A GENETIC ANALYSIS OF REPRODUCTION IN GROWTH SELECTED LINES OF CHICKENS

by

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Dissertation submitted to the Graduate Faculty of the
Virginia Polytechnic Institute and State University
in partial fulfillment of the requirements for the degree of

DOCTOR OF PHILOSOPHY

in

Genetics

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July, 1976
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ACKNOWLEDGEMENTS

A deepest sense of gratitude and sincere appreciation is expressed to Dr. P. B. Siegel for his guiding, encouraging, assisting and stimulating my thought processes during the course of this study.

Personal thanks are extended to Dr. H. P. Van Krey for his timely and stimulating discussions on the physiological aspects of this investigation and his critical review of this dissertation. Also, there were with Dr. Koos van Middelkoop of the Spelderholt Institute many enjoyable discussions on the central theme of this dissertation. I wish to thank Drs. R. A. Teekell and W. B. Gross for their constructive criticisms, and Drs. C. E. Howes, J. M. White and D. A. West for suggestions and guidance.

My special thanks are extended to Mrs. Kay Criner for her help and understanding and to Mrs. Janice Scearce for her assistance in various aspects of my work. I am indebted to Dr. R. G. Saacke, who was instrumental in deepening my interests in cytological studies and to Dr. C. W. Heald for his interest and guidance given in preliminary studies with electron microscopy. For the valuable assistance and cooperation, the author wishes to thank Mrs. Angela Lineen and others in the physiology and lactation laboratories of the Dairy Science Department.

I owe a debt of gratitude to my father and brothers for their moral encouragement, and to my sister, Yoshoda and her family for support in furthering my higher education.

Finally, special appreciation is expressed to my wife, Kranthi, for her patience, understanding and support during various critical moments which made this work possible and a pleasant task.

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INTRODUCTION

The primary problem in today's broiler industry is maintaining reasonably high levels of hatching egg production. A steady decline in reproductive fitness has accompanied intensive selection for growth. This is due to the apparent negative correlation between growth rate and reproductive fitness (e.g., McClung, 1958; Jaap et al., 1962; Siegel, 1963; Kinney and Shoffner, 1965; Ideta and Siegel, 1966; Merritt et al., 1966). Recent investigations indicate that fast growing broilers actually develop more ovarian follicles and, in reality, there may be a positive correlation between growth rate and ova production. Therefore, the problem appears to be an imbalance in the physiology of the bird that hinders in realizing eggs suitable for incubation. This is vividly portrayed by the phenomenon of Erratic Oviposition and Defective Egg Syndrome (EODES) that was first reported by Jaap and Muir (1968). There is a need for detailed studies of this syndrome and of the ovarian activity in fast and slow growing chickens. Such investigations should provide insights necessary to better understand the genetic and physiological causes of the correlated responses in reproduction associated with continuous selection for body weight.

There is a growing interest in exploiting the genetic properties of the sex-linked dwarfing gene in the production of commercial meat and egg stocks. Information is needed on the effect of this gene on growth and reproduction in various genetic backgrounds.

The underlying physiological bases of different ovulation and oviposition patterns in growth selected lines may be understood by examining the function of appropriate cells. Surprisingly, there is a dearth of information on the ultrastructure of avian adenohypophysis. In addition, karyotype analyses may be an aid in selection procedures.

The high weight (HW) and low weight (LW) lines developed at VPI & SU are ideal experimental populations to utilize in genetic and physiological analyses of the conditions described above. The first experiment of this dissertation was designed to measure ovarian activity and egg production in these lines. Oviposition patterns and the influence of photoperiod were also included. The second experiment attempted to estimate the genetic parameters of egg production patterns and the relationships between growth and reproductive traits. Thirdly, the effect of a single major gene, dw, on growth and reproduction was studied in divergent genetic backgrounds. An attempt was made in the fourth experiment to probe into the ultrastructure of the pituitary gland in an effort to understand further the genetic and environmental influences on cell function. Finally, karyotype analyses were made to evaluate its potential as a tool to supplement selection methods.

REVIEW OF LITERATURE

Growth and Egg Production Relationships

It is well known that a negative phenotypic relationship exists between body weight and egg production in chickens. Large negative genetic correlations of 0.31 and 0.51 were reported by Wyatt (1954) and Jerome et al. (1955) respectively, while a small negative estimate was calculated by von Krosig and Pirchner (1965). Hogsett and Nordskog (1958) reported widely differing values for their light (-0.24) and heavy strains (0.74) of egg-type chickens.

Jaap et al. (1962) using a randombred population of chickens, observed a positive correlation between 8-week body weight and the number, weight, and shape of eggs. Upon selection for body weight, however, the correlated response of egg production was negative with a decrease of one percent in egg production per 45 g gain in 8-week body weight. Other investigators (McClung, 1958; Kinney and Shoffner, 1965, 1967; Siegel, 1963; Ideta and Siegel, 1966) also found a negative relationship between juvenile body weight and egg production.

Although egg production in broilers decreases as a concomitant response to selection for growth rate, the converse may not be true (Jaap and Khan, 1972). In the first two generations of selection for increased normal egg production, they observed a significant increase in hatching egg production with no significant decline in 8-week body weight. Similar results were reported by van Middelkoop (1973b).

In a two-way selection experiment for juvenile body weight, Siegel (1963, 1970) observed a negative correlated response in the unselected traits, percentage hen-day egg production and age at sexual maturity. The diametrically opposing correlated responses for these traits were noticed with pullets from the HW line maturing earlier and laying at a lower rate than those from the LW line. Subsequent research by Udale (1972) suggested that arhythmic ovulations resulting in abnormal eggs might be a possible explanation for the relationship between body weight and egg production in these lines. Bidirectional selection for body weight in mice resulted in correlated response in ovulation rate and embryonic mortality in the same direction as the changes in the selected trait (Land, 1970). Further, there was a significant positive genetic correlation between body weight at 6 weeks of age and ovulation rate.

The paradox of body weight and egg production relationships in egg-type chickens was studied by Nordskog and Briggs (1968). They noted that lowering body weight by 100 g from an overall mean of 1500 g would increase the hen-housed egg production by 12 eggs on the genetic scale and decrease it by 18 eggs on the environmental scale. They proposed that the theoretical optimum body weight for each genetic group was largely dictated by environment. Restricted energy intake of broiler stocks during the growing period delays age at maturity, decreases adult body weight, and increases the number and weight of hatchable eggs (Peacock et al., 1968; Fuller et al., 1969, 1973).

Frankham and Doornenbal (1970) observed that in lines selected for egg production body weight was negatively correlated, while egg production was positively correlated with gonadotropin sensitivity. They suggested the possibility of selecting directly for gonadotropin sensitivity as a means of increasing egg production, since the heritability for gonadotropin sensitivity in broiler populations was high (Siegel and Siegel, 1964).

Ovulation and Oviposition

There is a brief time relationship between ovulation and oviposition, with oviposition being influenced by ovulation since the ruptured follicle influences the time of oviposition (Rothchild and Fraps, 1944). They also suggested that some "extra ovarian" light sensitive mechanism may be involved in the process of oviposition. More recently, Tanaka and Nakada (1974) and Tanaka (1976) showed that the ruptured follicle played a hormonal function in controlling oviposition. One may induce premature oviposition by the administration of LH (Fraps, 1942), acetylcholine and histamine (Weiss and Sturkie, 1952), and through the presence of foreign objects in the wall of the oviduct (Sykes, 1953; Lake and Gilbert, 1964). Huston and Nalbandov (1953) demonstrated that ovulatory peaks of LH could be blocked by placing a thread in the oviduct. Furthermore, they indicated that endogenous LH was not sufficient to affect ovulation.

Neural mechanisms are involved in ovulation and oviposition cycles (van Tienhoven, 1953). According to Fraps (1954), the neural

mechanisms that control LH peaks exhibit a diurnal periodicity in their threshold of response to excitation. Additionally, asynchronous ovulatory cycles involve neural, hormonal, and metabolic factors. Bastian and Zarrow (1955) observed that enforced wakefulness and increased activity delayed ovulation. Abnormal ovulatory cycles were due to an interaction between the 24 hour day-night rhyt-m and the rhythmic maturation of follicles. The first follicle of a sequence was considerably more sensitive to ovulation-inducing hormone than subsequent follicles (Fraps, 1946; Bastian and Zarrow, 1955).

Normal nesting behavior was observed by Wood-Gush (1963) to be essential for regular ovulation and oviposition rhythms. Wood-Gush and Gilbert (1965), suggested that oviduct has a relatively unimportant role in nesting behavior while the neuro-humoral events occuring in the mature and the post-ovulatory follicle probably played a dominant role in controlling subsequent nesting behavior.

Erratic Egg Syndrome

Jaap and Muir (1968) were the first to report erratic oviposition and defective egg syndrome (EODES) in chickens. They observed arhythmic sequences in daily egg laying patterns with a higher incidence of defective eggs in meat than egg-type pullets. They proposed that many idiosyncracies of ovulation and oviposition may be a result from a low sensitivity of fast growing chickens to environmental rhythms of a circadian nature. Arhythmic ovipositions by broiler-type pullets is often accompanied by improperly formed eggs.

Meat-type pullets had more follicles in rapid development stages, but produced fewer eggs than egg-type pullets (Jaap and Clancy, 1968). A similar observation was made in a comparison of meat and egg-type turkeys by Nester et al. (1970). Using a dye feeding technique, Jaap and Mohammadian (1969) estimated that the number of ovarian follicles in rapid development was 2.2 fewer than that actually found at autopsy. Possible causes of this discrepancy were internal laying and a progressive regression of developing follicles. The rate of internal laying estimated by the frequency of nestings without laying was 11.6 percent in a sample of Brown Leghorns (Wood-Gush and Gilbert, 1970). Bacon et al. (1972) observed a greater number of atretic follicles per hen in a strain of turkeys selected for increased growth rate than in an egg-type strain. The above findings were confirmed by Udale (1972) in our growth selected lines; more ova were lost by the HW than by LW pullets.

In an explanation for EODES, Jaap (1969) hypothesized that inheritance which favors rapid body growth (protein anabolism) may also favor a rapid synthesis of yolk lipoprotein precursors in the liver, and deposition in the ovarian follicles. Interestingly, however, Bacon et al. (1973) found no significant differences between egg and meat-type turkeys in the low density fraction (LDF) of the yolk lipoprotein precursors.

Additional evidence of defective eggs in broiler populations has been presented by van Middelkoop (1972a, 1972b, 1973b), Udale (1972). They concluded that defective eggs were related to a short

ovulation interval. Van Middelkoop and Simon (1970) and van Middelkoop (1971) studied the phenomenon of laying two shelled eggs each day, and showed that the first egg of a pair had additional calcification. The higher embryonic mortality (72-87%) in the first eggs was attributed to poor shell porosity rather than to either a delayed escape of carbon dioxide or to a prolonged stay of the egg at the body temperature (van Middelkoop, 1972a). A possible association between follicles developing in pairs and oviposition irregularities in turkeys was indicated by Nester and Bacon (1972), since ova of nearly identical weights are likely to be ovulated simultaneously (Buss, 1963). Delaying sexual maturity by decreasing day length markedly reduced the frequency of double yolk eggs, but did not influence the incidence of two eggs laid on the same day (van Middelkoop and Kuit, 1974). This is not surprising because the incidence of double-yolked eggs is greater during the early parts of the production period, while the laying of two eggs a day is relatively constant throughout the laying period (van Middelkoop, 1973b). These observations are consistent with those of Lowry and Abplanalp (1967) who also recorded a similar influence of delayed maturity on the frequency of double-yolked eggs.

Genetic Influences on Defective Eggs

Although extensive genetical analyses have been made on various reproductive traits in chickens, investigations on the mode of inheritence of abnormal ovulation and oviposition patterns have drawn little attention. The primary reason being that most of the abnormal eggs

are not noticed when the hens are maintained on the floor or in general cage operations. Secondly, there appears to be little genetic variation for defective eggs in egg-type stocks due to the prolonged selection for high normal egg production. The situation with broiler stocks is different. This is because selection for body weight in broilers resulted in a correlated response in increased ovarian activity, thereby creating an imbalance between ovarian and oviducal functions.

The genetic basis of double-yolked eggs was first studied by Lowry (1967) and Lowry and Abplanalp (1967, 1968) who reported a heritability estimate of 0.3. More recently, Sarvella (1975) reported a genetic basis for multiple ovulations. Adult body and egg weights increase as correlated responses to selection for double-yolked eggs (Lowry and Abplanalp, 1968). Van Middelkoop (1973b) also observed higher 8-week body weights as a consequence to selection for double-yolked eggs and the laying of two eggs a day. Although abnormal eggs may be classified into various categories, little was known about the heriditary predisposition and relationship with other traits until the report of van Middelkoop (1973b). He initiated a systematic selection experiment to study the inheritence of at least two important types of abnormal eggs, i.e. double-yolked eggs and the first egg of the pair of two eggs a day. The heritabilities of laying normal, double-yolked, and two eggs a day, in a broiler strain were estimated as 0.4 to 0.5, 0.3 to 0.5, and 0.5 to 0.7, respectively. The data indicated that the frequency of abnormal eggs tended to be

positively correlated with 8-week body weight and he hypothesized at least two different and independent genetic causes for the production of abnormal eggs. This was because selection for one type did not change the incidence of the other type.

Silber and Merat (1974) reported a moderate to high heritability (0.2 to 0.5) for broken eggs. The percentage of broken eggs was positively correlated with adult weight, albumen height and negatively correlated with shell thickness and age at first egg. Also the dw gene reduced the incidence of broken eggs. Van Middelkoop and Kuit (1974) confirmed the higher influence of genetic factors controlling abnormal egg production. Comparisons between pure lines and reciprocal crosses revealed an additive effect of the genes controlling the production of abnormal eggs.

Photoperiod, Growth and Fecundity

Light and Egg Production

Recent reviews of the effects of light on avian reproduction are by Morris (1968) and van Tienhoven and Planck (1973). Historically, Goodale (1924) questioned the "feeding effect" of increased photoperiod and hypothesized that artificial light operated directly to stimulate egg production rather than to stimulate greater feed consumption. Since the first report by Whetham (1933) that the anterior pituitary was involved in photoperiodic stimulation of reproduction, several studies (Bissonnette, 1931; Benoit, 1935, 1938, 1964; Rowan, 1938; Green and Harris, 1949) have established that light acts through

the visual system on hypothalamic-hypophyseal complex and regulates the activity of gonads. Benoit (1964) postulated that there may be two photoreceptors, one retinal and one hypothalamic, that are involved in photostimulation. Light not only stimulates the production and release of the hypothalamic-pituitary releasing factors, but may also act as a signal for synchronizing endogenous circadian rhythms.

Warren and Scott (1936) showed that under natural light the onset of darkness was a factor in the termination of the clutch. Oviposition, however, was distributed throughout the 24-hour period when chickens were maintained under continuous light. Similar observations were made by McNally (1946), and Payne et al. (1965). The rhythmic nature of oviposition under continued light was observed by McNally (1947), who noted that the feeding time determined the time of oviposition. Wilson and Abplanalp (1956) observed that egg production was higher for chickens maintained under intermittent lighting than for those exposed to continuous illumination. This may be due to photorefractoriness developing after the initial photostimulation obtained from continuous light (Farner and Follett, 1966). These results, however, do not agree with those of Moore and Mehrhof (1946) who noted greater egg production under continuous lighting than with periodic increments.

The effects of increasing light schedules on growth and reproduction of commercial layers was studied by King (1961). He found that short-day rearing followed by regular increments in photoperiod

during laying period was beneficial to egg production. Morris and Fox (1961) and Marr et al. (1962) presented evidence that the use of a regular increase in photoperiod throughout the laying year resulted in a greater egg yield than the use of a constant photoperiod. In contrast, increasing the photoperiod from 14 to 15 hours at the time of housing was not advantageous (Yeates, 1960; Smith and Noles, 1963; Noles and Smith, 1964). The age at which chickens become photosensitive has not been clearly established. Morris (1968) observed that chickens were more sensitive to photoperiodic changes as they approach maturity, and indicated that changes in photoperiod within the range of 8 to 16 hours had considerably more effect, while Morris and Fox (1958) reported that photoperiodism was of little consequence during the first 10 weeks of life. Chicks, however, may be sensitive to photoperiodic changes as early as 3 weeks posthatching (Morris, 1967).

Siegel et al. (1961, 1963) studied the effects of various lighting regimes on sexual maturity and egg production of Leghorn-type layers. Restricting the photoperiod to six hours during the growing period significantly retarded age at sexual maturity, but increased livability compared to 14-hours of illumination. Similar results were reported by Bowman and Jones (1961), King (1961), and Shutze et al. (1961). In general, females exposed to a 6 hour--3% increment lighting regime produced eggs at a lower rate in the early part of the production cycle, but equalled or surpassed the 14-hour lighting regime during the later stages of the cycle. Restriction of photoperiod during growth and the early part of the laying year

reduces egg size (Shutze et al., 1961; Siegel et al., 1963).

Light and Broiler Growth

Several investigations have shown that meat-type chickens are heavier at broiler age when exposed to continuous light than those exposed to intermittent photoperiods (Shutze et al., 1961; Krueger et al., 1962; Beane et al., 1962, 1965; Weaver and Siegel, 1968). In contrast, Cherry and Barwick (1962) viewed that light was not strictly necessary for growth. After experience in light to locate feed and water, chicks reared in the dark although lower in body weight at 6 weeks of age, were equal in weight at 10 weeks of age to those exposed to near continuous light. Continuous illumination may be advantageous to populations with rapid growth potentials because it increases feeding time (Siegel and Wood, 1964; Beane et al., 1965). In addition, it was demonstrated by Beane et al. (1965) that continuous illumination not only provided additional feeding time, but also have stimulated other factors involved in growth. They hypothesized a change in behavioral responses in feeding rhythms stimulated by continuous light. Lighting regimes did not effect feed efficiency.

According to Gore et al. (1969), an adequate dark period of inactivity following feeding plays a dominant role in broiler growth. In contrast, Forshee et al. (1970) suggested that a primary factor affecting superior growth was a uniform distribution of activity throughout a 24-hour period. Quarles and Kling (1974) did not

observe any advantage of continuous light on broiler growth at 4 and 7 weeks of age.

The effect of light intensity on broiler growth has been investigated by Barrot and Pringle (1951), Shutze et al. (1961), Skoglund and Palmer (1962), and Beane et al (1965). Continuous light at a single intensity promoted greater growth than at variable intensities of light. This suggests that factors other than the feeding time are involved in the growth of broilers exposed to continuous light. Further, body weights were heavier when broilers were exposed to low rather than high intensities of light. Weaver and Siegel (1968) studied the effects of the length of photoperiod and light intensity on feeding behavior, body weight, feed efficiency, and endocrine activity. Cockrels exposed to continuous light at a single low intensity were heavier at 8 weeks of age than those maintained under other lighting regimes. Testes were heavier in males exposed to continuous light, but no significant differences were observed in either anterior pituitary weights or the gonadotropic potency of this gland. Fluctuations in light intensities influenced feeding rhythms and other activity patterns, which in turn, affected growth.

Dwarfing Gene, dw

The mode of inheritence and some of the phenotypic effects of sex-linked recessive dwarf gene, dw, were first described by Hutt (1959). Individuals with this gene reproduce normally and are distinct from those exhibiting the autosomally inherited thyrogenous

dwarfism (Landauer, 1929; Upp, 1934) which is usually semilethal prior to sexual maturity.

Effects of dw Gene

Although dw does not retard growth during incubation, its retarding effect is evident by two weeks of age (Hutt, 1959). At 40 weeks of age the percentage reduction in body weight is 35 to 43 for dwdw males and 26 to 32 for dw females. The corresponding percentage reductions in shank length are 32 and 24. Although dwarfs laid fewer and smaller eggs than their normal sisters, Hutt (1959) found no conclusive evidence that dw influences age at sexual maturity. Furthermore, viability, fertility, and hatchability were not effected by dwarfism. Several reports (Bernier and Arscott, 1960, 1966, 1972; Arscott and Bernier, 1968; Merat, 1969; Prod'homme and Merat, 1969; Jaap and Mohammadian, 1969; Mohammadian, 1969, 1970; Ricard and Cochez, 1972; Udale, 1972; Jaap, 1971; and French and Nordskog, 1973) while elaborating on the effects of the dw gene, have also confirmed general observations presented in the initial paper by Hutt. In addition, from these publications one may find evidence to show that the dw gene delays sexual maturity.

Research with dwarf gene in stocks of different polygenic backgrounds reveal varying results. Jaap (1968) noted that the dw gene was not completely recessive because heterozygous males weighed 2.5 to 5 percent less than normal homozygotes. Chambers et al. (1974) reported similar observations. The dw gene reduces the body and egg

weight in egg-type and broiler stocks (Mohammadian, 1969), and in bidirectionally growth selected lines (Udale, 1972). The proportion of reduction, within experiments, was similar in these populations for each trait. This was in contradiction to a subsequent report by Mohammadian and Jaap (1972) that the weight depressing effect of dw gene was severe in egg-type populations compared to broilers, while its effect on skeletal size was similar in both types of fowl.

Mohammadian (1969) observed that there was a reduction in the frequency of defective eggs in dwarfs with a genetic background of 3/4 broiler and 1/4 egg-type. Also there was a decline in normal egg production as the proportion of broiler background increased. Furthermore, the dw gene reduced the rate of yolk deposition in the ovary (Jaap and Mohammadian, 1969). The yolk weight of mature follicles was 8 percent lower in dwarfs than in normals which is similar to the 10 percent reduction in egg weights noted by Hutt (1959). Similar observations were made by van Middelkoop (1973a).

Matings of DwDw sires with dw dams produced Dw daughters that were 2.8 percent heavier at 8 weeks of age than female progeny from DwDw sires mated to Dw dams (Mohammadian, 1970). The dwarf daughters from dwdw x dw matings were 4.4 percent lighter than dwarf daughters from dwdw x Dw matings. In addition, dwarf progency of both sexes were more variable than the normal offspring. Mohammadian and Jaap (1972) demonstrated that the body weight of heterozygous (Dwdw) sons from normal dams were similar to dominant homozygous (DwDw) sons, but that the 8-week body weight of Dwdw males from dwarf dams was slightly

suppressed in comparison to DwDw males from normal dams. This may be due to smaller egg size of dwarf dams. Wiley (1950), Tindell and Morris (1964), and Merritt and Gowe (1965) reported a highly positive relationship between egg weight and the juvenile body weight of individuals from these eggs. Although the dw allele reduced the size of eggs from broiler dams slightly less than it did those from egg-type dams (Mohammadian, 1970), fertility and hatchability were not effected (Jaap, 1971; Merat, 1971; French and Nordskog, 1973). Although the dw gene delayed sexual maturity by 13 days and reduced body weight at 20 weeks of age by 36 percent, Ricard and Cochez (1972) observed that the rate of lay for their dwarf broiler line was superior (66%) to that of normal females (60%). Also when maintained in cages, there were fewer broken eggs from dwarfs than from normal chickens.

Jaap and Muir, (1968) proposed that the dw gene reduced yolk deposition in the ovary probably by suppressing lipoprotein synthesis in the liver. This hypothesis was evaluated by van Middelkoop (1973a) using a line selected for an increased incidence of double yolked eggs, a line selected for laying two eggs per day, and a normal population. Dwarfs from the selected lines laid significantly more normal eggs than nondwarfs, while the inverse was observed in the normal line. This suggests that the action of the dw gene may differ depending upon the genetic background in which it is tested.

Physiology of Dwarfs

The physiological basis of sex-linked recessive dwarfism is poorly understood. Van Tienhoven et al. (1966) probed into the microstructure of the thyroid and anterior pituitary glands of dwarf and normal chickens. Thyroids from dwarfs contained less colloid than those of normal chickens, suggesting a lower secretion rate of thyroid hormone. This, however, was not the primary cause of dwarfism because the condition was not corrected by feeding 0.4 percent protomone. Histological examination of anterior pituitary glands from dwarf chickens showed normal presumptive gonadotropic and thyrotropic cells and a higher incidence of cells with large secretory droplets. They hypothesized that these cells were probably concerned with growth hormone. A pituitary bioassay by Bernier and Arscott (1972), however, did not support the hypothesis of van Tienhoven et al. (1966) that there was an accumulation of somatotropic hormone.

A hypothyroidic condition of dwarfs was observed by several investigators (Merat and Guillaume, 1969; Rajaratnam et al., 1969; Guillaume, 1972; Bernier and Arscott, 1972). Rajaratnam et al. (1969) studied the effect of two levels of protomone supplementation (.033% and .066%) on dwarfs and normal chickens. They concluded that low thyroxine secretion may be partly responsible for the smaller size of dwarfs and their reduced food intake. Although gains in body weight of dwarfs fed low levels of protomone was significantly higher than that of the untreated dwarf controls, normal pullets responded similarly in both groups. Additional evidence of hypothyroidism was

provided by Rajaratnam et al. (1971a, b) and Summers et al. (1970) who showed that dwarfs had significantly lower body temperatures, basal metabolic rates, oxygen consumption, and more carcass fat than non-dwarf chickens. Grandhi et al. (1975) demonstrated that dwarf birds had significantly lower thyroid activity but normal organifications, since the uptake of labelled iodine by dwarfs was significantly lower while the release rates were equal to normal birds.

Lipogenesis was greater in dwarfs than in normal chickens (Touchburn and Blum, 1972). Thus, the increased fat storage in dwarfs may be due to a combined effect of greater lipogenesis and decreased energy expenditure.

Comparisons between dwarf and normal White Leghorn and White Plymouth Rock chickens showed that the packed cell volumes and hemoglobin concentrations were higher in nondwarf than in dwarf chickens (Wood et al., 1971). Furthermore, dwarfs had significantly lower levels of blood sugar, total lipids, cholesterol, and free amino acids in their muscles than normal chickens (Guillaume, 1972). The modification of amino acid patterns in dwarfs was characteristic because there were significantly lower levels of threonine, serine, and glycine and a considerable decline in valine and isoleucine.

Nutritional Studies with Dwarfs

Numerous investigations have been conducted on the efficiency of feed conversion of dwarf chickens (Bernier and Arscott, 1960, 1972; Arscott and Bernier, 1968; Prod'homme and Merat, 1969; Guillaume, 1969;

Selvarajah et al., 1970; Summers, 1971; Quisenberry, 1972; French and Nordskog, 1973; Chambers et al., 1974; and Sutton et al., 1975). Dwarf females weighed 37 percent less, consumed 34 percent less feed, and required 16 percent less feed per dozen of 24 oz. eggs than their normal sisters (Bernier and Arscott, 1960). Feed conversion consistently favored the dwarfs at all ages ranging from 4 to 23 weeks (Arscott and Bernier, 1968). Prod'homme and Merat (1969) in a study involving broiler-type pullets observed that although the dwarfs laid almost the same number of eggs as their normal sisters, they consumed 26 percent less feed even after accounting for their smaller body and egg weights. Contrary to the above reports, Guillaume (1969) found that whereas dwarf pullets consumed as much feed as normal chickens of the same body weight, they had lower production rates. This observation was confirmed by French and Nordskog (1973) who compared dwarfs from a White Leghorn strain to normal chickens of the same body weight.

Proteins and caloric requirements of dwarfs were evaluated by Arscott and Bernier (1970), Guillaume (1969, 1971), Leclercq and Blum (1971), Summers (1971), and Quisenberry (1972). Generally dwarfs required rations with higher protein and lower calorie to protein ratios than nondwarf chickens. Also, rations with higher levels of calcium and phosphorus improved the egg production of dwarfs more than that of normal chickens. In contrast, Prod'homme and Merat (1969) failed to note any differences in egg production due to calcium-phosphorus supplementation. Energy levels of the diet and ambient temperature have a negative relationship with feed intake in

both the dwarfs and normal birds (Ahmed et al., 1974). From this, they concluded that chemostatic and thermostatic mechanisms were equally operative in both the stocks. Additionally, Simon (1972) observed that dwarfs could not adapt to discontinuous feeding as readily as normal chickens. This was because food intake under ad libitum feedings were not increased after fasting phases as was typical for nondwarf chickens.

Ultrastructural Studies

Anterior Pituitary Gland

The avian hypophysis is distinct from that of other vertebrates because the pars intermedia is not morphologically distinct. The pars distalis of most of the avian species, including the domestic fowl, is a bilobed structure (Rahn, 1939; Rahn and Painter, 1941; and Wingstran, 1951), with the lobes being morphologically and physiologically distinct (Mikami, 1958; Tixier-Vidal et al., 1962). The hypothalamic-hypophyseal vascular supply of the domestic fowl was described by Hasegawa (1956). Vitinus et al. (1964) and Mikami (1969) related the cytological and functional differentiation of the avian adenohypophysis to the characteristic distribution of hypophyseal portal vessels between the median eminence and the two lobes of pars distalis. They suggested that the function of the cephalic lobe may be controlled by the anterior median eminence while the function of the caudal lobe may be controlled by the posterior median eminence.

Although there have been extensive studies of the ultrastructure of the adenohypophysis in several mammalian species (Herlant, 1963, 1964), the pars distalis has been investigated in only a few avian species. Ultrastructural studies of the adenohypophysis of the domestic fowl (Payne, 1965; Mikami, 1958, 1960; Mikami et al., 1969), domestic mallard (Tixer-Vidal, 1963, 1965), Pekin duck (Tixier-Vidal and Banoit, 1962), pigeon (Tixier-Vidal and Assenmacher, 1966), White crowned sparrow (Mikami, 1969; Mikami, et al., 1969) and Japanese quail (Tixier-Vidal, et al., 1967, 1968) have revealed its functional cytology.

Using light microscopy, Purves and Griesbach (1951) presented a hypothesis regarding the functional types of pituitary cells. Identification of cell types in the pars distalis of birds was largely achieved by target organ removal and employing agents that stimulated or inhibited these organs. Identification of ACTH cells by adrenalectomy (Mikami, 1958, 1969), gonadotrophs by castration (Payne, 1965; Tixier-Vidal, 1965; Mikami et al., 1969), thyrotrophs by thyroidectomy and the feeding of thiouracil (Mikami, 1969; Mikami et al., 1969), and lactotrophs by photoperiodic control (Mikami et al., 1969) has greatly enhanced our understanding of the relationships between the structure and the function of these cells.

Payne (1965) demonstrated lipid accumulation in the gonadotrophs of pituitaries of aging and castrated chickens, and indicated that secretory granules and mitochondria played no part in their formation. Thiouracil feeding resulted in an unexpected granular accumulation

in the endoplasmic reticular vescicles of thyrotrophs.

Using both light and electron microscopy, Mikami (1969) and Mikami et al., (1969) confirmed the morphological and functional features of various cells in the pars distalis of White crowned sparrows and chickens. They studied the cytological changes that occurred after castration, adrenalectomy, and thyroidectomy. Castration remarkably increased the number and size of gonadotrophs with extensive vacuolation, while adrenalectomy affected the ACTH amphotrophs. Thyrotrophic cells in the cephalic lobe lost secretory granules and were transformed into large vacuolated thyroidectomy cells. Photostimulation increased the frequency of luteotrophs, while adrenalectomy and castration decreased the number of these cells. They also showed that gonadotrophs occurred in both the cephalic and caudal lobes, while thyrotrophs and amphotrophs were confined to cephalic lobe. In addition, the caudal lobe was characterized by the presence of large somatotrophs. In contrast, an "avian type" distribution of the basophils that recognizes the occurance of thyrotrophs in both lobes, and the restriction of FSH gonadotrophs to cephalic lobe and LH gonadotrophs to the caudal lobe was shown in mallard ducks (Tixier-Vidal et al., 1962; Tixier-Vidal, 1963, 1965) and Japanese quail (Tixier-Vidal et al., 1967). These contrasting reports may reflect differences in methodology and in interpretations rather than true differences among species. The resolvement of these controversies still remains. This is because the basic difficulties that persist in the emperical identification of cell types which

include the questions of the significance of differentiating criteria and the problems associated with the stage of development and the phase of activity. Therefore, the results are vulnerable to subjective judgment and should be interpreted with caution.

Karyotype Analysis

Research in the cytogenetics of the domestic fowl suffered a great lag until the past decade. This was because of a lack of satisfactory procedures for preparing material to provide accurate descriptions of the normal chromosome complement. Developments in methodology to display avian chromosomes (Owen, 1965; Fechheimer and Jeffe, 1966; Shoffner et al., 1967; Miller et al., 1971; Bloom et al., 1972), comparisons of chromosomal aberrations in different genetic stocks (Bloom, 1969, 1971, 1974; Miller et al., 1971; Fechheimer et al., 1972; Lodge et al., 1973), development of techniques for longitudinal differentiation of chromosomes and identification of facultative and constitutive heterochromatin (Wang and Shoffner, 1973, Stock et al., 1974; and Mateescu et al., 1974), and the induction of chromosomal rearrangements by X-rays and chemical mutagens (Kosin, 1944; Wooster et al., 1973; Boerger et al., 1973; and Shoffner, 1972) have greatly enhanced our understanding of the possible uses of cytogenetics as potential aids to supplement the quantitative genetic approaches used in poultry breeding programs. In addition, chicken karyotype studies can serve as a model for analysis and study of various etiological factors that contribute to pregnancy wastage in

various species. This is because of the ease with which large number of embryos of varying age can be harvested without sacrificing the dam and can be subjected to different treatments in controlled physiological states.

