Advances in Regulatory Mechanisms and Key Regulatory Factors of Poultry Feed Intake: Postprint

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Abstract

Feeding is the fundamental pathway for poultry to ingest nutrients and a prerequisite for their healthy growth. Feed intake and feeding behavior are closely correlated with poultry production performance. This article, by summarizing and analyzing the concepts, regulatory mechanisms, and primary regulatory factors of poultry feed intake, provides a theoretical foundation for in-depth investigation into the physiological regulatory mechanisms of poultry feeding, which holds significant importance for stimulating poultry appetite, enhancing feed intake, and advancing the development of animal husbandry.

Full Text

Research Progress on Regulation Mechanism and Main Regulatory Factors of Feed Intake in Poultry

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Abstract

Feeding is the fundamental pathway for poultry to acquire nutrients and a prerequisite for healthy growth. Feed intake and feeding behavior are closely correlated with poultry production performance. This paper summarizes and analyzes the concept, regulatory mechanisms, and main regulatory factors of poultry feed intake, providing a theoretical foundation for further research into

the physiological regulation mechanisms of poultry feeding. This work is significant for promoting poultry appetite, increasing feed intake, and advancing the development of animal husbandry.

Keywords: poultry; feed intake; mechanism; regulatory factors

1. Feed Intake

Poultry feed intake typically refers to the total amount of feed consumed by poultry within 24 hours [5]. Feed intake reflects the quantity of various nutrients obtained by poultry and serves as the basis for evaluating nutrient requirements and energy metabolism [6-7]. Feed intake includes ad libitum intake and actual intake [8]. Ad libitum intake refers to the total amount of feed consumed by individual animals or groups under free-access conditions, while actual intake refers to the total amount of feed consumed by animals under practical feeding conditions within a specific period [9]. Although feed intake can be expressed as the total weight of feed consumed in 24 hours, different feeds contain varying levels of dry matter, energy, and protein. Therefore, identical feed weights do not necessarily mean identical nutrient intake. Expressing intake in terms of energy is more accurate because the essence of poultry feeding is to meet energy requirements [9].

2. Regulatory Mechanisms of Feed Intake

2.1 Central Regulation

The central nervous system (CNS) is the key site for feed intake regulation in poultry. The CNS integrates and processes various complex food signals, then stimulates the feeding center to generate either satiety or hunger, thereby regulating the initiation or termination of feeding [10]. The hypothalamus is the most critical component of the feeding regulation center, achieving precise control of poultry feeding by sensing and integrating various peripheral stimuli [11]. Several hypothalamic nuclei participate in feed intake regulation, including the arcuate nucleus (ARC), paraventricular nucleus (PVN), ventromedial nucleus (VMN), dorsomedial nucleus (DMH), and lateral hypothalamic area (LHA) [12]. The feeding center, located in the lateral hypothalamic area, is diffuse and connected to other brain regions via nerve fibers, serving as the central site that stimulates feeding. The satiety center, primarily located in the ventromedial nucleus of the hypothalamus, is the central site that inhibits feeding, and its signals can project to the feeding center to suppress its excitation [13].

After peripheral feeding-related signals (such as insulin, leptin, glucose, etc.) reach the CNS, they are processed and integrated in the poultry hypothalamus, ultimately promoting feeding through the neuropeptide Y (NPY) and

agouti-related protein (AgRP) pathways, or inhibiting feeding through the pro-opiomelanocortin (POMC) and cocaine- and amphetamine-regulated transcript (CART) pathways [12,14] [Figure 1: see original paper]. The blood-brain barrier in the hypothalamic arcuate nucleus senses changes in peripheral blood signals, compares the strength of orexigenic and anorexigenic signals, processes and integrates these signals, and ultimately produces effects that either promote or inhibit poultry feeding [15].

