Direct and maternal genetic effects for preinflection point growth traits and humoral immunity in quail

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ABSTRACT Early growth traits in quails are considered as the growth performances before the inflection point which are genetically different from body weights (**BW**) at later stages. Moreover, in addition to growth performance, humoral immunity is moderately heritable and is considered in some breeding programs. However, estimating the direct genetic, particularly the maternal genetic correlations between growth and immunity in quail, are not studied sufficiently, which were the aims of the present study. The quails' BW were recorded at hatch (**BW0**) to 25 d of age with a 5-d interval and body weight gains (**BWG**) were measured as average growth performance of the birds in a 5-d period. Antibody titer against Newcastle disease virus (**IgN**) was measured through the hemagglutination inhibition (**HI**) test. For titration of anti-SRBC antibodies (**IgY** and **IgM**), a hemagglutination microtiter assay was used. In general, growth records in 4,181 birds and humoral immune responses in 1,023 birds were assigned to the study. The genetic parameters were estimated by single-trait analysis via Gibb's sampling. After finding the best model for each trait, multi-trait analysis was done to estimate the direct and maternal genetic correlations. Direct heritabilities (h^2) were estimated to be moderate for BW ($0.481−0.551$) and BWG ($0.524−0.557$), while h^2 for immune responses were low ($0.035−0.079$). Maternal environmental effect (c^2) was only significant for BW0, BW5, and BWG0-5. Maternal heritabilities (m^2) for BW and BWG were all lower than corresponding h^2, ranging from 0.072 (BW25) to 0.098 (BW0). The m^2 for IgN (0.098) was more than 2.5 times greater than h^2 (0.040) for this trait. Direct (r_a) and maternal (r_m) genetic correlations between IgN-BW, IgY-BW, and IgY-BWG were negative, while ra and rm for IgM-BW, IgN-BWG, and IgM-BWG were positive. The r_a between humoral immune responses were low to moderate and rm was significant only for IgY-IgM (0.339). Given positive genetic correlations in BWG-IgN and BWG-IgM as well as positive genetic correlations between both IgN and IgM with IgY, it is suggested that including the BWG in the breeding programs would directly result in the improvement of the birds' growth performance. It would also contribute indirectly to the improvement of the birds' humoral immune responses.

Key words: maternal genetic, genetic correlation, heritability, body weight gain, humoral immune response

INTRODUCTION

Compared with growth at later stages of life, it is suggested that the birds' early growth performance can be genetically controlled through some different sets of genes (Sewalem et al., 2002). Biologically, early growth in most tissues was caused by hyperplasia (increasing the rate of cell division); however, hypertrophy (increased cell size) in later stages of the animals' growth assumes major importance (Atchley and Zhu, 1997). According to the sigmoid pattern of growth in birds, early growth traits are considered as the growth performances before the inflection point (as the increasing phase of the growth) which are genetically different from body weights (**BW**) in later stages (growth traits after the inflection point as decreasing phase of the growth). Due to its economic importance, studying genetic bases of early growth are of interest in quail (Mohammadi-Tighsiah et al., 2018), and chicken (Cunningham et al., 1987; Carlborg et al., 2003). Therefore, some studies have been conducted to estimate the genetic and non-genetic parameters for early growth

performances of the birds (Khaldari et al., 2010; Mohammadi-Tighsiah et al., 2018).

Genetic and environmental variation between dams influence on progeny performance (phenotype) which is called maternal effects (Bijma, 2006). There are several reports on direct heritability (h2) for growth traits in poultry. However, maternal effects often have been less considered (Marks, 1996; Nestor et al., 2008; Lwelamira, 2012); while the importance of maternal genetic and environmental effects have been demonstrated on early growth traits in chickens (Mignon-Grasteau et al., 1999; Tongsiri et al., 2019). It is proposed that the maternal effects for traits that be measured at the early stages of life even might be more important than direct genetic effects (Hartmann et al., 2003; Aslam et al., 2011). Therefore, ignoring maternal genetic effect could result in overestimated error variance and bias in direct heritability (Ghorbani et al., 2013; Barbieri et al., 2015; Abou Khadiga et al., 2016). Rather than growth traits, Addison et al. (2010) reported the little effect of maternal antibodies on the immune response in the early stage of life (Addison et al., 2010).

