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Estimation of genetic parameters for growth performance and carcass traits in Mukota pigs

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The objective of the study was to determine genetic parameters for growth and carcass traits in Mukota pigs, maintained on a fibrous diet. Records (n = 1961) were obtained from a population housed at the University of Zimbabwe Farm (Harare, Zimbabwe) between January 1998 and August 2003. Backfat thickness was measured at 50 and 75 mm (K5 and K7.5), respectively. Carcass length (CL) was measured from the anterior edge of the first rib to the pubic bone using a measuring tape. Variance components were estimated using a model that accounted for direct, common environmental litter and maternal genetic effects, using average information restricted maximum likelihood. Heritability estimates for average daily gain from birth to weaning (ADGW) and average daily gain from weaning to 12 weeks (ADG1) were 0.15 and 0.27, respectively. Maternal genetic effects accounted for 2.6% of variation for ADG1. Heritability for average daily gain from 12 weeks to slaughter (ADG2) was 0.20. Common environmental litter effects accounted for 18% of phenotypic variance for cold dressed mass (CDM). Heritability estimates for CDM and CL were 0.32 and 0.62, respectively. Maternal genetic effects accounted for 10.5% of variance in CL. Heritability estimates for K5 and K7.5 were 0.64 and 0.40, respectively. The CDM was positively genetically correlated to K5, but negative to K7.5. The K5 and K7.5 had a high genetic correlation (0.88). Genetic correlations between ADGW and K5, K7.5 and CL were 0.30, 0.05 and 0.35, respectively. The existence of sufficient genetic variation makes genetic improvement for many growth and carcass traits in the Mukota breed possible through effective selection methods.

Keywords: carcass composition, genetic parameters, growth, pigs

Introduction

Sustainable conservation of animal genetic resources requires a thorough characterisation of the attributes and possible uses for a breed and development of niche markets for their products (Anderson, 2003). Local or indigenous pigs are scattered in many countries of southern Africa. Although the Mukota indigenous pigs are adapted to the local environment, their number are declining largely due to livestock production policies that prefer the use of fast-growing imported breeds. Local pigs grow slowly, with average daily gains less than 250 g/day (Kanengoni *et al.*, 2004). It has been demonstrated that Mukota pigs are able to utilise fibrous feeds better than European breeds (Kanengoni *et al.*, 2002; Ndindana *et al.*, 2002). It has also been established that indigenous pigs are better able to utilise high-tannin red sorghum than Large White pigs (Mushandu *et al.*, 2005) which is important in areas that receive low amounts of rainfall. In addition, growing Mukota pigs have been shown to be less susceptible to *Ascaris suum* (Zanga *et al.*, 2003). In addition, meat from Mukota pigs has been described as being organoleptically more acceptable to the rural people than meat from European breeds (Ndiweni and Dzama, 1995), although the reasons are not clear. Fat deposition in the Mukota is largely subcutaneous (Holness, 1991; Kanengoni *et al.*, 2004) and can easily be trimmed off and used as lard for cooking purposes. No known organised selection programmes are currently being carried out on indigenous pigs. Development of sound genetic improvement programmes based on indigenous pigs requires information on genetic parameters for traits of economic importance. In literature, there are few, if any, reports on genetic parameters for growth and carcass traits in indigenous Mukota pig populations in southern Africa. Chimonyo *et al.* (2006) reported genetic parameters of birth weight, litter weight and litter size in the Mukota pigs.

Several authors have incorporated maternal genetic effects in models for evaluating genetic merit of pigs for

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growth and carcass traits (e.g. Roehe, 1999; Kaufmann *et al.*, 2000; Chen *et al.*, 2002). Maternal effects are any maternal contributions that affect the phenotypic expression of offspring, excluding direct additive genes. Intrauterine environment, milk production, and care-taking are all maternal components that may be genetically and environmentally determined. Although maternal effects have long been established in reproduction traits, it is now generally agreed that they also account for a significant portion of variance for traits that are manifested relatively late in life such as post-weaning growth and carcass backfat (Solanes *et al.*, 2004). For example, Bryner *et al.* (1992) observed significant maternal genetic effects explaining 23% and 11% of phenotypic variation for growth rate and backfat thickness, respectively. Many studies have also reported negative genetic correlations between direct and maternal genetic effects (Ferraz and Johnson, 1993; Crump *et al.*, 1997; Chen *et al.*, 2002). Both maternal effects and genetic correlations should, therefore, be accounted for in developing models for genetic evaluation of pigs. The objective of the study was to determine genetic parameters for growth and carcass traits in Mukota pigs, including maternal genetic effects.

