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Hormones and Metabolism in Poultry

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1. Introduction

Poultry are domesticated species of birds. Birds were domesticated for eggs (chickens, ducks and geese), meat (chickens, ducks, geese, turkeys, ostriches, emus, pigeons and game birds) or other uses including feathers (ostriches, ducks and geese), leather (ostriches), specific oils (ostriches, emus), cock-fighting (chickens) and homing (pigeons). Table 1 summarizes the approximate time when and the location where the species were domesticated.

Poultry by global production	Domestication			Production in 2009	
	Species	When	Location(s)	Meat million metric tons	Eggs million metric tons
Chickens	Gallus gallus	5000 Before Common Era (BCE)	North East China	79.6	62.4
Turkeys	Meleagris gallopavo	200-2500 BCE	Meso-America	5.3	< 1.0
Ducks	Anas platyrinchos	3000 BCE	Fertile Crescent and East Asia/China	3.8	5.0[b]
Geese	Anser anser/Anser cynoides	3000 BCE	Fertile Crescent and East Asia/China	2.4[a]	
Pigeons	Columbia livia	3000 BCE	Fertile Crescent	< 1.0	< 1.0
Ostriches	Struthio camelus	1857	South Africa	< 1.0	< 1.0
Emus	Dromaius novaehollandiae	within last 100 years	Australia	< 1.0	< 1.0

[a] Goose and guinea fowl combined; [b] Eggs other than chicken – predominantly duck and goose

Table 1. Poultry – Their Domestication and Production (based on Scanes, 2011 and data from the Food and Agricultural Organization)

Investigations of the hormonal control of growth and/or metabolism in poultry have predominantly been performed using chickens (reviewed Scanes, 2009). Not only have such studies focused on the chicken as an important agricultural animal but also the chick embryo has long been a model for developmental biology. Moreover, the chicken is the model species for birds. There are numerically many fewer studies in turkeys, ducks and ostriches together with a substantial body of research in Japanese quail as another avian model species.

Research approaches have included ablation and replacement studies, assay of circulating hormone concentrations or gene expression in response to physiological perturbations, genetic models associated with a singe gene such a dwarf or obese chickens and genetic models produced by multi-generation selection for specific phenotypes such as fast or slow growth. There have been limited transgenic studies on the hormonal control of growth or metabolism. There is presently not a robust knock-out model in poultry.

2. Glucose homeostasis

Steady state or basal circulating concentrations of glucose are much higher in poultry than in mammals. Indeed the circulating concentrations of glucose reported in poultry species would be considered grossly hyperglycemic or symptomatic of diabetes mellitus in mammals. For instance, circulating concentrations of glucose were reported in chickens as being between 190 to 220 mg/dL (reviewed by Hazelwood, 1986) or more recently based on 15 studies as 234 ±11.8 (SEM) mg/dL [13 ± 0.7 mM](Scanes, 2008).

Physiological state	Change in circulating concentration of glucose mM	Reference
Fasting (24hours)	No change or - 1.2	Belo et al., 1976; Harvey et al., 1976; Hazelwood & Lorenz, 1959
Insulin Administration	- 6 mM	Harvey et al., 1976;
Glucagon administration	+ 12 mM	Harvey et al., 1976;

Table 2. Changes in circulating concentrations of glucose in chickens with physiological state or perturbation

Despite the difference in set point, circulating concentrations are maintained within tight limits by a series of homeostatic mechanisms (see table 2). The physiological mechanisms are discussed below. Table 3 summarizes changes in various metabolites during fasting in chickens.

3. Hormones controlling circulating concentrations of glucose and metabolism

Circulating concentrations are maintained within tight limits by a series of physiological mechanisms. Synthesis of fatty acid and triglycerides in poultry are anatomically separated in poultry. Lipogenesis occurs predominantly in the liver of poultry while adipose tissue is the site of triglyceride synthesis and breakdown or lipolysis. These processes are under the

Physiological state	Change in circulating concentration of glucose	Reference
Glucose	No change or -4mM or –1.2 mM	Belo et al., 1976; Harvey et al., 1976; Hazelwood & Lorenz, 1959
Beta Hydroxy butyrate	+1.2 µM	Belo et al., 1976
Alanine	+110 µM	Belo et al., 1976
Serine	+250 µM	Belo et al., 1976
Glycine	-150 µM	Belo et al., 1976
Heart Glycogen	- 8.3 µmoles per g	Hazelwood & Lorenz, 1959
Liver Glycogen	- 85 µmoles per g	Hazelwood & Lorenz, 1959

Table 3. Changes in circulating concentrations of glucose, other metabolites and tissue glycogen in chickens when fasted

control of metabolic hormones with pancreatic glucagon, arguably, the most important. The role of glucagon and glucagons-like peptides of intestinal origin has received less attention.

