

Effects of Quantitative Feed Restriction on Serum Triacylglycerol, Cholesterol and Growth Related Hormones in White Pekin Ducks

A.H. El-Far

Department of Biochemistry, Faculty of Veterinary Medicine, Damanhour University, Egypt

Abstract: This study was conducted to evaluate the effect of feed restriction on some biochemical parameters-related to growth in white Pekin duck. Therefore, fifty white Pekin ducks were allocated into two equal groups as *ad libitum* and feed restriction group, in which each one was arranged in five replicates. The birds were allowed to free feed access from 1st to 7th days and from 15th to 49th days of age and there was feed restricted from 8th to 14th days. The results indicated that feed restriction induced significant increase in serum corticosterone and significant decreases in serum triacylglycerol, cholesterol, duck growth hormone 1 and chicken insulin-like growth factor 1. It has been concluded that even if the feed restriction spares some economic feeding costs but it retards the growth of white Pekin ducks that retardation have been evidenced by the significant decreases in serum duck growth hormone 1 and chicken insulin-like growth factor 1.

Key words: Feed Restriction • Corticosterone • Duck Growth Hormone 1 • Chicken Insulin-Like Growth Factor 1

INTRODUCTION

Poultry production represents an important sector especially in the developing countries to meet household food demands and as additional sources of incomes [1]. The Food and Agriculture Organization of the United Nations (FAO) estimated the global population of domestic chickens and ducks at over 18 billion and 1 billion, respectively [2]. The poultry industry is dominated by commercial farms while in developing countries; production consists of village or backyard poultry, which is often extensive [3]. Poultry sector in Egypt is one of the major sources of animal protein supply [4]. Poultry production plays a major role in providing a large and cheap source of animal protein in Egypt, beside pure Egyptian breeds there were some local developed strains that established for both meat and egg production [5]. Duck meat production constitutes around 4.3% of the overall poultry meat available in the world [6]. The world production of duck meat was 3.78 million tons in 2008 and will continue to grow at a rate in excess of three percent per annum [7]. Current feeding strategies in growing Pekin ducks should aim at higher body weight gain, breast meat yield, better feed conversion ratio (FCR) and lower fat content of carcasses[8].

Feed represents 70% of the total costs in poultry production [9]. Therefore, different feed regimes were conducted in different research where the full feeding or feed restrictions were beneficial for ducks. *Ad libitum* feeding have led to increase of growth rates poultry; unfortunately this high growth rate is associated with increased body fat deposition, high mortality and high incidence of metabolic and skeletal disorders which have negative impact on broilers [10]. Therefore, there is a critical need to increase efforts to reduce some of these problems and reduced feed cost [11]. Quantitative feed restriction has been found to reduce body weights [12] and delay sexual maturity [13]. Feed restriction, which is denying the fast growing birds a full access to nutrients that are required for their normal growth and development, is categorized into quantitative and qualitative feed restriction [14]. The potential of feed restriction programs as management tools related to decreasing the incidence of metabolic disease, carcass fat deposition, reduce maintenance requirements and improvement of feed efficiency in broiler chickens production [15]. Also can be lead to economical saving in cost of feeding in broiler chicken production, thus may be usefulness for commercial broiler chicks production farms [16].

Our study aimed to investigate the effects of both *ad libitum* and quantitative feed restriction on white Pekin ducks through determination of triacylglycerol (TAG), cholesterol, corticosterone, duck growth hormone 1 (GH1) and chicken insulin-like growth factor 1 (IGF-1).

MATERIALS AND METHODS

Birds and Experimental Design: Fifty white Pekin ducklings were obtained from the French company, El-Sadat city, Egypt were used and housed in clean and well-ventilated open-sided house with concrete floor that had been previously disinfected by fumigation using formaldehyde gas produced by mixing formalin 40% with potassium permanganate powder at a ratio of 2:1. The birds were randomly allocated into two groups of twenty-five birds as *ad libitum* and feed restriction groups in which each group was arranged as five replicates. Ducklings were floor brooded at 33°C at the bird's level during first three days of age and then temperature reduced gradually till room temperature at 14 days of age. The house was provided by electric heaters in addition to incandescent lamps, fresh and clean wheat straw litter was used along a 24 hours constant light. The ingredient and chemical analysis of the basal diet were presented in Table (1) and (2), respectively [17].