Estimates of genetic parameters for humoral immunity in poultry indicate low to moderate (generally between 0.05 and 0.30) of direct (Sivaraman et al., 2005; Mohammadi-Tighsiah et al., 2018; Faraji-Arough et al., 2022) and low (generally < 0.10) of maternal heritabilities (van der Klein et al., 2015; Bao et al., 2016). Moreover, heritability estimations of the immunological traits in 3 Japanese quail populations reported being ranged from medium to high (0.25−0.44; Monika et al., 2020). Therefore, the birds' humoral immunity related parameters are usually considered as moderately heritable traits which are sometimes included in the breeding plans for the poultry (Sarker et al., 1999; Bovenhuis et al., 2002; Buitenhuis et al., 2004). To study genetic parameters for humoral immune responses in the poultry, Sheep Red Blood Cells (**SRBC**) (Van der Zijpp and Leenstra, 1980; Sarker et al., 1999; Maghsoudi et al., 2020), conventional vaccines (Lwelamira et al., 2009), or other pathogens (Leitner et al., 1992; Yonash et al., 1996; Kaiser et al., 2002) have been usually used for the immune system stimulation. Most of the studies have considered only the additive genetic basis of humoral immunity (Bovenhuis et al., 2002; Mohammadi-Tighsiah et al., 2018). Moreover, there are only a few studies investigating maternal effects and this area has not received adequate scholarly attention (Bao et al., 2016). As far as the researchers know, estimates of genetic parameters of maternal genetic/environmental effects for humoral immune responses have been often studied in chicken (*Gallus gallus*). Although, an interesting investigation with a new methodology worked on maternally derived antibodies in quail has been performed (Addison et al., 2010), however, reports on the contribution of maternal genetic effects on humoral immunity in quail are a rarity. Nevertheless, direct and maternal genetic effects of humoral immunity have recently been studied in addition to non-additive genetic effects (Faraji-Arough et al., 2022).

According to the resource allocation theory, there is a negative relationship between the growth and immunity performances of animals. However, studying this type of relationship is often limited to the phenotypic or direct genetic effect of growth and immunity (Dunnington and Siegel, 1996; Van Eerden et al., 2004; Mohammadi-Tighsiah et al., 2018); whereas maternal effects are rarely considered (Saino et al., 2002). The immune system of vertebrates is immature immediately after birth; hence, newborn chickens are completely dependent on the maternal immune system ability which is transferred to the offspring via the egg. Therefore, ignoring maternal effects may lead to an incomplete assessment of early growth and immunity relationships.

To the best of the authors' knowledge, there is no report to clarify the direct and maternal genetic relationship between early growth and humoral immunity traits in quail. Therefore, the aims of this study were first to estimate the genetic parameters for early growth traits including BW and body weight gains (**BWG**) as well as humoral immune responses against SRBC and Newcastle disease virus (**NDV**) through single-trait animal models based on inclusion/exclusion of maternal effects (genetic and environmental). After finding the best animal model for each trait, investigation of the genetic and non-genetic relationships between growth performances and humoral immune responses was performed using a multi-trait animal model.

MATERIAL AND METHODS

Birds

This study was conducted following the general ethical guidelines of the Animal Care and Use Committee, Faculty of Veterinary Medicine, University of Zabol, Zabol, Iran. Data used in the current study were obtained from a random-bred population of Japanese quail (*Coturnix coturnix japonica*). Data and pedigree information were collected over 6 generations from 2015 to 2017. Over generations, all the birds reared under the same breeding plan and management practices. In the first generation, a total number of 800 one-day-old quail chicks were identified with wing band and weighed immediately after hatch. All birds were reared in 20-group cages separately with 40 birds in each cage until d 45 of age. On d 20 of age, the birds were separated based on their sex. On d 45 of age, a total of 400 birds (200 birds of each sex) were randomly distributed in pairs of 1 male and 1 female breeder, and each pair was transferred to a new mating cage (200 cages in total with 2 birds in each cage) and mated for the first time as base population. Fertile eggs were collected and numbered from each cage according to the identification of breeding pairs from d 70 to 80 of the parents' age. Afterward, before the incubation phase, all the eggs were disinfected and stored in the egg storage room with 12°C to 16°C and 70% humidity. The next generations were made through 2 consecutive hatches with a 5-d interval. The same pattern was done through 6 consecutive generations.

The optimum temperature for birds was 38 degrees Celsius in d 1 and it was kept constant until the end of the first week. The ambient temperature decreased during the growth period until d 45, when it remained constant at 25°C. The ambient temperature was kept between 22°C and 25°C during the egg production period. Generally, about 25% of the birds were excluded from the study during the first week, due to natural mortality, locomotion problems, and general weakness.

All the birds had access to the water and were fed ad libitum throughout the experiment. The birds were fed a corn-based concentrate ration with 255 g/kg crude protein and 12.3 MJ/kg of ME from 1 to 45 d of age as a growth period diet and with 201 g/kg crude protein and 11.7 MJ/kg of ME after d 45 as laying period diet. A common light program of 20 h light and 4 h darkness was applied during the experiment.

