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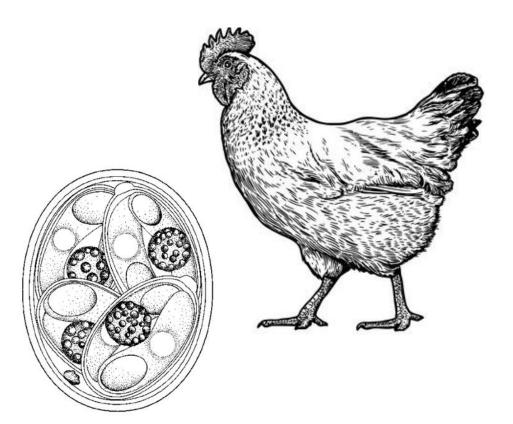
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Epidemiogenetic Modelling for Enhanced Coccidiosis Management in Poultry

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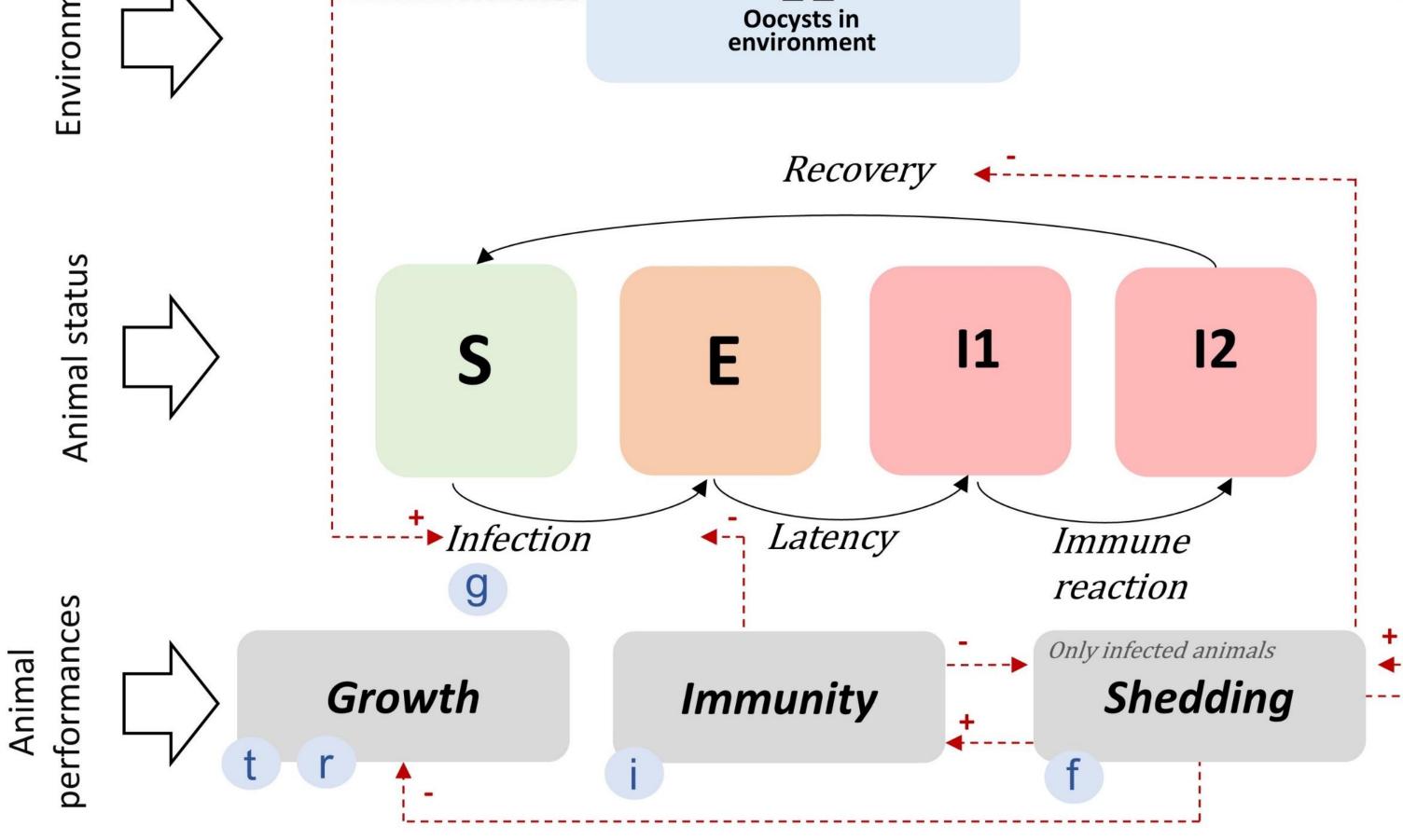
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Coccidiosis is a prevalent disease in poultry, caused by protozoan parasites belonging to the genus Eimeria. The growing resistance of Eimeria species to coccidiostatic drugs, coupled with the high costs of vaccines, stress the need for alternative intervention strategies against coccidiosis. In this context, genetic selection could play an important role. An individual's potential for transmitting the disease can be understood through several traits: (1) susceptibility, indicating the likelihood of infection upon contact with oocysts, (2) the intensity of oocyst shedding, and (3) the duration of shedding, influenced by the animal's immune response. Furthermore, the disease's impact on farm profitability also hinges on (4) an animal's tolerance to disease, defined as its capacity to maintain growth during infection, and (5) compensatory growth after infection. While much remains unknown about the genetic determinism of these five traits in poultry, existing knowledge regarding coccidiosis epidemiology enables us to model the effects of these traits on disease spread and impacts.

