

GLUCOSE TOLERANCE, PLASMA INSULIN, AND PLASMA  
GLUCAGON IN RELATION TO OBESITY IN CHICKENS

by

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## INTRODUCTION

Selection for increased body weight or increased growth rate has resulted in the excessive accumulation of body fat in broiler chickens. Excessive fat, particularly in the abdominal region, has obvious adverse effects on both production costs and consumer acceptability. The underlying mechanisms controlling fat deposition in the chicken, however, remain obscure.

Although selection for increased body weight is associated with increases in appetite, it is unlikely that increased fat deposition can be explained solely on the basis of food intake. Studies with mammalian models suggest that a number of metabolic defects associated with obesity develop prior to hyperphagia. Of these defects, abnormal glucose metabolism associated with hyperinsulinemia has been among the more consistently observed. The research reported in this thesis, therefore, was designed to compare glucose tolerance, plasma insulin, and plasma glucagon levels between fat and lean lines of chickens.



## REVIEW OF LITERATURE

Mammalian studies. The relationship between obesity and abnormal glucose metabolism was first observed in animals that were obese because of hypothalamic injury. Damage to the ventromedial hypothalamus results in increased food intake (Brobeck, 1946) and a gradual increase in body weight and body fat (Hetherington and Weil, 1940). Such animals have been shown to exhibit hyperinsulinemia which can be reduced to normal by fasting (Hales and Kennedy, 1964). Increased insulin release from the pancreas, however, occurs when caloric intake is restricted to that of normal animals (Goldman et al., 1974), indicating that the hyperinsulinemic response is not solely a function of food intake. Despite the hyperinsulinemia, blood glucose levels are often normal and a decreased responsiveness of muscle and liver tissue to insulin can be demonstrated several weeks after the onset of obesity (Le Marchand et al., 1978).

Hyperinsulinemia also appears to be consistently associated with genetic obesity. The autosomal recessive Zucker fatty rat (fa/fa), first described by Zucker and Zucker (1961), is hyperinsulinemic, hyperlipemic, and hyperphagic but has normal or only slightly elevated blood glucose levels (Stern et al., 1972; York et al., 1972). The normal blood glucose levels occurring concomitant with hyperinsulinemia has been attributed to insulin resistance.

The excessive adiposity of the Zucker fatty rat develops during the first week of life prior to the appearance of hyperphagia (Boulangue et al., 1979). Reductions in oxygen consumption and core temperature have also been observed at early ages (Planche et al., 1983) and it has been suggested that the increased adiposity is the result of a defect in energy expenditure. In addition, fatty rats exhibit an increase in lipoprotein lipase activity, which may contribute to fat cell hypertrophy observed during the development of obesity (Gruen et al., 1978).

Like the fatty rat, the obese mouse (ob/ob) is hyperinsulinemic (Bray and York, 1979). Plasma insulin increases by three to four weeks of age and reaches levels 50 times those of normal rats by six months of age; blood insulin levels subsequently decline at older ages. Unlike the Zucker fatty rat, however, obese mice are hyperglycemic as well as hyperinsulinemic, although it is unlikely that hyperglycemia is the cause of increased plasma insulin. Genuth (1969) reported that hyperinsulinemia occurred in the obese mouse regardless of whether blood glucose levels were elevated. Beloff-Chain et al. (1975) demonstrated that the intraperitoneal injection of glucose to obese mice resulted in a prolonged elevation of blood glucose which was associated with a decrease in circulating insulin levels.

It is unlikely that the hyperinsulinemia of obese mice is secondary to insulin resistance. Dubuc (1976) reported that altered pancreatic  $\beta$ -cell function, obesity, and abnormalities of somatic cell growth all preceded the onset of hyperglycemia and insulin resistance in obese mice. Insulin resistance, therefore, was apparently secondary to hyperinsulinemia. This conclusion was supported by studies showing that lowering serum insulin levels of obese mice by feed restriction (Cuendet et al., 1975), alloxan administration (Mahler and Szabo, 1971) or streptozotocin treatment (Batchelor et al., 1975) improved tissue sensitivity to insulin.

Although adult obese mice are hyperphagic, no differences in food intake between obese and normal mice occur at early ages (Lin et al., 1977). Yet, the young obese mouse is heavier and has much more body fat but less body protein than normal controls (Bergen et al., 1975; Lin et al., 1977). Hyperphagia, therefore, appears to be of secondary importance in the development of obesity in this recessive mutant. Indeed, the obese mouse resembles the fatty rat in that a thermogenic defect is detectable at very young ages (Kaplan and Leveille, 1974; Trayhurn et al., 1977; Thurlby and Trayhurn, 1978; Trayhurn and James, 1978).

Obesity has also been associated with hyperinsulinemia in diabetic mice (db/db). This autosomal recessive mutant

exhibits an overt obesity occurring shortly after weaning. A small increase in serum insulin has been detected as early as 10 to 12 days of age (Coleman and Hummel, 1974). Serum insulin increases slowly until weaning, then rises rapidly to levels six to ten times normal by two to three months of age. In some strains, the db allele results in severe hyperglycemia. In hyperglycemic strains, serum insulin tends to decrease after about three months of age and the animals lose weight before dying of diabetes at six to eight months of age. In other strains, hyperglycemia is less pronounced, the mice remain hyperglycemic, and the animals become grossly obese (Bray and York, 1979).

Hyperinsulinemia has also been documented in several milder forms of genetic obesity. Examples include the autosomal dominant yellow obese mouse ( $A^Y a$ ), the polygenic New Zealand obese mouse (NZO), and the Japanese KK mouse which probably exhibits a polygenic form of obesity (Bray and York, 1979). It is of interest that obesity appears to be most severe in recessive forms (ob/ob, db/db, and fa/fa). Hyperglycemia is severe in recessive forms of obesity in mice (ob/ob and db/db) but is not present in obese rats (hypothalamic obesity or fa/fa). The recently described  $db^{Pas}$  mouse, however, is an autosomal recessive mutant which exhibits gross obesity and severe hyperinsulinemia but normal or only slightly elevated blood glucose levels

(Aubert et al., 1985).

Bray and York (1979) postulated that control of insulin secretion by the central nervous system plays a pivotal role in the development of obesity. Hyperinsulinemia, which enhances lipogenesis and gluconeogenesis but decreases lipolysis, may have the net effect of shifting metabolism toward fat deposition at the expense of protein deposition. According to their hypothesis, the parasympathetic nervous system of individuals prone to obesity becomes hyperactive and exhibits exaggerated positive feedback responses. The sympathetic nervous system, in contrast, exhibits reduced responsiveness. Recent evidence for involvement of the central nervous system in the development of obesity was provided by observations that the hyperinsulinemia observed in preobese and obese fatty rats was partially mediated via the vagus nerve (Rohner-Jeanrenaud et al., 1983). Moreover, when diabetes was induced in both lean and obese rats by streptozotocin administration, insulin therapy resulted in equivalent weight gain and food intake (Stolz and Martin, 1982). These results suggest that increased insulin is required for the development of obesity. The obese rats, however, still exhibited higher percentages of body fat in comparison to the lean ones.

The relationship between glucagon secretion and obesity is not clear. Mayer (1953) first suggested that glucagon

might be involved in the etiology of obesity. Inoue et al. (1977) suggested that reduced activity of the sympathetic nervous system in hypothalamic-obese animals would result in increased plasma glucagon. Reports on plasma glucagon in response to lesioning of the ventromedial hypothalamus, however, have been variable. Inoue et al. (1977) found reduced levels of glucagon while Rohner (1978) found increased levels in hypothalamic-obese animals. Bernardis et al. (1977) made similar comparisons between lesioned and normal animals and found no differences in plasma glucagon. Comparisons of plasma glucagon levels between normal and genetically obese animals have also been inconsistent (Bray and York, 1979). Recent studies with humans, however, may explain some of these discrepancies. Plasma glucagon values differed among obese human subjects (Verga et al., 1984). In obese patients with normal glucose tolerance, glucagon values in plasma were reduced. In contrast, plasma glucagon values were higher in diabetic obese patients. Plasma glucagon responses, therefore, apparently differ with the specific type of obesity examined.

Avian studies. The regulation of glucose metabolism was once thought to differ markedly between birds and mammals. This conclusion was based on studies in which pancreatectomy had no effect on blood glucose or produced only a transient diabetes. It is now believed, however,

that the pancreatectomies in these early studies were only partial (see review by Sitbon et al., 1980). More recent experiments indicated that total pancreatectomy results in hypoglycemia in fasted-birds and hyperglycemia in fed-birds or in birds treated with glucose (Beeckman, 1956; Mialhe, 1958; Mikami and Ono, 1962; Sitbon, 1967). Differences in the control of plasma glucose by insulin and glucagon, therefore, are primarily quantitative rather than qualitative. The glucagon to insulin ratio, for example, is higher in birds than in mammals. Insulin, however, apparently lacks the antilipolytic properties in birds that it has in mammals (Sitbon et al., 1980).

In comparison to mammals, few models for studying obesity in birds have been discovered or developed. A mutant strain of chickens characterized by abnormally high fat deposition was described by Cole (1966). The obese birds were slightly smaller than normal, but differences in adiposity were observed at six to ten weeks of age. These obese chickens resembled obese rodents in their hypersensitivity to low environmental temperatures (van Tienhoven and Cole, 1962) and in their diminished sensitivity to insulin administration (Rudas et al., 1972). Higher plasma concentrations of glucose, total lipids, cholesterol, and phospholipids were also observed when obese chickens were compared to controls (Rudas et al., 1972).

The etiology of this syndrome appeared to be associated with hypothyroidism (Kite et al., 1969).

