Mixed Model Studies on Inheritance of Reproductive Traits in Laying Hens – A Bayesian Approach[1]

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ABSTRACT Segregation analyses performed for many livestock species indicate a mixed inheritance model of reproductive traits. Additionally, depending on the population, a given trait can be determined by a number of genes with large effects. Genetic backgrounds of hatchability and fertility in poultry are still not known sufficiently. The objectives of this study are to verify the hypothesis on segregation of single genes (1 vs. 2) affecting fertility and hatchability and to estimate a heritability of these traits. Records from 2,040 and 2,015 dams from full-pedigreed strains of Rhode Island Red (R33) and New Hampshire (N88) from a pedigree farm were analyzed. The percentage of fertilized eggs and the percentage of the eggs hatched of fertilized eggs were registered for dams only. Fertility was checked by candling on the eighth day of incubation. To obtain a binomial phenotypic scale, 10 eggs per dam were included into the analysis. Animal single-trait threshold models were used for the analysis of data. The first model included the effects of 2 single genes, 2 fixed effects of year and season, additive polygenic effects, and permanent environmental effects. In the second model, only 1 single gene effect was included. Additionally, the analysis based on the polygenic threshold model was also performed. The Gibbs sampling procedure was used. The significance of single gene effects was verified by highest posterior density regions. The obtained results clearly gave evidence for the segregation of 1 major gene for hatchability in strain R33. Furthermore, the mixed inheritance model can also be suggested for fertility in this strain. After the analysis, the polygenic heritabilities were very low (<0.11), whereas major polygenic heritability ranged from 0.05 to 0.12.

Key words: Gibbs sampling, laying hen, reproductive trait, single gene, threshold model

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INTRODUCTION

There are many reports on segregation of single loci determining egg production (Tuiskula-Haavisto et al., 2002), meat production (Rao et al., 2007), feed efficiency (Van Kaam et al., 1999), disease resistance (Bumstead, 1998), and skeletal integrity (Zhou et al., 2007). By contrast to these traits, a current knowledge on genetic determination of fertility and hatchability seems to be still relatively superficial. It is well known that reproductive characters are lowly heritable (Szwaczkowski, 2003). In consequence, selection effectiveness is decreased. Moreover, biological backgrounds of these traits are complex. Fertility can be considered as an interaction of male and female gametes to produce a viable zygote. In practice, the percentages of fertilized eggs are recorded for females. Reproductive traits become problematic in the statistical analysis, because they do not follow normal distribution. This can lead to underestimation of genetic parameters. Measurements of fertility are often biased. The percentage of fertilized eggs may be underestimated by embryonic mortality (Jassim et al., 1996).

Selection intensity may increase when information on the existence of single loci is included into genetic improvement programs (Lahav et al., 2006). At the moment, many major genes affecting reproductive ability are identified for livestock, mainly for sheep (Davis, 2005), cattle (Cobanoglu et al., 2005), and pigs (Rathje et al., 1997). Druyan and Cahaner (2007) suggested that 2 complementary dominant genes affect ascites resistance and ascites susceptibility in broilers.

Bayesian marker-free segregation analysis seems to be a very useful tool for detection of single loci (Kadarmideen and Janss, 2005). This approach supplies information on hypothetical genotypic effects and their frequencies. Both fertility and hatchability are recorded as binary traits. These traits are influenced by many genetic and environmental factors, so the threshold animal model seems to be the most adequate for their statistical analysis

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Table 1. Description of the data sets

Item	Strain R33	Strain N88
Number of pedigreed birds	3,070	4,073
Number of recorded birds		
Fertility	2,040	3,461
Hatchability	2,015	3,430
Fertility (%)	87.39	90.05
Hatchability (%)	61.23	70.95

(Sørensen et al., 1995; Bennewitz et al., 2007; Skotarczak et al., 2007). In this model, it is assumed that a categorical distribution of observations is determined by an unobserved continuous variable called liability. For the binary recorded traits, the threshold between 2 categories is set in point 0 and the variance component for liability is 1.

The objectives of this study were to verify the hypothesis on segregation of single genes (1 vs. 2) affecting fertility and hatchability and to estimate heritability of these traits.

MATERIALS AND METHODS

Material

Data of 2,040 and 2,015 dams from full-pedigreed strains of Rhode Island Red (R33) and New Hampshire (N88) from 1 pedigree farm located in Poland were analyzed.

