Inoue, K., Nishio, M., Takeda, M., Kishida, Y., Hirooka, H. (2026) Genomic autosomal contribution to total additive and non-additive genetic effects for carcass weight and beef marbling in Japanese Black cattle, *Proceedings 13th World Congress on Genetics Applied to Livestock Production* 23664: https://doi.org/10.31274/wcgalp.23664 ©2026 Inoue, et al. CC BY-NC-ND

Genomic autosomal contribution to total additive and non-additive genetic effects for carcass weight and beef marbling in Japanese Black cattle

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Introduction

Estimating non-additive genetic effects offers the potential to correct biases in genetic parameter and breeding value estimates, enabling more accurate prediction of animal performance. Neugebauer *et al*. (2010) and Tier and Meyer (2012) reported imprinting effects for carcass traits using pedigree-based imprinting models in beef cattle. In addition, large relative imprinting variances for beef marbling score (BMS) were found in different Japanese Black cattle populations (Inoue *et al*., 2020 & 2021; Okamoto *et al*., 2019). In recent years, the genotypic coding and construction of a genomic relationship matrix for an imprinting effect using single nucleotide polymorphism (SNP) data from animal-parents trios were developed by Nishio and Satoh (2015) in the context of genomic prediction. In addition, since variance components can now be estimated for each individual chromosome, it is possible to estimate the genetic contribution of each chromosome to a trait. Recently, Ogawa *et al*. (2022) estimated the autosomal contribution to total additive genetic variability of carcass traits in Japanese Black cattle. In this study, we utilized these methods to estimate the additive and non-additive genetic effects per chromosome for carcass weight (CW) and beef marbling score (BMS) in Japanese Black cattle.

Material & Methods

Data. The data used in this study were obtained from the carcass records (i.e., Animal Care and Use Committee approval was not necessary) of 1,908 Japanese Black steers and heifers fattened in Japan. The carcass records included CW and BMS, which is scored from 1 (poor) to 12 (abundant). BMS was evaluated between the sixth and seventh rib in accordance with the Japan Meat Grading Association (1988). Those 1,908 animals with records and their parents were genotyped using the GeneSeek GGP LD v4 SNP chip (Neogen GeneSeek Operations, NE, USA). The call rate of these animals was over 0.95. The SNP genotype data were imputed to the Illumina BovineSNP50 v2 BeadChip (Illumina, CA, USA) and then were phased by using Beagle ver. 4.0 (Browning and Browning, 2007) with pedigree information to distinguish the parental origin of the alleles. Finally, 34,481 SNPs on the 29 bovine cattle autosomes were used.

Variance component estimation. The following two statistical models were fitted to phenotypic values. Model 1 can be written as follows:

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z_a a} + \mathbf{Z_d d} + \mathbf{Z_i i} + \mathbf{e} \quad (1)$$

where $\mathbf{y}$ is a vector of the observed traits CW and BMS; $\mathbf{b}$ is a vector of fixed effects, including the sex of animals (two levels; steer and heifer), slaughter year (13 levels; 2006 to 2018), slaughter month (12 levels), carcass market (five levels), linear and quadratic covariates for age at slaughter (average 28.6 months); $\mathbf{a}$, $\mathbf{d}$, and $\mathbf{i}$ are vectors of additive genetic,

dominance, and imprinting effects of SNPs, respectively, and distributed as $\mathbf{a}\sim N(0,\mathbf{G_a}\sigma_a^2)$, $\mathbf{d}\sim N(0,\mathbf{G_d}\sigma_d^2)$, and $\mathbf{i}\sim N(0,\mathbf{G_i}\sigma_i^2)$, respectively; $\mathbf{e}$ is a vector of random residual effects as $\mathbf{e}\sim N(0,\mathbf{I}\sigma_e^2)$; and $\mathbf{X}$, $\mathbf{Z_a}$, $\mathbf{Z_d}$, and $\mathbf{Z_i}$ are known incidence matrices. $\mathbf{G_a}$, $\mathbf{G_d}$, and $\mathbf{G_i}$ are the additive genomic, dominance genomic, and imprinting genomic relationship matrices, respectively; $\mathbf{I}$ is an identity matrix; σ_a^2 is the additive genetic variance; σ_d^2 and σ_i^2 are variances due to the dominance and imprinting effects, respectively; and σ_e^2 is the residual variance. All other covariances between random effects were assumed to be zero. The genomic relationship matrices $\mathbf{G_a}$, $\mathbf{G_d}$, and $\mathbf{G_i}$ were calculated according to VanRaden (2008), Nishio and Satoh (2014) and Nishio and Satoh (2015), respectively, as follows:

