

**THE ROLE OF SOMATOTROPIC HORMONE IN THE ENDOCRINE
AND LOCAL CONTROL OF REPRODUCTIVE FUNCTION
IN THE DOMESTIC HEN (*Gallus domesticus* L.)**
(review)

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Abstract

Pituitary somatotrophic hormone (STH) has long been considered solely as a metabolic hormone that promotes the body growth and development until the puberty completion. However, studies in recent decades have shown that the area of STH action on the body is much wider (K.L. Hull et al., 2001; M. Lu et al., 2019). It has been established that, in addition to the pituitary gland, STH is produced in many cells and tissues, including nervous (C. Arámburo et al., 2014), immune (G. Mo et al., 2022) and reproductive (S. Harvey, 2010; M. Luna et al., 2014), where it plays an autocrine/paracrine role. The works of various researchers have shown that STH is involved in the regulation of a wide range of processes associated with the reproductive function in mammals and humans, such as sexual differentiation, puberty, steroidogenesis, gametogenesis and ovulation, as well as pregnancy and lactation (J.R. Silva et al., 2009; J. Devesa et al., 2019; C.W. Chang et al., 2022; X.Y. Zhou et al., 2023). On the contrary, much less is known about the participation of STH in the regulation of the reproductive system function in female birds. The present review article examines general information about the structural and functional properties of STH and its receptor (G.P. Baumann, 2009; M.J. Waters, 2016). The paper presents modern views on the mechanisms of activation of the hormone signaling pathways, key molecular mediators involved in its intracellular signal transmission, as well as the main mechanisms regulating STH synthesis/secretion (F. Dehkhoda et al., 2018; S.J. Frank, 2020; Y. Chhabra et al., 2021). Information is provided on the extrapituitary expression of STH and its receptor in the ovary and oviduct of the domestic hen (*Gallus domesticus* L.) which represents a convenient model for studying processes associated with reproduction in birds (M. Luna et al., 2014; A. Hrabia, 2015). The relationships of the STH concentration in the blood and the content of somatotrophic receptors in reproductive tissues with the functional activity and/or the developmental stage of the latter, as well as the age and reproductive status of the birds are considered (A. Hrabia et al., 2008; A. Hrabia et al., 2013; Smekalova et al., 2019). The review systematizes accumulated data on the STH role in the endocrine and local control of the hen reproductive function, namely, on the effect on reproductive tissues when administered to birds in vivo (A. Hrabia et al., 2011; H. Mohammadi et al., 2016; A. Hrabia, 2022) and on the steroidogenic activity, proliferation and apoptosis of follicular cells in vitro (A. Hrabia et al., 2012, 2014; A. Smekalova et al., 2020; A. Smekalova et al., 2021). The STH influence on the follicular cells of the domestic hen ovary has been shown to depend on the degree of the follicle maturity, the interaction of the follicle wall layers, the stage of the ovulatory cycle and the avian age (A. Smekalova et al., 2020; O.V. Aleynikova et al., 2021). Insulin-like growth factors are also considered as possible mediators of the STH effect on the hen reproductive system (O. Onagbesan et al., 2009; S.M. Ahumada-Solyrzano et al., 2016). It is concluded that STH plays a significant role in the regulation of follicles maturation, their preparation for ovulation and the formation of egg components, and it can also act as a survival factor for oviduct cells.

Keywords: somatotrophic hormone, receptors, intracellular signaling cascades, domestic hens, reproductive function, ovary, oviduct

Somatotropic hormone (STH), or growth hormone, is a polypeptide produced primarily in the somatotrophic cells of the anterior pituitary gland (adenohypophysis). For a long time, STH was considered only a metabolic hormone stimulating growth and development of the organism before the completion of its sexual maturation. However, in the last three decades, numerous evidences have been obtained indicating that the effect of STH on the body is pleiotropic [1-4]. In addition, STH is synthesized not only in the cells of the adenohypophysis, but in many other cells and tissues, including nervous [5], immune [6, 7] and reproductive [8-10]. Local production of the hormone indicates its autocrine/paracrine role in regulating the function of the corresponding cells, tissues and organs.

