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Genetic parameters of insect bite hypersensitivity in Dutch Friesian broodmares[1]

A. Schurink,*[2] B. J. Ducro,* H. C. M. Heuven,*† and J. A. M. van Arendonk*

*Animal Breeding and Genomics Centre, Wageningen University, PO Box 338, 6700 AH Wageningen, the Netherlands; and †Faculty of Veterinary Medicine, Utrecht University, PO Box 80163, 3508 TD Utrecht, the Netherlands

ABSTRACT: Insect bite hypersensitivity (IBH) is a seasonal allergic skin disease in horses caused by bites of certain *Culicoides* spp. The aim of our study was to investigate the maternal effect on IBH and to estimate the heritability and repeatability of IBH in the Dutch Friesian horse population. Data consisted of 3,453 Dutch Friesian broodmares with 3,763 visual observations on IBH clinical symptoms scored by 12 inspectors during organized foal inspections in 2004 and 2008. Nine percent of the mares (n = 310) were scored in both years. Mares descended from 144 sires and 2,554 dams and 26.2% of the dams (n = 669) had more than 1 offspring in the data set (range: 2 to 6). Insect bite hypersensitivity was analyzed as a binary trait with a threshold animal model with and without a maternal effect, using a Bayesian approach. Observed IBH prevalence in Dutch Friesian broodmare population was 18.2%. Heritability on the liability scale was 0.16 (SD = 0.06); heritability on the observed scale was 0.07; and repeatability was 0.89 (SD = 0.03). Maternal effect was 0.17 (SD = 0.06) and significantly differed from zero, although the animal model without a maternal effect fitted the data better. These results show that genetic and permanent environmental factors affect IBH in Dutch Friesian horses. The dam affected the IBH development of her offspring through an additive genetic influence but also by being part of their rearing environment.

Key words: *Culicoides* species, heritability, horse, insect bite hypersensitivity, maternal effect, summer eczema

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INTRODUCTION

Insect bite hypersensitivity (**IBH**) is a common allergic skin disease in horses caused by bites of certain *Culicoides* spp. Affected horses develop an intense pruritus, which results in self-inflicted trauma. Common clinical symptoms are therefore hair loss, excoriation, scaling, thickening of skin, and open wounds (Björnsdóttir et al., 2006; van den Boom et al., 2008). Welfare of affected horses is reduced, and some horses are unsuitable for riding and showing. Severely affected horses are occasionally euthanized (Anderson et al., 1988; Gortel, 1998).

The etiology of IBH is multifactorial in origin. The heritability of IBH on the observed binary scale was 0.08 in Icelandic horse populations and Dutch Shetland ponies (Table 1). Offspring from the same dam are subject to a common maternal environment during the first stages of their life and therefore often have similar phenotypes as a result of shared genes and maternal environment (Falconer and Mackay, 1996; Kruuk and Hadfield, 2007). Results from Unkel et al. (1987) suggest that a maternal effect might contribute to the development of IBH.

Susceptibility to allergies in humans is presumably highly influenced by events occurring early in life (Holt and Jones, 2000; Halken, 2003). For instance, early life and long-term exposure to farming environments has a strong protective effect against the development of allergy in humans (Holgate and Polosa, 2008). Rearing conditions during the first stages of the life of a horse are expected to affect the development of IBH later in life. The aim of our study was therefore to investigate the effect of rearing conditions during early life on IBH including the maternal effect (as part of rearing conditions) and to estimate the heritability and repeatability of IBH in the Dutch Friesian horse population.

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[2]Corresponding author: anouk3.schurink@wur.nl

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MATERIALS AND METHODS

Collection of IBH scores was a noninvasive method performed during routine foal inspections. Animal Care and Use Committee approval was therefore not required.

Population

Dutch Friesian broodmares were visually scored for IBH clinical symptoms by inspectors during organized foal inspections in 2004 and 2008. Data were a representative sample of the Dutch Friesian mare population because mares originated from 87 out of 90 regions (96.7%) across the Netherlands, where regions were defined based on 2-digit postal codes, and all mares with a foal were scored for IBH. Data were therefore an unselected sample of the population. Data from 3 mares with a missing value for age, month of scoring, or inspector were deleted. Data from 77 mares from mainly coastal regions without IBH (16 out of 87) were deleted because *Culicoides* spp. were probably absent within these regions (van Grevenhof et al., 2007), which enabled estimation of model effects. Number of observations per region varied from 3 to 700. Seventeen out of 71 regions (23.9%) contained less than 10 observations and 9 out of 71 regions (12.7%) contained over 100 observations. After editing, data consisted of 3,763 IBH scores on 3,453 Dutch Friesian broodmares (Table 2); 310 mares (9.0%) were scored in both years.