1. Individual stochastic epidemiological model

The stochastic part of the model described animal status gathered in 4 compartments: susceptible (S), exposed (E), infected without immunity rection (I1), infected with immunity reaction (I2). The rates of transition between compartments were calculated based on model parameters, oocysts in environment current animal performances and status and interindividual variability of animals.



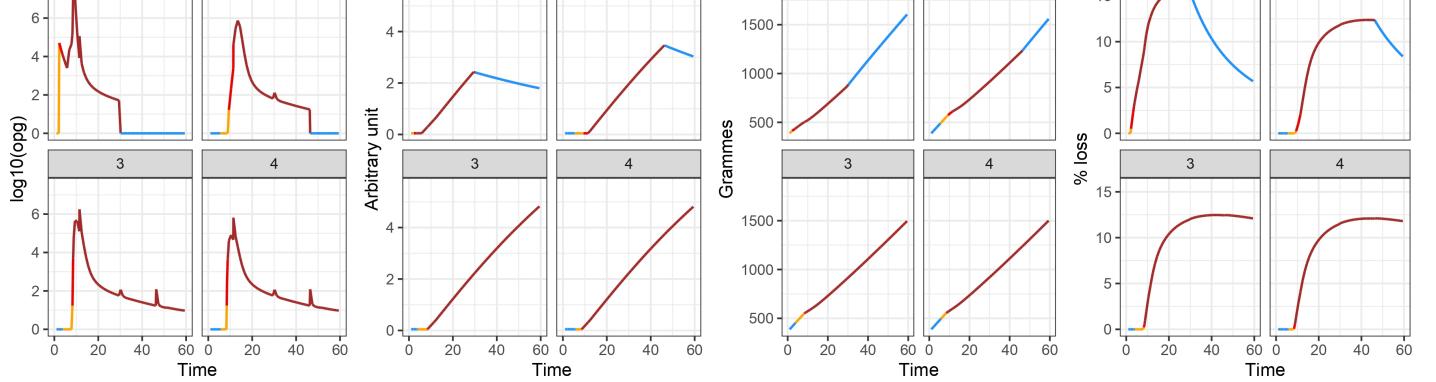
The model also comprised a deterministic part that described individual performances (growth, shedding rate, and immune reaction) and environment. Both stochastic and deterministic process were interconnected and influenced each other as described in the figure.

Normally distributed interindividual variability was included in the model for 5 traits: (g) susceptibility, indicating the likelihood of infection upon contact with oocysts, (f) the intensity of oocyst shedding, (i) the speed of the immune response, (t) animal's tolerance to disease and (r) compensatory growth.

