PERIPHERAL REGULATION OF FOOD INTAKE IN THE DOMESTIC FOWL

by

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(ABSTRACT)

Four studies were performed to examine peripheral factors involved with food intake regulation in the domestic fowl. In the first study, the mechanism by which tryptophan depresses food intake was clarified. Intraperitoneal injections of tryptophan methyl ester were demonstrated to inhibit feeding in Single Comb White Leghorn (SCWL) cockerels. Intragastric intubations of tryptophan inhibited food intake and decreased body temperature of SCWL cockerels. These results, in conjunction with previous findings, indicate that tryptophan's inhibitory influence on food intake is peripherally rather than centrally based. The second study explored the role of the duodenum in food intake regulation. Intraduodenal glucose loads had no effect on food intake of SCWL or Rock Cornish (RC) commercial broiler cockerels. In addition, splanchnicectomized birds did not respond to intraduodenal glucose infusions any differently than sham-operated

controls. Apparently, the duodenum does not play a significant role in food intake control in the fowl. Hepatic involvement in appetite regulation was examined in SCWL and RC cockerels in the third study. Amino acid solutions failed to influence food intake when infused intraportally in either strain of chicken. Relatively small glucose or lipid solutions depressed food intake significantly when infused intraportally in the SCWL birds but had no effect in the RC cockerels. The liver appears to be integrally involved in controlling food consumption in the SCWL chicken. In the final study, the existence of a "hunger" factor in the peripheral circulation of two lines of chickens divergently selected for body weight was explored. Intrahepatic infusions of plasma from food-deprived high-weight line chickens stimulated food intake of sated low-weight line chickens.

These studies indicate that peripheral mechanisms are important in regulating appetite in light-breed chickens such as the SCWL, however, such mechanisms in heavy-breed chickens such as the RC appear to be less sensitive. This desensitization in heavy-breed chickens suggests that genetic selection for increased growth has affected the food intake control systems.

DEDICATION

This dissertation is dedicated to the memory of Dr. Lacy. He introduced me to agriculture and education and is responsible for my interest and accomplishments in both. On top of that he was the best father in the world.

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INTRODUCTORY REMARKS

Advances in understanding food intake regulation in humans have been considerable in recent years. Research on factors controlling appetite has increased as more emphasis has been placed upon controlling body weight for health and cosmetic reasons. The alarming increase in the incidence of both obesity and anorexia in American society has further stimulated investigation of mechanisms regulating food consumption.

Interest in food intake regulation in agricultural animals has also been on the increase. Food consumption, of course, is a significant factor influencing growth. With the current trend toward expecting greater production from fewer animals, mechanisms affecting growth have come under close scrutiny. In addition, the ever rising cost of feeding domestic animals has necessitated the development of more effective and efficient feeding schemes. If knowledge of food intake regulatory mechanisms can be better developed, it might be possible to manipulate such mechanisms to affect growth to the advantage of animal producers. In the poultry industry, food intake manipulation should be especially beneficial. For example, mechanisms that inhibit food consumption could prove useful in limiting growth in broiler and turkey breeders. In

layers, it might be possible to induce molting in hens and control body size by influencing appetite. And, of course, considerable economic gains are possible if growth in meat-type poultry could be enhanced by stimulation of feeding.

Research in food intake regulation has evolved full cycle. Early theories, such as the oropharyngeal (von Haller, 1803) and the gastric sensations (Cannon and Washburn, 1912) hypotheses, enunciated the importance of peripheral mechanisms in appetite control. Then, with the advent of stereotaxic techniques, central theories came into vogue. The glucostatic (Mayer, 1953, 1955), lipostatic (Kennedy, 1950; Bates et al., 1955), aminostatic (Smyth et al., 1947

Mellinkoff et al., 1956), and the hypothalamic feeding and satiety centers (Hetherington and Ranson, 1939; Anand and Brobeck, 1951) hypotheses all emphasize the brain in regulating appetite. The brain serotonergic (reviewed by Blundell, 1977) and catecholaminergic (reviewed by Leibowitz, 1980) systems have been purported to be responsible for inhibition and stimulation of feeding, respectively. Certainly the central nervous system must play a critical role in food intake control; however, recent work has once again indicated that peripheral mechanisms are important, particularly in short-term regulation (reviewed by Novin, 1976; Novin and VanderWeele, 1977; Sawchenko and

Friedman 1979; Russek, 1981; Forbes, 1983). The role of peripheral control mechanisms may be especially significant if one is interested in manipulating appetite. Manipulation of central mechanisms is quite difficult due to the protective blood-brain barrier that insulates the brain from most blood-borne substances. Peripheral mechanisms may be more accessible and, therefore, of practical interest in influencing feeding behavior.

The purpose of this research was to study peripheral mechanisms regulating food intake in the domestic fowl. This dissertation will be presented as six chapters each of which has been or will be submitted for publication as a research paper or note. The first two chapters explore the nature of the inhibitory effect of tryptophan on food intake in the fowl. Chapter 3 deals with the role of the duodenum in food intake regulation. The involvement of hepatic mechanisms in appetite control is examined in Chapters 4 and 5. And the final chapter investigates the possibility of the existence of a hunger factor in the peripheral circulation of fasted birds.

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CHAPTER 1

INTRAPERITONEAL INJECTIONS OF TRYPTOPHAN INHIBIT FOOD INTAKE IN THE FOWL

INTRODUCTION

The brain serotonergic system has been shown to play an inhibitory role in feeding behavior (reviewed by Blundell, 1977). Central injections of serotonin (5-hydroxytryptamine, 5-HT) decreased food intake in rats (Goldman, et al., 1971; Lehr and Goldman, 1973; Kruk, 1973) and chickens (Denbow et al., 1983).

Normal functioning of the brain serotonergic system appears to be related to the availability of the amino acid tryptophan, the precursor of 5-HT. Large quantities of tryptophan in the diet of rats have been shown to increase brain levels of 5-HT (Fernstrom and Wurtman, 1971). Similarly, whole brain levels of 5-HT increased in response to orally administered tryptophan in the turkey (Lee and Britton, 1982). As might be expected, tryptophan has also been found to have an inhibitory effect on food intake. Dietary tryptophan inhibited feeding in rats (Ashley and Anderson, 1975), and oral intubations of tryptophan decreased food intake in the domestic fowl (Lacy et al., 1982).

Intraperitoneal (IP) administration of tryptophan has had mixed effects on food intake. Early studies in rats failed to show any effect (Barrett and McSharry, 1975; Weinberger et al. 1978) or only a mild inhibition of food intake (Fernstrom and Wurtman, 1972). More recently IP injections of tryptophan have resulted in a clear depression of food consumption in the rat (Latham and Blundell, 1979; Gibbons et al., 1981). In the present study, the effect of intraperitoneal injections of tryptophan on food intake in the chicken were examined.

MATERIALS AND METHODS

Two experiments were conducted. In the first, tryptophan solutions were tested in free-feeding subjects, and in the second, tryptophan was tested in subjects that had been fasted 24 hours. In each experiment, 20 Single-Comb White Leghorn chicks 16-weeks of age were used as the experimental animals.

The birds were housed in individual cages under constant light. Four levels of tryptophan (12.5, 25, 50 and 100 mg) and a saline control were tested in each of the animals using a replicated Latin Square design. The tryptophan treatments were prepared by dissolving L-tryptophan methyl ester (Sigma) in saline. They were injected into the birds IP in a volume of 1 ml. Tests began

between 0800 and 1000 hours. At least 48 hr separated the testing of each treatment in each bird.

Food intake was measured to the nearest g at 30, 60, 90, 120, 180 min and 24 hr post-injection. The effect of treatment on cumulative food consumption at each time period was tested for linear and quadratic responses using orthogonal polynomials.

RESULTS AND DISCUSSION

In Experiment 1, the tryptophan treatments caused a dose-dependent decrease in the food intake of the free-feeding chicks at all time periods (Table 1) as evidenced by a significant linear contrast ($P \leq 0.05$). Studies in rats have produced similar results. Fernstrom and Wurtman (1972), Latham and Blundell (1979) and Gibbons et al. (1981) all observed that tryptophan inhibits food intake in free-feeding rats.

Food intake of the 24-hr fasted chicks (Experiment 2) was unaffected by any of the tryptophan injections (Table 2) as demonstrated by the absence of a significant linear or quadratic response. Studies where tryptophan was administered to rats that had been fasted 18-hr (Barrett and McSharry, 1975) and 24-hr (Weinberger et al., 1978) also failed to show a significant effect on food intake. Latham and Blundell (1979) tested tryptophan in both free-feeding

and fasted rats and concluded that free-feeding animals are more sensitive models than food deprived animals for studying weak drug effects on food intake. They suggested that studies using fasted animals failed to detect the inhibitory action of tryptophan on feeding for this reason.

Apparently tryptophan affects food intake in the chicken in much the same way it does in the rat. These findings are consistent with existing data which link increased plasma tryptophan levels, increased brain 5-HT concentrations and food intake depression; however, attempts to affect the appetite of Leghorns with intracerebroventricular injections of tryptophan methyl ester have been unsuccessful (D. M. Denbow, personal communication). Recent studies on peripheral food intake regulatory mechanisms (Rezek and Novin, 1977; Deutsch and Gonzalez, 1980; Shurlock and Forbes, 1981a,b; Friedman and Sawchenko, 1984) have shown these to be more important than previously thought, and tryptophan may very well be working directly on amino acid receptors or through augmentation of 5-HT stores in the periphery.

Table 1. Mean cumulative food intake (g) by ad libitum fed Single-Comb White Leghorn chicks following intraperitoneal injections of tryptophan (trp).

	Food intake (g)					
Treatment	30 min	60 min	90 min	120 min	180 min	1440 min
Saline	6.4	9.8	12.2	14.3	19.5	94.6
12.5 mg trp	5.8	8.1	10.6	12.4	17.1	86.6
25 mg trp	6.1	8.0	11.4	13.4	18.4	96.6
50 mg trp	4.0	6.2	8.4	10.5	15.0	83.4
100 mg trp	3.8	5.6	7.8	9.9	14.2	81.2
SEM[1]	0.6	0.8	1.0	1.2	1.4	3.5
----------	F (1,72) Values					
Linear	13.45*	14.31*	10.80*	8.26*	8.72*	6.94*
Quadratic	2.02	0.60	0.75	0.32	0.27	0.02

*Mean cumulative food intake at each time period was tested for linear and quadratic responses using orthogonal contrasts. The linear contrast was significant ($P \leq 0.05$) at each time period.

[1]Standard error of the mean.

Table 2. Mean cumulative food intake by 24-hr fasted Single-Comb White Leghorn chicks following intraperitoneal injections of tryptophan (trp).

	Food Intake (g)					
Treatment	30 min	60 min	90 min	120 min	180 min	1440 min
Saline	10.8	15.0	18.8	22.0	29.8	110.4
12.5 mg trp	10.6	14.2	17.8	21.0	28.2	107.3
25 mg trp	11.4	15.4	19.0	22.2	29.9	112.0
50 mg trp	12.2	16.8	20.0	24.3	31.5	113.1
100 mg trp	11.7	15.5	19.0	22.0	29.6	104.9
SEM[1]	0.7	1.0	1.2	1.5	1.8	4.0
----------	F (1,72) Values					
Linear	0.64	0.82	0.26	0.17	0.09	0.63
Quadratic	0.33	0.98	0.58	0.41	0.54	0.00

Mean cumulative food intake at each time period was tested for linear and quadratic responses using orthogonal contrasts. Neither the linear nor the quadratic contrast were significant ($P \leq 0.05$).

[1]Standard error of the mean.

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CHAPTER 2

TRYPTOPHAN'S INFLUENCE ON FEEDING AND BODY TEMPERATURE IN THE FOWL

INTRODUCTION

Central administration of serotonin (5-hydroxytryptamine, 5-HT) has been shown to inhibit food intake in rats (Goldman et al., 1971; Lehr and Goldman, 1973; Kruk, 1973) and chickens (Denbow et al., 1982; Denbow et al., 1983). Tryptophan, the amino acid precursor of 5-HT, has also been shown to decrease feeding in a variety of species (Anderson and Ashley, 1977; Latham and Blundell, 1979; Gibbons et al., 1981; Lacy et al. 1982). Since dietary loads of tryptophan have been demonstrated to increase brain levels of 5-HT (Valzelli and Garattini, 1968; Fernstrom and Wurtman, 1971; Lee and Britton, 1982), it has been assumed that the mechanism by which tryptophan affected food intake involved changes in central 5-HT concentrations. Several studies, however, have suggested that peripheral amino acid receptors may be monitoring amino acid concentrations in the gut of the cat (Jeanningros, 1983) and liver of the dog, rat and rabbit (Russek 1970, 1971; Rezek and Novin, 1977) and that these peripheral receptors may be important mechanisms regulating appetite.

The objective of the present study was to clarify the site where tryptophan acts to inhibit food intake by examining the effect of tryptophan on body temperature. Serotonin administered centrally in chickens has been demonstrated to increase body temperature (Hillman et al., 1980; Nistico et al., 1980). Therefore, measuring the effect of tryptophan on body temperature might provide clues as to whether the effect of tryptophan on feeding is centrally or peripherally based in the chicken.