Growth Performance

tEarly growth performance of the birds was considered BW and BWG before the inflection point. Using mathematical modeling (Gompertz and Logistic functions) to study the growth pattern of Japanese quail, the inflection point was determined at d 25 of old (Faraji-Arough et al., 2018). Therefore, BW of the quails were recorded at hatch (**BW0**), and 5, to 25 d of age with 5-d interval included **BW5, BW10, BW15, BW20**, and **BW25**. Moreover, BWG were measured as the average growth performance of the birds in 5-d periods. Accordingly, BWG included in the analyses were **BWG0-5, BWG5-10, BWG10-15, BWG15-20**, and **BW20-25**.

Humoral Immune Responses

Over 6 generations, all the birds were vaccinated with NDV-B1 strain vaccine (Razi Co, Karaj, Iran) through intraocular administration on d 24 of age. Moreover, to assess humoral immune responses, each bird received 0.2 mL of 5% SRBC suspension (in sterile phosphate buffered saline; **PBS**) by intramuscular injection into the left breast muscle at d 31 and 38 of age. Blood samples were collected from the wing vein of the birds at d 45 of age using anticoagulant (**EDTA**) containing syringes and were immediately stored in ice. Then, blood samples were centrifuged at 2,500 × g for 10 min at 8°C, and the obtained plasma was stored at −80°C until antibody titration. Antibody titer against NDV (**IgN**) was measured through the hemagglutination inhibition (**HI**) test. For anti-SRBC (immunoglobulin Y, **IgY**; and immunoglobulin M, **IgM**) antibody titration, plasma samples were first heat-inactivated at 56°C to inhibition of the complement system. Next, resistant (IgY) or sensitive (IgM) antibodies to the 2-mercaptoethanol were measured through the treatment of 2-fold serial dilutions of plasma samples in PBS (0.01 mol/L, pH 7.4) with 1.4% 2-mercaptoethanol. Titers of IgY and IgM to SRBC antigen were expressed as log 2 of the reciprocal of the highest dilution in which hemagglutination was observed in U-bottom 96-well microtiter plates (Wegmann and Smithies, 1966). For each plasma sample, all the humoral immunity experiments were conducted in duplicate within 2 separate plates. In the current study, IgN, IgY, and IgM titers were considered as humoral immune responses of the birds. Descriptive statistics of the early growth traits and humoral immune responses of Japanese quail are shown in Table 1. A small number of the birds have higher values for IgY and IgM (Max = up to 9).

Genetic Parameters

First, 4 animal models were used for the genetic analysis of the traits (including 11 of growth performances and 3 humoral immune responses):

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z_1a} + \mathbf{e} \qquad \text{(Model 1)}$$

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z_1a} + \mathbf{Wc} + \mathbf{e} \qquad \text{(Model 2)}$$

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z_1a} + \mathbf{Z_2m} + \mathbf{e} \qquad \text{(Model 3)}$$

$$\mathbf{y} = \mathbf{Xb} + \mathbf{Z_1a} + \mathbf{Z_2m} + \mathbf{Wc} + \mathbf{e} \qquad \text{(Model 4)}$$

where $\mathbf{y}$ is a vector of observations for traits, and $\mathbf{b, a, m, c}$, and $\mathbf{e}$ are the vectors of fixed (sex, generation, and hatch which is nested in generation), direct genetic, maternal genetic, maternal environmental, and residual effects, respectively. Moreover, $\mathbf{X, Z_1, Z_2}$, and $\mathbf{W}$ are the design matrices relating to the corresponding effects, respectively. The prior distributions for the direct genetic, maternal genetic, maternal environmental, and residual effects assumed multivariate normal distributions with a mean of 0 and a variance of $A\sigma_a^2$, $A\sigma_m^2$, $\mathbf{I}c\sigma_c^2$, and $\mathbf{I}n\sigma_e^2$, respectively. Moreover, σ_a^2, σ_m^2, σ_c^2 and σ_e^2 are additive genetic, maternal genetic, maternal environmental, and residual variances, respectively, and $\mathbf{A}$ is the numerator relationship matrix. $\mathbf{Ic}$ and $\mathbf{In}$ are the

Table 1. Data characteristics of early growth traits and humoral immune responses in Japanese quail.

