The influence of genetic background versus commercial breeding programs on chicken immunocompetence

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ABSTRACT Immunocompetence of livestock plays an important role in farm profitability because it directly affects health maintenance. Genetics significantly influences the immune system, and the genotypic structure of modern fast-growing chickens has been changed, particularly after decades of breeding for higher production. Therefore, this study was designed to help determine if intensive breeding programs have adversely affected immunocompetence or whether the immune response profiles are controlled to greater extent by genetic background. Thus, 3 indigenous chicken populations from different genetic backgrounds and 2 globally available modern broiler strains, Ross 308 and Cobb 500, were evaluated for various aspects of immune response. These included antibody responses against sheep red blood cells and *Brucella abortus* antigen, as well as some aspects of cell-mediated immunocompetence by toe web swelling test and in vitro blood mononuclear cell proliferation. Significant differences ($P < 0.05$) in antibody responses to both antigens and cellular proliferation were observed among populations but not consistently between modern commercial strains versus the indigenous populations. In fact, the immune response profiles of Cobb 500 were similar to the indigenous populations, but varied compared with the other commercial strain. In addition, considerable variation was recorded between indigenous populations for all responses measured in this study. The results of this study suggest that the variation observed in immune responses between these strains of chickens is most likely due to differences in the genetic background between each strain of chicken rather than by commercial selection programs for high production.

Key words: immune response, indigenous chicken, commercial broiler, genetic background

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INTRODUCTION

Immune responses are crucial for optimal health and can be affected by several factors including host genetics. The effect of genetic background on the immune system has been studied in many vertebrate species, and heritability of some innate and adaptive immune response traits has been reported to range from 0.06 to 0.53 in chickens (Muir and Aggrey, 2003; Wijga et al., 2009), 0.19 to 0.41 in cattle (Thompson-Crispi et al., 2012), and 0.20 to 0.90 in pigs (Flori et al., 2011). For more than 60 yr, commercial chickens have been selected for production traits, such as higher BW. Consequently, the gene pool of commercial chickens has been modified by intensive breeding programs and this has been suggested to associate with decreased immunocompetence (Cheema et al., 2003; Hocking, 2010). Breeding programs of commercial chickens are trademarked company secrets; however, the genetic differences in these chickens is reflected in productive performance and variation in immune responses between commercial strains (Deif et al., 2007; Makram et al., 2010; Coble et al., 2011; Stear et al., 2012; Hong et al., 2012). Broiler chickens have been found to mount stronger cell-mediated immune response and have higher phagocyte activity of macrophages compared with the randombred population. However, in meat-type chickens, antibody responses are lower, suggesting that the immune response may be biased toward cell-mediated immunity in those strains (Cheema et al., 2003). The differences in immune responses also have been compared between broiler and layer chickens. Although both lines were commercially bred for higher production, significant differences were reported on the kinetic of antibody response, as well as the cell-mediated re-

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sponse (Koenen et al., 2002). Study of the immune response on other commercially bred bird species, such as the modern turkey, showed no adverse effect of commercial breeding on the most immune system indicators evaluated (Cheema et al., 2007).

In most of the above-mentioned studies, one genetic line was contrasted to one control line. Therefore, the objective of this study was to compare the immune response profiles of 2 commercial broiler strains and contrast them with those of 3 Iranian indigenous chicken breeds that are genetically distinct and have never undergone selection regimens for production. The hypothesis was that indigenous breeds would exhibit greater immune response than the commercial strains, demonstrating that modern commercial breeding programs for production may adversely affect immune response profiles.

MATERIALS AND METHODS

Chickens

Ross 308 and Cobb 500, which have similar production performance but different genetic backgrounds, were used in this study. One-day-old chicks from registered breeding farms with the same parental age were transferred to the research facility of the University of Tehran. One-day-old indigenous chicks were provided by the Iranian research centers on indigenous chickens from West Azarbaijan, Isfahan, and Mazandran provinces, which are located in the northwest, center, and north of Iran, respectively. Indigenous chicken were transferred to the same research facility as commercial chicks. The detailed history and genetic differences of Iranian indigenous chicken have been studied and reported previously (Shahbazi et al., 2007). To remove the effect of environment, the same management was applied to all birds.

