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INFLUENCE OF ASCITES SYNDROME ON GROWTH PATTERN OF CHICKENS REARED AT NORMAL OR COLD AMBIENT TEMPERATURE

Karim Hasanpur[1], Mohammadreza Nassiry[1♦], Ghasem Hosseini Salekdeh[2], Rasul Vaez Torshizi[3], Abbas Pakdel[4], Hasan Kermanshahi[1]

[1]Department of Animal Science, Faculty of Agriculture, Ferdowsi University of Mashhad, Iran
[2]Agricultural Biotechnology Research Institute of Iran (ABRII), Karaj, Iran
[3]Department of Animal Science, Faculty of Agriculture, University of Tarbiat Modares, Karaj, Iran
[4]Department of Animal Science, Isfahan University of Technology, Isfahan, Iran
♦Corresponding author: Nassiryr@um.ac.ir

Abstract

Ascites syndrome (AS) is a metabolic disorder usually seen in highly improved meat-type broiler strains. This syndrome causes major financial losses to the poultry industry. Previously it was believed that AS incidence was a side effect of intense selection for rapid growth rate (GR) or higher market weight. If this belief is the case, selection for further increase in GR will not be rational. However, it was later understood that there were significant genetic variations for both GR and susceptibility/resistance to AS; thus selective breeding could be helpful in diminishing the incidence of AS while improving GR. Furthermore, it was hypothesized that genes controlling the GR were not genetically dependent on those genes controlling susceptibility to AS. In the current research, we aimed to study the association of GR traits with AS% in a pure sire line. A total of 1458 1-day-old chicks from 67 sire families were used. The results revealed that ascitic chicks were not significantly superior in early GR traits (i.e. before day 28) than the healthy ones. At later ages, probably due to the commencement of the syndrome, the ascitic chicks were significantly lighter in body weight (BW) than their healthy counterparts. The lack of significant genetic correlations between the GR traits and AS% indicated that there was considerable scope for simultaneous selection of birds for increased BW and GR while controlling susceptibility to AS.

Key words: ascites syndrome, Richards function, genetic correlation, growth curve

Ascites syndrome (AS) is a metabolic disorder of meat-type lines/hybrids with improved feed conversion ratio (FCR) and rapid growth rate (GR) (Wideman, 2013; Druyan et al., 2008, 2009; Huchzermeyer, 2012; Franciosini et al., 2012). AS results in significant direct economic losses to the broiler industry due to (i) the ruined feed efficiency of the flock, (ii) higher mortality rate at farm, especially at later ages, (iii) higher mortality during the transport of the chicks to the slaughterhouse, and

(iv) higher condemnation of processed carcasses at slaughterhouse. Furthermore, because of the management activities which are usually implemented to control AS at the farm level (i.e. applying feed restriction regimes, alternative lighting programs, using feed additives such as antioxidants, vitamins, etc), the maximum growth potential of broilers could not be fully achieved (Julian, 1993; Solis de los Santos et al., 2005; Guo et al., 2007; Boostani et al., 2010). Therefore, the profitability of broiler farming lessens considerably.

It was traditionally believed that AS was a side effect of intense selection for growth-related traits, because AS was more prevalent in broiler chickens. Furthermore, it was claimed that the relative growth of chicken's internal organs (e.g. lung and heart) of heavy-weight lines was considerably lower than their unimproved ancestors (Vidyadaran et al., 1990; Scheele et al., 2005; Huchzermeyer, 2012). Therefore the cardiopulmonary capacity of the modern rapid-grower broilers has become unable to meet the increasing demand for oxygen which is triggered by higher GR.

Wideman (1998) suggested that AS develops in broilers in which GR exceeds the rate at which their pulmonary vascular capacity increases, but these broilers do not necessarily have to be the fastest growing broilers in a flock. This claim was supported by Decuypere et al. (2005), who stated that AS is caused by an impaired oxygen supply that cannot sustain the growth, rather than by an increased oxygen requirement per se. Other researchers tested the hypothesis whether the fastest growing broilers within a pure line were always the most susceptible ones (Druyan et al., 2007 a, 2008). They found that even within highly improved lines for growth-related traits, there were either susceptible or resistant individuals in the populations they studied. These researchers could not find any significant positive relationship between the growth-related traits and AS%/AS-related traits (Druyan et al., 2007 b).

AS and its indicator traits are moderate to highly heritable (de Greef et al., 2001; Pakdel, 2004). This indicates that genetic factors play a considerable role in the susceptibility of birds to AS. Indeed, it is hypothesized that the interaction between endogenous and environmental factors plays an important role in AS susceptibility (Closter, 2014; Hassanzadeh, 2009, 2010) and the heritability of this syndrome is higher under ascites-inducing conditions than under normal commercial conditions (Pakdel, 2004).

