South African Journal of Animal Science 2026, vol. 56(8)

Short communication: Genetic predisposition to wet carcass syndrome in sheep – evidence from a SNP-based heritability analysis

B. Bhika Kooverjee[1,2#], P. Soma[1], M.A. van der Nest[3], M.D. MacNeil[1,2,4], & F.W.C. Neser[2]

[1]Agricultural Research Council Animal Production, Irene, South Africa
[2]Department of Animal Sciences, University of the Free State, Bloemfontein, South Africa
[3]Forestry and Agricultural Biotechnology Institute, University of Pretoria, South Africa
[4]Delta G, 145 Ice Cave Road, MT 59301, Miles City, USA

(Submitted 24 November 2025; Accepted 03 June 2026; Published 04 August 2026)

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Abstract

Wet carcass syndrome (WCS) is a defect that occurs in sheep and is characterised by a wet, translucent appearance of the carcass, resulting in condemnation and industry losses. This defect is undetectable until the carcass has been chilled after slaughter, and genetic improvement has thus been proposed as a potential mitigation strategy. This study estimated the single nucleotide polymorphism-based heritability of WCS using a case-control population of South African Dorper, merino-type, and cross-bred sheep genotyped using the Illumina OvineSNP50 BeadChip. After quality control, 32 077 single nucleotide polymorphisms and 540 animals (164 cases, 376 controls) were retained. A genomic relationship matrix was constructed, and heritability was estimated using genomic-relatedness-based restricted maximum likelihood on the observed and liability scales. The liability-scale heritability was 0.30 ± 0.03, demonstrating a substantial additive genetic component underlying WCS susceptibility. These results provide foundational evidence that susceptibility to WCS has a genetic component and support future genome-wide association studies and genomic selection aimed at reducing the prevalence of this economically important condition.

Keywords: GREML, meat quality, ovine genomics, SNP-based heritability estimate

[#]Corresponding author: KooverjeeB@arc.agric.za

Wet carcass syndrome (WCS) was first reported in South African Dorper and Dorper cross-bred sheep in 1981 (Brock *et al.*, 1983). It only presents after slaughter, and is characterised by fluid accumulation that produces a carcass with a wet, translucent, and unaesthetic appearance (Van der Westhuizen *et al.*, 2019). Animals are therefore classified post-mortem as either affected or unaffected by WCS. Affected carcasses are considered unsuitable for human consumption (primarily to protect public health from zoonotic diseases, chemical residues, and food pathogens), with the meat consequently diverted to processed or pet-food markets, resulting in financial losses across the value chain (Hatting, 2023). Affected animals appear clinically normal before slaughter, making early diagnosis and management interventions infeasible. Recent reports indicate an increase in WCS cases

URL: www.sajas.co.za
ISSN 0375-1589 (print), ISSN 2221-4062 (online)
Publisher: South African Society for Animal Science
https://doi.org/10.17159/sajas.v56i08.01

over the past five years (National Animal Health Disease Information System, 2024), highlighting its growing significance. Although initially thought to be specific to the Dorper breed, WCS has now been observed in several South African breeds, such as merino-types, Meatmasters, and cross-bred sheep (Kooverjee *et al.*, 2025).

Van der Westhuizen *et al.* (2019) were the first to investigate the possibility of a genetic mechanism causing WCS. This relatively small study discovered two genetic markers associated with the condition, implying that genetics may contribute to the occurrence of WCS in South African sheep. The degree to which WCS is heritable is unknown. Heritability – the proportion of trait variation attributable to additive genetic effects – is central to the efficacy of selection (Yang *et al.*, 2017). For breeders, an estimate of heritability allows the prediction of the response to selection for desirable traits, including disease resistance. The advent of genomic technologies enables the estimation of heritability based on the analysis of single nucleotide polymorphism (SNP) genotypes using genomic relationship matrices. Methods such as genomic-relatedness-based restricted maximum likelihood (GREML), implemented in genome-wide complex trait analysis (GCTA), are now used for complex traits, including disease susceptibility (Yang *et al.*, 2011). Unlike pedigree-based approaches, SNP-based heritability does not rely on recorded kinships to form a relationship matrix, but instead uses relationships predicted from shared alleles.