Chickens were kept for 13 d to adapt to the environment before being randomly assigned to 1 of 3 treatment groups. Group 1 consisted of 60 chickens from each population (n = 60 × 5), which were randomly assigned into 2 subgroups to be immunized with 1 of 2 test antigens for assessment of antibody response. Group 2 consisted of 30 birds of each populations (n = 30 × 5) for assessment of toe web swelling test. Group 3 consisted of 30 chickens of each population (n = 30 × 5) for assessment of in vitro blood mononuclear proliferation. Groups 2 and 3 were not immunized.

Antibody Response

Group 1 chickens were immunized intravenously at 14 and 26 d of age with 0.1 mL of undiluted *Brucella abortus* antigen (**BA**; Razi Vaccine and Serum Research Institute, Karaj, Iran) or 0.1 mL of 20% sheep red blood cells (**sRBC**; Jahad Daneshgahi, Tehran, Iran) in PBS. The BA and sRBC are commonly used in chickens to assess immune response as T cell-independent and T cell-dependent antigens, respectively. Serum samples were collected at 4, 8, and 12 d postprimary (d 14) and postsecondary (d 26) immunization. The serum samples were heated at 56°C for 30 min to inactivate complement. Then, an agglutination assay was performed to measure total antibody (Zhou et al., 2001; Cheema et al., 2003). Briefly, a serial dilution (1:2) of each sample was made in a 96-well V-shaped bottom plate and incubated for 30 min at 37°C. Subsequently, 50 μL of 2% SRBC in PBS suspension or 1:10 diluted BA antigen in PBS, depending on the immunizing antigen, were added to each well. The agglutination titer was calculated based on the highest dilution showing 50% agglutination. To measure IgY, a similar agglutination procedure was used, with the addition of PBS supplemented with 0.01 *M* 2-mercaptoethanol to remove mercaptoethanol-sensitive immunoglobulin, IgM (Cheema et al., 2003). The IgM titer for each sample was calculated by subtracting the IgY titer from total antibody titer.

Toe Web Swelling Test

Toe web swelling test was utilized as an indicator of cell-mediated immune response. At 30 d of age, chickens of group 2 received a 100-μL intradermal injection of 100 μg of PHA-P (Sigma-Aldrich, St. Louis, MO) in the toe web (Corrier and DeLoach, 1990). Skin thickness at the spot of injection was measured by a thickness gauge (Insize Co., Suzhou, China) before injection (time 0) and at 24 and 48 h postinjection. The ratio of increase in skin thickness (r) was calculated by dividing the thickness at 24 (D_{24}) or 48 h postinjection (D_{48}) over the thickness at time 0 (D_0), ($r = D_{24\text{ or }48}{:}D_0$). Data on the ratio of the increase of skin thickness were used in the statistical analysis.

In Vitro Blood Mononuclear Cells Proliferation Assay

Blood mononuclear cells (**BMC**) were isolated at 18 and 30 d of age from group 3 and treated with concanavalin A (**Con A**), a lymphocyte mitogen as described previously (Miyamoto et al., 2002). Briefly, a mix of whole blood and RPMI1640 (Invitrogen, Carlsbad, CA) in equal volume were overlaid onto Lymphosep (Biosera, Uckfield, UK), and centrifuged at 800 × *g* for 30 min at 20°C. The isolated cells were washed 2 times with RPMI1640 and then resuspended in RPMI1640-GlutaMax (Invitrogen) supplemented with 10% fetal bovine serum (Biosera) and 1 × Penicillin-Streptomycin Solution (Biosera). Samples were cultured at the final concentration of 2.5×10^5 cells/well in 96-well tissue culture plate and immediately were treated by 12.5 μg/mL of Con A (Sigma-Aldrich). Samples were incubated for 48 h in a 41°C, 5% CO_2 chamber. Four hours before the end of the incubation, 20 μL of 10 mg/mL of

