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≪Research Note≫

# Effects of Fasting and Refeeding on the mRNA levels of Insulin-like Growth Factor-binding Proteins in Chick Liver and Brain

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The physiological functions of insulin-like growth factor-binding proteins (IGFBPs) in mammals have been evaluated in several studies. However, the physiological roles of IGFBPs in chickens have not yet been elucidated. In this study, we examined the effects of short-term (6 h) fasting and refeeding on the mRNA levels of IGFBPs in chick liver and brain. Eighteen 8-day-old chicks were weighed and allocated to three groups on the basis of body weight, and subjected to *ad libitum* feeding, 6 h of fasting, or 6 h of fasting followed by 6 h of refeeding. After the chicks were euthanized by decapitation, the liver and brain were excised, and the brain was dissected into six segments (telencephalon, optic lobes, cerebellum, rostral part of the brainstem, middle part of the brainstem, and caudal part of the brainstem). IGFBP mRNA levels were determined by qRT-PCR. Fasting significantly increased the mRNA levels of IGFBP-1 and -2 in the chick liver, and these changes were reversed by 6 h of refeeding. The mRNA levels of IGFBP-3 in the middle part of the brainstem and IGFBP-5 in the optic lobes were decreased by 6 h of fasting and were not reversed after 6 h of refeeding. These findings suggest that IGFBP-1 and -2 in the liver, IGFBP-3 in the middle part of the brainstem, and IGFBP-5 in the optic lobes may play physiological roles in response to short-term changes in the nutritional status of chicks.

**Key words**: brain, fasting, insulin-like growth factor, liver, refeeding

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## Introduction

Insulin-like growth factor-binding proteins (IGFBPs) are thought to function as carrier proteins in circulation, and locally expressed IGFBPs can inhibit and/or potentiate IGF activities in mammals (Duan and Xu, 2005). In addition, some IGFBPs show IGF-independent biological effects. For example, IGFBP-3 and -5 have nuclear localization sequences, and the nuclear transport protein importin-$\beta$ has been shown to mediate the translocation of both IGFBP-3 and -5 (Firth and Baxter, 2002). However, the physiological roles of IGFBPs in birds have not yet been fully investigated, although full-length cDNA sequences of IGFBP-1 to -5 are available in the GenBank database.

In genetically fat and lean chickens fasted for 48 h, among four circulating IGFBPs with molecular weights of 28, 34, 40, and 60 kDa, the 28-kDa, and to a lesser extent 34-kDa and 60-kDa IGFBPs were increased (Beccavin *et al*., 1999). In layer chickens, dietary protein restriction (Leili and Scanes, 1998), food deprivation (Leili *et al*., 1997), and dexamethasone (Leili and Scanes, 1998) affected the binding activity of plasma IGFBPs to IGF-1. Polymorphisms in *IGFBP-2* (Lei *et al*., 2005; Li *et al*., 2006; Leng *et al*., 2009) and *IGFBP-3* (Ou *et al*., 2009) may be related to growth and/or carcass traits. Kita *et al*. (2002) investigated the response of IGFBP-2 expression to variations in nutritional status in several tissues of layer chickens; they found that the mRNA level of IGFBP-2 in the liver was significantly increased after 2 days of food deprivation, and this increase was reversed after 6 h of refeeding. Thus, the expression of hepatic IGFBPs may influence the plasma level of IGFBPs, which in turn inhibit and/or potentiate IGF activities in chickens.

Recently, we found that central and peripheral administration of IGF-1 significantly suppressed food intake in chicks (Fujita *et al*., 2017). Additionally, we showed that the mRNA levels of IGF-1 in the liver were significantly increased upon refeeding and that the IGF-1 receptor is expressed throughout the brain (Fujita *et al*., 2017). Evidence demonstrates that IGF-1 crosses the blood-brain barrier (Reinhardt and Bondy, 1994; Armstrong *et al*., 2000; Nishijima *et al*., 2010). These

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findings suggest that hepatic IGF-1 functions as a satiety signal in the brain in chicks. The mRNA level of IGFBP-2 in the brain was significantly decreased after 2 days of food deprivation and this decrease was not reversed after 24 h of refeeding in layer chickens (Kita *et al*., 2002). Thus, it is possible that brain-expressed IGFBPs influence the function of circulating IGF-1 in the chicken brain. However, although the brainstem is proposed to contain the satiety center (Richards and Proszkowiec-Weglarz, 2007), the physiological functions of IGFBPs in the brainstem in chicks have not been investigated.

