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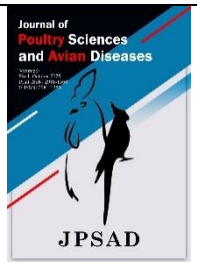

Dopaminergic Receptor Involvement in Insulin-Induced Anorexia in Broiler Chickens

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A B S T R A C T

The neurobiological mechanisms underlying appetite regulation and feeding behavior exhibit considerable complexity and interspecies variation. Among the key neurotransmitters implicated in the modulation of feeding behavior are dopamine and insulin, yet the interplay between these signaling molecules remains inadequately characterized. This investigation aimed to elucidate the interactions between insulin and the dopaminergic system in the context of appetite regulation in broiler-type chickens (Ross 308). Experimental protocols involved the intracerebroventricular (ICV) administration of insulin at doses of 2.5, 5, and 10 ng, respectively. Additionally, dopaminergic agents, including L-DOPA (a dopamine precursor) and receptor-specific antagonists SCH 23390 (D1), AMI-193 (D2), NGB 2904 (D3), and L-741,742 (D4), were administered alone or in combination with insulin (10 ng). Meal consumption was quantified cumulatively at 30-, 60-, and 120-minute intervals following the infusion. The findings revealed that insulin elicited a dose-dependent suppression of food intake ($p < 0.05$). Notably, the anorexigenic effect of insulin was attenuated by SCH 23390 (5 nmol) ($p < 0.05$), implicating D1 receptor-mediated pathways, whereas antagonists targeting D2, D3, and D4 receptors failed to modulate this response ($p > 0.05$). These results substantiate the critical role of D1 receptors in mediating insulin-induced anorexia in meat-type chickens, thereby advancing our understanding of the neurochemical interactions governing avian feeding behavior.

Keywords: *Dopaminergic receptors; Insulin; Feed intake; Broiler*

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1 Introduction

Appetite control in birds is managed through complex, multilayered homeostatic processes that require integrated communication between the central and peripheral nervous systems (CNS and PNS), as well as regulatory signals originating from peripheral organs such as the gastrointestinal tract, adipose tissue, and liver (1). While parallels exist between avian and mammalian systems—particularly in the localization of neural appetite-regulatory centers within the hypothalamus (2)—divergences, such as species-specific environmental influences on dietary selection, have been documented (3, 4). For instance, avian species exhibit distinct foraging behaviors and dietary preferences shaped by ecological and sensory cues, underscoring the evolutionary adaptation of appetite regulation to ecological niches (3). Central to this regulation are neural and hormonal mediators, whose identification and functional characterization are critical for elucidating the mechanistic basis of feeding behavior in birds (5). A deeper understanding of these pathways holds significant implications for avian physiology, agricultural productivity, and the management of metabolic disorders in poultry.

Among the hormones that have been extensively investigated in contemporary scientific literature, insulin has been recognized as a pivotal mediator in the regulation of caloric consumption and appetite. It is essential for the regulation and maintenance of glucose balance through the modulation of carbohydrate, lipid, and protein metabolism, as well as mitogenic activities (6). Beyond its peripheral metabolic functions, insulin acts as a neuroregulatory agent within the CNS, influencing energy balance, neuronal survival, and glucose metabolism (7). Its primary target, the arcuate nucleus of the hypothalamus, integrates peripheral energy signals to modulate energy expenditure and feeding behavior (8). In mammals, central insulin administration suppresses food intake via melanocortinergic pathways (9). Analogous studies in avian models demonstrate its capacity to upregulate pro-opiomelanocortin (POMC) and suppress neuropeptide Y (NPY) expression in laying hens, suggesting conserved yet nuanced roles across species (10). These findings highlight insulin's dual role as both a metabolic and neuroendocrine regulator, bridging systemic energy status with central control of appetite.

Dopamine, a neurotransmitter derived from L-DOPA, mediates a range of neurophysiological processes, including motivation, motor control, and interneuronal signaling. Its five receptor subgroups (D1–D5) are categorized into D1-like (D1, D5) and D2-like (D2, D3, D4) families, which differentially modulate intracellular signaling cascades (11). Avian and rodent studies suggest that dopaminergic signaling plays a role in appetite modulation, with evidence in chickens indicating that D1 and D2 receptors mediate hypophagic responses. In contrast, other subtypes appear to be uninvolved (12, 13). This receptor-specificity underscores the complexity of dopaminergic regulation and its potential as a therapeutic target for metabolic dysregulation (14). Notably, dopamine's role in reward pathways further positions it as a critical mediator of context-dependent feeding behaviors, linking metabolic needs with environmental stimuli (15).

