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Original Research Article

Effect of central and peripheral injection of prostaglandin E2 and F2α on feeding and the crop-emptying rate in chicks

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ABSTRACT

Prostaglandins (PGs) have been shown to cause several physiological changes in mammals including anorexia, awakening and sleeping, change in digestive function, and activation of the hypothalamus-pituitary-adrenal gland (HPA) axis. However, there is a paucity of information about the effect of PGs on physiological parameters in birds. The purpose of the present study was to clarify whether intracerebroventricular (ICV) and intraperitoneal (IP) injections of prostaglandin E2 (PGE2) and prostaglandin F2α (PGF2α) affect feeding, voluntary movement, crop-emptying rate, and corticosterone release in chicks (*Gallus gallus*). ICV injection of either PGE2 or PGF2α (2 and 4 μg) significantly decreased food intake in chicks. The anorexigenic effect was also observed after IP injection of the PGs. Voluntary movement was significantly suppressed by ICV injection of PGE2 or PGF2α, although the time-course change was different between the two. In contrast, IP injection of the PGs had no or less effect on voluntary movement. Both ICV and IP injection of PGE2 significantly retarded the crop-emptying rate, whereas PGF2α significantly lowered the crop-emptying rate only after IP injection. The plasma corticosterone concentration significantly increased after ICV and IP injection of PGE2, whereas PGF2α had no effect. These results suggest that central and peripheral PGs are involved in the regulation of appetite, voluntary movement, food passage in the digestive tract, and activation of the HPA axis in chicks, although the effects depend on the site of action and type of PGs.

1. Introduction

Lipopolysaccharide (LPS) is a component of the outer membrane of Gram-negative bacteria and causes several non-specific symptoms such as fever, somnolence, anorexia, adipsia, and dysfunction of the digestive tract in animals [1,2]. In mammals, these symptoms have been shown to be mediated by several kinds of bioactive molecules including prostaglandins (PGs). For example, LPS-induced anorexia and inhibition of gastric emptying are attenuated by indomethacin (IND), an inhibitor of a PG synthase, cyclooxygenase (COX), in rodents [3–5]. In addition, LPS treatment increases the production of PGs including prostaglandin E2 (PGE2) and prostaglandin F2α (PGF2α) in various tissues such as mouse embryo fibroblasts [6], human placental explants [7], and rat Kupffer cells [8]. Intraperitoneal (IP) injection of LPS increases plasma PGE2 concentration in rats [9]. Intravenous injection of LPS also increases serum concentration of 13,14-dihydro-15-keto-PGF2α, a metabolite of PGF2α, in porcine [10]. These results suggest that these kinds of PG are related to the effect of LPS in mammals. Indeed, central and peripheral injections of PGE2 and PGF2α have been shown to inhibit feeding behavior [11–15] and affect gastric emptying [16,17].

As in mammals, LPS induces hyperthermia, the anti-dipsogenic effect, somnolent activity, depression, retardation of crop emptying, and activation of the hypothalamus-pituitary-adrenal (HPA) axis in chickens (*Gallus gallus*) when administered centrally or peripherally [18–22]. In addition, LPS induces anorexia in chickens: the injection of LPS decreases food intake when administered both centrally and peripherally [18,19]. The anorexigenic effect is also mediated by PGs as reported by Johnson et al. [19] who showed that IP injection of IND attenuated LPS-induced anorexia, hyperthermia, and somnolence in young chickens. Intracerebroventricular (ICV) injection of IND also attenuated LPS-induced hyperthermia and somnolence but not anorexia [19]. These results indicated that LPS-induced anorexia and behavior are mediated by peripheral and central PGs. However, which PGs are related to these effects of LPS has not yet been investigated. It has been reported that intravenous injection of LPS increases plasma PGE2 concentration in 5-week-old chickens [23]. Similarly, IP injection of LPS increases serum PGE2 concentration in 16- and 20-day-old chickens

