

Genetic parameters of tick-infestation on lambs of the Norwegian White Sheep breed

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ABSTRACT: Sheep farmers in Norway experienced an increase in lamb loss on range pasture during the last decades. It is proposed that one reason for this increase in lamb loss in coastal areas is due to tick-borne fever (TBF) caused by *A. phagocytophilum* infection transmitted by the tick *Ixodes ricinus*. Within breed variation in response to an *Anaplasma phagocytophilum* infection suggest that genetic variation is present. Here genetic parameters of tick-count on lambs are estimated using data on 555 lambs of the Norwegian White Sheep breed from 6 different farms and a 10-generation pedigree. Results suggest that heritability for tick-count among Norwegian White Sheep was moderate to high and that tick-load may be reduced by selective breeding. The heritability estimates presented in this study may however be inflated. Further, the relationship between tick-count and robustness to TBF need further investigation and verification.

Keywords: sheep; ticks; robustness

Introduction

Sheep farming in Norway is based on grazing on unfenced rangeland and mountain pastures in summer. There has been an increase in lamb loss on such pastures from about 4.8% in 1990 to 8.3% in 2011 (Norwegian Forest and Landscape Institute (2013)). Sheep farming is facing a loss in production due to tick-borne disease. The tick *Ixodes ricinus* may transmit the bacteria *Anaplasma phagocytophilum*, and cause tick-borne fever (TBF) in sheep. TBF is stated as one of the main scourges in sheep farming in coastal areas (Stuen (2003)) and it is proposed to be an explanatory factor of the observed increase in lamb loss during the last decades. Lamb losses as high as 30% in a flock due to *A. phagocytophilum* infection are reported (Stuen and Kjølleberg (2000)) and the Norwegian Food Safety Authority considers restrictions of grazing on pastures with high losses due to the severe welfare problems (The Norwegian Food Safety Authority (2011)).

Tick-infestation in sheep is commonly controlled by chemical treatment (acaricides). The frequent use of such treatment is however associated with development of resistance against such treatment. An alternative strategy to control tick-infestation is to use genetically robust animals. Genetic variation in robustness is shown in many farmed species, where numerous diseases are involved (Bishop et al. (2010)) e.g., gastrointestinal nematode infections (Stear (1994)), mastitis (Rupp (2009)), footrot (Raadsma (2011)), ectoparasites (flies and lice) (Raadsma (1991)) (Pfeffer

(2007)) and scrapie (Dawson (1998)) in sheep. Various levels of host resistance to tick-infestation are found to occur in different breeds of cattle and have been implemented in breeding schemes (Utech et al. (1978); Lemos et al. (1985); Prayaga (2003)). Individual variation in response to *A. phagocytophilum* infection in sheep is evident and shown by Granquist et al. (2010) and Stuen et al. (2011). This variation in response to infection might include genetic variation in robustness to *A. phagocytophilum* infection. The risk of being infected increases with number of ticks infested as prevalence of *A. phagocytophilum* in ticks vary up to some 25% of ticks (Rosef (2009)). Hence, tick-count on lambs may to some degree reflect the susceptibility of *A. phagocytophilum* infection in lambs. To our knowledge, this is the first study where tick-counts of the *Ixodes ricinus* tick on sheep are used as a measure for genetic variation of robustness to tick-infestation in sheep.

Hence, the objective of this study is to identify possible within-breed genetic variation in tick-counts in lambs on tick-infested pastures.

Materials and Methods

Data recording. The study was conducted in 2011, 2012 and 2013 on 6 sheep farms in tick endemic areas on the west coast of Norway in Vestnes, Rauma and Fræna municipalities where ticks and losses to TBF have been observed earlier. Presence of ticks was confirmed by examining the pastures for questing ticks with the cloth lure method (Vassallo et al. (2000)) at the same day as counting ticks on the lambs. Tick counts were recorded on 555 lambs grazing on 12 different fenced pastures during spring. Lambs were sired by 78 rams that were mated with 283 dams. Tick-counts were repeatedly recorded on the same lambs at approximately 1 and 2 weeks after turn out to pasture from the winter indoor feeding period. Tick counts were observed on the head, armpit, and groin of the lamb.

Statistical analyses. (Co)variance components were estimated using maximum likelihood algorithm in ASReml (Gilmore et al. (2008)). Three alternative sire-dam mixed models were used; linear models on observed tick-count, linear model on natural logarithm [observed tick-count + 1] and Poisson model. Tick count could be viewed as a Poisson process, and an advantage with the Poisson model is its ability to account for higher variance at higher mean (Perez-Encizo et al. (1993)). The fixed variables of sex, age, rearing rank, farm and pasture was included in the

initial model. Sex, age and rearing rank was not significant and was removed from the models applied in the analyses. The random effects were sire (S), dam (D) and common environmental effect (C) and residual (e). To account for repeated measurement of the tick-count, permanent environmental effect (PE) was also fitted as a random effect. Phenotypic variance (V_P) was calculated as $(2*V_{SD} + V_C + V_{PE} + V_e)$, heritability (h^2) both observed scale from the linear models and liability scale from the Poisson model, common environmental effect (c^2) and repeatability (r) were calculated using the following formulas: $h^2 = (4*V_{SD})/V_P$, $c^2 = V_C/V_P$ and $r = (V_{PE} + 4*V_{SD})/V_P$. For the Poisson model, the residual variance was estimated as $1/\bar{\lambda}$, where the $\bar{\lambda}$ is the mean Poisson parameter, here mean tick count (Foulley et al. (1987), Olesen et al. (1994)).

