

## SARCOCYSTIS MIESCHERIANA: PHENOTYPIC AND GENETIC CHARACTERIZATION OF A DISEASE RESISTANCE MODEL IN SWINE.

Reiner, G.<sup>1</sup>, Hepp, S.<sup>1</sup>, Stankewitz, S.<sup>2</sup>, Hertrampf, B.<sup>1</sup>, Mackenstedt, U.<sup>3</sup>, Zahner, H.<sup>2</sup>

<sup>1</sup>: Dep. of Swine Diseases, University of Giessen, Germany; <sup>2</sup>: Institute for Parasitology, University of Giessen, Germany; <sup>3</sup>: Institute for Parasitology, University of Hohenheim, Germany.

### Introduction

We investigated the usability of *S. miescheriana*, as a model for disease resistance in porcine protozoan infection. With carnivores as definite hosts, *S. miescheriana* causes acute and chronic phases in susceptible pigs, leading to the formation of sarcocysts in skeletal and heart muscle tissues. In naturally infected swine, mild infections do not usually cause clinical signs, but weight gain and meat quality may be reduced over the whole fattening period. When we tested Pietrain and Chinese Meishan pigs for differences in susceptibility/resistance against sarcocystosis, clinical signs, loads of merozoites in muscle tissues and specific immune response after oral challenge discriminated well between both breeds (Reiner et al., 2002). Aiming towards the molecular basis of sarcocystis-susceptibility as a model for host-parasite interaction, we have set up a F2-crossbred model from both founder breeds. The present work describes clinical, clinical-chemical and haematological data, with definite clues for genetically determined differences in resistance/susceptibility against this parasitosis.

### Material and Methods

A number 139 F2-crossbreds set up from Chinese Meishan and European Pietrain breeds as founders, were challenged orally with a dose of 50,000 sporocysts per animal at an age of 12 weeks. The crossbreds were clinically examined on days 7 to 1 ante infectionem (a.i.), and on days 0, 7, 12 to 14, 21, 28, 35, 42, 45, 49, 56, 63 and 70 post infectionem (p.i.). Blood samples were collected on days 0, 14, 28 and 42 p.i., to be screened for a broad range of relevant haematological and clinical-chemical parameters. Each animal served as its own control and was further compared to a set of F2-control animals and to purebred Meishan and Pietrain pigs, each challenged and controlled.

Variance of traits was analysed with the Statistical package for Social Sciences (SPSS). Heritabilities for clinical, haematological and clinical-chemical values were evaluated with the CVE-version 4.2.5 (Groeneveld).

### Results

Clinical effects of *S. miescheriana* infection can be best clarified by the deviations of body temperatures after challenge from baseline a.i.. During second schizogony, a significant fever peak formed, with no differences between founder breeds and a smaller deviation in F2 pigs. A second fever peak became visible during 6<sup>th</sup> week p.i. in Pietrain and a part of the F2, but not in Meishan pigs. Numbers of merozoites, which had developed during the chronic state of infection until day 70, varied significantly between founder breeds and crossbreds, with Pietrain pigs showing 20 fold higher loads per g of muscle tissue than Meishan pigs (fig. 1). Merozoite loads of the F2 crossbreds were found to be at the average level

between their founder breeds. Variance within F2 pigs was mainly explainable by additive genetic effects, leading to heritabilities in the range of 0.75.

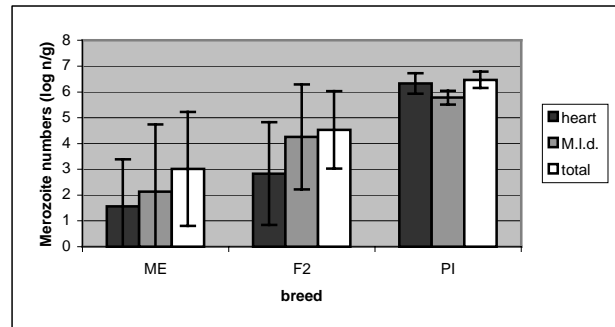
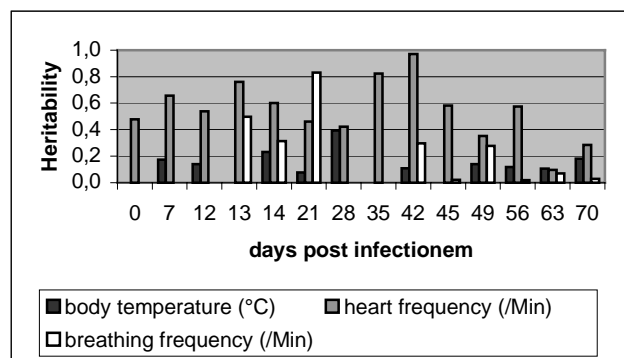


Figure 1: Numbers of merozoites per g of heart muscle or musculus longissimus dorsi (M.l.d.) and total numbers of both of Meishan (ME), F2-crossbreds (F2) and Pietrain (PI) pigs ( $x \pm s$ ).

Some clinical and clinical-chemical parameters showed moderate to high heritabilities too, mainly during acute (days 12-14) and chronic sarcocystosis (days 42-45).

Figure 2: Heritabilities for clinical traits, depending on



days post infectionem, as estimated from F2-crossbreds.

### Discussion

Clinical, clinical-chemical and haematological data produced a complex picture of sarcocystosis in swine, with definite clues for genetically determined differences in resistance/susceptibility against this parasite.

### Conclusion

The data highlight the suitability of this model to further analyse chromosomal regions, candidate genes and thus the molecular basis of host-parasite interaction in sarcocystosis.

### Acknowledgements

This research is funded by the German National Science Foundation (DFG).

### References

Reiner, G. et al. 2002. Vet. Parasitol. 106, 99-113.