Average age was 9.0 yr (SD = 4.2 yr) and varied from 3 to 25 yr (Table 2). Data from 3- and 4-yr-old-mares were grouped together (≤4 yr) as well as data from 20 yr and older mares (≥20 yr) because of few observations. Most mares were scored in July, August, and September; some mares were scored in June and October (Table 2).

Mares descended from 144 sires and 2,554 dams. Paternal half-sib groups ranged from 1 to 149 individuals. In total 669 dams (26.2%) had more than 1 offspring in the data set (range: 2 to 6). Pedigree was for 97.9% complete and used up to the 4th ancestral generation. Inbreeding coefficients were calculated using full pedigree data, but did not significantly affect IBH.

Trait Definition of IBH

Friesian broodmares were visually scored by 8 inspectors in 2004 and 4 inspectors in 2008. Number of mares scored per inspector varied from 13 (0.5%) to 1,361 (49.5%) in 2004 and from 49 (4.8%) to 425 (42.0%) in 2008. Scoring of IBH clinical symptoms was made as uniform as possible with help from a veterinarian during a training session in 2004. Inspectors visually scored the clinical symptoms (e.g., hair loss, thickened skin, bald spots, crusting, scaling, and excoriation) on IBH-sensitive locations such as the crest and base of the tail. Insect bite hypersensitivity status of mares was scored in 3 categories: 0 = absence of clinical symptoms, 1

Table 1. Overview of genetic analyses of insect bite hypersensitivity (IBH) in different horse populations

Population	Parents	Method[1]	IBH observation	Prevalence, %	Trait[2]	h^2_{obs} [3]	h^2_{liab} [3]
Unkel et al. (1987)							
984 Icelandic horses born in Germany	36 sires	LSM using PHS data	Owner	18.1	0, 1	0.08 (0.07)	
	362 dams				Scale	0.12 (0.08)	
Eriksson et al. (2008)							
1,250 Icelandic horses born in Sweden	33 sires	TSM	Owner	8.0[4]	0, 1	0.08	0.27 (0.17)
	942 dams				0, 1, 2	0.09	0.30 (0.19)
					0, 1, 2, 3	0.10	0.33 (0.19)
Schurink et al. (2009)							
6,073 Dutch Shetland ponies	984 sires	LAM	16 inspectors trained by a veterinarian	8.8	0, 1	0.08 (0.02)	0.24 (0.06)
	4,455 dams				0, 1, 2	0.08	

[1]LSM = linear sire model; PHS = paternal half-sib; TSM = threshold sire model; and LAM = linear animal model.
[2]0, 1: unaffected to affected; scale: judged according to the number of affected parts of the body, ranging from mild to severe where a horse is mildly affected when only tail or mane or tail and mane are affected, and severely affected when the entire body contains large affected areas; 0, 1, 2 (Eriksson et al., 2008): unaffected, mild IBH, moderate to severe IBH, respectively; 0, 1, 2, 3: unaffected, mild IBH, moderate IBH, severe IBH, respectively; 0, 1, 2 (Schurink et al., 2009): unaffected, dubious or mild symptoms, clear symptoms, respectively.
[3]h^2_{obs} = heritability on the observed scale; h^2_{liab} = heritability on the liability scale; in parentheses SE (linear model) or SD (threshold model), depending on analysis method used.
[4]Mild IBH: 4.7%; moderate IBH: 2.6%; severe IBH: 0.7%.

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Table 2. Data description

Variable	Year of scoring		Total
	2004	2008	
Observations,[1] n (%)	2,751 (73.1)	1,012 (26.9)	3,763
Observed IBH[2] prevalence, %	18.5	17.2	18.2
Mean age, yr (range)	8.8 (3 to 25)	9.3 (4 to 24)	9.0 (3 to 25)
Month of scoring, n (%)			
June	167 (6.1)	40 (4.0)	207 (5.5)
July	806 (29.3)	244 (24.1)	1,050 (27.9)
August	957 (34.8)	404 (39.9)	1,361 (36.2)
September	661 (24.0)	267 (26.4)	928 (24.6)
October	160 (5.8)	57 (5.6)	217 (5.8)

[1]Percentage of total number of observations.
[2]IBH = insect bite hypersensitivity.