Although many reports have confirmed that the diploid (2N) number of chromosomes in the chicken is 78, only 8 to 10 pairs of machrochromosomes can be consistently displayed (Yamashina, 1944; Newcomer, 1957, 1959; Krishnan, 1962; Schmid, 1962; Donnally and Newcomer, 1962). Of these, there is clear morphological distinction among 8 pairs of macrochromosomes. Although microchromosomes divide as regularly as macrochromosomes, there is variation in the number that may be attributed to negative heteropycnosis, fragmentation, depth of field, and problems associated with tissue processing (Krishnan, 1962). Microchromosomes are involved in nucleolar organization (Ohno et al., 1962). Schmid (1962) using a radioactive labelling technique, observed that microchromosomes replicated earlier than the macrochromosomes. A majority of birds, including the chicken, have a ZZ and ZW sex determining mechanism with the W chromosome having a small metacentric element in the size range of the ninth and tenth pairs of chromosomes (Schmid, 1962; Rothfels et al., 1963; Krishnan and Shoffner, 1966). The W chromosome can be easily identified because of its heterochromatic nature, repetitive DNA, and late replication in DNA synthesis (Bloom, 1974). Reciprocal translocations, duplications, and other chromosomal rearrangements are identifiable by banding techniques (Wang and Shoffner, 1973; Stock et al., 1974).

Chromosomal abberations in chicken karyotypes have been reported by several investigators. Newcomer (1959) induced a reciprocal translocation among chromosomes 1 and 2 by X-irradiation, and Ryan and Bernier (1968) reported spontaneous translocation involving the second and third largest chromosomes. Ohno et al. (1963) observed an adult triploid chicken with a left ovotestis and suggested that triploids could arise due to polyandry or polygyny. A number of spontaneously occuring triploids were observed by Hameed and Shoffner (1971). Subsequently, Wang and Shoffner (1973) showed that triploids could be produced by the production of diploid ova and diploid spermatozoa that were capable of union with normal haploid gametes.

A diploid-triploid mosaic embryo was identified by Bloom and Buss (1967). Triploidy was observed by Donner et al. (1969) in three embryos and a newly hatched chick. All were 3A·ZZZ. Trisomy-3, trisomy-4 and triploidy 3A·ZZZW in the chick embryos was first documented by Bloom (1970a). Cytological screening of 2056 embryos (Bloom, 1970b) revealed that 492 embryos were phenotypically abnormal, and of these 13 were haploids (A-Z) with a low incidence of mosaicism with diploid and triploid cells. Since no incidence of parthenogenetic development was observed in these stocks, the mosaics probably arose through a series of complex cellular events initiated by abnormal fertilization.

Karyotype analyses have been conducted for various populations of chickens (Miller et al., 1971; Fechhiemer et al., 1972; Bloom, 1972, 1974; and Lodge et al., 1973). A low frequency of chromosomal

abnormalities (1.4%) was observed at 16 hours incubation in Leghorn embryos by Lodge et al. (1973). These abnormalities included haploids, triploids, triple trisomy/triple monosomy mosaics, and a large deletion of chromosome 1. It was suggested that this mosaicism might be due to a simultaneous occurance of non-dysjunction for 3 chromosomes. It was further noted, that triple trisomy had a selective advantage over triple monosomy. Miller et al. (1971) observed chromosomal abnormalities in 12.7 percent of the embryos from a line of broilers that had undergone selection for rapid growth. This was in contrast to the 3.2 percent abnormalities observed in the randombred controls. Evidence from other species suggests that triploidy may be due to a hormonal imbalance (Carr, 1967). Such an imbalance could be especially important at the onset of lay which, in turn, suggests an association between the frequency of chromosomal abnormalities and EODES in broiler populations. Fechheimer et al. (1972) noted chromosomal aberrations in 1.5 percent of Leghorn and 11.9 percent of broiler-type embryos. This 8-fold difference was highly significant. The most frequent chromosomal abberation observed in broiler stocks was triploidy as the only cell type or as a mosaic cell line. This was followed by haploid-diploid mosaics. Furthermore, they observed a heterogeneous distribution of aberrant embryos within their broiler line. Similar observations were reported by Duber et al. (1973). Although there was no sire effect, pullets produced significantly more abberant embryos at the onset of lay than during the later ages of egg production; an observation consistent with that of Miller

et al. (1971).

In a karyotype analysis involving 4182 chick embryos from 10 strains and 5 strain crosses, Bloom (1972) observed significant differences in chromosomal aberration rates among populations. He postulated that genetic mechanisms were involved in the production of haploids. Subsequently, Bloom (1974) using the same stocks hypothesized that embryonic mortality to 4 to 5 days of incubation was primarily due to chromosomal aberrations. Meanwhile, Jaap and Fechheimer (1974) proposed that as much as 25 percent of the embryo mortality in chickens was the result of spontaneous heteroploidy.

Irradiation with X-rays (Zartman, 1971; Boerger et al., 1973; Wooster et al., 1973) and chemical mutagens (Shoffner, 1972) have been successfully utilized to produce viable chromosomal rearrangements in chickens. Zartman (1973) identified the pea comb locus on first chromosome by using a 1-Z interchange chromosome. The possibility of utilizing artificially constituted chromosomes in studies involving linkage relationships, in the coordination of cytological and genetic data, and in influencing genetic variation of quantitative traits was recently discussed by Shoffner, (1974).

EXPERIMENT I. OVULATION AND OVIPOSITION PATTERNS IN THE GROWTH SELECTED LINES

This experiment was designed to study the frequency and pattern of ovulation and oviposition in the lines of chickens selected for high and low juvenile body weight. Comparisons of daily egg production and ovarian activity were made between two lines maintained under two photoperiod regimes. Data were obtained in three trials that were conducted over three generations. The first two trials were designed to study the frequency of defective eggs and the relationship between egg production and ovarian activity in lines selected for high (HWS) and low (LWS) juvenile body weight. The third trial enabled comparisons among these selected and their respective relaxed lines for normal and defective egg production, plus growth and other reproductive parameters.

Materials and Methods

Genetic stocks and management: The selected populations used in this experiment were the S_{15}, S_{16} and S_{17} generations of the HWS and LWS lines. The respective relaxed lines that were used for the S_{16} and S_{17} generations were the R_3 and R_4 generations (R_0 being a sample taken from the S_{13} generation). The selection procedures used in the development of these lines were described by Siegel (1962, 1970). Briefly, bidirectional selection, based on individual phenotypic body weight at 8 weeks of age, was made from an original gene pool that

was derived from the crosses of 7 inbred lines of White Plymouth Rocks.

A generation was produced in March of each year. Chicks were reared under continuous light to 56 days of age and under natural lighting to 119 days of age. Thereafter, pullets received 14 hours of illumination per 24-hour day cycle. The specific experimental photoperiod for each trial will be described separately for that trial. The rations fed were the same formulation in all generations, and provided 1220 Kcal/lb of M.E. and 20% crude protein in chick rations and 1260 Kcal/lb of M.E. and 16% crude protein in breeder rations. Feeding schedules, however, differed each generation and will be described subsequently for each trial.

Egg production: Daily egg production was recorded on an individual bird basis from the date of first egg to January 1. Papers located beneath the laying cages were inspected for the presence of any defective eggs, membranes and yolk between 8:00 and 9:00 a.m. each day. Oviposits were classified according to the amount of shell deposition, number of yolks, presence of membranes, yolk, and shape. Eggs classified as double yolks were broken to insure the accuracy of classification. The following categories were used:

1. Normal (N)--a single yolked egg with an intact shell of normal thickness,

2. Double yolk (DY)--an egg with two yolks, with an intact and complete shell of normal thickness.

3. Membrane (M)--no visible calcification, but with membranes covering the yolk and albumen,

4. Broken (B)--apparently normal shelled eggs that were broken onto the dropping papers,

5. Soft shell (S)--an egg with incomplete shell calcification,

6. Remains (R)--albumen and yolk with ruptured membranes,

7. Deformed (d)--an egg with a flattened surface and corregated shell,

8. Yolk (Y)--yolk found on the dropping papers and occasionally smeared onto another egg (in some cases the vitelline membrane was visable),

9. Smalls (sm)--a very small egg with little or no yolk,

10. Twin membrane (TM)--an egg with two yolks covered only by membranes without any visible calcification,

11. Twin soft shelled (TS)--an egg with two yolks and partial calcification,

12. Triple yolks (TY)--an egg with three yolks having a normal shell.

Total defective eggs included Types 2 through 12. Although they may not reflect irregularities in ovulation and oviposition, broken eggs were considered defective. This was because HWS pullets laid significantly more broken eggs, and a genetic basis for this incidence was indicated by Carter (1971) and Silber and Merat (1974).

Trial 1 (S_{15} generation): A random sample of 58 HWS and 48 LWS pullets were maintained in individual cages under a constant photoperiod of 14 hours light and 10 hours darkness. Feed and water were provided ad lib. Females, within each line, were randomized into nine fixed treatment periods of 20, 40, 60, 80, 100, 120, 140, 160, and 180 days of lay from the date of first egg. Since the incidence of

defective eggs was mostly confined to early laying period, (0-80 days) this plan was discontinued after the first four treatment periods. Thus, on December 11, 1972 when the pullets were 258 days of age the remaining birds from both the lines that commenced production were exposed to continuous light and randomly assigned to five treatment groups of five birds per group. These pullets were sacrificed at weekly intervals for direct measurements of ovarian activity.

Autopsy: At the end of each fixed treatment period the pullets were weighed and sacrificed between 2:00 and 3:00 p.m. by a lethal intervenous injection of sodium pentobarbitol. The ovary was removed and developing follicles ($\geq$0.3 g) detached, counted, and individually weighed. Ruptured and atretic follicles were also counted and the weight of the ovary obtained.

Analyses: Data were analyzed by analysis of variance with lines and periods considered fixed main effects. The statistical model was:

$$Y_{ijk} = \mu + L_i + P_j + (LP)_{ij} + e_{ijk}$$

where, i = 1, 2 lines; j = 1, 2, ... 4 period; k = 1, 2, ... 5 pullets/line/period.

Bartlett's test of homogeneity of variance (Snedecor and Cochran, 1967) was used to test the variances among subclass groups. The percentage hen-day production (% HDP) of normal, defective, and total eggs were transformed to arc sin $\sqrt{\%}$ prior to analysis. Freeman-Tukey arc sins were obtained for proportions based on less than 50 observations (Mosteller and Youtz, 1961).

Trial 2 (S_{16} generation): This trial was designed to study the line-photoperiod interactions for ovarian activity and oviposition patterns. Data were obtained from random samples of 40 pullets from the HWS and the LWS lines. Pullets were randomized into four fixed periods of lay which consisted of 40, 80, 120, and 160 days from the date of first egg. The pullets were exposed to photoperiod treatments at the time of caging at 119 days of age. One group received a constant photoperiod of 14 hours illumination in a 24-hour cycle, while the other group starting at the same level received 3% increase in photoperiod per week to January 1. Sexual maturity was delayed by diet restriction. Pullets were fed on alternate days during the first 10 weeks after caging, and thereafter feed was restricted to approximately 60% of voluntary intake.

At the end of each fixed laying period pullets were sacrificed and ovarian activity studied. Autopsy procedures followed were those used in Trial 1. Data on the weights of the liver, thyroid, and pituitary glands were also obtained.

Analyses: The data were analyzed by analysis of variance with lines, photoperiodic treatments, and laying periods considered as fixed main effects. The statistical model was:

$$Y_{ijkl} = \mu + L_i + T_j + (LT)_{ij} + P_k + (LP)_{ik} + (TP)_{jk} + (LTP)_{ijk} + e_{ijkl}$$

where, i = 1, 2 lines; j = 1, 2 treatments; k = 1, 2 ... 4 periods and l = 1, 2 ... 5 individual/line/treatment and period combination.

The method of least squares was used because of disproportionate subclass numbers.

Trial 3 (S_{16} and S_{17} generations): Comparisons were made among selected and relaxed lines for the production of normal and defective eggs. Data were obtained on random samples of HWS, LWS, HWR and LWR lines from the date of first egg to January 1 of the respective year. Traits measured included body weight at 8, 24, 38 weeks of age, initial egg weights based on the first 3 eggs laid, and 35-week egg weight.

Photoperiod treatments were the same as those described in Trial 2. Feed restrictions in $S_{16}(R_3)$ generation were the same as those described for Trial 2, while in the $S_{17}(R_4)$ generation pullets received from the time of caging to January 1 about 60% of the feed under ad libitum situation.

Analyses: Analysis of variance was used within generations with lines and treatments considered as fixed main effects. The statistical model was:

$$Y_{ijk} = \mu + L_i + T_j + (LT)_{ij} + e_{ijk}$$

where, i = 1, 2, 3, 4 lines; j = 1, 2 treatments; k = 1, 2 ... r individuals.

In an attempt to evaluate the trends over generations the data from $S_{16}(R_3)$ and $S_{17}(R_4)$ generations were analyzed by a factorial analysis of variance using a fixed effects model with years, lines

and treatments as main variables. In both the cases an analysis by the method of least squares was used because of disproportionate subclass numbers. Duncan's Multiple Range Test was applied to test the differences between pairs of means.

A stepwise regression procedure was used to determine the effect of growth parameters on egg production patterns in all the lines in both the generations.

Results and Discussion

Trial 1

Sexual maturity: There was a highly significant difference between lines in the S_{15} generation for age at sexual maturity with pullets from the HWS line maturing 21.6 days earlier than those from the LWS line (Table 1). The difference between lines for age at sexual maturity was significant within 3 of the 4 periods and was consistent with earlier reports involving these lines (Siegel, 1962, 1970, 1974; Udale, 1972). Siegel et al. (1968) reported significant differences between HWS and LWS lines for gonadotropin content at fixed chronological ages that could explain the differences in age at sexual maturity.

Body weight: Pullets from the HWS line were significantly heavier than those from the LWS line in all comparisons (Table 1). This was expected because these lines had undergone bidirectional selection for body weight at 8 weeks of age (Siegel, 1974), and juvenile and adult body weight were highly correlated (Ideta and

Siegel, 1966). The important observation here, however, is that the differences in body weight occur not only at different chronological ages, but also at the same physiological ages. This suggests correlated responses on the physiological processes controlling reproduction to selection for juvenile body weight.

Egg production: Means and standard errors for number and for percentage hen-day production (% HDP) of normal, defective and total eggs are presented in Table 1. There were significant differences between lines for frequency and percentage of defective eggs, with values for HWS pullets being larger than those for LWS pullets. No such differences between lines were observed in normal egg production. When analyses were made within periods, LWS pullets produced significantly more normal eggs in the first period than HWS pullets, with no significant differences noted between lines in the subsequent periods. This was consistant with the observations of Udale (1972) who found that the pullets from the LWS line produced significantly more normal eggs than those from the HWS line during the first 28 days of egg production. The difference of 5.9 eggs between lines for total eggs approached significance ($0.05 \leq P \leq 0.10$).

Analyses of variance for egg production data are presented in Table 2. No significant differences were found among periods when egg production was expressed as % HDP. When expressed as actual numbers of eggs there were highly significant differences among periods for the number of normal and total eggs. These differences were primarily because of the time comparisons from 20 to 80 days.

Table 1. Means and standard errors[1] of various measures of female reproduction by lines and periods, S_{15} generation

Trait	Period (days) I (0-20) HWS	I (0-20) LWS	II (0-40) HWS	II (0-40) LWS
Age at 1st egg	181 ± 12[a]	208 ± 16[a]	160 ± 4[a]	188 ± 9[b]
No. of eggs				
Normal	11.0 ± 1.3[a]	14.7 ± 0.6[b]	25.4 ± 1.7[a]	26.8 ± 5.7[a]
Defective	2.2 ± 0.8[a]	1.0 ± 1.0[a]	8.6 ± 3.0[b]	0.4 ± 0.3[a]
Total	13.2 ± 1.4[a]	15.7 ± 1.5[a]	34.0 ± 2.0[b]	27.2 ± 5.5[a]
% HPD				
Normal	55.0 ± 6.5[a]	73.8 ± 3.2[b]	63.5 ± 4.2[a]	67.0 ± 14.2[a]
Defective	11.3 ± 3.8[a]	5.0 ± 5.0[a]	21.5 ± 7.6[b]	1.0 ± 0.6[a]
Total	66.2 ± 7.2[a]	78.7 ± 7.5[a]	85.0 ± 4.9[a]	67.5 ± 13.7[a]
No. of follicles				
Ruptured	6.0 ± 0.4[b]	3.5 ± 0.3[a]	6.0 ± 0.5[b]	3.6 ± 0.5[a]
Developing (> 0.3g)	9.2 ± 1.1[b]	4.5 ± 0.5[a]	8.6 ± 0.7[b]	4.0 ± 0.5[a]
Wt. of ovary				
Absolute (g)	45.4 ± 12.6[b]	19.9 ± 2.6[a]	46.5 ± 5.0[b]	19.7 ± 3.8[a]
Adjusted (g/100 g wt)	1.5 ± 0.3[a]	1.2 ± 0.2[a]	1.5 ± 0.1[a]	1.2 ± 0.2[a]
Body wt. at sacrifice (Kg)	3.0 ± 0.3[b]	1.7 ± 0.3[a]	3.1 ± 0.1[b]	1.5 ± 0.1[a]

Table 1 Cont.

Trait	Period (days) III (0-60) HWS	III (0-60) LWS	IV (0-80) HWS	IV (0-80) LWS	Overall difference HW-LW
Age at 1st egg	161 ± 7[a]	185 ± 3[b]	161 ± 5[a]	172 ± 2[b]	-21.6**
No. of eggs					
Normal	38.4 ± 3.3[a]	40.6 ± 3.8[a]	56.4 ± 4.4[a]	57.3 ± 3.4[a]	- 1.8
Defective	11.4 ± 3.6[b]	0.4 ± 0.3[a]	9.5 ± 1.3[b]	0.7 ± 0.5[a]	7.7**
Total	49.8 ± 1.7[b]	41.0 ± 3.8[a]	66.3 ± 5.6[b]	58.0 ± 3.8[a]	5.9†
% HDP					
Normal	64.0 ± 5.5[a]	67.6 ± 6.2[a]	71.0 ± 5.6[a]	71.7 ± 4.3[a]	- 5.7
Defective	19.6 ± 6.0[b]	0.7 ± 0.4[a]	11.9 ± 1.6[b]	0.9 ± 0.6[a]	14.3**
Total	83.0 ± 2.9[a]	68.3 ± 6.4[a]	82.9 ± 7.0[a]	72.5 ± 4.8[a]	9.7
No. of follicles					
Ruptured	5.6 ± 0.5[b]	3.2 ± 0.7[a]	5.2 ± 0.7[a]	3.5 ± 0.5[a]	1.2**
Developing	7.8 ± 0.7[b]	4.0 ± 0.3[a]	5.2 ± 1.1[a]	4.2 ± 0.2[a]	3.4**
Wt. of Ovary					
Absolute	46.1 ± 2.9[b]	23.5 ± 2.3[a]	29.7 ± 6.7[a]	23.8 ± 2.5[a]	19.2**
Adjusted	1.5 ± 0.1[a]	1.5 ± 0.1[a]	1.0 ± 0.2[a]	1.3 ± 0.1[a]	0.0
Body wt. at sacrifice	3.0 ± 0.1[b]	1.6 ± 0.1[a]	2.9 ± 0.2[b]	1.8 ± 0.1[a]	1.34**

[1]Within a period any two means on the horizonal with the same superscript are not significantly different ($P \leq 0.05$).

** $P \leq 0.01$

* $P \leq 0.05$

Table 2. Least squares analysis of variance for various measures of female reproduction, S_{15} generation

Source of Variation	df	Mean Squares									
		No. of Eggs			% Hen Day Production			No. of Follicles		Wt of Ovary	
		Normal	Defective	Total	Normal	Defective	Total	Ruptured	Developing	Absolute (g)	g/100 g body wt
Lines (L)	1	38	525**	281†	3004**	2992**	292	49.2**	122.6**	3995**	0.04
Periods (P)	3	3572**	30	4087**	106	116	38	0.6	9.1*	139	0.24
L * P	3	5	38†	61	24	24	219	0.4	8.3*	261†	0.20
Error	32	68	16	77	62	62	141	1.6	2.5	97	0.11

** $P \leq .01$

* $P \leq .05$

† $P \leq .10$

The lack of period effect for number of defective eggs indicates that defective egg production is not linear with days in production, but varies with stage in egg production cycle. The line by period interaction for % HDP of defective eggs was not significant, while it approached significance ($.05 \leq P \leq 0.10$) for the frequency of defective eggs. In the HWS line the number of defective eggs progressively increased to 40 days of egg production and tended to decline thereafter, while in the LWS line they decreased from 20 to 40 days and plateaued (Fig. 1). In view of this, periods were analyzed within lines. There were no significant differences in number of defective eggs among periods in the LWS line. In the HWS line, however, there was a significant difference between periods I and III in the frequency of defective eggs.

Since the effect of age at sexual maturity was eliminated by examining production during a fixed period, these data suggest that interactions exist between growth rate, frequency of defective eggs, and stage of the egg production cycle. The lack of significant interactions between lines and periods for normal and total eggs indicate that both lines responded similarly over time. During the first 80 days of production 95% of the HWS and 35% of the LWS pullets laid one or more defective eggs. This was significant according to Chi square analysis. Using the S_{14} generation of these same lines, Udale (1972) found that 87% of the HWS and 45% of the LWS pullets produced defective eggs, to 300 days of age, while only 76% of the HWS and 19% of LWS pullets showed this syndrome during the first 28

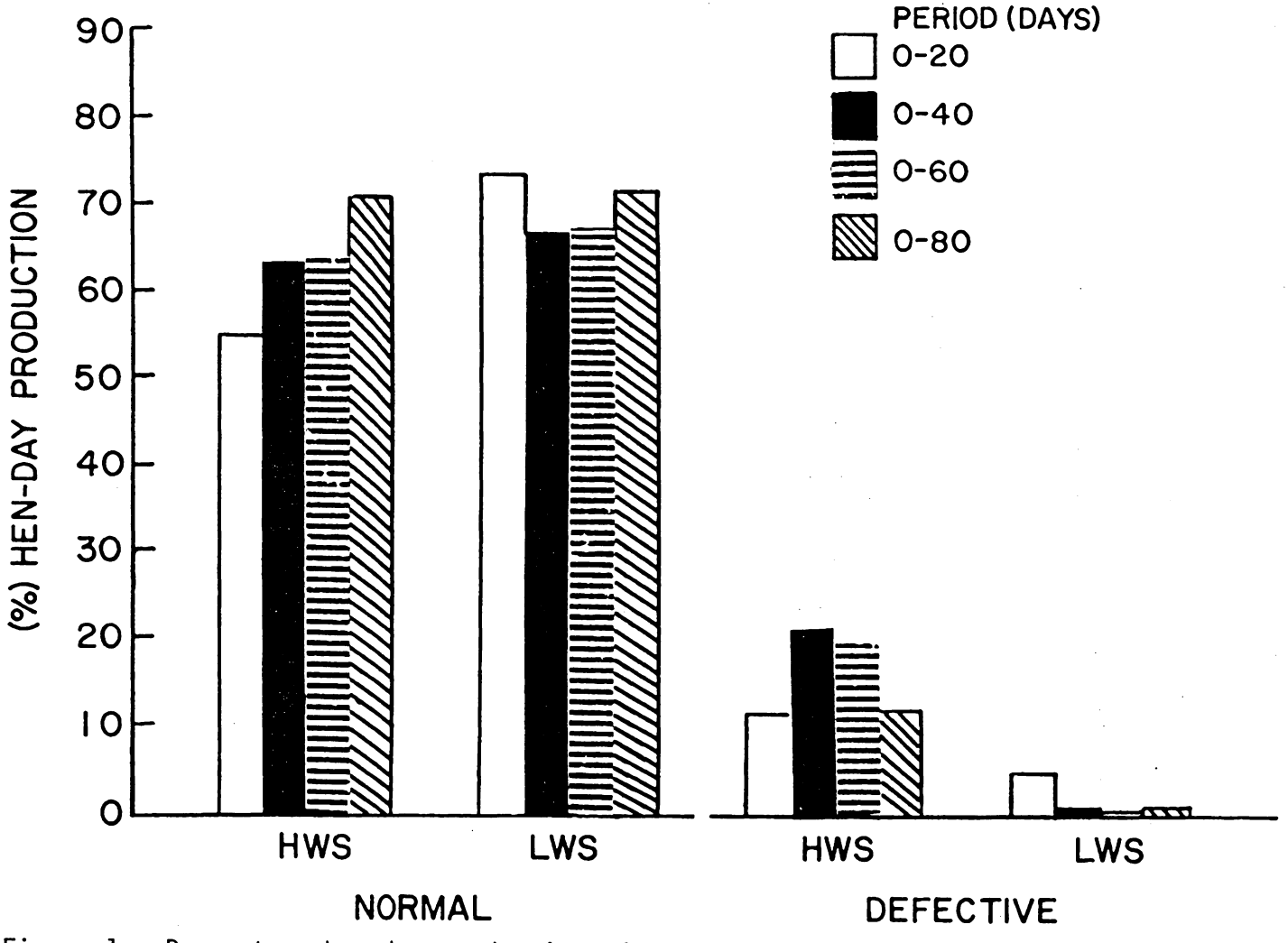


Figure 1. Percentage hen-day production of normal and defective eggs by lines and periods in the S_{15} generation.

days of egg production. This shows that the frequency of pullets laying defective eggs increases progressively after 28 days of lay. The results reported here are consistent with those of Udale (1972).

Examining the egg production data by 20-day periods using only those birds autopsyed (Table 3), revealed significant differences among lines for % HDP of defective eggs, and no differences in normal egg production. There was no significant period effect for % HDP of total eggs in the first two 20-day periods, while significant differences were present when analyzed for the first three and four 20-day periods. This was because the frequency of defective eggs increased for the period from 41-60 days of production. The significant line by period interaction was due to varying frequencies of defective eggs in HWS line in 20-day periods in comparison to the constancy of the LWS line.

Lighting: Comparisons of % HDP of normal, defective and total eggs after exposure of pullets to continuous illumination at 258 days of age are presented by weeks and lines in Table 4. There were highly significant differences between lines for the frequency and the % HDP of normal and total eggs (Table 5) with values for HWS pullets being larger than those for LWS pullets (Table 4). Again these differences may be primarily due to differences in the physiological ages (Table 5). No difference was observed between lines for frequency of defective eggs. Since the frequency of defective eggs had plateaued, the ovulatory rhythm for producing normal eggs was not influenced by the continuous illumination. Although the continuous

Table 3. Analyses of variance for percentage HDP of normal, defective and total eggs by periods, S_{15} generation

Source of Variation	df	Mean Squares		
		Normal	Defective	Total
		First two 20-day periods		
Lines (L)	1	21.9	1714.6**	1531.2
Periods (P)	1	82.4	145.7	31.2
L X P	1	111.3	16.3	151.2
Error	16	291.3	102.5	639.4
		First three 20-day periods		
Lines (L)	1	73.7	2317.0**	1613.3*
Periods (P)	2	6.4	335.4*	455.9
L X P	2	64.1	239.5	175.8
Error	24	118.9	73.2	308.3
		First four 20-day periods		
Lines (L)	1	19.3	1669.3**	1302.1†
Periods (P)	3	80.3	585.0**	436.8
L X P	3	47.9	304.9**	181.2
Error	40	147.6	33.8	328.5

** $P \leq .01$

* $P \leq .05$

† $P \leq .10$

Table 4. Means and standard errors of various measures of ovarian activity by lines, and weeks under continuous illumination[1], S_{15} generation

Week	Line	% HDP			No. Follicles		Wt of Ovary	
		Normal	Defective	Total	Ruptured	Developing	Absolute	g/100 g body wt.
I	HWS	68.6 ± 5.3	0	68.6 ± 5.3	6.2 ± 0.4	6.8 ± 0.4	48.4 ± 1.7	1.5 ± .03
	LWS	68.6 ± 2.9	0	68.6 ± 2.9	3.4 ± 0.2	4.8 ± 0.4	28.1 ± 2.7	1.6 ± .12
II	HWS	67.1 ± 6.6	0	67.1 ± 6.6	8.4 ± 0.4	8.4 ± 0.3	66.0 ± 2.8	2.0 ± .12
	LWS	59.5 ± 2.4	0	59.5 ± 2.4	4.3 ± 0.3	4.3 ± 0.7	26.6 ± 3.7	1.6 ± .25
III	HWS	70.5 ± 5.9	3.8 ± 2.2	74.3 ± 5.3	8.4 ± 0.9	8.2 ± 0.6	60.5 ± 5.2	1.8 ± .08
	LWS	58.1 ± 15.1	8.7 ± 8.7	66.7 ± 7.2	4.4 ± 0.5	4.4 ± 0.5	24.4 ± 5.7	1.5 ± .27
IV	HWS	63.6 ± 6.6	4.3 ± 2.6	67.9 ± 6.9	7.8 ± 0.7	7.0 ± 0.3	47.9 ± 3.2	1.4 ± .11
	LWS	45.0 ± 10.9	0	45.0 ± 10.9	3.8 ± 0.7	4.0 ± 0.3	20.5 ± 1.7	1.4 ± .22
V	HWS	77.1 ± 2.7	4.0 ± 1.5	81.1 ± 12.0	7.6 ± 0.7	6.2 ± 0.2	45.8 ± 4.6	1.4 ± .16
	LWS	54.3 ± 5.8	0	54.3 ± 5.8	3.6 ± 0.3	4.0 ± 0.3	23.4 ± 0.7	1.5 ± .08
Overall diff	(HWS-LWS)	14.9**	0.6	15.5**	3.2**	4.0**	30.3**	0-19

[1]The photoperiod was 24 hours light and 0 hours darkness

** $P \leq .01$

Table 5. Least squares analyses of variance of various female reproductive traits under continuous light, S_{15} generation

Source of variation	df	Mean Squares					
		No. of Eggs			% HD Production		
		Normal	Defective	Total	Normal	Defective	Total
Lines (L)	1	175**	1.25	206**	1210**	101	1238**
Weeks (W)	4	490**	2.89	538**	135	45	191
L X W	4	22	2.42	36	82	40	125
Error	39	17	2.19	13	153	40	102

Table 5. Cont.

Source of Variation	df	Mean Squares			
		No. of Follicles		Weight of Ovary	
		Ruptured	Developing	Absolute	/100 g body wt
Lines (L)	1	194.1**	127.2**	11288**	0.47
Weeks (W)	4	3.3	2.0	168	0.08
L X W	4	1.6	3.9*	265*	0.34
Error	39	1.8	1.1	74	0.16

** $P \leq 0.01$
*$P \leq 0.05$

light increased normal egg production in HWS line it decreased it in LWS line. At this stage of the laying cycle the ovarian and oviducal functions were probably at synchrony in the HWS line and the increased feeding time under continuous illumination was utilized with advantage even though diurnal rhythms in feeding activity exist (Siegel and Guhl, 1956). The differential response of these lines to continuous light may be due to photorefractoriness in the LWS line and photostimulation in HWS line. Photorefractoriness was first described by Riley (1936) and is characterized by a rapid regression of gonads under increased photoperiod which may be regarded as an evolutionary adaptive mechanism in migratory birds (Burger, 1947; Wolfson, 1952). Photorefractoriness was probably caused by a temporary inhibition of the appropriate hypothalemic centers from a negative feedback of gonadal hormones (Farner and Follett, 1966). The "light shock" was of sufficient severity to cause three of the LWS pullets to cease egg production. It was not possible to determine how long it would take for these pullets to regain photosensitivity because they were sacrificed at weekly intervals. There is also the possibility that these lines differ in the latency periods necessary to pass through a period of photostimulation to photorefractoriness. These latent periods may be more than five weeks in HWS and one to two weeks in the LWS line. Although the basic components of photoperiodic control mechanisms may be similar, they would have developed different performance characteristics in their different genetic backgrounds.

The pattern of % HDP displayed under continuous illumination by these lines at a common chronological age confirm the thesis (Osborne, 1952) that sudden changes in photoperiod play an important part in genotype-environment interactions. Since the pullets were exposed to several photoenvironments from hatching to 258 days it is difficult to draw conclusions without confounding the effects of previous photoperiods on subsequent performance. Although the literature on the mechanisms of photorefractoriness is mostly fragmentary and speculative, complex interactions between photoperiods, endogenous circadian components, and genetic backgrounds are expected. It would be interesting to conduct an experiment comparing these lines under continuous illumination from hatching through an entire laying cycle.

Ovarian activity: Means and standard errors for measures used to estimate ovarian activity are summarized by lines and periods in Table 1. Ovarian weight and number of developing and ruptured follicles were significantly greater in HWS than LWS pullets during each of the first three periods of lay and when all periods were pooled. This is consistent with the reports of Jaap and Mohammadian (1969) and Udale (1972) who found that the fast growing chickens had a significantly greater number of developing and ruptured follicles than the slow growing individuals. Similar findings were reported for turkeys by Nester et al. (1970). The lack of significant differences between lines in ovary weight, and number of developing and ruptured follicles after about 60 days of production indicates that the relationship between growth rate and ovarian activity is

primarily physiological. Bacon et al. (1972) using meat and egg-type turkeys, which differed during early production cycle in the frequency of growing and ruptured follicles, did not find significant differences during the later part of laying period. Such a relationship is also evident in our lines (Fig. 2).