2.2 Physical and Chemical Regulation

Various physical indicators of poultry feed, such as appearance and hardness, also provide important stimulation for feeding. Physical regulation is closely related to gastrointestinal volume and the volume of feed and chyme. After poultry consume feed, it is first stored in the crop, filling and stimulating crop receptors before entering the digestive tract, where it fills and stimulates pressure and osmotic pressure receptors in the gastrointestinal tract. These signals are ultimately transmitted via afferent nerves to the nerve center, causing poultry to terminate feeding [3]. Tension or emptying of the gastrointestinal tract creates pressure changes that form a negative feedback loop; the gastrointestinal tract senses these pressure changes and stops feeding [16].

Chemical regulation primarily modulates feed intake through changes in chyme composition and absorbed nutrient concentrations [9]. Substances such as glucose, volatile fatty acids, amino acids, and hormones can act directly on the hypothalamic feeding center by binding to receptors in the digestive tract or other locations, or indirectly affect the central feeding center by altering endocrine function and stored energy [17]. Among these, glucose and volatile fatty acids are the most important factors.

2.3 Short-term and Long-term Regulation

Short-term regulation refers to the control of each feeding activity, achieved by activating hormonal and neural signaling pathways in the gastrointestinal tract through feed or specific nutrients [18]. Nutrient content such as amino acids and physical properties of feed can directly act on neural receptors in the gastrointestinal tract or stimulate the production of appetite-related regulatory peptides, thereby activating specific signaling pathways that transmit satiety signals to the central integrative center [19].

Long-term regulation of feed intake refers to the long-term energy balance regulation in poultry [20]. This regulation depends on body energy reserves and fat stores, achieved through peripheral signaling molecules that activate hypothalamic neural and neuroendocrine pathways. The most typical theory regarding long-term feeding regulation is the lipostatic theory, which proposes that animals feed to maintain a certain amount of body fat reserves. When body fat is depleted, feed intake tends to increase to compensate for the loss [21] [Figure 2: see original paper].

3. Main Regulatory Factors

3.1 Orexigenic Factors

3.1.1 NPY Hypothalamic NPY in poultry is one of the main factors promoting feeding and plays an important role in feed intake and energy balance [22]. NPY is an endogenous appetite signal transduction factor that strongly stimulates animal feeding and regulates various appetite-promoting and appetite-inhibiting factors. It is currently recognized as the most important orexigenic hypothalamic neuropeptide [23]. NPY is concentrated in the arcuate nucleus of the hypothalamus and is transmitted via projection fibers to nuclei such as the paraventricular nucleus, ventromedial nucleus, lateral area, and dorsomedial nucleus to stimulate the feeding center [24]. NPY can regulate feeding at the central nervous system level and specifically stimulate carbohydrate intake in animals [25]. Blankenship et al. [26] reported that feed restriction significantly increased hypothalamic levels of NPY, AgRP, corticotropin-releasing hormone (CRH), and melanocortin 1 receptor (MC1R) in male and female quail, indicating that feed restriction induced hunger and enhanced appetite. Nakajima et al. [27] demonstrated that NPY significantly increased feed intake in mice. Katner et al. [28] found that intracerebroventricular injection of 5 g/ L NPY in Wistar rats also significantly increased feed intake. Saneyasu et al. [29] reported that central injection of NPY significantly increased feed intake in broiler chickens. NPY not only stimulates normal feeding but also exhibits time- and dose-dependent cumulative effects [30]. These studies indicate that NPY can promote animal appetite and feed intake, suggesting that exogenous NPY could be used as a short-term appetite regulatory factor in production practice to improve poultry appetite and feed intake.

3.1.2 AgRP AgRP is a neuropeptide produced by AgRP/NPY neurons in the hypothalamic arcuate nucleus. AgRP can promote animal feeding by selectively binding to melanocortin receptors and blocking the action of α-melanocyte-stimulating hormone (α-MSH) [31]. AgRP can also directly interact with melanocortin receptor 4 (MC4R) as an inverse agonist [32]. After competitively binding to melanocortin receptor 3 (MC3R) and MC4R, AgRP antagonizes α-MSH-mediated G protein activation, reduces intracellular cyclic adenosine monophosphate (cAMP) content, and thereby enhances animal appetite. Tachibana et al. [33] reported that AgRP increased feed intake in laying hens and reduced the inhibitory effect of α-MSH on feeding, demonstrating that AgRP can enhance poultry appetite and increase feed intake.