In the current study a highly AS-susceptible pure sire line was used to determine if there were significant genetic variations for AS% (i.e. resistant families) and growth-related traits in the population. Therefore, the objectives of the present study were (i) to fit some mathematical functions to the average BW data of ascitic and healthy chickens which were reared under cold rearing condition (CRC) or normal rearing condition (NRC) to select the most suitable function for fitting the individual curves, (ii) to study the potential differences in growth traits between the ascitic and healthy chickens, and (iii) to estimate genetic correlations between the growth traits and AS% to investigate the feasibility of combined selection for improved growth traits and reduced AS%.

Material and methods

Rearing protocol

In the current research, a total of 1458 1-day-old chicks from 67 paternal half-sib families of a pure meat-type sire line were used. All of the chicks were reared together in a house with normal commercial conditions (NRC) until day 20. On day 20 almost ten chicks from each family were transferred to another house with lower-than-standard temperature (CRC). The experiment was conducted in late winter and early spring, thereby it was possible to induce AS in susceptible chicks by exposing them to cool temperature. Some of the families did not have sufficient number of chicks to be divided into two houses. Therefore, only 461 chicks from 47 families were transferred to the cool house. The house temperature was 33–35ºC at days 1 and 2 and was gradually decreased by 1˚C every other day until 21ºC was achieved at day 21. In NRC the temperature was maintained at 19–21ºC from day 21 until the end of the rearing period (day 54). Whereas a fast-decreasing temperature regime was applied in the cool house at day 21. The temperature of this house was kept around 15 to 18ºC during daytime and 10 to 15ºC during nighttime until the end. Birds received 24 h light in the first 2 days followed by a 23 hours light and 1 hour darkness regime afterwards. In order to induce AS in the majority of the susceptible ones, the chicks were provided a three-phase diet with higher-than-catalogue energy levels (approximately 100 to 120 units). The energy (cal/kg of ME) and crude protein (%) contents of the starter, grower, and finisher diets were 3000 and 20.7, 3050 and 18.7, and 3100 and 17.6, respectively. The three diets were fed *ad libitum* from days 1 to 10, 11 to 24, and 25 to end, respectively.

Measurements

Body weights were measured once a week after 2 hours of starvation. The recording process was carried out in as short time as possible. All of the chicks that died after day 21 were necropsied and examined to determine the cause of death. Chicks with ascitic fluid in the abdominal cavity or hydropericardium were considered as having died from AS and therefore recorded as AS-susceptible. The few number of birds that died due to the other causes were excluded from the data analyses. In addition, all of the birds that survived until the end of rearing period were necropsied after killing by cervical dislocation. Those with abdominal ascitic fluid or hydropericardium were recorded as being AS-susceptible. The remaining chicks were considered as AS-resistant. The experimental protocols were reviewed and approved by the Animal Care Committee of the Ferdowsi University of Mashhad, Iran.

Differences of AS frequencies between NRC and CRC

To test for significance of ascites frequency under the two rearing conditions (i.e. NRC and CRC), the FREQ procedure of SAS with CHISQ test was used.

Growth curve functions

Four growth functions, namely Gompertz, Logistic, Richards, and Lopez were employed to fit the average growth curves of the chicks in different classes of health status (healthy or ascitic) and gender under the two rearing conditions. Fitting of the functions was carried out using Gauss-Newton algorithm of NLIN procedure of SAS. The mathematical forms of the functions were as follows (Darmani-Kuhi et al., 2003):

Gompertz: $W = W_0 \exp\{[1 - \exp(-bt)] \ln(W_f/W_0)\}$
Logistic: $W = W_0 W_f / [W_0 - (W_f - W_0)\exp(-bt)]$
Lopez: $W = (W_0 K^b + W_f t^b)/(K^b + t^b)$
Richards: $W = W_0 W_f / [W_0^n + (W_f^n - W_0^n)\exp(-bt)]^{1/n}$

where:

W – live weight,
t – time,
W_f – the final weight,
W_0 – the initial weight,
b, K and n – constants.

R-square, BIC and AIC statistics were used for evaluation of the goodness of fit of the functions. The values of these statistics were obtained using the AUTOREG procedure of SAS.