Emerging research highlights crosstalk between the insulin and dopaminergic systems, particularly under conditions of metabolic and psychological stress. Psychological stressors, such as depression, disrupt dopamine signaling and induce insulin resistance, illustrating bidirectional interactions between metabolic and neural pathways (16, 17). Conversely, insulin potentiates dopamine release in the nucleus accumbens via striatal cholinergic interneurons, suggesting a feedback loop that integrates energy status with reward-motivated behaviors (18). Molecular interactions include insulin-mediated regulation of dopamine reuptake (via dopamine transporter, DAT), modulation of dopamine catabolism (via monoamine oxidases, MAO), and alterations in neuronal firing frequency (19-21). These mechanisms collectively suggest that insulin may fine-tune dopaminergic activity to align feeding behavior with metabolic demands.

Despite these advances, the interplay between central dopaminergic pathways and insulin in avian feeding regulation remains unexplored. Broiler chickens, a key species in global poultry production, present a unique model for investigating these interactions due to their rapid growth rates and metabolic efficiency, which are heavily influenced by feed intake patterns. By elucidating how insulin-dopamine crosstalk modulates appetite in this species, the current study aimed to uncover novel neuroendocrine mechanisms that could inform strategies for optimizing feed efficiency and mitigating metabolic disorders in poultry. This investigation not only bridges a critical knowledge gap in comparative physiology but also aligns with broader efforts to enhance sustainable agricultural practices through precision nutrition.

2 Materials and Methods

2.1 Animals

This investigation examined the hypothesized interplay between insulin and dopaminergic systems in modulating central feeding mechanisms. A cohort of 288 one-day-old broiler chicks (Ross-308; Morghak Company, Tehran, Iran) was sourced from a commercial hatchery. Following a 2-day acclimatization period in group pens, subjects were randomly assigned to individual cages within electrically heated rearing units. Environmental conditions were maintained at 32 ± 1°C, 40–50% relative humidity, and a 23-hour light/1-hour dark photoperiod, aligning with established avian husbandry protocols (Olanrewaju et al., 2006). Throughout the experimental period, chicks were provided ad libitum access to a standardized starter diet (21% crude protein, 2850 kcal/kg metabolizable energy). At 5 days post-hatch, intracerebroventricular (ICV) injections were administered following a 3-hour fasting period (FD3), during which water remained freely available. All husbandry practices and experimental interventions adhered to the ethical standards outlined in the National Institutes of Health (USA) Guide for the Care and Use of Laboratory Animals and approved by the Islamic Azad University Institutional Animal Care and Use Committee.

2.2 Experimental Compounds

The experimental pharmacological agents utilized in this study comprised insulin, L-DOPA (dopamine precursor), SCH23390 (a selective antagonist of D1 receptors), AMI-193 (a selective antagonist of D2 receptors); NGB 2904 (a selective antagonist of D3 receptors), L-741,742 (a selective antagonist of D4 receptors), and Evans blue dye. All compounds were procured from Sigma-Aldrich (Sigma, USA). Initially, each drug was dissolved in absolute dimethyl sulfoxide (DMSO) and then diluted with a 0.85% isotonic solution containing Evans blue dye at a 1:250 dilution ratio before administration in experiments. The dosages for the injectable agents were determined based on protocols established in prior research (22, 23).