Selection has also been used to examine excessive fat deposition in domestic chickens. Lilburn et al. (1982a) divergently selected chickens for mature abdominal fat size. After eight generations of selection, significant differences in body weight and abdominal fat weight were apparent. Differences in body weight occurred before differences in feed intake, suggesting that differences in energy metabolism were associated with differences in fat deposition. Pair-feeding techniques, however, demonstrated that hyperphagia was a contributing factor. In a subsequent study (Lilburn et al., 1982b), it was revealed that hepatic lipogenesis of the obese chickens was elevated in comparison to values obtained with the lean birds. Plasma glucose, protein, triglyceride, and cholesterol concentrations did not differ between lines.

French workers also used polygenic methods to develop comparatively lean and fat chickens. Their approach produced fat (FL) and lean (LL) lines of similar body weight at 63 days of age (LeClercq et al., 1980). Selection resulted in substantial differences in body fat which were not accompanied by significant differences in feed intake. They suggested that the differences in adiposity were due to metabolic and hormonal effects rather than to differences in



the efficiency of energy utilization (Touchburn et al., 1981). No differences between lines were observed for maintenance energy requirements or for the efficiency of energy utilization (LeClercq and Saadoun, 1982). LeClercq (1983), however, reported that the LL chickens utilized dietary protein more efficiently than the FL birds. In addition, the FL chickens were characterized by comparatively high triglyceride, low amino acid, high phospholipid and high very low density lipoprotein concentrations in plasma. No significant differences between lines in body temperature were observed (Touchburn et al., 1981). Moreover, the FL chicken differed from most mammalian models of obesity in that the chicken did not become truly obese, hyperphagic or hyperinsulinemic and did not develop insulin resistance (LeClercq, 1984).

Based primarily on observations with the FL and LL lines of chickens, LeClercq (1984) proposed that an imbalance between plasma glucose and insulin, beginning very early in life, is responsible for obesity in chickens. According to this hypothesis, feeding induces a greater insulin release in obese chickens than in lean ones, causing a partition in metabolism favoring lipid synthesis at the expense of lean tissue growth. Data to support his hypothesis, however, are equivocal. Simon (1980) was unable to relate glucose tolerance to body fat content in

commercial broilers at two and four weeks of age. Simon and LeClercq (1982) observed lower plasma glucose in FL chickens than in LL ones, but these differences were not associated with differences in plasma insulin. During refeeding following a fast, however, plasma glucose levels were similar in both lines but plasma insulin was higher in FL than in LL chickens. In another study, differences in glucose tolerance between birds from these two lines were accompanied by consistent but nonsignificant differences in plasma insulin (Touchburn et al., 1981).

Direct selection for increased body weight almost invariably results in increased food intake and fat deposition (Lin, 1981; McLeod, 1982; McCarthy and Siegel, 1983). Chickens selected for high body weight exhibit many characteristics common to genetic obesity in rodents. Both metabolic differences and differences in feed consumption contribute to obesity due to selection, but hyperphagia is not necessarily a requisite (Nir, 1984). Reproductive difficulties are common (Siegel and Dunnington, 1985) and neuroendocrine mechanisms are apparently involved (Burkhart et al., 1983). Although lipogenesis may not be elevated in high-weight chickens, lipid turnover is reduced (Calabotta et al., 1983; 1985). No evidence has been obtained, however, showing that defective thermoregulatory capabilities are associated with obesity in high-weight

chickens (Dunnington and Siegel, 1984). Hormonal contributions to obesity in high-weight chickens have not been specifically examined.

## MATERIALS AND METHODS

Experimental subjects. The chicks used in this experiment were progeny from the S<sub>26</sub> generation of two lines developed from White Plymouth Rock germ plasm through divergent selection for high (HW) and low (LW) body weight at 56 days of age (Siegel, 1978). Contemporary progeny obtained from an F<sub>1</sub> cross (HL) between males from the HW line and females from the LW line as well as HW and LW chicks obtained from within line matings were used.

General procedures. The chicks were hatched, vaccinated for Marek's disease, and reared with sexes intermingled in litter-covered floor pens under a continuous photoperiod. A corn-soybean meal based diet adequate in all known nutrients was provided ad libitum.

At 21 days of age, 120 chicks from each of the three populations (HW, LW, HL) were sexed, wingbanded, and weighed. Within populations, the chicks were randomly divided into six groups of equal number and their plumage partially dyed to allow identification by groups. Each group was then randomly assigned for blood collection at either 0, 20, 40, 60, 80, or 100 minutes after the oral administration of glucose (2 g/kg body weight). All birds were fasted for approximately 24 hr prior to glucose administration. The groups designated to be bled at 0 minutes were, of course, not administered glucose.

Approximately 2 ml of blood were collected from each chick via the brachial vein.

Following blood collection, the chicks were returned to their assigned pens and maintained under the conditions described previously. At 42, 63, and 84 days of age, the groups were rerandomized and the procedures were repeated.

Plasma glucose, insulin and glucagon assays. All blood was collected in tubes containing sodium-heparin to prevent coagulation and sodium fluoride to inhibit glycolysis. The samples were placed on ice immediately after collection. Plasma was separated by centrifugation (1000 x g). Individual plasma samples were refrigerated overnight and then divided into three tubes for the subsequent determination of glucose, insulin, and glucagon concentrations. The samples for glucagon determinations were treated with a protease inhibitor (Trasyolol) to prevent glucagon degradation. Samples for glucagon and insulin assays were then frozen.

Plasma glucose was assayed in the Virginia Tech Poultry Science Department laboratories using the glucose oxidase method described by Raabo and Terkildsen (1960). Insulin and glucagon determinations were conducted at the Agricultural Research Center, United States Department of Agriculture in Beltsville, Maryland. The radioimmunoassay for insulin was developed by McMurtry et al. (1983) and

that for glucagon was described by Allen and McMurtry (1984).

Statistical analyses. The data were analyzed, within ages, by analyses of variance with population, sex, and sampling time (minutes after glucose administration) considered to be fixed effects. Prior to analysis, the insulin and glucagon values were subjected to square root transformations. The statistical model used was:

$$Y = \mu + P_i + S_j + T_k + (PS)_{ij} \\ + (ST)_{jk} + (PT)_{ik} + (PST)_{ijk} + e_{ijkl}$$

where  $i = 1, 2, 3$  populations,  $j = 1, 2$  sexes,  $k = 1, 2 \dots$   
 $\dots 6$  sampling times, and  $l = 1, 2 \dots n$  individuals. When significance was obtained, means were separated by Duncan's multiple range test.

## RESULTS AND DISCUSSION

Body weight. As anticipated, chicks from the HW line were much larger than those from the LW line, while the crosses were intermediate in weight (Table 1). Although sexual dimorphism for body weight existed, no effects of sex on any of the other traits examined were obtained. The results, therefore, were presented with sexes pooled.

Plasma glucose. Means and standard errors for plasma glucose at the various ages examined are presented in Tables 2-5. With the exception of the LW chicks at 21 days of age (Table 2), the chicks exhibited the expected increases in plasma glucose in response to the oral administration of glucose. Significant population by sampling time interactions, however, indicated that glucose homeostasis differed among populations.

At 21 days of age, chicks from the LW line had higher fasting plasma glucose levels than the HW or HL chicks (Figure 1). The LW chicks also exhibited fasting plasma glucose values higher than those for the HL chicks at 63 days of age. Fasting plasma glucose levels at 42 and 84 days of age were remarkably constant among populations. Regardless of population, fasting plasma glucose levels tended to decrease with age.

Although recessive forms of obesity in mice (ob/ob, db/db) are associated with severe hyperglycemia (Bray and

York, 1979), increased plasma glucose does not universally accompany obesity. The obese Zucker (fa/fa) rat is normoglycemic or exhibits only slightly elevated levels of plasma glucose (Stern et al., 1972; York et al., 1972). In less severe forms of genetic obesity in mice (NZO, KK, A<sup>Y</sup>a), hyperglycemia is moderate or lacking (Bray and York, 1979). Blood glucose levels in obese animals with lesions of the ventromedial hypothalamus are similar to those of controls (Steffens, 1970; Goldman et al., 1974).

In studies with chickens, heavy breeds have been shown to have higher plasma glucose levels than White Leghorns (Rudas et al., 1972; March, 1984). Comparisons of relatively fat and lean chickens within meat-type stocks, however, have failed to show that hyperglycemia is associated with obesity. Lilburn et al. (1982b) found no differences in plasma glucose between chickens from lean and fat lines. When chickens developed through selection for high (FL) and low (LL) abdominal fat weights were compared, plasma glucose was lower in the FL line (Touchburn et al., 1981; Simon and LeClercq, 1982). Comparisons between chicks from the HW and LW lines used in the present study have shown either no differences in plasma glucose or elevated levels in the LW chicks (Calabotta et al., 1985; Dunnington et al., 1985). Hyperglycemia, therefore, is apparently not characteristic of obesity in chickens.



Because of the general association between obesity and impaired glucose tolerance in mammals (Bray and York, 1979), the responses of the HW, HL, and LW chicks to the oral administration of glucose were compared at several ages. The dose administered, 2 g/kg body weight, was originally used in chickens by Simon (1980). Preliminary experiments showed that this dosage increased plasma glucose shortly after being administered to chicks from these particular populations.

With the exception of the LW chicks at 21 days of age, plasma glucose levels of chicks from all three populations increased significantly in response to glucose administration (Tables 2-5 and Figure 2). One possible explanation for the failure to observe a plasma glucose increase in the 21-day old LW chicks is that a peak in plasma glucose occurred in these chicks prior to the first sampling time 20 minutes postadministration. This would be supported by the observation that LW chicks have a higher rate of glucose uptake when compared to HW chicks (Walker et al., 1981). Alternatively, the failure to detect a peak in plasma glucose in these chicks could have been due to a false high value for fasting plasma glucose in the LW chicks (Figure 1).