Traits	N	Mean ± SD	Minimum	Maximum
BW0[1]	4181	8.138 ± 1.146	4.714	12.448
BW5	3097	14.157 ± 3.311	3.673	34.041
BW10	2422	27.628 ± 8.814	8.378	62.173
BW15	1907	52.916 ± 16.796	15.345	115.129
BW20	1737	81.316 ± 22.518	11.729	158.030
BW25	1597	113.854 ± 26.701	23.858	208.164
BWG0−5	3097	1.175 ± 0.608	−0.031	4.912
BWG5−10	2422	2.579 ± 1.389	−0.020	9.164
BWG10−15	1907	2.442 ± 4.659	−0.089	16.023
BWG15−20	1737	5.342 ± 2.225	−0.124	21.458
BWG20−25	1597	6.108 ± 2.215	−0.620	28.512
IgN	915	7.653 ± 2.063	1	12
IgY	1023	2.943 ± 1.422	0.5[2]	9
IgM	1023	1.266 ± 1.031	0.5	9

[1]BW: body weights of the birds at hatch (0), and 5, 10, 15, 20, and 25 d of age; BWG: body weight gain of the birds 0−5, 5−10, 10−15, 15−20, and 20−25 d period; IgN: antibody titers against NDV; IgY and IgM: immunoglobulin Y and M titers against SRBC, respectively.
[2]For all of the plasma samples, the humoral immunity experiments were conducted in duplicate; therefore, 0.5 value refer to mean of 1.0 + 0.0.

identity matrices with order equal to the number of dams and observations, respectively. The (co)variance components and genetic parameters for the studied traits were estimated by Gibb's sampling method using Gibbs3F90 (Misztal et al., 2002). Gibbs chains with 2,000,000 iterations were generated with an initial discard of 50,000 samples and saving every 100th sample. The Convergence diagnostics of the generated chains were performed by the Postgibbsf90 program (Misztal et al., 2002). To provide a comparison of models and to choose the best model for each trait, the deviance information criterion was used, and the model with the lowest deviance information criterion for each trait was identified as the favorable model. Having identified the most appropriate model for each trait, multivariate analyses were done to estimate direct and maternal genetic correlations.

RESULTS AND DISCUSSION

Direct and Maternal Genetic Parameters for Growth Traits

The best fitted model and estimates of direct heritability (h^2), the proportion of maternal environmental variance to the phenotypic variance (c^2), and maternal heritability (m^2) for early BW, BWG, and humoral immune responses of Japanese quail are shown in Table 2. The most complex model (Model 4) was assigned to the BW0 with all the possible random effects including genetic effects (direct and maternal) and maternal environmental effect. Also, Seraj et al. (2020) reported that the model including direct additive genetic, maternal additive genetic, and environmental maternal effects was the most appropriate model for hatch weight in 2 populations of quails (Seraj et al., 2020). The maternal genetic effect was significant for all the BW traits (from hatch to BW25) except for BW5, which were only significantly influenced by the maternal

environmental effect. Direct heritabilities for BW traits were moderately high and estimated from 0.481 (BW0) to 0.551 (BW10). The influence of the maternal environmental effect on BW0 and BW5 was very small (0.010 and 0.011, respectively). Maternal heritabilities of the BW traits were small too, and ranged from 0.072 (BW25) to 0.098 (BW0), with a decreasing trend observed from hatch to 25 d of old. However, Seraj et al reported that the direct heritability, maternal heritability, and the proportion of maternal environmental variance to phenotypic variance (c^2) of hatch weight were about 0.13, 0.48, and 0.06, respectively (Seraj et al., 2020). Relatively lower value of h^2 for BW0 (0.481) in the current study relates to the significance of both maternal genetic ($m^2 = 0.098$) and environmental effects ($c^2 = 0.010$) (Table 2). Significant random effects in our study were not the same as the estimates of (Barbieri et al., 2015) meat-type quail. They included the maternal genetic and environmental effects in the model for BW0 while for other BW traits only the maternal environment was included. However, they reported 0.03, 0.38, and 0.18 for h^2, c^2, and m^2 for BW0, respectively. After 4 generations of phenotypic selection and using pedigree information to estimate genetic parameters for 4-wk bodyweight (= BW28, comparable with BW25 in the current study), Khaldari et al. (2010) reported 0.26 and 0.11 for h^2 and c^2, respectively. In comparison with the results of the current study, dramatic higher importance of maternal effect for BW0 in the study of Barbieri et al. (2015), and lower h^2 (2 times) and higher c^2 (10 times) for BW28 in the study of Khaldari et al. (2010) was reported that might be due to the breed type, the number of generations, selection policies, pedigree structure, and climate. According to the results, there was not a considerable trend for heritabilities of BW. However, there was a decreasing trend observed for m^2 from hatch to d 25 of age. In Thai native chickens, maternal heritabilities declined when the age of the birds increased during the early growth traits (BW0 to BW20) (Tongsiri et al., 2019), which supports our estimates. Additionally, 2 previous studies reported that a model with additive direct and maternal genetic effects was the best model for evaluation of body weight traits except for birth weight (BW0). They indicated that for BW0 a model with additive direct, maternal genetic, and maternal permanent environmental effects was the best. They also suggested that maternal genetic effect is less important at later ages because the trends of direct genetic and maternal genetic effects from BW0 to BW42 were increasing and decreasing respectively (Lotfi et al., 2012; Ebrahimi et al., 2019). Another supportive result was from Lotfi et al, which reported that maternal effects were only significant on the BW0, and for later stages of life, the genes play dominated role to determine the BW in Japanese quail (Seraj et al., 2020). Moreover, estimation of variance components by Bayesian methods with a multiple-trait model for body weight traits came to the same conclusion about the pattern of direct and maternal effects in different ages in Japanese quails (Resende et al., 2005).