$$\mathbf{G_a}=\frac{\mathbf{M_a}\mathbf{M_a}'}{\sum_{j=1}^{m}2q_j(1-q_j)},$$

$$\mathbf{G_d}=\frac{\mathbf{M_d}\mathbf{M_d}'}{\sum_{j=1}^{m}\{2q_j(1-q_j)\}^2},$$

$$\mathbf{G_i}=\frac{\mathbf{M_i}\mathbf{M_i}'}{\sum_{j=1}^{m}2q_j(1-q_j)},$$

where $\mathbf{M_a}$, $\mathbf{M_d}$, and $\mathbf{M_i}$ are $n\times m$ matrices (n is the number of genotyped individuals and m is the number of SNPs). Considering two alleles $\mathrm{A_1}$ and $\mathrm{A_2}$ with frequencies $1-q_j$ and q_j, respectively, in the population, the elements of $\mathbf{M_a}$, $\mathbf{M_d}$, and $\mathbf{M_i}$ for the *i*th individual at the *j*th SNP were calculated as follows (Nishio and Satoh, 2015):

$$\mathbf{M_a}_{ij}=\begin{cases}2q_j & \mathrm{A_1A_1}\\ 2q_j-1 & \mathrm{A_1A_2(A_2A_1)},\\ 2q_j-2 & \mathrm{A_2A_2}\end{cases}$$

$$\mathbf{M_d}_{ij}=\begin{cases}-2q_j^2 & \mathrm{A_1A_1}\\ 2q_j(1-q_j) & \mathrm{A_1A_2(A_2A_1)},\\ -2(1-q_j)^2 & \mathrm{A_2A_2}\end{cases}$$

$$\mathbf{M_i}_{ij}=\begin{cases}0 & \mathrm{A_1A_1}\\ 1 & \mathrm{A_1A_2}\\ -1 & \mathrm{A_2A_1}\\ 0 & \mathrm{A_2A_2}\end{cases}.$$

Model 2 was as follows:

$$\mathbf{y}=\mathbf{Xb}+\mathbf{Z_a}\mathbf{a_1}+\mathbf{Z_a}\mathbf{a_2}+\mathbf{Z_d}\mathbf{d_1}+\mathbf{Z_d}\mathbf{d_2}+\mathbf{Z_i}\mathbf{i_1}+\mathbf{Z_i}\mathbf{i_2}+\mathbf{e}, \quad (2)$$

where $\mathbf{y}$, $\mathbf{b}$, and $\mathbf{e}$ are as in Model 1; $\mathbf{a_1}$, $\mathbf{d_1}$, and $\mathbf{i_1}$ are vectors of additive genetic, dominance, and imprinting effects of SNPs on a particular autosome, respectively; $\mathbf{a_2}$, $\mathbf{d_2}$, and $\mathbf{i_2}$ are vectors of additive genetic, dominance, and imprinting effects of SNPs on the remaining 28 autosomes, respectively. The distributions of those six vectors were assumed as follows: $\mathbf{a_1}\sim N(0,\mathbf{G_{a1}}\sigma_{a1}^2)$, $\mathbf{d_1}\sim N(0,\mathbf{G_{d1}}\sigma_{d1}^2)$, $\mathbf{i_1}\sim N(0,\mathbf{G_{i1}}\sigma_{i1}^2)$, $\mathbf{a_2}\sim N(0,\mathbf{G_{a2}}\sigma_{a2}^2)$, $\mathbf{d_2}\sim N(0,\mathbf{G_{d2}}\sigma_{d2}^2)$, and $\mathbf{i_2}\sim N(0,\mathbf{G_{i2}}\sigma_{i2}^2)$, where $\mathbf{G_{a1}}$ $(\mathbf{G_{a2}})$, $\mathbf{G_{d1}}$ $(\mathbf{G_{d2}})$, and $\mathbf{G_{i1}}$ $(\mathbf{G_{i2}})$ are the additive genomic, dominance genomic, and imprinting genomic relationship matrices calculated using by the genotype data of the SNPs on a particular autosome (on the remaining autosomes), respectively; σ_{a1}^2 (σ_{a2}^2), σ_{d1}^2 (σ_{d2}^2), and σ_{i1}^2 (σ_{i2}^2) are additive genetic, dominance, and imprinting variances explained by the SNPs located on a particular autosome (on the remaining autosomes) , respectively.

