Livestock Science 229 (2019) 56–63

Contents lists available at ScienceDirect

Livestock Science

journal homepage: www.elsevier.com/locate/livsci

Reaction norm models for the study of genotype × methionine + cystine level interaction in meat-type quail

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ARTICLE INFO

Keywords:
Genetic evaluation
Genetic parameter
Reaction norm model

ABSTRACT

The objective of this study was to use reaction norm models to study genotype × environment interactions of meat-type quails raised on different levels of digestible methionine + cystine. The weight gains of two meat-type quail strains (EV1 and EV2) were measured from 21 to 35 days of age. For EV1 strain the levels of digestible methionine + cystine were 0.60, 0.70, 0.80, 0.90 and 1.00%, for EV2 strain the levels were 0.61, 0.71, 0.81, 0.91 and 1.01%. Reaction norm models with Legendre polynomials (LP) varying from zero to four orders were tested, by using Bayesian methodology. The model of best fit was selected based on the criterions of Deviance Information and Posterior Model Probabilities (PMP). For the EV1 strain, the fourth-order LP model was the best fit for the weight gain description with different levels of digestible methionine + cystine for the mean trajectory, additive genetic effects and five classes of residual variance. For the EV2 strain, the first-order LP model was the best fit for the mean trajectory, fourth-order for additive genetic effects and five classes of residual variance. In both strains, genetic correlation estimates and reaction norm of the genotype in relation to dietary methionine + cystine indicated the existence of a genotype × dietary level of amino acids interaction. For EV1 strain, genetic correlations for weight gain in different levels of digestible methionine + cystine ranged from –0.98 to 0.84, while for EV2 strains these estimates varied from –0.98 to 0.94. Also, in general, for both strains the high density intervals of genetic correlations with 90% of samples included zero, indicating that genetic correlations between the weight gain in different levels of digestible methionine + cystine did not differed from zero. Furthermore, the reaction norms indicated that genotypes were not linearly related to tested dietary levels of amino acids. This absence of linearity implies that to verify the same response to selection in field that was predicted by the genetic evaluation, the nutritional levels used at the herds for selection candidates and their progenies must be the same.

1. Introduction

When designing breeding programs, in order to achieve genetic gain by artificial selection, it is generally assumed that the genotype of the individual will uniformly express itself, independent of the environment in which it and its progenies will be raised. However, quantitative traits present phenotypic plasticity or environmental sensibility, such that the mean phenotype from a population will change if there are changes in the environmental conditions to which it is exposed (Morgante et al., 2015).

A genotype × environment interaction occurs when a genotype presents different responses to environmental variations, and can lead to changes in both phenotype and the magnitude of additive and residual genetic variances (Calus et al., 2004; Mattar et al., 2011; Miranda et al., 2016). This interaction can be identified by fitting reaction norm models, and sensibility to environmental variation is quantified by the angular coefficient variance of the reaction norm. Thus, the regression that expresses the mean sensibility is assumed to be linear (Falconer, 1990). However, in the study of genotype sensibility to environmental variation, the assumption of linearity restricts the evaluation of the trait behavior in different environments, and thus, the capacity of certain genotypes to evolve under different environments (Gavrilets and Scheiner, 1993).

The genetic changes along an environmental gradient will depend on the extent to which a genotype can express different phenotypes depending on environmental conditions, that is, by how much and the

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https://doi.org/10.1016/j.livsci.2019.09.016
Received 28 August 2018; Received in revised form 15 July 2019; Accepted 13 September 2019
Available online 14 September 2019

manner in which the reaction norm will change along the environmental gradient (Gavrilets and Scheiner, 1993). The study of this behavior when different nutritional levels are used is important, given the relevance of the nutrition to the manifestation of satisfactory fitness of the quails, and considering that the costs with nutrition represent the most expressive cost of quail production (Griep Júnior et al., 2017). Still, regarding the different components of a diet, the methionine is the first limiting amino acid for birds in general (Kaur et al., 2008). Since in absence of this nutrient the protein synthesis is interrupted, is expected a big influence of it on the phenotypic manifestation.

Therefore, the aim of this study was to evaluate the genotype × environment interaction of the body weight gains during the final growing phase of meat-type quails fed different levels of dietary digestible methionine + cystine, using reaction norm models.

2. Material and methods

The weight gain records used in the present study were obtained from two strains of meat-type quails, which belong to the Breeding Program of Meat-type Quails, from the Universidade Federal de Minas Gerais. This experiment was approved under protocol #108/2013 of the Ethical Committee of Animal Usage. The quails (*Coturnix coturnix*) were raised in the Experimental Farm Professor Hélio Barbosa, located in Igarapé city, Minas Gerais State. A total of 65 males and 130 females of the EVI strain, and 73 males and 146 females of the EV2 strain, were mated to produce half-sibs families in a ratio of two females per male.

