Poultry Science Journal
ISSN: 2345-6604 (Print), 2345-6566 (Online) http://psj.gau.ac.ir
DOI: 10.22069/psj.2022.19507.1730

The Interaction of Dopaminergic System and $GABA_B$ Receptor in Feed Intake Regulation of Neonatal Chicken

Mona Hashemzadeh[1], Morteza Zendehdel[2], Vahab Babapour[2] & Negar Panahi[1]

[1]Department of Basic Sciences, Faculty of Veterinary Medicine, Science and Research Branch, Islamic Azad University, Tehran, Iran
[2]Department of Basic Sciences, Faculty of Veterinary Medicine, University of Tehran, 14155-6453, Tehran, Iran

Poultry Science Journal 2022, 10(2): 139-148

Keywords
Birds
Dopamine
Feed intake
GABAergic system

Corresponding author
Morteza Zendehdel
zendedel@ut.ac.ir

Article history
Received: September 23, 2021
Revised: July 15, 2022
Accepted: July 15, 2022

Abstract

Animal studies have shown the role of gamma amino butyric acid (GABAergic) and Dopaminergic systems in controlling appetite, but their interactions in birds have not yet been investigated . In this study, 6 experiments were carried out to investigate the interactions between GABArergic and dopaminergic systems in fresh laying hens (Each experiment included 4 groups, 11 chicks per group). Chicks received intracerebroventricular (ICV) injections after 3h of starvation in the following form: In 6 experiments; Each experiment has 4 groups. Group 1 (CON): Received Saline as a control group. Group 2 (DOP): Dopamine neurotransmitters include 125 nmol L-DOPA (levo-dihydroxyphenylalanine) as a dopamine precursor. 6-OHDA, 2.5 nmol as a dopaminergic neurotoxin. SCH23390, 5nmol. AMI-193, 5nmol. NGB2904, 6.4nmol and L-741742, 6nmol. (L-DOPA, 6-OHDA, D1 receptor, D2 receptor, D3 receptor and D4 receptor antagonists), respectively (each of them in one of the experiments). Group 3 (GABA): Baclofen, 0.2µm ($GABA_B$ agonist). Group 4 (DOP+GABA): Receive Baclofen simultaneously with any of the L-DOPA, 6-OHDA, D1, D2, D3 and D4 antagonists, respectively (each of them in one experiments). Cumulative consumption of feed (based on the percentage of body weight) was measured up to 120 minutes after the injection. According to the results, ICV injection of Baclofen alone significantly increased feed intake ($P < 0.05$). None of the dopamine receptors (L-DOPA, 6-OHDA, D1, D2, D3, D4, as well as dopamine synthesis inhibitors) did not affect feed intake ($P > 0.05$). The co-injection of the L-DOPA, 6-OHDA, D1-D4 receptor antagonists + baclofen ($P> 0.05$) showed no significant effect. The results of this study showed that dopaminergic and $GABA_B$ receptors had no interaction in FD3 neonatal layer-type chicken.

Introduction

The physiological importance of the feed intake control system in poultry science is highlighted by the genetic selection of chickens to obtain greater production of meat and egg on the one hand, and layer hens should have lower weight compared to the meat-type chickens on the other hand (Berthoud, 2006; Berridge *et al*., 2006). The environmental and biological factors can contribute to appetite regulation, but neurochemical mediators recognized as neurotransmitters in various parts of the central nervous system (CNS) such as the striatum, hypothalamus, amygdala and arcuate nucleus (ARC) are robust to the regulation of feed behavior (Duke, 1986; Denbow, 1994; Denbow, 1985; Rahmani *et al.*, 2021). The arcuate nucleus (ARC) of the hypothalamus contains at least two distinct groups of neurons managing appetite and energy balance, that includes orexigenic and anorexigenic neuropeptides. The orexigenic Neuropeptide such as Neuropeptide Y (NPY) and Agouti-related protein (AgRP) and the anorexigenic Neuropeptide including Pro opiomelanocortine (Agmo *et al*., 1996; Anderberg *et al*., 2014). These neuropeptides from the ARC,

Please cite this article as Mona Hashemzadeh, Morteza Zendehdel, Vahab Babapour & Negar Panahi. 2022. The Interaction of Dopaminergic System and $GABA_B$ Receptor in Feed Intake Regulation of Neonatal Chicken. Poult. Sci. J. 10(2): 139-148.

project to neurons (second order) in the paraventricular nucleus (PVN), ventromedial hypothalamus area (VMH), and lateral hypothalamic area (LHA) to orchestrate feed intake (Wang *et al*., 2012). Neurochemical systems, including the gamma amino butyric acid (GABA), cholinergic, opioid, and glutamatergic systems have also been shown to regulate feeding behavior (Zendehdel & Hassanpour, 2014; Shojaei *et al*., 2020; Motaghi *et al*., 2021). In addition, Dopamine receptors are distributed within the substantia nigra, ventral tegmental area (VTA), nucleus accumbence, hypothalamus, and PVN which are involved in perception, excitement, and feed intake regulation. (Zendehdel *et al*., 2019; Shahri, 2020; Zendehdel *et al*., 2016; Zendehdel *et al*., 2014). Dopamine (DA) has at least five types of receptors called D1, D2, D3, D4, and D5. It has been demonstrated that the effect of D1 receptor in the brain on feed intake has evolutionarily increased in broiler-type hens, while other receptors (D2-D4) may play no role in the regulation of appetite (Beaulieu & Gainetdinov, 2011). For instance, feed intake was reduced by the receptors of D1 and D2 in rats (Carew *et al*., 2004).