Results and Discussion

Descriptive statistics. The mean numbers of ticks on the lambs at the first and second tick-counting were 1.6 (SD = 2.4) and 1.2 (SD = 1.7). A number of lambs had zero tick-load (46.13% for the first count and 51.17% for the second count) as shown in Fig 1. Tick-counts ranged from 0 to 21 on the first counting and from 0 to 12 on the second counting. The tick (*Ixodes ricinus*) is a three host tick and the tick only attaches to the host for a period of 4 to 11 days (Macleod (1932)). Ticks may therefore attach and fall off between the points of time for the tick-count here. Other studies on tick-count as an indicator of robustness to ticks are mainly done on the *Rhipicephalus microplus* tick which is a one host tick and stays on the host for the whole life cycle. However, counting ticks twice at 7 days interval on the same lambs after they are exposed to ticks was considered an appropriate procedure for this study.

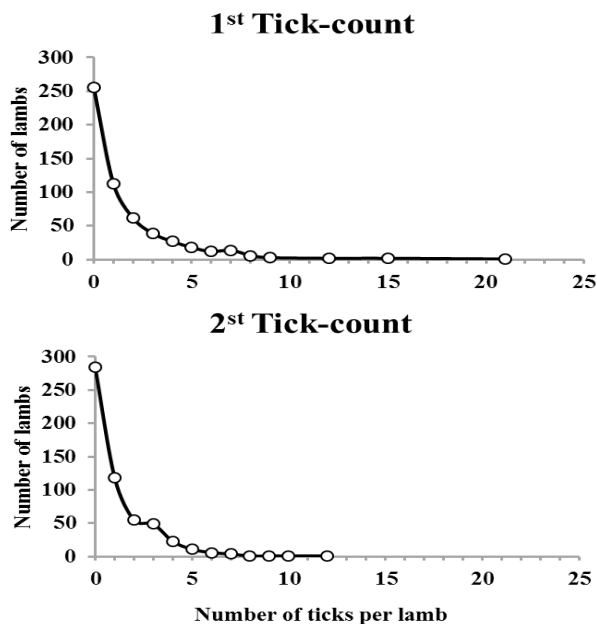


Figure 1. Distribution of the first and second tick-count per lam.

Tick-load on sheep will depend on the tick-load on pastures (unpublished data Grøva). There are a number of biological factors and physiological mechanisms involved in the attachment of ticks to a host, transfer of disease from tick to host and finally development of disease in an animal. Ticks feeding on resistant hosts show reduced weights when engorged, reduced fecundity and survival through moulting (Walker and Fletcher (1987)). It is suggested that site-resident sheep continually exposed to ticks acquire tick resistance (Ogden et al. (2001)). This resistance was measured by weighing engorged *Ixodes ricinus* nymphs from sheep resident in a tick-infested site compared to engorged nymphs from previously tick-naïve sheep. There is however little knowledge on the potential consequences this may have for the transmission of tick-borne diseases.

It is suggested that tick-resistant animals inhibit transmission of some tick-borne pathogens, including the bacteria *Francisella tularensis* (Wikel (1980)) and *Borrelia burgdorferi* sensu stricto (Wikel et al. (1997)) and the protozoon *Theileiria parva* (Fivaz, Norval and Lawrence (1987)). Previous studies on the prevalence of *A.phagocytophilum* in tick endemic areas suggest that prevalence of infection in sheep in areas with ticks is high (Stuen and Bergstrøm (2001)) Grøva et al. (2011)) and even up to 100% (Ogden et al. (1998)).

Genetic parameters. The h^2 for tick-count ranged from 0.32 to 0.59, depending on the models (Table 1). Variation in host resistance to ticks is also reported within cattle breeds and h^2 for tick infestation varied from 0.13 to 0.44 (Regatiano and Prayaga (2010)). Our estimates from the linear model is in the range of the previous study. Repeatability estimates ($r = 0.39$ to 0.69) were slightly higher than the estimated h^2 , indicating that V_{PE} was relatively lower than additive genetic variance and that the tick-count was a highly repeatable trait.

Table 1. Estimates of heritability (h^2), repeatability (r) and common environmental effect (c^2) of tick-count by three alternative models.

Model	h^2	r	c^2
Linear [observed]	0.37 ^{0.09}	0.39 ^{0.08}	0.00 ^{0.00}
Linear [ln(observed+1)]	0.32 ^{0.09}	0.37 ^{0.07}	0.00 ^{0.00}
Poisson	0.59 ^{0.10}	0.69 ^{0.09}	0.00 ^{0.00}

Superscript is a standard error of the estimate.

The h^2 for tick-count may however be inflated by common environmental effect. The tick-load of full-sib lambs is likely determined by the ewe's maternal ability. Therefore the phenotypic variance is expected to contain common environmental effect to full-sibs although the c^2 here was bounded to zero. This may be due to the small dataset which was not sufficient to disentangle the V_C from the V_S . Hence, no solid conclusion on the magnitude on h^2 can be drawn. However, the results (may) indicate that tick-count show genetic variation and that tick-load may there-

fore be reduced by selective breeding. The h^2 estimated from the linear models (0.32 to 0.37) was lower than h^2 from the non-linear Poisson model (0.59), which might be expected due to the difference in scale (observed scale versus liability scale). Anyhow, the goodness of fit of three different models should be compared with respect to its predictability. As a number of records were zero, an alternative zero-inflated Poisson model may fit the data better than regular Poisson model.

The success of selection for robustness to a disease depend on correctly recording of the phenotype. Before tick-count records can be selected for to improve robustness to TBF, further knowledge on covariation between tick number and pathogen transfer and disease development is needed.

Conclusion

Results suggest that heritability for tick-count among Norwegian White Sheep was moderate to high and that tick-load may be reduced by selective breeding. The heritability estimates presented in this study may however be biased upwards. More studies are needed to accurately estimate the heritability for tick-count.

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