

SELECTION FOR GENERAL IMMUNOCOMPETENCE IN CHICKENS

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SUMMARY

This experiment was designed to study selection for general immunocompetence by using a selection index. A total of 460 chicks from 20 sire families (randomly divided into 2 replicates of 10 sire families each) of a random breeding egg-laying chicken line (Ottawa strain 7) were used as the base population. Three *in vivo* assays (carbon clearance assay, PHA wing web assay and ELISA) were used to evaluate phagocytic activity (PI), cell-mediated response (WI) and antibody production to 2 vaccines, *Pasteurella multocida* and *Mycoplasma gallisepticum* (α -PM and α -MG), respectively. Heritabilities of these traits were estimated to be from 0.06 to 0.53. A selection index was constructed with the assumption that genetic correlations between these traits are negligible. The economic value of the traits were calculated to be negatively proportional to their respective heritability estimates on the assumption that equal selection pressure would be applied to each trait. After two generations of selection, significant differences were observed between high and low response sublines for anti-MG and WI. Analysis of additional generations will determine the feasibility of multi-trait selection for immunocompetence to increase general disease resistance in poultry.

INTRODUCTION

Genetic control of immune response and disease resistance is well-established in humans and other animal species. The presence of genetic variations in disease resistance provides the bases for the improvement of disease resistance by conventional selection methods. Resistance to specific diseases in farm animals has been demonstrated by genetic selection, but selection for resistance to every specific disease in farm animals may be impossible. Another approach to improve the resistance ability of farm animals is to select for general disease resistance. Among the most important of all the defensive mechanisms of animals is the immune response. Genetic control of the immune response of an individual consists of three major facets: phagocytosis, T-cell mediated immunity and antibody response. The coordination of these systems enables an individual to resist infection and disease. Biozzi *et al.* (1982) successfully selected for high and low levels of antibody production in mice using the bidirectional selection method. Divergent selection for antibody production to various antigens in chickens was also effective (Siegel *et al.*, 1980; Pevzner *et al.*, 1981; Cahaner *et al.*, 1986). Gyles *et al.* (1986) estimated the heritabilities of the antibody titers from 0% to 40%. Buschmann *et al.* (1985) investigated both cellular and humoral immunity of pigs and concluded that indirect selection for general disease resistance based on immune response traits may be possible.

The objectives of this experiment were to evaluate quantitative immunological parameters including heritabilities of, and phenotypic correlations among, immune response traits; to construct selection indices incorporating the three major components of the immune system; to study the feasibility of application of an immune response index selection to a commercial breeding program; and to provide genetically divergent populations

for subsequent testing of correlations of immune response and disease resistance by disease challenge.

MATERIAL AND METHODS

Animals. Ottawa strain 7 chickens from the Animal Research Center, Agriculture Canada, were used. This strain originated from four commercial stocks of White Leghorn chickens in North America and was used as the control strain for long-term selection for egg production (Gavora *et al.*, 1986). A total of 460 chickens were tested in the base population and about 500 birds were tested in subsequent generations. Selection of replicate lines was based on high (1H, 2H) or low (1L, 2L) immune response breeding values determined by a selection index.

In vivo assays. Three assays were used to evaluate the immunocompetence of the chickens. They were selected to be *in vivo* assays having no deleterious effects, and to be easy, quick, and economical so that sufficient numbers of chickens can be evaluated to obtain quality estimates of the genetic parameters needed in the construction of selection index. The enzyme-linked immunosorbant assay (ELISA) was used to evaluate antibody responses to *Pasteurella multocida* vaccine (α -PM) and *Mycoplasma gallisepticum* vaccine (α -MG). Vaccine to PM was administered at 5 weeks of age; to MG at 6 weeks of age. Blood samples were taken immediately pre- and 3 weeks post-immunization. Antibody levels were determined by ELISA test kits. The phytohemagglutinin (PHA) wingweb assay, carried out between 10 to 12 weeks of age, was used to evaluate *in vivo* cell-mediated immune response of chickens (van der Zijpp 1983, Lamont and Smyth 1984), which was expressed as a wingweb index (WI). The carbon clearance assay (Glick *et al.* 1964), carried out between 13 to 15 weeks of age, was used to evaluate *in vivo* phagocytic activity of chickens, which was measured by a phagocytic index (PI).

In vitro assays. Three *in vitro* assays were also selected to evaluate the three facets of the immune system. The plaque-forming cell assay (Jerne, 1963) was used to estimate antibody response to SRBC (PFC), the T-cell mitogenesis assay (in response to 2, 4, and 8 μ g of PHA-P) (van der Zijpp, 1983) was used to measure cell-mediated immunity (PHA2, PHA4, and PHA8), and monocyte/macrophage phagocytosis assay was used to determine the phagocytic activity of chickens (MONO). A total of 86 birds randomly selected from the base population were tested. These assays were compared and correlated with the three *in vivo* assays.