GABA is a major inhibitory neurotransmitter and is widely distributed in the CNS of vertebrates (Sivilotti & Nistri, 1991). GABA has three types of receptors include GABA$_A$, GABA$_B$, and GABA$_C$. Accumulating evidence shows that energy balance and eating behavior can be regulated by the GABAergic system in a broiler (Denbow, 1991), layer (Zendehdel *et al*., 2017; Bungo *et al*., 2003) and turkey (Zendehdel & Hassanpour, 2014). Moreover, intracerebroventricular (ICV) injection of GABA$_B$ agonist one of three GABA receptors that is a metabotropic receptor belongs to G-protein coupled receptors (Jonaidi *et al*., 2012) could amplify cumulative feed intake in rat and layer-type hens but not in broilers (Zendehdel *et al*., 2009).

Neuroanatomical studies about the dopaminergic (DAergic) and GABAergic systems have revealed a potential link between these mediators (Møller *et al*., 2019; Lopes *et al*., 2019). Based on published evidence, GABA$_B$ receptors modulate the firing pattern of DA neurons in the substantia nigra (Lacey *et al*., 1988). Furthermore, the DA firing rate has been reduced by systemic administration of GABA$_B$ receptor agonist baclofen in VTA (Erhardt *et al*., 2002; Nimitvilai *et al*., 2012). Recently, Baik (2021) demonstrated that the dopaminergic control of feed intake in the hypothalamic arcuate nucleus was located in the same place vesicular GABA transporter and GABA and dopamine were released togather in this area in rodents. Based on the literature, no evidence was observed regarding the interaction between GABAergic and dopaminergic systems on the intake of feed in domestic fowls. Moreover, there is a difference between the broilers and the layers with respect to central regulatory factors of feed intake in the brain. For example, agouti-related protein could amplify the consumption of the cumulative feed in layers but not in broilers (Denbow, 1994). Likewise, the effects of GABA$_B$ receptors on feeding behavior in layer-type birds are stronger than those in broilers (Zendehdel & Hassanpour, 2014). Accordingly, the aim of this study was to investigate the effects of the central GABA$_B$ receptors and DAergic system on the regulation of feed intake in feed-deprived layer chicken (FD3) after ICV injection.

Materials and Methods

Animals

A total of 264 one-day-old male layer-type chickens (Hy-line) were bought from a local hatchery (Morghak co. Iran). All chickens were kept at a temperature of 30±1ºC with 50±2% humidity. Birds at 2 days of age were randomly transferred to individual cages. Chickens were then allowed free access to drinking fresh water and fed a starter diet formulated to contain 21% crude protein and 2850 kcal/kg metabolizable energy. On the 5th day, three hours before the injection, the chickens were feed deprived (FD3) but allowed free access to drinking water. Animal handling and experimental procedures were performed according to the Guide for the Care and Use of Laboratory Animals by the National Institutes of Health (USA) and approved by the research committee, Faculty of Veterinary Medicine, Science and Research Branch, Islamic Azad University, Tehran, Iran (IAUSRB:2021.03.15).

Experimental drugs

L-DOPA (levo-dihydroxyphenylalanine as the precursor of dopamine), 6-OHDA (6-hydroxydopamine as dopaminergic neurotoxin), SCH23390 (D1 receptor antagonist), AMI-193 (D2 receptor antagonist), NGB2904 (D3 receptor antagonist), L-741,742 (D4 receptor antagonist), Baclofen (GABA$_B$ receptor agonist) and Evans blue were obtained from Sigma (US company and Tocris (UK company). Medicines were first dissolved in absolute DMSO (Dimethyl sulfoxide) and then diluted using 0.85% saline containing Evans blue at a ratio of 1:250. According to previous studies, DMSO with this ratio did not show a cytotoxic effect (Blevins *et al*., 2002; Qi *et al*., 2008). DMSO mixed with saline was served as a control solution for all experiments.

Intracerebroventricular injection procedure

Prior to the start of the study, the chickens were weighed and distributed among treatment groups based on their body weight so the mean body weight between treatment groups was as uniform as possible. To investigate the effects of GABA$_B$ receptors and

dopaminergic system on the central control of feed intake in layer chicken, six experiments were designed, each including four treatment groups within 11 replicates per group (n = 44 chickens per experiment). In each experiment, chickens received ICV injection once using a microsyringe (Hamilton, Switzerland) without anesthesia according to the techniques of Davis *et al*. (1979) and Furuse *et al*. (1997). In this method, the head of the birds was held using an acrylic device while the bill holder was at a 45° position and the calvarium was held as parallel to the surface of the table as possible (Van Tienhoven & Juhasz, 1962). An orifice was made in a plate where the skull over the right lateral ventricle was directly overlaid by this plate. Finally, the microsyringe was placed in the ventricle through this hole and the needle tip penetrated 4 mm below the skin of the skull (Jonaidi & Noori, 2012). It is well documented that no injection-induced physiological stress is observed in neonatal chicks when using this method (Saito *et al*., 2005). The volume of each ICV injection of vehicle or drug solution was 10 µL/chick. Right away after each injection of fD3, chickens were returned to their cages and received fresh feed and water (pre-weighted). Cumulative intake of feed (gr) was recorded 30, 60, and 120 min following the injection. Consumption of feed was computed as a percentage of body weight to reduce the effect of the body weight on the volume of feed intake. At the end of the experiments, to assess the injection accuracy, the birds were sacrificed by decapitation and the placement accuracy of the injection in the lateral ventricle was verified via the presence of Evans blue followed by slicing of the frozen brain tissue. In each group, 11 chickens received an injection, but just data of the birds were utilized for analysis where the dye was found in their lateral ventricle (11 birds per group). All experiments were performed from 8:00 A.M. until 3:30 P.M.