Material and methods

Animals

The Mukota pigs were housed at the University of Zimbabwe Farm, Harare, Zimbabwe, which is situated at 18°N and 30°E. The altitude is approximately 1300 metres above sea level and annual rainfall averages 800 mm. The pigs studied were part of a satellite population developed at the University of Zimbabwe Farm for research and breed characterisation. This was prompted by a significant decline in the population of Mukota pigs, which threatens loss of indigenous pig genes, largely through indiscriminate crossbreeding in the communal areas.

Four unrelated Mukota boars and 16 Mukota gilts were purchased from the Mutoko Communal Area, nearly 250 km to the north east of Harare, Zimbabwe, in October 1997, to develop a satellite population at the University of Zimbabwe Farm for genetic characterisation. All purchased pigs were deemed pure, based on phenotypic appearance and pedigree, as provided by the farmers, who were asked whether any form of crossbreeding with European breeds had occurred in the previous five generations. To increase diversity and broaden the genetic base, another six Mukota sows were purchased in 1998, from Mvuma, about 200 km south of Harare, and in 1999, five Mukota boars and five gilts were obtained from Mount Darwin, about 200 km north of Harare. Five Mukota boars were also selected from the herd and mated to gilts produced from sows that had been purchased from other farms to reduce inbreeding within the experimental population. Females were mated by natural service, in a way that avoided mating of close relatives. No more than one gilt was selected from the same litter. Sows were culled after eight parities. The population was maintained for five generations. More details on the structure of pig population and their management were described in Chimonyo *et al.* (2006).

Traits

Data from Mukota pigs farrowed from 53 sows between January 1998 and August 2003 were used. A total of 506 pigs with missing records were deleted, leaving 1961 records for analysis. Weight related traits analysed were average daily gain from birth to weaning (ADGW), average daily gain from weaning to 12 weeks (ADG1; g/day), average daily gain from 12 weeks to slaughter (ADG2; g/day) and weight of the dressed carcass after chilling at 4°C for 24 h (CDM). All weights were estimated using a portable scale (Kattleway, Marondera, Zimbabwe). Thickness of backfat was measured on the carcass at two points along the last rib at 50 and 75 mm from the mid line, and denoted as K5 and K7.5, respectively. Carcass length (CL) was measured from the anterior edge of the first rib to the pubic bone using a measuring tape.

Statistical analyses

The GLM procedure of the Statistical Analysis Systems Institute (2000) was used to test the significance of fixed effects, covariates (linear, quadratic and cubic) and any possible interactions among factors (to include in the mixed animal model). The fixed effects of sex of the pig, month of farrowing and parity of sow were significant ($P < 0.05$) for ADG1, ADG2, CDM, CL and both backfat measurements. None of the quadratic and cubic covariates was significant ($P > 0.05$). Weight at weaning was incorporated as a linear covariate for ADG1. The CDM, CL and backfat measurements were adjusted for the weight of pig at slaughter. All traits measured were assumed to be normally distributed. Variance-covariance components were estimated using AIREML (Gilmour *et al.*, 1995) using an animal model. The model used accounted for random direct genetic, common environmental litter and maternal genetic effects. In matrix form, the model is:

$$\mathbf{Y} = \mathbf{X}\beta + \mathbf{Z}_1\mathbf{u}_a + \mathbf{Z}_2\mathbf{u}_l + \mathbf{Z}_3\mathbf{u}_m + e;$$