= dubious or mild clinical symptoms, and 2 = clear clinical symptoms. Categories 1 and 2 were grouped together because percentage of mares with score 1 was limited (5.1%); resulting IBH scores were analyzed as a binary trait.

Statistical Analysis

Model. Data were analyzed with a threshold animal model. A threshold model assumes the presence of an underlying nonobservable continuous variable, known as liability. When liability is equal to or below a certain threshold, an animal will be unaffected; when the liability is above the threshold, an animal will be affected. In formula, this is

$$y_i = \begin{cases} 0 & \text{if } \lambda_i \leq \tau \\ 1 & \text{if } \lambda_i > \tau, \end{cases}$$

where $y_i = 0$ denotes an unaffected mare and $y_i = 1$ denotes an affected mare, depending on whether their liability (λ_i) exceeded the fixed threshold (τ) for the manifestation of IBH.

Fixed Effects. Because data were binary, a binomial distribution with an inverse probit link function was used. The linear predictor (η_{ijkl}) was

$$\eta_{ijkl} = \mu + \text{age}_i + \text{region}_j + \text{yearmonth}_k + \text{inspector}_l,$$

where μ = population mean on liability scale; age_i = fixed effect of the age of a Friesian broodmare in years (i = ≤4, 5, 6, …, ≥20); region_j = fixed effect of region (j = 1, 2, 3, …, 71); yearmonth_k = fixed combined effect of year and month of scoring (k = 1, 2, 3, …, 10); inspector_l = fixed effect of inspector (l = 1, 2, 3, …, 12). Fixed model effects were estimated using GLIMMIX procedure that fitted generalized linear mixed models (SAS Inst. Inc., Cary, NC).

Estimation of Variance Components

For the threshold models the underlying liabilities were sampled using Markov chain Monte Carlo methodology through a Gibbs sampling algorithm. The threshold and residual variance were set to, respectively, $\tau = 0$ and $\sigma_e^2 = 1$. Uninformative (flat) priors were assumed for all random model effects, being inverse χ^2 distribution. Shrinkage of fixed model effects was used to prevent poor convergence (Janss, 2008). Marginal posterior distributions of heritability, repeatability, and maternal effect were assessed using iBay software (Janss, 2008). An inverse probit link function, $\mu_i = \Phi(\eta_i)$, was used to relate the linear predictor (η_i), the IBH liability, to the conditional mean on the observed scale (α_i), where Φ is the cumulative distribution function of the standard normal distribution and α_i is the expected response on the observed scale (Kachman, 2000). The linear predictor (η_{ijklmn}) of the animal model was

$$\eta_{ijklmn} = \mu + \text{age}_i + \text{region}_j + \text{yearmonth}_k + \text{inspector}_l + \text{pe}_m + \text{animal}_m + \text{maternal}_n,$$

where fixed effects are as described previously; pe_m = the random permanent environment of the mth animal (m = 3,453) with $\sim N\left(0, \mathbf{I_{pe}}\sigma_{pe}^2\right)$; animal_m = the random additive genetic effect of the mth animal (m = 3,453) with $\sim N\left(0, \mathbf{A}\sigma_a^2\right)$; and maternal_n = the random maternal effect of the nth dam (n = 2,554) with $\sim N\left(0, \mathbf{I_m}\sigma_m^2\right)$, where $\mathbf{I_{pe}}$ and $\mathbf{I_m}$ are identity matrices of the appropriate dimensions; $\mathbf{A}$ is a matrix of additive genetic relationships among Friesian horses in our data; σ_{pe}^2 = permanent environmental variance; σ_a^2 = additive genetic variance; and σ_m^2 = maternal variance. Re-

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sidual variance of liabilities (σ^2_e) was fixed to 1, related to the inverse probit link function. Phenotypic variance for the animal model with maternal effect was $\sigma^2_p = \sigma^2_{pe} + \sigma^2_m + \sigma^2_a + \sigma^2_e$. Random effects were considered to be independent (no covariance). An animal model without the random maternal effect was run as well. Phenotypic variance for the animal model without maternal effect was $\sigma^2_p = \sigma^2_{pe} + \sigma^2_a + \sigma^2_e$.