In addition, in each experiment, we had four groups with 11 chickens in each. On day 5 of age, when the chicken was transferred to the individual cages, off feed procedure started at 8 A.M. 1st chicken was deprived of feed at 8:00 A.M, then 2nd chicken at 8:03 was deprived of feed, and so on 3rd chicken at 8.06, etc. Then in next three hours at 11 o'clock, chicken one, which was off feed for 3 hours, received the injection, and feed was provided for it. At 11.03, 2nd chicken and at 11.06 3rd chick and so on were injected and had feed. At 11:30, feed intake for chicken one was measured, 11.33 for chicken 2, and 11.36 for chicken three, etc. In each experiment, there were 44 chickens, so it was a highly complex procedure since after 60 min (at 12 o clock), chicken 1 was on 60 min post-injection while another chick was at 30 min post-injection. Bypassing the time, it got more complicated. At 1:00 pm, the feed intake for chicken 1 finished (after 120 min), at 1.03 for 2nd chick, 1.06 for 3rd chick, and so on. So, this timetable seems confusing, but all procedures have been done with very high seriousness. Also, each experiment was done one after another. Also, each bird was just used once in this study. Moreover, for the sake of clarity, it should be mentioned that the experiments were done on different days. Thus, the statement "All experimental procedures were done from 8:00 A.M. until 3:30 P.M" was replaced with "Each experiment was performed on chickens from 08:00 A.M. to 1:30 P.M. on the designated day for that experiment.

Feeding experiments

In this study, a total of 6 experiments were designed for 5 days old chicken, each with 4 treatment groups (n=48 per experiment). In experiment 1, four groups of FD3 birds received a either of the following Intracerebroventricular injections(A) control solution, (B) L-DOPA (DA precursor, 125 nmol), (C) Baclofen ($GABA_B$ receptor agonist, 0.2 µg), and (D) L-DOPA (125 nmol) + Baclofen (0.2 µg) spontaneously. In experiment 2, they received a dose of the ICV administration of (A) control solution, (B) 6-OHDA (as a dopaminergic neurotoxin, 2.5 nmol) (C) Baclofen (0.2 µg), and (D) 6-OHDA (2.5 nmol) + Baclofen (0.2 µg) spontaneously. In experiment 3, they received a dose of the ICV administration of (A) control solution, (B) SCH23390 (D1 receptor antagonist, 5 nmol) (C) Baclofen (0.2 µg) or (D) SCH23390 (5 nmol) + Baclofen (0.2 µg) spontaneously. In experiment 4, they received a dose of the ICV administration of (A) control solution, (B) AMI-193 (D2 receptor antagonist, 5 nmol) (C) Baclofen (0.2 µg) and (D) AMI-193 (5 nmol) + Baclofen (0.2 µg) spontaneously. In experiment 5, they received a dose of the ICV administration of (A) control solution, (B) NGB2904 (D3 receptor antagonist, 6.4 nmol), (C) Baclofen (0.2 µg) and (D) NGB2904 (6.4 nmol) + Baclofen (0.2 µg) spontaneously. In experiment 6, they received a dose of the ICV administration of (A) control solution, (B) L-741,742 (D4 receptor antagonist, 6 nmol), (C) Baclofen (0.2 µg) and (D) L-741,742 (6 nmol) + Baclofen (0.2 µg) spontaneously. Each experiment has done one after another. Each bird was injected once only. These doses of drugs were calculated based on previous studies (Zendehdel *et al*., 2017; Zendehdel *et al*., 2016; Jonaidi *et al*., 2002; Tajalli *et al*., 2006; Zendehdel *et al*., 2014) and our pilot experiments (unpublished data).

Statistical analysis

The data were expressed as mean ± *SEM* (standard error of the mean). a two-way repeated-measures (ANOVA) was used to assess cumulative intake of feed (as a percentage of body weight) SPSS 16.0 for Windows (SPSS, Inc., Chicago, IL, USA) was used for the data analysis. Tukey-Kramer test was used to

compare the means of any treatment showing the main effect by ANOVA. $P < 0.05$ was considered statistically significant.

Results

Figures 1. shows the effects of central GABA$_B$ receptors and DAergic system on cumulative intake of feed in FD3 neonatal layer-type chicken. ICV injection of the sub-effective dose of the L-DOPA (125 nmol) did not show any impact on the intake of feed ($P > 0.05$), while the effective dose of Baclofen (0.2 µg) considerably improved the intake of feed in the treatment groups as compared with controls ($P < 0.05$). Co-injection of the L-DOPA + Baclofen did not show any impact on feed intake in FD3 neonatal layer-type chickens as compared Baclofen group ($P>0.05$).

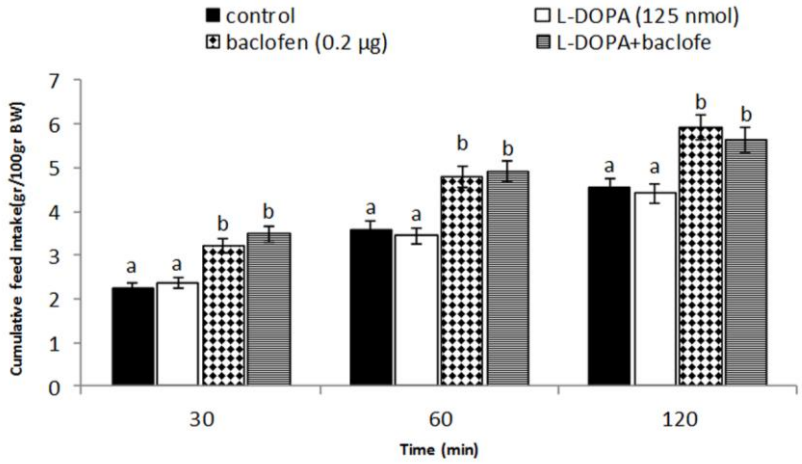

Figure 1. Impact of ICV administration of L-DOPA, Baclofen and their combination on cumulative intake of feed (g/100g BW) in neonatal layer-type chickens. LDOPA: dopamine precursor, Baclofen: GABA$_B$ receptor agonist. The data are presented as mean±*SEM*. Different letters (a and b) represent the statistically significant differences between treatment groups ($P<0.05$).