3-[4,5-dimethylthiazol-2-yl]-2,5- diphenyl tetrazolium bromide (Sigma-Aldrich) in PBS were added to each well. At the end of incubation, plates were centrifuged for 10 min at 1,000 × *g* in 4°C and supernatant was removed. To each well, 175 μL of 0.02 *M* HCl in isopropanol was added and plates were shaken for 3 min by microplate ELISA reader (Stat Fax 2000, Awareness Technology Inc., Palm City, FL). The optical density (**OD**) of samples were read at 550 nm and the proliferation index was calculated by dividing the average OD of 5 treated replicates of each sample by the average OD of 3 untreated replicates.

$$\text{Proliferation index} = \bar{X}_{\text{treated}} : \bar{X}_{\text{control}},$$

where $\bar{X}_{\text{treated}}$ is the mean of the OD of samples treated by Con A and $\bar{X}_{\text{control}}$ is the mean of the OD of samples with no treatment.

Statistical Analysis

Data for each measurement of immune response (antibody, nonspecific lymphoproliferation, and BMC proliferation) were analyzed independently with a GLM using PROC MIXED (SAS 9.1.3, SAS Institute, Cary, NC), which included repeated measures. Antibody data were based on agglutination titer, toe web swelling test was based on skin thickness increase, and BMC proliferation data were based on the proliferation index. The best structure was determined based on the variance-covariance matrix with the lowest Akaike information criterion. Covariance parameters were estimated using restricted maximum likelihood and a residual analysis conducted to test the assumptions of ANOVA using PROC UNIVARIATE. Normality was tested using the Shapiro-Wilk test. Residuals were plotted against explanatory variables to determine the need for data transformation. Data were transformed to the natural logarithm to normalize the distribution. The statistical model was

$$y_{ijk} = \mu + p_i + a_j + t_k + e_{ijk},$$

where y_{ijk} = antibody, nonspecific lymphoproliferation or BMC proliferation; μ = overall mean; p_i = fixed effect of the chicken population (5 strains); a_j = fixed effect of immunizing antigen (BA or sRBC); t_k = fixed effect of time; e_{ijk} = residual error.

The fixed effect of immunization (a_j) was removed from the model to analyze the results of toe web swelling test and BMC proliferation as birds were not immunized in these 2 experiments. Significant probability values were reported at $P \leq 0.05$. Interactions were tested, and nonsignificant ($P > 0.1$) interactions were removed. Least squares means were estimated, and a Tukey's test was used to compare contrasts between chicken populations and time points.

RESULTS

Antibody Response

The IgM responses against BA and sRBC were measured every 4 d after primary and secondary immunization for 12 d and the results are presented in Table 1. At d 4 postprimary immunization, the Isfahan and Urmia populations had the highest IgM response to BA, whereas the IgM titers for both commercial strains were less than 1. Although the Mazandran population had a titer of 0 on d 4 postprimary immunization, by d 8 they had the highest titer, which slightly increased by d 12. After secondary immunization, the Urmia population had the highest titer at all the time points compared with all other populations except at d 4, which was not significantly higher than Mazandaran and Cobb 500.

At d 4 after primary sRBC immunization, all the strains except Urmia reached the primary peak of IgM titer. The indigenous populations were among the highest and lowest responders between all populations at all time points. After secondary immunization, the highest IgM titer within the postsecondary immunization time points was observed at d 4 for all populations. Cobb500 and Mazandaran populations had the highest titer. Although there were no significant differences between the 3 other populations, Isfahan had the lowest titer (Table 1). The effect of population, antigen, and population × antigen interaction on IgM titer was statistically significant at all the time points.