The birds were naturally mated and kept on litter. The environmental conditions (e.g., feeding level) did not change considerably over time. Both populations were kept under long-term selection. The within-strain generation selection was based on the classical selection index described by Wężyk (1978). The five following production traits were included in the selection: initial egg production (until the 35th week), average egg weight between the 33rd and 35th week of age, body weight at the 18th week of age, and age at sexual maturity. Fertility was checked by candling on the eighth day of incubation. The percentage of fertilized eggs and the percentage of the eggs hatched of fertilized eggs were registered for dams only. These reproductive eggs were collected between the 45th and 54th week of age. In a binomial phenotypic scale, 10 eggs per dam were included into the analysis. The number of reproductive eggs studied was restricted by the algorithm applied.

The data were classified according to generation (8 levels) and hatch period (4 levels). Averages for studied traits fluctuated negligible over generations and hatch periods. Therefore, they are not shown in the present study. The brief description of the data sets is given in Table 1.

Methods

The following statistical models were considered:

$$\text{model 1: } \mathbf{u} = \mathbf{X}\boldsymbol{\beta} + \mathbf{ZW}\boldsymbol{\mu} + \mathbf{Za} + \mathbf{Sp} + \mathbf{e}$$

$$\text{model 2: } \mathbf{u} = \mathbf{X}\boldsymbol{\beta} + \mathbf{ZW}\boldsymbol{\mu} + \mathbf{ZV}\boldsymbol{\nu} + \mathbf{Za} + \mathbf{Sp} + \mathbf{e}$$

where $\mathbf{u}$ = a ($10s \times 1$) vector of unobserved liability; $\mathbf{X}$ = a ($10s \times b$) design matrix of nongenetic effects; $\boldsymbol{\beta}$ = a ($b \times 1$) vector of fixed effects of hatch period (4 levels) and generation (8 levels); $\mathbf{Z}$ = a ($10s \times q$) design matrix relating polygenic and single locus effects to observations; $\mathbf{W}$ and $\mathbf{V}$ = ($q \times 3$) random matrices containing information of genotype of each individual, each row of these matrices has 1 of the following forms: [1, 0, 0], [0, 1, 0], or [0, 0, 1] corresponding to genotypes *AA*, *Aa*, or *aa* and *BB*, *Bb*, or *bb*, respectively; $\boldsymbol{\mu} = [\mu_{AA}, 0, -\mu_{AA}]'$ and $\boldsymbol{\nu} = [\nu_{BB}, 0, -\nu_{BB}]'$ = the vectors in which the first element is the effect of dominant homozygote (e.g., *AA*), the effect of heterozygote (e.g., *Aa*) is assumed to be equal to 0 and the effect of recessive homozygote (e.g., *aa*) is the opposite of the effect of dominant homozygote; $\mathbf{a}$ = a ($q \times 1$) vector of random additive polygenic effects; $\mathbf{S}$ = a ($10s \times s$) design matrix relating environmental effects to observations; $\mathbf{p}$ = a ($s \times 1$) vector of permanent environmental effects; and $\mathbf{e}$ = a ($10s \times 1$) vector of random errors effects. Moreover,

Table 2. Posterior means (and standard deviations) of allele frequencies, effects of dominant homozygotes, and single gene variances[1]

Strain	Model	f_A	f_B	μ_{AA}	μ_{BB}	σ^2_{GA}	σ^2_{GB}
R33	1	0.308 (0.108)	—	0.287 (0.127) [0.024; 0.505]	—	0.038 (0.028) [0; 0.093]	—
	2	0.367 (0.114)	0.612 (0.110)	0.241 (0.114) [0.017; 0.452]	0.221 (0.139) [0; 0.484]	0.031 (0.026) [0; 0.085]	0.031 (0.033) [0; 0.101]
N88	1	0.414 (0.091)	—	0.198 (0.094) [0.009; 0.372]	—	0.022 (0.018) [0; 0.0586]	—
	2	0.537 (0.108)	0.442 (0.091)	0.15 (0.097) [0; 0.338]	0.076 (0.06) [0; 0.206]	0.015 (0.016) [0; 0.050]	0.004 (0.006) [0; 0.019]

[1]Values in brackets are 95% highest posterior density regions for effects of dominant homozygotes and single gene variances for fertility. f_A and f_B = frequencies of alleles *A* and *B*; μ_{AA} and μ_{BB} = effects of dominant homozygotes of *AA* and *BB*; σ^2_{GA} and σ^2_{GB} = single gene variances.

s denotes the number of recorded hens, and q is equal to the number of individuals included in the pedigree.