When circulating concentrations of glucose are elevated, there is a rapid increase in the rate of secretion of insulin from the pancreatic islet beta (β) cells and a concomitant rise in the circulating concentrations of insulin. The effects of insulin include the following:

- Increase in glucose uptake by muscle, liver and adipose tissue with glucose accumulating as glycogen together with increases in triglyceride in adipose tissue
- Increase in lipogenesis

When circulating concentrations of glucose are depressed, there is rapid increases secretion of glucagon from the pancreatic islet alpha (α) or A cells and a concomitant rise in the circulating concentrations of glucagon. The effects of glucagon include the following:

- Increased lipolysis in adipose tissue
- Decreased muscle and liver glycogen
- Decreased glucose utilization
- Increased gluconeogenesis
- Decrease in lipogenesis.

Other important hormones controlling metabolism in poultry include the avian adrenal glucocorticoid, corticosterone, and the thyroid hormone, triiodothyronine (T_3).

3.1 Insulin

The structure of chicken pro-insulin is long established (Perler et al., 1980). The amino-acid sequence for ostrich and chicken insulin are identical (Evans et al., 1988). The structure of insulin is identical in Pekin ducks, Muscovy ducks and domestic geese (Chevalier et al., 1996). Duck insulin has a lower potency and binding affinity to the mammalian insulin receptor compared to chicken insulin (Constans et al., 1991).

Insulin is released at times of surplus glucose, for instance following post prandial absorption of nutrients from the small intestine (DeBeer et al., 2009). The factors controlling insulin secretion are summarized in table 4. Insulin secretion from chicken pancreas *in situ* is increased by elevated glucose concentrations; this being potentiated by glucagon (King & Hazelwood, 1976). Similarly, elevated glucose concentrations increase insulin from chicken B islets *in vitro* (Datar et al., 2006). The stimulatory effect of glucagon is unlikely to be physiological as circulating concentrations of glucagon are very low when high insulin secretion is evident as, for instance, is seen following feeding (DeBeer et al., 2008). Circulating concentrations of insulin increased by the glucocorticoid, dexamethasone (Song et al., 2011). Similarly in ducks, insulin secretion is increased by glucose or arginine or oleic acid or glucagon (Foltzer & Miahle, 1980).

Stimulus	Insulin	Glucagon	Reference
Glucose	↑↑↑	↓↓	Chicken: Lanslow et al., 1970; King & Hazelwood, 1976; Honey et al., 1980 Duck: Foltzer & Miahle, 1980
Arginine	↑	↑	Chicken: King & Hazelwood, 1976; Honey et al., 1980 Duck: Foltzer & Miahle, 1980
Oleic Acid	↑	?	Chicken: Lanslow et al., 1970; King & Hazelwood, 1976; Duck: Foltzer & Miahle, 1980
Insulin	Not applicable	--	Chicken: Honey & Weir, 1979 Duck: Sitbon & Mialhe, 1978.
Glucagon	↑ in the presence of high glucose	Not applicable	Chicken: King & Hazelwood, 1976; Duck: Foltzer & Miahle, 1980
Somatostatin	↓	↑	Duck: Strosser et al., 1980
Corticosterone	↑↑	?	Chicken: Song et al., 2011
Epinephrine	↓	?	Chicken: Lanslow et al., 1970;

Table 4. Control of insulin and glucagon secretion in chickens and ducks

Despite the very high ambient circulating concentrations of glucose in poultry, insulin still plays an important role in the control of carbohydrate and to some extent also lipid metabolism. Insulin acts to increase energy storage as glycogen (liver and muscles) and triglyceride in adipose tissue (see figure 1). The physiological role of insulin in poultry is supported by the elevated circulating concentrations of glucose observed after fed chickens receive antisera against insulin (Simon et al., 2000).

3.1.1 Carbohydrate metabolism

Insulin evokes a movement of glucose from the blood. For instance, when administered to chickens insulin will induce a decline in circulating concentrations of glucose, albeit to levels that would be considered hyperglycemic in mammals (Hazelwood & Lorenz, 1959).

Chickens respond to insulin with a depression in circulating concentrations of glucose (Hazelwood & Lorenz, 1959) but are relatively insensitive to insulin (Vasilatos-Younken, 1986; Edwards et al., 1999). Insulin increases glucose uptake by skeletal muscle (M. fibularis longus) *in vitro* as indicated 2 deoxy--D-[1,2-3H]-glucose uptake (Zhao et al., 2009). In ducks, insulin increase in glucose uptake by skeletal muscle sarcolemmal vesicles while also inducing translocation of GLUT-4 like proteins from intracellular pools to the sarcolemma (Thomas-Delloye et al., 1999).