Various research groups [11-13] have convincingly demonstrated the effect of STH on the reproductive system of female mammals. Considerable attention is paid to the involvement of this hormone in the regulation of female fertility [14-16] and reproductive aging [17-19]. However, there is only fragmentary information on the influence of STH on the reproductive system in other classes of vertebrates.

The purpose of this review was to briefly analyze the known information on somatotrophic hormone and the mechanisms of its action. We generalized and systematized the currently available data on the involvement of STH in the regulation of reproductive function in the domestic chicken (*Gallus domesticus* L.) as the most convenient model for studying reproduction in birds.

Somatotropic hormone: structure, functions, regulation of secretion, receptors and intracellular signaling pathways. The main information on the structural and functional properties of STH, its receptors and the mechanisms of intracellular signaling was obtained in mammals.

Two hypothalamic hormones that mainly regulate the pulsatile release of STH in the pituitary gland are STH-releasing hormone (STH-RH; stimulation) and somatostatin (inhibition). Several short and long feedback loops decrease STH secretion. Increased blood levels of this hormone and insulin-like growth factor-1 (IGF-1), produced by the liver in response to it, cause the release of somatostatin and inhibit the release of STH-RH and STH [20].

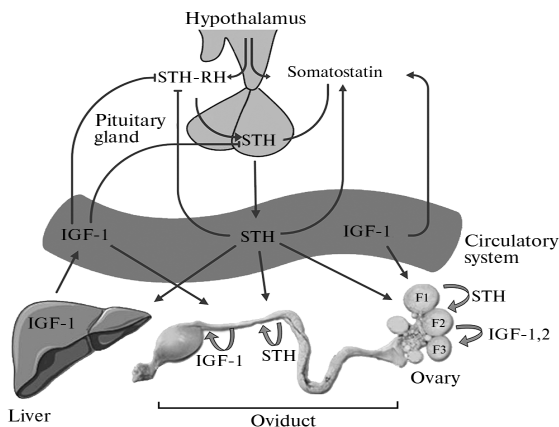


Fig. 1. Regulation of central and local production of somatotrophic hormone (STH) in chickens: IGF-1 is insulin-like growth factor-1, IGF-1,2 are insulin-like growth factors-1 and -2, STH-RH is STH-releasing hormone, F1, F2, F3 are preovulatory follicles in the order of their ovulation.

Ultrashort feedback is also distinguished, through which somatostatin and STH modulate their own release (autocrine regulation) [20, 21]. The regulation scheme of STH synthesis/secretion in the domestic chicken is similar to that in mammals [22], and the reproductive organs serve as an extrapituitary source of STH, where its autocrine/paracrine action is realized, partially mediat-

ed by IGF-1 or IGF-2 (Fig. 1). In addition to STH-RH, there are other neuropeptides and hormones that can stimulate the release of STH in chickens, such as thyrotropin-releasing hormone (TRH), glucagon-like peptide 1 (GLP1), ghrelin, neuropeptide W, pituitary adenylate cyclase-activating polypeptide (PACAP), leptin, and gonadotropin-releasing hormone (GnRH) [21-23].

The STH gene is located on chromosome 27 of the domestic chicken and consists of five exons and four introns similar to the STH gene of mammals [24]. Polymorphisms found in different regions of this gene exert associations with egg and meat productivity [25-28].

In chickens, like in mammals, there are many structural forms of STH, differing in molecular weight, the ratio of which changes depending on the growth and development of the organism. The appearance of the hormone isoforms is due to different splicing patterns and post-translational modifications, such as glycosylation, phosphorylation, dimerization, and oligomerization [29-31]. However, only the 22 kDa STH of chicken is able to bind to its receptors in a radioreceptor assay [32].

The biological activity of STH is realized only after its association with the receptor in the plasma membrane of the target cell, as it is confirmed by the postnatal dwarfism in case of a defect in the STH receptor gene in humans and domestic chickens [33, 34]. Somatotropic receptors are present in almost all tissues where the pleiotropic actions of STH occurs. These include the regulation of metabolism, postnatal growth, cognitive function, the functioning of the immune, cardiac, genitourinary and reproductive systems, and the intestine. The hormone exerts these actions primarily through changes in gene expression initiated by activation of its membrane receptor and subsequent intracellular signaling [35]. However, some physiological effects of STH are realized indirectly, through the induction of IGF-1 or IGF-2.