In total 10 independent chains of 210,000 cycles per chain were run, with a burn-in period of 10,000 cycles. Every 500th sample was stored giving a total of 4,000 samples. Saving every 100th sample hardly affected the results. Analysis of within and between chain variance for variance components estimates indicated an effective number of samples of at least 347. Natural logarithm of Bayes factor was used to compare models (Janss, 2008). Bayes factor is penalized for the number of parameters in the model. The model with greater Bayes factor is therefore better (Kass and Raftery, 1995; Janss, 2008).

Calculations of Parameters Derived from Variance Components

The correlation between repeated observations within a Friesian broodmare, known as repeatability (r), was calculated as

$$r = \frac{\sigma^2_a + \sigma^2_{pe}}{\sigma^2_p},$$

where all variance components are as described previously. The numerator included σ^2_m for the animal model with maternal effect.

Heritability on the liability scale (h^2_{liab}) was calculated as

$$h^2_{liab} = \frac{\sigma^2_a}{\sigma^2_p},$$

where all variance components are as explained previously. To enable comparison with results from previous studies on the genetic background of IBH, heritability on the liability scale was transformed to heritability on the observed scale (h^2_{obs}) according to Dempster and Lerner (1950):

$$h^2_{obs} = \frac{z^2 h^2_{liab}}{p(1-p)},$$

where z = the ordinate of a standard normal distribution at the threshold corresponding to the fraction of affected mares, which is $p = 0.182$.

Maternal effect was calculated as

$$\text{maternal effect} = \frac{\sigma^2_m}{\sigma^2_p},$$

where all variance components are as explained previously.

Calculations of parameters derived from estimated variance components was needed to obtain features of marginal posterior distributions (mean, SD, 95% highest posterior density region) of repeatability, heritability, and maternal effect by computing from each Gibbs sample the corresponding value of repeatability, heritability, or maternal effect and subsequently using these values when the posterior distributions were summarized.

RESULTS AND DISCUSSION

Observed IBH Prevalence

Observed IBH prevalence was 18.5% in 2004 and 17.2% in 2008, on average 18.2% (Table 2). Prevalence in Friesian broodmares descending from affected dams or maternal granddams was greater (respectively 21.3 and 17.6%) than prevalence of mares descending from unaffected dams or maternal granddams (respectively 17.9 and 12.2%). An increased prevalence in horses descending from affected dams was also found in Icelandic horses (Eriksson et al., 2008) and Shetland broodmares (Schurink et al., 2009). Prevalence in paternal half-sib groups with more than 20 members varied from 0 to 34.9%. Prevalence in paternal half-sib groups in an Icelandic horse population varied from 0 to 30% (Eriksson et al., 2008) and in a Shetland broodmare population from 0 to 37% (Schurink et al., 2009).

Associated Effects

Combined effect of year and month of scoring ($P = 0.003$), inspector ($P < 0.0001$), and region within the Netherlands ($P = 0.043$) had a significant effect on IBH prevalence. Age of the mare had a suggestive effect ($P = 0.078$).

Year and Month of Scoring. Predicted prevalence increased with month of scoring in 2004 from 14.0% in June to 23.7% in October. Predicted prevalence of month of scoring in 2008 fluctuated: 7.8% in June, 7.3% in July, 16.4% in August, 18.1% in September, and 3.2% in October. Distinct annual trends in IBH prevalence might be related to differences in presence and activity of *Culicoides* spp. between years and months, which could be caused by differences in environmental conditions (Anderson et al., 1993; Takken et al., 2008).

Inspector. Predicted prevalence of inspector varied from 11.6 to 35.0%. Within a region, 1 to 10 inspectors scored the Friesian broodmares. In 3 out of 71 regions

