Decreasing fat deposition in broiler chickens by using a beta-adrenergic agonist "salbutamol"

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The objective of this study was to reduce fat deposition in broiler chickens using a synthetic compound of beta-adrenergic agonist. A total number of 200 day-old unsexed Hubbard broiler chicks was equally allotted at random to 5 treatments each of 2 equal replicates.1 A beta-adrenergic agonist drug (salbutamol) was supplemented in the diet at the levels of zero , 50 and 100 mg / kg diet either from 1 or 21 days until 42 days of age. Growth performance was weekly recorded during the experimental period. Relative weight of adipose tissue and some related blood constituents were evaluated at 21 and 42 days of age. Body weight and gain were significantly decreased throughout the experimental period in birds received salbutamol from 1 day of age as compared to those of 21 days or the control. At 21 days of age , relative weights of abdominal, gizzard, sartorial and neck fat of treated birds were insignificantly lower than the control, only a significant decrease was observed in breast and total fat in the level of 50 mg salbutamol / kg diet. At 42 days of age, the most effective doses of salbutamol in decreasing the relative weight of adipose tissue were 50 and 100 mg / kg diet for 6 weeks as compared to the other treatments. Significant effect was only found in sartorial and total fat in the level of 100 mg salbutamol / kg diet for 6 weeks. Plasma glucose, triglycerides and very low density lipoprotein (VLDL) levels were significantly increased at 21 days of age in treated chicks. However, at 42 days of age, only high density lipoprotein (HDL) was significantly the lowest in the plasma of chicks treated with 100 mg salbutamol regardless the time of treatment initiation. It is apparent that the beta-adrenergic agonist can exert a pronounced effect on reduction and re-distribution of the adipose tissues in broiler chickens.

Keywords: broiler chickens; fat deposition; beta-adrenergic agonist; salbutamol

Introduction

Continuous attempts to increase growth rate of meat-type chickens in poultry industry have been accompanied by excessive fatness which is not desired by most of consumers. Therefore, B-adrenergic agonists were found to enhance leanness in livestock species and would not likely represent a credible risk to the consumers of edible tissues of properly treated animals(Smith,1998). The effects of such compounds on performance characteristics are well established in domestic animals including chickens(Buttery and Dawson, 1987).

The objective of this study was to reduce fat deposition in broiler chickens using a synthetic compound of B-adrenergic agonists, salbutamol sulphate.

Materials and methods

A total number of 200 day-old unsexed Hubbard broiler chicks was equally allotted at random to 5 treatments each of 2 replicates. A B-adrenergic agonist drug (salbutamol sulphate) was supplemented in the basal diet at levels of zero, 50 and 100 mg / kg diet either from 1 or 21 days until 42 days of age. Birds were housed in standard cages of nature ventilated house. Vaccination, hygiene and lighting

programmes were performed on the birds as recommended. They received a basal starter diet(21.7%CP and 3000 Kcal / kg ME) for the first 3 weeks, and a basal finisher diet(18%CP and 3200 Kcal / kg ME) from 3 to 6 weeks of age. Feed and water were provided ad. lib.

Live body weight and weight gain were weekly recorded. At 21 and 42 days of age 8 birds of each treatment were randomly taken for carcass fat measurements according to Cahaner et al.(1986) and for the assay of plasma glucose, triglycerides, high density lipoprotein(HDL) and very low density lipoprotein(VLDL) using available commercial kits. Data were analyzed by analysis of variance using the general model of SAS(1998).

Results and discussion

Growth performance: Table(1) shows that salbutamol decreased live body weight in treatments 2 and 4 throughout the whole experimental period. This means that the time of treatment initiation is a limiting factor that can affect live body weight. This finding is consistent with the observations of Takahashi et al.(1993) and inconsistent with the results of Dalrymple et al.(1984). The decrease in live body weight through the first 3 weeks of age in treatments 2 and 4 was also reflected on body weight gain(Table 1).From 3 to 4 weeks, treatment 2 had nearly the same weight of the control, however, it decreased again and joined treatment 4 from 4 to 5 weeks of age. From 5 to 6 weeks of age , there were no significant differences in weight gain among all treatments. This was in agreement with Hamano et al.(1999), however, it was in disagreement with Duquette et al.(1988). Because of the growth promoting effect of B-adrenergic agonists is likely to be dependent on type, dosage and possibly strain of broilers, these factors may account for the apparent discrepancies among the published data.