where $\mathbf{Y}$ is a vector of n observations, β is a vector of the fixed effects of year of farrowing (6 years), month of farrowing (12 months per year), parity (eight parities) and sex (male and female). All fixed factors included in the model were significant ($P > 0.05$) based on the GLM analysis. Parity was incorporated in all models, since it was found to be significant in influencing post−weaning growth performance. Vectors of random effects included environmental effects $\mathbf{u}_l \sim N_{q1}(\mathbf{0}, \mathbf{I}\sigma_l^2)$ that were common to pigs within each of q_l litters, direct $\mathbf{u}_a$ and maternal genetic effects $\mathbf{u}_m$ of q_2 pigs, and residual environmental effects $e \sim N_n(\mathbf{0}, \mathbf{I}\sigma_e^2)$. All random effects are assumed to be sampled from a normal distribution with a zero mean and variance-covariance structure of:

$$v\begin{bmatrix} u_l \\ u_a \\ u_m \\ u_e \end{bmatrix} = \begin{bmatrix} I\sigma_l^2 & 0 & 0 & 0 \\ 0 & A\sigma_a^2 & A\sigma_{\mathrm{am}} & 0 \\ 0 & A\sigma_{\mathrm{am}} & A\sigma_m^2 & 0 \\ 0 & 0 & 0 & I\sigma_e^2 \end{bmatrix}$$

The implied genetic models are infinitesimal (Bulmer, 1980), where the base population is assumed to be unselected, non–inbred and unrelated. Vectors of the direct and maternal genetic effects were assumed to be distributed as:

$$\begin{bmatrix} \overset{\sim}{u_a} \\ u_m \end{bmatrix} \mathrm{N}(\mathbf{0}, \mathbf{G}_{\mathrm{am}} \otimes \mathbf{A}), \text{ where } \mathbf{G}_{\mathrm{am}} = \begin{bmatrix} \sigma_a^2 & \sigma_{\mathrm{am}} \\ \sigma_{\mathrm{am}} & \sigma_m^2 \end{bmatrix}$$

where **I** and **A** are the identity and numerator relationship matrices, respectively. The $\mathbf{G}_{\mathrm{am}}$ is the genetic (co) variance matrix between direct and maternal effects and ⊗ denotes the direct product. Permanent environmental effects were assumed to be uncorrelated with direct additive genetic effects. Incidence matrices **X** and $\mathbf{Z}_i$ (i = 1, 2, 3) link the fixed and random effects, respectively, with corresponding records in vector **Y**.

Genetic and phenotypic correlations were estimated using a multi–trait model. All traits were analysed together in one model.

Results

Summary statistics and levels of significance for the fixed factors

Table 1 shows summary statistics for ADGW, post–weaning growth performance and carcass traits. Levels of significance for the fixed factors are in Table 2. Sex of pig affected ($P < 0.05$) ADG2 but not ADG1 ($P > 0.05$). Weight at weaning, incorporated as a covariate, significantly affected ($P < 0.05$) ADG1. There was a significant interaction ($P < 0.05$) between sex of pig and month on backfat thickness.

Table 1 *Summary statistics for growth and carcass traits of Mukota pigs (n = 1961) raised in a common herd in Zimbabwe*[†]

Trait	Mean	s.d.	Minimum	Maximum
ADGW (g)	177.2	112.3	80.6	261.5
ADG1 (g)	321.1	107.06	114.0	544.1
ADG2 (g)	383.8	157.35	153.6	594.3
CDM (kg)	21.8	4.23	12.1	37.8
K5 (mm)	11.4	1.69	6.0	20.4
K7.5 (mm)	14.9	1.79	6.0	23.0
CL (mm)	508.1	43.28	402	621

[†] Abbreviations are: ADGW = average daily gain from birth to weaning; ADG1 = ADG from weaning to 12 weeks of age; ADG2 = ADG from 12 weeks of age to slaughter; CDM = cold dressed mass, measured after 24 h at 4°C; CL: carcass length measured from the anterior edge of the first rib to the pubic bone; K5 = backfat at 50 mm from the mid line along the last rib; K7.5 = backfat thickness at 75 mm from the mid line along the last rib.