The RKHS function in BGLR statistical package (Pérez and de los Campos, 2014) was used for obtaining posterior distributions of variance components. For each analysis, a single Markov chain with 15,000 samples was generated, with the initial 5,000 samples discarded as burn-in. Finally, variance components were determined based on the remaining 10,000 samples.

Results & Discussion

Posterior means of the variance components and ratios of variance for carcass traits estimated from the all autosomes (Model 1) are shown in Table 1. The dominance (σ_d^2) and imprinting (σ_i^2) variance components for the traits were small. The proportions of the dominance variance to the phenotypic variance (h_d^2) and imprinting variance to the phenotypic variance (h_i^2) were also small, being 0.08 (0.02) and 0.06 (0.01) for CW and 0.09 (0.03) and 0.05 (0.01) for BMS, respectively.

In studies using pedigree-based imprinting models, large imprinting variances for BMS were reported in different populations in Japanese Black cattle (Inoue *et al*., 2020 & 2021; Okamoto *et al*., 2019). In contrast to the previous pedigree-based studies, small imprinting variances and h_i^2 for BMS were estimated by using genomic relationship matrices in the present study. There were large differences between the imprinting effects for BMS from pedigree-based imprinting models and from genomic information.

Table 1. Posterior means of the variance components and ratios of variance for carcass traits estimated from the all autosomes (Model 1).

Trait[1]	Parameter[2] (s.e.)							
	σ_p^2	σ_e^2	σ_a^2	σ_d^2	σ_i^2	h_a^2	h_d^2	h_i^2
CW	2417.7	850.2	1246.5	187.7	133.3	0.52	0.08	0.06
(kg)	(96.0)	(81.8)	(117.8)	(55.3)	(32.1)	(0.03)	(0.02)	(0.01)
BMS	4.238	1.829	1.825	0.387	0.197	0.43	0.09	0.05
(No.)	(0.163)	(0.175)	(0.197)	(0.132)	(0.061)	(0.04)	(0.03)	(0.01)

[1] CW = carcass weight, BMS = beef marbling score.

[2] σ_p^2 = phenotypic variance, σ_e^2 = residual variance, σ_a^2 = additive genetic variance, σ_d^2 = dominance variance, σ_i^2 = imprinting variance, $h_a^2 = \sigma_a^2/\sigma_p^2$, $h_d^2 = \sigma_d^2/\sigma_p^2$, $h_i^2 = \sigma_i^2/\sigma_p^2$.

Proportion of additive, dominance, and imprinting genetic variances explained by each autosome to the correspond total genetic variances (additive: $\sigma_{a1}^2/(\sigma_{a1}^2+\sigma_{a2}^2)$, dominance: $\sigma_{d1}^2/(\sigma_{d1}^2+\sigma_{d2}^2)$, and imprinting: $\sigma_{i1}^2/(\sigma_{i1}^2+\sigma_{i2}^2)$) obtained by using Model 2 are shown in Figure 1. The proportions of individual autosome variance within the total genetic variance for additive genetic effect ($\sigma_{a1}^2/(\sigma_{a1}^2+\sigma_{a2}^2)$) of CW were relatively large in chromosomes 6, 7, 8, and 14 (Figure 1a). The large proportions in chromosomes 6, 8, and 14 could be reflected the influence of the *CW-2* (chromosome 6), *CW-3* (chromosome 8), and *CW-1* (chromosome 14) genes. In contrast to CW, no chromosome-specific characteristics were observed in BMS. Ogawa *et al*. (2022) also reported the large autosomal contribution to total additive genetic variability of CW in chromosomes 6, 8, and 14 in the different population of Japanese Black cattle.

In the dominance and imprinting variance proportions, no clear chromosome-specific characteristics were observed for either trait (Figure 1b and 1c, respectively). However, both variances showed a tendency to decrease in size along chromosome length (number of SNPs), from chromosome 1 to chromosome 29. These results suggest that different polygenic function may be at work between additive and non-additive genetic effects.

