

EFFECTS OF β -AGONIST L-644,969 ON ENERGY METABOLISM IN BROILERS

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ABSTRACT

Effects of β -agonist L-644,969 on energy metabolism were evaluated in broiler chickens. The chickens were allocated into twelve cages with six chickens per cage during the first week and three chickens in the following five weeks of the experiment. The β -agonist L-644,969 was given at the doses of 0 (T0), 0.25 (T1) and 1.0 ppm (T2). Energy metabolism evaluation showed that chickens given the highest dose (1.0 ppm) increased the total heat production, resulting in a decrease in retained energy. A further change in treated chickens was the decrease in retained fat energy (T0=369, T1=311 and T2=275 kJ/kgW^{0.75}/d) without the change in retained protein energy. It was concluded that L-644,969 was an effective repartitioning agent in growing chickens.

(Key Words: β -agonist, Energy Balance, Energy Metabolism, Heat Production, Retained Energy).

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PENGARUH β -AGONIST L-644,969 TERHADAP METABOLISME ENERGI PADA BROILER

INTISARI

Pengaruh β -agonist L-644,969 terhadap metabolisme energi dievaluasi pada broiler. Tujuh puluh dua broiler tersebut dibagi dalam 12 kandang koloni sehingga masing-masing kandang terdiri dari 6 ekor pada minggu pertama percobaan dan dikuangi menjadi 3 ekor pada 5 minggu berikutnya. β -agonist L-644,969 diberikan pada dosis 0 (T0), 0,25 (T1), dan 1,0 ppm (T2). Evaluasi pada metabolisme energi menunjukkan bahwa broiler yang diberi dosis 1 ppm β -agonist L-644,969 meningkat total produksi panasnya, yang berakibat pada penurunan energi yang ditimbun dalam tubuh. Perubahan lebih lanjut adalah pada penurunan energi dalam bentuk lemak (T0=369, T1=311 and T2=275 kJ/kgW^{0.75}/d) tanpa adanya perubahan penimbunan energi dalam bentuk protein. Dapat disimpulkan bahwa L-644,969 efektif dalam fungsinya sebagai zat pengubah energi dalam bentuk lemak menjadi energi dalam bentuk protein pada broiler.

Introduction

Some studies have shown that dietary administration of β -adrenergic agonist improved growth rate, feed efficiency and carcass composition in pigs (Moser *et al.*, 1986) and broilers (Dalrymple *et al.*, 1984). The increase in protein deposition was primarily as a result of a decrease in protein degradation and followed by a reduced fat deposition without additional feed consumption (Reeds *et al.*, 1986). The most recent study showed in broilers that L-644,969 increased protein retention and decreased fat retention, and consequently the total energy retention was reduced (Xiyi *et al.*, 1994). Similar results were also found in cattle (Chwalibog *et al.*, 1996). Although the effects of β -adrenergic agonist on performance characteristics and energy metabolism are well established in pigs and ruminants, only a few studies have addressed the effect of β -adrenergic agonist on energy metabolism in growing chickens. Thus, the objective of this study was to investigate the effects of β -agonist, L-644,969 on protein balance and energy metabolism in growing chickens.

Materials and Methods

Male broiler chickens, Ross Cobb 208 obtained from a commercial hatchery, were used in this experiment. Seventy-two day-old chicks were allocated into twelve cages with six chicks per cage during the first week and three chicks in the following five weeks of the experiment. The cages were equipped with feeders and two hanging nipple drinkers supplying water through pipes positioned at the back of the cages. Droppings fell through wire mesh floors into the dropping trays. The diameter of the wire used in the first period was 1.0 cm while 2.0 cm was used in the later periods. The temperature in the cages was maintained at 34-36°C for the first week and was reduced at the rate of about 3 - 4°C per week until it reached 21°C at four weeks of age. The relative humidity was 60 to 70%.

Chickens were fed a commercial diet (Table. 1) and given *ad libitum* for a five-day collection period. Food residues were weighed, mixed and used for dry matter (DM) determination to correct the differences in DM content between food and food residues. Droppings were collected daily before feeding and analysed for DM, crude protein (CP), fat, ash and energy according to methods described by Thorbek (1980), and the values of gross

Table 1. Chemical composition of the experiment

Chemical composition	Quantity
Gross energy	16.8 MJ
Dry matter	88.8%
Crude protein	22.5%
Fat	5.0%
Ash	7.0%
<i>Amino acids</i>	
Lysin	12.8 g/kg
Methionin	6.1 g/kg
Cystin	3.3 g/kg
<i>Vitamins</i>	
A	12 IU/g
D3	3 IU/g
E	35 mg/kg
<i>Mineral mix</i>	
Copper	20 mg/kg
Selenium	0.2 mg/kg
Avilmacin	10 mg/kg
Salinomysin natrium	60 mg/kg

The diet composition: cereals, oil seed meals and their by-products, animal products, legumes and their by-products, fish meals and oil with ethoxyquin stabilizer

energy (GE) were obtained by means of calorimetric bombs.