Heritability estimates for several economically important disease and fitness traits have shown the value of genetic improvement. For example, reports have indicated that resistance to gastrointestinal nematodes (commonly assessed using faecal egg counts, $h^2 = 0.2–0.4$) and foot rot ($h^2 = 0.2–0.3$) have low to moderate levels of heritability, indicating meaningful additive genetic variation and potential response to selection (Bishop & Morris, 2007; Nieuwhof *et al.*, 2008). More recently, genome-wide SNP studies have further supported the genetic basis of parasite resistance and flystrike susceptibility, enabling genomic selection approaches in sheep breeding programmes (Forbes *et al.*, 2014; Pickering *et al.*, 2015). Bovine congestive heart failure, like WCS, is only observed after slaughter and has an estimated heritability of approximately 0.36 (Buchanan *et al.*, 2023). These examples support the broader principle that disease incidence may be reduced through selection when additive genetic variance exists, particularly when genomic information is combined with phenotypes recorded from relatives or at slaughter.

Wet carcass syndrome was analysed as a binary trait because carcasses are classified at inspection as either affected or unaffected, resulting a dichotomous phenotype suitable for case-control analysis. For such traits, observed-scale heritability is affected by ascertainment bias when cases are oversampled (Srivastava *et al.*, 2023). Case-control datasets are typically enriched for affected individuals, resulting in the observed phenotypic variance being skewed. This bias can be corrected using GREML, which assumes an underlying normally distributed liability for the disease (Yang *et al.*, 2017). This allows the transformation of heritability estimates from the observed scale to the liability scale, yielding biologically meaningful values that reflect population-level risk (Lee *et al.*, 2011).

Despite the economic implications of WCS, its underlying genetic architecture remains poorly understood. In particular, it is unclear whether susceptibility to WCS is influenced by additive genetic effects that could be exploited through selection. Therefore, the aim of this study was to quantify the SNP-based heritability of WCS in South African sheep using genome-wide SNP data, in order to determine the extent to which additive genetic variation contributes to the condition. It was hypothesised that WCS susceptibility is influenced by additive genetic effects captured by SNP markers, resulting in the SNP-based heritability being significantly greater than zero. Establishing the magnitude of heritability for WCS is an essential first step towards evaluating selection as a mitigation strategy. This short communication reports the first estimate of SNP-based heritability for WCS in sheep.

A case-control design combined with SNP-based GREML was chosen to enable the robust estimation of the additive genetic variance for this relatively rare condition that manifests post slaughter, while accounting for ascertainment bias and leveraging realised genomic relationships. For this purpose, the genotype data of a population that included both sheep that exhibited WCS and sheep that were unaffected were used to estimate the heritability on both the observed and liability scales using the GREML method. The results provide the foundational evidence needed to assess the potential for genetic solutions to this costly industry problem.

Ethical approval was granted by the Agricultural Research Council Animal Production Ethics Committee (APIEC 23/07). This study included the genotypic data of 164 sheep confirmed with WCS post-slaughter, 82 contemporaneous unaffected controls (as classified during carcass inspection), and

294 additional genotyped reference animals from external pure-bred populations with no recorded evidence of WCS. The confirmed WCS sheep and their contemporaries originated from multiple South African regions (Northern Cape, Free State, Mpumalanga, Eastern Cape, and Gauteng) and represented several breeds. These included Dorper (n = 150), Meatmaster (n = 1), Van Rooyen (n = 2), White Dorper (n = 2), South African Mutton Merino (n = 2), and Merino sheep (n = 41). Cross-breeds such as Dorper × Meatmaster and additional cross-bred animals (n = 47) whose breed composition was unknown were also included. All animals were under 12 months of age and were classified as A-grade lambs.

Data for an additional 294 animals with SNP genotypes were obtained from the Grootfontein Agricultural Development Institute and Dzomba *et al.* (2021). These data were from presumably unafflicted animals and included Afrino (n = 51), Black Head Persian (n = 14), Dohne Merino (n = 50), Dorper (n = 12), Meatmaster (n = 48), Merino (n = 91), Namakwa-Afrikaner (n = 10), South African Merino (n = 8), and South African Mutton Merino (n = 10) sheep. The *Ovis aries* Oar_v4.0 (GCF_000298735.2) genomic sequence was selected as the reference genome for all analyses. The sample size (>500 animals) is comparable to previous SNP-based heritability studies (Arzik *et al.*, 2022) and is sufficient to detect moderate additive genetic variance using GREML, although larger datasets would improve precision. Using the genomic relationship matrix accounted for population structure potentially arising from multiple breeds and regions, with approximately normal distribution of relatedness values indicating minimal stratification bias.