MATERIALS AND METHODS

Ten Single-Comb White Leghorn (SCWL) cockerels 24 weeks of age were used as experimental animals. The birds were maintained in individual cages under constant light. Food and water were available ad libitum except as noted.

The birds were randomly assigned to two groups of five birds each. One group was intragastrically intubated with 1.25 g of tryptophan suspended in 10 mls of distilled water, while the other group served as a control and was intubated with 10 mls of distilled water. Both the tryptophan suspension and the distilled water were warmed to 40°C prior to administration to ensure that the temperature of these solutions did not influence body temperature. Intubations were carried out using a 10 cc syringe with a 15 cm length of plastic tubing attached. The tubing was directed toward

the caudal esophagus of a bird and the test solution was deposited at that point. Forty-eight hours later the experiment was repeated in exactly the same manner except the treatment groups were reversed. Data from the two replicates were combined.

Experiments were conducted between 1115 and 1700 hours. At the beginning of the experiments food was taken away from the birds, thermistor probes (YSI Model 402 or 423) were inserted rectally, secured with tape, and attached to a telethermometer (YSI Model 46 TUC). Two hours later the birds were intubated with either the tryptophan suspension or the distilled water. A base-line body temperature measurement was recorded immediately prior to the intubations and every 30 min thereafter for a total of 210 min. Body temperatures were measured to the nearest 0.1°C. Thirty minutes post-intubation the birds were provided access to food. Food intake to the nearest g was then recorded every 30 min for 180 minutes. Cumulative food intake and changes in body temperature were examined by analysis of variance.

RESULTS AND DISCUSSION

Food intake of the SCWL cockerels was significantly depressed ($P \leq 0.05$) by tryptophan at all of the time periods (Table 1). The inhibition is similar to that seen in

previous experiments with chickens (Lacy et al., 1982) and rats (Anderson and Ashley, 1977).

Body temperature at all the time periods was also significantly decreased ($P \leq 0.05$) by the tryptophan treatment (Table 1). Under certain conditions, declines in body temperature have been shown following central 5-HT treatment. Such conditions have included high dose levels of 5-HT (Brittain and Handley, 1967; Myers, 1968), the specific site of injection within the central nervous system (Crawshaw, 1972; Pyornila and Hissa, 1979), low ambient temperature (Bligh and Cottle, 1969) or the particular 5-HT salt used (Hillman et al., 1980). Under most conditions; however, central administration of 5-HT leads to an increase in body temperature in chickens (Marley and Nistico, 1975; Hillman et al., 1980; Nistico et al., 1980) and many mammalian species (reviewed by Myers and Waller, 1977). This is the opposite of the results obtained in the present experiment.

When 5-HT has been administered peripherally to mammals (reviewed by Myers and Waller, 1977) a decrease in body temperature has usually resulted. It is thought this fall in body temperature is due to the effect that 5-HT exerts on peripheral systems such as the respiratory (Asboe-Hansen and Wegelius, 1956) and cardiovascular system (Ginzel and Kottegoda, 1954; Page, 1968). Since 5-HT does not readily

cross the blood-brain barrier, it is not likely that 5-HT administered peripherally has a significant effect in the central nervous system.

In the present experiment tryptophan decreased body temperature as peripherally administered 5-HT has been shown to do. Therefore, it may be that orally administered tryptophan is affecting body temperature in the chicken peripherally rather than centrally. The possible peripheral effect of tryptophan on body temperature fosters speculation as to whether the effect of tryptophan on food intake may also be peripherally based. In an earlier study (Lacy et al., 1984), it was found that orally administered tryptophan had no effect on tonic immobility, a behavior known to be very sensitive to brain 5-HT manipulation (Maser et al., 1975; Harston et al., 1976; Wallnau and Gallup, 1977; Hennig, 1980). It was postulated at that time that tryptophan might not be influencing food intake by increasing brain 5-HT concentrations. The results of the present experiment do not disprove the assumption that tryptophan inhibits food intake via its influence on central 5-HT levels but do suggest that the role of peripheral mechanisms should be investigated.

Table 1. Mean cumulative food intake (g) and rectal temperature change (°C) of Single-Comb White Leghorn cockerels in response to intragastric intubations of tryptophan (trp) or water.

	Time post-intubation[1]						
Trt	30 min	60 min	90 min	120 min	150 min	180 min	210 min
				Food intake (g)			
Water	--	8.2	12.8	14.0	17.5	19.1	23.0
Trp	--	4.8*	7.4*	9.2*	10.2*	11.3*	13.6*
SEM[2]	--	0.9	1.1	1.3	1.3	1.2	1.6
				Δ Rectal temperature (°C)			
Water	0.06	0.06	0.00	-0.16	-0.12	-0.15	-0.13
Trp	-0.27*	-0.69*	-0.70*	-0.78*	-0.83*	-0.69*	-0.67*
SEM[2]	0.09	0.15	0.14	0.12	0.16	0.17	0.17

[1]Birds were fasted 2 hr pre-intubation and given access to food 30 min post-intubation.

[2]Standard error of the mean.

*Significantly different from control ($P \leq 0.05$).

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CHAPTER 3

INTRADUODENAL GLUCOSE INFUSIONS DO NOT AFFECT FOOD INTAKE IN THE FOWL

INTRODUCTION

Animals are thought to consume food to satisfy energy requirements. Since the major energy constituent in the diets of many animals is carbohydrate, it has been proposed that glucose or some component of the glycolytic chain may be a key factor monitored by mechanisms controlling food intake. Since glucose is absorbed for the most part in the duodenum and anterior small intestine, this area of the gastrointestinal tract has been looked to as a putative food intake regulatory site.

Numerous studies have demonstrated the inhibitory effects of intraduodenal glucose infusions on feeding in mammals (Ehman et al., 1971; Novin et al., 1974; Cabanac and Fantino, 1977; Stephens, 1980); however, published information on whether this is true in birds is limited. Sonoda and Makino (1980) reported that intracrop glucose loads decreased food intake more slowly in splanchnicectomized (nerves to the duodenum and small intestine severed) cockerels than in sham operated controls. They concluded that the celiac plexus of the splanchnic nerve carries satiety signals from the digestive tract to

the brain. Although this splanchnicectomy study suggests that glucose in the duodenum or small intestine may influence food intake in the fowl, the only report that has directly examined the effect of intraduodenal glucose infusions on food intake in birds (Shurlock and Forbes, 1981) found that duodenal glucose loads did not affect food intake in cockerels.

Two important questions need to be examined to clarify whether duodenal glucose loads affect feeding in birds. The first concerns the concentration and/or quantity of glucose administered intraduodenally. In the rabbit it has been demonstrated that small, isotonic glucose infusions suppress food intake, whereas larger quantities of glucose actually enhance feeding (Rezek et al., 1975). Shurlock and Forbes (1981) infused rather large and hypertonic glucose solutions in their experiment with chickens; therefore, smaller, less concentrated solutions should be tested. The second question concerns whether intraduodenal infusions of glucose in denervated and sham-operated birds would yield results like those observed following the intracrop infusions in the study by Sonoda and Makino (1980). The objective of the present study was to answer these questions.

MATERIALS AND METHODS

General. A series of seven experiments were performed. The first six examined the effect of intraduodenal glucose infusions on food intake in the fowl, and the final experiment tested the effect of intraduodenal glucose loads in splanchnicectomized birds. In all experiments chicks were housed in individual cages under continuous light. Food and water were available ad libitum unless otherwise noted.

Duodenal cannulae were implanted while birds were under sodium pentobarbitol anesthesia (25 mg/kg body weight). Feathers were removed from the right side of each bird and a 5 cm incision was made between the cloaca and sternum. The duodenum was exposed and a small incision made in the descending limb. A polyethylene cannula (i.d. 1.6 mm, o.d. 2.1 mm) with a flared end was inserted through the incision into the duodenum. The duodenal incision was closed and the cannula secured with a purse string suture. The extraduodenal portion of the cannula was encased in Tygon® tubing (i.d. 4.8mm, o.d. 6.3mm) for protection, passed under the skin and exteriorized in the thoracic region of the bird. Incisions were closed with silk thread. The cannula was attached to a swivel system at the top of the cage via the protective tubing, which allowed infusions to be made without handling the subjects. Birds were allowed a 4-6 day recovery period before experiments were initiated.

Experiment 1. Eight Single-Comb White Leghorn (SCWL) cockerels 12 weeks of age were fitted with duodenal cannulae. Four treatments were tested in each of the eight birds using a replicated Latin Square design. Tests were conducted between 0800-1200 hr with at least 24 hr separating testing of treatments. Treatments tested in this first experiment were 10 ml of a 5% glucose solution, a 0.9% saline solution, a 1.8% urea solution and an uninjected control. The glucose, urea and saline solutions were isotonic at 290 mOsm/liter. Osmolarity of solutions was determined by freezing point depression. The treatments were infused into the birds via the duodenal cannulae over a 30 min period using a multi-channel syringe pump. Subjects were provided food ad libitum prior to and immediately following the infusions but were not fed during the infusions. Food intake was measured every 30 min post-infusion for 2 hours. Cumulative food intake at each time period was examined by analysis of variance for replicate, day, bird and treatment effects. When a significant treatment effect ($P \leq 0.05$) was observed, Duncan's Multiple Range Test was used to determine which treatments differed.

Experiment 2. Another group of eight SCWL cockerels 12 weeks of age were implanted with duodenal cannulae. Experiment 1 was repeated with the exception that the treatments tested were an uninjected control, 10 ml of 10%

glucose, 10 ml of 1.8% saline, and 10 ml of 3.5% urea. The three solutions were isosmotic at 580 mOsm/liter.

Experiments 3-6. A third group of 8 SCWL cockerels 12 weeks of age received duodenal cannulae. Four treatments, 10 ml of 20% glucose, 10 ml of 3.5% saline, 10 ml of 7% urea and an uninjected control, were tested in these birds using the same procedures as in Experiment 1. Osmolarity of the solutions tested was 1160 mOsm/liter. This experiment was also repeated in 24-hr fasted SCWL cockerels 12 weeks of age (Experiment 4) and in ad libitum fed (Experiment 5) and 24-hr fasted (Experiment 6) Rock Cornish (RC) commercial broiler cockerels 6 weeks of age.

Experiment 7. Twenty-four SCWL cockerels 12 weeks of age were anesthetized with sodium pentobarbitol (25 mg/kg body weight). Feathers were removed from the right side of the birds and an incision made between the vertebral ribs. The plexus celiacus and plexus celiacus accessorius nerves were located. In 12 of the birds these nerves were severed (splanchnicectomy) while in the other 12 birds the nerves were left intact. Incisions were closed with silk thread. Duodenal cannulae were then implanted in both groups of birds. Subjects again were given 4-6 days to recover.

In separate trials, glucose concentrations ranging from 20 to 80% and volumes from 5 to 10 ml were tested in both groups of birds. Infusions were made over a 30 min period

with a multi-channel syringe pump. Food intake was monitored as before and cumulative food intake of the splanchnicectomized and sham-operated control cockerels was examined by analysis of variance. Significance implies $P \leq 0.05$.

RESULTS AND DISCUSSION

Experiment 1. Neither the isotonic 5% glucose nor the urea solution had an effect on the food intake of ad libitum fed SCWL cockerels (Table 1); however, the 0.9% saline treatment did significantly inhibit food intake at 60 min post-infusion. Although Rezek et al. (1975) found that small volumes of isotonic glucose were much more potent inhibitors of feeding in free fed rabbits than larger glucose loads, this did not appear to be the case in the chicken.

Experiment 2. The 10% glucose and isosmotic saline solutions significantly depressed ($P \leq 0.05$) food intake at 60 min post-infusion when compared with the uninjected control (Table 2). Although glucose inhibited feeding in this experiment, it appears that the osmolarity of the glucose solution was a factor since the glucose, saline and urea treatments never differed significantly.

Shurlock and Forbes (1981) hypothesized that osmotic mechanisms located in the duodenum might be affecting food

intake in the fowl. Glucose loads are absorbed very rapidly from the small intestine, and this has led to speculation that glucose may be removed from the gut before osmoreceptors can be activated. Since urea is also readily absorbed from the intestine but has no nutritive value, it was used (along with NaCl and the uninjected control) to test the effects of glucose on food intake. The urea treatment did not differ statistically from the glucose or saline treatments at any of the time periods; therefore, it does not appear that rapid uptake of glucose from the duodenum influenced the results.

Experiment 3. In this experiment 20% glucose and isosmotic solutions of urea and saline were infused to investigate further a possible osmotic effect. At 120 min post-infusion saline significantly depressed food intake (Table 3) when compared to the uninjected control, but glucose and urea treatments were not inhibitory. Food intake responses resulting from saline, glucose and urea administration were not significantly different from each other.

Experiments 4-6. Shurlock and Forbes (1981) demonstrated that mildly fasted birds were more sensitive to intraduodenal glucose loads. Therefore, as a further test of the role of glucose and the duodenum in food intake regulation, 24 hr fasted SCWL (Experiment 4) and ad libitum

fed (Experiment 5) and 24 hr fasted (Experiment 6) commercial broilers were infused with the 20% glucose and the isosmotic saline and urea solutions. The results obtained (Tables 4-6) were similar to those obtained with the ad libitum fed SCWL cockerels; glucose did not affect food intake. Inhibition of feeding due to hypertonicity was observed earlier (30-min versus 120-min post-infusion) in the fasted SCWL cockerels than in the ad libitum fed SCWL birds. It seems logical that a relatively empty gastro-intestinal tract would be more sensitive to hyperosmotic infusions. The fasted broilers did not show any sensitivity to the hyperosmotic treatments, and the reason for this cannot be explained.