According to the goodness of fit, the best function (i.e. Richards) was chosen to be fitted for individual and average family growth curves. After fitting the individual and average family growth curves, GR of all individuals and families for consecutive weekly intervals were obtained using the estimated BWs as below:

$$GR = \left(\frac{W_j - W_i}{d_j - d_i} \right)$$

where W_j and W_i are the estimated BWs at day j (d_j) and day i (d_i) of age, respectively.

Furthermore, weight and time at inflection point (W_{IP} and T_{IP}, respectively) were calculated using the primary parameters of the Richards function as below:

$$T_{IP} = \frac{1}{b} \ln \left(\frac{W_f^n - W_0^n}{nW_0^n} \right), \quad W_{IP} = \frac{W_f}{(n+1)^{1/n}}$$

Differences of growth curve parameters and growth rates between the ascitic and healthy chickens

After fitting the individual curves, data of estimated hatch weight (W0), final asymptotic weight (Wf), weight and time at the inflection point (WIP and TIP, respectively), as well as GR traits at different weekly intervals were estimated for each chick as mentioned above. Therefore, they were used as new growth-related traits for analyses. Mixed Procedure of SAS was used to analyze the mentioned data using the following model:

$$y = \mu + Sex + HS + Sex{*}HS + Sire + e$$

where sex and HS (health status) were considered as fixed effects, but sire and residual were considered as random effects.

Tukey multiple-range method was chosen for comparing the least square mean differences of the ascitic and healthy chicks.

Genetic correlations between AS% and growth curve parameters and growth rate traits

Frequency of AS for each family was calculated as percentage of ascitic chicks to the total number of chicks (AS%) in that family which were reared at CRC. Family mean values of weekly BW traits were calculated from chicks which were reared at NRC. As described above, the family mean values were subjected to be fitted by Richards function in order to obtain the average family growth curve parameters as well as the family average GR traits.

The estimates of pairwise correlation between AS% and family mean values of the GR traits or growth curve parameters served as approximate estimates of genetic correlation between the traits (Falconer and Mackay, 1996; Druyan et al., 2007 b). The CORR procedure of SAS was used to estimate the correlations.

Results

Severe cold stress, which was implemented to induce AS in the susceptible individuals, was tremendously successful. The frequency of AS was significantly ($P<0.0001$) higher in CRC (28.63%; 132 out of 461 chicks) than in NRC (3.11%; 31 out of 997 chicks).

Table 1. The BIC values of growth functions in fitting the average curves of healthy and ascitic chickens reared under cold rearing condition (CRC) or normal rearing condition (NRC)

Function	CRC				NRC			
	males		females		males		females	
	ascitic	healthy	ascitic	healthy	ascitic	healthy	ascitic	healthy
Gompertz	101.9	96.3	98.3	99.4	79.8	87.4	79.2	82.9
Logistic	105.2	97.0	95.3	100.2	69.8	74.5	72.8	68.3
Richards	101.9	94.9	94.8	98.8	54.9	74.3	69.5	67.9
Lopez	101.7	96.1	96.7	99.3	72.7	84.8	75.2	79.5

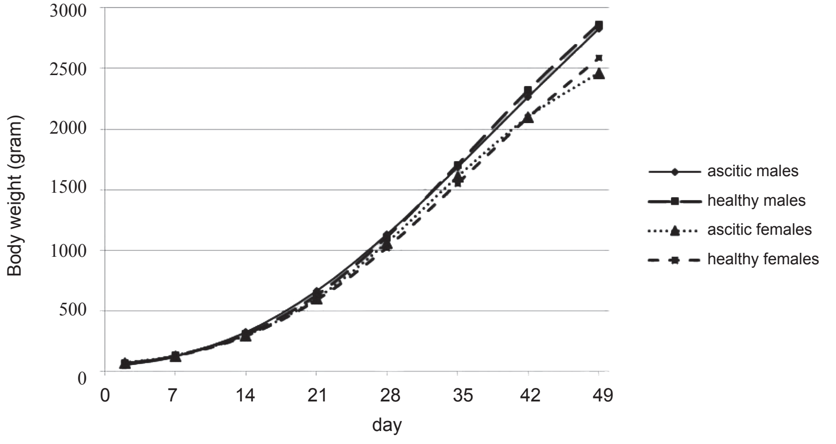

Figure 1. The growth curves of chickens split up into sex and health status (reared under cold rearing conditions)

