Aspects of Animal Health Economics in the finisher pig production

- with emphasis on leg disorders

Ph.D. Thesis

Tina Birk Jensen

University of Copenhagen
Faculty of Life Sciences
Department of Large Animal Sciences
2008
This PhD thesis has been carried out as part of my PhD education at University of Copenhagen, Department of Large Animal Sciences. The thesis was initiated in 2005 and has been funded by University of Copenhagen, Danish Institute of Agricultural Sciences and Danish Meat Association. I am indeed grateful for having been given the opportunity to work on the thesis that, in many ways, has expanded my horizons, and put a new perspective into my previous education in Veterinary Medicine.

I have had the privilege to be supervised by a number of excellent people to whom I am very thankful. First of all I would like to thank my main supervisor Professor Hans Houe for qualified guidance and help with keeping the focus for the thesis. I would like to give a special thank to Senior Scientist Nils Toft for his many great ideas, encouragement and enthusiasm throughout the process. I am also very grateful to Senior Scientist Niels Peter Baadsgaard for inspiration and for providing insight into practical applications of the thesis in the pig production, and to Senior Scientist Søren Østergaard for valuable discussions during my PhD study. I wish to express my sincere gratitude to Professor Anders Ringgaard Kristensen for his interest, and great help and support in this thesis.

During the past 3 years, I have been part of the Research School for Animal Production and Health (RAPH), where I have had the pleasure to attend a number of interesting courses and meet other PhD students working in related areas. Thanks to all fellow PhD students and coordinators of RAPH. I have also had the possibility to attend meetings with the CEPROS project: “Decision support system for Animal Health Economics within the pig production”, which has provided a valuable aid to my understanding of Animal Health Economics.

In the spring of 2007, I had the opportunity to visit the Animal Health Economics group at University of Utrecht. I am very grateful to Associate Professor Henk Hogeveen for his time and fruitful discussions during my stay.

I would like to thank the Danish Pig Production for providing data for my thesis, and in particular thanks to Gert Ohl and Jørgen Håberg for practical help. A number of people have helped with experts opinions to the thesis. I gratefully acknowledge: Øyestein Angen (National Veterinary Institute), Marie Erika Busch
(Danish Meat Association), Svend Haugegaard (National Veterinary Institute), Tim Kåre Jensen (National Veterinary Institute), Sven Erik Jorsal (National Veterinary Institute), Bente Jørgensen, Elisabeth Okholm Nielsen (Danish Meat Association), Jens Peter Nielsen (University of Copenhagen) and Helle Stege (University of Copenhagen) for their willingness to participate.

I am indeed thankful to Helle Halkjær Kristensen for great support and for taking interest in my thesis. I would also like to acknowledge Cecile Cornou for help with Latex and Lene Elisabeth Buelund for help with the front page. Thanks to all colleagues at Population Biology for an excellent working environment, and finally, a great thank to new and old friends at LIFE who indeed made the past 3 years memorable.
CONTENTS

0.1 Summary ................................................. 9
0.2 Sammendrag .......................................... 11

1 General Introduction ............................... 13
  1.1 The aim of the thesis .............................. 14
  1.2 Outline of the thesis .............................. 15

2 Animal Health Economics ......................... 17
  2.1 Definition and concepts ............................ 17
     2.1.1 The effect of disease .......................... 18
     2.1.2 Disease control ............................... 19
     2.1.3 Utility value of the livestock producer ...... 20
  2.2 The framework for economic analysis in AHE .... 20
     2.2.1 Modeling approaches .......................... 21

3 Object Oriented Bayesian Networks ............... 25
  3.1 Bayesian networks .................................... 25
     3.1.1 Example of a Bayesian network .......... 25
     3.1.2 Previous work using Bayesian networks ...... 26
  3.2 Object orientation ................................... 27
  3.3 Bayesian networks and object-orientation ....... 28

4 Lameness in finishers - a review .................. 30
  4.1 Infectious arthritis ................................. 30
     4.1.1 Mycoplasma hyosynoviae ......................... 30
     4.1.2 Erysipelothrix rhusiopathiae ................. 31
     4.1.3 Haemophilus parasuis .......................... 32
     4.1.4 Streptococcus suis ................................ 33
     4.1.5 Risk factors for infectious arthritis .......... 33
CONTENTS

4.2 Osteochondrosis ........................................... 34
  4.2.1 Description of osteochondrosis in finishers ............. 34
  4.2.2 Risk factors for osteochondrosis ....................... 34

4.3 Injuries to the limb and claw ............................. 35
  4.3.1 Description of limb and claw injuries ................. 35
  4.3.2 Risk factors for injuries to the leg and claw .......... 36

5 Materials and Methods ................................... 43
  5.1 Data sources and data collection ......................... 43
    5.1.1 The boar test station - background .................. 43
    5.1.2 Description of the production system ............... 44
    5.1.3 Production records .................................. 44
    5.1.4 Disease records .................................... 45
    5.1.5 Local abattoir ...................................... 45
    5.1.6 Veterinary laboratory ............................... 45
  5.2 Data management ......................................... 46
    5.2.1 Objective 1 ........................................ 46
    5.2.2 Data control ........................................ 47
    5.2.3 Objective 2 ........................................ 47
    5.2.4 Data control ........................................ 48
  5.3 Statistical analysis for Objectives 1 and 2 ............... 48
  5.4 Objective 3 ............................................ 50
    5.4.1 Qualitative structure of the model .................. 51
    5.4.2 Materials for the quantitative part of the model .... 53
    5.4.3 Methodology for the construction of the nodes ....... 54

6 Results ...................................................... 57
  6.1 Objective 1 (Manuscript 1) ............................... 57
  6.2 Objective 2 (Manuscript 2) ............................... 59
  6.3 Objective 3 (Manuscript 3) ............................... 59
    6.3.1 Modeling scenarios .................................. 60
    6.3.2 Results ............................................. 60

7 General discussion and conclusion ........................ 62
  7.1 Overall framework ....................................... 62
  7.2 Objectives 1 and 2 ...................................... 63
    7.2.1 Results from Objectives 1 and 2 ..................... 63
    7.2.2 Performance variables for the Objectives 1 and 2 ... 64
    7.2.3 Data used for Objectives 1 and 2 ................. 65
  7.3 Objective 3 ............................................. 66
    7.3.1 OOBN model ........................................ 66
    7.3.2 Results from the model .............................. 67
    7.3.3 Use of expert opinions and model validation ....... 68
  7.4 Conclusions of the thesis ............................... 69
## CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>Perspectives</td>
<td>71</td>
</tr>
<tr>
<td>9</td>
<td>Manuscript 1: The effect of lameness on productivity</td>
<td>73</td>
</tr>
<tr>
<td>9.1</td>
<td>Introduction</td>
<td>74</td>
</tr>
<tr>
<td>9.2</td>
<td>Materials and methods</td>
<td>75</td>
</tr>
<tr>
<td>9.2.1</td>
<td>Study design</td>
<td>75</td>
</tr>
<tr>
<td>9.2.2</td>
<td>Description of the production system</td>
<td>75</td>
</tr>
<tr>
<td>9.2.3</td>
<td>The data</td>
<td>76</td>
</tr>
<tr>
<td>9.2.4</td>
<td>Data control</td>
<td>77</td>
</tr>
<tr>
<td>9.2.5</td>
<td>Statistical analysis</td>
<td>77</td>
</tr>
<tr>
<td>9.3</td>
<td>Results</td>
<td>80</td>
</tr>
<tr>
<td>9.3.1</td>
<td>Descriptive results</td>
<td>80</td>
</tr>
<tr>
<td>9.3.2</td>
<td>Mean daily weight gain</td>
<td>80</td>
</tr>
<tr>
<td>9.3.3</td>
<td>Feed conversion ratio</td>
<td>81</td>
</tr>
<tr>
<td>9.4</td>
<td>Discussion</td>
<td>81</td>
</tr>
<tr>
<td>9.4.1</td>
<td>Lameness treatments</td>
<td>81</td>
</tr>
<tr>
<td>9.4.2</td>
<td>Records of non-lameness treatments</td>
<td>83</td>
</tr>
<tr>
<td>9.4.3</td>
<td>Consequences of lameness</td>
<td>84</td>
</tr>
<tr>
<td>9.4.4</td>
<td>Weight at 4 weeks</td>
<td>84</td>
</tr>
<tr>
<td>9.4.5</td>
<td>Breed</td>
<td>84</td>
</tr>
<tr>
<td>9.4.6</td>
<td>Model validation</td>
<td>85</td>
</tr>
<tr>
<td>9.4.7</td>
<td>The data</td>
<td>85</td>
</tr>
<tr>
<td>9.5</td>
<td>Conclusion</td>
<td>86</td>
</tr>
<tr>
<td>10</td>
<td>Manuscript 2: The association between disease and profitability</td>
<td>90</td>
</tr>
<tr>
<td>10.1</td>
<td>Introduction</td>
<td>91</td>
</tr>
<tr>
<td>10.2</td>
<td>Materials and Methods</td>
<td>92</td>
</tr>
<tr>
<td>10.2.1</td>
<td>The data</td>
<td>92</td>
</tr>
<tr>
<td>10.2.2</td>
<td>Data management</td>
<td>92</td>
</tr>
<tr>
<td>10.2.3</td>
<td>Data control</td>
<td>93</td>
</tr>
<tr>
<td>10.2.4</td>
<td>Statistical analysis</td>
<td>94</td>
</tr>
<tr>
<td>10.3</td>
<td>Results</td>
<td>96</td>
</tr>
<tr>
<td>10.3.1</td>
<td>Descriptive analysis</td>
<td>96</td>
</tr>
<tr>
<td>10.3.2</td>
<td>Statistical analysis</td>
<td>96</td>
</tr>
<tr>
<td>10.3.3</td>
<td>Sensitivity analysis</td>
<td>97</td>
</tr>
<tr>
<td>10.3.4</td>
<td>Excluded boars</td>
<td>97</td>
</tr>
<tr>
<td>10.4</td>
<td>Discussion</td>
<td>100</td>
</tr>
<tr>
<td>10.4.1</td>
<td>Treatment during the finishing period</td>
<td>100</td>
</tr>
<tr>
<td>10.4.2</td>
<td>Pathological findings</td>
<td>101</td>
</tr>
<tr>
<td>10.4.3</td>
<td>Breed</td>
<td>101</td>
</tr>
<tr>
<td>10.4.4</td>
<td>Weight at 4 weeks</td>
<td>102</td>
</tr>
<tr>
<td>10.4.5</td>
<td>Model evaluation</td>
<td>102</td>
</tr>
<tr>
<td>10.4.6</td>
<td>The effect of disease at herd level</td>
<td>103</td>
</tr>
<tr>
<td>10.4.7</td>
<td>Conclusion</td>
<td>104</td>
</tr>
</tbody>
</table>
II Manuscript 3: An object-oriented Bayesian network 106

11.1 Introduction . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 107
11.2 Materials and Methods . . . . . . . . . . . . . . . . . . . . . . . 108
11.2.1 The qualitative structure of the model . . . . . . . . . . . 109
11.2.2 Elicitation of probabilities . . . . . . . . . . . . . . . . . 113
11.2.3 Modeling methods . . . . . . . . . . . . . . . . . . . . . . 117
11.2.4 Modeling scenarios . . . . . . . . . . . . . . . . . . . . . . 118
11.3 Results from the model . . . . . . . . . . . . . . . . . . . . . . . 120
11.4 Discussion . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 123
11.4.1 Economic benefit of diagnostic examination . . . . . . . . 123
11.4.2 The qualitative structure of the model . . . . . . . . . . . 124
11.4.3 Probabilities used in the model . . . . . . . . . . . . . . . . 125
11.4.4 Future research and conclusion . . . . . . . . . . . . . . . . 126

A Appendix A: Disease codes 131

B Appendix B: Elicitation of probabilities 133

B.1 Introduction . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 133
B.2 Risk Index . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 135
B.2.1 Common structure of a risk index complex . . . . . . . . . 135
B.2.2 A model for the probabilities of the risk index . . . . . . . 135
B.2.3 A model for the probabilities of the diseases . . . . . . . . 137
B.2.4 Parameter needs . . . . . . . . . . . . . . . . . . . . . . . 137
B.2.5 Elicitation of probabilities . . . . . . . . . . . . . . . . . . 138

\begin{itemize}
\item Obtaining the indirect quantitative information \hfill 138
\item Combining the quantitative information into a data set \hfill 139
\item Fitting a linear model \hfill 140
\item Optimizing the fit \hfill 141
\item Final adjustment of parameters \hfill 141
\end{itemize}

B.3 Inherited complex . . . . . . . . . . . . . . . . . . . . . . . . . . 143
B.3.1 Probabilities for the Inherited complex . . . . . . . . . . . 143
B.3.2 Methodology for constructing the nodes . . . . . . . . . . . 146

\begin{itemize}
\item Prior estimates \hfill 143
\item Conditional probabilities elicited from experts \hfill 145
\item Conditional probabilities elicited from the literature \hfill 145
\item Gain node \hfill 146
\item Inherited node \hfill 147
\item OCD and OCM \hfill 152
\end{itemize}

B.4 Physical complex . . . . . . . . . . . . . . . . . . . . . . . . . . 154
B.4.1 Probabilities for the Physical complex . . . . . . . . . . . 154
B.4.2 Methodology for constructing the nodes . . . . . . . . . . . 155

\begin{itemize}
\item Marginal distributions \hfill 154
\item Conditional probabilities elicited from experts \hfill 155
\item Physical node \hfill 155
\end{itemize}
<table>
<thead>
<tr>
<th>CONTENTS</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>ClawWall, ClawSole and Fracture</td>
<td>161</td>
</tr>
<tr>
<td>B.5 Infectious complex</td>
<td>162</td>
</tr>
<tr>
<td>B.5.1 Probabilities for the Infectious complex</td>
<td>163</td>
</tr>
<tr>
<td>Prior estimates</td>
<td>163</td>
</tr>
<tr>
<td>Conditional probabilities elicited from experts</td>
<td>163</td>
</tr>
<tr>
<td>B.5.2 Methodology for constructing the nodes</td>
<td>166</td>
</tr>
<tr>
<td>Infectious node</td>
<td>166</td>
</tr>
<tr>
<td>Myco, Erysip, Haemo and Strep</td>
<td>174</td>
</tr>
<tr>
<td>B.6 The PigLame node</td>
<td>175</td>
</tr>
<tr>
<td>B.6.1 Final adjustment of the PigLame node</td>
<td>175</td>
</tr>
<tr>
<td>B.7 Sensitivities and Specificities</td>
<td>175</td>
</tr>
<tr>
<td>C Appendix C: R-codes</td>
<td>179</td>
</tr>
</tbody>
</table>
0.1 Summary

This thesis addresses Animal Health Economics in the finisher pig production. It investigates the effect of endemic diseases on performance in individual finisher pigs and develops a model that can estimate the most likely cause of leg disorders at herd level.

Animal Health Economics is a research area that deals with economic consequences of animal disease. The research area can be divided into 3 overall tasks:

1. To quantify the financial effects of animal disease.
2. To develop methods for optimizing decisions when individual animals, herds or populations are affected.
3. To determine the costs and benefits of disease control measures.

The thesis focuses on the economical implications in regard to the disease complex: leg disorders in finishers. Leg disorders may reduce the productivity among pigs and increase the costs of medicine expenditures. Moreover, leg disorders can have a negative effect on animal welfare. The thesis covers parts of the 3 tasks in Animal Health Economics. The objectives of the thesis are:

1. To investigate the effect of leg disorders on the productivity in individual pigs.
2. To investigate the association between different disease indicators and the profitability in individual pigs.
3. To develop a mathematical model that can estimate the most likely cause of leg disorders in finisher herds.

The thesis is composed of 11 chapters and 3 appendices. Chapters 1-8 provide general background information, an overview of the used methodologies and obtained results, followed by discussion and perspectives. Chapters 9-11 present the 3 manuscripts prepared for the thesis. Appendix A gives an overview of disease codes, Appendix B describes how the probabilities have been elicited for the model in Objective 3 and Appendix C presents the R codes for Objective 3.

A general introduction to the thesis is presented in Chapter 1. This chapter includes a presentation of the objectives and an outline of the thesis. Chapter 2 describes the research area: Animal Health Economics and presents the general framework for economic analysis in Animal Health Economics. In order to provide background information for the object-oriented Bayesian network used as the
modeling method for Objective 3, Chapter 3 describes the theory behind Bayesian networks in general and of object-oriented Bayesian networks in particular. Chapter 4 reviews the existing literature on lameness in finishers with emphasis on 3 major causes: Infectious arthritis, physical injuries and osteochondrosis.

A thorough description of the materials and methods used in the thesis is provided in Chapter 5. This includes both a presentation and description of the data set used for Objectives 1 and 2, and a description of the materials and the methodology for constructing the object-oriented Bayesian network model in Objective 3. An overview of the results from the 3 objectives is given in Chapter 6. In Manuscript 1: “The effect of lameness treatments and treatments for other health disorders on the weight gain and feed conversion in boars at a Danish test station” it has been shown that boars with records of lameness have a significant reduction in the mean daily weight gain. However, no significant effect on the feed conversion ratio is shown. Contrary, it is found that boars with records of non-lameness treatments have a significant negative effect on both production variables. Manuscript 2: “The association between disease and profitability in individual finishing boars at a test station” has shown that boars treated parenterally and orally have a significant negative effect on the profit margin. Also boars with pathological findings at slaughter are negatively associated with the profit margin; however, this effect is influenced by the breed of the boar. In Manuscript 3: “An object oriented Bayesian network modeling the causes of leg disorders in finisher herds”, an object-oriented Bayesian network model has been constructed that can estimate risk indexes for 3 major cause-categories of leg disorders using information from 2 different levels: the herd and individual pigs. In order to illustrate the behaviour of the model, the benefit of performing diagnostic examinations of individual pigs are investigated in 2 fictitious herds with different herd characteristics related to the risk of leg disorders (e.g. purchase policy, production type and stocking density in pens). The results show that the benefit of performing diagnostic tests will depend on the individual herd. For example, given a certain prevalence of infectious leg disorders, the extended diagnostic examinations of individual pigs are much more beneficial in a herd with prior evidence indicating low-risk of infectious leg disorders than in a herd with evidence indicating high-risk of infectious leg disorders.

A discussion of the results from the thesis and a conclusion are found in Chapter 7. This chapter includes a discussion of the overall framework of the work of the thesis, the data used for the analyses and the construction and use of the object-oriented Bayesian network model. Finally, Chapter 8 provides a perspective of the work for this thesis.
0.2 Sammendrag

Denne afhandling omhandler husdyrsundhedsøkonomi i relation til slagtesvineproduktionen. Afhandlingen undersøger effekten af endemiske sygdomme i forhold til produktiviteten af individuelle slagtesvin, og udvikler en model der kan estimere risikoen for 3 forskellige årsager til benproblemer i slagtesvinebesætninger.

Husdyrsundhedsøkonomi er et forskningsområde, der fokuserer på de økonomiske konsekvenser af sygdomme i husdyrproduktionen. Forskningsområdet kan opdeles i 3 forskellige elementer:

1. Kvantificering af de finansielle omkostninger af sygdomme i husdyrproduktionen.
2. Udvikling af metoder, der kan optimere beslutninger, når individuelle husdyr, besætninger eller populationer er berørt af sygdom.

Denne afhandling fokuserer på de økonomiske konsekvenser af sygdomsomrekset: Benproblemer hos slagtesvin. Benproblemer kan reducere produktiviteten hos slagtesvin samt øge behandlingsomkostningerne. Endvidere kan benproblemer have en negativ indflydelse på dyrevelfærd. Afhandlingen dækker dele af de 3 elementer i husdyrsundhedsøkonomi. De specifikke formål med afhandlingen er:

1. At undersøge effekten af benproblemer i forhold til produktiviteten hos individuelle slagtesvin.
2. At undersøge sammenhængen mellem forskellige sygdomsindikatorer og profitabiliteten hos individuelle slagtesvin.
3. At udvikle en matematisk model der kan estimere den mest sandsynlige årsag til benproblemer i slagtesvinebesætninger.


En general introduktion til afhandlingen, bestående af en præsentation af delformålene samt afhandlingens struktur, fremstilles i Kapitel 1. Kapitel 2 giver en


CHAPTER 1

GENERAL INTRODUCTION

The economic importance of disease in the finisher pig production has been recognized at various levels in society. Previously, attention has been drawn towards the economic consequences of epidemic diseases (Meuwissen et al., 1999; Nielen et al., 1999). Epidemic diseases may cause economic losses to the farmer due to sudden deaths and decreased productivity among the livestock. Epidemics, such as classical swine fever, can have huge consequences for the sector and the national economy primarily due to foreign trade restrictions (Dijkhuizen et al., 1991).

Likewise, focus has been placed on the occurrence of zoonotic diseases in the pig production e.g. salmonellosis. Zoonotic diseases may not affect animals clinically, but these diseases pose a risk in regard to the consumer health, which will lower the demand for animal products (Nørgaard, 2000). So far, only limited efforts have been made to study the economic implications of endemic diseases in finisher herds. Certain endemic diseases are constantly present in the finisher pig production with a low prevalence, which can lead to a reduction in the product quality and inefficient use of resources (Dijkhuizen et al., 1991).

This PhD thesis primarily focuses on the economic implications in regard to the endemic disease complex: leg disorders in finisher herds. Leg disorders are in the thesis defined as any lesion or dysfunction of the leg or claw that might give rise to clinical signs of lameness. The motivation for selecting leg disorders is the fact that the disease complex may reduce the productivity among affected animals and increase the workload due to the physical handling of lame pigs, which has also been recognized by others (van Dijk et al., 1984; Woltmann et al., 1995). Moreover, it has been shown that lameness is the third most frequent cause of treatment among finishers (Christensen et al., 1994). Hence, the cost of medicine and veterinary service cannot be neglected. Leg disorders can be painful for pigs, and hence, the disease complex has a negative effect on animal welfare (Busch et al., 2003). In today’s society, rising incomes and changing social preferences
has increased the public’s attention to the quality of animal products as well as animal welfare (McInerney, 1998). Therefore, leg disorders among finishers may cause a reduction in consumer’s demand for pork, which indicates the importance of considering animal welfare in economic analyses (McInerney, 1991).

The thesis falls within the area of Animal Health Economics (AHE). AHE is a research area focusing on the economic consequences of animal disease. The purpose of AHE is to provide “a framework of concepts, procedures and data to support the decision making process in optimizing animal health management” (Dijkhuizen and Morris, 1997). As described by Dijkhuizen and Morris (1997), research in AHE can be divided into 3 overall tasks:

1. Quantifying the economic effects of animal disease.
2. Developing methods for optimizing decisions when individual animals, herds or populations are affected.
3. Determining the profitability of specific disease control and health management programs and procedures.

In AHE, consequences of animal disease can be studied and evaluated at different levels: the animal level, the farm level, the sector level and the national level. Depending on the level studied, concepts and definitions from micro or macro economics are applied (Howe, 1985). As this thesis will focus on the implications of disease in the finisher pig production, concepts and theories from micro economics will be presented.

The present thesis will cover parts of the 3 tasks in AHE with focus on quantifying the effect of endemic disease, i.e. leg disorders, in the finisher pig production. Secondly, the thesis will develop a model that can estimate the risk of different causes of leg disorders in a finisher herd. This will increase the certainty about the cause of leg disorders, which is an elementary step towards developing a decision support model for leg disorders in finisher herds.

1.1 The aim of the thesis

The aim of the work of this thesis is to investigate the influence of endemic disease, and in particular leg disorders, on the performance of finisher pigs. This is done by the following 2 objectives:

Objective 1 To investigate the effect of leg disorders on the productivity in individual pigs.

Objective 2 To investigate the association between different disease indicators and the profitability in individual pigs.

Secondly, the aim is to develop a method that can estimate the most likely cause of leg disorders in a finisher herd. Hence, the third objective of the thesis is:
1.2 Outline of the thesis

Objective 3 To develop a mathematical model that can estimate the most likely cause of leg disorders in finisher herds.

The empirical analyses of this thesis (Objectives 1 and 2) are based on existing data sets. As the main focus of this thesis has been on AHE and modeling, it has been prioritized not to collect specific data for the purpose of Objectives 1 and 2. However, it has been chosen to obtain expert opinions for the purpose of the modeling part (Objective 3).

1.2 Outline of the thesis

The thesis contains 11 chapters and 3 appendices. Chapter 2 provides an overview of the principles and concepts of AHE. Moreover, the framework for economic analysis is described in Chapter 2. Chapter 3 presents the theory behind Bayesian networks in general and of object-oriented Bayesian networks in particular. A review of the existing literature on lameness in finishers is given in Chapter 4. Chapter 5 presents the materials and methods for the 3 objectives, and Chapter 6 gives an overview of the main results of the thesis. Chapter 7 discusses and links the results from the 3 objectives in order to provide a conclusion, and finally, a perspective of the work of the thesis is found in Chapter 8. Chapters 9-11 contains the Manuscripts 1-3, which correspond to Objectives 1-3. Appendix A gives an overview of the disease codes used in Objectives 1 and 2. Appendix B provides a thorough description of the elicitation of probabilities for the model in Objective 3, and the R codes are finally presented in Appendix C.

References


2.1 Definition and concepts

Animal Health Economics (AHE) has been the subject of increased interest over the past years. The research area combines principles and concepts from 2 different sciences: social sciences and natural sciences. Strictly speaking, AHE is economics applied to animal health issues (Howe and Christiansen 2004). Economics is a social science focusing on the wellbeing of humans. It deals with how society manages its scarce resources and how to allocate resources in an optimal way that fulfills human wants (Mankiw 2004). Contrary, research in animal health has the origin in natural sciences, e.g. Veterinary Medicine and Animal Sciences, where animal health and wellbeing is in focus. Combining the 2 sciences, AHE is a research area which focuses on those aspects of animal health that affect human benefit.

In the livestock production, animals are kept for the production of e.g. meat, milk and wool consumed by humans. Certain resources, e.g. land, livestock, feed and labour are necessary for the production of livestock products. The transformation of resources to animal products, as well as the consumption of these products by humans, can be illustrated using the basic economic model (McInerney 1987) (Fig. 2.1).

From an economic perspective, the livestock products and the resources used are associated with a particular value. The value is determined by the preferences of people and the availability of the particular good (McInerney 1987). Often, the value of a good is measured in monetary units. However, other goods involved in the economic framework can have a value that is more difficult to measure in monetary units (e.g. labour time and animal welfare). Though these goods do not have a market price, they still represent an economic value with an associated opportunity cost that is of importance in the evaluation of market transactions (McInerney)
It has been argued that Economics is an integrated part of Veterinary Epidemiology (Howe, 1989). Both sciences focus on populations of humans or livestock animals, and share a common interest in the physical transformation process of animals (Howe, 1988). However, as Veterinary Epidemiology and Economics belong to 2 different sciences, the concept of disease differs. From a veterinary point of view, disease is a “finite abnormality of structure or function with an identifiable pathological or clinicopathological basis, and with a recognizable syndrome of clinical signs” (Blood and Studdert, 1988). In AHE, disease is only relevant if it disturbs the transformation of resources and hence affects human benefit in some way. Hence, a disease that does not disturb the transformation of resources will not be considered a problem in AHE (McInerney, 1988a, 1996).

### 2.1.1 The effect of disease

When disease (as defined by an animal health economist) is introduced to a herd it can have direct as well as indirect consequences (McInerney, 1987, 1988a). In the finisher pig production, the direct effects can be measured as increased mortality, reduced growth rate and/or deterioration in the feed efficiency. Fewer products can be produced using the same amount of resources and, hence, extra resources must be used in the production. Fig. 2.2 illustrates an example of the direct effects of disease for a finisher pig enterprise.

The cost of feed represents the major part of the total cost of production (Losinger, 1998). Hence, finishers with a poor feed conversion ratio (FCR) due to disease can have a negative impact on the farm economy. Additionally, reduced mean daily weight gain (MDWG) caused by disease may increase the time period for finishers to reach the target slaughter weight. As illustrated in Fig. 2.2, this can increase the number of days in the finisher unit, and hence, the cost of feed. Moreover, a reduction in the MDWG among finishers can potentially influence the number of fattening rounds per year, which can affect the herd profitability. However, it should be emphasized that the effect of a reduced MDWG among finishers will depend on the production system (sectioned and continuous production) in the herd. Yet, other important consequences of disease can be increased mortality among finishers and expenditures to control strategies (health costs) (Fig. 2.2). Mortality
Figure 2.2: Examples of the direct effects of disease on the profit margin for a finisher pig enterprise. Factors that can be influenced by disease is presented in boxes. Modified after Rougoor et al. (1996).

cause a direct negative effect on the profit margin (PM) due to foregone revenues as well as costs in regard to the use of feed and purchasing costs.

Solely measuring the direct effects of disease will simply be a financial assessment of the impact of animal disease (McInerney, 1988a,b). However, the indirect effects of disease can involve the livestock producer as well as other groups in society. These effects can be related to consequences in regard to animal welfare, environmental sustainability, food safety and restrictions in trading of animal products. As the indirect effects of animal disease are highly important for society, both the direct and indirect effects must be included, or at the least considered, in the economical evaluation of animal disease (McInerney, 1996; James, 2005).

2.1.2 Disease control

In order to restore the production, it is necessary to implement strategies that will reduce the negative effect of disease (Howe, 1985; McInerney, 1987, 1988a; McInerney et al., 1992). Often, there are a number of alternative control strategies against disease, which can have different costs and time-horizons. Veterinary interventions, such as curative treatment (medication), are operational interventions with a short time horizon. Interventions with a mid-term time horizon (tactical interventions) can be related to preventive measures and management adjustments (e.g. changes in the feeding or bio-security procedures), and strategic interventions can be considered as interventions with a long time horizon (e.g. building or reconstructing the barn) (Saatkamp et al., 2001). Indeed, control measures against disease pose expenditures for the livestock producer. Therefore, it is necessary to weigh the cost of each control strategy with the benefit incurred, measured by the reduction in production losses (McInerney et al., 1992).
2.1.3 Utility value of the livestock producer

One of the main goals in AHE is to help in the decision making process when selecting among different disease control strategies (Dijkhuizen et al., 1995). The strategy that minimizes the total losses (the sum of production losses and control expenditures) is considered to be the most profitable strategy (McInerney et al., 1992), and has traditionally been the favorable choice of action. In the selection of an optimal control strategy, the preferences of the individual livestock producer can vary. Hence, it has previously been shown that farmer’s preferences and attitudes can widely differ (Austin et al., 2005). Though a high profitability is often the goal for the livestock producer, other preferences, such as good working condition, good animal welfare and leisure time may exist, which can influence the decision making process for the livestock producer. These preferences can in a technical term be described as attributes and the combination of different attributes can represent the overall utility of the livestock producer (Edwards-Jones, 2006; Kristensen et al., 2007). Hence, in the selection of the optimal control strategy, it is the goal to maximize the utility of the individual producer. Yet, it should be emphasized that the farmer can face certain restraints e.g., legal restraints, production quotas and limited capacities of land and buildings, which also must be taken into account in the decision making process (Kristensen et al., 2007).

2.2 The framework for economic analysis in AHE

Traditionally, the means of supporting decisions for the livestock producer has been through general recommendations and standards. Recommendations will often ignore the differences in preferences and constraints of livestock producers. Indeed, the use of norms and standards is often sub-optimal for the individual pig producer (Kristensen et al., 2007). The use of models in AHE has, therefore, become advantageous. A model based system can combine information from various sources. Furthermore, it is possible to take the individual herd conditions into account and represent the uncertainty of different scenarios, thus devising an optimal strategy for the specific farm (Kristensen et al., 2007).

McInerney (2001) illustrated a simplified framework for economic analysis in AHE. This framework can basically be divided into a biological part and an economic part (Fig. 2.3). A number of risk factors will influence the occurrence of disease, which can have certain negative effects on the productivity (e.g. reduced weight gain, decreased feed efficiency and mortality). An understanding of the relation between risk factors, disease and disease effects are essential elements in the biological part of the framework. The reduced productivity can lead to a reduction in the outcome for the farmer, and hence, a reduction in human benefit. The link between disease effects and the farmer outcomes is the economic part of the framework. Control strategies against disease can be implemented in order to restore the production to the disease free level and hence increase human benefit. The strategies can be directed at the risk factors (e.g. management adjustment), the
2.2 The framework for economic analysis in AHE

disease (e.g. preventive treatment) or the disease effects (e.g. curative treatment) (Fig. 2.3).

Figure 2.3: The framework for a model in AHE (McInerney, 2001)

This framework illustrates the importance of achieving a biological understanding regarding the disease in question, and in particular to obtain knowledge regarding the cause of disease, when constructing a model in AHE. Hence, it is necessary to identify the risk factors that influence the occurrence of disease and the production parameters that will be affected. Additionally, it is necessary to outline the different control actions (if they exist) that can eradicate or diminish disease and, thereby, restore production (McInerney, 2001).

Based on the framework, quantitative information regarding risk factors and their effects must be obtained. Moreover, information about how the effects of disease influence the production variables, and hence the farmer outcome, must be known (March, 1999; Rushton et al., 1999). Often, this information originate from research studies in veterinary epidemiology (Mlangwa and Samui, 1996). However, a common problem in the model-building-process is lack of information quantified in the literature (James, 2005). Hence, in case no information exists or in case data is too expensive to be collected and analyzed, it is possible to take advantage of expert opinions. Expert opinions can be used to produce quantitative assessments of causal relations, and a number of different approaches to obtain expert opinions can be used (van der Fels-Klerx et al., 2002). Though the elicitation of expert opinions can be associated with uncertainty, it is shown to be a useful way of obtaining inputs to models in AHE (van der Fels-Klerx et al., 2002).

2.2.1 Modeling approaches

Different mathematical modeling approaches can be used in order to combine the biological and economic part of the framework (Dijkhuizen et al., 1995). The choice of modeling method depends on the purpose and nature of the objectives.
The intention of some models is to find optimal solutions and hence support decisions in AHE. Other models focus on the biological part with the intention of representing new knowledge that can improve the understanding of a biological system. For the former, optimization models determine the optimal solution for a given herd taken the individual herd conditions into account (Dijkhuizen et al., 1995). Simulation models mimic the real life and illustrate the effect of different control strategies based on different “what-if” scenarios (Dijkhuizen et al., 1995; Pla, 2007). Principally, models in AHE can be classified based on different criteria, such as, whether or not they take time into account (dynamic models versus static models) and whether or not they are able to represent uncertainty (stochastic models versus deterministic models) (Dijkhuizen et al., 1995; Kristensen et al., 2007; Pla, 2007). It is essential to evaluate models by using sensitivity analyses, and hence, find parameters of great importance for the model output (Dijkhuizen et al., 1995). Moreover, it is important to validate the model output by estimating the degree of agreement between the model and the results in existing herds (Sørensen et al., 1995).

Decisively, AHE is an interdisciplinary research area combining aspects of Economics, Veterinary Epidemiology, Animal Sciences, Statistics and mathematical modeling. For the purpose of Objective 3, an object-oriented Bayesian network (OOBN) is chosen as this methodology combines information from different sources and present the variables of interest under uncertainty (Jensen, 2001). As all readers might not be familiar with OOBN, the next chapter describes the theory behind this methodology.

References


REFERENCES


A Bayesian network (BN) model is used for the investigation of Objective 3. This chapter describes the general framework of BN, and how a BN can be modeled using an object-oriented approach.

3.1 Bayesian networks

A Bayesian network is a static and probabilistic model that uses conditional probabilities to describe interdependencies between causal relations. The network consists of nodes and directed edges between nodes. Together, the nodes and directed edges must form a directed acyclic graph. A directed edge indicates the causal relation between any two nodes. Each node has a finite set of mutually exclusive states, and can be defined as either a parent node or a child node [Jensen, 2001].

3.1.1 Example of a Bayesian network

In order to illustrate the theory behind BN, the relationship between disease and a diagnostic test will be used as a simple example of a BN (Fig. 3.1). The result of a diagnostic test can help in estimating the probability of the disease. The disease (D) can either be present or not (D+/D-) and the diagnostic test (T) can be positive or negative (T+/T-). Hence, each of the nodes: Disease and Test can be in 2 mutually exclusive states. As the disease will decide the outcome of the diagnostic test, the direction of the causal edge is from disease to diagnostic test. Therefore, Disease is a parent of Test and Test is a child of Disease.

After the construction of the causal graph, it is necessary to provide the probabilities to be used in the BN. For nodes without parents, the probabilities are based
on prior knowledge. Hence, for the node Disease, the marginal distribution can be based on previous knowledge regarding the distribution of the particular disease in Denmark. For the node Test, it is necessary to obtain the probability for each state of the node given the state of the parent node. Table 3.1 illustrates the conditional probability table (CPT) for the node Test.

Table 3.1: Conditional probability table (CPT) for the node Test as it will appear in a Bayesian network

<table>
<thead>
<tr>
<th></th>
<th>Test+</th>
<th>Test-</th>
<th>Sum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disease+</td>
<td>$P(T+</td>
<td>D+)$</td>
<td>$P(T-</td>
</tr>
<tr>
<td>Disease-</td>
<td>$P(T+</td>
<td>D-)$</td>
<td>$P(T-</td>
</tr>
</tbody>
</table>

In this example, $P(T+|D+)$ is equivalent to the sensitivity of the test and $P(T-|D-)$ is equivalent to the specificity of the test. These probabilities are often known from literature. However, usually it is more interesting to know the probability of a disease given the outcome of the diagnostic test, in other words, the positive predictive value (PPV) of the disease. Bayes’ Theorem can be used to provide this estimate.

$$PPV = P(D+|T+) = \frac{P(T+|D+)P(D+)}{P(T+|D+)P(D+) + P(T+|D-)P(D-)} \quad (3.1)$$

Hence, information can flow in the opposite direction of the causality (i.e. the direction of the arrows), which is a characteristic feature of BN models (Jensen, 2001).

### 3.1.2 Previous work using Bayesian networks

In general, BNs are used to estimate new probabilities that are either unobservable in real life or very costly to observe. Hence, these nodes are called hypothesis nodes in the terminology of BN (Jensen, 2001).
Over the years, BN models have been recognized as a valid method for the physician in estimating the probability of a diagnosis in human medicine (Kahn et al., 1997). However, only few studies have used BN models for research within veterinary medicine. A BN model for *Mycoplasma hyopneumoniae* in finisher herds was developed by Otto and Kristensen (2004). The purpose of that study was to calculate consequences of possible control strategies against *Mycoplasma hyopneumoniae*. Recently, a large BN model for the finisher pig production has been developed. This model estimated consequences of alternative control strategies against a number of underlying diseases associated with gastrointestinal and respiratory disorders (Otto, 2006). Moreover, McKendrick et al. (2000) used a BN to estimate the likelihood of various diseases in sub-Saharan cattle in the presence and absence of certain clinical signs. The previous studies used evidence from either the herd (Otto and Kristensen, 2004; Otto, 2006) or individual animals (McKendrick et al., 2000) for the specification of new probabilities. Using evidence from both the herd as well as from a number of individual animals will easily result in a very large and complex structure of the BN. A possible solution to this problem is to apply an object-oriented structure that will ease the specification of the BN.

### 3.2 Object orientation

An object-oriented structure can be used to encapsulate a complex model, and make a hierarchical structure that can ease the specification of the BN model. The objects and classes are the main components in the object-oriented structure of a model. Each object has an entity with an identity, state and behaviour and is assigned to a class. A class describes a group of objects with similar attributes, behaviour and operations (Bangsø and Wuillemin, 2000; Bangsø, 2004).

In the previous example, it is possible to create a class designed for a pig. This class can be characterized using the attributes: Disease and Test. The Pig class can be used to create a number of pig objects, each of which will have exactly the same structure as the class. Each object of the Pig class will have a unique identity and present a unique state of the Disease and Test. As it is possible for a model to have different classes, a class designed for a herd can be another class in this example. This class can be characterized using the attributes: herd risk factor (Risk) and disease prevalence (Prev).

Two features of object-orientation can ease the specification of a large model. First, classes are created in order to provide a model that can be used for a number of similar objects. This reduces the complexity of a model when repetitive objects are needed for the specification of the model. Secondly, classes can be organized in a hierarchical way. Hence, a class can be divided into a superclass and subclasses, where the subclasses inherit the structure of the superclass. Whenever a change is made in the superclass, the same change will appear in each of the subclasses. The concept of inheritance eases the modeling process of a complex model (Bangsø, 2004).
3.3 Bayesian networks and object-orientation

An object-oriented Bayesian network (OOBN) model is a BN model applying an object-oriented approach. In the previous example, an OOBN model can be constructed that consists of 2 classes: a Herd class and a Pig class. Fig. 3.2 illustrates the structure of the Pig class, and Fig. 3.3 illustrates an example of the Herd class with 3 pig objects.

![Figure 3.2: The structure of the Pig class.](image)

![Figure 3.3: The structure of the Herd class with 3 pig objects. Only the nodes Prev* from the pig objects are visible in the Herd class.](image)

The probability of disease for each pig is influenced by the prevalence of disease in the herd. Basically, the Herd class (Fig. 3.3) do not know anything about
the Pig class (Fig. 3.2). The only nodes from the pig objects that can be seen in the Herd class are the nodes \( \text{Prev}^* \).

In an OOBN model, each node can be characterized as an input node, output node or protected node \( \text{[Bangsø, 2004]} \). Input nodes represent nodes that are parents of nodes inside instances of the class, whereas output nodes are nodes that are parents of nodes outside instances of the class. Protected nodes represent nodes that can only have parents and children inside the class. The input nodes and output nodes can be seen from the outside, and hence, form the interface of the OOBN model. A fourth type of node is the reference node. The reference node is a node outside the class that is identical to a parent node inside the class. Hence, the reference node can be described as a pointer that provides evidence to the OOBN. Therefore, all reference nodes are classified as input nodes \( \text{[Bangsø, 2004]} \).