After birth, the quails were identified by leg-banding, weighed, and their pedigree information was recorded. Families of each strain were distributed into 10 masonry boxes, measuring 1.20-m wide × 1.50-m long × 25-cm high, with wood shavings for litter, and heating bells, which were turned on for 24 h each day, until the quails were 21-days old. From day 22 onwards, the heating bells were turned off and the birds remained under natural lighting. Progenies of each mating were distributed into the boxes, using an average density for EV1 strain of 28 quails/m^2 and for EV2 strain, 35 quails/m^2. Water and food were provided *ad libitum* during the whole experimental period.

The quail growing period was divided into an initial phase, from birth to 21 days, and a final phase, from 22 to 35 days. The quails were weighed at birth and then weekly from 21 to 35 days. Sexing was performed at 28 days, when feathers were completely differentiated between males and females. From birth to 21-days old, the quails were fed a standard growth diet containing 25% crude protein and 2900 kcal of metabolizable energy (Table 1s). From day 22, which corresponded to the beginning of the experimental period, different experimental diets were provided for each experimental unit to create an environmental gradient. These included five levels of apparent digestible methionine + cystine (0.60, 0.70, 0.80, 0.90, and 1.00% for the EV1 strain; 0.61, 0.71, 0.81, 0.91, and 1.01% for the EV2 strain; Table 2s). The experimental diets were formulated based on the nutritional composition of ingredients presented by Rostagno et al. (2011), and the nutritional requirements were in line with those recommended by the National Research Council (NRC, 1994), with the exception of digestible lysine (Ferreira, 2015), and digestible methionine + cystine (the subject of this study). Apparent digestibility of the studied amino acids were determined by an ileal digestibility assay with meat-type quails from the EV1 and EV2 strains, as described by Ferreira (2015).

To edit the database for each strain, data from quails with the following records were considered in the analyses: sire, dam, sex, and weight at 21 and 35 days of age. Weight gain in the second growth phase was calculated as the difference between weight at 35 and 21 days of age. The databases were constituted by 522 and 632 weight gain records of EV1 and EV2 strains, respectively. The pedigree was composed of seven quail generations of each strain, selected for weight at 35 days of age. The pedigree matrix for the EV1 strain was composed by 2230 animals, and for the EV2 strain, by 4164 animals. The database structure is presented in Table 1.

Weight gain during the second growth phase was analyzed using the single trait random regression animal model, considering sex and a mean trajectory as a fixed effect and the additive genetic effects as random effect. Orthogonal Legendre polynomials (LP) were used to model the mean trajectories and the additive genetic random effects.

The model can be described as follows:

$$y_{ijk} = s_i + \sum_{l=0}^{K_b} \phi_l(N_j^*) b_l + \sum_{m=0}^{K_a} \phi_m(N_j^*) a_{k_m} + e_{ijk},$$

where y_{ijk} represents the trait phenotype; s_i is the effect of sex i of the animal k; N_j^* is the digestible methionine + cystine level j standardized in the −1 to 1 interval; ϕ_l represents the l-th order LP model for digestible methionine + cystine level j used to fit the mean trajectories; ϕ_m represents m-th order LP model for the digestible methionine + cystine level j used to fit the additive genetic random effect; K_b andK_a represent the LP orders used to model the mean trajectory and the direct additive genetic effects, respectively; b_l is the l-th regression coefficient of the effect of digestible methionine + cystine on weight gain; a_{k_m} represents the additive genetic random regression coefficient of the animal k and is associated with the m-th order LP model; and e_{ijk} represents the associated error at each observation.

In matrix notation, the model above can be described as follows:

$$y = X\beta + Za + e,$$

where y represents the vector containing the observed phenotypes. This vector was assumed as $y|\beta, a, G_0, R_0 \sim N(X\beta + Za, IR_0)$, where G_0 is the covariance matrix of the regression coefficients of order $K_n + 1$, so that $\begin{bmatrix} \sigma^2_{b_0} & & & \\ \sigma_{b_0 b_1} & \sigma^2_{b_1} & & \\ \vdots & \vdots & \ddots & \\ \sigma_{b_0 b_n} & \sigma_{b_1 b_n} & \cdots & \sigma^2_{b_n} \end{bmatrix}$, where n is the order of the LP used, and R_0 is the residual variance matrix for the traits, such that $R_0 = \begin{bmatrix} \sigma^2_{e_1} & 0 & & \\ 0 & \sigma^2_{e_2} & & \\ 0 & 0 & \ddots & \\ 0 & 0 & \cdots & \sigma^2_{e_p} \end{bmatrix}$, where p represent the number of classes of residual variance considered in the model; X and Z are the incidence matrix for β and a, respectively; β is the vector of non-additive systematic effects, so that $\beta \sim$ constant; a is the vector containing the solution for random regression coefficients of the additive genetic effects. This vector was assumed as $a|A, G_0 \sim N(0, G_0 \otimes A)$, where A represents Wright relationship matrix, N is the normal distribution and ⊗ is the direct product of the operator between the matrices; and e is a vector of residuals associated with each observation. It was assumed that $e|I, R_0 \sim N(0, R_0)$, being I an identity matrix in which the order and number of observations are the same.