3.1.3 Orexin (ORX) Orexin, also known as the feeding factor, is distributed only in the hypothalamic feeding center, particularly the feeding center. ORX encodes two neuropeptides, ORXA and ORXB, whose main functions are to regulate animal feed intake and energy balance [34]. Székely et al. [35] reported

that intracerebroventricular injection of ORXA in mice caused voracious appetite after 30 minutes. ORX can also stimulate digestive juice secretion and promote gastric motility [16]. ORX and NPY have synergistic effects in promoting feeding. López et al. [36] reported that ORX injection significantly increased NPY mRNA expression in rats. However, some studies in poultry found that hypothalamic ORX did not affect chicken feed intake [37]. Further research on poultry ORX is needed to clarify its regulatory mechanisms in poultry and its relationship with other hormones and neuropeptides.

3.1.4 Ghrelin Ghrelin is a small peptide discovered in gastric endocrine cells and the hypothalamic arcuate nucleus. Ghrelin has multiple functions, including stimulating growth hormone release from the anterior pituitary, enhancing animal appetite, regulating energy balance, and promoting gastric acid secretion [38]. Kamegai et al. [39] reported that ghrelin significantly increased feed intake and mRNA expression of NPY and AgRP in mice. Asakawa et al. [40] demonstrated that ghrelin promoted gastrointestinal motility and gastric acid secretion in rats. These studies indicate that ghrelin can enhance appetite and increase feed intake in mammals. However, avian ghrelin differs structurally from mammalian ghrelin and has different regulatory effects on feeding [41]. Furuse et al. [42] found that intracerebroventricular injection of ghrelin significantly inhibited feeding behavior in chicks. Saito et al. [43] reported that intracerebroventricular injection of ghrelin in 4-day-old chicks promoted corticosterone secretion in a dose- and time-dependent manner and inhibited feed intake. Ocłoń et al. [44] reported that intraperitoneal injection of ghrelin caused anorexia in broiler chickens. Geelissen et al. [45] also reported that ghrelin reduced feed intake in chickens. These studies demonstrate that ghrelin can inhibit feed intake in poultry. The effect of ghrelin on poultry feeding is a complex regulatory process, and its specific mechanisms require further in-depth research.

3.2 Anorexigenic Factors

3.2.1 POMC POMC is one of the important hypothalamic appetite-inhibiting factors. Avian POMC consists of 256 amino acid residues and is mainly distributed in the hypothalamus, anterior pituitary, and adipose tissue [46]. Pro-opiomelanocortin plays an important role in feeding behavior, body weight, and energy homeostasis regulation. POMC is the precursor of melanocortin (MC), α-MSH, lipotropin, and β-endorphin. MC binds to melanocortin receptors (MCRs) and plays important roles in regulating animal feeding, energy metabolism, epidermal pigment deposition, sebaceous gland secretion, and reproduction [47]. MC4R is particularly important in controlling appetite and body weight homeostasis [47]. Studies have shown that intracerebroventricular injection of MC4R agonists can inhibit feeding, while injection of selective antagonists leads to hyperphagia [48]. Klovins et al. [49] found that when central POMC expression is activated in mammals, its product α-MSH reduces feeding by activating the sympathetic nervous system

via melanocortin receptors MC3R and MC4R. Zendehdel et al. [50] reached the same conclusion in broiler experiments.

3.2.2 CART CART is widely distributed in the central nervous system, peripheral nervous system, and peripheral tissues, particularly in the hypothalamus and gastrointestinal tract [51]. Studies have shown that CART can reduce animal feed intake in a dose-dependent manner [52]. Tachibana et al. [53] reported that intracerebroventricular injection of CART significantly inhibited feeding behavior in fasted broiler chickens. Research indicates that CART likely inhibits feeding by affecting gastric acid secretion and gastric emptying, thereby causing mechanical stimulation of visceral organs and transmitting satiety signals [52]. Okumura et al. [54] found that CART reduced feed intake in rats while also decreasing gastric juice and acid secretion and slowing gastric emptying. These studies demonstrate that CART can reduce feed intake in poultry by acting on both the hypothalamus and gastrointestinal tract to decrease appetite and feed digestion rate.