Experiment 7. Sonoda and Makino (1980) infused 10 ml of a 50% glucose solution into the crop of splanchnicectomized and intact Leghorn cockerels. They concluded that food intake of the intact birds was inhibited more rapidly by the glucose infusions, and the duodenum or small intestine was sending satiety signals via the splanchnic nerve to the brain. In the present experiment, infusing volumes and concentrations of glucose intraduodenally at, above and below those used in their experiment, no significant difference between the food intake of splanchnicectomized and intact subjects. Data from one of these experiments (40% glucose, 5 ml volume) are presented in Table 7.

A closer examination of the study by Sonoda and Makino (1980) revealed that separate experiments were performed on their splanchnicectomized and sham-operated birds. Unfortunately the two groups of birds used as controls in these two experiments (which apparently were birds that had not undergone any type of surgery) responded quite differently in the two experiments. Therefore, their results appear to be open to alternative interpretations. Results of the present experiment show that intraduodenal glucose infusions do not affect food intake of splanchnicectomized birds any differently than intact birds.

General Discussion. Intraduodenal glucose infusions do not appear to affect food intake in the domestic fowl. This is true whether isotonic 5% glucose or more hypertonic 10% or 20% glucose solutions are infused. Hyperosmotic solutions infused into the duodenum of the chicken did inhibit food intake, especially in fasted SCWL chickens. The mechanism or physiological significance of this osmotic effect is not known and deserves examination. The present study could not confirm that splanchnicectomized birds responded any differently to intraduodenal glucose loads than sham-operated controls. Thus, it appears that the conclusion of Shurlock and Forbes (1981) that the duodenum of the fowl is not involved in food intake regulation through a gluco-sensitive mechanism is correct.

Nevertheless, the possibility of other visceral organs playing a role in appetite control has not been eliminated.

Table 1. Mean cumulative food intake (g) of ad libitum fed Single-Comb White Leghorn cockerels following intraduodenal infusions of 5% glucose (10 ml volume).

	Food intake (g)			
Treatments	30 min	60 min	90 min	120 min
Uninjected	5.5	8.2[ab]	10.2	12.8
Urea	7.0	9.9[a]	11.3	14.0
Saline	5.5	7.1[b]	8.7	11.1
Glucose	5.6	8.9[ab]	10.6	13.2
SEM[1]	0.5	0.6	0.6	0.8

Treatments within a column with differing superscripts are significantly different ($P \leq 0.05$).

[1]Standard error of the mean.

Table 2. Mean cumulative food intake (g) of ad libitum fed Single-Comb White Leghorn cockerels following intraduodenal infusions of 10% glucose (10 ml volume).

	Food intake (g)			
Treatments	30 min	60 min	90 min	120 min
Uninjected	6.5	9.6[a]	12.9	15.1
Urea	6.8	9.0[ab]	11.5	13.5
Saline	5.4	8.1[b]	11.2	13.9
Glucose	5.5	8.1[b]	10.1	11.9
SEM[1]	0.4	0.4	0.8	0.9

Treatments within a column with differing superscripts are significantly different ($P \leq 0.05$).

[1]Standard error of the mean.

Table 3. Mean cumulative food intake (g) of ad libitum fed Single-Comb White Leghorn cockerels following intraduodenal infusions of 20% glucose (10 ml volume).

	Food intake (g)			
Treatments	30 min	60 min	90 min	120 min
Uninjected	7.4	11.5	13.4	15.9[a]
Urea	8.0	11.1	12.2	14.0[ab]
Saline	5.8	8.8	9.1	10.4[b]
Glucose	5.9	8.9	10.2	11.9[ab]
SEM[1]	0.9	1.0	1.2	1.3

Treatments within a column with differing superscripts are significantly different ($P \leq 0.05$).

[1]Standard error of the mean.

Table 4. Mean cumulative food intake (g) of ad libitum fed Rock-Cornish (broiler) cockerels following intraduodenal infusions of 20% glucose (10 ml volume).

	Food intake (g)			
Treatments	30 min	60 min	90 min	120 min
Uninjected	6.5^{ab}	9.4^{a}	11.9^{a}	14.6^{a}
Urea	8.2^{a}	10.9^{a}	13.1^{a}	14.5^{a}
Saline	4.4^{b}	5.8^{b}	8.2^{b}	10.6^{b}
Glucose	8.0^{a}	10.5^{a}	11.8^{a}	14.5^{a}
SEM[1]	0.7	0.8	1.0	1.1

Treatments within a column with differing superscripts are significantly different ($P \leq 0.05$).

[1]Standard error of the mean.

Table 5. Mean cumulative food intake (g) of 24-hr fasted Single-Comb Leghorn cockerels following intraduodenal infusions of 20% glucose (10 ml volume).

	Food intake (g)			
Treatments	30 min	60 min	90 min	120 min
Uninjected	12.1[a]	18.1[a]	24.0	27.8
Urea	12.5[a]	17.2[a]	22.1	25.2
Saline	8.0[b]	12.4[b]	17.6	21.8
Glucose	12.0[a]	18.8[a]	23.1	27.9
SEM[1]	0.9	1.1	1.8	2.0

Treatments within a column with differing superscripts are significantly different ($P \leq 0.05$).

[1]Standard error of the mean.

Table 6. Mean cumulative food intake (g) of 24-hr fasted Rock-Cornish (broiler) cockerels following intraduodenal infusions of 20% glucose (10 ml volume).

	Food intake (g)			
Treatments	30 min	60 min	90 min	120 min
Uninjected	11.1	18.4	23.2	30.2
Urea	11.8	18.8	23.1	27.4
Saline	9.9	16.2	22.1	28.4
Glucose	10.9	18.2	23.8	28.9
SEM[1]	1.6	2.0	2.2	2.4

No significant treatment differences.

[1]Standard error of the mean.

Table 7. Mean cumulative food intake (g) of ad libitum fed splanchnicectomized and sham-operated control Single-Comb White Leghorn cockerels following intraduodenal infusions of 40% glucose (5 ml volume).

	Food intake (g)			
Treatments	30 min	60 min	90 min	120 min
Control	2.8	5.4	8.0	10.3
Splanchnicectomized	3.8	5.8	8.0	9.6
SEM[1]	0.6	0.7	0.8	0.9

No significant treatment differences.

[1]Standard error of the mean.

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CHAPTER 4

EFFECT OF INTRAHEPATIC GLUCOSE INFUSIONS ON FEEDING IN HEAVY AND LIGHT BREED CHICKS

INTRODUCTION

Several studies have demonstrated that food intake regulatory mechanisms in heavy and light breeds of chickens may differ (Denbow et al., 1981, 1982, 1983; Lacy et al., 1982; Burkhart et al., 1983). The majority of these studies have focused on central nervous system regulation. Although central mechanisms certainly play a critical role, peripheral aspects may also be important in short-term regulation of food intake.

Due to its strategic location and its role in the maintenance of blood glucose levels, the mammalian liver has received much attention as a possible site of food intake regulation. The hepatostatic theory of food intake was postulated by Russek (1963) as a system to regulate carbohydrate reserves. Glucose has been shown to inhibit food intake when infused into the hepatic circulation of rats (Campbell and Davis, 1974; Booth and Jarman, 1976), rabbits (Novin et al., 1974) and dogs (Russek, 1970). The validity of this theory, however, has been questioned by a number of investigators (Stephens and Baldwin, 1974; Bellinger et al., 1976; Strubbe et al., 1977; Woo et al.,

1979; Louis-Sylvestre, 1981). One of the more serious criticisms has been that inordinately large volumes or non-physiological concentrations of glucose have been necessary to successfully inhibit food intake via hepatic infusion or intraperitoneal injection (Bellinger, 1981).

Very little is known regarding the role peripheral regulatory mechanisms may play in food intake in the fowl. Recently, in a thorough series of experiments, Shurlock and Forbes (1981) showed that glucose solutions infused in large volumes into the hepatic circulation of an egg-laying strain of chicken decreased food intake.

The first objective of this study was to establish whether or not the liver functions in short-term regulation of food intake in the chicken when small volumes of isotonic glucose are infused directly into the hepatic circulation. The second objective was to investigate the possibility of differential sensitivities or responses to such glucose infusions in relatively fast-growing (broiler) and slow-growing (egg-laying) strains of chickens.

MATERIALS AND METHODS

Five separate experiments were performed. In all experiments the birds were housed in individual cages and exposed to continuous light. Food and water were available ad libitum, unless otherwise noted. Birds were fed chick

starter mash (20% crude protein, 2684 kcal/kg metabolizable energy) both prior to and during the experiments.

Experiment 1. The effects of intrahepatic glucose infusions on food intake were examined in free-feeding Single-Comb White Leghorn (SCWL) cockerels. Indwelling cannulae were implanted into the hepatic portal circulation of SCWL cockerels which were 12 weeks of age. Sodium pentobarbital (25 mg per kg body weight) was used to anesthetize birds prior to surgery.

Feathers were removed from the right abdominal region of the chicks, and a 6-cm incision was made between the cloaca and last sternal rib. The coccygeo-mesenteric vein was incised and a Silastic® cannula (i.d. 0.76 mm, o.d. 1.65 mm) was inserted and threaded toward the liver. The cannula was secured with ligatures such that the tip rested approximately 3 mm from the right hepatic portal vein, immediately adjacent to the liver. The extra-venal portion of the Silastic® cannula was encased in Tygon® tubing (i.d. 4.8 mm - o.d. 6.3 mm) for protection, passed under the skin and exteriorized in the thoracic region of the bird. Incisions were sutured with silk thread.

The protective Tygon® tubing and cannula were attached to a swivel system located at the top of the cage. This arrangement allowed infusions to be made without handling the subjects. The birds were allowed 4 to 6 days to recover

from surgery before the experiments began. Cannulae were flushed daily with heparinized (1000 USP units/ml) saline to insure patency.

Four treatments (10-ml of 5% glucose, 10-ml of 0.9% saline, 20-ml of 5% glucose and 20-ml of 0.9% saline) were tested in each of the eight SCWL birds using a replicated 4 x 4 Latin square design. All four solutions were isotonic at 300 mOsm/liter. Solutions were infused into the hepatic circulation over a 30-min period using a syringe pump. At least 24-hrs separated each test. Subjects had free access to food until the infusions began. Food was made available to the birds again as soon as the 30-min infusion ended. Feeders were weighed every 30 min for 3 hrs post-infusion. Analysis of variance was employed to determine bird, day and treatment effects, and Duncan's multiple range test was utilized to identify significant treatment differences.

Experiment 2. The effects of intrahepatic glucose infusions were examined in free-feeding Rock Cornish (RC) commercial broiler cockerels. The subjects were 9 weeks of age. Hepatic cannulae were implanted in eight RC cockerels using the method described in Experiment 1. The treatments and injection schedule used in Experiment 1 were also applied here. Again, a replicated 4 x 4 Latin square design was utilized and day, bird and treatment effects were examined by analysis of variance.

Experiment 3. The effects of intrahepatic glucose infusions were studied in 16-hr fasted SCWL cockerels. The birds were 15 weeks of age. The same cannulation procedure, injection schedule, treatments, design and analysis used in Experiment 1 were used here, except the birds were denied food for 16-hrs prior to infusion.

Experiment 4. The effects of intrahepatic glucose infusions were observed in 16-hr fasted RC cockerels. The subjects were 6 weeks of age. Again, the same methods, treatments, design and analysis employed in Experiment 1 were used in this experiment.

Experiment 5. The effects of intrajugular glucose infusions on the food intake of SCWL cockerels were tested. Eight SCWL cockerels 12 weeks of age served as subjects. Following anesthesia, feathers were removed from the right neck region of the birds, and a 5-cm longitudinal incision was made there. The right jugular vein was carefully dissected from the vagus nerve. A Silastic® cannula (i.d. 0.76 mm, o.d. 1.65 mm) was inserted into the vein, passed toward the heart to the level of the superior vena cava and secured. As in the hepatic experiments, the Silastic® cannula was encased in a protective Tygon® sheath, passed under the skin, exteriorized in the thoracic region and attached to a swivel system at the top of the cage. Again, incisions were sutured with silk thread, 4 to 6 days were

permitted for recovery and cannulae were flushed daily with heparinized saline.

Experimental design, treatments and infusion schedule were as described previously in the hepatic studies. The experiment was performed in free-feeding SCWL cockerels only. Rationale will be provided in the Results and Discussion.

RESULTS AND DISCUSSION

Experiment 1. In the free-feeding SCWL cockerels, both the 10-ml and 20-ml 5% glucose treatments caused a significant decrease in food intake when compared to the 10-ml saline treatment at all of the time periods, except 60 and 150 min (Table 1). Food intake was also depressed by the 20-ml saline treatment, however. Carpenter and Grossman (1983) recently reported a similar saline induced decrease in food intake in the rat. A saline mediated depression in food intake was cause for concern, because isotonic saline infusions were being utilized as a control in our experiments. Nevertheless, since the 10-ml glucose treatment significantly depressed food intake over and above the inhibition caused by the 10-ml saline treatment, it appears that glucose was very satiating.