Using the notation of OOBN models, the nodes: Risk, Disease and Test are characterized as protected nodes in the example. The prevalence node (Prev) is a reference node (and therefore input node) pointing at the referenced nodes \( \text{Prev}^* \) in the pig objects (which are the only nodes seen in the Herd class). Information regarding the herd prevalence of disease (prev) will, therefore, come from 2 sources: the herd risk factors and the disease status of individual pigs.

References


CHAPTER 4
LAMENESS IN FINISHERS - A REVIEW

In general, lameness in finishers represents a variety of clinical signs overall characterized as a deviation in the normal gait and posture. Clinical signs of lameness are often caused by disorders i.e. a physical derangement of the leg and/or feet. This review will focus on the underlying causes of the common leg disorders seen in the finisher pig production: 1. Infectious arthritis caused by pathogens (e.g. *Mycoplasma hyosynoviae*, *Erysipelothrix rhusiopathiae*, *Haemophilus parasuis* and *Streptococcus suis*) 2. Osteochondrosis and 3. Physical injuries to the leg and feet. For each leg disorder, a description of clinical manifestations and pathological findings will be given. A brief comment on the pathogenesis will be added to the description of each pathogen causing infectious arthritis, while the main emphasis will be on the possible risk factors that can predispose to the occurrence of leg disorders and eventual lameness in finishers. Hence, a description of risk factors for infectious arthritis, osteochondrosis and physical injuries are given in separate sections, while an overview of the risk factors is presented in Table 4.1.

4.1 Infectious arthritis

4.1.1 Mycoplasma hyosynoviae

*Mycoplasma hyosynoviae* is a swine-specific mycoplasma that survives in the environment ([Friis] 1987, [Kobisch and Friis] 1996, [Hagedorn-Olsen et al.] 1999a). Arthritis caused by *Mycoplasma hyosynoviae* is often produced by an intranasal exposure. A systemic phase of the infection usually lasts between 1 and 2 weeks, and the microorganism can spread through the blood to the synovial membrane and other serous membranes. After an incubation time of 4 and 9 days, infected pigs
Table 4.1: Summery of the most important risk factors for the 3 cause-categories of leg disorders in finisher herds

<table>
<thead>
<tr>
<th>Cause category</th>
<th>Factor</th>
<th>Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infectious</td>
<td>Immunity</td>
<td>Affect the risk</td>
</tr>
<tr>
<td></td>
<td>Purchase policy</td>
<td>Mixing of piglets increase the risk</td>
</tr>
<tr>
<td></td>
<td>All in all out</td>
<td>Decrease the risk</td>
</tr>
<tr>
<td></td>
<td>Herd size</td>
<td>Increase with increasing size</td>
</tr>
<tr>
<td></td>
<td>Pen density</td>
<td>Increase with higher density</td>
</tr>
<tr>
<td></td>
<td>Floor type</td>
<td>Affect the risk</td>
</tr>
<tr>
<td></td>
<td>Bedding</td>
<td>The use of straw increase the risk</td>
</tr>
<tr>
<td>Osteochondrosis</td>
<td>Breed</td>
<td>Landrace increase the risk</td>
</tr>
<tr>
<td></td>
<td>Daily weight gain</td>
<td>Increase with increasing weight gain</td>
</tr>
<tr>
<td></td>
<td>LMP</td>
<td>Increase with increasing percentage</td>
</tr>
<tr>
<td></td>
<td>Feeding strategy</td>
<td>Ad libitum increase the risk</td>
</tr>
<tr>
<td></td>
<td>Gender</td>
<td>Castrate increase the risk</td>
</tr>
<tr>
<td></td>
<td>Exercise</td>
<td>Exercise decrease the risk</td>
</tr>
<tr>
<td></td>
<td>Trauma</td>
<td>Injuries increase the risk</td>
</tr>
<tr>
<td>Physical</td>
<td>Floor type</td>
<td>Slatted floors increase the risk</td>
</tr>
<tr>
<td></td>
<td>Quality of the floor</td>
<td>Affect the risk</td>
</tr>
<tr>
<td></td>
<td>Bedding</td>
<td>Straw decrease the risk</td>
</tr>
<tr>
<td></td>
<td>Pen density</td>
<td>Increase with higher density</td>
</tr>
</tbody>
</table>

can develop clinical signs of arthritis (Hagedorn-Olsen et al., 1999a). The pigs will either recover spontaneously after approximately 1 to 2 weeks or become chronically infected (Friis, 1987). Hence, *Mycoplasma hyosynoviae* can be isolated from the tonsils of both chronically infected and healthy pigs (Friis et al., 1991). These pigs can infect pen-mates and moreover be at risk of a re-infection (Friis, 1987; Hagedorn-Olsen et al., 1999a). Clinical signs are usually observed in growing-finishing pigs (40-100 kg). These signs can vary but most often lameness of the hind legs is seen and a dog-sitting position can be observed. Occasionally, the lameness is acute and the affected pig will have a rise in temperature (Hagedorn-Olsen et al., 1999a; Friis, 1987). In severe outbreaks of *Mycoplasma hyosynoviae*, morbidity rates between 10 and 50% have been noted (Kobisch and Friis, 1996). *Mycoplasma hyosynoviae* has also been isolated from joints from pigs with no history of lameness (Nielsen et al., 2001). Thus, although *Mycoplasma hyosynoviae* is present in a herd, it may not always cause arthritis among finishers in the herd (Hagedorn-Olsen et al., 1999b). The macroscopic pathological findings are mainly non-purulent arthritis with moderate to severe oedema and thickening of the synovial membrane. Serohaemorrhagic or serofibrinous synovial fluid can be found in joints from affected pigs (Hagedorn-Olsen et al., 1999b).

4.1.2 Erysipelothrix rhusiopathiae

*Erysipelothrix rhusiopathiae* is a gram positive rod that can affect a number of species. Usually, pigs in the age interval from 3 months to 3 years become in-
Lameness in finishers - a review

32

fected (Wood and Henderson, 2006). The bacterium survives well in soil, slurry
and water, and several serotypes of Erysipelothrix rhusiopathiae have been iso-
lated (Norrung et al., 1987). Studies have shown that Erysipelothrix rhusiopathiae
is the most frequently isolated pathogen in infected joints (Hariharan et al., 1992;
Smith and Morgan, 1997). Among finishers sent to slaughter, the occurrence of
arthritis caused by Erysipelothrix rhusiopathiae in Danish finishers has previously
been estimated to be 0.2 per 1000 carcasses examined (Mousing et al., 1997). The
bacterium enters the body through different routes, e.g. skin lesions, the upper
respiratory tract or the gastro-intestinal tract and cause acute, subacute or chronic
infection. Erysipelothrix rhusiopathiae has been isolated from the tonsils of chron-
ically infected pigs as well as from healthy pigs (Takahashi et al., 1989). It is be-
lieved that the enzyme, neuramidase, is important in the pathogenesis of erysipelas
(Wood and Henderson, 2006).

In the acute and subacute stages, pigs will suffer from bacteraemia and show
clinical signs of a generalized infection. Pigs with clinical signs of acute erysipelas
are usually lethargic with a rise in the body temperature, and sudden death falls
may occur. The limbs are painful and swollen and the feet are carried under the
center of the body. Rhomboid urticarial lesions can be seen in the skin after 2 to 3
days. Arthritis may follow the acute erysipelas and is a common manifestation of
the chronic stage. Affected pigs may have a stiff gait and swollen joints. Moreover,
clinical signs of cardiac insufficiency are occasionally seen (Wood and Henderson,
2006).

Post mortem investigation of joints from chronically affected pigs often reveals
a hyperaemic and thickened synovial membrane, and characteristic fringes can pro-
trude into the joint cavity. There can be an excessive amount of synovial fluids, and
the lymph nodes draining the affected joint are usually enlarged and oedematous
(Butenschon et al., 1995; Wood and Henderson, 2006).

4.1.3 Haemophilus parasuis

Haemophilus parasuis is a gram negative rod that can be found in the upper respi-
ratory tract of finisher pigs (Smart et al., 1989). Fifteen serovars have been isolated
and the bacterium can give rise to the disease complex called Glässers disease
(Angen et al., 2004; Nedbalcova et al., 2006). From the upper respiratory tract, the
bacterium can invade into the system. However, the pathogenesis has not yet been
fully described (Nedbalcova et al., 2006). Usually the disease affects pigs between
2 weeks and 4 months. In the acute stage of Glässers disease, pigs are lethargic and
can have an increase in the body temperature. In naive herds, the mortality may
be as high as 50% (Nicolet, 1992). Respiratory distress, tremor and incoordina-
tion can be observed, and cyanosis can appear on the skin of the peripheral parts
of the body. Often affected pigs show clinical signs of lameness. The joints may
be swollen, hot and painful on palpation (Nicolet, 1992). The bacterium has also
been isolated as a secondary microorganism in the lung tissue from pigs that suffer
from pneumonia (Rapp-Gabrielson et al., 2006). Serofibrinous or fibrino-purulent
4.1 Infectious arthritis

Exudates on mucosal membranes are characteristic findings at necropsy, and pleuritis, pericarditis, peritonitis, meningitis and arthritis are often found (Nicolet [1992]; Nedbalcova et al. [2006]).

4.1.4 Streptococcus suis

*Streptococcus suis* is a gram positive coccus that can be found in the upper respiratory tract as well as in the genital and alimentary tract of pigs (Higgens and Gottschalk [2006]). Several serotypes of *Streptococcus suis* have been isolated. In a Danish study, serotype 2 and 7 represented more than 75% of isolated serotypes (Boetner et al. [1987]). *Streptococcus suis* can affect a number of animal species and may give rise to infection in humans. Invasion of the bacterium from the upper respiratory tract into the blood may also occur, and several virulence factors seem to be important in the pathogenesis (Gottschalk and Segura [2000]). The highest incidence of clinical signs are usually seen in young pigs (from 2 to 10 weeks), and the morbidity appear to be low (< 5%). The early clinical signs are increased body temperature followed by septicaemia. Inappetence, depression and shifting lameness can also be observed. A characteristic feature of *Streptococcus suis* is clinical signs of meningitis (Clifton-Hadley et al. [1986]). At necropsy, fibrinous polyserositis is often observed, and fibrinous or fibrino-purulent synovitis may be found in affected joints (Clifton-Hadley et al. [1986]).

4.1.5 Risk factors for infectious arthritis

A number of risk factors can predispose to the occurrence of arthritis caused by infectious pathogens. These factors can be related to the characteristics of the pathogen, the animal and the environment. The immunity of individual pigs, the weight of muscles and the amount of stress exposed to the animal can be important for the occurrence of infectious arthritis (Ross et al. [1971]; Hagedorn-Olsen et al. [1999b]). Mixing of pigs from different herds can increase the risk of introducing infectious pathogens causing arthritis (Smart et al. [1989]). Also the size of the herd can be important. In a case control study of factors associated with arthritis, the case herds were in general larger than the control herds (Heinonen et al. [2007]). It has been found that sectioned production can reduce the occurrence of *Mycoplasma hyopneumoniae* induced pneumonia (Nielsen et al. [2000]). Hence, the production type (continuous versus sectioned) may also affect the risk of introducing pathogens causing infectious arthritis. Conditions in the pens, such as the stocking density, the floor type and bedding have also been related to infectious arthritis in the finisher pig production (Kobisch and Fries [1996]; Smith and Morgan [1997]; Thacker [2006]). However, only few of the above mentioned studies have quantified the importance of the risk factors on the occurrence of infectious arthritis.
4.2 Osteochondrosis

4.2.1 Description of osteochondrosis in finishers

Osteochondrosis is a disturbance of the endochondral ossification in the joint cartilage as well as the epiphyseal plates (Grøndalen 1974a). The initial state is a focal thickening of the cartilage followed by necrosis occurring in the deep cartilage layer. These focuses do not undergo mineralization and further ossification. Dissecting lesions can develop leading to the formation of a cartilage flap or fragment (Jørgensen et al. 1995). Osteochondrosis often occurs bilaterally and changes are usually seen in the condyles of the femur and humerus and in the physeal growth plate of the ulna (Jørgensen et al. 1995; Jørgensen and Andersen 2000). Three different manifestations of lesions have been described by Ytrehus (2004): osteochondrosis latens, osteochondrosis manifesta and osteochondrosis dissecans. Osteochondrosis latens (OCL) is characterized as a “focal necrosis of cartilage canals and adjacent epiphyseal growth cartilage of the resting zone, not protruding into underlying bone and hence not visible on macroscopic and microscopic examination”. Osteochondrosis manifesta (OCM) is characterized as “a focus of necrotic growth cartilage partially surrounded by the advancing ossification front and hence visible on macroscopic inspection of cut section or radiographic examination as uneven or thickened cartilage protruding into the underlying bone”. Finally, osteochondrosis dissecans (OCD) is characterised as “lesions of fissured/fractured articular cartilage overlying a focus of necrotic growth cartilage protruding into the underlying bone”. In a study of 9,696 finishers in 4 herds, the prevalence of pigs with macroscopic osteochondrotic lesions in joints was above 65% (Busch et al. 2007a). The prevalence of pigs with OCD is usually lower, and has been shown to be 7 and 14% (Ytrehus et al. 2004; Busch et al. 2007a).

The correlation between osteochondrosis and lameness has previously been investigated. Jørgensen et al. (1995) found a significant association between OCD in the elbow joint and leg disorder problems such as “stiff movement”. Other studies have also shown a relation between severe joint lesions and clinical signs of lameness (Brennan and Aherne 1986; Lundeheim 1987). However, this disagrees with a number of studies that did not find any correlation between lameness and osteochondral changes in the joints (Reiland et al. 1978; Fredeen and Sather 1978; Jørgensen 1995).

4.2.2 Risk factors for osteochondrosis

Risk factors associated with osteochondrosis are mainly related to the intrinsic features of the pig. A heritable component is associated with the occurrence of osteochondrosis, and a heritability of 0.2-0.5 has been found (Lundeheim 1987; Stern et al. 1995). Among purebred pigs, Landrace has the highest degree of leg weakness and osteochondrosis compared to other breeds (Grøndalen 1974b; Lundeheim 1987; Jørgensen and Vestergaard 1990; Jørgensen and Andersen 2000).
4.3 Injuries to the limb and claw

The differences in the occurrences of osteochondrosis among breeds can be due to differences in the exterior and in the joint and bone shape of the breeds (Grøndalen, 1974b). A high daily weight gain may predispose to leg weakness and osteochondrosis (Ketland, 1978; Goedegebuure et al., 1980; Carlson et al., 1988). Busch et al. (2007b) found that for each 100 gram increase in the daily weight gain, the risk of osteochondrotic lesions increased by approximately 20% (OR = 1.14 – 1.21). Rapid growth rate causes mechanical stress to the joint cartilage which consequently can increase the risk of osteochondrosis. Hence, lowering the growth rate would be beneficial to prevent osteochondrosis in finishers (Arnbjerg, 2007).

A genetic correlation between the meat quality and osteochondrosis has been found (Stern et al., 1995; Kadarmideen et al., 2004). It has been shown that the risk of osteochondrosis can increase by 3-5% (OR = 1.03 – 1.05) for each percentage point increase in the lean meat percentage (Busch et al., 2007b).

The feeding strategy used in the finisher pig production can also be important for the occurrence of leg weakness and osteochondrosis. Pigs fed ad libitum have a higher degree of leg weakness compared to pigs fed on a restricted diet (Lepine et al., 1985; Jørgensen, 1995). This can be explained by the fact that pigs fed ad libitum tend to have a faster growth rate which will increase the risk of osteochondrosis. Stern et al. (1995) showed that pigs fed a low protein diet (13.1% crude protein) had more favorable osteochondrosis scores compared to pigs fed a high protein diet (18.5% crude protein). A number of minerals (calcium and phosphorus) as well as vitamins (vitamin A, C, D and biotin) have been suggested to be involved in the development of osteochondrosis. However, no significant effect of any of these vitamins and minerals on osteochondrosis has been found (Brennan and Aherne, 1986; Nakano et al., 1987).

There is a difference in the incidence and in the severity of leg weakness and osteochondrosis between genders. Leg weakness and osteochondrosis have been shown to be more pronounced in castrates and boars compared to females (Sather and Fredeen, 1982; van der Wal et al., 1983; Lundehan, 1987; Stern et al., 1995; Ytrehus et al., 2004; Jørgensen and Nielsen, 2005). Hence, castrates have 1.17-1.31 higher odds (OR = 1.17 – 1.31) for osteochondrotic lesions compared to females (Busch et al., 2007b). Other factors such as the joint conformation, mechanical stress, lack of exercise and trauma can also be involved in the development of leg weakness and osteochondrosis (Nakano et al., 1987; Nakano and Aherne, 1988; Petersen, 1998; Ytrehus et al., 2004).

4.3 Injuries to the limb and claw

4.3.1 Description of limb and claw injuries

Injuries to the leg and feet can lead to clinical signs of lameness. Fractures of the limb and ruptures of muscles and tendons will often result in acute lameness. However, injuries due to violent trauma occur sporadically in finisher herds. In a large
study of emergency culling and mortality in 1319 growing finishing pigs, luxation and fracture was diagnosed in only one case (0.07%) (Baumann and Bilkei, 2002). Contrary, lesions to the claw are common findings among finisher pigs. Penny et al. (1963) found 2080 of 3195 finishers (65%) with lesions of the claw. In a more recent study, 3974 finishing pigs from 21 units were examined and 94% had at least one foot lesion (Mouttotou et al., 1997). The lesions can have different clinical manifestations. In the volar area of the feet, erosions to the sole, toe and heel can be observed, whereas lesions to the white line and cracks in the horn are common manifestations in the wall of the claw (Penny et al., 1963). Opportunistic bacteria from the environment can invade the claw lesions and give rise to secondary infections. In a study by Mouttotou et al. (1997) sole lesions (62%) and white line lesions (55%) appeared with the highest prevalence. It was found that lateral digits were more severely affected with lesions than medial digits and, the hind claws more commonly affected compared to the front claws (Mouttotou et al., 1998a).

Another common manifestation in finisher pigs is adventitious bursitis, which is a soft tissue swelling containing serous fluids and lined with fibroblasts. Bursitis arises in the subcutaneous connective tissue and are usually located on the hind limbs below the hock. In England, the prevalence of bursitis in finishers has been shown to be in the range from 51 to 85% (Mouttotou et al., 1998a,b). Bursitis rarely cause clinical signs of lameness, however, an association between bursitis and foot lesions has been found (Mouttotou et al., 1998a).

### 4.3.2 Risk factors for injuries to the leg and claw

The construction of the pen and the quality of the floor are important for the occurrence of injuries to the leg and claw. Pigs on fully slatted (RR = 2.26) and partially slatted floors (RR = 1.92) have a higher frequency of lameness compared to pigs housed on deep bedding (Nielsen et al., 2003). Furthermore, slatted floors increase the risk of sole and heel erosion compared to solid floors with straw-bedding (Scott et al., 2006; Mouttotou et al., 1999a). Yet, another study showed that pigs on concrete solid floors without bedding had the highest risk of claw lesions compared to straw-bedded pens and fully slatted floors (Jørgensen, 2003). The quality of the floor, such as rough sharp edges, abrasiveness, slipperiness and the width of the slats, are also important for the development of claw lesions and bursitis (Smith and Morgan, 1998; Rähse and Hoy, 2007).

The supply of straw can protect pigs from injuries to the leg and claw and straw bedding will, therefore, reduce the prevalence of these lesions (Kelly et al., 2000; Mouttotou and Green, 1999; Mouttotou et al., 1999b). Hence, bedding covering the whole pen (OR = 0.11) or only the lying area (OR = 0.29) has been shown to reduce the risk of bursitis compared to pigs in pens with no bedding (Mouttotou et al., 1999b). Straw bedding can also have a negative effect on the claws of pigs. Pigs on straw bedding abrade their claws slowly and this can increase the risk of wall lesions and toe erosions (Mouttotou et al., 1999a; Scott et al., 2006).

High stocking density and poor hygiene in pens can increase the risk of injuries
to the leg and claw (Jørgensen, 2003; Mouttotou et al., 1999b). In 4 sow herds with high stocking density (<2 m² per sow) among pregnant sows, the relative risk of claw infection was 2.1 times higher than in 11 herds with a lower stocking density (≥2 m² per sow) (Gjein and Larssen, 1995). Finally, it has been suggested that intrinsic factors such as claw size, variation in the weight of the hooves, and nutritional and genetic factors, are important for the development of claw lesions (Mouttotou and Green, 1999).

References


REFERENCES


Mouttotou, N., Hatchell, F. M., Green, L. E., 1999a. Foot lesions in finishing pigs and their associations with the type of floor. Veterinary Record 144, 629–632.


Mouttotou, N., Sterry, J., Green, L. E., 1998b. Cohort study of the association between adventitious bursitis of the hock and the age at slaughter and carcase quality of the pigs on one farm. Veterinary Record 142, 52–55.


Penny, R. H. C., Osborne, A. D., Wright, A. I., 1963. The causes and incidence of lameness in store and adult pigs. Veterinary Record 75, 1225–1235.


5.1 Data sources and data collection

The investigation of Objectives 1 and 2 was based on empirical data. A number of existing datasets from the Danish Meat Association, Danish Pig Production and University of Copenhagen were attained and evaluated for the selection of suitable data for Objectives 1 and 2. Basically, it was required that the chosen data set met the following criteria:

1. The collection of data should be completed.
2. Both clinical and production variables should be available.
3. Registration should be on animal level.

In the evaluation process, data from the Danish boar test station: “Bøgildgaard” fulfilled all requirements, and was considered to be the most appropriate dataset for the purpose of the objectives. Therefore, data from “Bøgildgaard” was selected for the investigation of Objectives 1 and 2.

5.1.1 The boar test station - background

The boar test station is owned and run by the Danish Pig Production. The purpose of the boar test station is to evaluate purebred boars of the breeds: Duroc, Hampshire, Landrace and Yorkshire and select boars for collection of semen for use for artificial insemination. Hence, a selection index is calculated for each boar based on the breeding values for different traits (e.g. daily weight gain, feed conversion, lean meat percentage (LMP), litter size and longevity). Approximately, 5000 boars are tested at the station annually, and 20% of the boars with the highest index scores are selected for artificial insemination.
5.1.2 Description of the production system

Danish breeding herds deliver four-week old piglets to the boar test station. Piglets that arrive at the test station in the same week are initially allocated in one section in the early weaning unit. The early weaning section has 8 sections each with 4 pens and all pens have fully slatted floors. Twenty-eight piglets of the same breed are placed together in one pen, and after 7 days, 14 piglets with the lowest weight are removed from each pen and allocated to a pen in another section. Each piglet is individually identified with an ear tag at arrival. Vaccination against *Mycoplasma hyopneumoniae* (Stellamune®) and *Actinobacillus pleuropneumoniae* (Haemo Shield®) is carried out when the piglets are 4 and 6 weeks, respectively. After approximately 6 weeks in the early weaning section, boars in the same batch are moved to the finisher unit. The finisher unit comprise of 16 sections with 8 pens. These pens have solid concrete floors with bedding, and feed and water are given ad libitum. A graphical depiction of the production system in “Bøgildgaard” is shown in Fig. 5.1.

![Graphical depiction of the production system at the test station](image)

Figure 5.1: A graphical depiction of the production system at the test station

5.1.3 Production records

The technical staff from the test station record the weight of each piglet at arrival, and at various intervals during the fattening period. In the finisher unit, feed is given through a single feeder. Each time a boar enters the feeder, the time and the feed intake is registered in a central data base.
5.1 Data sources and data collection

5.1.4 Disease records

All boars are inspected daily by the technical staff and boars with clinical signs of disease are treated medically. The diagnoses are recorded according to a pre-specified coding system that corresponds to the clinical diagnosis. In Appendix A, an overview of the disease codes are given. Basically, boars are only given a diagnosis in case they are treated. Hence, when a disease is recorded, the medical product and the amount of medicine used in the treatment are also recorded.

5.1.5 Local abattoir

Boars not selected for AI are slaughtered at the local abattoir “Herning slagteri”. Here, veterinarians and technicians assess the carcasses by visual examination and record all pathological lesions based on the recording system used by Danish Crown. The pathology codes used at the abattoir are presented in Appendix A. Records regarding the LMP and the slaughter weight are also available from the local abattoir.

5.1.6 Veterinary laboratory

Boars that either die unassisted or are euthanized due to severe clinical signs, are investigated at the veterinary laboratory in Kjelleup. Here, all pathological diagnoses are recorded by a veterinarian. Table 5.1 and Fig. 5.2 give an overview of the origin of data used for Objectives 1 and 2.

<table>
<thead>
<tr>
<th>Initial database</th>
<th>Origin of data</th>
<th>Variable(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breed database</td>
<td>Herd of origin</td>
<td>Breed of the boar (Duroc, Hampshire, Landrace, Yorkshire)</td>
</tr>
<tr>
<td>Weight database</td>
<td>Bøgildgaard</td>
<td>Weight of the boar recorded at various intervals</td>
</tr>
<tr>
<td>Disease database</td>
<td>Bøgildgaard</td>
<td>Clinical diagnosis based on a pre-specified coding system. Medical product used in the treatment</td>
</tr>
<tr>
<td>Feed database</td>
<td>Bøgildgaard</td>
<td>Feed intake recorded repeatedly for each boar</td>
</tr>
<tr>
<td>Slaughter database</td>
<td>Abattoir</td>
<td>Meat inspection record based on a pre-specified coding system. Carcass weight of the boar. Lean meat percentage</td>
</tr>
<tr>
<td>Pathological database</td>
<td>Laboratory</td>
<td>Pathological diagnosis</td>
</tr>
</tbody>
</table>
5.2 Data management

5.2.1 Objective 1

Data from the boar test station: Bøgildgaard was used to generate new variables for the purpose of Objective 1. Hence, 2 production variables were generated: Mean daily weight gain (MDWG) and feed conversion ratio (FCR).

\[ \text{MDWG} = \frac{W_{\text{End}} - W_{\text{Start}}}{D} \left( \frac{\text{Kg}}{\text{Day}} \right) \]  \hspace{1cm} (5.1)

Where

- \( W_{\text{Start}} \) was the weight of the boar when entering the finisher unit (kilograms), at 10 weeks of age.
- \( W_{\text{End}} \) was the weight at the time for leaving the finisher unit (kilograms).
- \( D \) was the number of days in the finisher unit.

The FCR measured the number of feed units (FU) used per kilogram weight gain and the FCR for each boar was calculated using the following formula:

\[ \text{FCR} = C \times \left( \frac{\text{Feed}}{W_{\text{End}} - W_{\text{Start}}} \right) \left( \frac{\text{FU}}{\text{Kg weight gain}} \right) \]  \hspace{1cm} (5.2)
5.2 Data management

Where

• Feed was the total amount of feed intake (kilograms) in the period from entering to leaving the finisher unit.

• C was the FU per kg of feed (C = 1.05).

Two explanatory variables were furthermore generated for Objective 1: Lameness treatments and Non-lameness treatments. Lameness treatments represented the number of treatments each boar had received against lameness during the fattening period. The variable was divided into 5 categories: 1-3 lameness treatments, 4 lameness treatments, 5 lameness treatments, more than 5 lameness treatments and no lameness treatments. All treatment records other than lameness were recoded as “non-lameness treatments”. Boars were divided into 2 groups according to whether or not they had records of “non-lameness treatments” during the fattening period.

5.2.2 Data control

Boars that had been in the finisher unit for a period of at least 70 days were included in Objective 1. Hence, from February 2002 to December 2004, a total of 12,073 boars had been at the test station for at least 70 days. During the fattening period, 113 of the boars died, and 1484 boars had missing values on the feed intake. These boars were consequently excluded from the study. The continuous variables: weight at 4 weeks, FCR and MDWG were checked for extreme values and frequency distributions were used to identify potential illegal values for the categorical variables. Three boars had biological extreme values for the FCR (> 12(\frac{FU}{kg})), and it was decided to exclude these 3 boars from the analyses.

5.2.3 Objective 2

Data from the boar test station: “Bøgildgaard” as well as data from the slaughterhouse was used to generate new variables for the purpose of Objective 2. Hence, the performance variable: profit margin (PM) was generated for each boar in the interval from 30 kg until slaughter. This variable represented the actual profit received for each animal.

\[
PM = (\text{Carcass weight} \times \text{price}) - (FU \times \text{price}) - (\text{medicine}) - (\text{piglet price}) \tag{5.3}
\]

Where

• Carcass weight was the weight of the boar when slaughtered and price represented the price per kg carcass.

• FU was the total amount of feed units consumed by each boar during the finishing period and price was the price per FU.
• Medicine represented the total expenses for medicine during the finishing period.

• Piglet price corresponded to the price of a piglet adjusted according to its actual weight.

Oral treatments were given in the drinking water when a majority of boars in a pen showed clinical signs of pneumonia or gastro-intestinal disorders. Contrary, parenteral treatments were given as injections to individual boars as treatment against disease (e.g. pneumonia, lethargy and lameness). New variables were generated that represented the disease status for each boar. These were: pathological recordings at slaughter (yes/no), oral treatments in the finishing period (yes/no) and parenteral treatments in the finishing period (yes/no).

5.2.4 Data control

During the period from July 2002 to December 2004, 7306 boars from the test station were recorded at the slaughterhouse. A total of 575 boars were excluded by the technical staff during the fattening period, primarily due to a low slaughter weight. Furthermore, 954 had missing values on either the feed intake or the LMP. Weight at 4 weeks and PM were checked for extreme values, and the categorical variables were checked for potential illegal values.

5.3 Statistical analysis for Objectives 1 and 2

The analyses of Objectives 1 and 2 were performed using multivariable hierarchical models (PROC MIXED) in (SAS) [2002]. For both objectives the hierarchical structure of the data is depicted in Fig. 5.3.

![Herd of origin](Pen) Pig

Figure 5.3: The hierarchical structure of the data for Objectives 1 and 2

Herd of origin and boars placed in the same pen (indicated by a unique batch number) were included as random effects in the analyses in order to allow for the
variation between herds of origin and batch. The initial model included all potential risk factors and the biological plausible interactions of fixed effects. An overview of the risk factors included in Objectives 1 and 2 is given in Table 5.2.

Table 5.2: Overview of the outcome and explanatory variables for Objectives 1 and 2

<table>
<thead>
<tr>
<th>Objective</th>
<th>N⁴</th>
<th>Outcome</th>
<th>Explanatory variables</th>
</tr>
</thead>
<tbody>
<tr>
<td>Objective 1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Part 1</td>
<td>10473</td>
<td>MDWG</td>
<td>Number of lameness treatments. Non-lameness treatments (yes/no). Breed (Duroc, Hampshire, Landrace, Yorkshire). Weight at 4 weeks</td>
</tr>
<tr>
<td>Part 2</td>
<td>10473</td>
<td>FCR</td>
<td>Number of lameness treatments. Non-lameness treatments (yes/no). Breed (Duroc, Hampshire, Landrace, Yorkshire). Weight at 4 weeks</td>
</tr>
<tr>
<td>Objective 2</td>
<td>5777</td>
<td>PM</td>
<td>Pathological record (yes/no). Oral treatments (yes/no). Parenteral treatments (yes/no). Breed (Duroc, Hampshire, Landrace, Yorkshire). Weight at 4 weeks</td>
</tr>
</tbody>
</table>

⁴Number of pigs included in the study

As an example, the initial model for MDWG in Objective 1 was expressed as:

\[
Y_{ijklmn} = \mu + A_i + B_j + C_k + D_{xijk} + AB_{ij} + AC_{ik} + BC_{jk} + v_l + \omega_m + \epsilon_{ijklmn}
\]

Where

- \( Y_{ijklmn} \) was the MDWG for boar \( n \) in the \( i \)th lameness treatment, with the \( j \)th records of non-lameness treatment in the \( k \)th breed from the \( l \)th herd of origin and placed in the \( m \)th batch.
- \( \mu \) was the intercept.
- \( A_i \) was the fixed effect of the \( i \)th lameness treatment (i: 1-3, 4, 5, >5, no lameness treatments).
- \( B_j \) was the fixed effect of the \( j \)th records of non-lameness treatment (\( j \): yes, no).
- \( C_k \) was the fixed effect of the \( k \)th breed (\( k \): Duroc, Hampshire, Landrace, Yorkshire).
- \( D_{xijk} \) was the estimate of the weight at four weeks for the \( i \)th lameness treatment, the \( j \)th records of non-lameness treatment and the \( k \)th breed.
- \( AB_{ij} \) was the interaction between the \( i \)th lameness treatment and the \( j \)th records of non-lameness treatment.
• $AC_{ik}$ was the interaction between the $i$th lameness treatment and the $k$th breed.

• $BC_{jk}$ was the interaction between the $j$th records of non-lameness treatment and the $k$th breed.

• $v_l$ was the random effect of herd of origin.

• $\omega_m$ was the random effect of batch.

• $\epsilon_{ijklmn}$ was the residuals.

A backwards elimination strategy was applied to reduce the interaction terms and fixed effects. For all analyses, a significance level of 5% was used to exclude factors (Ersbøll et al., 2004).

Three assumptions exist for the use of analysis of variance (Ersbøll et al., 2004). These are:

1. Data should be normally distributed.

2. All observations should have equal variances.

3. Observations must be independent.

To assess the fulfillment of the assumptions, QQ-plots of residuals and plots of residuals versus predicted values were evaluated in all 3 analyses. Each level of the categorical variables was supposed to be more alike in regard to production compared to any other level, and therefore, different levels of the categorical variables were assumed to have different variances. Hence, among the categorical variables used in Objectives 1 and 2, variance heterogeneity was expected. Akaike’s Information Criteria (AIC) was used to validate whether the estimation of dispersion effects between the categorical variables gave a better model fit. Hence, for all 3 analyses, it was shown that the optimal variance included dispersion effects of the categorical variables.

Sensitivity analyses were carried out in Objective 2 in order to investigate the effect of changes in prices on the results. Hence, the analysis was repeated for each 10% change in the price of feed, medicine, kg carcass weight and 30 kg piglet, and evaluated in the interval from -50% to +50%.

5.4 Objective 3

The methodology used for the investigation of Objective 3 was based on a normative modeling approach. Hence, an object-oriented Bayesian network (OOBN) model was built in order to estimate the most likely cause of leg disorders in a finisher herd. The following section describes the qualitative structure of the OOBN model, and presents the materials and methods used for the quantitative part of the model.
5.4 Objective 3

5.4.1 Qualitative structure of the model

The causal structure of the OOBM model was based on knowledge from the literature (Chapter 4). Three major cause-categories of leg disorders were identified as being the most common in finisher herds: “Physical”, “Inherited” and “Infectious”. Hence, strategies for prophylaxis, treatment and control of leg disorders would depend on the underlying cause-category. The purpose of the OOBM model was to estimate a risk index for each of the 3 cause-categories. The risk index was defined on an arbitrary scale from 0 to 9, where the interpretation of 0 was: “poor risk” of the particular cause category and the interpretation of 9 was: “high risk” of the cause-category. The cause-category “Infectious” described the risk index of leg disorders due to arthritis caused by infectious pathogens (Mycoplasma hyosynoviae, Erysipelothrix rhusiopathiae, Haemophilus parasuis, and Streptococcus suis). The cause-category “Physical” described the risk index of leg disorders due to injuries to the leg or claw, primarily caused by conditions in the surroundings. Finally, the cause-category “Inherited” described the risk index of leg disorders due to osteochondrotic lesions. Hence, the 3 major cause-categories of leg disorders represented hypothesis nodes in the OOBM model.

A Herd class and a Pig class were identified as the basic components in the object-oriented structure of the OOBM model. The Herd class was used to create one object, and the Pig class was used to create several objects. Evidence about the herd could influence the risk index for the 3 cause-categories. Hence, the risk of infectious leg disorders was influenced by the herd size, the type of production (sectioned or continues production) and the number of herds from which the piglets were purchased. The type of floor, supply of straw and the stocking densities in the pens would influence the risk of both infectious and physical causes of leg disorders. Finally, the breed of the pigs (purebred and cross breed) and the estimated daily weight gain (600, ..., 1000 g/day) were assumed to influence the risk of inherited leg disorders. Fig. 5.4 depicts the causal structure of the Herd class. A thorough description of each node is given in Manuscript 3 (Table 11.1).

In the Pig class, the node “ObsLame” stated whether or not a selected pig (observed outside the pen) showed clinical signs of lameness. “PigLame” represented the true state of lameness for the individual pig. The relation between the 2 nodes: “PigLame” and “ObsLame” depended on the sensitivity and the specificity of the clinical observation of lameness. Available results from diagnostic tests would add information to each object in the Pig class. The diagnostic tests were specified as clinical examination (inspection and palpation of joints or claws), pathological examination of joints or claws and bacteriological examination of joint fluids. Hence, for each diagnostic test the estimated sensitivity and specificity were taken into account. Fig. 5.5 illustrates the causal structure of the Pig class, and a thorough description of each node is given in Manuscript 3 (Table 11.2). Based on evidence from the Herd class as well as information regarding the individual pigs in the Pig class, it was possible to estimate a risk index for each of the 3 cause-categories of leg disorders.
Figure 5.4: Depiction of the Herd class of the OOBN model

Figure 5.5: Depiction of the Pig class of the OOBN model
5.4 Objective 3

5.4.2 Materials for the quantitative part of the model

For the quantitative part of the model, the probabilities originated from 2 sources: results from published literature and expert opinions. Initially, the literature was reviewed for quantitative information to be used in the model. In this process, results from literature were evaluated in regard to the sample size used and the study population. Only results that were representative for Danish finisher herds were used as input in the model.

However, the literature could only provide a limited part of the quantitative information needed in the model. Therefore, 9 Danish experts within the field of leg disorder in finisher pigs were asked to assess the quantitative information for the model. Specific probabilities to be assessed were given to one or more experts with professional knowledge within a particular area, and altogether, each expert was asked to estimate between 10 and 96 conditional probabilities.

As the nodes: “Physical”, “Infectious” and “Inherited” were hypothesis nodes, and hence, not directly observable, it was not possible to quantify the links between information nodes in the Herd class and the 3 cause-category nodes. Instead, it was possible to obtain information regarding the causal links between the information nodes and the individual leg disorders in the Pig class. Fig. 5.6 presents the causal links that were elicited from experts in regard to the hypothesis node “Physical”. For the elicitation of the probabilities, the method developed by van der Gaag et al. (2001) was used. When 2 or more experts answered similar questions, the resulting probabilities used in the model were merely the average of the individual elicitation.

An example of a probability to be elicited is given in Fig. 5.7.

Figure 5.6: Causal links elicited from experts in regard to the hypothesis node: “Physical”. Edges shown with bold arrows are links from which we do not have quantitative information. Edges shown with dashed arrows are the quantitative information that were obtained.
Consider 100 pigs examined individually at a herd visit. The herd has a high stocking density in the pens. How often do you, during the examination, expect to find a pig with a fracture?

<table>
<thead>
<tr>
<th>Always(almost)</th>
<th>100</th>
</tr>
</thead>
<tbody>
<tr>
<td>Usually</td>
<td>85</td>
</tr>
<tr>
<td>Often</td>
<td>75</td>
</tr>
<tr>
<td>As often as not</td>
<td>50</td>
</tr>
<tr>
<td>Sometimes</td>
<td>25</td>
</tr>
<tr>
<td>Once in a while</td>
<td>15</td>
</tr>
<tr>
<td>(Almost)never</td>
<td>0</td>
</tr>
</tbody>
</table>

Figure 5.7: Example of the elicitation of probabilities using expert opinions. The conditional probability \( P(\text{Fracture}|\text{high stocking density}) \) was obtained by asking the above question.

### 5.4.3 Methodology for the construction of the nodes

In the following, a general description of the methodology for constructing the risk indexes for the 3 cause-category nodes in the Herd class, and for constructing the leg disorder nodes in the Pig class will be given. For a thorough description of the methodology, readers are referred to Appendix B.

Fig. 5.8 shows the general layout of a subgraph modeling the effects of a number of herd level risk factors \( R_1, R_2, \ldots, R_n \) on a number of distinct diseases \( D_1, D_2, \ldots, D_m \) at pig level. As illustrated in the figure, it was assumed that the effects of the risk factors always were expressed through the risk index, \( I \).

For each hypothesis node, the risk index was defined based on a linear equation that quantified the total effect of the information nodes (risk factors) on the leg disorders in question. Initially, the risk indexes were assumed to be continuous. Due to the fact that it is not possible for discrete child nodes (i.e. the leg disorders nodes) to have continuous parent nodes [Jensen, 2001], the nodes were eventually modeled as discrete variables, where 10 distinct levels (0–9) were distinguished. The general model for the risk index for a given configuration \((i_1, \ldots, i_n)\) of the risk factors \((R_1, \ldots, R_n)\) was defined with the following model

\[
I_{i_1 \ldots i_n} = \mu + \rho_{i_1}^1 + \rho_{i_2}^2 + \ldots + \rho_{i_n}^n + e_{i_1 \ldots i_n}, \quad (5.4)
\]

where

- \( I_{i_1 \ldots i_n} \) was the resulting risk index.
Figure 5.8: A number of herd level risk factors, $R_1, \ldots, R_n$, influencing a number of pig level diseases $D_1, \ldots, D_m$ through a common risk index $I$.

- $\mu$ was an intercept.
- $\rho_{i_k}^k$ was the systematic effect of state $i_k$ of risk factor $k$.
- $e_{i_1 \ldots i_n} \sim N(0, \sigma_e^2)$ was a random residual.