β -Agonist administration

The β -adrenergic agonist, L-644,969 (R,R isomer of 6-amino-alpha{[(1-methyl-3-phenylpropyl) amino] methyl}-3-pyridine methanol dihydrochloride), was provided by Merck & Co., Inc. (Rahway, NJ), and the amount of L-644,969 was given orally in water dilution with a micro pipette injection. The treatment levels of β -agonist were control, i.e. 0 ppm (T0), together with 0.25 ppm (T1) and 1.0 ppm (T2) based on the food intake of the preliminary experimental series.

Respiration experiments

A 22 to 24-hour respiration measurement was made in the middle of each five-day collection period using an open-air circulation respiration unit. In the respiration chambers,

the temperature and humidity are automatically controlled. The air flow in the system is measured by the differential pressure principle, and the composition of outgoing air is measured using an infrared gas analyser Uras (Hartmann and Braun, Germany) for carbon dioxide and a paramagnetic analyser Magnos (Hartmann and Braun, Germany) for oxygen. The gas exchange was calculated from the difference between the concentration of atmospheric gas entering the chamber and the gas leaving the chamber. The difference in gas concentration was multiplied by the rate of flow at which the gas was withdrawn from the chamber. Heat production (HE) was calculated according to Brouwer's equation (1965).

HE (kJ) =

$$(\text{O}_2, \text{l consumption} \times 16.18) + (\text{CO}_2, \text{l production} \times 5.02)$$

The total heat production was not corrected for systemic experimental errors due to lost ammonia during the time of dropping collection and biological errors due to

excretion of endogenous and non-protein nitrogen. However, a 10% underestimation of urine nitrogen would only influence the contribution of protein oxidation to the total heat production by 1-2% (Chwalibog et al., 1992). Body retained energy (RE) was calculated as the difference between metabolizable energy (ME) and HE. The energy retained as protein (RPE) was calculated from the N retention multiplied by factors 6.25 and 23.86 kJ/g, and the energy retained as fat (RFE) was RE minus RPE.

Measurements and statistical analysis

The measurements included nitrogen balance, gaseous exchange, heat production, metabolizable energy and energy balance. All measurements are reported in relation to metabolic body weight (W , $\text{kg}^{0.75}$). The statistical analysis used was an unbalanced ANOVA procedure appropriate for factorial arrangement of treatments in completely randomized block design using GLM

procedure (SAS Institute, 1988). Duncan's multiple range test was used to compare treatment means. The mathematical description of an observation is given by:

$$Y_{ijk} = \mu + B_i + J_j + (BJ)_{ij} + g_{ijk}$$

μ = treatment mean

B_i = effect of period i

J_j = effect of treatment j

$(BJ)_{ij}$ = interaction between period and treatment

g_{ijk} = experimental error

Results and Discussion

Data on gaseous exchange and energy balance is summarized in Table 2. Oxygen consumption and CO_2 production were significantly different between control and treatment groups ($P < 0.05$). The higher dose of L-644,969 (T2) increased both O_2 consumption and CO_2 production compared with T0 but statistically was not different to T1. The increase in O_2 consumption was

Table 2. Means of oxygen (O_2) consumption, carbon dioxide (CO_2) production, respiration quotient (RQ), gross energy (GE), metabolizable energy (ME), heat production (HE), retained energy (RE), retained protein energy (RPE), retained fat energy (RFE)

	T0 (0 ppm)	T1 25	T2 (1.0 ppm)	SEM	Significance
n	24	25	29		
CO_2 , l/d	33.3 a	34.7 ab	35.5 b	192	**
O_2 , l/d	35.6 a	37.2 ab	38.9 b	262	**
RQ	0.93	0.93	0.92	0.4	ns
GE, kJ/d	1951	1923	1911	1334	ns
ME, kJ/d	1483	1462	1443	1031	ns
HE, kJ/d	743 a	776 ab	807 b	459	**
RE, kJ/d	740 a	686 ab	636 b	897	**
RPE, kJ/d	373	380	367	351	ns
RFE, kJ/d	367 a	306 b	269 b	629	**

a-b Means within a row followed by different Letters are significantly different

** ($P < 0.01$); ns, not significant

followed by increased CO_2 production as the dose of β -agonist was increased. Therefore, the values of respiratory quotients (RQ) among the groups were not different. The range of RQ was between 0.93 and 0.92.