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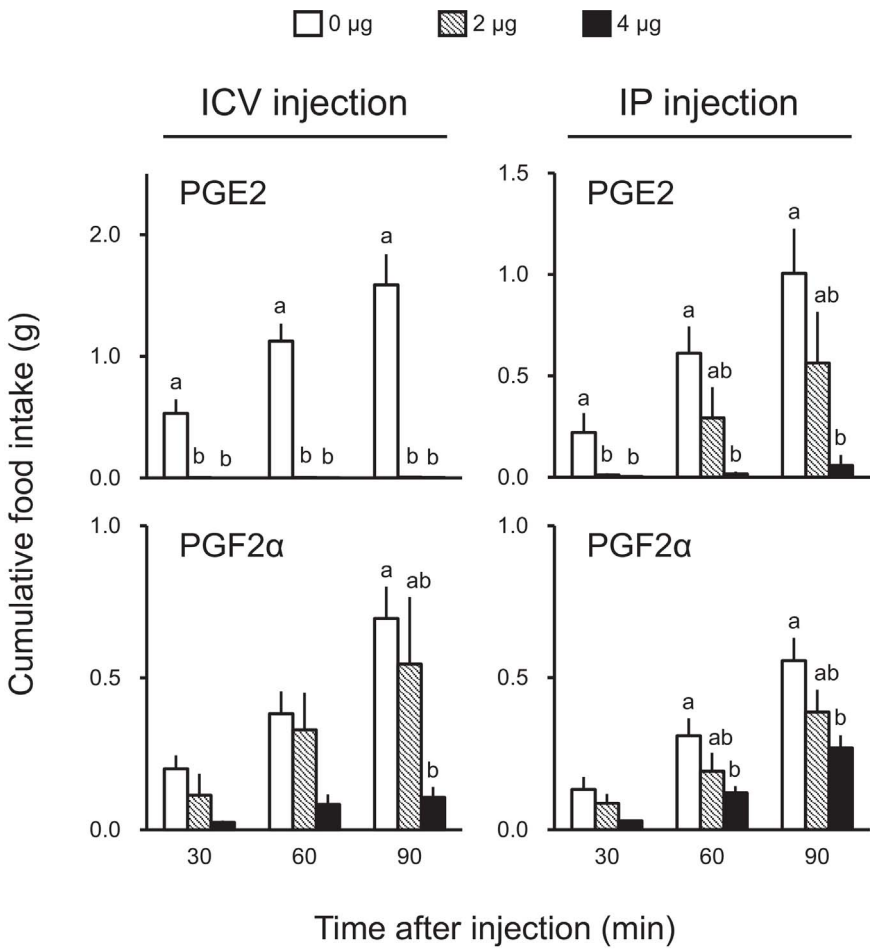

Fig. 1. Effect of ICV or IP injection of PGE2 and PGF2α on food intake in chicks. In the PGE2 study, the number of chicks in the 0 (control), 2, and 4 μg groups were 8, 8, and 9 for ICV injection, and 6, 6, and 7 for IP injection, respectively. In the PGF2α study, the number of chicks in the 0, 2, and 4 μg groups were 9, 9, and 8 for ICV injection, and 10, 10, and 9 for IP injection, respectively. Data are expressed as mean ± SEM. Groups with different letters within a time period are significantly different ($P < 0.05$).

[21], suggesting that PGE2 is a strong candidate to mediate the effect of LPS in chickens. To our knowledge, the kinetics of other PGs after LPS treatment has not yet been investigated in chickens, but it is possible that PGF2α is produced and released by LPS as reported in mammals.

In the present study, we investigated the effect of ICV and IP injections of PGE2 and PGF2α on feeding behavior in chicks. In addition, PG-induced voluntary movement was also investigated to clarify how PGs affect feeding behavior. The role of PGs in crop emptying and plasma corticosterone concentration was also examined.

2. Materials and methods

2.1. Animals

Day-old male layer chicks (*Gallus gallus*, White Leghorn, Julia) were purchased from a local hatchery (Nihon Layer, Gifu, Japan) and raised in a room kept at 30 °C with continuous lighting. A commercial diet (crude protein: 24%, metabolizable energy: 3050 kcal/kg; Toyohashi Feed Mills Co. Ltd, Aichi, Japan) and water were available ad libitum to the chicks. Chicks were transferred to the experimental cage at least two days before each experiment to accustom experimental condition. They were individually reared one day prior to each experiment. Before the experiment, body weight was measured and then chicks were distributed into experimental groups so that the average body weight was as uniform as possible between treatment groups. The chicks were maintained in accordance with the recommendations of the National Research Council [24]. This study was approved by the Committee of Animal Care and Use in Ehime University, Japan (No. 08-o3-10).

2.2. Drugs and injections

All injections were given between 0800 and 1000. For ICV injection, PGE2 (Tokyo Chemical Industry, Tokyo, Japan) and PGF2α (Cayman Chemical Company, MI, USA) were dissolved in a normal saline solution containing 0.1% Evans Blue dye. The vehicle only was used for the control treatment. ICV injections were given according to a previously reported method [25]. In brief, the head of the chick was inserted into an acrylic box with a hole in the top plate. The injection coordinates were 3 mm anterior to the coronal suture, 1 mm lateral from the sagittal suture, and 3 mm deep targeting the left lateral ventricle. Anatomical landmarks were determined visually and by palpation. The solution was injected through the hole using a microsyringe at a volume of 10 μl per chick. The injection procedure is rapid and does not result in additional stress to neonatal chicks judging from food intake and corticosterone release data [26,27]. At the end of each experiment, the chicks were euthanized with an overdose of pentobarbital. The brain was then removed to confirm the accuracy of injection. Any chicks that did not show the presence of Evans Blue dye in the lateral ventricle were not used for analyses.

For IP injection, PGE2 and PGF2α were dissolved in a normal saline and the vehicle alone was used for the control treatment. These solutions were injected into the abdominal cavity at a volume of 0.2 ml per chick.

2.3. Effect of PGE2 and PGF2α on feeding behavior

In the ICV injection study, 6-day-old chicks were ICV injected with 0