Fat deposition: Table(2)indicates that salbutamol decreased the relative weights of total and different parts of adipose tissue at 21 days of age. However, significant effect was only detected in total and breast fat, where the lowest relative weights were found in 50 mg / kg diet treatment. At 42 days of age (Table 3)the most effective doses of salbutamol in decreasing weight %of adipose tissues were 50 and 100 mg / kg diet for 6 weeks. Significant effect was only observed in total and sartorial fat, where the lowest weight% were recorded in 100 mg / kg diet for 6 weeks. These results may indicate that salbutamol should be supplied from the first day of age to show its effect on body fat metabolism. B-adrenergic agonists, like glucagons, are lipolytic agents and may act on avian adipocyte(Campbell and Scanes, 1985 b). These lipolytic agents exert their effect by increasing intracellular cAMP levels which activate hormone sensitive lipase via phosphorylation of triacylglycerol lipase then triacylglycerol degraded to free fatty acids. However, the response of broiler chickens to B-adrenergic agonists is relatively small compared to those of ruminants(Yang and McElligott,1989).

Plasma constituents: Table(4)shows that salbutamol increased plasma glucose, triglycerides, HDL and VLDL at 21 days of age. Significant effect was only noticed in the levels of plasma glucose, triglycerides and VLDL. However, at 42 days of age salbutamol decreased plasma glucose and VLDL(Table 5). In addition, triglycerides level was increased only when treatment initiation was from one day post-hatching regardless of salbutamol dose. Significant effect of salbutamol was only detected on decreasing HDL with the dose of 100 mg / kg diet regardless of the time of treatment initiation.

The depression in plasma VLDL observed at 6 weeks of age may suggest that B-adrenergic agonists inhibit lipogenesis or at least the transport of triglycerides from the liver to adipose tissues and vice versa at this age(Buyse et al. 1991). However, data on triglycerides in the present study are inconsistent with the observations of Buyse et al. (1991). Data of plasma constituents at 21 days of age oppose the theory of Griffin et al.(1982)that plasma VLDL, LDL and triglycerides concentrations are positively correlated with body fat. The observations on glucose are consistent with those of Eisemann et al.(1988) who found that the increase in plasma glucose due to B-adrenergic agonists effect is transient.

Table 1 Live body weight and body weight gain of broiler chicks as influenced by salbutamol.

Age (week)	Treatments					±SD	Overall	Prob.
	(1) 0mg/6wks	(2) 50mg/6wks	(3) 50mg/3wks	(4) 100mg/6wks	(5) 100mg/3wks			
				Live body weight (g)				
0	36.2^{b}	36.74^{b}	39.57^{a}	39.47^{a}	39.46^{a}	3.27	38.37	**
1	146.76^{a}	124.42^{b}	144.78^{a}	121.65^{b}	147.22^{a}	18.45	136.7	**
2	383.54^{a}	315.90^{b}	379.16^{a}	303.35^{b}	378.36^{a}	49.60	350.9	**
3	721.80^{a}	615.45^{b}	701.92^{a}	593.83^{b}	708.07^{a}	81.48	664.6	*
4	1085.45^{a}	970.15^{b}	1041.53^{a}	906.75^{c}	1049.5^{a}	108.19	1014.0	**
5	1494.05^{a}	1329.29^{b}	1428.8^{a}	1261.41^{b}	1448.08^{a}	153.59	1397.2	**
6	1848.45^{a}	1702.80^{b}	1858.56^{a}	1683.74^{b}	1898.03^{a}	191.85	1796.4	**
				Body weight gain (g)				
0-1	110.56^{a}	87.68^{c}	105.21^{b}	82.18^{c}	107.76^{a,b}	17.92	98.14	**
1-2	241.78^{a}	191.48^{b}	234.38^{a}	181.7^{b}	231.14^{a}	33.95	214.13	**
2-3	333.2^{a}	299.55^{c,b}	322.76^{a,b}	290.48^{c}	329.71^{a}	42.63	313.6	**
3-4	363.65^{a}	354.7^{a}	339.61^{a,b}	312.92^{b}	341.43^{a,b}	58.45	343.98	*
4-5	408.6^{a}	359.14^{b}	387.27^{a,b}	354.66^{b}	398.58^{a}	66.15	382.06	**
5-6	354.4	373.51	429.76	422.33	449.95	77.98	380.59	NS
0-6	1812.2^{a}	1666.06^{b}	1818.99^{a}	1644.27^{b}	1858.57^{a}	160.0	1760.0	**

