A biological network describing infection with *Mycoplasma hyopneumoniae* in swine herds.

L. Otto[1] and C.S. Kristensen[2*]. [1]Danish Research Institute of Food Economics, Denmark and Research Centre for the Management of Animal Production and Health (CEPROS). [2]The National Committee for Pig Production, DANICH BACON AND MEAT COUNCIL, Denmark.

Summary

We present a biological model for infection with *Mycoplasma hyopneumoniae* (Mycoplasma) in slaughter pig production based on a Bayesian network. The model describes the various risk factors for Mycoplasma. Together with prevalence of infection based on veterinary examinations this leads to a probabilistic/stochastic determination of the severity of infection with Mycoplasma based on the inherent biological uncertainty. We use the model to calculate how the risk factors and our evidence of the herd influence our view on the severity of Mycoplasma, and how our view changes when we gather more evidence.

Introduction

Enzootic pneumonia causes economic losses in the pig industry worldwide, and since the main agent involved is *M. hyopneumoniae* (Mycoplasma) (Ross, 1999) we have modeled this infection in slaughter pig production. The model is based on already published results found in the literature, combined and evaluated by experts (Nielsen et al., 2000).

Objectives

Our aim is to describe a biological model for Mycoplasma in slaughter pig production with parameters and probabilities found in the literature and appraised by experts. The model includes herd-specific information on risk factors, herd productivity and information to determinate the present severity of infection.

Materials and methods

The model describes the *steady state equilibrium* between the various risk factors the herd is exposed to and the severity of Mycoplasma within the herd. Thus the model is not intended for acute outbreak of Mycoplasma. The model is a static Bayesian network (Jensen, 1996, 2001), i.e. a stochastic model where all interdependence is described by conditional probabilities. Input to the model is the state of various risk factors and the results of veterinary examinations as they become available. Some of the variables are easily accessible, e.g. the size of the herd, others require special effort, e.g. serum samples, post mortem examination.

The output of the model is a probability distribution for the severity of Mycoplasma. The biological variation is therefore described by the effects of the risk factors and the evidence of examinations and observations in the form of probabilities.

The variables in the model can attain one of a finite number of states or levels, typically 4 or 5; for instance either «yes» or «no»; or either «zero», «low», «middle», or «high». If the state of a variable in the model is unknown the distribution on the states of that variable is calculated dependent of the states for the other variables in the model. Therefore a Bayesian network has been used.

When we undertake a veterinary examination (a clinical examination, a serological examination, and/or a post mortem examination) we gather information of the herd.
When we make a change in a control strategy, we cannot say what will happen with certainty to a given variable, but we can say how the probabilities for the states of the variable will change. When we set up the parameters in the model we treated the various variables as independent, i.e. without parametric interactions. This is not an ideal situation, but is frequently a default due to lack of published interactions in the empirical literature (Nielsen et al., 2000).

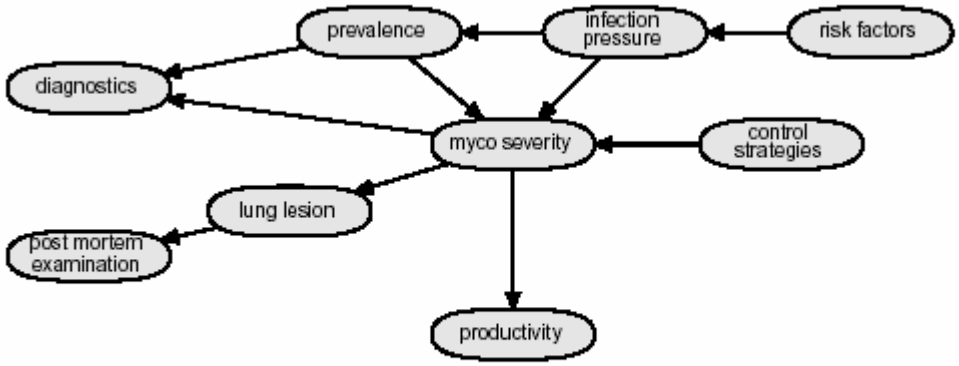

Fig. 1. Causal connections in the biological decision support model of Mycoplasma in slaughter pig production