At all ages, the glucose tolerance curves for the HW chicks, in comparison to those from the LW line (Figure 2),

were characteristic of a diabetic-like condition (Ganong, 1977). At all ages, the peak in plasma glucose in response to glucose administration was higher in the HW than in the LW chicks. Plasma glucose also tended to return to fasting levels more rapidly in the LW than in the HW chicks following glucose intubation. The HL chicks exhibited similar but less pronounced responses as those exhibited by the HWs.

The glucose tolerance responses observed, which were similar to responses reported for lean and obese rodents (Bray and York, 1979), were inconsistent with results obtained with the French FL and LL lines. Simon and LeClercq (1982) observed no differences in peak plasma glucose levels between FL and LL birds during refeeding or force-feeding following a fast. The FL birds also exhibited faster glucose disposal rates than the LL birds until 57 days of age, after which the rates were similar (Touchburn et al., 1981; Simon and LeClercq, 1982). It was concluded that the FL chicken differed from the obese Zucker rat in that increased fat deposition was not associated with impaired glucose tolerance (LeClercq, 1984). The results reported here, however, indicated that increased fat deposition due to selection for rapid growth was associated with impaired glucose tolerance. Moreover, it may be relevant that increased fat deposition in the FL line does

not develop into a true obesity in mature individuals (LeClercq, 1984), while selection for increased growth does (McCarthy and Siegel, 1983).

Plasma insulin. At 21 days of age, no significant differences in plasma insulin levels were obtained (Table 6). However, observation numbers at this age were limited. Insulin levels in many of the plasma samples were below the sensitivity of the assay (19 pg/ml). In addition it was not possible to collect sufficient blood for assay from some of the individuals at this age.

Fasting levels of plasma insulin did not differ significantly among populations at any age examined (Tables 6-9). Although obese mammals are consistently hyperinsulinemic (Bray and York, 1979), the hyperinsulinemia is not always apparent under fasting conditions. Fasting reduces plasma insulin levels to normal in hypothalamic-obese animals and in some forms of genetic obesity (Bray and York, 1979), but insulin remains elevated under fasted conditions in other models of genetic obesity (Beloff-Chain et al., 1975; Lavine et al., 1975). With chickens, no significant differences in fasting plasma insulin levels were observed between FL and LL chicks (Touchburn et al., 1981; Simon and LeClercq, 1982) and LeClercq (1984) concluded that plasma insulin in the FL chicks was normal or only slightly elevated in either fed or fasted states.

At 42 and 63 days of age, significant population by sampling time interactions indicated that plasma insulin responses differed among populations (Tables 7 and 8). For the HW and HL chicks, significant increases in plasma insulin were observed 20 minutes after glucose administration (Figure 3). This increase persisted through 40 minutes but returned to fasting levels by 60 minutes postadministration. In the LW chicks at these ages, no significant increases in plasma insulin were observed in response to glucose administration.

In contrast to the results at 42 and 63 days of age, changes in plasma insulin in response to glucose intubation did not differ significantly among the HW, HL, and LW chicks at 84 days of age; the sampling time by population interaction was not significant (Table 9). All populations exhibited pronounced increases in plasma insulin after glucose was administered, with peak values occurring 40 minutes postadministration (Figure 3). Values for plasma insulin 60, 80, and 100 minutes postadministration did not differ significantly from fasting values (Table 9).

An increased insulin release in response to a meal or a glucose load has been associated with both genetic (Stern et al., 1972; Bryce et al., 1977), and hypothalamic obesity (Steffens, 1970; Inoue et al., 1977). The results reported here indicate that, at least at ages up to 63 days, HW

chicks exhibited similar hyperinsulinemic responses. Moreover, the present results were similar to those reported for the French FL and LL lines, where FL chicks exhibited significantly higher plasma insulin than LL chicks in response to a meal or a glucose load up to 56 days of age (Touchburn et al., 1981; Simon and LeClercq, 1982). At 17 weeks of age, however, no differences between FL and LL chickens were observed. No differences in plasma insulin levels were observed among HW, HL, and LW chicks at 84 days of age (Table 9).

Plasma glucagon. Values for plasma glucagon at the various ages measured are presented in Tables 10-13. Significant differences among populations in response to glucose administration were observed, but the lack of population by sampling time interactions indicated that changes in plasma glucagon levels resulting from glucose intubation were similar among populations.

At all ages, plasma glucagon levels were higher in the HW and HL chicks than in chicks from the LW line. No differences in plasma glucagon between the HW and HL chicks, however, were obtained at any age. These population differences were consistent in fasted states and at the various times examined subsequent to glucose intubation.

Comparisons of plasma glucagon concentrations between lean and obese individuals of several species have been

inconsistent. Plasma glucagon levels have been reported to be higher in obese than in control mice (Lavine et al., 1975; Mahler et al., 1976) lower in fasted obese Zucker (fa/fa) rats in comparison to fasted lean rats (Eaton et al., 1976; Bryce et al., 1977), and not different between obese and non-obese humans (Kalkhoff et al., 1973; Schade and Eaton, 1974; Verga et al., 1984). Comparisons of plasma glucagon levels between fat and lean chickens have not been previously reported.

Although significant differences in plasma glucagon resulted from glucagon intubation at 42, 63, and 84 days of age (Tables 11-13), the responses varied at different ages (Figure 4). These variable responses followed no consistent trends, making interpretation difficult. Glucose intubation would be expected to decrease plasma glucagon levels (Samols et al., 1969; Karmann and Mialhe, 1976). Only at 63 days of age, however, was such a pattern apparent although a small but significant reduction in plasma glucagon did occur shortly after glucose administration to fasted chicks at 84 days of age (Table 13).

Interrelationships. The principal objective of this study was to determine whether excessive fat deposition in chickens developed through selection for rapid growth was associated with impaired glucose tolerance and hyperinsulinemia. Studies with both mammalian (Bray and

York, 1979) and avian (LeClercq, 1984) models suggest that hyperinsulinemia is a key factor in the development of obesity. Because of the relationships between insulin and glucagon in regulating carbohydrate metabolism, plasma glucagon concentrations were also measured.

When glucose was administered orally to fasted chicks from the LW line at 21 days of age, no significant increase in plasma glucose was observed (Table 2). At older ages, glucose intubation resulted in significant increases in plasma glucose in the LW chicks (Tables 3-5), but the increases were less pronounced than the increases observed in the HW chicks (Figure 2). The LW chicks also tended to clear excess glucose from the blood more rapidly than the HW chicks. This enhanced ability of the LW chicks, in comparison to those from the HW line, to metabolize glucose was not associated with comparable increases in the levels of circulating insulin (Figure 3). Only at 84 days of age did a significant increase in plasma insulin occur in the LW chicks in response to glucose intubation (Table 5).

Subsequent to 21 days of age, glucose intubation into fasted HW chicks stimulated insulin release into the circulatory system (Figure 2). Although the plasma of the HW chicks contained as much or more insulin as their LW counterparts, the HWs still exhibited impaired glucose tolerance (Figure 2). It appeared, therefore, that the

sensitivity of peripheral tissue to insulin activity was diminished in the HW chicks.

The results obtained with the HL chicks were also suggestive of insulin resistance. The HL chicks consistently secreted more insulin into the blood in response to glucose intubation than did the LWs (Figure 3), but the LW chicks were better able to maintain appropriate levels of blood glucose (Figure 2). Moreover, the HL chicks generally exhibited glucose tolerance responses intermediate to those for the HW or LW chicks (Figure 2), but plasma insulin responses more closely resembled those of the HW chicks (Figure 3). The intermediate responses of the crosses would suggest additive genetic effects for glucose tolerance, while the preponderance towards the HW line suggests dominance for insulin response.

Plasma glucagon has not been examined in relation to adiposity in chickens. Unfortunately, the only consistent observation in this study was that plasma glucagon was higher in the HW than in the LW chicks (Tables 10-13). Plasma glucagon levels in the crosses (HL) were similar to those of the HWs, suggesting nonadditive genetic effects for this trait. The significance of these differences in plasma glucagon is not known.

Glucagon displays a potent action in birds. Minute amounts increase blood glucose and free fatty acids through



the stimulation of gluconeogenesis and lipolysis (Hazelwood, 1976). The higher levels of circulating glucagon in the HW chicks may have contributed to the increase in plasma glucose observed in response to glucose intubation. The high plasma glucose levels in diabetic individuals, for example, is probably the result of increased glucagon secretion as well as the failure of glucose to be cleared from the blood because of insulin insufficiency (Ganong, 1977). The increased plasma glucagon levels in the fasted HW chicks, however, did not result in increases in fasting levels of plasma glucose (Figure 1).

Regardless of population, plasma glucagon levels in response to glucose administration were erratic and inconsistent (Figure 4). The available data for birds indicate that both glucose and insulin administration decreases glucagon secretion (Karmann and Mialhe, 1976; Sitbon and Mialhe, 1978). Only at 63 days of age, however, did plasma glucagon levels decrease substantially in response to glucose intubation, and this decrease did not occur until 60 minutes postadministration. Similarly, plasma glucagon levels did not appear to be consistently influenced by plasma insulin levels. Moreover, when insulin-glucagon ratios were examined (data not presented), these ratios did not appear to be associated with the differences in plasma glucose observed between lines. The

metabolic effects of glucagon in maintaining glucose homeostasis in the fowl, therefore, appear to be worthy of further investigation.

General discussion. Based primarily on comparisons between chickens developed through selection for high (FL) or low (LL) abdominal fat weights, LeClercq (1984) proposed that an imbalance between plasma glucose and insulin, beginning very early in life (Simon and LeClercq, 1982), contributes to obesity in the chicken. According to his hypothesis, insulin release in fat chicks following a meal or glucose load is greater than that induced in lean ones. The higher insulin secretion causes metabolism to shift in favor of lipid synthesis. The results reported in this thesis were generally supportive of this concept.

