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Incidence and heritability of ovine pneumonia, and the relationship with production traits in New Zealand sheep

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ABSTRACT

The cost of ovine pneumonia and pleurisy to the New Zealand economy is high, with the majority of loss through slower growth and reduced carcass value at slaughter. Farm management practices and vaccine development have traditionally been the main focus for prevention of pneumonia. The objective of this study was to estimate the heritability of pneumonia in New Zealand lambs, and investigate the genetic relationship with key production traits.

The lungs of 11,437 lambs from pedigree-recorded flocks were scored for the presence and severity of pneumonic lesions at slaughter. On average 28% of lambs had pneumonic lesions at slaughter, with 7% showing severe lesions. The incidence of pleurisy in these animals was 6%. Heritability estimates for pneumonic lesions and pleurisy were 0.07 ± 0.02 and 0.02 ± 0.01, respectively. There was a significant positive genetic correlation between pneumonic lesions and faecal egg count (0.30 ± 0.13). Animals with pneumonic lesions had grown faster from birth to weaning, and slower from weaning to slaughter than animals without lesions.

This study has shown that there is a heritable component to pneumonia in sheep. Including more data from pedigree-recorded flocks with severe pneumonia and a high incidence of pleurisy will enable more accurate estimates of genetic parameters, and subsequent correlations with production and disease traits. This would be aided by routine recording of pneumonia in lungs at slaughter by processing plants.

1. Introduction

Respiratory diseases, including pneumonia, are common in most livestock around the world. The economic cost of pneumonia to the New Zealand sheep industry is high and consists of three main factors; direct losses of stock on farm, reduced weight gains and wool production from sub-clinical animals, and condemnation and downgrading of carcasses at slaughter (West et al., 2009). A study by Goodwin-Ray et al. (2008c) estimated the annual cost of pneumonia to the New Zealand sheep industry to be NZD$1.36 to $3.31 per lamb, with the prevalence of pneumonia during February through April having the greatest impact on cost. However, this is a conservative estimate of the cost to the industry given the study did not include the costs associated with pneumonia related mortalities on farm. Pneumonia has been reported to have a significant effect on lamb growth rate (Kirton et al., 1976; Alley, 1987; Goodwin et al., 2004). The extent of the loss depends on the study; the first study of its kind in New Zealand sheep by Kirton et al. (1976) showed that when adjusted for weaning weight moderate to severe pneumonia reduced carcass weights by 450 g. Alley (1987) found that carcass weights of lambs with experimentally-induced chronic non-progressive pneumonia were on average 1.5 kg lighter than controls over a 2 month period. Goodwin et al. (2004) addressed the relationship between severity of pneumonia and average daily gain (ADG) in the month prior to slaughter, finding that when more than 20% of the lung surface area was affected by pneumonia, ADG decreased from 136 to 65 g/day.

In sheep respiratory disease is etiologically complex, resulting from an interaction of the infectious agents (bacterial, mycobacterial, and viral) with the host's defence mechanisms, which are often compromised by environmental factors (Alley, 2002). In New Zealand the term chronic non-progressive pneumonia (CNP) is typically used to describe subclinical pneumonia, which is widespread among lambs (Goodwin et al., 2004). Acute pneumonia occurs more sporadically, and occurs in sheep of all ages. The disease has a rapid onset and the first sign is often the death of the affected animal, although recovery with chronic lesions may also occur (Alley, 2002). Animals that have subclinical pneumonia and those that have survived clinical pneumonia may develop pleurisy, where the lungs adhere to the chest wall.

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Clinical diagnosis of pneumonia is difficult, with accurate diagnosis requiring post-mortem examination of the lungs; hence the animal needs to be euthanised. Traditionally, the main focus for prevention of pneumonia has been farm management practices and vaccine development. A case-control study by Goodwin-Ray et al. (2008a) identified shearing lambs at weaning, breeding replacement ewes on farm and contact with other flocks through the purchase of lambs post-weaning as practises likely to increase pneumonia prevalence. Vitamin B12 treatments at time of docking and weaning, as well as set stocking lambs post-weaning, were seen as protective practises. Lambs slaughtered later in the season versus at or around the time of weaning are at much higher risk, but this is more likely to be a consequence of slower growth because of pneumonia than a cause of pneumonia. A vaccine based on the serotype S1 strain of *Mannheimia haemolytica*, the primary cause of lung damage, has been shown to provide cross-protection against S2 strains in an experimental challenge (Zheng et al., 2015). The evaluation of a commercial vaccine containing antigens of *M. haemolytica* and *Pasteurella trehalosi* (Ovipast Plus, Intervet) under field conditions showed no difference in prevalence of pneumonic lesions or ADG between the placebo-treated and vaccinated lambs (Goodwin-Ray et al., 2008b).

Improvement of animal health through genetic selection can provide a complementary approach to disease control (Stear et al., 2001; Bishop and Morris, 2007; Berry et al., 2011). Baird et al. (2012) developed a pneumonic lesion scoring system in the processing plant (chain speed), and estimated the heritability of the consolidated pneumonic lesion score in pedigree-recorded lambs to be 0.12 ± 0.06. This is comparable to estimates of heritability of bovine respiratory disease (BRD) resistance in cattle, which range from 0.04 to 0.26 (Muggli-Cockett et al., 1992; Snowder et al., 2005; Schneider et al., 2010).