Table 2. Genetic parameters ± SD for body weight traits (BW), body weight gains (BWG) and humoral immune responses of Japanese quail based on the best fitted models.

Traits	Model	$h^2 \pm$ SD	$c^2 \pm$ SD	$m^2 \pm$ SD
BW0[1]	4	0.481 ± 0.057	0.010 ± 0.005	0.098 ± 0.024
BW5	2	0.519 ± 0.059	0.011 ± 0.003	–
BW10	3	0.551 ± 0.061	–	0.090 ± 0.022
BW15	3	0.528 ± 0.036	–	0.088 ± 0.021
BW20	3	0.527 ± 0.061	–	0.081 ± 0.020
BW25	3	0.525 ± 0.066	–	0.072 ± 0.023
BWG0-5	2	0.551 ± 0.060	0.009 ± 0.004	–
BWG5-10	3	0.557 ± 0.059	–	0.087 ± 0.024
BWG10-15	3	0.524 ± 0.065	–	0.091 ± 0.021
BWG15-20	3	0.526 ± 0.061	–	0.085 ± 0.022
BWG20-25	1	0.531 ± 0.063	–	–
IgN	3	0.040 ± 0.030	–	0.098 ± 0.054
IgY	3	0.079 ± 0.054	–	0.075 ± 0.037
IgM	1	0.035 ± 0.023	–	–

[1]BW: body weights of the birds at hatch (0), and 5, 10, 15, 20 and 25 d of age; BWG: body weight gain of the birds 0−5, 5−10, 10−15, 15−20, and 20−25 d period; IgN: antibody titers against NDV; IgY and IgM: immunoglobulin Y and immunoglobulin M titers against SRBC, respectively. Direct heritability, proportion of maternal environmental to the phenotypic variance and maternal heritability are shown as h^2, c^2, and m^2, respectively.

Heritability estimates for BWG were moderate and almost similar for all traits, ranging from 0.526 (BWG15−20) to 0.557 (BWG5−10). The maternal environment only significantly influenced BWG0−5 and was estimated very low ($c^2 = 0.009$). Although BWG20−25 was influenced only with direct additive genetics effect ($h^2 = 0.531$), maternal heritability was significant for BWG5−10 (0.087), BWG10−15 (0.091), and BWG15−20 (0.085). Comparatively, BWG (also recognized as average daily gain, ADG) has been less studied than BW traits in poultry. BWG has been mostly evaluated as a supplementary trait to study poultry feed efficiency (Van Eerden et al., 2004); hence, it has been rarely studied as an independent trait (Mohammadi-Tighsiah et al., 2018). Moreover, studying genetic aspects of BWG is of importance due to the trade-off between growth and immunity (Rauw, 2012). Except for BWG20-25, the results of the current study showed the significance of maternal genetic and/or environmental effects on early growth traits in Japanese quail. To the best of the authors' knowledge, reports on maternal effects (genetic and environmental) on BWG in poultry are absent.

Nevertheless, measurement simplicity, linear relationship between BWG and feed efficiency (in particular residual feed intake), and their trade-off with immune system performance suggested inclusion of the birds' BWG in the breeding programs. However, due to the statistical significance of maternal genetic effect on BWG (except BWG0-5 and BWG20-25 in the current study), maternal breeding values for these traits should be taken into account.

Direct and Maternal Genetic Parameters for Humoral Immunity

Estimates of genetic parameters for humoral immune responses are presented in Table 2. Titers of immunoglobulins against NDV (IgN) were significantly affected by maternal genetics ($m^2 = 0.098$) whereas the heritability estimate for IgN was 0.040 (m^2 being 2.5 times greater than h^2). Heritabilities for IgY and IgM were low and estimated as 0.079 and 0.035, respectively. Maternal heritability for IgY was almost equal to direct heritability ($m^2 = 0.075$). However, maternal genetic and environmental effects did not significantly influence IgM.