Skip-a-day deprivation of feed is a quantitative method applied by removing feed for 24 hour periods during the starter period. All ducks were allowed to free access to feed from 1st to 7th days and 15th to 49th days of age, water provided at all time. Ducks were feed restricted from 8th to 14th days of age i.e. *ad libitum* feeding for a day followed by starvation for a day [18].

Table 1: Ingredient composition (%) of the basal diet.

Ingredient	Composition (%)
Ground yellow corn	63.1
Soya bean meal (44% CP)	28.2
Corn gluten meal (60% CP)	4.3
Vegetable oil *	0.6
Dicalcium phosphate **	1.8
Ground limestone	1.1
Common salts	0.4
Mineral and Vitamin Premix	0.3
Lysine	0.1
Methionine	0.1

* Vegetable oil composed of soybean oil, cottonseed oil and sunflower oil.

**Dicalcium phosphate contains 18% phosphorus, 23% calcium.

Table 2: The calculated chemical analysis (%) of the basal diet on DM basis.

Nutrients	(%)
ME Kcal/Kg diet	2655.47
Crude protein	20.36
Ether extract	3.387
Crude fiber	3.4
Calcium	0.999
Available phosphorus	0.469
Sodium	0.173
Manganese (mg/Kg)	192.77
Lysine	1.066
Methionine	0.452
Methionine + cystine	0.798

Biochemical Analysis: Blood samples were collected from the wing vein at 7th, 14th and 49th days of experiment. Blood samples were centrifuged at 3000 RPM for 5 min to separate clear sera. Collected serum samples were subjected to biochemical analysis of TAG [19] and cholesterol [20]. In addition, chicken corticosterone, GH1 and IGF-1 ELISA kits were purchased from Cusabio (<http://www.cusabio.com/>). The UNICO 2100 UV-Spectrophotometers, ELx800 Absorbance Microplate Reader and other laboratory equipment aids were used for biochemical study.

Statistical Analysis: The obtained data was statistically analyzed by t-test by SAS software 1996 [21].

RESULTS

The effects of feed regime either *ad libitum* or restricted feed on serum TAG, cholesterol, corticosterone, GH1 and IGF-1 of Pekin ducks were showed in Tables (3& 4) in which feed restriction induced a significant decrease ($P<0.05$) in serum TAG and cholesterol at 14th days (The end of feed restriction period of experiment) when compared with *ad libitum* group. In contrary, the levels of corticosterone the stress biomarker were significantly increase ($P<0.05$). Meanwhile, the levels of serum GH1 and IGF-1 were significant decrease ($P<0.05$) at 14th day in relation to control one (Table, 4).

DISCUSSION

Diet restriction decreased the concentrations of plasma lipids [22] and reduced the basal metabolism [23]. The turnover of adipose tissue TAG is responsive to changes in macronutrient intake in which fasting

Table 3: Mean values of serum TAG and cholesterol levels in *ad libitum* and feed-restricted groups of white Pekin ducks

Groups	TAG (mg/dl)			Cholesterol (mg/dl)		
	7 th day	14 th day	49 th day	7 th day	14 th day	49 th day
<i>Ad libitum</i>	93±4.19 ^a	104.5±2.53 ^a	102.29±2.61 ^a	173.37±5.82 ^b	169.84±9.09 ^a	179.3±0.46 ^b
Feed restricted	91±3.72 ^a	68.13±0.13 ^b	99.83±6.58 ^a	175.79±7.32 ^b	134.95±4.41 ^b	176.4±2.55 ^b

Means within the same column carrying different letters are significantly different between groups (p<0.05)

Table 4: Mean values of serum corticosterone, GH1 and IGF-1 levels in *ad libitum* and feed-restricted groups of white Pekin ducks

Groups	Corticosterone (ng/ml)			GH1 (ng/ml)			IGF-1 (pg/ml)		
	7 th day	14 th day	49 th day	7 th day	14 th day	49 th day	7 th day	14 th day	49 th day
<i>Ad libitum</i>	52.66±7.08 ^a	68.04±4.55 ^b	77.68±7.02 ^a	73±3.92 ^a	84.73±4.81 ^a	102.55±3.81 ^a	151.18±0.11 ^a	151.27±0.13 ^a	151.1±0.07 ^a
Feed restricted	56.12±5.64 ^a	112.68±3.82 ^a	76.09±7.01 ^a	70.27±5.3 ^a	34.78±3.93 ^b	93.15±4.79 ^a	151.2±0.19 ^a	137.19±0.08 ^b	151.02±0.09 ^a