Experiment 2. When glucose was infused into the liver of free-feeding RC cockerels, no effect was observed (Table

2). Other food intake studies have also shown this insensitivity in heavy breeds. Nir et al. (1978) found that the food intake of light breed chickens could be increased 70% over ad libitum fed levels by force-feeding techniques, while the food intake of heavy breed chickens could be increased only 13%. Lacy et al. (1982) found that in SCWL chicks, intragastric administration of tyrosine and tryptophan increased and decreased food intake, respectively, but had a lesser or no effect in broilers. These results strongly suggest that fast-growing birds are less sensitive to factors affecting food intake.

The specific reason that the broilers were unresponsive to hepatic glucose infusions is difficult to determine. Burkhart et al. (1983) found that lesions of the ventromedial hypothalamus caused hyperphagia in a genetically selected low-weight line of chickens (Siegel, 1978) but failed to do so in the selected high-weight line of birds. Based on these findings, it is possible that the broilers did not respond to glucose infusions in the present experiment because selection for rapid growth resulted in their hypothalamic appetite regulatory centers becoming less sensitive to signals from hepatic receptors. Denbow et al. (1981), however, found that epinephrine injected into the lateral cerebral ventricle of broilers stimulated feeding, indicating that at least some of the appetite regulatory

centers in the broiler are functional. Thus, the possibility also exists that the unresponsiveness noted in the broiler is due to peripheral insensitivity at the level of the hepatic receptors.

Experiment 3. Shurlock and Forbes (1981) reported that fasting of cockerels (egg-laying strain) for 21 hr accentuated the decrease in food intake observed following intrahepatic glucose infusions. Novin et al. (1974) concluded that hepatic-portal glucose was effective in reducing the food intake of deprived rabbits but not of free-feeding animals. In the present experiment, however, when the glucose infusions were tested in SCWL chicks that had been fasted for 16 hr, no difference between the glucose and saline infusions was found (Table 3).

Although these results appear to conflict with those of Shurlock and Forbes (1981) and Novin et al. (1974), others have reported similar findings. Robinzon and Snapir (1983) found intracerebroventricular glucose injections inhibited food intake in satiated White Leghorn cockerels but had no effect if the birds were fasted 24 hours. Latham and Blundell (1979) utilizing rats and Lacy et al. (1982) using chickens found that tryptophan inhibited feeding in free-feeding but had no effect in fasted animals. Denbow et al. (1982) obtained similar results when chickens were injected intracerebroventricularly with serotonin. These researchers

all suggested that a free-feeding animal was a more sensitive model for the testing of inhibitory food intake regulatory mechanisms.

Experiment 4. When the test solutions were infused in fasted RC cockerels, no effect was seen (Table 4).

Experiment 5. To determine if the inhibition of food intake in the free-feeding SCWL birds was attributable to hepatic glucose levels specifically, the infusions were repeated in birds fitted with jugular cannulae. Since only the free-feeding SCWL birds exhibited a response in the hepatic experiments, they were utilized as the experimental subjects. No differences in food intake were observed when glucose was infused intrajugularly (Table 5). Shurlock and Forbes (1981) showed this same lack of response in their experiment.

In summary, the results of these experiments demonstrated that relatively small volumes of isotonic glucose infused intra-hepatically inhibited food intake in free-feeding SCWL chicks but had no effect when administered intrajugularly. This finding supports the contention that an hepatic glucostatic food intake regulatory mechanism exists in light breed chickens (Shurlock and Forbes, 1981). In contrast to the results of Shurlock and Forbes (1981), food intake of fasted light breed chickens was unaffected by hepatic glucose infusions indicating that fasted animals are

less sensitive to factors that depress feeding. Heavy breed chicks, both free-feeding and fasted, were not influenced by the glucose infusions.

Table 1. Mean cumulative food intake (g) by ad libitum fed Single-Comb White Leghorn cockerels following intrahepatic infusions of glucose or saline.

Treatment	Food intake (g)					
	30 min	60 min	90 min	120 min	150 min	180 min
Glucose 20 ml	3.0[a]	4.6[a]	5.5[a]	7.0[a]	9.2[a]	10.6[a]
Saline 20 ml	4.4[a]	6.2[a]	7.0[ab]	7.6[a]	9.4[a]	10.4[a]
Glucose 10 ml	3.8[a]	5.0[a]	5.5[a]	6.2[a]	7.5[a]	8.1[a]
Saline 10 ml	6.4[b]	8.0[a]	9.5[b]	11.0[b]	13.7[a]	15.5[b]
SEM[1]	0.6	0.9	1.0	1.1	1.6	1.6

Means within a column with dissimilar superscripts are significantly different ($P \leq 0.05$).

[1]Standard error of the mean.

Table 2. Mean cumulative food intake (g) by ad libitum fed Rock Cornish cockerels following intrahepatic infusions of glucose or saline.

Treatment	Food intake (g)					
	30 min	60 min	90 min	120 min	150 min	180 min
Glucose 20 ml	9.6	13.2	15.5	18.1	20.4	26.2
Saline 20 ml	7.0	11.1	14.5	18.2	21.0	24.1
Glucose 10 ml	9.2	14.0	18.1	21.8	25.6	29.1
Saline 10 ml	10.8	13.2	17.8	21.6	23.9	26.9
SEM[1]	1.1	2.1	2.7	3.4	3.8	4.8

Treatments did not differ significantly ($P \leq 0.05$).

[1]Standard error of the mean.

Table 3. Mean cumulative food intake (g) by 16-hr fasted Single-Comb White Leghorn cockerels following intrahepatic infusions of glucose or saline.

Treatment	Food intake (g)					
	30 min	60 min	90 min	120 min	150 min	180 min
Glucose 20 ml	13.1	23.6	32.9	38.4	45.8	49.1
Saline 20 ml	13.1	23.5	33.4	39.5	46.5	48.8
Glucose 10 ml	11.4	20.2	27.9	34.1	39.1	44.0
Saline 10 ml	13.0	22.1	30.8	36.4	42.8	47.5
SEM[1]	1.0	1.8	2.3	2.6	3.2	3.2

Treatments did not differ significantly ($P \leq 0.05$).

[1]Standard error of the mean.

Table 4. Mean cumulative food intake (g) by 16-hr fasted Rock Cornish cockerels following intrahepatic infusions of glucose or saline.

Treatment	Food intake (g)					
	30 min	60 min	90 min	120 min	150 min	180 min
Glucose 20 ml	8.8	16.8	24.6	32.1	39.0	47.1
Saline 20 ml	10.5	17.0	23.2	31.6	39.4	47.1
Glucose 10 ml	10.8	20.0	26.5	34.2	40.8	48.6
Saline 10 ml	8.9	17.9	26.2	33.8	41.4	49.6
SEM[1]	0.6	1.2	1.5	1.7	1.5	1.8

Treatments did not differ significantly ($P \leq 0.05$).

[1]Standard error of the mean.

Table 5. Mean cumulative food intake (g) by ad libitum fed Single-Comb White Leghorn cockerels following intrajugular infusions of glucose or saline.

Treatment	Food intake (g)					
	30 min	60 min	90 min	120 min	150 min	180 min
Glucose 20 ml	2.9	3.1	3.1	3.1	3.2	3.4
Saline 20 ml	3.4	3.9	4.0	4.2	4.2	4.6
Glucose 10 ml	3.5	4.0	4.9	5.1	5.1	5.8
Saline 10 ml	3.4	4.0	4.1	4.5	4.5	4.6
SEM[1]	0.4	0.5	0.6	0.6	0.6	0.6

Treatments did not differ significantly ($P \leq 0.05$).

[1]Standard error of the mean.

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CHAPTER 5

FOOD INTAKE IN THE DOMESTIC FOWL: EFFECT OF INTRAHEPATIC INFUSION OF LIPID AND AMINO ACID SOLUTIONS

INTRODUCTION

The glucostatic (Mayer, 1953), lipostatic (Kennedy, 1950) and aminostatic (Smyth et al., 1947) theories of food intake state that the central nervous system monitors the concentration of particular nutrients and regulates appetite accordingly. During the last two decades, an increasing number of studies have shown that peripheral mechanisms may also be monitoring nutrient concentrations and aiding in the control of food intake (reviewed by Novin and VanderWeele, 1977).

It has been suggested that the mammalian liver has the ability to monitor blood glucose (Russek, 1963; Campbell and Davis, 1974; Booth and Jarman, 1976; Novin and VanderWeele, 1977) or some metabolite of the glycolytic chain such as pyruvate (Russek, 1981). Recently, hepatic glucose infusions were shown to inhibit food intake in the fowl (Shurlock and Forbes, 1981). Few studies have been reported on the capability of the liver to sense nutrients other than glucose, and these experiments have resulted in conflicting conclusions (Russek, 1970; Stephens and Baldwin, 1974; Bellinger et al., 1977).

The objective of the present study was to determine if peripheral food intake regulatory mechanisms sensitive to plasma amino acid or lipid concentrations exist in the liver of the domestic fowl.

MATERIALS AND METHODS

Experimental Design Five separate experiments were conducted. Eight birds were utilized in each experiment with a different group of birds being used for every experiment. In an experiment, four treatments were tested in each of the eight birds using a replicated Latin Square design. Tests were conducted between 0800 - 1200 hr with at least 24 hr separating a test. The treatments tested in the various experiments are described below. Birds were housed in individual cages, under continuous light and with food and water available ad libitum.

Experiment 1. Single-Comb White Leghorn (SCWL) cockerels 11 weeks of age were used as subjects. Indwelling Silastic® cannulae (i.d. 0.76mm, o.d. 1.65mm) were implanted into the hepatic portal circulation and exteriorized dorsally in the mid-thoracic region (as described in Chapter 4). Cannulae were attached to swivel systems at the top of each cage so that infusions could be made without handling the birds. The chicks were allowed 4 to 6 days to recover following surgery before the experiment was begun. Cannulae

were flushed daily with 1 ml of heparinized (1000 USP units/ml) saline to maintain patency.

A commercially prepared amino acid solution, 10% FreAmine III® (American McGraw), served as the stock solution from which the treatment solutions were prepared (see Table 1 for description of 10% FreAmine III®). The treatments were an uninjected control or 5 ml of either a 3.33% amino acid solution, a 1.67% amino acid solution, or a 1.01% saline solution. All solutions were isosmotic at 320 mOsm/liter.

The solutions were infused into the portal circulation of the birds via the hepatic cannulae over a 30-min period using a multi-channel syringe pump. The animals were provided food ad libitum prior to the infusion but were not fed during the infusion. They were given access to feed again immediately following the 30-min infusion.

Experiment 2. Single-Comb White Leghorn cockerels 11 weeks of age served as subjects. The effects of amino acids infused into the general circulation were tested in this experiment. Indwelling Silastic® cannulae (i.d. 0.76mm, o.d. 1.65mm) were implanted into the right jugular vein of each bird, passed under the skin and exteriorized in the mid-thoracic region (as described in Chapter 4). The injection procedures and treatments were the same as in Experiment 1.

Experiment 3. SCWL cockerels 11 weeks of age were fitted with hepatic cannulae as in Experiment 1. In this experiment the treatments were an uninjected control and 1 ml of a 20% lipid solution, 10% lipid solution, or 1.05% saline. The osmolarity of both lipid treatments and of the 1.05% saline solution was 340 mOsm/liter. A commercially produced lipid solution, 20% Liposyn® (Abbott Laboratories), was used for the 20% lipid treatment (see Table 2 for description of 20% Liposyn®). The 10% lipid treatment was obtained by mixing equal amounts of 20% Liposyn® and 1.05% saline. The solutions were infused into the hepatic circulation of the cockerels in the same manner as in Experiment 1.

Experiment 4. SCWL cockerels 11 weeks of age were fitted with jugular cannulae as in Experiment 2. The treatments used in Experiment 3 were then tested in these birds to determine the effects of lipids infused into the general circulation.

Experiment 5. Hepatic cannulae were implanted in commercial broiler Rock-Cornish (RC) cockerels 6 weeks of age. Experiment 3 was repeated in these birds to determine if lipid infusions would affect food intake in this heavy breed of bird. The volume of the infusions was increased in this experiment to 5 ml to compensate for the larger body weight of the broilers.

Data Collection and Analysis. Each experiment was analyzed separately. In all of the experiments cumulative food consumption was measured to the nearest g at 1 hr intervals for 3 hours. At each of the time periods orthogonal contrasts were used to test for linear and quadratic responses and to determine whether the saline and uninjected controls differed from each other. The saline and uninjected control treatments were combined when testing for the linear and quadratic responses. Since the RC birds grew at a much faster rate than the SCWL birds, it was necessary to test the lipid solutions on them at an earlier age. This resulted in age and stock being confounded. Similarly, the intrajugular and intrahepatic infusions could not be administered to the same birds; therefore, different groups of birds were used for these experiments. Nevertheless, it is doubtful that either of these problems had any significant impact upon the results of the study.

RESULTS AND DISCUSSISON

Experiments 1 and 2. Amino acid solutions, whether infused intrahepatically (Experiment 1) or intrajugularly (Experiment 2), had no effect on food intake in the SCWL cockerels (Table 3). The amino acid solutions used may seem to be relatively dilute (3.33% and 1.67%) and small in volume (5 ml), but the maximum concentration of amino acid

solution was dictated by osmolarity constraints. The stock 10% FreAmine III® solution had an osmolarity of 900 mOsm/liter, which is far in excess of avian blood (300 mOsm/liter). The strongly adverse effect hyperosmolarity has on food intake is well documented (McCleary, 1953; Bellinger et al., 1977).