RESULTS

Heritability. Heritabilities of the immunological traits were estimated from the sire components by $h^2 = 4\sigma_s^2 / (\sigma_s^2 + \sigma_d^2 + \sigma_e^2)$ (Falconer, 1981) for the base population and for subsequent generations when more data became available so that they could be updated for the next generation of selection (Table 1). The variance components were estimated by both analysis of variance (ANOVA) and maximum likelihood (ML) methods.

Selection index. The coefficients (Table 4) in the selection index were calculated from the normal equation $Pb = Ga$; where P was the phenotypic variance and covariance matrix (Table 2); G was the genetic variance and covariance matrix (Table 3); a was the vector of 'weight' of the immunological traits (Table 1) and was calculated as $a_i = T/h_i^2$, where

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 van der Zijpp, A. J., 1983. Poultry Sci. 62:205-211.

Table 1. Heritability estimates and economic weight of base population.

Trait	h^2_{ANOVA}	h^2_{ML}	a
α -PM	0.15	0.12	4.53
α -MG	0.53	0.50	1.28
PI	0.28	0.28	2.41
WI	0.06	0.06	11.33

Table 3. Genetic variance and covariance matrix of base population.

	PI	WI	α -PM	α -MG
PI	.0060	0.0	0.0	0.0
WI		0.0119	0.0	0.0
α -PM			0.0162	0.0
α -MG				1.29

Table 5. Least square means of *in vivo* immunological traits.

generation	2				3			
	PI	WI	α -PM	α -MG	PI	WI	α -PM	α -MG
1H	0.353 ^b	1.56 ^{bc}	0.525 ^{ab}	1.26 ^{bc}	0.338	1.64 ^a	/	2.50 ^b
1L	0.430 ^a	1.61 ^{ab}	0.587 ^a	1.11 ^c	0.349	1.39 ^b	/	2.26 ^b
2H	0.376 ^b	1.49 ^c	0.512 ^{ab}	1.74 ^a	0.350	1.43 ^b	/	2.96 ^a
2L	0.374 ^b	1.69 ^a	0.407 ^b	1.46 ^b	0.353	1.47 ^{ab}	/	2.55 ^b

Values within each column with no common superscripts are significantly different (P<.05).

Table 6. Phenotypic correlations between *in vivo* and *in vitro* assays.

	WI	PI	α -PM	α -MG
MONO	-0.37 ^{**}	0.19	0.11	0.21
PFC	-0.11	-0.15	0.09	0.05
PHA8	-0.12	0.11	0.05	0.05
PHA4	-0.22 [*]	0.11	0.01	0.07
PHA2	-0.23 [*]	0.14	-0.03	0.18

^{*}P<.05

^{**}P<.01

Table 2. Phenotypic variance and covariance matrix of base population.

	PI	WI	α -PM	α -MG
PI	.0215	.00039	-.00048	.0252
WI		.199	-.0088	.0419
α -PM			.108	.0769
α -MG				2.434

Table 4. Coefficients of selection indexes used in each generation of selection.

trait	gen 1	gen 2	gen 3
PI	-0.133	1.447	0.357
WI	0.834	1.851	0.665
α -PM	0.258	1.287	/
α -MG	0.686	1.173	0.785

Table 7. Phenotypic correlations between immunological traits measured by *in vivo* assays.

	WI	α -PM	α -MG
PI	0.006	-0.01	0.11
WI		-0.06	-0.06
α -PM			0.15 ^{**}

^{**}P<.01

$T = h_{PI}^2 + h_{WI}^2 + 1/2(h_{\alpha-PM}^2 + h_{\alpha-MG}^2)$; i was any of the traits.

Least square means. Table 5 presents the least square means of sublines after each generation of selection.

Correlations. Phenotypic correlations between *in vivo* and *in vitro* assays were calculated for the base population (Table 6). Phenotypic correlations between immunological traits were also calculated (Table 7).

Production performance. The production performance of the selected sublines were monitored in the experiment. No significant differences between sublines were observe (results not shown).

DISCUSSION

Heritabilities of the antibody responses of the base population were estimated from 0.12 for α -PM to 0.53 for α -MG. Estimates from the pooled data of subsequent generations ranged from 0.25 to 0.46. These values are similar to the estimates obtained by Gyles *et al.* (1986). There were no references available on the heritability estimates of cell-mediated response and phagocytosis. Their heritabilities were estimated to be from 0.06 to 0.36, implying that greater environmental components are present and selection progress will be slow. The results after two generations of selection showed that selection for antibody response, especially for α -MG which had the highest heritability estimate, was indeed more successful than selection for the other two traits.

Phenotypic correlations between immunological traits measured by *in vivo* assays were generally low in value, similar to the results obtained for another egg-laying line (Cheng and Lamont, 1988). Correlations between *in vitro* assays showed similar trend (results not shown). These data suggest that simultaneous improvement in the three facets of the immune system by index selection should be possible. There were significant negative correlations between *in vivo* PHA injection assay and *in vitro* PHA mitogenesis assay (Table 6), although both assays are thought to measure cell-mediated immune response. This may be due to the involvement of other cells in *in vivo* assay.

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