All animals were genotyped using the Illumina OvineSNP50 BeadChip, as described by Kooverjee *et al.* (2025). Quality control was performed in PLINK v1.9 (Purcell *et al.*, 2007) and GCTA v1.93 (Yang *et al.*, 2011). Single nucleotide polymorphisms (SNPs) that exhibited a minor allele frequency <0.01, a genotype call rate <95%, or significant deviation from Hardy-Weinberg equilibrium ($P < 1 \times 10^{-6}$) were excluded. Individuals were removed if their genotype call rate was below 90%. After quality control, 32 077 SNPs and 540 animals remained for subsequent analyses. To reduce redundancy due to linkage disequilibrium, linkage disequilibrium-pruning was applied using a sliding window approach (50 kb window, 10-SNP step, $r^2 = 0.2$ threshold).

A genomic relationship matrix (GRM) was constructed using autosomal SNPs following the approach of Yang *et al.* (2011):

$$G_{ij} = \frac{1}{M}\sum_{k=1}^{M}\frac{(x_{ik} - 2P_k)(x_{jk} - 2P_k)}{2P_k\,(1 - P_k)}$$

where: G_{ij} represents the genetic relationship between individuals *i* and *j*, x_{ik} is the genotype of individual *i* at SNP *k* (coded as 0, 1, or 2), P_k is the allele frequency of SNP *k*, and *M* is the number of SNPs.

The SNP-based estimate of heritability was calculated using the GREML approach, implemented in GCTA via a linear mixed model including batch effects as fixed effects (Yang *et al.*, 2011). The linear mixed model assumes that the phenotype (disease status) is influenced by an additive genetic component and residual variance, as in Henderson (1984):

$$y = X\beta + g + e$$

where: *y* is the phenotype vector (0 = control, 1 = case), $X\beta$ represents the fixed effects (genotyping batch), *g* is the random genetic effect distributed as N (0, $G\sigma_g^2$), and *e* is the residual term distributed as N (0, $I\sigma_e^2$).

The significance of the estimated genetic variance component (σ_g^2) was assessed using a likelihood ratio test (LRT), comparing the full model to a null model without the genetic effect. Statistical significance was set at $\alpha = 0.05$.

For quantitative traits, the scale of measurement is the same as the scale on which heritability is expressed. For disease traits, however, the phenotypes (afflicted and unafflicted cases) were measured on the 0 to 1 scale and heritability is most readily interpreted on a scale of liability (Lee *et al.*, 2011). Liability models facilitate an understanding and comparison of the relative importance of genetic and environmental risk factors that shape different clinically important outcomes, through the estimation of the liability-scale heritability (Ojavee *et al.*, 2022). The liability model is a statistical convention, not a proven biological mechanism for WCS.

Since the trait has a binary distribution, heritability was first estimated on the observed scale ($h^2{}_{Obs}$) and then transformed to the liability scale ($h^2{}_L$) to account for the ascertainment bias resulting from the oversampling of afflicted cases that is inherent in case-control studies. This transformation assumes an underlying normally distributed liability for disease, with a threshold corresponding to the population disease prevalence (Lee *et al.*, 2011):

$$h^2{}_L = \frac{(h^2{}_{Obs} \times K^2 (1-K)^2)}{Z^2 \times P(1-P)}$$

where: *K* is the population prevalence (*K* = 0.05), *P* is the sample prevalence (*P* = 154/540 = 0.3048), and *z* is the standard normal density at the liability threshold.

The value of K was calculated from data presented in reports of the National Animal Health Disease Information System for ten years from 2015 to 2024. The number of cases is likely to have been underreported, and the size of the relevant population is unknown. Thus, the value of $h^2{}_L$ that is reported herein is probably an underestimate of its true value.

The GRM displayed an approximately normal distribution of pairwise relatedness values, indicating minimal close relatedness and a suitable population structure for GREML application. Heritability estimated on the observed scale was 0.53 ± 0.06 (Table 1), indicating that a substantial proportion of variation in WCS status within the case-control sample is explained by additive genetic effects captured by SNP markers. After transforming to the liability scale, the estimate of heritability was reduced to 0.30 ± 0.03, indicating that nearly 30% of the variation in WCS susceptibility is attributable to additive genetic effects captured by common SNPs. This moderate heritability suggests that genetic factors contribute meaningfully to WCS risk, although environmental influences and other non-additive effects also play a substantial role. Thus, additive genetic factors may have a role in determining whether or not an individual sheep develops WCS.