In a preliminary experiment we tested a larger volume (10 ml) of the amino acid solutions. No significant effect was observed when 10 ml were administered intrahepatically; however, both of the amino acid solutions and the saline solution significantly decreased food intake compared to the uninjected control when 10 ml were infused intrajugularly. Such a volume effect has been observed previously (Carpenter and Grossman, 1983a). It was for this reason the smaller 5 ml volume was used in the present experiment.

The amount of amino acids in 5 ml of the 3.33% solution was calculated to be just slightly less than the amount of protein these birds consumed in their normal diet during an average 30 min period, which should be a sufficient quantity of amino acid to affect food intake if a physiological aminostatic mechanism exists in the liver.

Numerous studies have shown that amino acids influence food intake. For example, diets containing high concentrations of protein or excessive amounts of a single amino acid decreased food consumption in rats and chickens

(reviewed by Harper et al., 1970). Subcutaneous injections of amino acids in suckling rats (Mozes et al., 1983) and intragastric infusions of amino acids in suckling pigs (Houpt et al., 1983) depressed subsequent milk intake. Amino acids administered orally to adult rats depressed food intake (Geliebter, 1979).

Russek (1970) inhibited the food intake of rats with intraperitoneal injections of amino acids. In addition he was able to decrease feeding in fasted dogs by infusing ammonium chloride intraportally (Russek, 1971). He hypothesized that the liver was monitoring ammonium groups and could sense amino acids in the portal blood and regulate feeding. Rezek and Novin (1977) demonstrated that both casein and protein metabolites inhibited food intake in rats when infused intraportally. Nevertheless, the lack of an effect following hepatic amino acid infusions, as reported in our experiment, has also been reported previously. Stephens and Baldwin (1974) were unable to influence the food intake of pigs with intraportal infusions of amino acid solutions, even when large quantities were utilized. Similarly, Bellinger et al. (1977) failed to influence feeding in dogs with hepatic infusions of amino acids.

Stephens and Baldwin (1974), when relating their work in the pig to earlier studies by Russek (1970; 1971) in the dog, suggested that the omnivorous pig may not be as

sensitive to amino acids as the carnivorous dog. The chicken is also considered somewhat omnivorous, and, thus, the same reasoning may apply to the lack of an amino acid effect reported in the present experiment.

Experiment 3. When lipid was administered to SCWL cockerels via the hepatic cannulae, a significant linear response ($P \leq 0.05$) was demonstrated for cumulative food intake at the 2 and 3 hr readings (Table 4), indicating that intrahepatic lipid infusions inhibit food intake in a dose-dependent manner. The saline treatment did not affect food intake as compared to the uninjected control. These results indicate that the SCWL is sensitive to very small amounts of lipid administered intrahepatically.

Several studies in rats have also shown that peripherally administered lipids have a considerable influence on food intake. For example, it is believed that rats meter gastric triglyceride content (Deutsch and Gonzalez, 1980). Intravenous lipid injections decreased food intake in normal rats (Carpenter and Grossman, 1983a) and negative correlations have been observed between food intake and plasma fat metabolites in rats recovering from reversible obesity (Carpenter and Grossman, 1983b). Orally or intragastrically administered corn oil decreased food intake in rats (Ramirez and Friedman, 1983). Glick (1980), Brief and Davis (1982) and Wirtshafter and Davis (1977)

observed that glycerol decreased food intake in rats, and they suggested that glycerol might be the critical substance monitored by the central nervous system in regulating food intake and body weight. Dissenting views also exist, however (Ramirez and Friedman, 1982).

Differing effects of short-, medium- and long-chain triglycerides on food intake have received recent attention (Bach et al., 1980 Maggio and Koopsman, 1982; Friedman et al., 1983; Edens and Friedman, 1984). Short-chain triglycerides, which are absorbed and enter the portal circulation directly, inhibit the food intake of rats faster than medium-chain triglycerides. Medium-chain triglycerides in turn inhibit food intake faster than long-chain triglycerides, which in the mammal must be taken up by the lymphatic system before reaching the circulation. The delayed effect of the long-chain triglycerides suggests that circulating lipid levels do have an impact on food intake in mammals. In birds, short-, medium- and long-chain triglycerides are all absorbed directly into the portal circulation (Conrad and Scott, 1942; Bensadoun and Rothfeld, 1972); therefore, an examination of the effects of triglycerides of differing lengths on food intake might provide information concerning lipid mediated appetite regulation.

Not all food intake studies have shown that lipids inhibit feeding. Alimentary infusions of neutral fats did not influence food intake in the pig (Stephens, 1980). Geliebter (1979) found that fat administered intragastrically did not affect food intake as much as equicaloric loads of carbohydrate or protein. Circulating free fatty acid concentrations have been negatively correlated with satiety and positively correlated with hunger (Walker and Remley, 1970).

Heparin, which was used in our experiments to keep the hepatic and jugular cannulae open, can affect lipolysis (reviewed by Engelberg, 1977). Endogenous heparin appears to be an essential cofactor for the lipoprotein lipase enzymatic system which is the major physiological mechanism involved in removal of triglyceride from the blood. Therefore, it is possible that lipolysis and triglyceride removal were enhanced when cannulae were flushed with heparin. Nevertheless, since all subjects received identical heparin infusions, the lipid induced inhibition of feeding observed was likely due to the lipid treatment and not the heparin.

Experiment 4. Food intake of the SCWL cockerels was not affected by intrajugular lipid infusions (Table 4). Thus, lipid treatment that inhibited food intake when infused intrahepatically failed to do so when administered

intrajugularly. It would appear that the effect observed in Experiment 3 is specific to the site of infusion and that the liver of the fowl is capable of monitoring circulating lipid levels and regulating food intake accordingly.

Experiment 5. Food intake of RC chicks was not affected by intrahepatic lipid or saline infusions (Table 5). Even though the volume of lipid infused into the RC cockerels was five times the amount infused into the SCWL cockerels, no significant depression in food intake was observed (in a preliminary experiment 5 ml of 20% lipid decreased food intake in SCWL cockerels by 75%). The reason for this lack of effect is equivocal. It may be that the liver of the broiler is not responsive to lipid stimulation or that the central nervous system is insensitive to neural or humoral signals from the liver.

Several other studies have shown that appetite control mechanisms in fast-growing birds lack sensitivity. Nir et al. (1978) observed that food intake of ad libitum fed light-breed chicks could be increased 70% by force-feeding, but intake of heavy-breed chicks could be increased only 13%. Ventromedial hypothalamic lesions caused hyperphagia in genetically selected low weight line chickens but had no effect in high weight line chickens (Burkhart et al., 1983). Intragastric intubations of tyrosine and tryptophan increased and decreased food intake, respectively, in SCWL

cockerels; the same amino acid intubations had a lesser or no effect in broilers (Lacy et al., 1982). Glucose infused intrahepatically in SCWL chicks inhibited food intake while the same infusions in broilers did not (see Chapter 3).

Egg-type chickens are selected for high egg production at a minimal body weight while meat-type chickens are selected for increased body weight. Much of the improvements in body weight have come about through increased food consumption (reviewed by McCarthy and Siegel, 1983). Evidence of the difference in body weights between the two types of chickens is seen in this experiment; the RC chicks average body weight at 6 weeks of age was 1116 ± 68 g while the SCWL chicks at 11 weeks of age weighed 989 ± 55 g. The differences in feed consumption are as dramatic in that the RC controls consumed 36.6 g while the SCWL controls 11.2 g for the 3 hr time interval, demonstrating the great differences in feed intake.

Previous work has indicated the liver plays an important role in the regulation of food intake in the domestic fowl (Shurlock and Forbes 1981). The present experiments suggest that food intake of SCWL cockerels is not influenced by intraportal amino acid infusions. Feeding in SCWL cockerels, however, was significantly inhibited by hepatic portal infusions of lipid. Such an inhibition was not observed in SCWL cockerels infused with lipid

intrajugularly, suggesting that the liver is monitoring plasma lipid levels and adjusting appetite accordingly. Food intake of broiler chickens was not influenced by hepatic lipid infusions. This finding supports the accumulating evidence that genetic selection for increased body weight has desensitized appetite regulatory mechanisms.

Table 1. Composition of 10% FreAmine III® (American McGraw).[1]

Essential Amino Acids	mg per 1 ml 10% FreAmine®
L-isoleucine	6.90
L-leucine	9.10
L-lysine acetate	10.20 (7.25 free base)
L-methionine	5.30
L-phenylalanine	5.60
L-threonine	4.00
L-tryptophan	1.50
L-valine	6.60
Nonessential Amino Acids	
L-alanine	7.10
L-arginine	9.50
L-histidine	2.80
L-proline	11.20
L-serine	5.90
aminoacetic acid	14.00
L-cysteine $HCl \cdot H_2O$	≤0.24
Other	
Phosphoric acid	1.15
Sodium bisulfite (antioxidant)	≤1.00

[1]Acetic acid used to adjust pH to approximately 6.5. Osmolarity approximately 950 mOsm/liter.

Table 2. Composition of 20% Liposyn® (Abbott Laboratories).[1]

Components	mg per 1 ml 20% Liposyn®
Safflower oil[2]	200
Egg phosphatides (emulsifier)[3]	12
Glycerin	25

[1]Sodium hydroxide used to adjust pH to approximately 8.3. Osmolarity approximately 340 mOsm/liter.

[2]Safflower oil is a mixture of neutral triglycerides. The major component fatty acids are linoleic (77%), oleic (13)%, palmitic (7%) and stearic (2.5%).

[3]Egg phosphatides are primarily a mixture of phospholipids isolated from egg yolk. Emulsification results in fat particles approximately 0.4μ in diameter similar to natural occurring chylomicrons.

Table 3. Mean cumulative food intake (g) by ad libitum fed Single-Comb White Leghorn cockerels following intrahepatic and intrajugular infusions of FreAmine® (5 ml volume).

	Food intake (g)					
	Intrahepatic			Intrajugular		
Treatment	1 hr	2 hr	3 hr	1 hr	2 hr	3 hr
Uninjected	3.5	5.9	8.1	4.6	5.9	6.2
Saline	4.0	7.1	9.9	4.8	7.0	7.0
1.67% FreAmine®	4.4	6.8	10.1	4.8	7.1	7.6
3.33% FreAmine®	4.0	6.0	8.8	4.1	6.2	7.6
SEM[1]	0.8	1.1	1.4	1.0	1.6	1.3
----------F (1,18) Values----------						
Linear contrast[2]	0.51	0.14	0.05	0.23	0.02	0.42
Quadratic contrast[2]	0.00	0.51	2.67	0.05	0.32	0.21
Uninjected vs saline	0.11	0.23	1.72	0.01	0.47	0.18

[1]Standard error of the mean.

[2]Uninjected control and saline treatment were combined when testing for linear and quadratic responses.

Table 4. Mean cumulative food intake (g) by ad libitum fed Single-Comb White Leghorn cockerels following intrahepatic and intrajugular infusions of Liposyn® (1 ml volume).

	Food intake (g)					
	Intrahepatic			Intrajugular		
Treatment	1 hr	2 hr	3 hr	1 hr	2 hr	3 hr
Uninjected	5.1	8.4	11.2	3.5	8.0	9.8
Saline	5.1	7.9	9.6	3.9	6.4	8.5
10% Liposyn®	2.8	5.5	7.4	3.2	5.8	8.8
20% Liposyn®	3.0	4.1	4.9	4.6	6.1	8.0
SEM[1]	1.1	1.5	1.4	0.9	1.0	1.3
----------F (1,18) Values----------						
Linear contrast[2]	2.57	4.81*	10.32*	0.77	0.72	0.51
Quadratic contrast[2]	1.78	0.56	0.55	0.55	0.84	0.00
Uninjected vs saline	0.00	0.06	0.66	0.09	1.26	0.47

[1]Standard error of the mean.

[2]Uninjected control and saline treatment were combined when testing for linear and quadratic responses.

*Linear contrast was significant ($P \leq 0.05$) at this time period indicating that intrahepatic Liposyn inhibited food intake in a dose-dependent manner.

Table 5. Mean cumulative food intake (g) by ad libitum fed Rock-Cornish cockerels following intrahepatic infusions of Liposyn® (5 ml volume).

Treatment	Food Intake (g)		
	1 hr	2 hr	3 hr
Uninjected	20.0	29.0	36.6
Saline	24.9	35.0	44.5
10% Liposyn®	22.6	29.9	36.5
20% Liposyn®	20.1	27.5	35.2
SEM[1]	2.8	3.9	5.0
----------F (1,18) values----------			
Linear contrast	0.47	0.90	0.74
Quadratic contrast[2]	0.09	0.02	0.16
Uninjected vs. saline[2]	1.56	1.20	1.22

[1]Standard error of the mean.

[2]Uninjected control and saline treatment were combined when testing for linear and quadratic responses.