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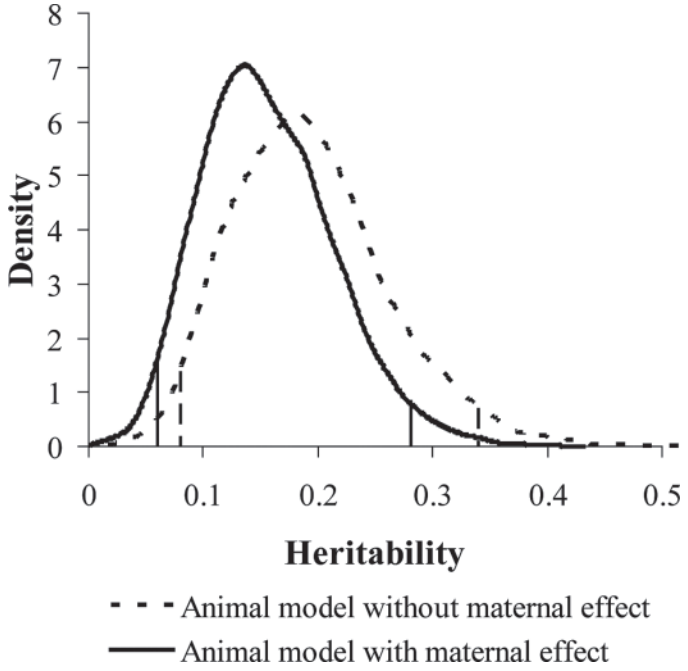

Figure 1. Estimated marginal posterior distribution of heritability on the liability scale (95% highest posterior density regions between vertical lines) estimated with an animal model with (solid line) and without a maternal effect (dashed line).

only 1 inspector per region scored the mares (n = 15). In 16 regions, 7 to 10 inspectors per region scored 2,786 mares (74.0% of all observations). Inspector and region within the Netherlands were therefore not confounded. Thus, differences in IBH prevalence between inspectors were not explained by regional differences in IBH prevalence. Scoring of IBH clinical symptoms was uniformed with help from a veterinarian during a training session in 2004, but only a portion of the inspectors was able to join. Because IBH scoring was a subjective method, inspector was included in the analysis.

Region. Predicted prevalence in regions with more than 20 observations varied from 1.2 to 28.6%. Most low-risk regions were found in coastal areas of the Netherlands, whereas most high-risk regions were found in the provinces Gelderland, Noord-Brabant, Overijssel, and Limburg. This finding agrees with results from van Grevenhof et al. (2007) and van den Boom et al. (2008) for various horse breeds (e.g., Friesian horses, Icelandic horses, Haflinger horses, Warmblood horses, and Shetland ponies). Regional differences in IBH prevalence were observed by other researchers (e.g., Broström et al., 1987; van Grevenhof et al., 2007; Eriksson et al., 2008; van den Boom et al., 2008). van Grevenhof et al. (2007) found that habitat and climate factors, which included information about vegetation, soil type, rainfall, number of warm and cold days within regions of the Netherlands, significantly affected IBH prevalence in a region. Environmental conditions within regions affect the presence and activity of *Culicoides* spp. and thereby the observed IBH prevalence.

Age. Predicted prevalence in Friesian broodmares up to 4 yr was 27.9%. Predicted prevalence increased

Table 3. Direct additive genetic variance (σ_a^2), permanent environmental variance (σ_{pe}^2), maternal variance (σ_m^2), phenotypic variance (σ_p^2); posterior mean, posterior SD, and 95% highest posterior density region (HPDR) of heritability on the liability scale (h_{liab}^2), repeatability (r) and maternal effect of insect bite hypersensitivity; heritability on the observed scale (h_{obs}^2); estimates are shown for an animal model without and with a maternal effect

	Variance component[1]				h_{liab}^2			r[3]		Maternal effect	
Model	σ_a^2	σ_{pe}^2	σ_m^2	σ_p^2	Mean (SD)	95% HPDR	h_{obs}^2 [2]	Mean (SD)	95% HPDR	Mean (SD)	95% HPDR
Animal model without maternal effect	1.78	6.43	—	9.21	0.19 (0.07)	0.08 to 0.34	0.09	0.88 (0.03)	0.82 to 0.93	—	—
Animal model with maternal effect	1.51	5.43	1.71	9.65	0.16 (0.06)	0.06 to 0.28	0.07	0.89 (0.03)	0.83 to 0.93	0.17 (0.06)	0.05 to 0.30

[1]Animal model without maternal effect: $\sigma_p^2 = \sigma_{pe}^2 + \sigma_a^2 + \sigma_e^2$; animal model with maternal effect: $\sigma_p^2 = \sigma_{pe}^2 + \sigma_m^2 + \sigma_a^2 + \sigma_e^2$, where $\sigma_e^2 = 1$, related to the inverse probit link function.

[2]Heritability on the observed scale after transformation according to Dempster and Lerner (1950): $h_{obs}^2 = \frac{z^2 h_{liab}^2}{p(1-p)}$, where z = the ordinate of a standard normal distribution at the threshold corresponding to the fraction of affected mares, which is $p = 0.182$.