2.3 Injection Procedure

During each experimental session, birds received a single ICV injection administered via a Hamilton microsyringe (Hamilton, Switzerland) without the use of anesthesia, consistent with previously established methodologies (24, 25). To stabilize the head, a custom-made acrylic apparatus was used, positioning the beak at a 45° angle and aligning the calvarium parallel to the table surface. A small aperture was created in a plate located directly above the right lateral ventricle of the skull. Through this opening, the needle of the microsyringe was carefully inserted approximately 4 mm beneath the skin to reach the ventricle. This technique has been demonstrated to reduce injection-related physiological stress in early post-hatch chickens (26). Each bird was administered a single 10 µL injection containing either a control solution or the experimental compound. In experiment 1, FD 3 chickens were ICV injected with (A) saline, (B) insulin (2.5 ng), (C) insulin (5 ng), and (D) insulin (10 ng). In experiment 2, ICV infusion of (A) saline, (B) L-DOPA (125 nmol), (C) insulin (10 ng), and (D) L-DOPA (125 nmol) + insulin (10 ng) was applied to the birds. In experiment 3, ICV injections included (A) saline, (B) SCH23390 (5 nmol), (C) insulin (10 ng), and (D) SCH23390 (5 nmol) + insulin (10 ng). In experiment 4, the birds received infusions of (A) saline, (B) AMI-193 (5 nmol), (C) insulin (10 ng), and (D) AMI-193 (5 nmol) + insulin (10 ng). In experiment 5, chickens were injected with (A) saline, (B) NGB2904 (6.4 nmol), (C) insulin (10 ng), and (D) NGB2904 (6.4 nmol) + insulin (10 ng). In experiment 6, ICV injections included (A) saline, (B) L-741,742 (6 nmol), (C) insulin (10 ng), and (D) L-741,742 (6 nmol) + insulin (10 ng). The classification of experimental groups and drugs injected into each group are presented in Table 1. Following the injection, the birds were promptly returned to their cages, where they had free access to pre-weighed food and water. Food intake was cumulatively measured at 30-, 60-, and 120-minute intervals after infusion. To adjust for variations in body size, food consumption data were normalized and expressed as a percentage of the bird's body weight (% BW). At the end of the experimental procedures, the accuracy of the infusion site was confirmed post-mortem by decapitating the birds and examining frozen brain sections for the presence of Evans blue dye within the lateral ventricle. Although 12 birds were injected per group, only those with verified dye localization in the lateral ventricle (typically between 9 and 12 per group) were included in the final dataset.

Table 1. The treatment protocols implemented in Experiments 1 through 6

Experiments	Groups			
	A	B	C	D
1	CS*	Insulin (2.5 ng)	Insulin (5 ng)	Insulin (10 ng)
2	CS	L-DOPA (125 nmol)	Insulin (10 ng)	L-DOPA + Insulin (125 nmol) + (10 ng)
3	CS	SCH23390 (5 nmol)	Insulin (10 ng)	SCH23390 + Insulin (5 nmol) + (10 ng)
4	CS	AMI-193 (5 nmol)	Insulin (10 ng)	AMI-193 + Insulin (5 nmol) + (10 ng)
5	CS	NGB2904 (6.4 nmol)	Insulin (10 ng)	NGB2904 + Insulin (6.4 nmol) + (10 ng)
6	CS	L-741,742 (6 nmol)	Insulin (10 ng)	L-741,742 + Insulin (6 nmol) + (10 ng)

CS: control solution (containing Evan's blue); L-DOPA: a dopamine precursor; SCH23390: a D1 receptor antagonist; AMI-193: a D2 receptor antagonist; NGB2904: a D3 receptor antagonist; L-741,742: a D4 receptor antagonist.

2.4 Statistical Analysis

Food consumption data, calculated as a % BW, were analyzed via a two-way repeated-measures ANOVA implemented in SPSS. Results are presented as Mean±SEM. In cases where ANOVA revealed statistically significant main effects, pairwise group comparisons were conducted using Tukey's post hoc test. A significance level of $p \leq 0.05$ was considered for all analyses.

3 Results

In experiment 1, the results reveal that insulin doses of 5 ng and 10 ng significantly reduced total food intake compared to the control group ($p < 0.05$). In contrast, the 2.5 ng dose did not produce a significant change ($p > 0.05$) (Figure 1). Additionally, a dose-dependent decline in cumulative meal consumption was observed following insulin treatment.

In Experiment 2, insulin administration (10 ng) significantly reduced food intake ($p < 0.05$), whereas L-DOPA alone did not alter food consumption significantly ($p > 0.05$). Moreover, co-administration of L-DOPA with insulin did not alter the anorexigenic effect of insulin ($p>0.05$) (Figure 2). These findings indicate that while insulin suppresses feeding, L-DOPA does not modulate this insulin-induced suppression.