The Bayesian methods with Gibbs sampling algorithm have been used to the estimation of unknown parameters in the above introduced models. The known formulas for the ordinary threshold animal model have been adapted for the case when repeated observations were collected for 1 individual. Improper prior uniform distributions were assumed for vectors $\boldsymbol{\beta}$, $\boldsymbol{\mu}$, and $\boldsymbol{\nu}$. The following multivariate normal distributions were assumed for the random vectors: $\mathbf{a} \sim N(\mathbf{0}, \mathbf{A}\sigma_a^2)$, where $\mathbf{A}$ = a $(q \times q)$ relationship matrix, $\mathbf{p} \sim N(\mathbf{0}, \mathbf{I}_s\sigma_p^2)$, $\mathbf{e} \sim N(\mathbf{0}, \mathbf{I}_{10s})$. The inverted χ^2 distribution was taken as a prior distribution of variance components σ_a^2 and σ_p^2.

Because it is well known, the conditional posterior distributions for vectors $\boldsymbol{\beta}$, $\boldsymbol{\mu}$, $\boldsymbol{\nu}$, $\mathbf{a}$, and $\mathbf{p}$ in the presented models are normal with means equal to the solutions of the appropriate mixed model equations (Sørensen and Gianola, 2002). In the considered case, the number of the mixed model equations is 10 times higher than the number of observed individuals. Therefore, the following formulas were used to calculate the expected values and variances in every step of Gibbs sampling procedure; for the vector of fixed effects $\boldsymbol{\beta}$:

$$\beta_i \sim N[(\mathbf{x}_i'\mathbf{x}_i)^{-1}\mathbf{x}_i'(\mathbf{u} - \mathbf{X}_{-i}\boldsymbol{\beta}_{-i} - \mathbf{ZW}\boldsymbol{\mu} - \mathbf{ZV}\boldsymbol{\nu} - \mathbf{Za} - \mathbf{Sp}); \quad (\mathbf{x}_i'\mathbf{x}_i)^{-1}]$$

where $\mathbf{x}_i$ = the ith column of $\mathbf{X}$; $\mathbf{X}_{-i}$ = matrix $\mathbf{X}$ without the ith column; $\boldsymbol{\beta}_{-i}$ = vector $\boldsymbol{\beta}$ without the ith element, $i = 1,...,b$; for the vector of additive genetic effects $\mathbf{a}$:

$$a_i \sim N\left[(\mathbf{z}_i'\mathbf{z}_i + \frac{1}{\sigma_a^2}A_{i,i})^{-1}[\mathbf{z}_i'(\mathbf{u} - \mathbf{X}\boldsymbol{\beta} - \mathbf{ZW}\boldsymbol{\mu} - \mathbf{ZV}\boldsymbol{\nu} - \mathbf{Sp}) - \frac{1}{\sigma_a^2}\mathbf{A}_{i,-i}\mathbf{a}_{-i}]; \quad \left(\mathbf{z}_i'\mathbf{z}_i + \frac{1}{\sigma_a^2}A_{i,i}\right)^{-1}\right]$$

where $\mathbf{z}_i$ = the ith column of $\mathbf{Z}$; $A_{i,i}$ = the element of $\mathbf{A}^{-1}$ in the ith row and ith column; $\mathbf{A}_{i,-i}$ = the ith row of matrix $\mathbf{A}^{-1}$ without the ith element; $\mathbf{a}_{-i}$ is vector $\mathbf{a}$ without the ith element, $i = 1,...,q$. In both formulas, the element $\mathbf{ZV}\boldsymbol{\nu}$ should be used in model 2 only.

Further, for the elements of vectors $\boldsymbol{\mu}$ and $\boldsymbol{\nu}$, the following assumptions were made:

$$\mu_{AA} \sim N\left((\mathbf{w}_1'\mathbf{w}_1 + \mathbf{w}_3'\mathbf{w}_3)^{-1}[(\mathbf{zw})_1'(\mathbf{zw})_1 - (\mathbf{zw})_3'(\mathbf{zw})_3] \; (\mathbf{u} - \mathbf{X}\boldsymbol{\beta} - \mathbf{ZV}\boldsymbol{\nu} - \mathbf{Za} - \mathbf{Sp}); (\mathbf{w}_1'\mathbf{w}_1 + \mathbf{w}_3'\mathbf{w}_3)^{-1}\right)$$

where $\mathbf{w}_1'$ and $\mathbf{w}_3'$ = the 1st and 3rd column of matrix $\mathbf{w}_1$ and $(\mathbf{zw})_1'$ and $(\mathbf{zw})_3'$ = the 1st and 3rd column of matrix $\mathbf{ZW}$, respectively.

