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Epidemiological and Genetical Studies in Norwegian Pig Herds

V. Estimates of Heritability and Phenotypic Correlations of the most Common Diseases in Norwegian Pigs

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Lingaas, F. and K. Rønningen: Epidemiological and genetical studies in Norwegian pig herds. V. Estimates of heritability and phenotypic correlations of the most common diseases in Norwegian pigs. Acta vet. scand. 1991, 32, 115–122. – A genetic analysis was performed on disease registrations from 70 Norwegian pig herds. This analysis was complicated by the small degree of cross classification of dams and sires across herds, and the relatively sparse use of artificial insemination. Herd effects seemed to be responsible for much of the variation in disease frequencies. The estimated heritabilities were relatively low. The results of this investigation also revealed positive phenotypic correlations between the investigated diseases.

swine; MMA-syndrome; mastitis; metritis; neonatal diarrhoea; arthritis; scrotal hernia; disease recording; health cards.

Introduction

Though no outbreaks of any serious infectious diseases have occurred in the Norwegian pig population for many years (*Central Bureau of Statistics in Norway* 1978–87), there has been a steady increase in the number of reported cases of the most common production diseases (*Landsrådet for husdyrkontrollen* 1986). Most of the treatments registered in association with piglet production concern 6 common diseases (*Lingaas & Rønningen* 1990), and attention has therefore to be focused on these if total disease incidence is to be lowered. If a program to reduce disease incidence is to be effective, various components such as improvement of environmental factors, immunprophylaxis and selection need to be integrated in an optimal manner.

Studies on the heritability of diseases in sows and piglets based on disease recordings in commercial herds, have, as far the authors are aware, not been performed previously. Some genetic parameters have been estimated for respiratory diseases in fatteners (*Lundeheim* 1979, *Lundeheim* 1986). These studies were, however, performed in a limited number of specialized herds. The importance of selection is still uncertain as information on several genetic parameters is lacking from commercial herds. The present study was carried out to estimate the heritability of some important diseases and their correlations.

Material and methods

Material

The present material consisted of clinical pig disease registration made in connection with a project to test a disease recording scheme based on health cards. The project which lasted for 3 years from 1984 to 1986, involved 70 herds, all of which participated in the Norwegian herd performance record-

ing scheme (*Landsrådet for husdyrkontrollen* 1986). No further selection of herds was made. Most of the herds had combined production, i.e. feeding their own piglets through to slaughter, while about 25 % of the farms produced only weaner pigs. A more detailed description of the health card registrations has been given elsewhere (*Lingaas & Rønningen* 1990).

Statistical methods

Heritabilities and correlations were estimated according to Hendersons method III using Harvey's LSMLMW program (*Harvey* 1985). Because of the small degree of cross classification of dams and sires across herds, several different statistical models were used:

Model 1. Sire cross classified with herd

$Y_{ijklmn} = \mu + R_i + S_{ij} + B_k + M_l + L_m + e_{ijklmn}$

where

Y_{ijklmn} = the reported disease status (1 or 0) on the individual

μ = Least squares mean

R_i = effect of breed (i)

S_{ij} = effect of sire (j) within breed (i)

B_k = effect of herd (k)

M_l = effect of month (l)

L_m = effect of number of piglets (m) in the litter

e_{ijklmn} = effect of random error

Model 2. Model without herd, but with sow

$Y_{ijklmn} = \mu + T_i + S_{ij} + D_{ijk} + M_l + L_m + e_{ijklmn}$

where

Y_{ijklmn} = the reported disease status (1 or 0) on the individual

μ = Least squares mean

T_i = effect of i'th herd status, where 1 = breeding herds, 2 = combined herds, 3 = weaner herds

S_{ij} = effect of j'th sire within herd status

D_{ijk} = effect of sow (k) within sire (j) within herd status (i)

M_l = effect of month (l)

L_m = effect of number of piglets (m) in the litter

e_{ijklmn} = effect of random error

Model 3. Model based on herd-residuals

$Y_{ijklmn} = \mu + R_i + S_{ij} + D_{ijk} + M_l + L_m + e_{ijklmn}$

where

Y_{ijklmn} = the reported disease status on the individual measured as deviation from herd mean

μ = Least squares mean

R_i = effect of breed (i)

S_{ij} = effect of sire (j) within breed (i)