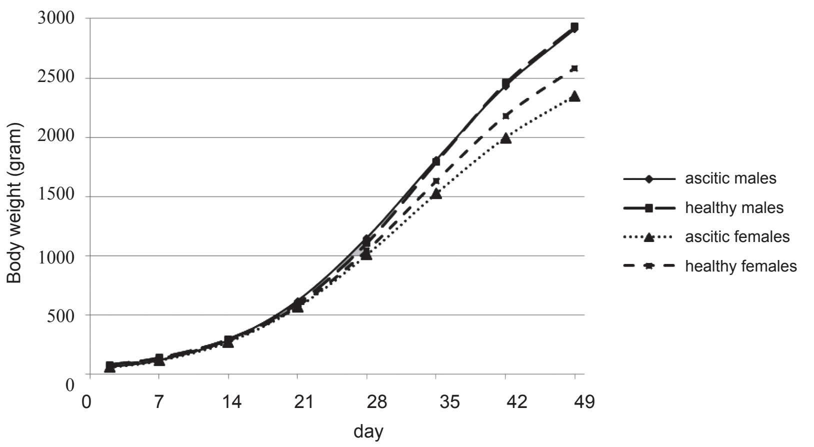

Figure 2. The growth curves of chickens split up into sex and health status (reared under normal rearing conditions)

All functions were accurate enough in fitting the average growth curves of chicks in different health status and gender classes with R-square values ranging from 0.9953 for Logistic function to 0.9999 for Richards function. Comparing the functions by BIC, AIC and R-square statistics revealed that the Richards function outperformed others. This function was followed by Lopez, Logistic and Gompertz functions, respectively. The goodness of fit results (BIC statistic) are presented in Table 1. Also, growth curves of ascitic males, ascitic females, healthy males and healthy females fitted by the Richards function are shown in Figures 1 and 2 for chicks reared at CRC and NRC, respectively.

The Richards function was used further to fit the individual and average family growth curves. Therefore the growth curve parameters and GR traits were estimated for them separately. The convergence criterion met for approximately 96% of the individual curves. The remaining 4% of the curves did not converge successfully even after using numerous priors for several times. All of the average family growth curves converged successfully.

Table 2. Least-square mean values of growth curve parameters and estimated growth rates of ascitic versus healthy chickens reared under cold rearing condition

Trait#	Male				Female			
	healthy	ascitic	DIFF %##	P-Value	healthy	ascitic	DIFF %	P-Value
W_0	57.1	55.4	3.2	NS	56.0	60.2	–7.0	NS
W_f	3600	3322	8.4	NS	3269	2586	26.4	<0.0001
n	0.98	1.26	–22.6	NS	1.09	1.82	–40.1	0.004
b	0.118	0.147	–20.1	NS	0.127	0.194	–34.3	0.002
W_{IP}	1701	1574	8.0	0.027	1554	1315	18.2	<0.0001
T_{IP}	34.4	32.6	5.6	0.004	34.3	31.3	9.7	<0.0001
GR_1	10.3	10.6	–2.8	NS	9.7	9.7	–0.1	NS
GR_2	23.8	24.9	–4.4	NS	22.2	22.1	0.2	NS
GR_3	45.0	47.2	–4.6	NS	41.4	42.0	–1.4	NS
GR_4	71.4	73.4	–2.8	NS	65.2	66.8	–2.3	NS
GR_5	90.1	86.0	4.7	NS	81.7	73.9	10.5	0.001
GR_6	85.3	73.1	16.6	0.001	76.8	53.5	43.6	<.0001
GR_7	63.5	50.4	26.2	0.001	56.9	33.9	67.8	<.0001

W_0 and W_f are weight at hatch and final body weight; n and b are constants; T_{IP} and W_{IP} are time and weight at inflection point; GR_1 to GR_7 are average growth rate at week 1 to week 7.

(Healthy-ascitic)/ascitic*100.

Table 3. Least-square mean values of growth curve parameters and estimated growth rates of ascitic versus healthy chickens reared under normal rearing condition

Trait#	Male				Female			
	healthy	ascitic	DIFF %##	P-Value	healthy	ascitic	DIFF %	P-Value
W_0	52.9	49.6	6.6	NS	51.1	47.9	6.7	NS
W_f	4064	3747	8.5	NS	3601	2520	42.9	0.002
n	0.86	1.04	–17.7	NS	0.78	1.34	–41.7	0.033
b	0.107	0.131	–18.7	NS	0.102	0.162	–37.2	0.001
W_{IP}	1881	1761	6.8	NS	1629	1252	30.1	0.001
T_IP	35.9	34.3	4.6	NS	34.9	30.9	13.0	0.001
GR_1	10.0	9.7	3.0	NS	9.9	9.5	4.3	NS
GR_2	23.6	23.4	0.8	NS	23.0	22.3	3.2	NS
GR_3	45.3	46.3	–2.3	NS	42.9	41.1	4.4	NS
GR_4	72.8	77.0	–5.5	NS	66.5	62.0	7.3	NS
GR_5	94.5	97.2	–2.8	NS	82.4	71.1	15.8	0.003
GR_6	95.8	86.0	11.4	NS	79.6	61.0	30.4	<0.0001
GR_7	76.6	60.7	26.2	NS	62.2	38.0	63.7	<0.0001

W_0 and W_f are weight at hatch and final body weight; n and b are constants; T_{IP} and W_{IP} are time and weight at inflection point; GR_1 to GR_7 are average growth rate at week 1 to week 7.