Heritability estimates

Table 3 shows the genetic parameter estimates for ADGW, ADG1 and ADG2. The direct and maternal genetic effects for ADGW were 0.15 ± 0.005 and 0.20 ± 0.005, respectively. The direct heritability for ADG1 was 0.27 ± 0.003. Maternal genetic effects accounted for 3% (±0.3%) of phenotypic variance. The genetic correlation between direct and maternal genetic effects was significantly different from zero at −0.417 ± 0.071. The litter effect for ADG2 was low and the direct heritability was 0.20 ± 0.001. Maternal genetic effects accounted for 3.1% of the phenotypic variance. For ADG1, there was a negative correlation between direct and maternal genetic effects.

Common environmental litter effects for CDM accounted for 18% of the phenotypic variation (Table 3). The heritability estimate was 0.32 ± 0.002. Maternal genetic effects accounted for 2.7% of variance, and there was a negative correlation between direct and maternal genetic effects ($r_{am} = -0.281 \pm 0.047$). The heritability estimate for CL was 0.62 ± 0.001. Litter effects and maternal genetic effects accounted for only 4 and 10.5% of the phenotypic variance, respectively. The genetic correlation between direct and maternal effects for CDM was negative ($r_{am} = -0.482 \pm 0.009$).

Common environmental litter effects were marginal for both K5 and K7.5 measurements (Table 3). The heritability estimate for K5 was 0.64 ± 0.001 and the maternal genetic influence was 4%. The heritability for K7.5 was 0.40 ± 0.001.

Genetic and phenotypic correlations

Estimates of genetic and phenotypic correlations are in Table 4. There was a significant ($P < 0.05$) genetic correlation between pre–weaning growth rate and ADG1. The genetic correlation between ADGW and ADG2 was, however, negative. The ADG1 and ADG2 were positively correlated, both phenotypically ($r_p = 0.32 \pm 0.002$) and genetically ($r_g = 0.35 \pm 0.015$). Standard errors for genetic correlations for growth traits tended to be larger than for their phenotypic counterparts.

The CDM had a positive genetic correlation with the K5 backfat measurement ($r_g = 0.23 \pm 0.048$). The phenotypic correlation was also positive. Although CDM was phenotypically positively correlated with K7.5 ($r_p = 0.23 \pm 0.026$), the genetic correlation was negative ($r_g = -0.34 \pm 0.013$). Both the genetic and phenotypic correlations between CDM and CL were positive and relatively large ($r_g = 0.53 \pm 0.046$; $r_p = 0.66 \pm 0.017$). The correlations between CL and both backfat measurements were positive. Genetic correlations between these traits were higher than their phenotypic counterparts. The two backfat measurements were highly correlated ($r_g = 0.88 \pm 0.026$; $r_p = 0.79 \pm 0.009$).

Table 2 *Levels of significance for the fixed factors the analysis of Mukota pigs reared in a common herd*[†]

	Fixed factors						Covariates			
Trait	S	M	P	P × M	S × M	P × S × M	β_1	β_2	β_3	β_4
ADGW (g)	‡	‡	‡	‡	‡	‡	*	§	§	§
ADG1 (g/day)	‡	*	*	*	**	*	*	§	*	§
ADG2 (g/day)	**	‡	‡	*	**	*	‡	§	‡	§
CDM (kg)	**	‡	‡	*	*	*	‡	§	‡	*
CL (mm)	‡	‡	‡	*	‡	§	§	§	§	‡
K5 (mm)	‡	*	‡	*	**	§	§	§	§	‡
K7.5 (mm)	‡	*	‡	*	*	§	§	§	§	‡

[†] Abbreviations are: S = sex of pig; M = month of farrowing; P = parity of sow; ADGW = average daily gain from birth to weaning; ADG1 = ADG from weaning to 12 weeks of age; ADG2 = ADG from 12 weeks of age to slaughter; CDM = cold dressed mass; measured after 24 h at 4°C; CL = carcass length measured from the anterior edge of the first rib to the pubic bone; K5 = backfat at 50 mm from the mid line along the last rib; K7.5 = backfat thickness at 75 mm from the mid line along the last rib; β_1 = individual weight at birth; β_2 = average litter weight at birth; β_3 = weight at weaning; β_4 = weight at slaughter.
[‡] Not significant ($P > 0.05$).
[§] Not included in the model.

