Genetic resistance to respiratory diseases in pigs

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Background

Infections in the respiratory tract are of frequent occurrence in pig production herds. These diseases are leading to decreased production, will jeopardise animal welfare and lead to increased costs. The increased costs are mainly caused by decreased growth rate, increased feed conversion ratio and higher mortality.

Catarrhal pneumonia, often caused by Mycoplasma hyopneumonia, will in some herds lead to chronic respiratory problems. Another more serious infection in the lungs are caused by the bacteria Actinobacillus pleuropneumonia (App) which could lead to severe problems of the lung function and also lead to pericarditis.

In earlier studies it has been shown that lung diseases have low heritability [Henryon et al, 2001, Lundeheim et.al, 1979]. In a large project, carried out in Denmark, lung lesions were registered at slaughter for 10.000 pigs from 12 sire groups. The results showed that there were large differences in offspring groups both for Mycoplasma and Actinobacillus infections. It was also shown that production traits, mainly growth rate, were correlated to lung problems. In a study from Japan genetic parameters in Landrace pigs were analysed and heritability for Mycoplasma was estimated to 0.14.

Lung diseases are almost impossible to diagnose and measure in breeding herds. Therefore a study was performed where lung lesion data from slaughtered pigs in ordinary pig production herds was used and where the pedigree of the pigs were known.

The objective of this study was to estimate the additive genetic variation in lung diseases in pigs, measured as lung lesions from slaughter pigs and to estimate the correlation to some main production traits. Response to selection and possible ways to implement a trait regarding lung diseases in production herds in a breeding program was also studied.

Materials and methods

Lung lesions in pig production

During 2004-2006 almost 9500 pigs were studied from birth to slaughter. Each pig was individually scored for 4 different lung diseases at the slaughter line by specially trained technicians and vets and slaughter weight and meat% were also registered.
Data were collected from one integrated production herd and 5 adjacent slaughter pig producing units. The 535 sows in the study were all LY-hybrids and the 170 boars used were among the best and youngest Duroc AI boars. In total 952 litters were produced. The data in the sow herd were registered individually regarding matings, AI-boars used, farrowings, parity numbers and litter size. The Duroc boars were used within 2-3 weeks period and 6-10 litters per boar were produced. More than half (52%) of the total number of sows produced two or more litters in this study. The parities were well spread over parity 1 to 5 with some litters from parity 6-8. During the slaughter production period (30 kg-) the pigs were kept in 6 different slaughter herds/stables.

Table 1. Data structure, sires and dams

Parameter	Mean	Std. dev.
No. of offspring per sire	73,8	40,4
No. of offspring per dam	23,4	14,9

All pigs were slaughtered in the same slaughter house. At slaughter, meat% and slaughter weight were registered (Table 2), as well as the lung lesions. The lungs were examined individually and scored by a veterinarian or technician. Lung data from the lung examined pigs were identified regarding identity and pedigree and used in the further analysis. The lung diseases examined were Catarrhal pneumonia, Pleuritis and Pleuropneumonia and Pericarditis as a 0-1 trait where 0 indicates no lung lesions and 1 indicates lung lesions found. The examination was done at the slaughter line and only one veterinarian or technician per slaughter day was scoring the lungs.

Table 2. Slaughtered pigs phenotypic values.

Trait	Mean	Std. dev.
Age at slaughter	171,1	15,8
Slaughter weight	80,2	6,8
Daily gain birth-slaughter	619,6	71,1
Meat%	61,3	2,5

Methods

The genetic parameters were estimated using a multitrait animal model

$$Y=Xb+Zu+a+e \qquad (2)$$

where the traits in Y are pleuritis, growth rate and meat content and the fixed effects in X consist of herd, parity, sex, technician and starting weight. The random effects in Z are litter and date of slaughter. The genetic component of the animal is $a \sim N(0,\sigma_a^2\mathbf{A})$ where **A** is the relationship matrix and e is the residual, b and u are vectors of proper order.
The model was estimated using VCE6 (Groenefeldt et. al. 2002).

Results

Incidence of lung lesions

Four types of lung lesions were measured at slaughter. One of them, pleuritis, had a very high prevalence (Table 3). A pig often had more than one lung lesion but rarely all four. Out of all pigs 4336 pigs just had scorings for pleuritis, 690 had both pleuritis and pericarditis. In total 14.2% had both catarrhal pneumonia and at least one of the other three lesions. Due to the low frequencies pneumonia, pleuropneumonia and pericarditis were not included in the genetic study.

Table 3. Prevalence of lung lesions

Parameter	Mean
Catarrhal pneumonia	0,143
Pleuritis	0,538
Pleuropneumonia	0,0127
Pericarditis	0,099

Environmental effects
Pigs were slaughtered from 6 different herds/stables. The frequencies of pleuritis were generally high in all herds. Catarrhal pneumonia was considerably high in some herds, in spite of vaccination early in life in the sow herd. Significances of fixed effects and variances of random effects were calculated where sire, litter and date at slaughter, nested within technician, were considered random. For the fixed effects herd, technician, sex, parity and growth rate were found to be significant. Litter size, slaughter weight and meat% were not significant (SAS Glimmix).

Table 4. Fixed effects, significance table

Fixed effects	**Number of levels**	**Catarrhal pneumonia 0-1**	**Pleuritis 0-1**
Class variables:			
Technician	7	***	***
Herd	6	***	**
Parity	8	n.s.	**
Sex	2	***	n.s.
Growth rate	1	***	***
Slaughter weight	1	n.s.	n.s.
Meat%	1	n.s.	n.s.
Litter size, born alive	1	n.s.	n.s.

There was a clear tendency that first parity offspring had higher probability of performing lung lesions for pleuritis. For catarrhal pneumonia this relationship was not found. Meat% did not phenotypically seem to affect the lung lesion. Growth rate significance indicates that increased lung lesions decrease daily gain (or the other way around).

Genetic components
In this multitrait model (2) data from production herds and breeding herds were used together. Lung lesion data came from the production herds. Data for growth rate and meat content used came from offspring in field testing and performance testing in the nucleus herds. The common ancestor were the 170 Duroc boars used in the study and all their relatives in the database. The calculated heritability for pleuritis was low. The genetic correlation found between pleuritis, growth rate was slightly positive and for meat content the genetic correlation was negative.

Table 6. Heritabilities and correlations. The heritabilities (diagonal) and genetic correlations (above the diagonal).

	Chronic pleuritis	Growth rate	Meat%
Chronic pleuritis	0,06	+0,07	-0,22
Growth rate		0,22	-0,10
Meat%			0,41

Discussion and conclusion

This study established that additive genetic variation for lung diseases is low but would be possible to use in breeding evaluation. The estimated heritabilities were in the same range as other studies published. There are some genetic correlations to production traits that are important and will affect genetic gain if the trait is implemented in the breeding program. The genetic correlation between pleuritis and meat content was -0.22 indicating that high prelvalence of lung diseases is genetically correlated to low meat content. On the other hand, in this study it was a weak genetic relationship between growth rate and pleuritis.

This study shows that breeding for resistance to pleuritis can be organised by transferring information from production herds to the breeding evaluation of the nucleus population. This could be organized by using reference production herds to collect lung lesion data. With known pedigree of the pigs in the reference herds it is possible to use the data in breeding evaluation of the sires. When calculating the timeframe per generation it is possible to generate breeding values for AI sires by using lung lesion information from production herds before selection of next generation of sons.

References

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