In experiment 2 (figure 2), ICV administration of the sub-effective dose of the 6-OHDA (2.5 nmol) did not have a substantial effect on feeding behavior ($P>$ 0.05) while hyperphagia was observed by the injection of the Baclofen (0.2 µg) compared to controls ($P<0.05$). Co-administration of the 6-OHDA + Baclofen considerably enhanced GABA$_B$ receptors agonist-induced feed intake in FD3 neonatal layer-type chicken ($P<0.05$) as compared with controls.

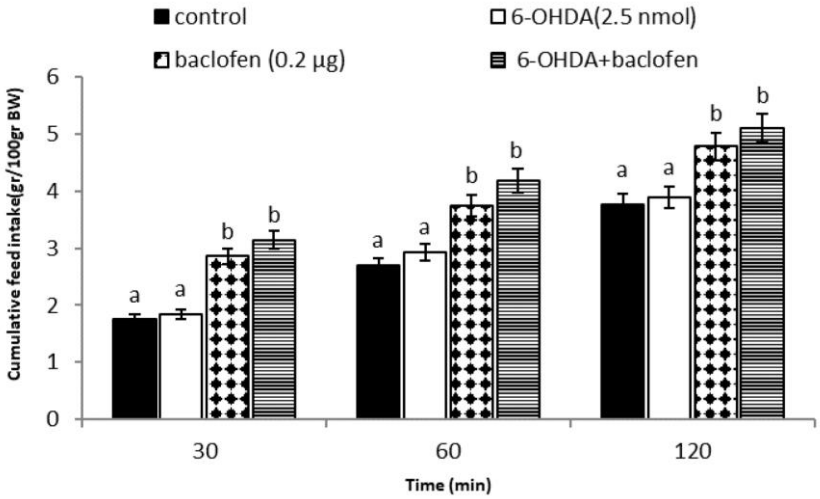

Figure 2. Impact of ICV administration of 6-OHDA, Baclofen and their combination on cumulative intake of feed (g/100g BW) in neonatal layer-type birds. 6-OHDA: a dopaminergic neurotoxin, Baclofen: GABA$_B$ receptor agonist. Data are presented as mean ± *SEM*. Different letters (a-b) denote the statistically significant differences between treatment groups ($P < 0.05$)

The results obtained from experiment 3 (figure 3) demonstrated that no substantial effect was found by ICV administration of the sub-effective dose of the SCH23390 (5 nmol) compared to controls ($P > 0.05$). ICV administration of the Baclofen (0.2 µg) enhanced feed intake in FD3 neonatal layer-type chickens as compared with controls ($P < 0.05$). Also, co-injection of the SCH23390 + Baclofen dramatically induced hyperphagia in FD3 neonatal layer-type chickens as compared with controls ($P < 0.05$).

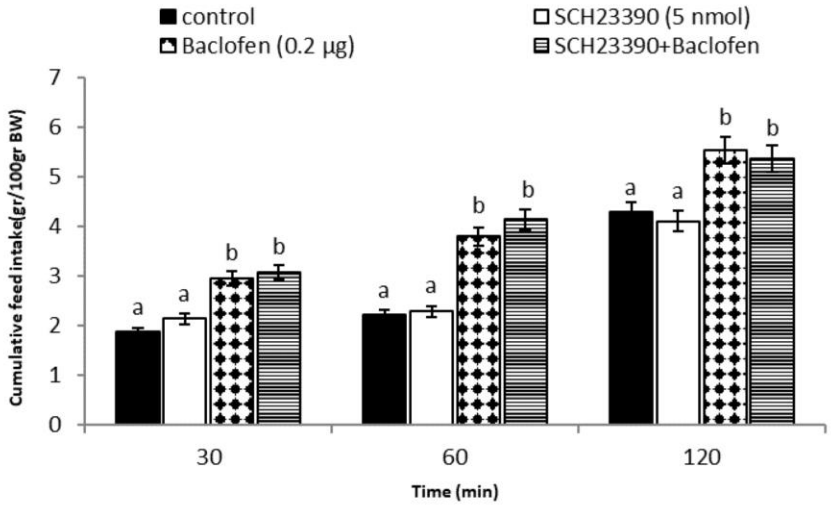

Figure 3. Impact of ICV administration of SCH23390, Baclofen and their combination on cumulative intake of feed (g/100g BW) in neonatal layer type birds. SCH23390: D1 receptor antagonist, Baclofen: $GABA_B$ receptor agonist. The data are presented as mean ± *SEM*. Different letters (a-b) represent the statistically significant differences between treatment groups ($P < 0.05$)

In experiment 4 (figure 4), ICV injection of a sub-effective dose of the AMI-193 (5 nmol) did not show any impact on feeding behavior ($P > 0.05$), while Baclofen (0.2 µg) dramatically enhanced intake of feed as compared with controls ($P < 0.05$). Co-injection of the AMI-193 + Baclofen did not show any impact on feed intake in FD3 neonatal layer-type chickens compared to the Baclofen group ($P > 0.05$).

