The Role of Central Catecholaminergic Systems in Regulation of Food Intake of Chicks

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To clarify catecholaminergic systems on the regulation of food intake in the neonatal chick, we examined the effects of intracerebroventricular (ICV) injection of prazosin (α1-adrenoceptor antagonist), yohimbine (α2-adrenoceptor antagonist) and benserazide (an inhibitor of L-aromatic amino acid decarboxylase). We found that food intake was significantly suppressed by ICV injection of yohimbine (25 and 50μg) over 60 min (P<0.05). Any doses of prazosin (1.25, 2.5 and 5.0μg) did not alter food intake of chicks (P>0.05). ICV administration of benserazide induced a hypophagia after 60 min postinjection (P<0.05). It is suggested that catecholaminergic systems play an important role in the neural regulation of food intake in chicks, especially through α2-adrenoceptor.

Key words: yohimbine, prazosin, benserazide, feeding behavior, chicken

Introduction

Many neurotransmitters are of importance in the regulation of food intake in birds, i.e., serotonin (Denbow et al., 1982, 1983), GABA (Denbow et al., 1991) and opioid (Steinman et al., 1987). The noradrenergic system also plays an important role in the control of feeding behavior in the domestic fowl (Denbow et al., 1981, 1983). In general, it has been known that central α1-adrenoceptors function to suppress feeding and α2-adrenoceptors enhanced feeding behavior in mammals (Wellman et al., 1993). Sleight et al. (1988) found that the α2-adrenoceptor antagonist, idazoxan, increased food intake in rats when given alone. On the other aspect, the results have been obtained in rat prefrontal cortex slices, where the noradrenergic inhibition of excitatory postsynaptic potentials was abolished by α1-antagonist prazosin, but not by

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the $\alpha_z$-antagonist yohimbine (Law-Tho et al., 1993). The role of the noradrenergic system in modulating in birds has been less elucidated. Recently, we observed that ICV injection of clonidine ($\alpha_2$-adrenoceptor agonist) increased food intake in chicks, and paradoxically, food intake of satiety chicks was enhanced by the central injection of fusaric acid (an inhibitor of dopamine-$\beta$-hydroxylase) in a dose-dependent manner (Bungo et al., 1999).

To expand our current knowledge regarding the role of the catecholaminergic systems in food intake, the present study was designed to assess by blocking both $\alpha_1$- and $\alpha_2$-adrenoceptor and inhibiting L-aromatic amino acid decarboxylase.

**Materials and Methods**

Day old male broiler chicks (Cobb; Mori Hatchery, Fukuoka, Japan) were housed in a windowless room at a constant temperature of 28°C and continuous lighting. The birds were given free access to a commercial starter diet (Toyohashi Feeds and Mill Co. Ltd., Aichi, Japan) and water. They were maintained in accordance with the recommendations of the National Research Council (1985). Birds (2-day-old) were distributed into experimental groups (10 birds per group) based on their body weight such that the average body weight was as uniform as possible for each treatment. Intracerebroventricular (ICV) injection was done according to Davis et al. (1979) and the solutions (10 $\mu$l) were administered using a microsyringe. Yohimbine hydrochloride, prazosin hydrochloride and benserazide were purchased from Katayama (Osaka, Japan), Funakoshi (Tokyo, Japan) and Sigma (St. Louis, MO, USA), respectively. These solutions were dissolved in 0.1% Evans Blue solution, which were prepared in 0.85% saline.

Experiment 1 was conducted to determine if food intake of the neonatal chick was affected by blocking the $\alpha_2$-adrenoceptor. In preliminary trials, we found that lower doses (0.4, 2 and 10 $\mu$g) had no effect on food intake in the neonatal chick. Therefore, higher levels of yohimbine were used in this experiment. Birds were injected ICV with saline or yohimbine (25 and 50 $\mu$g). Food intake was measured at 60 min after ICV injection.

Experiment 2 was done to determine whether food intake of the neonatal chick was affected by antagonizing the $\alpha_1$-adrenoceptor. Birds were injected ICV with saline or prazosin (1.25, 2.5 and 5 $\mu$g). Food intake was measured at 60 min after ICV injection.

Experiment 3 was conducted to determine whether food intake in the neonatal chick was affected by inhibiting the L-aromatic amino acid decarboxylase. Birds were injected ICV with saline or benserazide (2, 10 and 50 $\mu$g). Food intake was measured at 30, 60 and 120 min after ICV injection.

At the end of the experiment, birds were sacrificed by decapitation, after which the location of the injection was confirmed. Data from the individuals that were not verified by the presence of Evans Blue dye in the lateral ventricle were deleted. Therefore, the number of birds used was as follows: Experiment 1: control, 9; 25 $\mu$g, 8 and 50 $\mu$g, 9. Experiment 2: control, 9; 1.25 $\mu$g, 9; 2.5 $\mu$g, 9 and 5 $\mu$g, 10. Exper-
Data were subjected to one-way ANOVA by the General Linear Model procedure using a commercially available package (SAS, 1985) and comparisons between means were made using Duncan's multiple range test. The results are presented as mean ± SEM.