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CHAPTER 6

FOOD INTAKE RESPONSES IN SELECTED HIGH AND LOW-WEIGHT LINE COCKERELS TO PLASMA INFUSIONS FROM FASTED FOWL

INTRODUCTION

Numerous studies have indicated that a satiety factor exists in the blood of sated animals. For example, plasma from sated goats and sheep reduced feeding in hungry rats when injected intraperitoneally (Baile, 1971). In both rats (Davis et al., 1969) and sheep (Seoane et al., 1972), transfusions from sated animals reduced food intake of fasted animals. Furthermore, transfusions from 1-4 hr fasted rats into hungry recipients demonstrated that the longer the donors were fasted the less the food intake of recipients was depressed (Davis et al., 1971). Knoll (1979, 1984) isolated a component from human, rat, rabbit and goose serum purported to play an inhibitory role in the regulation of food intake. Skewes et al. (1984) demonstrated that a constituent of chicken plasma inhibits food intake in the fowl.

The existence of a blood-borne hunger factor to complement the well documented satiety factor is equivocal. Food intake was stimulated in sated sheep cross-circulated with blood from fasted sheep (Seoane et al., 1972), and

blood transfusions from 5-hr fasted rats stimulated the food intake of recipient rats (Davis et al., 1971). On the other hand, neither plasma from fasted sheep nor blood from fasted goats had an effect on food intake of fasted rats when injected intraperitoneally (Baile, 1971). Similarly, Davis et al. (1969) reported that food intake of sated rats could not be elevated by transfusions from fasted rats. Recently, Strickler (1984) reviewed the literature on hunger and satiety and suggested there may be no stimulus for hunger. He reasoned that hunger may be due to the disappearance of inhibitory factors.

The objective of this study was to determine if food intake of free-feeding chickens could be stimulated by intrahepatic infusions of plasma collected from fasted chickens. The experiment was performed with genetically selected high- and low-weight line birds to test for the possibility of a differential effect or a genetic predilection.

MATERIALS AND METHODS

Stock. The subjects used in the experiment were five high-weight (HW) line cockerels and five low-weight (LW) line cockerels 12 weeks of age. These birds were progeny from parent lines of White Plymouth Rock chickens that had undergone 26 generations of divergent selection for

body weight at 8 weeks of age (Dunnington and Siegel, 1985). Body weights of the cockerels at the time of the experiment were 347 ± 45 for the LW and 991 ± 125 for the HW line. Birds were housed in individual cages under continuous illumination with food and water available ad libitum.

Cannulizations. Birds were anesthetized with sodium pentobarbital (25 mg/kg body weight). Indwelling Silastic® cannulae (i.d. 0.76 mm, o.d. 1.65 mm) were implanted into the hepatic portal circulation and exteriorized dorsally in the mid-thoracic region (as described in Chapter 4). Cannulae were attached to swivel systems at the top of each cage so that infusions could be made without handling the birds. The cockerels were allowed 4-6 days to recover following surgery before the experiment was begun. Cannulae were flushed daily with 1-ml of heparinized saline (1000 USP units/ml) to maintain patency.

Treatments. Both the HW line and LW line birds were infused with four plasma treatments and a saline control via the intrahepatic cannulae. The plasma treatments were from ad libitum fed LW line chickens, 24-hr fasted LW line chickens, ad libitum fed HW line chickens and 24-hr fasted HW line chickens. The volume of the infusions was 7-ml for the LW line and 20-ml for the HW line cockerels (20 ml/kg average body weight). Infusions were made over a 30 minute period using a multi-channel syringe pump. Subjects were

provided access to food prior to infusion but were not fed during infusion.

Experimental Design and Analysis. The five treatments were tested in each of the five HW line birds and LW line birds using a Latin Square design. Tests were conducted between 0800-1200 hr with 24-hr separating each test. Food intake of the birds was measured hourly for 3-hr post-infusion. Cumulative food intake at each hr was tested within line for day, bird and treatment effects by analysis of variance. Duncan's multiple range test was used to examine significant ($P \leq 0.05$) treatment differences.

RESULTS AND DISCUSSION

No statistical differences in food intake were observed when the plasma treatments and saline were infused intrahepatically into LW line cockerels until 3-hr post-infusion (Table 1). At that time, food intake of LW line birds treated with plasma from fasted HW line birds was significantly higher ($P \leq 0.05$) than that of LW line birds infused with plasma from LW fed, HW fed or LW fasted birds. The saline treatment did not differ statistically from any of the plasma treatments. None of the plasma treatments had an effect on the food intake of HW line cockerels.

In several previous studies, attempts to modify feeding of sated animals with blood components from fasted donors

were unsuccessful (Davis et al., 1969; Baile, 1971). The results of the present experiment are in agreement with those of Seoane et al. (1972) who transfused plasma from fasted sheep into sated sheep and observed increased appetite in the sated group.

The failure of plasma from fasted subjects to enhance feeding when infused into HW birds was not surprising. It has been shown consistently that heavy-breed chickens are less susceptible to factors controlling food intake than light-breed chickens. Burkhart et al. (1983) demonstrated that ventromedial hypothalamic lesions caused hyperphagia in LW hens but had no effect in HW line hens. Lacy et al. (1982) found that tyrosine stimulated food intake in light-breed chickens but did not increase food consumption in heavy-breed birds. Nir et al. (1978) demonstrated that heavy-breed birds normally consume food at a volume close to the total capacity of their GI tract (87%), while light-breed birds eat more to satisfy metabolic needs and normally filled a relatively small percentage (30%) of their gastrointestinal tract capacity. Siegel (1978) concluded that genetic variation was still present in the HW line but that physiological limits were blocking its expression.

The major difference between feeding behavior in the HW and LW line birds has been reported to be that HW line birds consume more meals with a concomitant decrease in between

meal interval (Barbato et al., 1980). Thus, the HW birds appear to be in a rather constant state of hunger. This feeding characteristic causes one to speculate that if a "hunger" factor exists in the plasma of the fowl, it might be more prevalent in HW line chickens. Results of the present experiment where food intake of the LW line birds was stimulated by plasma from fasted HW line birds but was unaffected by plasma from fasted LW line birds indicate that a hunger factor (or absence of satiety factors) may be more concentrated in the plasma of HW line chickens. In addition, it appears the diminution of appetite regulatory mechanisms observed in heavy-breed birds is the result of receptor insensitivity rather than a lack of appropriate signals. This suggestion has been made in previous studies. Burkhart et al. (1983) proposed that selection for increased body weight had resulted in a desensitization of hypothalamic satiety mechanisms, and hepatic satiety mechanisms in heavy-breed birds have been purported to be less sensitive than those in light-breed birds (see Chapter 4).

The saline treatment was included in the present experiment to address the question of whether a hunger factor(s) exists in the plasma of fasted birds or whether appetite stimulation by such plasma is simply due to the absence of a satiety factor(s). If plasma from fasted birds

and saline affected food intake similarly, this would suggest that stimulation of food intake was caused by the lack of blood-borne satiety factors. On the other hand, if plasma from fed birds affected food intake similarly to saline, this would indicate that the stimulation was due to a hunger factor(s). Unfortunately in our experiment, food intake of the saline treated birds did not differ statistically from that of birds treated with the plasma from fasted or fed birds; therefore, the question cannot be answered at this time.

In conclusion, the results of this experiment demonstrate that some property of plasma from fasted birds stimulates food intake. The question of whether this stimulatory property is a humoral factor or whether it is simply that satiety factors are absent remains unanswered. If future studies determine that a factor exists that is specific for appetite stimulation; the possibilities of practical application may warrant examination.

Table 1. Mean cumulative food intake (g) by ad libitum fed high- and low-weight line cockerels following intrahepatic infusions of plasma from fed and fasted high-weight (HW) and low-weight (LW) line birds

	Food intake (g)					
	Recipient line					
	LW			HW		
Plasma donor	1 hr	2 hr	3 hr	1 hr	2 hr	3 hr
LW fasted	5.8	6.4	8.6[b]	18.4	26.0	29.0
LW fed	6.4	7.6	9.0[b]	18.4	24.6	26.6
HW fasted	8.4	11.4	13.6[a]	18.4	25.8	28.4
HW fed	5.8	7.8	9.6[b]	14.6	20.2	26.4
Saline	7.4	8.6	10.6[ab]	21.1	26.8	29.4
SEM[1]	1.1	1.1	1.1	2.1	2.3	2.6

Means within a column with differing superscripts are significantly different ($P \leq 0.05$).

[1]Standard error of the mean.

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GENERAL SYNTHESIS

The six chapters which comprise this dissertation examined various aspects of peripheral regulation of food intake in chickens. These experiments confirm that food intake control is multi-faceted.

Intravenous infusion of amino acid mixtures did not appear to affect appetite; however, peripheral administration of tryptophan did inhibit food intake. Intraperitoneal injections of tryptophan methyl ester caused a significant reduction in food intake of ad libitum fed SCWL cockerels. Since central administration of tryptophan methyl ester is known not to affect food intake in the fowl (D. M. Denbow, personal communication), it appears that tryptophan's effect on food intake is peripherally rather than centrally based as previously assumed. Intragastric intubations of tryptophan depressed both food consumption and body temperature further supporting the hypothesis that this amino acid is inhibiting food intake via peripheral mechanisms.

The duodenum was found not to play a significant role in food intake regulation in the fowl. The liver, on the other hand, was shown to be very sensitive to small loads of glucose or lipid. Intrahepatic glucose or lipid solutions significantly depressed food intake of Single-Comb White

Leghorn cockerels while intrajugular infusions of these substances did not. Thus, it appears that the liver of the Leghorn chicken is capable of monitoring circulating glucose and lipid levels and adjusting food consumption in respect to these nutrients. Intrahepatic glucose or lipid infusions had no affect on the food intake of Rock Cornish broiler cockerels. This finding supports the accumulating evidence that genetic selection for increased growth has affected appetite control systems.

The existence of an appetite stimulating factor in the plasma of food-deprived chickens was tested for by infusing plasma from ad libitum fed and 24-hr fasted high-weight (HW) and low-weight (LW) line donors into ad libitum fed HW and LW recipients. Infusions of plasma from fasted HW line chickens increased food intake of sated LW line recipients, suggesting that a "hunger" factor is present in the plasma of fasted birds that stimulates food consumption. Although this finding implies that LW line birds may be more sensitive to appetite control signals, it also indicates that hunger signals are not only present in HW birds but are perhaps more concentrated than in the LW line.

As happens so often in scientific research the results of these studies produced more questions than answers. Numerous factors appear to be involved in peripheral regulation of food intake and the significance and possible application of these factors remain to be determined.

The results of the tryptophan experiments provoke several inquiries. The gastro-intestinal tract is probably not the site where tryptophan is depressing appetite. Whether the liver is the sensitive site is not known and warrants further investigation. In addition to identifying the site of action, exploration of the mechanism by which tryptophan inhibits food intake could be worthwhile. The decrease in body temperature resulting from oral tryptophan administration might have application in heat stress situations where lowering body temperature would be advantageous. It would also be interesting to know what the effects of long-term consumption of a diet high in tryptophan would be on body weight, fat composition, and reproductive efficiency of both egg-laying and meat-type chickens

Although, the duodenum was ruled out as having an important function in food intake regulation, other portions of the gastro-intestinal tract may be involved in appetite control. Several studies have suggested that the crop may play an appetite regulatory role in birds (Polin and Wolford, 1973; Shurlock and Forbes, 1981). Recently the stomach has been shown to be sensitive to various nutrients and to regulate food intake accordingly in mammals (Deutsch, et al., 1978; Deutsch and Gonzalez, 1980). Perhaps the proventriculus serves a similar function in birds.

The considerable sensitivity of the liver of the Leghorn to lipid was unexpected and promotes speculation regarding the role of lipids in appetite regulation in this egg-laying breed of bird. Very high blood levels of lipid are characteristic of hens producing eggs and "fatty liver syndrome," where large amounts of fat accumulate in the liver, can be a serious problem in laying birds. How the normal and "fatty liver syndrome" laying hen respond to intrahepatic lipid infusions should be examined. Another area that may warrant study is the role of short-, medium- and long-chain triglycirides in food intake regulation and possible application of this information to body weight restriction situations.

Identification of the putative "hunger factor" in the plasma of fasted HW line cockerels would be useful. One might speculate that glucose, insulin or glucagon could be involved; however, analysis of these substances in fasted HW and LW line birds suggests that this is probably not the case (N. A. Sinsigalli, personal communication). Plasma lipid concentrations and neutral amino acid ratios (Anderson, 1979) should be looked at.

In conclusion, manipulation of food intake to increase or decrease growth in light-breed chickens like the SCWL are conceivable. In fast-growing fowl, such as the broiler and HW line bird, it appears that peripheral food intake

regulatory mechanisms are desensitized. Therefore, it is likely that manipulation of feeding behavior in meat-type fowl will prove difficult.

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APPENDIX A

Appendix A Table 1. Analysis of variance for food intake of fed Single-Comb White Leghorn chicks injected intraperitoneally with tryptophan methyl ester.