The IgY responses to BA and sRBC were measured at the same time points as IgM. Results of the agglutination assay are presented in Table 2. The Isfahan population was the only group to mount an IgY response against BA antigen at d 4 postprimary immunization. At d 8 postprimary immunization, Ross 308 had significantly higher IgY titer than all other populations, and this was maintained for 4 d before secondary immunization (d 12). Conversely, after the secondary immunization, the IgY titers in Ross 308 significantly decreased to about half of the other populations, indicating a higher primary versus secondary response in this strain. At d 8 postsecondary immunization, the Isfahan population had the highest IgY response and at d 12 the Mazandaran and Isfahan populations were significantly higher than other populations. The effect of population, antigen, and population × antigen interaction were significant at all the time points, the same as IgM.

The kinetics of antibody response to BA in Ross 308 was unique among all other strains, and the classical immunoglobulin isotype switching, a mechanism mediated by cytokines that cause an activated B cell to secrete an isotype of immunoglobulin other than IgM to benefit from a different effector mechanism, was not observed in that population (Kindt et al., 2006). The kinetics of antibody response against sRBC dif-

Table 1. Least squares means of IgM (mercaptoethanol-sensitive) anti-sheep red blood cell (sRBC) and anti-*Brucella abortus* (BA) antibody titer[1] of commercial broilers and indigenous chicken ecotype

		Source of variation[2]					
		Days PPI[3]			Days PSI[4]		
Population	Antigen	4[5]	8	12	4	8	12
Ross 308	BA	0.41^{ab*}	0.60^{a*}	1.07^{a}	1.97^{a}	1.43^{a}	1.00^{a*}
Cobb 500	BA	0.79^{ac*}	2.67^{b}	3.47^{bc*}	3.50^{bd*}	2.07^{a}	1.07^{a}
Urmia	BA	1.37^{c}	4.07^{c*}	4.20^{c*}	4.62^{cd*}	2.77^{b*}	1.87^{b*}
Isfahan	BA	1.27^{c*}	2.97^{bd}	3.40^{b*}	2.53^{a}	1.57^{a*}	0.67^{a}
Mazandaran	BA	0.00^{b*}	4.11^{cd*}	4.17^{c*}	3.73^{d*}	1.73^{a}	0.80^{a*}
Ross 308	sRBC	4.00^{a}	1.73^{ac}	0.60^{a}	2.70^{a}	2.10^{a}	1.63^{ac}
Cobb 500	sRBC	4.14^{a}	3.40^{b}	1.83^{b}	5.34^{b}	2.13^{a}	0.77^{b}
Urmia	sRBC	0.93^{b}	1.87^{ac}	0.53^{a}	3.31^{a}	1.60^{bd}	1.13^{c}
Isfahan	sRBC	5.90^{c}	3.10^{bc}	1.63^{b}	2.53^{a}	0.77^{c}	0.53^{bc}
Mazandaran	sRBC	2.83^{c}	2.37^{c}	0.87^{a}	5.00^{b}	2.13^{a}	2.46^{d}

$^{a–d}$Within a column for the same antigen, means with no common superscripts indicate significant difference between population ($P < 0.05$).

[1]Titers were obtained from the agglutination assay.

[2]The R^2 value of the statistical model was 0.50.

[3]PPI = postprimary immunization.

[4]PSI = postsecondary immunization.

[5]Numbers represent the number of days after each immunization.

*Significant differences within each time point and population between 2 antigens ($P < 0.05$).

fered from that observed for BA (Tables 1 and 2). No populations had IgY titer against sRBC of greater than 1 before the second immunization. Both commercial strains, Ross 308 and Cobb 500, had significantly lower IgY responses compared with the indigenous chickens at d 4 postsecondary immunization. Again, the Isfahan population had the highest response at d 8 postsecondary immunization and both the Isfahan and Urmia population had higher responses against sRBC at 12 d after secondary immunization. The classical immunoglobulin class switching was only observed in Isfahan and Urmia populations.