D_{ijk} = effect of sow (k) within sire (j) within breed (i)

M_l = effect of month (l)

L_m = effect of number of piglets (m) in the litter

e_{ijklmn} = effect of random error

Weighted means of heritability

In some cases where there was lack of cross-classification of data, it was necessary to perform the analyses within subsets of the material. The results from the analyses within the sub-populations were then combined to give pooled estimates. Each of the single estimates was weighted according to the inverse of the variance in the following way:

$$h^2_{Pooled} = \frac{(h_1)^2/V_1 + (h_2)^2/V_2 + ... + (h_n)^2/V_n}{1/V_1 + 1/V_2 + ... + 1/V_n}$$

where h^2 = heritability, and V = the variance of the estimate.

The variance on the pooled estimate was calculated according to *Armitage & Berry* (1987).

The heritability of a mean

Estimates of the heritability of diseases in sows and piglets using models which do not include the effect of dam will actually be

estimates of means, each value being based on a variable number of observations.
The heritability of values based on means is given by:

$$h_m^2 = \frac{n}{1+(n-1)*t} * h^2$$

where t is the intraclass correlation (repeatability) and n is the number of observations in the mean (*Falconer* 1981). This formula was used to adjust the estimated heritabilities in cases where the estimates were based on repeated measurements on the same individual. The number of observations was known, and the heritability based on single observations could be estimated for different repeatabilities.
Estimates based on the binomial variable were transformed to correspond to those estimated directly from normally distributed data as outlined by *Dempster et al.* (1950).

Results

Estimates of heritability based on sires of the sows

Weighted estimates of the heritabilities of the six most important diseases as estimated in different herd categories are given in Table 1. As can be seen, the estimated heritabilities are small.

Table 1. The heritability (h^2) and standard error (s.e.) of 6 frequent diseases estimated by means of paternal half-sib correlation. (Sires used in 2 or more herds. Model 1).

	Weighted estimate $h^2 \pm$ s.e.
MMA-syndrome	.101 (.030)
Mastitis	.186 (.035)
Metritis	.152 (.033)
Neonatal diarrhoea	.029 (.086)
Arthritis (piglets)	.164 (.094)
Scrotal hernia	.102 (.089)

Table 2. Heritabilities for diseases in piglets estimated as paternal half-sib correlation. (Model 1).

	Weighted estimate $h^2 \pm$ s.e.
Neonatal diarrhoea	.053 (.036)
Arthritis	* –
Scrotal hernia	.141 (.039)

* = negative variance components.

Estimates of heritabilities based on the sires of the litters

When considering diseases in the piglets, it is of interest to compare the estimates based on the sires of the sow with the corresponding estimates based on the sires of the litters. The estimates, based on the sires of the litters, are shown in Table 2.
When estimating the heritability of arthritis in piglets by paternal half-sib correlation, the variance components were negative and it was not possible to compare them with the estimates in Table 1. As regards neonatal diarrhoea and scrotal hernia, however, there was good correspondence between the separate estimates.

Estimates of heritability derived from repeated measurements on the same animal

The estimates in Tables 1 and 2 have not been adjusted for the effect of repeated measurements on the same individual (sow), and are therefore probably too high. The mean number of recordings per sow in the material was 2.2. Earlier studies have shown that most of the important diseases in pigs show a tendency to recur (*Jorsal* 1983, *Lingaas* 1991b). This may indicate that the repeatability is greater than 0.5. Using the formula for heritability based on mean values, and repeatability values of 0.5, 0.7 and 0.9, it is possible to obtain estimates of heritabilities based on single observations. The results are given in Table 3.

Table 3. Adjusted estimates of heritability of single observations using the formula for the heritability of a mean. Non-adjusted estimates from Table 1.

	Non-adjusted h^2	Repeatability 0.5	0.7	0.9
MMA-syndrome	0.101	0.073	0.084	0.095
Mastitis	0.186	0.135	0.156	0.175
Metritis	0.152	0.111	0.127	0.144
Neonatal diarrhoea	0.029	0.021	0.024	0.027
Arthritis	0.164	0.119	0.137	0.155
Scrotal hernia	0.102	0.074	0.085	0.096

This table shows that heritabilities adjusted for repeated measurements in this case, depending on the repeatability, varies from about 70 % and upwards of the non-adjusted values.