$$\nu_{BB} \sim N\left((\mathbf{v}_1'\mathbf{v}_1 + \mathbf{v}_3'\mathbf{v}_3)^{-1}[(\mathbf{zv})_1'(\mathbf{zv})_1 - (\mathbf{zv})_3'(\mathbf{zv})_3] \; (\mathbf{u} - \mathbf{X}\boldsymbol{\beta} - \mathbf{ZW}\boldsymbol{\mu} - \mathbf{Za} - \mathbf{Sp}); (\mathbf{v}_1'\mathbf{v}_1 + \mathbf{v}_3'\mathbf{v}_3)^{-1}\right)$$

where $\mathbf{v}_1$ and $\mathbf{v}_3$ = the 1st and 3rd column of matrix $\mathbf{V}$ and $(\mathbf{zv})_1$ and $(\mathbf{zv})$ = the 1st and 3rd column of matrix $\mathbf{ZV}$. In every step of Gibbs sampling procedure, it was assumed that $\mu_{AA} > 0$ and $\nu_{BB} > 0$.

The following conditional posterior normal distribution was assumed for the unobservable liability: $u_i \sim N[\mathbf{x}_i^R\boldsymbol{\beta} + (\mathbf{zw})_i^R\boldsymbol{\mu} + (\mathbf{zv})_i^R\boldsymbol{\nu} + \mathbf{z}_i^R\mathbf{a} + \mathbf{s}_i^R\mathbf{p}; \quad 1]$, where $\mathbf{x}_i^R$ = the ith row of matrix $\mathbf{X}$; $(\mathbf{zw})_i^R$ = the ith row of matrix $\mathbf{ZW}$; $(\mathbf{zv})_i^R$ = the ith row of matrix $\mathbf{ZV}$; $\mathbf{z}_i^R$ = the ith row of matrix $\mathbf{Z}$, $i = 1,...,10s$. The expression $(\mathbf{zv})_i^R\boldsymbol{\nu}$ should be omitted in model 1.

Moreover, the following inverted χ^2 distributions were assumed for variance components:

$$p(\sigma_a^2 \mid \boldsymbol{\beta}, \mathbf{a}, \mathbf{p}, \mathbf{u}, \mathbf{G}, \sigma_p^2) \propto (\sigma_a^2)^{-\left(\frac{q+\nu_a}{2}+1\right)} \exp\left(-\frac{\mathbf{a}'\mathbf{A}^{-1}\mathbf{a} + v_aS_a^2}{2\sigma_a^2}\right)$$

and

$$p(\sigma_p^2 \mid \boldsymbol{\beta}, \mathbf{a}, \mathbf{p}, \mathbf{u}, \mathbf{G}, \sigma_a^2) \propto (\sigma_p^2)^{-\left(\frac{s+\nu_p}{2}+1\right)} \exp\left(-\frac{\mathbf{p}'\mathbf{p} + v_pS_p^2}{2\sigma_p^2}\right),$$

where $\nu_a, S_a^2, \nu_p, S_p^2$ = the hyperparameters (ν_a and ν_p are degrees of freedom, S_a^2 and S_p^2 are scale parameters).

According to the suggestions of Guo and Thompson (1994), the elements of the unknown genotypes table $\mathbf{G}$ were generated from the following formula:

$$p(G_i \mid \boldsymbol{\beta}, \mathbf{a}, \mathbf{p}, \mathbf{u}, \sigma_a^2, \sigma_p^2, \mathbf{G}_{-i}) \propto \left[\prod_{o_i} P(G_{o_i} \mid G_i, G_{m_i})\right] P(G_i \mid G_{s_i}, G_{d_i}) \exp\left(-\frac{[\mathbf{z}_i'(\mathbf{u} - \mathbf{X}\boldsymbol{\beta} - \mathbf{ZW}\boldsymbol{\mu} - \mathbf{ZV}\boldsymbol{\nu} - \mathbf{Za} - \mathbf{Sp})]^2}{2}\right)$$

where G_i = the genotype of the ith individual; $\mathbf{G}_{-i}$ = a table of the genotypes of all individuals excluding the ith individual; G_{o_i} = the genotype of the progeny of the ith individual; G_{m_i} = the genotype of the ith individual's mate; G_{s_i}, G_{d_i} are the genotypes of the ith individual's parents, $i = 1,...,q$. When the individual is not observed, the last term will be substituted by 1. Moreover, the element $\mathbf{ZV}\boldsymbol{\nu}$ was used in model 2 only. It should be stressed that in model 2, the table $\mathbf{G}$ was generated for each matrix $\mathbf{W}$ and $\mathbf{V}$ separately. In both models in the first step of Gibbs sampling, it was assumed that matrices $\mathbf{W}$ and $\mathbf{V}$ have