Results

As shown in Fig. 1, food intake was significantly (P < 0.05) suppressed by ICV injection of yohimbine (25 and 50 µg) over 60 min.

The effect of central injection of prazosin is shown in Fig. 2. Food intake was not affected significantly by any levels of prazosin (1.25-5 µg) when compared with the

![Fig. 1](image1.png)

**Fig. 1.** Food intake over a 60 min period after ICV injection of yohimbine or saline in chicks. Means with different letters are significantly different at P < 0.05. Numbers of birds used were: 9 (saline), 8 (25 µg) and 9 (50 µg), respectively. Values are means ± SEM.

![Fig. 2](image2.png)

**Fig. 2.** Food intake over a 60 min period after ICV injection of prazosin or saline in chicks. Numbers of birds used were: 9 (saline), 9 (1.25 µg), 9 (2.5 µg) and 10 (5 µg), respectively. Values are means ± SEM.
saline control ($P > 0.05$).

Fig. 3 gives the effect of central injection of benserazide in chicks. ICV administration of benserazide did not influence food intake at 30 min postinjection. Thereafter, benserazide significantly suppressed food intake of chicks ($P < 0.05$).

Discussion

The present findings demonstrated that ICV injection of yohimbine inhibited food intake of broiler chicks (Fig. 1). There may be two possibilities for explaining this result. The first is that yohimbine-induced hypophagia is directly mediated by postsynaptic $\alpha_2$-adrenoceptor (see Fig. 4). It has been reported that the stimulation of postsynaptic $\alpha_2$-adrenoceptor enhances feeding behavior (Schlemmer et al., 1981; Goldman et al., 1985; Wellman et al., 1993). We also found a similar response using clonidine ($\alpha_2$-adrenoceptor agonist) in neonatal chicks (Bungo et al., 1999). The second is that the anorexic effect of yohimbine was due to a synergic effect of $\alpha_2$-adrenoceptor and other sub-receptors such as $\alpha_1$, $\beta_1$- and $\beta_2$-adrenoceptors, because other sub-receptors were stimulated by endogenous noradrenaline even under $\alpha_2$-adrenoceptor being blocked by yohimbine (Fig. 4). As regarding $\alpha_1$-adrenoceptor, Wellman et al. (1993) reported that the stimulation of $\alpha_1$-adrenoceptor inhibited feeding behavior in rats. Hypophagia mediated by $\alpha_1$-adrenoceptor may not be a critical factor in the central nervous system of chicks, because central injection of prazosin did not affect food intake (Fig. 2). However, it is premature to conclude in the chick that the significance of a function for $\alpha_1$-adrenoceptor as a synergism of both $\alpha_1$- and $\alpha_2$-adrenoceptors that has been proposed by Wellman et al. (1993), since only single antagonist for each $\alpha$-adrenoceptor was used in the present study. Similarly, the relationships between $\alpha$- and $\beta$-adrenoceptor on food intake were unclear. These remain to be clarified in future.

Previously, we found that fusaric acid, an inhibitor of dopamine-$\beta$-hydroxylase (Fig. 4), stimulated food intake of chicks (Bungo et al., 1999). Our next experiment was, therefore, designed to elucidate whether deficient of noradrenaline, which was induced by benserazide, elicits food intake of the chick. However, benserazide, an inhibitor of L-aromatic amino acid decarboxylase (Fig. 4), showed a depression, but not a stimulation of food intake in chicks (Fig. 3). It is hypothesized that this effect was due to the depletion of dopamine mediated by benserazide, but it is difficult to conclude that dopamine itself directly affected food intake of chicks. For instance, Denbow et al. (1981, 1983) reported that ICV injection of dopamine did not affect food intake of chickens. We have also confirmed that central injection of dopamine (0-100μg) did not alter food intake of the neonatal chick (unpublished data). The effect of benserazide was not rapidly observed in the present study. This result implied that the inhibition of noradrenaline synthesis resulted from the depletion of dopamine may involve in the hypophagia.

Further experiments will be required to conclude the synergism between sub-
Fig. 3. Cumulative food intake of chicks injected ICV with saline or three levels of benserazide (2, 10 and 50 µg). Means with different letters at each time are significantly different at $P<0.05$. Numbers of birds used were: 10 (saline), 10 (2 µg), 10 (10 µg) and 8 (50 µg), respectively. Values are means ± SEM.

Fig. 4. Proposed central mechanisms of α-adrenergic systems in food intake of chicks (based on Wellman et al. (1993)); DA, dopamine; NA, noradrenaline. NA is synthesized from l-tyrosine by each enzyme and is released to the synaptic cleft. Released NA binds each receptor.
adrenoceptor on food intake of chicks. However, the results described here suggest that noradrenergic systems have an important role for feeding, especially through α2-adrenoceptor.

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