Phenotypic correlations between ADGW and carcass traits were weak and were not different from zero (Table 4). The magnitude of the genetic correlation between ADGW and K5 was larger than the correlation between ADGW and K7.5 (0.30 ± 0.028 versus 0.05 ± 0.021). The genetic correlation between CL and ADGW was positive ($r_g = 0.35 \pm 0.085$). The phenotypic correlations between ADG1 and backfat thickness measurements were positive, whereas the genetic correlations were negative. Both genetic and phenotypic correlations between ADG2 and backfat measurements (both K5 and K7.5) were positive. The phenotypic correlations were, in both cases, higher than genetic correlations. Both ADG1 and ADG2 were positively correlated to CDM; the phenotypic correlations being stronger than their genetic counterparts.

Discussion

Mukota pigs, which have been demonstrated to adapt to survive under rural low input production systems, have been shown to exhibit relatively low growth rates (Kanengoni *et al.*, 2004). The low growth rates, however, could be an advantage in that they do not require large amounts of concentrate feeds. This study aimed at estimating the genetic contribution to growth rate and carcass traits in these pigs. Heritability estimates obtained in this study were similar to those reported for Australian pigs (Hermesch *et al.*, 2000a).

The observed common environmental litter effects obtained in this study, which represent a non−genetic likeness between sibs in the same litter, were lower than literature values (Hermesch *et al.*, 2000b; Peskovicova *et al.*, 2002). Peskovicova *et al.* (2002) also reported differences in the heritability estimates based on−farm test records *versus* on−station record. The estimates for these two production systems in the Czech Republic were 0.18 and 0.36, respectively. These heritability estimates are comparable to those obtained for ADG2 in the present study.

Litter effects obtained in this study were lower than those reported in literature. Ferraz and Johnson (1993) reported that between 5 and 7% of the variation in backfat and ADG were due to common environmental effects in Landrace and Large White pigs. Chen *et al.* (2002) reported estimates of 8 to 12% for backfat across breeds. Crump *et al.* (1997) reported estimates of 5 and 6% in backfat and ADG, respectively, while Johnson *et al.* (1999) reported

Table 3 *Estimates of the proportion of phenotypic variance explained by direct genetic* (h^2), *maternal genetic* (m^2) *and common environmental litter effects* (l^2) *of the correlation between direct and maternal genetic effects* (r_{am}) *and of the phenotypic variance* $\left(\sigma_p^2\right)$ (± s.e.) *for traits analysed in a population of African Mukota pigs reared in a common herd*[†]

	h^2	m^2	l^2	r_{am}	σ_p^2
ADGW (g)	0.15 ± 0.005	0.20 ± 0.005	0.03 ± 0.006	− 0.35 ± 0.361	12 612
ADG1 (g)	0.27 ± 0.003	0.03 ± 0.003	0.03 ± 0.003	− 0.42 ± 0.071	11 471
ADG2 (g)	0.20 ± 0.001	0.03 ± 0.001	0.03 ± 0.001	− 0.50 ± 0.026	24 688
CDM (kg)	0.32 ± 0.002	0.03 ± 0.002	0.18 ± 0.002	− 0.28 ± 0.047	17.97
K5 (mm)	0.64 ± 0.001	0.04 ± 0.001	0.01 ± 0.001	− 0.31 ± 0.007	2.91
K7.5 (mm)	0.40 ± 0.001	0.03 ± 0.001	0.01 ± 0.001	− 0.25 ± 0.005	2.72
CL (mm)	0.62 ± 0.001	0.11 ± 0.001	0.04 ± 0.001	− 0.48 ± 0.009	1858

[†] Abbreviations are: S = sex of pig; M = month of farrowing; P = parity of sow; ADGW = average daily gain from birth to weaning; ADG1 = ADG from weaning to 12 weeks of age; ADG2 = ADG from 12 weeks of age to slaughter; CDM = cold dressed mass; measured after 24 h at 4°C; CL = carcass length measured from the anterior edge of the first rib to the pubic bone; K5 = backfat at 50 mm from the mid line along the last rib; K7.5 = backfat thickness at 75 mm from the mid line along the last rib.