In experiment 3, hypophagia was observed after ICV injection of insulin (10 ng) ($p < 0.05$), whereas SCH23390 alone at 5 nmol had no significant effect ($p > 0.05$). Importantly, co-administration of SCH23390 with insulin significantly lessened insulin's suppressive impact on feeding ($p<0.05$) (Figure 3), suggesting that dopamine D1 receptors partially mediate insulin's hypophagic action in broilers.

In experiment 4, ICV injection of insulin (10 ng) significantly decreased food intake ($p < 0.05$), whereas AMI-193 alone at 5 nmol did not affect meal consumption ($p > 0.05$). Co-infusion of AMI-193 with insulin did not alter insulin's suppression of feeding ($p > 0.05$) (Figure 4), indicating that dopamine D2 receptor blockade does not influence insulin's effect on food intake in this context.

In experiment 5, hypophagia was observed after ICV injection of insulin (10 ng) ($p < 0.05$), whereas NGB-2904 at 6.4 nmol alone did not affect feeding ($p > 0.05$). Combined treatment with NGB-2904 and insulin did not significantly modify the anorexic effect of insulin ($p>0.05$) (Figure 5), suggesting dopamine D3 receptors are not involved in insulin feeding suppression in broiler chickens.

In experiment 6, the administration of insulin (10 ng) significantly reduced food intake ($p < 0.05$), but L-741,742 at 6 nmol alone did not alter meal consumption ($p > 0.05$). Co-infusion of L-741,742 with insulin did not significantly affect insulin's anorexigenic action ($p>0.05$) (Figure 6), indicating that dopamine D4 receptor antagonism does not modify insulin-induced feeding suppression in broilers.

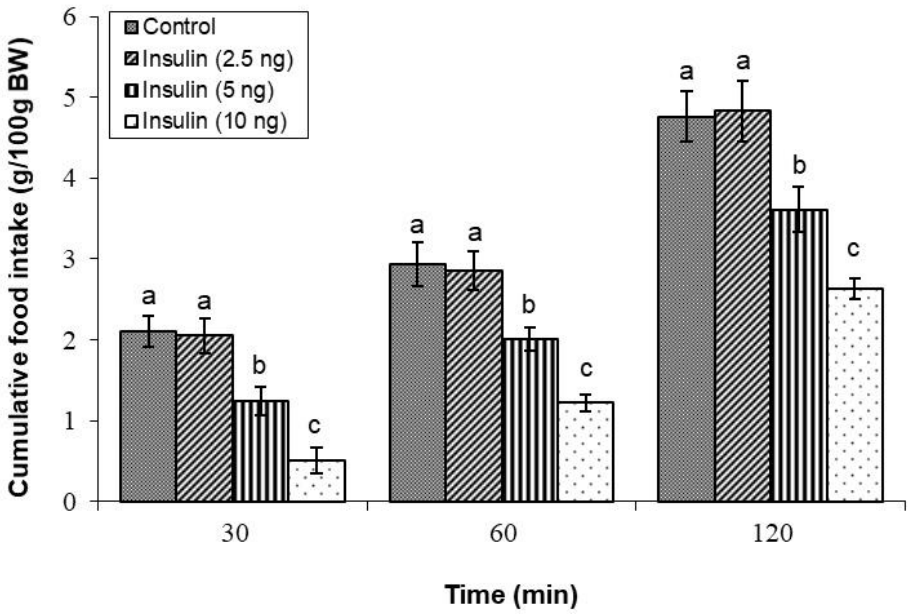

Figure 1. Effect of ICV infusion of insulin (2.5, 5 and 10 ng) on cumulative food intake in broilers (n=48). The results are presented as Mean±SEM. Statistically significant differences between treatment groups are indicated by heterogenous letters (a, b, and c) with a significance level of $p<0.05$.