Based on the measurements of O_2 consumption and CO_2 production, HE showed the same pattern among the groups. The heat production of chickens treated with β -agonist at the dose of 0.25 ppm was higher than the control group, and the increase of the dose up to 1.0 ppm showed further significant increase in HE ($P < 0.01$).

The means of GE were not different between the groups, although the values tended to decrease as the dose of β -agonist was increased. However, the means of HE and RE were significantly different between the groups ($P < 0.01$). The highest RE was 747 kJ/d in the control group, while β -agonist treatment caused reductions of 55 and 99 kJ in T1 and T2, respectively. The variation between T0 and T1 was not significantly different, but the larger reduction between T0 and T2 was significantly different ($P < 0.01$). The total RE is the sum of energy retained in protein (RPE) and in fat (RFE) as measured in the balance and respiration experiments (Table 2). RPE was not different but RFE of the treated chickens was significantly different from the control ($P < 0.01$).

In the control group, CO_2 production and O_2 consumption were lower than in treated animals. The pattern of increase was in parallel to the increase in the dose of L-644,969. Therefore, RQ was constant since the equation of RQ is the ratio of CO_2 production to O_2 consumption. The values of RQ were between 0.93 and 0.92, being higher than measured by Thorbek and Chwalibog (1984) in growing chickens and by Chwalibog (1985) in laying hens. The same pattern was also found for HE being increased as the dose of β -agonist was increased. It seems that the immediate effect of L-644,969 was an increase in HE followed by the decrease in the total RE. Similar increases in HE were also reported by

Xiyi et al. (1994) when broilers were fed clenbuterol. However, Aijun and Farrell (1991) were unable to detect any differences in HE in broilers fed a diet containing cimaterol.

In the present study, an improvement of RPE was shown when the data were adjusted to the equal IP. The results showed that L-644,969 increased RPE, but the higher level of β -agonist did not further increase RPE ($T_0=372$, $T_1=383$ and $T_2=376$ kJ/kg $\text{LW}^{0.75}$; $\text{SEM}=51.8$; $P < 0.05$). Since the RFE was the only other part of retained energy beside RPE, the increase in the value of RFE/RE would decrease the value of RPE/RE (Figure 1 and 2), indicating an increased protein deposition and decreased fat deposition as the dose of β -agonist was increased. The review by Reeds and Mersmann (1991) showed that β -agonists could reduce fat deposition by mean of lipolysis in treated animals.

Conclusion

Finally, the increase in partial efficiency of ME for retained protein energy and the decrease in partial efficiency of ME for retained fat energy in L-644,969 treated broilers indicated that β -agonist is an effective repartitioning agent in growing broilers. This study indicates that β -agonist function as a repartitioning agent is by increasing protein utilization and decreasing fat deposition in chickens.

References

- Aijun, Y and D. J. Farrel. 1991. Energy exchange of broilers, selected for fatness and leanness, on diets with and without a repartioning agent. *Brit. Poult. Sci.*, 32(2):337-342.
- Brouwer, E. 1965. Report on sub-committee on constants and factors. In: *Symposium on Energy Metabolism*. EEAP Pub., 14:441-443.
- Chwalibog, A. 1985. *Studies on Energy Metabolism in Laying Hens*. Report