It was a model assumption that there were no interactions between the risk factors and that the effects of the risk factors were additive. Each leg disorder node in the Pig class was modeled using a logistic regression. Hence, the general model of an arbitrary pig to have the $k$th leg disorder for a given state $i_I$ of the risk index $I$ was defined as:

$$Y_{i_I}^k = \delta^0_k + \delta^1_k i_I,$$

Where

- $Y_{i_I}^k$ was the logistic transformation of the conditional probability of an arbitrary pig to have the leg disorder $k$ ($k$: yes, no).
- $\delta^0_k$ was the intercept indicating the base prevalence of the leg disorder $k$.
- $\delta^1_k$ was the slope which indicated the sensitivity to changes in the risk level of the herd.

Based on the probabilities elicited from experts and literature the parameter estimates for Eqs. (5.4) and (5.5) were determined in such a way that they created the best possible fit to the conditional probabilities under the constraint that all effects of the risk factors were expressed through a common hypothesis node. A combination of fitting a linear model and applying a general optimization function based on Nelder-Mead algorithm was used (Nelder and Mead, 1965). All analyses
were carried out in R ([R Development Core Team][1] [2006]). The linear model was fitted by the `lm` function and the Nelder-Mead algorithm was called through the `optim` function.

**References**


6.1 Objective 1 (Manuscript 1)

A total of 10,473 boars were included in Objective 1. During the finishing period, 4% of the boars had records of lameness treatments whereas 65% of the boars had records of non-lameness treatments. Lameness treatments among finishers were significantly associated with the MDWG at the 5% significance level ($p < 0.0001$). However, lameness treatments were not significantly associated with the FCR ($p = 0.14$). Boars with 1 to 3 lameness treatments had a significantly lower MDWG compared to boars with no lameness treatments. This corresponded to a relative reduction of 3%. The MDWG of boars with 4 and 5 lameness treatments did not differ significantly from boars with no lameness treatments. Boars with more than 5 lameness treatments had the largest reduction in the MDWG, which corresponded to a relative reduction of 4% compared to boars with no lameness treatments.

Non-lameness treatments in the finishing period were significantly associated with both the MDWG and the FCR. Non-lameness treatments caused a reduction in the MDWG of 6%. Moreover, there was an increase in the FCR of 0.04 FU per kg live weight due to non-lameness treatments. This was equivalent to a relative increase in the FCR of 2%. The explanatory factors: weight at 4 weeks and breed were significantly associated with both the MDWG and FCR. Duroc had a significantly higher MDWG whereas Hampshire had a significantly lower MDWG compared to other breeds. In the pair wise comparison of breed, Duroc and Yorkshire had a significantly lower FCR compared to Hampshire and Landrace. In both analyses, herd of origin explained approximate 1% and batch 14-16% of the total random variation. Table 6.1 summarizes the results from Objective 1.
### Table 6.1: Summary of the results from Objective 1

<table>
<thead>
<tr>
<th>Variable</th>
<th>Result</th>
<th>Specification of the effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lameness treatment</td>
<td>Significantly associated with MDWG ($p &lt; 0.0001$)</td>
<td>1-3 lameness treatments reduce the MDWG by 3% More than 5 lameness treatments reduce the MDWG by 4%</td>
</tr>
<tr>
<td></td>
<td>Not significantly associated with FCR ($p = 0.14$)</td>
<td></td>
</tr>
<tr>
<td>Non-lameness treatment</td>
<td>Significantly associated with MDWG ($p &lt; 0.0001$)</td>
<td>Non-lameness treatments reduce the MDWG by 6%</td>
</tr>
<tr>
<td></td>
<td>Significantly associated with FCR ($p &lt; 0.0001$)</td>
<td>Non-lameness treatments increase the FCR by 2%</td>
</tr>
<tr>
<td>Breed</td>
<td>Significantly associated with MDWG ($p &lt; 0.0001$)</td>
<td>Duroc has the highest MDWG. Hampshire has the lowest MDWG</td>
</tr>
<tr>
<td></td>
<td>Significantly associated with FCR ($p &lt; 0.0001$)</td>
<td>Duroc and Yorkshire have the lowest FCR. Hampshire and Landrace have the highest FCR</td>
</tr>
<tr>
<td>Weight at 4 weeks</td>
<td>Significantly associated with MDWG ($p &lt; 0.0001$)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Significantly associated with FCR ($p = 0.0281$)</td>
<td></td>
</tr>
</tbody>
</table>
6.2 Objective 2 (Manuscript 2)

A total of 5,777 boars were included in Objective 2. During the finishing period, 3,539 boars (61%) were treated against disease. Of boars that were treated, 3,074 (87%) had received oral treatments and 1,257 (36%) had received parenteral treatments. Moreover, 985 (17%) boars had pathological findings at slaughter. The analysis showed that oral and parenteral treatments were significantly associated with the PM at the 5% significance level. Boars that received parenteral treatments had a reduction in the PM of 17% compared to boars that were not treated parenterally. Oral treatments caused a reduction in the PM of 7%. The analysis showed a significant interaction between pathological findings and breed ($p = 0.005$). Hence, pathological findings caused a reduction in the PM of 4, 6, 20 and 8% for boars of the breeds Duroc, Hampshire, Landrace and Yorkshire, respectively. Moreover a significant association between weight at 4 weeks and PM was found. Herd of origin explained 1%, batch explained 9% whereas the residuals explained 90% of the total random variation. Table 6.2 summarizes the results from the analysis.

The effects of parenteral treatments, oral treatments and pathological findings were relatively robust in regard to changes in feed price and price of a 30 kg piglet. However, price per kg carcass weight appeared to influence the effect of all 3 disease indicators. The effect of oral and parenteral treatments on PM was sensitive to changes in medicine prices (Manuscript 2, Figs. 10.1-10.3).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Result</th>
<th>Specification of the effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parenteral treatment</td>
<td>Significantly associated with PM ($p &lt; 0.0001$)</td>
<td>Reduction in the PM of 17%</td>
</tr>
<tr>
<td>Oral treatment</td>
<td>Significantly associated with PM ($p &lt; 0.0001$)</td>
<td>Reduction in the PM of 7%</td>
</tr>
<tr>
<td>Pathological finding</td>
<td>Significant interaction between pathological finding and breed ($p &lt; 0.005$)</td>
<td>Duroc: reduction in the PM of 4 %. Hampshire: reduction in the PM of 6 %. Landrace: reduction in the PM of 20 %. Yorkshire: reduction in the PM of 8 %</td>
</tr>
<tr>
<td>Weight at 4 weeks</td>
<td>Significantly associated with PM ($p &lt; 0.0001$)</td>
<td></td>
</tr>
</tbody>
</table>

6.3 Objective 3 (Manuscript 3)

Two fictitious herds were used to investigate the behaviour of the OOB model. Hence, the level of information required in order to predict the most likely cause of leg disorders in each herd was investigated.
6.3.1 Modeling scenarios

Ten different scenarios were investigated in 2 fictitious herds with different herd characteristics. In each herd, 20% of the pigs were lame due to infectious arthritis caused by *Mycoplasma hyosynoviae*. Hence, the disease status was similar for the 2 herds. A total of 50 randomly selected pigs were observed for lameness outside the pen. It was assumed that infectious arthritis was the only problem in the herds. Herd 1 was defined as a low risk herd with the following characteristics: sectioned production, low pen densities (> 0.65m$^2$ space per pig in a pen), solid floors, no supply of straw in the pens, production of own piglets and delivering of 2000 finishers to the slaughter house, annually. Herd 2 was a high risk herd characterized as: delivering of 6000 finishers annually, high pen densities (< 0.65m$^2$ space per pig in a pen), partially slatted floors, sparse supply of straw, continuous production and purchase of piglets from several supply herds. Table 6.3 presents the 10 different scenarios used in the investigation.

6.3.2 Results

Figures for the cause-categories: “Physical”, “Inherited” and “Infectious” are presented in Manuscript 3 (Figs. 11.5, 11.6 and 11.7). The figures illustrate the risk indexes for the cause-categories in each scenario for the low-risk and the high-risk herd. A strong belief in a particular cause-category was presented by a high risk index, and hence, a right shift in the curve, whereas a low risk index indicated a poor belief in a particular cause-category, and hence, a left shift in the curve.

Comparing the 2 herds, it was possible to illustrate the change in knowledge when information at herd level and pig level was added to the model. Moreover, it was possible to see the effect of selecting different groups of animals for diagnostic examinations. In the low-risk herd, it was not possible to differentiate the cause-categories in Scenarios 1-3. When clinical examinations of lame pigs were carried out (Scenario 4), infectious leg disorders appeared with a higher risk than physical and inherited leg disorders. For the high-risk herd, infectious leg disorders tended to be the most likely cause after entering herd evidence in the model (Scenario 2). This tendency was even more pronounced after examining 50 randomly selected pigs for lameness outside the pen (Scenario 3). Hence, less information on animal level was required for the high-risk herd than for the low-risk herd. For the diagnostic examinations, 2 different populations of pigs were chosen. In Scenarios 4-6 (Fig. 11.6), diagnostic examinations were performed in pigs showing clinical signs of lameness, whereas in Scenarios 7-9 (Fig. 11.7), diagnostic examinations were performed in all (50) pigs. Selecting all pigs for diagnostic examination gave the best estimate of the most likely cause of leg disorders in a herd.
Table 6.3: Overview of the 10 scenarios used to illustrate the behaviour of the object-oriented Bayesian network model

<table>
<thead>
<tr>
<th>Scenario</th>
<th>Herd evidence</th>
<th>Obslame</th>
<th>Pig evidence</th>
<th>Results from diagnostic test</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>No</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>Yes</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>Yes</td>
<td>10 yes 40 no</td>
<td>-</td>
<td>Lame pigs (n = 10) examined clinically. Clinical signs of Myco in lame pigs. No clinical signs of other leg disorders.</td>
</tr>
<tr>
<td>4</td>
<td>Yes</td>
<td>10 yes 40 no</td>
<td>-</td>
<td>Lame pigs (n = 10) examined clinically, pathologically and bacteriologically. Evidence of Myco in all tests for lame pigs. No evidence of other leg disorders.</td>
</tr>
<tr>
<td>5</td>
<td>Yes</td>
<td>10 yes 40 no</td>
<td>-</td>
<td>Lame pigs (n = 10) examined clinically, pathologically and bacteriologically. Evidence of Myco in all tests for lame pigs. No evidence of other leg disorders.</td>
</tr>
<tr>
<td>6</td>
<td>Yes</td>
<td>10 yes 40 no</td>
<td>-</td>
<td>Lame pigs (n = 10) examined clinically, pathologically and bacteriologically. Evidence of Myco in all tests for lame pigs. No evidence of other leg disorders.</td>
</tr>
<tr>
<td>7</td>
<td>Yes</td>
<td>10 yes 40 no</td>
<td>-</td>
<td>All pigs (n = 50) examined clinically. Clinical signs of Myco in lame pigs. No clinical signs of other leg disorders.</td>
</tr>
<tr>
<td>8</td>
<td>Yes</td>
<td>10 yes 40 no</td>
<td>-</td>
<td>All pigs (n = 50) examined clinically and pathologically. Evidence of Myco in both tests for lame pigs. No evidence of other leg disorders.</td>
</tr>
<tr>
<td>9</td>
<td>Yes</td>
<td>10 yes 40 no</td>
<td>-</td>
<td>All pigs (n = 50) examined clinically, pathologically and bacteriologically. Evidence of Myco in all tests for lame pigs. No evidence of other leg disorders.</td>
</tr>
<tr>
<td>10</td>
<td>Yes</td>
<td>10 yes 40 no</td>
<td>-</td>
<td>All pigs (n = 50) examined with perfect tests where the SE and SP equal 1.</td>
</tr>
</tbody>
</table>
CHAPTER 7
GENERAL DISCUSSION AND CONCLUSION

The thesis has investigated different aspects of AHE in the finisher pig production. The focus has been on investigating the effect of disease on performance in individual pigs and on constructing a model that can estimate a risk index for 3 cause-categories of leg disorders in finisher herds. This chapter will discuss the 3 objectives based on the framework for economic analysis in AHE. Secondly, the results from Objectives 1 and 2 will be discussed in relation to the existing literature and together with the data used for the data analyses. Moreover, the chapter will discuss the construction and use of the OOBN model (Objective 3). Finally, the conclusions of the thesis are summarized.

7.1 Overall framework

The framework for economic analyses in AHE illustrated by [McInerney, 2001], serves as a suitable background for the discussion of the 3 objectives in this thesis (Fig. 7.1). Basically, this framework consists of 2 parts: a biological part that focus on the link between risk factors, disease and disease effects in a production herd, and an economic part that focus on the link between the disease effects and the farmer’s outcome. In regard to the framework for economic analysis, Objective 1 has focused on the link between disease and disease effects, whereas Objective 2 has focused both on the relation between disease and disease effects as well as the farmer’s outcome.

Objectives 1 and 2 investigated the effect of different disease indicators on the performance in individual pigs. This was done in order to provide accurate estimates of the effects of disease in pigs. For disease where no interactions among pigs are assumed, the effect of disease on animal level can be used to calculate the
effect of disease at herd level. The construction of models in AHE requires information regarding disease and, in particular, how disease affects the performance in the herd. Information regarding the effect of disease in individual animals can, therefore, serve as input into future decision support systems in AHE. Hence, it can be said that Objectives 1 and 2 add information to both the biological and economic part of the framework in AHE.

Leg disorder in finishers is a disease complex comprising different disorders that can cause clinical signs of lameness. The eventual choice of a strategy against leg disorders must rely on available information about the underlying cause in a herd. Implementing a control strategy against leg disorders, without knowing the cause of the problem, may cause losses in regard to production due to no effect of the strategy, and due to the unnecessary expenditures of the control strategy. Therefore, it is important to know the most likely cause of leg disorders in a particular herd, and furthermore, to know the level of information required in order to estimate the most likely cause of leg disorders. In this thesis, an OOBN model was developed that estimates risk indexes for 3 major cause-categories of leg disorders using information from the herd and from individual pigs. Based on the framework for economic analysis in AHE (Fig. 7.1), Objective 3 has focused on the biological part of the framework in AHE, and in particular, focused on increasing the certainty about the cause of disease, which is elementary for performing economic analyses at herd level.

7.2 Objectives 1 and 2

7.2.1 Results from Objectives 1 and 2

The results of Objective 1 showed that 1-3 lameness treatments and more than 5 lameness treatments were significantly associated with a reduction in the MDWG.
Hence, 1-3 lameness treatments caused a reduction of 3%, and more than 5 lameness treatments caused a reduction of 4% compared to boars with no lameness treatments. Other studies have also found a negative effect of lameness on MDWG (van Dijk et al. 1984; Zoric et al. 2003; Johansen et al. 2004). However, no effect of lameness treatments on the FCR was found. This agrees with Huang et al. (1995) who did not find any significant correlation between leg weakness and FCR. Contrary, treatment for other diseases than lameness reduced the MDWG by 6% and increased the FCR by 2% compared to boars without treatment for other diseases. As the prevalence of boars with lameness treatments and treatments for other health disorders was 4% and 65%, respectively, it can be concluded that lameness contributed to a minor part of the total losses caused by disease (Manuscript 1).

Oral and parenteral treatments as well as pathological findings caused a significant reduction in the PM. Hence, oral treatments caused a reduction in the PM of 7% whereas parenteral treatments caused a reduction in the PM of 17%. A number of studies have investigated the effect of disease (e.g. pneumonia and gastrointestinal disorders) on productivity (Straw et al. 1989; McOrist et al. 1997); however, no studies have investigated the effect of disease on PM including several disease indicators concomitantly. The lower effect of oral treatments can be explained by the fact that some boars with records of oral treatments were treated without showing clinical signs of disease. Other studies have shown a positive effect of antibiotics on the performance of pigs (Partanen et al. 2002). Hence, antibiotics in the drinking water could have improved the productivity of the boars.

The effect of pathological findings on the PM varied between breeds. This can be due to genetic variation for resistance to clinical and subclinical diseases among different breeds (Henryon et al. 2001). The effect of oral and parenteral treatments and pathological findings on the PM was sensitive in regard to the price per kg carcass weight. This can be explained by differences in the slaughter weight among boars with and without disease. Also the medicine price influenced the effect of oral and parenteral treatments, where only diseased boars were treated (Manuscript 2).

### 7.2.2 Performance variables for the Objectives 1 and 2

In Objectives 1 and 2, different performance variables were used to investigate the influence of disease in individual pigs. Objective 1 investigated the effect of disease on the production variables: MDWG and FCR. These production variables are known to have economical importance for the pig enterprise (Fig. 2.2). Increase in the mortality rate among pigs is another important economic consequence of disease; however, this variable was not included in Objective 1.

The effect of disease on FCR and mortality will have a direct effect on the PM for the herd. Hence, deterioration in the FCR in finishers will increase the expenditures for feed, which have a direct effect on the herd profitability. Likewise an increase in the mortality rate will affect the total revenue and hence the profitability for the pig producer. On the other hand, the effect of MDWG on the PM will de-
7.2 Objectives 1 and 2

Objectives 1 and 2 depend on the production strategy used in the herd. In herds that deliver finishers to the slaughterhouse based on a specific target slaughter weight, which is the case in a continuous production system, the effect of a reduction in the MDWG will result in an increase in the number of feeding days. Consequently, this will increase the costs of feed for the livestock producer, and hence, affect the profitability. Contrary, in herds that deliver pigs at specific time intervals, which is the case in a sectioned production system, the effect of reduced MDWG among finishers will result in a number of pigs that do not reach the target slaughter weight within the desired time interval. This will have a negative influence on the total revenue and hence on the herd profitability. Information regarding the production strategy (i.e. continuous or sectioned production), in addition to the pig producer’s ability and willingness to cope with reduced growth among the pigs, should be taken into account when evaluating the influence of a reduced MDWG caused by disease on herd profitability.

Yet, Objective 2 investigated the influence of disease on the PM directly. In the calculation of the PM, the total revenue and the variable costs of medicine, feed and 30 kg piglet were included. There might have been other variable costs of importance, such as transportation costs and labour costs (Figure 2.2), however, due to lack of information, these variables were not taken into account. The prices used in the calculation of the PM were based on prices at a specific point in time. As prices can vary depending on time and site, sensitivity analyses were performed for all prices used in the calculation of the PM (Manuscript 2).

7.2.3 Data used for Objectives 1 and 2

Data used for Objectives 1 and 2 originated from a Danish boar test station. As information on both disease and production was available on animal level, data from the test station was considered to be suitable for the purpose of Objectives 1 and 2. However, certain limitations exist when using data from the boar test station to draw conclusions on production herds in general.

First of all, certain routines and strategies at the boar test station differed compared to the finisher pig production in general. The technical staff observed the boars daily and treated all boars with clinical signs of disease. As the boars at the test station were valuable animals, it is likely that the threshold for treating the boars was lower compared to the threshold for treating pigs in the production herds. In a study by Petersen et al. (2004), variation among individual observers performing clinical evaluations of animals was found. Hence, the lack of standardization and validation of the technical staff may have contributed to misclassification bias in the studies. The pens in the station had low stocking densities and the feed was provided in individual feeders. The production system could have reduced the competition among the purebred boars and, consequently, improved the production of the boars.

Secondly, all disease indicators used in Objectives 1 and 2 were based on treatment records. It was not possible to obtain additional information regarding the
disease, e.g. the severity of the clinical signs or results from other diagnostic tests. It is likely that the effect of treatment could have a positive effect on the productivity of individual boars, which consequently would have reduced the total effect of disease. Contrary, it is possible that the low threshold for treating boars at the test station would have induced more expenses to medicine compared to the finisher production in general. Conclusively, the results from Objectives 1 and 2 are likely to be conservative compared to the effect of disease that is found in typical Danish production herds.

7.3 Objective 3

In current models in AHE, the ways and strategies of obtaining information regarding the disease level in a herd are often not considered. Usually, models in AHE estimate the effect of different control strategies against a particular disease assuming that the disease level in a herd is known. However, to plan and conduct an effective control strategy against endemic disease, it is necessary to obtain information about the disease level in the herd. The purpose of Objective 3 was to develop a mathematical model that can estimate the most likely cause of leg disorders in a finisher herd. Control strategies against leg disorders will depend on the underlying cause-category. Hence, a common intervention against infectious leg disorders will be curative treatment of individual pigs. This can be classified as an operational intervention with a short time horizon. Secondly, re-constructing the floors in pens can be considered as a strategic intervention against physical leg disorders (e.g. injuries to the claw) which have a long term horizon (Saatkamp et al., 2001). Finally, changing the feeding strategy, and hence the growth rate, can be an intervention against osteochondrosis. This intervention can be considered as tactical with a mid-term time horizon. As interventions against leg disorders can have different costs and time horizons, it is essential to know the most likely cause of leg disorders in order to select and implement an effective control strategy.

7.3.1 OOBN model

An Bayesian network (BN) model was used for the purpose of Objective 3. The model estimated the risk of 3 cause-categories of leg disorders in a finisher herd at a specific point in time, which made a static BN model suitable. In general, BNs express the relations between causal links as conditional probability distributions (Jensen, 2001). Therefore, it is possible to combine information in the BN model and represent the uncertainty of the variables modeled. Yet, another characteristic of BN model is the fact that variables can be estimated, which are not observable in real life or very costly to observe (Jensen, 2001). Hence, the estimation of these variables is possible even if evidence of one or more information node is missing. Due to these characteristics, a BN model was found suitable for the purpose of Objective 3.
Previously, a BN model has been constructed for *Mycoplasma hyopnemoniae* in finisher herds. Here, information at herd level was used as input to the model (Otto and Kristensen [2004]). The model in Objective 3 used information from both the herd and individual pigs for the estimation of the most likely cause of leg disorders at herd level. As information to the model originated from 2 different levels, the Bayesian network was modeled using an object-oriented structure. This eased the specification of the model when several pigs were included in the model concomitantly. The OOBN model can be considered to be a suitable method of combining information from 2 different levels when estimating the cause of leg disorders. Hence, evidence about the individual herd (e.g. the production policy and the pen construction) will be available prior to a herd visit. This evidence can provide information about the cause of leg disorders in the herd. In order to increase the level of information about leg disorder it can be necessary to obtain information about individual pigs. This requires observation of pigs, and probably selection of a number of pigs for further diagnostic examinations. Individual pigs can be examined in different ways: observed outside the pens, examined clinically (inspection and palpation) inside the pen, and finally pigs can be euthanized, and examined pathologically and bacteriologically. The results from diagnostic tests will add information to the cause of leg disorder at herd level. Hence, each additional diagnostic test will potentially increase the certainty about the cause-category. However, diagnostic tests will pose extra expenditures for the pig producer. As an example, the price of a pathological and bacteriological examination of one pig is 75 € and 44 €, respectively (Danish Pig Production [2008]). Therefore, it is crucial to know the number of diagnostic tests to carry out in order to estimate the cause of leg disorder with a desired certainty.

The OOBN model presented in this thesis can be used as a help to decide on the level of information that is needed in an individual herd, in order to estimate the most likely cause-category of leg disorders under uncertainty. The model will be able to help answering the following questions: Is it necessary to investigate individual pigs in the herd? Which diagnostic test(s) should be performed? How should pigs for diagnostic examination be selected? How many pigs should be selected for diagnostic examination? The level of information (e.g. the number of diagnostic tests) required, in order to increase the certainty of the disease level in a herd should indeed be taken into account in future analysis within AHE.

### 7.3.2 Results from the model

The impact of different levels of information on the ability to distinguish the most likely cause of leg disorders at herd level was investigated. Hence, 10 different scenarios of information were compared in 2 fictitious herds with characteristics indicating low-risk and high-risk for infectious leg disorders. Fifty randomly selected pigs were investigated for lameness (yes/no) outside the pen. In both herds, 20% of the selected pigs showed clinical signs of lameness. All lame pigs were positive for infectious arthritis caused by *Mycoplasma hyosynoviae* and negative
for any other leg disorder. In the high-risk herd, information regarding the herd characteristics as well as information regarding lameness in 50 randomly selected pigs (observed outside the pen) was sufficient in order to estimate the most likely cause-category. However, in the low-risk herd, it was necessary to perform, at least a thorough clinical examination of lame pigs (inside the pen) in order to estimate the cause of leg disorders. As information from clinical examinations of individual pigs changed the knowledge of the cause-category in the low-risk herd, there was more economic benefit in performing diagnostic tests of individual pigs in the low-risk herd compared to the high-risk herd. However, a quantification of the economic benefit was not carried out in this thesis.

7.3.3 Use of expert opinions and model validation

The disorders included in the OOBN model do not represent a complete list of leg disorders in finishers. However, it is believed that the leg disorders included in the model are the most common disorders seen in the finisher pig production. Expert opinions were primarily used for the elicitation of the probabilities due to limited quantitative information regarding the causal links in the literature. A method described by van der Gaag et al. (2001) was used for the elicitation of the probabilities. To ensure that the individual experts had a thorough understanding of the problem to be evaluated, the problem at hand was discussed with the experts before and after the elicitation. When more than one expert elicited the same question, the resulting probabilities used in the model were an average of the individual elicitations (van der Gaag et al., 2001). However, the presence of disagreement among the individual experts was not taken into account in the generation of the resulting probabilities. In the elicitation of the probabilities a combination of qualitative and quantitative methods were used, which may have jeopardized the validity of the resulting probabilities for the model. In a previous study, that used expert panels for the quantification of variables, the individual assessments were weighted according to the expertise of the experts (van der Fels-Klerx et al., 2002). Weighting the individual experts according to their expertise in this study may have yield more valid estimates for the model.

As more information, from either data analyses or expert opinions becomes available in the future, the Bayesian framework offers a readily applied approach to update the probabilities in the model. Before the model can be applied in finisher herds, it is necessary to explore the relationship between the model output and every single input parameter as described by Kjærulff and van der Gaag (2000). Moreover, it is necessary to perform a thorough validation of the model in real finisher herds.
7.4 Conclusions of the thesis

A significant negative effect of lameness on the MDWG among boars at a test station was found. However, no effect of lameness on the FCR was shown. Boars with records of non-lameness treatments had a significant negative effect on both MDWG and FCR. Disease indicators, measured as oral and parenteral treatments, had a significant negative effect on the PM of individual boars. Parenteral treatments were associated with a larger reduction in the PM than oral treatments. Pathological findings at slaughter were also associated with a negative effect on the PM, however, this effect was influenced by the breed of the boar.

A model was constructed that estimated risk indexes for 3 major cause-categories of leg disorders in finisher herds. The model can be used to investigate the benefit of performing diagnostic tests in finisher herds. Hence, the results from 2 fictitious herds showed that the value of performing diagnostic test would depend on the individual herd. Therefore, it is necessary to include calculations of diagnostic tests in economic models. Sensitivity analyses and validation of the model in finisher herds are needed before the model can be applied in real finisher herds.

References


REFERENCES


From the information that can be obtained from the OOBN model, it may be possible in the future, to construct a decision support system that can estimate the effect of different control strategies against leg disorders in finisher herds. However, before a decision support model can be constructed, it is necessary to obtain information regarding the effect of leg disorders on performance and to identify all potential control strategies against leg disorders as well as their costs. Indeed, a great challenge is to obtain valid information about the effect of different leg disorders on the production variables (e.g. MDWG, FCR and mortality). Objective 1 investigated the effect of the number of lameness treatments on the MDWG and FCR. As no further diagnostic information regarding lameness was available at the boar test station, lameness was handled as an integrated disease entity in Objective 1. However, the effect of lameness on the productivity will most likely differ depending on the underlying cause. E.g. fractures and infectious arthritis can affect the individual animal systemically and cause a negative effect on the performance. On the contrary, leg disorders such as osteochondrosis and lesions to the claw are often localized lesions that may only affect the performance slightly. Hence, before it is possible to construct a decision support system for leg disorders in finisher herds, it is necessary to carry out further data analyses in order to obtain valid information regarding the effect of different leg disorders on the productivity of pigs.

From the conclusions of this thesis, it can be argued that the financial consequences of leg disorders in the finisher pig production are minor compared to other endemic disease complexes. Studies in AHE usually focus on endemic diseases in production herds that cause a potential negative effect on the productivity (MDWG, FCR and mortality), and hence affect the herd profitability considerably. The purpose of most models in AHE is, therefore, to help the livestock producer in selecting an effective control strategy against disease that will maximize the herd
profitability. For leg disorders in the finisher pig production, it might be shown that control strategies against leg disorders are more expensive than the benefit gained from reducing the production losses caused by leg disorders. As an example, reconstructing the floor in pens as an intervention for reducing the prevalence of claw lesions might not favour the profitability for the pig producer. It would, therefore, be difficult to encourage the pig producer to plan and conduct strategies against leg disorders based on solely financial grounds. However, the pig producer may have other preferences such as improving animal welfare, good working conditions and extra leisure time that should be considered (Kristensen et al., 2007). Each of these preferences may influence the overall utility of the livestock producer.

When selecting a control strategy against endemic diseases, it is basically the goal to maximize the utility of the livestock producer. The motivation for implementing control strategies against leg disorders could, therefore, be based on a wish from the pig producers to fulfill his preference for good animal welfare in the herd. In the future, it may be possible to integrate different preferences in the overall framework for economic analysis in AHE. This will eventually enable the livestock producer to make a decision regarding disease, e.g. leg disorders, based on different preferences that altogether will maximize his utility value.

References

CHAPTER 9

THE EFFECT OF LAMENESS TREATMENTS AND TREATMENTS FOR OTHER HEALTH DISORDERS ON THE WEIGHT GAIN AND FEED CONVERSION IN BOARS AT A DANISH TEST STATION

Tina Birk Jensen, Niels Peter Baadsgaard, Hans Houe, Nils Toft, Søren Østergaard

Livestock Science 112, 34-42

Abstract: Lameness in finishers can cause economic losses to farmers and lameness affects the welfare of pigs. In order to study the economic losses, we investigated the effect of lameness on productivity, measured as the mean daily weight gain (MDWG) and the feed conversion ratio (FCR), and evaluated the importance of these effects. The study design was observational of a cross sectional type and data was collected from a Danish boar test station during February 2002 and December 2004. A total of 10,473 boars were included in the study. We adapted a quantitative interpretation of lameness, using the number of lameness

1Reprinted with permission from Elsevier Science
treatments of the individual animal, and generated the new variable: “lameness
treatments”. All treatments other than lameness were recoded as “non-lameness
treatments”. Multivariable hierarchical analyses were performed to assess the as-
sociation between the risk factors: number of lameness treatments, records of non-
lameness treatments (yes/no), breed (Duroc, Hampshire, Landrace, Yorkshire) and
weight at 4 weeks with each of the outcome variables: MDWG and FCR. In order
to improve the assumption of normality, we used a quadratic transformed MDWG
and an inverse transformed FCR in the analyses. Lameness treatments had a signifi-
cant effect on the transformed MDWG ($p < 0.0001$). Boars with one to three lame-
teness treatments had a significant reduction in the MDWG, which corresponded to a
reduction of 27 gram per day. Boars with four and five lameness treatments did not
have a significant reduction in the MDWG. More than five lameness treatments
causd the largest reduction in the MDWG corresponding to 40 gram per day.
There was no significant association between lameness treatments and the trans-
formed FCR ($p = 0.14$). Records of non-lameness treatments, breed and weight at
4 weeks were all significantly associated with the transformed MDWG and FCR.
Boars with records of non-lameness treatments had a reduction in the MDWG of
56 gram per day and an increase in the FCR of 0.04 feed units per kilogram live
weight. At the test station, the prevalence of boars with records of lameness treat-
ments was 4% whereas the prevalence of records of non-lameness treatments was
65%.

**Keywords**: Boars; Lameness treatments; Non-lameness treatments; Weight
gain; Feed conversion ration

### 9.1 Introduction

Lameness in finishers can have a negative influence on the farmer’s economy. In
addition, lameness is known to have a negative impact on the welfare of pigs
(Busch et al., 2003). The economic losses are caused by increased work load, due
to physical handling of pigs, medical treatment costs and reduced productivity in
the pigs. The consequences of reduced productivity in slaughter pig herds are often
split into decreasing returns due to a reduction in the daily weight gain of finishers
and increased costs of feed as a result of poorer feed conversion (Ridgeon, 1988).
Information on the effect of lameness on the mean daily weight gain (MDWG)
and the feed conversion ratio (FCR) is, therefore, important when evaluating the
economic consequences of lameness. The effect of lameness on productivity - and
hence economy, has been investigated in other studies. In a study by Christensen
et al. (1994), lameness was the third most frequent cause of treatment and lameness
represented 11% of all treatments given to finishers. The effect of lameness on the
weight gain in piglets, has previously been examined (Zoric et al., 2003; Johansen
et al., 2004). Johansen et al. (2004) investigated risk factors for low daily weight
gain in piglets. The MDWG was reduced by 38 gram per day among piglets treated
for arthritis compared to clinically healthy pigs. In another study of fattening pigs, leg disorders reduced the MDWG by 67 gram per day (van Dijk et al., 1984). Woltmann et al. (1995) evaluated the gait soundness in barrows and gilts at the weight of approximately 100 kg. The gait of each pig was scored on a scale from 1, representing extreme leg weakness, to 8, representing superior leg structure. For each unit increase in the soundness score there was an increase in the finishing phase daily weight gain of 29 gram per day. On the contrary, Nielsen et al. (2001) did not find a significant reduction in the MDWG among 3-5 month old pigs with clinical signs of lameness. The feed intake is rarely measured in individual pigs, and hence few studies have addressed the effect of lameness on the FCR. In a study by Huang et al. (1995), no significant correlation was found between leg weakness and the FCR.

The causes of lameness have been investigated in previous studies. Infectious arthritis can lead to clinical signs of lameness, and Mycoplasma hyosynoviae, Erysipelothrix rhusiopathiae and Streptococcus spp. are most frequently isolated in affected animals (Friis et al., 1992; Hariharan et al., 1992; Buttenschon et al., 1995; Smith and Morgan, 1997; Nielsen et al., 2001). Osteochondrosis is a non-infectious degenerative condition of the cartilage and the bone, and osteochondrosis can cause lameness in finishers (Grøndalen, 1974; Nakano et al., 1987). Furthermore, clinical signs of lameness can be caused by traumatic limb and claw injuries (Gjein and Larssen, 1995; Mouttotou and Green, 1999).

The objective of this study was to adapt a quantitative interpretation of lameness using the number of lameness treatments and to assess the impact and importance of lameness in finishers on productivity, measured by MDWG and FCR. The impact was assessed by means of a multivariable model including the explanatory variables: non-lameness treatments, breed and weight at 4 weeks.

9.2 Materials and methods

9.2.1 Study design

From February 2002 to December 2004, data was collected from a Danish boar test station owned and run by the Danish Pig Production / Danish Meat Association. The study design was observational of a cross sectional type.

9.2.2 Description of the production system

The boar test station received four-week old boars of the breeds: Duroc, Hampshire, Landrace and Yorkshire from Danish breeding herds. All piglets that arrived at the test station in the same week were allocated in one section in the early weaning unit. In each of the 8 sections, there were 4 pens with fully slatted floors. Initially, 28 piglets of the same breed were placed together in one pen. After seven days, the technical staff removed 14 piglets with the lowest weight from each pen.
These piglets were allocated to a pen in another section. Each piglet was individually identified with an ear tag at arrival. At 5 weeks of age, the piglets were vaccinated against *Mycoplasma hyopneumoniae* (Stellamune®) and one week later against *Actinobacillus pleuropneumoniae* (Haemo Shield®). After approximately 6 weeks in the early weaning section, boars were moved to the finisher unit. Boars remained in the same batch in the finisher unit. This unit comprised of 16 sections with eight pens. These pens had solid concrete floors with bedding, and feed and water was given ad libitum. Feed was given through a single feeder, and time, duration and feed consumption was recorded for each individual visit of the boar. The main components of the feed were wheat, barley and soybean, and consisted of 16.2% crude protein. All boars were inspected daily by the technical staff and boars with clinical signs of disease were treated medically. The treatments and the related diagnoses were recorded according to a pre-specified coding system.

The weight of each boar was recorded by the technical staff at arrival, and at regular intervals during the fattening period. When the boars reached slaughter weight (at approximately 100 kilograms) a selection index was calculated for each boar based on the breeding values for different traits (e.g. MDWG, FCR, lean meat percentage, litter size and longevity). Twenty percent of the boars were selected for collection of semen for artificial insemination, and the remaining 80% were slaughtered.

### 9.2.3 The data

Boars that had been in the finisher unit for a period of at least 70 days were included in the present study. Individual treatments were given to boars that showed clinical signs of arthritis (e.g. deviation from the normal gait and posture and/or swelling of joints). These boars were injected with lincomycin, penicillin or streptomycin and given the treatment code: lameness. However, the aetiology of the lameness was not investigated further, and in the records, there were no distinctions between lameness occurring in one or more extremities. Based on the number of lameness treatments, boars in the finisher unit were divided into five groups: One to three lameness treatments (*n* = 163), four lameness treatments (*n* = 55), five lameness treatments (*n* = 156), more than five lameness treatments (*n* = 59) and no lameness treatments (*n* = 10,040).

During the study period, there was a high prevalence of boars with treatment records for diseases other than lameness. The most prevalent causes for treatment were pneumonia (32%), diarrhoea (28%), lethargy (17%) and unspecified treatments (13%). Collective medication was administered in the drinking water against diarrhoea, pneumonia and lethargy in 84, 44 and 22% of the times, respectively. In these cases, all boars in a pen were given a treatment code. As a consequence, records of diarrhoea, pneumonia and lethargy represented treatment against a large variety of clinical signs, and hence, the diagnostic criteria were more uncertain than for lameness. Moreover, there was a low prevalence of boars treated individually for: meningitis (0.6%), tail bite (0.6%) abscesses (0.4%) and eczema (0.2%).
We considered the overall treatment against non-lameness diseases as a covariate in this study. Hence, all treatment records other than lameness were recoded as “non-lameness treatments”. Boars were divided into two groups according to whether or not they had records of “non-lameness treatments” during the fattening period. The MDWG measures the live weight gain per day and the MDWG for each boar was calculated using the following formula:

$$\text{MDWG} = \frac{W_{\text{End}} - W_{\text{Start}}}{D} \left( \frac{\text{Kg}}{\text{day}} \right)$$

(9.1)

$W_{\text{Start}}$ is the weight of the boar when entering the finisher unit (kilograms), at ten weeks of age, $W_{\text{End}}$ is the weight at the time for leaving the finisher unit (kilograms) and $D$ is the number of days in the finisher unit.

The FCR measures the number of feed units (FU) used per kilogram live weight and the FCR for each boar was calculated using the following formula:

$$\text{FCR} = C \times \left( \frac{\text{Feed}}{W_{\text{End}} - W_{\text{Start}}} \right) \left( \frac{\text{FU}}{\text{kg}} \right)$$

(9.2)

Feed is the total amount of feed intake (kilograms) in the period from entering to leaving the finisher unit, and $C$ is the FU per kilogram of feed ($C = 1.05$).

### 9.2.4 Data control

One hundred and thirteen boars died during the fattening period and 1484 boars had missing values on the feed intake. These boars were, consequently, excluded from the study. Frequency distributions of the categorical variables: lameness treatments, records of non-lameness treatments and breed were used to identify potentially illegal values. The continuous variables: weight at four weeks, FCR and MDWG were checked for extreme values. Except for 3 boars which had biological extreme values of the FCR (>12 \(\text{FU/Kg}\)), the 95% confidence interval of the FCR was in the interval [2.35; 2.36]. It was decided to exclude these 3 boars from further analyses.

### 9.2.5 Statistical analysis

Two multivariable hierarchical models were constructed to assess the association between the risk factors: lameness treatments, records of non-lameness treatments, weight at four weeks and breed with each of the outcome variables: FCR and MDWG. The statistical software system SAS version 9.1 (Proc mixed) was used (SAS 2002). Variance heterogeneity was expected among the different breeds and the different levels of lameness treatments and records of non-lameness treatments.

