# Developmental Patterns and Neuroendocrine Mechanisms of Thermoregulation in Young Poultry: Postprint

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**Date:** 2018-12-24T00:00:00+00:00

## Abstract

From egg incubation to early post-hatch period, the thermobalance of avian embryos and chicks is sequentially constrained by the high thermal conductivity of eggs and embryos, the diffusion resistance of oxygen across the eggshell and chorioallantoic membrane, the developmental degree of the thyroid gland, and the metabolic capacity of young chicks. It is not until the late stage of egg incubation that avian embryos exhibit thermogenic responses to cold stimulation. Around hatching, chicks already possess well-developed cutaneous and core temperature sensing functions, and hypothalamic neurons exhibit high sensitivity to cold. Central hormones such as hypothalamic thyrotropin-releasing hormone and corticotropin-releasing hormone have important effects on thermogenesis in chicks; the hypothalamic-pituitary-thyroid axis begins to function from the mid-incubation period and interacts with the hypothalamic-pituitary-adrenal axis in the later stages, yet direct evidence for thyroid hormones and corticosterone regulating body temperature development in neonatal chicks has not been found to date. This review summarizes the developmental patterns and neuroendocrine mechanisms of thermoregulatory function in chicks, aiming to provide references for the regulation of thermal comfort in poultry, particularly regarding central nervous system plasticity.

## Full Text

### Ontogenic Pattern of Avian Thermoregulation and Underlying Neuroendocrine Mechanisms

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**Abstract**

From the onset of incubation through early post-hatch life, the thermoregulatory capacity of avian embryos and neonates is sequentially constrained by the high thermal conductance of eggs and embryonic tissues, the diffusion resistance of oxygen across the eggshell and chorioallantoic membrane, the developmental status of the thyroid gland, and the metabolic capacity of the hatchlings. Not until the late stages of incubation do avian embryos exhibit thermogenic responses to cold stimuli. Around the time of hatching, chicks possess well-developed peripheral and core temperature sensing capabilities, with hypothalamic neurons displaying high sensitivity to cold. Central hormones such as hypothalamic thyrotropin-releasing hormone and corticotropin-releasing hormone exert significant influence on thermogenesis in chicks. The hypothalamic-pituitary-thyroid (HPT) axis becomes functional during mid-incubation and interacts with the hypothalamic-pituitary-adrenal (HPA) axis toward the later stages, though direct evidence for thyroid hormones and corticosterone regulating temperature development in newborn chicks remains elusive. This review synthesizes the ontogenic patterns of thermoregulatory function and underlying neuroendocrine mechanisms in avian neonates, aiming to provide insights for modulating thermal comfort in poultry, particularly regarding the plasticity of the central nervous system.

**Keywords:** thermoregulation; neuroendocrine; ontogeny; avian embryo; neonatal birds

The temperature of avian embryos is subject to external environmental conditions, with the thermoregulatory system undergoing continuous development throughout incubation and not achieving mature function until several weeks post-hatch [1-3]. Unlike most wild birds (which are altricial, characterized by short incubation periods, naked and unfeathered bodies at hatching, closed eyes, inability to live independently, requiring nest retention for warmth and parental feeding for an extended period, typified by Passeriformes), domestic poultry such as chickens and ducks are precocial (featuring longer incubation periods, feathered bodies at hatching, open eyes, strong legs, and the ability to follow parents and forage shortly after down drying, typified by Anseriformes and Galliformes), possessing substantial thermogenic capacity from birth [4]. The maturation of thermoregulatory function in avian neonates begins with embryonic nervous system development, proceeds through the differentiation of heat production and dissipation mechanisms (activating these neuronal controllers), and concludes with post-hatch growth that alters the surface-to-volume ratio and insulation properties, thereby enhancing heat conservation [3,5-7]. Thermoregulatory responses during the perinatal period significantly influence subsequent

thermobalance and production performance [8-12]; to elucidate the epigenetic mechanisms underlying this acquired environmental adaptation, it is essential to first understand the developmental patterns and regulatory mechanisms of the thermoregulatory system during incubation and early life.

## 1 Developmental Stages of Thermoregulatory Function in Avian Neonates

From the onset of incubation until the maturation of thermoregulatory function after hatching, avian neonates must progress through four distinct stages (Figure 1 [Figure 1: see original paper]) [3,13] to achieve independence from ambient temperature dependence.