Inverted Wishart distributions were assumed a priori for the (co) variance matrixes of the regression coefficients and residual variances. Thus, $G_0 \sim W^{-1}(\Sigma^2_a, n_a)$ and $R_0 \sim W^{-1}(\Sigma^2_e, n_e)$, where Σ^2_a, Σ^2_e, n_a, and n_e represent the hyperparameters of the inverted Wishart distributions. Flat distributions were assumed for the hyperparameters of location and scale, that is, the values were assumed for these parameters to consider the priories as non-informative ones. Information on *a posteriori* complete conditional distributions has been previously described (Sorensen and Gianola, 2002).

Complete conditional distribution samples of the systematic components were obtained via a Gibbs sampler using the GIBBS3F90 program (Misztal et al., 2015). To analyze the EVI strain, chains of 880,000, with an initial discard of 80,000 samples, and covariance component values sampled every 20 cycles were considered. For EV2 strain analyses, chains of 1,800,000 cycles were considered, with initial discard of 200,000 samples and covariance component values sampled every 20 cycles. The chain size used to analyze each strain was defined

Table 1
Summary statistics[a] for weight gain of meat-type quails of EV1 and EV2 strains fed with different levels of methionine + cystine digestible apparent.

EV1 strain Level (%)	N	NS	ND	$\bar{X}$	SD	EV2 strain Level (%)	N	NS	ND	$\bar{X}$	SD
0.60	102	52	68	95.34	16.81	0.61	145	69	92	91.83	21.28
0.70	114	59	77	91.68	16.86	0.71	147	68	91	82.60	15.16
0.80	93	51	62	109.89	14.18	0.81	126	65	82	101.31	16.40
0.90	107	55	68	97.76	15.01	0.91	105	62	81	101.28	26.43
1.00	106	58	70	103.43	14.88	1.01	108	62	75	101.81	15.50

[a] N = number of observations; NS = number of sires with progeny; ND = number of dams with progeny; $\bar{X}$ = average, sd = standard deviation.

in preliminary analyses according to the Raftery and Lewis (1992) method, which is available in the BOA package (Smith, 2005) of R software (R DEVELOPMENT CORE TEAM, 2016). The definitive chain convergence was observed and evaluated by the Heidelberger and Welch (1983) criterion (available in the same program) and visual inspection of the sampled values in each iteration.

Representation of the random regression model was defined as follows: $LEG_{k_b k_a e}$, where *LEG* represents the LP; and k_b, k_a, and e represent the LP order used to fit the growth mean trajectory, the additive genetic random effects, and the number residual variance classes considered in the model, respectively.

The fitted models were compared by the deviance information criterion (DIC), as proposed by Spiegelhalter et al. (2002):

$$DIC = 2\overline{D}(\theta) - D(\bar{\theta})$$

where $\overline{D}(\theta)$ represents *a posteriori* estimation of Bayesian deviations; and $\overline{D}(\theta) = E_{\theta|y}[D(\theta)]$ and $D(\bar{\theta})$ indicate the fitting quality of model $D(\bar{\theta}) = -2\log p(y|\theta)$. The models presenting the lowest DIC values were chosen, since lower values are associated with models that present better fit, considering the number of estimated parameters. In this way, lower DIC values represent models that best fit the data and are less standardized.

The superiority of models that presented best fit over the others for each strain was also evaluated by calculating the Posterior Model Probabilities (PMP), using the approximation presented by Wilberg and Bence (2008). PMP allowed a rescale of DIC differences among models and was given by:

$$p(M_s|y) = \frac{e^{-(\Delta_s/2)}}{\sum_{s=1}^{S} e^{-(\Delta_s/2)}}$$

where $p(M_s|y)$is the posterior probability of model s (s = 1,2,…,S) be the best model among the candidate models (s = 9 for EV1 strain and s = 10 for EV2 strain); Δ_s is the DIC difference between model s and the best model.

Comparison of the models began with the mean trajectory fitting, followed by fitting of additive genetic random effects, and, finally, for residual variance classes. Initially, the four-order LP model for additive genetic effect and five residual variance classes were used to assess the mean trajectory. Then, the LP orders were varied and the mean trajectory was adjusted from one to four-order LP, the aim being to determine the best model for the mean trajectory fitting. After adjusting the mean trajectory, the procedure was repeated for the additive genetic effect. The LP order used in the model that best fitted the mean trajectory was fixed, varying the LP order used to adjust the additive genetic effect from one to four, and five residual variance classes were fixed. Finally, the number of residual variance classes was adjusted, in accordance with the behavior of the residual variance observed *a posteriori* in the previous models.