The STH receptor belongs to the class I cytokine receptor family, which includes more than 30 members. The somatotropic receptor is a 638-amino acid protein with one extracellular domain, a transmembrane domain that crosses the plasma membrane once, and a cytoplasmic intracellular domain (Fig. 2, A). The extracellular domain consists of upper and lower modules which are similar to fibronectin III and act as a ligand-binding site [35-38]. STH receptors are capable of releasing their extracellular domain into the blood, producing circulating STH-binding protein which regulates the availability of the hormone for binding to membrane receptors [39].

Activation of somatotropic receptors requires the formation of a complex consisting of one hormone molecule and two receptor molecules. Such an oligomeric complex is formed by binding of STH to the receptor homodimer which is formed either constitutively or after the inducing effect of the hormone [40, 41]. The interaction of STH with receptors occurs in two stages: first, the hormone binds via its "site 1" motif to one receptor molecule, causing conformational changes in the second receptor, then it binds to it via its "site 2" motif. The GH receptor does not have its own tyrosine kinase activity, so further signal transduction requires binding of the intracellular domain, namely its conserved Box1 motif, to the cytoplasmic tyrosine kinase Janus 2 (JAK2) [42].

Binding of GH brings the parallel transmembrane domains of the receptors into a cross-orientation, which causes separation of the lower part of the transmembrane helices (see Fig. 2, A). Since the tyrosine kinase JAK2 is bound to the Box1 motif located in close proximity to the inner side of the membrane, receptor activation leads to separation of the two associated JAK2s and, in particular, to the removal of the inhibitory pseudokinase domain [43]. Transactivation of the two kinase domains occurs and tyrosine phosphorylation is initiated

of the cytoplasmic domain of both receptors and downstream effectors such as STAT1, STAT3, and STAT5 (signal transducers and activators of transcription) which are a group of transcription factors that mediate most of the genomic effects of STH (see Fig. 2, B). Phosphorylation leads to the formation of STAT dimers and their translocation to the nucleus where they regulate target gene expression. Termination of signaling involves proteins of the SOCS (suppressor of cytokine signaling) family, the suppressors of cytokine signaling which, due to the presence of the SH2 domain, compete for STAT receptor binding sites and inactivate JAK2. In addition, this signaling cascade can be inactivated by a number of protein tyrosine phosphatases that catalyze the dephosphorylation of the receptor and its substrates at phosphotyrosine residues [37].

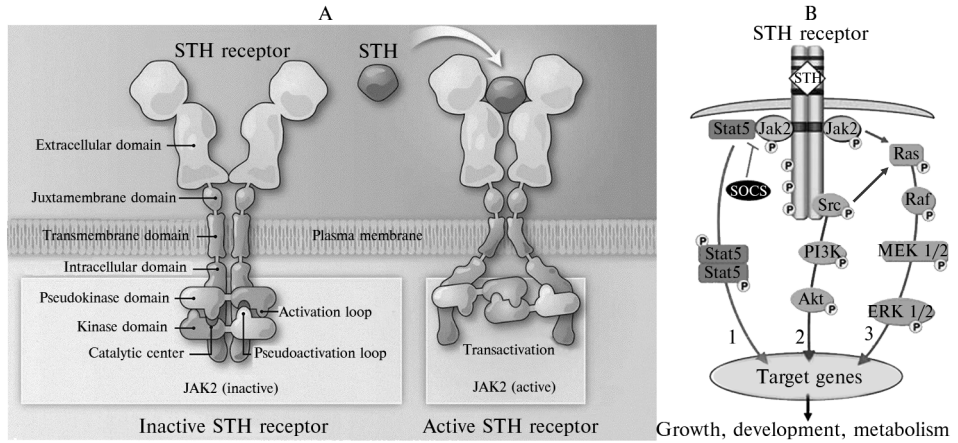


Fig. 2. Model of growth hormone (GH) receptor activation (A) and GH receptor-induced intracellular signaling (B): JAK2 is Janus 2 tyrosine kinase, STAT5 is a signal transducer and activator of transcription, SOCS is a suppressor of cytokine signaling, Ras is a membrane-bound protein that is the first to participate in the transmission of the RAS-RAF-MEK-ERK pathway signaling cascade, Src is a proto-oncogene tyrosine kinase, PI3K is phosphatidylinositol-3, Akt is a protein kinase B. Adapted from [38] and [43].