*P<0.05 **P<0.01 NS: not significant

a, b, c, Mean values with no common superscripts within rows differ significantly

Table 2 Relative weights (%) of adipose tissue parts from salbutamol-treated broiler chicks at 21d of age.

Adipose tissue	Treatments			Prob.
	Control	50mg/kg diet	100mg/kg diet	
Abdominal	0.94	0.78	0.80	NS
	±0.13	±0.14	±0.10	
Gizzard	0.58	0.45	0.38	NS
	±0.13	±0.05	±0.03	
Sartorial	1.35	0.98	0.96	NS
	±0.17	±0.26	±0.09	
Neck	0.97	0.80	0.84	NS
	±0.04	±0.14	±0.08	
Breast	0.55a	0.21b	0.35a,b	*
	±0.10	±0.07	±0.08	
Total fat	4.41a	3.23b	3.36a,b	*
	±0.33	±0.49	±0.28	

*P<0.05 NS: not significant

a.b Mean values with no common superscripts within rows differ significantly

Table 3 Relative weights (%) of adipose tissue parts from salbutamol-treated boilers at 42days of age.

Adipose tissue	Treatments					Prob.
	(1) 0mg/6 wks	(2) 50mg/6 wks	(3) 50mg/3 wks	(4) 100mg/6 wks	(5) 100mg/3 wks	
Abd. (1)	1.65	1.39	1.63	1.43	1.79	NS
Giz.	0.88	0.62	0.91	0.66	0.86	NS
Sart.	$1.59^{a,b}$	1.40^{b}	1.84^{a}	1.29^{b}	1.95^{a}	**
Nec.	1.29	0.97	1.02	0.93	1.00	NS
Bre.	0.57	0.52	0.50	0.41	0.61	NS
Mes.	0.52	0.31	0.48	0.37	0.57	NS
Tot.	$6.54^{a,b}$	$5.29^{a,b}$	$6.40^{a,b}$	5.16^{b}	6.78^{a}	*

*P<0.05 **P<0.01 NS: not significant

(1) Abd.=abdominal, Giz.=gizzard, Sart.=sartorial, Nec.=neck, Bre.=breast, Mes.=mesenteric, Tot.=total fat

Table 4 Effect of salbutamol supplementation on some plasma metabolites in broiler chicks at 21days of age.

Parameter	Treatments			SD±	Prob.
	control	50mg/kg diet	100mg/kg diet		
Glu (1)	294.14[b]	401.13[a]	471.57[a]	115.57	*
TG	28.87[b]	56.0[a]	43.87[a]	15.63	**
HDL	194.88	225.43	216.88	30.40	NS
VLDL	5.77[b]	11.20[a]	9.20[a]	3.14	**

*P<0.05 **P<0.01 NS: not significant

a.b,c Mean values with no common superscripts within rows differ significantly

(1) GLU=glucose (mg /dl),TG=triglycerides (mg /dl),HDL=high density lipoprotein (mg /dl),VLDL=very low density lipoprotein(mg /dl)

Table 5 Effect of salbutamol supplementation on some plasma metabolites in broiler chicks at 42days of age.