(Healthy-ascitic)/ascitic*100.

Least square means of growth curve parameters and GR traits are presented in Tables 2 and 3 for the CRC and NRC chicks, respectively. The results revealed that estimated weight at hatch did not differ significantly between the ascitic and healthy chicks. However, the asymptotic final weight was markedly different between the chicks of the two health status classes, but it was significant only in female chicks ($P<0.05$).

As shown in Figures 1 and 2, the growth pattern of healthy chicks was different from the ascitic ones, either in CRC or NRC. Compared to the ascitic chicks, the inflection point of healthy chicks occurred at later ages (2 to 4 days) and with higher weight, especially in female chicks ($P<0.05$; Tables 2 and 3). Although being greater in values, the time and weight at the inflection point of healthy males were not significantly different from those of ascitic ones in NRC.

Growth rates of ascitic chicks in the early weeks of age were not significantly higher than those of healthy ones, neither in NRC nor in CRC. In later ages (weeks 5, 6, and 7), however, the growth rates were significantly lower in ascitic chicks than in their healthy counterparts (4.7 to 26.7% in males and 10.5 to 67.8% in females).

Table 4. Mean and standard deviation of growth rates and growth curve parameters# as well as genetic correlation of ascites frequency with them

	W_0	W_f	n	b	T_{IP}	W_{IP}	GR_1##	GR_2	GR_3	GR_4	GR_5	GR_6	GR_7
Mean	58.7	3411	0.944	0.112	34.5	1650	9.8	22.4	42.6	69.1	89.1	86.1	63.5
S.D.	9.4	577	0.464	0.035	1.7	156	0.7	2.1	3.7	4.6	5.6	6.1	12.2
R$	–0.28	0.03	–0.22	–0.20	–0.08	–0.12	0.02	0.13	0.12	–0.02	–0.23	–0.21	0.02

W_0 and W_f are weight at hatch and final body weight; n and b are constants; T_{IP} and W_{IP} are time and weight at inflection point; GR_1 to GR_7 are average growth rate at week 1 to week 7.

Ascites frequency of the families was recorded in chickens reared under AS-inducing conditions (CRC) whereas the body weight data were recorded from the corresponding family members reared under normal rearing conditions (NRC).

$ R indicates the genetic correlation of predicted growth rates and parameters with ascites frequency; none of the correlations were statistically significant ($P>0.05$).

Genetic correlations between AS% and the GR traits and growth curve parameters are presented in Table 4. These correlations ranged from –0.28 for weight at hatch to 0.13 for GR at week 2. Except for final weight, the other growth curve parameters had negative correlations with AS%. Genetic correlations between AS% and GR traits at early weeks of age were positive, whereas these two groups of traits were negatively correlated at later ages. None of the genetic correlations were statistically significant ($P>0.05$).

Discussion

Since the used sire line was under intense selection for higher market weight over the past few years, it is considerably heavier than the other existing sire lines or hybrids in Iran (Azizian et al., 2013). However, AS is prevalent in this line and, in some cases, causes death in up to 10% of the chicks when reared under normal commercial conditions. In the current research, we aimed to study the feasibility of genetic improvement of the mentioned line for resistance to AS and improved GR traits simultaneously. In our previous work, using a small number of chicks of the same line, we found that there was no significant evidence of a positive association between growth-related traits and AS% (results not shown). Indeed if the mentioned results could be reacquired, this could convince us to design a new breeding program to select the selection candidates for improved GR traits but reduced AS frequency, simultaneously. Therefore, we designed the present experiment to study the relationship of these two groups of traits further and more precisely.

As mentioned above, the measurements of BWs took place once a week, thereby estimating GR of chicks accurately in the intervals between two consecutive weekly measurements was of great importance. Furthermore, a number of chicks in some families, especially in NRC, remained unmeasured in some instances and, therefore, had missing data. In order to use the majority of the data in the analyses we decided to use an accurate function for estimating missing data as well as to estimate GR and growth curve parameters precisely. Although the Gompertz model is a function of choice in many of the studies (Faridi et al., 2011), we aimed to choose the best function from four widely used functions which fits to our own data the best. Therefore, we employed the Gompertz, Logistic, Richards, and Lopez functions. According to the goodness of fit results (Table 1) the Richards function outperformed others and, therefore, was chosen for the fitting of individual and average family growth curves.