Source of variation	df	Sum of squares	F value
30 min			
Rep	3	23.79	1.10
Day	4	133.00	4.63
Bird (Rep)	16	235.76	2.05
Trt	4	120.90	4.21
Error	72	517.30	
Total	99	1030.75	
Linear contrast	1	96.60	13.45*
Quadratic contrast	1	14.50	2.02
60 min			
Rep	3	154.59	3.80
Day	4	118.86	2.19
Bird (Rep)	16	403.12	1.86
Trt	4	228.16	4.21
Error	72	976.18	
Total	99	1880.91	
Linear contrast	1	194.04	14.31*
Quadratic contrast	1	8.18	0.60
90 min			
Rep	3	165.39	2.50
Day	4	101.30	1.15
Bird (Rep)	16	383.36	1.09
Trt	4	289.90	3.29
Error	72	1584.80	
Total	99	2524.75	
Linear contrast	1	237.62	10.80*
Quadratic contrast	1	16.61	0.75

120 min			
Rep	3	182.51	2.22
Day	4	101.84	0.93
Bird (Rep)	16	470.08	1.07
Trt	4	282.94	2.58
Error	72	1976.42	
Total	99	3013.79	
Linear contrast	1	226.84	8.26*
Quadratic contrast	1	8.88	0.32
180 min			
Rep	3	359.63	3.25
Day	4	266.14	1.81
Bird (Rep)	16	589.36	1.00
Trt	4	404.54	2.75
Error	72	2651.72	
Total	99	4271.39	
Linear contrast	1	321.31	8.72*
Quadratic contrast	1	10.12	0.27
1440 min			
Rep	3	15249.40	20.18
Day	4	8084.14	8.02
Bird (Rep)	16	14422.64	3.58
Trt	4	3106.54	3.08
Error	72		
Total	99		
Linear contrast	1	1749.36	6.94*
Quadratic contrast	1	4.18	0.02

*Linear contrast significant P≤0.05.

Appendix A Table 2. Analysis of variance for food intake of fasted Single-Comb White Leghorn chicks injected intraperitoneally with tryptophan methyl ester.

Source of variation	df	Sum of squares	F value
30 min			
Rep	3	45.84	1.38
Day	4	127.24	2.88
Bird (Rep)	16	965.60	5.46
Trt	4	35.24	0.80
Error	72	796.32	
Total	99	1970.24	
Linear contrast	1	7.03	0.64
Quadratic contrast	1	3.65	0.33
60 min			
Rep	3	181.00	3.01
Day	4	131.26	1.64
Bird (Rep)	16	1219.76	3.80
Trt	4	77.56	0.97
Error	72	1443.98	
Total	99	3053.56	
Linear contrast	1	16.53	0.82
Quadratic contrast	1	19.56	0.98
90 min			
Rep	3	595.15	6.59
Day	4	392.54	3.26
Bird (Rep)	16	1309.44	2.72
Trt	4	48.94	0.41
Error	72	2167.72	
Total	99	4513.79	
Linear contrast	1	7.80	0.26
Quadratic contrast	1	17.49	0.58

120 min			
Rep	3	1132.40	8.17
Day	4	433.66	2.35
Bird (Rep)	16	1256.16	1.70
Trt	4	118.86	0.64
Error	72	3327.08	
Total	99	6268.16	
Linear contrast	1	8.00	0.17
Quadratic contrast	1	18.98	0.41
180 min			
Rep	3	1906.64	9.42
Day	4	1168.10	4.33
Bird (Rep)	16	1684.96	1.56
Trt	4	110.50	0.41
Error	72	4859.80	
Total	99	9730.00	
Linear contrast	1	5.78	0.09
Quadratic contrast	1	36.16	0.54
1440 min			
Rep	3	14653.00	15.03
Day	4	2649.04	2.04
Bird (Rep)	16	27498.64	5.29
Trt	4	917.14	0.71
Error	72	23391.02	
Total	99	69108.84	
Linear contrast	1	206.04	0.63
Quadratic contrast	1	0.60	0.00

*Linear contrast significant P≤0.05.

APPENDIX B

Appendix B Table 1. Analysis of variance for food intake and temperature change of Single-Comb White Leghorn intubated with tryptophan

Source of variation	df	Sum of squares	F value
	Food intake		
60 min			
Trt	1	57.80	7.84*
Error	18	141.20	
Total	19	199.00	
90 min			
Trt	1	145.80	13.96*
Error		188.00	
Total	19	333.80	
120 min			
Trt	1	115.20	6.53*
Error	18	317.60	
Total	19	432.80	
150 min			
Trt	1	266.45	12.75*
Error	18	376.10	
Total	19	642.55	
180 min			
Trt	1	304.20	12.09*
Error	18	453.00	
Total	19	757.20	
210 min			
Trt	1	441.80	13.42*
Error	18	592.40	
Total	19	1034.20	

		Temperature change	
30 min			
Trt	1	0.55	6.51*
Error	18	1.50	
Total	19	2.05	
60 min			
Trt	1	2.81	13.14*
Error	18	3.85	
Total	19	6.66	
90 min			
Trt	1	2.45	12.53*
Error	18	3.52	
Total	19	5.97	
120 min			
Trt	1	1.92	13.10*
Error	18	2.64	
Total	19	4.56	
150 min			
Trt	1	2.52	10.23*
Error	18	4.44	
Total	19	6.96	
180 min			
Trt	1	1.46	4.76*
Error	18	5.51	
Total	19	6.97	
210 min			
Trt	1	1.46	5.18*
Error	18	5.06	
Total	19	6.52	

*Treatments differ significantly $P \leq 0.05$.

APPENDIX C

Appendix C Table 1. Analysis of variance for food intake of fed Single-Comb White Leghorn cockerels infused intraduodenally with 6% glucose.

Source of variation	df	Sum of squares	F value
30 min			
Rep	1	0.78	0.36
Day	3	19.09	2.95
Bird (Rep)	6	11.19	0.86
Trt	3	12.84	1.99
Error	18	38.81	
Total	31	82.72	
60 min			
Rep	1	13.78	5.44
Day	3	57.84	7.62
Bird (Rep)	6	36.94	2.43
Trt	3	31.84	4.19*
Error	18	45.56	
Total	31	185.97	
90 min			
Rep	1	28.12	8.80
Day	3	94.25	9.83
Bird (Rep)	6	78.88	4.12
Trt	3	29.25	3.05
Error	18	57.50	
Total	31	288.00	
120 min			
Rep	1	16.53	3.36
Day	3	112.09	7.59
Bird (Rep)	6	78.69	2.67
Trt	3	35.59	2.41
Error	18	88.56	
Total	31	331.47	

*Treatments differ significantly $P \leq 0.05$.

Appendix C Table 2. Analysis of variance for food intake of fed Single-Comb White Leghorn cockerels infused intraduodenally with 10% glucose.

Source of variation	df	Sum of squares	F value
30 min			
Rep	1	11.28	6.81
Day	3	22.34	4.50
Bird (Rep)	6	7.94	0.08
Trt	3	11.59	2.33
Error	18	29.81	
Total	31	82.96	
60 min			
Rep	1	10.12	8.48
Day	3	101.75	28.40
Bird (Rep)	6	32.88	4.59
Trt	3	11.75	3.28*
Error	18	21.50	
Total	31	178.00	
90 min			
Rep	1	0.50	0.11
Day	3	144.62	10.30
Bird (Rep)	6	47.88	1.70
Trt	3	30.62	2.18
Error	18	84.25	
Total	31	307.88	
120 min			
Rep	1	0.03	0.00
Day	3	237.09	11.70
Bird (Rep)	6	45.94	1.13
Trt	3	43.09	2.13
Error	18	121.56	
Total	31	447.72	

*Treatments differ significantly $P \leq 0.05$.

Appendix C Table 3. Analysis of variance for food intake of fed Single-Comb White Leghorn cockerels infused intraduodenally with 20% glucose.

Source of variation	df	Sum of squares	F value
30 min			
Rep	1	60.50	8.75
Day	3	21.15	1.02
Bird (Rep)	6	68.00	1.64
Trt	3	29.75	1.43
Error	18	124.50	
Total	31	304.00	
60 min			
Rep	1	242.00	28.90
Day	3	16.12	0.64
Bird (Rep)	6	122.38	2.44
Trt	3	50.62	2.01
Error	18	150.75	
Total	31	581.88	
90 min			
Rep	1	288.00	26.89
Day	3	18.50	0.58
Bird (Rep)	6	122.50	1.91
Trt	3	88.25	2.75
Error	18	192.75	
Total	31	710.00	
120 min			
Rep	1	357.78	26.77
Day	3	17.84	0.45
Bird (Rep)	6	219.44	2.74
Trt	3	139.34	3.48*
Error	18	240.56	
Total	31	974.96	

*Treatments differ significantly $P \leq 0.05$.

Appendix C Table 4. Analysis of variance for food intake of fed Rock-Cornish cockerels infused intraduodenally with 20% glucose.

Source of variation	df	Sum of squares	F value
30 min			
Rep	1	2.53	0.59
Day	3	39.09	3.04
Bird (Rep)	6	48.69	1.90
Trt	3	76.09	5.92*
Error	18	77.06	
Total	31	243.46	
60 min			
Rep	1	32.00	5.57
Day	3	47.25	2.74
Bird (Rep)	6	45.50	1.32
Trt	3	131.25	7.61*
Error	18	103.50	
Total	31	359.50	
90 min			
Rep	1	66.12	7.56
Day	3	101.25	3.86
Bird (Rep)	6	113.88	2.17
Trt	3	105.25	4.01*
Error	18	157.50	
Total	31	544.00	
120 min			
Rep	1	55.12	5.93
Day	3	123.12	4.42
Bird (Rep)	6	118.25	2.12
Trt	3	92.12	3.30*
Error	18	167.25	
Total	31	555.88	

*Treatments differ significantly $P \leq 0.05$.

Appendix C Table 5. Analysis of variance for food intake of fasted Single-Comb White Leghorn cockerels infused intraduodenally with 20% glucose.

Source of variation	df	Sum of squares	F value
30 min			
Rep	1	3.78	0.60
Day	3	33.09	1.76
Bird (Rep)	6	91.19	2.42
Trt	3	107.34	5.71*
Error	18	112.81	
Total	31	348.22	
60 min			
Rep	1	32.00	3.17
Day	3	113.25	3.74
Bird (Rep)	6	245.00	4.05
Trt	3	201.75	6.67*
Error	18	181.50	
Total	31	773.50	
90 min			
Rep	1	116.28	4.75
Day	3	324.34	4.41
Bird (Rep)	6	411.94	2.80
Trt	3	192.84	2.62
Error	18	441.06	
Total	31	1486.46	
120 min			
Rep	1	215.28	6.45
Day	3	426.09	4.26
Bird (Rep)	6	645.19	3.22
Trt	3	197.84	1.98
Error	18	600.81	
Total	31	2085.22	

Appendix C Table 6. Analysis of variance for food intake of fasted Rock Cornish cockerels infused intraduodenally with 20% glucose.

Source of variation	df	Sum of squares	F value
30 min			
Rep	1	9.03	2.74
Day	3	42.34	4.28
Bird (Rep)	6	489.44	24.76
Trt	3	14.59	1.48
Error	18	59.31	
Total	31	614.72	
60 min			
Rep	1	52.53	12.64
Day	3	154.09	12.64
Bird (Rep)	6	776.94	31.16
Trt	3	30.34	2.43
Error	18	74.81	
Total	31	1088.72	
90 min			
Rep	1	171.12	18.93
Day	3	549.12	20.24
Bird (Rep)	6	877.75	16.18
Trt	3	11.12	0.41
Error	18	162.75	
Total	31	1771.88	
120 min			
Rep	1	185.28	9.53
Day	3	651.84	11.17
Bird (Rep)	6	1020.94	8.75
Trt	3	34.34	0.59
Error	18	350.06	
Total	31	2242.46	

Appendix C Table 7. Analysis of variance for food intake of splanchnicectomized and control Single-Comb White Leghorn cockerels infused intraduodenally with glucose.

Source of variation	df	Sum of squares	F value
30 min			
Trt	1	6.00	1.48
Error	22	89.33	
Total	23	95.33	
60 min			
Trt	1	0.67	0.12
Error	22	119.17	
Total	23	119.93	
90 min			
Trt	1	0.00	0.00
Error	22	224.00	
Total	23	224.00	
120 min			
Trt	1	3.38	0.29
Error	22	259.58	
Total	23	262.96	

APPENDIX D

Appendix D Table 1. Analysis of variance for food intake of fed Single-Comb White Leghorn cockerels infused intrahepatically with glucose.

Source of variation	df	Sum of squares	F value
30 min			
Rep	1	8.00	2.46
Day	3	34.25	3.51
Bird (Rep)	6	24.50	1.26
Trt	3	50.25	5.15*
Error	18	58.50	
Total	31	175.50	
60 min			
Rep	1	5.28	0.80
Day	3	157.09	7.95
Bird (Rep)	6	64.44	1.63
Trt	3	55.59	2.81
Error	18	118.56	
Total	31	400.97	
90 min			
Rep	1	10.12	1.28
Day	3	279.00	11.79
Bird (Rep)	6	90.88	1.92
Trt	3	85.50	3.61*
Error	18	142.00	
Total	31	607.50	
120 min			
Rep	1	22.78	2.50
Day	3	433.34	15.87
Bird (Rep)	6	129.44	2.37
Trt	3	105.59	3.87*
Error	18	163.81	
Total	31	854.97	
150 min			
Rep	1	91.12	4.74
Day	3	677.62	11.74
Bird	6	136.25	1.18
Trt	3	162.62	2.82
Error	18	346.25	
Total	31	1413.88	

180 min			
Rep	1	69.03	3.28
Day	3	1001.34	15.86
Bird (Rep)	6	171.44	1.36
Trt	3	231.59	3.67*
Error	18	378.81	
Total	31	1852.22	

*Treatments differ significantly P≤0.05.