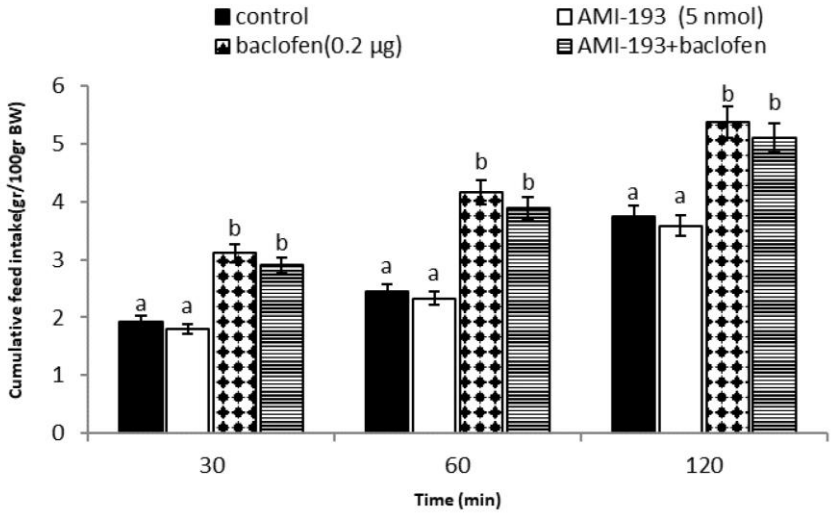

Figure 4. Effect of ICV administration of AMI-193, Baclofen and their combination on cumulative intake of feed (g/100g BW) in neonatal layer–type birds. AMI- 193: D2 receptor antagonist, Baclofen: $GABA_B$ receptor agonist. The data are presented as mean ± *SEM*. Different letters (a and b) denote the statistically significant differences between treatment groups ($P < 0.05$).

As shown in Figure 5, ICV administration of NGB2904 (6.4 nmol) did not show any impact on the consumption of feed compared to controls ($P > 0.05$), while hyperphagia was found by ICV

administration of the Baclofen (0.2 µg) ($P < 0.05$). Co-administration of the NGB2904 + Baclofen did not have any impact on the cumulative intake of feed induced by Baclofen in FD3 neonatal layer-type birds ($P > 0.05$).

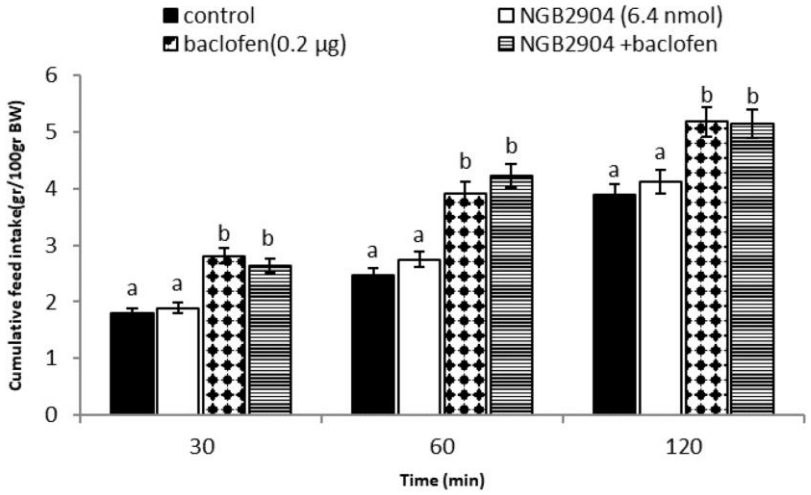

Figure 5. Impact of ICV administration of NGB2904, Baclofen and their combination on cumulative intake of feed (g/100g BW) in neonatal layer-type chickens. NGB2904: D3 receptor antagonist, Baclofen: GABA$_B$ receptor agonist. The data are presented as mean ± *SEM*. Different letters (a and b) show the statistically significant differences between treatment groups ($P < 0.05$).

In experiment 6, ICV administration of a sub-effective dose of the L-741,742 (6 nmol) did not have any impact on feed consumption compared to controls ($P > 0.05$). No significant effect on feed intake was found by a combination of the L-741,742 +Baclofen (0.2 µg) in FD3 neonatal layer-type chickens as compared with controls ($P > 0.05$) (figure 6).

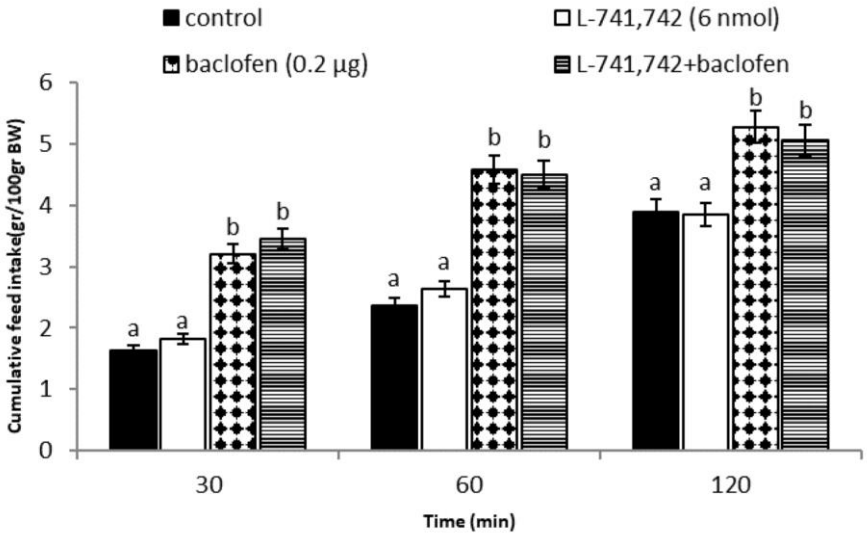

Figure 6. Impact of ICV administration of L-741,742, Baclofen and their combination on cumulative intake of feed (g/100g BW) in neonatal layer-type chickens. L-741,742: D4 receptor antagonist, Baclofen: GABA$_B$ receptor agonist. The data are presented as mean ± *SEM*. Various letters (a and b) represent the statistically significant differences between treatment groups ($P < 0.05$).