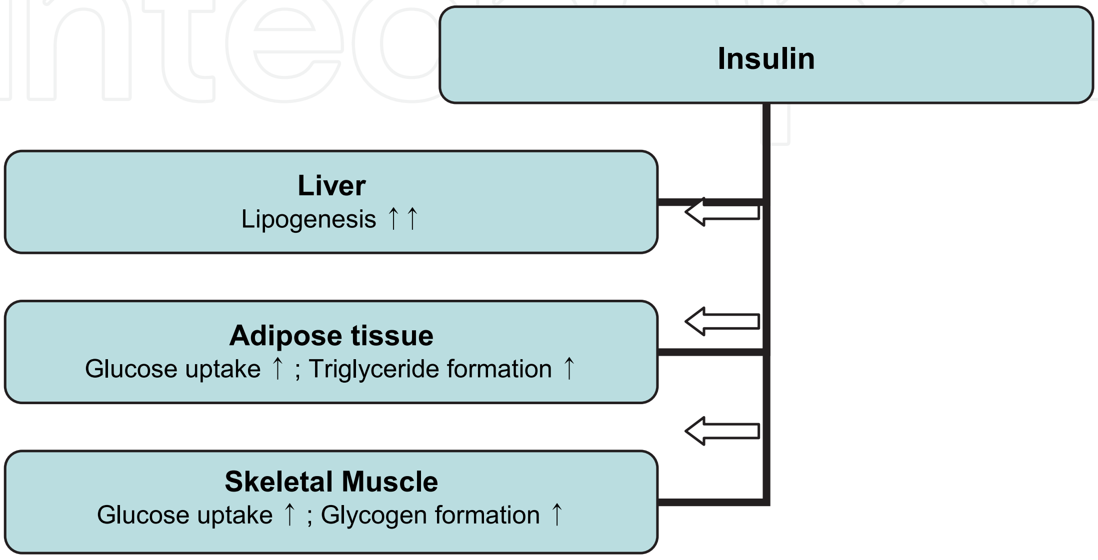

Fig. 1. Schema of physiological role of insulin in poultry

3.1.2 Lipid metabolism

Insulin can influence lipid metabolism but the physiological significant of these effects is uncertain. Fatty acid synthesis occurs predominantly in the liver of poultry and is under the control of metabolic hormones. Lipogenesis is increased *in vitro* in the presence of insulin together with T_3 (Goodridge, 1973; Wilson et al., 1986). Insulin also stimulates free fatty acid uptake by duck hepatocytes (Gross and Mialhe, 1984). However, insulin does not suppress the lipolytic effects of glucagon on chicken adipose tissue (Langslow & Hales, 1969).

3.2 Glucagon

In poultry, the major role of glucagon is to modulate carbohydrate and lipid metabolism to provide readily utilizable energy, at times of nutritional restriction. Ostrich glucagon is identical to duck glucagon (Ferreira et al., 1991).

Glucagon effects the mobilization of glucose and fatty acids from storage into the circulation together with decreasing glucose utilization (see figure 2). As might be expected, in the absence of feeding, as in regimens where chickens are fed on alternative days, there are large increases in the circulating concentrations of glucagons between meals (DeBeer et al., 2009). Surprisingly in view of its release during fasting, glucagon decreases food intake when administered centrally to chicks (Honda et al., 2007). The factors controlling glucagon secretion are summarized in table 4. Broadly, glucagon secretion is inhibited by glucose but stimulated by amino-acids. For instance, in ducks, glucagon secretion is increased by the amino-acid, arginine (Foltzer & Miahle, 1980).

3.2.1 Carbohydrate metabolism

Glucagon administration increases the circulating concentration of glucose (see table 2)(chicken: Harvey et al. 1978; turkey: McMurtry et al., 1996; ducks: Foltzer & Miahle, 1980). This is due to increases in hepatic glycogenolysis and gluconeogenesis together with decreased glucose utilization, for instance for fatty acid synthesis (Wilson et al., 1986). Glucagon has been found to stimulate gluconeogenesis, for instance, in the perfused chicken liver (Sugano et al., 1982).