LeClercq (1984) also compared the Zucker rat (fa/fa) to the FL chicken and concluded that the FL chicken differs in that it does not become truly obese and does not exhibit hyperphagia, hyperinsulinemia, insulin resistance, or excessive weight gain. He hypothesized that genes for true obesity were absent from his original stock, or there is some form of metabolic regulation that prevents the development of true obesity in the chicken. The inference is that excessive fat deposition in chickens differs in nature from obesity in mammals. The results reported here were not consistent with this concept.

In critiquing LeClercq's (1984) conclusion that obesity in birds differs from that in mammals, it is important to consider the basis of selection for the FL and LL chickens. The FL chicken was developed through selection for abdominal fat weight (LeClercq, 1984). Growth rate was not a selection criterion, but rather was restricted by selection. Most meat-type chickens, however, have been developed through selection for rapid growth and increased body fat has accompanied the increased growth rate (Siegel, 1984). It is a common practice in poultry husbandry to restrict the feed of reproductive populations of meat-type chickens to alleviate reproductive difficulties associated with excessive fat deposition (McCarthy and Siegel, 1983). It appears, therefore, that meat-type chickens do exhibit true obesity and that the FL chicken is atypical of meat-type chickens in general.

Although LeClercq (1984) demonstrated that the FL chicken is not hyperphagic, increased body weights in chickens due to selection for rapid growth are highly correlated with increases in feed intake (McCarthy and Siegel, 1983; Nir, 1984). Similar correlations have been reported for most strains of obese mice and rats (Bray and York, 1979). Hyperphagia, however, is not a requisite for obesity. Non-hyperphagic obesity following hypothalamic lesioning of White Leghorns was reported by Sonoda (1978).

Pair-feeding did not abolish differences in carcass fat between comparatively fat and lean chickens (Lilburn et al., 1982a; 1982b; LeClercq, 1984). Similar observations have been reported in mammals (Bray and York, 1979).

In rodents, metabolic deviations associated with obesity consistently appear prior to the onset of hyperphagia (Bray and York, 1979), indicating that hyperphagia is a secondary factor in the development of obesity. It is not clear whether similar patterns occur in chickens. Correlated changes in food intake and growth in birds are pronounced during the first week posthatching (Marks, 1980; Barbato et al., 1982). Lilburn et al. (1982a), however, reported that differences in body weight between lean and obese selected lines occurred prior to differences in feed intake, and Simon and LeClercq (1982) observed metabolic differences between their FL and LL lines by the end of embryonic development. These latter observations suggest that the relationships between hyperphagia and obesity is similar in mammals and birds.

Although fasting levels of insulin do not appear to differ between fat and lean chicks (Tables 6-9; Touchburn et al., 1981; Simon and LeClercq, 1982), an increased insulin release in response to a meal or a glucose load occurs in fat chicks (Figure 2; Touchburn et al., 1981; Simon and LeClercq, 1982). Since meat-type chickens tend to eat near

gut capacity (Nir, 1984) and would normally have a full gut, it is probable that they would generally be in a hyperinsulinemic state. It has not been demonstrated, however, that this hyperinsulinemic response persists in chickens at older ages. No differences in insulin release were observed between FL and LL chicks at 17 weeks of age (LeClercq, 1984). In the study reported here, changes in plasma insulin in response to glucose intubation did not differ significantly between the HW and LW chicks at 84 days of age. The relationship between hyperinsulinemia and age should be further examined. Nevertheless, it appears clear that, at least at younger ages, chickens resemble mammals in that the development of obesity is associated with hyperinsulinemia.

Finally, plasma insulin levels (Figure 3) and glucose tolerance curves (Figure 2) suggested that peripheral tissues of HW chicks exhibited a diminished sensitivity to insulin activity. Evidence supportive of insulin resistance in obese chickens was provided by Rudas *et al.* (1972). Similar insulin resistance in FL chicks at eight weeks of age has also been reported (Touchburn *et al.*, 1981; Simon and LeClercq, 1982), but other studies indicated that insulin resistance did not occur in these chickens, even as adults (LeClercq and Simon, 1982; Simon and LeClercq, 1982).

Contrary to the conclusion of LeClercq (1984), obesity

in chickens appears to be similar to that occurring in mammalian species. LeClercq's (1984) hypothesis that hyperinsulinemia is involved in the development of obesity in chickens, however, appears valid. Accordingly, it can be speculated that feeding in meat-type chickens stimulates an enhanced release of insulin into the circulatory system. This increased insulin secretion could be potentiated by the propensity of heavy chickens to eat more meals than their lighter weight counterparts (Barbato et al., 1980). An associated insulin resistance impairs and delays glucose clearance from the blood, resulting in abnormal glucose tolerance curves. Increased plasma glucagon levels in heavy chickens might also contribute to increases in plasma glucose resulting from feeding. Moreover, insulin resistance would reduce glucose utilization in muscle cells, resulting in a decreased efficiency of protein deposition (LeClercq, 1983). On the other hand, increased glucose would be available to lipogenic sites in the liver and adipose tissue. Either lipogenesis would be increased (LeClercq and Saadoun, 1982; Lilburn et al., 1982b; Calabotta, et al., 1983; Saadoun and LeClercq, 1983), lipolysis inhibited (Calabotta et al., 1983; 1985) or both. The net effect would be a shift in metabolism toward fat deposition at the expense of protein deposition.

Table 1. Means and standard errors for body weights of HW, HL, and LW chicks at 21, 42, 63, and 84 days of age<sup>1</sup>

Age	Population					
	HW		HL		LW	
-d-			-g-			
21	(104)	250 ± 39 <sup>A</sup>	(113)	157 ± 29 <sup>B</sup>	(113)	63 ± 14 <sup>C</sup>
42	(120)	775 ± 111 <sup>A</sup>	(118)	511 ± 96 <sup>B</sup>	(115)	142 ± 49 <sup>C</sup>
63	(120)	1344 ± 203 <sup>A</sup>	(120)	887 ± 179 <sup>B</sup>	(117)	276 ± 109 <sup>C</sup>
84	(118)	1895 ± 326 <sup>A</sup>	(116)	1269 ± 239 <sup>B</sup>	(112)	469 ± 141 <sup>C</sup>

<sup>1</sup>Means in the same row with different superscripts differed significantly ( $P \leq .05$ ). Numbers in parentheses indicate the number of observations.

Table 2. Means and standard errors for plasma glucose at 0, 20, 40, 60, 80, and 100 minutes after the administration of glucose to fasted HW, HL, and LW chicks at 21 days of age<sup>1</sup>

Min	Population			$\bar{x}$
	HW	HL	LW	
	-mg/100 ml-			
0 (12)	230 ± 11 <sup>Bc</sup>	(18) 234 ± 9 <sup>Bc</sup>	(21) 299 ± 12 <sup>Aa</sup>	260
20 (20)	420 ± 15 <sup>Aa</sup>	(17) 362 ± 15 <sup>Ba</sup>	(18) 303 ± 7 <sup>Ca</sup>	364
40 (17)	324 ± 10 <sup>Ab</sup>	(20) 295 ± 11 <sup>ABb</sup>	(17) 269 ± 11 <sup>Bb</sup>	296
60 (17)	206 ± 8 <sup>Acd</sup>	(19) 195 ± 6 <sup>Ad</sup>	(19) 186 ± 8 <sup>Ac</sup>	195
80 (20)	194 ± 7 <sup>Ade</sup>	(19) 182 ± 9 <sup>Ad</sup>	(20) 193 ± 9 <sup>Ac</sup>	190
100 (18)	174 ± 7 <sup>Ae</sup>	(19) 180 ± 6 <sup>Ad</sup>	(18) 187 ± 6 <sup>Ac</sup>	180
$\bar{x}$	261	239	240	

<sup>1</sup>Means in the same row with the same upper case superscript and means in a column with the same lower case superscript were not significantly different ( $P \leq .05$ ). The time by population interaction was significant. Numbers in parentheses indicate the number of observations.



Table 3. Means and standard errors for plasma glucose at 0, 20, 40, 60, 80, and 100 minutes after the administration of glucose to fasted HW, HL, and LW chicks at 42 days of age<sup>1</sup>

Min	Population			$\bar{x}$
	HW	HL	LW	
	-mg/100 ml-			
0 (21)	242 ± 5 <sup>Ab</sup>	(20) 245 ± 8 <sup>Ac</sup>	(20) 243 ± 10 <sup>Ab</sup>	243
20 (20)	367 ± 11 <sup>Aa</sup>	(19) 347 ± 11 <sup>Aa</sup>	(19) 285 ± 16 <sup>Ba</sup>	334
40 (20)	377 ± 16 <sup>Aa</sup>	(20) 307 ± 8 <sup>Bb</sup>	(19) 235 ± 8 <sup>Cb</sup>	307
60 (20)	267 ± 10 <sup>Ab</sup>	(20) 240 ± 6 <sup>Ac</sup>	(20) 259 ± 8 <sup>Aab</sup>	255
80 (20)	254 ± 7 <sup>Ab</sup>	(20) 250 ± 8 <sup>Ac</sup>	(20) 241 ± 7 <sup>Ab</sup>	248
100 (19)	248 ± 7 <sup>Ab</sup>	(19) 239 ± 7 <sup>Ac</sup>	(17) 228 ± 8 <sup>Ab</sup>	238
$\bar{x}$	292	271	249	

<sup>1</sup>Means in the same row with the same upper case superscript and means in a column with the same lower case superscript were not significantly different ( $P \leq .05$ ). The time by population interaction was significant. Numbers in parentheses indicate the number of observations.

