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# Genetic and non-genetic indirect effects for bite mark traits in group housed mink

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**ABSTRACT:** Social interactions among individuals are abundant, both in wild and domestic populations. With social interactions, genes of an individual may affect the trait values of other individuals, a phenomenon known as Indirect Genetic Effects (IGEs). IGEs can be estimated using linear mixed models. Here we show that IGE contribute substantial heritable variation in bite mark scores in group housed mink. Furthermore we investigated whether IGEs depend on relatedness between interacting individuals or on their sex. Current IGE-models assume that individuals interact equally to all group members irrespective of relatedness. Kin selection theory predicts that an individual will interact differently with family members vs. non-family members. We showed that mink interact differently either due to sex or the family relationship with their group mates. Our results show that IGEs are very important for welfare in mink, and there are good prospects for genetic improvement.
**Keywords:** mink; Bite mark; IGEs

## Introduction

Social interactions among individuals are common in both plants and animals (Frank (1998)). In agriculture, these interactions can have significant effects on production and welfare traits. For example, social interactions may result in mortality due to cannibalism in laying hens (Muir (1996)) and in bite marks in mink (Moller et al. (2003)). Because of social interaction an individual's genes may affect the trait values of its interacting partner, a phenomenon known as Indirect Genetic Effects IGE, also referred to as associative effects (Moore et al. (1997); Muir (2005)).

IGEs may change heritable variation and can change the rate and direction of response to selection (Griffing (1967)). IGEs can be estimated using linear mixed models. However, current models assume that individuals interact equally to all group members whether the group member is family or not. Kin selection theory predicts that an individual will interact differently with family members vs. non-family members. However, there is no empirical study that has showed whether individual interact differently with family members and non-family members.

This paper gives estimated genetic parameters for bite mark traits in group housed mink, showing that IGE contribute the majority of heritable variation. The study includes evaluation of bite marks on neck, body, and tail region of the body as well as the average of these regions on mink in group housing. Furthermore, we provide empirical evidence that mink to interact differently either because of their sex, or because of family relatedness.

## Material and method

We took bite mark records during pelting; hence, it gives an indication of the aggression received by the individual over the period of time prior to pelting. A total of 1969 mink descending from 136 sires and 349 dams was used in our analysis. Two male siblings and two female siblings were placed in 2 storey cage in years 2009, 2010, and 2011. The number of bite marks was subjectively measured on the scale 1 to 9.

**Statistical methods.** We compared four models using the Akaike information criterion (AIC):

**Model 1.** Usual animal model

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Za} + \mathbf{Wg} + \mathbf{e}$$

where $\mathbf{y}$ a vector of is observed bite-mark scores; $\mathbf{b}$ is a vector of fixed effects, with incidence matrix $\mathbf{X}$ linking observations to fixed effects, $\mathbf{a}$ is a vector of the breeding values which were assumed to follow a normal distribution, $\mathbf{a} \sim N\left(0, \mathbf{A}\sigma_a^2\right)$, with incidence matrix $\mathbf{Z}$ linking observations on individuals to their breeding values, and $\mathbf{A}$ is the numerator relationship matrix, $\mathbf{g}$ is a vector of the random cage effects with, $\mathbf{g} \sim N\left(0, \mathbf{I}_g\sigma_g^2\right)$, $\mathbf{I}_g$ is an identity matrix of appropriate dimension, with incidence matrix $\mathbf{W}$ for groups and, $\sigma_g^2$ is group variance, $\mathbf{e}$ is a vector of residuals.

**Model 2**. Usual animal model extended with an environmental covariance between family members present in the same group (non-genetic effect of family or sex recognition):

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Za} + \mathbf{Wg} + \mathbf{Vk} + \mathbf{e}$$

where, $\mathbf{V}$ is an incidence matrix for family x groups and $\mathbf{k}$ a vector of group*family effects. Hence,

$$\mathbf{k} \sim N(\mathbf{0}, \mathbf{K} \otimes \mathbf{I}_g), \text{where } \mathbf{K} = \begin{bmatrix} \sigma^2_{f1} & 0 \\ 0 & \sigma^2_{f2} \end{bmatrix}$$

$\sigma^2_{f1}$ , is the variance common to male siblings in the same group and $\sigma^2_{f2}$ is the variance common to female siblings in the same group. Other elements were the same as in model 1.