3.2.3 Cholecystokinin (CCK) CCK is a gastrointestinal polypeptide hormone that causes gallbladder contraction and promotes enzyme secretion in pancreatic juice. It is widely present in the digestive system, central and peripheral nervous systems, and has an inhibitory effect on animal feeding [55-56]. CCK stimulates pancreatic enzyme secretion and gallbladder contraction, participates in regulating gastrointestinal motility, and induces satiety [57]. At least two types of CCK receptors exist in poultry: CCK-A and CCK-B. After binding to receptors, CCK transmits signals via the vagus nerve to activate POMC neurons in the nucleus of the solitary tract, which send satiety signals through their receptors (MC4R) to produce appetite-suppressing effects [58]. Under physiological conditions, feed entering the gastrointestinal tract causes physical and chemical stimulation of the digestive tract, leading to peripheral CCK release. Gastric distension stimulates circulating CCK to bind to CCK-A receptors on gastric vagal nerves, converting the signal into neural impulses that are transmitted to the nucleus of the solitary tract (NTS) in the medulla oblongata, thereby inhibiting the hypothalamic feeding center [59]. Savory et al. [60] reported that CCK can reduce feed intake in chickens in a dose-dependent manner.

3.2.4 Leptin Leptin is a protein hormone encoded by the obese gene, with avian adipose tissue and liver being the main sites of leptin synthesis [61]. Leptin reduces animal appetite, improves energy metabolism efficiency, and increases energy expenditure. Studies have shown that both short-term high-dose and long-term low-dose leptin injections can reduce hypothalamic NPY mRNA expression while increasing POMC mRNA expression associated with anorexia [62-63]. Boswell et al. [64] reported that leptin regulates feed intake by modulating the expression of AgRP and POMC genes. Cassy et al. [65] demonstrated that leptin significantly reduced feed intake in 56-day-old laying hens. Denbow

et al. [66] reported that intracerebroventricular injection of recombinant human leptin protein significantly inhibited feed intake in 42-day-old broiler chickens. Dridi et al. [67] found that leptin significantly reduced hypothalamic mRNA expression of NPY and ORX in 21-day-old broiler chickens. These studies indicate that leptin signaling can enhance POMC/CART neuronal activity while reducing NPY/AgRP neuronal activity, thereby decreasing poultry feed intake.

3.2.5 Insulin Insulin is produced and secreted by pancreatic β-cells, can penetrate into cerebrospinal fluid, and insulin receptors exist in key hypothalamic regions, playing an important role in animal feed intake regulation. Studies have shown that insulin binding to its receptors can regulate ATP-sensitive potassium channel (KATP) activity in POMC neurons of the hypothalamic VMN, further affecting intracellular reactive oxygen species (ROS) levels through the phosphatidylinositol 3-kinase (PI3K) signaling pathway to modulate energy status and appetite [68]. Insulin entering the CNS can bind to corresponding receptors on arcuate nucleus neurons, activate insulin receptor signaling, inhibit NPY/AgRP neuronal expression, promote POMC neuronal synthesis and secretion, and suppress animal feeding. Insulin can also work together with leptin to inhibit AMP-activated protein kinase activity in the hypothalamus, increase acetyl-CoA carboxylase activity, raise malonyl-CoA activity, and reduce poultry feed intake [69-70]. However, Liu et al. [71] reported that insulin injection did not affect appetite in broiler chickens.

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4. Conclusion

Although the regulatory mechanism of poultry feed intake is extremely complex and not yet fully understood, significant progress has been made in recent years with advances in science and technology, leading to the identification of key regulatory factors and signaling pathways. In-depth investigation of feed intake regulatory mechanisms using gas chromatography-mass spectrometry (GC-MS) and multi-omics technologies is crucial for improving poultry appetite, developing new feed attractants, increasing feed intake, and enhancing production performance.

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