensitivity to changes in susceptible population size. Values gathered from literature concerning animal turnover in US dairy herds were utilized, and rates of transmission from disease states were calculated from retrospective fecal culture data from herds in New York and Pennsylvania.

Results

The model conformed with the expectation that aggressive test-and-cull strategies do not result in successful elimination of MAP in a short time frame. Model output illustrated relative insensitivity of infectious transmission to the presence of high-shedding animals. These high-shedding animals, which may be contributing a bacterial load many orders of magnitude greater than low-shedding animals, were not necessarily responsible for disease maintenance at a herd level. Instead, the model indicated that sustained transmission at a low level could only occur if high-shedding animals were of no more than one order of magnitude greater infectious contribution than low-shedding animals. When calf-to-calf transmission was introduced to the model, a greater disparity in infectiousness between high and low shedders led to a stable low-level prevalence of MAP.

Significance

Multiple levels of contagiousness among infected adult animals and introduction of the contribution of MAP bacteria from infected calves was able to explain the maintenance of low prevalence infections in herds. Herd-level MAP control programs which remove high-shedding animals but allow low-shedding animals to remain in a herd may be less efficacious at MAP elimination than is desirable. Although previous experimental studies supported the potential of infectious transmission among young calves, further research is needed to verify existence of a pool of infectious young animals. If this group of animals is diagnostically identifiable and contributing to maintenance of disease, elimination strategies will need to address calf management practices to include decreasing risk of exposure to other infected calves.