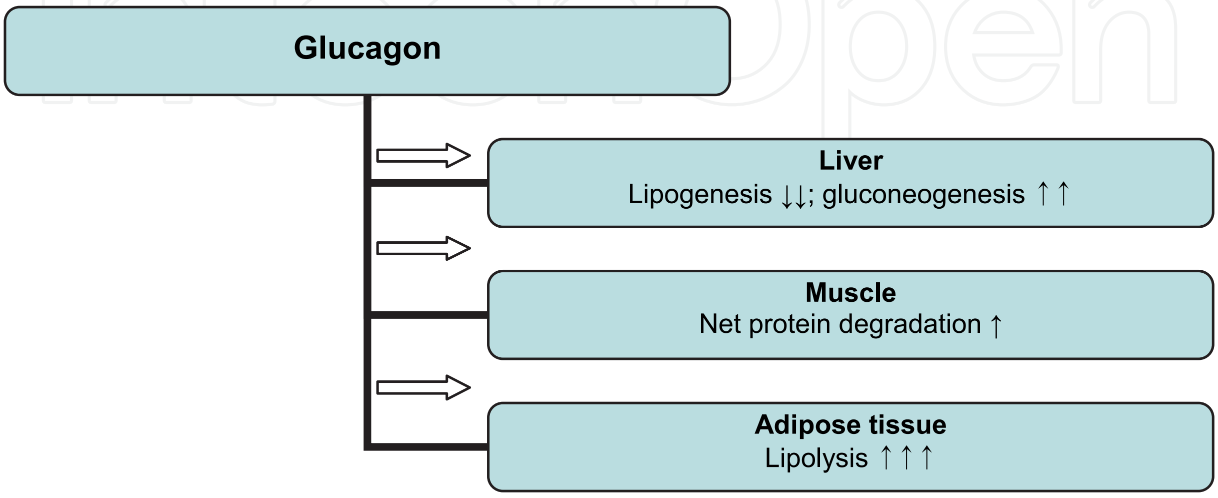

Fig. 2. Schema of physiological role of glucagon in poultry

3.2.2 Lipid metabolism

Glucagon suppresses lipogenesis in chicken hepatocytes in vitro (Wilson et al., 1986). Glucagon is the major lipolytic hormone in birds (Langslow & Hales, 1969) with its effect decreased in the presence of GH (e.g. Campbell & Scanes, 1987).

3.3 Protein metabolism

Glucagon increases gluconeogenesis and presumably net protein degradation in stores such as muscle. In ducks, glucagon increases both circulating concentrations of amino-acids (Foltzer & Miahle, 1980).

3.4 Thyroid hormones

As in mammals, the thyroid glands produce thyroxine (T_4). This is converted to the active Form, T_3. There is further deiodination and inactivation of T_3 by the T_3-degrading type III deiodinase. This enzyme is decreased in the presence of growth hormone (GH) (Darras et al., 1993). Metabolic effects of T_3 chickens include the following:

- T_3 increases heat production. Exposure of chickens to cool environmental temperatures has been reported to increase circulating concentrations of T_3, heat production and expression of uncoupling proteins (UCPs) (Collin et al., 2003). Thyroid hormones increase thermogenesis by a mechanism involving UCPs. Expression of UCPs in chickens is related to circulating concentrations of thyroid hormones, particularly T_3 (Collin et al., 2005).
- The amount of adipose tissue in chickens is related to thyroid status with increased adipose tissue in hypothyroid chickens treated with methimazole and reduced adipose

- tissue in hyperthyroid chickens, receiving T_3 administration chronically (Decuypere et al., 1987)
- T_3, together with insulin, elevates the rate of lipogenesis in chicken hepatocytes *in vitro* (Wilson et al., 1986)
- Thyroid hormones influence metabolism in the small intestine with T_4 *in vitro* for 72 hours increasing glucose active transport in the duodenum of chick embryos (Black, 1988)
- T_3 decreasing the expression of genes related to obesity including brain-derived neurotrophic factor (BDNF), leptin receptor (LEPR), pro-opiomelanocortin (POMC), thyrotropin releasing hormone (TRH), and agouti-related protein (AGRP) in chicken hypothalamic neurons in vitro (Byerly et al., 2009).

3.5 Corticosterone

Corticosterone is the major glucocorticoid in birds. It has marked effects on carbohydrate, lipid and protein metabolism. Production of corticosterone is stimulated by ACTH and other hormones including calcitonin (e.g. Nakagawa-Mizuyachi et al., 2009). The glucocorticoid receptor (GR) is expressed in multiple tissues in the chicken including the liver and anterior pituitary gland (Porter at al., 2007). The activation of receptor and its translocation into the nucleus is stimulated by corticosterone and this is blocked by the specific glucocorticoid antagonist, ZK98299, when the chicken GR is expressed in Cos-7 cells (Proszkowiec-Weglarz & Porter, 2010). Hepatic expression of the glucocorticoid receptor in chickens is reported to be inversely correlated with circulating concentrations of corticosterone (Marelli et al., 2010) suggesting the corticosterone down regulates expression of its own receptor..

3.5.1 Carbohydrate metabolism

Effects of corticosterone on carbohydrate metabolism include the following:

- Increasing circulating concentrations of glucose (Lin et al., 2007; Jiang et al., 2008; Yuan et al., 2008; Zhao et al., 2009) although depressed circulating concentrations of glucose have also been reported (Gao et al., 2008)
- Increasing circulating concentrations of insulin (Jiang et al., 2008; Yuan et al., 2008; Zhao et al., 2009)
- Increasing glucose uptake as indicated 2 deoxy--D-[1,2-3H]-glucose uptake by fibularis longus muscle *in vitro* (Zhao et al., 2009)
- Decreasing glucose uptake response to either insulin or nitric oxide as indicated 2 deoxy--D-[1,2-3H]-glucose uptake by fibularis longus muscle in vitro (Zhao et al., 2009)
- Decreasing circulating concentrations of nitric oxide (Zhao et al., 2009)
- Increased breast muscle glycogen (Lin et al., 2007) but lower levels have also been reported (Gao et al., 2008).

Despite the marked effects on glucose metabolism, circulating concentrations of lactate are unchanged in chickens receiving glucocorticoid administration (Lin et al., 2007).