### 1.1 Arrhenius Limitation Period

The first stage occurs before pipping, when the neural control system and peripheral heat production and dissipation mechanisms (effectors) are underdeveloped, rendering the embryo incapable of thermoregulation. Although metabolic rate increases progressively with embryonic age, it remains relatively low and directly dependent on ambient temperature [14]. When environmental temperature decreases, the egg cools and embryonic metabolism diminishes instantaneously. In chicken embryos, for example, metabolic rate (measured as oxygen consumption) at 24°C is only 40% of that at 37°C. This relationship conforms to the Arrhenius equation describing temperature effects on chemical reaction rate constants, hence the term "Arrhenius limitation period." The temperature coefficient Q10 (based on the exponential relationship between embryonic metabolic rate and temperature, where the logarithm of the former varies linearly with the latter, representing the fold change in metabolic rate per 10°C temperature increase) is approximately 2 [15].

### 1.2 Oxygen ($O_2$) Conductance Limitation Period

The second stage extends from pipping to hatching, during which the embryo becomes covered with skin and feathers, and the brain and skeletal muscle tissues (particularly leg muscles) exhibit vital activity [16]. Because the embryo remains within the egg, heat production is constrained by the eggshell (external diffusion barrier) and chorioallantoic membrane (internal diffusion barrier) that impede oxygen uptake. When egg temperature decreases, oxygen consumption increases if the embryo' s basal metabolic rate is below the $O_2$ transport limit; however, if basal metabolic rate has already reached this limit, oxygen consumption remains stable. Nevertheless, if embryonic heat production is insufficient to elevate body temperature above ambient temperature for a sustained period, the egg cools; as egg temperature declines, the thermoregulatory system gradually "shuts down,"reverting to the Arrhenius limitation period [17]. Unlike precocial species, altricial birds do not develop effective thermoregulatory systems before hatching and thus remain in the Arrhenius limitation period without experiencing the $O_2$

conductance limitation period [18].

### 1.3 Thermogenic Limitation Period and Homeothermic Period

The third stage spans from hatching to approximately 10 days post-hatch, when metabolic rate is no longer limited by the eggshell as the site of gas exchange shifts from the chorioallantoic membrane to the lungs, enabling maximal heat production under adequate $O_2$ conditions [19]. During this period, however, the thermogenic capacity of most chicks remains insufficient to counteract heat loss due to the immaturity of liver, digestive tract, and other tissues, as well as limited thyroid activity, preventing them from achieving thermal independence. Some poultry species, such as mallard ducks and brush turkeys, exhibit robust thermoregulatory ability from hatching and can bypass the "thermogenic limitation period," proceeding directly to the "homeothermic period" [20]. The final stage commences at 10–13 days post-hatch and extends into adulthood [1,21]. Through coordinated actions of insulation (e.g., subcutaneous fat), heat production, heat dissipation (e.g., lungs, kidneys, blood vessels), and neuroendocrine systems, chicks achieve complete physiological, morphological, and behavioral adaptation to environmental temperature fluctuations, ultimately establishing thermobalance.

## 2 Neural Mechanisms Underlying the Development of Thermoregulatory Function in Avian Neonates

Similar to mammals, the neural thermoregulatory system in birds comprises three components: temperature sensation and afferent pathways, central nervous system integration of thermal signals, and effector pathways that initiate autonomic and behavioral adjustments.

### 2.1 Formation of Thermosensation in Avian Neonates

Birds possess temperature receptors in peripheral tissues such as skin, tongue, and beak, as well as in deep-body organs including abdominal viscera [22]. The differentiation of the peripheral nervous system in chickens begins early in incubation, with sensory fibers from the facial ganglion entering the brain at 2–3 days of embryonic age [18]. By day 8 of incubation, chicken embryos exhibit increased or decreased heat production within a short period (<16 min) when ambient temperature is reduced or elevated by 0.3°C [23]. Myelination of peripheral nerves occurs around day 11 of incubation [18].

Around hatching, researchers have indirectly confirmed the presence of peripheral and deep-body thermoreceptors through local or whole-body cooling and heating experiments. For instance, cloacal cooling induces shivering in 1-week-old chicks, while low temperatures increase nitric oxide (NO) release from the chorioallantoic membrane and hypothalamus, reducing activity in neonatal chicks [24-26]. From the onset of pipping (3–4 days before hatching), embryos can communicate vocally with each other, producing distress calls [27]. When

Muscovy duck eggs are immersed in cool water (20–22°C), distress vocalizations increase; upon rewarming, these calls decrease [28]. These findings demonstrate that avian neonates possess well-developed temperature perception from birth, and their inability to maintain constant body temperature during the neonatal period (cold susceptibility) is unrelated to thermosensory deficits.