Toe Web Swelling Test

As an indicator of cell-mediated immune response, a toe web swelling test was performed and the results are presented in Figure 1. The greatest increase in skin thickness was observed in Cobb 500 strain at 24 h postinjection, compared with all other populations. The Isfahan and Mazandaran populations had the lowest response at both time points, 24 and 48 h. After 48 h, the Ross 308, Cobb 500, and Urmia populations had significantly higher proliferation response compared with the Isfahan and Mazandaran populations.

Table 2. Least squares means of IgY (mercaptoethanol-resistant) anti-sheep red blood cell (sRBC) and anti-*Brucella abortus* (BA) antibody titer[1] of commercial broilers and indigenous chicken ecotypes

		Source of variation[2]					
		PPI[3]			PSI[4]		
Population	Antigen	4[5]	8	12	4	8	12
Ross 308	BA	0.00^{a*}	4.07^{a*}	4.30^{a*}	3.07^{a}	2.44^{a}	2.21^{a*}
Cobb 500	BA	0.00^{a}	0.44^{b*}	2.37^{b*}	6.37^{b*}	5.13^{b*}	4.37^{b*}
Urmia	BA	0.00^{a}	0.07^{c}	1.47^{c*}	6.07^{b}	5.40^{b*}	4.47^{b*}
Isfahan	BA	0.30^{b}	1.96^{d*}	2.83^{b*}	6.00^{b}	6.13^{c*}	5.28^{c*}
Mazandaran	BA	0.00^{a}	0.53^{bc*}	2.20^{b*}	6.63^{b*}	5.33^{b*}	4.70^{bc*}
Ross 308	sRBC	0.38^{a}	0.03^{a}	0.17^{a}	2.69^{a}	1.79^{a}	1.17^{a}
Cobb 500	sRBC	0.07^{b}	0.03^{a}	0.23^{a}	3.45^{a}	2.97^{a}	1.90^{b}
Urmia	sRBC	0.07^{b}	0.30^{a}	0.10^{a}	6.11^{b}	3.83^{b}	3.07^{c}
Isfahan	sRBC	0.08^{b}	0.50^{a}	0.97^{b}	6.00^{b}	4.90^{c}	3.55^{c}
Mazandaran	sRBC	0.00^{b}	0.00^{a}	0.00^{a}	4.27^{c}	3.00^{a}	2.23^{b}

$^{a–d}$Within a column for the same antigen, means with no common superscripts indicate significant difference between population ($P < 0.05$).

[1]Titers were obtained from the agglutination assay.

[2]The R^2 value of the statistical model was 0.80.

[3]PPI = postprimary immunization.

[4]PSI = postsecondary immunization.

[5]Numbers represent the number of days after each immunization.

*An asterisk indicates a significant difference within each time point and population between 2 antigens ($P < 0.05$).