The effect of STH on target cells is also realized through other intracellular signaling pathways (see Fig. 2, B) [44]. The binding of STH to its receptors activates kinases of the Src family independently of JAK2 [45]. This leads to the activation of the RAS (rat sarcoma virus) protein and the initiation of a signaling cascade associated with MAPK (mitogen-activated protein kinase) and controlling cell proliferation, differentiation, apoptosis and other processes (RAS-RAF-MEK-ERK pathway) [42]. Other signaling pathways activated by STH have also been described, including the activation of phosphatidylinositol 3-kinases (PI3K), protein kinase B/Akt and protein kinase C [46-48]. It is still unknown whether STH activates its main signaling pathway Jak2-STAT in domestic chicken cells, but the involvement of signaling cascades associated with ERK and protein kinase Akt in mediating the action of this hormone in chickens has been demonstrated [49, 50].

Somatotropic hormone and reproductive function of chickens. It has now been established that STH plays the role of a positive modulator of the reproductive function of female mammals, affecting the growth, development and resistance to apoptosis of various organ structures of the reproductive system [1, 51-53]. Many cells of this system exert extrapituitary STH expression [9, 54, 55]. The reproductive function of chickens is also a target for the action of STH which serves not only as an endocrine, but also as a paracrine (autocrine) regulator in reproduction [56].

Expression of STH and its receptors in the reproductive organs of chickens.

STH production has been detected in the domestic chicken ovary, indicating its ability to locally influence ovarian function [56-58]. STH mRNA was detected in the stroma of the chicken ovary starting from 10 weeks of age, but at a significantly lower concentration than in the pituitary gland. Expression of this mRNA occurred in small follicles of 1-4 mm in diameter after their formation at 14 weeks of age and in all larger follicles of 4-30 mm in diameter developing after 16 weeks of age. Immunoreactivity to STH was also evident in the ovarian stroma starting from 10 weeks of age and in all follicles (1-4 mm and 4-30 mm), confirming the expression of the corresponding protein, but it was more intense in the granulosa layer than in the theca layer. As the follicles matured, this immunoreactivity increased in the granulosa layer and significantly decreased in the thecal layer [56]. Similar differences in the expression level of GH mRNA were found between granulosa and theca cells [58]. In addition, nine GH protein isoforms were detected in the follicular wall, the ratio of which changed with follicle development, being probably associated with modulation of hormone activity in the ovary [58].

The oviduct of chickens also is an extrapituitary site of GH gene expression [10, 57, 59]. Using RT-PCR, GH mRNA was detected in all functional parts of the oviduct, and its concentration increased from the albumen part to the vagina [57]. In addition, ELISA test determined immunoreactivity to GH in proteins isolated from all parts of the oviduct [10]. The GH content was constant throughout its length, gradually decreasing with the age of the hens. At the age of 48 weeks, the GH concentration in the tissues of the oviduct was only 25% of that in 13-week-old hens. However, due to more than 5-fold increase in the oviduct mass with age, between 13 and 48 weeks, the total GH production also increased. However, the GH concentration per 1 mg protein did not differ from this parameter in other extra-pituitary tissues (e.g., testes and bursa of Fabricius) of adult hens. Therefore, the level of extra-pituitary GH production is comparable to the amount of hormone produced by the smaller pituitary gland. Using polyacrylamide gel electrophoresis and Western blotting, several structural isoforms of GH were detected in the oviduct, as in the ovary [10].