Parameter	Treatments					SD±	Prob.
	(1) 0mg/6 wks	(2) 50mg/6 wks	(3) 50mg/3 wks	(4) 100mg/6 wks	(5) 100mg/3 wks		
GLU	201.75	182.0	183.50	170.83	178.3	19.6	NS
TG	79.0	85.5	70.62	84.66	78.14	16.9	NS
HDL	28.75[a]	30.87[a]	25.5[a,b]	18.34[b]	17.4[b]	10.1	*
VLDL	16.15	15.57	14.12	14.87	15.62	3.81	NS

*P<0.05 **P<0.01 NS: not significant

a.b,c Mean values with no common superscripts within rows differ significantly

References

BUTTERY, P.J., DAWSON, L. (1987) The mode of action of beta-agonists as manipulators of carcass composition, in:HANRAHAN, J.P. (Ed.) beta-agonists and their effects on animal growth, carcass quality, pp29-43,London.

BUYSE, J., DECUYPERE, E., HUYGHEBAERT, G. and HERREMANS, M. (1991) The effect of clenbuterol on growth performance and on plasma hormone and metabolites levels of broilers. Poultry Science **70**: 993-1002.

CAHANER, A., NITSAN, Z. and NIR,I. (1986) Weight and fat content of adipose and non adipose tissue in broilers selected for or against abdominal adipose tissue. Poultry science **65**: 215-222.

CAMPBELL, R.M.and SCANES, C.G. (1985 b) Adrenergic control of lipogenesis and lipolysis in the chicken in vitro. Comp. Physiol. Biochem. **82 c**: 137-142.

DALRYMPLE, R.H., BAKER, P.K., GINGHER, P.E., INGLE, D.L.,PPENSACK, M. and RICKS, C.A. (1984) A repartitioning agent to improve performance and carcass composition of broilers. Poultry Science **63**:2376-2383.

DUQUETTE, P.F., RICKES,E.L., OLSON,G., CAPIZZI,T.P. and CONVEY, E.M. (1988) Growth and carcass composition of broilers fed beta-adrenergic agonist, L640-033,pp 173-177 in: Beta agonists and their effects on animal growth and carcass quality. J.P. Hanrahan, Elsevier Applied Science, London, England and New York, NY.

EISEMANN, J.H., HUNTINGTON, G.B. and FERREL, C.L. (1988) Effect of dietary clenbuterol on metabolism of the hindquarter in stress. J. Anim. Sci. **66**: 342.

GRIFFIN, H.D. and WHITEHEAD, C.C. (1982) Plasma lipoprotein concentration as an indicator of fatness in broilers: development and use of a simple assay for plasma very low density lipoproteins. Brit. Poult. Sci. **23**: 307-313.

HAMANO, Y., OKADA, S. and ANAKA, T. (1999) Effects of thiamine and clenbuterol on body composition, plasma metabolites and hepatic oxygen consumption in broiler chicks. Brit. Poult. Sci. **40**: 127-130.

S.A.S. (1998) SAS Users Guide: Statistics. Statistical Analysis Institute, Inc., Cary, NC.

SMITH, D.J. (1988) The pharmacokinetics, metabolism and tissue residues of B-adrenergic agonists in livestock. J. Anim. Sci. **76**: 173-194.

TAKAHASHI, T., AKIBA, Y. and HORIGUCHI, M. (1993) Effect of beta-adrenergic agonist, (clenbuterol) on performance, carcass composition, hepatic microsomal mixed function oxidase and antibody production in female broilers treated with or without corticosterone. Brit. Poult. Sci. **34**: 167-175

YANG, Y.T. and McELLIGOTT, M.A. (1989) Multiple actions of beta-adrenergic agonists on skeletal muscle and adipose tissue. Biochem. J. **261**: 1-10.