Table 4. Means and standard errors for plasma glucose at 0, 20, 40, 60, 80, and 100 minutes after the administration of glucose to fasted HW, HL, and LW chicks at 63 days of age<sup>1</sup>

Min	Population			$\bar{x}$
	HW	HL	LW	
	-mg/100 ml-			
0 (20)	178 ± 5 <sup>ABc</sup>	(21) 169 ± 4 <sup>Bd</sup>	(20) 189 ± 4 <sup>Abc</sup>	178
20 (20)	248 ± 7 <sup>Aa</sup>	(20) 251 ± 8 <sup>Aa</sup>	(20) 216 ± 9 <sup>Ba</sup>	238
40 (20)	263 ± 8 <sup>Aa</sup>	(19) 247 ± 9 <sup>Aa</sup>	(20) 199 ± 5 <sup>Bab</sup>	236
60 (20)	220 ± 6 <sup>Ab</sup>	(20) 206 ± 4 <sup>Ac</sup>	(20) 180 ± 5 <sup>Bc</sup>	202
80 (20)	263 ± 14 <sup>Aa</sup>	(20) 220 ± 8 <sup>Bbc</sup>	(17) 211 ± 8 <sup>Ba</sup>	232
100 (20)	214 ± 7 <sup>Bb</sup>	(20) 232 ± 6 <sup>Aab</sup>	(20) 206 ± 4 <sup>Bab</sup>	217
$\bar{x}$	231	220	200	

<sup>1</sup>Means in the same row with the same upper case superscript and means in a column with the same lower case superscript were not significantly different ( $P \leq .05$ ). The time by population interaction was significant. Numbers in parentheses indicate the number of observations.

Table 5. Means and standard errors for plasma glucose at 0, 20, 40, 60, 80, and 100 minutes after the administration of glucose to fasted HW, HL, and LW chicks at 84 days of age<sup>1</sup>

Min	Population			$\bar{x}$
	HW	HL	LW	
	-mg/100 ml-			
0 (20)	161 ± 4 <sup>Ae</sup>	(21) 159 ± 4 <sup>Ad</sup>	(19) 165 ± 5 <sup>Ac</sup>	162
20 (20)	188 ± 3 <sup>Ad</sup>	(20) 186 ± 4 <sup>Ac</sup>	(20) 188 ± 4 <sup>Ab</sup>	187
40 (18)	227 ± 5 <sup>Ab</sup>	(18) 230 ± 5 <sup>Aa</sup>	(19) 208 ± 6 <sup>Ba</sup>	222
60 (20)	251 ± 6 <sup>Aa</sup>	(17) 200 ± 6 <sup>Bb</sup>	(18) 181 ± 5 <sup>Cb</sup>	212
80 (20)	204 ± 5 <sup>Ac</sup>	(22) 191 ± 4 <sup>Bbc</sup>	(19) 174 ± 4 <sup>Cbc</sup>	190
100 (20)	176 ± 5 <sup>Ad</sup>	(18) 169 ± 4 <sup>Ad</sup>	(17) 164 ± 5 <sup>Ac</sup>	170
$\bar{x}$	201	188	180	

<sup>1</sup>Means in the same row with the same upper case superscript and means in a column with the same lower case superscript were not significantly different ( $P \leq .05$ ). The time by population interaction was significant. Numbers in parentheses indicate the number of observations.

Population

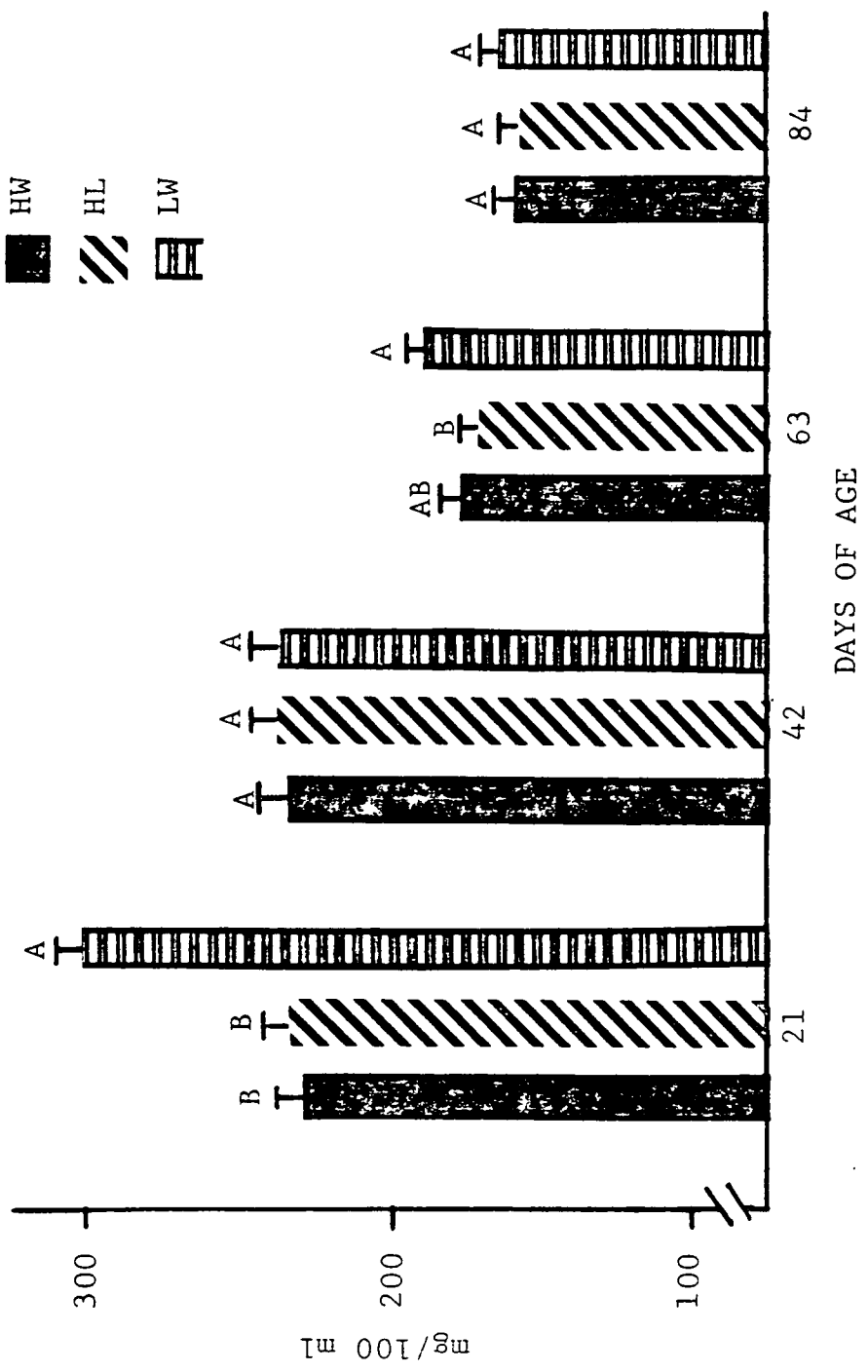
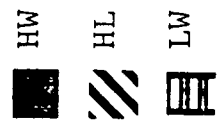


Figure 1. Fasting plasma glucose levels of HW, HL, and LW chicks at 21, 42, 63, and 84 days of age. Values are means  $\pm$  S.E.M. Within ages, means with the same letter did not differ significantly ( $P > .05$ ).