Table 4 *Estimates of genetic (above diagonal) and phenotypic correlations (below diagonal) among pre-weaning growth performance, post-weaning growth rate and carcass traits from a multi-trait analysis of data from a population of African Mukota pigs reared in a common herd*[†]

	Trait						
Trait	ADGW	ADG1	ADG2	CDM	K5	K7.5	CL
ADGW		0.58 ± 0.121	−0.29 ± 0.124	0.08 ± 0.005	0.30 ± 0.028	0.05 ± 0.021	0.35 ± 0.085
ADG1	0.04 ± 0.026		0.35 ± 0.015	0.45 ± 0.001	−0.21 ± 0.049	−0.32 ± 0.014	0.55 ± 0.043
ADG2	−0.21 ± 0.026	0.32 ± 0.002		0.68 ± 0.018	0.29 ± 0.053	0.34 ± 0.013	0.47 ± 0.060
CDM	0.01 ± 0.003	0.62 ± 0.001	0.74 ± 0.002		0.23 ± 0.048	−0.34 ± 0.013	0.53 ± 0.046
K5	0.03 ± 0.026	0.35 ± 0.024	0.34 ± 0.024	0.35 ± 0.024		0.88 ± 0.026	0.41 ± 0.022
K7.5	−0.01 ± 0.028	0.23 ± 0.026	0.23 ± 0.026	0.23 ± 0.026	0.79 ± 0.009		0.43 ± 0.002
CL	0.04 ± 0.032	0.67 ± 0.017	0.65 ± 0.017	0.66 ± 0.017	0.35 ± 0.022	0.26 ± 0.024	

[†] Abbreviations are: ADGW = average daily gain from birth to weaning; ADG1 = ADG from weaning to 12 weeks of age; ADG2 = ADG from 12 weeks of age to slaughter; CDM = cold dressed mass, measured after 24 h at 4°C; CL = carcass length measured from the anterior edge of the first rib to the pubic bone; K5 = backfat at 50 mm from the mid line along the last rib; K7.5 = backfat thickness at 75 mm from the mid line along the last rib.

litter effects of 13% in Large White boars. Although reports on estimates for common environmental litter effects for CDM are scarce, these effects were quite substantial in the current study.

Additive maternal genetic effects contributed 4% of the phenotypic variation for K5, which is in the same range as those reported for lean growth rate by Chen *et al.* (2002). The maternal genetic effect contribution to ADG was similar to findings of Chen *et al.* (2002). The negative genetic correlation between direct and maternal genetic effects is consistent with previous literature findings (Ferraz and Johnson, 1993; Crump *et al.*, 1997; Chen *et al.*, 2002). Ignoring maternal genetic effects, as is common in several models used before (e.g. Fernández *et al.*, 2003), has the consequence of producing biased heritability estimates with wide standard errors or reduced precision (Solanes *et al.*, 2004). The heritability of CL was 0.53, which agrees with earlier reports (Lopez−Serrano *et al.*, 2000). The high maternal influence on CL was unexpected, and difficult to explain. It is possible that there was some degree of confounding between litter and maternal effects, since cross fostering was rare and pigs of the same litter would be raised together until slaughter. When litter or maternal effects were removed from the model, no marked differences in estimates of heritability and their standard errors were detected. However, the model that contain direct, litter and maternal genetic effects had lower residual error variances than models containing direct and litter effects only and was, therefore, used. There is, however, a need to obtain additional estimates from large data sets that are better designed to separate these effects.