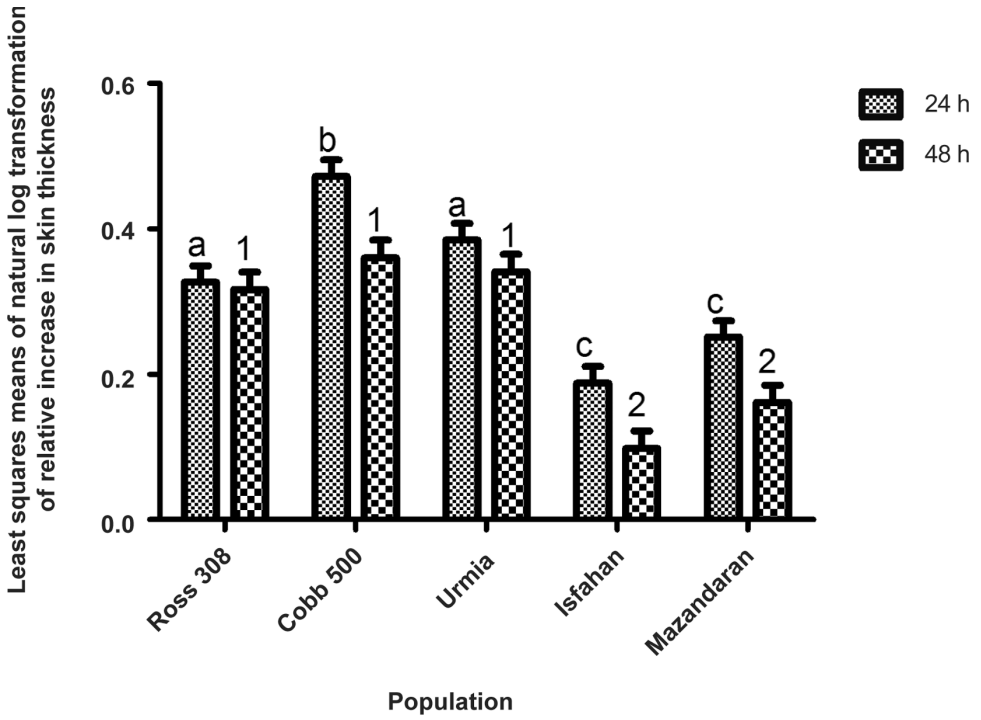

Figure 1. Least squares means of the relative increase in skin thickness at 24 and 48 h post-phytohemagglutinin-protein injection. Different letters (a–c) above each column indicate a significant difference at 24 h between populations ($P < 0.05$). Different numbers (1, 2) above each column indicate a significant difference at 48 h between populations ($P < 0.05$).

In Vitro Blood Mononuclear Cell Proliferation Assay

The results of the proliferation assay are presented in Figure 2. At both time points, 14 and 28 d of age, a large difference was observed between the commercial strains in response to Con A. At d 14, whereas the Cobb 500 population had the highest proliferation response to Con A, Ross 308 had the lowest response between all populations. Between indigenous populations, Mazandaran chickens showed significantly higher response than the 2 other populations. The effect of age was significant in this experiment. The stimulation index increased significantly from 14 to 28 d of age in the Ross 308, Urmia, and Isfahan populations. At the second time point, the Cobb 500 and Urmia populations had the highest proliferation response compared with other populations, whereas Ross 308 had the lowest response.

DISCUSSION

Since the late 20th century, many studies have been conducted to explore the effect of intensive breeding programs, based primarily on production, on the immune system performance of commercial chicken strains (Han and Smyth, 1972; Martin et al., 1988; Qureshi and Havenstein, 1994; Bacon et al., 2000; Cheema et al., 2007). A meta-analysis study, mostly on resistance to Marek's disease (**MD**), indicated a significant decrease of immune function, as measured by mortality rate after challenging with MD virus or antibody response to sRBC, or both, in a chicken line that had been experimentally selected for higher BW (Van der Most et al., 2011). Immunocompetence of Arbor Acres and Ross 308 strains, which have been selected on a commercial scale, were compared in 2 studies with the Athens-Canadian Randombred Control chicken population, which had not undergone any recent selection program. The authors concluded that some immunological traits, such as antibody response to sRBC in modern broilers, are negatively affected by genetic selection for higher production performance in chickens, whereas there is either no effect or a positive effect on inflammatory and cell-mediated immune response (Qureshi and Havenstein, 1994; Cheema et al., 2003).

Although the above-mentioned studies found negative associations of breeding for enhanced production with immune response in chickens, this is in contrast to the finding of the current study. These differences may be attributed to the various experimental designs and study limitations, including the absence of relevant control randombred populations with similar genetic background to commercial modern broilers. This limitation becomes apparent when only one commercial breed is contrasted to just one control breed. The second problem is the unavailability of detailed procedures of commercial breeding programs, when experimentally selected chickens were used in the studies to show the effect of breeding program on immune response. The current study attempts to overcome the first limitation by evaluating the effect of genetic background on