[3]Repeatability calculated using animal model without maternal effect: $r = \frac{\sigma_a^2 + \sigma_{pe}^2}{\sigma_p^2}$, and using animal model with maternal effect: $r = \frac{\sigma_a^2 + \sigma_{pe}^2 + \sigma_m^2}{\sigma_p^2}$.

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Table 4. Comparison of the absence or presence of observed insect bite hypersensitivity (IBH) clinical symptoms in 2004 and 2008 for Friesian broodmares with repeated observations in number of mares and percentage (in parentheses)

IBH clinical symptoms 2004	IBH clinical symptoms 2008		
	Absent	Present	Total
Absent	228 (85.1)	40 (14.9)	268 (100)
Present	26 (61.9)	16 (38.1)	42 (100)

to 38.8% in 8-yr-old mares and subsequently decreased to 18.1% in 20 yr and older mares. Although often an increase in prevalence with age was found (e.g., Halldórdsóttir and Larsen, 1991; Steinman et al., 2003), our results might reflect an effect of selection, where affected Friesian broodmares are culled from breeding.

Repeatability, Heritability, and Maternal Effect

Estimates of variance components, repeatability, heritability, and maternal effect for the animal model with and without maternal effect are shown in Table 3. Estimated marginal posterior distributions of heritability on the liability scale and maternal effect are shown in Figures 1 and 2. Estimates of repeatability, heritability, and maternal effect will be described and discussed separately in the subsequent sections.

Repeatability. Investigation of uncorrected repeated observations on 310 Friesian broodmares (Table 4) showed that 78.7% of the mares (n = 244) had a similar IBH observation in 2004 and 2008. However, 14.9% (n = 40) of mares without clinical symptoms in 2004 had developed clinical symptoms in 2008. Also, 61.9% (n = 26) of mares with clinical symptoms in 2004 did not have clinical symptoms in 2008. Schurink et al. (2009) analyzed uncorrected phenotypes of Dutch Shetland broodmares and found that 5.2% (n = 76) of mares without symptoms in the first year had developed symptoms in the second year and 72.8% (n = 75) of mares with symptoms in the first year did not have symptoms in the second year. When exposed to *Culicoides* spp., clinical symptoms rarely disappear with age (Anderson et al., 1988). Owners possibly started applying preventive measures after a mare showed IBH, which resulted in the reduction of clinical symptoms. Information on application of preventive measures by owners was not available to confirm the impact. Year and month of scoring, region, inspector, and age of the mares were known and contributed to changes in IBH status of Friesian broodmares between years. The model for estimation of repeatability (and all other parameters) corrected for these effects.

Mean repeatability estimate in Friesian broodmares was 0.89 in the animal model with a maternal effect and 0.88 in the animal model without a maternal effect (Table 3). Insect bite hypersensitivity is considered to be a permanent condition (Anderson et al., 1991). A high repeatability was therefore expected. Repeatability in Dutch Shetland broodmares was 0.30 (SE = 0.02; Schurink et al., 2009). However, repeatability estimate of Schurink et al. (2009) is likely underestimated due to limited possibilities to correct for fixed effects and confounding of genetic effects, region and inspector.

Heritability. Mean heritability estimate on the liability scale was 0.16 in the animal model with a maternal effect and 0.19 in the animal model without a maternal effect (Table 3). Mean heritability estimate on the liability scale estimated with a sire and sire-dam model were respectively 0.17 (SD = 0.08) and 0.21 (SD = 0.12; data not shown). Heritability significantly differed from 0, because 95% highest posterior density regions excluded 0. Mean heritability on the observed scale was 0.07 in the animal model with a maternal effect and 0.09 in the animal model without a maternal effect (Table 3). Heritability on the liability scale is less compared with estimates from Eriksson et al. (2008) and Schurink et al. (2009; Table 1). Estimates of heritability on the observed scale depend on prevalence within a population, which was 18.2% in Friesian broodmares, 18.1% in German-born Icelandic horses (Unkel et al., 1987), 8.0% in Swedish-born Icelandic horses (Eriksson et al., 2008), and 8.8% in Shetland mares (Schurink et al., 2009; Table 1). Heritability on the observed scale in Friesian broodmares equals estimates from Unkel et al. (1987), Eriksson et al. (2008), and Schurink et al. (2009), despite differences in recording of phenotype, prevalence, analysis method used, and investigated breed and population (Table 1).