Adjustment of ovarian weight for differences in body weight eliminated the differences between lines and periods. This suggests that follicular growth may not be the function of body growth at a particular physiological age. It is hypothesized that differences in neural thresholds for gonadotropin sensitivity may exist to account for the differences in follicular development observed between these lines. Jaap and Clancy (1968) failed to find significant differences in circulating estrogen levels between broiler and Leghorn strains known to differ in the number of follicles under rapid development. Using S_5 and S_6 generation pullets from these lines, Siegel et al. (1968) found significant differences between lines for adjusted ovarian weights at various chronological ages. This suggests differences in target organ sensitivity to gonadotropins (Siegel and Siegel, 1964; Siegel and Van Krey, 1968).

The effect of the duration of continuous light on the number of growing and ruptured follicles was not significant (Tables 4 and 5). The significant line-week interaction for the growing follicles and ovary weight was due to differences among weeks within HWS pullets and a consistency among weeks for the LWS pullets. Adjusted ovary weights did not differ among lines, which was consistent with that

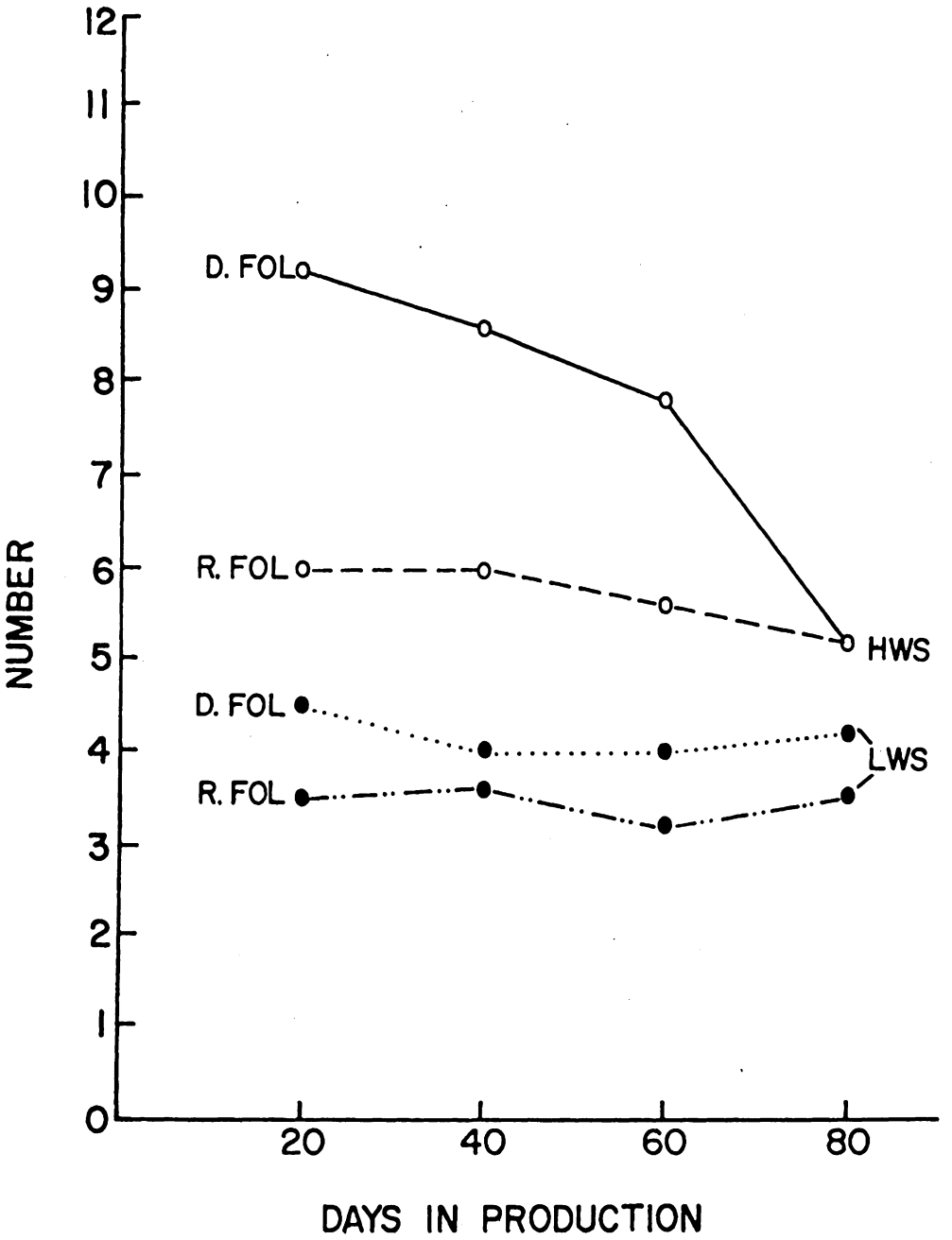


Figure 2. Frequency of developing and ruptured follicles after specific days in egg production, by lines in the S_{15} generation.

observed under a photoperiod of 14L:10D.

Trial 2

Means and analyses of variance for all quantitative traits measured in this trial are summarized in Tables 6 to 9 and 10 to 12, respectively. Differences between an increasing photoperiod and a light regime of 14L:10D were not significant for any of the traits measured in the S_{16} generation, except for ovarian weight in HWS line. Data were pooled over light treatments for subsequent discussions because the effect of photoperiodic treatments was significant in only 1 of 25 comparisons and for none of the interactions involving it and other main variables. Should details be desired, the means and standard errors of various reproductive parameters and gland weights are summarized by subclass groups in Appendix Tables 1 to 4.

There were significant differences between lines for all traits measured except % HDP of normal eggs (Tables 10, 11, and 12). The HWS pullets matured earlier and had heavier body, gland, and organ weights than LWS pullets. In addition the % HDP of defective and total eggs was greater for HWS than LWS pullets. Details of the comparisons between lines during the various production periods follow.

Age and weight at 1st egg: Differences among laying periods within lines were not significant for either age or weight at first egg (Table 8), indicating that representative samples of the populations were used in each period. The HWS pullets matured significantly earlier and weighed more at sexual maturity than the LWS pullets (Tables 7 and 8). These relationships under a restricted feeding

Table 6. Means of various traits by lines and photoperiods, S_{16} generation

Trait	HWS			LWS		
	Photoperiod[1]		diff	Photoperiod[1]		diff
	I	II	I-II	I	II	I-II
Age at 1st egg (days)	192	195	- 3	208	207	1
Body wt at 1st egg (g)	2835	2915	-80	1532	1539	-7
%HDP						
Normal	71.6	66.1	5.5	63.7	64.5	-0.8
Defective	9.2	13.5	- 4.3	0.1	0.5	-0.4†
Total	80.7	79.4	1.3	63.9	65.0	-1.1
No. of Follicles						
Ruptured	7.0	7.5	- 0.5	4.2	4.1	0.1
≥ 0.3 g	6.5	6.8	- 0.3	4.0	4.2	-0.2
≥ 0.1 g	16.9	16.4	0.5	10.0	9.7	0.3
Wt. of						
Ovary (g)	48.8	56.7	- 7.9*	24.7	26.2	-1.5
Liver (g)	65.0	68.0	- 3.0	32.0	33.2	-1.2
Thyroid (mg)	150.3	136.9	13.4	67.8	64.5	3.3
Pituitary (mg)	9.8	9.1	0.7	6.1	6.7	-0.6

[1]Photoperiod I = 14L:10D
II - 14L:10D with 3% increment/week

* $P \leq .05$

† $P \leq .10$

Table 7. Means for various traits by lines, S_{16} generation

Trait	HWS	LWS	diff HWS--LWS
Age at 1st egg (days)	193	207	- 14 **
Body wt. at 1st egg (g)	2880	1535	1345 **
%HDP			
Normal	68.4	64.1	4.3
Defective	11.6	0.3	11.3**
Total	79.9	64.4	15.5**
No. of Follicles			
Ruptured	6.5	3.9	2.6**
$\geq$ 0.3 g	6.7	4.1	2.6**
$\geq$ 0.1 g	16.6	9.8	6.8**
Wt. of			
Ovary (g)	53.3	25.4	27.9**
Liver (g)	66.7	32.5	34.2**
Thyroid (mg)	142.7	66.2	76.5**
Pituitary (mg)	9.4	6.4	3.0**
Adjusted Weights			
Ovary (g)	1.66	1.45	0.21*
Liver (g)	2.06	1.88	0.88**
Thyroid (mg)	4.46	3.80	0.66**
Pituitary (mg)	0.29	0.37	- 0.08**

** $P \leq 0.01$

* $P \leq 0.05$

Table 8. Meanslof age and weight at 1st egg and % hen day egg production by lines and periods (S_{16} generation).

Traits	Line	Laying Period (days) I (0-40)	II (0-80)	III (0-120)	IV (0-160)
Age at 1st egg (days)	HWS	194 [a]	193 [a]	196 [a]	191 [a]
	LWS	201 [b]	206 [b]	208 [b]	215 [b]
Body wt at 1st egg (g)	HWS	3009 [a]	2923 [a]	2740 [a]	2823 [a]
	LWS	1499 [b]	1517 [b]	1486 [b]	1651 [b]
%HDP					
Normal	HWS	62.5[a]	70.8[a]	71.7[a]	69.1[a]
	LWS	62.3[a]	68.4[a]	67.0[a]	56.3[a]
Defective	HWS	21.7[a]	13.3[ad]	3.3[c]	6.6[cd]
	LWS	0.3[b]	0.1[b]	0.4[b]	0.6[b]
Total	HWS	83.8[a]	84.0[a]	75.0[a]	75.7[a]
	LWS	62.5[b]	68.5[b]	62.7[b]	56.9[b]

[1]Means, for a given trait, with the same superscript are not significantly different ($P \leq .05$).

Table 9. Means[1] of ovarian traits and gland weights by lines and periods (S_{16} generation).

Traits		Days from Date of 1st Egg 40	80	120	160
No. of Follicles					
Ruptured	HWS	6.7[a]	6.8[a]	6.1[a]	6.5[a]
	LWS	4.0[c]	3.9[c]	4.9[c]	4.9[c]
≥ 0.3 g	HWS	7.7[a]	7.2[a]	6.1[b]	5.4[c]
	LWS	4.3[d]	4.4[d]	3.8[d]	3.7[d]
≥ 0.1 g	HWS	16.7[a]	17.9[a]	15.8[a]	15.8[a]
	LWS	9.1[b]	9.5[b]	9.7[b]	11.2[b]
Wt of					
Ovary (g)	HWS	53.2[a]	55.6[a]	54.7[a]	49.2[a]
	LWS	23.2[b]	27.7[b]	25.9[b]	24.8[b]
Liver (g)	HWS	64.5[a]	62.9[a]	74.5[b]	56.4[a]
	LWS	35.5[c]	33.2[cd]	30.5[d]	30.8[cd]
Thyroid (mg)	HWS	169.6[a]	132.8[b]	145.9[ab]	121.4[b]
	LWS	66.2[c]	69.5[c]	62.8[c]	66.5[c]
Pituitary (mg)	HWS	10.1[a]	9.8[a]	8.8[a]	8.6[a]
	LWS	5.9[b]	6.8[b]	6.3[b]	6.5[b]

[1]Means, for a given trait, with the same superscript are not significantly different ($P \leq 0.05$).

program were consistant with those observed under ad libitum feeding in Trial 1 demonstrating that a negative correlated response of sexual maturity to selection for body weight at eight weeks of age exists under both feeding programs.

Egg production: Pullets from the HWS line had a significantly greater % HDP of defective and total eggs than those from the LWS line (Table 7). The LWS pullets had a very low but uniform distribution of defective eggs throughout, while in the HWS line the incidence was significantly higher initially and changed over time (Table 8). The contrasting pattern observed between these lines was very similar to that described in Trial 1, and resulted in a significant line by period interaction. It is interesting that although the change in feeding program caused the age at first egg to differ significantly in the S_{15} and S_{16} generations, the defective egg production curves were similar in both generations (Fig. 3). Fuller et al. (1973) was able to reduce the incidence of defective eggs and thereby increased the number of settable eggs in broiler stocks by delaying sexual maturity through energy restriction and decreasing photoperiods. Van Middelkoop and Kuit (1974) reported that increasing age at sexual maturity by decreasing day length and restricted day length and restricted feeding would reduce the frequency of double yolk eggs, but not the incidence of two eggs a day. The results suggest specific genetic factors are expressed at a specific physiological rather than chronological age. This implies a genetic basis for erratic ovulation and oviposition patterns. Estimates of genetic parameters for these

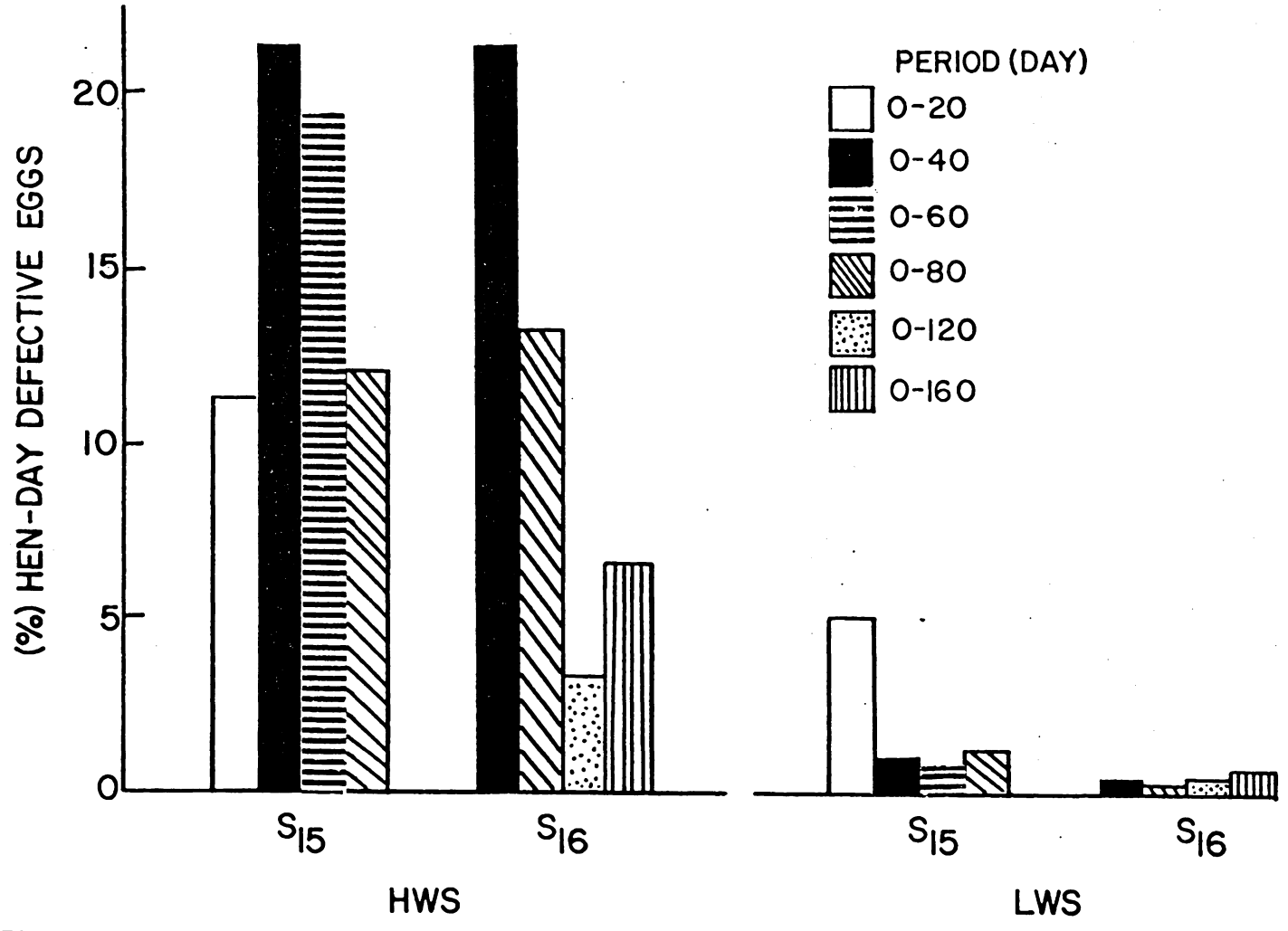


Figure 3. Percentage hen-day production of defective eggs by lines and periods in the S_{15} and S_{16} generations.

factors will be discussed subsequently in this dissertation.

When total egg production was examined, no significant differences were found between periods in either line, whereas significant differences existed between lines in all periods (Table 8). This lack of difference between periods for % HDP of total eggs in HWS line, while significant differences in the frequency of defective eggs were evident, was due mainly to the compensatory decrease in normal egg production when defective eggs were produced. This explanation cooraborates the observations of Jaap and Mohammadian (1969) and Udale (1972).

Ovarian activity: Although significant differences between lines were found for all traits measured, differences among periods were significant only for the number of follicles under rapid development (Tables 7 and 9). The number of rapid growing follicles observed in HWS pullets decreased significantly at 120 and 160 days of lay from that at 40 and 80 days of lay. This was in contrast to the constancy noted in the LWS line. The decline in the HWS line over time was partly due to atresia of the growing follicles. One or more atretic follicles were observed in the ovaries of 13 of the 16 HWS pullets sacrificed at 120 and 160 days after the onset of egg production. The incidence of pullets exhibiting atretic follicles progressively increased from 5 of 8 at 120 days to 8 of 8 at 160 days of lay. Chi-square analysis revealed that HWS pullets had significantly more atretic follicles than LWS pullets. Udale (1972) did not find differences in the incidence of atretic follicles between these lines during the first 28 days of lay. While no differences could be

detected between lines during early production (Nester et al., 1970), in the middle of the laying period the number of atretic follicles was significantly greater in rapid growing than in egg-type turkeys (Bacon et al., 1972). This is consistent with Udale's (1972) and our results and indicates that the incidence of atretic follicles increases during the egg production cycle. Gilbert (1960) suggested that a hormonal imbalance might be the cause of this condition and that reactivation of the follicles undergoing atrecia was also possible. Normal nesting behavior without oviposition, however, was observed when atresia of the matured follicle occured (Wood-Gush and Gilbert, 1970). The significant differences between the HWS and LWS lines for the number of developing, ruptured, and atretic follicles indicate that selection for body weight has resulted in correlated changes not only in gonadotropin secretory capabilities (Siegel et al., 1968), but perhaps also in neural thresholds of the hypothalamic-pituitary-gonadal complex.

A pullet was classified as an internal layer when egg material was observed in the body cavity during autopsy. Chi-square analysis of these data revealed a significantly greater number of internal layers among HWS than LWS pullets. Udale (1972), from data obtained from S_{14} generation HWS and LWS pullets, concluded that 35% of the developing follicles were lost between the ovary and cage floor. Although only limited data are available here, there is suggestive evidence that both internal laying and atresia of the developing follicles contribute to the discripancy between ovulation and egg production. Buss (1963), Gilbert (1967), and Bacon and Cherms (1968)

observed that ova of nearly identical weights would be ovulated at the same time and thus incorporated in the same egg. Various types of abnormal eggs may result when the time interval between ovulations is less than 21 hours (van Middelkoop, 1972). Thus, follicles weighing within 1 g of each other may be considered to be developing in pairs. The incidence of ova developing in pairs was significantly higher in HWS than in LWS pullets, and is consistent with the other observations discussed previously for these lines.

Weights of glands: There were highly significant differences between lines for liver, thyroid, and pituitary weights. In all cases the absolute weights were heavier for HWS than LWS pullets (Table 7). When adjusted for body weight the relationship between lines was similar for liver and thyroid weight but reversed for pituitary weight.

The larger absolute and adjusted liver weights in HWS than in LWS pullets may reflect an increase in liver lipids, mainly triglycerides (Balnave, 1968). Various studies have established that liver is the site for the synthesis of phospholipids and phosphoproteins (Flickinger and Rounds, 1956; Vanstone et al., 1957; Greengard et al., 1965; Heald and McLachlan, 1965) which are influenced by estrogens. Since phosphoprotein is released into the blood soon after synthesis (Greengard et al., 1965) and is exclusively of hepatic origin, it is not reasonable to assume that liver weight expresses a capability to synthesize yolk proteins and lipids. The liver, however, may act as the storage depot for triglycerides which are used in the synthesis of lipoproteins that is triggered by complex hormone potentials (largely)

Table 10. Least squares analysis of variance of age and weight at sexual maturity and % hen day egg production (S_{16} generation).

Source of Variation	df	Mean Squares				
		Age at Maturity	Body wt X 10^3	% hen day production		
				Normal	Defective	Total
Line (L)	1	3,596**	31,812**	187	3,196**	2,356**
Treatment (T)	1	22	47	77	228	12
L X T	1	82	28	54	60	9
Period (P)	3	78	53	92	328**	152
L X P	3	264	114	58	211*	21
T X P	3	145	44	24	51	124
L X T X P	3	426	65	98	48	159
Error	58	351	48	75	62	70

** $P \leq 0.01$

* $P \leq 0.05$

Table 11. Least squares analysis of variance of traits to measure ovarian activity (S_{16} generation

Source of Variation	df	Mean Squares					
		No. of follicles			Wt. of ovary		Wt. of yolk in follicles ≥ 0.3 g
		Ruptured	≥ 0.3 g	≥ 0.1 g	Absolute	Adjusted for body wt	
Line (L)	1	121.00**	116.30**	804.0**	13358**	57.9**	9751**
Treatment (T)	1	0.44	1.16	2.1	405*	72.3*	410
L X T	1	0.02	0.08	0.09	189	15.8	212
Period (P)	3	0.63	8.20**	4.1	89	21.1	266
L X P	3	0.49	2.09	12.6	30	4.3	109
T X P	3	1.12	0.21	9.9	11	2.9	22
L X T X P	3	1.30	0.54	14.5	18	1.8	31
Error	58	0.78	0.95	11.3	83	12.3	83

** $P \leq 0.01$

* $P \leq 0.05$

Table 12. Least squares analysis of variance of liver, thyroid and pituitary weights (S_{16} generation).

Source of Variation	df	Mean Squares					
		Weights (absolute)			Weights (adjusted for body wt)		
		Liver	Thyroid	Pituitary	Liver	Thyroid	Pituitary
Line (L)	1	21449**	108856**	154.4**	62**	795**	10.8**
Treatment (T)	1	39	1390	0.1	18	125	0.5
L X T	1	2	587	5.5	1	21	1.2
Period (P)	3	65	1808*	3.2	48**	698**	1.8
L X P	3	236*	2235**	4.5	27*	183	0.4
T X P	3	140	365	1.6	8	174	0.8
L X T X P	3	96	611	5.6	9	66	1.0
Error	58	64	478	3.7	8	88	1.1

** $P \leq 0.01$

* $P \leq 0.05$

estrogens) at different stages of egg production cycle.

The thyroids of HWS pullets were significantly larger than those of LWS pullets both on an absolute and an adjusted basis. There was also an age effect. The HWS pullets had significantly larger thyroids after 20 days of lay than at subsequent ages, while values were similar for all ages measured in the LWS line (Table 9). This was the cause of the significant line by period interaction for absolute thyroid weight.

The larger pituitary mass of HWS pullets in comparison to LWS pullets (Table 7) indicates differences in the potential to secrete gonadotropins. This difference is not surprising because these lines are known to differ in various reproductive parameters. The lower adjusted pituitary mass of HWS than LWS pullets is consistent with the observations of Farrington and Mellen (1967) and Siegel et al. (1968). The latter authors, using S_5 and S_9 generation pullets from the HWS and LWS lines, bioassayed the pituitary glands for gonadotropins. They found that the pituitary secretory capabilities for gonadotropins, although similar in both lines on a per unit basis, were different because of the greater pituitary mass in the HWS than in the LWS line.

Relationships among traits: Phenotypic correlations among various measures of reproduction were calculated within lines (Table 13). Emphasis in this discussion will be given to those correlations enclosed in the boxes because of their direct relevance to EODES. The correlations of ovarian weight with number and weight of growing

follicles were large and highly significant. Correlation coefficients were very similar for both lines with ovarian weight having a greater relationship with the weight rather than the number of follicles. These high relationships were expected because weight of the ovary is a function of these two variables.

The correlations among various components of egg production differed considerably in these lines. The high phenotypic correlation of 0.99 between % HDP of normal and total eggs in the LWS line resulted from the low frequency of defective eggs in that line. The association between these traits in the HWS line, while significant and positive (0.32), was of a considerably lower magnitude than that calculated for the LWS line. This is indicative of the higher frequency of defective eggs as a component of total egg production in the HWS line. The significant negative correlation of -0.65 between % HDP of normal and defective eggs in the HWS line supports the view that production of defective eggs is at the expense of normal egg production.

There was a significant negative correlation of age at sexual maturity with % HDP of normal and total egg production in the LWS line indicating that normal egg production may be improved by lowering age at sexual maturity. The lowering of the age at sexual maturity may have an opposite effect in HWS line. This is because the correlation between this trait and % HDP of normal eggs was essentially zero and its correlation with % HDP of defective eggs was negative and approached significance ($0.05 \leq P \leq 0.1$). Furthermore, in the HWS line, the % HDP of defective eggs was positively correlated with number and weight of

Table 13. Product moment correlation coefficients[1] between various traits (S_{16} generation).

Code	Trait	Code 1	2	3	4	5	6	7	8	9
1	Age at 1st egg		.07	-0.28†	-.27	.10	-.24	-.15	-.15	-.29†
2	%HDP normal eggs	-.36*		-.65**	.32†	-.07	-.16	-.21	-.20	-.03
3	%HDP defective eggs	.12	-.25		.51**	-.08	.55**	.42*	.39*	-.09
4	%HDP total eggs	-.34*	.99**	-.21		-.19	.50**	.30†	.27	-.15
5	No. ruptured follicles	.18	.17	-.05	.17		.05	-.07	-.04	-.06
6	No. growing follicles	-.13	.31†	-.15	.30†	.09		.67**	.65**	.21
7	Wt. of ovary	.05	.21	-.05	.21	.10	.63**		.99**	.33†
8	Wt. of growing follicles	.04	.21	-.04	.21	.11	.61**	.99**		.39*
9	Wt. of liver	-.42*	.19	-.19	.18	-.10	.29†	.10	.14	

** P ≤ .01 * P ≤ .05 †P ≤ .10

[1]Values for the HWS line is above and LWS line below the diagonal.

growing follicles and ovary weight while no such relationship existed in the LWS line. It is difficult to explain the lack of a significant correlation between the number of ruptured follicles and egg production in either line. Although the number of ruptured follicles reflect the relative ovulation rates, the rate of regression may mask this relationship. Egg production in itself is not a good index of the ovulation rate because of internal laying (Wood-Gush and Gilbert, 1970) and the counting of multiple yolked eggs as one.

Multiple regression equations were calculated within lines using stepwise regression procedures, to explain further the interrelationships between various traits in the two lines. The dependent variables were age and body weight at sexual maturity, percentage HDP of normal eggs, and the number of developing and ruptured follicles. In the HWS line 62.5% of variation in the dependent variable was accounted by only two variables, namely the percentage HDP of normal eggs and number of developing follicles. In contrast, these two independent variables accounted only 7.3% of the variation in the percentage HDP of defective eggs in the LWS line where there is little surplus yolk production. Other independent variables accounted for a nonsignificant amount of variation in the dependent variable in both the lines, suggesting that the relationship among variables is quite different in the HWS and LWS line.

Trial 3

Mortality: The frequency of mortality from caging to 300 days of age by lines and generations is presented in Table 14. A chi-square

Table 14. Incidence of mortality among HWS, LWS, HWR, and LWR lines from caging to 300 days of age, in S_{16} and S_{17} generations.

Line	$S_{16}(R_3)$			$S_{17}(R_4)$		
	Alive	Dead	% Mortality[2]	Alive	Dead	% Mortality[1]
HWS	159	38	23.9^{b}	142	8	5.6^{a}
HWR	75	18	24.0^{bc}	94	6	6.4^{a}
LWS	208	24	11.5^{a}	144	6	4.2^{a}
LWR	96	13	13.5^{ac}	82	2	2.4^{a}

[1] Percentages in a column with the same superscript are not significantly significant ($P \leq 0.05$).

[2] Chi-square test of independence was done on the actual frequencies.

test of independence revealed significantly higher mortality among HWS than LWS pullets in the $S_{16}(R_3)$ generation. No significant difference, however, was detected either between the two relaxed lines or between the selected and relaxed lines of a respective direction. There was no difference in mortality among lines in $S_{17}(R_4)$ generation. The differential mortality among generations may be attributed to the fact that the birds were vaccinated for Marek's Disease in $S_{17}(R_4)$ but not in $S_{16}(R_3)$ generation. Han and Smyth (1972) reported a greater resistence to Marek's Disease (MD) in slow rather than fast growing lines of chickens. Additional evidence of a negative genetic correlation between resistence to MD and body weight comes from the studies of Friers et al. (1972), and Gavora et al. (1974) who suggested that in view of the genetic antagonism between growth rate and resistence to Marek's Disease, the effectiveness of vaccination may be decreased in populations under selection for high growth rate. The higher incidence of mortality among HWS pullets in the S_{16} generation may also be due in part to the severe energy restriction during the first ten weeks after caging. Thus genotype-environment interactions appear to be important for the incidence of mortality.

Effect of photoperiod: There were no consistent differences between a progressively increasing and a constant light regime of 14L:10D, for egg production traits and age at maturity (Appendix Tables 5 and 6). The inconsistencies are reflected by significant generation-line-treatment interactions (Appendix Table 7). These results are consistent with those of Smith and Noles (1963) and Noles and Smith

(1964) who obtained no beneficial effect on reproductive traits by increasing the day length beyond 14 to 15 hours per 24-hour period. Although, the upper limit of response has not been clearly defined, Morris (1967) was of the opinion that a photoperiod of over 16 hours was probably of no advantage.

The requirement of the chicken for light intensity is in the nature of a fixed threshold, with a maximum stimulus of the photoperiodic mechanism when the light intensity is above 5 luxes (Morris, 1967). The light intensity used in this experiment ranged from 2.7 to 5.6 luxes at bird level which may explain in part for the inconsistencies in photoperiodic effects on reproductive traits.

Body and egg weights were largely uneffected by the increasing photoperiod (Table 6). This is probably because the effect of light on growth and reproduction is mediated through different physiological pathways. Light influences reproductive processes via the visual-hypothalamic complex (Benoit and Assenmacher, 1959; Farner, 1959; van Tienhoven, 1961), whereas its effect on growth is primarily due to its influence on the pattern of activity and feeding time (Shutze et al., 1961). Under a restricted feed intake, longer photoperiods may not be beneficial either for body or egg weights, which are suspected to be influenced by pleiotropic effects (Siegel, 1963). Because of the inconsistencies and the lack of differences of growth and reproductive parameters among photoperiodic effects, the data were pooled for subsequent comparisons among lines.

Body and egg weights: There were significant differences among lines for body weights at 8, 24 and 38 weeks of age, with the weights for the relaxed lines being intermediate to those of the selected lines (Table 15). This was expected because of the bidirectional selection for 8-week body weight and the high genetic correlations between this trait and body weight at 24 and 38 weeks of age (Siegel, 1963, 1970; Ideta and Siegel, 1966).

Both the initial and the 35-week egg weights followed the trend of body weights in the HW lines, while in the LW lines there were no differences between the selected and relaxed lines. Significant correlations between body weight and egg weight are well documented (Hale, 1961; Merritt and Gowe, 1962; Jaap et al., 1962, Siegel, 1963; Ideta and Siegel, 1966).

Age at sexual maturity: As expected the restricted feeding schedure in the $S_{16}(R_3)$ generation effectively delayed the age at sexual maturity. It is interesting to note that in both generations pullets from the relaxed lines matured earlier than those from their respective selected lines. Information, however, on the optimum body weight for early maturity is lacking.

Normal and defective eggs: Means and standard errors of egg production traits by lines and generations are presented in Table 16. The HWS and LWS lines did not differ significantly in percentage HDP of normal eggs in the S_{16} generation. The percentage HDP of normal eggs from the R_3 pullets of HWR line were significantly higher while those from the LWR line were significantly lower than that of their

respective selected lines. The results for this trait in the next generation were slightly different in that, while no significant difference existed between the selected and their relaxed lines, values for the high weight lines (HW) were superior to those of low weight lines (LW). Irrespective of the statistical significance, the pullets from the HWR line produced more normal eggs than those from the HWS line. This may possibly be due to noting the "first eggs" which were extra-calcified (Foster, 1970; van Middelkoop and Simons, 1970; van Middelkoop, 1972a) as normal in both the generations. The second egg is compressed sided and would be classified as defective (van Middelkoop and Siegel, 1976). Such an error in classification would result in higher values for the HWR line, if this line truely produced a greater number of "first eggs" than the HWS line. Such was the case in the S_{18} HWS and R_5 HWR lines when such eggs were classified as a distinct group (van Middelkoop and Siegel, 1976). The significantly higher percentage hen-day production of normal eggs in the HWS line compared to the LWS line in S_{17} generation may not be a contradiction with the results in Trial 1, since these data pertain to the laying period to January 1, rather than for a fixed physiological age as was the case in Trial 1.