There is a growing viewpoint that corticosterone acts via induction of insulin resistance. Evidence for corticosterone inducing insulin resistance comes from the consistent increasing in circulating concentrations of both glucose and insulin (see above e.g. Yuan et al., 2008) and the decreased glucose uptake evoked by insulin as indicated 2 deoxy--D-[1,2-3H]-glucose uptake by fibularis longus muscle in vitro (Zhao et al., 2009)

3.5.2 Protein metabolism

The effects on corticosterone on protein metabolism include the following effects reported in chickens:

- Depressing body weight, and particularly breast muscle weight, following chronic administration of corticosterone (Lin et al., 2006)
- Increasing net breakdown of muscle protein due to both decreased synthesis and increased degradation (discussed in more detail under corticosterone and growth)
- Decreasing circulating concentrations of total amino-acids (Gao et al., 2008) and increased concentrations of urate (Lin et al., 2007). These are indicative of both increases in deamidation of amino-acids and consequently of gluconeogenesis (Lin et al., 2007)
- Increasing gluconeogenesis by perfused liver (Kobayashi et al., 1989)
- Reductions in super-oxide dismutase activity (Lin et al., 2009).

3.5.3 Lipid metabolism

Corticosterone has marked effects on lipid metabolism including:

- Increased liver weight (Jiang et al., 2008)
- Increased hepatic lipogenesis (Lin et al., 2006; Yuan et al., 2008)
- Increased abdominal and subcutaneous adipose weight (Bartov, 1982; Buyse et al., 1987; Jiang et al., 2008; Yuan et al., 2008)
- Increased circulating concentrations of non-esterified fatty acids (NEFA) (Jiang et al., 2008; Yuan et al., 2008)
- Increased circulating concentrations of triglyceride and very low density lipoprotein (VLDL) (Jiang et al., 2008)
- Increased adipose lipo-protein lipase (LPL) (Jiang et al., 2008; Yuan et al., 2008).

3.5.4 Immune effects of corticosterone

Administration of corticosterone to chicken results in reductions in the weights (and weights as a percentage of body weight) of the bursa Fabricius and spleen (Shini et al., 2008) Other effects include an initial transitory improvement of the antibody response to infectious bronchitis virus (IBV) vaccination followed by a marked impairment of the response to IBV (Shini et al., 2008)/ Other effects including increasing the heterophil to lymphocyte (H/L) ratio in the circulation (Shini et al., 2009). Corticosterone increases expression interleukins -1beta, IL-6, IL-10, IL-12alpha and IL-18 while decreasing that of chemokine C-C motif ligand (CCL)16 and transforming growth factor-beta4 in heterophils in the circulation of chickens (Shini et al., 2010).

3.5.5 Other metabolic effects of corticosterone

Other metabolic effects of corticosterone in chickens include the following:

- Increasing expression sodium and glucose co-transporter 1 (SGLT-1 vitamin D-dependent calcium-binding protein-28,000 molecular weight (CaBP-D28k), and peptide transporter 1 (PepT-1) mRNA in the duodenum (Hu et al., 2010)
- Increasing expression of genes related to obesity in the chicken hypothalamus including brain-derived neurotrophic factor (BDNF), neuropeptide Y and agouti-related protein (AGRP) (Byerly et al., 2009)
- Depressing adenosine deaminase activity in all regions of the chicken gastro-intestinal tract except the proventriculus (Bhattacharjee et al., 2009).

3.6 Other hormones and metabolism

3.6.1 Estrogen and metabolism

Estrogen has some effects on metabolism. Estrogen increases adiposity in poultry. For instance, synthetic estrogens increase adipose tissue in chickens (Carew & Hill, 1967; Snapir et al., 1983). Moreover, the anti-estrogen, tamoxifen, decreases adiposity in female chickens (Rozenboim et al., 1989; 1990). In addition, estrogens are responsible for the dramatic increase in the hepatic synthesis of the yolk lipo-proteins (Reviewed: Scanes et al., 2004).

3.6.2 Ghrelin and metabolism

Lipogenesis in the chicken liver is increased by ghrelin as indicated by expression of fatty acid synthase (Buyse et al., 2009). Moreover, ghrelin reduces the respiratory quotient in young chickens (Geelissen et al., 2006).

3.6.3 Growth Hormone (GH) and metabolism

Both native and biosynthetic growth hormone (GH) *per se* can stimulate lipolysis *in vitro* (Campbell & Scanes, 1985). Moreover, GH inhibits glucagon stimulated lipolysis (Campbell & Scanes, 1986).

3.6.4 Somatostatin and metabolism

The major gastro-intestinal hormone, somatostatin is reported to be a potent inhibitor of glucagon stimulated lipolysis with chicken adipose tissue (Di Scala et al., 1985).

4. Hormonal control of growth

In poultry, the two major hormones required for the full expression of growth are GH and T_3. Both require the anterior pituitary gland. GH is directly synthesized by somatotrophs in the caudal lobe of the anterior pituitary gland in poultry. T_3 is produced by monodeiodination of the thyroid hormones, thyroxine (T_4). In turn, secretion of T_4 is stimulated by the anterior pituitary hormone, thyrotropin (thyroid stimulating hormone TSH). Moreover, the circulating concentrations of T_3 are maintained by GH reducing deactivation by T3-degrading type III deiodinase (Darras et al., 1993).