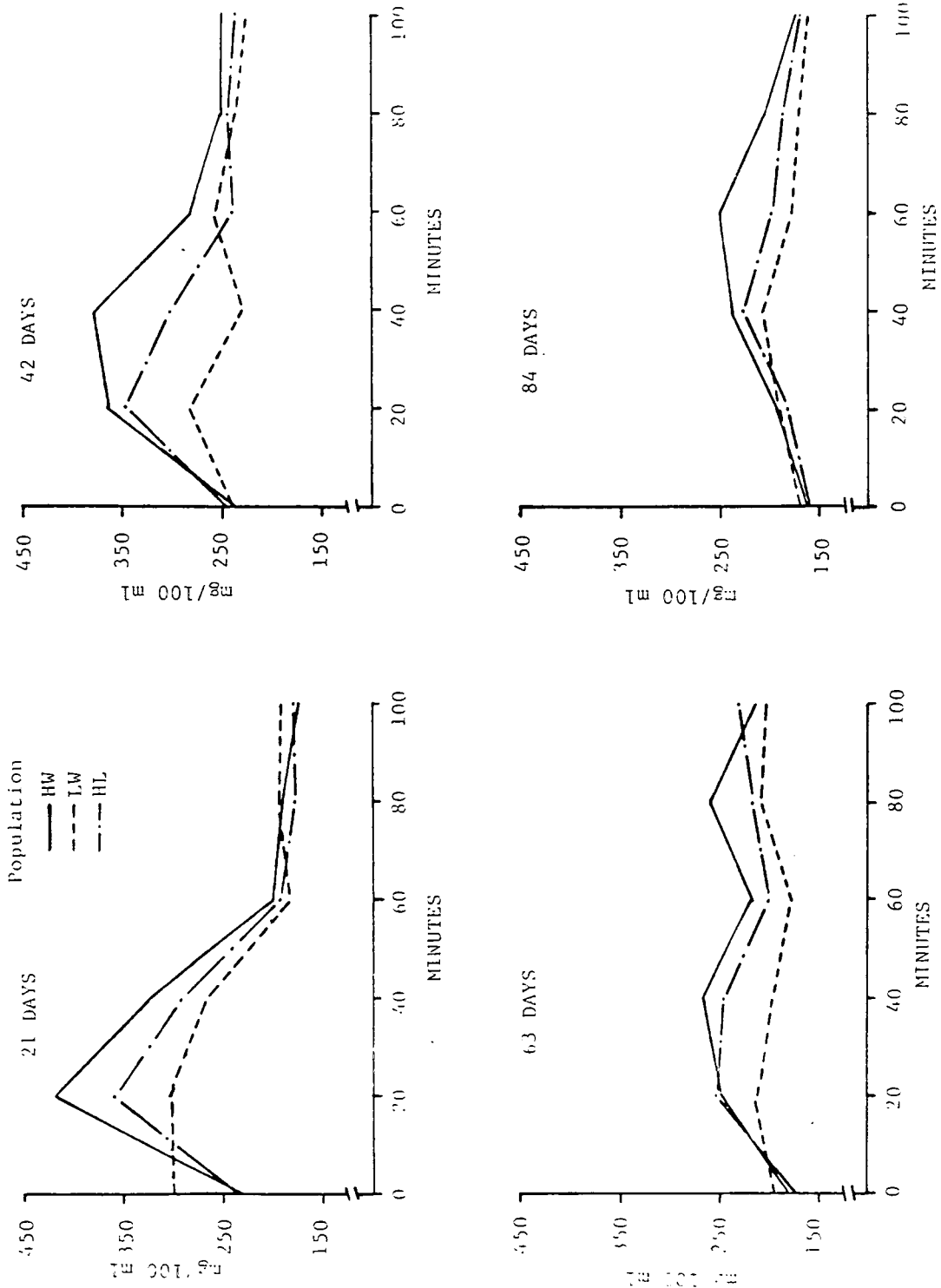


Figure 2. Glucose tolerance for HW, HL, and LW chicks at 21, 42, 63, and 84 days of age

Table 6. Means and standard errors for plasma insulin at 0, 20, 40, 60, 80, and 100 minutes after the administration of glucose to fasted HW, HL, and LW chicks at 21 days of age<sup>1</sup>

Min	Population			$\bar{x}$
	HW	HL	LW	
	-pg/ml-			
0	(0) --	(0) --	(2) 128 ± 107	128 <sup>a</sup>
20	(6) 104 ± 37	(3) 12 ± 4	(1) 11	67 <sup>a</sup>
40	(3) 31 ± 17	(3) 23 ± 6	(4) 53 ± 28	37 <sup>a</sup>
60	(4) 20 ± 6	(2) 6 ± 5	(8) 28 ± 7	22 <sup>a</sup>
80	(1) 14	(1) 115	(2) 20 ± 9	42 <sup>a</sup>
100	(4) 21 ± 6	(1) 25	(4) 48 ± 20	34 <sup>a</sup>
$\bar{x}$	50 <sup>A</sup>	26 <sup>A</sup>	44 <sup>A</sup>	

<sup>1</sup>Means in the same row with the same upper case superscript and means in a column with the same lower case superscript were not significantly different ( $P \leq .05$ ). The time by population interaction was not significant. Numbers in parentheses indicate the number of observations.

Table 7. Means and standard errors for plasma insulin at 0, 20, 40, 60, 80, and 100 minutes after the administration glucose to fasted HW, HL, and LW chicks at 42 days of age<sup>1</sup>

Min	Population			$\bar{x}$
	HW	HL	LW	
	-pg/ml-			
0	(4) 57 ± 19 <sup>Ac</sup>	(6) 163 ± 63 <sup>Ab</sup>	(7) 100 ± 28 <sup>Aa</sup>	112
20	(20) 375 ± 61 <sup>Aa</sup>	(18) 497 ± 94 <sup>Aa</sup>	(6) 131 ± 33 <sup>Ba</sup>	392
40	(17) 261 ± 48 <sup>Aab</sup>	(17) 158 ± 26 <sup>ABb</sup>	(9) 76 ± 10 <sup>Ba</sup>	181
60	(9) 69 ± 8 <sup>Ac</sup>	(9) 69 ± 12 <sup>Ab</sup>	(9) 65 ± 7 <sup>Aa</sup>	68
80	(6) 104 ± 21 <sup>Abc</sup>	(7) 87 ± 15 <sup>Ab</sup>	(3) 57 ± 19 <sup>Aa</sup>	88
100	(7) 65 ± 11 <sup>Ac</sup>	(11) 108 ± 28 <sup>Ab</sup>	(3) 117 ± 20 <sup>Aa</sup>	95
$\bar{x}$	220	221	89	

<sup>1</sup>Means in the same row with the same upper case superscript and means in a column with the same lower case superscript were not significantly different ( $P \leq .05$ ). The time by population interaction was significant. Numbers in parentheses indicate the number of observations.

Table 8. Means and standard errors for plasma insulin at 0, 20, 40, 60, 80, and 100 minutes after the administration of glucose to fasted HW, HL, and LW chicks at 63 days of age<sup>1</sup>

Min	Population			$\bar{x}$
	HW	HL	LW	
	-pg/ml-			
0	(9) 174 ± 61 <sup>Ab</sup>	(13) 171 ± 53 <sup>Ab</sup>	(17) 208 ± 33 <sup>Aa</sup>	185
20	(19) 385 ± 104 <sup>Aa</sup>	(19) 313 ± 47 <sup>Aa</sup>	(14) 182 ± 58 <sup>Aa</sup>	304
40	(20) 290 ± 35 <sup>Aa</sup>	(16) 354 ± 51 <sup>Aa</sup>	(15) 161 ± 31 <sup>Ba</sup>	272
60	(16) 113 ± 12 <sup>Ab</sup>	(19) 103 ± 19 <sup>Ab</sup>	(14) 154 ± 45 <sup>Aa</sup>	121
80	(14) 142 ± 27 <sup>Ab</sup>	(14) 148 ± 32 <sup>Ab</sup>	(13) 92 ± 13 <sup>Aa</sup>	128
100	(16) 126 ± 24 <sup>Ab</sup>	(17) 143 ± 45 <sup>Ab</sup>	(18) 135 ± 29 <sup>Aa</sup>	135
$\bar{x}$	218	207	157	

<sup>1</sup>Means in the same row with the same upper case super-script and means in a column with the same lower case super-script were not significantly different ( $P \leq .05$ ). The time by population interaction was significant. Numbers in parentheses indicate the number of observations.



Table 9. Means and standard errors for plasma insulin at 0, 20, 40, 60, 80, and 100 minutes after the administration of glucose to fasted HW, HL, and LW chicks at 84 days of age<sup>1</sup>

Min	Population			$\bar{x}$
	HW	HL	LW	
	-pg/ml-			
0 (12)	131 ± 30	(14) 172 ± 43	(12) 120 ± 22	143 <sup>c</sup>
20 (18)	204 ± 53	(18) 252 ± 47	(17) 251 ± 50	236 <sup>b</sup>
40 (15)	351 ± 79	(18) 428 ± 73	(16) 298 ± 48	362 <sup>a</sup>
60 (19)	206 ± 41	(14) 166 ± 26	(12) 207 ± 81	194 <sup>bc</sup>
80 (17)	177 ± 29	(19) 135 ± 15	(14) 180 ± 51	162 <sup>bc</sup>
100 (16)	155 ± 38	(13) 272 ± 55	(10) 268 ± 62	223 <sup>bc</sup>
$\bar{x}$	205 <sup>A</sup>	240 <sup>A</sup>	224 <sup>A</sup>	

<sup>1</sup>Means in the same row with the same upper case superscript and means in a column with the same lower case superscript were not significantly different ( $P \leq .05$ ). The time by population interaction was not significant. Numbers in parentheses indicate the number of observations.