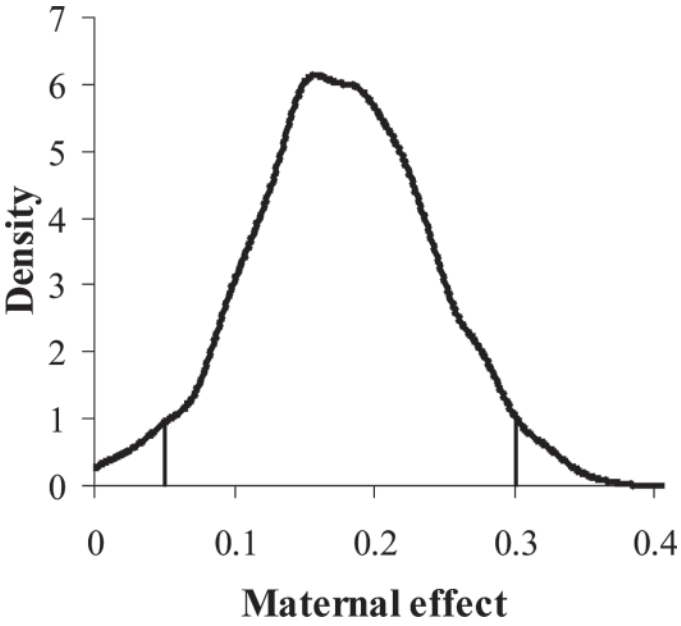

Figure 2. Estimated marginal posterior distribution of maternal effect (95% highest posterior density region between vertical lines).

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Maternal Effect. Mean maternal effect significantly differed from 0 and composed 17% of the variance (Table 3), which is comparable in size with the variation explained by genetic factors (i.e., heritability). Mean maternal effect estimated with a sire and sire-dam model composed, respectively, 17 and 21% of the variance (data not shown). Besides a direct additive genetic influence, the dam also affected the IBH development of her offspring by being part of their rearing environment. Unkel et al. (1987) suggested a similar effect of a common maternal effect on IBH (0.20) in Icelandic horses because ANOVA using full-sib data indicated that IBH heritability based on dam variance was greater ($h^2_d = 1.04$) compared with heritability based on sire variance ($h^2_s = 0.23$). The common maternal effect estimated by Unkel et al. (1987) included both maternal genetic and maternal environmental effects.

Possible causes of a maternal effect might be related to maternal features such as transfer of various immunological substances via colostrum, to environmental aspects experienced during early life such as habitat related to allergen exposure, to management by breeders such as feeding or stabling, and to genetic ability. Susceptibility to allergies is presumably highly influenced by events occurring early in life (Holt and Jones, 2000; Halken, 2003). Maternal equine IgE antibodies against *Culicoides* allergens are passively transferred from IBH-affected mares to their foals via colostrum. However, consequences of transferred maternal equine IgE are unknown (Wagner et al., 2006; Marti et al., 2009).

Environmental conditions experienced by maternal (half)-sibs during early life, such as allergen exposure related to habitat, or management by breeders, such as feeding or stabling, might result in maternal (half)-sibs with similar phenotypes caused by shared genes, but also due to shared environment. Region of the dam was known for 30.0% (n = 1,130) of the Friesian broodmares. In total, 57.7% (652 out of 1,130) of the broodmares were kept within the same region as their dam. Maternal effect might therefore include a regional effect because some progeny remain within the same region of their dam, although fixed effect of region within the Netherlands remained significant. Maternal effect might also represent a farm effect, but estimates of maternal effect differed between dams within a farm.

Trait Definition of IBH

Friesian broodmares with dubious or mild clinical symptoms (category 1) and clear clinical symptoms (category 2) were considered to be affected and IBH scores were analyzed as a binary trait. When only Friesian broodmares with clear clinical symptoms (category 2) were considered to be affected, heritability, repeatability, and maternal effect were in the range of estimates presented in Table 3 (data not shown). Heritability in Dutch Shetland broodmares was equal when IBH was analyzed as a binary trait or on a 0, 1, 2 scale (Schurink et al., 2009). Eriksson et al. (2008) classified severity of IBH symptoms in Icelandic horses into several categories (mild-severe) and found that heritability was greater ($h^2_{liab} = 0.33$) compared with heritability of IBH analyzed as a binary trait ($h^2_{liab} = 0.27$). However, SD were large (Table 1), and all 95% highest posterior density regions overlapped (Eriksson et al., 2008). The benefit of scoring severity of IBH symptoms for genetic research is limited because only phenotypes of affected horses, the minority of a population, are subdivided. The majority of horses in a population are unaffected (often $> 80\%$; i.e., without clinical symptoms) and therefore have a similar phenotype, although sensitivity to IBH in unaffected horses can differ greatly. Research on IBH will therefore benefit from a reliable diagnostic or DNA test that can distinguish horses according to their sensitivity to IBH, independent of observed clinical symptoms.