Estimates of heritability when ignoring herd effects

Herd effects are usually considerable, a fact that was more evident when heritabilities were estimated without herd in the model. The results are given in Table 4. The heritabilities estimated with this model (Model 2) were medium sized.

Estimates of heritabilities based on residuals (deviations from herd mean)

Using the herd-residuals, it was possible to adjust for herd and dam in the same model. This approach was used on the material derived from herds which shared sires with at least 1 other herd. Estimates were also made using all sires regardless of the number of herds in which they were used. Results are given in Table 5. The estimates of heritabilities were in the range from 0.7 % to 3.8 % for the MMA-syndrome, metritis and piglet arthritis. For the other 3 diseases, Model 3 resulted in negative variance components.

Table 4. Estimates of heritability of 6 common diseases. Paternal (sires of sows) half-sib correlation, model without herd (Model 2).

	h^2	± s.e.
MMA-syndrome	.275	(.048)
Mastitis	.218	(.045)
Metritis	.231	(.046)
Neonatal diarrhoea	.036	(.066)
Arthritis (piglets)	.630	(.102)
Scrotal hernia	.048	(.066)

Transformation of heritabilities to the underlying normally distributed variable

Dempster et al. (1950) showed that it is possible to transform estimates of categorial data to the theoretical heritability based on a normal distributed scale. In the present material transformation according to *Dempster* in several cases resulted in too high heritabilities, which probably partly were due to the low disease frequencies.

Estimates of phenotypic correlations

The phenotypic correlations between the different diseases are shown in Table 6. This

Table 5. Estimates of heritability of 6 common diseases. The estimates are based on paternal (sires of sows) half-sib correlation, model 3 (herd-residuals). Standard errors given in parentheses.

Sires used in Number of observations	Model 3 > 1 herd 5596	Model 3 All sires 7280
MMA-syndrome	.007 (.024)	0.21 (.026)
Mastitis	–	–
Metritis	.019 (.026)	.029 (.026)
Neonatal diarrhoea	–	–
Arthritis (piglets)	.038 (.050)	.002 (.048)
Scrotal hernia	–	–

Table 6. Estimates of phenotypic correlations between diseases – Model 1.

		Mastitis	Metritis	Neonatal diarrhoea	Arthritis	Scrotal hernia
MMA-syndrome	a	0.206	0.112	0.171	–0.009	–0.051
	b	0.114	0.070	0.125	0.021	–0.017
Mastitis	a		0.118	0.062	0.121	0.042
	b		0.031	0.062	–0.020	0.025
Metritis	a			–0.008	0.114	0.026
	b			0.032	–0.027	0.104
Neonatal diarrhoea	a				–0.025	0.001
	b				0.041	–0.001
Arthritis	a					–0.009
	b					–0.008

a: Specialized production; piglet production and breeding herds.
b: Ordinary combined production.
All the correlations were significantly different from zero at the 1 % level.

table shows that the phenotypic correlations between the separate diseases are positive or near zero.

Discussion

The analysis of disease registrations from Norwegian pig herds is associated with certain methodological difficulties. These are mainly due to small herd size, and a relatively sparce use of artificial insemination, and a consequently low degree of cross classification of sires between herds. Another important factor is the short farrowing interval which usually results in each sow producing 2 litters every year. It is therefore necessary to take account of the effect of the sow when analyzing data. Models which do not take the sow into consideration will in fact estimate the heritability of the disease based on mean values.

A good model for estimation of genetical and environmental effects would be a model including the effect of breed, sire within breed, dam within sire and breed as well as the effect of herd and other environmental factors. Because the dams are not used in more than 1 herd (not cross classified with herd), this model could not be applied. In some cases, the degree of cross classification of sires over herds was also a limiting factor. Herd and herd status both seem to exert a major influence on disease incidence (*Lingaas* 1991a), and it is therefore, necessary to consider herd effects when estimating the genetic parameters. As there is no cross classification between sows and herds, however, it is not possible to consider both sow and herd in the same model, the consequence being that the sow effect must be deleted from the optimal model. Alternatively, analyses could be performed on "blocks" of short time span, less than half a year, each sow thereby usually just contributing information from one farrowing. This would, however, further adversely affect the degree of cross classification between sire and herd. This means that no single model is entirely satisfactory, and it is therefore necessary to analyze the data employing several alternative models, all with minor disadvantages. This is in accordance with earlier analyses on pig data (*Standal* 1977).