Discussion

This study was designed to investigate the probable effects of GABA$_B$ receptors and DAergic systems on feedintake regulation in neonatal layer-type chickens. So, we used a sub-effective dose of dopamine to achieve the real effect of GABA$_B$ receptors on dopamine receptors in little layer birds. Obtained data in experiments revealed that ICV injection of

Baclofen ($GABA_B$ receptors agonists) solely increased, while the sub-effective dose of dopaminergic receptors was ineffective in cumulative feed intake in FD3 neonatal chicks. It has been shown that $GABA_B$ receptors are neuronal regulators of feed intake in rats (Chen *et al*., 2015), turkeys (Denbow, 1991), sheep and pigs (Denbow *et al*., 1983). However, it is also clear that significant functional differences are observed between the meat-type and layer-type chickens in terms of the regulation of feed intake (Alizadeh *et al*., 2015). For example, ICV administration of Baclofen did not show any impact on the intake of feed in broilers (Jonaidi *et al*., 2002; Tajalli *et al*., 2006; Zendehdel *et al*., 2009). It is proposed that GABA receptors act in several mechanisms in a feeding system. In fact, it may inhibit the ventromedial hypothalamus (VMH) as satiety center. GABA may act through loss of inhibition caused by feeding centers of the brain using the inhibitory GABAergic projection from the medial accumbens to the lateral hypothalamic area (LHA). Finally, direct excitatory effect of GABA can have an orexigenic impact on some areas of the brain like the hypothalamus (Jonaidi *et al*., 2002; Zendehdel *et al*., 2017). There are interesting evidence suggesting that D1 receptor stimulation may promote satiety in rats (Terry *et al*., 1995). D1 receptors are considered anorexigenic factors, while D3 and D4 receptors did not affect broiler cockerels (Zendehdel & Hassanpour, 2014; Emadi *et al*, 2021) and central administration of DA showed no effect on Leghorn and Turkey chicks (Denbow *et al*., 1983). However, the results of this study showed that ICV injection of DA receptors (D1, D2, D3, D4, L-DOPA, and 6-OHDA) in a dose-dependent manner did not modify feed intake in FD3 neonatal layer-type chicks (Figures 1, 2). Nevertheless, DA modulates the release of lots of neurotransmitters in appetite control in various regions of the central nervous system. A previous study examined an available interaction between D1 dopaminergic and $GABA_A$ receptors regarding the feed intake regulation of neonatal layers (Hashemzadeh *et al*., 2018). It has been identified, that cannabinoids may alter DA transmission via long-term mediated synaptic depression of GABAergic neurons in the ventral tegmental area, substantia nigra, basal ganglia, and Striatal projection (Fernández-Ruiz *et al*., 2010). However, the GABA agonist, muscimol, led to dopamine-dependent increases in locomotion, aggression, and the intake of feed in the VTA (Scheel-Krüger *et al*., 1980). GABAergic generation of intense appetitive eating behavior was the independence of modulatory dopamine signals within the nucleus acumbance of rats (Richard *et al*., 2013). In agreement with our study, Bungo *et al*. (2010) reported that ICV injection of dopaminergic receptors had no effect on feed intake in layer-type chicken, but suggesting that dopamine might have interaction with other neurotransmitters in the central regulation of appetite in layer-type birds (Bungo *et al*., 2010). The results of the present study indicated that $GABA_B$ induced hyperphagia, but there was no modulation by the DAergic system in layer-type chicks.

However, humoral and neural pathways of highly integrated regulatory mechanisms exist for the regulation of appetite involving complex interplay between peripheral tissues and the central nervous system. Within the CNS, the hypothalamus and neurotransmitters can contribute to both appetite regulation and energy balance (Richards *et al*., 2010). This study examined the effects of the interaction of the dopaminergic system and $GABA_B$ receptors on the regulation of feed intake in layer–type chickens. A better understanding of the neural circuits related to monitoring feed intake and energy balance and how their expression can be regulated by both nutritional and hormonal stimuli can provide new insights into current layer breeding and management practices. However, further investigation is required to elucidate the underlying cellular and molecular signaling pathways in the interconnection between GABAergic and DAergic systems on feeding behavior in neonatal layer chicks.

Acknowledgements

The authors thank Dr. Shahin Hassanpour for his co-operation during the research as well as for authorship and revision of the current manuscript.

References

Agmo A, Belzung C & Giordano M. 1996. Interactions between dopamine and GABA in the control of ambulatory activity. Journal of Neural Transmission, 193: 025-034. DOI: 10.1007/ BF01291783

Alizadeh A, Zendehdel M, Babapour V, Charkhkar S & Hassanpour S. 2015. Role of cannabinoidergic system on feed intake in neonatal layer-type chicken. Veterinary Research Communications, 39: 151–157. DOI: 10.1007/s11259-015-9636-3

Anderberg RH, Anefors C, Bergquist F, Nissbrandt H & Skibicka KP. 2014. Dopamine signaling in the amygdala, increased by feed ingestion and GLP-1, regulates feeding behavior. Physiology & Behavior, 136: 135-144. DOI: 10.1016/ j.physbeh.2014.02.026

Baik JH. 2021. Dopaminergic control of the feeding circuit. Endocrinology and Metabolism, 36: 229-239. DOI: 10.3803/EnM.2021.979

Beaulieu JM & Gainetdinov R. 2011. The physiology, signaling, and pharmacology of

dopamine receptors. Pharmacological Reviews, 63(1): 182-217. DOI: 10.1124/pr.110.002642

Berridge CW, Stratford TL, Foote SL & Kelley AE. 2006. Distribution of dopamine β-hydroxylase-like immunoreactive fibers within the shell subregion of the nucleus accumbens. Synapse, 26(3): 239-241. DOI: 10.1002(SICI)10 98-2396(199711)27:3<230::AID -SYN8>3.0.CO; 2-E

Berthoud HR. 2006. Homeostatic and non-homeostatic pathways involved in the control of feed intake and energy balance. Nutrition & Metabolism, 14(5): 106S-299S. DOI: 10.1038/ oby.2006.308

Blevins JE, Stanley BG & Reidelberger RD. 2002. DMSO as a vehicle for central injections: tests with feeding elicited by norepinephrine injected into the paraventricular nucleus. Biochemistry and Behavior, 71: 277-282. DOI: 10.1016/s0091-3057 (01)00659-1