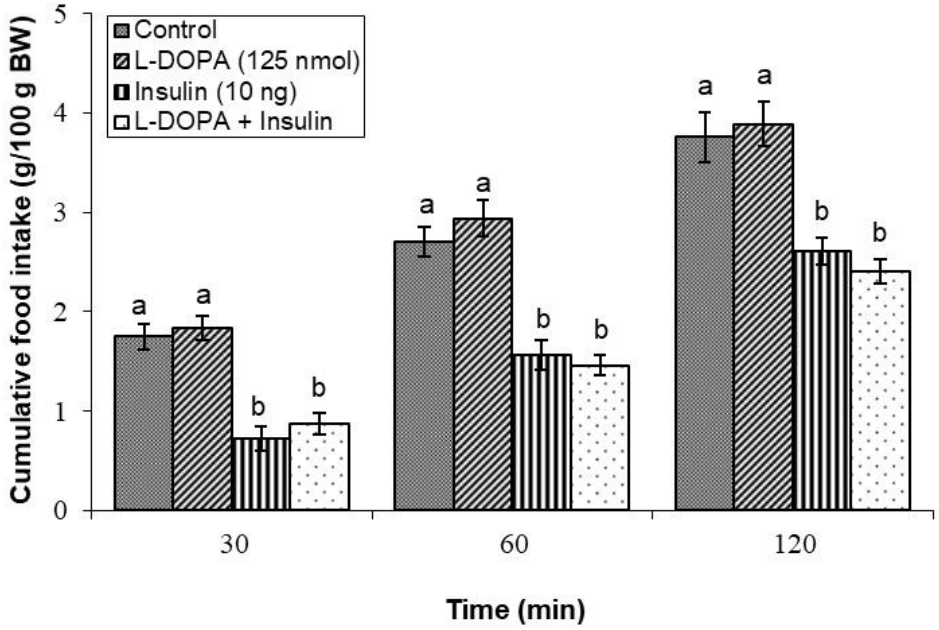

Figure 2. Effect of ICV infusion of L-DOPA (125 nmol), insulin (10 ng), and their combined treatment on cumulative food intake in broilers (n=48). The results are presented as Mean±SEM. Statistically significant differences between treatment groups are indicated by heterogenous letters (a and b) with a significance level of $p<0.05$.

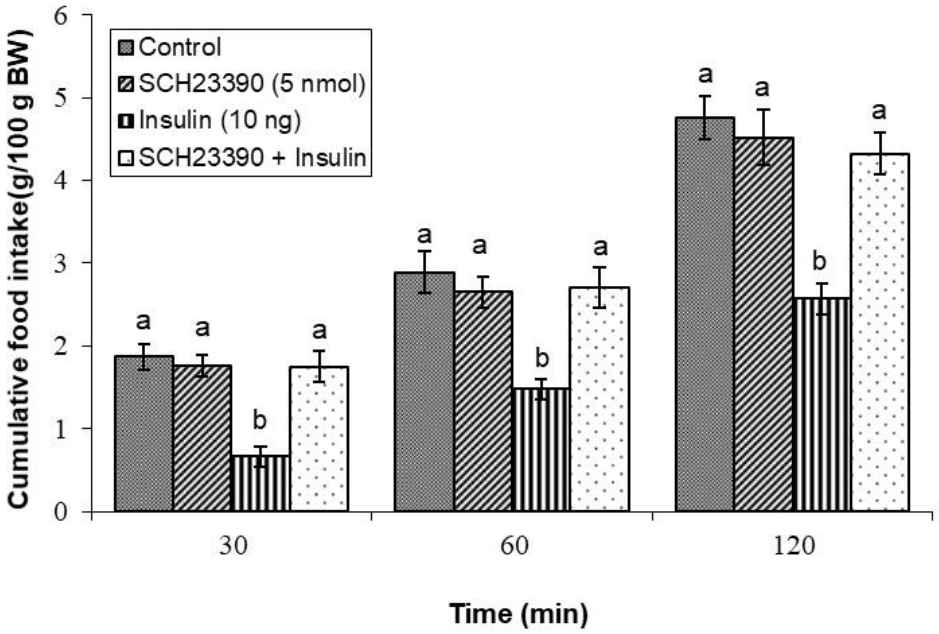

Figure 3. Effect of ICV infusion of SCH 23390 (5 nmol), insulin (10 ng), and their combined treatment on cumulative food intake in neonatal chicken (n=48). The results are presented as Mean±SEM. Statistically significant differences between treatment groups are indicated by heterogenous letters (a, b, and c) with a significance level of $p<0.05$.