After selecting the best-fitted model to describe the weight gain of meat-type quails in both studied strains, the posterior mean and high posterior density intervals with 90% of the samples (HPD90) of genetic parameters for individuals raised on different dietary levels of digestible methionine + cystine were estimated as follows:

$$r_{a_{(j,j')k}} = \frac{\mathrm{cov}_{a_{(j,j')k}}}{\sqrt{\sigma^2_{a_{(j)k}} \times \sigma^2_{a_{(j')k}}}}; \quad h^2_{(j)k} = \frac{\sigma^2_{a_{(j)k}}}{\sigma^2_{P_{(j)k}}} \text{ and } CV_{a_{(j)k}} = \frac{\sqrt{\sigma^2_{a_{(j)k}}}}{\overline{X}_{(j)k}},$$

where r_a represents the genetic correlation; cov_a is the genetic covariance; σ^2_a denotes the additive genetic variance; h^2 refers to heritability; CV_a is the coefficient of additive genetic variation; σ^2_p is the phenotypic variance; $\overline{X}$ denotes the average quail weight gain; j and j' are the levels of digestible methionine + cystine in the quail diet; and k, represent the interaction from which the estimates were obtained. The HPD90 was obtained from the cumulative distribution of samples at the end of each interaction and represents the shortest interval to which 90% of the sampled values are contained, assuming that they follow a unimodal distribution (Plumer et al., 2016).

The estimated genetic values for each quail with different levels of digestible methionine + cystine were obtained from matrixes containing the solutions of regression coefficients and the LP values defined for each level, enabling the genetic behavior of individuals with different levels of amino acids to be estimated; that is, the sensibility of each animal to the tested environment.

3. Results

The models that best explained the weight gain of EV1 and EV2 quails in the second phase of their development were distinct, according to the DIC (Table 2). For the EV1 strain, the fourth-order LP model could describe the mean trajectories of weight gain along with environmental gradient. In contrast, for EV2, the first-order LP model

Table 2
Number of regression coefficients of the Legendre polynomials for the mean trajectory (K_b), direct additive genetic effect (K_a), number of residual variances classes (e), Deviance Information Criterion (DIC) and Posterior Model Probability (PMP), for random regression models used in the analyses of weight gain in meat-type quail of EV1 and EV2 strains. The values in bold indicate the models of best fit for each strain.

	Model	K_b	K_a	e	DIC	PMP
EV1 strain	LEG_{145}	2	5	5	3938.76	≈0.00
	LEG_{245}	3	5	5	3752.71	≈0.00
	LEG_{345}	4	5	5	3866.39	≈0.00
	LEG_{415}	5	2	5	4018.98	≈0.00
	LEG_{425}	5	3	5	4164.93	≈0.00
	LEG_{435}	5	4	5	4089.54	≈0.00
	LEG_{441}	5	5	1	4132.58	≈0.00
	LEG_{444}	5	5	4	4003.48	≈0.00
	LEG_{445}	5	5	5	**3090.16**	**≈1.00**
EV2 strain	LEG_{115}	2	2	5	5574.67	≈0.00
	LEG_{125}	2	3	5	5407.43	≈0.00
	LEG_{135}	2	4	5	5333.39	≈0.00
	LEG_{141}	2	5	1	5301.41	≈0.00
	LEG_{142}	2	5	2	5240.90	≈0.00
	LEG_{143}	2	5	3	5277.23	≈0.00
	LEG_{145}	2	5	5	**5225.84**	**≈1.00**
	LEG_{245}	3	5	5	5263.39	≈0.00
	LEG_{345}	4	5	5	5243.26	≈0.00
	LEG_{445}	5	5	5	5356.57	≈0.00

was sufficient to describe the mean weight gain with different levels of digestible methionine + cystine, showing that the behavior of mean weight gain along the environmental gradient was linear in this strain. In both strains the selected models were far superior in relation to the other tested models for modeling the weight gain in meat type quails, once for EV1 and EV2 strains the PMP for the model with lowest DIC was 100% superior of the second lowest DIC model.

The fourth-order LP model best described the additive genetic effects of quail weight gain in both strains, according to DIC values (Table 2). The heterogeneous residual variance models were better fitted than those considering homogeneity of residual. The model with five classes of residual variance best-fitted both strains, according to the proposed criterion for model comparison (Table 2). Thus, the fourth-order LP model could best describe the weight gain in meat-type quail of EV1 strain in terms of dietary digestible methionine + cystine after fitting the mean trajectory and the additive genetic random effects with five residual variance classes. By its turn, the model used to describe response under study in quails from EV2 strain was linear LP for fitting the mean trajectory, four-order LP for fitting the additive genetic random effects and five residual variance classes.

The weight gain averages, estimated by the selected model in terms of the amino acid level provided, and those observed in quails raised on diets with different levels of digestible methionine + cystine, can be observed in Fig. 1. Overall, the fit of the estimated values to the average weight gain observed in each group with the selected models were of good quality.

The variances, covariances, and correlations between the random regression coefficients of the direct additive genetic effects for EV1 and EV2 strains can be observed in Table 3. The intercept and linear coefficient of each model are not correlated, and the correlation between the intercept and the third regression coefficient was positive, and that between the intercept and the fourth regression coefficient was negative for the additive genetic effects in both strains. The correlation between the intercept and the second regression- coefficient (linear) differed between strains. It did not differ from zero in the model selected to describe EV1 quail weight gain, and was positive in the model selected for the EV2 strain. Correlations between the other regression coefficients did not differ in the selected models for the EV1 and EV2 strains. They were negative between the first- and third- regression coefficients, and between the second and the fourth coefficients for each model, and did not differ from zero between the other coefficients (Table 3).