Bungo T, Izumi T, Kawamura K, Takagi T, Ueda H & Furuse M. 2003. Intracerebroventricular injection of muscimol, baclofen or nipecotic acid stimulates feed intake in layer-type, but not meat-type, chicks. Brain Research, 993 (1-2): 235-238. DOI: 10.1016/j.brainres.2003.09.017

Bungo T, Yanagita K & Shiraishi J. 2010. Feed intake after infusion of noradrenalin, dopamine or its precursor into the lateral ventricles in neonatal chicks. Journal of Animal and Veterinary Advances, 9(4): 660-663. DOI: 10.3923/javaa. 2010.760.763

Carew LB, Evarts KG & Alster FA. 2004. Growth, feed intake, and plasma thyroid hormone levels in chicks fed dietary excesses of essential amino acids. Poultry Science, 6: 204 205. DOI: 10.1093/ ps/77.2.295

Chen Z, Xie J, Hu MY, Tang J, Shao ZF & Li MH. 2015. Protective effects of γ -aminobutyric acid (GABA) on the small intestinal mucosa in heat-stressed wenchang chicken. Journal of Animal & Plant Sciences, 25(1): 78-87.

Davis JL, Masuoka DT, Gerbrandt LK & Cherkin A. 1979. Autoradiographic distribution of Lproline in chicks after intracerebral injection. Physiology & Behavior, 22:693-695. DOI: 10.1016/0031-9384(79) 90233-6

Denbow DM. 1985. Feed intake control in birds. Neuroscience & Biobehavioral Reviews, 9(2): 223-32. DOI: 10.1016/0149-7634(85)90047-8

Denbow DM. 1991. Induction of feed intake by a GABAergic mechanism in the turkey. Physiology & behavior, 49(3): 485-488. DOI: 10.1016/0031-9384(91)90268-s

Denbow DM. 1994. Peripheral regulation of feed intake in poultry. conference: feed intake regulation: neuropeptides, circulating factors and genetics. The Journal of nutrition, 124 (8): 1349s-1354s. DOI: 10.1093/jn/124.suppl_8.1349S

Denbow DM, Van Krey HP, Lacy MP & Dietrick TJ. 1983. Feeding, drinking and temperature of leghorn chicks: effects of ICV injections of biogenic amine. Physiology & Behavior, 31: 85-90. DOI: 10.1016/0031-9384(83)90100-2

Duke GE. 1986. Alimentary canal: anatomy, regulation of feeding, and motility. In: Avian physiology. Sturkie PD. Academic Press. Pages, 130-166. DOI: 10.1016/S1055-937X(99)80036-X

Emadi L, Jonaidi H, Nazifi S, Khasti H, Rohani E & Kaiya H. 2021. The effects of central ghrelin on serum parameters related to energy metabolism in neonatal chicks. Iranian Journal of Veterinary Medicine, 16(2): 110-118. DOI: 10.22059/ ijvm. 2021.325585.1005182

Erhardt S, Mathé JM, Chergui K, Engberg G & Svensson TH. 2002. GABAB receptor-mediated modulation of the firing pattern of ventral tegmental area dopamine neurons in vivo. Archives of Pharmacology, 365:173–180. DOI: 10.2174/157015906775203020

Fernández-Ruiz J, Hernández M & Ramos JA. 2010. Cannabinoid–dopamine interaction in the pathophysiology and treatment of CNS disorders. CNS Neuroscience & Therapeutics, 16(3): e72-e91. DOI: 10.1111/j.1755-5949.2010.00144.x

Furuse M, Matsumoto M, Saito N, Sugahara K & Hasegawa S. 1997. The central corticotropin-releasing factor and glucagon-like peptide-1 in feed intake of the neonatal chick. European Journal of Pharmacology, 339: 211-214. DOI: 10.1111/asj.12619

Hashemzadeh M, Zendehdel M, Babapour V & Panahi N. 2018. Interaction between central GABAA receptor and Dopaminergic system on feed intake in neonatal chicks: role of D1 and GABAA receptors. International Journal of Neuroscience, 128(4):361-368. DOI: 10.1080/ 00207454.2017.1383908

Jonaidi H, Abbassi L, Yaghoobi MM, Denbow DM, Kamali Y & Shojaei B. 2012. The role of GABAergic system on the inhibitory effect of ghrelin on feed intake in neonatal chicks. Neuroscience Letters, 520:82-86. DOI: 10.1016 /j.neulet.2012.05.036

Jonaidi H, Babapour V & Denbow DM. 2002. GABAergic control of feed intake in the meat type chickens. Physiology & Behavior, 76: 465-468. DOI: 10.1016/S0031-9384(02)00692-3

Jonaidi H & Noori Z. 2012. Neuropeptide Y-induced feeding is dependent on GABAA receptors in neonatal chicks. Journal of Comparative Physiology A, 198:827–832. DOI: 10.1007/ s00359-012-0753-y

Lacey MG, Mercuri NB & North RA. 1988. On the potassium conductance increase activated by

GABAB and dopamine D2 receptors in rat substantia nigra neurones. The Journal of Physiology, 401(1): 437-453. DOI: 10.1113/jphysiol.1988.sp017171

Lopes EF, Roberts BM, Siddorn RE, Clements MA & Cragg SJ. 2019. Inhibition of nigrostriatal dopamine release by striatal GABAA and GABAB receptors. The Journal of Neuroscience, 39(6):1058–1065. DOI: 10.1523/JNEUROSCI.2028-18

Møller A, Thomsen KT, Brooks1 DJ, Mouridsen K, Blicher JU, Hansen KV & Lou HC. 2019. Attenuation of dopamine-induced GABA release in problem Gamblers. Brain and Behavior. 1-7. DOI: 10.1002/brb3.1239

Motaghi M, Jonaidi H, Bashiri B & Noori Gooshki S. 2021. Purinergic regulation of feed and fat intakes in broiler's central nervous system. Iranian Journal of Veterinary Medicine, 15(4): 404-410. DOI: 10.22059/ijvm.2021.309901.1005127