To further evaluate the influence of differences in precocity on the length of laying period egg production data were examined for the first 28, 56 and 84 days of production in the S_{17} generation (Table 17). No significant differences were found between the HWS and LWS lines for percentage HDP of normal eggs at each of the

physiological ages considered. This suggests that the line differences in Trial 1 may be due to sampling. These results are also in contrast to those of Udale (1972) who reported a significantly higher percentage HDP of normal eggs in the LWS line during the first 28 days of lay. For the defective egg-types and total yolks, there was good agreement among the results for the laying period to January 1 and the three physiological ages.

The data on the percentage HDP of two distinct classes of defective eggs, namely the double yolk (DY) and broken (B) eggs, were analyzed by Chi-square because of the low incidence and the abnormal frequency distributions. Although the DY eggs relate to anamolies in ovarian activity and ovulation rhythms, the B eggs may be influenced by oviposition patterns, laying behavior and temperament of the bird, inherent characteristics of the egg shell and cage floor. The percentage HDP of DY eggs was significantly higher in the HW than in the LW lines in both the generations. The HWS pullets produced significantly more DY eggs than those from the HWR line, while no significant difference existed between LWS and LWR lines. This implies that the frequency of ova developing in pairs should be significantly more in HWS pullets compared to HWR pullets since mature follicles of near identical weights are ovulated simultaneously, resulting in multiple yolked eggs (Gilbert, 1969; Buss, 1963). Although we have autopsy evidence to show that HWS pullets have significantly more ova developing in pairs compared to LWS pullets (Trial 1 and 2), similar data on the relaxed lines are not available.

Table 15. Means and standard errors[1] of various traits in the $S_{16}(R_3)$ and $S_{17}(R_4)$ generations

Trait	Line	Generations $S_{16}(R_3)$	Generations $S_{17}(R_4)$
Body weight (g)			
8 weeks			
	HWS	1033 ± 9.0^{d}	1036 ± 6.0^{d}
	HWR	933 ± 15.0^{c}	922 ± 7.6^{c}
	LWS	399 ± 5.4^{a}	415 ± 5.1^{a}
	LWR	464 ± 5.8^{b}	483 ± 6.2^{b}
24 weeks			
	HWS	2402 ± 28^{d}	2920 ± 22^{d}
	HWR	2242 ± 28^{c}	2615 ± 21^{c}
	LWS	1283 ± 15^{a}	1353 ± 15^{a}
	LWR	1438 ± 16^{b}	1458 ± 20^{b}
38 weeks			
	HWS	3176 ± 42^{d}	3441 ± 27^{c}
	HWR	2848 ± 84^{c}	3117 ± 40^{b}
	LWS	1738 ± 15^{a}	1738 ± 18^{a}
	LWR	1802 ± 27^{b}	1762 ± 22^{a}
Egg weight (g)			
Initial			
	HWS	42.6 ± 0.8^{c}	40.3 ± 0.4^{c}
	HWR	39.3 ± 0.6^{b}	35.7 ± 0.4^{b}
	LWS	36.7 ± 0.4^{a}	35.0 ± 0.4^{ab}
	LWR	36.2 ± 0.4^{a}	34.0 ± 0.5^{a}
35 weeks			
	HWS	51.6 ± 0.9^{c}	51.9 ± 0.3^{c}
	HWR	49.2 ± 0.4^{b}	48.3 ± 0.4^{b}
	LWS	42.0 ± 0.4^{a}	41.3 ± 0.3^{a}
	LWR	42.5 ± 0.3^{a}	41.6 ± 0.4^{a}
Age at maturity			
	HWS	195 ± 1.7^{b}	172 ± 1.2^{b}
	HWR	189 ± 2.2^{a}	167 ± 1.7^{a}
	LWS	200 ± 2.1^{c}	195 ± 1.4^{d}
	LWR	192 ± 2.2^{ab}	180 ± 1.9^{c}
Shell thickness			
Initial	HWS	10.84 ± 0.14^{a}	
	LWS	10.68 ± 0.12^{a}	
35 weeks	HWS	11.93 ± 0.15^{a}	
	LWS	11.82 ± 0.10^{a}	

[1]Within a generation the means for a trait with the same superscript are not significantly different. ($P \leq 0.05$).

The consistent differences between the HWS and the HWR lines in the production of DY eggs provide a good illustration of the effect of relaxing selection for growth on follicular development and ovulation patterns. It may be hypothesized that genes controlling growth rate and rate of yolk synthesis are pleiotropic. Thus, relaxed selection, while stabilizing the selected trait, also has a correlated effect on yolk synthesis. A simple model is proposed to explain the physiological basis of yolk production patterns in the HWS and HWR lines. If yolk synthesis and deposition in the ovarian follicles are directly proportional to growth rate, then the HWS and HWR lines should differ in the rate of yolk synthesis because of difference in growth rate. This difference in growth rate (Table 15) would mean that HWS pullets deposit yolk in the follicles at a faster rate than HWR pullets. Faster yolk deposition may disturb the follicular hierarchy in the ovary leading to paired development, and resulting in the simultaneous ovulation and/or the ovulation in closer succession of two or more mature follicles (Gilbert, 1969). This also implies that the time interval between two successive follicles reaching maturity may be shorter in the HWS than in the HWR line. Although large numbers of abnormal eggs are caused by a rapid succession of ovulations, the "first eggs" are characterized by an ovulation interval of about 19 to 20 hours (van Middelkoop, 1972). Thus this model not only explains the higher incidence of DY eggs in the HWS line but also the possible higher frequency of extra-calcified eggs in the HWR line. Although these inferences may have the limitation of not knowing the precise

Table 16. Means and standard errors[1] of egg production traits by lines and generations

Trait	Line	Generations $S_{16}(R_3)$	$S_{17}(R_4)$
Percentage Hen Day Production			
Normal eggs	HWS	68.98 ± 1.34^b	67.45 ± 1.02^b
	HWR	73.00 ± 1.40^c	70.45 ± 1.24^b
	LWS	67.11 ± 0.87^b	63.87 ± 1.01^a
	LWR	62.84 ± 1.26^a	62.39 ± 1.12^a
Double-yolked eggs			
	HWS	0.92 ± 0.11^c	1.38 ± 0.14^c
	HWR	0.50 ± 0.13^b	0.92 ± 0.15^b
	LWS	0.04 ± 0.02^a	0.02 ± 0.01^a
	LWR	0.04 ± 0.02^a	0.01 ± 0.01^a
Broken eggs			
	HWS	1.35 ± 0.22^b	1.76 ± 0.19^d
	HWR	1.60 ± 0.30^b	1.25 ± 0.21^c
	LWS	0.14 ± 0.04^a	0.17 ± 0.04^b
	LWR	0.13 ± 0.04^a	0.05 ± 0.02^a
Total defective eggs			
	HWS	6.88 ± 0.88^c	7.36 ± 0.59^c
	HWR	4.86 ± 0.73^b	4.96 ± 0.45^b
	LWS	0.62 ± 0.13^a	0.97 ± 0.37^a
	LWR	0.52 ± 0.09^a	0.38 ± 0.08^a
Total yolks			
	HWS	76.78 ± 1.16^c	76.20 ± 1.02^b
	HWR	78.35 ± 1.15^c	76.33 ± 1.12^b
	LWS	67.77 ± 0.88^b	64.87 ± 0.89^a
	LWR	63.40 ± 1.24^a	62.78 ± 1.13^a

[1]Within a generation the means for a trait with the same superscript are not significantly different ($P \leq 0.05$).

ovulation timing and the oviducal term of the ovum, the model is consistent with the hypothesis proposed by Jaap (1969) that selection for growth (protein anabolism) also stimulates lipoprotein anabolism in the liver and its subsequent deposition in ovarian follicles.

The paired follicular development and the production of significantly more DY eggs in the HWS line does not necessarily imply greater yolk synthesis. This is because the paired development of ova tends to disturb the hierarchical organization of growing follicles and creates a lag in follicular development. This was evidenced in both generations by the nonsignificant difference between the HWS and the HWR lines for percentage HDP of total yolks. Udale (1972) showed that follicular development was about 8 hours longer in the HW than LW lines and his data and ours (Table 17) show greater yolk production in the HW than LW lines. In a population of White Leghorns, Lowry and Abplanalp (1968) found that double-yolk eggs markedly increased due to selection, without a significant increase in total yolk production. In contrast, van Middelkoop (1973b) reported in a broiler population a correlated increase in total yolks consequent to selection for double-yolk eggs.

There were significant differences in percentage HDP of broken eggs with the incidence greater for HW than for LW pullets (Table 15). Although, specific causes of egg breakage were not established, genetic factors, drop height, and the effective mass of the cage floor seem to have important effects (Carter, 1971). The effective mass of the cage floor assumes higher values for heavier birds which is exclusively due

Table 17. Means and standard errors[1] of egg production traits for 28, 56 and 84 days of production by lines, in S_{17} generation

Trait	Line	Days in Production 28 days	56 days	84 days
Percentage Hen-Day Production				
Normal eggs				
	HWS	17.9 ± 0.3^a	38.1 ± 0.6^a	57.7 ± 0.2^a
	HWR	19.1 ± 0.4a	40.6 ± 0.7^b	61.0 ± 0.7^b
	LWS	18.1 ± 0.5^a	38.2 ± 0.6^a	57.1 ± 0.8^a
	LWR	18.7 ± 0.5^a	37.4 ± 0.8^a	54.8 ± 1.0^a
Double yolked eggs				
	HWS	1.0 ± 0.1^c	1.6 ± 0.1^c	1.8 ± 0.2^c
	HWR	0.7 ± 0.1^b	1.1 ± 0.2^b	1.3 ± 0.2^b
	LWS	0.0 ± 0.0^a	0.0 ± 0.0^a	0.0 ± 0.0^a
	LWR	0.0 ± 0.0^a	0.0 ± 0.0^a	0.0 ± 0.0^a
Broken eggs				
	HWS	0.6 ± 0.1^d	1.0 ± 0.1^d	1.5 ± 0.1^d
	HWR	0.4 ± 0.1^c	0.8 ± 0.1^c	1.1 ± 0.2^c
	LWS	0.1 ± 0.0^b	0.1 ± 0.0^b	0.2 ± 0.0^b
	LWR	0.0 ± 0.0^a	0.0 ± 0.0^a	0.1 ± 0.0^a
Total Defective eggs	HWS	4.5 ± 0.3^c	7.1 ± 0.6^c	8.5 ± 0.7^c
	HWR	3.0 ± 0.3^b	4.7 ± 0.5^b	5.9 ± 0.6^b
	LWS	0.4 ± 0.1^a	0.5 ± 0.1^a	0.6 ± 0.1^a
	LWR	0.3 ± 0.1^a	0.4 ± 0.1^a	0.5 ± 0.1^a
Total Yolks				
	HWS	23.4 ± 0.4^b	46.8 ± 0.7^b	68.1 ± 0.9^b
	HWR	22.9 ± 0.4^b	46.5 ± 0.7^b	68.2 ± 1.1^b
	LWS	18.5 ± 0.5^a	38.7 ± 0.6^a	57.8 ± 0.8^a
	LWR	19.0 ± 0.5^a	37.9 ± 0.8^a	55.3 ± 1.0^a

[1] Within a column the means for a trait with the same superscript are not significnatly different ($P \leq 0.05$).

to body weight, and this may in part be the cause of the higher frequency of egg breakage in the HW lines. Similarly, the higher frequency of egg breakage of S_{17} generation HWS pullets than R_4 generation HWR pullets may be attributed to the former being heavier than the latter. The mean drop height was one of the important sources of variation for the crack incidence in eggs (Tyler and Geake, 1958). The contribution of mean drop height to egg breakage is, however, difficult to assess in the absence of data on the laying behavior of the bird. Although the birds from the HW lines were generally taller than those from the LW lines, this variable need not necessarly effect the incidence of egg breakage, provided the bird squats at the time of oviposition. Silber and Merat (1974) found that the correlation between the percentage broken eggs and shank length was low, suggesting that the behavior of the bird at laying was more important than its height. Although pullets from the HWS line consistently exhibited a higher incidence of broken eggs than those from the LWS line, there were no differences among lines for shell thickness either at the commencement of egg production or at the age of 35 weeks (Table 16). These data, available for S_{16} generation only, provide indirect support to the reports of Anderson et al. (1970) and Carter (1970) that the incidence of cracks is largely independent of shell thickness.

When the comparisons were made for the frequency of total defective eggs, irrespective of the etiological causes, pullets from the HWS line produced significantly more total defective eggs than those from the HWR line (Table 16). This difference may again be due

Table 18. R^2 values[1] when the dependent variable is %HDP of normal eggs with various independent variables

Independent variable	$S_{16}(R_3)$				$S_{17}(R_4)$			
	HWS	HWR	LWS	LWR	HWS	HWR	LWS	LWR
Body weight at:								
8 weeks	2.77	0.16	0.14	0.38	1.51	0.99	0.02	9.92
24 weeks	0	10.10	2.42	1.67	0.59	1.45	6.91	0.48
38 weeks	1.20	1.19	7.40	0.05	0.13	0.54	3.36	1.53
Age at maturity	0.43	8,82	0.15	0.17	0.34	0.44	1.38	1.06
Egg weight:								
Initial	0.15	0.27	0.01	2.53	2.60	0.02	1.20	0.32
35 weeks	0	1.72	1.65	1.61	0.39	4.50	2.46	0.13
%HDP total defective eggs	8.86	9.23	1.16	2.03	10.09	23.17	1.40	0.06

1 = R^2 X 100

to the failure to delineate extra-calcified from normal eggs. The LWS and LWR lines, while not different from each other, produced significantly fewer total defective eggs than the HW lines in both the generations.

Stepwise regression procedures were used to assess the relative importance of body weight, egg weight, age at sexual maturity and percentage HDP of total defective eggs in the production of normal eggs. R^2 values were largely inconsistent and low in all lines in both generations (Table 18). It is traditionally believed that delaying sexual maturity age offers significant beneficial effect on the production of normal eggs. The multiple regression data indicate no influence of sexual maturity age on the rate of normal egg production. This was also evident by the nonsignificant difference in the percentage HDP of normal eggs even when there were significant differences in ages at first egg. Similarly, egg weight was a poor measure of the variability in percentage HDP of normal eggs.

Summary

The first two trials were conducted to study the oviposition patterns and ovarian activity at various physiological ages in S_{15} and S_{16} generation pullets from lines selected didirectionally for juvenile growth. In the third trial, correlated responses in egg production patterns were studied in the $S_{16}(R_3)$, and $S_{17}(R_4)$ generations of the selected lines and their respective relaxed lines when maintained under two nutritional and photoperiodic environments.

The ovulation and oviposition patterns differed among lines with the HWS pullets exhibiting a higher frequency of arhythmic ovulations and production of defective eggs at each of the physiological ages studied. A delaying of sexual maturity did not significantly change the pattern of defective egg production over a fixed period of lay. The pattern of defective egg production also differed between the selected lines. These data were supported by autopsy data with HWS pullets showing more developing, ruptured and atretic follicles, a higher frequency of internal laying and simultaneous development of two or more ovarian follicles than LWS pullets. The possible physiological mechanisms involved in the differences among lines were discussed in view of the differences in certain gland weights. The strong negative relationship between normal and defective eggs found in HWS line were supported by multiple regression analysis.

Increasing photoperiod beyond 14 hours in a 24-hour period did not have any consistent effect on growth and reproductive parameters. Relaxed selection resulted in fewer double-yolked and total defective eggs in the high line, while no significant effect was noticed in low line. A model correlating protein anabolism for growth and lipoprotein anabolism for yolk synthesis was presented and the significance of these on egg production patterns was discussed.

EXPERIMENT II. HERITABILITY ESTIMATES, PHENOTYPIC, GENETIC AND ENVIRONMENTAL CORRELATIONS

It has become increasingly evident that erratic egg syndrome is a characteristic feature of many broiler populations. The influences of heredity on this syndrome are still ambiguous as are the genetic and environmental correlations between growth rate, yolk production and egg production patterns. This experiment was designed to provide estimates of genetic parameters of and between growth traits and erratic and normal ovulation patterns.

Materials and Methods

Genetic stocks and management: The development of the lines, selection procedures and management practices were discussed in Experiment I. The genetic parameters reported here were estimated from S_{16} and S_{17} generation data for two lines (HWS and LWS) divergently selected for 8-week body weight and their respective relaxed lines (HWR, LWR) in the R_3 and R_4 generations.

Although the formulations of rations were the same in both generations the feeding levels differed. Pullets in $S_{16}(R_3)$ generation were fed on alternate days during the first ten weeks after caging, followed by daily restricted feeding thereafter. Caging was at 119 days of age. This was in contrast to daily restricted feeding to about 2/3 of *ad libitum* from caging to January 1, in $S_{17}(R_4)$ generation.

Hence, the feed restriction may be referred to as severe in $S_{16}(R_3)$ and moderate in $S_{17}(R_4)$ generations.

Measurement of traits: Body weights were obtained at 8 and 24 weeks of age. Measurements were to the nearest g at 8 weeks and nearest 10 g at 24 weeks of age. Age at sexual maturity was measured as the number of days from hatching to the date of first egg. Egg production was recorded daily on an individual bird basis between 8:00 and 9:30 a.m. from the date of first egg to January 1. Both normal and various types of defective eggs were recorded. Since the frequencies of specific defective egg-types were very low, they were pooled for analysis. The analyses were also performed on total yolks because of the multiple ovulations and incorporation of two or more yolks in the same egg. In view of the controversy as to whether broken eggs are defective, analyses were made with broken eggs included and excluded in the frequency of defective eggs. The term used when broken eggs were excluded was ovulatory defective. To correct for the differences in precocity (Bray et al., 1960), percentage hen-day production (HDP) was used rather than the number of eggs laid. Percentages were transformed to arc sin $\sqrt{\%}$ before analysis. Individual egg weights in g, were obtained for the first three eggs laid by a pullet (initial egg weight) and for those laid during a period of three consecutive days when the pullets reached 35 weeks of age.

Analyses: A hierarchical mating design was used to produce progeny for data to calculate genetic parameters. Heritabilities, based on paternal and maternal half-sib and full-sib correlations were estimated each generation from variance component analysis (King and Henderson, 1954; Lerner, 1958; Dickerson, 1961).

The statistical model was:

$$X_{ijkl} = \mu + L_i + S_{ij} + D_{ijk} + E_{ijkl}$$

where X_{ijkl} was the measurement on the lth offspring of the kth dam mated to jth sire within the ith line. All the elements in the model except μ are uncorrelated variables with zero mean and variances of σ^2_L, σ^2_S, σ^2_D, and σ^2_E.

Heritabilities were computed for the two upward (HW) and the two downward (LW) lines separately since they represented divergent genotypes. Analyses were also performed for all lines for a combined estimate. Theoretical estimates of genetic, phenotypic and environmental correlations were calculated each generation by the procedure developed by Hazel et al. (1943) using analysis of variance and covariance in a nested design. The genetic and environmental correlations were based on equal weight of both sire and dam components of variance and covariance.

Results and Discussion

Heritability estimates: Estimates of heritability were obtained for various traits on within a line-generation basis from paternal

and maternal half-sib and full-sib correlations. (Appendix Table 8). There were wide fluctuations among the estimates making the interpretations difficult. This may also be due to considerable confounding of line differences with genetic differences among sires and dams. The discrepancies between heritability estimates calculated from paternal and maternal half-sib correlations might in part be due to non-additive genetic effects and sire-dam interactions (King and Henderson, 1954). Actually the estimates from the paternal half-sib correlations are most reliable if sufficient number of degrees of freedom are available in the estimate of sire component and the sex-linked effects and epistatic variance are not large. On the other hand the estimates from the maternal half-sib correlations are complicated by dominance and maternal effects. The most reliable estimates under the present circumstances are those based on full-sib correlations because they are essentially an average of the estimates from sire and dam components. These estimates also correct somewhat for the variability of estimates obtained from either of the half-sib correlations and for the dominance, maternal and sex-linked effects. Furthermore, since the estimates obtained in this experiment were primarily to categorize the heritabilities as high, moderate or low, those based on full sib correlations were largely utilized for discussion. In general these estimates (Table 19) agree quite well with those reported in the literature (Table 20) which are the unweighted averages of published heritability estimates of the pertinent traits (Kinney, 1969).

Reproduction traits: The combined heritability estimate of 0.36 for age at sexual maturity is within the range of estimates reported by other investigators, and is similar to the average heritability of 0.38 in the first four generations of selection (Siegel, 1963). The variation in the estimates among the two generations may be attributed to genotype-environment interactions since feeding regimes were different. The mean heritability estimate of 0.24 for percentage HDP of normal eggs is within the range of published estimates and is in close agreement with the average estimate of 0.21 obtained during the first four generations of selection (Siegel, 1963). The estimates for percentage HDP of normal eggs and yolks were consistantly lower for the HW than the LW lines (Table 19). Van Middelkoop (1973) obtained relatively high heritability estimates of normal egg and yolk production both from the component analysis and parent-offspring regression methods.

Comparisons of heritability estimates for percentage HDP of normal eggs and yolks may explain in part the genetic-influences on the production of defective eggs. This should be more so in the HW lines which have a high frequency of defective eggs. Heritabilities of normal eggs and yolks were similar in $S_{16}(R_3)$ generation but seem to vary in $S_{17}(R_4)$ generation with the estimate for percentage HDP of yolks being higher than the corresponding estimate for normal eggs. The unweighted average heritabilities over the generations for these two traits were very similar suggesting that the genetic basis controlling total yolk production may not be different from that

Table 19. Heritability estimates based on full-sib correlations for various traits by lines, generations and combined

Trait	Line	Generation S_{16}	Generation S_{17}	Unweighted mean
Age at 1st egg	HW	.22 ± .18	.47 ± .17	.35
	LW	.22 ± .14	.55 ± .21	.39
	Combined	.20 ± .13	.52 ± .13	.36
%HDP				
Normal	HW	.03 ± .15	.21 ± .14	.12
	LW	.36 ± .17	.40 ± .17	.38
	Combined	.19 ± .10	.30 ± .11	.24
Total defective	HW	.46 ± .24	.21 ± .13	.34
	LW	.25 ± .15	.40 ± .17	.33
	Combined	.48 ± .15	.26 ± .10	.37
Ovulation defective	HW	.36 ± .22	.22 ± .14	.29
	LW	.20 ± .13	.26 ± .15	.23
	Combined	.39 ± .13	.23 ± .10	.31
Yolks	HW	.03 ± .14	.37 ± .16	.20
	LW	.35 ± .17	.47 ± .20	.41
	Combined	.17 ± .10	.41 ± .12	.29
Body weight at:				
8 weeks	HW	.84 ± .24	.31 ± .16	.58
	LW	.55 ± .18	.52 ± .19	.54
	Combined	.77 ± .19	.40 ± .12	.58
24 weeks	HW	.62 ± .20	.20 ± .15	.41
	LW	.18 ± .12	.24 ± .16	.21
	Combined	.44 ± .12	.20 ± .11	.32
Egg weight				
Initial	HW	.41 ± .19	.27 ± .15	.34
	LW	.11 ± .12	.28 ± .16	.20
	Combined	.31 ± .18	.27 ± .17	.29
35 weeks	HW	.61 ± .26	.53 ± .21	.57
	LW	.48 ± .18	.58 ± .22	.53
	Combined	.57 ± .20	.54 ± .22	.55

Table 20. Average heritability estimates of various traits reported in the literature[1]

Trait	Heritability		
	4S	4D	2(S + D)
Age at 1st egg	0.39	0.25	0.32
Rate of normal egg production to Jan 1	0.10	0.30	0.18
Body weight-- 8 weeks	0.39	0.61	0.42
Body weight-- 24 weeks	0.53	0.56	0.51
Initial (early) egg weight	0.57	0.65	0.67
Mature egg weight	0.58	0.54	0.58

[1]Taken from Kinney (1969)

controlling normal egg production. Similar conclusions were drawn by van Middelkoop (1973). The frequency of internal laying may, however, confound the picture since there was indirect evidence of considerable internal laying by HWS pullets (Trial 1 and 2 of Experiment I). Wood-Gush and Gilbert (1970) estimated that about 12% of all the yolks ovulated are lost by internal laying. Thus the picture may be different if total yolk production were measured rather than just those ova picked-up by the oviduct. The slightly higher heritability estimate for percentage HDP of yolks in the $S_{17}(R_4)$ generation may be indicative of specific genetic factors controlling the overproduction of yolks than the chicken's capacity to incorporate them into normal eggs.

Although the phenotypic distribution of defective egg production is discontinuous on the visible scale (P-scale) an underlying continuous distribution of causal factors on the invisible scale (X-scale) is expected. This continuous distribution could be the levels of gonadotrophins and/or neural thresholds to hormone action. The appearance of the trait on the P-scale depends upon its surpassing a threshold on the X-scale. Because of the quantitative inheritance but discontinuous distribution on the P-scale it is not possible to estimate genetic parameters by simple statistical methods requiring normality of distribution. Hence no attempt was made to estimate the heritability of individual defective egg-types. The logic of pooling all defective eggs is because of a common cause, namely the ovulation interval which may be continuously distributed. Additionally the estimation of genetic parameters of this composit trait may have

practical application in breeding. As mentioned earlier, estimates were made with broken eggs included (total defective) and with broken eggs excluded (ovulation defective).

Although the heritability estimates for percentage HDP of defective eggs are not precise, the consistency in the magnitude of these estimates suggest that this composit trait is moderate to highly heritable. Van Middelkoop (1973) reported relatively high realized heritabilities for two specific types of abnormal eggs namely the double yolk (DY) and extracalcified (EC) eggs. Comparison of pure lines and their reciprocal crosses also revealed higher influence of additive effects of genes controlling abnormal egg production (van Middelkoop and Kuit, 1974). The heritability estimates of total defective eggs were somewhat higher than those of ovulation defectives (Table 19) suggesting that broken eggs may mask other defective egg types when defective arthymic ovulation patterns are studied.

Body and egg weight: The mean heritability estimates of 8-week body weight were larger than the realized heritability reported by Siegel (1963). These estimates from full sib correlations include considerable amount of dominance and maternal variances as indicated by the larger estimates from maternal than paternal half-sib correlations (Appendix Table 8). This is consistent with Yao (1961) and Siegel (1962) who found that both dominance and maternal effects influence juvenile body weight. The mean heritability estimate of 0.32 for 24-week body weight is lower than the 0.46 reported by Siegel (1963) and the 0.51 average reported in the literature (Table 20).

There are very few published reports of heritability estimates of initial egg weight. Although the mean heritability of 0.29 for initial egg weight was close to 0.34 estimated by King and Henderson (1958) for early egg weight it should be remembered that there is considerable variation in age at maturity and that the age-environment interaction may influence the magnitude of this estimate. The mean heritability estimate of 0.55 for a 35-week egg weight is in close agreement with the average of 0.58 reported in the literature (Table 20).

Correlations among traits: Genetic, phenotypic and environmental correlations among various traits were obtained each generation and are presented in Tables 21, 22, and 23 respectively. With few exceptions the genetic correlations were greater than the phenotypic correlations. This further emphasizes the need of genetic and environmental correlations, since the phenotypic correlation in itself is often a deceptive statistic in a breeding program. Although the genetic correlations were in general agreement with most other estimates obtained from component analyses (Kinney, 1969) they are of limited value unless similar relationships among these traits are found over several generations.

Age at sexual maturity and egg production patterns: Although the phenotypic correlations between age at maturity and percentage HDP of normal eggs or yolks were negative in both the generations, the corresponding genetic correlations were positive in $S_{16}(R_3)$ and negative in $S_{17}(R_4)$ generations. Thus, early sexual maturity was associated with higher rate of normal eggs and total yolk production

in $S_{17}(R_4)$ generation when the feed restriction was moderate, while the effect was opposite under relatively severe feed restriction in $S_{16}(R_3)$ generation. This may provide evidence for a genotype-environment interaction. Ideta and Siegel (1966) reported the evidence of correlation between genotypes and environments in the earlier generations of selection. Although negative genetic correlations between age at maturity and rate of egg production has been reported on several occasions (King, 1961; Hale, 1961; Kinney and Shoffner, 1965; Kinney and Lowe, 1968; Merritt, 1968) positive correlations are infrequent. While these data suggest an effect of the feeding program on this correlation they do not provide information on the optimum level of energy restriction to maximize normal egg production by delayed sexual maturity. Negative environmental correlations in $S_{16}(R_3)$ generation suggest that the nongenetic factors which delay sexual maturity also reduce the percentage HDP of normal eggs and total yolks. The negative phenotypic correlations among these traits in the two generations may be largely due to negative environmental correlations.

Although the genetic correlation between percentage HDP of defective gees and age at sexual maturity was negative in both generations, the higher estimates in S_{17} generation suggest that moderate feed restriction throughout the laying period may be beneficial in reducing the incidence of ovulation defective eggs. Although the environmental correlations between age at maturity and percentage ovulation defective eggs are too small to be on any practical significance, they do indicate

Table 21. Genetic correlations[1] among reproductive, body and egg weight traits in S_{16} and S_{17} generations

No	Trait	1	2	3	4	5	6	7	8
1	Age at 1st egg		-.25	-.51	-.36	-.22	.08	.51	-.16
2	%HDP Normal	.39		.11	.88	.20	.33	-.37	-.34
3	%HDP Ovulation defective	-.20	-.26		.56	.20	.04	-.19	.24
4	%HDP Yolks	.44	.93	.28		.17	.25	-.43	-.33
5	Body weight 8 weeks	-.42	.39	.16	.36		.78	.29	.58
6	Body weight 24 weeks	-.55	.14	.17	.20	.73		.79	.43
7	Initial egg weight	.36	-.13	-.14	-.14	.24	.18		.89
8	35 week egg weight	-.04	-.07	-.12	-.08	.52	.67	.77	

The values for S_{17} generation above and S_{16} generation below diagonal.

[1] $r_{GG} = \frac{(Sxy + Dxy)}{(Sx + Dx)\ (Sy + Dy)^{1/2}}$

Table 22. Phenotypic correlations[1] among reproductive, body and egg weight traits in S_{16} and S_{17} generations

No	Trait	1	2	3	4	5	6	7	8
1	Age at 1st egg		-.13	-.13	-.14	-.25	-.16	.44	.06
2	%HDP Normal	-.11		-.31	.85	.14	.10	-.07	-.12
3	%HDP Ovulation defective	-.00	-.31		.17	.04	.07	-.03	.11
4	%HDP Yolks	-.08	.81	.04		.12	.11	-.04	-.02
5	Body weight 8 weeks	-.20	.10	-.04	.08		.47	-.04	.13
6	Body weight 24 weeks	-.39	.11	.09	.13	.40		.02	.06
7	Initial egg weight	.49	-.00	-.03	-.04	.03	.01		.51
8	35 week egg weight	.06	-.03	-.06	-.06	.18	.17	.56	

The values for S_{17} generation above and S_{16} generation below diagonal

[1] $r_{pp} = \frac{Sxy + Dxy + Exy}{(Sx + Dx + Ex)\,(Sy + Dy + Ey)^{\frac{1}{2}}}$

Table 23. Environmental correlations[1] among reproductive, body and egg weight traits in S_{16} and S_{17} generations

No	Trait	1	2	3	4	5	6	7	8
1	Age at 1st egg		-.05	.08	.04	-.28	-.26	.42	.30
2	%HDP Normal	-.23		-.46	.84	.11	.03	.05	-.01
3	%HDP Ovulation defective	.08	-.34		.03	-.02	.07	.02	.06
4	%HDP Yolks	-.19	.77	-.04		.08	.06	.16	.13
5	Body weight 8 weeks	-.09	-.11	-.32	-.10		.36	-.20	-.24
6	Body weight 24 weeks	-.34	.10	.04	.10	-.08		-.19	-.11
7	Initial egg weight	.45	.03	.03	-.01	-.21	-.05		.26
8	35 week egg weight	.12	.01	-.01	-.05	.34	-.16	.42	

The values for S_{17} generation above and S_{16} generation below diagonal

$$^{1}r_{EE} = \frac{Exy - Sxy - Dxy}{(Ex - Sx - Dx)(Ey - Sy - Dy)^{\frac{1}{2}}}$$

that the environmental forces that influence sexual maturity have no major influence on the production of defective eggs. This, reiterates the observation in Trial 1 and 2 of Experiment I that the defective egg production curves were essentially similar in S_{15} and S_{16} generation pullets, although they differed in age at maturity. The negative phenotypic correlation between age at maturity and normal eggs in S_{17} generation is consistent with the report of Fuller et al. (1969) who obtained more normal and fewer defective eggs by delaying sexual maturity following restricted feeding. Van Middelkoop and Kuit (1974) also obtained significantly more normal eggs and yolks by delaying sexual maturity due to decreasing photoperiod.