Thus, the analyses were performed using a log-linear variance model, allowing for dispersion effects of residual variances between the categorical variables. To allow for the variation between herds of origin and between batches at the test
Table 9.1: Descriptive statistics of the categorical risk factors included in the study of the mean daily weight gain (Kg/Day)

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>Mean daily weight gain Mean (Kg/Day)</th>
<th>S.D.</th>
<th>S.E.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>10,473</td>
<td>0.884</td>
<td>0.126</td>
<td>0.001</td>
</tr>
<tr>
<td>Lameness treatments</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-3</td>
<td>163</td>
<td>0.839</td>
<td>0.119</td>
<td>0.009</td>
</tr>
<tr>
<td>4</td>
<td>55</td>
<td>0.846</td>
<td>0.126</td>
<td>0.017</td>
</tr>
<tr>
<td>5</td>
<td>156</td>
<td>0.847</td>
<td>0.118</td>
<td>0.009</td>
</tr>
<tr>
<td>&gt;5</td>
<td>59</td>
<td>0.820</td>
<td>0.124</td>
<td>0.016</td>
</tr>
<tr>
<td>No treatments</td>
<td>10,040</td>
<td>0.886</td>
<td>0.126</td>
<td>0.001</td>
</tr>
<tr>
<td>Non-lameness treatments</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>yes</td>
<td>6792</td>
<td>0.868</td>
<td>0.133</td>
<td>0.002</td>
</tr>
<tr>
<td>No</td>
<td>3681</td>
<td>0.914</td>
<td>0.106</td>
<td>0.002</td>
</tr>
<tr>
<td>Breed</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duroc</td>
<td>3258</td>
<td>0.926</td>
<td>0.113</td>
<td>0.002</td>
</tr>
<tr>
<td>Hampshire</td>
<td>1757</td>
<td>0.790</td>
<td>0.131</td>
<td>0.003</td>
</tr>
<tr>
<td>Landrace</td>
<td>2637</td>
<td>0.898</td>
<td>0.113</td>
<td>0.002</td>
</tr>
<tr>
<td>Yorkshire</td>
<td>2821</td>
<td>0.880</td>
<td>0.117</td>
<td>0.002</td>
</tr>
</tbody>
</table>

a Standard deviation  
b Standard error of the mean

station, we included herd of origin and batch as random effects in the models. The initial models included all risk factors and the biological plausible interactions of fixed effects as well as local dispersion effects for all levels of fixed effects. To reduce the fixed effects a backwards elimination strategy, using a significance level of 5% to exclude factors, was applied (Ersbøll et al., 2004). Akaike’s Information Criterion (AIC) was used to reduce the dispersion and random effects. Confounding was assessed by comparing the p-values and the parameter estimates when the models were reduced from having all fixed effects in the models at the same time, to models where one fixed variable was excluded. A change in the parameter estimates of more than 20% was used as an indication of confounding. The initial model for MDWG and FCR was:

\[ Y_{ijklmn} = \mu + A_i + B_j + C_k + D_{ijkl} + AB_{ij} + AC_{ik} + BC_{jk} + v_l + \omega_m + \epsilon_{ijklmn} \]

\[ Y_{ijklmn} \] was the MDWG or the FCR for boar n in the i-th lameness treatment, with the j-th records of non-lameness treatment in the k-th breed from the l-th herd of origin and placed in the m-th batch. The \( \mu \) represented the intercept, \( A_i \) was the fixed effect of the i-th lameness treatment (i: 1-3, 4, 5, >5, no treatments), \( B_j \) was the fixed effect of the j-th records of non-lameness treatment (j: yes, no) and \( C_k \) was the fixed effect of the k-th breed (k: Duroc, Hampshire, Landrace, Yorkshire). \( D_{ijkl} \) was the estimate of the weight at four weeks for the i-th lameness treatment, the j-th records of non-lameness treatment and the k-th breed. \( AB_{ij} \) was the interaction between the i-th lameness treatment and the j-th records of non-
9.2 Materials and methods

lameness treatment. $AC_{ik}$ was the interaction between the $i$th lameness treatment and the $k$th breed and $BC_{jk}$ was the interaction between the $i$th records of non-lameness treatment and the $k$th breed. The random effect of herd of origin was $\nu_l \sim N(0, \sigma_\text{Herd}^2)$, and the random effect of batch was $\omega_m \sim N(0, \sigma_\text{Batch}^2)$. The residuals, $\epsilon_{ijklmn} \sim N(0, \sigma_e^2 \exp(U\delta))$, were Normal distributed within each combination of lameness treatments, records of non-lameness treatments and breed with $\sigma_e^2$ as the common intercept term of the residual variance, $U$ as the design matrix reflecting the combination of the categorical variables and $\delta$ as an 8-dimensional vector of estimated parameters.

Residual plots, and plots of residuals and predicted values, were used to evaluate the model assumptions for each analysis. Box-Cox analyses were performed to explore the benefit of a Box-Cox transformation of the outcomes on the model fit (Dohoo et al., 2003). Finally, the influence of extreme values was assessed by running non-parametric analyses and comparing the results from the parametric and non-parametric analyses.

Table 9.2: Descriptive statistics of the categorical risk factors included in the study of the feed conversion ratio ($\frac{FU}{Kg}$).

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>Feed conversion ratio Mean ($\frac{FU}{Kg}$)</th>
<th>S.D. $^a$</th>
<th>S.E. $^b$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>10,473</td>
<td>2.357</td>
<td>0.213</td>
<td>0.002</td>
</tr>
<tr>
<td>Lameness treatments</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-3</td>
<td>163</td>
<td>2.386</td>
<td>0.191</td>
<td>0.015</td>
</tr>
<tr>
<td>4</td>
<td>55</td>
<td>2.358</td>
<td>0.226</td>
<td>0.030</td>
</tr>
<tr>
<td>5</td>
<td>156</td>
<td>2.356</td>
<td>0.204</td>
<td>0.016</td>
</tr>
<tr>
<td>&gt;5</td>
<td>59</td>
<td>2.390</td>
<td>0.201</td>
<td>0.026</td>
</tr>
<tr>
<td>No treatments</td>
<td>10,040</td>
<td>2.356</td>
<td>0.214</td>
<td>0.002</td>
</tr>
<tr>
<td>Non-lameness treatments</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>yes</td>
<td>6792</td>
<td>2.371</td>
<td>0.229</td>
<td>0.003</td>
</tr>
<tr>
<td>No</td>
<td>3681</td>
<td>2.330</td>
<td>0.117</td>
<td>0.003</td>
</tr>
<tr>
<td>Breed</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duroc</td>
<td>3258</td>
<td>2.325</td>
<td>0.205</td>
<td>0.004</td>
</tr>
<tr>
<td>Hampshire</td>
<td>1757</td>
<td>2.424</td>
<td>0.214</td>
<td>0.005</td>
</tr>
<tr>
<td>Landrace</td>
<td>2637</td>
<td>2.403</td>
<td>0.236</td>
<td>0.005</td>
</tr>
<tr>
<td>Yorkshire</td>
<td>2821</td>
<td>2.308</td>
<td>0.117</td>
<td>0.003</td>
</tr>
</tbody>
</table>

$^a$Standard deviation  
$^b$Standard error of the mean
9.3 Results

9.3.1 Descriptive results

A total of 10,473 boars from 84 Danish breeding herds had records on production and disease and among these, 433 boars (4%) had records of lameness treatments and 6792 boars (65%) had records of non-lameness treatments during the fattening period. Of boars that were treated for lameness, 293 were treated for both lameness and non-lameness diseases (data not shown). The boars were kept in 1013 individual batches at the test station. The mean weight at four weeks was 8.45 kg (S.D. = 1.28). Descriptive statistics of the categorical variables included in the initial analyses are in Tables 9.1 and 9.2.

9.3.2 Mean daily weight gain

The result of the Box-Cox analysis showed that the quadratic transformation of the MDWG improved the normality of the residuals considerably and, therefore, we used this transformation in the analysis of the MDWG. The reduced model included lameness treatments, records of non-lameness treatments, breed and weight at four weeks at the 5% significance level (Table 9.3). No significant effects were found for the interaction terms and confounding was not present. Based on AIC, there was no benefit of reducing the initial model with the dispersion effects. In the pair wise comparison between the 5 lameness groups, boars with one to three lameness treatments had a significantly lower MDWG compared to boars with no lameness treatments. However, the MDWG of boars with four and five lameness treatments did not differ significantly from boars with no lameness treatments. The lowest MDWG was seen among boars with more than five lameness treatments and the MDWG was significantly lower than boars with five lameness treatments and boars with no lameness treatments. Boars with records of non-lameness treatments had a significant reduction in the MDWG. Duroc had a significantly higher MDWG whereas Hampshire had a significantly lower MDWG compared to other breeds. The total random variation was the sum of the following components: $\sigma^2_{\text{Herd}} + \sigma^2_{\text{Batch}} + \sigma^2_{\epsilon}[\exp(U\delta)]$. The residual variation for the breed Duroc, treated 1-3 times for lameness and also treated for non-lameness diseases, was calculated as: $\exp(\delta_{\text{Duroc}} + \delta_{1-3 \text{ lameness treatments}} + \delta_{\text{Non-lameness treatments}})\sigma^2_{\epsilon} = \exp(0.031 + (-0.182) + 0.266) 0.027 = 0.030$. In this case, the total random variation was 0.037, and hence herd of origin explained 1%, batch explained 16% whereas the residuals explained 81% of the total random variation. Thus, it appeared that the random effect of batch explained a larger part of the random component than did herd of origin (Table 9.3). Evaluating the dispersion effects of residual variances showed that non-lameness treatments contributed to the largest variation in the residuals (data not shown).
9.4 Discussion

9.3.3 Feed conversion ratio

According to the Box-Cox analysis an inverse transformation of the FCR was optimal and inspection of residuals suggested that this transformation improved the assumption of normality considerably. The multivariable analysis showed no significant association between the lameness treatments and the transformed FCR ($p = 0.14$) (Table 9.4). However, records of non-lameness treatments, breed and weight at four weeks were significantly associated with the transformed FCR at the 5% significance level. The interaction terms were non-significant and confounding was not present either. Boars with records of non-lameness treatments had a significantly higher FCR compared to boars without these treatments. In the pair wise comparison of breed, Duroc and Yorkshire had a significantly lower FCR compared to Hampshire and Landrace. Comparing the random effects, batch explained a larger part of the random component than did herd of origin (Table 9.4). Hence, for the breed Duroc treated for non-lameness diseases, batch explained 14% of the total random variation whereas herd of origin explained 0.7%. Using AIC, the optimal residual variance structure included dispersion effects for breed as well as records of non-lameness treatments. Non-lameness treatments had the largest effect on the residual variances (data not shown). Despite the data transformation, the residual plot indicated a few outliers. Performing the nonparametric analysis did not change the statistical significance of risk factors, and this suggested that the model fitted data adequately.

9.4 Discussion

9.4.1 Lameness treatments

In this study, lameness treatments were significantly associated with the MDWG. Other studies have found a similar effect of lameness on the MDWG (van Dijk et al., 1984; Zoric et al., 2003; Johansen et al., 2004). Boars with one to three lameness treatments had a significant reduction in the quadratic transformed MDWG compared to boars with no lameness treatments. As the models worked on the transformed scale, we performed a back transformation to the original scale, using the least squares means, in order to discuss the effect of lameness on productivity. Boars with one to three lameness treatments had a reduction in the MDWG of 27 gram per day which corresponds to a relative reduction in the MDWG of 3%. Boars with more than five lameness treatments had the largest reduction in the MDWG, corresponding to a reduction in the MDWG of 40 gram per day. Hence, more than five lameness treatments caused a relative reduction in the MDWG of 4% compared to boars with no lameness treatments. The number of lameness treatments can be interpreted as an indicator of the duration and/or the severity of the clinical signs of lameness. An increased period of clinical signs of lameness could have a systemic effect on the boars, and this can explain why the highest reduction in the MDWG was seen among boars with more than five lameness treatments. No
Table 9.3: Estimates of the effects of the model describing the quadratic transformed mean daily weight gain \( \left( \frac{K_g}{\text{Day}} \right)^2 \)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Estimate</th>
<th>S.E.</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>0.693</td>
<td>0.017</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Lameness treatments 1-3</td>
<td>-0.047(^{bc})</td>
<td>0.013</td>
<td></td>
</tr>
<tr>
<td>Lameness treatments 4</td>
<td>-0.027(^{abc})</td>
<td>0.021</td>
<td></td>
</tr>
<tr>
<td>Lameness treatments 5</td>
<td>-0.019(^{ab})</td>
<td>0.013</td>
<td></td>
</tr>
<tr>
<td>Lameness treatments &gt;5</td>
<td>-0.069(^{c})</td>
<td>0.019</td>
<td></td>
</tr>
<tr>
<td>No treatments</td>
<td>0(^{a})</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-lameness treatments yes</td>
<td>-0.097</td>
<td>0.005</td>
<td></td>
</tr>
<tr>
<td>Non-lameness treatments no</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breed</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duroc</td>
<td>0.090(^{c})</td>
<td>0.011</td>
<td></td>
</tr>
<tr>
<td>Hampshire</td>
<td>-0.150(^{b})</td>
<td>0.012</td>
<td></td>
</tr>
<tr>
<td>Landrace</td>
<td>0.021(^{a})</td>
<td>0.011</td>
<td></td>
</tr>
<tr>
<td>Yorkshire</td>
<td>0(^{a})</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight at 4 weeks</td>
<td>0.019</td>
<td>0.002</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Random component</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\sigma^2_{\text{Herd of origin}})</td>
<td>0.0005</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\sigma^2_{\text{Batch}})</td>
<td>0.006</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\delta_{1-3} \text{ lameness treatments})</td>
<td>-0.182</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\delta_{4} \text{ lameness treatments})</td>
<td>-0.304</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\delta_{5} \text{ lameness treatments})</td>
<td>-0.292</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\delta_{&gt;5} \text{ lameness treatments})</td>
<td>-0.456</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\delta_{\text{Non-lameness treatments}})</td>
<td>0.266</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\delta_{\text{Duroc}})</td>
<td>0.031</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\delta_{\text{Hampshire}})</td>
<td>-0.108</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\delta_{\text{Landrace}})</td>
<td>-0.086</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\sigma^2_{\epsilon})</td>
<td>0.027</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Estimates with different letters as superscripts are significantly different \( p \leq 0.05 \)
Table 9.4: Estimates of the effects of the model describing the inverse transformed feed conversion ratio \( \left( \frac{FU}{Kg} \right)^{-1} \)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Estimate</th>
<th>S.E.</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>0.446</td>
<td>0.003</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Non-lameness treatments</td>
<td>&lt;0.0001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>yes</td>
<td>-0.007</td>
<td>0.0009</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breed</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duroc</td>
<td>-0.003(^a)</td>
<td>0.002</td>
<td></td>
</tr>
<tr>
<td>Hampshire</td>
<td>-0.020(^b)</td>
<td>0.002</td>
<td></td>
</tr>
<tr>
<td>Landrace</td>
<td>-0.015(^c)</td>
<td>0.002</td>
<td></td>
</tr>
<tr>
<td>Yorkshire</td>
<td>0(^a)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight at 4 weeks</td>
<td>-0.0007</td>
<td>0.0003</td>
<td>0.0281</td>
</tr>
<tr>
<td>Random component</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\sigma^2) Herd of origin</td>
<td>0.00001</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\sigma^2) Batch</td>
<td>0.0002</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\delta) Non-lameness treatments</td>
<td>0.340</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\delta) Duroc</td>
<td>0.569</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\delta) Hampshire</td>
<td>0.359</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\delta) Landrace</td>
<td>0.823</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\sigma^2) (\epsilon)</td>
<td>0.0005</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Estimates with different letters as superscripts are significantly different \(p \leq 0.05\)

Significant effect of lameness on the FCR was found. This is in agreement with the study of [Huang et al. (1995)](1995) who found no significant correlation between leg weakness and the FCR. However, in our study, there was a tendency of the FCR to increase when boars were treated for lameness. Boars were categorized into groups according to the number of lameness treatments. The effect of treatment may have influenced the MDWG and the FCR of the individual boars. In a study on Mycoplasma in pigs, lincomycin had a positive effect on both the FCR and the MDWG ([Lukert and Mulkey (1982)](1982)). As antibiotics, and in particular lincomycin, were used in the treatment of lameness, it is possible that the effect of treatment can explain the tendency of increased MDWG and decreased FCR among boars with four and five lameness treatments.

### 9.4.2 Records of non-lameness treatments

We included treatment against non-lameness diseases as a dichotomized variable in the analyses. Non-lameness treatments had a negative effect on both the quadratic transformed MDWG and the inverse transformed FCR. Non-lameness treatments caused a reduction in the MDWG of 56 gram per day corresponding to a relative reduction in the MDWG of 6%. Moreover, there was an increase in the FCR of 0.04 FU per kilogram live weight due to non-lameness treatments, which was equivalent to a relative increase in the FCR of 2%. The use of antibiotics as treatment against
non-lameness diseases, and in particular the collective administration of antibiotics in the drinking water, could have affected the MDWG -and FCR. Thus, the effects caused by non-lameness treatments were probably more prominent than shown in this study. The interaction term between lameness treatments and non-lameness treatments were non-significant in the two analyses. This suggests that the association between lameness treatments and the MDWG -and FCR did not depend on whether or not boars were treated for non-lameness diseases.

9.4.3 Consequences of lameness

At the boar test station, the prevalence of boars with records of lameness treatments was 4%, whereas the prevalence of boars with records of non-lameness treatments was 65%. Due to the low prevalence of lameness in this herd, the economic effect of lameness was considered to be a minor part of the total losses due to disease. For production herds in general, the importance of lameness will depend on the prevalence of lameness in the herd. Moreover, the economic impact of a reduction in the MDWG will depend on the production type of the finisher herd (e.g. sectioning or continuous production). Hence, the production type should be taken into consideration when evaluating the economic consequences of lameness. In this study we focused on the effect of lameness on productivity and hence economy. However, lameness is known to be painful, and therefore, the animal welfare should be considered when evaluating the consequences of lameness. Other factors to take into account could be the cost of treatments and the extra labour time used in regard to lameness. However, it was not possible to measure these factors in this study.

9.4.4 Weight at 4 weeks

We included weight at 4 weeks in the analyses due to an expectation that weight at this age was related to the productivity later in the fattening period. We found a significant association between weight at 4 weeks and the MDWG as well as the FCR. Genetic differences in the production potential between boars can explain this association. It is also likely that boars with a high weight at 4 weeks were less susceptible to infectious diseases which could have an effect on the productivity in the fattening period.

9.4.5 Breed

Breed was a significant risk factor in the analysis of both the MDWG and the FCR. The differences in the productivity between the four breeds are overall in concordance with the results from the Danish Pig Production (The National Committee for Pig Production 2005). Lundheim (1987) found a genetic correlation between growth rate and leg weakness. It is possible that the association between lameness and MDWG shown in this study was influenced by the genetics of the individual boar. However, the genetic correlation was not investigated further.
9.4 Discussion

9.4.6 Model validation

Models for continuous data are based on the assumptions of normality of the residuals and homoscedasticity of the variances (Dohoo et al., 2003). To improve the model fit we performed Box-Cox analyses in order to see if any transformation of the outcome would improve the normality for the residuals. A quadratic transformation of the MDWG and an inverse transformation of the FCR improved the normality of the residuals considerably. However, the Box-Cox transformation is only one type of transformation, and other transformations of the outcomes may be relevant (Dohoo et al., 2003). Allowing the local effects of residual variances between the different breeds and the different level of lameness treatments and records of non-lameness treatments confirmed that the variances of the categorical variables differed. As records of non-lameness treatments represented treatment for diseases where the diagnostic criteria was uncertain, the effect of non-lameness treatments contributed to the largest variation in the residual variances.

Herd of origin and batch were included in the models as random effects. This was due to the assumption that boars originating from different herds and kept in separate batches had different risk of infection. It has been shown that the risk of infection increases when piglets from multiple herds are mixed together (Hege et al., 2002). However, in this study, the herd of origin explained only a limited part of the random component in the analyses of both the MDWG -and FCR. Boars in the finisher unit had been at the test station for a longer period and most likely obtained a similar health status. This can explain the limited importance of herd of origin seen in this study.

Due to the changes in the growth rate and the feed intake for pigs in the fattening period, we included only boars that had been in the finisher unit for at least 70 days. This was done in order to make the comparison of the MDWG and the FCR between boars more accurate.

9.4.7 The data

Data used in this study was from a boar test station and originally collected for other purposes. In general, there is little information regarding the effect of lameness on the productivity which can be due to difficulties in getting data on both disease and production on animal level. For the purpose of our study, data from the boar test station was considered appropriate due to records on treatments as well as records on weight gain and feed intake. However, there are certain limitations when generalizing the results from this study to the production herds in general. The technical staff observed the boars daily, and boars with clinical signs of disease were recorded and treated medically. It is likely that the threshold for treating boars was lower compared to the threshold for treating pigs in production herds. Moreover, the purebred boars were placed in pens with low stocking densities and individual feeders. Hence, in the production herds, we would anticipate even greater effects of lameness and non-lameness diseases on the MDWG and
the FCR. The inter-observer variation and the threshold for treating boars were not evaluated among the technical staff. Petersen et al. (2004) evaluated the agreement among observers performing a standard clinical examination of finishers. Despite training and standardization of the observers in that study, there appeared to be variation among the individual observers. The lack of standardization and validation of the treatment records was a limitation in our study. In other studies, regarding the effect of health disorders on productivity, the clinical signs have been evaluated with higher accuracy (Scheidt et al., 1990; Straw et al., 1990). Straw et al. (1990) examined the effect of pneumonia on the daily weight gain and the feed efficiency in 20 pigs from 12 weeks to finish. During the fattening period, the severity of pneumonia was evaluated using a coughing score and a radiographic pneumonia score. At slaughter the percentage of lesions in each lung was also determined. Hence, in that study it was possible to estimate the effect of lung lesions on productivity with a high accuracy. Woltmann et al. (1995) evaluated clinical signs of leg disorders in barrows and gilts at 100 kg. The clinical signs were evaluated thoroughly by the use of a gait score, and the association between the gait score and the daily weight gain was investigated. In our study, the lameness was observed during the entire finishing period. Moreover, we used the number of lameness treatments as a quantitative measure of lameness. Even though the clinical signs of lameness were assumed to be easily recognizable, no gait score or further information on the severity of lameness existed. Nor did we have any information on the exact aetiology of the lameness. In future studies, the clinical registrations of lameness should be standardized and the aetiological cause of lameness should be verified in order to increase the accuracy of the effect of lameness on performance. Other risk factors to be included in future studies could be information on clinical diagnoses in the early weaning unit and the genetics of the individual boar.

9.5 Conclusion

Lameness treatments had a significant negative effect on the MDWG. No significant effect of lameness treatments on the FCR was found. However, boars with records of non-lameness treatments had a significant negative effect on both the MDWG and the FCR. Because of the low prevalence of lameness at the boar test station, the economic losses due to lameness must be considered to be a minor part of the total losses due to disease.

Acknowledgements

We wish to thank Annette Kjær Erbsøll for advice in this study and the Danish Meat Association for providing data.
References


REFERENCES


CHAPTER 10

THE ASSOCIATION BETWEEN DISEASE AND PROFITABILITY IN INDIVIDUAL FINISHING BOARS AT A TEST STATION

Tina Birk Jensen, Niels Peter Baadsgaard, Hans Houe, Nils Toft, Søren Østergaard

Livestock Science (in press) [1]
doi: 10.1016/j.livsci.2007.12.003

Abstract: Endemic diseases in finisher herds are considered to be costly for the pig producer. We investigated the effect of diseases on the profit margin using data from a Danish boar test station (n = 5,777) collected from July 2002 to December 2004. Boars reaching a target slaughter weight of at least 80 kilogram were included in the study. Oral and parenteral treatments were used as indicator of disease in the finishing period and, pathological lesions were used as indicator of disease at slaughter. Profit margin was calculated individually for each boar as the difference between the total revenue and the variable costs. A multivariable hierarchical model was constructed to investigate the association between the risk factors: oral treatment (yes/no), parenteral treatment (yes/no), pathological findings (yes/no), breed (Duroc, Hampshire, Landrace, Yorkshire) and weight at 4 weeks with the outcome variable: profit margin. The results showed that treatment in the finishing period had a negative effect on the profit margin. According to the

[1] Reprinted with permission from Elsevier Science
least square means estimates, boars that were treated parenterally had a reduction in the profit margin of 2.24 €. This corresponded to a reduction in the profit margin of 17%. Boars treated orally had a reduction of 0.88 €, which corresponded to a reduction in the profit margin of 7%. Pathological findings, breed and weight at 4 weeks were also significantly associated with the profit margin. The effect of pathological findings was influenced by breed and caused a reduction in the range from 0.54 to 2.41 € (corresponding to a reduction in the profit margin ranging from 4 to 20%). The results were robust to changes in price of a 30 kilogram piglet and, relatively robust in regard to changes in the feed price. However, price per kilogram carcass weight appeared to influence the economic effect of oral and parenteral treatment and pathological findings on the profit margin. The effect of oral and parenteral treatments was also sensitive to changes in medicine prices.

**Keywords:** Boar test station; Growing-finishing period; Profitability; Treatments; Pathological findings

### 10.1 Introduction

The costs of disease in the finisher pig production are associated with reduced productivity, increased cost for veterinary services and medicine and, increased labour time. The effect of disease on productivity, e.g. mean daily weight gain (MDWG), feed conversion ratio (feed units per kilogram weight gain) (FCR) and mortality, is often used to estimate the economic consequences of disease. Reduction in the MDWG can increase the time period for finishers to reach slaughter weight, which can have an effect on the number of fattening rounds per year and hence the annual returns. The cost of feed is an important expenditure in finisher herds and represents the major part of the total cost of production [Losinger (1998)]. Finishers with a poor FCR due to disease will, therefore, have a negative impact on the farm economy. The profit margin (PM) represents the difference between the total revenue and the variable costs [Rougoor et al. (1996)]. Reduction in the PM is, therefore, a direct estimate of the economic consequences of disease.

The profitability in pig production is, usually, measured and evaluated on the herd level. A large variation in the profitability can be found among pig producers [Edwards et al. (1989)]. Some of this variation may be attributed to the effect of different production diseases on the PM. To estimate the economical benefit of reducing the prevalence of diseases at herd level, it is feasible to assess the consequences of individual pigs, assuming that disease in an individual pig does not affect the PM of other pigs in the herd. This will allow detailed estimated effects of diseases on the PM in individual pigs to be used for aggregated herd level calculations. Previous studies have investigated the effect of disease, e.g. pneumonia and gastrointestinal disorder, on productivity [Straw et al. (1989) McOrist et al. (1997)]. Rougoor et al. (1996) developed a framework for calculating the cost of disease based on the PM in both sow and -finisher herds. However, to our knowledge, no studies have investigated the effect of disease on the PM in individual
pigs, including several indicators of disease concomitantly.

The objective of this study was to investigate the impact of disease on the PM using data from individual boars at a Danish boar test station. Treatments were used as indicators of disease in the finishing period and, records of pathological lesions were used as an indicator of disease at slaughter. The objective was studied by means of a multivariable hierarchical analysis. The explanatory variables comprised breed and weight at 4 weeks.

10.2 Materials and Methods

10.2.1 The data

Data was collected from boars at a Danish boar test station owned and run by the Danish Pig Production/Danish Meat Association. The data collection period was July 2002 to December 2004. The boar test station received four-week old purebred boars of the breeds: Duroc, Hampshire, Landrace, Yorkshire from Danish breeding herds. The production system at the boar test station has been described previously (Henryon et al., 2001, Jensen et al., 2007).

During the finishing period (30 kg until slaughter), boars were housed in the finisher unit. The unit comprised 16 sections with 8 pens. Normally, 14 boars were placed in each pen. Feed was given through a single feeder, and time, duration and feed consumption was recorded for each individual visit of the boar. The main components of the feed were wheat, barley and soybean, and consisted of 16.2% crude protein. All boars were inspected daily by the technical staff, and boars with clinical signs of disease were treated medically. The treatments and the related diagnoses were recorded according to a pre-specified coding system. Boars with severe clinical signs were either euthanized or died unassisted. These boars were investigated pathologically by a veterinarian. The technical staff recorded the weight of each boar at regular intervals during the finishing period. For each boar at the test station, a selection index was calculated based on the breeding values for different production traits (e.g. MDWG, FCR, lean meat percentage (LMP), litter size and longevity). Approximately 20% of the boars were selected for collection of semen for artificial insemination (AI). Boars not selected for AI were slaughtered at the local abattoir. Veterinarians and technicians at the abattoir assessed the carcasses by visual examination and recorded all pathological lesions.

10.2.2 Data management

Boars that were not selected for AI but slaughtered at the local abattoir were included in the study. Records regarding the slaughter weight, LMP and pathological findings were available from the slaughtered boars, and these data was merged with the data from the test station. Pathological findings at slaughter and treatment records during the finishing period were used as indicators of disease. Boars were
Materials and Methods

divided into two groups according to whether or not they had any pathological findings at slaughter. During the finishing period, antibiotic treatment against disease was given either by injection or treatment was administered in the drinking water as collective treatment at the pen level. Oral treatments were primarily used against diarrhoea and pneumonia whereas parenteral treatments were used against a number of disease complexes, e.g., pneumonia, lethargy and lameness. We constructed two dichotomous variables: oral and -parenteral treatment, and we grouped boars according to whether or not they had any records of oral or -parenteral treatments during the finishing period.

PM was calculated for each boar in the interval from 30 kg until slaughter. This variable represented the actual profit received for each animal.

\[
PM = (\text{Carcass weight} \times \text{price}) - (\text{FU} \times \text{price}) - (\text{medicine}) - (\text{piglet price}) \quad (10.1)
\]

Changes in prices may have a great influence on the PM during the period of the study. In order to standardize this variable according to changes in prices, we used fixed values of prices corresponding to the market price in week 10 of 2007 (Danish Pig Production, 2007).

The product of carcass weight and price represented the total revenue for each boar. Carcass weight was the weight of the boar when slaughtered and price represented the price per kg carcass (1.09 €). The basic price per kg carcass was given for carcass weights in the interval from 70 to 86 kg. Outside this interval, the price was reduced with 0.013 € per deviating kg. Furthermore, there was a penalty for a LMP below 60 and a premium for a LMP above 60 of -/+ 0.013 € per percentage point, respectively. Hence, for each boar an individual price per kg carcass could be calculated.

The total cost of feed during the finishing period was calculated as the product of feed units (FU) and feed price. FU was the total amount consumed by each boar during the finishing period and feed price was the price per FU (0.15 €/FU).

Piglet price corresponded to the price of a piglet at 30 kg (43 €). In the calculation of the PM, the piglet price was adjusted according to the actual weight for each piglet. For each piglet above or below 30 kg, there was a change in the piglet price of +/- 0.6 € per kg.

Medicine represented the total expenses for medicine during the finishing period. A total of 14 different medical products were used during the study period. Prices of these products were fixed and based on the prices from the federation of the Danish Industry of Veterinary Medicine (Veterinærmedicinsk Industiforening, 2006). These prices can be obtained from the corresponding author upon request.

10.2.3 Data control

Five hundred and seventy-five boars were excluded by the technical staff during the finishing period, primarily due to a low growth rate and, hence slaughter weight. These boars were consequently excluded from the study population, as we wanted
all boars to reach a target finishing weight of at least 80 kg. A total of 2% of the boars died unassisted or were euthanized during the study period. According to the pathological investigation, this was primarily due to leg disorders and lung lesions. Furthermore, 954 had missing values on either the feed intake or the LMP. Frequency distributions of the categorical variables: oral treatment, parenteral treatment, pathological findings and breed were used to identify potentially illegal values. The continuous variables: weight at 4 weeks and PM were checked for extreme values.

10.2.4 Statistical analysis

We constructed a multivariable hierarchical model to assess the association between the potential risk factors: oral treatment, parenteral treatment, pathological findings, breed and weight at 4 weeks with the outcome variable: PM. The statistical software SAS version 9.1 (Proc Mixed) was used (SAS [2002]) for the analysis. Variance heterogeneity was expected among the different breeds as well as between the groups with and without oral or parenteral treatment or pathological findings at slaughter. The analysis was performed using a log-linear variance model, allowing for dispersion effects of residual variances between the categorical variables. To allow for the variation between herds of origin and between boars placed in the same pen (indicated by a unique batch number), we included both variables as random effects in the analysis. The initial model included all potential risk factors and the biological plausible interactions of fixed effects as well as local dispersion effects for all levels of fixed effects. To reduce the fixed effects a backwards elimination strategy, using a significance level of 5% to exclude factors, was applied (Ersbøll et al., 2004). Akaike’s Information Criterion (AIC) was used to reduce the dispersion and random effects. Confounding was assessed by comparing the p-values and the parameter estimates when the model was reduced from having all fixed effects in the model at the same time, to models where one fixed variable was excluded. A change in the parameter estimates of more than 20 % was used as an indication of confounding. The initial model was:

\[ Y_{ijklmno} = \mu + A_i + B_j + C_k + D_l + E_{ijkl} + AB_{ij} + AC_{ik} \\
+ AD_{il} + BC_{jk} + BD_{jl} + CD_{kl} + \psi_m + \omega_n + \epsilon_{ijklmno} \]

Where

- \( Y_{ijklmno} \) was the PM for the oth boar with the rth oral treatment, the jth parenteral treatment, the kth pathological finding in the lth breed from the mth herd of origin and placed in the nth batch.
- \( \mu \) was the intercept.
- \( A_i \) was the fixed effect of the ith oral treatment (i: yes, no).
10.2 Materials and Methods

- $B_j$ was the fixed effect of the $j$th parenteral treatment ($j$: yes, no).
- $C_k$ was the fixed effect of the $k$th pathological finding ($k$: yes, no).
- $D_l$ was the fixed effect of the $l$th breed ($l$: Duroc, Hampshire, Landrace, Yorkshire).
- $E_{ijkl}$ was the estimate of the weight at 4 weeks for the $i$th oral treatment, the $j$th parenteral treatment, the $k$th pathological finding and the $l$th breed.
- $AB_{ij}$ was the interaction between the $i$th oral treatment and the $j$th parenteral treatment.
- $AC_{ik}$ was the interaction between the $i$th oral treatment and the $k$th pathological finding.
- $AD_{il}$ was the interaction between the $i$th oral treatment and the $l$th breed.
- $BC_{jk}$ represented the interaction between the $j$th parenteral treatment and the $k$th pathological finding.
- $BD_{jl}$ was the interaction between the $j$th parenteral treatment and the $l$th breed.
- $CD_{kl}$ was the interaction between the $k$th pathological finding and the $l$th breed.

The random effect of herd of origin was $\nu_m \sim N(0, \sigma^2_{\text{Herd}})$, and the random effect of batch was $\omega_n \sim N(0, \sigma^2_{\text{Batch}})$. The residuals, $\epsilon_{ijklmno} \sim N(0, \sigma^2_{\epsilon}[\exp(U\delta)])$, were assumed to be Normal distributed within each combination of oral treatment, parenteral treatment, pathological finding and breed with $\sigma^2_{\epsilon}$ as the common intercept term of the residual variance, $U$ as the design matrix reflecting the combination of the categorical variables and $\delta$ as an 6-dimensional vector of estimated parameters.

Sensitivity analyses were performed in order to investigate the influence of changes in prices on the results. Prices are known to be highly site and time dependent. Hence, for changes in the price per kg carcass weight, feed, medicine and 30 kg piglet, we repeated the analysis and, evaluated the results with respect to differences between parenteral treatment (yes/no), oral treatment (yes/no) and pathological findings (yes/no). The evaluation was based on the range of variation in these differences. The analyses were performed for each 10% change in prices and evaluated in the interval from -50% to +50%. The model assumptions were evaluated visually by QQ-plots of residuals and plots of residuals versus predicted values. The influence of extreme values was assessed by running the analysis without the extreme values and comparing the results from the analysis with and without the extreme values.
10.3 Results

10.3.1 Descriptive analysis

A total of 5,777 boars from 81 Danish breeding herds were included in the present study. The boars were kept in 819 individual batches. The mean weight at four weeks was 8.43 kg (S.D. = 1.28). At slaughter, 985 boars had pathological findings (17%). Pleuritis was the most frequent pathological finding and present in more than 9% of boars in this study. During the finishing period, 3,539 boars (61%) were treated against disease. Of boars that were treated, 3,074 (87%) had received oral treatment and 1,257 (36%) had received parenteral treatment. A total of 792 (22%) had received both oral and parenteral treatment. The data control did not lead to further exclusion of data. Descriptive statistics of the categorical risk factors and the variables included in the calculation of PM are shown in Table 10.1.

Table 10.1: Descriptive statistics of the categorical risk factors and the variables included in the calculation on the profit margin (PM) (€)

<table>
<thead>
<tr>
<th>Variable</th>
<th>N</th>
<th>Mean (S.D.)</th>
<th>PM (S.D.)</th>
<th>Revenue (S.D.)</th>
<th>Feed (S.D.)</th>
<th>Medicine (S.D.)</th>
<th>30 kg pig (S.D.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>5777</td>
<td>13.28 (5.19)</td>
<td>81.88 (6.04)</td>
<td>24.88 (2.56)</td>
<td>1.03 (1.24)</td>
<td>42.68 (1.02)</td>
<td></td>
</tr>
<tr>
<td>Oral treatment</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>3074</td>
<td>12.56 (5.23)</td>
<td>81.99 (6.19)</td>
<td>25.00 (2.61)</td>
<td>1.69 (1.16)</td>
<td>42.74 (1.08)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>2703</td>
<td>14.09 (5.01)</td>
<td>81.75 (5.87)</td>
<td>24.75 (2.49)</td>
<td>0.29 (0.82)</td>
<td>42.62 (0.94)</td>
<td></td>
</tr>
<tr>
<td>Parenteral treatment</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>1257</td>
<td>11.38 (5.50)</td>
<td>81.47 (6.37)</td>
<td>24.95 (2.69)</td>
<td>2.43 (1.48)</td>
<td>42.71 (1.08)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>4520</td>
<td>13.80 (4.97)</td>
<td>81.99 (5.94)</td>
<td>24.86 (2.52)</td>
<td>0.64 (0.80)</td>
<td>42.68 (1.00)</td>
<td></td>
</tr>
<tr>
<td>Pathology record</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>985</td>
<td>11.86 (5.97)</td>
<td>80.83 (6.76)</td>
<td>24.78 (2.58)</td>
<td>1.38 (1.43)</td>
<td>42.81 (1.09)</td>
<td></td>
</tr>
<tr>
<td>No</td>
<td>4792</td>
<td>13.57 (4.96)</td>
<td>82.09 (5.86)</td>
<td>24.91 (2.56)</td>
<td>0.96 (1.18)</td>
<td>42.66 (1.00)</td>
<td></td>
</tr>
<tr>
<td>Breed</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duroc</td>
<td>1806</td>
<td>12.82 (5.11)</td>
<td>81.07 (5.90)</td>
<td>24.57 (2.44)</td>
<td>0.89 (1.05)</td>
<td>42.79 (1.09)</td>
<td></td>
</tr>
<tr>
<td>Hampshire</td>
<td>983</td>
<td>14.94 (4.99)</td>
<td>83.61 (6.35)</td>
<td>25.00 (2.53)</td>
<td>1.15 (1.42)</td>
<td>42.51 (0.87)</td>
<td></td>
</tr>
<tr>
<td>Landrace</td>
<td>1454</td>
<td>12.19 (5.53)</td>
<td>81.84 (6.09)</td>
<td>25.80 (2.61)</td>
<td>1.12 (1.31)</td>
<td>42.73 (1.06)</td>
<td></td>
</tr>
<tr>
<td>Yorkshire</td>
<td>1534</td>
<td>13.77 (4.73)</td>
<td>81.74 (5.73)</td>
<td>24.31 (2.41)</td>
<td>1.04 (1.23)</td>
<td>42.63 (0.96)</td>
<td></td>
</tr>
</tbody>
</table>

*aCarcass weight × price per kilogram carcass weight (€)
*bTotal amount of feed units × price per feed unit (€)
*cExpenditures for medicine (€)
*dPrice for piglet at 30 kg (€)

10.3.2 Statistical analysis

The multivariable analysis showed that the fixed effects: oral treatment, parenteral treatment, pathological findings, breed and weight at 4 weeks were significantly associated with the PM at the 5% significance level (Table 10.2). We used the least square means estimates to quantify the effect of the three disease indicators on the PM. Boars that received parenteral treatment had a reduction in the PM of 2.24 € compared to boars that were not treated parenterally. This corresponded to a relative reduction of 17%. Boars treated orally had a reduction in the PM of 0.88
10.3 Results

€, corresponding to a relative reduction of 7%. The analysis showed a significant interaction between pathological finding and breed ($p = 0.005$). According to the least square means (data not shown), pathological findings caused a reduction in the PM of 0.54 € for boars of the breed Duroc, which corresponded to a relative reduction of 4%. Pathological findings at slaughter in Hampshire, Landrace and Yorkshire reduced the PM by 0.85 €, 2.41 € and 1.02 €, respectively, corresponding to a relative reduction of 6, 20 and 8%.

The total random variation was the sum of the following components: $\sigma^2_{\text{Herd}} + \sigma^2_{\text{Batch}} + \sigma^2_{\epsilon}\{\exp(U\delta)\}$. As an example, the residual variation of the breed Duroc, with oral and parenteral treatments during the finishing period but no pathology findings at slaughter was calculated as: $\exp(\delta_{\text{Oral}} + \delta_{\text{Parenteral}} + \delta_{\text{Duroc}}) \sigma^2_{\epsilon} = \exp(0.06 + 0.16 + 0.22)15.36 = 23.85$. Thus, in this case the total random variation was 0.24 + 2.53 + 23.85 = 26.62, i.e., herd of origin explained 1%, batch explained 9% whereas the residuals explained 90% of the total random variation. Thus, the random effect of batch explained a larger part of the random component than did herd of origin (Table 10.2). No indication of confounding was found in this analysis. The AIC showed that the optimal variance included dispersion effects for the four categorical variables. The residual plots indicated a few outliers. Repeating the analysis without the outliers did not change the statistical significance of risk factors, which suggested that the model for the PM fitted data adequately.

10.3.3 Sensitivity analysis

Sensitivity analyses were performed to investigate the influence of changes in prices on the differences in PM for parenteral treatment (yes/no), oral treatment (yes/no) and pathological findings (yes/no) (Figs. 10.1-10.3). The latter was performed for each of the 4 breeds but only depicted for Yorkshire (Fig. 10.3). The graphs illustrate the range of variation in the effect of disease on PM for percentage changes in prices. The effects of parenteral treatment, oral treatment and pathological findings were relatively robust in regard to changes in feed price and price of a 30 kg piglet. However, price per kg carcass weight appeared to influence the effect of oral and -parenteral treatment and pathological findings on PM. The effect of oral and -parenteral treatment on PM was also sensitive to changes in medicine prices.