**Model 3.** The usual direct-indirect animal model (Muir (2005)), extended with a random group effect (Bergsma et al. (2008)):

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z}_D\mathbf{a}_D + \mathbf{Z}_S\mathbf{a}_S + \mathbf{Wg} + \mathbf{e},$$

$$\begin{bmatrix} \mathbf{a}_D \\ \mathbf{a}_S \end{bmatrix} \sim MVN(\mathbf{0}, \mathbf{G} \otimes \mathbf{A}),$$

in which

$$\mathbf{G} = \begin{bmatrix} \sigma^2_{A_D} & \sigma_{A_{DS}} \\ \sigma_{A_{DS}} & \sigma^2_{A_S} \end{bmatrix}$$ $\otimes$ indicates the Kronecker product.

Other elements were the same as in model 1. The $\mathbf{Z}_S\mathbf{a}_S$ accounts for heritable indirect effects and $\mathbf{Wg}$ accounts for non-heritable indirect effects. This model assumes IGE are the same for family members and non-family members.

**Model 4**. Following Alemu et al. (2014)) this model is similar to model 3 but here IGE differ for family vs. non- family members and a non-genetic covariance between family members in the same group is included.

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z}_D\mathbf{a}_F + \mathbf{Z}^u_S\mathbf{a}_S + \mathbf{Wg} + \mathbf{Vk} + \mathbf{e},$$

where $\mathbf{Z}_D$ is a known incidence matrix for direct genetic effects, and $\mathbf{Z}^u_S$ is a known incidence matrix for indirect genetic effects on group members belonging to the other family (hence, subscript *u* indicates "unrelated"), and $\mathbf{a}_F$ is a vector of random family breeding values (this includes the direct genetic effect and the IGE to family members; see Alemu et al. 2014), and $\mathbf{a}_S$ is a vector of indirect genetic effects on the members of the other family, with

$$\begin{bmatrix} \mathbf{a}_F \\ \mathbf{a}_S \end{bmatrix} \sim MVN(\mathbf{0}, \mathbf{G}_r \otimes \mathbf{A}),$$

in which

$$\mathbf{G}_r = \begin{bmatrix} \sigma^2_{A_F} & \sigma_{A_{FS}} \\ \sigma_{A_{FS}} & \sigma^2_{A_S} \end{bmatrix}.$$

All the other elements are the same as in model 2.

## Result and Discussion

As shown in Table 1, the AIC of model 4 is lower than that of the other models. Thus, model 4 fitted the data better than other models. This is true for neck, body, tail, and total bite mark score. Therefore there is systematic interaction between the two female siblings and between the two male siblings in a group. Table 2 shows the estimated genetic parameters from model 4. The IGE on unrelated individuals of the other sex contribute a substantial amount of heritable variation, about 13% to 20% of the total heritable variation for bite marks in the different regions of the body. Moreover, there was a strong positive correlation between family breeding value and IGE to stranger (0.79 to 0.93), which further increased total heritable variance. Thus, most of the heritable variation in bites mark score relates to IGEs.

**Table 1. Model comparison for bite mark (bm) using AIC[1](Akaike information criterion)**

| Model | Neck bm | Body bm | Tail bm | Total bm |
|---|---|---|---|---|
| 1 | 78.41 | 71.04 | 54.00 | 65.90 |
| 2 | 41.5 | 65.52 | 29.50 | 39.20 |
| 3 | 32.00 | 0.62 | 7.54 | 10.64 |
| 4 | 0.00 | 0.00 | 0.00 | 0.00 |

[1]AIC value of best model according to AIC was set to zero as reference. AIC = 2× # parameters – 2 × log-likelihood; thus smaller values indicate a better model.

## Conclusion

We have shown that IGEs make a large contribution to the heritable variation in bite mark scores in mink. The total genetic variance for response to selection was 45% of phenotypic variance ($T^2$ for total bm; Table 2), indicating good prospects for genetic improvement. Moreover, our results show that the interactions differ systematically between the two male siblings and the two female siblings within a cage. Therefore, estimation of genetic parameters for group housed mink needs to take in to account such systematic differences to avoid biased estimates of the genetic parameters and suboptimal response to selection.