Conclusions

The proportion of individual autosome additive variance within the total genetic variance of CW were relatively large for chromosomes 6, 7, 8, and 14, reflecting the influence of major gene. In contrast to CW, no chromosome-specific characteristics were observed in BMS. In the dominance and imprinting variance proportions, no clear chromosome-specific characteristics were observed for either trait.

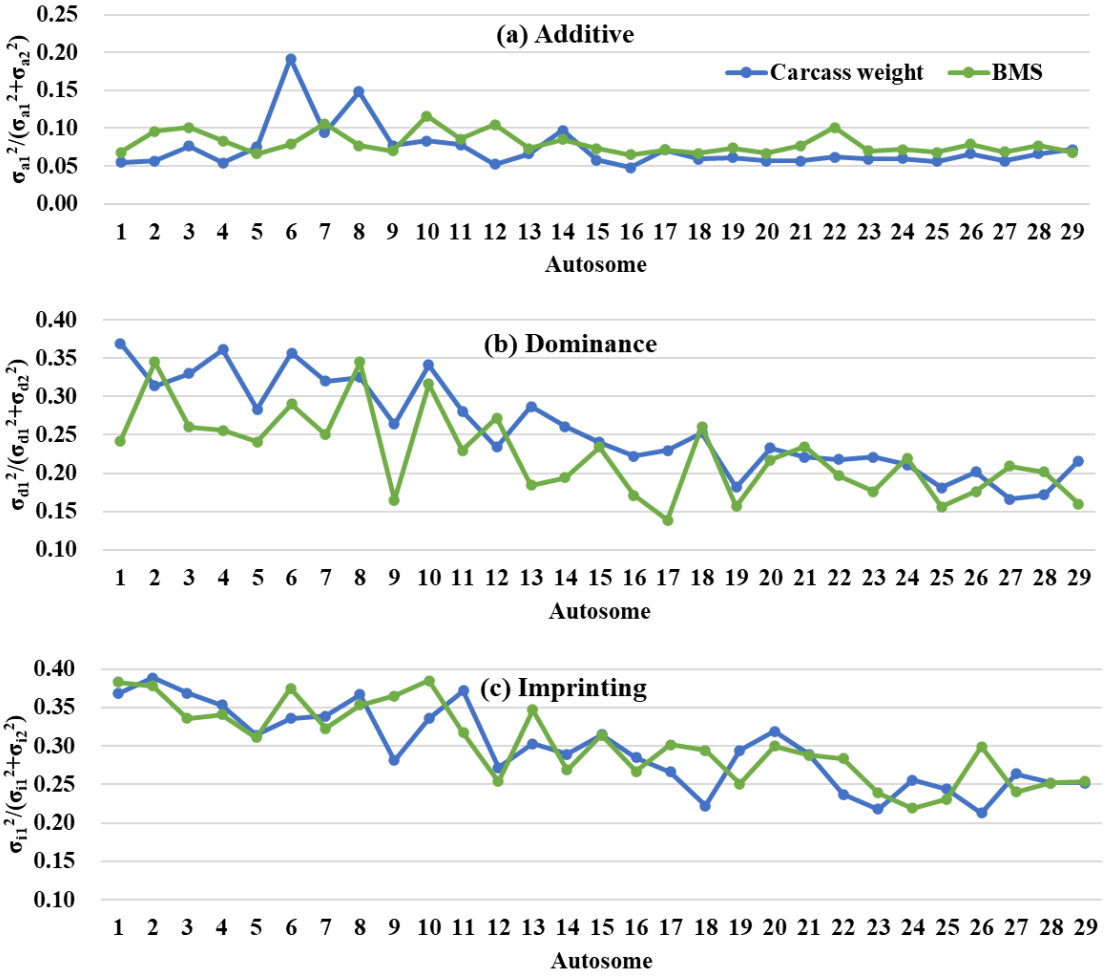

Figure 1. Proportion of additive (a), dominance (b) and imprinting (c) genetic variances explained by each autosome to the correspond total genetic variances (a: $\sigma_{a1}^2/(\sigma_{a1}^2+\sigma_{a2}^2)$, b: $\sigma_{d1}^2/(\sigma_{d1}^2+\sigma_{d2}^2)$ and c: $\sigma_{i1}^2/(\sigma_{i1}^2+\sigma_{i2}^2)$) obtained by using Model 2.

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