A posteriori estimates of additive, residual, and phenotypic genetic variances for weight gain in EV1 quails presented an irregular trajectory along the environmental gradient (Fig. 2). However, no significant difference was observed in variance along the environmental gradient as a consequence of wide HPD90. The behavior of the phenotypic variance in the EV1 strain was similar to the behavior of additive genetic variance. In relation to EV2, the additive genetic variance did not alter significantly along the environmental gradient. In turn, the residual variance between the levels presented higher discrepancy along the environmental gradient. Phenotypic variance in the EV2 strain was similar to the residual variance (Fig. 2).

Table 4 shows *a posteriori* average estimates of heritability and additive genetic variation coefficients for quails of both strains raised on each level of digestible methionine + cystine. Heritability varied from low to high between the different levels of digestible methionine + cystine provided to the quails. These values did not differ among quails of the EV1 strain. In the EV2 strain, heritability for weight gain in quails raised on 0.91% digestible methionine + cystine was smaller than the observed for quails raised on 0.71%. The other heritability values observed in quails of EV2 strain were intermediate and did not differ with the level of dietary amino acids. The variation coefficients for direct additive genetic values of weight gain in meat-type quail did not differ along the dietary level of amino acids provided for each strain, and presented moderate to high values, considering phenotypic variations up to 26% in both strains (Table 4).

In general, the *posteriori* means of genetic correlations for weight gain in different levels of methionine + cystine in each strain were less than 1.00 (Table 5). The genetic correlations for weight gain among EV1 quails raised on different levels of digestible amino acids were, in general, equal to zero, except for the correlation between the first and the third levels, and between the first and the fourth levels, which were negative. In the EV2 strain, the behavior of the correlations varied from high and negative, to high and positive along the environmental gradient.

The reaction norms of weight gain for the 10 sires from the EV1 and EV2 strains that have contributed with the highest number of progenies in the tested environments are shown in Fig. 3. The genetic value of quails changed with different levels of digestible methionine + cystine. However, there was no standard behavior for these changes. There were changes in the classification order of the individuals, according to the level of digestible methionine + cystine provided.

4. Discussion

Several studies using reaction norm models to evaluate the genotype × environment interaction for traits of economic interest in different species have considered the reaction norm to be linear, without testing more complex models (Mattar et al., 2011; Cardoso and Tempelman, 2012; Mota et al., 2015; Ambrosini et al., 2016; Miranda et al., 2016). The assumption of a linear reaction norm can lead to the use of models with worse fitting when describing trait variability, which can impair the monitoring of genetic change under study in the tested environments (Gavrilets and Scheiner, 1993).

Models with unsuitable fitting have less accurate parameter estimates, since they can present properties that do not appropriately reflect the model properties that better describe the measured trait along

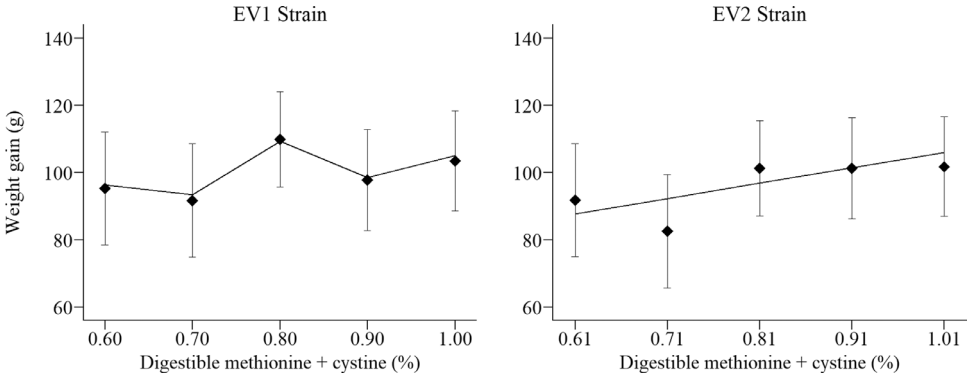

Fig. 1. Observed means (points) and estimated (lines) weight gain of quails of EV1 and EV2 strains, estimated by LEG_{445} and LEG_{145} models, respectively. Standard deviations of observed means are represented by bars.

Table 3
Posteriori means of variances (diagonal - in bold), covariances (below diagonal) and correlations (above diagonal) (and highest posterior interval with 90% of samples) of random regression coefficients[a] of the models selected to describe weight gain in meat-type quail of EV1 and EV2 strains according to the dietary methionine + cystine digestible apparent level.