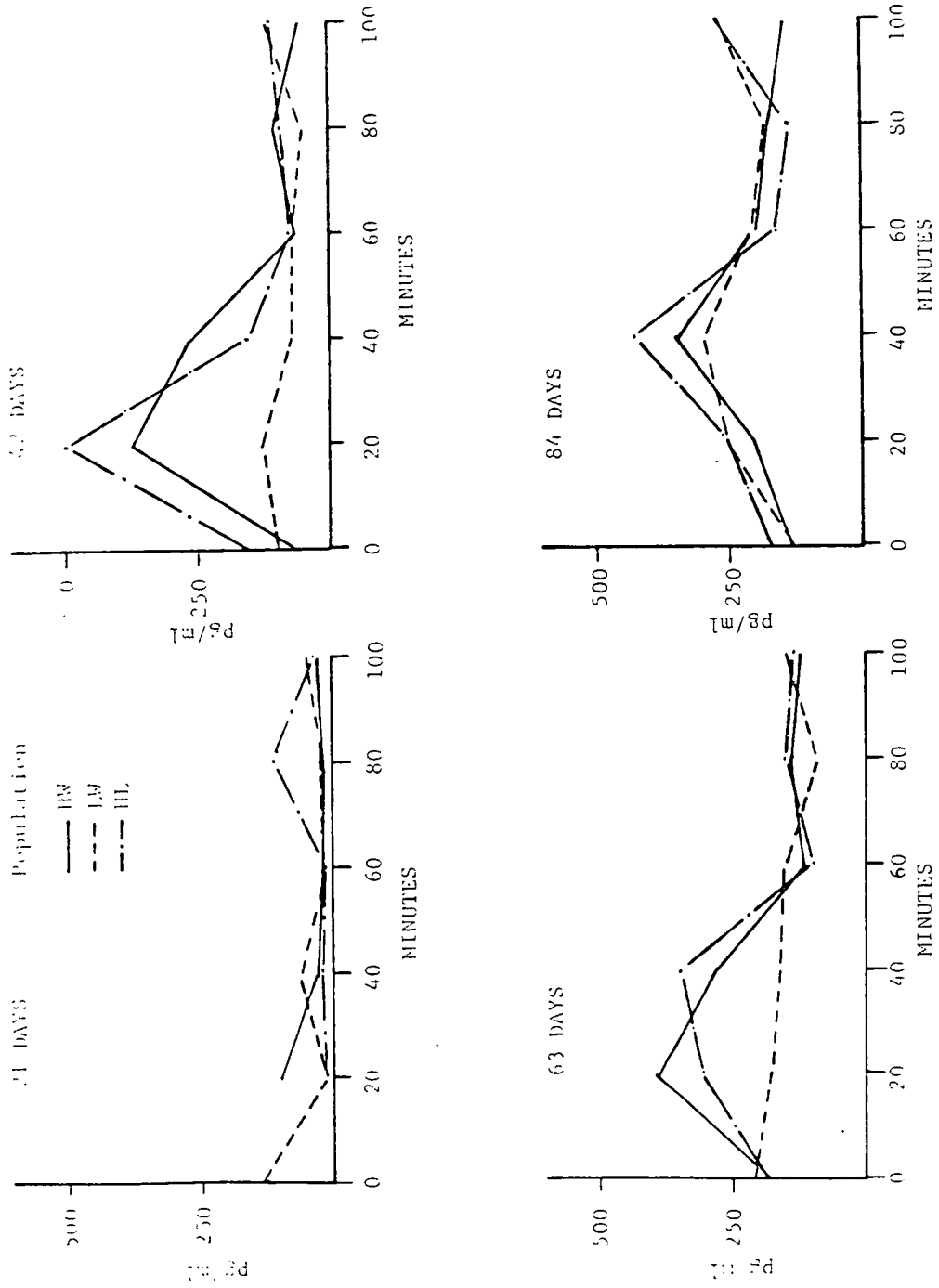


Figure 4. Plasma insulin responses of HW, HL, and LW chicks to the oral administration of glucose at 21, 42, 63, and 84 days of age.

Table 10. Means and standard errors for plasma glucagon at 0, 20, 40, 60, 80, and 100 minutes after the administration of glucose to fasted HW, HL, and LW chicks at 21 days of age<sup>1</sup>

Min	Population			$\bar{x}$
	HW	HL	LW	
	-pg/ml-			
0 (10)	941 ± 168	(13) 668 ± 48	(5) 549 ± 93	744 <sup>a</sup>
20 (19)	839 ± 68	(14) 614 ± 54	(11) 629 ± 108	715 <sup>a</sup>
40 (17)	781 ± 78	(15) 934 ± 99	(7) 847 ± 139	852 <sup>a</sup>
60 (16)	981 ± 89	(11) 840 ± 105	(4) 826 ± 106	911 <sup>a</sup>
80 (14)	996 ± 92	(5) 758 ± 72	(5) 661 ± 172	877 <sup>a</sup>
100 (11)	750 ± 59	(13) 774 ± 82	(7) 565 ± 166	718 <sup>a</sup>
$\bar{x}$	880 <sup>A</sup>	766 <sup>A</sup>	671 <sup>B</sup>	
$\bar{x}^*$	29.14	27.15	24.65	

<sup>1</sup>For main effects, means in a row or column having the same superscript were not significantly different ( $P \leq .05$ ). The time by population interaction was not significant. Numbers in parentheses indicate the number of observations.

\*Square root.

Table 11. Means and standard errors for plasma glucagon at 0, 20, 40, 60, 80, and 100 minutes after the administration of glucose to fasted HW, HL, and LW chicks at 42 days of age<sup>1</sup>

Min	Population						$\bar{x}$
	HW		HL		LW		
	-pg/ml-						
0 (20)	860 ± 38	(19)	782 ± 40	(10)	759 ± 54		809 <sup>C</sup>
20 (18)	782 ± 30	(19)	854 ± 27	(7)	659 ± 102		793 <sup>C</sup>
40 (19)	808 ± 22	(18)	939 ± 50	(12)	793 ± 57		853 <sup>bc</sup>
60 (19)	1015 ± 60	(18)	1039 ± 83	(13)	879 ± 81		988 <sup>a</sup>
80 (18)	921 ± 32	(16)	909 ± 35	(11)	703 ± 73		864 <sup>bc</sup>
100 (15)	914 ± 43	(16)	951 ± 66	(13)	831 ± 59		903 <sup>ab</sup>
$\bar{x}$	882 <sup>A</sup>		910 <sup>A</sup>		783 <sup>B</sup>		

<sup>1</sup>For main effects, means in a row or column having the same superscript were not significantly different ( $P \leq .05$ ). The time by population interaction was not significant. Numbers in parentheses indicate the number of observations.

Table 12. Means and standard errors for plasma glucagon at 0, 20, 40, 60, 80, and 100 minutes after the administration of glucose to fasted HW, HL, and LW chicks at 63 days of age<sup>1</sup>

Min	Population									
	HW			HL			LW			$\bar{x}$
	-pg/ml-									
0 (20)	1011 ± 44	(18)	942 ± 37	(15)	928 ± 42				964 <sup>b</sup>	
20 (20)	1082 ± 44	(17)	1119 ± 34	(16)	899 ± 65				1039 <sup>a</sup>	
40 (20)	940 ± 36	(16)	984 ± 28	(17)	929 ± 47				950 <sup>b</sup>	
60 (19)	605 ± 22	(20)	570 ± 18	(19)	527 ± 19				567 <sup>d</sup>	
80 (20)	639 ± 18	(19)	625 ± 19	(16)	589 ± 20				619 <sup>c</sup>	
100 (20)	603 ± 24	(20)	585 ± 13	(19)	526 ± 12				572 <sup>d</sup>	
$\bar{x}$	815 <sup>A</sup>			788 <sup>A</sup>			721 <sup>B</sup>			

<sup>1</sup>For main effects, means in a row or column having the same superscript were not significantly different ( $P \leq .05$ ). The time by population interaction was not significant. Numbers in parentheses indicate the number of observations.

Table 13. Means and standard errors for plasma glucagon at 0, 20, 40, 60, 80, and 100 minutes after the administration of glucose to fasted HW, HL, and LW chicks at 84 days of age<sup>1</sup>

Min	Population						$\bar{x}$
	HW		HL		LW		
	-pg/ml-						
0 (20)	590 ± 13	(21)	573 ± 12	(18)	525 ± 16		564 <sup>a</sup>
20 (19)	552 ± 23	(20)	546 ± 12	(19)	482 ± 12		527 <sup>b</sup>
40 (18)	562 ± 20	(18)	551 ± 20	(17)	478 ± 23		531 <sup>b</sup>
60 (20)	520 ± 29	(17)	521 ± 31	(15)	538 ± 34		525 <sup>b</sup>
80 (20)	599 ± 24	(22)	589 ± 12	(17)	534 ± 17		576 <sup>a</sup>
100 (17)	530 ± 23	(18)	588 ± 12	(16)	502 ± 21		542 <sup>ab</sup>
$\bar{x}$	559 <sup>A</sup>		563 <sup>A</sup>		509 <sup>B</sup>		

<sup>1</sup>For main effects, means in a row or column having the same superscript were not significantly different ( $P \leq .05$ ). The time by population interaction was not significant. Numbers in parentheses indicate the number of observations.

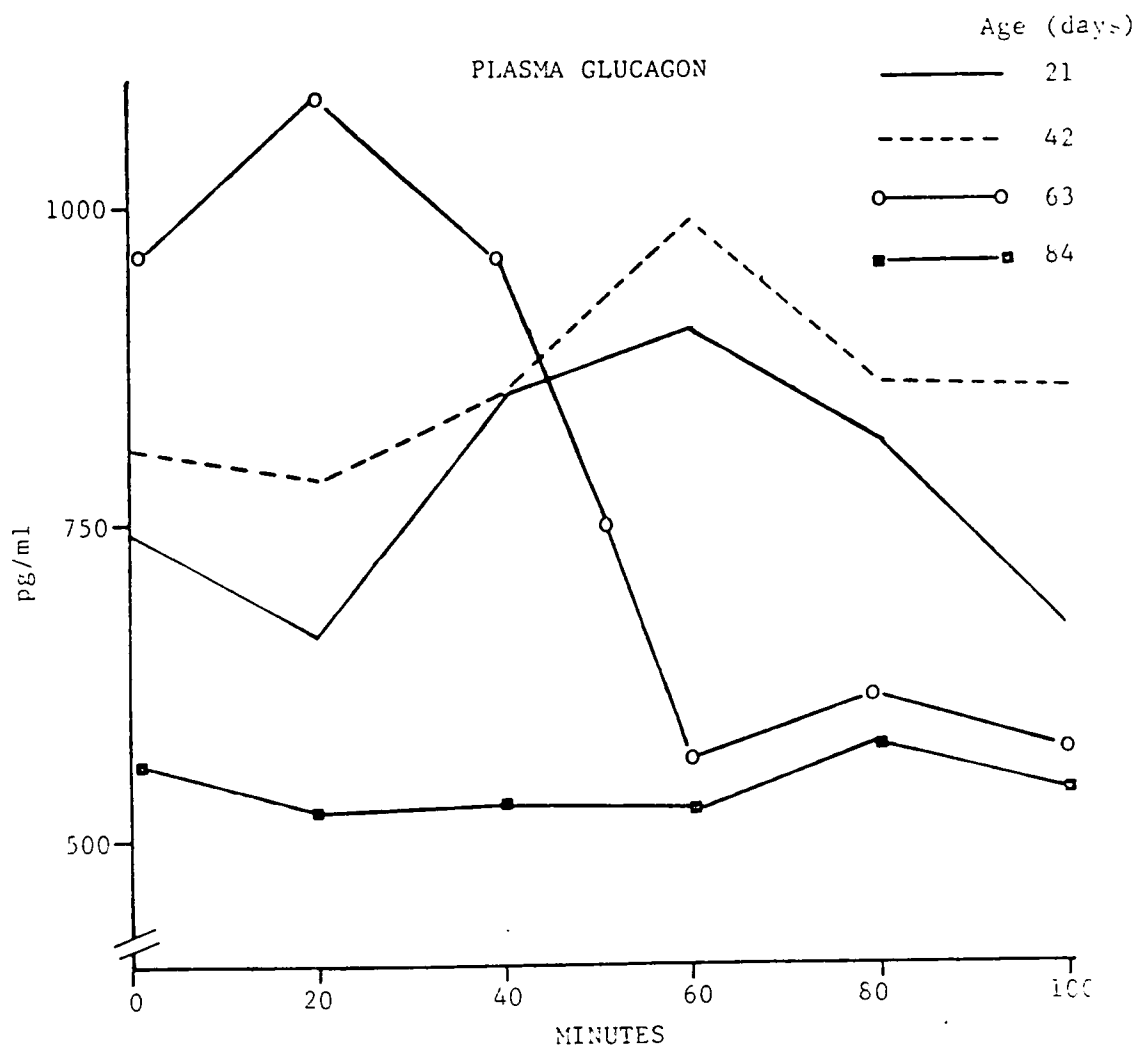


Figure 4. Plasma glucagon responses to the oral administration of glucose to fasted chicks at 21, 42, 63, and 84 days of age.