Means within the same column carrying different letters are significantly different between groups (p<0.05)

increases lipolysis [24]. Dietary restriction had increases in AMP-activated protein kinase, an enzyme that stimulates glucose uptake and fatty acid oxidation while decreasing lipid synthesis [25]. Nutrient restriction induced a greater decrease in the plasma glucose that, coupled with increase in fat mobilization from adipose tissue, was sufficient to meet the nutrient needs for animal [26]. Fat mobilization induces a body weight loss whereas the concentrations of plasma phospholipids, total cholesterol and low-density lipoprotein decreased [27]. Total plasma lipids, triacylglycerols, cholesterol and high-density lipids were lower in the feed restricted chickens [28].

Corticosterone is a 21-carbon steroid hormone of the corticosteroid type produced in the cortex of the adrenal glands in rodents and other non-human animals, which act primarily as glucose sparing through the increment of lipid oxidation and protein degradation [29]. Feed restriction resulted in higher corticosterone [30]. Corticosterone both inhibits protein synthesis and degrades protein that pronounced by a slower feather growth and an extended period of poor flight [31]. The catabolic effects of glucocorticoids on muscle protein metabolism are well known. It is generally agreed that glucocorticoids inhibit muscle protein synthesis and stimulate muscle protein breakdown [32]. Once in the nucleus, the glucocorticoid receptor activates the expression of two target genes, encoding REDD1 and KLF15 [33] that finally induces branched-chain amino acid degradation in muscles [34]. Glucocorticoids modulate the secretion of GH [35] and directly inhibit growth hormone effects at target tissues by inhibiting IGF-1 and other growth factors. The effects of stress on the growth axis may account for the delay in growth [36, 37].

Growth primarily involves cell proliferation, but also may result from cell hypertrophy. The primary hormones involved with growth are GH, triiodothyronine (T₃) and IGF-1 [38]. Feed restriction induces changes in mRNA expression of the GH/IGF-I and thyroid hormone receptors [39] and changes the growth hormone-insulin-like growth factor-1 [40]. The continuous exposure to glucocorticoids has a continuous catabolic effect that associated with reduced weight gain and nitrogen retention [41]. The greater sensitivity of the intestine to the catabolic actions of corticosterone may be related to the interaction of corticosterone with IGF-I. Intestinal tissue is especially sensitive to the anabolic action of IGF-1 [42].

CONCLUSION

From the obtained data we can conclude that, the feed restriction induced a great stress on white Pekin ducks during growth period which evidenced by the significant increase in serum corticosterone and significant decreases in serum TAG, cholesterol, GH1 and IGF-1.

REFERENCES

- Conan, A., F.L. Goutard, S. Sorn and S. Vong, 2012. Biosecurity measures for backyard poultry in developing countries: a systematic review. BMC Veterinary Research, 8: 240.
- FAO, 2012. Faostat.Production.Live animals.<http://faostat.fao.org/site/573/default.aspx#ancor>
- Sonaiya, E.B. and S.E.J. Swan, 2004. Small-scale poultry production. Rome: FAO.