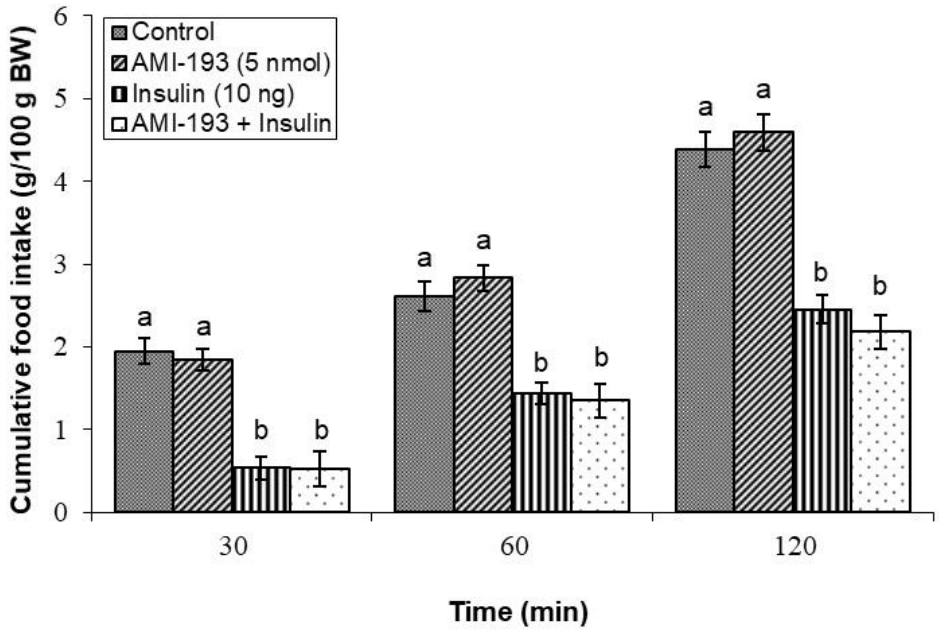

Figure 4. Effect of ICV infusion of AMI-193 (5 nmol), insulin (10 ng), and their combined treatment on cumulative food intake in broilers (n=48). The results are presented as Mean±SEM. Statistically significant differences between treatment groups are indicated by heterogenous letters (a and b) with a significance level of $p<0.05$.

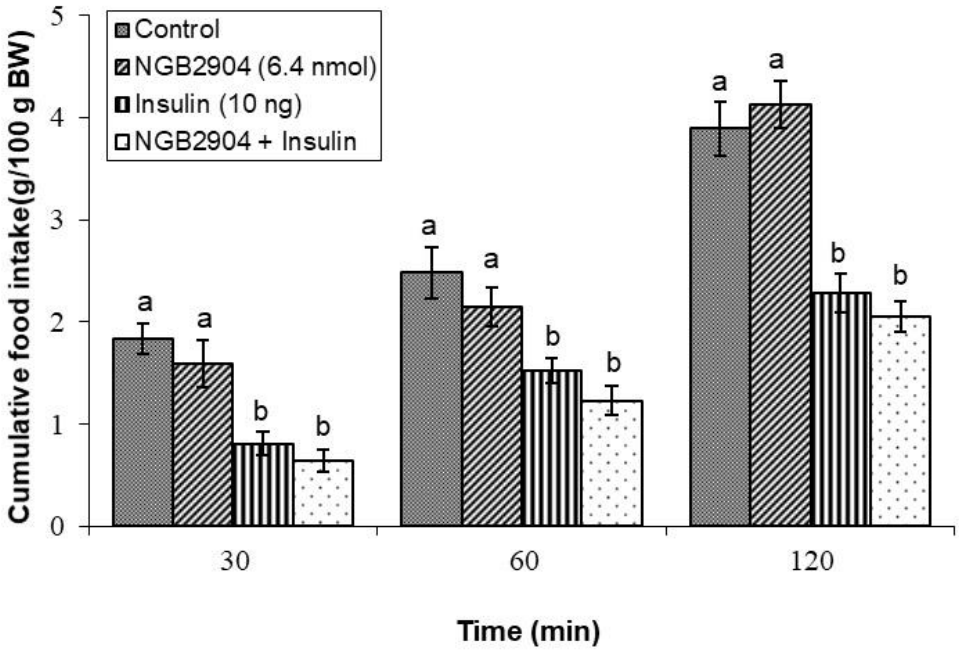

Figure 5. Effect of ICV infusion of NGB 2904 (6.4 nmol), insulin (10 ng), and their combined treatment on cumulative food intake in broilers (n=48). The results are presented as Mean±SEM. Statistically significant differences between treatment groups are indicated by heterogenous letters (a and b) with a significance level of $p<0.05$.