Both Gompertz and Logistic functions represent smooth sigmoid behavior with a fixed point of inflection ($0.368{*}W_f$ and $0.5{*}W_f$, respectively), whereas Richards function encompass sigmoid behavior with flexible point of inflection (Darmani--Kuhi et al., 2003). The Lopez equations, like the Richards, provide a flexible growth function capable of describing sigmoidal and diminishing returns behavior (Lopez et al., 2000). Therefore, our secondary aim was to assess the ability of functions with flexible point of inflection with those with fixed point of inflection in fitting the average growth curves. The ranks of the functions in goodness of fit (using AIC, BIC and R-square statistics; results not shown) indicated that functions with flexible point of inflection (i.e. Richards and Lopez functions) were more suitable for fitting the growth curves. This could be due, in some degree, to the variable inflection point of growth curves in ascitic vs. healthy and male vs. female chicks.

As can be seen in Figures 1 and 2, the estimated weights (by Richards function) of ascitic males were a bit higher than those of healthy ones at ages before 35 days. The slightly higher weight of ascitic chicks can still convince us to accept the hypothesis that the fastest grower chicks are the most susceptible ones to AS. However, this pattern was not observed in the female chicks, especially in NRC. Under these rearing conditions the ascitic females were lighter throughout the rearing period than the healthy ones. In ages later than day 28, probably due to the onset of the syndrome, the GR of ascitic chicks diminished gradually until the end of the rearing period. Such that, at later days of rearing period the BW of ascitic chicks was much lower than that of healthy ones. These findings are in agreement with the reports of Roush and Wideman (2000). In that study, compared to the ascitic and pre-ascitic chickens, the growth rate of clinically normal birds was higher for almost the entire rearing period, particularly at ages later than 28 days.

The statistical differences of growth curve parameters and GR traits between the ascitic and healthy chicks confirmed the aforementioned results which were based solely on the schematic comparison of the curves of healthy vs. ascitic chicks. As can be seen in Tables 2 and 3, the GR of ascitic chicks in the early weeks of ages were not significantly higher than that of their healthy counterparts. Therefore, these results support the hypothesis of Wideman (1998). This hypothesis claims that although AS develops in broilers with non-efficient cardiopulmonary system, these are not necessarily the fastest growing broilers in the flock. In addition to the results of the current study, this hypothesis was also supported previously by the findings of Roush and Wideman (2000) and Druyan et al. (2007 b).

Comparing the growth curve parameters of ascitic vs. healthy chicks revealed that the healthy chicks had curves with inflection point happening at later ages as compared to the curves of ascitic ones. This means that selection based on growth curve parameters, particularly time at the inflection point (T_{IP}), can probably eliminate the challenges which arise from rapid-growth on juvenile chicks and thereby can reduce the frequency of AS. To be a useful selection criterion, such an indicator should be heritable and truly genetically associated with AS resistance or susceptibility and easy to measure (Druyan et al., 2007 b). However, the heritability of time at inflection point was estimated to be small (0.11 in NRC and 0.15 in CRC), therefore the great genetic progress for this trait is not expected. Also, the genetic correlation of time at inflection point and 42-day BW was slightly negative ($r= -0.06$; $P= 0.69$). Therefore, selection based on such indicator trait can somewhat compromise the selection for more important traits such as GR and meat yield in the long term.

The growth pattern of ascitic chicks was not suitable and the ascitic chicks grew less efficiently during the rearing period (if not died), therefore it seems that the higher frequency of AS can ruin the FCR of the flock. It means that reducing the frequency of ascitic individuals in this population will probably have a positive side effect such as an improved feed efficiency. Wideman (2013) reported that after 14 generations of divergent selection to generate AS-susceptible and AS-resistant lines, the resistant line was more efficient in feed utilization than the susceptible line.

