Genetic Variations Of Aggression In Thai Indigenous Chicken

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Introduction

Aggression is habitually related to competition for survival, reproduction and also social behavior purposes (Duncan, 1998). In poultry production, aggression causes increased stress, injury, and lead to mortality. However, the aggressive behavior is the most characteristic needs in Fighting chicken. In Southeast Asia, such as Thailand, Vietnam, Burma, Malaysia, etc., most fighting chicken are selected and trained from Indigenous chicken. Selection for high aggression has been challenged because of the complicate genetic control and trait measurements. The genetic variation of aggressive behavior in chicken has been seldom reported either in terms of quantitative or molecular study. Monoamines have been found associated to aggressive behavior Berman and (Coccaro, 1998). Tryptophanhydroxylase (TPH) related to serotonin synthesis in the brain (Popova, 2008). In addition, some reports revealed that polymorphisms of the intron of this gene associated to the aggressive in broilers (Shea et al., 1990) and human (New et al., 1998). Dopamine is another monoamine involving in several behavior in domestic animals. Flisikowski et al. (2008) reported that point mutations at 5' flanking and exon 2 of dopamine receptor D4 (DRD4) associated with the pecking behavior. The objective of this study was to estimate the genetic parameters and genetic polymorphisms of some candidate genes associated with the aggression in Thai Indigenous chicken.

Material and methods

Animals and data. A total of 3,345 Thai Indigenous chicken from two generations were examined for aggressive and non-aggressive behaviors. Records were obtained from the Research and Development Network Center for Animal Breeding (Native Chicken), Khon Kaen University, Thailand.

DNA Isolation, PCR and SSCP. Blood samples from aggression (n = 74) and non-aggression (n = 50) chicken were collected for isolating genomic DNA. TPH and DRD4 primers were designed from Genbank accession number U26428 and FJ217173, respectively, using PRIMER III Primer 3 (http://wwwgenome.wi.mit.edu/cgi-bin/primer/primer3.cgi). The reaction was carried out in a total volume of 10 µl containing: 50 ng of genomic DNA (1 µl of 50 ng/µl genomic DNA), 1xPCR buffer (1 µl of 10xPCR buffer), 1 mM dNTP (1 µl of 10 mM dNTP), 5 mM MgCl2 (1 µl of 50 mM MgCl2), 0.5 mM

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of each primer (1 μl of 5 mM of each primer) and 0.5 unit of Taq DNA polymerase (Promega, San Diego, CA). Reactions were performed in a 96-well thermal cycler (GeneAmp PCR System 9600, Perkin-Elmer Applied Biosystems, CA) according to the following cycling profile: initial denaturation at 94 ^{o}C for 5 min.; 35 cycles of 94 ^{o}C for 30 s., 60 or 64 ^{o}C 45 s, and 72 ^{o}C 45 s.; and final extension at 72 ^{o}C for 5 min. Single Strand Conformation Polymorphism (SSCP) of the PCR products were continued to perform in 5% non-denaturing polyacrylamide gel electrophoresis (40% acrylamide:bis-acrylamide **(** 19:1, Bio-Rad Laboratories**)** with 0.2% glycerol)

Statistical analyses. (Co)variance components for Aggressive behavior (AB) was analyzed by restricted maximum likelihood (REML) using AIREMLF90 (Misztal et al., 2002) in BLUPF90-WINPAK (Duangjinda et al., 2010). The analysis model was described as follows:

$$\boldsymbol{y} = \boldsymbol{X\beta} + \boldsymbol{Za} + \boldsymbol{Wc} + \boldsymbol{Sf} + \boldsymbol{\varepsilon}, \qquad Var\begin{bmatrix} \boldsymbol{a} \\ \boldsymbol{p} \\ \boldsymbol{f} \\ \boldsymbol{\varepsilon} \end{bmatrix} = \begin{bmatrix} \boldsymbol{A}\sigma_a^2 & 0 & 0 & 0 \\ 0 & \boldsymbol{I}\sigma_c^2 & 0 & 0 \\ 0 & 0 & \boldsymbol{F}\sigma_f^2 & 0 \\ 0 & 0 & 0 & \boldsymbol{I}\sigma_e^2 \end{bmatrix}$$