10.3.4 Excluded boars

Boars excluded from the study population represented a distinct group with a mean PM of -10.07 €. The model assumptions could not be fulfilled when these boars were included in the study population. Performing a non-parametric analysis of all boars did not affect the overall results to a considerable degree. The only change in significance was that oral treatment came out non-significant in this analysis.
Table 10.2: Estimates of the effects of the model describing the profit margin (€)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Estimate</th>
<th>S.E.</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>11.80</td>
<td>0.53</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Oral treatment</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>-0.88</td>
<td>0.16</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>No</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parenteral treatment</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>-2.24</td>
<td>0.17</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>No</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pathological finding</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>-1.02</td>
<td>0.34</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>No</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breed</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duroc</td>
<td>-0.98</td>
<td>0.28</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Hampshire</td>
<td>0.92</td>
<td>0.33</td>
<td></td>
</tr>
<tr>
<td>Landrace</td>
<td>-1.48</td>
<td>0.30</td>
<td></td>
</tr>
<tr>
<td>Yorkshire</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight at 4 weeks</td>
<td>0.37</td>
<td>0.06</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Breed×Pathology</td>
<td></td>
<td></td>
<td>0.005</td>
</tr>
<tr>
<td>Duroc×Pathology(yes)</td>
<td>0.48</td>
<td>0.48</td>
<td></td>
</tr>
<tr>
<td>Hampshire×Pathology(yes)</td>
<td>0.16</td>
<td>0.58</td>
<td></td>
</tr>
<tr>
<td>Landrace×Pathology(yes)</td>
<td>-1.39</td>
<td>0.54</td>
<td></td>
</tr>
</tbody>
</table>

Random component

<table>
<thead>
<tr>
<th>Variable</th>
<th>Estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\sigma_{\text{Herd of origin}}^2$</td>
<td>0.24</td>
</tr>
<tr>
<td>$\sigma_{\text{Batch}}^2$</td>
<td>2.53</td>
</tr>
<tr>
<td>$\delta_{\text{Parenteral treatment}}$</td>
<td>0.16</td>
</tr>
<tr>
<td>$\delta_{\text{Oral treatment}}$</td>
<td>0.06</td>
</tr>
<tr>
<td>$\delta_{\text{Pathology}}$</td>
<td>0.36</td>
</tr>
<tr>
<td>$\delta_{\text{Duroc}}$</td>
<td>0.22</td>
</tr>
<tr>
<td>$\delta_{\text{Hampshire}}$</td>
<td>0.14</td>
</tr>
<tr>
<td>$\delta_{\text{Landrace}}$</td>
<td>0.35</td>
</tr>
<tr>
<td>$\sigma_{\epsilon}^2$</td>
<td>15.36</td>
</tr>
</tbody>
</table>
10.3 Results

Figure 10.1: Sensitivity analyses for the effect of parenteral treatment on the profit margin (€), i.e. the expected difference in PM for a boar received parenteral treatment compared to a similar boar who did not receive parenteral treatment. The analysis was performed for a 10% change (in the interval +/- 50%) for each of the following prices: Kilogram carcass weight, feed, 30 kilogram piglet and medicine.

Figure 10.2: Sensitivity analyses for the effect of oral treatment on the profit margin (€), i.e. the expected difference in PM for a boar received oral treatment compared to a similar boar who did not receive oral treatment. The analysis was performed for a 10% change (in the interval +/- 50%) for each of the following prices: Kilogram carcass weight, feed, 30 kilogram piglet and medicine.
Figure 10.3: Sensitivity analyses for the effect of pathological findings on the profit margin (€), for the breed Yorkshire, i.e. the expected difference in PM for a boar with pathological findings and a similar boar with no pathological findings. The analysis was performed for a 10% change (in the interval +/- 50%) for each of the following prices: Kilogram carcass weight, feed, 30 kilogram piglet and medicine.

In order to investigate the importance of these boars, we repeated the analysis for this separate group only (summary results are given in the following). The results showed that the potential risk factors: oral treatment, pathological findings and weight at 4 weeks were not significantly associated with PM at the 5% significance level. However, parenteral treatment and breed had a significant effect on the PM. Boars treated parenterally had a reduction in the PM of 4.33 €, which corresponded to a reduction of 57% compared to boars without any parenteral treatment. Hampshire caused a reduction in the PM of 73%, 36% and 95% compared to Duroc, Landrace and Yorkshire, respectively.

10.4 Discussion

10.4.1 Treatment during the finishing period

Boars from the study population that were treated parenterally had a reduction in the PM of 17%. From the descriptive analysis, it appeared that the reduction in the PM was similar to the average expenditures of medicine for boars treated parenterally, indicating that the reduction in the PM merely reflected the medicine expenditures. It is also possible that disease prevented boars from reaching target slaughter weight, which consequently could decrease the total revenue. Alternatively, the number of days in the finishing unit could be prolonged which increased the cost.
of feed for the individual boar. Decrease in the total revenue and/or increase in the feed cost could also explain the reduction in the PM seen among boars treated parenterally. Oral treatments represented collective administration of antibiotics in the drinking water during the finishing period. In this study, boars treated orally had a reduction in the PM by 7%. It was a general practise to administer antibiotics in the drinking water when a majority of boars in a pen showed clinical signs of pneumonia or diarrhoea. Hence, some boars with records of oral treatments were treated without showing clinical signs of disease. Administration of antibiotics in the drinking water can have a positive effect on the productivity (e.g. MDWG and FCR) (Partanen et al. 2002). The positive effect on productivity and, the fact that some boars were treated without showing clinical signs of disease could explain the limited effect of oral treatment seen in this analysis.

10.4.2 Pathological findings

Pathological findings at slaughter had a significant negative effect on the PM, however, the effect varied between breeds. This can be due to the fact that the four breeds had different immune status influenced by the genetics of the individual boar. In a study by Henryon et al. (2001), genetic variation for resistance to clinical and subclinical diseases was found in growing pigs. However, the genetic influence was not investigated further in our study. Pathological findings could lead to local condemnation of tissue at the abattoir, which would reduce the carcass weight and, thereby, the total revenue. This could explain the reduction in the PM seen among boars with pathological findings. Pathological findings represented disease close to slaughter. Pigs without lesions at slaughter may have had lesions earlier in the growing period (Straw et al. 1983). Hence, it is possible that some boars in this study had lesions earlier in the period which healed up and, therefore, were not detected at slaughter.

More than 9% of boars had records of pleuritis at slaughter. The prevalence of chronic pleuritis on the chest wall recorded by meat inspection, has previously been estimated to be above 20% (Christensen and Enøe 1999; Cleveland-Nielsen et al. 2002). In order to investigate the effect of pleuritis on the PM further, we divided the pathological findings into pleuritis (yes/no) and other pathology records (yes/no). The effect of pleuritis and other pathology records on the PM was found to be similar (data not shown). Due to this finding, as well as the fact that the sensitivity of pathological recordings at slaughterhouses is known to be low (Christensen and Enøe 1999), we decided to use an overall estimate of pathological findings in our study.

10.4.3 Breed

Hampshire was the most profitable breed in the study of boars reaching a slaughter weight of at least 80 kg. The association between breed and productivity has been investigated in a previous study (Jensen et al. 2007). In that study, Hampshire had
the lowest MDWG whereas Duroc had the highest MDWG. Moreover, it was found that Hampshire and Landrace had a significantly higher FCR compared to Duroc and Yorkshire. The breed Hampshire is known to have a high LMP (The National Committee for Pig Production [2005]), which would result in a larger proportion of the living weight used for human consumption. Contrary, it was shown that Hampshire was the least profitable breed among boars excluded from the study population. This was due a very low carcass weight among Hampshire in this group.

10.4.4 Weight at 4 weeks

We found a significant effect of weight at 4 weeks on PM suggesting that a high weight at 4 weeks would lead to an increase in the PM. The result agrees with a number of other studies that have shown a significant association between weaning weight and performance in the finishing period (Mahan and Lepine [1991], Jensen et al. [2007]).

10.4.5 Model evaluation

In the calculation of the PM, we included the total revenue for each boar and costs of medicine, feed and 30 kg piglet. These costs were considered to be variable costs as they would differ with the individual boar. However, not all variable costs were included in the calculation. Due to lack of information, we did not include cost of transportation and labour in the calculation of the PM. However, we believe that the costs included in the calculation of the PM are the most important variable costs in the finisher production. The risk of infection increases when piglets from multiple herds are mixed together (Hege et al. [2002]). At the test station, piglets arrived from 81 different breeding herds. During the post weaning phase, piglets from different herds were allocated to pens according to their weight. We assumed that boars originating from different herds and kept in different batches had different risk of disease and therefore, herd of origin and batch were included as random effects in the model. Herd of origin explained a limited part of the random component. As boars had been at the test station since the age of 4 weeks, they had most likely obtained a similar health status, which can explain the limited importance of herd of origin. Allowing the local effects of residual variances between the different breeds, parenteral treatment, oral treatment and pathological findings confirmed that the variances of the categorical variables differed. As pathological findings represented different findings at slaughter, the effect of this variable contributed to the largest variation in the residual variances.

In order to perform sensitivity analyses of the prices included in the study, we repeated the analysis for each 10% change in the price per kg carcass weight, feed, medicine and 30 kg piglet. The percentage interval was chosen in order to compare the sensitivity in regard to changes in each of the four prices. The effect of parenteral treatment, oral treatment and pathological findings on the PM was robust
to changes in the price of a 30 kg piglet and, relatively robust in regard to changes in the price of feed. The latter suggests that the total feed consumption was more or less the same for pigs with and without disease. Varying the medicine prices had a large influence on the difference between boars with and without oral and parenteral treatments, where only the treated boars had an effect on the PM due to changing the medicine prices. The effect of parenteral treatment, oral treatment and pathological findings on the PM was sensitive to changes in price per kg carcass weight, which can be explained by differences in the slaughter weight between pigs with and without disease. Hence, boars with pathological findings had a reduction in the slaughter weight of 0.92 kg, boars with parenteral treatment had a reduction in the slaughter weight of 0.72 kg and, boars with oral treatment had an increase in the slaughter weight of 0.18 kg. The estimated sensitivities provide input for adjusting the results of the analysis to local prices.

10.4.6 The effect of disease at herd level

The results from this study were based on individual boars reaching a target slaughter weight of at least 80 kilogram. For a finisher pig enterprise, the profitability is always evaluated on the group or -herd level due to lack of identification of individual pigs. Without identification and recordings of individual performance, the effect of disease on pig level can be used to estimate the effect of disease at herd level, assuming independence among the pigs.

Boars excluded from the study population represented a separate group of boars with a very low mean PM. Among these boars, it was shown that boars treated parenterally had a significant reduction in the PM. In production herds, this group of pigs comprise wasting pigs placed in relief pens. During the study period, two percent of the boars were euthanized or died unassisted during the study period due to severe clinical signs. These boars would contribute to a substantial loss for the finisher pig production, as they pose a direct loss in regard to the use of feed as well as purchasing costs. Moreover, dead pigs represent an opportunity cost as they occupy a space that otherwise could be used for a pig reaching slaughter weight. Other studies have recognized the importance of wasting and dead pigs on herd profitability [Wolff et al., 2006]. Hence, when estimating the overall effect of disease at herd level, the consequences of these pigs must be taken separately into account.

Boars at the test station were placed in pens with low stocking densities and individual feeders. Furthermore, the threshold for treating boars was lower compared to the threshold for treating pigs in production herds. Hence, it is conceivable that our estimates are conservative compared to the effects in typical production herds. Though, we can not generalize the results to production herds in general this study provides minimum estimates of the influence of disease on the profitability in individual pigs.
10.4.7 Conclusion

Disease had a significant negative effect on the PM in individual boars reaching a slaughter weight of at least 80 kilograms. Parenteral treatment was associated with a larger reduction in the PM than oral treatment. Also pathological findings at slaughter were associated with a negative effect on the PM. The effect of pathological findings was influenced by breed. The results were shown to be robust to changes in the price of a 30 kg piglet and relatively robust in regard to the price of feed. However, price of kg carcass weight and medicine appeared to influence the analysis results.

Acknowledgements

We wish to thank the Danish Pig Production for providing data.

References


Danish Pig Production, 2007. Notering. Danish Pig Production, Copenhagen, Denmark.
URL http://www.dansksvineproduktion.dk/Notering


REFERENCES


CHAPTER 11

AN OBJECT-ORIENTED BAYESIAN NETWORK MODELING THE CAUSES OF LEG DISORDERS IN FINISHER HERDS

Tina Birk Jensen, Anders Ringgaard Kristensen, Nils Toft, Niels Peter Baadsgaard, Søren Østergaard, Hans Houe

Submitted for journal publication

Abstract: The implementation of an effective control strategy against disease in a finisher herd requires knowledge regarding the disease level in the herd. A Bayesian network was constructed that can estimate risk indexes for 3 cause-categories of leg disorders in a finisher herd. The cause-categories of leg disorders were divided into physical causes (e.g. fracture and claw lesions), infectious causes (arthritis caused by infectious pathogens) and inherited causes (osteochondrosis). Information about the herd (e.g. the herd size, floor type and number of suppliers) and information about individual pigs (e.g. results from diagnostic tests) were used to estimate the most likely cause of leg disorders at herd level. As information to the model originated from 2 different levels, we used an object-oriented structure in order to ease the specification of the Bayesian network. Hence, a Herd class and a Pig class comprised the basic components of the object-oriented structure. The causal structure of the model was based on evidence from published literature. The conditional probabilities used in the model were elicited from experts
within the field and from the published literature. To illustrate the behaviour of the model, we investigated the benefit of performing diagnostic examinations of individual pigs in 2 fictitious herds with different herd characteristics related to the risk of leg disorders (e.g. purchase policy, production type and the stocking density in pens). In order to estimate the most likely cause of leg disorders at herd level, it was shown that the benefit of performing diagnostic examination of individual pigs would depend on the characteristics of the 2 herds.

Keywords: Bayesian networks; Risk factors; Diagnostic test procedures; Leg disorders; Finisher herds.

11.1 Introduction

Endemic diseases in finisher herds cause severe losses for the livestock producer. To plan and conduct an effective control strategy at herd level, it is necessary to have sufficient information regarding disease in the herd. Different sources can provide information about the disease level in a herd. Before a herd visit, the veterinarian can obtain information about the individual herd. Hence, the health status, the pen construction and the production policy can provide information about the risk of disease at herd level. During a herd visit, the veterinarian may observe a number of pigs clinically. The purpose of clinical examination of individual pigs is twofold: to find the most likely cause of disease, and maybe rule out other causes, and to quantify the magnitude of the problem. Moreover, the herd veterinarian may select a number of animals for further diagnostic examinations, e.g. pathological and bacteriological examination. Hence, information about disease can derive from evidence about the herd and from diagnostic examinations of individual pigs. To allow the decision maker to predict and chose the most effective control strategy, it is important to know the level of information required, in order to obtain knowledge about the disease status at herd level. Indeed, diagnostic examinations pose extra expenditure for the livestock producer, and therefore, it is essential to know which animals to select for diagnostic examinations and how many animals to test.

A common disease complex to be diagnosed in the finisher pig production is leg disorders. Leg disorders in finisher pigs (30-100 kg) can affect the productivity, and hence, the economy for the pig producer (Jensen et al., 2007), and cause welfare problems for the pigs (Busch et al., 2003). In this context, leg disorders are any lesion or dysfunction in the leg or joint that may cause clinical signs of lameness, whereas lameness is defined as deterioration in the gait and posture. The major manifestations or mechanisms behind leg disorders consist of arthritis caused by infectious pathogens (e.g. Mycoplasma hyosynoviae, Erysipelothrix rhui- siopathiae, Haemophilus parasuis and Streptococcus sp) (Hariharan et al., 1992; Buttenschon et al., 1995; Nielsen et al., 2001), physical injuries such as fractures and lesions to the claw wall (Mouttotou et al., 1997), and osteochondrosis, which may be influenced by the genetics of the pig (Grøndalen, 1974; Lundheim, 1987).
Hence, based on knowledge from the literature we identified 3 cause-categories of leg disorders in finishers: “Infectious”, “Physical” and “Inherited”. Strategies for prophylaxis, treatment and control of leg disorders in finisher herds will depend on the underlying cause-category. For example, antibiotic treatment will be used against infectious arthritis, supply of straw to pens and re-constructing the floor can be used against physical injuries, and controlling the boar semen and the growth rate of pigs can be actions against osteochondrosis.

In human medicine, Bayesian networks have been used in the diagnosis of disease, and hence, as decision support when selecting an appropriate treatment (Kahn et al., 1997; Burnside, 2005). For the finisher pig production, Otto and Kristensen (2004) constructed a Bayesian network for risk factors for infections with *Mycoplasma hyopneumoniae*. However, only few studies have used Bayesian networks to estimate the disease status in the livestock production. As information about a particular disease status at the herd level derives from both the herd characteristics as well as individual animals, the construction of Bayesian network can become very complex. An object-oriented approach can impose a hierarchical structure for the Bayesian network model, which can ease the specification of the model.

The overall objective of this study was to build a Bayesian network model that can estimate the risk of 3 cause-categories of leg disorders in finisher herds using information from the herd and individual pigs. The causal structure of the model was based on published results from literature, and the probabilities for the quantitative part of the model were elicited from published literature and expert opinions. In this study, causality was used in a broad meaning by including both specific biological causes as well as risk factors. In order to illustrate a potential application of the model, it was also the objective to estimate the benefit of performing diagnostic examinations of individual pigs when estimating the cause of leg disorders at herd level under uncertainty.

### 11.2 Materials and Methods

The model was a static Bayesian network model made for a single herd. The programming of the model was done in JAVA using the API of the general Bayesian network tool “Esthauge Limid Software System”\(^1\).

In general, a Bayesian network is a probabilistic expert system where all interdependencies are described using conditional probability distributions. The model must form a directed acyclic graph consisting of nodes and edges and, the directions of the edges represent the biological causalities. The Bayesian network allows information to flow in the opposite direction of the causality, i.e. if there is a causal direction from a node A to a node B, it is possible to obtain information of the node A by entering evidence in the node B (Jensen, 2001). As information to this model derived from both the herd characteristics and evidence about individual pigs, we

\(^1\)http://www.esthauge.dk
11.2 Materials and Methods

used an object-oriented structure for the model in order to ease the specification of the Bayesian network model. The objects were instances of 2 classes: the Herd class and the Pig class, which were the basic components in the object-oriented structure. Each of the 2 classes represented objects that shared the same structure, behaviour and attributes, as is the case in general (Bangsø, 2004). The Herd class created one object with a number of entities (e.g. stocking density, purchase policy and herd size), and the Pig class was used to create several objects and each object had entities representing animal specific information (e.g. clinical signs of lameness, gender and results from diagnostic tests).

The background for the causal structure of the model was based on evidence from the literature. On the 1st of February 2007, a literature search was performed in the databases: Agris, Agricula and CAB for the time period from 1970 to 2007. The search history included the following words being part of either the title and/or the abstract: (“pig” or “swine” or “sow” or “gilt” or “barrow” or “boar” or “porcine” or “piglet”) and (“foot” or “feet” or “leg” or “limb” or “locomotion” or “claw” or “joint” or “toe” or “digit”) and (“osteochondrosis” or “arthrosis” or “arthritis” or “epiphysiolysis” or “leg disorder” or “leg weakness” or “lameness” or “lame” or “lesion”) and (“epidemiology” or “prevalence” or “incidence” or “mortality” or “risk” or “occurrence” or “ratio”). Articles where the first author of this paper identified a relevant abstract were included. Additionally, some articles were found from the reference list of relevant articles.

11.2.1 The qualitative structure of the model

The nodes of interest in the model were the 3 hypothesis nodes representing the cause-categories of leg disorders: “Infectious”, “Inherited” and “Physical”. In the terminology of Bayesian networks, hypothesis nodes represent nodes where the event is not observable in real life (Jensen, 2001).

For each of the 3 hypothesis nodes, a risk index was defined on an arbitrary scale from 0 to 9, where the interpretation of 0 was “low risk” of the particular cause-category and the interpretation of 9 was “high risk” of the cause-category. Hence, the cause-category “Infectious” described the risk index of leg disorders due to arthritis (e.g. swollen and painful joints, stiff gait or lameness) caused by the infectious pathogens: *Mycoplasma hyosynoivae*, *Erysipelothrix rhusiopathiae*, *Haemophilus parasuis* and *Streptococcus suis*. The cause-category “Physical” described the risk index of leg disorders due to leg or claw injuries primarily caused by conditions in the surroundings. These injuries were specified as fractures of the leg, lesions to the claw wall (white line lesions and wall lesions) and lesions to the claw sole (sole, toe and heel erosion). Finally, the cause-category “Inherited” described the leg disorders due to osteochondrotic lesions, specified as osteochondrosis manifesta (OCM) and osteochondrosis dissecans (OCD) as described by (Ytrehus, 2004).

Evidence to the model was the observed state of the information nodes from the Herd class, and the diagnostic evidence regarding individual pigs from the Pig
Each node in the model was discrete and represented a finite number of states, typically from 2 to 10 states (e.g., yes/no or no straw/sparse straw/deep bedding). The causal structure of the Herd class is depicted in Fig. 11.1 and a description of the variables is given in Table 11.1.

For the Herd class, evidence regarding the nodes: “Production” (sectioned or continuous production), “Purchase” (number of suppliers of piglets) and “Herd-size” (number of pigs slaughtered annually) would influence the risk index for infectious leg disorders. The nodes: “Floor” (floor type in the pen), “Straw” (use of bedding) and “PenDen” (stocking density in the pen) would influence the risk index for both infectious and physical causes of leg disorders. Finally, “Breed” (purebred and crossbred) and “Gain” (daily weight gain: 600, . . . ,1000 g/day) were assumed to affect the risk index for inherited leg disorders.

For the Pig class, evidence regarding the nodes: “ObsLame” (yes/no) on whether or not the selected pig showed lameness was included in the node “ObsLame”. The true state of lameness for the individual pig was presented in the latent node “PigLame”, and the relation between the 2 nodes: “PigLame” and “ObsLame” depended on the sensitivity and the specificity for the
11.2 Materials and Methods

Table 11.1: Description of the nodes in the Herd class of the object-oriented Bayesian network modeling the causes of leg disorders in finisher herds

<table>
<thead>
<tr>
<th>Node name</th>
<th>Node type</th>
<th>Explanation</th>
<th>States</th>
</tr>
</thead>
<tbody>
<tr>
<td>PenDen</td>
<td>Information node</td>
<td>Stocking density of pens</td>
<td>Low (&gt; 0.65m$^2$) /High (&lt; 0.65m$^2$)</td>
</tr>
<tr>
<td>Floor</td>
<td>Information node</td>
<td>Type of floor in pens</td>
<td>Solid/Partially slatted /Fully slatted</td>
</tr>
<tr>
<td>Straw</td>
<td>Information node</td>
<td>Supply of straw to pens</td>
<td>No /Sparse /Deep bedding</td>
</tr>
<tr>
<td>HerdSize</td>
<td>Information node</td>
<td>Pigs slaughtered annually</td>
<td>1-1000 /1001-3000 /3001-5000 /5001-5000</td>
</tr>
<tr>
<td>Production</td>
<td>Information node</td>
<td>Type of production</td>
<td>Sectioned /Continuous</td>
</tr>
<tr>
<td>Purchase</td>
<td>Information node</td>
<td>Number of supply farms</td>
<td>Zero /One /&gt;one</td>
</tr>
<tr>
<td>Breed</td>
<td>Information node</td>
<td>The breed of pigs</td>
<td>Crossbred /Purebred</td>
</tr>
<tr>
<td>FeedStrat</td>
<td>Information node</td>
<td>Feeding strategy</td>
<td>Ad libitum /Restricted</td>
</tr>
<tr>
<td>Gain</td>
<td>Information node</td>
<td>Daily weight gain of pigs</td>
<td>600g /700g /800g /900g /1000g</td>
</tr>
<tr>
<td>Physical</td>
<td>Hypothesis node</td>
<td>Risk index for Physical</td>
<td>0/1/2/3/4/5/6/7/8/9</td>
</tr>
<tr>
<td>Infectious</td>
<td>Hypothesis node</td>
<td>Risk index for Infectious</td>
<td>0/1/2/3/4/5/6/7/8/9</td>
</tr>
<tr>
<td>Inherited</td>
<td>Hypothesis node</td>
<td>Risk index for Inherited</td>
<td>0/1/2/3/4/5/6/7/8/9</td>
</tr>
</tbody>
</table>

Figure 11.2: The Pig class of the object-oriented Bayesian network modeling the cause of leg disorders in finisher herds
Table 11.2: Description of the nodes in the Pig class of the object-oriented Bayesian network modeling the causes of leg disorders in finisher herds

<table>
<thead>
<tr>
<th>Node name</th>
<th>Node type</th>
<th>Explanation</th>
<th>States</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fracture</td>
<td>Hypothesis node</td>
<td>Fracture of the leg</td>
<td>Yes /No</td>
</tr>
<tr>
<td>ClawWall</td>
<td>Hypothesis node</td>
<td>White line lesion /Wall lesion</td>
<td>Yes /No</td>
</tr>
<tr>
<td>ClawSole</td>
<td>Hypothesis node</td>
<td>Sole /Toe /Heel erosion</td>
<td>Yes /No</td>
</tr>
<tr>
<td>Myco</td>
<td>Hypothesis node</td>
<td>Infectious arthritis caused by Myco</td>
<td>Yes /No</td>
</tr>
<tr>
<td>Strep</td>
<td>Hypothesis node</td>
<td>Infectious arthritis caused by Strep</td>
<td>Yes /No</td>
</tr>
<tr>
<td>Erysi</td>
<td>Hypothesis node</td>
<td>Infectious arthritis caused by Erysi</td>
<td>Yes /No</td>
</tr>
<tr>
<td>Haemo</td>
<td>Hypothesis node</td>
<td>Infectious arthritis caused by Haemo</td>
<td>Yes /No</td>
</tr>
<tr>
<td>OCM</td>
<td>Hypothesis node</td>
<td>Osteochondrosis manifesta</td>
<td>Yes /No</td>
</tr>
<tr>
<td>OCD</td>
<td>Hypothesis node</td>
<td>Osteochondrosis dissecans</td>
<td>Yes /No</td>
</tr>
<tr>
<td>Gender</td>
<td>Information node</td>
<td>Gender of the pig</td>
<td>Female /Castrate</td>
</tr>
<tr>
<td>LMP</td>
<td>Information node</td>
<td>Lean meat percentage</td>
<td>57 /58 /59 /60 /61 /62 /63</td>
</tr>
<tr>
<td>PigLame</td>
<td>Hypothesis node</td>
<td>True state of lameness</td>
<td>Yes /No</td>
</tr>
<tr>
<td>ObsLame</td>
<td>Information node</td>
<td>Observed lameness</td>
<td>Yes /No</td>
</tr>
<tr>
<td>C1-C9</td>
<td>Information node</td>
<td>Clinical examination</td>
<td>Yes /No</td>
</tr>
<tr>
<td>P1-P9</td>
<td>Information node</td>
<td>Pathological examination</td>
<td>Yes /No</td>
</tr>
<tr>
<td>B1</td>
<td>Information node</td>
<td>Bacteriological examination for Myco</td>
<td>Yes /No</td>
</tr>
<tr>
<td>B2</td>
<td>Information node</td>
<td>Bacteriological examination for Strep</td>
<td>Yes /No</td>
</tr>
<tr>
<td>B3</td>
<td>Information node</td>
<td>Bacteriological examination for Erysi</td>
<td>Yes /No</td>
</tr>
<tr>
<td>B4</td>
<td>Information node</td>
<td>Bacteriological examination for Haemo</td>
<td>Yes /No</td>
</tr>
</tbody>
</table>

Myco: Mycoplasma hyosynoviae, Erysi: Erysipelothrix rhusiopathae, Haemo: Haemophilus parasuis, Strep: Streptococcus suis
11.2 Materials and Methods

clinical observation of lameness. Available results from further diagnostic tests were used to specify the leg disorders in the Pig class with a level of precision depending on the applied diagnostic tests. These diagnostic tests were: clinical examination (inspection and palpation of joints or claws), pathological examination of joints or claws, and bacteriological examination of joint fluids. Hence, for each diagnostic test, the sensitivity and specificity were taken into account. Based on available results from diagnostic tests as well as further information regarding the individual pig, e.g. the gender (“Gender”) of the pig and the lean meat percentage (“LMP”) recorded at slaughter, it was possible to reason against the causal direction, and hence, estimate the probabilities for the specific leg disorders. Hence, probabilities for the individual leg disorders would add information to the risk indexes for the cause-categories at herd level.

11.2.2 Elicitation of probabilities

Prior information about finisher herds in Denmark was used to define the marginal distributions of the information nodes in the Herd class and the nodes: Gender and LMP in the Pig class (Jensen et al., 2008). The conditional probabilities in the model were elicited from 2 sources: results from published literature found from the previously described literature search, and expert opinions. Conversion of the quantitative results from the literature, presented by odds ratios, to conditional probabilities in the model has been described by Otto and Kristensen (2004). Tables [11.3 and 11.4] summarizes the literature used as background for the quantitative part of the model, and specifies, whether the causal relations were quantified by experts or based on results from published literature.

As only a limited number of the causal links was quantified in the literature, the probabilities for the OOBN model were mainly quantified by expert opinions. Nine experts were selected based on their specialist knowledge of leg disorders in finishers. Each expert had established experience through international research publications within specific areas of the leg disorder complex or through extensive practical experience with leg disorders. As the nodes: “Physical”, “Infectious” and “Inherited” were hypothesis nodes, and hence, not directly observable, it was not possible to quantify the links between the information nodes in the Herd class and the 3 hypothesis nodes. Instead, it was possible to obtain information regarding the causal links between the information nodes and the individual leg disorders in the Pig class (Fig. [11.3]). Additionally, we asked experts to elicit the sensitivity and specificity of each diagnostic test in the Pig class and to quantify the probabilities of clinical signs of lameness given each of the underlying leg disorder.

For the elicitation of the probabilities, we used the method developed by van der Gaag et al. (2001). Hence, we asked a specific question to be evaluated and marked on a probability scale from 0-100%. Segments of verbal anchors were added to the probability scale as a help for experts with little experience in dealing with probabilities (Fig. [11.4]).

The probabilities to be elicited were not distributed randomly among experts.
<table>
<thead>
<tr>
<th>Parent node</th>
<th>Child node(s)</th>
<th>Key reference</th>
<th>Source for the probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Floor type</td>
<td>ClawWall, ClawSole, Fracture</td>
<td>Mouttotou et al. (1999a), Scott et al. (2006)</td>
<td>Experts</td>
</tr>
<tr>
<td>Straw</td>
<td>ClawWall, ClawSole, Fracture</td>
<td>Mouttotou et al. (1999b), Kelly et al. (2000)</td>
<td>Experts</td>
</tr>
<tr>
<td>HerdSize</td>
<td>Myco, Erysi, Strep, Haemo</td>
<td>Heinonen et al. (2007)</td>
<td>Experts</td>
</tr>
<tr>
<td>Production</td>
<td>Myco, Erysi, Strep, Haemo</td>
<td>Nielsen et al. (2000)</td>
<td>Experts</td>
</tr>
<tr>
<td>Purchase</td>
<td>Myco, Erysi, Strep, Haemo</td>
<td>Smart et al. (1989)</td>
<td>Experts</td>
</tr>
<tr>
<td>Gain</td>
<td>OCM, OCD</td>
<td>Arnbjerg (2007), Literature</td>
<td></td>
</tr>
<tr>
<td>FeedStrat</td>
<td>Gain</td>
<td>Ramaekers et al. (1995)</td>
<td>Experts</td>
</tr>
<tr>
<td>Gain</td>
<td></td>
<td>The National Committee for Pig Production (2005)</td>
<td>Experts</td>
</tr>
</tbody>
</table>

Myco: Mycoplasma hyosynoviae, Erysi: Erysipelothrix rhusiopathiae, Haemo: Haemophilus parasuis, Strep: Streptococcus suis
Table 11.4: Information used as background for the qualitative and quantitative part of the object-oriented Bayesian network model. The Pig class.

<table>
<thead>
<tr>
<th>Parent node</th>
<th>Child node(s)</th>
<th>Key reference</th>
<th>Source for the probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>LMP</td>
<td>OCM, OCD</td>
<td>Stern et al. (1995), Busch et al. (2007)</td>
<td>Literature</td>
</tr>
<tr>
<td>Gender</td>
<td>OCM, OCD</td>
<td>Stern et al. (1995), Busch et al. (2007)</td>
<td>Literature</td>
</tr>
<tr>
<td>B1-B4</td>
<td>Myco, Erysi, Strep, Haemo</td>
<td>Experts</td>
<td></td>
</tr>
<tr>
<td>P1-P9</td>
<td>ClawWall, ClawSole, Fracture Myco, Erysi, Strep, Haemo OCM, OCD</td>
<td>Experts</td>
<td></td>
</tr>
<tr>
<td>C1-C9</td>
<td>ClawWall, ClawSole, Fracture Myco, Erysi, Strep, Haemo OCM, OCD</td>
<td>Experts</td>
<td></td>
</tr>
<tr>
<td>ObsLame</td>
<td>PigLame</td>
<td>Baadsgaard and Jørgensen (2003)</td>
<td>Literature</td>
</tr>
</tbody>
</table>

Myco: Mycoplasma hyosynoviae, Erysi: Erysipelothrix rhusiopathiae, Haemo: Haemophilus parasuis, Strep: Streptococcus suis

Figure 11.3: Causal links elicited from experts in regard to the hypothesis node: “Physical”. Edges shown with bold arrows are links from which we do not have quantitative information. Edges shown with dashed arrows are the quantitative information we can obtain.
Consider 100 pigs examined individually at a herd visit. The herd has a high stocking density in the pens.

How often do you, during the examination, expect to find a pig with a fracture?

<table>
<thead>
<tr>
<th>Probability</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Always(almost)</td>
<td>100</td>
</tr>
<tr>
<td>Usually</td>
<td>85</td>
</tr>
<tr>
<td>Often</td>
<td>75</td>
</tr>
<tr>
<td>As often as not</td>
<td>50</td>
</tr>
<tr>
<td>Sometimes</td>
<td>25</td>
</tr>
<tr>
<td>Once in a while</td>
<td>15</td>
</tr>
<tr>
<td>(Almost)never</td>
<td>0</td>
</tr>
</tbody>
</table>

Figure 11.4: Example of the elicitation of the probabilities using expert opinions. The conditional probability $P(\text{Fracture} | \text{high stocking density})$ was obtained by asking the above question.
Instead, specific probabilities were given to one or more experts with professional knowledge within a particular area. Consequently, each expert was asked to estimate between 10 and 96 conditional probabilities. When 2 or more experts answered similar questions, the resulting probabilities used in the model were merely the average of the individual elicitations.

### 11.2.3 Modeling methods

For each of the 3 hypothesis node: “Physical”, “Infectious” and “Inherited”, the risk index was defined based on a linear equation that quantified the total effect of the information nodes (risk factors) on the leg disorders in question. Though, the model assumed a continuous risk index, we modeled it as a discrete variable where we distinguished 10 distinct levels from 0-9. This was due to the fact that it is not possible for discrete child nodes (i.e. the leg disorders nodes) to have continuous parent nodes [Jensen, 2001].

The general model for the risk index for a given configuration \((i_1, \ldots, i_n)\) of the total effect of risk factors \((R_1, \ldots, R_n)\) was defined with the following model

\[
I_{i_1 \ldots i_n} = \mu + \rho_1^{i_1} + \rho_2^{i_2} + \ldots + \rho_n^{i_n} + e_{i_1 \ldots i_n}, \tag{11.1}
\]

where

- \(I_{i_1 \ldots i_n}\) was the resulting risk index.
- \(\mu\) was an intercept.
- \(\rho_k^{i_k}\) was the systematic effect of state \(i_k\) of risk factor \(k\).
- \(e_{i_1 \ldots i_n} \sim N(0, \sigma_e^2)\) was a random residual.

It was a model assumption that there were no interactions between the risk factors and that the effects of the risk factors were additive.

Each leg disorder node in the Pig class was modeled using a logistic regression. Hence, the general model of an arbitrary pig to have the \(k\)th leg disorder for a given state \(i_I\) of the risk index I was defined as:

\[
Y_{i_I}^k = \delta_k^0 + \delta_k^{1i_I}, \tag{11.2}
\]

Where

- \(Y_{i_I}^k\) was the logistic transformation of the conditional probability of an arbitrary pig to have the leg disorder \(k\) (\(k: \text{yes, no}\)).
- \(\delta_k^0\) was the intercept indicating the base prevalence of the leg disorder \(k\).
- \(\delta_k^1\) was the slope which indicated the sensitivity to changes in the risk level of the herd.
Based on the probabilities elicited from experts and literature, the parameter estimates for Eqs. (11.1) and (11.2) were determined in such a way that they created the best possible fit to the conditional probabilities under the constraint that all effects of the risk factors were expressed through a common hypothesis node. A combination of fitting a linear model and applying a general optimization function based on Nelder-Mead algorithm was used (Nelder and Mead, 1965). All analyses were carried out in R (version 2.3.1) (R Development Core Team, 2006). The linear model was fitted by the \texttt{lm} function and the Nelder-Mead algorithm was called through the \texttt{optim} function. For a thorough description of the modeling methods of each node, readers are referred to Jensen et al. (2008).

### 11.2.4 Modeling scenarios

To demonstrate the behaviour of the Bayesian network model, we investigated the impact of different levels of information required on our ability to distinguish the most likely cause of leg disorders at herd level. Hence, we compared 10 different scenarios in 2 fictitious herds with different herd characteristic but the same prevalence of leg disorders. In both herds, a total of 50 randomly selected pigs were observed for lameness outside the pen, and we assumed that 20% of the selected pigs showed clinical signs of lameness. Moreover, we assumed that lame pigs were positive for \textit{Mycoplasma hyosynoviae} when further diagnostic tests (clinical, pathological and bacteriological examinations) were performed.

From the literature it is known that a number of herd risk factors can influence the risk of arthritis caused by infectious pathogens (Smith and Morgan, 1997). Hence, Herd 1 was defined as a low-risk herd with sectioned production, low pen densities (>0.65m$^2$ space per pig in a pen), solid floors and no supply of straw in the pens. Moreover, we assumed that Herd 1 produced own piglets and delivered 2000 finishers to the slaughter house, annually. Herd 2 was a high-risk herd producing 6000 finishers annually. The densities in the pens were high (<0.65m$^2$ space per pig in a pen) with partially slatted floors and sparse supply of straw. The production was continuous and piglets were purchased from several supply herds.

For each herd, we investigated and compared the following 10 scenarios.

1. No evidence about the herd or pigs (default value).
2. Herd evidence available.
3. Herd evidence available and 50 randomly selected pig investigated for lameness (outside the pen).
4. Herd evidence available and lame pigs examined clinically (inside the pen).
5. Herd evidence available and lame pigs examined clinically and pathologically.
6. Herd evidence available and lame pigs examined clinically, pathologically and bacteriologically.
7. Herd evidence available and all pigs examined clinically.

8. Herd evidence available and all pigs examined clinically and pathologically.

9. Herd evidence available and all pigs examined clinically, pathologically and bacteriologically.

10. Herd evidence available and perfect knowledge about all pigs regarding the individual leg disorders (SE = 1 and SP = 1 for all diagnostic tests).

An overview of the 10 scenarios is given in Table 11.5.

Table 11.5: Overview of the 10 scenarios used to illustrate the behaviour of the object-oriented Bayesian network model

<table>
<thead>
<tr>
<th>Scenario</th>
<th>Herd evidence</th>
<th>Pig evidence</th>
<th>Results from diagnostic test</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>No</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>Yes</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>Yes</td>
<td>10 yes 40 no</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>Yes</td>
<td>10 yes 40 no</td>
<td>Lame pigs (n = 10) examined clinically. Clinical signs of Myco in lame pigs. No clinical signs of other leg disorders.</td>
</tr>
<tr>
<td>5</td>
<td>Yes</td>
<td>10 yes 40 no</td>
<td>Lame pigs (n = 10) examined clinically and pathologically. Evidence of Myco in both tests for lame pigs. No evidence of other leg disorders.</td>
</tr>
<tr>
<td>6</td>
<td>Yes</td>
<td>10 yes 40 no</td>
<td>Lame pigs (n = 10) examined clinically, pathologically and bacteriologically. Evidence of Myco in all tests for lame pigs. No evidence of other leg disorders.</td>
</tr>
<tr>
<td>7</td>
<td>Yes</td>
<td>10 yes 40 no</td>
<td>All pigs (n = 50) examined clinically. Clinical signs of Myco in lame pigs. No clinical signs of other leg disorders.</td>
</tr>
<tr>
<td>8</td>
<td>Yes</td>
<td>10 yes 40 no</td>
<td>All pigs (n = 50) examined clinically and pathologically. Evidence of Myco in both tests for lame pigs. No evidence of other leg disorders.</td>
</tr>
<tr>
<td>9</td>
<td>Yes</td>
<td>10 yes 40 no</td>
<td>All pigs (n = 50) examined clinically, pathologically and bacteriologically. Evidence of Myco in all tests for lame pigs. No evidence of other leg disorders.</td>
</tr>
<tr>
<td>10</td>
<td>Yes</td>
<td>10 yes 40 no</td>
<td>All pigs (n = 50) examined with perfect tests where the SE and SP equal 1.</td>
</tr>
</tbody>
</table>
11.3 Results from the model

Figs. [11.5][11.6] and [11.7] depict the risk indexes for the hypothesis nodes: “Physical”, “Inherited” and “Infectious” for each scenario in both the low-risk and the high-risk herd.

<table>
<thead>
<tr>
<th>Scenario</th>
<th>Low-risk herd</th>
<th>High-risk herd</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Default</td>
<td><img src="#" alt="Graph 1" /></td>
<td><img src="#" alt="Graph 2" /></td>
</tr>
<tr>
<td>2 Herd evidence</td>
<td><img src="#" alt="Graph 3" /></td>
<td><img src="#" alt="Graph 4" /></td>
</tr>
<tr>
<td>3 ObsLame (yes/no)</td>
<td><img src="#" alt="Graph 5" /></td>
<td><img src="#" alt="Graph 6" /></td>
</tr>
</tbody>
</table>

Figure 11.5: Risk indexes for the cause-categories: “Physical”, “Inherited” and “Infectious” in a low-risk and a high-risk herd. Scenarios 1-3.