In the present study, we examined the effects of short-term (6 h) fasting and refeeding on the mRNA levels of IGFBPs in chick liver and brain. Our findings suggest the physiological importance of IGFBP-1 and -2 in the liver, IGFBP-3 in the middle part of the brainstem, and IGFBP-5 in the optic lobes under the fasting and/or refeeding condition in chicks.

## Materials and Methods

One-day-old male broiler chicks (ROSS 308) were purchased from a local hatchery (Ishii, Tokushima, Japan). They were given free access to water and a commercial chick starter diet (Nippon Formula Feed, Kanagawa, Japan). This study was approved by the Institutional Animal Care and Use Committee and was carried out according to the Kobe University Animal Experimentation Regulation.

Eighteen 8-day-old chicks were weighed and divided in three groups on the basis of body weight, and they were euthanized by decapitation after 0 or 6 h of fasting, or 6 h of refeeding after 6 h of fasting. The whole brains were collected, preserved in RNAlater® tissue storage reagent (Sigma-Aldrich, St. Louis, MO, USA), and dissected into six segments (telencephalon, optic lobes, cerebellum, rostral part of the brainstem, middle part of the brainstem, and caudal part of the brainstem) as described previously (Aoki *et al*., 2017). Total RNA extraction and cDNA synthesis were conducted using Sepasol-RNA I (Nacalai Tesque, Kyoto, Japan) and ReverTra Ace® qPCR RT Master Mix with gDNA Remover (Toyobo, Osaka, Japan) as described previously (Honda *et al*., 2015a). cDNAs of chicken IGFBP -1, -2, -3, -4, and -5 (GenBank accession numbers: NM_001001294, NM_205359, NM_001101034, NM_204353, and XM_422069, respectively) were amplified with the following primers: IGFBP-1 sense, 5′-CCC AAC TGT AAC AAG AAT GGA TTT T-3′; IGFBP-1 antisense, 5′-CGG AAT CTC CAT CCA GTG AAG-3′; IGFBP-2 sense, 5′-AAT GGG CAG CGT GGA GAG T-3′; IGFBP-2 antisense, 5′-CTG GAT CAC CTT CCC ATG GA-3′; IGFBP-3 sense, 5′-ATC AGG CCA TCC CAA GCT T-3′; IGFBP-3 antisense, 5′-GAT GTG CTG TGG AGG CAA ATT-3′; IGFBP-4 sense, 5′-GAG CAC CCC AAC AAC AGC TT-3′; IGFBP-4 antisense, 5′-CCG TTG TTG ATG CGC TTT G-3′; IGFBP-5 sense, 5′-CAA GGC CGA ACG GGA AT-3′; IGFBP-5 antisense, 5′-TCC TCC GTC ATC TCC GAT GT-3′. cDNA of ribosomal protein S17 (GenBank accession number: NM_204217), as an internal standard, was amplified with previously reported primers (Honda *et al*., 2015b). mRNA levels were quantified in duplicate using an Applied Biosystems 7300 Real-Time PCR system and SYBR® Premix Ex Taq™ II (Tli RNaseH Plus) (Takara Bio, Shiga, Japan) according to the supplier's recommendations.

Data were analyzed with one-way analysis of variance and Fisher's protected least significant differences post-hoc test. All statistical analyses were performed using the commercial package StatView version 5 (SAS Institute, Cary, NC, USA).

## Results and Discussion

As shown in Table 1, 6 h of fasting significantly increased the mRNA levels of IGFBP-1 and -2 in the chick liver, and these changes were reversed by 6 h of refeeding. The changes in IGFBP-2 are in agreement with the results of a study in 6-week-old layer chickens, in which hepatic IGFBP-2 mRNA expression was significantly increased after 2 days of food deprivation and this increase was reversed after 6 h of refeeding (Kita *et al*., 2002). Beccavin *et al*. (1999) reported that 48 h of fasting significantly increased plasma 28-kDa and 34-kDa IGFBPs in male lean chickens and suggested that these proteins are the equivalents of mammalian IGFBP-1 and -2, respectively. These findings suggest that the transcriptional regulation of hepatic IGFBP-1 and -2 may play a physiological role in response to a change in nutritional status induced by short-term fasting and refeeding in broiler chicks.