### 2.2.1 Brain Structures

The preoptic-anterior hypothalamus (PO/AH) serves as the central coupling unit between thermoreceptors and effectors, containing not only cold-sensitive and warm-sensitive neurons [29-31] that finely regulate normal temperature variations but also a small population of "temperature guardian neurons" [30-31] that maintain brain temperature between 36–42°C during thermal dysregulation. Brain development in chicks initiates at day 2 of incubation, continues throughout the entire incubation period, and extends until three weeks post-hatch [32]. Diencephalic differentiation begins at day 3, and by day 9, the diencephalic anatomy reaches the level of neonatal chicks [33]. Precocial chicks exhibit more extensive brain growth before hatching compared to altricial species [34], which may contribute to their superior thermoregulatory capacity during the neonatal period.

The temperature sensitivity of the hypothalamus in neonatal Muscovy ducks undergoes stage-specific changes: from embryonic day 28 to post-hatch day 5, the PO/AH contains a higher proportion of cold-sensitive neurons (30%) and fewer warm-sensitive neurons (5%); between post-hatch days 5–10, the proportion of cold-sensitive neurons declines (14%) while that of warm-sensitive neurons increases (15%) [30,35]. In adult Pekin ducks, cold-sensitive and warm-sensitive neurons represent 6.2% and 58.3% of the population, respectively [36]. These findings indicate that during maturation of thermoregulatory function, hypothalamic cold sensitivity decreases while warm sensitivity increases. Both body temperature and brain temperature in chicks follow power functions of age, rising at different rates (body temperature > brain temperature) and reaching adult levels by approximately day 10 [37]. During this period, the temperature differential increases linearly with age, likely regulated by the development of the ophthalmic rete (an extracranial vascular network in the head that cools blood flowing to the brain through arteriovenous heat exchange) [38].

### 2.2.2 Spinal Cord

The avian spinal cord contains cold-sensitive and warm-sensitive neurons, with the cervical and thoracic spinal cord participating in temperature sensation [22]. Beyond perceiving local temperature, the spinal cord serves as a conduit for afferent thermal signals from the body surface and core to the central nervous system and integrates some temperature signals. Spinal cord differentiation in chicken embryos begins during the first week of incubation [39]. Synapses are first observable at day 5 of incubation, electrophysiological activity emerges from days 4–5, and spinal tracts are well-developed by day 7. Although the funda-

mental spinal architecture is established during incubation, whether it possesses mature temperature signal transmission and integration functions around the time of hatching remains unclear.

### 2.3 Effector Pathways for Thermoregulation

The final triggering of thermoregulatory responses depends on well-developed effector pathways that interact with sympathetic and parasympathetic nervous systems to activate endocrine organs, specifically the thyroid and adrenal glands [40]. In chickens, most motor neurons (efferent neurons that transmit information from spinal cord and brain to muscles and endocrine glands, controlling effector organ activity) are formed by day 6 of incubation. Muscle reflexes reflecting local proprioceptive efferent activity capacity are first observable at day 10 of incubation [39].

## 3 Endocrine Mechanisms Underlying the Development of Thermoregulatory Function in Avian Neonates

Since the 1960s, research on hormonal roles in the development of thermoregulatory function in avian neonates has primarily focused on the hypothalamic-pituitary-thyroid (HPT) axis. From late incubation (day 18 in chickens) onward, the hypothalamic-pituitary-adrenal (HPA) axis demonstrates interactive effects with the HPT axis [41].

### 3.1 Hypothalamus, Pituitary, and Thyroid

Between days 6.5–13.5 of incubation, the number of thyrotropin-releasing hormone (TRH)-positive perikarya in hypothalamic neurons of chicken embryos gradually increases [42]. Hypothalamic function begins to mature between days 11.5–16.0 of incubation. From day 14 of incubation to hatching, hypothalamic TRH levels increase tenfold, while somatostatin (SRIH) levels only double; from pipping to hatching, TRH levels in the chicken embryo hypothalamus increase twofold [43]. Thyrotropic cells are first observable in the distal pituitary of chicken embryos at day 6.5 [44]. Thyrotropin (TSH) levels decrease from day 14 of incubation to hatching but gradually increase from birth to adulthood [43].

Thyroid differentiation in chickens begins with evagination from the base of the pharynx, splitting into two lobes by day 5 of incubation. By day 8, the gland becomes uniformly vascularized; by day 10, sinusoidal vascular development reaches its maximum. Thyroid follicles appear between days 10–11, thyroid weight increases proportionally with body weight from days 10–15, and histological structure is fully developed by day 15 [45-46]. During mid-to-late incubation, the thyroid glands of precocial chicks (e.g., chickens and quail) exhibit functional activity, including iodine uptake and synthesis of thyroglobulin and thyroid hormones, whereas altricial chicks (e.g., pigeons) show functional activity only after hatching [47].