Body weight and egg production patterns: Genetic correlations between juvenile body weight and egg production patterns were consistently positive and higher than the phenotypic correlations. The positive genetic correlation between juvenile body weight and percentage HDP of normal eggs while in contrast with the consistently negative relation reported by others (Dilland et al., 1953; McClung, 1958; Merritt and Gowe, 1962; King et al., 1963; Siegel, 1963, 1970; Kinney and Shoffner, 1965; Ideta and Siegel, 1966; van Middelkoop, 1973), is consistent with some estimates. For example, Krueger et al. (1952) and Hale (1961) obtained correlation estimates of 0.13 and 0.12 respectively, between juvenile body weight and normal eggs. Jaap et al. (1962) obtained a positive genetic correlation between 8-week body weight and percentage normal eggs. The positive genetic correlations between 24-week body weight and normal egg production is consistent

with the observations of Hicks (1958) and Hogsett and Nordskog (1958) who obtained similar correlations in heavy breeds of chickens. Interestingly, however, Hicks (1958) obtained a positive genetic correlation between mature body weight and egg production when egg production was poor and a negative genetic correlation when the environment favored higher egg production. The positive genetic correlations between body weight at both ages and egg production patterns may be attributed to the nutritional environments which caused the environmental covariances to decrease resulting in positive genetic covariances. It is interesting that the phenotypic correlations between body weight and egg production patterns were rather similar in both the generations. The environmental correlations between juvenile body weight and percentage HDP of normal eggs was negative in $S_{16}(R_3)$ generation but positive in $S_{17}(R_4)$ generation indicating erratic environmental influence on the relationship between these traits.

Positive genetic correlations between growth rate and percentage HDP of defective eggs poses problems for those involved with increasing broiler weight and reducing the frequency of defective eggs. Van Middelkoop (1973) reported positive genetic correlations between 8-week body weight and the production of DY and EC eggs. The low magnitude of phenotypic and environmental correlations among these traits suggest that under restricted feeding regimes defective egg production is largely independent of growth rate. Therefore, in view of high heritability for this composit trait a genetic approach to the problem of defective eggs would be appropriate.

Egg weight and egg production patterns: Generally the high production of normal eggs and yolks was genetically associated with reduced initial and mature egg weights, whereas there was a slight tendency for environmental effects to increase both the egg production and egg weight. The negative genetic correlations between percentage HDP of normal eggs and egg weight were in agreement with Jerome et al. (1956), King (1961), Jaap et al. (1962), Kinney and Lowe (1968), Merritt (1968) and Kinney et al. (1968). The genetic correlations between egg weight and defective egg production were largely negative, and the phenotypic and environmental correlations were low.

Relationships among egg production patterns: The correlations among egg production patterns are likely to be confounded by part-whole relationships and because an increase in the incidence of some tends to decrease the incidence of others. The genetic, environmental and phenotypic correlations between the percentage HDP of normal eggs and yolks were positive and high. This was expected since they represent part-whole relationships and was consistent with the observations of van Middelkoop (1973) who reported positive genetic and phenotypic correlations between these traits. The genetic correlations between the percentage HDP of defective eggs and yolks were positive and higher than the phenotypic correlations. Although this may again be due to part-whole relationships, the correlations were smaller than those between the normal eggs and yolks, suggesting the involvement of factors other than the overproduction of yolk. Examples of such factors may be levels of gonadotrophins, the neural thresholds and

ovarian sensitivity to gonadotrophins.

Perhaps the most interesting correlations are those between normal and defective eggs. While the phenotypic and environmental correlations were negative in both the generations, the genetic correlation was negative in the $S_{16}(R_3)$ generation only. The positive genetic correlation in $S_{17}(R_4)$ generation was due to large positive sire covariance component. Van Middelkoop (1973) also reported negative genetic and phenotypic correlations between normal and DY or EC eggs. It may be argued that total defective eggs of this experiment falls between the two extremes, namely the DY and the EC eggs, and hence the relationships between normal and defective eggs in these two studies seems considtent. The negative relationship between normal and defective eggs was largely environmental as indicated by higher negative environmental correlations than either phenotypic and genetic correlations.

Age at maturity, body and egg weights: Negative genetic correlations between juvenile body weight and age at maturity were consistent with those reported by Merritt and Gowe (1962), Siegel (1963, 1970), Kinney and Shoffner (1965), Ideta and Siegel (1966), Kinney and Lowe (1968), and Bhuvanendran and Merritt (1972). However, Peeler et al. (1955) and Hale (1961) obtained negative estimates from dam components and positive values from the sire and combined estimates. The phenotypic and environmental correlations between these traits were negative, but lower than the genetic correlations and are in agreement with those reported by Peeler et al. (1955) and Hale (1961). The genetic,

environmental and phenotypic correlations between age at maturity and body weight at 24 weeks of age were generally similar in magnitude and direction to those between 8-week body weight and age at maturity. This may be due to a high genetic correlation between body weights at 8 and 24 weeks of age. The environmental correlations between body weight and age at maturity suggest that sexual maturity may be controlled by manipulating growth by environmental means.

Positive genetic correlations between juvenile and mature body weights is well documented (Wyatt, 1954; Peeler et al., 1955; Hale, 1961; Jaap et al., 1962; Siegel, 1963, 1970; Kinney and Shoffner, 1965; Ideta and Siegel, 1966; Merritt, 1968). These correlations suggest that the genes that influence juvenile growth also have considerable effect on weight at maturity and represent part-whole relationships. The genetic correlations were higher than the phenotypic and environmental correlations. Furthermore, the low environmental correlations between the body weights at two ages suggest that environmental forces that are operative on growth during broiler age are not necessarily effective at 24 weeks of age.

Early sexual maturity appears to be associated with smaller egg size, with the genetic and environmental influences being similar. Age at maturity was positively correlated with initial egg weight genetically, phenotypically and environmentally. The positive relationship between age at maturity and 35-week egg weight seems largely due to environmental effects.

Genetic correlations between body and egg weight were positive and consistently higher than the phenotypic correlations, whereas the environmental correlations were negative. Similar relationships among these traits were reported by King (1961), Merritt and Gowe (1962) and King et al. (1963). Positive genetic and phenotypic correlation among these traits are in accordance with those reported in the literature. Siegel (1963) suspected pleiotrophy as the primary cause of the correlated response of post juvenile body and egg weights when selection was practiced for juvenile body weight. The magnitude and direction of both genetic and environmental correlations support the above hypothesis of a strong genetic relationship between body and egg weights. The negative environmental correlations demonstrate the existence of non-genetic factors that contribute to increased juvenile and mature body weights but lower initial and adult egg weights.

Estimates of genetic parameters may be biased because of non-additive genetic variation and genotype-environment interactions. There was evidence of such interactions in this study. The relative importance of such effects may be indicated by the discrepancy between the actual phenotypic correlation and that expected from the genetic and environmental correlations (Jaap et al., 1962). The expected phenotypic correlations were computed by the following formula given by Falconer (1960).

$$r_{PP} = h_x h_y r_{GG} + e_x e_y r_{EE}$$

where r_{PP} was the expected phenotypic correlation between trait x and

Table 24. Genetic and environmental correlations based on sire components with actual and expected phenotypic correlations among various traits by generations

Correlations between	r_{GG}		r_{EE}		r_{PP}		$E(r_{PP})$[1]	
	S_{16}	S_{17}	S_{16}	S_{17}	S_{16}	S_{17}	S_{16}	S_{17}
Age at 1st egg and %HDP of								
Normal	.04	- .41	-.20	-.06	-.11	-.13	-.12	-.13
Defective	.33	-1.52	-.08	.10	-.00	-.13	.11	-.14
Yolks	.17	- .50	-.19	.02	-.08	-.14	-.08	-.13
8-week body weight and %HDP of								
Normal	.44	.36	-.05	.10	.10	.14	.08	.14
Defective	- .22	- .31	-.08	.04	-.04	.04	-.12	.00
Yolks	.29	- .04	-.03	.12	.08	.12	.05	.08
24-week body weight and %HDP of								
Normal	.15	.08	.10	.07	.11	.10	.10	.17
Defective	-1.17	.13	.30	.06	.09	.07	-.09	.06
Yolks	.21	- .08	.11	.12	.13	.11	.12	.06

[1] $E(r_{pp}) = h_x h_y r_{GG} + e_x e_y r_{EE}$

y with heritabilities of h^2_x and h^2_y. The e_x and e_y were the square roots of $1-h^2_x$ and $1-h^2_y$ respectively. The r_{GG} and r_{EE} were the genetic and environmental correlations between traits x and y based on sire variance-covariance components. The heritabilities were also based on sire components.

The most pertinent correlations from the view point of the central theme of this dissertation are those between the egg production patterns and body weight and age at maturity traits. The actual and expected phenotypic correlations (Table 24) were in general agreement with regard to correlations of the percentage HDP of normal eggs and yolks with age at maturity or body weight traits. Discrepancies between actual and expected phenotypic correlations seems evident for correlations between percentage HDP of defective eggs with body weight and age at maturity, suggesting that non-additive genetic effects may contribut to environmental correlations among these traits.

Summary

Genetic parameters estimated from intraline genetic analysis based on full sib correlations were in general agreement with most of the published estimates. Phenotypic data suggested that common genetic factors control both the production of normal eggs and total number of yolks. The heritability estimates of the total defective eggs were of a moderate to high magnitude. The genetic relationship between age at maturity and normal egg production varied under two nutritional environments suggesting genotype-environment interactions.

The environmental forces that delay sexual maturity had no significant effect on the production of defective eggs. This plus the magnitude of the heritabilities suggest that a genetic approach to the problem of defective eggs is appropriate. The positive association between the production of normal eggs and total yolks was largely genetic, whereas the negative relationship between normal and defective eggs was largely environmental. The small phenotypic and environmental correlations between body weight and defective eggs suggested that under restricted feeding regimes defective egg production was largely independent of growth rate. Evidence suggesting the contribution of non-additive genetic effects to environmental correlations among defective eggs and growth rate was presented.

EXPERIMENT III. EFFECT OF SEX-LINKED RECESSIVE DWARF GENE, *dw* ON GROWTH AND REPRODUCTION INDIVERSE GENETIC BACKGROUNDS

Although considerable efforts are being made to exploit the genetic properties of sex-linked dwarfism in commercial poultry production, no definite conclusions have emerged on its usefulness in diverse stocks. This may be due to the nature of the various interactions between the *dw* gene and the specific genetic backgrounds of the testing populations. This experiment was designed to evaluate the effect of a single major gene, *dw* on growth and reproduction in lines selected bidirectionally for juvenile growth from a common gene pool.

Materials and Methods

Breeding scheme: Heterozygous (*Dwdw*) males from a commercial broiler line were mated to a sample of S_{13} generation females from the high (HWS) and low (LWS) lines selected for 8-week body weight (Siegel, 1962, 1970). The males from such matings were progeny tested to evaluate their genotype at dwarf locus. *Dwdw* males were backcrossed to the females of the respective line in S_{14} generation to produce B_1 generation progeny. Similar backcrosses were made to the S_{15}, S_{16} and S_{17} generations to produce the B_2, B_3 and B_4 generations. This repeated backcrossing provided normal and dwarf progeny with 50, 75, 87.5, 93.8 and 96.8% of the inheritence of the

selected line in the F_1 through the B_4 generations, respectively. For all practical purposes the B_3 and B_4 generations provided data to evaluate the effect of this gene in divergent genetic backgrounds influencing juvenile growth.

Management and traits measured: Chicks were obtained by two hatches in March of each year. Feeding and management procedures from hatching to caging were uniform through all the generations and were described in Experiment I. The birds were caged at 119 days of age and were fed rations with 16% protein and 1260 Kcal/lb of M.E. From the B_2 generation feeding was restricted after caging to approximately 60% of ad libitum in normal pullets of both lines and dwarf pullets of HW line. The LW dwarfs were provided ad libitum feeding throughout. A constant light regime of 14 hours light and 10 hours darkness was maintained during the laying period.

The traits measured in each of the backcross generations are presented in Table 25. Egg production was recorded daily on individual bird basis from the date of first egg to January 1. Eggs were classified as normal, double yolk, broken and other types of defective eggs. Initial and 35-week egg weights were obtained in the manner outlined in Experiment I. Shell thickness of these eggs was also measured at these times. Data on egg quality traits were obtained in the B_3 generation only following the usual procedures.

Analysis: The body size and reproductive traits were analyzed within dam lines by analysis of variance using a nested design. The

Table 25. Growth and reproductive traits measured in each of the backcross generations (marked x)

Trait	Generations			
	B_1	B_2	B_3	B_4
Body weight (g)				
4 weeks	x	x	x	x
8 weeks	x	x	x	x
12 weeks	x	x	x	x
16 weeks	x	x	x	x
20 weeks	x	x	x	x
24 weeks	x	x	x	x
32 weeks	x	x	x	x
40 weeks	x	x	x	x
Shank length (mm)				
12 weeks	x	x	x	x
16 weeks	x	x	x	x
40 weeks	x	x	x	x
Age at 1st egg	x	x	x	x
%HDP				
Normal	x	x	x	x
Defective		x	x	x
Yolks		x	x	x
Egg weight (g)				
Initial		x	x	
35 weeks	x	x	x	
Shell thickness (10^{-4} mm)				
Initial		x	x	
35 weeks		x	x	
Specific gravity			x	x
Haugh units			x	

statistical model was:

$$X_{ijklm} = \mu + H_i + S_{ij} + D_{ijk} + G_{ijkl} + E_{ijklm}$$

where X_{ijklm} was the measurement on the mth offspring of the lth genotype from Kth dam mated to jth sire in the ith hatch. The percentages of HDP of eggs were transformed to arc sin $\sqrt{\%}$ prior to analysis.

Results and Discussion

Mortality: The frequency of mortality in the four backcross generations is presented by lines and genotypes in Table 26. Mortality was independent of line and genotype in three of the four generations. The higher incidence of mortality among normal pullets in B_2 generation may be due to their lower resistence to Marek's Disease (MD), since the birds were not vaccinated against this disease in the first two backcross generations. This implies that the dw gene confers resistence to MD, an observation similar to Meurier (1971). Comparisons of dwarfs from the two lines, however, also suggest that the dw gene in itself may not confer any special resistence to MD, but that its action depends on the background genotype. Although the dwarfs were more resistent than the normal pullets significant differences in mortality were evident in dwarfs of divergent strains (Meurier, 1971). Hence the negative correlation between growth rate and resistence to MD (Han and Smyth, 1972; Friers et al., 1972) may not hold when the growth rate was below a threshold value. This, and the severe energy restriction during the first 10 weeks after caging in B_2 generation might have

Table 26. Incidence of mortality [1,2] among normal (Dw) and dwarf (dw) pullets from the HW and LW lines from caging to 300 days of age in the B_1 through B_4 generations

Genotype	B_1		B_2		B_3		B_4	
	No.	% Mortality	No.	% Mortality	No.	% Mortality	No.	% Mortality
HW Dw	37	13.5[a]	36	30.6[b]	52	9.6[a]	53	17.0[a]
DW dw	12	8.3[a]	21	0 a	50	8.0[a]	49	6.1[a]
LW Dw	44	6.8[a]	44	22.7[b]	50	6.0[a]	42	7.1[a]
LW dw	46	10.9[a]	39	18.0[a]	45	4.4[a]	28	0.0[a]

[1]The values with the same superscript in each generation are not significantly different ($P \leq .05$).

[2]Chi-square test of independence was done on the actual frequencies.

contributed to differential mortality among the genotypes. It should be pointed out that Chambers et al. (1974) did not observe significant differences in laying house mortality between dwarf and normal pullets. Perhaps the inconsistencies in results are due to genotype-environment interactions.

Body size traits: Means and standard errors for various traits studied in B_1, B_2, B_3 and B_4 generations are presented by lines and genotypes in Appendix Tables 9, 10, 11 and 12, respectively. To minimize the effects of mortality, data were analyzed for those pullets surviving to the end of the experiment. Comparisons between dwarfs and their normal sibs were made by expressing the means for dwarfs as a percentage of normal pullets. Such comparisons for the F_1 through B_4 generations are summarized in Table 27.

There was a decrease in absolute body weights as the amount of specific selected inheritence increased through the backcrossing. (Appendix Tables 9 - 12). This was expected because the dw gene was introduced into these lines by Dwdw sires from a commercial broiler strain that had heavy broiler weights.

The relative depressive effect of the dw gene on body weight was cumulative with age to 12 weeks of age, after which it remained relatively constant (Table 27). An age effect on the dimorphism of morphological traits in dwarf and normal pullets is well documented (Hutt, 1959; Mohammadian, 1969; Bernier and Arscott, 1972; Polkinhorne and Lowe, 1973). Although this pattern was similar in both lines in all backcross generations, the effect was about 10% more in LW than in the HW line

after 12 weeks of age. A differential effect of dw gene on growth rate in fast and slow growing strains of chickens was reported in several investigations (Merat, 1969; Mohammadian and Jaap, 1972; French and Nordskog, 1973). The results obtained here show that this differential effect was not present in the F_1 generation, appeared to a degree in the B_1 and the B_2 generations and stabilized in the subsequent generations.

It is rather surprising that the dwarf effect on the body weight at 4 and 8 weeks of age was similar in both lines in all backcross generations, since there is a suggestive evidence that depressive effect of the dw gene on growth is more severe in the genetic background of slow growth. Leporé et al. (1963) showed that the high and low weight lines were characterized by divergent anabolic environments from the embryonic stage, with the former being more efficient in the utilization of basic amino acids. Although the dwarf and normal chicks had similar weights at hatching (Hutt, 1959; French, 1971), the former had larger yolk sacs and less body tissue (Guillaume, 1969) than the lattet due to a slower absorption of yolk (Merat and Guillaume, 1969). This suggests that the action of dw gene not only depends upon the specific genetic background, but also the physiological age of the bird.

A more meaningful comparison between Dw and dw alleles in divergent genetic backgrounds can be made from the data of B_3 and B_4 generations where the specific background genotype approximated 95%. The average depressive effect due to dw gene on body weight and shank length in the two lines at various ages is shwon in Figure 4. The effect of dw

on body weight was similar in both lines to eight weeks of age. Thereafter, the effect of the dw gene in the LW line continued its sharp rise to 12 weeks of age and then gradually plateaued between 20 and 24 weeks of age. In contrast, in the HW line, the dwarfing effect was maximum at 16 weeks of age and then tended to decline. Thus the depressive effect on body weight which was 6% more in LW than HW line at 16 weeks of age, progressively increased to 32 weeks of age when this difference between lines was about 20%. There is an indication to suggest that depressive effect of the dw gene on growth rate has peaked before the onset of sexual maturity.

The dw gene reduced the shank length in the HW line by about 25% in F_1 and also subsequent generations. This was consistent with observations in a fast growing broiler line by Mohammadian and Jaap (1972) who observed no change in the effect on shank length after the first backcross generation. In the LW line, the effect of the dw gene was similar to that of HW line in F_1 and B_1 generations. Thereafter it tended to increase and by the B_4 generation the depressive effect of dw gene on shank length was about 6% more in the LW than in the HW line (Table 27, Figure 4). This was consistent with the observations of French and Nordskog (1973) who reported that the depressive effect of the dw gene on shank length was 5% more in their slow than fast growing Leghorn line. Comparisons of relative effects on dwarfism on body growth and shank length (Figure 4) indicated that the depressive effect on protein anabolism was about 6% more than on skeletal growth in the HW line and 16% in the LW line. Although the physiological

Table 27. Comparisons[1] between dwarf and normal pullets for growth and reproduction traits in F_1, B_1, B_2, B_3 and B_4 generations

Trait	HW					LW				
	F_1	B_1	B_2	B_3	B_4	F_1	B_1	B_2	B_3	B_4
Body weight										
4 weeks	80	91	85	88	87	89	86	65	88	89
8 weeks	80	82	77	79	76	79	77	78	75	76
12 weeks	--	72	68	68	71	--	65	64	63	64
16 weeks	67	70	68	64	69	67	65	61	59	65
20 weeks	67	71	70	72	70	66	63	60	55	59
24 weeks	--	75	70	73	70	--	61	55	55	55
32 weeks	67	76	67	74	74	65	65	53	50	57
40 weeks	70	75	68	71	70	65	67	51	53	54
Shank length										
12 weeks	--	76	78	77	76	--	75	73	73	74
16 weeks	74	74	76	76	77	74	75	68	69	71
40 weeks	76	75	78	78	79	75	75	68	71	72
Age at 1st egg	120	110	102	97	97	115	102	106	102	104
Percentage HDP										
Normal	153	127	106	107	108	103	80	57	49	47
Defective	45	--	21	16	16	19	--	60	30	94
Yolks	126	--	93	96	95	97	--	57	49	50
Egg weight										
Initial	104	--	96	89	--	96	--	89	90	--
35 weeks	90	99	95	95	--	88	90	88	85	--
Shell thickness										
Initial	--	--	99	93	--	--	--	89	88	--
35 weeks	--	--	102	103	--	--	--	99	89	--

[1](Dwarf/Normal) x 100

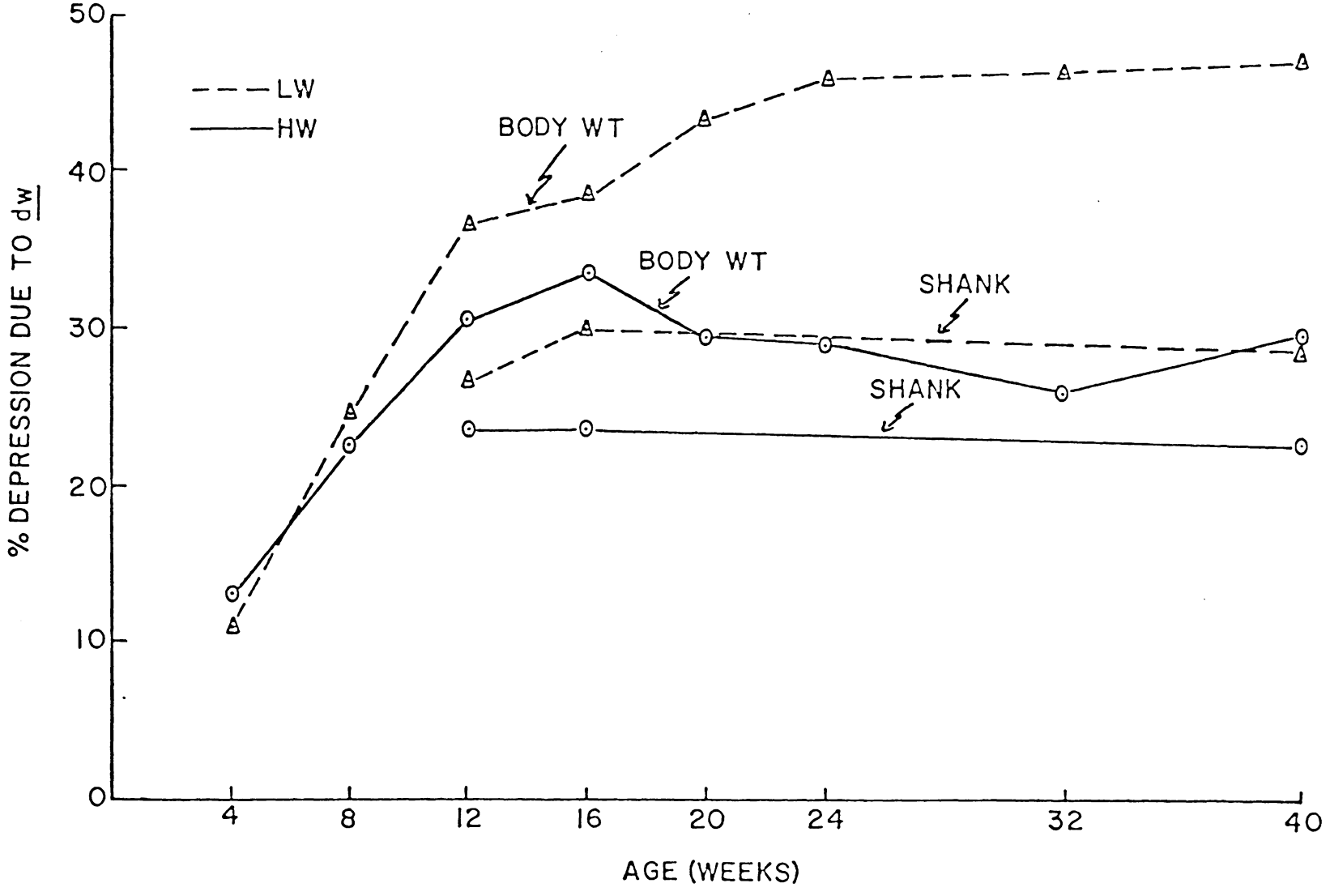


Figure 4. Percentage depression due to dw gene on body size traits at various ages, by lines. (Average of B_3 and B_4 generations).

mechanisms of gene action are not known, pituitary growth hormone is suspected to play part in dwarfism (van Tienhoven et al., 1966).

The relative contribution of hatch, sire, dam and genotype to the total variation in each trait is summarized for the B_2, B_3 and B_4 generations in Tables 28, 29 and 30, respectively. In B_1 generation all dam families were not represented by both dwarf and normal progeny and hence analysis of variance was not performed. Genotype was the only main effect consistently contributing to the total variation in body size traits in all the generations.

Reproductive traits: Means and standard errors for various reproductive traits in the B_1, B_2, B_3 and B_4 generations are presented by lines and genotypes in Appendix Tables 9 to 12, and comparisons among dwarf and normal pullets are summarized in Table 27.

Age at first egg: Dwarfism delayed sexual maturity in both lines in the F_1, B_1 and B_2 generations with the effect being slightly higher in the HW than in the LW line (Table 27). As the inheritence for rapid growth increased through successive backcrossing, the depressive effect of the dw gene was reduced. On the other hand, the dwarfing effect on sexual maturity was rather consistent after the F_1 generation in a genetic background of the slower growth (Figure 5). Interestingly, the LW dwarfs that did not lay their first egg were those that weighed less than 1000 g at this age. The true effect of dw gene could, however, be evaluated from the B_3 and B_4 generation data when the background genotype comprised of about 95% of the selected line. In these generations HW dwarfs matured earlier than their normal sisters, but this

Table 28. Percentage of total variation for various body size and reproductive traits that were contributed by hatch, sire, dam, and genotype in the B_2 generation

Trait	HW				LW			
	Hatch	Sire	Dam	Genotype	Hatch	Sire	Dam	Genotype
Body weight								
4 weeks	0	5	0	58	59	0	1	19
8 weeks	0	10	0	78**	0	0	0	59
12 weeks	0	1	0	93**	3	10	0	76**
16 weeks	0	0	0	95**	4	4	0	83**
20 weeks	0	0	0	93**	0	4	0	89**
24 weeks	0	0	0	92**	1	4	0	89**
32 weeks	0	1	0	92**	0	5	0	90**
40 weeks	0	2	0	91**	0	1	0	91**
Shank length								
12 weeks	0	0	0	98**	0	4	0	93**
16 weeks	0	0	0	99**	3	1	0	94**
40 weeks	0	0	0	98**	1	2	0	95**
Age at 1st egg	0	12	26	0	0	3	0	13
Percentage HDP								
Normal	0	0	32	0	0	1	0	84**
Defective	0	15	0	58†	14	0	0	28
Yolks	3	0	19	0	0	2	0	83**
Egg weight								
Initial	10	4	8	25	0	18	31	0
35 weeks	2	0	0	60†	0	26	0	55*
Shell thickness								
Initial	47	0	0	18	1	0	19	8
35 weeks	27	0	15	0	0	0	45	0

**P $\leq$ .01 *P $\leq$ .05 † P $\leq$ .10

Table 29. Percentage of total variation for various body size and reproductive traits that were contributed by hatch, sire, dame and genotype in B_3 generation

Trait	HW				LW			
	Hatch	Sire	Dam	Genotype	Hatch	Sire	Dam	Genotype
Body weight								
4 weeks	11	0	0	45	3	3	0	62
8 weeks	0	7	0	83**	0	4	0	70*
12 weeks	0	4	0	90**	1	5	0	83**
16 weeks	4	4	0	76**	4	6	0	80**
20 weeks	0	8	0	80**	0	8	0	86**
24 weeks	0	2	0	85**	0	5	0	89**
32 weeks	0	3	0	85**	0	5	0	89**
40 weeks	2	3	0	88**	0	5	0	88**
Shank length								
12 weeks	0	6	0	91**	0	5	0	92**
16 weeks	0	6	0	91**	0	5	0	93**
40 weeks	0	6	0	92**	1	4	0	64
Age at 1st egg	2	22	14	2	0	19	0	0
Percentage HDP								
Normal	2	13	0	24	0	6	0	85**
Defective	0	9	0	56	2	9	0	0
Yolks	3	9	0	38	0	6	0	86**
Egg weight								
Initial	0	27	0	26	0	11	0	48
35 weeks	0	9	0	54	4	0	0	57†
Shell thickness								
Initial	0	4	0	10	0	6	0	69**
35 weeks	0	5	9	23	0	0	29	0
Specific gravity	0	5	0	19	0	2	0	23
Haugh units	0	38	7	1	0	20	40	11

** $P \leq .01$ *$P \leq .05$ † $P \leq .10$

Table 30. Percentage of total variation for various body size and reproductive traits that were contributed by hatch, sire, dam and genotype in B_4 generation

Trait	HW				LW			
	Hatch	Sire	Dam	Genotype	Hatch	Sire	Dam	Genotype
Body weight								
4 weeks	12	6	0	50	0	9	0	26
8 weeks	3	1	0	90**	0	14	0	65**
12 weeks	7	0	0	87**	9	20	0	58**
16 weeks	0	0	0	95**	2	22	0	48
20 weeks	0	0	0	91**	4	25	0	61**
24 weeks	0	0	0	93**	0	26**	0	69**
32 weeks	0	0	0	92**	0	25	0	43
40 weeks	0	0	0	90**	0	30*	0	60**
Shank length								
12 weeks	1	1	0	95**	0	18	0	76**
16 weeks	0	3	0	95**	0	24	0	72**
40 weeks	0	1	0	98**	0	29	0	69**
Age at 1st egg	0	0	48	0	0	18	0	57*
Percentage HDP								
Normal	0	0	2	7	0	26	0	55*
Defective	0	0	0	69*	0	2	0	10
Yolks	0	5	0	2	0	29	0	56**
Specific gravity	0	3	0	1	0	0	65*	0

**P $\leq$ .01 *P $\leq$ .05

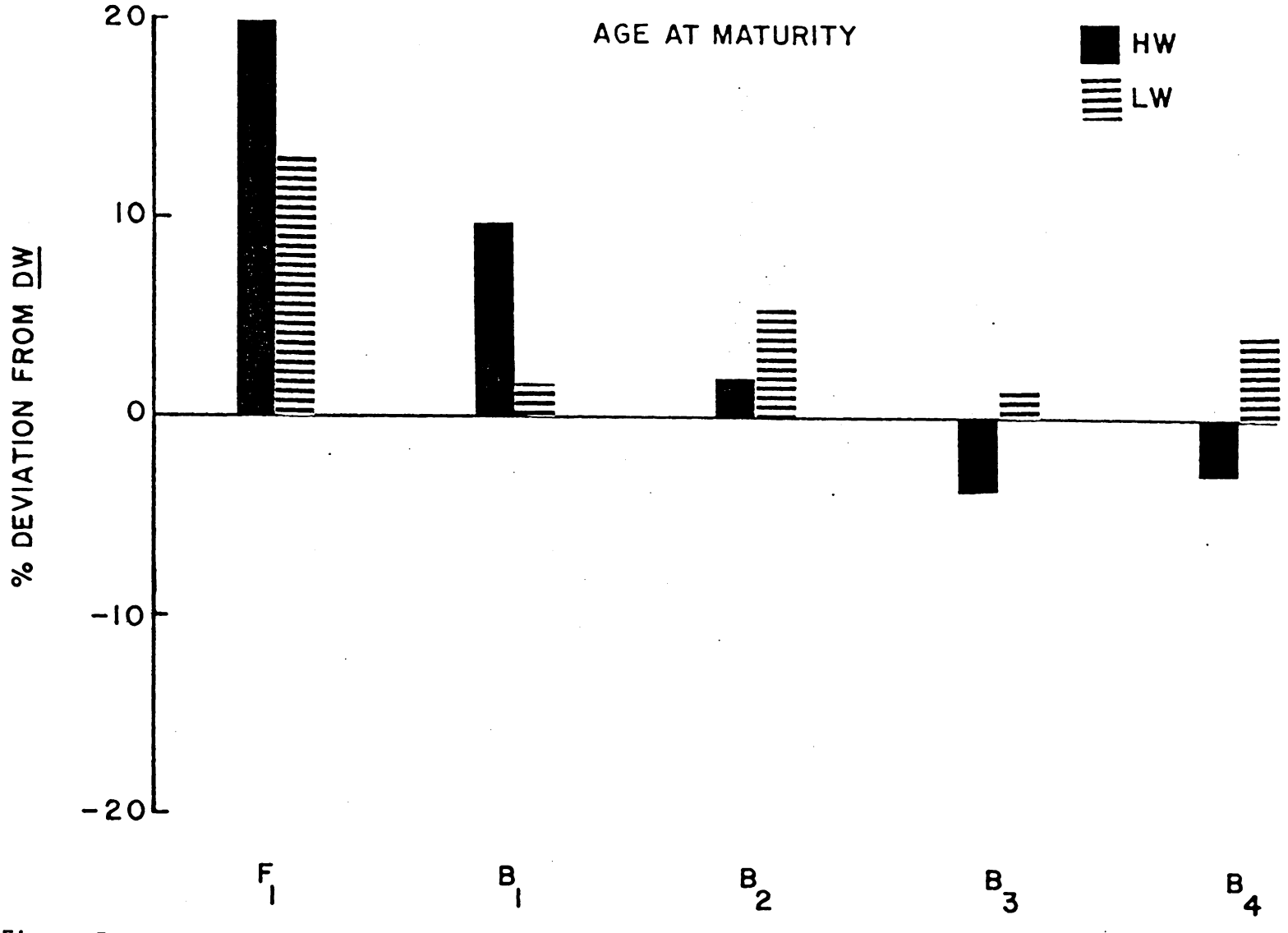


Figure 5. Percentage devation of dwarfs from normal pullets for age at maturity, by lines and generations.

difference was not statistically significant. This observation is in contrast with the rather consistent reports of delayed sexual maturity in dwarfs obtained in other studies (Hutt, 1959; Selvarajah, 1971; French, 1971; Merat, 1971; Richard and Cochez, 1971; Bernier and Arscott; 1972; van Middelkoop, 1973; Polkinghorne and Lowe, 1973). Delayed sexual maturity of dwarfs has been attributed to their lower social ranks when reared in intermingled flocks with normal pullets (Merat, 1971; Udale, 1972). This is because age at sexual maturity is negatively correlated with social rank (Tindell and Craig, 1959). Such confounding of social stratification was not present in this experiment because dw and Dw pullets were maintained in individual cages after 19 weeks of age. Thus, without the effects of social competition, the effect of dw reported by others was not observed after the B_2 generation in the HW line.