In mammals, the availability of blood IGFs to the receptors of target cells is limited by binding to IGFBPs. In chickens,

Table 1. **Effects of fasting and refeeding on the mRNA levels of insulin-like growth factor-binding proteins (IGFBPs) in chick liver**

| | Feeding | Fasting | Refeeding |
|---|---|---|---|
| IGFBP-1 | 1.00±0.12[a] | 4.81±1.91[b] | 1.07±0.26[a] |
| IGFBP-2 | 1.00±0.14[a] | 2.72±0.78[b] | 0.58±0.05[a] |
| IGFBP-3 | 1.00±0.08 | 0.93±0.07 | 0.80±0.12 |
| IGFBP-4 | 1.00±0.20 | 0.64±0.07 | 0.59±0.05 |
| IGFBP-5 | 1.00±0.12 | 0.70±0.06 | 0.95±0.17 |

Values are means±SEM of six chicks in each group. Values with different letters are significantly different ($P<0.05$).

Table 2. **Effects of fasting and refeeding on the mRNA levels of insulin-like growth factor-binding proteins (IGFBPs) in chick brain**

| | Feeding | Fasting | Refeeding |
|---|---|---|---|
| Telencephalon | | | |
| IGFBP-1 | 1.00±0.09 | 1.15±0.07 | 0.97±0.09 |
| IGFBP-2 | 1.00±0.04 | 1.11±0.10 | 0.95±0.05 |
| IGFBP-3 | 1.00±0.04 | 1.00±0.06 | 0.95±0.04 |
| IGFBP-4 | 1.00±0.06 | 0.90±0.09 | 1.10±0.07 |
| IGFBP-5 | 1.00±0.07 | 1.11±0.13 | 0.99±0.08 |
| Optic lobes | | | |
| IGFBP-1 | 1.00±0.09 | 1.01±0.12 | 0.94±0.11 |
| IGFBP-2 | 1.00±0.07 | 0.92±0.09 | 0.89±0.14 |
| IGFBP-3 | 1.00±0.06 | 0.86±0.04 | 0.88±0.07 |
| IGFBP-4 | 1.00±0.14 | 0.94±0.11 | 0.91±0.13 |
| IGFBP-5 | 1.00±0.03[a] | 0.85±0.08[ab] | 0.68±0.08[b] |
| Cerebellum | | | |
| IGFBP-1 | 1.00±0.13 | 1.22±0.05 | 1.05±0.21 |
| IGFBP-2 | 1.00±0.04 | 1.04±0.12 | 1.08±0.09 |
| IGFBP-3 | 1.00±0.12 | 0.97±0.07 | 1.12±0.10 |
| IGFBP-4 | 1.00±0.15 | 1.17±0.13 | 1.23±0.12 |
| IGFBP-5 | 1.00±0.11 | 1.26±0.07 | 1.00±0.14 |
| Rostral part of the brainstem | | | |
| IGFBP-1 | 1.00±0.06 | 1.05±0.10 | 0.81±0.07 |
| IGFBP-2 | 1.00±0.05 | 1.02±0.04 | 1.24±0.17 |
| IGFBP-3 | 1.00±0.04 | 0.94±0.03 | 0.95±0.04 |
| IGFBP-4 | 1.00±0.13 | 1.18±0.05 | 1.07±0.10 |
| IGFBP-5 | 1.00±0.04 | 0.98±0.07 | 1.01±0.08 |
| Middle part of the brainstem | | | |
| IGFBP-1 | 1.00±0.08 | 1.02±0.06 | 0.89±0.10 |
| IGFBP-2 | 1.00±0.04 | 0.97±0.05 | 0.89±0.07 |
| IGFBP-3 | 1.00±0.04[a] | 0.84±0.02[b] | 0.84±0.02[b] |
| IGFBP-4 | 1.00±0.11 | 0.87±0.07 | 0.94±0.07 |
| IGFBP-5 | 1.00±0.07 | 0.78±0.05 | 0.80±0.07 |
| Caudal part of the brainstem | | | |
| IGFBP-1 | 1.00±0.09 | 1.05±0.03 | 0.84±0.09 |
| IGFBP-2 | 1.00±0.07 | 1.10±0.18 | 0.90±0.07 |
| IGFBP-3 | 1.00±0.04 | 0.93±0.03 | 0.87±0.04 |
| IGFBP-4 | 1.00±0.11 | 1.13±0.09 | 1.32±0.16 |
| IGFBP-5 | 1.00±0.13 | 0.98±0.07 | 0.94±0.06 |