Evidence for the importance of anterior pituitary hormones in the growth of poultry (see figure 3) comes from ablation and replacement therapy studies. In young chickens, hypophysectomy depressed growth (body weight or skeletal growth) with growth rate being partially restored with either GH or T_3 replacement therapy (King & Scanes, 1986; Scanes et al., 1986). Similarly in young turkeys, hypophysectomy reduced growth rate but no effects of GH are observed (Proudman et al., 1994).

4.1 Growth Hormone (GH) and Growth

Dwarf chickens exhibit markedly reduced growth (Scanes et al., 1983) due to lack of GH receptors (Burnside et al., 1991; Agarwal et al., 1994) and the reduced circulating concentrations of T_3 (Scanes et al., 1983). While GH may be essential for growth, additional exogenous GH have either no (chickens: Cogburn et al. 1989, Cravener et al., 1989; Rosebrough et al., 1991; turkeys: Bacon et al., 1995) or only a small positive effect on poultry growth (Leung et al., 1986; Vasilatos-Younken et al., 1988; Scanes et al., 1990) with the latter potentially transitory. Instead, it may be hypothesized that the set points for GH/IGF-I

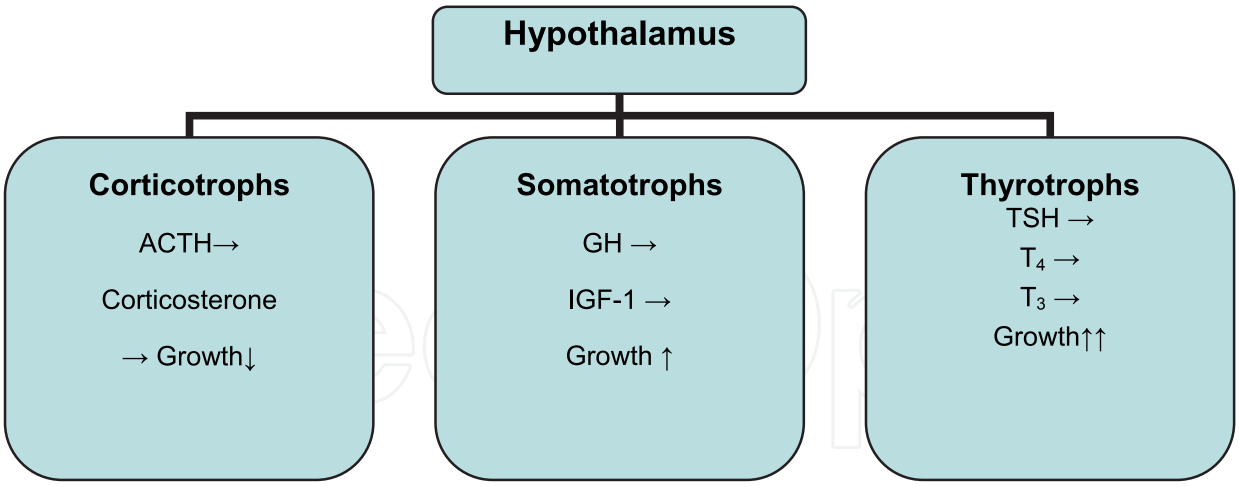

Fig. 3. Schema of physiological role of growth hormone in poultry

mediated growth are tightly controlled to insure optimal growth. It is argued that excess weight/size would be selected heavily against birds because of the energy requirements for flight.

GH acts specifically on the growth of immune tissues in birds. In birds, there is special separation between T and B cells during development in respectively the thymus and bursa Fabricius. In young chickens, hypophysectomy depresses thymus growth with GH partially overcoming this effect (King and Scanes, 1986; Johnson et al., 1993).

4.1.1 Control of GH synthesis and release

Release of GH from somatotrophs in the chicken pituitary is controlled by the following:

- The number of somatotrophs;
- The amount of GH available to be released, which is in turn dependant on GH gene expression and translation (GH synthesis);
- Stimulatory control by hypothalamic peptides such as GH releasing hormone (GHRH), thyrotropin releasing hormone (TRH), ghrelin and pituitary adenylate cyclase-activating peptide (PACAP) together with possibly leptin;
- Inhibitory control by the hypothalamic and peripheral somatostatin, together with negative feedback by hormones whose release/synthesis are increased by GH, namely insulin-like growth factor 1 (IGF-1) and triiodothyronine (T_3);
- The stimulatory and inhibitory effects depend upon both the concentrations of the stimulator or inhibitor and the responsiveness of somatotrophs to them. Not all chicken somatotrophs respond to all secretagogues; some respond to both GHRH and PACAP (85%), or to GHRH and TRH (73%) or to GHRH and leptin (51%) or to GHRH and ghrelin (21%) (Scanes et al., 2007).

Expression of the GH gene is inhibited by T_3 or IGF-1 in chickens (Radecki et al., 1994; Scanes et al., 1999).