In the HW line, none of the main effects made any significant contribution to the total variation in age at sexual maturity in either of the generations, while in the LW line the only significant main effect was the genotype in B_4 generation (Tables 28 to 30). The significant sire effects that were found in the LW line in F_1 generation (Udale, 1972) was not observed in the subsequent backcross generations.

Normal eggs and yolks: Egg production data were analyzed on percentage hen-day basis to minimize effects of the differences in precocity and length of laying period among the genotypes. The percentage HDP of normal eggs was consistently higher for HW dwarf than for normal pullets in all generations (Table 27). The normal egg production

of dwarfs was 53% higher than their normal sisters in the F_1 generation, but as the genome of the selected line increased in subsequent generations the rate of normal egg production decreased and stabilized after B_2 generation (Figure 6). This may be due to population differences and epistatic interactions of a varied nature between the dw gene and the background genotype. The genotype effect, however, was not significant when analyzed within hatch, sire, and dam basis (Tables 28 to 30) because of the higher variation among individuals within a genotype. When analyzed by t-test, however, the dwarfs seem to have produced significantly more normal eggs in F_1, B_1 and B_4 generations. This is consistent with the observations of Ricard and Cochez (1972) who reported significantly higher normal egg production in dwarfs than in the normal pullets (65.8% vs 60.4%). Interestingly, the mature body weights in their study and that of ours are very similar and suggest that mature body weight of 2.2 to 2.5 kg in broiler dwarf pullets may be optimum for efficient egg production. Prod'homme and Merat (1969) also reported that dw gene does not reduce the rate of egg production in broiler dams which have attained a mature body weight of about 2.0 kg. The dw gene reduced the rate of yolk formation in the ovary but did not reduce normal egg production in pullets weighing 2.6 kg at 36 weeks of age (Jaap and Mohammadian, 1969). This, however, cannot be generalized for all populations.

In the LW line, the dwarfs were equivalent to their normal sisters in F_1 generation, but with successive backcrosses, the capacity of the dwarfs for yolk synthesis and normal egg production were reduced

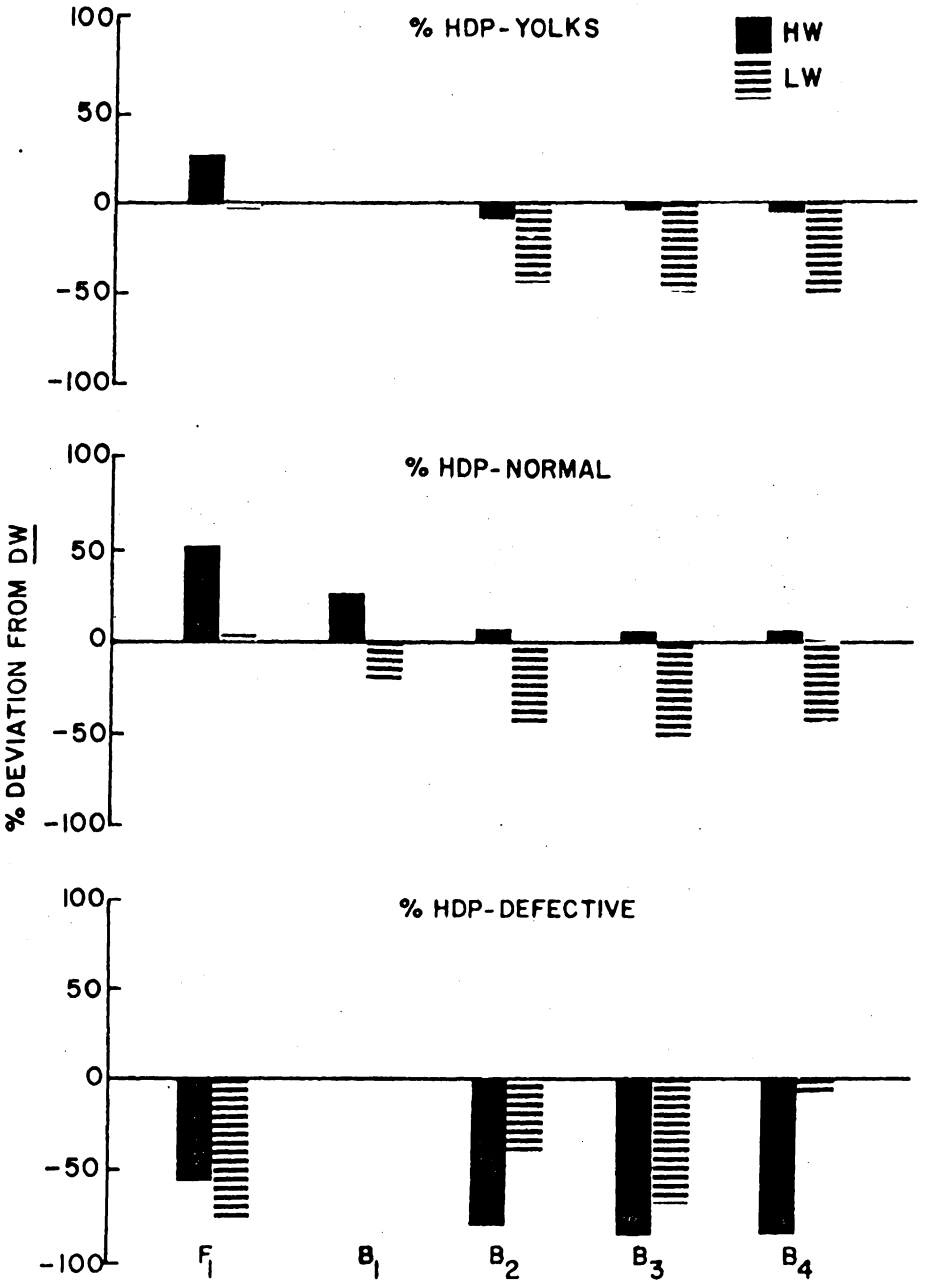


Figure 6. Percentage deviation of dwarfs from normal pullets for %HDP of yolks, normal and defective eggs, by lines and generations.

so that by the B_3 generation, they were producing 50% as many yolks and normal eggs as their normal sisters (Figure 6). This differential response reflects the nature of dwarf gene effect in divergent genetic backgrounds. The observation that the dw gene is beneficial in stocks with excess yolk synthesis and arhythmic ovulation rates (van Middelkoop, 1973) is consistent with the data reported here. On the other hand, this gene may have an opposite effect in stocks with optimum yolk synthesis and coordination between ovarian and oviductal functions. Van Middelkoop (1973) reported a similar response due to dw gene in his superovulating DY line and N line selected for normal egg production.

Genotype contributed significantly to the total variation of % HDP of normal eggs and yolks in all the backcross generations in the LW line (Tables 28 to 30). Comparisons of the means for these traits (Appendix Tables 9 to 12) showed significant differences among the genotypes, with the Dw pullets producing significantly more normal eggs and yolks than dw pullets in all generations.

The most interesting observation was the relationship between the rate of normal egg and yolk production in Dw and dw pullets within each line. In the HW line, the difference among Dw and dw pullets for normal egg production was primarily because there were more Dw pullets that were poor layers (Figure 7). Similarly, the difference among these genotypes for yolk production was due to more Dw pullets producing a greater number of yolks (Figure 8). These frequency distributions for Dw pullets shifter the mean to the left for normal eggs and to the right for yolk production with reference to the means

for the dw pullets. The apparent difference in the frequency distributions for normal eggs and yolks of nondwarf pullets is associated with the significantly higher frequency of defective eggs. Contrary to this, no significant variation seems to exist in the frequency distributions of normal eggs and yolks in dwarf pullets. This was because of their low frequency of defective eggs.

The patterns for Dw and dw genotypes of the LW line were similar. These frequency distributions for various genotypes drawn on the same scale facilitate comparisons. The most pertinent comparisons may be between the HW dwarf and LW normal genotypes. Since the body weights of these genotypes are comparable, they represent the proper material to compare the effect on reproductive parameters of the major dw gene and polygenes that both influencing growth rate. This idea was evaluated by French and Nordskog (1973) who compared normal and dwarf pullets from the large and small Leghorn lines. They reported no significant beneficial effect of dw gene on any of the reproductive parameters or feed efficiency. The lower egg production of the LW normal pullets was mainly due to some birds producing very few eggs which is consistent with that found here. A body weight threshold seems to be a factor for the initiation and maintenance of normal egg production. It is interesting to note that none of the LW dwarfs reached the mean normal egg production of their normal sisters.

Defective eggs: The dwarf pullets, in the HW line, produced significantly fewer defective eggs than their normal sibs in all generations (Figure 6). Genotype effect approached significance in

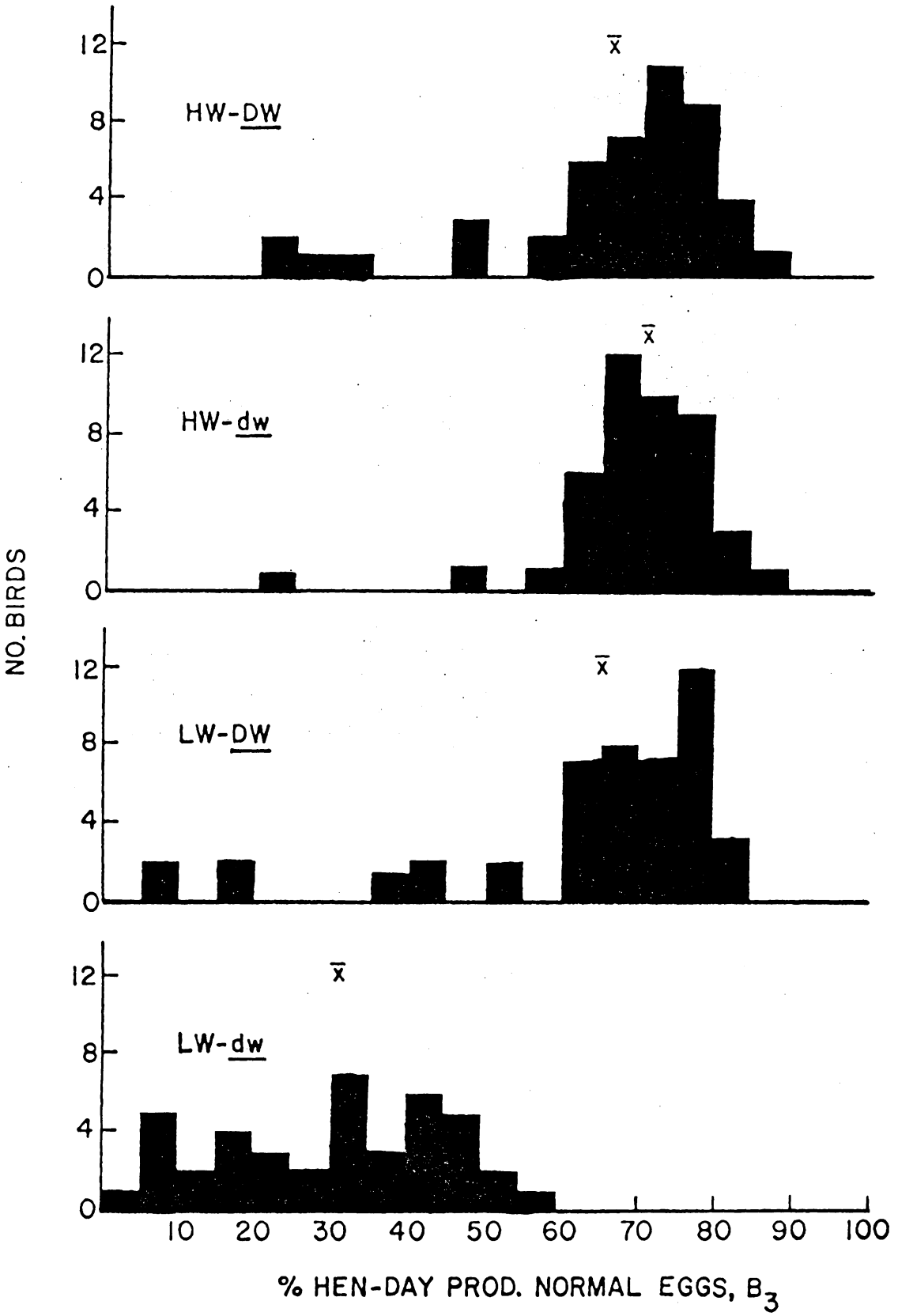


Figure 7. Frequency distribution of %HDP of normal eggs to 300 days of age by lines and genotypes (B_3 generation).

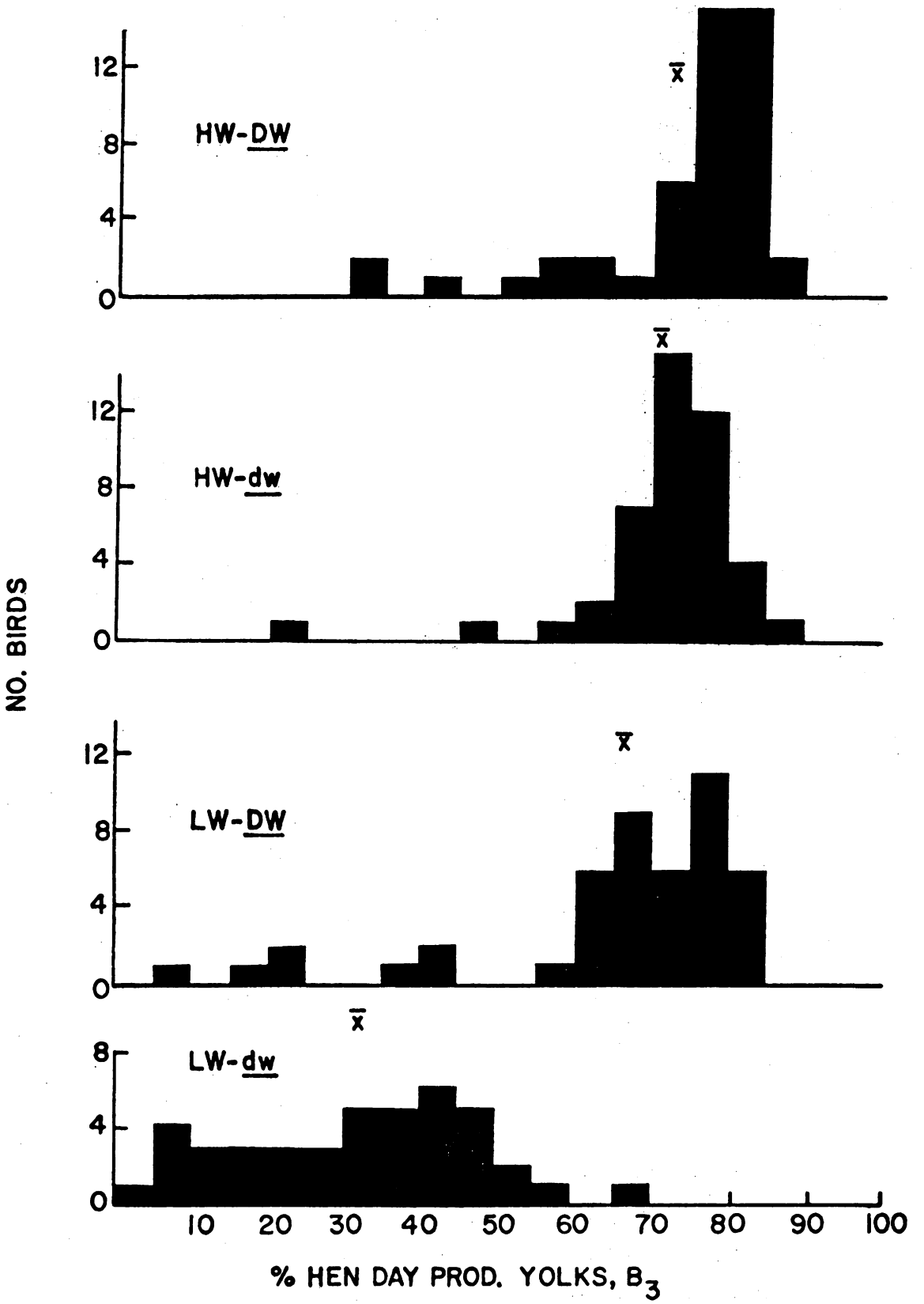


Figure 8. Frequency distribution of %HDP of yolks to 300 days of age by lines and genotypes (B_3 generation).

the B_2 generation (Table 28), were not significant in B_3 generation (Table 29), and were significant in the B_4 generation (Table 30). This is consistent with the observations of Jaap (1969), Jaap and Mohammadian (1969) and Mohammadian (1971) who reported that dw alleviates the defective egg syndrome in broiler pullets presumably by a depressive effect on lipoprotein anabolism, which results in fewer follicles under rapid development. This control of follicular development may be partially responsible for rhythmic ovulation patterns and the production of normal eggs. Although there was some indication to show that the dw gene increases somewhat the frequency of defective eggs in the LW line, the main effect of genotype was not significant in any of the backcross generations. Extremely low frequency of defective eggs in this line precludes any meaningful conclusions to be reached at this time.

Separate analyses were made in the B_2, B_3 and B_4 generations to test for differences among genotypes for various types of defective eggs (Table 31). Because of the low frequency and abnormal distribution, genotype differences were tested by Chi-square. The pattern of defective egg production was similar in the three backcross generations. The HW normal pullets produced significantly more DY and B eggs compared to their dwarf sisters in all the generations. The low frequency of DY eggs in HW dwarfs is consistent with the hypothesis that the dw gene depresses yolk synthesis and thus the simultaneous development of two or more follicles (Jaap and Mohammadian, 1969). Difference in the frequencies of broken eggs among the normal and dwarf pullets of

Table 31. Means and standard errors[1] for various types of defective eggs by lines and genotypes in the B_2, B_3 and B_4 generation

Trait	Genotype	Generations B_2	B_3	B_4
%HDP of eggs				
Double-yolk	HW-Dw	1.72 ± .58^b	.90 ± .10^b	1.20 ± .30^b
	dw	.07 ± .07^a	.20 ± .10^a	.20 ± .10^a
	LW-Dw	.40 ± .10^a	.10 ± .10^a	.10 ± .10^a
	dw	0	0	0
Broken				
	HW-Dw	3.51 ± .76^c	1.20 ± .20^b	2.40 ± .40^b
	dw	.78 ± .31^b	.30 ± .10^a	.60 ± .10^a
	LW-Dw	.10 ± .10^a	.10 ± .10^a	.30 ± .10^a
	dw	.10 ± .10^a	.20 ± .10^a	.40 ± .20^a
Ovulatory defective				
	HW-Dw	7.40 ± 1.70^b	5.50 ± 1.10^b	7.70 ± 1.10^b
	dw	1.50 ± .50^a	.90 ± .30^a	1.00 ± .20^a
	LW-Dw	.90 ± .20^a	1.60 ± .40^a	3.20 ± 1.1 ^{b}
	dw	.50 ± .10^a	.40 ± .10^a	2.90 ± 1.1 ^{b}
Total defective				
	HW-Dw	10.90 ± 2.10^c	8.60 ± 1.60^b	10.1 ± 1.1 ^{c}
	dw	2.30 ± .70^b	1.20 ± .30^a	1.6 ± .3 ^{a}
	LW-Dw	1.00 ± .30^a	1.70 ± .40^a	3.5 ± 1.1 ^{b}
	dw	.60 ± .20^a	.60 ± .20^a	3.3 ± 1.0 ^{b}

[1]Means within a line and generation with the same superscript are not significantly different ($P \leq 0.05$).

HW line may better be resolved by understanding the relationship with body size and reproductive traits.

The frequencies of pullets that produced various types of defective eggs are presented in Table 32 for each genotype. Differences among the genotypes within a line were tested by Chi-square. It is clear that a significantly greater number of HW normal pullets produced various types of defective eggs than the HW dwarfs. No significant differences were observed in the relative frequencies of LW normal and LW dwarf pullets producing various defective egg-types. Thus, it is evident that high frequencies of these defective egg types in HW normal pullets were not due to a few pullets producing more defective eggs, but because of a larger number of pullets producing these defective eggs.

Correlations: Because of the low frequency and abnormal distribution of broken and ovulatory defective eggs, Spearman's rank correlations were calculated between these traits and various body size and reproductive traits (Table 33).

The correlations between percentage HDP of broken eggs and other traits that were more consistently significant for the HW normal pullets than for other genotypes. For the HW normal pullets broken eggs were positively correlated with 24-week body weight and ovulatory defective eggs and negatively with % HDP of normal eggs and shell thickness of eggs laid at 35 weeks of age. These correlations reflect the importance of genetic factors, egg shell characteristics and effective mass of cage floor in the incidence of broken eggs. Negative

Table 32. Frequency of pullets by genotypes that produced various types of defective eggs in B_2, B_3 and B_4 generations

Genotype	N	DY	B	O-def.	T-def.
			1973 (B_2)		
HW-$\underline{Dw}$	25	13	21	24	22
HW-$\underline{dw}$	21	1	7	13	10
($\underline{HW}$-$\underline{Dw}$)-($\overline{HW}$-$\underline{dw}$)		12*	14**	11**	12*
LW-$\underline{Dw}$	34	8	3	15	15
LW-$\underline{dw}$	28	0	2	11	10
($\underline{LW}$-$\underline{Dw}$)-($\overline{LW}$-$\underline{dw}$)		8*	1	4	5
			1974 (B_3)		
HW-$\underline{Dw}$	47	27	30	43	43
HW-$\underline{dw}$	44	8	16	26	20
($\underline{HW}$-$\underline{Dw}$)-($\overline{HW}$-$\underline{dw}$)		19*	14**	17*	23*
LW-$\underline{Dw}$	46	5	5	27	25
LW-$\underline{dw}$	42	0	8	17	12
($\underline{LW}$-$\underline{Dw}$)-($\overline{LW}$-$\underline{dw}$)		5	3	10	13*
			1975 (B_4)		
HW-$\underline{Dw}$	42	21	34	41	37
HW-$\underline{dw}$	47	7	19	33	25
($\underline{HW}$-$\underline{Dw}$)-($\overline{HW}$-$\underline{dw}$)		14**	15**	8**	12**
LW-$\underline{Dw}$	37	4	7	22	17
LW-$\underline{dw}$	38	0	6	20	17
($\underline{LW}$-$\underline{Dw}$)-($\overline{LW}$-$\underline{dw}$)		4	1	2	0

**P $\leq$ .01

*P $\leq$ .05

Table 33. Spearman's Rank Correlation coefficients between percentage HDP of broken eggs and ovulatory defective eggs with various other traits in B_2 and B_3 generations

Trait	Genotype	r between %HDP broken and B_2	r between %HDP broken and B_3	r between %HDP O-defective and B_2	r between %HDP O-defective and B_3
Body weight at 24 weeks	HW-Dw	0.28	0.31*	0.17	0.21
	dw	0.26	0.33*	0.13	0.36*
	LW-Dw	0.05	0.07	0.21	0.20
	dw	-0.18	-0.02	-0.35†	-0.26†
Shank length at 40 weeks	HW-Dw	0.14	0.18	0.42*	0.24
	dw	0.24	0.21	0.51**	0.38**
	LW-Dw	0.00	0.23	0.31†	0.35*
	dw	-0.02	0.10	-0.30	-0.11
%HDP of eggs Normal	HW-Dw	-0.57**	-0.38**	-0.65**	-0.66**
	dw	-0.10	-0.24	-0.18	-0.28†
	LW-Dw	-0.06	-0.20	-0.01	-0.11
	dw	-0.26	-0.16	0.06	-0.41**
Ovulatory defective	HW-Dw	0.43*	0.35*	-	-
	dw	0.40†	0.22	-	-
	LW-Dw	0.40*	0.08	-	-
	dw	0.11	0.09	-	-
Egg weight at 35 weeks	HW-Dw	-0.31	-0.22	-0.04	-0.27†
	dw	0.14	0.17	-0.08	0.35*
	LW-Dw	-0.40*	0.14	0.00	0.04
	dw	-0.40*	-0.05	0.30	-0.31†
Shell thickness at 35 weeks	HW-Dw	-0.53**	-0.36**	-0.32	-0.32*
	dw	-0.33	0.16	-0.34	0.18
	LW-Dw	-0.11	0.09	-0.03	0.08
	dw	0.00	-0.24	0.18	-0.33*

**P $\leq$ .01

*P $\leq$ .05

†P $\leq$ .10

correlations between broken and normal eggs suggest that egg breakage is not proportional to the frequency of laying, but depends upon the inherent characteristics of individual birds. The poor layers are liable to have more broken eggs. Positive correlations between broken and ovulatory defective eggs indicate that broken eggs may often be of thin and soft shell and occupy a transitory place between a soft shelled and normal egg. It is legitimate to expect a negative correlation between egg breakage and shell thickness. The effective mass of the cage floor increases in proportion to weight of the bird and may influence egg breakage due to a greater impact when the egg is dropped on the cage floor. The role of these factors on the incidence of cracks in eggs was evaluated by Carter (1971). It is rather interesting that shank length did not influence egg breakage reaffirming the earlier view that egg breakage depends upon the laying behavior of the bird.

In the HW line, the percentage HDP of ovulatory defective eggs was positively correlated with shank length and negatively correlated to the normal eggs and shell thickness. Although the relationship between shank length and ovulatory defective eggs cannot be explained, if a true relationship exists it may be used as one of the selection criteria to reduce the incidence of defective eggs in dwarf breeder pullets. The negative correlation between normal and defective eggs indicates that defective eggs are produced at the cost of normal eggs in HW normal pullets. On the other hand, the low and nonsignificant correlation among these traits in the HW dwarfs may be due to low

frequency of defective eggs. The negative correlation between ovulatory defective eggs and shell thickness is in agreement with the etiology of defective egg production.

Although the correlations in HW dwarfs were not significant and consistent among generations, they followed similar trends to those observed for the HW normal pullets. The correlations in LW line may not be reliable for any meaningful inferences, not only because of their inconsistencies among generations, but also due to prominent zero class.

Egg weight, shell thickness and egg quality: None of the main effects including the genotype was significant for initial egg weight in both lines, and did not show any consistent trends over generations (Tables 28 and 29). The individual variation within a genotype was more than the variation between genotypes. The depressive effect of dw gene on initial egg weight seemed similar in both lines. Also, the effect of genotype on 35-week egg weight was significant for the B_2 and B_3 generations in both lines with the normal pullets producing significantly heavier eggs than the respective dwarf pullets. The dwarf effect, however, appeared to be slightly more in the LW than the HW line. This is consistent with the observations of French and Nordskog (1973) who stated that the relative reduction in egg weight due to the dw gene was greater in their small than in their large Leghorn line (12.6% vs 7.5%). Although the effect on egg weight was similar to that on body weight and shank length, the magnitude of depression was significantly less. The dwarf effect on 35-week egg weight in the LW

line was similar to that reported in egg-type stocks (Hutt, 1959; Prod'homme and Merat, 1969; Selvarajah et al., 1970).

In the HW line the dw gene reduced the initial shell thickness but not shell thickness or specific gravity of eggs at 35 weeks of age. The depressive effect of the dw gene on initial shell thickness seemed more severe in the LW than the HW line resulting in shell thickness being the primary cause of defective and broken eggs in LW dwarf pullets.

There was no difference between dwarf and nondwarf pullets for haugh units in either the HW or LW line (Table 29).

Summary

The effect of the sex-linked recessive gene, dw, on growth and reproductive traits were evaluated in the genetic backgrounds of populations with high and low growth potentials. Data for the B_3 and B_4 generations received the greatest emphasis in comparisons between dwarfs and their normal sisters because of the additional backcrossing. There was a suggestive evidence that the depressive effect of the dw gene is more severe on body weight than on skeletal growth and in the genetic background of populations exhibiting slower rather than faster growth. Differential effects among lines on reproductive traits were also observed, with HW dwarfs maturing earlier and having a higher production of normal eggs than nondwarfs from this line.

Detrimential effects of the dw gene were noted in the LW line. Evidence was obtained that corroborated results from other studies

regarding the action of dwarfing gene in bringing the ovarian and oviducal functions into synchrony. It is concluded that the dw gene may be beneficial in stocks with excessive yolk synthesis and arhythmic ovulations, while it has detrimental effects in those with optimum ovarian activity. Within a population of dwarf pullets, egg breakage was largely independent of shank length. The data on growth and reproductive parameters in dwarf and normal pullets in the four backcross generations indicate that the effects of the dw gene are largely dictated by the specific interactions with the background genotype and the presence of genetic modifiers.

EXPERIMENT IV. ULTRASTRUCTURAL STUDIES OF ADENOHYPOPHYSICAL GONADOTROPHS IN GROWTH SELECTED LINES OF CHICKENS

Several independent studies (Jaap and Muir, 1968; Udale, 1972; van Middelkoop,1973a; Reddy and Siegel, 1976) have shown that hens of meat-type stocks exhibit a high rate of yolk synthesis along with erratic rhythms of follicular maturation and ovulation. This would suggest that populations genetically divergent for growth and reproductive parameters may differ in the frequency and/or activity of gonadotrophs in the pars distalis. Therefore, the ultrastructural characteristics of these cells may provide information necessary to understand the basis for the divergent ovarian activity in these lines.

This experiment was designed to study the relations between egg production patterns and gonadotrophic cell activity at the ultrastructural level in S_{16} generation pullets of lines selected for high and low juvenile body weight.

Materials and Methods

The pullets utilized for this experiment are those described in Trial 2 of Experiment I. Anterior pituitary glands were removed from samples of HWS and LWS pullets 40, 80, 120, and 160 days after the laying of the first egg.

Pullets were anaesthetized by pentobarbital sodium, the cervical region dissected, and 2 ml of 2% glutaraldehyde injected into the carotid artery in an effort to fix the anterior pituitary gland in situ. After 2 to 3 minutes, the bird was decapitated and the skull

opened to expose the anterior pituitary gland. The pars distalis was separated into the cephalic and the caudal lobes, and 1 mm^3 tissue blocks were fixed at 5°C in 3% glutaraldehyde adjusted to a pH of 7.2 to 7.4 in 0.1 M phosphate buffer (Luce, 1966). After fixation for 1 to 2 hours the tissue was transferred to 0.1 M phosphate buffer and held at room temperature overnight. The tissue was then postfixed in 1% osmium tetroxide in 0.1 M phosphate buffer for 1 hour followed by dehydration in a graded ethanol series. Embedding was in Epon 812. The fixation, dehydration and embedding materials and schedules are presented in the appendix. Sections were cut with glass knives on a Portor-Blum MT-2 ultramicrotome, stained with uranyl acetate and lead citrate (Venable and Coggeshall, 1965) and examined with an RCA-EMU-3H electron microscope using a voltage of 50 kV.

Ultrastructural characteristics of cell-types: Although there may be variations among avian species, the topographical distribution of various cell-types is fairly well established in the domestic fowl. There are at least five cell-types in the pars distalis; somatotrophs (ST), luteotrophs (LT), gonadotrophs (GT), corticotrophs (CT), and the undifferentiated chromophobe cells (C), (Payne, 1965; Mikami, 1969). Amin and Gilbert (1970), however, could not characterize corticotrophs in the pars distalis of the domestic fowl. While the gonadotrophs were distributed at random in both the lobes, the somatotrophs were located exclusively in the caudal lobe, luteotrophs primarily in the cephalic lobe, while thyrotrophs and corticotrophs were confined to the cephalic lobe. With the light microscope, these

cell-types may be identified by the tintorial affinities of their secretory granules. Granule size, form and density served as the most single useful criterion for cell identification under the electron microscope. The characteristics of cell organelles, especially those of the rough endoplasmic reticulum (RER), the Golgi complex (GC) and the mitochondria (M) further aided in the proper identification of the cell-types. The ultra structural characteristics used to differentiate between cell-types are briefed in Table 34.

In this study, only the gonadotrophic cell activity of the two lines under two photoperiodic treatments was considered.

Morphological differences among gonadotrophs: Although morphological distinctions can be made between the two types of gonadotrophs, functional differentiation is difficult. Both types were observed as a group, since both FSH and LH were suspected to be involved in the manifestation of the Erratic Ovulation and Defective Egg Syndrome (EODES). The RER is characteristic in two cell-types, those presumably secreting FSH and LH. In Type-A cells the RER is comprised of cisternal profiles along the periphery of the cell or are scattered through the cytoplasmic matrix, and are surrounded by ribosomes. The Golgi complex is extensively developed and contains numerous dense granules of variable size and shape. In the Type-B cells the RER is a vesicular type with moderate Golgi apparatus. The Type-B cells contain secretory granules of variable size (150-200 mμ) and are relatively less dense.