Table 3. Posterior means (and standard deviations) of allele frequencies, effects of dominant homozygotes, and single gene variances[1]

Strain	Model	f_A	f_B	μ_{AA}	μ_{BB}	σ^2_{GA}	σ^2_{GB}
R33	1	0.547 (0.071)	—	0.494 (0.068) [0.354; 0.625]	—	0.121 (0.032) [0.058; 0.183]	—
	2	0.574 (0.065)	0.684 (0.128)	0.528 (0.051) [0.462; 0.679]	0.114 (0.076) [0; 0.264]	0.135 (0.027) [0.082; 0.188]	0.008 (0.01) [0; 0.030]
N88	1	0.528 (0.115)	—	0.056 (0.048) [0; 0.165]	—	0.003 (0.004) [0; 0.013]	—
	2	0.375 (0.108)	0.381 (0.114)	0.063 (0.046) [0; 0.163]	0.158 (0.064) [0.02; 0.28]	0.003 (0.003) [0; 0.013]	0.013 (0.009) [0; 0.001]

[1]Values in brackets are 95% highest posterior density regions for effects of dominant homozygotes and single gene variances for fertility. f_A and f_B = frequencies of alleles A and B; μ_{AA} and μ_{BB} = effects of dominant homozygotes of AA and BB; σ^2_{GA} and σ^2_{GB} = single gene variances.

the following form: $\mathbf{W} = \mathbf{V} = [\mathbf{0}:\mathbf{1}:\mathbf{0}]$. Further, for the major gene, Mendelian transmission probabilities were assumed.

To estimate the genotypes for the individuals, the frequencies of alleles among the founders groups are required. The frequencies were generated from the beta distribution according to the following formulas (Kadarmideen and Janss, 2005):

$$f(f_A \mid \boldsymbol{\beta}, \mathbf{a}, \mathbf{u}, \sigma^2_a, \sigma^2_p, \mathbf{G}) \propto f_A^{n_A} (1 - f_A)^{n_a}$$

$$f(f_B \mid \boldsymbol{\beta}, \mathbf{a}, \mathbf{u}, \sigma^2_a, \sigma^2_p, \mathbf{G}) \propto f_B^{n_B} (1 - f_B)^{n_b}$$

where n_A and n_a = the number of alleles A and a and n_B and n_b = the number of alleles B and b, respectively, in the group of founders.

The significance of the major gene effects was checked on the basis of the highest posterior density regions (**HPDR**; Scott, 1992), which were built for the major gene variance components $\sigma^2_{GA} = 2f_A(1 - f_A)\mu^2_{AA}$ and $\sigma^2_{GB} = 2f_B(1 - f_B)\mu^2_{BB}$. For example, if the 95% HPDR for σ^2_{GA} included value 0, it was stated that the effect of AA genotype is not significant.

Moreover, additional analyses in a submodel were conducted. The submodel did not contain the major genes effect (i.e., it had the following form: $\mathbf{u} = \mathbf{X}\boldsymbol{\beta} + \mathbf{Za} + \mathbf{Sp} + \mathbf{e}$). The estimation of parameter $\boldsymbol{\beta}$, $\mathbf{a}$, $\mathbf{p}$, $\mathbf{u}$, σ^2_a, and σ^2_p in the submodel was carried out in a similar way as in models 1 and 2, but in the formulas for conditional posterior distributions, the components containing $\boldsymbol{\mu}$ and $\boldsymbol{\nu}$ were omitted.