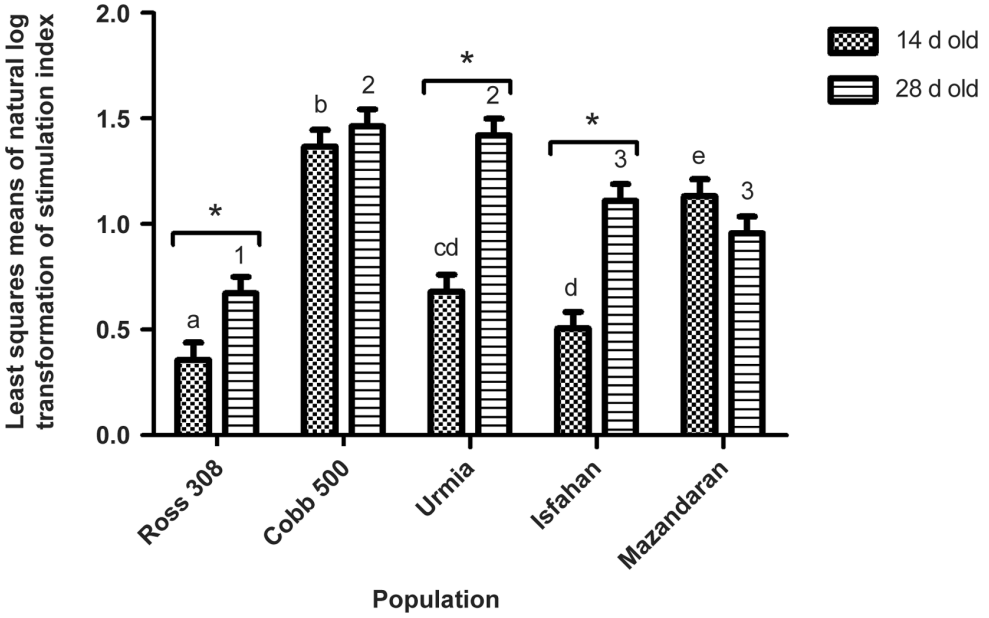

Figure 2. Least squares means of the stimulation index of chicken blood mononuclear cells at 14 and 28 d of age when stimulated by concanavalin A. Different letters (a–e) above each column indicate a significant difference at the age of 14 d ($P < 0.05$). Different numbers (1–3) above each column indicate a significant difference at the age of 28 d ($P < 0.05$). *An asterisk represents a significant difference within each population between 2 time points ($P < 0.05$).

a range of host defense mechanism in 5 populations of chickens, including 2 commercial strains and 3 indigenous populations. This design allows for the comparison of the influence of genetic background on immune response traits between strains with different backgrounds, as well as the effect of the breeding program on immune response, by comparing overall differences in commercial versus indigenous chickens.

Antibody response is important in control of a wide range of economically important pathogens, such as *Escherichia coli* and *Salmonella*. Therefore, antibody titer can be used as one index of immunocompetence (Liu et al., 2001; Lynne et al., 2006; Thompson-Crispi et al., 2013). The results of the current study did not indicate consistently higher or lower titer of IgM or IgY against BA in commercials broilers versus indigenous populations, whereas significant differences were observed between Ross 308 and other populations (Tables 1 and 2). These results are in contrast with the previous studies that compared antibody responses against sRBC in Athens-Canadian Randombred Control chickens with Arbor Acres and Ross 308 (Qureshi and Havenstein, 1994; Cheema et al., 2003) where the commercial breeds showed the lower response. These differences may be partly due to the nature of the test antigen because in the previous studies, sRBC were used as the antigen. The BA antigen is a polysaccharide and the antibody response mechanisms against polysaccharides are T cell-independent rather than T cell-dependent responses that govern sRBC (Karaca et al., 1999). Thus, it may be possible that the T cell-independent antibody response pathway is not necessarily negatively associated with selection for higher BW due to similar response to BA between Cobb 500 and indigenous populations.