Model Comparison

The animal model with and without maternal effect obtained similar heritability and repeatability estimates (Table 3); 95% highest posterior density region of heritability and repeatability overlapped. Clément et al. (2001) simulated data to investigate the effect of model adequacy on the estimation of genetic parameters for traits affected by maternal effects and found that heritability was overestimated when a maternal (genetic or genetic and environmental) effect existed but was neglected. Similar results were obtained by Kruuk and Hadfield (2007) in a simulated ungulate population. True direct additive genetic variance was 0.3, and true maternal environmental variance was 0.2. When maternal environmental effect was neglected, direct additive genetic variance was overestimated (0.52). Our results (Table 3) show that IBH heritability estimated with an animal model without maternal effect was only slightly greater (0.19) compared with heritability estimated with an animal model with maternal effect (0.16).

Heritability, repeatability, and maternal effect significantly differed from 0; all 95% highest posterior density regions excluded 0 (Table 3; Figures 1 and 2). However, the natural logarithm of Bayes factor was greater for the animal model without a maternal effect. The animal model without a maternal effect therefore fitted the data better, though data including more dams with at least 2 offspring will be required to demonstrate a significant improvement of the model when including a maternal effect and to separate maternal genetic and maternal environmental effects.

Conclusions and Implications

Insect bite hypersensitivity is a heritable trait in the Dutch Friesian horse population. Repeatability of IBH scores is high (0.89). Maternal effect on IBH (0.17) is

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comparable in size with the heritability estimate (0.16). Besides an additive genetic influence, the dam also affected the IBH development of her offspring by being part of their rearing environment.

Although selection against IBH is possible, a traditional breeding program based only on scoring clinical symptoms is inefficient. Progeny testing is needed to reliably estimate the genetic potential of stallions. However, progeny testing will seriously increase generation interval because offspring have to be old enough (at least 3 yr of age) and exposed to *Culicoides* spp. to be able to show IBH clinical symptoms. Genomic information can determine the genetic potential of an individual more reliably, at a younger age, and independent of the presence of *Culicoides* spp. Including genomic information in a breeding program against IBH will improve response to selection and thereby decrease the number of affected horses more rapidly compared with a traditional breeding program based on progeny testing.

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Erratum to "Genetic parameters of insect bite hypersensitivity in Dutch Friesian broodmares" (J. Anim. Sci. 89:1286–1293)

A. Schurink, B. J. Ducro, H. C. M. Heuven, and J. A. M. van Arendonk

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The μ symbol appeared incorrectly as the α symbol twice on page 1288 in the last sentence of the section shown below. The symbols are shown correctly below. The journal regrets the error.

Estimation of Variance Components

For the threshold models the underlying liabilities were sampled using Markov chain Monte Carlo methodology through a Gibbs sampling algorithm. The threshold and residual variance were set to, respectively, $\tau = 0$ and $\sigma_e^2 = 1$. Uninformative (flat) priors were assumed for all random model effects, being inverse χ^2 distribution. Shrinkage of fixed model effects was used to prevent poor convergence (Janss, 2008). Marginal posterior distributions of heritability, repeatability, and maternal effect were assessed using iBay software (Janss, 2008). An inverse probit link function, $\mu_i = \Phi\left(\eta_i\right)$, was used to relate the linear predictor (η_i), the IBH liability, to the conditional mean on the observed scale (μ_i), where Φ is the cumulative distribution function of the standard normal distribution and μ_i is the expected response on the observed scale (Kachman, 2000).

Schurink, A., B. J. Ducro, H. C. M. Heuven, and J. A. M. van Arendonk. 2011. Genetic parameters of insect bite hypersensitivity in Dutch Friesian broodmares. J. Anim. Sci. 89(5):1286–1293.

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