Values are means±SEM of six chicks in each group. Values with different letters are significantly different ($P<0.05$).

the 28-kDa IGFBP (the equivalent of mammalian IGFBP-1) released by hepatoma cells inhibits exogenous IGF-1-stimulated amino acid uptake by the hepatoma cells (Duclos *et al*., 1998). Kita *et al*. (2002) reported that the response of *IGFBP-2* gene expression to variations in nutritional status was rapid and differed in several tissues of layer chickens, which would help modulate the growth-promoting effect of circulating IGF-I through IGF-IGFBP complex formation. In the present study, the mRNA levels of hepatic IGFBP-1 and -2 were significantly increased by 6 h of fasting in broiler chicks. Therefore, it seems likely that the upregulation of hepatic *IGFBP-1* and *-2* expression may contribute to the suppression of IGF effects under the fasting condition in chickens.

Nagao *et al*. (2001) reported that intravascular administration of insulin reversed the fasting-induced increase in hepatic IGFBP-2 mRNA in layer chickens. Plasma insulin reportedly significantly decreases within 6 h of fasting in broiler chickens (Krestel-Rickert *et al*., 1986; Christensen *et al*., 2013; Saneyasu *et al*., 2017). Krestel-Rickert *et al*. (1986) reported that 4 h of fasting significantly decreased the plasma insulin level, and this decrease was returned to pre-fasted concentration within 15 min of feeding in broiler chickens. Bigot *et al*. (2003) reported that 30 min of re-feeding significantly increased plasma insulin in 3-week-old broiler chickens. It is therefore likely that plasma insulin may be involved in the fasting- and refeeding-induced changes in IGFBP-2 mRNA in the liver in broiler chicks.

The mRNA levels of most IGFBPs throughout the brain were not changed by fasting and refeeding (Table 2). However, IGFBP-3 mRNA in the middle part of the brainstem was significantly decreased by 6 h of fasting, and this change

was not reversed by 6 h of refeeding. The mRNA level of IGFBP-5 in the optic lobes was significantly decreased after 6 h of refeeding. In mammals, IGFBP-3 and -5 are associated with the cell surface and extracellular matrix and modulate IGF actions in the cells (Clemmons, 2001; Firth and Baxter, 2002). In addition, IGFBP-3 and -5 show IGF-independent biological effects (Firth and Baxter, 2002). The physiological importance of IGFBPs in the brain is not fully understood in both mammals and birds. However, it is possible that IGFBP-3 and -5 play important roles in the neurons of the middle part of the brainstem or optic lobes in response to fasting-induced changes in nutritional status in chickens.

We previously demonstrated that intravascular administration of IGF-1 significantly suppressed food intake in chicks (Fujita *et al*., 2017). The dorsal vagal complex (DVC) and arcuate nucleus (ARC) receive and integrate peripheral satiety signals in mammals (Williams and Elmquist, 2012). In chickens, the rostral and caudal parts of the brainstem include the DVC and infundibular nucleus (the avian equivalent of mammalian ARC), respectively (Kuenzel and Masson, 1988). In addition, we have reported that the IGF-1 receptor is expressed in these parts of the brain in chicks (Fujita *et al*., 2017). The plasma IGF-1 concentration was significantly decreased by 48 h of fasting and reversed by 48 h of refeeding in layer chickens (Kita *et al*., 2002). The effects of short-term fasting on the plasma IGF-1 concentration in chickens had not been investigated. However, McMurtry *et al*. (1996) demonstrated that the half-life of free IGF-1 was 5.17±0.27 min in broiler chickens. We previously confirmed that hepatic IGF-1 mRNA levels were significantly decreased by 6 h of fasting in 8-day-old broiler chicks (Fujita *et al*., 2017) and by 4 h of fasting in 14-day-old broiler chicks (unpublished data). These findings suggest that the plasma IGF-1 concentration may be significantly decreased under the 6-h fasted condition, which in turn stimulates appetite in broiler chicks. On the other hand, the mRNA levels of IGFBPs in the rostral part and the caudal part of the brainstem were not influenced by 6 h of fasting and refeeding in chicks (Table 2). It is therefore possible that IGFBPs in these parts are not involved in appetite regulation, at least in the experimental conditions used in this study.