On the basis of the estimated variance components, the following genetic parameters were calculated:

$$\text{major gene heritability: } h^2_G = \frac{\sigma^2_{GA} + \sigma^2_{GB}}{\sigma^2_a + \sigma^2_{GA} + \sigma^2_{GB} + \sigma^2_p + 1},$$

$$\text{polygenic heritability: } h^2_a = \frac{\sigma^2_a}{\sigma^2_a + \sigma^2_{GA} + \sigma^2_{GB} + \sigma^2_p + 1},$$

$$\text{total heritability: } h^2_t = \frac{\sigma^2_a + \sigma^2_{GA} + \sigma^2_{GB}}{\sigma^2_a + \sigma^2_{GA} + \sigma^2_{GB} + \sigma^2_p + 1}, \text{ and}$$

$$\text{repeatability: } r = \frac{\sigma^2_a + \sigma^2_{GA} + \sigma^2_{GB} + \sigma^2_p}{\sigma^2_a + \sigma^2_{GA} + \sigma^2_{GB} + \sigma^2_p + 1}.$$

In the above formulas, σ^2_{GB} is omitted in model 1, and σ^2_{GA} and σ^2_{GB} are omitted in the submodel.

The simulation studies were performed previously to verify the properties of the implemented algorithm (Skotarczak et al., 2007).

In each analyzed case, 1,000,000 rounds of Gibbs sampling were conducted. The first 400,000 steps were discarded as a burn-in period. The important results were collected from every 10th iteration. The means of the posterior distributions were calculated as the point estimators of the unknown parameters.

RESULTS AND DISCUSSION

Estimated posterior means and standard deviations of allele frequencies, single gene variance, dominant homozygote effects, as well as their HDPR for fertility and hatchability are listed in Tables 2 to 5, respectively. The HDPR for single gene variance were used as criteria for segregation of major genes. From this perspective, the obtained results clearly gave evidence for the segregation of a major gene for hatchability in strain R33 (Table 3). It should be stressed that the same result was confirmed in 2 models (with 1 and 2 single gene effects). Major gene variance is the main criterion for mixed model inheritance under Bayesian marker free segregation analysis (Janss et al., 1995). It was successfully applied by Kadarmideen and Janss (2005) to detect a segregation of major gene for osteochondrosis in pigs. However, by contrast to reproductive traits, heritability of osteochondrial diseases is relatively high. Two posterior densities for single gene variances for hatchability for strain R33 are presented in Figures 1 and 2. In both the illustrated cases, the posterior mode was close to the posterior mean.

Table 4. Posterior means (and standard deviations) of additive and permanent environmental variances; estimates of heritability and repeatability coefficients for fertility[1]

	Strain R33						Strain N88					
Models	σ_a^2	σ_p^2	h_G^2	h_a^2	h_t^2	r	σ_a^2	σ_p^2	h_G^2	h_a^2	h_t^2	r
Model 1	0.102 (0.052)	0.440 (0.046)	0.024	0.064	0.089	0.367	0.055 (0.029)	0.486 (0.035)	0.014	0.035	0.049	0.360
Model 2	0.102 (0.048)	0.421 (0.049)	0.039	0.064	0.103	0.369	0.069 (0.029)	0.478 (0.036)	0.012	0.044	0.056	0.361
Submodel	0.178 (0.054)	0.553 (0.047)	—	0.103	—	0.422	0.072 (0.03)	0.490 (0.036)	—	0.046	—	0.360

[1] σ_a^2 = additive polygenic variance; σ_p^2 = permanent environmental variance; h_G^2 = major gene heritability; h_a^2 = additive polygenic heritability; h_t^2 = total heritability; r = repeatability.

A number of single loci were identified in poultry (Hocking, 2005). The genes with the largest effects (for instance, sex-linked dwarfing gene) were detected by the use of simple statistical tools. However, genes identified recently have relatively small effects. Therefore, more sophisticated approaches for detection of quantitative trait loci were developed (Kadarmideen and Janss, 2005). The identification of loci, especially for low heritable traits, becomes more difficult. Thus, by definition of heritability, participation of genetic components in phenotypic variability of a given trait is very small as well. Furthermore, as it has already been mentioned, the studied traits are modeled on an unobservable underlying liability scale. Investigations carried out in livestock populations exhibited segregation of more than 1 gene for some traits (e.g., Davis, 2005) depending on population. In the present study, both strains were originated from 2 pure breeds. Hence, various gene pools were also observed. Fertility and hatchability were not directly included into the genetic improvement program, but they were recorded for dams. Both negative and positive correlations between egg production and reproduction traits can be found in literature (Gowe et al., 1993; Schmidt et al., 1994). It results from various gene pools and different breeding goals across populations as well as from different definitions of traits. Therefore, genetic variability of these characters depends on the population.