Table 34. Ultrastructural characteristics used to differentiate cell-types in *pars distalis* of *Gallus domesticus*

Characteristic	Acidophils		Basophils		
	STH	LTH	TSH	GT-A	GT-B
1. Location	Caudal lobe	Cephalic lobe (principally) (primarily)	Cephalic lobe	Both lobes	Both lobes
2. Secretory granules					
a. size (mμ)	350-400	400-450	50-100	200	150-350
b. Form	Spherical	Oval and polymorphic	Spherical	Spherical	Polymorphic
c. Density	Very dense	Very dense	Dense	Dense	Less dense
3. Golgi-apparatus	Less developed and fragmented	Circular Prominant lammelar	Less developed	Prominent lamellar and vesicular	Small and less developed
4. Endoplasmic reticulum	Less developed and cisternal often obscured by dense branules	Prominent lamellar	Less developed and vesicular	Prominent cisternal	Prominent vesicular
5. Mitochondria	Large spherical	Large elongated	Small rods	Small rods	Large elongated

The protein secreting activity of the gonadotrophs was scored on an arbitrary scale in the range of (+) to (+ + + +). This was based on the extent and complexity of the RER, the Golgi apparatus, and the distribution of secretory granules. Similarly, lysosomal digestion of secretory granules was estimated by the number and size of lytic bodies.

Ultrastructural Observations

HWS pullets: Data pertaining to ovarian activity, egg production and gonadotrophic cell activity for individual birds of the HWS line are presented in Table 35. The most conspicuous feature of the gonadotrophs from these birds was the moderate to well developed rough endoplasmic reticulum and the large and extensive Golgi complex. Depending on the type of gonadotrophic cell, the RER was both of the lamellar (Plate 1, 2b, and 2c) and vesicular (Plate 2a) types. In most cases the RER was fragmented and scattered in the cytoplasmic matrix. Nebenkern formations which are a characteristic feature of most of the protein secreting cells was very rare in these cells. In a few instances the RER cisternae was masked by the presence of secretory granules, while in others the ribonucleoprotein particles were distributed free in the cytoplasmic matrix. Numerous smooth surfaced vesicles could be seen between the functional elements of RER and Golgi cisternae, which may constitute the so called "shuttling system" for transport of the synthesized product from the RER to the Golgi apparatus. There was consistent morphological evidence of budding of immature secretory granules from the inner Golgi lamellae, suggesting

Table 35. Ovarian activity, egg production and functional cytology of the gonadotrophs of pullets from the HWS line

Pullets No.	Days in lay	No. of Follicles at Autopsy			%HDP[1] of Eggs		Activity of Gonadotrophs	
		Developing	Ruptured	Atretic	Normal	Defective	Synthesis	Crinophagy
				Photoperiod I (14L:10D)				
323C	40	7	7	-	5.3	40	+ + + +	+ +
521G	80	5	8	-	69	23	+ + +	+
381A	80	6	6	-	76	-	+ +	
441E	120	7	7	-	86	-	+ + +	
322J	120	5	6	1	65	18	+ +	
320C	160	7	5	1	80	2	+ + +	
380C	160	6	6	2	76	6	+ + +	±
				Photoperiod II (14L:10D, 3%Δ)				
421A	40	6	9	-	33	58	+ + +	
502J	40	7	9	-	55	48	+ + +	
502F	40	6	7	-	75	5	+ +	
420E	80	9	8	-	55	21	+ + +	+
361C	80	6	9	-	56	36	+ + +	+ +
461C	80	8	6	-	76	4	+ + +	+
302A	120	8	5	2	42	1	+ + +	+ + +
483B	120	6	6	-	71	1	+ +	
400G	160	6	5	1	66	12	+ +	

[1]HDP = Hen-day production

an active state of the cell (Plates 3 and 6). This may be the most dependable evidence for the Golgi origin of secretory granules. In most cases the central core of cytoplasm encompassed by the Golgi apparatus contained numerous polymorphic secretory granules of variable density, indicating that secretory granules were being formed in several steps. Morphological evidence of several secretory granules aggregating into larger polymorphous granules was abundant (Plates 3 and 12).

The specific activity of the GT-cells was based not only on the ultrastructural characteristics of the protein synthetic apparati (RER and GC), but also on the frequency and distribution of secretory granules. Thus, heavy granulation represented a "storage" phase and sparce granulation was interpreted as a "secretory" or "regranulation" phase. Some of the secretory granules were non-electron dense due to a transitory phase of condensation. Occasionally the GT cells (Plate 4) resembled early castration cells as described by Payne (1965) for the domestic fowl and by Mikami et al. (1969) for White Crowned Sparrows. In a few instances, the Golgi lamellae were fragmented and surrounded by smooth forming vesicles resulting in many transitional stages between Golgi lamellae and smooth vesicles (Plate 5). Mitochondria, though few, were located in the region of the Golgi complex and presumably were involved in energy metabolism during biosynthetic activity. In these cases the cristae of the mitochondria were highly developed (Plate 6). Ichikawa (1965) reported that mitochondria move into this Golgi zone during the most active synthetic stage and some

of the mitochondria show considerable deformations in their ultrastructure. Morphological findings suggested that secretion of granules by the GT-cells was by exocytosis coupled with endocytosis. The mature secretory granules were often very close to the plasma membrane and a few endocytic vacuoles could be found in this region (Plates 5 and 7).

The frequency of lytic bodies and multivesicular bodies in the GT cells was high. There was considerable variation in their size and shape (Plates 7 and 8). The lytic process was primarily autophagic in nature. The multivacuolar dense bodies were rather irregular in outline and contained 1-10 vacuoles. These vacuoles probably represented undigested lipid droplets because lysosomal fractions are poor in lipases and phospholipases (Tappel and deDuve, 1963). It is not until the lipid droplets are released into the cytoplasm that they are brought into contact with the lipid splitting enzymes. There was morphological evidence that these lipid droplets were being released from the dense bodies (Plate 7). The multivacuolated dense bodies correspond to the lipochondria in the pancreatic exocrine cells described by Baker (1951) and to "vacuolated bodies" in renal tubules described by Miller and Palade (1964). Occassionally multivesicular bodies containing aggregating granules of low density were encountered in cells showing evidence of intracellular digestion of secretory protein (crinophagy).

A qualitative analyses of the secretory status, of pituitary GT cells, based on the amount and complexity of the protein synthetic apparatus, indicated that 11 out of 16 pullets showed evidence of

hyperactivity in GT cells. Six pullets with hyperactive gonadotrophs in the anterior hypophysis exhibited evidence of arhythmic ovulations that resulted in a significantly higher production of defective eggs. An extensive lysosomal digestion of secretory product was evident in 7 out of 16 pullets. No gross ultrastructural differences were observed in pullets from the two photoperiodic treatments and different physiological ages studied.

LWS pullets: The overall gonadotrophic activity, as evidenced by ultrastructure of the protein synthetic apparatus and by the granule distribution, was lower in the LWS pullets (Plates 9 to 11). The RER was less elaborate, and the Golgi apparatus was less conspicuous when compared with cells from HWS pullets of comparable physiological ages. Gross ultrastructure revealed fewer aggregating granules and "shuttling" vesicles in the core of cytoplasm (Plate 12).

Of the 16 pituitaries studied, four showed evidence of hyperactive gonadotrophs (Table 36). It was not possible to associate hyperactivity of gonadotrophs with arhythmic ovulation pattern (Plates 12 and 13) since the frequency of defective eggs was considerably lower in the LWS line. There was evidence of lysosomal digestion of secretory products in three of 16 pullets (Plates 14 and 15). Crinophagy tended to be positively related with cellular activity. Photoperiods did not effect the ultrastructure of GT cells in this line.

Comparison between lines: Chi-square analyses showed that the frequency of pullets with hyperactive gonadotrophs was significantly higher in the HWS than in the LWS line. No significant difference,

Table 36. Ovarian activity, egg production and functional cytology of the gonadotrophs of pullets from the LWS line

Pullets	Days in lay	No. of Follicles at Autopsy			%HDP[1] of Eggs		Activity of Gonadotrophs	
		Developing	Ruptured	Atretic	Normal	Defective	Synthesis	Crinophagy
				Photoperiod I (14L:10D)				
781A	40	4	4	-	78	-	+ + +	
803B	40	3	3	-	52	-	+ +	
621F	80	3	4	-	62	-	+ +	
723A	80	5	5	-	71	0	+ +	
660D	120	4	3	1	60	1.7	+ + +	+ ±
642F	120	4	4	-	67	0	+ +	
722J	160	4	3	-	61	0	+ +	
580A	160	4	5	1	41	0	+ +	
				Photoperiod II (14L:10D 3%Δ)				
641B	40	4	3	-	30	2.5	+ +	
781E	40	4	4	-	75	0	+ + +	
660J	80	4	4	-	69	-	+ +	
782A	80	4	5	-	81	-	+ + +	±
761B	120	5	4	-	79	0.8	+ +	
582I	120	4	3	-	58	0.8	+ +	
760	160	3	4	-	64	0	+ +	+ +
741B	160	3	2	-	53	0.6	+ +	

[1]HDP = Hen-day production.

however, was found in the incidence of crinophagy. Qualitative analyses of the ultrastructure of GT-cells from the two lines suggest a higher degree of activity in HWS line at all physiological ages. Increasing photoperiods did not have any significant effect on the GT-cell activity in either line.

Discussion

Anterior pituitary glands from genetically divergent lines known to have different growth and reproductive parameters provide interesting biological material for probing ultrastructurally. The main result from this experiment was to demonstrate that the gonadotrophs from HWS line pullets exhibited a greater degree of activity than did those from the LWS line. The evidence was based on the morphology of the protein synthetic apparatus. The general scheme of the secretory process in the anterior pituitary gland followed the classical concepts established by Palade and his coworkers (Siekevitz and Palade, 1960; Caro and Palade, 1964; Jamieson and Palade, 1966, 1967a,b, 1971; Redman et al., 1966; Castle et al., 1972; and Palade, 1975). According to their scheme the secretory proteins are synthesized on ribosomes, transferred to endoplasmic reticulum cisternae, and then transported to the Golgi apparatus, where they are condensed and subsequently secreted by exocytosis.

Although the differences between the HWS and LWS lines in gonadotrophic activity are based on qualitative observations, they are consistent with the data on egg production and ovarian activity.

The HWS pullets had more ovarian follicles under rapid development than LWS pullets did and the ovulation patterns of the HWS line was arhythmic. This suggests that selection for growth (protein anabolism) may trigger not only lipoprotein anabolism in the liver, but also protenaceous secretions from various glands. Thus, the excess secretions from the gonadotrophic cells in the HWS line may cause surges in the blood levels of these hormones affecting follicular growth and maturation. Various explanations for the arhythmic ovulations in fast growing meat type chickens include neural thresholds and target organ sensitivity to gonadotrophic hormones (Jaap and Muir, 1968; Van Krey and Siegel, 1968). Although the pituitaries from the HWS line were heavier than those from the LWS line on an absolute basis, the difference disappeared when adjusted for body weight. This was consistent with observations made in the earlier generations that led Siegel et al. (1968) to conclude that the primary correlated response to selection for juvenile body weight was the size of the pituitary rather than its unit potency. The ultrastructural observations in this experiment demonstrate that selection has also influenced the differentiation of gonadotrophic cells at the level of the genome. It has been traditionally assumed that the hormones produced by the cells of the anterior pituitary are stored as granules in the cytoplasm for a variable period of time before they are discharged into the circulation. The ultrastructural observations suggest that another pathway exists for the turnover of secretory protein. This intracellular disposal mechanism termed as "crinophagy" was described first in

mammotrophs of the rat pituitary by Smith and Farquhar (1966), but further work has shown that it probably occurs in all the cells of anterior pituitary (Farquhar, 1971). The high incidence of crinophagy in gonadotrophs in this study may be interpreted as due to greater synthesis and/or lower rate of secretion. The role of Ca^{++} ion and energy in the process of exocytosis is well established (Douglas and Rubin, 1963). Since secretion from cells of the anterior hypophysis is largely by exocytosis, a calcium deficiency or debt at the cellular level could slow down the rate of secretion and induce an intracellular disposal of secretory products by lysosomes. Smith and Farquhar (1966) demonstrated that suppressing LTH secretion by separating the young from lactating mothers induced crinophagy in mammotrophs. The use of lysosomes for degrading excess secretory proteins may be a protective mechanism against lysosomal hydrolases, which might be at work along the secretory pathway beginning with the RER (Palade, 1975). It was hypothesized by Smith and Farquhar (1966) that lysosomes operate at the catabolic end of protein turnover and constitute a regulatory mechanism to compensate for an oversynthesis of the secretory products. Lytic bodies occur in all types of cells in the adenohypophysis of normal animals. Thus, it would appear that the secretory products may, depending on the fluctuations in secretory activity, be more or less continuously channeled through the lysosomal pathway. The higher incidence of crinophagy, in addition to the greater activity of gonadotrophs in HWS pullets suggest that fluctuations in cell activity were more common in that line.

Summary

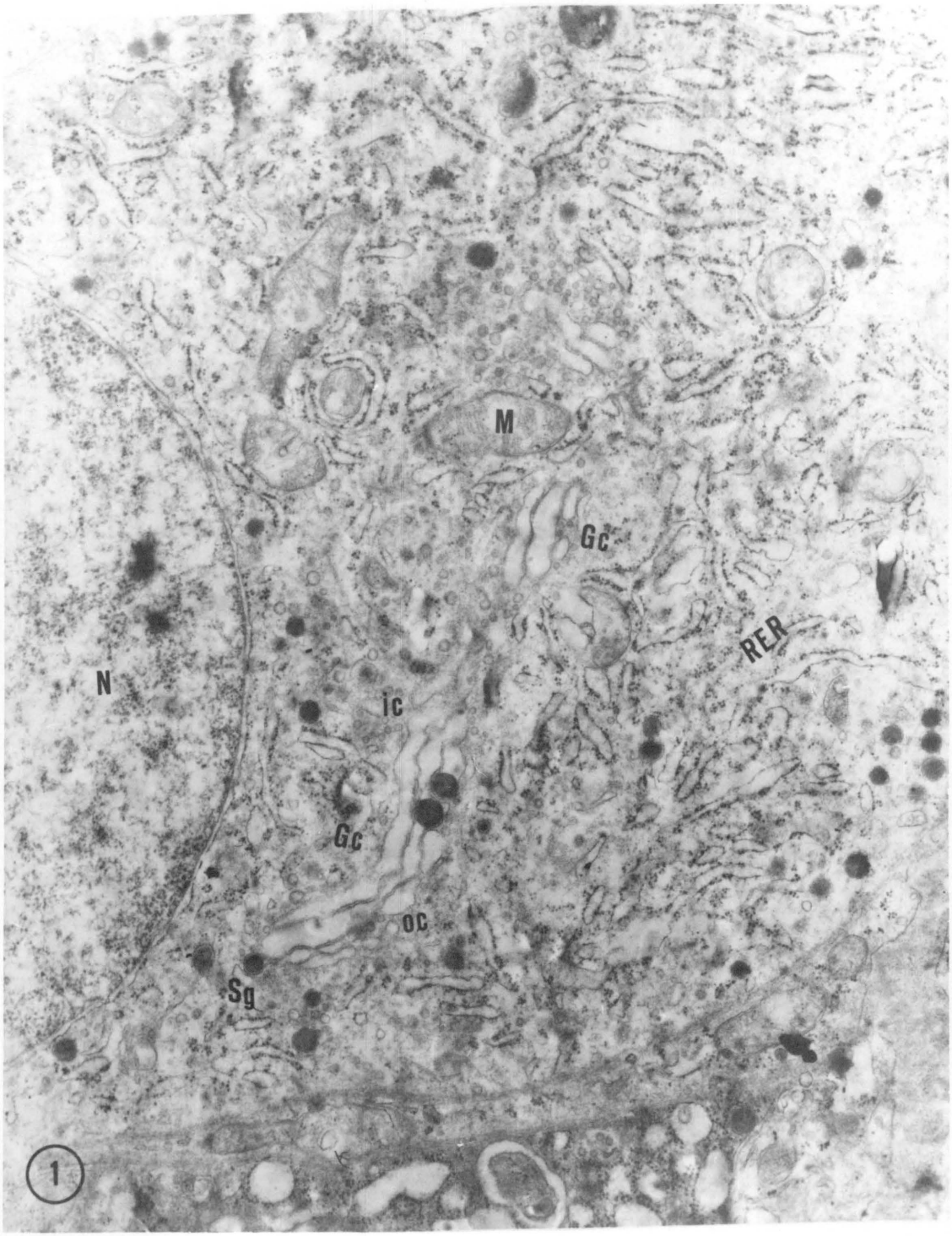
The ultrastructural studies of pituitary gonadotrophs, based on the degree of development and the complexity of the protein synthetic apparatus and on the secretory granule distribution indicated a higher degree of synthetic activity in HWS pullets when compared to the LWS pullets. This was true at all physiological ages studied. The incidence of crinophagy was also greater and more conspicuous in HWS line. Crinophagy tended to be positively related to cell activity in both lines. Increasing photoperiods did not have any significant effect on the ultrastrucutre of GT-cells in either line. It was concluded that ovarian activity, ovulation patterns, and the defective egg syndrome were due to fluctuations in GT-cell activity and/or in the relasing mechanism.

GLOSSARY OF SYMBOLS

Ag	Aggregating granule	LB	Lytic body
Av	Autophagic vacuole	Li	Lipid droplet
cem	centromere	M	Mitochondria
CH	Chromophobe cell	mv	Multivesicular body
cm	Cell membrane	N	Nucleus
DB	Dense body	oc	outer Golgi cisternae
GC	Golgi complex	r	Ribosomes
GD	Golgi dictyosome	RER	Rough Endoplasmic Reticulum
GT-A	Gonadotroph A cell	SER	Smooth Endoplasmic Reticulum
GT-B	Gonadotroph B cell	Sg	Secretory granule
ic	Inner Golgi cisternae	ST	Somatotroph
L	Lysosome	sv	smooth vesicle

Plate 1: A GT-A cell from HWS pullet A320C

The rough endoplasmic reticulum (RER) is highly developed, with short flattened profiles that are scattered in the cytoplasmic matrix. The Golgi complex (GC) is juxtanuclear, with many secretory granules forming in the inner Golgi cisternae (ic). There are only a few mature secretory granules with no evidence of accumulation. The cell seems to be in a state of rapid synthesis, condensation and release of secretory product.

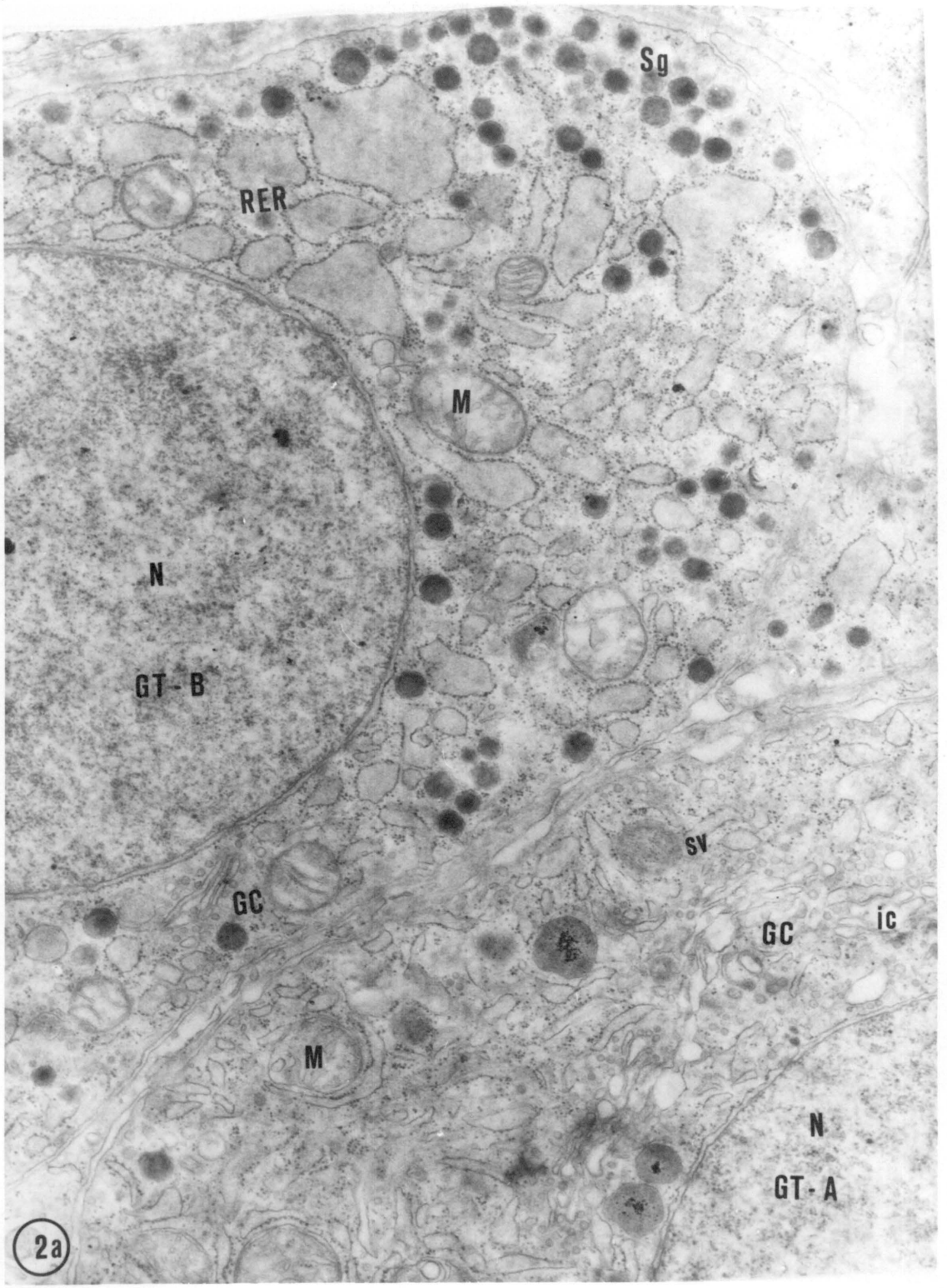


x26,700

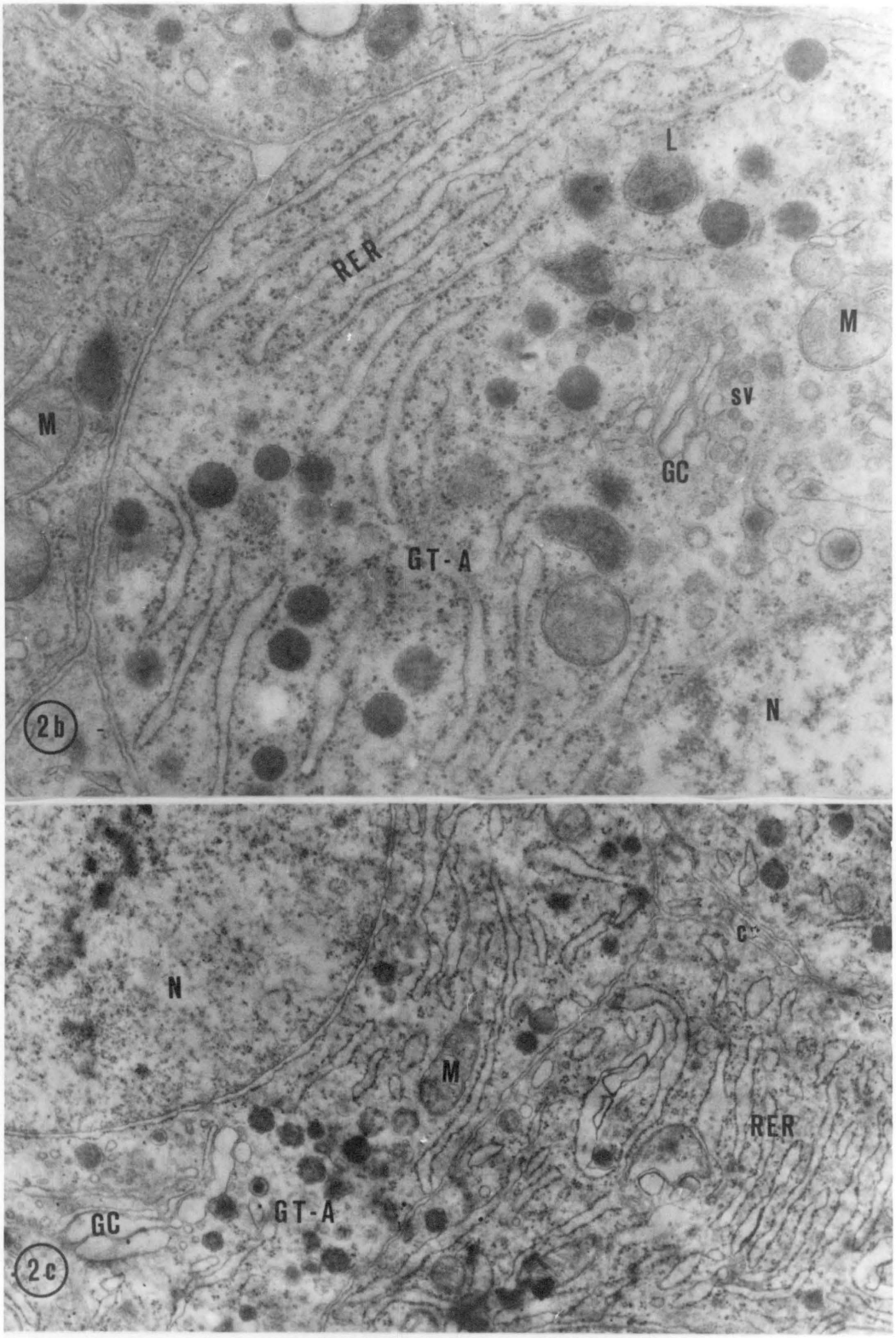
Plate 2a, b, c: GT-cells of A and B types from HWS pullet A323C

Two types of GT-cells are seen, but it is not possible to characterize their gonadotropic activity. It is assumed that two types of cells are probably differentiated to secrete FSH and LH. The rough endoplasmic reticulum (RER) in the upper cell is of a vesicular type whose Golgi complex (GC) is less developed and situated juxtanuclear. The secretory granules (Sg) are largely polarized and near the plasma membrane.

The lower cell has an extensively developed juxtanuclear Golgi complex (GC). Many profiles of forming granules are seen in the inner Golgi cisternae (ic). There are a few smooth surfaced "Shuttling"vesicles (sv) between the Golgi zone and the short scattered lamellae of RER. The rough endoplasmic reticulum (RER) in plates 2b and 2c is also lamellar type and is extensive with active ribosomes.



x15,100



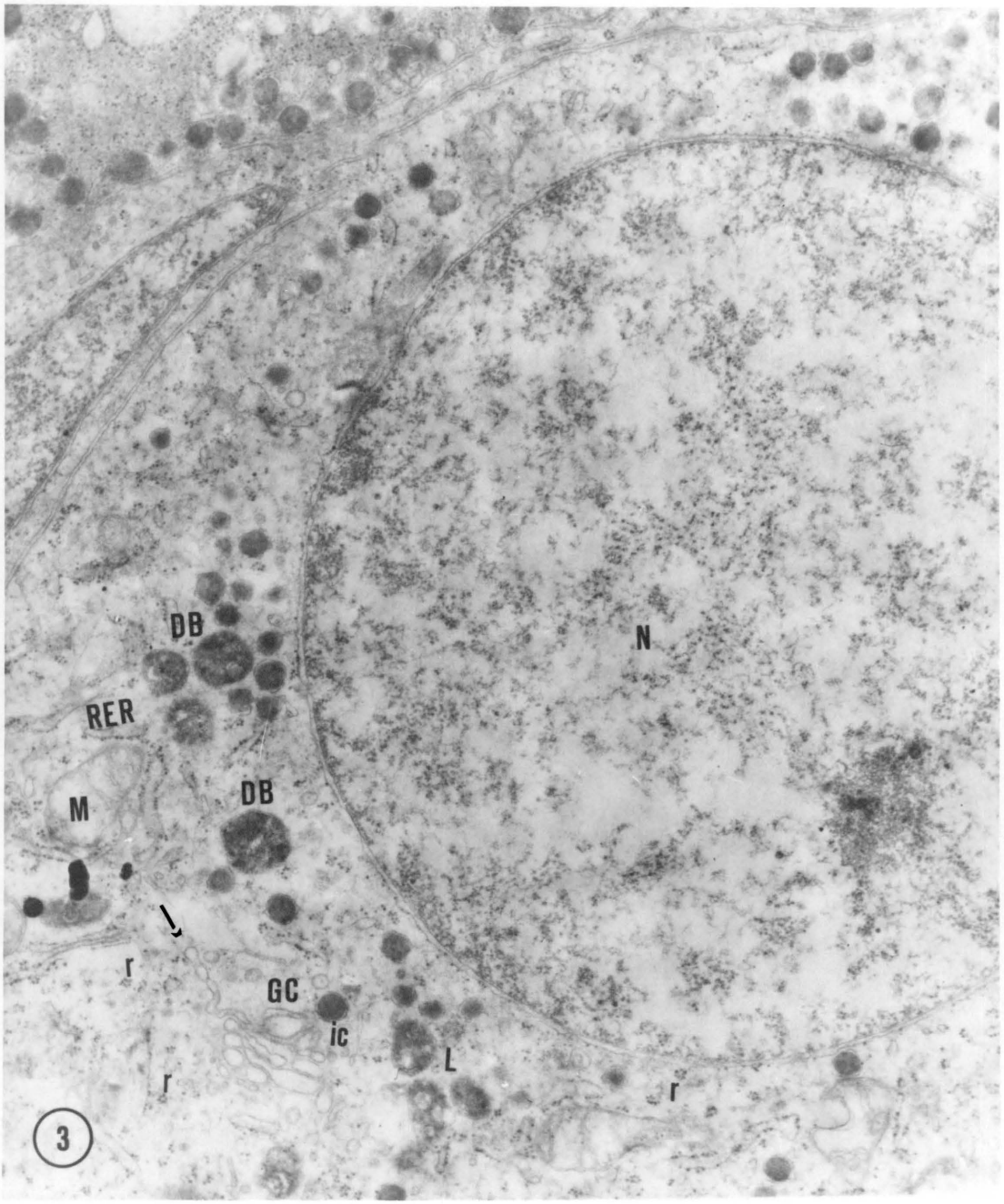
2b 2c

38,000 17,900

Plate 3: A GT cell from HWS pullet A441E

This cell has a moderate rough endoplasmic reticulum (RER) and an active Golgi complex (GC). Numerous ribosomes (r) are apparent throughout the cytoplasmic matrix. There is clear evidence of immature secretory granules pinching off (arrow) from the inner Golgi cisternae (ic). Typically few lysosomes (L) are seen in the Golgi zone.

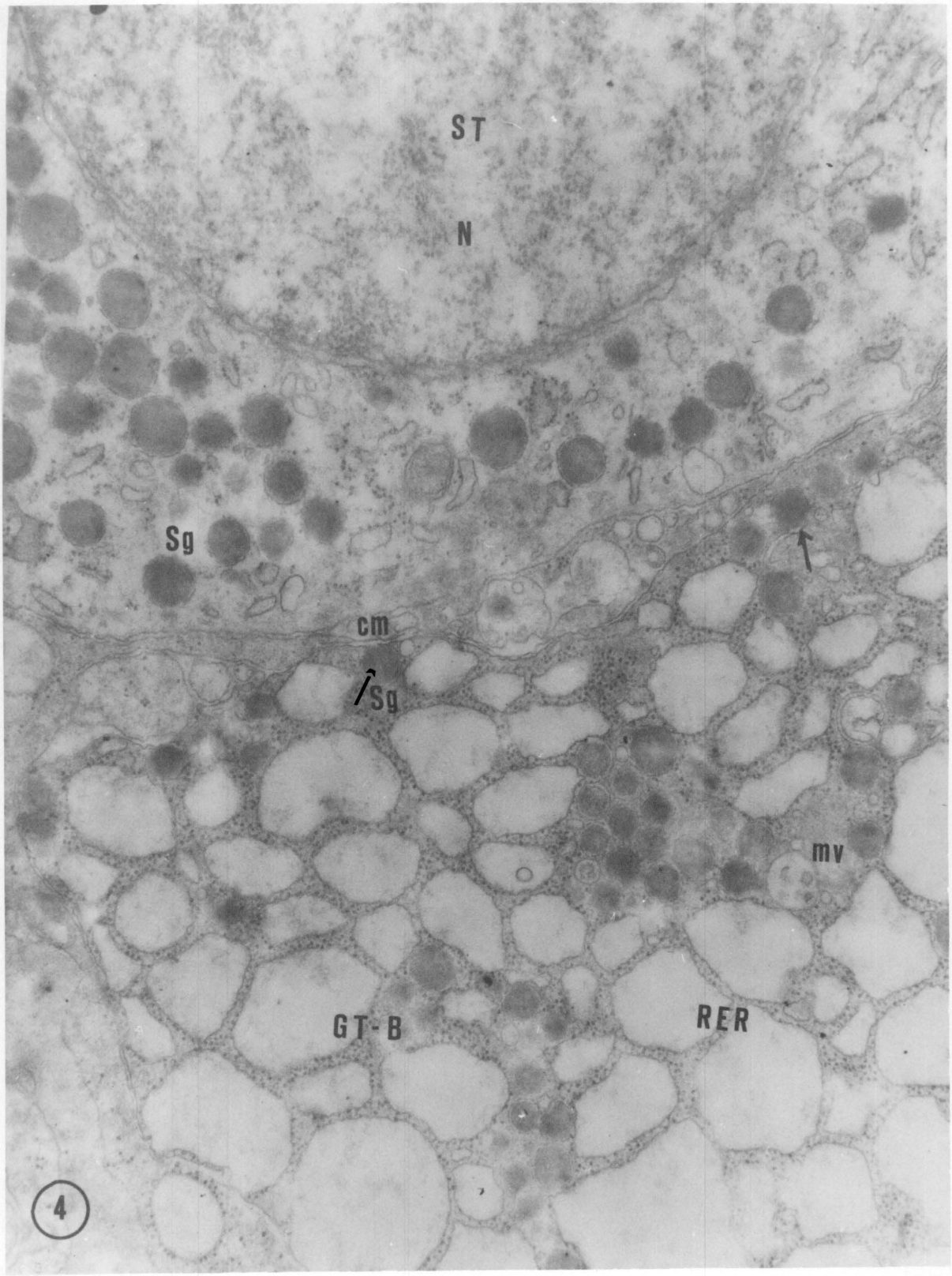
The bird showed normal ovulation and oviposition patterns in the first 120 days of lay.



x24,900

Plate 4: A GT-B cell from HWS pullet A381A

The rough endoplasmic reticulum (RER) is vesicular and highly distended. The secretory granules (Sg) are less dense and smaller in comparison to the secretory granules of the somatotroph (ST) seen in the upper portion of the plate. The GT-B cell is interpreted to be very actively secreting. The secretory granules are lined (arrows) against the cell membrane (cm). A small multivesicular body (mv) can also be seen.