By comparing the different scenarios, it was possible to illustrate changes in knowledge regarding the most likely cause-category at herd level. For both the low- and the high-risk herd, the model predicted a mean default value of approximately 4.5 for each cause-category when no information was included in the model (Scenario 1, Fig. [11.5]). Hence, a high risk of a particular cause-category was presented by right shift in the figures whereas a low risk of a particular cause-category was shown as left shift in the figures. For the low-risk herd, it was not possible to differentiate the most likely cause-category of leg disorders in Scenarios 1-3.
11.3 Results from the model

<table>
<thead>
<tr>
<th>Scenario</th>
<th>Low-risk herd</th>
<th>High-risk herd</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>Clinic ex. (lame pigs)</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Clinic and path ex. (lame pigs)</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Clinic, path. and bac. ex. (lame pigs)</td>
<td></td>
</tr>
</tbody>
</table>

Figure 11.6: Risk indexes for the cause-categories: “Physical”, “Inherited” and “Infectious” in a low-risk and a high-risk herd. Scenarios 4-6.
<table>
<thead>
<tr>
<th>Scenario</th>
<th>Low-risk herd</th>
<th>High-risk herd</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>Clinic ex.</td>
<td>Clinic ex.</td>
</tr>
<tr>
<td></td>
<td>(all pigs)</td>
<td>(all pigs)</td>
</tr>
<tr>
<td>8</td>
<td>Clinic and</td>
<td>Clinic and</td>
</tr>
<tr>
<td></td>
<td>path. ex.</td>
<td>path. ex.</td>
</tr>
<tr>
<td></td>
<td>(all pigs)</td>
<td>(all pigs)</td>
</tr>
<tr>
<td>9</td>
<td>Clinic, path.</td>
<td>Clinic, path.</td>
</tr>
<tr>
<td></td>
<td>and bac. ex.</td>
<td>and bac. ex.</td>
</tr>
<tr>
<td></td>
<td>(all pigs)</td>
<td>(all pigs)</td>
</tr>
<tr>
<td>10</td>
<td>Perfect knowledge</td>
<td>Perfect knowledge</td>
</tr>
</tbody>
</table>

Figure 11.7: Risk indexes for the cause-categories: “Physical”, “Inherited” and “Infectious” in a low-risk and a high-risk herd. Scenarios 7-10.
11.4 Discussion

However, when lame pigs were further clinically examined (Scenario 4, Fig. 11.6), infectious leg disorders appeared to have the highest risk index, which consequently indicated that infectious leg disorders were the most likely cause in the herd. This indication was even stronger after lame pigs were investigated both clinically and pathologically (Scenario 5, Fig. 11.6). For the high-risk herd, infectious leg disorders already appeared to have the highest risk index after entering herd evidence in the model (Scenario 2, Fig. 11.5). Observing lameness in 50 randomly selected pigs (Scenario 3, Fig. 11.5) increased the risk index for infectious leg disorders even further. Hence, less information on animal level was required for the high-risk herd than for the low-risk herd. For the diagnostic examinations, 2 different populations of pigs were chosen. In Scenarios 4-6 (Fig. 11.6), diagnostic examinations were performed on pigs showing clinical signs of lameness, whereas in Scenarios 7-9 (Fig. 11.7), diagnostic examinations were performed on all (50) animals. Selecting all pigs for diagnostic examinations (Scenarios 8 and 9, Fig. 11.7) predicted a low risk for both physical and inherited leg disorders and a high risk for infectious leg disorders. Hence, these scenarios gave the best estimate of the most likely cause in both herds. However, a special case was seen when all pigs were examined clinically (Scenario 7, Fig. 11.7). Despite the fact that no pigs had clinical signs of OCM and OCD, inherited leg disorders appeared to have the highest risk index in both herds. This was due to the very low sensitivities and specificities of OCM (0.19/0.19) and OCD (0.33/0.20) given by experts (Jensen et al., 2008).

11.4 Economic benefit of diagnostic examination

We have presented a Bayesian network model that can estimate risk indexes for 3 cause-categories of leg disorder in finisher herds. Others have previously used a Bayesian network model to describe the influence of herd risk factors on the severity of *Mycoplasma hyopnemoniae* infection in finisher herds (Otto and Kristensen, 2004). However, only evidence at herd level was used as input in that model. Our model used information from 2 sources: evidence regarding the particular herd as well as evidence about individual pigs, in the estimation of the most likely cause of leg disorders. Hence, to ease the specification of the Bayesian network an object-oriented structure was used.

11.4.1 Economic benefit of diagnostic examination

The OOBN model can predict the impact of different levels of information in order to estimate the most likely cause of leg disorders in a herd. Basically, the costs of disease can be split into production losses (reduced growth rate, increased feed consumption and increased mortality) and intervention expenditures (McInerney et al., 1992). A successful intervention can, thereby, reduce the prevalence of disease, and hence, the production losses. However, implementing a control strategy
(e.g. treatment) without knowing the cause of disease will increase the risk of choosing an ineffective control strategy. Literally, this can cause losses in regard to production (no effect of treatment) as well as medicine expenditures. Hence, a successful intervention requires valid information regarding the cause and the level of the disease in question. By performing diagnostic tests of individual animals, it is possible to increase the certainty of the most likely cause, and thereby, reduce the risk of choosing an ineffective control strategy. It is well known that diagnostic tests pose an extra expenditure for the farmer. The price of a pathological examination is 75 € per pig and the price of a bacteriological examination is 44 € per pig [Danish Pig Production, 2008]. Therefore, it is desirable to estimate the minimum number of diagnostic tests to be carried out in a herd, in order to increase the certainty of the cause to a level wanted by the decision maker i.e. farmer and the veterinarian. Hence, the OOBN model can be helpful when the decision maker has to decide whether or not to perform diagnostic examinations of individual pigs, in order to obtain information about the disease status in the herd.

This study investigated the behaviour of the model by comparing 10 different scenarios in 2 fictitious herds. It is likely that the disease prevalence would differ between herds with different characteristics. Moreover, some of the presented scenarios may not be of practical relevance to the finisher pig production. However, the scenarios serve as a convenient illustration of the level of information needed, in order to estimate the most likely cause of leg disorder in 2 herds with different characteristics. For the high-risk herd, infectious leg disorders already tended to have the highest risk index when herd evidence was included in the model (Scenarios 2 and 3). This was not the case in the low-risk herd. In this herd, it was necessary to perform further diagnostic examinations of individual pigs in order to estimate the most likely cause of leg disorders. It appeared that more information at pig level was necessary in the low-risk herd compared to the high-risk herd. This was due to the fact that information at herd level for the low-risk herd indicated poor risk for infectious arthritis, whereas information from individual pigs changed this prior knowledge considerably. Therefore, the benefit of additional information at pig level (further diagnostic tests) was greatest for the low-risk herd.

11.4.2 The qualitative structure of the model

The 3 cause-categories: “Physical”, “Inherited” and “Infectious” represented different leg disorders in finisher herds. The reason to integrate leg disorders into 3 cause-categories was primarily due to the fact that the control strategies would depend on the underlying cause-category.

The differential diagnoses of leg disorders presented in the model were not exhaustive. There may be other causes of leg disorders in finishers, e.g. muscle ruptures and nerve compression that may cause problems in finisher herds. Yet, we believe that the disorders presented in the model are the most common leg disorders seen in the Danish finisher pig production. Moreover, it is possible that other risk factors could influence the occurrence of leg disorders. It has been stated that
tail bite can increase the risk of other infectious diseases (Schrøder-Petersen and Simonsen [2001]). This can be due to the fact that tail biting affects the immunity of pigs and can serve as entrance for infectious pathogens. However, the experts could not quantify any differences in the probability of infectious arthritis for a pig with and without a tail bite. Consequently, we chose not to include injuries (e.g., tail bite and claw lesions) as risk factors for infectious arthritis in the model. The individual leg disorders were specified using different diagnostic tests. For the bacteriological examination, we assumed that a positive finding would indicate clinical arthritis. Previously, *Mycoplasma hyosynoviae* has been found in joints from pigs with no clinical signs of arthritis (Nielsen et al. [2001]). The fact that a pathogen can be present latently in the joint was not considered in the model. The model is supposed to include pigs during the entire finishing period (30-100 kg). However, some leg disorders may be more prominent in certain age intervals, e.g., the incidence of osteochondrosis might be highest in the final part of the finishing period. In the definition of the individual leg disorders, we did not explicitly distinguish between the acute or chronic state of the disorder. It is likely that the state of the disorders could influence the outcome of the model. Hence, the state of each disorder and the age of the pigs should be included in future models.

### 11.4.3 Probabilities used in the model

The majority of probabilities in the model were quantified using expert opinions. The experts were selected based on their specialist knowledge in the field. It is important to be aware of possible bias when using expert opinions. Specifically, experts should have a thorough understanding of the problem to be evaluated, and they must be capable of responding to questions using probabilities (van der Fels-Klerx et al. [2002]). In order to encompass these problems, we discussed the problem at hand with experts before and after the elicitation. Moreover, we added segments of verbal anchors to the probability scale as a help for the experts to elicit the probabilities, as described by van der Gaag et al. (2001). The group of experts used for the elicitation in this study had different expertise within the field. When similar questions were asked to 2 or more experts, the experts did not always agree. However, the resulting probability used in the model was the average of the individual answers. Van der Fels-Klerx et al. (2002) used a heterogeneous expert panel for the quantification of continuous variables. In that study, the individual expert assessments were weighted according to the expertise of the individual experts. Weighting the individual experts according to their expertise could potentially have produced more valid estimates for our model.

The risk indexes were defined based on a linear equation that quantified the total effect of risk factors on the leg disorders in question. We assumed that the effect of the risk factors was additive and that no interactions existed among the different risk factors. This assumption was made out of convenience, since interaction terms would drastically increase the complexity of the specification of conditional probabilities of the model. However, it should be emphasized that from a method-
ological point of view, interactions could easily be handled by adding interaction terms in Eq. (11.1). If estimates on the interaction terms become available in the future, these should be taken into account in the model.

11.4.4 Future research and conclusion

This study has presented an object-oriented Bayesian network model that estimates risk indexes for 3 major cause-categories of leg disorders in finisher herds using information from the herd and from individual pigs. An extended validation of the network model in real finisher herds is needed in the future. Sensitivity analyses should be carried out in order to investigate the robustness of the model; e.g., investigate the relationship between the model output and every single input parameter, as described by Kjærulff and van der Gaag (2000).

Ten alternative ways of conducting information were investigated in 2 fictitious herds. The results showed large differences in the obtained information regarding the underlying cause. It was shown that the benefit of performing diagnostic examinations would depend on the individual herd. Hence, the model can be used to predict the level of information required in order to estimate the most likely cause of leg disorders in a herd. We plan to integrate the model in a decision support system that can estimate the cost-effectiveness of diagnostic examinations in finisher herds.

Acknowledgements

The authors wish to thank Professor Linda van der Gaag for advise on the elicitation of expert opinions for this study. Furthermore, the following people are thanked for providing expert opinions to the model: Øystein Angen (National Veterinary Institute), Marie Erika Busch (Danish Pig Production), Svend Haugegaard (National Veterinary Institute), Tim Kåre Jensen (National Veterinary Institute), Sven Erik Jorsal (National Veterinary Institute), Bente Jørgensen, Elisabeth Okholm Nielsen (Danish Meat Association), Jens Peter Nielsen (University of Copenhagen), Helle Stege (University of Copenhagen).

References


REFERENCES


URL http://www.dansksvineproduktion.dk/Services/Laboratorium/Priser%20paa%20undersoegelser.html


URL http://www.prodstyr.ihh.kvl.dk/oobn/


Mouttotou, N., Hatchell, F. M., Green, L. E., 1999a. Foot lesions in finishing pigs and their associations with the type of floor. Veterinary Record 144, 629–632.


REFERENCES


# APPENDIX A

## DISEASE CODES

Table A.1: Pre-specified coding list for diseases used at the boar test station

<table>
<thead>
<tr>
<th>Disease code</th>
<th>Explanation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Respiratory disease</td>
</tr>
<tr>
<td>2</td>
<td>Respiratory disease</td>
</tr>
<tr>
<td>4</td>
<td>Nasal disorders</td>
</tr>
<tr>
<td>5</td>
<td>Diarrhoea</td>
</tr>
<tr>
<td>6</td>
<td>Abscess</td>
</tr>
<tr>
<td>7</td>
<td>Abscess in foot</td>
</tr>
<tr>
<td>8</td>
<td>Lameness</td>
</tr>
<tr>
<td>13</td>
<td>Convulsion / Meningitis</td>
</tr>
<tr>
<td>15</td>
<td>Paralysis</td>
</tr>
<tr>
<td>16</td>
<td>Lethargic</td>
</tr>
<tr>
<td>18</td>
<td>Eczema</td>
</tr>
<tr>
<td>19</td>
<td>Tail bite</td>
</tr>
<tr>
<td>20</td>
<td>Ear tag inflammation</td>
</tr>
<tr>
<td>22</td>
<td>Other</td>
</tr>
<tr>
<td>25</td>
<td>Navel hernia</td>
</tr>
<tr>
<td>32</td>
<td>Cryptochism</td>
</tr>
<tr>
<td>33</td>
<td>Hernia</td>
</tr>
<tr>
<td>45</td>
<td>Other</td>
</tr>
</tbody>
</table>
Table A.2: Pre-specified coding list for diseases used at the abattoir

<table>
<thead>
<tr>
<th>Disease code</th>
<th>Explanation</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>Acute pericarditis</td>
</tr>
<tr>
<td>11</td>
<td>Chronic pericarditis</td>
</tr>
<tr>
<td>16</td>
<td>Atrophic rhinitis</td>
</tr>
<tr>
<td>17</td>
<td>Abscess in head</td>
</tr>
<tr>
<td>18</td>
<td>Abscess in neck</td>
</tr>
<tr>
<td>21</td>
<td>Chronic pneumonia</td>
</tr>
<tr>
<td>23</td>
<td>Pleuritis</td>
</tr>
<tr>
<td>24</td>
<td>Lung disease 2</td>
</tr>
<tr>
<td>26</td>
<td>Lung disease 1</td>
</tr>
<tr>
<td>31</td>
<td>Chronic colitis</td>
</tr>
<tr>
<td>34</td>
<td>Torsion of spleen</td>
</tr>
<tr>
<td>41</td>
<td>Chronic peritonitis</td>
</tr>
<tr>
<td>42</td>
<td>Hernia</td>
</tr>
<tr>
<td>43</td>
<td>Abscess in the peritoneum</td>
</tr>
<tr>
<td>51</td>
<td>Chronic renal infection</td>
</tr>
<tr>
<td>56</td>
<td>Cryptochism</td>
</tr>
<tr>
<td>62</td>
<td>Chronic arthritis</td>
</tr>
<tr>
<td>63</td>
<td>Abscess in foot</td>
</tr>
<tr>
<td>64</td>
<td>Osteomyelitis</td>
</tr>
<tr>
<td>65</td>
<td>Acute fracture</td>
</tr>
<tr>
<td>66</td>
<td>Chronic fracture</td>
</tr>
<tr>
<td>67</td>
<td>Luxation of joint</td>
</tr>
<tr>
<td>68</td>
<td>Abscess in the caudal part</td>
</tr>
<tr>
<td>69</td>
<td>Tail bite</td>
</tr>
<tr>
<td>71</td>
<td>Scar tissue</td>
</tr>
<tr>
<td>72</td>
<td>Deviating color of skin</td>
</tr>
<tr>
<td>73</td>
<td>Excema</td>
</tr>
<tr>
<td>79</td>
<td>Tumor</td>
</tr>
<tr>
<td>88</td>
<td>Barn diagnosis</td>
</tr>
</tbody>
</table>
APPENDIX B

ELICITATION OF PROBABILITIES IN AN OBJECT-ORIENTED BAYESIAN NETWORK FOR LEG DISORDERS IN FINISHER PIGS

Tina Birk Jensen, Anders Ringgaard Kristensen, Nils Toft

B.1 Introduction

This appendix serves as background for the article: “An object-oriented Bayesian network modeling the causes of leg disorders in finisher herds”. Fig. B.1 and B.2 illustrate the causal structure of the Herd class and the Pig class of the model.

The appendix contains all probabilities used in the model, and describes the methodology for modeling the individual nodes. Section B.2 describes the common methodology for modeling the risk indexes for the 3 nodes. Section B.3 - B.5 present the probabilities and the resulting parameter estimates used for each of the nodes: “Inherited”, “Physical” and “Infectious”. Section B.6 describes the modeling of the “PigLame” node, and finally, the sensitivities and specificities used in the model are specified in Section B.7. The model can be downloaded at: http://www.prodstyr.ihh.kvl.dk/oobn/
Appendix B: Elicitation of probabilities

Figure B.1: The Herd class of the OOBN model

Figure B.2: The Pig class of the OOBN model
B.2 A Risk Index complex

This section will describe the common methodology for modeling the 3 risk indexes “Inherited”, “Physical” and “Infectious”.

B.2.1 Common structure of a risk index complex

Fig. [B.3] shows the general layout of a subgraph modeling the effects of a number of herd level risk factors $R_1, R_2, \ldots, R_n$ on a number of distinct diseases $D_1, D_2, \ldots, D_m$ at pig level. As illustrated in the figure, it is assumed that the effects of the risk factors always is expressed through the risk index, $I$.

![Diagram](image)

Figure B.3: A number of herd level risk factors, $R_1, \ldots, R_n$ influencing a number of pig level diseases $D_1, \ldots, D_m$ through a common risk index $I$.

All nodes of Fig. [B.3] represent discrete variables with a finite number of mutually exclusive states. Thus, we shall refer to a specific state of e.g. $R_k$ as $i_k$. We shall use the notation $|R_k|$ for the number of states defined for $R_k$. In other words, $i_k \in \{1, \ldots, |R_k|\}$. For the risk factors each state typically refers to a natural level of the risk factor (i.e. for the risk factor $R_k = \text{“Breed”}$, we have $i_k \in \{\text{“Purebred”}, \text{“Crossbred”}\}$). For the diseases the state space is simply for the $k$th disease, $j_k \in \{\text{“No”}, \text{“Yes”}\}$ corresponding to absence or presence of the disease, respectively.

B.2.2 A model for the probabilities of the risk index

The idea of the risk index, $I$, is to quantify the total effect of the risk factors on the diseases in question. The general model for the risk index for a given configuration $(i_1, \ldots, i_n)$ of the risk factors $(R_1, \ldots, R_n)$ is

$$I_{i_1 \ldots i_n} = \mu + \rho_1^{i_1} + \rho_2^{i_2} + \ldots + \rho_n^{i_n} + e_{i_1 \ldots i_n},$$

(B.1)
where

- $I_{i_1 \ldots i_n}$ is the resulting risk index.
- $\mu$ is an intercept.
- $\rho_{i_k}^k$ is the systematic effect of state $i_k$ of risk factor $k$.
- $e_{i_1 \ldots i_n} \sim \mathcal{N}(0, \sigma_e^2)$ is a random residual.

The model (B.1) assumes that there are no interactions between the risk factors and that the effects are additive. This assumption is made out of convenience, since interaction terms would drastically increase the complexity of the specification of conditional probabilities of the model. Since the elicitation of conditional probabilities in most cases is based on expert knowledge using methods developed by van der Gaag et al. (2001), it would make the elicitation process very complicated if interactions were included. It should, however, be emphasized that from a methodological point of view, interactions could easily be handled just by adding interaction terms in (B.1).

Even though the model (B.1) assumes a continuous risk index, we model it as a discrete variable where we distinguish 10 distinct levels from 0 to 9. The interpretation of 0 is: “poor risk” of the particular cause category and the interpretation of 9 is: “strong risk” of the cause category. Furthermore, we require by definition, that the prior mean of the risk index (before any observations are made) must be 4.5.

For a given state $i_I$, $0 < i_I < 9$, of $I$ we interpret the value as the midpoint of the interval from $i_I - 0.5$ to $i_I + 0.5$. Thus state $i_I$ is interpreted as $i_I - 0.5 < I \leq i_I + 0.5$. The states $i_I = 0$ and $i_I = 9$ are interpreted as $I \leq 0.5$ and $I > 8.5$, respectively.

The conditional probability distribution $(I \mid R_1, \ldots, R_n)$ (for $I$ regarded as a continuous variable) is according to Eq. (B.1):

$$(I \mid R_1, \ldots, R_n) \sim \mathcal{N}(\mu_{i_1 \ldots i_n}, \sigma_e^2),$$

where $\mu_{i_1 \ldots i_n} = \mu + \rho_{i_1}^1 + \rho_{i_2}^2 + \ldots + \rho_{i_n}^n$. When $I$ is transformed to the discrete form, we simply calculate the conditional probability for state $i_I$ given the configuration $(i_1, \ldots, i_n)$ of the risk factors $(R_1, \ldots, R_n)$ as

$$P(i_I \mid i_1, \ldots, i_n) = \begin{cases} 
\Phi \left( \frac{0.5 - \mu_{i_1 \ldots i_n}}{\sigma_e} \right), & i_I = 0, \\
\Phi \left( \frac{i_I + 0.5 - \mu_{i_1 \ldots i_n}}{\sigma_e} \right) - \Phi \left( \frac{i_I - 0.5 - \mu_{i_1 \ldots i_n}}{\sigma_e} \right), & 0 < i_I < 9, \\
\Phi \left( \frac{8.5 - \mu_{i_1 \ldots i_n}}{\sigma_e} \right), & i_I = 9
\end{cases},$$

where $\Phi$ denotes the distribution function of the standard normal distribution.

\[B.2\]
B.2 Risk Index

Table B.1: Parameter needs for specification of the probabilities of a risk index complex.

<table>
<thead>
<tr>
<th>For each risk factor, $R_k, k \in {1, \ldots, n}$</th>
<th>Parameter</th>
<th>Index values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marginal distribution of states $P(i_k)$</td>
<td>$i_k \in {1, \ldots,</td>
<td>R_k</td>
</tr>
<tr>
<td>Additive effect of state $i_k$ on the risk index $\rho^k_{i_k}$</td>
<td>$i_k \in {1, \ldots,</td>
<td>R_k</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>For the risk index, $I$</th>
<th>Parameter</th>
<th>Index values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>$\mu$</td>
<td></td>
</tr>
<tr>
<td>Conditional standard deviation</td>
<td>$\sigma_e$</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>For each disease, $D_k, k \in {1, \ldots, m}$</th>
<th>Parameter</th>
<th>Index values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>$\delta^0_k$</td>
<td></td>
</tr>
<tr>
<td>Slope</td>
<td>$\delta^1_k$</td>
<td></td>
</tr>
</tbody>
</table>

B.2.3 A model for the probabilities of the diseases

As concerns the diseases, $D_1, \ldots, D_m$, the logistic transform $Y^k_{i_I} = \ln \frac{p_k}{1-p_k}$ of the conditional probability, $p$, of an arbitrary pig to have the $k$th disease (i.e. $j_k = \text{"Yes"}$) is, for a given state $i_I$ of the risk index $I$, modeled as

$$Y^k_{i_I} = \delta^0_k + \delta^1_k i_I,$$

(B.3)

where $\delta^0_k$ and $\delta^1_k$ are parameters specific for the $k$th disease. Accordingly, we have that the probability of the pig being diseased is

$$P(j_k = \text{"Yes"} \mid i_I) = \frac{1}{1 + e^{-Y^k_{i_I}}},$$

(B.4)

and the corresponding probability of the pig being healthy is just

$$P(j_k = \text{"No"} \mid i_I) = 1 - P(j_k = \text{"Yes"} \mid i_I).$$

(B.5)

The model (B.3) ensures (through $\delta^0_k$) that different diseases may have different base prevalence for the same value of the risk index and (through $\delta^1_k$) that they may be more or less sensitive to changes in the risk level of the herd.

It should be noticed, that the level of the risk factor on the 0 to 9 scale (i.e. the parameter $\mu$) may be set arbitrarily by simultaneous adjustment of the intercepts $\delta^0_1, \ldots, \delta^0_m$ of the $m$ diseases. It is, therefore, easy to ensure in the final calibration of the model that the prior mean of the risk index (before any observations are made) is 4.5.

B.2.4 Parameter needs

In order to specify all the probabilities of the complex, we need the information specified in Table B.1. When those parameters are available, the conditional probabilities of $I$ and $D_1, \ldots, D_m$ are calculated as described in the previous sections.
B.2.5 Elicitation of probabilities

Obtaining the indirect quantitative information

As illustrated in Fig. B.3, the effects of the risk factors on the diseases are assumed to be entirely expressed through a risk index node. Since this node is a conceptual latent model construct, it will not be possible to find any direct help in the literature for elicitation of the parameters of Table B.1. Neither will it be possible to ask experts for help, since the concept of a risk index is just a trick to model the influence of the risk factors in a consistent and compact way. The parameters have therefore been identified in an indirect way described in the following.

Instead of searching the literature and asking the experts about the causal edges of Fig. B.3, quantitative information about the direct effects of each of the risk factors on each of the diseases is requested from the experts and/or the literature. This is illustrated through the dashed arrows of Fig. B.4.

![Diagram of risk index complex](image)

Figure B.4: The risk index complex as defined in Fig. B.3. Causal edges shown with bold arrows are links from which we do not have quantitative information from the literature or expert opinions. Edges shown as dashed arrows are the quantitative information we can obtain.

Thus, for each of the dashed arrows from risk factor $R_i$ to disease $D_j$ of the figure, literature or experts will provide us with a conditional probability table specifying $P(D_j | R_i)$. In addition, a marginal probability table $P(R_i)$ is obtained from the literature, the experts or industry data bases.

Based on those probabilities, the parameters of Table B.1 are determined in such a way that they create the best possible fit to the expert/literature supplied conditional probabilities under the constraint that all effects of the risk factors are expressed through the common risk index (the hypothesis variable $I$) as shown in Fig. B.3. A combination of fitting a linear model and applying a general optimization function based on the Nelder-Mead algorithm is used (Nelder and Mead).
Combining the quantitative information into a data set

The first step is to combine the quantitative information into a data set which is afterwards used for estimation of the parameters of Table B.1.

For each combination of risk factor \( R_k \) and disease \( D_k' \), a mean prevalence \( P_\mu(D_k', R_k) \) may be determined as

\[
P_\mu(D_k', R_k) = \sum \Pr(D_k' = \text{"Yes"} \mid R_k = i_k) \Pr(R_k = i_k).
\]

Since we are dealing with expert supplied information we cannot expect full consistency with the same mean prevalence of the disease based on all risk factors. In other words, for a disease \( D_k' \) and two risk factors \( R_{k_1} \) and \( R_{k_2} \), we expect that

\[
P_\mu(D_k', R_{k_1}) \neq P_\mu(D_k', R_{k_2}).
\]

We therefore need to choose one of the risk factors and define it as the base. It is natural to choose the risk factor of which we have the highest confidence in the probabilities. We shall in the following refer to the chosen base as \( R_{k_0} \).

For each disease we now combine the conditional probability tables of all risk factors into a common conditional probability table using the following assumptions:

- The base, \( R_{k_0} \), defines the common mean of the prevalence.
- The risk factors are mutually independent.
- The effects of the risk factors are additive on the logistic scale.

In the following we shall throughout use the symbol \( \hat{Y} \) to denote the logistic transform of a corresponding probability \( P \). In other words, \( \hat{Y} = \ln(P/(1-P)) \), and for instance, the notation \( \hat{Y}(D_k' = \text{"Yes"}) \) denotes the logistic transform of the probability \( P(D_k' = \text{"Yes"}) \). Similarly, we let \( \hat{Y}_\mu \) denote the logistic transform of the mean prevalence \( P_\mu \).

Using the above assumptions we may calculate the logistic transform of the combined conditional probability table for the disease \( D_k' \) as follows:

\[
\hat{Y}(D_k' = \text{"Yes"} \mid i_1, \ldots, i_n) = \hat{Y}_\mu(D_k', R_{k_0}) + \sum_{k=1}^{n} [\hat{Y}(D_k' = \text{"Yes"} \mid i_k) - \hat{Y}_\mu(D_k', R_k)],
\]

where \( (i_1, \ldots, i_n) \) is a configuration of \( (R_1, \ldots, R_n) \). This is done for all configurations and all diseases and the result is a data set as shown in Table B.2.
Table B.2: Example of a data set for estimation of the parameters of Table B.1. It is assumed that there are two diseases and three risk factors each having two states. A simplifying notation $Y_{k,i_1,...,i_n}$ is used for $Y(D_k = "Yes" \mid i_1, \ldots, i_n)$.

<table>
<thead>
<tr>
<th>$Y_{k,i_1,...,i_n}$</th>
<th>Disease (k)</th>
<th>$R_1$</th>
<th>$R_2$</th>
<th>$R_3$</th>
</tr>
</thead>
<tbody>
<tr>
<td>$Y_{1,111}$</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>$Y_{1,112}$</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>$Y_{1,121}$</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>$Y_{1,122}$</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>$Y_{1,211}$</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>$Y_{1,212}$</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>$Y_{1,221}$</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>$Y_{1,222}$</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>$Y_{2,111}$</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>$Y_{2,112}$</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>$Y_{2,121}$</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>$Y_{2,122}$</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>$Y_{2,211}$</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>$Y_{2,212}$</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>$Y_{2,221}$</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>$Y_{2,222}$</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

Fitting a linear model

Having constructed a data set as shown in Table B.2 a linear model is fitted as follows:

$$
Y_{k,i_1,...,i_n} = \mu + \alpha_k + \rho^1_{i_1} + \rho^2_{i_2} + \ldots + \rho^n_{i_n} + \epsilon_{k,i_1,...,i_n} = \\
= \mu_k + \rho^1_{i_1} + \rho^2_{i_2} + \ldots + \rho^n_{i_n} + \epsilon_{k,i_1,...,i_n}, \tag{B.7}
$$

where

- $Y_{k,i_1,...,i_n}$ is the logistic transform of the probability of diseases $k$. The value is known from the first column of Table B.2.
- $\alpha_k$ is the fixed effect of disease $k$.
- $\mu_k = \mu + \alpha_k$ is the disease specific intercept.
- $\rho^k_{i_k}$ is the fixed effect of state $i_k$ of risk factor $R_k$, $k = 1, \ldots, n$.
- $\epsilon_{k,i_1,...,i_n}$ is the residual.

Comparing the model (B.7) with the parameter needs of Table B.1 we notice that the additive effects of the risk factors $\rho^k_{i_k}$ are directly determined (even though they must be adjusted appropriately in order to transform them from the logistic scale.
B.2 Risk Index

assumed in Eq. (B.7) to the arbitrary 0-9 scale of the risk factor). The two intercepts \( \mu \) and \( \delta^0_k \) from Table B.1 correspond, in principle, directly to the parameters \( \mu \) and \( \alpha_k \) of model (B.7) even though (as previously mentioned) the intercepts can be set arbitrarily.

The only parameters from Table B.1 that are not determined by fitting the model (B.7) are the disease specific slopes \( \delta^1_k \). In order to fit the slopes, we shall introduce an additional disease specific parameter \( \lambda_k \) and modify the model to

\[
Y_{k,i_1...i_n} = \mu_k + \lambda_k (\rho_{i_1}^1 + \rho_{i_2}^2 + \ldots \rho_{i_n}^n) + \epsilon_{k,i_1...i_n},
\]

where the \( \lambda_k \) parameters ensure that the sensitivity towards changes in the risk factors differs between diseases. The model (B.8) expresses the possible presence of interactions between the risk index (i.e. the sum of the risk factor effects) and the diseases. Thus, the values \( \lambda_1 = \ldots = \lambda_m = 1 \) correspond to a situation without interactions.

Optimizing the fit

The values of \( \lambda_k \) cannot be determined directly by regression analysis, but for given values of \( \lambda_k \), we may rearrange Eq. (B.8) to

\[
\frac{Y_{k,i_1...i_n}}{\lambda_k} = \frac{\mu_k}{\lambda_k} + \frac{\rho_{i_1}^1}{\lambda_k} + \frac{\rho_{i_2}^2}{\lambda_k} + \ldots \frac{\rho_{i_n}^n}{\lambda_k} + \frac{\epsilon_{k,i_1...i_n}}{\lambda_k}.
\]

Now, define

\[
Y_{k,i_1...i_n}(\lambda_k) = \frac{Y_{k,i_1...i_n}}{\lambda_k}, \quad \mu_k(\lambda_k) = \frac{\mu_k}{\lambda_k}, \quad \text{and} \quad \epsilon'_{k,i_1...i_n} = \frac{\epsilon_{k,i_1...i_n}}{\lambda_k}.
\]

We may then express the latter model as

\[
Y_{k,i_1...i_n}(\lambda_k) = \mu_k(\lambda_k) + \rho_{i_1}^1 + \rho_{i_2}^2 + \ldots \rho_{i_n}^n + \epsilon'_{k,i_1...i_n}
\]

which, for known \( \lambda_k \), is an ordinary linear model, where the parameters \( \mu_k(\lambda_k) \) and \( \rho_{i_1}^1, \ldots, \rho_{i_n}^n \) may be estimated by standard methods.

In order to find the value set \( (\lambda_1, \ldots, \lambda_m) \) resulting in the best fit of the model (B.9) a general purpose optimization by use of the Nelder-Mead algorithm is performed. A function fitting the model (B.9) and returning the residual sum of squared residuals is defined in R as shown in Appendix C (Fig. C.2). A call to the built-in R function \texttt{optim} then determines the parameters \( \lambda_1, \ldots, \lambda_m \) minimizing the sum of squares of the residuals. As initial values we use \( \lambda_1 = \ldots = \lambda_m = 1 \).

The optimized values of \( \lambda_1, \ldots, \lambda_m \) enable us to determine the slope parameters \( \delta^1_k \) of Table B.1 as shown in the next section.

Final adjustment of parameters

The final adjustment of parameters has the following steps:

1. Choose a disease \( k' \) as the base reference:
Appendix B: Elicitation of probabilities

(a) Define a lower and an upper bound for the logistic transform of the prevalence in a herd. Denote them as $b_l$ and $b_u$, respectively.

(b) Identify $b_l$ with risk index 0 and $b_u$ with risk index 9.

(c) Define $b_l$ as an initial value of the intercept, i.e. put $\delta_{k'}^0 = b_l$.

(d) Define an initial slope $\delta_{k'}^1$ as $\delta_{k'}^1 = \frac{b_u - b_l}{9}$ (because the risk index varies from 0 to 9).

2. For each disease, $D_k$, $k \in \{1, \ldots, m\}$:

(a) Define the preliminary intercept to $\hat{\delta}_k^0 = \lambda_k (\delta_{k'}^0 + \mu_k (\lambda_k') - \mu_{k'} (\lambda_{k'}'))$.

(b) Redefine the slope to $\delta_{k'}^1 = \lambda_{k'} \delta_{k'}^1$.

(c) Calculate the conditional probabilities by use of Eqs. (B.4) and (B.5).

3. For each risk factor, $R_k$, $k \in \{1, \ldots, n\}$:

(a) For each state $i_k \in \{1, \ldots, |R_k|\}$ define the additive effect as $\rho_{i_k}^k = \hat{\rho}_{i_k}^k / \delta_{k'}^1$, where the $\hat{\rho}_{i_k}^k$ are the estimated values fitted by the model (B.9).

4. For the risk index $I$:

(a) Define an initial intercept $\pi$ as $\pi = (\mu_{k'} (\lambda_{k'}) - \delta_{k'}^0) / \delta_{k'}^1$, where $\mu_{k'} (\lambda_{k'})$ is estimated from the model (B.9).

(b) Calculate the conditional probability table of the risk index as described by Eqs. (B.2).

When all probabilities have been entered into the model, and it has been compiled, the prior mean $E(I)$ of the risk index is found, and the intercept of $I$ is adjusted in order to have a prior mean of 4.5. If an adjustment of $\Delta \mu = 4.5 - E(I)$ is necessary, we define the final intercept $\mu$ of Eq. (B.1) as

$$\mu = \pi + \Delta \mu.$$ 

The intercepts for the individual diseases $\delta_1^0, \ldots, \delta_m^0$ are adjusted accordingly as

$$\delta_k^0 = \hat{\delta}_k^0 - \Delta \mu \delta_{k'}^1$$

for $k = 1, \ldots, m$. Finally, the conditional probability tables of $I$ and $D_1, \ldots, D_m$ are recalculated.
B.3 The Inherited complex

This section will describe the methodology for modeling the Herd class node “Inherited” and “Gain”, and the Pig class nodes “OCM” and “OCD”. Fig. B.5 shows a subfigure of the model representing the Inherited complex. The core of the complex corresponds directly to the general layout shown in Fig. B.3 with “Breed” and “Gain” as risk factors at herd level and the two disease nodes “OCD” and “OCM” at pig level. The procedure used for this part of the complex is completely along the general guidelines of Section B.2.

On the other hand, the present complex is slightly more complicated due to the presence of causal edges from “Breed” and “Feed strat” to “Gain” at herd level (the encircled part), and the causal edges from “Gender” and “LMP” to “OCD” and “OCM” at pig level. Those edges are dealt with separately.

Figure B.5: The Inherited complex. Causal edges shown with bold arrows are links from which we do not have quantitative information from the literature or expert opinions. Edges shown as dashed arrows are the quantitative information we can obtain. The encircled part contributes to the estimation of the node Gain.

B.3.1 Probabilities for the Inherited complex

Prior estimates

Initially, the marginal distributions for Breed, Feeding strategy, lean meat percentage (LMP) and Gender are defined based on prior knowledge regarding the Danish finisher production (Tables B.3-B.6).
Table B.3: Marginal distribution of Breed

<table>
<thead>
<tr>
<th>Breed</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crossbreed</td>
<td>0.99</td>
</tr>
<tr>
<td>Purebreed</td>
<td>0.01</td>
</tr>
</tbody>
</table>

Table B.4: Marginal distribution of Feeding strategy

<table>
<thead>
<tr>
<th>Feeding strategy</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ad libitum</td>
<td>0.50</td>
</tr>
<tr>
<td>Restricted</td>
<td>0.50</td>
</tr>
</tbody>
</table>

Table B.5: Marginal distribution of lean meat percentage (LMP)

<table>
<thead>
<tr>
<th>LMP</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤ 57</td>
<td>0.16</td>
</tr>
<tr>
<td>58</td>
<td>0.11</td>
</tr>
<tr>
<td>59</td>
<td>0.15</td>
</tr>
<tr>
<td>60</td>
<td>0.16</td>
</tr>
<tr>
<td>61</td>
<td>0.15</td>
</tr>
<tr>
<td>62</td>
<td>0.11</td>
</tr>
<tr>
<td>≥ 63</td>
<td>0.16</td>
</tr>
</tbody>
</table>

Table B.6: Marginal distribution of Gender

<table>
<thead>
<tr>
<th>Gender</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female</td>
<td>0.50</td>
</tr>
<tr>
<td>Castrate</td>
<td>0.50</td>
</tr>
</tbody>
</table>
**B.3 Inherited complex**

**Conditional probabilities elicited from experts**

The conditional probabilities $P(OCM|\text{Breed})$ and $P(OCD|\text{Breed})$ have been elicited from expert opinions. The resulting probabilities are shown in the Tables B.7 and B.8. Experts have also provided prior estimates regarding the daily weight gain for purebred and crossbred pigs, and for pigs fed ad libitum and restricted. Hence, the daily weight gain of crossbred pigs fed ad libitum is estimated to be 873 gram per day. Purebred pigs are believed to have a reduction in the daily weight gain of 50 gram compared to crossbred pigs, and pigs fed ad libitum tend to have an increase in the daily weight gain of 50 gram compared to pigs on restricted feeding. These estimates will be used for the construction of the Gain node.

**Table B.7: Conditional probability table (CPT) for $P(OCM|\text{Breed})$**

<table>
<thead>
<tr>
<th>OCM</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cross breed</td>
<td>0.32</td>
<td>0.68</td>
</tr>
<tr>
<td>Pure breed</td>
<td>0.33</td>
<td>0.67</td>
</tr>
</tbody>
</table>

**Table B.8: Conditional probability table (CPT) for $P(OCD|\text{Breed})$**

<table>
<thead>
<tr>
<th>OCM</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cross breed</td>
<td>0.79</td>
<td>0.21</td>
</tr>
<tr>
<td>Pure breed</td>
<td>0.82</td>
<td>0.18</td>
</tr>
</tbody>
</table>

**Conditional probabilities elicited from the literature**

From a study by Busch et al. (2007) the conditional probabilities $P(OCM|\text{Gain})$, $P(OCD|\text{Gain})$ have been elicited. The result of this study showed that for each 100 gram increase in the average daily weight gain, the odds are 1.14 higher ($OR = 1.14$) for developing OCM and 1.21 higher ($OR = 1.21$) for developing OCD in the finishing period. The method of transforming the odds ratio to a probability has been described in Otto and Kristensen (2004). We have,

$$P_{yes} = \frac{(P_{no}/(1 - P_{no}))OR}{1 + (P_{no}/(1 - P_{no}))OR}, \quad (B.10)$$

where $P_{no}$ is the probability of OCM or OCD when a pig has no increase in the ADG. OR is the odds ratio for OCM or OCD when the pig has an increase in the ADG of 100 g. It is necessary to specify $P_{no}$ for both OCM and OCD. For this study we choose the following probabilities for OCM and OCD, respectively: $P_{no} = 0.5$ and $P_{no} = 0.2$.

An ADG of 600g will be used as the baseline value in these calculations, and the probabilities for OCM and OCD for different values of ADG are calculated...
using Formula B.10. Hence, the probabilities of OCM and OCD for the following values of ADG: $P_{600g}$, $P_{700g}$, $P_{800g}$, $P_{900g}$, $P_{1000g}$ are shown in Tables B.9 and B.10.