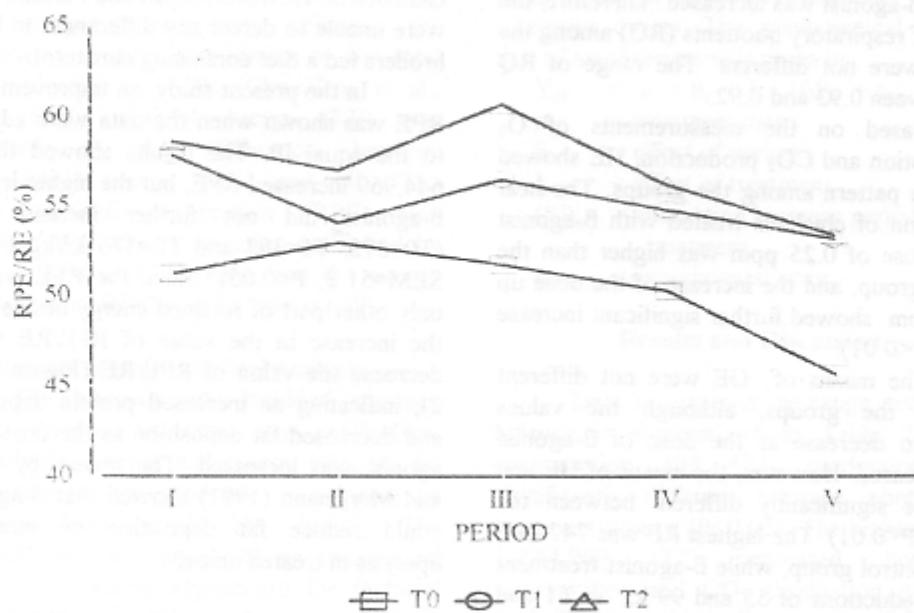


Figure 1. Energy retained in protein (RPE) in relation to total retained energy (RE).

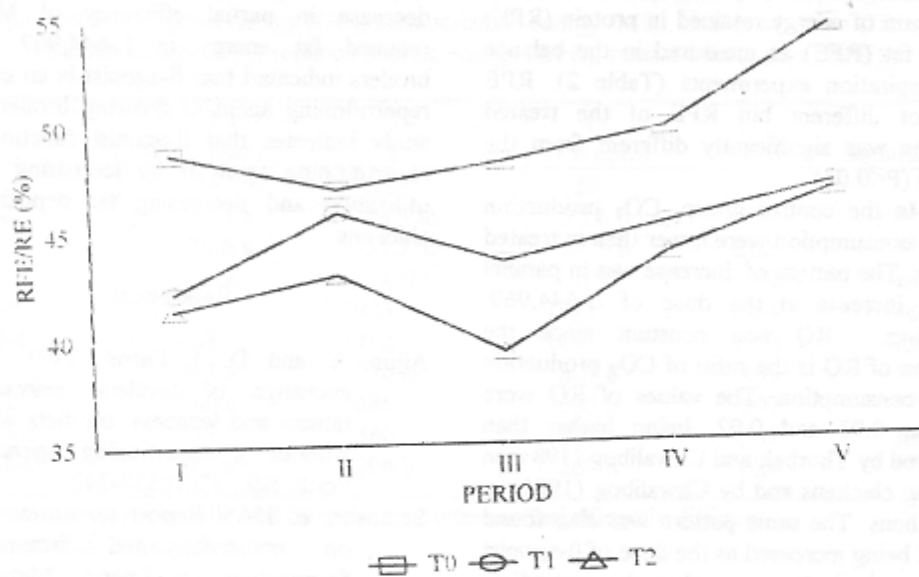


Figure 2. Energy retained in fat (RFE) in relation to total retained energy (RE).

- from the National Institute of Animal Science, Denmark. 578:48pp.
- Chwalibog, A., K. Jensen and K. Thorbek. 1996. Quantitative protein and fat metabolism in bulls calves treated with β -adrenergic agonist. Arch. Anim. Nut., 49:159-167.
- Chwalibog, A., K. Jakobsen, S. Heckel and G. Thorbek. 1992. Estimation of quantitative oxidation and fat retention from carbohydrate, protein and fat in growing pigs. J. Anim. Phy. and Anim. Nut., 68: 123-135.
- Dalrymple, R. H., P. K. Baker, P. E. Ginger, D. L. Ingle, J. M. Pensack and C. A. Ricks. 1984. A repartioning agent to improve performance and carcass composition of poultry. Poult. Sci., 63: 2376-2383.
- Moser, R. L., R. H. Dalrymple, S. G. Cornelius, J. E. Pettigrew and C. E. Allen. 1986. Effect of cimaterol (CL-263,780) as repartioning agent in the diet for finishing pigs. J. Anim. Sci., 62:21-26.
- Reeds, P. J. and H. J. Mersmann. 1991. Protein and energy requirments of animals treated with β -adrenergic agonist: A discussion. J. Anim. Sci. 69:1542-1550.
- SAS Institute. 1988. SAS/STAT Guide for Personal Computer. Version 6 Ed.(Cary, NC, SAS Institute Inc.).
- Thorbek, G. and A. Chwalibog. 1984. Influence of environmental temperature on nitrogen and energy metabolism in growing chickens. Arch. Exp. Vet. and Med., 38: 458-466.
- Xiyi, M.; J. Shihou, Y. Xiufang, L. Yingjun and S. Qun. 1994. Influence of clenbuterol on energy and protein metabolis in broilers. Proceedings of the 13th Symposium on Energy Metabolism, EAAP Publ., 76: 285-288.