Genetic Parameter Estimates for Egg and Body Weights of Indigenous Chicken in Kenya

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Introduction

The importance of indigenous chicken (IC) production in African countries has been well documented (Guèye, 2000). However, meat and egg production of IC are low compared to commercial chicken breeds and hybrids. However, in Kenya no improvement attempt has ever focused on selection yet selection strategies have been used to improve growth and egg production of IC in Iran (Kamali et al. 2007). Improvement through selection requires estimates of genetic parameters. The objective of this study was to obtain heritability and correlation (genetic and phenotypic) estimates of egg weight and body weights at various ages.

Material and methods

Data source. The study was conducted at the National Animal Husbandry Research Centre (NAHRC), Naivasha. Thirty one cocks were randomly sampled from a random mating flock collected from various ecological regions of the country. Each cock was allocated between 5 and 10 unrelated and randomly sampled hens and housed together in deep litter pens. Each egg collected was labelled to identify its cock family and stored for at most 14 days before incubation. After the 2nd candling, each egg was placed into an individual compartment for hatching. At hatch, chicks were wing tagged and reared in electric brooders up to 6 weeks and in deep litter pens thereafter. Sex was determined by physiological appearance on the 8th and 14th weeks of age. Standard commercial feeds and clean water were supplied *ad libitum* at all ages. Egg weight (EW) and body weights at hatch (BW0), 2 (BW2), 4 (BW4), 6 (BW6), 12 (BW12), 14 (BW14), 16 (BW16) and 26 (BW26) weeks of age were measured using a digital weighing scale.

Statistical analyses. Significant fixed effects and covariates were determined using the general linear model procedures (SAS 1998). (Co)variance components were simultaneously estimated using the DFREML programme (Meyer 1998) fitting a multiple trait sire model. In the analysis of EW, hatch (1, …, 7) was the only fixed effect fitted while for subsequent

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body weights an additional fixed effect of sex was added. BW0 was fitted as a covariate in the analysis of BW2 to BW6. Heritability estimates (h^2) were calculated as:

$$h^2 = 4*s^2_s / s^2_p$$

where s^2_s and s^2_p are the sire and phenotypic variances, respectively. The genetic and phenotypic correlations were estimated as:

$$r_{y(1,2)} = s_{s(1,2)} / v(s^2_{y(1)}\, s^2_{y(2)})$$

where $r_{y(1,2)}$ is the genetic or phenotypic correlation between trait 1 and 2, $s_{s(1,2)}$ is the sire covariance between trait 1 and 2 and $s^2_{y(1)}$ and $s^2_{y(2)}$ are the sire or phenotypic variances of trait 1 and 2, respectively

Results and discussion

The genetic parameter estimates are presented in Table 1. The heritability estimate for EW (0.98) was comparable to 0.91 reported by Poggenpoel et al. (1996) and Magothe et al. (2006). However, it was higher than the range of 0.26 to 0.68 reported by Nurgiartiningsih et al. (2004) and Kamali et al. (2007). Although EW is highly heritable, the unselected population in this study is expected to exhibit high additive genetic variation hence the high heritability estimate. However, non-additive, maternal additive and maternal environmental effects also highly contribute to EW variation (Bunter and Cloete 2004). Maternal additive and environmental effects were not accounted for and therefore may have been confounded with the sire variance, leading to overestimation of the heritability.

The heritability estimates for body weights were within the ranges reported in literature (Chambers 1990). However, the estimates increased from 0.51 for BW2 to 0.65 for BW4 and thereafter reduced gradually to 0.11 for BW26. The increase in heritability from week 2 to week 4 and subsequent decrease thereafter may be attributed to increased growth promoters and inhibitors gene actions, respectively (Baéza et al. 2001; Geraert 2004). However, the decreasing estimates may also be explained by increases in phenotypic and residual variances as expected in time series growth data. Selection for BW12 has been used to improve growth and laying traits of native chickens in Iran (Kamali et al. 2007).

Table 1: Estimates of genetic parameters[a]

	EW	BW2	BW4	BW6	BW12	BW14	BW16	BW26
EW	**0.98**	-0.13	-0.14	-0.15	0.05	0.17	0.25	0.58
BW2	-0.02	**0.51**	0.99	0.99	0.89	0.84	0.78	0.52
BW4	-0.03	0.14	**0.65**	0.99	0.85	0.80	0.74	0.47
BW6	-0.03	0.13	0.14	**0.51**	0.83	0.78	0.72	0.44
BW12	0.01	0.09	0.10	0.09	**0.35**	0.99	0.97	0.80
BW14	0.02	0.08	0.09	0.08	0.08	**0.31**	0.99	0.87
BW16	0.03	0.08	0.08	0.07	0.08	0.08	**0.29**	0.92
BW26	0.05	0.03	0.03	0.03	0.04	0.04	0.04	**0.11**

[a] Heritabilities on the diagonal, genetic and phenotypic correlations above and below diagonal, respectively.