Where $\boldsymbol{y}$ is the vector of aggressive behavior, $\boldsymbol{\beta}$ is a vector of fixed effects of contemporary group (CG) of genration and hatch of birth, sex, and age, $\boldsymbol{a}$ is the vector of random additive genetic effects, $\boldsymbol{c}$ is the vector of random maternal permanent environment effects, $\boldsymbol{f}$ is the vector of random parental dominance effects, $\boldsymbol{\varepsilon}$ is the vector of random residual effects and $\boldsymbol{X}$, $\boldsymbol{Z}$,$\boldsymbol{W}$ and $\boldsymbol{S}$ are the known incidence matrices, $\boldsymbol{A}$ is the additive numerator relationship matrix, $\boldsymbol{F}$ is the parental dominance relationship matrix, σ_a^2, σ_c^2, σ_f^2 and σ_e^2 are the additive, maternal permanent environment, parental dominance and residual variances, respectively.

The association between aggressive behavior and SSCP patterns of TPH and DRD4 genes was tested by Chi-square.

Results and discussion

Estimates of genetic parameters and of aggressive behavior in Thai Indigenous chicken was shown in table 1. This study showed that heritability value for aggression was relatively low (0.11) compared to production traits. Maternal permanent variance ratio about 10% showed that this effect had similar magnitude to additive genetics on aggressive variation. Interestingly, this study revealed that non-additive genetics played a major role controlling the variations of chicken aggression. It was found that about 40% of this behavior was controlled by dominance. Therefore, crossing between specific sire and dam to get the

preference offspring genetics, so called “mate selection”, should be considered in breeding program for improving aggressive behavior in Thai native chicken.

Table 1: Estimates of genetic components of aggressive behavior for Thai Indigenous chicken

Parameter[α]	Estimate	S.E.
σ_a^2	0.0238	
σ_c^2	0.0223	
σ_d^2	0.0868	
σ_e^2	0.0823	
h^2	0.1104	0.0039
c^2	0.1036	0.0072
d^2	0.4036	0.0065

[α] σ_a^2 = additive genetic variance, σ_c^2 = maternal permanent variance, σ_d^2 = dominance variance = $4\sigma_f^2$, σ_e^2 = error variance, h^2 = heritability, c^2 = maternal permanent environment variance ratio, d^2 = dominance variance ratio.

SSCP polymorphisms of in TPH intron and DRD4 exonII showed genetic variations in monoamine genes. The associations of TPH pattern 2 and DRD4 pattern 3 with the aggression were significantly found (Table 2). Dennis et al. (2008) reported that different levels of serotonin found in high and low aggressive chicken lines. In addition, Cheng and Muir (2007) also reported that the variations of blood cathecolamines including serotonin and dopamine were genetically different in high and low aggression chicken lines.

Table 2: Proportion of SSCP pattern associated with aggressive behavior in Thai Indigenous chicken

	N	Agressivee	Non-aggressive	χ^2	Significant
TPH					
- Pattern 1	111	0.688	0.312	15.66	**
- Pattern 2	13	0.583	0.417	0.36	NS
DRD4					
- Pattern 1	20	0.667	0.333	2.22	NS
- Pattern 2	23	0.513	0.487	0.02	NS
- Pattern 3	42	0.833	0.167	18.67	**

Conclusion

This study reported genetic parameters for aggressive behavior in Thai Indigenous chicken. The heritability and maternal permanent environment variance ratio is about 10% while the dominance variance ratio is about 40%. It implied that mothering ability in terms of maternal permanent environment and the non-additive genetic effect plays major roles in controlling the aggression in offspring. The selection for this trait should be stressed on mating selection instead of direct breeding value selection. This study also found the genetic variation in TPH and DRD4 genes and the association with the aggression in Thai indigenous chicken which might be useful for marker assisted selection in breeding program.

Acknowledgments

The authors thank the Research and Development Network Center for Animal Breeding (Native Chicken) for providing the data and Agricultural Biotechnology Center for Sustainable Economy for partial funding in this project.

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