Estimates of genetic parameters for IgN showed more importance of maternal genetic ($m^2 = 0.098$) than direct genetic effect ($h^2 = 0.040$). It is demonstrated that maternally transferred antibodies against NDV were depleted by 10 d of age in commercial broiler chicks (Gharaibeh and Mahmoud, 2013). However, the results of this study might be different from indigenous and less domesticated poultry. Indeed, a mother may have genetically appropriate potential to produce antibodies, but this does not effectively lead to environmentally affect the commercial broiler chicks' humoral immune performance. Whereas we measured IgN titers at 45 d of age (21 d after vaccination), significant m2 refers to the maternal genetic potential against NDV in Japanese quail. On the other hand, mothers with higher maternal breeding values can provide a better immunological environment for their offspring. Therefore, it seems that chickens are more dependent on the maternal genetic potential than their direct additive genetics to improve their humoral immune responses. Another aspect to consider may be the potential interference of maternal antibodies with developing their immune responses to vaccination. Addison et al, examined how differences in neonatal antibodies (IgY) influence immunity at the 2 stages of young and adult in Japanese quail and they reported little evidence for an influence of maternal antibodies on the immune response in the early stage of life (Addison et al., 2010). However, the importance of maternal effect is as equal as a direct additive genetic effect in the case of IgY in our study ($h^2 = 0.079$ vs. $m^2 = 0.075$).

Variation between nature of the studied immune system (adaptive or innate), type of breeds/strains, and time/type of immunization may lead to various heritability estimates in different studies for anti-SRBC immunoglobulin titers. Moreover, humoral immunity in poultry is conveniently partitioned to innate (natural) and adaptive performances; hence, estimates of genetic parameters for these 2 types of humoral immunity would be principally different due to the impact of pathogen/antigen in adaptive humoral immune responses (Bao et al., 2016; Mohammadi-Tighsiah et al., 2018). Study of genetic parameters of anti-SRBC immunoglobulin titers in a medium heavy layer chickens resulted in higher values for direct and maternal heritabilities than our estimates ($h^2 = 0.17$ and $m^2 = 0.15$), because the mean of antibody titer tends to be increased in the highly selected population (Wijga et al., 2009). The heritability estimates for the corresponding trait in a control, H (high SRBC titers), and L (low SRBC titers) lines after 18 generations of divergent selection were 0.203, 0.151, and 0.276, respectively (Bovenhuis et al., 2002). In other words, to select higher immune performances, the effect of genetics was indirectly reduced. Therefore, just emphasizing on increased immune performances in breeding plans may lead to increasing the non-heritable environmental effects. Hence, simultaneous planning to improve growth and immunity would be beneficial.

Many studies have emphasized the significance of maternal effects at the early stages of life. However, maternal effects are still significant for the immune performance of the birds, whereas the birds' immunity is evaluated at later ages. Experimental studies in poultry have revealed that maternally transferred antibodies provide the chick with resistance (Price, 1998). However, chickens receive different levels of antibodies (variation in the chicks' humoral immunity) due to the variation in responses of mothers against antigens. Probably, this part of variation may appear in maternal heritability (Wijga et al., 2009), and somehow in maternal environmental variances as a proportion of total variance (Berghof et al., 2015).

Direct and Maternal Genetic Correlations Between Growth and Immunity

Direct genetic (r_a), maternal genetic (r_m), and environmental (re) correlations between humoral immune responses and BW are shown in Table 3. Estimates of r_a and r_m for BW with IgN and IgY, showed SDs higher than absolute values of correlations, suggested none of the negative correlations were significantly different from 0. Except for IgM-BW, all the genetic and non-genetic correlations between IgN-BW and IgY-BW were negatively estimated. The range of direct genetic correlation between IgN and BW traits was from −0.068 (IgN-BW15) to −0.253 (IgN-BW10). However, there was no a sensible trend for additive genetic correlations with age. Due to the insignificancy of maternal genetics for IgM, r_m was not estimated between BW and IgM. Maternal genetic correlations between IgN-BW0, IgN-BW10, and IgN-BW25 were lower than the corresponding r_a (from −0.018 to −0.104) while r_m was greater than r_a between IgN-BW15 (−0.118 vs. −0.068), and IgN-BW20 (−0.104 vs. −0.089). In general, r_a between IgY-BW were lower than IgN-BW, while both of them were negative and ranged between −0.078 (IgY-BW10) and −0.116 (IgT-BW5). The highest maternal genetic correlation was estimated between IgY and BW25 (−0.309). There was no significant trend between maternal genetic correlations with increasing age. Rather than IgN and IgY, direct additive genetic correlation estimates between IgM and BW were positive. The highest and lowest value was 0.806 (IgM-BW0) and 0.378 (IgM-BW15), respectively.