As mentioned above, the segregation of 1 major gene is suggested for hatchability in strain R33. This trait has a complex background influenced by many genetic and environmental factors. Hatchability is a sum of embryo survivals in a certain period of time—the present study from the eighth to the 21st day of incubation. In general, the ratio of embryonic mortality for chicken is relatively high (Liptoi and Hidas, 2006). It can be strongly affected by recessive genotypes and chromosomal abnormalities. A number of genes determining mortality of embryos were presented by Liptoi and Hidas (2006). It should be noted that the majority of them were identified in first decades of the 20th century, for instance the so-called

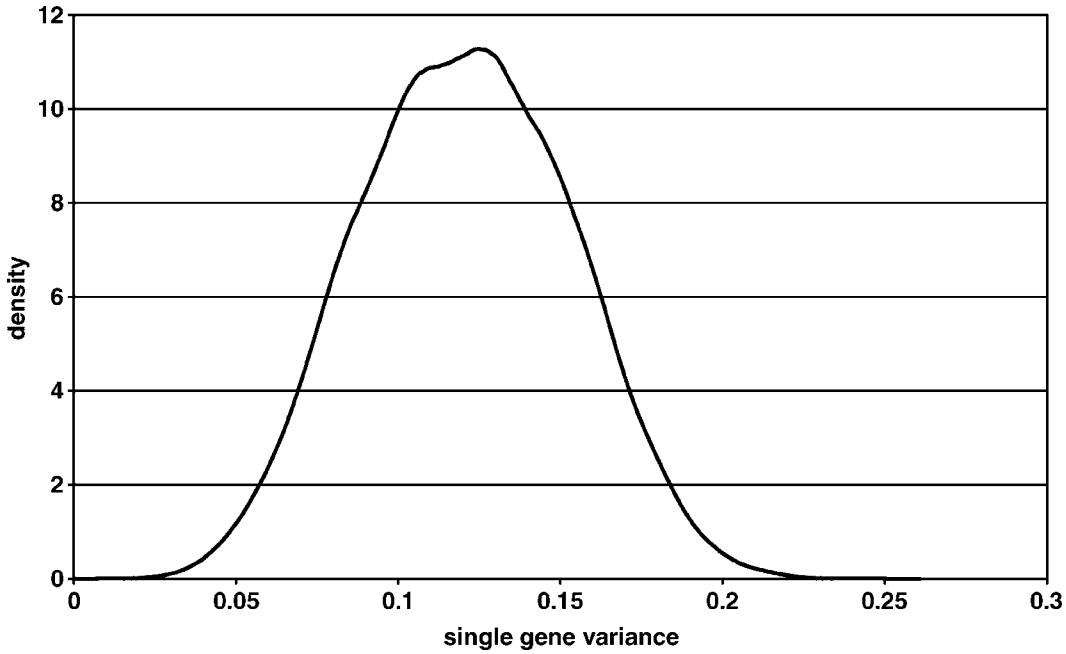

Figure 1. Posterior density of single gene variance σ_{GA}^2 in model 1 for hatchability (strain R33).