In accordance with previous reports claiming no true association between the growth-related traits and AS% (Wideman, 1998; Druyan et al., 2007 b), we did not observe any significant genetic relationship between the two groups of traits either. Genetic correlation between AS% and growth curve parameters and GR revealed that there was no merely positive genetic relationship between the rapid GR and AS incidence. Although correlations of early GR traits (e.g. GR at weeks 1, 2, and 3) with AS incidence were positive, the remaining correlations were negative. None of the genetic correlations, however, were statistically significant ($P>0.05$), indicating that these correlations were not statistically different from zero. Genetic correlations among growth-related traits and AS-related traits were studied by a number of researchers (de Greef et al., 2001; Moghadam et al., 2001; Pakdel, 2004). Under NRC, Moghadam et al. (2001) found a positive genetic correlation between the AS and BW traits. Under AS-inducing conditions, however, de Greef et al. (2001) and Pakdel (2004) reported a negative genetic correlation between AS-related traits and BW. In addition, Pakdel (2004) estimated a weak but positive genetic correlation among AS-related traits measured under AS-inducing condition and BW measured under NRC. The weakly positive genetic correlation between the AS and some early GR traits could, in a long time, hinder the acquisition of the highest amount of genetic progress for the growth-related traits. Pavilidis et al. (2007) reported that the AS-resistant line was on average 163 g lighter at 42 days of age than the AS-susceptible line. It is theoretically adopted that integrating additional traits in breeding goal can reduce the genetic progress for all of the traits. Pakdel (2004), studying the different breeding strategies for combined selection of BW and AS, reported that the genetic progress for BW could be reduced by almost 17% in a breeding strategy which both BW and AS-indicator traits included in the breeding goal as compared to the alternative strategy that included only BW in the breeding goal. This researcher also mentioned that using genetic markers for selection against AS% can lessen the amount of reduction in genetic response when the two traits were integrated in the breeding goal. This idea was supported by Rabie et al. (2005) and Closter (2014) who finely mapped significant QTLs for AS-related traits. Nowadays the appearance of modern genotyping and sequencing technologies can assist poultry breeders further in implementing such breeding strategies (Burks and Rhods, 2011).

The general conclusion of the current study is that although AS has partly been controlled in widely used commercial lines and is no longer considered a significant risk factor in them (Bishop et al., 2010), it is still a major problem of the heavy pure sire line which was used in the current study. The GR at early ages of the chicks were not significantly different between the ascitic and healthy chicks, neither in NRC nor in CRC. However the time at inflection point of growth curve of healthy chicks happens almost 2–4 days later than that of their ascitic counterparts, indicating that the growth curves of healthy chicks were more suitable than the growth curves of ascitic ones. Therefore, selective breeding to modify growth curves can, in a long time, influence the frequency of AS incidence in the population effectively, with limited negative effect on the genetic progress of growth-related traits. The GR and growth curve parameters were not significantly correlated with the incidence of AS. However, early GR traits were weakly but not significantly correlated with AS incidence. If the relying genetic correlation was truly positive, even weakly, it could hinder the acquisition of maximum potential genetic progress for growth-related traits in the long term in the case in which both of the traits are integrated in the breeding program.

References

Azizian M., Rahimi S., Kamali M.A., Karimi Torshizi M.A., Zobdeh M.R. (2013). Comparison of the susceptibility of six male broiler hybrids to ascites by using hematological and pathological parameters. J. Agr. Sci. Tech., 15: 517–525.

Bishop S.C., Axford R.F.E., Nicholas F.W., Owen J.B. (2010). Editors. Breeding for disease resistance in farm animals. Cambridge, USA, CABI publication, 3rd ed., 362 pp.

Boostani A., Ashayerizadeh A., Mahmoodian Fard H.R., Kamalzadeh A. (2010). Comparison of the effects of several feed restriction periods to control ascites on performance, carcass characteristics and hematological indices of broiler chickens. Braz. J. Poultry Sci., 12: 171–177.

Burks J.R., Rhoads D.D. (2011). Sequence analysis of the angiotensin II type 1 receptor (*AGTR1*) gene for mutations contributing to pulmonary hypertension in the chicken (*Gallus gallus*). Inquiry, 12: 49–59.

Closter A.M. (2014). Quantitative genetic analysis of ascites in broilers. PhD Diss. Wageningen University, The Netherlands.

Darmani-Kuhi H., Kebreab E., Lopez S., France J. (2003). An evaluation of different growth functions for describing the profile of live weight with time (age) in meat and egg strains of chicken. Poultry Sci., 82: 1536–1543.

Decuypere E., Hassanzadeh M., Buys N., Buyse J. (2005). Further insights into the susceptibility of broilers to ascites. Vet. J., 169: 319–320.

De Greef K.H., Janss L.L.G., Vereijken A.L.J., Pit R., Gerritsen C.L.M. (2001). Disease-induced variability of genetic correlations: Ascites in broilers as a case study. J. Anim. Sci., 79: 1723–1733.

Druyan S., Ben-David A., Cahaner A. (2007 a). Development of ascites-resistant and ascites-susceptible broiler lines. Poultry Sci., 86: 811–822.