4.2 Thyroid hormones and growth

In poultry, normal growth rate requires critical or optimal concentrations of T_3 and perhaps T_4 also. Administration of T_3 to dwarf chicks to restore normal circulating concentrations of T_3 produces some increase in growth rate (Marsh et al., 1984; Bowen et al., 1987). However, T_3 administration to chickens with circulating concentrations within the normal range

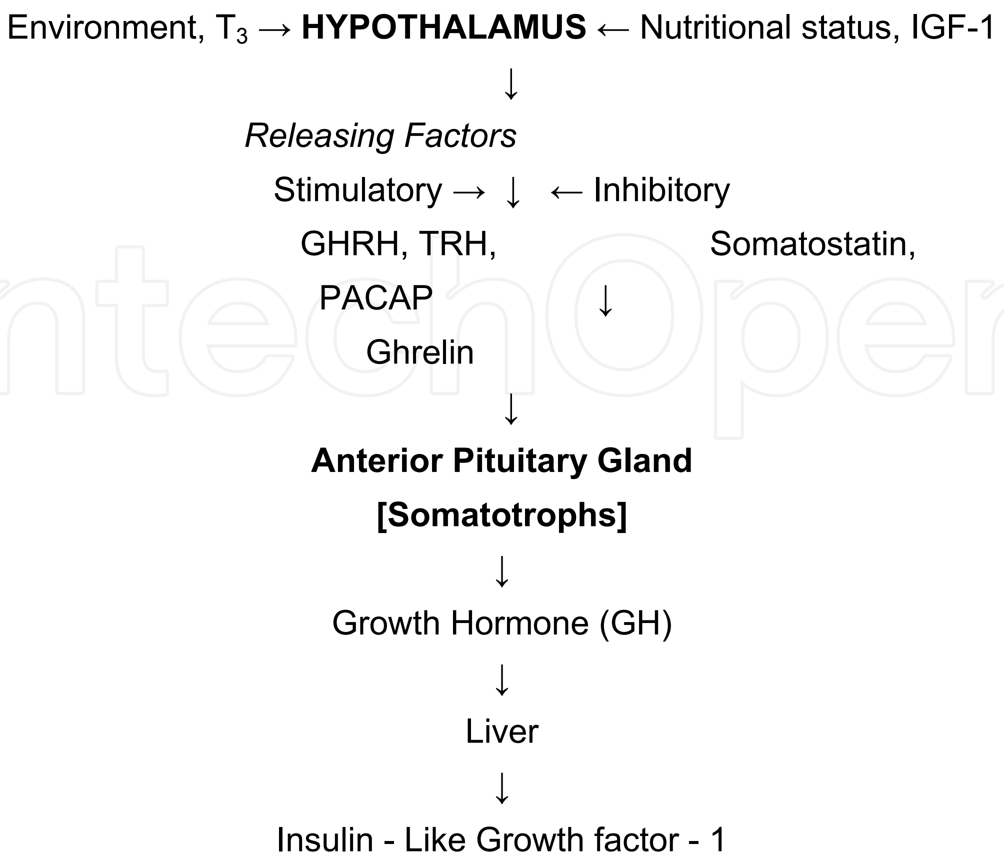

Fig. 4. The hypothalamic growth hormone – insulin-like growth factor 1 – growth axis in poultry

depresses growth rate (Marsh et al., 1984; Bowen et al., 1987). Thyroid ablation by the goitrogen methimazole results in markedly lower growth rates (Chaisson et al., 1979; Decuypere et al., 1987) and circulating concentrations of IGF-1(Decuypere et al., 1987; Rosebrough et al., 2003). Growth rates of chickens are depressed by T3 administration and to a less extent T4 (Decuypere et al., 1987). This would support the concept that normal growth rate in poultry depends on a physiological "set- point".

Other growth related effects of thyroid hormones include the following:

- T_3 increases the growth rate of young hypophysectomised chickens (Scanes et al., 1986);
- Thyroid hormones induce development of the small intestine with thyroxine *in vitro* for 72 hours increasing microvillar growth and the rate of mitosis in the epithelia in chick embryo duodena (Black, 1978) and glucose active transport in the duodenum of chick embryos (Black, 1988);
- T3 decreases GH secretion by effects at both the levels of the anterior pituitary and the hypothalamus. For instance, T3 increases the expression of both type 2 and 5 somatostatin receptor sub-types (De Groef et al., 2007) and reduces the expression of thyrotropin releasing hormone (TRH) both in vivo and in vitro in chicken hypothalamic neurons (Byerly et al., 2009).