Results

An overview of the model can be seen in figure 1.The risk factors that we include in the model are only risk factors with published quantification of effects. In this matter, we use the evidence as documented in Nielsen et al. (2000). Changes in a control strategy or in any risk factor will change the biological state of the herd, i.e. the prevalence and the severity of Mycoplasma (figure 1). The biological state on the other hand determines the productivity. Various risk factors that are shown in the literature to influence the severity of Mycoplasma are collected in the variable risk factors. The variable infection pressure collects the effects from the risk factors and influences the number of animals that are infected, prevalence, and how severely they are affected, myco severity. The farmer can indirectly influence the productivity by either changing the risk factors, like take exceptional measures to prevent the pigs from getting infected at all (change production system), or through control strategies like medication. The more accurately we know the myco severity of infection the more accurately can we determine how changes in risk factors and control strategies are expected to change the productivity. Part of this knowledge is only indirectly available through various diagnostics and post mortem examination.
We will try to give an idea on how the model works. We consider a low risk herd characterized by all in–all out production, a small herd size and no buying of piglets from the outside world. With the terminology from Bayesian network we say that we have evidence that the state level for the variable size is at the first level. In a stochastic language we can say that the size of the herd is in the range 1–1000 with probability 100% and in the other ranges, the other states, with probability 0%. We

mark this in the model by writing 100% for herd size in the range 1–1000 and 0% in the other possibilities for herd size. We mark the other characteristics of the herd correspondingly. Thus we have indicated 100% in the model for all in–all out production, and 100% for zero purchase policy corresponding to no buying of piglets from the outside. Without further evidence the model predict the probability of zero true prevalence in the herd to 63% and the probability of zero severity of Mycoplasma, myco severity, to 56%. If we were to make a serological examination, the results from the model says that we should expect the result of zero serological prevalence with a probability of 51% and of high serological prevalence with 9% – the difference between the results for serological prevalence and true prevalence is due to the sensitivity and specificity of the serological method. Then we gather evidence from a serological examination (we go out and take blood samples) and find a zero serological prevalence. We indicate 100% probability for the zero level of serological prevalence in the model. Now the probability of zero severity of Mycoplasma, myco severity has increased to 83% and zero true prevalence has increased to 93% from 63%. Thus the knowledge that a serological investigation shows zero serological prevalence let us conclude that the probability of zero true prevalence has increased from 63% to 93% and that the probability of zero severity of Mycoplasma, myco severity has increased from 56% to 83%.

Discussion

Not all needed information could be found in the literature. One problem is that there is no information of infection pressure and therefore absolutely no information on how infection pressure influences myco severity. In our model infection pressure and severity of Mycoplasma are considered as latent variables. Another problem is that most research shows how the risk factors influence lung lesion and thus only show the reduced or compressed causal connections without effects on prevalence and severity. We have seen that by utilizing published research results we were able to build a Bayesian network that can lead us to a more precise description of a herd. This also lead us to a more precise knowledge of how new information and evidence changes our knowledge of the herd. This is the first step on the road to sound economic advice and economic decision support where biological uncertainty is treated in a biological valid and consistent manner. The model describes how uncertainty propagates through the risk factors to the severity of Mycoplasma and to lung lesions.

References

Jensen, F. V., 1996. An introduction to Bayesian Networks. UCL Press, London.

Jensen, F. V., 2001. Bayesian Networks and Decision Graphs. Springer Verlag, NewYork.

Nielsen, J. P., Bækbo, P., Jensen, C. S., Jorsal, S. E., Sørensen, V., December 2000. Mycoplasma hyopneumoiae infection: A review of pathogenesis, diagnosis, risk factors and biological effects as input for economic modelling. Animal health economics CEP97-5, Research Centre for the Management of Animal Production and Health (CEPROS), Foulum, DK-8830 Tjele, Denmark.