Druyan S., Shlosberg A., Cahaner A. (2007 b). Evaluation of growth rate, body weight, heart rate, and blood parameters as potential indicators for selection against susceptibility to the ascites syndrome in young broilers. Poultry Sci., 86: 621–629.

Druyan S., Hadad Y., Cahaner A. (2008). Growth rate of ascites-resistant versus ascites-susceptible broilers in commercial and experimental lines. Poultry Sci., 87: 904–911.

Druyan S., Shinder D., Shlosberg A., Cahaner A., Yahav S. (2009). Physiological parameters in broiler lines divergently selected for the incidence of ascites. Poultry Sci., 88: 1984–1990.

Falconer D.S., Mackay T.F.C. (1996). Introduction to quantitative genetics. Harlow, UK, Longman Group Ltd, 4th ed., 366 pp.

Faridi A., Mottaghitalab M., Rezaee F., France J. (2011). Narushin-Takma models as flexible alternatives for describing economic traits in broiler breeder flocks. Poultry Sci., 90: 507–515.

Franciosini M.P., Tacconi G., Leonardi L. (2012). Ascites syndrome in broiler chickens. Vet. Sci. Res., 3: 60–66.

Guo X., Zhang H., Yin Q.Q., Cheng W., Jiang Y.B. (2007). Study on mechanism of ascites syndrome of broilers. Am. J. Anim. Vet. Sci., 2: 62–65.

Hassanzadeh M. (2009). New approach for the incidence of ascites syndrome in broiler chickens and management control of the metabolic disorders. Int. J. Poultry Sci., 8: 90–98.

Hassanzadeh M. (2010). Endogenous and environmental factors interactions that contribute to the development of ascites in broiler chickens: a review. Int. J. Vet. Res., 4: 117–126.

Huchzermeyer F.W. (2012). Broiler ascites: a review of the ascites work done at the poultry section of the Onderstepoort Veterinary Institute 1981–1990. World Poultry Sci. J., 68: 41–50.

Julian J.R. (1993). Ascites in poultry. Avian Pathol., 22: 419–454.

Lopez S., France J., Dhanoa M.S., Mould F., Dijkstra J. (2000). A generalized Michaelis-Menten equation for the analysis of growth. J. Anim. Sci., 78: 1816–1828.

Moghadam H.K., McMillan I., Chambers J.R., Julian R.J. (2001). Estimation of genetic parameters for ascites syndrome in broiler chickens. Poultry Sci., 80: 844–848.

Pakdel A. (2004). Genetic analysis of ascites-related traits in broilers. PhD Diss. Wageningen University, The Netherlands.

Pavlidis H.O., Balog J.M., Stamps L.K., Hughes J.D., Huff W.E., Anthony N.B. (2007). Divergent selection for ascites incidence in chickens. Poultry Sci., 86: 2517–2529.

Rabie T.S.K.M., Crooijmans R.P.M.A., Bovenhuis H., Vereijken A.L.J., Veenendaal T., van der Poel J.J., van Arendonk J.A.M., Pakdel A., Groenen M.A.M.

(2005). Genetic mapping of quantitative trait loci affecting susceptibility in chicken to develop pulmonary hypertension syndrome. Anim. Genet., 36: 468–476.

Roush W.B., Wideman R.F. (2000). Evaluation of broiler growth velocity and acceleration in relation to pulmonary hypertension syndrome. Poultry Sci., 79: 180–191.

Scheele C.W., van der Klis J.D., Kwakernaak C., Dekker R.A., van Middelkoop J.H., Buyse J., Decuypere E. (2005). Ascites and venous carbon dioxide tensions in juvenile chickens of highly selected genotypes and native strains. World Poultry Sci. J., 61: 113–129.

Solis de los Santos F., Tellez G., Farnell M.B., Balog J.M., Anthony N.B. (2005). Hypobaric hypoxia in ascites resistant and susceptible broiler genetic lines influences gut morphology. Poultry Sci., 84: 145–148.

Vidyadaran M.K., King A.S., Kassim H. (1990). Quantitative comparisons of lung structure of adult domestic fowl and red jungle fowl. Avian Pathol., 19: 51–58.

Wideman R.F. (1998). Causes and control of ascites in broilers. In: National Mtg. Poultry Health and Processing. Delmarva Poultry Industry Inc. Georgetown, DE. (33) pp. 56–85.

Wideman R.F. (2013). Pulmonary arterial hypertension (ascites syndrome) in broilers: a review. Poultry Sci., 92: 64–83.

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