In summary, we examined the effects of fasting and refeeding on IGFBP expression in the liver and brain. Our findings suggest that IGFBP-1 and -2 in the liver, IGFBP-3 in the middle part of the brainstem, and IGFBP-5 in the optic lobes may play physiological roles in response to nutritional changes in broiler chicks.

## References


Aoki K, Kondo M, Okuda M, Saneyasu T, Honda K and Kamisoyama H. Identification, expression analysis, and functional characterization of peptide YY in chickens (*Gallus gallus domesticus*). General and Comparative Endocrinology, 242: 11−17. 2017.

Armstrong CS, Wuarin L and Ishii DN. Uptake of circulating insulin-like growth factor-I into the cerebrospinal fluid of normal and diabetic rats and normalization of IGF-II mRNA content in diabetic rat brain. Journal of Neuroscience Research, 59: 649−660. 2000.

Beccavin C, Chevalier B, Simon J and Duclos MJ. Circulating insulin-like growth factors (IGF-I and -II) and IGF binding proteins in divergently selected fat or lean chickens: effect of prolonged fasting. Growth Hormone & IGF Research, 9: 187−94. 1999.

Bigot K, Taouis M and Tesseraud S. Refeeding and insulin regulate S6K1 activity in chicken skeletal muscles. Journal of Nutrition, 133: 369−373. 2003.

Butt AJ and Williams AC. IGFBP-3 and apoptosis-a licence to kill? Apoptosis, 6: 199−205. 2001.

Christensen K, McMurtry JP, Thaxton YV, Thaxton JP, Corzo A, McDaniel C and Scanes CG. Metabolic and hormonal responses of growing modern meat-type chickens to fasting. British Poultry Science, 54: 199−205. 2013.

Clemmons DR. Use of mutagenesis to probe IGF-binding protein structure/function relationships. Endocrine Reviews, 22: 800−817. 2001.

Daughaday WH, Kapadia M, Yanow CE, Fabrick K and Mariz IK. Insulin-like growth factors I and II of nonmammalian sera. General and Comparatve Endocrinology, 59: 316−325. 1985.

Duan C and Xu Q. Roles of insulin-like growth factor (IGF) binding proteins in regulating IGF actions. General and Comparatiive Endocrinology, 142: 44−52. 2005.

Duclos MJ, Insulin-like growth factor-I (IGF-1) mRNA levels and chickens muscle growth. Journal of Physiology and Pharmacology, 56: 25−35. 2005.

Duclos MJ, Chevalier B, Remignon H, Ricard FH, Goddard C and Simon J. Divergent selection for high or low growth rate modifies the response of muscle cells to serum or insulin-like growth factor-I *in vitro*. Growth Regulation, 6: 176−184. 1996.

Duclos MJ, Chevalier B, Upton Z, and Simon J. Insulin-like growth factor-I effect on chicken hepatoma cells (LMH) is inhibited by endogenous IGF-binding proteins. Growth Hormone & IGF Research, 8: 97−103. 1998.

Firth SM and Baxter RC. Cellular actions of the insulin-like growth factor binding proteins. Endocrine Reviews, 23: 824−854. 2002.

Fujita S, Honda K, Hiramoto D, Gyu M, Okuda M, Nakayama S, Yamaguchi M, Saneyasu T and Kamisoyama H, Central and peripheral administrations of insulin-like growth factor-1 suppress food intake in chicks. Physiology & Behavior, 179: 308−312. 2017.

Gordon SH. Effects of day-length and increasing day length programs on broiler welfare and performance. World's Poultry Science Journal, 50: 269−282. 1994.

Honda K, Saneyasu T, Okuda M, Uemura T and Kamisoyama H. Glucagon and neuromedin U suppress food intake in broiler chicks. Journal of Poultry Science, 52: 268−278. 2015a.

Honda K, Shimatani T, Aoki K, Yamaguchi T, Kondo M, Saneyasu T and Kamisoyama H. Glucagon-like peptide-2 functions as anorexigenic peptide not only in the central nervous system but also in the peripheral circulation in broiler chicks. Journal of Poultry Science, 52: 183−187. 2015b.

Honda K, Kondo M, Hiramoto D, Saneyasu T and Kamisoyama H. Effects of continuous white light and 12 h white-12 h blue light-cycles on the expression of clock genes in diencephalon, liver, and skeletal muscle in chicks. Comparative Biochemistry and Physiology Part A: Molecular & Integrative Physiology, 207: 73−78. 2017.