EV1 strain	a_0	a_1	a_2	a_3	a_4
a_0	**176.18**	0.17	0.37	0.41	−0.78
	(87.20;265.50)	(−0.30;0.59)	(−0.06;0.84)	(0.03;0.82)	(−0.98;−0.60)
a_1	18.96	**61.26**	−0.19	−0.70	0.03
	(−25.15;63.86)	**(11.60;107.30)**	(−0.82;0.46)	(−0.96;−0.45)	(−0.54;0.58)
a_2	44.94	−15.96	**88.26**	0.21	−0.65
	(−17.01;106.30)	(−63.34;32.03)	**(18.35;150.00)**	(−0.37;0.76)	(−0.96;−0.37)
a_3	28.99	−30.79	9.73	**29.16**	−0.25
	(−94.37;60.98)	(−56.18;−2.91)	(−20.91;38.36)	**(10.37;46.68)**	(−0.74;0.19)
a_4	−85.78	2.95	−52.11	−10.38	**68.00**
	(−136.30;−32.85)	(−31.58;35.91)	(−99.29;−5.26)	(−31.03;9.77)	**(27.74;107.60)**
EV2 strain	a_0	a_1	a_2	a_3	a_4
a_0	**72.30**	−0.28	0.76	0.55	−0.74
	(35.95;107.40)	(−0.79;0.22)	(0.59;0.94)	(0.24;0.87)	(−0.92;−0.55)
a_1	−20.33	**77.59**	0.21	−0.71	−0.25
	(−57.50;17.73)	**(19.80;132.70)**	(−0.33;0.82)	(−0.98;−0.45)	(−0.79;0.24)
a_2	65.73	21.47	**101.92**	−0.08	−0.86
	(21.71;108.90)	(−35.14;75.95)	**(32.92;171.60)**	(−0.58;0.36)	(−0.98;−0.73)
a_3	31.84	−43.89	−9.00	**48.92**	0.00
	(8.64;54.98)	(−76.83;−9.90)	(−43.74;25.57)	**(22.17;74.21)**	(−0.42;0.44)
a_4	−63.08	−25.02	−87.34	2.36	**100.31**
	(−102.00;−23.72)	(−75.59;26.40)	(−144.60;−27.76)	(−28.31;32.11)	**(41.79;155.90)**

[a] a_0 = intercept; a_1 = first-degree coefficient; a_2 = second-degree coefficient; a_3 = third-degree coefficient; a_4 = fourth-degree coefficient.

the environments (Morrissey and Liefting, 2016). Besides testing more parameterized models and determining the model with the best fitting according to suitable criteria, the increased prediction accuracy of genetic parameters can be altered by adjusting the non-genetic systematic effects included in the model, since these factors can lead to a correlation between the phenotypes due to non-genetic factors (Schaeffer, 2004). This fitting in random regression models is also known as mean trajectory. In the present study, the mean trajectory fitting was accomplished through fixed regression, which considered all possible non-genetic effects that could influence the phenotype from one or more quail groups (Table 2). As in a random regression model, the curve that describes the observed phenotypes is represented by a mean trajectory plus a set of random regression coefficients that defines the individual deviations related to additive genetic effects. The suitable fitting for this trajectory (Fig. 1) is essential in order to properly estimate the other effects that explain phenotype variations.

Investigations of phenotypic variation in natural and domesticated populations of plants and animals commonly study variance components (Gupta and Lewontin, 1982). Any attempt to understand the phenotype of individuals for quantitative traits in populations without knowing the reaction norms obtained by well-fitted models is incomplete, since the variance analysis mistakes environmental and

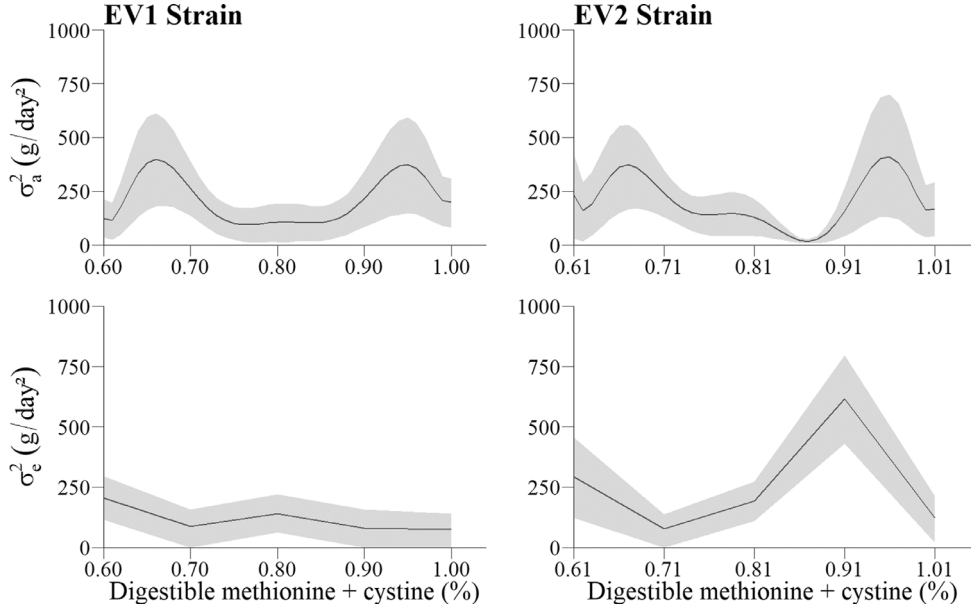

Fig. 2. Genetic additive (σ^2_a), residual (σ^2_e) and phenotypic (σ^2_p) (lines) variances and highest posterior density interval with 90% of samples (gray) of weight gain in meat-type quails from EV1 and EV2 strains, estimated by LEG_{445} and LEG_{145} models, respectively.