Table B.9: Conditional probability table (CPT) for $P(OCM|Gain)$

<table>
<thead>
<tr>
<th>OCM</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>600g</td>
<td>0.50</td>
<td>0.50</td>
</tr>
<tr>
<td>700g</td>
<td>0.47</td>
<td>0.53</td>
</tr>
<tr>
<td>800g</td>
<td>0.43</td>
<td>0.57</td>
</tr>
<tr>
<td>900g</td>
<td>0.40</td>
<td>0.60</td>
</tr>
<tr>
<td>1000g</td>
<td>0.37</td>
<td>0.63</td>
</tr>
</tbody>
</table>

Table B.10: Conditional probability table (CPT) for $P(OCD|Gain)$

<table>
<thead>
<tr>
<th>OCD</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>600g</td>
<td>0.80</td>
<td>0.20</td>
</tr>
<tr>
<td>700g</td>
<td>0.77</td>
<td>0.23</td>
</tr>
<tr>
<td>800g</td>
<td>0.73</td>
<td>0.27</td>
</tr>
<tr>
<td>900g</td>
<td>0.69</td>
<td>0.31</td>
</tr>
<tr>
<td>1000g</td>
<td>0.65</td>
<td>0.35</td>
</tr>
</tbody>
</table>

**B.3.2 Methodology for constructing the nodes**

**Gain node**

The Gain node has 5 states corresponding to 600, 700, ..., 1000g average daily gain for the herd, respectively, as shown in Tables B.9 and B.10.

The values for the Gain node can immediately be provided from the expert opinions.

**Standard deviation between farms ($\sigma$)** 70

**Average gain ($\mu$)** 873

**Effect of pure breed** -50

**Effect of restricted feeding** -50

Based on this information the conditional distribution of the Gain node is calculated from a normal distribution with the specified average (corrected for effects of pure breed and/or restricted feeding) and the specified standard deviation.

The following intervals define the states of the node Gain:
The conditional probability for the state “600g” is calculated as

\[ P(600g) = \Phi \left( \frac{650 - \mu_c}{70} \right) \]

where \( \Phi \) is the distribution function of the standard normal distribution, and \( \mu_c \) is the corrected mean. Accordingly, the conditional probability for the state “700g” is calculated as

\[ P(700g) = \Phi \left( \frac{750 - \mu_c}{70} \right) - P(600g) \]

and similarly for other states.

**Inherited node**

The combined conditional probability tables (CPT) for OCM and OCD given Breed and Gain, \( P(\text{OCM}|\text{Breed, Gain}) \) and \( P(\text{OCD}|\text{Breed, Gain}) \), are calculated as described in Section B.2.5 with Eq. (B.6) as the central formula. We must decide on the base risk, \( R_{b0} \), and we choose “Breed” for calculation of the base prevalence of both diseases, “OCD” and “OCM”. The combined CPTs are then calculated on the logistic scale as defined in Eq. (B.6). The R-code for the development of the combined CPTs is given in Appendix C (Fig. C.1), and the resulting combined probability values for the two disease nodes are shown in Table B.11 which corresponds directly to Table B.2.

Using the values of Table B.11 we fit a regression model corresponding to Eq. (B.7) for the “Inherited” node describing the relation between the logistic values of the combined probabilities and the effects: Disease (OCM, OCD), Breed (Crossbred, Purebred) and Gain (600-1000g). Hence the regression model is defined as:

\[
Y_{k,\text{i}1\text{i}2} = \mu_k + \alpha_k + \rho_{11} x_{k,\text{i}1\text{i}2} + \rho_{12}^2 x_{k,\text{i}1\text{i}2} + \epsilon_{k,\text{i}1\text{i}2},
\]

where

- \( Y_{k,\text{i}1\text{i}2} \) is the logistic value of the probability of inherited leg disorders for pigs with Disease \( k \), the Breed \( \text{i1} \) and the daily gain \( x_{k,\text{i}1\text{i}2} \). The values of \( Y_{k,\text{i}1\text{i}2} \) are shown in the leftmost column of Table B.11.
Table B.11: The combined conditional probabilities for OCM and OCD given Breed and Gain. The probabilities are presented on the logistic scale, $Y_{k, i_1 i_2}$, calculated directly from Eq. (B.6) as well as on the corresponding probability scale, $P(k | i_1 i_2)$.

| $Y_{k, i_1 i_2}$ | $P(k | i_1 i_2)$ | Disease, $k$ | Breed, $i_1$ | Gain, $i_2$ | OCM/OCD |
|------------------|------------------|--------------|--------------|------------|---------|
| 0.5531           | 0.6349           | OCM          | Crossbred    | 600        |
| 0.6732           | 0.6622           | OCM          | Crossbred    | 700        |
| 0.8350           | 0.6974           | OCM          | Crossbred    | 800        |
| 0.9586           | 0.7228           | OCM          | Crossbred    | 900        |
| 0.5075           | 0.6242           | OCM          | Purebred     | 600        |
| 0.6277           | 0.6520           | OCM          | Purebred     | 700        |
| 0.7894           | 0.6877           | OCM          | Purebred     | 800        |
| 0.9130           | 0.7136           | OCM          | Purebred     | 900        |
| -1.6259          | 0.1644           | OCD          | Crossbred    | 600        |
| -1.4479          | 0.1903           | OCD          | Crossbred    | 700        |
| -1.2342          | 0.2254           | OCD          | Crossbred    | 800        |
| -1.0397          | 0.2612           | OCD          | Crossbred    | 900        |
| -1.8173          | 0.1398           | OCD          | Purebred     | 600        |
| -1.6393          | 0.1626           | OCD          | Purebred     | 700        |
| -1.4256          | 0.1938           | OCD          | Purebred     | 800        |
| -1.2311          | 0.2260           | OCD          | Purebred     | 900        |
In the regression analysis, the values of $x_{k,i_1,i_2}$ are set to 0, 1, ..., 5 corresponding to 600, 700, ..., 1000 g average daily gain.

The fit of the regression is afterwards optimized by the introduction of disease specific factors, $\lambda_1 = \lambda_{\text{OCD}}$ and $\lambda_2 = \lambda_{\text{OCM}}$, ensuring disease specific sensitivities towards changes in the risk factors as described in Section B.2.5. For the optimal $\lambda$-factors the parameter estimates of a model like the one shown in (B.9) are used.

Having performed the optimization, the sum of the squared residuals is 0.03999251 compared to 0.03999465 for the initial values $\lambda_{\text{OCD}} = \lambda_{\text{OCM}} = 1$. It can, therefore, be concluded that the optimized model gives a slightly better model fit. The result of the optimized model is given in Table B.12 and the R code is presented in Fig. C.2 (Appendix C). The optimized model gives the following values for $\lambda$:

$$\lambda_{\text{OCD}} = 1.001084$$

$$\lambda_{\text{OCM}} = 1.089095$$

<table>
<thead>
<tr>
<th>Table B.12: Result of the optimized model</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parameter</td>
</tr>
<tr>
<td>------------</td>
</tr>
<tr>
<td>Intercept, OCD</td>
</tr>
<tr>
<td>Intercept, OCM</td>
</tr>
<tr>
<td>Breed(Pure breed)</td>
</tr>
<tr>
<td>Gain</td>
</tr>
</tbody>
</table>

We are now ready to adjust the parameters as described in Section B.2.5:

1. As the base reference disease, $k'$, we choose OCD, and

   (a) Define the lower bound for the prevalence in a herd as 7% and the upper bound as 30%. On the logistic scale this leads to a lower bound of $b_l = -2.6$ and an upper bound of $b_u = -0.8$

   (b) On the arbitrary 0 to 9 scale of the risk index, we identify the value 0 with -2.6 on the logistic scale, and the value 9 with -0.8 on the logistic scale as shown in Table B.13
(c) We define the initial intercept for OCD as $\delta_0^{OCD} = y = -2.6$.
(d) We define the initial slope for OCD as $\delta_1^{OCD} = \frac{b_u - b_l}{9} = \frac{-0.8 - (-2.6)}{9} = 0.2$.

2. For each disease, OCD and OCM we:

(a) Define the preliminary intercept:
   i. $\hat{\delta}_0^{OCD} = \lambda_{OCD} \delta_0^{OCD} = 1.001084 \times (-2.6) = -2.6028$.
   ii. $\hat{\delta}_0^{OCM} = \lambda_{OCM} (\delta_0^{OCD} + \mu_{OCM} (\lambda_{OCM} - \mu_{OCD} (\lambda_{OCD})) = 1.089095 \times (-2.6 + 0.54046 - (-1.62414)) = -0.4742$.

(b) Redefine the slopes:
   i. $\delta_1^{OCD} = \lambda_{OCD} \delta_1^{OCD} = 1.001085 \times 0.2 = 0.2002$.
   ii. $\delta_1^{OCM} = \lambda_{OCM} \delta_1^{OCD} = 1.089095 \times 0.2 = 0.2178$.

(c) The resulting conditional probability tables of OCD and OCM given risk index may then be calculated by Eqs. (B.4) and (B.5).

3. For each risk factor, “Breed” and “Gain”, we define the additive effects from the estimates of Table B.12.

“Breed” The effect of “Crossbred” is $\rho_1^1 = 0$ according to the table, and the corrected effect of “Purebred” is $\hat{\rho}_2^1 = \frac{\hat{\rho}_2^1}{\delta_1^{OCD}} = -0.0.11872 / 0.2 = -0.5932$.

“Gain” A linear effect of gain was assumed, and the estimated coefficient $\hat{\rho}_2^2$ is known from the table. This leads to the following additive effects of “Gain”:
- **600 g:** $\hat{\rho}_2^2 = 0$.
- **700 g:** $\hat{\rho}_2^2 = \frac{\hat{\rho}_2^2}{\delta_1^{OCD}} = 0.16738 / 0.2 = 0.8369$.
- **800 g:** $\hat{\rho}_2^2 = 2 \frac{\hat{\rho}_2^2}{\delta_1^{OCD}} = 2 \times 0.16738 / 0.2 = 1.6738$.
- **900 g:** $\hat{\rho}_2^2 = 3 \frac{\hat{\rho}_2^2}{\delta_1^{OCD}} = 3 \times 0.16738 / 0.2 = 2.5107$.
- **1000 g:** $\hat{\rho}_2^2 = 4 \frac{\hat{\rho}_2^2}{\delta_1^{OCD}} = 4 \times 0.16738 / 0.2 = 3.3476$.

4. For the “Inherited” node (the risk index) we

(a) Define the initial intercept as $\mu = (\mu_{OCD} (\lambda_{OCD}) - \delta_0^{OCD}) / \delta_1^{OCD} = (-1.62414 - (-2.6)) / 0.2 = 4.8793$.

(b) The resulting conditional probability table of the “Inherited” node may now be calculated according to Eq. (B.2) assuming a standard deviation of $\sigma_e = 1.0$.

Having entered all parameters and compiled the model, it turned out that the prior mean of the “Inherited” node, was $E(\text{Inherited}) = 6.9244$. Since this prior mean by definition must be 4.5, we notice that $\Delta \mu = 4.5 - 6.9244 = -2.4244$. 

B.3 Inherited complex

Table B.13: The arbitrary scale used in the Inherited node and the corresponding logistic scale. Hence, 7% corresponds to the index 0 and 30% corresponds to the index 9. The corresponding logistic values of 7% and 30%, respectively, are \( b_l = -2.6 \) and \( b_u = -0.8 \).

<table>
<thead>
<tr>
<th>Risk index</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
</tr>
</thead>
<tbody>
<tr>
<td>Logistic</td>
<td>-2.6</td>
<td>-2.4</td>
<td>-2.2</td>
<td>-2.0</td>
<td>-1.8</td>
<td>-1.6</td>
<td>-1.4</td>
<td>-1.2</td>
<td>-1.0</td>
<td>-0.8</td>
</tr>
</tbody>
</table>

Thus, according to Section B.2.5, the final intercept of the “Inherited” node becomes \( \mu = \overline{\mu} + \Delta \mu = 4.8793 + (-2.4222) = 2.4549 \).

With reference to Eq. (B.1) the parameters for calculation of the CPT of the “Inherited” risk index may be summarized as follows:

**Intercept:** \( \mu = 2.4549 \).

**Effect of breed:**
- **Crossbred:** \( \rho_1^1 = 0 \).
- **Purebred:** \( \rho_2^1 = -0.5932 \).

**Effect of daily gain:**
- 600 g: \( \rho_1^2 = 0 \).
- 700 g: \( \rho_2^2 = 0.8369 \).
- 800 g: \( \rho_3^2 = 1.6738 \).
- 900 g: \( \rho_4^2 = 2.5107 \).
- 1000 g: \( \rho_5^2 = 3.3476 \).

**Standard deviation of the residual:** \( \sigma_e = 1.0 \).

For a given configuration of the parent nodes, the conditional distribution of the inherited node is calculated from a normal distribution in the same way as described for the Gain node. The conditional mean is calculated from the information on “Breed” and “Gain” using the effects specified above. The conditional standard deviation of the risk index basically express the precision of the expert knowledge. This standard deviation is specified as input to the model, and the default value used is a standard deviation of 1.0 (measured on the arbitrary scale from 0 to 9).

As an example, we will consider the situation where evidence regarding Purebred and a daily weight gain of 800g is entered in the model. In this situation the corrected mean of the risk index for “Inherited” is

\[
\mu_c = 2.4549 + (-0.5932) + 1.6738 = 5.9599
\]

and this value will be used as the corrected mean for the given situation.

The following intervals define the states of the “Inherited” node:
The conditional probability for the state “0” is calculated as
\[
P(0) = \Phi\left(\frac{0.5 - \mu_c}{1.0}\right)
\]
where \(\Phi\) is the distribution function of the standard normal distribution, and \(\mu_c\) is the corrected mean. Accordingly, the conditional probability for the state “1” is calculated as
\[
P(1) = \Phi\left(\frac{1.5 - \mu_c}{1.0}\right) - P(0)
\]
and similarly for other states.

**OCD and OCM**

Because of the additional parents “Gender” and “LMP” of the “OCD” and “OCM” nodes we need to extend the standard disease model presented in Eq. (B.3).

From the literature, we know that for each percentage point increase in the LMP the OR for OCD is 1.05 (Busch et al., 2007). Thus, on the logistic scale, the effect of one percentage point increase in the LMP is \(\ln 1.05 = 0.04879\). The node LMP is defined to have 6 states corresponding to an LMP of 57, 58, \ldots, 63.

From the literature, we know that castrates have 1.31 higher odds (OR = 1.31) for developing OCD compared to females. On the logistic scale, an OR of 1.31 corresponds to a difference between the two genders of \(\ln 1.31 = 0.2700\). The full model for the OCD node therefore becomes
\[
Y_{i,i_1,i_2}^{\text{OCD}} = \delta_{i_1}^{OCD} + \delta_{i_2}^{OCD} i_f + \delta_{\text{OCD},i_1}^{OCD} + \delta_{\text{OCD}}^{3}(x_{i_2} - 57), \quad (B.12)
\]
where
- \(Y_{i,i_1,i_2}^{\text{OCD}}\) is the logistic transform of the probability of OCD for a pig of gender \(i_1\), an LMP of \(x_{i_2}\) in a herd with risk index \(i_f\).
- \(\delta_{OCD}^0\) is the intercept.
- \(\delta_{OCD}^1 = 0.2002\) is the linear effect of risk index (as determined in the previous section).
B.3 Inherited complex

- \( \delta^2_{OCD,i} \) is the additive effect of gender (\( i \in \{F, C\} \), i.e. female or castrate), where \( \delta_{OCD,F}^2 = -0.2700/2 = -0.1350 \) and \( \delta_{OCD,C}^2 = 0.27003/2 = 0.1350 \) (since the difference must be 0.2700 as mentioned above).

- \( \delta^3_{OCD} = 0.04879 \) is the linear effect of LMP as defined above.

In the final adjustment of the intercept for the OCD node we assign the expert provided levels to an LMP of 60% and to average values across genders (or 3 percentage points above the lowest value). Thus the previously determined preliminary intercept \( \hat{\delta}_{OCD}^0 = -2.6028 \) must be adjusted accordingly to \( \delta_{OCD}^0 = \delta_{OCD}^0 - 3\delta_{OCD}^3 = -2.6028 - 3 \times 0.04879 = -2.7492 \). As mentioned in previous section, the prior mean of the risk index had to be adjusted by \( \Delta \mu = -2.4244 \). Thus, according to Section B.2.5, the final intercept must be adjusted to \( \tilde{\delta}_{OCD}^0 = \delta_{OCD}^0 - \Delta \mu \delta_{OCD}^1 = -2.7492 - (-2.4244) \times 0.2002 = -2.2638 \).

We may now summarize the information used for the CPT of the “OCD” node as follows:

**Intercept:** \( \delta_{OCD}^0 = -2.2638 \).

**Linear effect of risk index:** \( \delta_{OCD}^1 = 0.2002 \).

**Effect of gender:**
- **Female:** \( \delta_{OCD,F}^2 = -0.1350 \).
- **Castrate:** \( \delta_{OCD,C}^2 = 0.1350 \).

**Linear effect of LMP:** \( \delta_{OCD}^3 = 0.04879 \).

From the literature, we know that for each percentage point increase in the LMP the OR for OCM is 1.03 (Busch et al., 2007). On the logistic scale the effect of one percentage point increase in the LMP is \( \ln 1.03 = 0.029559 \).

Busch et al. (2007) found that castrates have 1.17 higher odds \( (OR = 1.17) \) for developing OCM compared to females. On the logistic scale, an OR of 1.17 corresponds to a difference between the two genders of \( \ln 1.17 = 0.1570 \). The full model for the OCM node therefore becomes

\[
Y_{i,t,i_1i_2}^{OCM} = \delta_{OCM}^0 + \delta_{OCM}^1 i + \delta_{OCM,F}^2 + \delta_{OCM,C}^2 + \delta_{OCM}(x_{i_2} - 57). \quad (B.13)
\]

Using exactly the same procedure as for the “OCD” node, we may therefore adjust the intercept (for effect of LMP and \( \Delta \mu \)) and summarize the information used for the CPT of the “OCM” node as follows:

**Intercept:** \( \delta_{OCM}^0 = -0.0348 \).

**Linear effect of risk index:** \( \delta_{OCM}^1 = 0.2178 \).
Effect of gender:

**Female**: $\delta_{\text{OCM,F}}^2 = -0.0785$.

**Castrate**: $\delta_{\text{OCM,C}}^2 = 0.0785$.

**Linear effect of LMP**: $\delta_{\text{OCM}}^3 = 0.029559$.

### B.4 The Physical complex

This chapter will describe the methodology for modeling the Herd class node “Physical” and the Pig class nodes “ClawWall”, “ClawSole” and “Fracture”. Fig. B.6 shows a subfigure of the model representing the Physical complex. As it is seen, the structure is in this case completely analogous to Fig. B.3.

![Physical complex diagram](image)

Figure B.6: The Physical complex. Causal edges shown with bold arrows are links from which we do not have quantitative information from the literature or expert opinions. Edges shown as dashed arrows are the quantitative information we can obtain.

### B.4.1 Probabilities for the Physical complex

**Marginal distributions**

Initially, the marginal distributions for Straw, Floor and PenDen are defined based on prior knowledge regarding the distribution of herds in Denmark (Table B.14 - B.16).
Table B.14: Marginal distribution of supply of straw

<table>
<thead>
<tr>
<th>Straw</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>0.45</td>
</tr>
<tr>
<td>Sparse</td>
<td>0.45</td>
</tr>
<tr>
<td>Deep</td>
<td>0.10</td>
</tr>
</tbody>
</table>

Table B.15: Marginal distribution of floor type

<table>
<thead>
<tr>
<th>Floor</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Solid</td>
<td>0.25</td>
</tr>
<tr>
<td>Partially slatted</td>
<td>0.25</td>
</tr>
<tr>
<td>Fully slatted</td>
<td>0.50</td>
</tr>
</tbody>
</table>

Conditional probabilities elicited from experts

All conditional probabilities have been elicited from expert opinions. The resulting probabilities are shown in Tables B.17 - B.25

B.4.2 Methodology for constructing the nodes

Physical node

The combined CPT’s for “ClawWall”, “ClawSole” and “Fracture” given “Straw”, “PenDen” and “Floor” (i.e. \( P(\text{ClawWall}|\text{Straw}, \text{PenDen}, \text{Floor}) \), \( P(\text{ClawSole}|\text{Straw}, \text{PenDen}, \text{Floor}) \) and \( P(\text{Fracture}|\text{Straw}, \text{PenDen}, \text{Floor}) \)) are calculated as described in Section B.2.5 with Eq. (B.6) as the central formula. We must decide on the base risk, \( R_{b0} \), and we choose “Floor” for calculation of the base prevalence of all three diseases, “ClawWall”, “ClawSole” and “Fracture”. The combined CPTs are then calculated on the logistic scale as defined in Eq. (B.6). The R-code for the development of the combined CPTs is given in Appendix C, (Fig. C.3), and an excerpt of the resulting combined probability values for the three diseases are shown in Table B.26 which corresponds directly to Table B.2.

Using the values of Table B.26 we fit a regression model corresponding to Eq. (B.7) for the “Physical” node describing the relation between the logistic values of the combined probabilities and the effects: Disease(ClawWall, ClawSole, Fracture), Straw(No, Sparse, Deep), PenDen(Low, High), Floor(Solid, Partially slatted, Fully slatted). Hence, the regression model is defined as:

Table B.16: Marginal distribution of pen density

<table>
<thead>
<tr>
<th>Pen density</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>0.70</td>
</tr>
<tr>
<td>High</td>
<td>0.30</td>
</tr>
</tbody>
</table>
Table B.17: Conditional probability table (CPT) for $P(\text{ClawWall}|\text{Straw})$

<table>
<thead>
<tr>
<th>ClawWall</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>0.580</td>
<td>0.420</td>
</tr>
<tr>
<td>Sparse</td>
<td>0.577</td>
<td>0.423</td>
</tr>
<tr>
<td>Deep</td>
<td>0.793</td>
<td>0.207</td>
</tr>
</tbody>
</table>

Table B.18: Conditional probability table (CPT) for $P(\text{ClawSole}|\text{Straw})$

<table>
<thead>
<tr>
<th>ClawSole</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>0.577</td>
<td>0.423</td>
</tr>
<tr>
<td>Sparse</td>
<td>0.570</td>
<td>0.430</td>
</tr>
<tr>
<td>Deep</td>
<td>0.793</td>
<td>0.207</td>
</tr>
</tbody>
</table>

Table B.19: Conditional probability table (CPT) for $P(\text{Fracture}|\text{Straw})$

<table>
<thead>
<tr>
<th>Fracture</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>0.995</td>
<td>0.005</td>
</tr>
<tr>
<td>Sparse</td>
<td>0.995</td>
<td>0.005</td>
</tr>
<tr>
<td>Deep</td>
<td>0.997</td>
<td>0.003</td>
</tr>
</tbody>
</table>

Table B.20: Conditional probability table (CPT) for $P(\text{ClawWall}|\text{PenDen})$

<table>
<thead>
<tr>
<th>ClawWall</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>0.633</td>
<td>0.367</td>
</tr>
<tr>
<td>High</td>
<td>0.603</td>
<td>0.397</td>
</tr>
</tbody>
</table>

Table B.21: Conditional probability table (CPT) for $P(\text{ClawSole}|\text{PenDen})$

<table>
<thead>
<tr>
<th>ClawSole</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>0.630</td>
<td>0.370</td>
</tr>
<tr>
<td>High</td>
<td>0.567</td>
<td>0.433</td>
</tr>
</tbody>
</table>

Table B.22: Conditional probability table (CPT) for $P(\text{Fracture}|\text{PenDen})$

<table>
<thead>
<tr>
<th>Fracture</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>0.995</td>
<td>0.005</td>
</tr>
<tr>
<td>High</td>
<td>0.993</td>
<td>0.007</td>
</tr>
</tbody>
</table>
Table B.23: Conditional probability table (CPT) for \( P(\text{ClawWall}|\text{Floor}) \)

<table>
<thead>
<tr>
<th>ClawWall</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Solid</td>
<td>0.690</td>
<td>0.310</td>
</tr>
<tr>
<td>Partially slatted</td>
<td>0.583</td>
<td>0.417</td>
</tr>
<tr>
<td>Fully slatted</td>
<td>0.507</td>
<td>0.493</td>
</tr>
</tbody>
</table>

Table B.24: Conditional probability table (CPT) for \( P(\text{ClawSole}|\text{Floor}) \)

<table>
<thead>
<tr>
<th>ClawSole</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Solid</td>
<td>0.690</td>
<td>0.310</td>
</tr>
<tr>
<td>Partially slatted</td>
<td>0.573</td>
<td>0.427</td>
</tr>
<tr>
<td>Fully slatted</td>
<td>0.503</td>
<td>0.497</td>
</tr>
</tbody>
</table>

Table B.25: Conditional probability table (CPT) for \( P(\text{Fracture}|\text{Floor}) \)

<table>
<thead>
<tr>
<th>Fracture</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Solid</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>Partially slatted</td>
<td>0.995</td>
<td>0.005</td>
</tr>
<tr>
<td>Fully slatted</td>
<td>0.993</td>
<td>0.007</td>
</tr>
</tbody>
</table>

\[
Y_{k,i_1,i_2,i_3} = \mu + \alpha_k + \rho_{i_1}^1 + \rho_{i_2}^2 + \rho_{i_3}^3 + \epsilon_{k,i_1,i_2,i_3}
\]

Y_{k,i_1,i_2,i_3} is the logistic value of the probability of physical leg disorders for pigs with Disease \( k \), placed in pens with Straw \( i_1 \), PenDen \( i_2 \) and Floor \( i_3 \). The values of \( Y_{k,i_1,i_2,i_3} \) for ClawWall are shown in the leftmost column of Table B.26 (similar values are calculated for ClawSole and Fracture).

- \( \mu \) is the intercept.
- \( \alpha_k \) is the fixed effect of Disease (\( k \): ClawWall, ClawSole, Fracture).
- \( \mu_k = \mu + \alpha_k \) is the disease specific intercept (\( k \): ClawWall, ClawSole, Fracture).
- \( \rho_{i_1}^1 \) is the fixed effect of Straw (\( i_1 \): No, Sparse, Deep).
- \( \rho_{i_2}^2 \) is the fixed effect of PenDen (\( i_2 \): Low, High).
Table B.26: An excerpt of the combined conditional probabilities for “ClawWall”, “ClawSole” and “Fracture” given “Straw”, “PenDen” and “Floor” (only the values for “ClawWall” are shown). The probabilities are presented on the logistic scale, $Y_{k,i_1i_2i_3}$, calculated directly from Eq. (B.6) as well as on the corresponding probability scale, $P(k|i_1i_2i_3)$.

| $Y_{k,i_1i_2i_3}$ | $P(k|i_1i_2i_3)$ | Disease, $k$ | Straw, $i_1$ | PenDen, $i_2$ | Floor, $i_3$ |
|------------------|-----------------|-------------|-----------|-------------|-------------|
| -0.7562          | 0.3195          | ClawWall    | No        | Low         | Solid       |
| -0.2912          | 0.4277          | ClawWall    | No        | Low         | Partially slatted |
| 0.0159           | 0.5040          | ClawWall    | No        | Low         | Fully slatted  |
| 0.0159           | 0.3477          | ClawWall    | No        | High        | Solid       |
| -0.1640          | 0.4591          | ClawWall    | No        | High        | Partially slatted |
| 0.1431           | 0.5357          | ClawWall    | No        | High        | Fully slatted  |
| -0.7439          | 0.3222          | ClawWall    | Sparse    | Low         | Solid       |
| -0.2789          | 0.4307          | ClawWall    | Sparse    | Low         | Partially slatted |
| 0.0282           | 0.5071          | ClawWall    | Sparse    | Low         | Fully slatted  |
| -0.6168          | 0.3505          | ClawWall    | Sparse    | High        | Solid       |
| -0.1517          | 0.4621          | ClawWall    | Sparse    | High        | Partially slatted |
| 0.1554           | 0.5388          | ClawWall    | Sparse    | High        | Fully slatted  |
| -1.7765          | 0.1447          | ClawWall    | Deep      | Low         | Solid       |
| -1.3115          | 0.2122          | ClawWall    | Deep      | Low         | Partially slatted |
| -1.0044          | 0.2681          | ClawWall    | Deep      | Low         | Fully slatted  |
| -1.6494          | 0.1612          | ClawWall    | Deep      | High        | Solid       |
| -1.1844          | 0.2343          | ClawWall    | Deep      | High        | Partially slatted |
| -0.8772          | 0.2937          | ClawWall    | Deep      | High        | Fully slatted  |
B.4 Physical complex

- \( \rho_{i3}^2 \) is the fixed effect of Floor \((i_3:\text{Solid, Partially slatted, Fully slatted})\).
- \( \epsilon_{ki123i} \) is the residual.

The fit of the regression is afterwards optimized by the introduction of disease specific factors, \( \lambda_1 = \lambda_{\text{ClawWall}} \), \( \lambda_2 = \lambda_{\text{ClawSole}} \) and \( \lambda_3 = \lambda_{\text{Fracture}} \), ensuring disease specific sensitivities towards changes in the risk factors as described in Section B.2.5. For the optimal \( \lambda \)-factors the parameter estimates of a model like the one shown in (B.9) are used.

Having performed the optimization, the sum of the squared residuals is 0.9907146 compared to 0.9963956 for the initial values \( \lambda_{\text{ClawWall}} = \lambda_{\text{ClawSole}} = \lambda_{\text{Fracture}} = 1 \). It can, therefore, be concluded that the optimized model gives a slightly better model fit. The result of the optimized model is given in Table B.27. Fig. C.4 (Appendix C) presents the R code for the the optimization of the Physical complex.

Hence, the optimized model gives the following values for \( \lambda \):

- \( \lambda_{\text{ClawWall}} = 0.9026038 \)
- \( \lambda_{\text{ClawSole}} = 1.0569546 \)
- \( \lambda_{\text{Fracture}} = 1.1057546 \)

Table B.27: Result of the optimized model for the “Physical” node.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Symbol</th>
<th>Estimate</th>
<th>Std. error</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept, ClawWall</td>
<td>( \mu_{\text{ClawWall}}(\lambda_{\text{ClawWall}}) )</td>
<td>-0.83784</td>
<td>0.05649</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Intercept, ClawSole</td>
<td>( \mu_{\text{ClawSole}}(\lambda_{\text{ClawSole}}) )</td>
<td>-0.79338</td>
<td>0.04892</td>
<td>0.368</td>
</tr>
<tr>
<td>Intercept, Fracture</td>
<td>( \mu_{\text{Fracture}}(\lambda_{\text{Fracture}}) )</td>
<td>-5.54686</td>
<td>0.04892</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Straw, Sparse supply</td>
<td>( \hat{\rho}_1^2 )</td>
<td>0.01817</td>
<td>0.04892</td>
<td>0.712</td>
</tr>
<tr>
<td>Straw, Deep bedding</td>
<td>( \hat{\rho}_3^1 )</td>
<td>-0.85073</td>
<td>0.04892</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Pen density, High</td>
<td>( \hat{\rho}_2^3 )</td>
<td>0.24576</td>
<td>0.03994</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Floor, Partially slatted</td>
<td>( \hat{\rho}_2^2 )</td>
<td>0.40293</td>
<td>0.04892</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Floor, Fully slatted</td>
<td>( \hat{\rho}_3^3 )</td>
<td>0.71216</td>
<td>0.04892</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

We are now ready to adjust the parameters as described in Section B.2.5:

1. As the base reference disease, \( k' \), we choose ClawWall, and

   (a) Define the lower bound for the prevalence in a herd as 10% and the upper bound as 80%. On the logistic scale this leads to a lower bound of \( b_l = -2.2 \) and an upper bound of \( b_u = 1.4 \).

   (b) On the arbitrary 0 to 9 scale of the risk index, we identify the value 0 with -2.2 on the logistic scale, and the value 9 with 1.4.

   (c) We define the initial intercept for ClawWall as \( \delta_{\text{ClawWall}}^{(0)} = b_l = -2.2 \)

   (d) We define the initial slope for ClawWall as \( \delta_{\text{ClawWall}}^{(1)} = \frac{b_u - b_l}{9} = \frac{1.4 - (-2.2)}{9} = 0.4 \).
2. For each disease, ClawWall, ClawSole and Fracture we:

(a) Define the preliminary intercept:
   i. \[ \hat{\delta}_{0}^{\text{ClawWall}} = \lambda_{\text{ClawWall}} = 0.9026038 \times (-2.2) = -1.9857. \]
   ii. \[ \hat{\delta}_{0}^{\text{ClawSole}} = \lambda_{\text{ClawSole}} + \mu_{\text{ClawSole}} \left( \hat{\delta}_{0}^{\text{ClawWall}} \right) = \left( 1.0569546 \times (-2.2) - 0.79338 \right) = -2.2783. \]
   iii. \[ \hat{\delta}_{0}^{\text{Fracture}} = \mu_{\text{Fracture}} \left( \hat{\delta}_{0}^{\text{ClawWall}} \right) = \left( 1.1057546 \times (-2.2) - 5.54686 \right) = -7.6397. \]

(b) Redefine the slopes:
   i. \[ \delta_{1}^{\text{ClawWall}} = \lambda_{\text{ClawWall}} \hat{\delta}_{1}^{\text{ClawWall}} = 0.9026038 \times 0.4 = 0.3610. \]
   ii. \[ \delta_{1}^{\text{ClawSole}} = \lambda_{\text{ClawSole}} \hat{\delta}_{1}^{\text{ClawSole}} = 1.0569546 \times 0.4 = 0.4228. \]
   iii. \[ \delta_{1}^{\text{Fracture}} = \mu_{\text{Fracture}} \hat{\delta}_{1}^{\text{Fracture}} = 1.1057546 \times 0.4 = 0.4423. \]

(c) The resulting conditional probability tables of ClawWall, ClawSole and Fracture given risk index may then be calculated by Eqs. (B.4) and (B.5).

3. For each risk factor, “Straw”, “PenDen” and “Floor”, we define the additive effects from the estimates of Table B.27:

   **“Straw”**: The effects of the various states are as follows (values from Table B.27):
   - No: \[ \rho_{1}^{1} = 0. \]
   - Sparse: \[ \rho_{2}^{1} = \rho_{2}^{1} / \hat{\delta}_{\text{ClawWall}} = 0.01817 / 0.4 = 0.0454. \]
   - Deep: \[ \rho_{3}^{1} = \rho_{3}^{1} / \hat{\delta}_{\text{ClawWall}} = -0.85073 / 0.4 = -2.1268. \]

   **“PenDen”**: The effects of the various states are as follows (values from Table B.27):
   - Low: \[ \rho_{1}^{2} = 0. \]
   - High: \[ \rho_{2}^{2} = \rho_{2}^{2} / \hat{\delta}_{\text{ClawWall}} = 0.24576 / 0.4 = 0.6144. \]

   **“Floor”**: The effects of the various states are as follows (values from Table B.27):
   - Solid: \[ \rho_{1}^{3} = 0. \]
   - Partially slatted: \[ \rho_{2}^{3} = \rho_{2}^{3} / \hat{\delta}_{\text{ClawWall}} = 0.40293 / 0.4 = 1.0073. \]
   - Fully slatted: \[ \rho_{3}^{3} = \rho_{3}^{3} / \hat{\delta}_{\text{ClawWall}} = 0.71216 / 0.4 = 1.7804. \]

4. For the “Physical” node (the risk index) we

   (a) Define the initial intercept as \[ \mu = \left( \mu_{\text{ClawWall}} \lambda_{\text{ClawWall}} - \hat{\delta}_{0}^{\text{ClawWall}} \right) / \hat{\delta}_{1}^{\text{ClawWall}} = (-0.83784 - (-2.2)) / 0.4 = 3.4054. \]

   (b) The resulting conditional probability table of the “Physical” node may now be calculated according to Eq. (B.2) assuming a standard deviation of \( \sigma_e = 1.0 \).
Having entered all parameters and compiled the model, it turned out that the prior mean of the “Physical” node, was \( E(\text{Physical}) = 4.54 \). Hence, it is no necessary to perform any adjustment of the intercepts. In other words, \( \Delta \mu = 0 \) implying that \( \mu = \overline{\mu} = 3.4054 \).

With reference to Eq. (B.1) the parameters for calculation of the CPT of the “Physical” risk index may be summarized as follows:

**Intercept:** \( \mu = 3.4054 \).

**Effect of Straw:**
- No: \( \rho_1^1 = 0 \).
- Sparse: \( \rho_2^1 = 0.0454 \).
- Deep: \( \rho_3^1 = -2.1268 \).

**Effect of PenDen:**
- Low: \( \rho_1^2 = 0 \).
- High: \( \rho_2^2 = 0.6144 \).

**Effect of Floor:**
- Solid: \( \rho_1^3 = 0 \).
- Partially slatted: \( \rho_2^3 = 1.0073 \).
- Fully slatted: \( \rho_3^3 = 1.7804 \).

**Standard deviation of the residual:** \( \sigma_e = 1.0 \).

For a given configuration of the parent nodes, the conditional distribution of the Physical node is calculated from a normal distribution in the same way as described for the Inherited node in Section B.3.

**ClawWall, ClawSole and Fracture**

Since, in this case, \( \Delta \mu = 0 \), the preliminary intercepts \( \delta_{\text{ClawWall}}^0, \delta_{\text{ClawSole}}^0 \) and \( \delta_{\text{Fracture}}^0 \) are equal to the final intercepts, \( \delta_{\text{ClawWall}}^0, \delta_{\text{ClawSole}}^0 \) and \( \delta_{\text{Fracture}}^0 \), respectively. The CPTs of the three nodes are calculated by use of the logistic model (B.3).

**Summary of the ClawWall node**

**Intercept:** \( \delta_{\text{ClawWall}}^0 = -1.9857 \).

**Linear effect of risk index:** \( \delta_{\text{ClawWall}}^1 = 0.3610 \).

**Summary of the ClawSole node**

**Intercept:** \( \delta_{\text{ClawSole}}^0 = -2.2783 \).
Appendix B: Elicitation of probabilities

Linear effect of risk index: $\delta_{\text{ClawSole}} = 0.4228$.

Summary of the Fracture node

Intercept: $\delta_{\text{Fracture}} = -7.6397$.

Linear effect of risk index: $\delta_{\text{Fracture}} = 0.4423$.

B.5 The Infectious complex

This section will describe the methodology for modeling the Herd class node “Infectious” and the Pig class nodes “Myco”, “Erysip”, “Haemo” and “Strep”. Fig. B.7 shows a subfigure of the model representing the Infectious complex. The structure is completely analogous to the general Fig. B.3.

Figure B.7: The Infectious complex. Causal edges shown with bold arrows are links from which we do not have quantitative information from the literature or expert opinions. Edges shown as dashed arrows are the quantitative information we can obtain.
B.5 Infectious complex

B.5.1 Probabilities for the Infectious complex

Prior estimates

The marginal distributions of three of the risk factors, “Straw”, “Floor” and “Pen-Den” have already been presented in Tables B.14 - B.16 of Section B.4. The remaining distributions for “Production”, “Purchase” and “HerdSize” are defined based on prior knowledge regarding the distribution of herds in Denmark (Tables B.28 - B.30).