4. AbdelGaied, S. and H.H. Bakri, 2009. An economic evaluation for the impacts of spreading of bird flu on poultry sector in Egypt. World Journal of Agricultural Sciences, 5(3): 264-269.
5. Taha, A.E., M.A. El-Edel, H.F. El-Lakany and R.S. Shewita 2012. Growth Performance and Immune Response against Newcastle and Avian Influenza Vaccines in Egyptian Chicken Strains. Global Veterinaria, 9(4): 434-440.
6. USDA, 2006. (United States Department of Agriculture) International Egg and Poultry Review, pp: 09-17.
7. FAO, 2010. Poultry meat and eggs: agribusiness handbook. Viale delle Terme di Caracalla.
8. Hall, A.D. and D.M. Martin, 2006. Where next with duck meat production? International Hatchery Practice, 20(6): 7-8.
9. Willems, O.W., S.P. Miller and B.J. Wood, 2013. Assessment of residual body weight gain and residual intake and body weight gain as feed efficiency traits in the turkey (*Meleagris gallopavo*). Genetics Selection Evolution, 45: 26.
10. Zubair, A.K. and S. Leeson, 1996. Changes in body composition and adipocyte cellularity of male broilers subjected to varying degrees of early-life feed restriction. Poult. Sci., 75: 719-728.
11. Sarvestani, T.S., N. Dabiri, M.J. Agah and H. Norollahi, 2006. Effect of Pellet and Mash Diets Associated with Biozyme Enzyme on Broilers Performance, International Journal of Poultry Science, 5(5): 485-490.
12. Fattori, T.R., H.R. Wilson, R.H. Harms, F.B. Mather, R.D. Miles and G.D. Butcher, 1993. Response of broiler breeder females to feed restriction below recommended levels. Characterizing the onset of sexual maturity. Poult. Sci, 72: 2044-2051.
13. Krishnappa, P., G. Devegowda, G.R. Lokanath and B.S. Ramappa, 1992. Effect of restricted feeding on subsequent performance of broiler breeder dams. Indian Poult. Sci., 27(1): 29-31.
14. Fanooci, M. and M. Torki, 2010. Effects of Qualitative Dietary Restriction on Performance, Carcass Characteristics, White Blood Cell Count and Humoral Immune Response of Broiler Chicks. Global Veterinaria, 4(3): 277-282.
15. Sahraei, M., 2012. Feed Restriction in Broiler Chickens Production: A Review. Global Veterinaria, 8(5): 449-458.
16. Sahraei, M. and M.H.M. Hadloo, 2012. Effect of Physical Feed Restriction in Finisher Period on Carcass Traits and Broiler Chickens Performance. Global Veterinaria, 9(2): 201-204.
17. Ayoub, M.M., A.H. El-Far, N.M. Taha, M.A. Korshom, A.A. Mandour, H.S. Abdel-Hamid and M.S. El-Neweshy, 2011. The Biochemical Protective Role of Some Herbs against Aflatoxicosis in Ducklings: II. *Nigella sativa*, Lucrari Stiintifice, 55: 68-77.
18. Tan, B.J. and S. Ohtani, 2000. Effect of different early feed restriction regimens on performance, carcass composition and lipid metabolism in male ducks. Anim. Sci J., 71: 586-593.
19. Fossati, P. and L. Prencipe, 1982. Serum Triacylglycerols Determined Colorimetrically with an Enzyme that Produces Hydrogen Peroxide, Clin. Chem., 28(10): 2077-2080.
20. Zak, B., R.C. Dickenman, E.G. White, H. Burnett and P.J. Cherney, 1954. Rapid estimation of free and total cholesterol. Am. J. Clin. Pathol, 24(11): 1307-1315.
21. SAS, 1996. Statistical Analysis System. Users Guide Statistics, SAS Institute Cary, North Carolina.
22. Vidal, J., 2002. Updated review on the benefits of weight loss. Int. J. Obes. Relat. Metab. Disord. 26 Suppl, 4: S25-S28.
23. Van Weyenberg, S., M. Hesta, J. Buyse and G.P. Janssens, 2008. The effect of weight loss by energy restriction on metabolic profile and glucose tolerance in ponies. J Anim Physiol Anim Nutr (Berl), 92(5): 538-545.
24. Varady, K.A., D.J. Roohk, Y.C. Loe, B.K. McEvoy-Hein and M.K. Hellerstein, 2007. Effects of modified alternate-day fasting regimens on adipocyte size, triacylglycerol metabolism and plasma adiponectin levels in mice. Journal of Lipid Research, 48: 2212-2219.
25. Richter, E.A. and N.B. Ruderman, 2009. AMPK and the biochemistry of exercise: implications for human health and disease. Biochem J, 418(2): 261-275.
26. Bjerre-Harpoth, V., N.C. Friggens, V.M. Thorup, T. Larsen, B.M. Damgaard, K.L. Ingvarsten and K.M. Moyes, 2012. Metabolic and production profiles of dairy cows in response to decreased nutrient density to increase physiological imbalance at different stages of lactation. J. Dairy Sci, 95: 2362-2380.