Table 4
Posteriori means of heritability (h^2) and additive genetic variation coefficient (CV_a) (and highest posterior interval with 90% of samples) for weight gain during the second growth period of meat-type quail of EV1 and EV2 strains, fed with different levels of methionine + cystine digestible apparent.

EV1 strain Level (%)	h^2	CV_a	EV2 strain Level (%)	h^2	CV_a
0.60	0.37 (0.13;0.62)	0.11 (0.07;0.16)	0.61	0.43 (0.09;0.75)	0.16 (0.08;0.24)
0.70	0.74 (0.53;1.00)	0.17 (0.13;0.22)	0.71	0.75 (0.54;1.00)	0.19 (0.14;0.23)
0.80	0.42 (0.09;0.73)	0.09 (0.05;0.13)	0.81	0.40 (0.15;0.63)	0.11 (0.07;0.15)
0.90	0.72 (0.42;1.00)	0.15 (0.10;0.19)	0.91	0.20 (0.06;0.34)	0.12 (0.07;0.17)
1.00	0.72 (0.45;1.00)	0.13 (0.09;0.18)	1.01	0.56 (0.22;0.92)	0.12 (0.07;0.17)

Table 5
Posteriori means of genetic correlations of weight gain (and highest posterior interval with 90% of samples) between meat-type quails in the second growth period of EV1 and EV2 strains raised in different levels of methionine + cystine digestible apparent[a].

	EV1 strain 0.60	0.70	0.80	0.90
0.70	−0.31 (−0.77;0.13)			
0.80	−0.68 (−0.97;−0.38)	−0.02 (−0.63;0.60)		
0.90	−0.48 (−0.92;−0.03)	0.40 (0.00;0.83)	0.15 (−0.43;0.74)	
1.00	0.34 (−0.12;0.80)	0.35 (−0.07;0.79)	0.03 (−0.56;0.63)	0.22 (−0.30;0.74)
	EV2 strain 0.61	0.71	0.81	0.91
0.71	−0.19 (−0.74;0.28)			
0.81	0.17 (−0.44;0.74)	−0.14 (−0.59;0.25)		
0.91	−0.28 (−0.85;0.25)	−0.05 (−0.49;0.35)	−0.96 (−1.00;−0.91)	
1.01	0.15 (−0.47;0.75)	0.44 (0.01;0.94)	0.12 (−0.44;0.69)	−0.17 (−0.77;0.38)

[a] EV1 strain = 0.60; 0.70; 0.80; 0.90 e 1.00% of methionine + cystine digestible apparent; EV2 strain = 0.61; 0.71; 0.81; 0.91 e 1.01% of methionine + cystine digestible apparent.

genetic factors (Lewontin, 1974). In fact, the major discrepancy of phenotypic variability observed with different levels of dietary digestible methionine + cystine can be attributed to sudden variations in the tested environments, as observed by comparing the behavior of additive, residual, and phenotypic variances from the EV2 strain (Fig. 2). However, the phenotypic behavior cannot be solely attributed to heterogeneity of residual variance, since changes in genetic variance can also influence phenotypic behavior, as observed in the EV1 strain (Fig. 2). The irregular behavior of additive, residual, and phenotypic variance also directly affects the behavior of heritability (Table 4).

Heritability is the standard measurement of potential genetic change in a population (Pigliucci, 2008). In populations under artificial selection, the genetic change of a certain trait through generations is obtained by selection and it is seen as genetic gain. Moderate and high values of heritability were found for weight gain in quails from both strains (Table 4), which indicates that it is possible to select for the trait under study and to obtain genetic gains, because this is a trait determined mainly by additive genetic factors. In addition, as heritability in each strain, in general, does not alter along the environment gradient, it is assumed that all groups created under different levels are equally susceptible to the genotype × environmental interaction effect (Raidan et al., 2015). Only the heritabilities for weight gain of the quails raised under 0.91% of digestible methionine + cystine was significantly lower of the heritability of the gain for quails raised under 0.71% of the same amino acid, this fact could be interpreted as an accidental finding, that could be explained for the cryptic variance accumulated over the years of selection that made the estimated genetic variance for the quails raised under 0.71% of digestible methionine + cystine more expressive than the genetic variance at 0.91% level (Gibson and Dworkin, 2004).

Additionally, another important measurement used to evaluate the genetic change potential of certain populations is the additive variation coefficient (CV_a) (Houle, 1992). If jointly evaluated, the heritability and CV_a make it possible to infer specifically about the potential of genetic changings of the populations under study. Joint analysis of coefficient values from the additive genetic variation and heritability indicates that genetic variability exists for quail weight gain in the second growth phase. In this way, the high genetic variability observed for weight gain in both quail strains, indicates that selection using weight-gain criterion makes it possible to obtain genetic gains across generations (Table 4). These values are more expressive when considering that the quails under study belong to a breeding stock previously selected for weight at 35 days, which involves a trait positively correlated with weight gain.