2. Outcome of the model in 4 animals

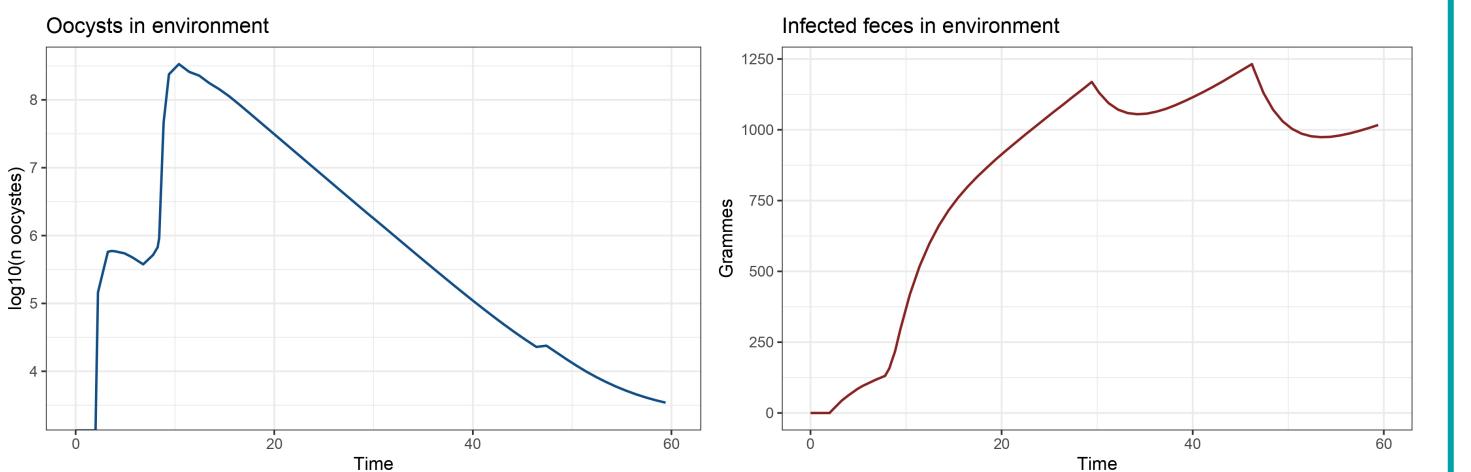


3. Estimate the impact of the variability of the 5 traits on coccidiosis epidemiology



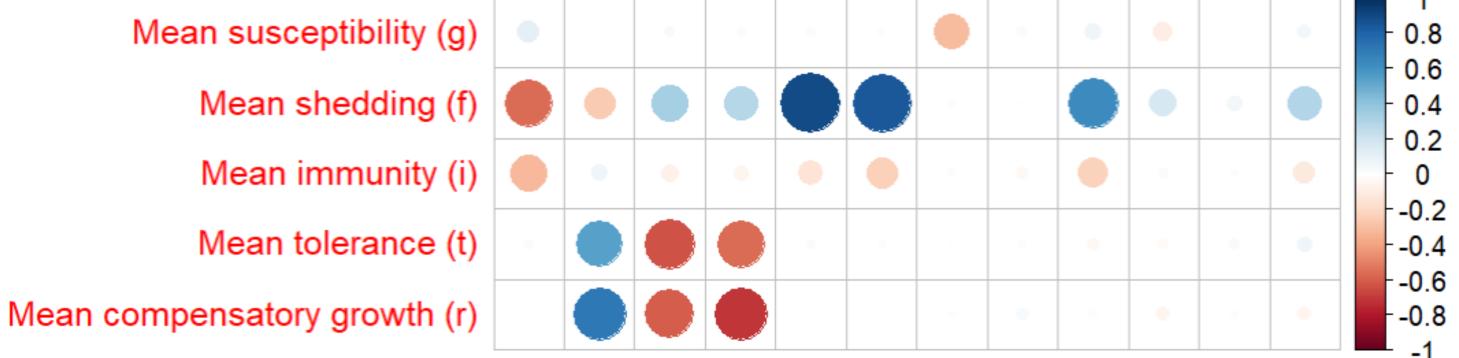
Animal status

- Susceptible (S) - Exposed (E) - Infected without immune reaction (I1) - Infected with immune reaction (I2)



An epidemic is simulated among four chickens in a shared environment. At time = 0 (10 days of age), one chicken ingests 5,000 oocysts. The upper figures display the status, shedding rate, immune response, weight, and weight loss (percentage compared to theoretical weight) from day 0 to day 60 post-infection. The lower figures shows the number of oocysts and infected feces in the environment. No interindividual variation is included in this example.





Several epidemics were simulated among ten chickens. At 10 days of age, one chicken ingested 5,000 oocysts. Interindividual variability was included in the model for five traits: (g) susceptibility, (f) intensity of oocyst shedding, (i) speed of the immune response, (t) tolerance, and (r) compensatory growth. The values were randomly assigned based on a normal distribution, with a standard deviation of 0.2 and a mean of either -0.5, 0, or 0.5 for each trait, resulting in 3^5 parameter combinations. Each combination was simulated three times. The figure shows correlations between the mean value of the five traits and the outcome of the model,

Conclusion

This simulation model, based in a literature review of coccidiosis epidemiology, enhances our understanding of how individual variability influences disease circulation on a farm and its economic impact. The results highlight traits such as tolerance, compensatory growth, and shedding rate as particularly promising, guiding our future research in genetic selection to improve coccidiosis management. In the future, we plan to conduct animal experiments to refine our model parameters and further explore the genetic determinants of these individual traits,

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