4.3 Insulin like growth factor-1 and growth

There is strong evidence that the effects of GH and thyroid hormones are mediated by hepatic production of insulin-like growth factor-1 (IGF-1). Circulating concentrations of IGF-

1are markedly decreased in hypophysectomized young chickens with GH partially reversing this effect (Huybrechts et al., 1985; Lazarus and Scanes, 1988). GH also elevates plasma IGF-1 in intact adult chickens (Scanes et al., 1999). Moreover, IGF-I release from chicken hepatocytes *in vitro* is elevated in the presence of GH and synergistically with GH and insulin (Houston & O'Neill, 1991). Circulating concentrations of IGF-1 are reduced by chronic methimazole administration with concentrations partially restored by T3 administration (Rosebrough and McMurtry, 2003). Chicks treated with the goitrogens, propylthiouracil, have depressed growth rate, circulating concentrations of IGF-I and hepatic expression of IGF-I (Tsukada et al., 1998) and T_4 administration partially restoring these parameters (Tsukada et al., 1998).

There is a report that the administration of IGF-I stimulate growth rate in chickens (Tomas et al., 1998). Moreover, there are increases in skeletal muscle mass and elevated rates of protein synthesis (Conlon & Kita, 2002) and depressed rates of degradation (Tomas et al., 1998). The effect of IGF-1 on chick growth has not been observed in other studies (McGuinness & Cogburn, 1991; Huybrechts et al., 1992; Tixier-Boichard et al., 1992). One mechanism by which, glucocorticoid hormones depress growth is by depressing IGF-1; circulating concentrations of IGF-1 have recently been observed to be decreased by the glucocorticoid, dexamethasone, in chickens (Song et al., 2011).

4.4 Other hormones and growth

Other hormones such as the adrenal cortical hormone, corticosterone, estradiol and testosterone can have marked effects on growth.

4.4.1 Corticosterone and growth

Glucocorticoids including the endogenous avian steroid, corticosterone, and the synthetic dexamethasone depress growth in chickens (Li et al., 2009; Hu et al., 2010; Song et al., 2011).

- Decreased skeletal muscle weight (Yuan et al., 2008; Song et al., 2011)
- Increased protein degradation as indicated by increases in the concentrations of 3-methyl histidine in both pectoralis and femoris muscles (Dong et al., 2007)
- Increases muscle proteolysis (Gao et al., 2008)
- Reduced skeletal protein synthesis as indicated by the RNA:protein ratio (Dong et al., 2007) Decreasing the growth of the small intestine in chickens although the effect is of a small magnitude than that with overall growth as weight of the small intestine relative to body weight is increased (Hu et al., 2010)
- Increases expression of myostatin (Song et al., 2011).
- Depressing duodenal and jejunal villus height and crypt depth (Hu et al., 2010)

Corticosterone plays the pivotal role in inducing functioning somatotropes during late embryonic development (Dean and Porter, 1999; Porter et al., 2001).

4.4.2 Estrogen and growth

Estrogen has effects on the growth of specific organs being responsible for the massive growth of the oviduct during sexual maturation (reviewed: Scanes et al., 2004). Estrogens play an important role in the formation of the calcium storing tissue, medullary bone, at the time of sexual maturation. Formation of medullary bone matrix is stimulated by estradiol and testosterone in immature male quail chicks with mineralization requiring vitamin D3 (Takahashi et al., 1983). Estradiol, in combination with testosterone, is has found to stimulate proliferation of chicken medullary osteoblasts and inhibit their apoptosis (Chen et al., 2010). Moreover, medullary bone formation is suppressed when aromatase, critical for estradiol synthesis, is inhibited (Deng et al., 2010).

4.4.3 Glucagon-like peptide 2 (GLP-2) and growth

Glucagon-like peptide 2 increases growth in chickens (Hu et al., 2010).

4.4.4 Testosterone and growth

The major circulating androgen in birds is testosterone. Testosterone acting via conversion to 5α Dehydro-testosterone (DHT) depresses growth in chickens (Fennell & Scanes, 1992a) while stimulating that of turkeys (Fennell & Scanes, 1992b).Androgens in combination with estrogens induce the formation of medullary bone at the time of sexual maturation (e.g. Chen et al., 2010).

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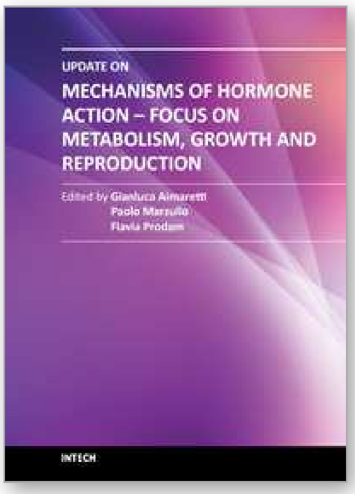
UPDATE ON

MECHANISMS OF HORMONE ACTION – FOCUS ON METABOLISM, GROWTH AND REPRODUCTION

Edited by Gianluca Aimaretti
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Flavia Prodam

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The purpose of the present volume is to focus on more recent aspects of the complex regulation of hormonal action, in particular in 3 different hot fields: metabolism, growth and reproduction. Modern approaches to the physiology and pathology of endocrine glands are based on cellular and molecular investigation of genes, peptide, hormones, protein cascade at different levels. In all of the chapters in the book all, or at least some, of these aspects are described in order to increase the endocrine knowledge.

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