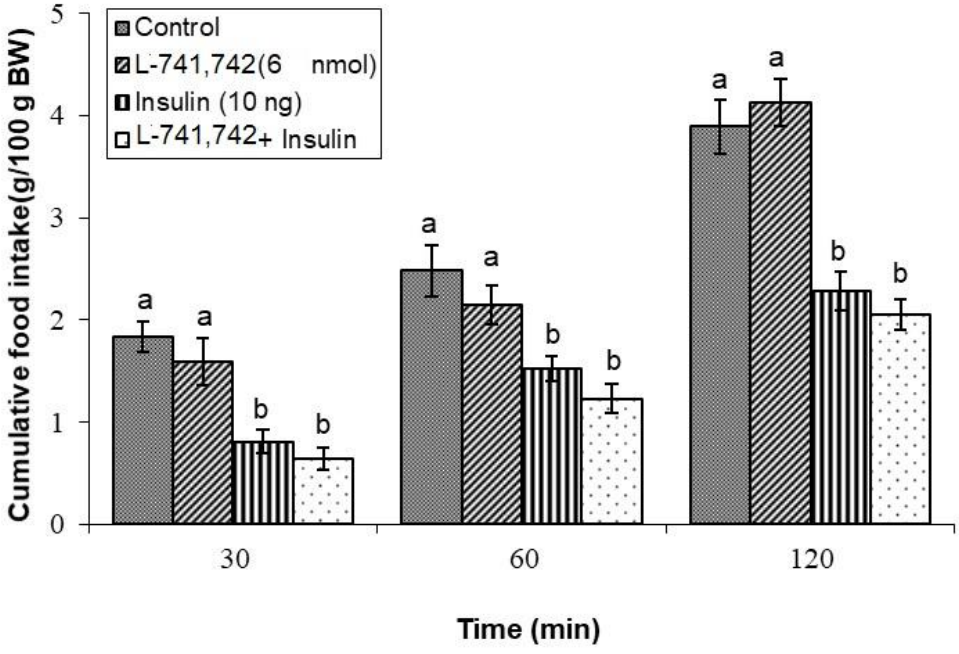

Figure 6. Effect of ICV infusion of L-741,742 (6 nmol), insulin (10 ng), and their combined treatment on cumulative food intake in broilers (n=48). The results are presented as Mean±SEM. Statistically significant differences between treatment groups are indicated by heterogenous letters (a and b) with a significance level of $p<0.05$.

4 Discussion & Conclusion

The current study corroborates previous findings that central infusion of insulin exerts a significant anorexigenic effect in Ross-308 broiler chickens. Consistent with earlier research in both avian and mammalian models, ICV infusion of insulin resulted in a marked reduction in meal consumption (9, 10). This supports the concept that insulin functions as a hypophagic neuropeptide, influencing feeding behavior through central nervous system pathways.

Insulin's anorexigenic action has been extensively documented in rodent models, where it modulates hypothalamic neurons, particularly those expressing POMC and NPY, which are key regulators of energy homeostasis (9). Similar mechanisms appear to operate in birds, as ICV insulin administration in layer chicks stimulates POMC expression and suppresses NPY expression, thereby reducing food intake (10). These neuropeptide-mediated pathways likely contribute to the central effects of insulin observed in broiler chickens. However, the literature also reveals strain-dependent variability in insulin's impact on feeding behavior. For instance, Shiraishi et al. (2011) reported that insulin did not notably affect meal consumption in Chunky broiler chickens, contrasting with the hypophagic response observed in Ross-308 broilers in the current study (27). This discrepancy may be attributed to genetic differences that influence insulin receptor sensitivity or the efficacy of downstream signaling within hypothalamic circuits. Such strain-specific responses underscore the complexity of neuroendocrine regulation in avian species and highlight the necessity of considering genetic background when interpreting insulin's central effects on feeding behavior.