Reproductive tissues serve not only as extrapituitary producers of GH but also as a target for this hormone. Expression of the GH receptor gene was detected in the ovary of broiler chickens aged 4 to 16 weeks [60]. The presence of mRNA and immunoreactivity to the GH receptor was detected in the stroma of the developing ovary and in the walls of follicles of different sizes in sexually mature chickens. The expression of the corresponding mRNA and protein was more intense in the granulosa layer than in the theca layer, as was the case with the hormone itself [56]. Radioreceptor analysis showed that with the growth of preovulatory follicles in laying hens, the number of GH-binding sites in granulosa cells increases, while in theca cells decreases [61]. The presence of the STH protein and its receptor was detected in cultured chicken granulosa cells, confirming the possibility of activation of the latter by a locally produced hormone [58].

The expression of the somatotropic receptor was also detected in the chicken oviduct. The STH receptors and their mRNA were revealed in the infundibulum of the oviduct, its protein part, isthmus, uterine part and in the vagina [10, 62]. Immunoreactivity to the STH receptor was mainly observed in the mucosa and was lowest in the oviduct infundibulum [62].

It should be noted that STH reduces the mRNA content of its receptors in the liver of chickens [63]. However, it is still unknown what effect this hormone, coming from the circulatory system or synthesized locally, will have on the expression of somatotropic receptors in the reproductive organs.

Physiological significance of STH for the functioning of the reproductive sys-

tem. As is known, the blood content of STH depends on age, and its decrease can be the cause of some functional disorders. However, the advisability of administering this hormone to elderly people or animals is still a controversial issue [64]. It has been demonstrated that short-term exposure to STH in elderly patients with reduced ovarian reserve increases the effectiveness of ovarian hyperstimulation, and its long-term exposure in aged rodents has a beneficial effect on some organs and components of the metabolic system [19, 65]. It has also been shown that weakening or blocking intracellular STH signaling is associated with an increase in the lifespan of individuals of different species [66, 67]. Many researchers consider the age-related decrease in the blood concentration of this hormone as a protective mechanism that reduces the metabolic activity of cells and increases their resistance to oxidative stress which is one of the main causes of aging, including reproductive aging [64]. In young hens, the onset of the egg-laying period coincides with an increase in the blood concentration of STH [68], and with further cessation of egg-laying, its production decreases [69]. In reproductively aged laying hens, an increased content of STH in the blood plasma was associated with a reduction in the egg-laying cycle [70]. Thus, the effect of STH on the reproductive function may be dual in nature and depend on the age, physiological state and reproductive status of the birds. It should be emphasized that at the body level, the final effects of STH will be determined by its interaction with other reproductive and metabolic hormones, and with local paracrine factors involved in the regulation of the reproductive system functioning [1, 57].

In the blood of laying hens, daily fluctuations in the concentration of STH associated with the ovulatory cycle occur. Its maximum increase was noted 2 h after ovulation, then the STH level remained elevated for another 4 h, which is probably due to the metabolic needs for yolk synthesis [71]. In addition, the production of STH by the granulosa layer increased during folliculogenesis, and the concentration of its receptors in granulosa cells was highest at the final stage of the largest yellow follicle development [56, 61]. This suggests that STH may play a significant role in the regulation of follicle maturation and their preparation for ovulation in hens.

Expression of STH receptors was higher in the albumen part, isthmus, and uterus compared to the infundibulum of the hens' oviduct [62]. As is known, these segments of the oviduct are characterized by high metabolic activity associated with the formation of egg components, i.e., 25-30 g of albumen and 5-6 g of shell. In addition, STH immunoreactivity was observed only in the mucous membrane of the oviduct, and not in the stroma consisting of muscles and connective tissue [72]. These data may indicate the participation of STH in the control of the production of egg components.

The effect of STH on reproductive tissues when administered to birds in vivo. A number of studies confirm the important role of STH in controlling reproductive function in female birds. Administration of this hormone to laying hens resulted in an increase in the number of small follicles in the ovary [57]. Injections of recombinant chicken STH to pullets resulted in a several-fold increase in ovarian weight one week before puberty. Unlike the control group, most treated hens had yellow hierarchical follicles in the ovary, indicating the participation of STH in the selection of small follicles into the preovulatory hierarchy [57, 73].

Administering STH to pullets during puberty increased the levels of progesterone and estradiol in the ovary [72]. This treatment also stimulated cell proliferation and suppressed apoptosis in the ovarian stroma and in white and small yolk follicles. Thus, STH may participate in the regulation of growth, development and steroidogenic activity of pre-hierarchical follicles of hens.