Kanatani M, Sugimoto T, Nishiyama K and Chihara K. Stimulatory

effect of insulin-like growth factor binding protein-5 on mouse osteoclast formation and osteoclastic bone-resorbing activity. Journal of Bone and Mineral Research, 15: 902−910. 2000.

Kita K, Nagao K, Taneda N, Inagaki Y, Hirano K, Shibata T, Yaman MA, Conlon MA and Okumura J. Insulin-like growth factor binding protein-2 gene expression can be regulated by diet manipulation in several tissues of young chickens. Journal of Nutrition, 132: 145−151. 2002.

Kuenzel WJ and Masson MA. Stereotaxic Atlas of the Brain of the Chick (*Gallus domesticus*). The Johns Hopkins University Press, Baltimore and London. 1988.

Krestel-Rickert DH, Baile CA and Buonomo FC. Changes in insulin, glucose and GH concentrations in fed chickens. Physiol & Behavior, 37: 361−363. 1986.

Lei MM, Nie QH, Peng X, Zhang DX and Zhang XQ. Single nucleotide polymorphisms of the chicken insulin-like factor binding protein 2 gene associated with chicken growth and carcass traits. Poultry Science, 84: 1191−1198. 2005.

Leng L, Wang S, Li Z, Wang Q, and Li H. A polymorphism in the 3'-flanking region of insulin-like growth factor binding protein 2 gene associated with abdominal fat in chickens. Poultry Science, 88: 938−942. 2009.

Li ZH, Li H, Zhang H, Wang SZ, Wang QG and Wang YX. Identification of a single nucleotide polymorphism of the insulin-like growth factor binding protein 2 gene and its association with growth and body composition traits in the chicken. Journal of Animal Science, 84: 2902−2906. 2006.

McMurtry JP, Francis GL, Upton Z, Walton PE, Rosselot G, Caperna TJ and Brocht DM. Plasma clearance and tissue distribution of labelled chicken and human IGF-I and IGF-II in the chicken. Journal of Endocrinology, 150: 149−160. 1996.

McMurtry JP, Francis GL and Upton Z. Insulin-like growth factors in poultry. Domestic Animal Endocrinology, 14: 199−229. 1997.

Nagao K, Aman Yaman M, Murai A, Sasaki T, Saito N, Okumura J and Kita K. Insulin administration suppresses an increase in insulin-like growth factor binding protein-2 gene expression stimulated by fasting in the chicken. British Poultry Science, 42: 501−504. 2001.

Nishijima T, Piriz J, Duflot S, Fernandez AM, Gaitan G, Gomez-Pinedo U, Verdugo JM, Leroy F, Soya H, Nuñez A and Torres-Aleman I. Neuronal activity drives localized blood-brain-barrier transport of serum insulin-like growth factor-I into the CNS. Neuron, 67: 834−846. 2010.

Ou JT, Tang SQ, Sun DX and Zhang Y. Polymorphisms of three neuroendocrine-correlated genes associated with growth and reproductive traits in the chicken. Poultry Science, 88: 722−727. 2009.

Parvin R, Mushtaq MMH, Kim MJ and Choi HC. Light emitting diode (LED) as a source of monochromatic light: a novel lighting approach for behavior, physiology and welfare of poultry, World's Poultry Science Journal, 70: 543−556. 2014.

Reinhardt RR and Bondy CA. Insulin-like growth factors cross the blood-brain barrier. Endocrinology, 135: 1753−1761. 1994.

Richards MP and Proszkowiec-Weglarz M. Mechanisms Regulating Feed Intake, Energy Expenditure, and Body Weight in Poultry. Poultry Science, 86: 1478−1490. 2007.

Saneyasu T, Shiragaki M, Nakanishi K, Kamisoyama H and Honda K. Effects of short term fasting on the expression of genes involved in lipid metabolism in chicks. Comparative Biochemistry and Physiology Part B: Biochemistry and Molecular Biology, 165: 114−118. 2013.

Scanes CG. Perspectives on the endocrinology of poultry growth and metabolism. General and Comparative Endocrinology, 163: 24−32. 2009.

Williams KW and Elmquist JK. From neuroanatomy to behavior: central integration of peripheral signals regulating feeding behavior. Nature Neuroscience, 15: 1350−1355. 2012.