**Table 2. Estimated parameters for bite mark (bm) from the best model[1] according to AIC**

| Parameter | Neck bm | Body bm | Tail bm | Total bm |
|---|---|---|---|---|
| $\hat{\sigma}^2_{AF}$ | 0.49±0.13 | 0.87±0.20 | 0.69±0.17 | 5.41±1.16 |
| $\hat{\sigma}_{AF,S}$ | 0.22±0.06 | 0.40±0.09 | 0.25±0.06 | 2.19±0.0.48 |
| $\hat{\sigma}^2_{AS}$ | 0.15±0.04 | 0.30±0.07 | 0.11±0.04 | 1.10±0.31 |
| $\hat{r}_{AF,Su}$ | 0.84±0.13 | 0.79±0.10 | 0.87±0.13 | 0.92±0.1121 |
| $\hat{\sigma}^2_{AT}$ | 1.70±0.38 | 3.090±0.61 | 1.89±4.2 | 16.21±2.90 |
| $^2\hat{\rho}$ | 0.05±0.04 | 0.02±0.06 | 0.06±0.04 | 0.09±0.05 |
| $\hat{\rho}_m$ | -0.0001±0.04 | -0.10±0.08 | -0.12±0.05 | -0.09±0.05 |
| $\hat{\rho}_f$ | 0.40±0.05 | 0.06±0.08 | 0.01±0.08 | 0.2±0.06 |
| $\hat{\sigma}^2_{em}$ | 1.3±0.12 | 3.0±0.24 | 2.55±0.2 | 12.84±1.1 |
| $\hat{\sigma}^2_{ef}$ | 2.20±0.19 | 3.40±0.28 | 5.38±0.4 | 23.030±1.83 |
| $\hat{\sigma}^2_P$ | 3.5±0.11 | 4.95±0.16 | 5.30±0.17 | 30.20±1.00 |
| $\hat{h}^2_F$ | 0.14±0.05 | 0.17±0.03 | 0.13±0.03 | 0.18±0.03 |
| $\hat{T}$ | 0.49±0.14 | 0.62±0.14 | 0.36±0.8 | 0.54±0.11 |

$\hat{\sigma}^2_{AF}$ is the family breeding value variance, $\hat{\sigma}^2_{AS}$ the indirect genetic effect to non-family members variance, $\hat{\sigma}_{AF,S}$ the covariance between family breeding value and IGE to non-family members, $r_{AF,S}$ the correlation between family breeding value and IGE to non-family members, $\hat{\sigma}^2_P$ the phenotypic variance, $\hat{h}^2_F$ the family heritable variation, and $\hat{T}^2$ the total heritable variation.

[1] $\mathbf{y} = \mathbf{Xb} + \mathbf{Z}_D\mathbf{a}_F + \mathbf{Z}^u_S\mathbf{a}_S + \mathbf{Wg} + \mathbf{Vk} + \mathbf{e}$

[2]We fitted a cage and cage*sex covariance. The result here is expressed as the correlation between cage mates and cage mates of the same sex.

$$\rho = \frac{\hat{\sigma}^2_g}{\hat{\sigma}^2_{g*s} + \hat{\sigma}^2_g + 0.5*\left(\hat{\sigma}^2_{e_m} + \hat{\sigma}^2_{e_f}\right)},$$

$$\rho_f = \frac{\hat{\sigma}^2_{g*sf}}{\hat{\sigma}^2_{g*s} + \hat{\sigma}^2_g + 0.5*\left(\hat{\sigma}^2_{e_m} + \hat{\sigma}^2_{e_f}\right)},$$

where, $\hat{\sigma}^2_g$ refers cage variance , $\hat{\sigma}^2_{g*s}$ cage*sex variance.

$$\rho_m = \frac{\hat{\sigma}^2_{g*sm}}{\hat{\sigma}^2_{g*s} + \hat{\sigma}^2_g + 0.5*\left(\hat{\sigma}^2_{e_m} + \hat{\sigma}^2_{e_f}\right)}$$

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