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APPENDIX



Appendix Table 1. Analysis of variance for traits presented in the text<sup>1</sup>

Source of Var.	df	Mean Squares			
		21-day wt. (329)	42-day wt. (353)	63-day wt. (357)	84-day wt. (346)
Time	5	1253	7212	11187	35379
Line	2	916711**	11580686**	33401089**	57499926**
TxL	10	478	6824	11095	29517
Sex	1	24632**	661474**	3140392**	8635592**
TxS	5	1123	5779	25919	17447
LxS	2	3312*	68480**	310064**	842505**
TxLxS	10	232	7364	27231	43400
Error (n-35)		765	5838	17841	32458

<sup>1</sup>Number in parentheses represents the number of observations (n).

\*(P ≤ .05).

\*\* (P ≤ .01).

Appendix Table 2. Analysis of variance for traits presented in the text (21 day-old chicks)<sup>1</sup>

Source of var.	df	Mean Squares		
		Plasma glucose (329)	Plasma insulin <sup>2</sup> (49)	Plasma glucagon <sup>2</sup> (197)
Time	5	293487**	15.4	66.7
Line	2	13907**	11.0	229.6**
TxL	10	18883**	15.0	39.1
Error	(n-17)	1729	9.3	35.7

<sup>1</sup>Number in parentheses represents the number of observations (n).

<sup>2</sup>Square root transformations were used for analysis.

\*(P ≤ .05).

\*\* (P ≤ .01).

Appendix Table 3. Analysis of variance for traits presented in the text (42 day-old chicks)<sup>1</sup>

Source of var.	df	Mean Squares		
		Plasma glucose (353)	Plasma insulin <sup>2</sup> (168)	Plasma glucagon <sup>2</sup> (281)
Time	5	91346**	457.9**	68.2**
Line	2	55576**	126.3**	134.6**
TxL	10	16827**	51.0*	12.2
Error	(n-17)	1764	24.2	11.8

<sup>1</sup>Number in parentheses represents the number of observations (n).

<sup>2</sup>Square root transformations were used for analysis.

\*(P ≤ .05).

\*\* (P ≤ .01).

Appendix Table 4. Analysis of variance for traits presented in the text (63 day-old chicks)<sup>1</sup>

Source of var.	df	Mean Squares		
		Plasma glucose (357)	Plasma insulin <sup>2</sup> (283)	Plasma glucagon <sup>2</sup> (331)
Time	5	33246**	315.8**	840.5**
Line	2	28368**	70.5	69.4**
TxL	10	5893**	55.1*	8.5
Error (n-17)		1027	25.9	5.7

<sup>1</sup>Number in parentheses represents the number of observations (n).

<sup>2</sup>Square root transformations were used for analysis.

\*(P ≤ .05).

\*\* (P ≤ .01).

Appendix Table 5. Analysis of variance for traits presented in the text (84 day-old chicks)<sup>1</sup>

Source of var.	df	Mean Squares		
		Plasma glucose (346)	Plasma insulin <sup>2</sup> (274)	Plasma glucagon <sup>2</sup> (332)
Time	5	30876**	270.0**	12.6**
Line	2	12809**	30.0	44.0**
TxL	10	4109**	27.5	4.9
Error (n-17)		446	33.1	3.5

<sup>1</sup>Number in parentheses represents the number of observations (n).

<sup>2</sup>Square root transformations were used for analysis.

\*(P ≤ .05).

\*\* (P ≤ .01).

Appendix Table 6. Means and standard errors for insulin:glucagon ratios at 21 and 42 days of age (data not presented in text)<sup>1</sup>

Age	Time	Population			$\bar{x}$
		HW	HL	LW	
-d-	-min-				
21	0	---	---	(1) .51	.51 <sup>a</sup>
	20	(6) .13±.04	(2) .15±.01	---	.10 <sup>b</sup>
	40	(3) .06±.03	(3) .04±.01	(2) .02±.01	.04 <sup>b</sup>
	60	(4) .02±.01	(1) .001	(2) .03±.01	.02 <sup>b</sup>
	80	(1) .02	---	---	.02 <sup>b</sup>
	100	(3) .04±.02	---	---	.04 <sup>b</sup>
	$\bar{x}$	.07 <sup>A</sup>	.03 <sup>A</sup>	.12 <sup>A</sup>	
42	0	(4) .08±.03	(6) .23±.11	(4) .08±.03	.14 <sup>b</sup>
	20	(18) .52±.08	(18) .59±.11	(6) .22±.05	.51 <sup>a</sup>
	40	(16) .35±.06	(15) .18±.04	(9) .11±.02	.23 <sup>b</sup>
	60	(8) .07±.01	(8) .09±.02	(7) .09±.02	.08 <sup>b</sup>
	80	(6) .13±.03	(6) .09±.03	(2) .08±.02	.11 <sup>b</sup>
	100	(6) .07±.02	(10) .13±.04	(3) .13±.004	.11 <sup>b</sup>
	$\bar{x}$	.29 <sup>A</sup>	.27 <sup>A</sup>	.12 <sup>B</sup>	

<sup>1</sup>Means in the same row with the same upper case superscript and means in a column with the same lower case superscript were not significantly different ( $P \leq .05$ ). The time by population interaction was not significant. Numbers in parentheses indicate the number of observations.

Appendix Table 7. Means and standard errors for insulin:glucagon ratios at 63 and 84 days of age (data not presented in text)<sup>1</sup>

Age	Time	Population			$\bar{x}$	
		HW	HL	LW		
-d- -min-						
63	0	(9)	.16±.05 (12)	.19±.06 (14)	.25±.05	.21 <sup>a</sup>
	20	(19)	.37±.10 (17)	.32±.06 (11)	.24±.08	.32 <sup>a</sup>
	40	(20)	.31±.04 (15)	.34±.05 (14)	.18±.03	.29 <sup>a</sup>
	60	(16)	.18±.02 (19)	.18±.04 (13)	.29±.08	.21 <sup>a</sup>
	80	(14)	.24±.04 (14)	.24±.06 (13)	.15±.02	.21 <sup>a</sup>
	100	(16)	.22±.05 (17)	.25±.08 (18)	.26±.05	.24 <sup>a</sup>
	$\bar{x}$		.26A	.25A	.23A	
84	0	(12)	.23±.05 (14)	.30±.08 (12)	.23±.04	.26 <sup>c</sup>
	20	(17)	.37±.10 (18)	.46±.08 (16)	.47±.10	.43 <sup>b</sup>
	40	(15)	.65±.16 (18)	.77±.11 (15)	.62±.11	.68 <sup>a</sup>
	60	(19)	.39±.07 (14)	.30±.04 (11)	.45±.17	.38 <sup>bc</sup>
	80	(17)	.30±.05 (19)	.23±.03 (12)	.31±.09	.28 <sup>bc</sup>
	100	(14)	.31±.08 (13)	.46±.09 (10)	.57±.14	.43 <sup>b</sup>
	$\bar{x}$		.38 <sup>A</sup>	.43 <sup>A</sup>	.45 <sup>A</sup>	

<sup>1</sup>Means in the same row with the same upper case superscript and means in a column with the same lower case superscript were not significantly different ( $P \leq .05$ ). The time by population interaction was not significant. Numbers in parentheses indicate the number of observations.

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GLUCOSE TOLERANCE, PLASMA INSULIN, AND PLASMA  
GLUCAGON IN RELATION TO OBESITY IN CHICKENS

by

Nancy A. Sinsigalli

(ABSTRACT)

Relationships among glucose tolerance, plasma insulin, and plasma glucagon were examined in chicks developed through selection for high (HW) and low (LW) body weight, and in F<sub>1</sub> crosses (HL) between HW males and LW females. At 21, 42, 63, and 84 days of age, chicks from each population were intubated with glucose (2 g/kg body weight) following a 24-hr fast. Blood was collected at 20 minute intervals up to 100 minutes postadministration.

At all ages, the LW chicks were better able to metabolize glucose than their HW counterparts, while the HLs exhibited intermediate responses. Impaired glucose tolerance in the HWs and HLs was not associated with insulin insufficiency; the HWs and HLs, in comparison to the LWs, were hyperinsulinemic at 42 and 63 days of age and plasma insulin levels did not differ among populations at 21 or 84 days of age. Plasma glucagon responses to glucose administration were inconsistent, but plasma glucagon levels were consistently higher in the HWs and HLs than in the LWs.

It was concluded that excessive fat deposition in chickens selected for rapid growth is associated with hyperinsulinemia and insulin resistance.