According to the resource allocation theory, negative estimates for r_a and r_m between BW-IgN and BW-IgY were expected. Despite agreeing with a recent study on Japanese quail Mohammadi-Tighsiah et al. (2018), moderately low to high positive (0.378−0.806) estimates for BW-IgM were unexpected. We measured secondary humoral immune response against SRBC where the IgM

(mean = 1.266) titers became lower than IgY (mean = 2.943) (Table 1). In general, the IgM titer is higher than IgY in primary immunization against SRBC, and in the second immunization, the titers of IgY become larger (Kaiser and Balic, 2015), while the IgM remains at the initial state. In the literature, reports on the estimation of heritabilities for immune performances are scarce. Furthermore, the genetic association between initial body weight and immunity has rarely been studied (Zhang et al., 2018).

Direct genetic (r_a), maternal genetic (r_m), and environmental (r_e) correlations between humoral immune responses and BWG are shown in Table 4. Except for IgY, genetic (direct and maternal) and environmental correlations between BW with IgN and IgM were positive. Direct additive genetic correlations between IgN-BWG are varied from moderate (IgN-BWG0−5 = 0.425) to low (IgN-BWG20−25 = 0.015). A decreasing trend for r_a was considered with age. Maternal genetic correlations between IgN with BWG10−15 (0.230) and BWG15−20 (0.218) are 40% and 9.6% were greater than corresponding r_a (0.164 and 0.104, respectively). The results of Table 4 showed a moderately negative direct genetic correlation between IgY and BWG0−5 (−0.435). All the estimated maternal genetic correlations were lower than the corresponding r_a, which were varied from −0.049 (IgY-BWG10−15) to −0.086 (IgY-BWG5−10). Like BW, genetic and non-genetic correlations between IgM and BWG were positive. BWG0-5 and BWG5-10 were moderately correlated with IgM (0.549 and 0.513, respectively) while BWG20−25 was poorly correlated (0.145). As the authors know, there is not any estimate for the maternal genetic correlation between the birds' humoral immune responses and BWG in the literature. Therefore, the current study can be considered as the first report.

Direct and Maternal Genetic Correlations Between Humoral Immune Responses

Correlations between humoral immune responses (titers of IgN, IgY, and IgM) of Japanese quail are shown in Table 5. All the correlations were positively estimated. A 0.538 direct additive genetic correlation has been estimated between IgN and IgM. Maternal genetic correlation between IgN-IgY (0.339) was more than 2.5 times stronger than the corresponding r_a where r_a was estimated as 0.180. Given that there are various methods to record the immunity performances (Bao et al., 2016), as well as the nature of humoral immunity (adaptive or innate) recording the humoral immune system performance, is fundamentally different from traits such as BW and BWG. As a result, reports on the birds' humoral immune performance (as phenotype) in different studies may be highly variable, which can directly affect the estimates of genetic parameters. For an instance, the genetic correlation between natural IgY-IgM in a commercial layer chicken strain was estimated from 0.02 to 0.89 (Bao et al., 2016). Other factors,

Table 3. Correlations ± SD between body weight traits (BW) and humoral immune responses in Japanese quail.

Trait 1	Trait 2	ra ± SD	rm ± SD	re ± SD
IgN[1]	BW0	−0.245 ± 0.307	−0.109 ± 0.197	−0.058 ± 0.062
	BW5	−0.122± 0.284	–	−0.054 ± 0.052
	BW10	−0.253 ± 0.345	−0.104 ± 0.194	−0.053 ± 0.058
	BW15	−0.068 ± 0.371	−0.118 ± 0.207	−0.067 ± 0.058
	BW20	−0.089 ± 0.308	−0.104 ± 0.191	−0.086 ± 0.057
	BW25	−0.115 ± 0.309	−0.018 ± 0.265	−0.082 ± 0.058
IgY	BW0	−0.108 ± 0.249	−0.074 ± 0.211	−0.042 ± 0.063
	BW5	−0.116 ± 0.291	–	−0.043 ± 0.055
	BW10	−0.078 ± 0.286	−0.072 ± 0.210	−0.039 ± 0.062
	BW15	−0.086 ± 0.308	−0.059 ± 0.213	−0.086 ± 0.063
	BW20	−0.084 ± 0.297	−0.105 ± 0.222	−0.075 ± 0.065
	BW25	−0.096 ± 0.260	−0.109 ± 0.207	−0.061 ± 0.061
IgM	BW0	0.806 ± 0.280	–	–
	BW5	0.744 ± 0.233	–	–
	BW10	0.512 ± 0.248	–	–
	BW15	0.378 ± 0.280	–	–
	BW20	0.418 ± 0.267	–	–
	BW25	0.392 ± 0.266	–	–

[1]IgN: antibody titers against NDV; IgY and IgM: immunoglobulin Y and M titers against SRBC, respectively; BW: body weights of the birds at hatch (0), and 5, 10, 15, 20 and 25 d of age.