The main disadvantages when working with Model 1 is that the same sow could contri-

bute more than 1 observation to the material. When estimating the heritability of diseases as half-sib correlation (paternal half-sib), the mean relationship between sows is set at 0.25. In cases where each animal is represented by more than 1 observation in the data set, the mean genetic relationship between half-sib groups will be greater than 0.25, and this leads to biased estimates. It is possible to adjust the estimates using the formula for heritability based on mean values.

Model 2 does not consider herd effects. This model will therefore overestimate the genetic effects, especially in cases with small herds and sparce use of artificial insemination as some of the herd effect will appear to be the effect of genetic differences between sires. Such overestimation means that the heritability estimates given in Table 4 are too high. Even if the herds and the sire groups are small, an adjustment for herd does not seem to be disadvantageous (*Henderson* 1974).

One possibility for dealing with the problem of lack of cross classification between sires and herds is to use models based on herd residuals. It has, however, been shown that models based on residuals may be biased (*Van Vleck et al.* 1961), and estimates of heritability based on residuals may lead to underestimation of heritabilities (*Syrstad* 1966, *Eijke* 1974a,b).

Estimating heritabilities on binomial variables often leads to underestimation compared to cases where heritability can be estimated directly on a continuous, normally distributed scale (*Dempster et al.* 1950, *Van Vleck* 1972). Transformation to the normally distributed values will usually give accurate estimates as long as the incidence of disease in greater than 15–20 % (*Van Vleck* 1972, *Olausson & Rønningen* 1975). The estimated incidences in this material are lower than this, and the transformation will therefore be biased. The factor z used for many diseases in this material were high, and some of the corresponding heritabilities estimated on a normal distributed scale were higher than 1.

When selecting for various traits it is important to know the effect on disease resistance. If there are positive biological genetic correlations between performance traits and disease resistance selection for increased production will also improve disease resistance, and vice versa. The genetic correlations between performance traits and disease resistance are not well known in pigs. However, there are indications that pigs with the K-88 receptor in the intestine show a higher daily weight gain than pigs without the receptor (*Edfors-Lilja et al.* 1986). In cattle there is positive statistical genetic correlation between milk production and the incidence of mastitis (*Syväjärvi et al.* 1986, *Madsen et al.* 1987), and a positive statistical genetic correlation between milk production and acetonemia has also been reported (*Gröhn et al.* 1986). Though the present material was too small to estimate genetic correlations, most of the phenotypic correlation were positive. Further studies on genetic correlations should therefore be performed.

The heritabilities of diseases, as estimated in the present study, seem to be low. This finding agrees with other reports on the heritability of clinical diseases (*Lush et al.* 1948, *Lundeheim* 1979, Solbu 1984). It is therefore important to base selection on information from progeny, and half- and/or full sibs if satisfactory accuracy is to be obtained. Selection based on full sibs and half sibs would probably be the best where no progeny testing is performed. When selection is based on half sibs, data from a large number of half sibs is required to achieve satisfactory accuracy. Siblings will usually comprise a

valuable source of information for the selection of breeding pigs, and selection based on indexes could increase accuracy without prolonging the generation interval. In this context, the use of an animal model would be an effective way of using pedigree information to increase accuracy (*Henderson* 1977). A heritability of 6 % would give an R^2 of 6 % when 20 half sibs, 11 % with 50 half sibs and 15 % with 100 half sibs. The corresponding R^2 based on progeny testing would be 24 %, 44 % and 60 %, respectively, and disease recording would then have to encompass a great number of herds if reasonable breeding progress for disease resistance in pigs is to be achieved.

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Sammendrag

Epidemiologiske og genetiske studier av sjukdommer i norske svinebesetninger. V. Arvbarheter og korrelasjoner for noen viktige sjukdommer hos svin.

På grunnlag av sjukdomsregistreringer i 70 svinebesetninger er det utført genetiske undersøkelser for seks vanlig forekommende sjukdommer hos svin. Datastrukturen, med en liten grad av krysssklassifisering av data over besetning, gjorde analysene kompliserte. Flere alternative statistiske modeller ble derfor brukt i analysene. De estimerte arvbarhetene var forholdsvis lave. Det ble påvist signifikante, men lave, positive fenotypiske korrelasjoner mellom de undersøkte sjukdommene.

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