Whereas all phenotypic correlations were low (0.01 to 0.14), genetic correlations ranged from low (0.05) to near unity (0.99). Magothe et al. (2006) reported low phenotypic correlations similar to the current study. However, medium to high correlations have been reported (Lwelamira et al. 2009). Phenotypic correlations of less than unity are indicators of genotype by environment (GxE) interactions and the lower the correlations, the higher the interactions (Settar et al. 1999). The population in this study comprised of various genotypes with differing growth patterns as reported by Magothe et al. (2010). Both phenotypic and genetic correlations between EW and BW2, BW4 and BW6 were negative. This may be due to the confounding effects of maternal influences on EW as indicated earlier. In addition, fitting BW0 as a covariate for juvenile weights appears to have indirectly accounted for some maternal effects as suggested by Bunter and Cloete (2004). Therefore, the genetic correlations between EW and juvenile body weights in this study may, to a larger extend be indicating correlations between EW maternal additive effects and juvenile body weights direct additive effects. Comparable negative genetic correlations between maternal and direct additive genetic effects in chickens have been reported (Prado-Gonzalez et al. 2003; Norris and Ngambi 2006). Furthermore, the positive and increasing genetic correlation between EW and body weights at 12, 14, 16and 26 weeks of age appear to support this argument. In addition, the genetic correlations between body weights at various ages were all positive and decreased with age from near unity to moderate. These estimates and their decrease with age agree with reports on turkeys and chicken (Kranis et al. 2006; Lwelamira et al. 2009).

Conclusion

From the parameters obtained, it is concluded that juvenile, growing and mature body weights of IC in Kenya can be improved through selection. The moderate heritability coefficient of BW12, lack of maternal influences and high genetic correlation with both juvenile and mature body weights suggests this to be a possible selection point. However, since one of the positive attributes of IC is their adaptation to harsh environmental conditions, it may also be important to consider GxE interactions and inclusion of fitness traits in a breeding programme for their improvement.

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References

Baéza, E., Williams, J., Guémené, D. et al. (2001). *Reprod. Nutr. Dev.,* 41: 173–179.

Bunter, K.L. and Cloete, S.W.P. (2004). *Livest. Prod. Sci*., 91: 9–22.

Chambers, J.R. (1990). Elsevier, Amsterdam, The Netherlands, Pages 559–643.

Geraert, E. (2004). *Int. J. Dev. Biol.,* 48: 271–274.

Guèye, E.F. (2000). *Outlook Agric*., 29: 129–136.

Kamali, M.A., Ghorbani, S.H., Sharbabak, M.M. *et al.* (2007). *Brit. Poult. Sci.,* 48: 443–448.

Kranis, A., Hocking, P.M., Hill, W.G. *et al.* (2006). *Brit. Poult. Sci.,* 47: 685–693.

Lwelamira, J., Kifaro, G.C. and Gwakisa, P.S. (2009). *Trop. Anim. Health and Prod.,* 41: 51–59.

Magothe, T.M., Muhuyi, W.B. and Kahi, A.K. (2006). In *Proc 8th WCGALP, Belo Horizonte, Minas Gerais, Brazil, 13-18 August.*

Magothe, T.M., Muhuyi, W.B. and Kahi, A.K. (2010). *Trop. Anim. Health and Prod.,* 42: 173–183.

Meyer, K. (1998). *DFREML user's manual,* University of New England, Armidale, Australia.

Norris, D. and Ngambi, J.W. (2006). *Trop. Anim. Health and Prod.,* 38: 605–609.

Nurgiartiningsih, V.M.A., Mielenz, N., Preisinger, R. et al. (2004). *Brit. Poult. Sci.,* 45: 604–610.

Poggenpoel, D.G., Ferreira, G.F., Hayes, J.P. *et al.* (1996). *Brit. Poult. Sci*., 37: 743–756.

Prado-Gonzalez, E.A., Ramirez-Avila, L. and Segura-Correa, J.C. (2003). *Livest. Res. for Rural Dev*., 15.

SAS. (1998). *User's Guide, Version 6.12*. SAS Institute Inc., Cary, NC.

Settar, P., Yalcin, S., Turkmut, L. *et al.* (1999). *Poult. Sci.,* 78: 1353–1358.