The objective of this study was to expand on previous work to estimate the heritability of pneumonic lesions in New Zealand lambs, and, in addition, investigate the genetic relationship with key production traits

2. Materials and methods

The work reported here was undertaken using records sourced from New Zealand sheep breeders and stored in the Sheep Improvement Limited database (SIL, www.sil.co.nz). The animals were managed in accordance with the provisions of the New Zealand Animal Welfare Act 1999, and the New Zealand Codes of Welfare developed under sections 68–79 of the Act.

2.1. Animals

The lungs from a total of 11,437 ram and ewe lambs from one North Island (Flock H) and eight (Flocks A–G and I) Southland and Otago pedigree-recorded flocks were scored for the presence and severity of pneumonic lesions. Data collection was carried out from January to May. Animals from flocks I and E were selected for slaughter by weight and body condition score at 3 different time points. Pneumonic lesions were only recorded in lambs slaughtered at the second and third time points, as preliminary studies had shown little to no lesions in the lungs of lambs slaughtered at weaning. All other flocks were fixed-date slaughters, and primarily took place between February and March. Animals were predominantly composites of the main dual-purpose sheep breeds used in New Zealand, including Romney, Coopworth, Perendale, and Texel.

2.2. Pneumonia scoring system

The extent of the pneumonic lesions within the lungs was determined post-slaughter at the processing plant, ensuring the lungs were fresh. For lambs born 2008–2010 this was determined by removing the lungs from the processing chain and visually assessing each of the five lobes. Each lobe was scored on a 0–5 scale, indicating the percentage of surface area affected, where 0 = no lesions present; 1 = small lesions; 2 = approximately 25% of the lobe affected; 3 = approximately 50% of the lobe affected; 4 = approximately 75% of the lobe affected and 5 = 100% of the lobe affected. Pneumonic lesions were defined as compacted, dark purple-red areas of the lung that were firm to touch (Fig. 1B and C). The sum of the individual lobe scores was calculated and recorded as a 'total lesion score' (TLS). The TLS system is not appropriate for recording pneumonic lesions at chain-speed on the processing chain at the plant, where up to 10 animals per minute may be slaughtered. A refined scoring system was devised by Baird et al. (2012) and was implemented for recording pneumonic lesions at chain-speed from the progeny born in 2011 onwards. This refined TLS system, termed 'consolidated pneumonia score' (CPS), has a range from 0 to 2, where 0 = no lesions present; 1 = any individual lobe with up to 50% of the lobe affected and 2 = any individual lobe with greater than 50% of the lobe affected (Fig. 1). Animals that had been scored under the TLS system were retrospectively given CPS values, and CPS was used for all subsequent analysis.

2.3. Production Traits

Carcass weight (CWT), and primary and secondary carcass fault data, including pleurisy (PLEUR) were obtained from the meat processing plant. The carcass measurements of depth of tissue 110 mm off the mid line in the region of the 12th rib (CGRM) and carcass butt circumference (CBUTT) were measured the day post-slaughter, before the further processing of the carcass. All other data, including Julian date of birth (JDOB), birth rearing rank (BRR; born as a single, twin or triplet, and raised as a single, twin or triplet), age of dam AOD; (1 year old, 2 years old, or older), birth weight (BWT), weaning weight (WWT), weaning weight mob, pre-slaughter weight (PRESLTWT), pedigree, date of slaughter and breed were obtained from Sheep Improvement Limited (SIL) records.

2.4. Statistical analysis

Data cleaning consisted of removal of records with 1) missing values for sire information or weaning weight, and 2) contemporary groups containing less than 5 observations. Weight and slaughter traits were adjusted using SIL standard practice (S-A. Newman, personal communication) to account for relationships between contemporary group mean and variance, using the equation: $aTr = (Tr/Trm)^{*}oTrm$ where *Trm* is the mean for the contemporary group, and *oTrm* is the overall mean for the trait (Brown et al., 2005). Contemporary group (CG) was specific to each trait, and was defined as flock, birth year, sex, weaning mob and trait/slaughter mob combination. The traits adjusted using this approach were BWT, WWT, PRESLTWT, CWT, CBUTT and CGRM. Summer faecal egg count values are a repeatable trait, and were estimated from two samples, taken several days apart. These were distinguished as *Nematodirus* (NEM1) or 'other trichostrongyles' (FEC1; primarily *Teladorsagia* spp, *Trichostrongylus* spp and *Cooperia curticei*). Both FEC1 and NEM1 values were log transformed before analysis ($\ln(X+25)$).

To account for lack of challenge in some contemporary groups (CGkill; sex, birth year, flock, weaning mob and kill date), data was set to missing if the mean CPS in that contemporary group was below 0.1. This is assuming that animals in the contemporary groups where CPS was low were not sufficiently exposed to stressors and pathogens to express a phenotype. To adjust for heteroscedasticity, CPS (initially scored as 0, 1 or 2) was scaled using the formula $CPSa = CPS/SQRT(CPSm^{*}(2-CPSm))$, where *m* is the