It has been established that STH participates in the formation of egg albumen and shell. Injections of the hormone for 3 weeks to laying hens at the end of the egg-laying period did not affect the egg production, but significantly improved the shell quality [74]. A later study showed that the simultaneous administration of STH and testosterone in the late phase of egg laying causes an increase in egg production and improves egg quality, namely egg weight, the height of the dense protein layer, and the shell density [75]. Treatment of hens with STH during puberty led to a significant increase in the mRNA expression of ovalbumin, the main component of egg albumen, and ovocalyxin-32 and ovocalyxin-36 which are part of the eggshell matrix [59].

The effect of STH on the oviduct of birds is apparently also associated with the regulation of apoptotic cell death, since injections of this hormone for several weeks during puberty in hens caused inhibition of cell apoptosis and a decrease in the expression and activity of apoptosis markers caspases 2 and 3 in the albumen section of the oviduct. STH did not affect the expression of genes encoding anti-apoptotic proteins, such as Bcl-2 or survivin [57, 59]. In general, these data allow us to consider STH as a survival factor in the hen oviduct.

In addition, STH injections during the pause in egg-laying caused by starvation led to an increase in the concentration of sex steroid hormones in the blood and the oviduct tissues [76]. Changes occur in the expression of steroid hormone receptors and individual egg proteins in different parts of the oviduct. Therefore, STH can play a significant role in determining the rate of regression and renewal of the oviduct during molting, and also regulate its secretory activity.

The effect of STH in vitro on the functional activity of follicular cells. In vitro studies have obtained rather contradictory data on the effect of STH on the steroidogenic activity of the structural components of the hen ovary. The hormone stimulated estradiol secretion by whole pre-hierarchical follicles, but inhibited estradiol secretion and increased progesterone production by yellow hierarchical follicles during sexual maturity of birds [77, 78]. During the peak of egg laying, the effect of exogenous and locally produced STH on granulosa cells of the second largest preovulatory follicle (F2) dose-dependently resulted in an increase in progesterone synthesis which was due to an increase in the expression of cytochrome P450_{scc}, an enzyme that cleaves the side chain of cholesterol [79]. A. Hrabia et al. [78] failed to detect any effect of STH at the start of egg laying either on progesterone secretion by granulosa layer explants or on estradiol secretion by thecal layer fragments of the three largest preovulatory follicles (F3-F1). STH decreased LH-stimulated estradiol secretion by the thecal layer of F3-F1 follicles isolated 22 h before ovulation.

A later study showed that the effect of STH on the steroidogenic activity of follicular layers depends on their paracrine interaction, the degree of maturity of the preovulatory follicle, and the age/reproductive status of the laying hens. In the absence of the thecal layer, STH increased progesterone secretion by the granulosa layer of F1 and F2 follicles in young hens at the peak of egg production and suppressed this secretion in the case of the F1 follicle in aged hens with reduced egg production [80]. In the presence of the thecal layer, STH stimulated progesterone production by the granulosa layer of the F1 follicle in both young and reproductively aged laying hens. Furthermore, when theca explants were cultured individually, GH did not significantly affect testosterone production in both F1 and F2 follicles in young high-laying hens, and decreased it 2-fold in F1 follicles in older hens at the end of the laying period [81]. In contrast, when theca and granulosa explants were cultured together, GH increased testosterone secretion by the thecal layer of F1 follicles and decreased this secretion by the thecal layer of F2 follicles in both young and old hens.

Incubation of granulosa cells from pre-hierarchical follicles with recombinant chicken GH or conditioned medium containing predominantly the 15 kDa ovarian GH isoform resulted in a dose-dependent increase in cell proliferation [79]. Moreover, both exogenous and locally produced STH induced phosphorylation of Erk1/2 kinase in granulosa cells which is associated with the signaling pathway that promotes cell survival and proliferation. In addition, a stimulating effect of STH in vitro on the proliferative activity of granulosa and theca cells from the most mature preovulatory follicle of laying hens was revealed [82]. Under these conditions, the hormone also decreased the expression of the proapoptotic protein Bax in granulosa cells and increased this expression in theca cells.