Table 1 Estimates of single nucleotide polymorphism-based heritability for wet carcass syndrome from a case-control study (± standard error)

Scale	Genetic variance	Residual variance	Phenotypic variance	h^2 estimate
Observed	0.09 ± 0.01	0.08 ± 0.01	0.17± 0.01	0.53 ± 0.06
Liability	–	–	–	0.30 ± 0.03

The LRT provided strong evidence for a significant genetic component (LRT statistic = 141.859, $P < 1 \times 10^{-10}$). These findings indicate that variation in WCS occurrence is not solely explained by random processes, but that additive genetic factors contribute to susceptibility and can be detected using genomic markers. Transformation to the liability scale adjusts for the over-representation of cases in the case-control sample, providing an estimate of underlying risk that is more comparable to the broader population (Ojavee *et al.*, 2022). However, this framework assumes a common latent susceptibility across animals, whereas the true risk for WCS may also differ according to breed composition, management system, physiological status, and environmental conditions. Consequently, the heritability estimate should be interpreted as an average population-level parameter rather than a uniform risk across all sheep.

This study provides the first estimate of heritability for WCS in sheep and demonstrates that the condition has a significant additive genetic component. The liability-scale heritability estimate of 0.30 places WCS among other moderately heritable disease traits in sheep, such as faecal egg count and foot rot resistance (Nieuwhof *et al.*, 2008; Pickering *et al.*, 2015; Forbes *et al.*, 2024). These levels of heritability are considered sufficient to support genomic selection strategies. Given that WCS manifests post-slaughter and is likely influenced by pre-slaughter stress (Birhanu, 2020), metabolic status, and carcass handling conditions, the moderate heritability observed here is consistent with a complex trait influenced by both biological and environmental processes.

Although the liability-scale heritability estimated here is moderate, it is important to highlight that genomic tools can remain highly effective even when the true underlying heritability is low. Several

livestock studies show that genomic prediction can enhance selection accuracy for traits with heritability values as low as 0.03 to 0.10, for mastitis resistance in dairy cattle (Gaddis *et al.*, 2020), or 0.07 to 0.29, for resistance to bovine respiratory disease (Snowder *et al*., 2005). This is because genomic data capture realises genetic relationships among individuals, enabling the more accurate use of information from relatives compared to pedigree-based approaches (Clark *et al.*, 2012). This then becomes important for traits that cannot be measured directly on breeding candidates, including those measured post-mortem and disease-status traits. For example, the genomic information from siblings and progeny increased the prediction accuracy for carcass traits in beef cattle (Fernandes Júnior *et al.*, 2016), parasite resistance in sheep (Forbes *et al.*, 2024), and porcine reproductive and respiratory syndrome resistance in pigs (Rowland *et al.*, 2012). Across species, studies consistently demonstrate that even low heritability estimates do not hinder meaningful genetic progress when high density SNP data is available, along with phenotypes that can be linked through relatives. Interpretation of the present results should take into account the fact that the true population prevalence of WCS is uncertain and likely lower than in this case-control sample, influencing the liability-scale estimates. The multi-breed dataset may also introduce population structure, although mitigated through the use of a GRM, while residual stratification and environmental variation may still affect the estimated variance components.

Given the inability to diagnose WCS before slaughter, the identification of a substantial genetic contribution represents an important step forward. The results confirm that selection for reduced susceptibility is biologically feasible, and justify future research focused on identifying genomic regions associated with WCS susceptibility. Future studies should further evaluate breed-specific susceptibility and genotype-by-environment interactions contributing to WCS expression. Such work may include genome-wide association studies and the development of a genomically derived estimated breeding value to assist breeders in reducing the incidence of WCS.

Acknowledgements

The authors would like to thank the Technology Innovation Agency (API012403000010) and Red Meat Research and Development South Africa (API012403000003\) for funding this work. We would like to acknowledge the Cavalier and KLK abattoirs for making the samples available for this study. The authors are grateful to the Grootfontein Agricultural Development Institute and Dzomba *et al.* (2021) for sharing the sheep reference SNP data.

Author contributions

M.M. first hypothesised a genetic basis for wet carcass syndrome. B.B.K. and P.S. contributed to the acquisition of data. B.B.K. and M.v.d.N. analysed the data. B.B.K., P.S., and M.v.d.N. conceptualised the manuscript. B.B.K. drafted and wrote the initial version of the manuscript. P.S., M.v.d.N., F.N., and M.M. reviewed and revised the manuscript. All authors have read and approved the finalised manuscript.

Conflict of interest declaration

The authors have no conflict of interests to declare.

Data availability statement

The datasets presented in this article are not readily available because they are the property of South Africa sheep producers, and the information in these datasets is commercially sensitive. Requests to access the datasets should be directed to the corresponding author.

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