Nimitvilai S, Arora SD, McElvain AM & Brodie SM. 2012. Reversal of inhibition of putative dopaminergic neurons of the ventral tegmental area: Interaction of GABAB and D2 receptors. Neuroscience, 226: 29–39. DOI: 10.1016/j.neuroscience.2012.08.045

Qi W, Ding D & Salvi RJ. 2008. Cytotoxic effects of dimethyl sulphoxide (DMSO) on cochlear organotypic cultures. Hearing Research, 236: 52-60. DOI: 10.1016/j.heares.2007.12.002

Rahmani B, Ghashghayi E, Zendehdel, M, khodadi M & Hamidi B. 2021. The crosstalk between brain mediators regulating feed intake behavior in birds: A Review. International Journal of Peptide Research and Therapeutics, 27: 2349–2370. DOI: 10.1007/s10989-021-10257-1

Richard JM, Plawecki AM & Berridge KC. 2013. Nucleus accumbens GABAergic inhibition generates intense eating and fear that resists environmental retuning and needs no local dopamine. European Journal of Neuroscience, 37(11):1789-802. DOI: 10.1111/ejn.12194

Richards MP, Rosebrough RW, Coon CN & McMurtry JP. 2010. Feed intake regulation for the female broiler breeder: In theory and in practice. Journal of Applied Poultry Research, 19: 182–193. DOI: 10.3382/japr.2010-00167

Saito ES, Kaiya H, Tachibana T, Tomonaga S, Denbow DM, Kangawa K, Furuse M. 2005. Inhibitory effect of ghrelin on feed intake is mediated by the corticotropin-releasing factor system in neonatal chicks. Regulatory Peptides, 125: 201-208. DOI: 10.1016/j.regpep.2004.09.003

Scheel-Krüger J, Arnt J, Magelund G, Olianas M, Przewlocka B & Christensen AV. 1980. Behavioural functions of GABA in basal ganglia and limbic system. Brain Research Bulletin, 5(2): 261-267. DOI: 10.1016/0361-9230(80)90043-X

Shahri Sh. 2020. The effects of dopamine and glutamate agonists on brain histology and feed intake of quails exposed to environmental heat stress. Journal of Infertility and Reproductive Biology, 8(3):57-60. DOI: 10.47277/JIRB/8(3)/57

Shojaei M, Yousefi A, Zendehdel M & Khodadadi M. 2020. Feed intake regulation in birds: the role of neurotransmitters and hormones. Iranian Journal of Veterinary Medicine, 14(1): 100-115. DOI: 10.22059/IJVM.2019.285059.1005006

Sivilotti L & Nistri A. 1991. GABA receptor mechanisms in the central nervous system. Progress in Neurobiology, 36(1): 35–92. DOI: 10.1016/0301-0082(91)90036-z

Tajalli S, Jonaidi H, Abbasnejad M & Denbow DM. 2006. Interaction between nociceptin/orphanin FQ (N/OFQ) and GABA in response to feeding. Physiology & Behavior, 89: 410-413. DOI: 10.1016/j.physbeh.2006.07.009

Terry P, Gilbert DB & Cooper SJ. 1995. Dopamine receptor subtype agonists and feeding behavior. Obesity Research, 3(l4): SlSS-S23S. DOI: 10.1002/j.1550-8528.1995.tb00221.x

Van Tienhoven A & Juhasz LP. 1962. The chicken telencephalon, diencephalon and mesencephalon in sterotaxic coordinates. Journal of Comparative Neurology, 118: 185-197. DOI: 10.1002/cne.901180205

Wang S, Khondowe P, Chen S,Yu J, Shu G, Zhu X, Wang L, Gao P, Xi Q, Zhang L & Jiang Q. 2012. Effects of "Bioactive" amino acids leucine, glutamate, arginine and tryptophan on feed intake and mRNA expression of relative neuropeptides in broiler chicks. Journal of Animal Science and Biotechnology, 3: 27. DOI: 10.1186/2049-1891-3-27

Zendehdel M, Baghbanzadeh A, Babapour V & Cheraghi J. 2009. The effects of bicuculline and muscimol on glutamate-induced feeding behavior in broiler cockerels. Journal of Comparative Physiology A, 195: 715-720. DOI: 10.1007/s00359-009-0446-3

Zendehdel M, Ebrahimi YS, Hassanpour S & Koohi K. 2019. Interaction of the dopaminergic and Nociceptin/Orphanin FQ on central feed intake regulation in chicken. British Poultry Science, 60(6): 317-322. DOI: 10.1080/00071668.2019.1596225

Zendehdel M, Ghashghayi E, Hassanpour Sh, Baghbanzadeh A & Jonaidi H. 2016. Interaction between opioidergic and dopaminergic systems on feed intake in neonatal layer type chicken. International Journal of Peptide Research and Therapeutics, 22: 83–92. DOI: 10.1007/s10989-015-9486-4

Zendehdel M, Hasani K, Babapour V, Mortezaei S, Khoshbakht Y & Hassanpour Sh. 2014. Dopamine-induced hypophagia is mediated by D1

and 5HT-2c receptors in chicken. Veterinary Research Communications, 38: 11–19. DOI: 10.1007/s11259-013-9581-y

Zendehdel M, Hassanpour Sh. 2014. Central regulation of feed intake in mammals and birds: a review. Eurotransmitter, 1: e251. DOI: 10.14800/nt.251

Zendehdel M, Tirgari F, Shohre B, Deldar H & Hassanpour S. 2017. Involvement of GABA and cannabinoid receptors in central feed intake regulation in neonatal layer chicks: Role of CB1 and GABAA receptors. British Journal of Poultry Science, 19(2): 51-60. DOI: 10.1590/1806-9061-2016-0438