Thus, the available data suggest the participation of STH in the regulation of follicle growth and development in domestic chickens by modulating the steroidogenic and proliferative activity of follicular cells, as well as their resistance to apoptosis. Moreover, the nature of the hormonal effect depends on the age of the bird, the stage of follicle maturation, the type of follicular cells and their distant interaction.

Insulin-like growth factors as possible mediators of the action of STH on the reproductive system of chickens. Some biological effects of STH on target cells are mediated by insulin-like growth factors (IGFs), which are synthesized in many tissues in response to GH stimulation [83]. In recent decades, sufficient evidence has accumulated that the IGF system (IGF-1, IGF-2, IGF receptors, IGF-binding proteins) plays an important role in the regulation of ovarian function in mammals [13, 53, 84]. In the chicken ovary, all members of the IGF family have been identified, which are involved in the regulation of steroidogenesis, proliferation, cell differentiation, and follicle selection (85, 86). Expression of IGF-1 and IGF-2 mRNA was detected in ovarian cells of 4-week-old chickens (60) and preovulatory follicles of adult chickens, and this expression was higher in theca cells than in granulosa cells (85). In birds, both IGF-1 and IGF-2 bind to the same receptor, IGF-1R (IGF receptor type 1). IGF-1R protein and mRNA have been detected in the immature ovary of 4-week-old chickens (60) and in the granulosa and thecal cells of developing follicles in adult chickens (85, 87). The concentration of IGF-1R mRNA in granulosa cells is higher than in theca cells and increases with follicular growth.

Thus, the granulosa layer represents the main site of IGF action in hierarchical follicles, and theca cells are the main source of IGF, confirming a local paracrine/autocrine action of IGF in the avian ovary. In vitro studies showed that GH was able to induce IGF-1 production by granulosa cells of prehierarchal follicles (79) and large preovulatory follicles (85). It is believed that the stimulating effect of GH on estradiol secretion by prehierarchal follicles, as well as on progesterone production by granulosa cells of preovulatory follicles, may be mediated by IGF-1 (77, 85). At the same time, administration of GH to chickens during puberty resulted in a significant decrease in the expression of IGF-1 mRNA in the wall of vitelline and large yellow follicles, as well as IGF-2 mRNA in the ovarian stroma and preovulatory follicles (57). In contrast, this expression was increased in the liver, indicating a tissue-specific effect of GH. Such differences could be due to paracrine interactions between the thecal and granulosa layers in intact follicles, which took place in vivo experiments.

Incubation of granulosa cells from pre-hierarchical follicles with IGF-1 and GH led to an increase in their proliferative activity, and immunoneutralization of IGF-1 completely suppressed the growth-stimulating effect of GH. This suggests that GH stimulated cell proliferation through local regulation of IGF-1 gene expression [79].

In the oviduct of quail and chicken, components of the IGF system (IGF-1, IGF-1R, and IGF-binding protein-2) were also detected, and paracrine/autocrine effects of IGF-1 were observed, indicating the possibility that the latter mediates some of the effects of GH [88, 89].

Thus, the currently available information indicates the participation of somatotrophic hormone (STH) in the endocrine and paracrine/autocrine control of the reproductive organs in domestic chicken. The pattern of changes in the blood STH content and expression of somatotrophic receptors and STH production in the cells of the ovary and oviduct during their ontogenetic development indicates a significant role of this hormone in regulating the maturation of follicles, their preparation for ovulation and the production of egg components. Studies on the introduction of STH to birds in vivo and the effect of STH on ovarian and oviduct cells in vitro revealed a hormonal effect on the growth and development of follicles in domestic chickens, which is realized through the modulation of the steroidogenic and proliferative activity of follicular cells and their resistance to apoptosis. These studies have shown that somatotrophic hormone can act as a survival factor for oviductal cells, determining the rate of regression and renewal of the oviduct during molting, and regulating the secretory activity of the latter. Most information on the mechanisms of STH action on the female reproductive tract cells was obtained in mammals and requires confirmation in birds. Further investigations should be aimed at elucidating the intracellular signaling pathways activated by STH in ovarian and oviductal cells of chickens, possible paracrine mediators and factors modulating the direct effect of the hormone on cells.

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