27. Sejersen, H., M.T. Sorensen, T. Larsen, E. Bendixen and K.L. Ingvarsen, 2013. Liver protein expression in young pigs in response to a high-fat diet and diet restriction. *J. Anim. Sci.*, 91: 147-158.
28. Rajman, M., M. Jurani, D. Lamosova, M. Macajova, M. Sedlackova, L. Kost'a, D. Jezova and P. Vyboh, 2006. The effects of feed restriction on plasma biochemistry in growing meat type chickens (*Gallus gallus*). *Comp Biochem Physiol A Mol Integr Physiol.*, 145(3): 363-71.
29. Johnson, B.N. and B.K. Yamamoto, 2010. Chronic Stress Enhances the Corticosterone Response and Neurotoxicity to 3,4-Methylenedioxy-methamphetamine (MDMA): The Role of Ambient Temperature. *JPET*, 335: 180-189.
30. Soleimani, A.F., I. Zulkifli, A.R. Omar and A.R. Raha, 2011. Neonatal feed restriction modulates circulating levels of corticosterone and expression of glucocorticoid receptor and heat shock protein 70 in aged Japanese quail exposed to acute heat stress. *Poult Sci.*, 90(7): 1427-34.
31. Auclair, D., D.R. Garrel, Z.A. Chaouki and L.H. Ferland, 1997. Activation of the ubiquitin pathway in rat skeletal muscle by catabolic doses of glucocorticoids. *Am J Physiol.*, 272: C1007-1016.
32. Romero, L.M., D. Stochlic and J.C. Wingfield, 2005. Corticosterone inhibits feather growth: Potential mechanism explaining seasonal down regulation of corticosterone during molt. *Comparative Biochemistry and Physiology Part A: Molecular & Integrative Physiology*, 142: 65-73.
33. Shimizu, N., N. Yoshikawa, N. Ito, T. Maruyama, Y. Suzuki, S. Takeda, J. Nakae, Y. Tagata, S. Nishitani, K. Takehana, M. Sano, K. Fukuda, M. Suematsu, C. Morimoto and H. Tanaka, 2011. Crosstalk between glucocorticoid receptor and nutritional sensor mTOR in skeletal muscle. *Cell Metab*, 13: 170-182.
34. Waddell, D.S., L.M. Baehr, J. van den Brandt, S.A. Johnsen, H.M. Reichardt, J.D. Furlow and S.C. Bodine, 2008. The glucocorticoid receptor and FOXO1 synergistically activate the skeletal muscle atrophy-associated MuRF1 gene. *Am. J. Physiol. Endocrinol. Metab*, 295: E785-E797.
35. Gherardo, M. and G. Andrea, 2013. Glucocorticoids and the regulation of growth hormone secretion. *Nature Reviews Endocrinology*, 9: 265-276.
36. Unterman, T.G. and L.S. Phillips, 1985. Glucocorticoid effects on somatomedin and somatomedin inhibitors. *J Clin Endocrinol Metab*, 61: 618-626.
37. O'Connor, T.M., D.J. O'Halloran and F. Shanahan, 2000. The stress response and the hypothalamic-pituitary-adrenal axis: from molecule to melancholia. *QJM.*, 93(6): 323-333.
38. Koyuncu, C.E., S.T. Yildirmak, M. Temizel, T. Ozpacaci, P. Gunel, M. Cakmak and Y.G. Ozbanazi, 2013. Serum Resistin and Insulin-Like Growth Factor-1 Levels in Patients with Hypothyroidism and Hyperthyroidism. *Journal of Thyroid Research*, 2013, Article ID 306750, 6 pages.
39. Li, Y., X. Yang, Y. Ni, E. Decuypere, J. Buyse, N. Everaert, R. Grossmann and R. Zhao, 2012. Early-age feed restriction affects viability and gene expression of satellite cells isolated from the gastrocnemius muscle of broiler chicks. *J Anim Sci Biotechnol.*, 3(1): 33.
40. Dessauge, F., V. Lollivier, B. Ponchon, R. Bruckmaier, L. Finot, S. Wiart, E. Cutullic, C. Disenhaus, S. Barbey and M. Boutinaud, 2011. Effects of nutrient restriction on mammary cell turnover and mammary gland remodeling in lactating dairy cows. *J Dairy Sci.*, 94(9): 4623-4635.
41. Brownlee, K.G., P.C. Ng, M.J. Henderson, M. Smith, J.H. Green and P.R. Dear, 1992. Catabolic effect of dexamethasone in the preterm baby. *Arch Dis Child*, 67: 1-4.
42. Steeb, C.B., J.F. Trahair, F.M. Tomas and L.C. Read, 1994. Prolonged administration of IGF peptides enhances growth of gastrointestinal tissues in normal rats. *Am J Physiol*, 266: G1090-G1098.