The antibody response against sRBC also did not result in a clear-cut differentiation between commercial strains versus indigenous populations, except at the first time point after postsecondary immunization (Table 2). Again, these results contradict results of the study by Cheema et al. (2003), which reported lower IgY titer against sRBC in commercial strains compared with randombred populations. An important difference between the studies relates to the effect of age on immune system maturation and the time points when immune response was examined. Studies on completion of spleen colonization and cytokine expression in early days posthatch revealed more reactivity of the spleen cells to T cell-dependent antigens. Significantly higher antibody responses were noted in chicken that were immunized between 12 and 14 d of age in contrast to chickens that were immunized at 7 d of age or younger (Dunon et al., 1997; Mast and Goddeeris, 1999; Abdul-Careem et al., 2007). In the current study, chickens were immunized at 14 d of age, whereas in the other study chickens received the first dose of antigen at 6 d of age (Cheema et al., 2003), which may explain the lower IgY titer of the primary response. Hence, the variation of IgY against sRBC as a T cell-dependent antigen may align with maturation of the immune system of younger chickens.

The other notable finding in antibody response against sRBC was a distinct variation in titer of IgM at all time points between indigenous populations (Table 1). These results are in agreement with the study by Baelmans et al. (2005) who reported large variation in

antibody response against sRBC in indigenous chickens originating from India, Tanzania, and Bolivia.

Although some of the results from the current study indicate lower antibody titers for one commercial strain versus one indigenous population at a certain time point (i.e., at 8 d PPI, IgY response against BA between Cobb 500 and Isfahan population, or at 8 d PPI, IgY anti sRBC between Ross 308 and Isfahan population), the high-low pattern was not consistent between other populations. Overall the findings indicated greater differences between the 2 commercial strains. By including all commercial and indigenous populations in the comparison, genetic background seems to affect antibody response more than selection programs. In addition, classical antibody isotype switching after secondary immunization with BA did not happen in the Ross 308 strain. Secondary immunization with sRBC activated the isotype switching only in 2 indigenous populations, Urmia and Isfahan. These results suggest that intense breeding programs are not likely the source of variation in the kinetics of antibody response because no similar kinetics within either commercial strains or indigenous populations were observed.

In the current study, a toe web swelling test was used to compare the capacity of T cell response between chicken populations, as T cells play a key role in adaptive immune response. The results indicate that the Cobb 500 had the highest response, which is similar to findings by Makram et al. (2010; Figure 1). In that study, the same assay was used to compare 4 commercial chicken strains including Avian, Arbor Acres, Hubbard, and Cobb, and the results showed that the Cobb chickens had the highest response compared with all other strains (Makram et al., 2010). The current study also showed the Urmia population had slightly higher response than Ross 308 and significantly lower than Cobb 500 at 24 h post-PHA-P injection and there was no significant difference between these 3 populations at the second time point (Figure 1). These results do not support the proposed hypothesis that intensive commercial breeding for production has adverse effects on immunity but rather the idea that the genetic background has a greater effect on the immune system than selection for higher BW, at least for this indicator of cell-mediated immune response.

In addition to the toe web swelling test, an in vitro BMC proliferation assay was used as a second indicator of T cell proliferation. The results of the in vitro experiment at 28 d of age were similar to the toe web swelling test results at 24 h as Cobb 500 and Urmia populations had the highest proliferation response such that the ranking of population were similar (Figure 2). The only difference was observed in the Ross 308 strain that had the lowest stimulation index when BMCs were stimulated by ConA, but it was among high responders when PHA-P was used in toe web swelling test. Several reasons may contribute to this difference: first, the potency of mitogen, Con A versus PHA-P; and second, many factors in the chemotaxis pathway such as adhesive molecules on the surface of endothelial cells are absent in the in vitro experiment, which play a crucial role in response to the toe web swelling test.

In conclusion, by comparing differences within and between 2 commercial strains and 3 indigenous populations of chickens, the results of this study suggest that the genetic background of the strain plays a more significant role in the regulation of immune responses than the effect of intensive selection programs for increased production. Similarly, immunocompetence studies of turkeys using randombred populations with similar genetic background found no significant differences between commercial and randombred control populations in a turkey pullet population in antibody response against sRBC (Li et al., 2000; Cheema et al., 2007). Therefore intensive commercial breeding programs do not appear to adversely affect immunocompetence in poultry.

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