However, high heritability values and additive variation coefficients, which have not varied with the dietary amino acid levels, indicate that the genetic gain obtained in each generation would be the same regardless of the selection and raising environment of the quails. Conversely, the results obtained in this study indicate that the expected genetic change by generation would be different in each environment, considering the same population of selected individuals as parents of the next generation. Fig. 3 shows that different sires would be selected as superior for each environment, which constitutes the environmental gradient; thus, the ranking of individuals changed along the environmental gradient, which suggests the presence of a

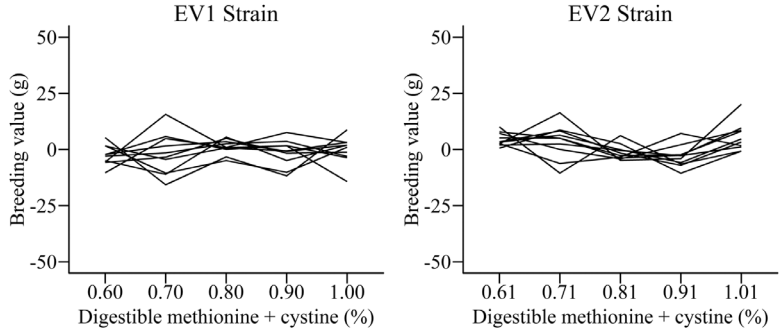

Fig. 3. Reaction norms from additive genetic values of weight gain in the second growth phase of 10 breeders, which have contributed with the highest number of progenies in EV1 and EV2 quail strains.

genotype × environment interaction. It is important to highlight that the Fig. 3 reports the estimated breeding value trajectory for each animal along the different environments, which is important to infer about genotype × environment interaction. It does not necessarily represent the behavior of the observed weight gain in each environment – which has a different behavior that reflects in the observed means for average daily gain (Fig. 1).

A genotype × environment interaction can be observed through the correlation of the genetic values and regression coefficients, which were, in general, non-different from zero (Tables 3 and 5) (Falconer, 1990). A genotype × environment interaction for performance traits in meat-type quails was observed by Mota et al. (2015). In terms of artificial selection, the presence of such an interaction indicates that the selection and raising environments of the quails must be the same, making it necessary to design specific genetic breeding programs for each selection/raising environment (Mota et al., 2015).

In a study with beef cattle, Raidan et al. (2015) suggested that changes in the intensity of the selection accomplished in the different groups can control for losses caused by the presence of a genotype × environmental interaction, after response analyses to direct and indirect selection, without restricting the animals raising and selecting environment to the same one. However, this type of compensation could not be applied to all levels in the present study, since non-significant genetic correlations were observed among quail groups raised in certain environmental groups, in both strains. The presence of genetic correlations that did not differ from zero, suggest the absence or reduction of a correlated response (indirect response) to selection. This makes it impossible to correct the losses caused by a genotype × environment interaction by changes in the selection intensity practiced on the livestock.

The correlations were assumed to be non-significant because the HPD90 contains the zero value. It is possible to observe that this intervals are large, so it is not possible to assume with precision that the average daily gain between environments are non-associated traits. However, it is possible to infer that the values of correlations were not significant. Under this perspective, the genetic correlations estimated in the present study, for both strains, only propose that different groups of genes are partially responsible for the phenotype expression in the different environments (Falconer and Mackay, 1996) – what does not mean that all the genes differed of each other in function of the environment.

The results of the present study reveal the importance of considering all genetic parameters estimated by the reaction norm models before regarding the presence of a genotype × environment interaction as a barrier to the accomplishment of artificial selection. Taking into account that, in some cases, it is necessary to recommend specific selection programs for livestock with restricted selection and raising environment of the quails.

5. Conclusions

There is an interaction between quail genotypes from the EV1 and EV2 strains and digestible methionine + cystine levels in the diet. In the presence of this interaction, the use of reaction norm models with high order Legendre polynomials are suitable, since they make possible to verify that the weight gain behavior in relation to digestible methionine + cystine levels is not necessarily linear.

Declaration of Competing Interest

The authors declare no conflicts of interest.

Acknowledgements

The authors acknowledge the Brazilian funding agencies FAPEMIG ('Fundação de Amparo à Pesquisa do Estado de Minas Gerais'), CNPq ('Conselho Nacional de Desenvolvimento Científico e Tecnológico') for financial support and CAPES ('Coordenação de Aperfeiçoamento de Pessoal de Nível Superior') for a fellowship to Gabriela C. Gouveia. In addition, the authors express appreciation to 'Ajinomoto do Brasil Ind.', for providing the amino acids utilized in the experiment. Finally yet importantly, the authors thanks to Professor José Miguel Ortega for providing access to Sagarana server allowing the database processing.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.livsci.2019.09.016.

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