At the intracellular level, insulin's metabolic and anorexigenic functions are largely mediated by the phosphoinositide 3-kinase (PI3K)/AKT signaling pathway. Inhibition of this pathway has been shown to abrogate most insulin-mediated effects, including regulation of glucose metabolism and appetite control (28). In avian models, administration of insulin antiserum suppresses hepatic AKT phosphorylation, indicating that insulin signaling in the liver complements its central actions in maintaining energy balance (29). These observations emphasize the integrated nature of insulin signaling across central and peripheral tissues in the regulation of feeding. Nutritional status and diet composition further modulate insulin's effects on feeding behavior. Studies in rodents demonstrate that insulin's anorexigenic impact is more pronounced in animals consuming high-fat diets compared to those on low-fat diets (30). Chronic administration of insulin analogs, such as detemir, has been shown to reduce meal consumption and adiposity in high-fat-fed rats, suggesting that insulin signaling adapts to metabolic conditions to regulate energy intake (31). Although such diet-dependent effects have not been extensively studied in poultry, they may contribute to variability in insulin responsiveness observed among different strains and feeding regimens.

The present study not only confirms the anorexigenic effects of centrally administered insulin on food intake in Ross-308 broiler chickens but also investigates the mediating role of dopaminergic receptors in these effects. Specifically, we demonstrate that co-administration of a D1 dopamine receptor antagonist significantly attenuates the hypophagic response induced by insulin, highlighting the critical involvement of dopaminergic signaling pathways in modulating insulin's central regulation of feeding behavior.

In both mammalian and avian species, feeding behavior is significantly controlled by the dopaminergic system. Previous research has shown that ICV infusion of dopamine and its precursor L-DOPA reduces meal consumption in broiler cockerels (23). Similarly, in rodent models, activation of dopamine receptors has been linked to dose-dependent hypophagia; ICV infusion of D1 and D2 receptor agonists decreases feeding, while blockade of these receptors alters feeding responses (32). Genetic studies further support this role, as D1 receptor knockout mice exhibit diminished operant responding for sucrose rewards, and D2 receptor-deficient mice show delayed acquisition of reward-based tasks, indicating impaired motivational feeding behavior (33).

Given the pivotal roles of insulin and dopamine in energy homeostasis, their interaction represents a crucial regulatory axis. Insulin has been shown to modulate the dopaminergic system through multiple molecular mechanisms. First, insulin regulates dopamine uptake by inducing the expression of the DAT, thereby influencing synaptic dopamine availability. Second, insulin modulates the function and half-life of dopamine by regulating dopamine-degrading enzymes, such as MAOs and DAT. Third, insulin affects the firing rates of both dopaminergic and cholinergic neurons, which are integral to reward and feeding circuits (19-21).

Moreover, diet-induced insulin resistance impairs dopamine synthesis by reducing the expression of tyrosine hydroxylase (TH), the enzyme that limits the rate of

dopamine synthesis (34). This suggests that insulin signaling is essential for maintaining dopaminergic tone in the brain. Supporting this, mice lacking insulin receptors, specifically on dopaminergic neurons, display increased body weight due to hyperphagia, highlighting the importance of insulin sensitivity within dopaminergic pathways for normal feeding regulation and weight control (21).

Administering insulin directly into the ventral tegmental area (VTA) of rats leads to a reduction in food-anticipatory behavior by suppressing excitatory synaptic inputs to dopamine-producing neurons; however, this inhibitory effect is weakened under conditions of elevated insulin levels (hyperinsulinemia) (35, 36). These data collectively indicate that insulin modulates dopaminergic neurotransmission to regulate feeding motivation and energy balance.

The results obtained from broiler chickens support this comprehensive framework, indicating that the appetite-suppressing effect of insulin is, to some extent, reliant on functional D1 receptor signaling. The attenuation of insulin-induced hypophagia by D1 receptor antagonist implies that dopamine receptor activation is necessary for the full expression of insulin's central effects on feeding. This interaction may represent a conserved neuroendocrine mechanism across vertebrates, linking metabolic signals with reward-related neural circuits to fine-tune energy intake and regulation.

In conclusion, the current investigation offers strong evidence that dopaminergic D1 receptors play a critical role in mediating insulin-induced reductions in meal consumption in broilers. These results advance our understanding of the neurochemical pathways underlying appetite regulation and highlight potential targets for modulating feeding behavior in poultry production.

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Conflict of Interest

The authors declare no competing interests.

Author Contributions

All authors equally contributed to this study.

Data Availability Statement

Data are available from the corresponding author upon reasonable request.

Ethical Considerations

This study was approved by the local ethics committee for animal experiments of Islamic Azad University, Garmsar Branch.

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