Appendix D Table 2. Analysis of variance for food intake of fed Rock Cornish cockerels infused intrahepatically with glucose.

Source of variation	df	Sum of squares	F value
30 min			
Rep	1	38.28	3.62
Day	3	197.09	6.21
Bird (Rep)	6	121.19	1.91
Trt	3	59.34	1.87
Error	18	190.31	
Total	31	606.22	
60 min			
Rep	1	236.53	6.84
Day	3	216.34	2.09
Bird (Rep)	6	316.94	1.53
Trt	3	36.84	0.36
Error	18	622.06	
Total	31	1428.72	
90 min			
Rep	1	488.28	8.27
Day	3	280.09	1.58
Bird (Rep)	6	340.94	0.96
Trt	3	73.59	0.42
Error	18	1063.06	
Total	31	2245.97	
120 min			
Rep	1	648.00	7.17
Day	3	197.12	0.73
Bird (Rep)	6	449.38	0.83
Trt	3	98.12	0.36
Error	18	1627.25	
Total	31	3019.88	
150 min			
Rep	1	657.03	5.79
Day	3	203.84	0.60
Bird (Rep)	6	712.69	1.05
Trt	3	145.84	0.43
Error	18	2043.06	
Total	31	3762.47	

180 min			
Rep	1	657.03	3.52
Day	3	257.34	0.46
Bird (Rep)	6	976.94	0.87
Trt	3	101.59	0.18
Error	18	3356.81	
Total	31	5349.72	

Appendix D Table 3. Analysis of variance for food intake of fasted Single-Comb White Leghorn cockerels infused intrahepatically with glucose.

Source of variation	df	Sum of squares	F value
30 min			
Rep	1	34.03	4.27
Day	3	124.59	5.21
Bird (Rep)	6	619.44	12.94
Trt	3	17.59	0.74
Error	18	143.56	
Total	31	939.22	
60 min			
Rep	1	72.00	2.87
Day	3	409.75	5.21
Bird (Rep)	6	1256.50	12.94
Trt	3	59.25	0.74
Error	18	452.00	
Total	31	2249.50	
90 min			
Rep	1	108.78	2.48
Day	3	1145.09	8.70
Bird (Rep)	6	1599.94	6.08
Trt	3	150.34	1.14
Error	18	789.31	
Total	31	3793.47	
120 min			
Rep	1	258.78	4.92
Day	3	1386.59	8.78
Bird (Rep)	6	2297.69	7.27
Trt	3	134.09	0.85
Error	18	947.56	
Total	31	5024.72	
150 min			
Rep	1	442.53	5.53
Day	3	1642.84	6.84
Bird (Rep)	6	2531.69	5.27
Trt	3	270.09	1.12
Error	18	1440.81	
Total	31	6327.97	

180 min			
Rep	1	344.53	4.23
Day	3	1619.09	6.62
Bird (Rep)	6	2427.44	4.96
Trt	3	130.84	0.54
Error	18	1467.31	
Total	31	5989.22	

Appendix D Table 4. Analysis of variance for food intake of fasted Rock Cornish cockerels infused intrahepatically with glucose.

Source of variation	df	Sum of squares	F value
30 min			
Rep	1	9.03	2.68
Day	3	19.59	1.94
Bird (Rep)	6	310.69	15.39
Trt	3	26.59	2.63
Error	18	60.56	
Total	31	426.47	
60 min			
Rep	1	52.53	4.90
Day	3	31.09	0.97
Bird (Rep)	6	577.94	8.99
Trt	3	52.34	1.63
Error	18	192.81	
Total	31	906.72	
90 min			
Rep	1	175.78	9.79
Day	3	70.59	1.31
Bird (Rep)	6	659.19	6.12
Trt	3	55.34	1.03
Error	18	323.31	
Total	31	1284.21	
120 min			
Rep	1	242.00	11.01
Day	3	87.38	1.33
Bird (Rep)	6	956.88	7.26
Trt	3	38.12	0.58
Error	18	395.50	
Total	31	1719.88	
150 min			
Rep	1	288.00	15.25
Day	3	146.75	2.59
Bird (Rep)	6	1314.50	11.60
Trt	3	30.25	0.53
Error	18	340.00	
Total	31	2119.50	

180 min			
Rep	1	496.12	19.64
Day	3	286.75	3.78
Bird (Rep)	6	1715.88	11.32
Trt	3	36.00	0.47
Error	18	454.75	
Total	31	2989.50	

Appendix D Table 5. Analysis of variance for food intake of fed Single-Comb White Leghorn cockerels infused intrajugularly with glucose.

Source of variation	df	Sum of squares	F value
30 min			
Rep	1	0.30	0.03
Day	3	9.09	2.75
Bird (Rep)	6	7.69	1.16
Trt	3	1.84	0.56
Error	18	19.81	
Total	31	38.47	
60 min			
Rep	1	0.12	0.07
Day	3	16.75	3.14
Bird (Rep)	6	8.88	0.83
Trt	3	4.25	0.80
Error	18	32.00	
Total	31	62.00	
90 min			
Rep	1	0.03	0.01
Day	3	19.84	2.53
Bird (Rep)	6	19.69	1.25
Trt	3	12.34	1.57
Error	18	47.06	
Total	31	98.97	
120 min			
Rep	1	0.00	0.00
Day	3	16.50	2.16
Bird (Rep)	6	21.00	1.38
Trt	3	16.75	2.20
Error	18	45.75	
Total	31	100.00	
150 min			
Rep	1	0.03	0.01
Day	3	14.34	1.86
Bird (Rep)	6	21.19	1.37
Trt	3	14.59	1.89
Error	18	46.31	
Total	31	96.47	

180 min			
Rep	1	0.03	0.01
Day	3	13.34	1.33
Bird (Rep)	6	27.44	1.36
Trt	3	22.59	2.25
Error	18	60.31	
Total	31	123.72	

APPENDIX E

Appendix E Table 1. Analysis of variance for food intake of Single-Comb White Leghorn cockerels infused intrahepatically and intrajugularly with amino acids.

Source of variation	df	Sum of squares	F value
	Intrahepatic		
1 hr			
Rep	1	0.28	0.06
Day	3	33.84	2.30
Bird (Rep)	6	19.44	0.66
Trt	3	3.09	0.21
Error	18		
Total	31		
Linear contrast	1	2.52	0.51
Quadratic contrast	1	0.01	0.00
Uninjected vs. saline	1	0.56	0.11
2 hr			
Rep	1	6.12	0.62
Day	3	88.38	2.99
Bird (Rep)	6	53.25	0.90
Trt	3	8.62	0.29
Error	18	177.50	
Total	31	333.88	
Linear contrast	1	1.33	0.14
Quadratic contrast	1	5.04	0.51
Uninjected vs. saline	1	2.25	0.23
3 hr			
Rep	1	105.12	2.50
Day	3	1626.75	12.87
Bird (Rep)	6	1010.88	4.00
Trt	3	187.00	1.48
Error	18	758.25	
Total	31	3688.00	
Linear contrast	1	2.08	0.05
Quadratic contrast	1	112.67	2.67
Uninjected vs. saline	1	72.25	1.72

Intrajugular

1 hr			
Rep	1	10.12	1.39
Day	3	27.62	1.26
Bird (Rep)	6	122.75	2.81
Trt	3	2.12	0.10
Error	18	131.25	
Total	31	293.88	
Linear contrast	1	1.69	0.23
Quadratic contrast	1	0.37	0.05
Uninjected vs. saline	1	0.06	0.01
2 hr			
Rep	1	45.12	4.22
Day	3	19.38	0.60
Bird (Rep)	6	184.25	2.87
Trt	3	8.62	0.27
Error	18	192.50	
Total	31	449.88	
Linear contrast	1	0.19	0.02
Quadratic contrast	1	3.38	0.32
Uninjected vs. saline	1	5.06	0.47
3 hr			
Rep	1	18.0	1.42
Day	3	36.25	0.46
Bird (Rep)	6	170.50	0.87
Trt	3	10.25	0.18
Error	18	228.50	
Total	31	463.50	
Linear contrast	1	5.33	0.42
Quadratic contrast	1	2.67	0.21
Uninjected vs. saline	1	2.25	0.18

Appendix E Table 2. Analysis of variance for food intake of Single-Comb White Leghorn cockerels infused intrahepatically and intrajugularly with lipid.

Source of variation	df	Sum of squares	F value
	Intrahepatic		
1 hr			
Rep	1	3.12	0.33
Day	3	12.75	0.45
Bird (Rep)	6	12.88	0.23
Trt	3	40.75	1.45
Error	18	168.50	
Total	31	238.00	
Linear contrast	1	24.08	2.57
Quadratic contrast	1	16.67	1.78
Uninjected vs. saline	1	0.00	0.00
2 hr			
Rep	1	1.53	0.09
Day	3	54.34	1.02
Bird (Rep)	6	32.69	0.31
Trt	3	96.34	1.81
Error	18	319.06	
Total	31	503.97	
Linear contrast	1	85.33	4.81*
Quadratic contrast	1	10.01	0.56
Uninjected vs. saline	1	1.00	0.06
3 hr			
Rep	1	1.53	0.10
Day	3	34.09	0.71
Bird (Rep)	6	94.69	0.99
Trt	3	184.34	3.84
Error	18	287.81	
Total	31	602.47	
Linear contrast	1	165.02	10.32*
Quadratic contrast	1	8.76	0.55
Uninjected vs. saline	1	10.56	0.66

		Intrajugular	
1 hr			
Rep	1	24.50	4.03
Day	3	44.38	2.43
Bird (Rep)	6	67.88	1.86
Trt	3	8.62	0.47
Error	18	109.50	
Total	31	254.88	
Linear contrast	1	4.69	0.77
Quadratic contrast	1	3.38	0.75
Uninjected vs. saline	1	0.56	0.09
2 hr			
Rep	1	6.12	0.73
Day	3	36.38	1.45
Bird (Rep)	6	235.25	4.69
Trt	3	23.62	0.94
Error	18	150.50	
Total	31	451.88	
Linear contrast	1	6.02	0.72
Quadratic contrast	1	7.04	0.84
Uninjected vs. saline	1	10.56	1.26
3 hr			
Rep	1	12.50	0.95
Day	3	8.25	0.21
Bird (Rep)	6	307.00	3.88
Trt	3	13.00	0.33
Error	18	237.25	
Total	31	578.00	
Linear contrast	1	6.75	0.51
Quadratic contrast	1	0.00	0.00
Uninjected vs. saline	1	6.25	0.47

*Linear contrast significant P≤0.05.

Appendix E Table 3. Analysis of variance for food intake of Rock Cornish cockerels infused intrahepatically with lipid.

Source of variation	df	Sum of squares	F value
1 hr			
Rep	1	87.78	1.44
Day	3	162.84	0.89
Bird (Rep)	6	627.69	1.72
Trt	3	129.09	0.71
Error	18	1094.31	
Total	31	2102.72	
Linear contrast	1	28.52	0.47
Quadratic contrast	1	5.51	0.09
Uninjected vs. saline	1	95.06	1.56
2 hr			
Rep	1	731.53	6.12
Day	3	533.34	1.49
Bird (Rep)	6	1450.44	2.02
Trt	3	254.34	0.71
Error	18	2151.56	
Total	31	5121.22	
Linear contrast	1	108.00	0.90
Quadratic contrast	1	2.34	0.02
Uninjected vs. saline	1	144.00	1.20
3 hr			
Rep	1	1069.53	5.27
Day	3	720.34	1.18
Bird (Rep)	6	2087.69	1.72
Trt	3	430.09	0.71
Error	18	3649.81	
Total	31	7957.47	
Linear contrast	1	150.52	0.74
Quadratic contrast	1	31.51	0.16
Uninjected vs. saline	1	248.06	1.22

APPENDIX F

Appendix F Table 1. Analysis of variance for food intake of low- and high weight chickens infused with plasma from fed and fasted donors.

Source of variation	df	Sum of squares	F value
	High-weight recipients		
1 hr			
Day	4	168.64	1.84
Bird	4	186.64	2.04
Trt	4	104.24	1.14
Error	12	274.32	
Total	24	733.84	
2 hr			
Day	4	287.84	2.63
Bird	4	257.84	2.36
Trt	4	137.84	1.26
Error	12	327.92	
Total	24	1011.44	
3 hr			
Day	4	307.76	2.27
Bird (Rep)	4	472.96	3.49
Trt	4	38.16	0.28
Error	12	406.08	
Total	24	1244.96	

Low-weight recipients

1 hr			
Day	4	30.16	1.18
Bird	4	132.56	5.20
Trt	4	25.36	0.99
Error	12	76.48	
Total	24	264.56	
2 hr			
Day	4	31.76	1.31
Bird	4	185.36	7.67
Trt	4	70.16	2.90
Error	12	72.48	
Total	24	359.76	
3 hr			
Day	4	9.84	0.43
Bird	4	262.64	11.53
Trt	4	80.24	3.52*
Error	12	68.32	
Total	24	421.04	

*Treatments differ significantly $P \leq 0.05$.

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