Table 4. Correlations ± SD between average daily gains (ADGs) and humoral immune responses in Japanese quail.

Trait 1	Trait 2	$r_a \pm$ SD	$r_m \pm$ SD	$r_e \pm$ SD
IgN[1]	BWG0−5	0.425 ± 0.114	−	0.468 ± 0.128
	BWG5−10	0.234 ± 0.262	0.108 ± 0.102	0.040 ± 0.059
	BWG10−15	0.164 ± 0.243	0.230 ± 0.130	0.066 ± 0.052
	BWG15−20	0.104 ± 0.270	0.218 ± 0.122	0.043 ± 0.041
	BWG20−25	0.015 ± 0.281	−	0.030 ± 0.043
IgY	BWG0−5	−0.435 ± 0.166	−	−0.495 ± 0.128
	BWG5−10	−0.340 ± 0.216	−0.086 ± 0.104	−0.071 ± 0.066
	BWG10−15	−0.401 ± 0.198	−0.049 ± 0.127	−0.097 ± 0.056
	BWG15−20	−0.095 ± 0.302	−0.079 ± 0.106	−0.015 ± 0.046
	BWG20−25	−0.167 ± 0.252	−	−0.004 ± 0.048
IgM	BWG0−5	0.549 ± 0.176	−	0.055 ± 0.237
	BWG5−10	0.513 ± 0.170	−	0.057 ± 0.057
	BWG10−15	0.272 ± 0.212	−	0.002 ± 0.051
	BWG15−20	0.216 ± 0.272	−	0.034 ± 0.043
	BWG20−25	0.145 ± 0.246	−	0.027 ± 0.044

[1]IgN: antibody titers against NDV; IgY and IgM: immunoglobulin Y and M titers against SRBC, respectively; BWG: body weight gain of the birds at 0−5, 5−10, 10−15, 15−20 and 20−25 d' period.

Table 5. Correlations ± SD between humoral immune responses of Japanese quail.

Trait 1	Trait 2	$r_a \pm$ SD	$r_m \pm$ SD	$r_e \pm$ SD
IgN*	IgY	0.180 ± 0.334	0.339 ± 0.315	0.014 ± 0.049
	IgM	0.538 ± 0.283	−	0.082 ± 0.042
IgY	IgM	0.127 ± 0.347	−	0.690 ± 0.581

*IgN: antibody titers against NDV; IgY and IgM: immunoglobulin Y and M titers against SRBC, respectively.

such as breed/strain, pedigree structure, and climate, may also influence the genetic parameters of the birds' humoral immune responses. Genetic correlation for IgY-IgM was recently estimated as 0.34 for Japanese quail (Mohammadi-Tighsiah et al., 2018) which was approximately 2 times greater than our estimate ($r_m = 0.127$). They included information of 3 generations in their study (compared with including information of 6 generations in the current study); therefore, estimating the maternal genetic was not a possible and significant estimate of the maternal genetic correlation between IgN and IgY ($r_m = 0.339$) may have led to this difference. However, the higher estimate for r_m showed more importance of correlated maternal additive genetic effect than the direct additive genetic effect for co-expression of IgN and IgY.

CONCLUSION

Due to its significant effects on early growth and humoral immunity, the maternal effect is suggested to be included in the animal models. Heritability estimates for early growth traits propose that selection for these traits will appropriately improve the birds' body weight and BWG. However, due to lower heritability estimates, genetic improvement based on avian humoral immunity is not readily achieved. Considering positive genetic correlations for BWG-IgN and BWG-IgM as well as the positive genetic correlations between both IgN and IgM with IgY, it is suggested that including the BWG in the breeding programs will result in the direct improvement of the birds' growth performance and indirect improvement of the birds' humoral immune responses. As a result, in most studies BW of birds are usually considered as a representative trait of the birds' growth in both breeding studies and breeding programs, while our study showed that BWG, at least at early stages of life, should not be ignored due to relationship with humoral immunity.

ACKNOWLEDGEMENTS

DISCLOSURES

The authors declare no conflicts of interest.

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