Table B.28: Marginal distribution of Production

<table>
<thead>
<tr>
<th>Production</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Continuous</td>
<td>0.50</td>
</tr>
<tr>
<td>Sectioned</td>
<td>0.50</td>
</tr>
</tbody>
</table>

Table B.29: Marginal distribution of Purchase

<table>
<thead>
<tr>
<th>Purchase</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>Own piglets</td>
<td>0.50</td>
</tr>
<tr>
<td>Purchase from one</td>
<td>0.40</td>
</tr>
<tr>
<td>Purchase from more than one</td>
<td>0.10</td>
</tr>
</tbody>
</table>

Table B.30: Marginal distribution of HerdSize

<table>
<thead>
<tr>
<th>HerdSize</th>
<th>Probability</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-1000</td>
<td>0.50</td>
</tr>
<tr>
<td>1000-3000</td>
<td>0.40</td>
</tr>
<tr>
<td>3000-5000</td>
<td>0.10</td>
</tr>
<tr>
<td>&gt;5000</td>
<td>0.10</td>
</tr>
</tbody>
</table>

Conditional probabilities elicited from experts

All conditional probabilities has been elicited from expert opinions. The resulting probabilities are shown in Tables B.31 - B.54.
Table B.31: Conditional probability table (CPT) for \( P(\text{Myco} | \text{PenDen}) \)

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>0.991</td>
<td>0.009</td>
</tr>
<tr>
<td>High</td>
<td>0.981</td>
<td>0.019</td>
</tr>
</tbody>
</table>

Table B.32: Conditional probability table (CPT) for \( P(\text{Erysip} | \text{PenDen}) \)

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>0.997</td>
<td>0.003</td>
</tr>
<tr>
<td>High</td>
<td>0.997</td>
<td>0.003</td>
</tr>
</tbody>
</table>

Table B.33: Conditional probability table (CPT) for \( P(\text{Haemo} | \text{PenDen}) \)

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>0.997</td>
<td>0.003</td>
</tr>
<tr>
<td>High</td>
<td>0.996</td>
<td>0.004</td>
</tr>
</tbody>
</table>

Table B.34: Conditional probability table (CPT) for \( P(\text{Strep} | \text{PenDen}) \)

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>0.997</td>
<td>0.003</td>
</tr>
<tr>
<td>High</td>
<td>0.992</td>
<td>0.008</td>
</tr>
</tbody>
</table>

Table B.35: Conditional probability table (CPT) for \( P(\text{Myco} | \text{Floor}) \)

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Solid</td>
<td>0.989</td>
<td>0.011</td>
</tr>
<tr>
<td>Partially slatted</td>
<td>0.986</td>
<td>0.014</td>
</tr>
<tr>
<td>Fully slatted</td>
<td>0.986</td>
<td>0.014</td>
</tr>
</tbody>
</table>

Table B.36: Conditional probability table (CPT) for \( P(\text{Erysip} | \text{Floor}) \)

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Solid</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>Partially slatted</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>Fully slatted</td>
<td>0.995</td>
<td>0.005</td>
</tr>
</tbody>
</table>
### Table B.37: Conditional probability table (CPT) for \( P(\text{Haemo|Floor}) \)

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Solid</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>Partially slatted</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>Fully slatted</td>
<td>0.996</td>
<td>0.004</td>
</tr>
</tbody>
</table>

### Table B.38: Conditional probability table (CPT) for \( P(\text{Strep|Floor}) \)

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Solid</td>
<td>0.997</td>
<td>0.003</td>
</tr>
<tr>
<td>Partially slatted</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>Fully slatted</td>
<td>0.996</td>
<td>0.004</td>
</tr>
</tbody>
</table>

### Table B.39: Conditional probability table (CPT) for \( P(\text{Myco|Straw}) \)

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>0.986</td>
<td>0.014</td>
</tr>
<tr>
<td>Sparse</td>
<td>0.986</td>
<td>0.014</td>
</tr>
<tr>
<td>Deep bedded</td>
<td>0.981</td>
<td>0.019</td>
</tr>
</tbody>
</table>

### Table B.40: Conditional probability table (CPT) for \( P(\text{Erysip|Straw}) \)

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>0.997</td>
<td>0.003</td>
</tr>
<tr>
<td>Sparse</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>Deep bedded</td>
<td>0.997</td>
<td>0.003</td>
</tr>
</tbody>
</table>
Table B.41: Conditional probability table (CPT) for P(Haemo|Straw)

<table>
<thead>
<tr>
<th>Haemo</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>0.997</td>
<td>0.003</td>
</tr>
<tr>
<td>Sparse</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>Deep bedded</td>
<td>0.997</td>
<td>0.003</td>
</tr>
</tbody>
</table>

Table B.42: Conditional probability table (CPT) for P(Strep|Straw)

<table>
<thead>
<tr>
<th>Strep</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>0.997</td>
<td>0.003</td>
</tr>
<tr>
<td>Sparse</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>Deep bedded</td>
<td>0.997</td>
<td>0.003</td>
</tr>
</tbody>
</table>

Table B.43: Conditional probability table (CPT) for P(Myco|HerdSize)

<table>
<thead>
<tr>
<th>Myco</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-1000</td>
<td>0.985</td>
<td>0.015</td>
</tr>
<tr>
<td>1000-3000</td>
<td>0.985</td>
<td>0.015</td>
</tr>
<tr>
<td>3000-5000</td>
<td>0.985</td>
<td>0.015</td>
</tr>
<tr>
<td>&gt; 5000</td>
<td>0.985</td>
<td>0.015</td>
</tr>
</tbody>
</table>

B.5.2 Methodology for constructing the nodes

Infectious node

The combined CPT’s for “Myco”, “Erysip”, “Haemo” and “Strep” given “Pen-Den”, “Floor”, “Straw”, “Production”, “HerdSize” and “Purchase” are calculated as described in Section B.2.5 with Eq. (B.6) as the central formula. We must decide on the base risk, $R_{k0}$, and, again, we choose “Floor” for calculation of the base prevalence of all four. The combined CPTs are then calculated on the logistic scale as defined in Eq. (B.6). The R-code for the development of the combined CPTs is given in Appendix C, (Fig C.5), and an excerpt of the resulting combined probability values for the three diseases are shown in Table B.55, which corresponds directly to Table B.2.

Using the values of Table B.55 we fit a linear model corresponding to Eq. (B.7) for the “Infectious” node describing the relation between the logistic values of the combined probabilities and the effects: Disease(Myco, Erysip, Haemo, Strep), PenDen(Low, High), Floor(Solid, Partially slatted, Fully slatted), Straw(No, Sparse, Deep), HerdSize(1-1000, 1000-3000, 3000-5000, >5000), Production (Sectioned,
Table B.44: Conditional probability table (CPT) for $P(\text{Erysip} \mid \text{HerdSize})$

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-1000</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>1000-3000</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>3000-5000</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>&gt; 5000</td>
<td>0.996</td>
<td>0.004</td>
</tr>
</tbody>
</table>

Table B.45: Conditional probability table (CPT) for $P(\text{Haemo} \mid \text{HerdSize})$

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-1000</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>1000-3000</td>
<td>0.997</td>
<td>0.003</td>
</tr>
<tr>
<td>3000-5000</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>&gt; 5000</td>
<td>0.996</td>
<td>0.004</td>
</tr>
</tbody>
</table>

Table B.46: Conditional probability table (CPT) for $P(\text{Strep} \mid \text{HerdSize})$

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-1000</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>1000-3000</td>
<td>0.997</td>
<td>0.003</td>
</tr>
<tr>
<td>3000-5000</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>&gt; 5000</td>
<td>0.996</td>
<td>0.004</td>
</tr>
</tbody>
</table>

Table B.47: Conditional probability table (CPT) for $P(\text{Myco} \mid \text{Production})$

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sectioned</td>
<td>0.991</td>
<td>0.009</td>
</tr>
<tr>
<td>Continuous</td>
<td>0.986</td>
<td>0.014</td>
</tr>
</tbody>
</table>

Table B.48: Conditional probability table (CPT) for $P(\text{Erysip} \mid \text{Production})$

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sectioned</td>
<td>0.997</td>
<td>0.003</td>
</tr>
<tr>
<td>Continuous</td>
<td>0.997</td>
<td>0.003</td>
</tr>
</tbody>
</table>

Table B.49: Conditional probability table (CPT) for $P(\text{Haemo} \mid \text{Production})$

<table>
<thead>
<tr>
<th></th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sectioned</td>
<td>0.997</td>
<td>0.003</td>
</tr>
<tr>
<td>Continuous</td>
<td>0.996</td>
<td>0.004</td>
</tr>
</tbody>
</table>
Table B.50: Conditional probability table (CPT) for \( P(\text{Strep}|\text{Production}) \)

<table>
<thead>
<tr>
<th>Strep</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sectioned</td>
<td>0.997</td>
<td>0.003</td>
</tr>
<tr>
<td>Continuous</td>
<td>0.996</td>
<td>0.004</td>
</tr>
</tbody>
</table>

Table B.51: Conditional probability table (CPT) for \( P(\text{Myco}|\text{Purchase}) \)

<table>
<thead>
<tr>
<th>Myco</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Own piglets</td>
<td>0.985</td>
<td>0.015</td>
</tr>
<tr>
<td>Purchase from one</td>
<td>0.985</td>
<td>0.015</td>
</tr>
<tr>
<td>Purchase from more than one</td>
<td>0.976</td>
<td>0.024</td>
</tr>
</tbody>
</table>

Table B.52: Conditional probability table (CPT) for \( P(\text{Erysip}|\text{Purchase}) \)

<table>
<thead>
<tr>
<th>Erysip</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Own piglets</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>Purchase from one</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>Purchase from more than one</td>
<td>0.996</td>
<td>0.004</td>
</tr>
</tbody>
</table>

Table B.53: Conditional probability table (CPT) for \( P(\text{Haemo}|\text{Purchase}) \)

<table>
<thead>
<tr>
<th>Haemo</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Own piglets</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>Purchase from one</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>Purchase from more than one</td>
<td>0.995</td>
<td>0.005</td>
</tr>
</tbody>
</table>

Table B.54: Conditional probability table (CPT) for \( P(\text{Strep}|\text{Purchase}) \)

<table>
<thead>
<tr>
<th>Strep</th>
<th>No</th>
<th>Yes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Own piglets</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>Purchase from one</td>
<td>0.996</td>
<td>0.004</td>
</tr>
<tr>
<td>Purchase from more than one</td>
<td>0.995</td>
<td>0.005</td>
</tr>
</tbody>
</table>
Table B.55: An excerpt of the combined conditional probabilities for “Myco”, “Erysip”, “Haemo” and “Strep” given “PenDen”, “Floor”, “Straw”, “Production”, “HerdSize” and “Purchase” (only some of the values for “Myco” are shown). The probabilities are presented on the logistic scale, $Y_{k,i_1,i_2,i_3,i_4,i_5,i_6}$, calculated directly from Eq. (B.6) as well as on the corresponding probability scale, $P(k| i_1,i_2,i_3,i_4,i_5,i_6)$. 

<table>
<thead>
<tr>
<th>$Y_{k,i_1...i_6}$</th>
<th>Disease.</th>
<th>PenDen</th>
<th>Floor</th>
<th>Straw</th>
<th>Production</th>
<th>HerdSize</th>
<th>Purchase</th>
</tr>
</thead>
<tbody>
<tr>
<td>-5.1319</td>
<td>Myco</td>
<td>Low</td>
<td>Solid</td>
<td>No</td>
<td>Sectioned</td>
<td>1-1000</td>
<td>One herd</td>
</tr>
<tr>
<td>-5.1319</td>
<td>Myco</td>
<td>Low</td>
<td>Solid</td>
<td>No</td>
<td>Sectioned</td>
<td>1-1000</td>
<td>&gt; one herd</td>
</tr>
<tr>
<td>-5.1319</td>
<td>Myco</td>
<td>Low</td>
<td>Solid</td>
<td>No</td>
<td>Sectioned</td>
<td>1000-3000</td>
<td>One herd</td>
</tr>
<tr>
<td>-5.1319</td>
<td>Myco</td>
<td>Low</td>
<td>Solid</td>
<td>No</td>
<td>Sectioned</td>
<td>1000-3000</td>
<td>&gt; one herd</td>
</tr>
<tr>
<td>-5.1319</td>
<td>Myco</td>
<td>Low</td>
<td>Solid</td>
<td>No</td>
<td>Sectioned</td>
<td>3000-5000</td>
<td>One herd</td>
</tr>
<tr>
<td>-5.1319</td>
<td>Myco</td>
<td>Low</td>
<td>Solid</td>
<td>No</td>
<td>Sectioned</td>
<td>3000-5000</td>
<td>&gt; one herd</td>
</tr>
<tr>
<td>-5.1319</td>
<td>Myco</td>
<td>Low</td>
<td>Solid</td>
<td>No</td>
<td>Sectioned</td>
<td>&gt; 5000</td>
<td>Own piglets</td>
</tr>
<tr>
<td>-5.1319</td>
<td>Myco</td>
<td>Low</td>
<td>Solid</td>
<td>No</td>
<td>Sectioned</td>
<td>&gt; 5000</td>
<td>One herd</td>
</tr>
<tr>
<td>-5.1319</td>
<td>Myco</td>
<td>Low</td>
<td>Solid</td>
<td>No</td>
<td>Sectioned</td>
<td>&gt; 5000</td>
<td>&gt; one herd</td>
</tr>
</tbody>
</table>

Continuous) and Purchase(Own, One herd, More than one herd). Hence, the regression model is defined as:

$$Y_{k,i_1i_2i_3i_4i_5i_6} = \mu + \alpha_k + \rho_1^{i_1} + \rho_2^{i_2} + \rho_3^{i_3} + \rho_4^{i_4} + \rho_5^{i_5} + \rho_6^{i_6} + \epsilon_{k,i_1i_2i_3i_4i_5i_6} (B.15)$$

where

- $Y_{k,i_1i_2i_3i_4i_5i_6}$ is the logistic value of the probability of infectious leg disorders for pigs with Disease $k$ placed in placed in pens with PenDen $i_1$, Floor $i_2$ and Straw $i_3$ in a herd with HerdSize $i_4$, Production $i_5$ and Purchase $i_6$. Part of the values of $Y_{k,i_1i_2i_3i_4i_5i_6}$ for Myco are shown in the lefmost column of Table B.55 (similar values are calculated for Erysip, Haemo and Strep).

- $\mu$ is the intercept.

- $\alpha_k$ is the fixed effect of Disease ($k$: Myco, Erysip, Haemo, Strep).

- $\mu_k = \mu + \alpha_k$ is the disease specific intercept ($k$: Myco, Erysip, Haemo, Strep).

- $\rho_1^{i_1}$ is the fixed effect of PenDen ($i_1$: Low, High).

- $\rho_2^{i_2}$ is the fixed effect of Floor ($i_2$: Solid, Partially slatted, Fully slatted).

- $\rho_3^{i_3}$ is the fixed effect of Straw ($i_3$: No, Sparse, Deep).

- $\rho_4^{i_4}$ is the fixed effect of HerdSize ($i_4$: 1-1000, 1000-3000, 3000-5000, >5000).
Appendix B: Elicitation of probabilities

- \( \rho_5^i \) is the fixed effect of Production (\( i_5 \): Sectioned, Continuous).
- \( \rho_6^i \) is the fixed effect of Purchase (\( i_6 \): Own, One herd, >one herd).
- \( \epsilon_{k,i1i2i3i4i5i6} \) is the residual.

The fit of the regression is afterwards optimized by the introduction of disease specific factors, \( \lambda_1 = \lambda_{\text{Myco}}, \lambda_2 = \lambda_{\text{Erysip}}, \lambda_3 = \lambda_{\text{Haemo}} \) and \( \lambda_4 = \lambda_{\text{Strep}} \), ensuring disease specific sensitivities towards changes in the risk factors as described in Section [B.2.5]. For the optimal \( \lambda \)-factors the parameter estimates of a model like the one shown in (B.9) are used. Hence, Fig. [C.6] (Appendix C) presents the R code for the optimization of the Infectious complex.

Having performed the optimization, the sum of the squared residuals is 120.3751 compared to 120.5360 for the initial values \( \lambda_{\text{Myco}} = \lambda_{\text{Erysip}} = \lambda_{\text{Haemo}} = \lambda_{\text{Strep}} = 1 \). It can, therefore, be concluded that the optimized model gives a slightly better model fit. The result of the optimized model is given in [B.36]. The optimized model gives the following values for \( \lambda \):

- \( \lambda_{\text{Myco}} = 1.0850923 \)
- \( \lambda_{\text{Erysip}} = 0.9905036 \)
- \( \lambda_{\text{Haemo}} = 1.0146171 \)
- \( \lambda_{\text{Strep}} = 1.0692615 \)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Symbol</th>
<th>Estimate</th>
<th>Std. error</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept, Myco</td>
<td>( \mu_{\text{Myco}}(\lambda_{\text{Myco}}) )</td>
<td>-4.72907</td>
<td>0.02470</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Intercept, Erysip</td>
<td>( \mu_{\text{Erysip}}(\lambda_{\text{Erysip}}) )</td>
<td>-6.11292</td>
<td>0.01804</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Intercept, Haemo</td>
<td>( \mu_{\text{Haemo}}(\lambda_{\text{Haemo}}) )</td>
<td>-6.10667</td>
<td>0.01804</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Intercept, Strep</td>
<td>( \mu_{\text{Strep}}(\lambda_{\text{Strep}}) )</td>
<td>-6.16568</td>
<td>0.01804</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Pen density, High</td>
<td>( \hat{\rho}_1^3 )</td>
<td>0.50751</td>
<td>0.01275</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Floor, Partially slatted</td>
<td>( \hat{\rho}_2^3 )</td>
<td>0.13252</td>
<td>0.01562</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Floor, Fully slatted</td>
<td>( \hat{\rho}_3^3 )</td>
<td>0.18856</td>
<td>0.01562</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Straw, Sparse supply</td>
<td>( \hat{\rho}_3^3 )</td>
<td>0.21582</td>
<td>0.01562</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Straw, Deep bedding</td>
<td>( \hat{\rho}_3^3 )</td>
<td>0.07692</td>
<td>0.01562</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Herd size, 1000-3000</td>
<td>( \hat{\rho}_3^4 )</td>
<td>-0.14527</td>
<td>0.01804</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Herd size, 3000-5000</td>
<td>( \hat{\rho}_3^4 )</td>
<td>-0.00093</td>
<td>0.01804</td>
<td>0.959</td>
</tr>
<tr>
<td>Herd size, &gt;5000</td>
<td>( \hat{\rho}_3^4 )</td>
<td>-0.00093</td>
<td>0.01804</td>
<td>0.959</td>
</tr>
<tr>
<td>Production, Continuous</td>
<td>( \hat{\rho}_6^3 )</td>
<td>0.25560</td>
<td>0.01275</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Purchase, One herd</td>
<td>( \hat{\rho}_6^i )</td>
<td>-0.00070</td>
<td>0.01562</td>
<td>0.964</td>
</tr>
<tr>
<td>Purchase, More than one herd</td>
<td>( \hat{\rho}_6^i )</td>
<td>0.23117</td>
<td>0.01562</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

We are now ready to adjust the parameters as described in Section [B.2.5].

1. As the base reference disease, \( k' \), we choose Myco, and
(a) Define the lower bound for the prevalence in a herd as 0.01% and the upper bound as 5% bound of $b_l = -9.2$ and an upper bound of $b_u = -2.9$.
(b) On the arbitrary 0 to 9 scale of the risk index, we identify the value 0 with -9.2 on the logistic scale, and the value 9 with -2.9.
(c) We define the initial intercept for Myco as $\delta_{Myco}^0 = b_l = -9.2$
(d) We define the initial slope for Myco as $\delta_{Myco}^1 = b_u - b_l = -2.9 - (-9.2) = 0.7$

2. For each disease, Myco, Erysip, Haemo and Strep we:
(a) Define the preliminary intercept:
   i. $\hat{\delta}_{Myco}^0 = \lambda_{Myco}(\delta_{Myco}^0 + \mu_{Myco}(\lambda_{Myco})) = 1.0850923 \times (-9.2) = -9.9828$
   ii. $\hat{\delta}_{Erysip}^0 = \lambda_{Erysip}(\delta_{Myco}^0 + \mu_{Erysip}(\lambda_{Erysip}) - \mu_{Myco}(\lambda_{Myco})) = 0.9905036 \times (-9.2 - 6.11292 - (-4.72907)) = -10.4833$
   iii. $\hat{\delta}_{Haemo}^0 = \lambda_{Haemo}(\delta_{Myco}^0 + \mu_{Haemo}(\lambda_{Haemo}) - \mu_{Myco}(\lambda_{Myco})) = 1.0146171 \times (-9.2 - 6.10667 - (-4.72907)) = -10.7322$
   iv. $\hat{\delta}_{Strep}^0 = \lambda_{Strep}(\delta_{Myco}^0 + \mu_{Strep}(\lambda_{Strep}) - \mu_{Myco}(\lambda_{Myco})) = 1.0692615 \times (-9.2 - 6.16568 - (-4.72907)) = -11.3733$
(b) Redefine the slopes:
   i. $\delta_{Myco}^1 = \lambda_{Myco}(\delta_{Myco}^1 + \mu_{Myco}(\lambda_{Myco})) = 1.0850923 \times 0.7 = 0.7596$
   ii. $\delta_{Erysip}^1 = \lambda_{Erysip}(\delta_{Myco}^1 + \mu_{Erysip}(\lambda_{Erysip}) = 0.9905036 \times 0.7 = 0.6934$
   iii. $\delta_{Haemo}^1 = \lambda_{Haemo}(\delta_{Myco}^1 + \mu_{Haemo}(\lambda_{Haemo}) = 1.0146171 \times 0.7 = 0.7102$
   iv. $\delta_{Strep}^1 = \lambda_{Strep}(\delta_{Myco}^1 + \mu_{Strep}(\lambda_{Strep}) - \mu_{Myco}(\lambda_{Myco})) = 1.0692615 \times 0.7 = 0.7485$
(c) The resulting conditional probability tables of Myco, Erysip, Haemo and Strep given risk index may then be calculated by Eqs. (B.4) and (B.5).

3. For each risk factor, “PenDen”, “Floor”, “Straw”, “HerdSize”, “Production” and “Purchase” we define the additive effects from the estimates of Table B.27:

   **“PenDen”**: The effects of the various states are as follows (values from Table B.56):
   Low: $\rho_1^1 = 0$
   High: $\rho_2^1 = \hat{\rho}_2^1/\delta_{Myco} = 0.50751/0.7 = 0.7250$

   **“Floor”**: The effects of the various states are as follows (values from Table B.56):
   Solid: $\rho_2^2 = 0$
   Partially slatted: $\rho_2^2 = \hat{\rho}_2^2/\delta_{Myco} = 0.13252/0.7 = 0.1893$
Fully slatted: \( \hat{\alpha}^2 = \frac{\hat{\alpha}^2}{\bar{\delta}_{\text{Myco}}} \times 0.18856/0.7 = 0.2694. \)

**“Straw”:** The effects of the various states are as follows (values from Table B.56):

- **No:** \( \hat{\alpha}^3 = 0. \)
- **Sparse:** \( \hat{\alpha}^3 = \frac{\hat{\alpha}^3}{\bar{\delta}_{\text{Myco}}} = 0.21582/0.7 = 0.3083. \)
- **Deep:** \( \hat{\alpha}^3 = \frac{\hat{\alpha}^3}{\bar{\delta}_{\text{Myco}}} = 0.07692/0.7 = 0.1099. \)

**“HerdSize”:** The effects of the various states are as follows (values from Table B.56):

- **1-1000:** \( \hat{\alpha}^4 = 0. \)
- **1000-3000:** \( \hat{\alpha}^4 = \frac{\hat{\alpha}^4}{\bar{\delta}_{\text{Myco}}} = -0.14527/0.7 = -0.2075. \)
- **3000-5000:** \( \hat{\alpha}^4 = \frac{\hat{\alpha}^4}{\bar{\delta}_{\text{Myco}}} = -0.00093/0.7 = -0.0013. \)
- **> 5000:** \( \hat{\alpha}^4 = \frac{\hat{\alpha}^4}{\bar{\delta}_{\text{Myco}}} = -0.00093/0.7 = -0.0013. \)

**“Production”:** The effects of the various states are as follows (values from Table B.56):

- **Sectioned:** \( \hat{\alpha}^5 = 0. \)
- **Continuous:** \( \hat{\alpha}^5 = \frac{\hat{\alpha}^5}{\bar{\delta}_{\text{Myco}}} = 0.25560/0.7 = 0.3651. \)

**“Purchase”:** The effects of the various states are as follows (values from Table B.56):

- **Own piglets:** \( \hat{\alpha}^6 = 0. \)
- **One herd:** \( \hat{\alpha}^6 = \frac{\hat{\alpha}^6}{\bar{\delta}_{\text{Myco}}} = -0.00070/0.7 = -0.0010. \)
- **> one herd:** \( \hat{\alpha}^6 = \frac{\hat{\alpha}^6}{\bar{\delta}_{\text{Myco}}} = 0.23117/0.7 = 0.3302. \)

4. For the “Physical” node (the risk index) we

(a) Define the initial intercept as \( \mu = (\mu_{\text{Myco}}(\lambda_{\text{Myco}}) - \bar{\delta}_{\text{Myco}})/\bar{\delta}_{\text{Myco}} = (-4.72907 - (-9.2)) / 0.7 = 6.3870. \)

(b) The resulting conditional probability table of the “Physical” node may now be calculated according to Eq. (B.2) assuming a standard deviation of \( \sigma_e = 1.0. \)

Having entered all parameters and compiled the model, it turned out that the prior mean of the “Infectious” node, was E(Infectious) = 7.0833. Since this prior mean by definition must be 4.5, we notice that \( \Delta \mu = 4.5 - 7.0833 = -2.5833. \) Thus, according to Section B.2.5 the final intercept of the “Infectious” node becomes \( \mu = \mu + \Delta \mu = 6.3870 + (-2.5833) = 3.8037. \)

**Intercept:** \( = \mu = 3.8037. \)

**Effect of PenDen:**

- **Low:** \( \hat{\alpha}^1 = 0. \)
High: $\rho_1^2 = 0.7250$.

Effect of Floor:
- Solid: $\rho_1^2 = 0$.
- Partially slatted: $\rho_2^2 = 0.1893$.
- Fully slatted: $\rho_3^2 = 0.2694$.

Effect of Straw:
- No: $\rho_1^3 = 0$.
- Sparse: $\rho_2^3 = 0.3083$.
- Deep: $\rho_3^3 = 0.1099$.

Effect of HerdSize:
- 1-1000: $\rho_1^4 = 0$.
- 1000-3000: $\rho_2^4 = -0.2075$.
- 3000-5000: $\rho_3^4 = -0.0013$.
- >5000: $\rho_4^4 = -0.0013$.

Effect of Production:
- Sectioned: $\rho_1^5 = 0$.
- Continuous: $\rho_2^5 = 0.3651$.

Effect of Purchase:
- Own piglets: $\rho_1^6 = 0$.
- One herd: $\rho_2^6 = -0.0010$.
- > one herd: $\rho_3^6 = 0.3302$.

Standard deviation of the residual: $\sigma_e = 1.0$.

For a given configuration of the parent nodes, the conditional distribution of the Infectious node is calculated from a normal distribution in the same way as described for the Gain node in Section B.3.2. The conditional mean is calculated from the information on Straw, PenDen, Floor, HerdSize, Production and Purchase using the effects specified above. The conditional standard deviation of the risk index basically express the precision of the expert knowledge. This standard deviation is specified as input to the model, and the default value used is a standard deviation of 1.0 (measured on the arbitrary scale from 0 to 9).
Appendix B: Elicitation of probabilities

Myco, Erysip, Haemo and Strep

The construction of the nodes for “Myco”, “Erysip”, “Haemo” and “Strep” is based on a linear regression model on the logistic scale (cf. Eq. (B.3)). The intercept of the regression model indicates the expected prevalence of “Myco”, “Erysip”, “Haemo” and “Strep” in a random herd and the slope represent the effect of the risk index for the cause-category: “Infectious”.

The slopes $\delta_1^{\text{Myco}}, \delta_1^{\text{Erysip}}, \delta_1^{\text{Haemo}}$ and $\delta_1^{\text{Strep}}$ have already been determined in the previous section together with preliminary intercepts $\hat{\delta}_0^{\text{Myco}}, \hat{\delta}_0^{\text{Erysip}}, \hat{\delta}_0^{\text{Haemo}}$ and $\hat{\delta}_0^{\text{Strep}}$. The final step is to correct the intercepts for $\Delta \mu$ as described in Section B.2.5. Thus,

\[
\delta_0^{\text{Myco}} = \hat{\delta}_0^{\text{Myco}} - \Delta \mu \delta_1^{\text{Myco}} = -9.9828 - (-2.5833) \times 0.7596 = -8.0205,
\]
\[
\delta_0^{\text{Erysip}} = \hat{\delta}_0^{\text{Erysip}} - \Delta \mu \delta_1^{\text{Erysip}} = -10.4833 - (-2.5833) \times 0.6934 = -8.6920,
\]
\[
\delta_0^{\text{Haemo}} = \hat{\delta}_0^{\text{Haemo}} - \Delta \mu \delta_1^{\text{Haemo}} = -10.7322 - (-2.5833) \times 0.7102 = -8.8975
\]
and
\[
\delta_0^{\text{Strep}} = \hat{\delta}_0^{\text{Strep}} - \Delta \mu \delta_1^{\text{Strep}} = -11.3733 - (-2.5833) \times 0.7485 = -9.4397.
\]

Summary of the Myco node

**Intercept:** $\delta_0^{\text{Myco}} = -8.0205$.

**Slope:** $\delta_1^{\text{Myco}} = 0.7596$

Summary of the Erysip node

**Intercept:** $\delta_0^{\text{Erysip}} = -8.6920$.

**Slope:** $\delta_1^{\text{Erysip}} = 0.6934$

Summary of the Haemo node

**Intercept:** $\delta_0^{\text{Haemo}} = -8.8975$.

**Slope:** $\delta_1^{\text{Haemo}} = 0.7102$

Summary of the Strep node

**Intercept:** $\delta_0^{\text{Strep}} = -9.4397$.

**Slope:** $\delta_1^{\text{Strep}} = 0.7485$
B.6 PigLame node

This section will present the construction of the PigLame node. The probabilities of lameness given each of the leg disorders have been elicited from experts (Table B.57). The property of “noisy or gate” for Bayesian networks (Jensen, 2001) is used for the construction of the “PigLame” node. As an example of the “noisy or gate”, Fig. B.8 illustrates the relation between the leg disorders: Fracture (yes/no), ClawWall (yes/no), ClawSole (yes/no) and PigLame (yes/no).

Lesions to the claw wall can cause clinical signs of lameness unless an inhibitor prevents it. The probability of the inhibitor is defined as 

$$P(\text{Lameness}_{\text{no}}|\text{ClawWall}) = q_{\text{ClawWall}}.$$ 

Likewise, the probabilities of the inhibitors for ClawSole and Fracture are 

$$q_{\text{ClawSole}} \text{ and } q_{\text{Fracture}},$$ 

respectively. Assuming that the inhibitors for ClawWall, ClawSole and Fracture are independent, it is possible to calculate the probability of no lameness given the occurrence of Fracture, ClawWall and ClawSole as the product of the individual inhibitors:

$$P(\text{Lameness}_{\text{no}}|\text{Fracture, ClawWall, ClawSole}) = q_{\text{ClawWall}}q_{\text{ClawSole}}q_{\text{Fracture}}$$ 

Therefore, the probability of lameness given the occurrence of Fracture, ClawWall, ClawSole is:

$$P(\text{Lameness}_{\text{yes}}|\text{Fracture, ClawWall, ClawSole}) = 1 - (q_{\text{ClawWall}}q_{\text{ClawSole}}q_{\text{Fracture}})$$ 

All probabilities for the CPT of the PigLame node are obtained in a similar way.

B.6.1 Final adjustment of the PigLame node

Based on the expected prevalence of lameness in the finisher pig production, we want the default value of the PigLame model to estimate approximately 5% lame pigs before any observations are made. However, the model estimate a default value of lame pigs in a random herd to be approximately 50%. This is due to high conditional probabilities for 

$$P(\text{Lameness}|\text{OCM}), P(\text{Lameness}|\text{OCD}), P(\text{Lameness}|\text{ClawWall}) \text{ and } P(\text{Lameness}|\text{ClawSole})$$ 

combined with high prevalence of these diseases. As these probabilities are way too high compared to what we expect to find in real herds, we scale the conditional probabilities in order to obtain the required default value.

B.7 Sensitivities and Specificities

The sensitivity and the specificity for each diagnostic test in the Pig class are elicited from either expert opinions or the literature. The values are given in Table B.58.
Table B.57: Probabilities given by experts

<table>
<thead>
<tr>
<th>Probability</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>P(Lameness</td>
<td>OCD)</td>
</tr>
<tr>
<td>P(Lameness</td>
<td>OCM)</td>
</tr>
<tr>
<td>P(Lameness</td>
<td>ClawWall)</td>
</tr>
<tr>
<td>P(Lameness</td>
<td>ClawSole)</td>
</tr>
<tr>
<td>P(Lameness</td>
<td>Fracture)</td>
</tr>
<tr>
<td>P(Lameness</td>
<td>Myco)</td>
</tr>
<tr>
<td>P(Lameness</td>
<td>Erysip)</td>
</tr>
<tr>
<td>P(Lameness</td>
<td>Haemo)</td>
</tr>
<tr>
<td>P(Lameness</td>
<td>Strep)</td>
</tr>
</tbody>
</table>

Figure B.8: “Noisy Or” for the Physical complex
### Table B.58: Sensitivities and specificities

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myco clinic</td>
<td>0.76</td>
<td>0.88</td>
<td>Expert</td>
</tr>
<tr>
<td>Myco Pathology</td>
<td>0.57</td>
<td>0.60</td>
<td>Expert</td>
</tr>
<tr>
<td>Myco bacteriology</td>
<td>0.61</td>
<td>0.92</td>
<td>Expert</td>
</tr>
<tr>
<td>Strep clinic</td>
<td>0.77</td>
<td>0.90</td>
<td>Expert</td>
</tr>
<tr>
<td>Strep pathology</td>
<td>0.60</td>
<td>0.56</td>
<td>Expert</td>
</tr>
<tr>
<td>Strep bacteriology</td>
<td>0.78</td>
<td>0.89</td>
<td>Expert</td>
</tr>
<tr>
<td>Erysip clinic</td>
<td>0.71</td>
<td>0.92</td>
<td>Expert</td>
</tr>
<tr>
<td>Erysip pathology</td>
<td>0.69</td>
<td>0.73</td>
<td>Expert</td>
</tr>
<tr>
<td>Erysip bacteriology</td>
<td>0.52</td>
<td>0.81</td>
<td>Expert</td>
</tr>
<tr>
<td>Haemo clinic</td>
<td>0.64</td>
<td>0.87</td>
<td>Expert</td>
</tr>
<tr>
<td>Haemo pathology</td>
<td>0.45</td>
<td>0.46</td>
<td>Expert</td>
</tr>
<tr>
<td>Haemo bacteriology</td>
<td>0.66</td>
<td>0.78</td>
<td>Expert</td>
</tr>
<tr>
<td>Claw wall clinic</td>
<td>0.80</td>
<td>0.90</td>
<td>Expert</td>
</tr>
<tr>
<td>Claw wall pathology</td>
<td>0.96</td>
<td>0.97</td>
<td>Expert</td>
</tr>
<tr>
<td>Claw sole clinic</td>
<td>0.70</td>
<td>0.93</td>
<td>Expert</td>
</tr>
<tr>
<td>Claw sole pathology</td>
<td>0.97</td>
<td>0.97</td>
<td>Expert</td>
</tr>
<tr>
<td>Fracture clinic</td>
<td>0.92</td>
<td>0.19</td>
<td>Expert</td>
</tr>
<tr>
<td>Fracture pathology</td>
<td>0.89</td>
<td>0.96</td>
<td>Expert</td>
</tr>
<tr>
<td>OCM clinic</td>
<td>0.19</td>
<td>0.19</td>
<td>Expert</td>
</tr>
<tr>
<td>OCM pathology</td>
<td>0.96</td>
<td>0.89</td>
<td>Expert</td>
</tr>
<tr>
<td>OCD clinic</td>
<td>0.33</td>
<td>0.20</td>
<td>Expert</td>
</tr>
<tr>
<td>OCD pathology</td>
<td>0.95</td>
<td>0.94</td>
<td>Expert</td>
</tr>
<tr>
<td>Lame obs</td>
<td>0.76</td>
<td>0.96</td>
<td>[Baudsgaard and Jørgensen, 2003]</td>
</tr>
</tbody>
</table>
References


APPENDIX C

R CODES

```r
pCombined = function(X, Y, x, y, useX) {
  baseX = averageProb(X, x)
  baseLogitX = logit(baseX)
  baseY = averageProb(Y, y)
  baseLogitY = logit(baseY)
  logitX = logit(X[,2])
  devX = logitX - baseLogitX
  logitY = logit(Y[,2])
  devY = logitY - baseLogitY
  res = matrix(rep(NA, length(x)*length(y)*2), length(x)*length(y))
  c = 0
  for (i in 1:length(x)) {
    for (j in 1:length(y)) {
      c = c + 1
      if (useX) {
        res[c,2] = invLogit(baseLogitX + devX[i] + devY[j])
      } else {
        res[c,2] = invLogit(baseLogitY + devX[i] + devY[j])
      }
      res[c,1] = 1 - res[c,2]
    }
  }
  return(res)
}
```

Figure C.1: Inherited complex. R code for creating the combined probabilities.
findFit = function(lambda, data) {
  data$cLogit = data$Logit
  for (i in 1:length(data$Dis)) {
    data$cLogit[i] = data$Logit[i] / lambda[data$OCM[i]+1]
  }
  parms = lm(cLogit ~ factor(OCM)+ factor(Breed) +Gain, data)
  res = sum(parms$residuals**2)
  return(res)
}

lambda = c(1, 1)
findFit(lambda, data)

getParms = function(lambda, data) {
  data$cLogit = data$Logit
  for (i in 1:length(data$Dis)) {
    data$cLogit[i] = data$Logit[i] / lambda[data$OCM[i]+1]
  }
  parms = lm(cLogit ~ factor(OCM)+ factor(Breed) +Gain, data)
  return(parms)
}

parms = getParms(lambda, data)
parms
summary(parms)

opt = optim(lambda, findFit, NULL, method = "Nelder-Mead", lower = -Inf, upper = Inf,
control = list(maxit = 5000), hessian =FALSE, data)
opt

parms = getParms(opt$par, data)
parms
summary(parms)

Figure C.2: R code for the optimization of the Inherited complex.
# Combined probabilities for ClawWall, ClawSole, Fracture given Straw, Density
# and Floor

# The base for argument i is used if useV = i
pCombined = function(X, Y, Z, x, y, z, useV) {
  bases = c(NA, NA, NA)
  baseX = averageProb(X, x)
  baseLogitX = logit(baseX)
  bases[1] = baseLogitX
  baseY = averageProb(Y, y)
  baseLogitY = logit(baseY)
  bases[2] = baseLogitY
  baseZ = averageProb(Z, z)
  baseLogitZ = logit(baseZ)
  bases[3] = baseLogitZ
  logitX = logit(X[,2])
  devX = logitX - baseLogitX
  logitY = logit(Y[,2])
  devY = logitY - baseLogitY
  logitZ = logit(Z[,2])
  devZ = logitZ - baseLogitZ
  res = matrix(rep(NA, length(x)*length(y)*length(z)*2), length(x)*length(y)*length(z))
  c = 0
  for (i in 1:length(x)) {
    for (j in 1:length(y)) {
      for (k in 1:length(z)) {
        c = c + 1
        res[c,2] = invLogit(bases[useV] + devX[i] + devY[j] + devZ[k])
        res[c,1] = 1 - res[c,2]
      }
    }
  }
  return(res)
}

Figure C.3: Physical complex. R code for creating the combined probabilities.
```r
findFit = function(lambda, data) {
  data$cLogit = data$Logit
  for (i in 1:length(data$Dis)) {
    data$cLogit[i] = data$Logit[i] / lambda[data$DIS[i]+1]
  }
  parms = lm(cLogit ~ factor(DIS)+ factor(Straw) + factor(Density)+ factor(Floor), data)
  res = sum(parms$residuals**2)
  return(res)
}

lambda = c(1, 1, 1)
findFit(lambda, data)

getParms = function(lambda, data) {
  data$cLogit = data$Logit
  for (i in 1:length(data$Dis)) {
    data$cLogit[i] = data$Logit[i] / lambda[data$DIS[i]+1]
  }
  parms = lm(cLogit ~ factor(DIS)+ factor(Straw) + factor(Density)+ factor(Floor), data)
  return(parms)
}

parms = getParms(lambda, data)
summary(parms)

opt = optim(lambda, findFit, NULL, method = "Nelder-Mead", lower = -Inf, upper = Inf, control = list(maxit = 5000), hessian =FALSE, data)
opt

parms = getParms(opt$par, data)
summary(parms)
```

Figure C.4: R code for the optimization of the Physical complex.
# Combined probabilities for MH, ER, HP, SS given Straw, Density
# Floor, Herdsize, Production, Purchase
# The base for argument $i$ is used if useV = $i$

```
pCombined = function(X, Y, Z, A, B, E, x, y, z, a, b, e, useV) {
  bases = c(NA, NA, NA, NA, NA, NA)
  baseX = averageProb(X, x)
  baseLogitX = logit(baseX)
  bases[1] = baseLogitX
  baseY = averageProb(Y, y)
  baseLogitY = logit(baseY)
  bases[2] = baseLogitY
  baseZ = averageProb(Z, z)
  baseLogitZ = logit(baseZ)
  bases[3] = baseLogitZ
  baseA = averageProb(A, a)
  baseLogitA = logit(baseA)
  bases[4] = baseLogitA
  baseB = averageProb(B, b)
  baseLogitB = logit(baseB)
  bases[5] = baseLogitB
  baseE = averageProb(E, e)
  baseLogitE = logit(baseE)
  logitX = logit(X[,2])
  devX = logitX - baseLogitX
  logitY = logit(Y[,2])
  devY = logitY - baseLogitY
  logitZ = logit(Z[,2])
  devZ = logitZ - baseLogitZ
  logitA = logit(A[,2])
  devA = logitA - baseLogitA
  logitB = logit(B[,2])
  devB = logitB - baseLogitB
  logitE = logit(E[,2])
  devE = logitE - baseLogitE
  res = matrix(rep(NA, length(x)*length(y)*length(z)*length(a)*length(b)*length(e)*2),
              length(x)*length(y)*length(z)*length(a)*length(b)*length(e))
  c = 0
  for (i in 1:length(x)) {
    for (j in 1:length(y)) {
      for (k in 1:length(z)) {
        for (l in 1:length(a)) {
          for (m in 1:length(b)) {
            for (n in 1:length(e)) {
              c = c + 1
              res[c,1] = 1 - res[c,2]
            }
          }
        }
      }
    }
  }
  return(res)
}
```

Figure C.5: Infectious complex. R code for creating the combined probabilities.
findFit = function(lambda, data) {
  data$cLogit = data$Logit
  for (i in 1:length(data$Dis)) {
    data$cLogit[i] = data$Logit[i] / lambda[data$DIS[i]+1]
  }
  parms = lm(cLogit ~ factor(DIS)+ factor(PenDen) + factor(Floor)+ factor(Straw2) +
             factor(Herdsize2) + factor(Production2) +  factor(Purchase2), data)
  res = sum(parms$residuals**2)
  return(res)
}

lambda = c(1, 1, 1, 1)
findFit(lambda, data)

getParms = function(lambda, data) {
  data$cLogit = data$Logit
  for (i in 1:length(data$Dis)) {
    data$cLogit[i] = data$Logit[i] / lambda[data$DIS[i]+1]
  }
  parms = lm(cLogit ~ factor(DIS)+ factor(PenDen) + factor(Floor)+ factor(Straw2) +
             factor(Herdsize2) + factor(Production2) +  factor(Purchase2), data)
  return(parms)
}

parms = getParms(lambda, data)
parms
summary(parms)

opt = optim(lambda, findFit, NULL, method = "Nelder-Mead", lower = -Inf, upper = Inf,
            control = list(maxit = 5000), hessian =FALSE, data)
opt

parms = getParms(opt$par, data)
parms
summary(parms)

Figure C.6: R code for the optimization of the Infectious complex.