Backfat thickness in large−framed pigs is usually measured at the P2 position, a position 65 mm from the mid line along the last rib. Heritability at the P2 position seems to correspond well with the K5 position in Mukota pigs. The heritability was higher for K5 than for K7.5, suggesting that, when selection intensity and accuracy are the same, more gain in leanness is likely to be achieved for selecting Mukota pigs using the former. Chen *et al.* (2002) reported a heritability of 0.48 for Yorkshire, Duroc, Hampshire and Landrace breeds, while Peskovicova *et al.* (2002) also reported a similar value in the Czech Landrace. Kanengoni *et al.* (2004) also indicated that the positions which are used for assessing carcass grades in large−framed pigs seem inappropriate for small−framed pigs, such as the Mukota. Such traits with economic value should, therefore, be included in selection programmes.

That both genetic and phenotypic correlation coefficients were of the same sign and magnitude suggests that the genetically and environmentally (residual) correlations are likely to share the same pattern (Roff, 1996). Residual correlations observed in this study were low to moderate in comparison to the genetic correlations, which agree with De Vries *et al.* (1994) and Kemm *et al.* (1995).

Genetic correlations among backfat measurements were lower than those reported for Australian pigs (Hermesch *et al.*, 2000b). When genetic correlations are not significantly different from unity, it suggests that the two traits are under the influence of the same genes. In other words, selection for improvement in one trait will result in improvement in the other trait at relatively the same rate of genetic change (Kyriazakis and Whittemore, 2006). The high genetic correlation between K5 and K7.5, therefore, indicates that either position could be included in a selection programme designed to reduce backfat in the Mukota. The negative genetic correlation between CDM and K7.5 is difficult to explain. The negative genetic correlation between ADG1 and backfat measurements agrees with Hermesch *et al.* (2000b), where pigs were fed *ad libitum*. The genetic correlations obtained in this study agree with literature values that were based on *ad libitum* feeding systems (Lo *et al.*, 1992; Ducos *et al.*, 1993; Mrode and Kennedy, 1993; Cameron and Curran, 1994).

The positive genetic correlation between backfat thickness and ADG2 is consistent with literature (Falconer, 1981; Li and Kennedy, 1994; Hermesch *et al.*, 2000c; Kyriazakis and Whittemore, 2006). These researchers observed a positive genetic relationship between growth rate during the last 4 weeks to slaughter with backfat thickness. The genetic correlation between growth rate and leanness has been shown to be more favourable in young pigs, when feed intake capacity is limited (Campbell *et al.*, 1986; McPhee *et al.*, 1988; Von Felde *et al.*, 1996). The unfavourable positive genetic correlation between ADG2 and backfat is, therefore, an indication that lean deposition in pigs at that age is increasing at a decreasing rate, whereas fat deposition is increasing at an increasing rate or that the pigs are physiologically more mature (Holness, 1991; Kyriazakis and Whittemore, 2006). In such cases, the extra energy consumed is deposited as fat tissue. Von Felde *et al.* (1996) also reported that feed intake at the beginning of the growing period had a more favourable genetic correlation with leanness than feed intake over the entire growing period. Since pigs were fed in groups, it was not economically and physically possible to track feed consumption for each pig. It was, therefore, not possible to determine genetic relationships of growth rate and feed conversion efficiency.

For traits for which the genetic correlation between maternal and direct effects is negative, methods of selection accounting for both components would result in greater economic response to long−term selection than selection based only on direct animal effects. The maternal genetic effects for ADGW and ADG1 were 0.20 and 0.03, respectively. The genetic correlation between direct and maternal genetic effects for ADGW, ADG1 and ADG2 were negative. Common environmental litter effects for these traits were low. Heritability estimates for carcass traits were high, while their maternal genetic effects were small, except for CL. The CDM was positively correlated to K5 ($r_g = 0.23$), but negatively correlated to K7.5 ($r_g = -0.34$).

In conclusion, the existence of sufficient genetic variation makes the genetic improvement for many growth and carcass traits in the Mukota breed possible through effective selection methods.

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