Tyrosine iodination is observable in chicken embryo thyroids between days 9–11 of incubation, coinciding with the appearance of thyroid follicles [48]. Thyroxine (T4) has been detected at day 6.5 of incubation [49], though it remains uncertain whether this originates from maternal sources [50]. Although the thyroid cannot concentrate iodine until at least day 5, iodine uptake becomes markedly enhanced by days 11–13 [48]. Plasma levels of triiodothyronine (T3) and T4 are low during mid-incubation, then gradually increase, peaking at hatching, subsequently declining, and stabilizing from day 10 post-hatch onward [51-52].

During early incubation (days 3–10), the adenohypophysis can regulate the thyroid autonomously, but later (after day 16), hypothalamic involvement becomes necessary [53]. The pituitary is regulated by TRH from day 6.5 onward, becoming increasingly sensitive after day 13.5 [54]. Similarly, thyroid activity shifts from autonomous regulation to pituitary dependence from day 11.5 onward, with progressively stronger responsiveness to TSH. In contrast to precocial chicks, the pituitary of altricial chicks lacks biological function during embryonic development and exerts no influence on thyroid activity [47].

### 3.2 HPT Axis and Thermoregulation

In low-temperature environments (30°C), chicken embryos exhibit sustained metabolic responses from the time of membrane detachment [55]. Under cold exposure (20°C), intraperitoneal injection of 300  g/kg T3 or T4 in 1-day-old chicks slows the rate of rectal temperature decline, demonstrating the thermogenic effect of thyroid hormones [56]. By 14 days post-hatch, quail thyroid follicular cell height reaches adult levels, coinciding with substantially enhanced cold tolerance [57]. Thyroid hormones regulate basal metabolic rate (obligatory thermogenesis) by increasing mitochondrial oxygen consumption and promoting ATP generation.

Exogenous thyroid hormone administration enables chicks to resist cold stress. However, under normal temperatures, intraperitoneal T3 injection elicits thermogenic effects only in chickens older than 2 weeks, proving ineffective in chicks younger than 1 week [58-60]. This may be attributed to high endogenous T3 levels in neonates saturating receptor binding sites, or to underdevelopment of muscle tissue or T3 signaling pathway components. Nevertheless, blocking peripheral T4-to-T3 conversion with iopanoic acid causes rectal temperature decline in 1-day-old chicks [61], confirming T3' s role in thermoregulation of newborn chicks.

Intracerebroventricular TRH injection increases body temperature, respiratory rate, oxygen consumption, and carbon dioxide ($CO_2$) production in 1–2-week-old chicks without significantly altering thyroid hormone levels [60], suggesting TRH' s thermogenic effect is independent of T3 or T4 and may directly regulate energy metabolism. However, intravenous TRH administration does elevate thyroid hormone levels [62]. The precise mechanisms of TRH-mediated thermoregulation in avian neonates require further investigation.

### 3.3 HPT Axis and HPA Axis

Intracerebroventricular administration of corticotropin-releasing hormone (CRH) significantly elevates rectal temperature (at 10, 30, and 60 min post-injection), oxygen consumption, $CO_2$ production, and heat production in 2–4-day-old chicks [58,63], suggesting HPA axis involvement in avian thermoregulation. In chickens, CRH and somatostatin (SRIH) influence TSH release. Components resembling ovine corticotropin-releasing factor (CRF) are detectable in perikarya of periventricular hypothalamic neurons as early as day 14 of incubation [64-65]. On day 18 of incubation, injection of ovine CRH into chicken embryos increases not only plasma corticosterone levels but also plasma T3 and T4 levels [66]. Since TSH levels rise concurrently, CRH may act on the thyroid by modulating TSH secretion [67]. From day 14 of incubation, chicken pituitary thyrotropes exhibit responsiveness to CRH [68], possibly due to high expression of CRH receptor 2 (with low abundance of CRH receptor 1) [69].

## 4 Summary

The susceptibility of chicks to cold is common knowledge in the poultry industry, yet the underlying scientific question remains: what are the physiological mechanisms governing the transition from ectothermy to endothermy in avian neonates? Answering this question is fundamental to leveraging the plasticity of early central nervous system development for modulating thermal comfort in poultry. Over the past decades, the spatiotemporal developmental patterns of key neural structures and endocrine components related to thermoregulation in avian embryos and neonates have been established at the tissue level of the hypothalamus, spinal cord, and thyroid. With the completion of quantitative trait loci (QTL) identification and chromosomal mapping for avian thermoregulatory control [70-71], future research employing emerging high-throughput genomic sequencing technologies such as high-density single nucleotide polymorphism (SNP) chips promises to elucidate the functional connections and signaling networks underlying the genesis and development of these components at cellular and molecular levels.

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*Note: Figure translations are in progress. See original paper for figures.*