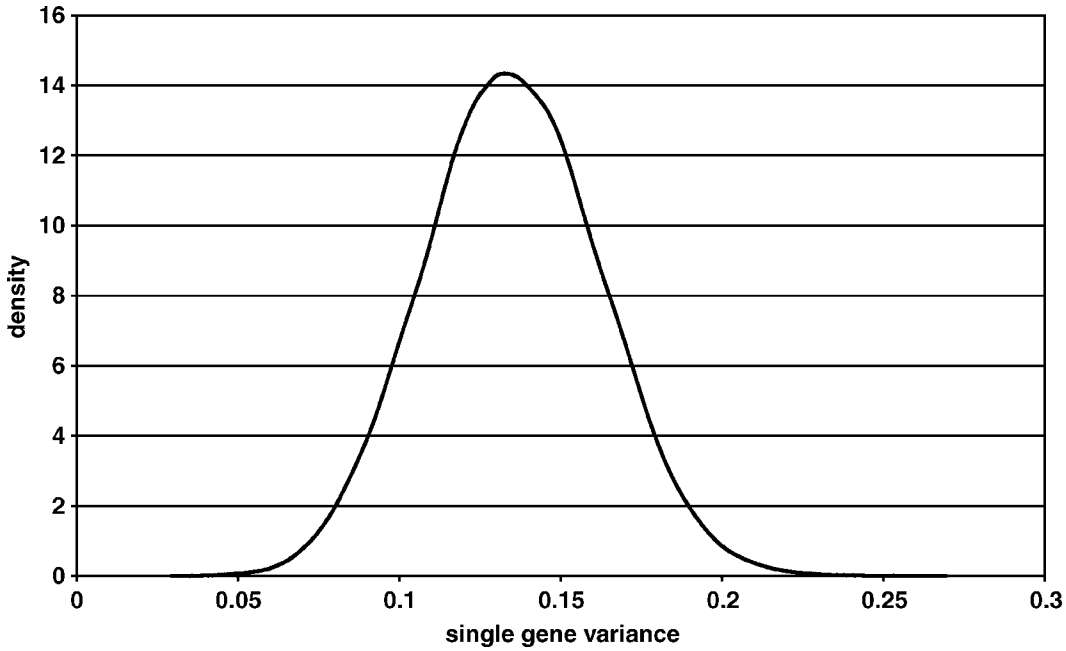

Figure 2. Posterior density of single gene variance σ^2_{GA} in model 2 for hatchability (strain R33).

creeper gene. Hence, they were usually rejected from populations. However, in the last decades, new mutations have been also identified. Delany et al. (1994) found the variation of rRNA gene copy number, which affected early embryonic mortality in chicken. A number of lethal and sublethal genes were described for other animal species. So, the arguments presented above seem to confirm the existence of single loci for hatchability.

It should be recalled that fertility was examined on the eighth day of incubation. Therefore, the percentage of fertilized eggs is underestimated by early embryo mortality. Although the 95% HPDR for single gene variance of fertility of strain R33 included zero, this criterion for genotypic mean (for 1 locus) suggested a possibility of segregation of a single gene. Szwaczkowski et al. (2006), based on the Bayesian linear model, found a single locus responsible for the percentage of fertilized eggs in the population studied (R33). In the present paper, estimated allele frequencies were 0.308 for fertility and 0.547 for hatchability, with relatively large standard deviations. However, Beaumont et al. (1997) estimated the positive genetic correlations between susceptibilities to the different stages of embryonic death. Is this the same locus with various effects over time? The hypothesis can be verified by detailed molecular segregation and linkage analysis.

The results correspond with estimates of additive polygenic variances for strain R33 (see Tables 4 and 5). When single locus effects were omitted, the estimates of polygenic variance increased strongly, especially for hatchability. In the case of this trait, a similar tendency was also registered for permanent environmental variance estimates. Of course, it considerably influenced heritability estimates. Relatively large participation of major gene variance in total variance compared with polygenic heritability should be stressed. This confirms a segregation of 1 biallelic single locus for hatchability in this strain.

For the second population (N88), the estimates of variance components, and in consequence, the genetic parameters, were balanced for the 3 models used. Contrary to

Table 5. Posterior means (and standard deviations) of additive and permanent environmental variances; estimates of heritability and repeatability coefficients for hatchability[1]

	Strain R33						Strain N88					
Models	σ^2_a	σ^2_p	h^2_G	h^2_a	h^2_t	r	σ^2_a	σ^2_p	h^2_G	h^2_a	h^2_t	r
Model 1	0.018 (0.014)	0.09 (0.021)	0.098	0.015	0.113	0.186	0.048 (0.014)	0.084 (0.014)	0.003	0.042	0.045	0.119
Model 2	0.009 (0.008)	0.08 (0.02)	0.116	0.007	0.123	0.188	0.037 (0.014)	0.081 (0.012)	0.014	0.033	0.047	0.118
Submodel	0.104 (0.03)	0.263 (0.023)	—	0.076	—	0.268	0.048 (0.014)	0.086 (0.013)	—	0.042	—	0.118

[1] σ^2_a = additive polygenic variance; σ^2_p = permanent environmental variance; h^2_G = major gene heritability; h^2_a = additive polygenic heritability; h^2_t = total heritability; r = repeatability.

strain R33, it indicates no segregation of single genes determining fertility and hatchability.

Although heritability varies across populations, models, and methods, a number of authors (Foerster, 1993; Brah et al., 1999; Bennewitz et al., 2007) concluded that reproductive traits are lowly heritable. Moreover, the heritability for fertility is usually lower than for hatchability. Despite different measurements of these characters, the results correspond with reports by many authors (Sapp et al., 2005; Bennewitz et al., 2007).

Finally, further analysis based on the molecular study should be suggested to localize the locus responsible for hatchability.

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