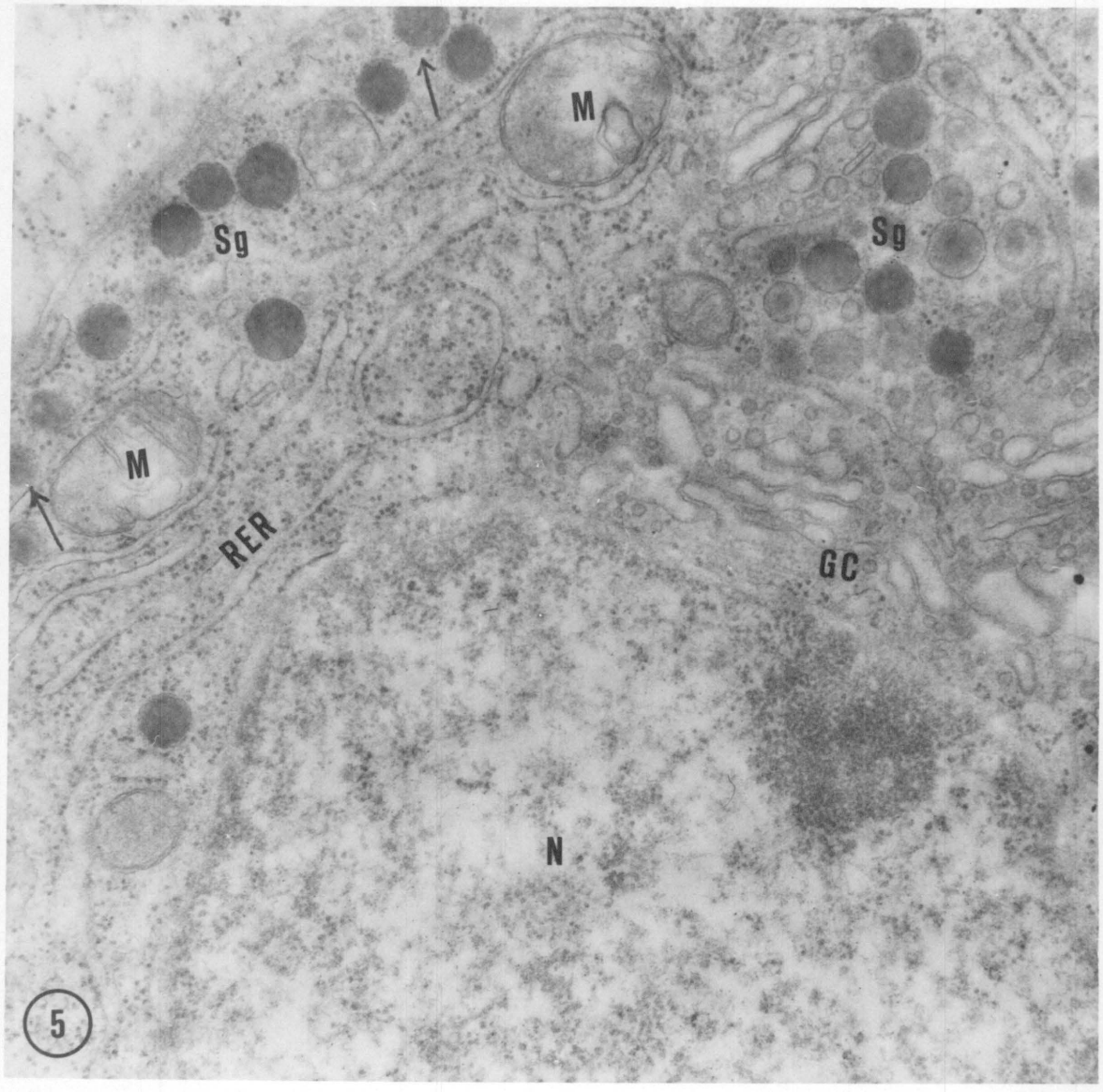


x42,100

Plate 5: A GT cell from HWS pullet A502J

The Golgi complex (GC) is extensive and forms a circular profile of lamellae. The core of cytoplasm circumscribed by the Golgi complex is literally filled with secretory granules in various stages of condensation. The mature secretory granules (Sg) are seen along the cell membrane (arrows) indicating the active secretory status of the cell. The rough endoplasmic reticulum (RER) consists of flattened cisternae studded with ribosomes.

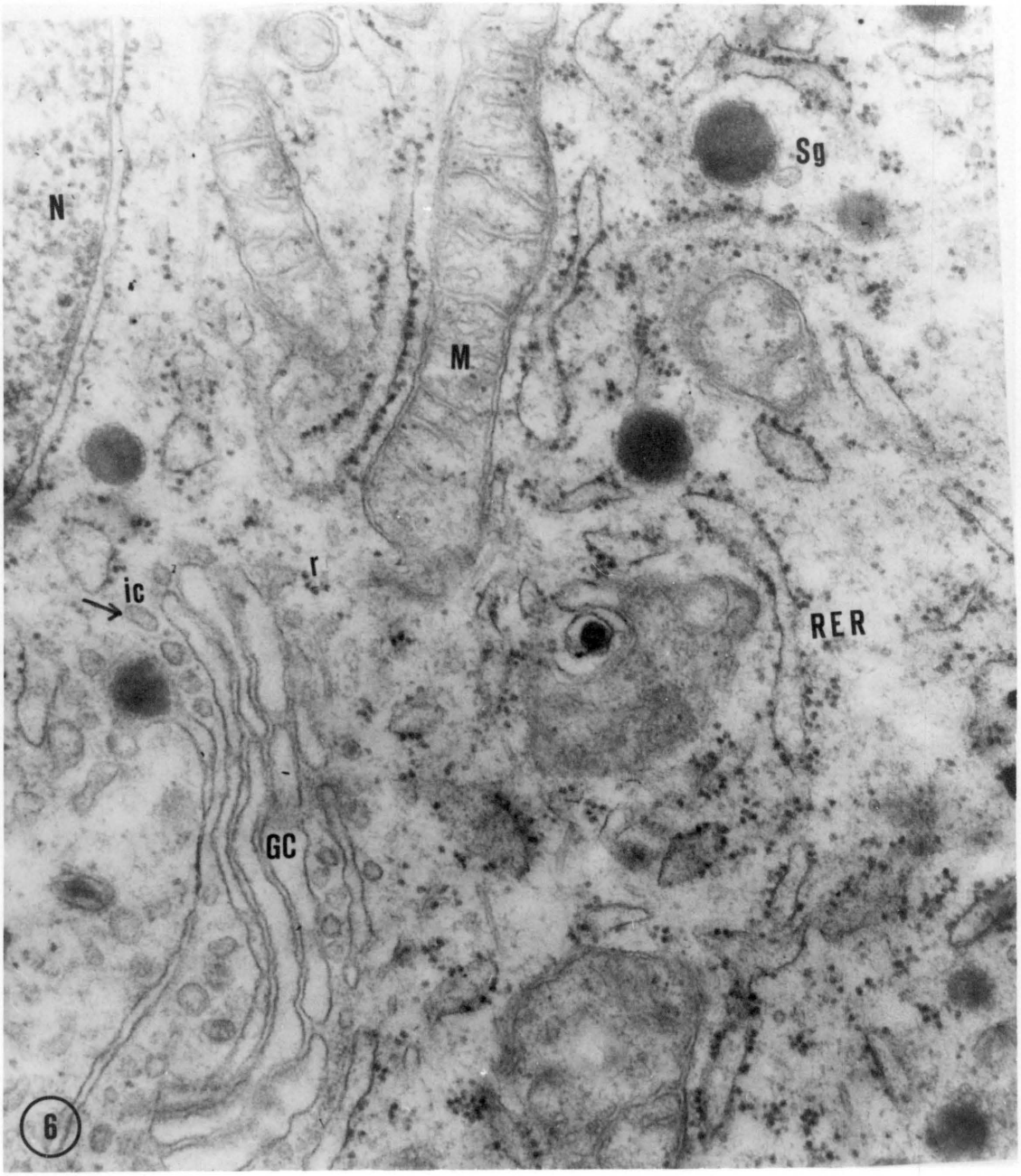
This bird exhibited evidence of arhythmic ovulation pattern in the first 40 days of lay.



x27,500

Plate 6: Portions of a GT-A cell from HWS pullet A323C

Portions of Golgi complex (GC) and of elongated mitochondria (M) are seen. Mitochondria are presumably involved in energy metabolism during the transport and condensation of secretory products from rough endoplasmic reticulum (RER) to the Golgi. Short RER lamellae covered by ribosomes are scattered in the cytoplasmic matrix. In addition there are free ribsomes (r) in the cytoplasmic matrix. Immature granules emerging from inner cisternae (ic) could be seen (arrow).

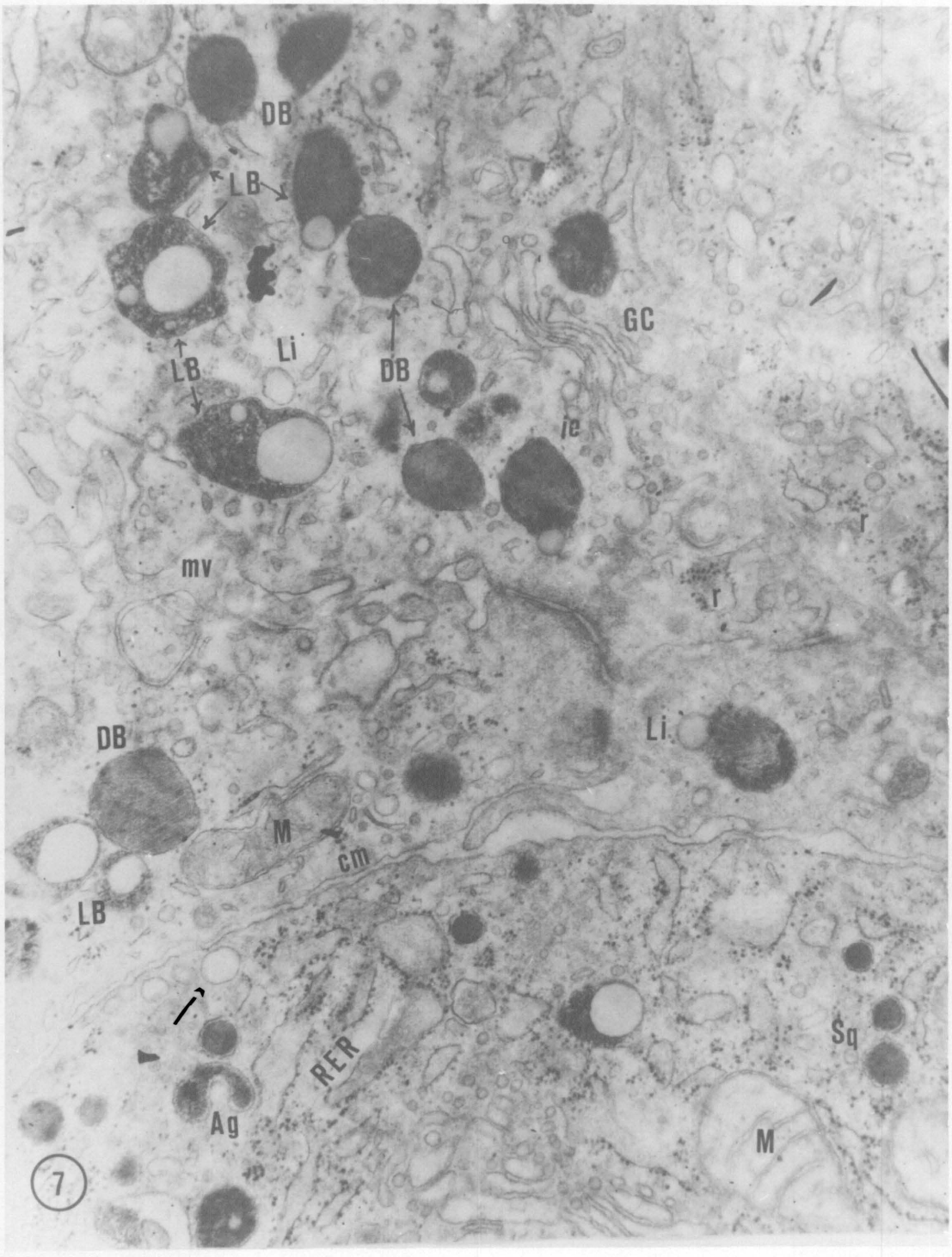


x61,000

Plate 7: Portions of a GT cell from HWS pullet A323C

Numerous dense bodies (DB) and lytic bodies (LB) of several morphological stages are seen. These structures occupy a major portion of the cell. There is also evidence of lipid droplets (Li) separating from lytic bodies. Few free lipid droplets can also be seen in the cytoplasm. The Golgi complex (GC) is active with profiles of forming granules. A few endocytic vesicles could be seen near the cell membrane (arrow).

This bird showed evidence of arhythmic ovulation patterns in the first 40 days of lay.

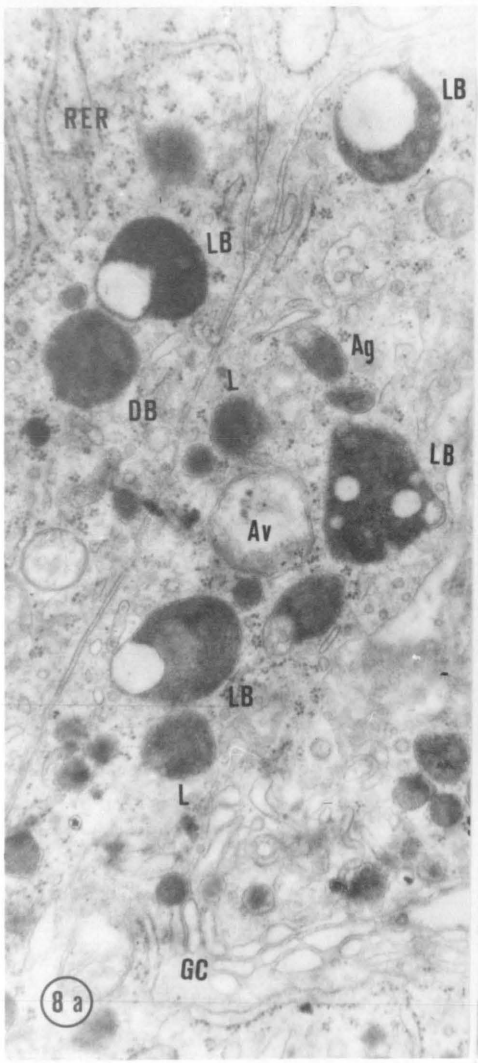


x36,700

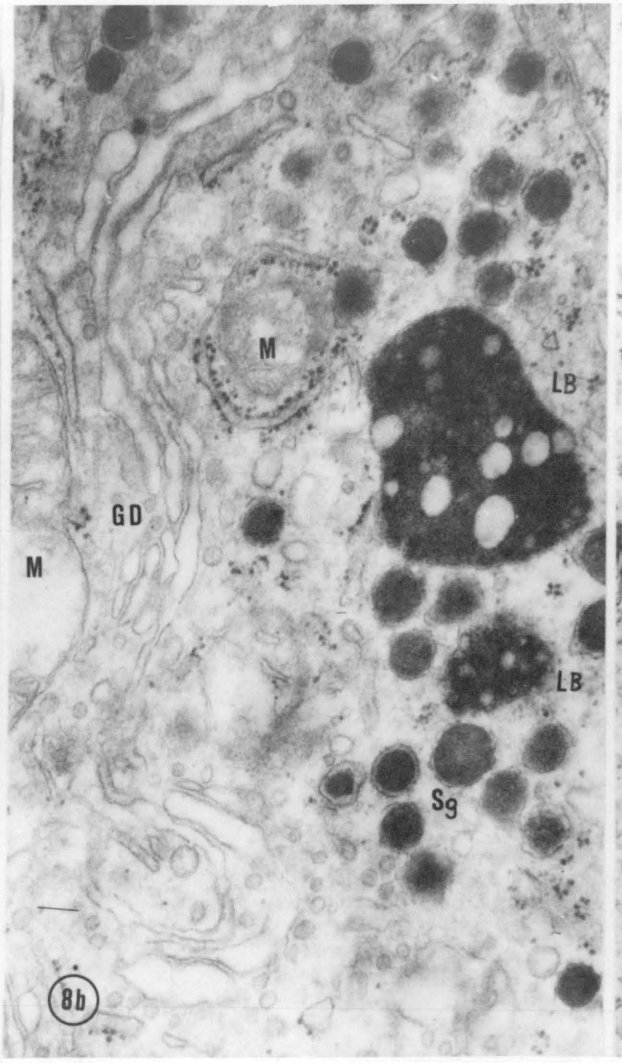
Plate 8 a, b, c: Portions of three GT cells from HWS pullet A302A

The Golgi complex (GC) is extensively developed with the evidence of granule formation in these areas, and at various stages of condensation. Numerous lytic bodies (LB) of several morphological types are present in all these cells. The cell in the middle has extensive Golgi dictyosome. There are indications that synthesis and autolysis of secretory products are occuring simultaneously in these cells. The two multivacular lytic bodies near the nucleus and an autophagic vacuole and two dense bodies in the active Golgi zone in 8c cell are evidence of such an occurance. The rough endoplasmic reticulum (RER) consists of short flattened cisternae. The Golgi areas have polymorphous granules in all these cells to depict transitory stages of condensation.

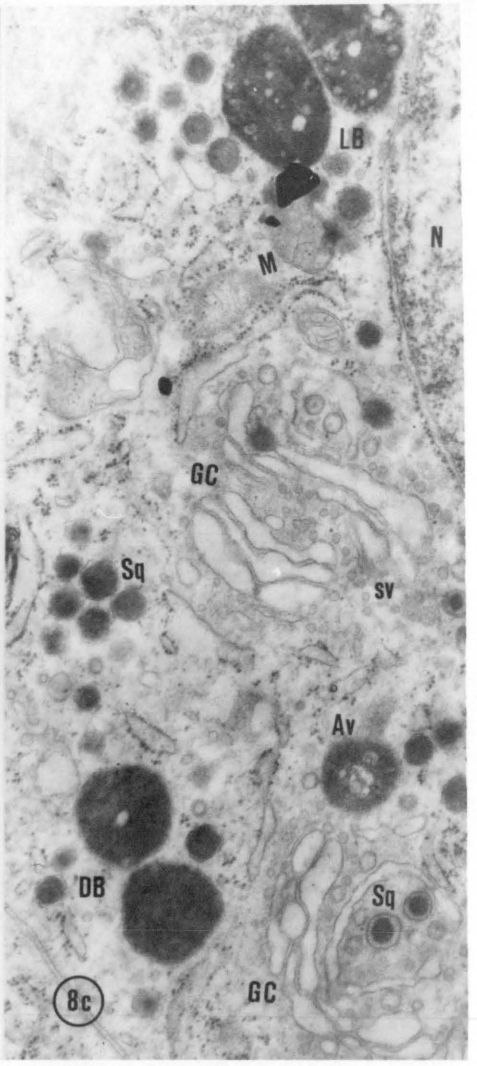
This bird was sacrificed after 120 days of lay. The egg production record and autopsy data relate well to the ultrastructure of the GT cells. This bird which stopped normal egg production about 20 days before sacrifice had more than the average number of growing and ruptured follicles. In addition, there was evidence of follicular atrisia. This bird was not out of production but was instead an obligate interval layer.



x29,700



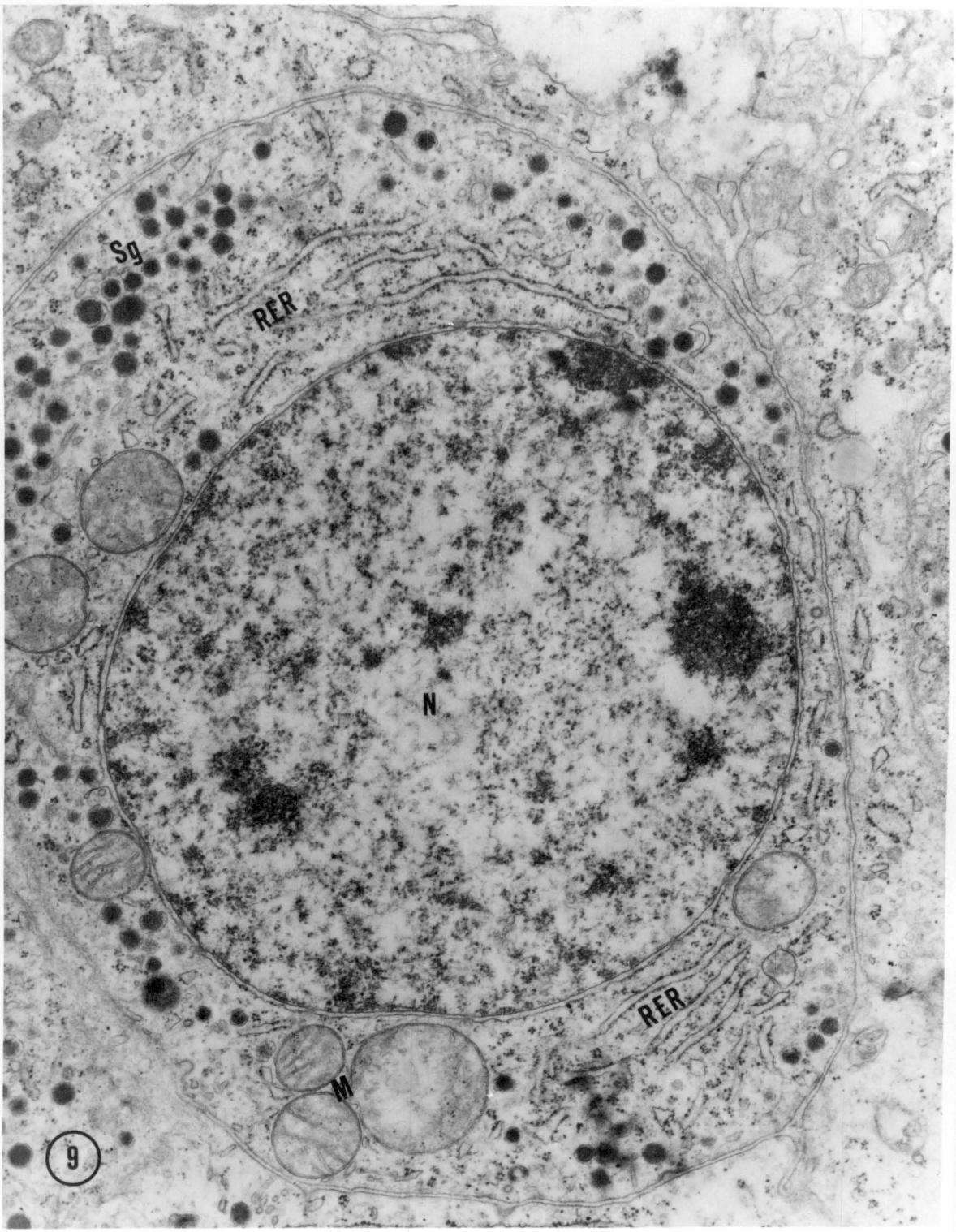
x45,000



x28,700

Plate 9: A GT cell from LWS pullet A602F.

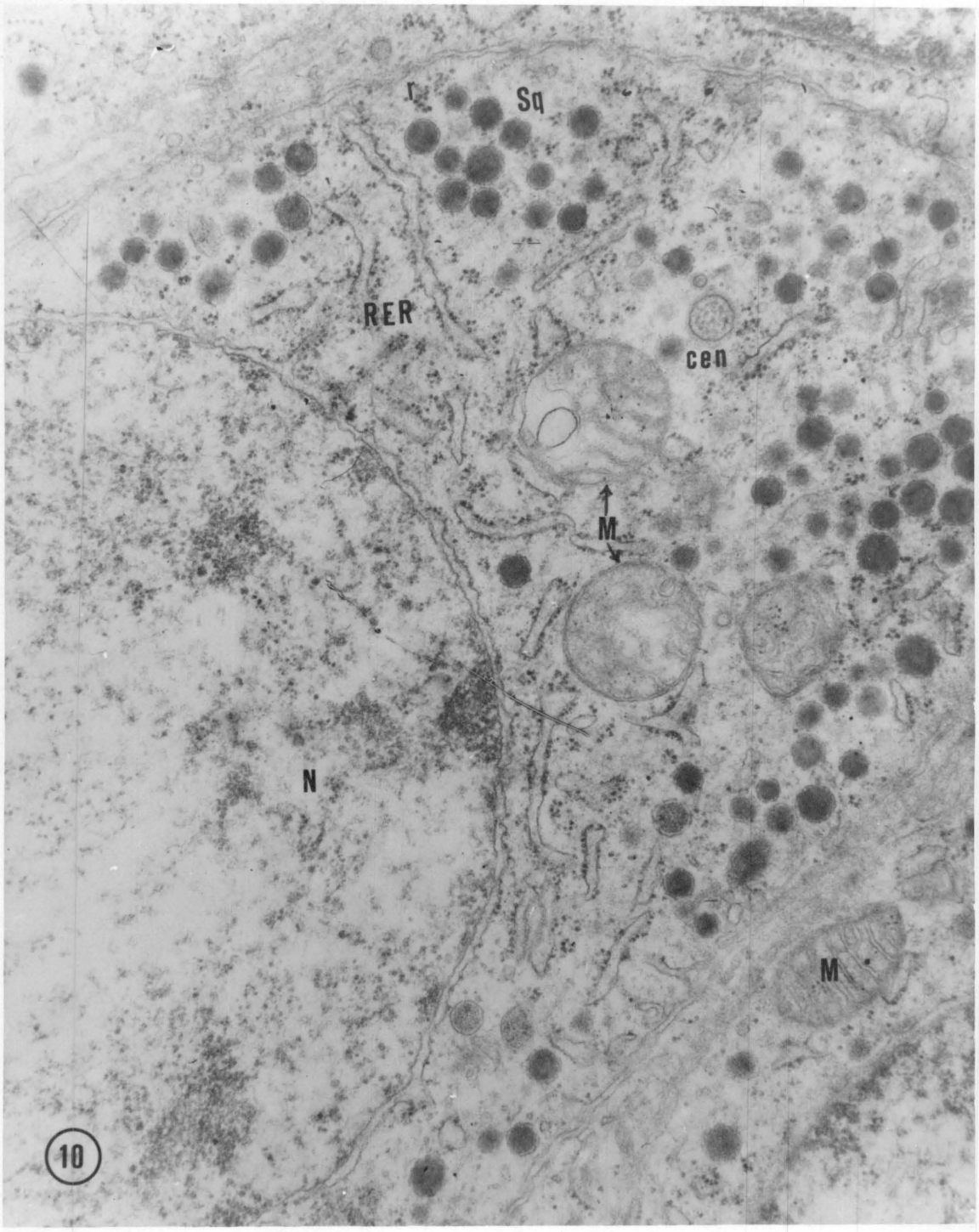
The protein synthetic apparatus including the rough endoplasmic reticulum (RER), Golgi complex and granule (Sg) distribution indicate a moderate cellular activity. The nucleus occupy large portions of the cell.



x25,900

Plate 10: A GT-A cell from LWS pullet A803B.

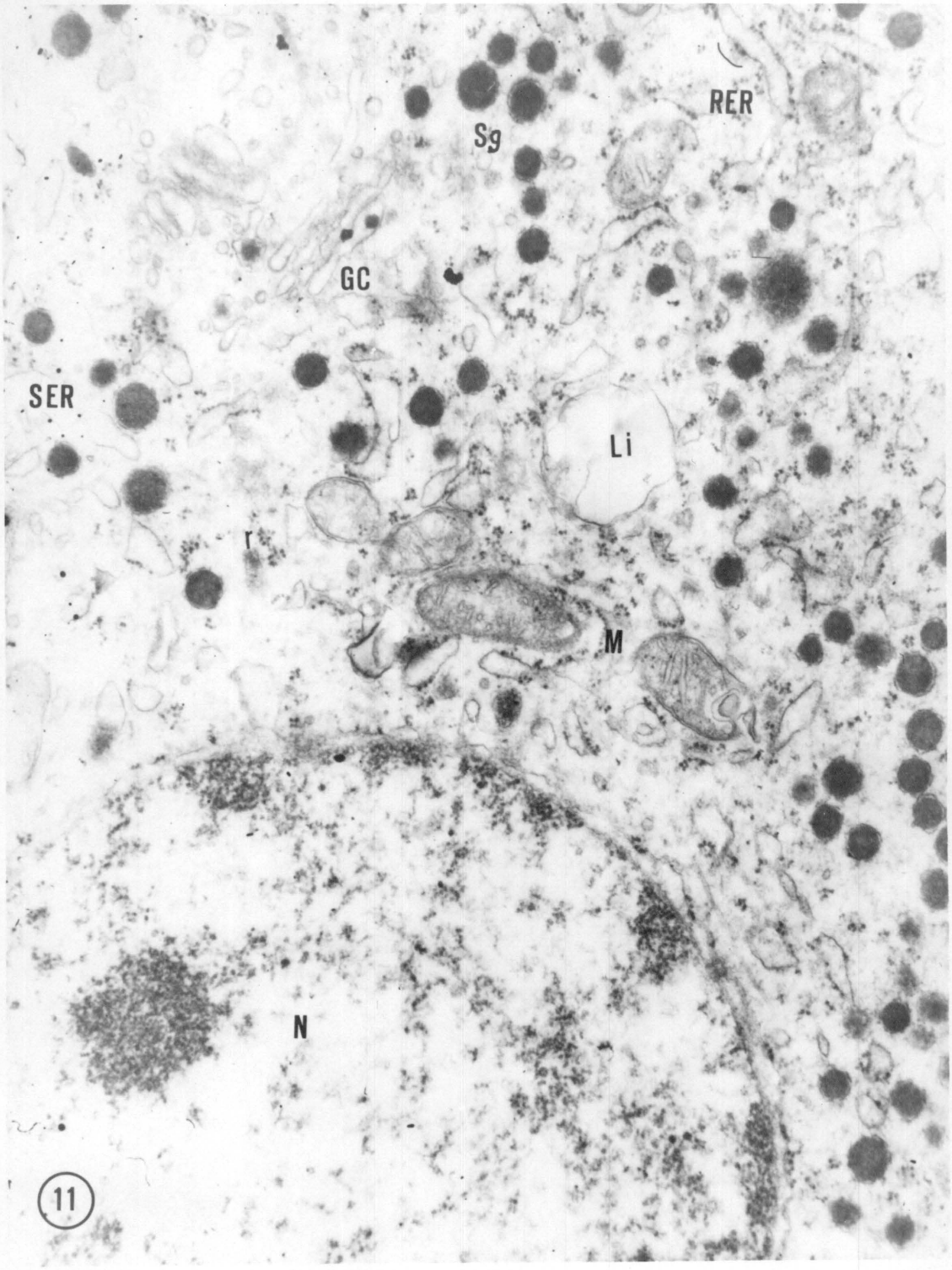
The rough endoplasmic reticulum (RER) cisternae are short and scattered. The Golgi is poorly developed and there are a very few smooth vesicles between the RER and the Golgi. Continuity between the outer leaflet of nuclear envelope and RER is seen (arrow). Also visible is a centriole (cen).



x25,700

Plate 11: A GT cell from LWS pullet A660J.

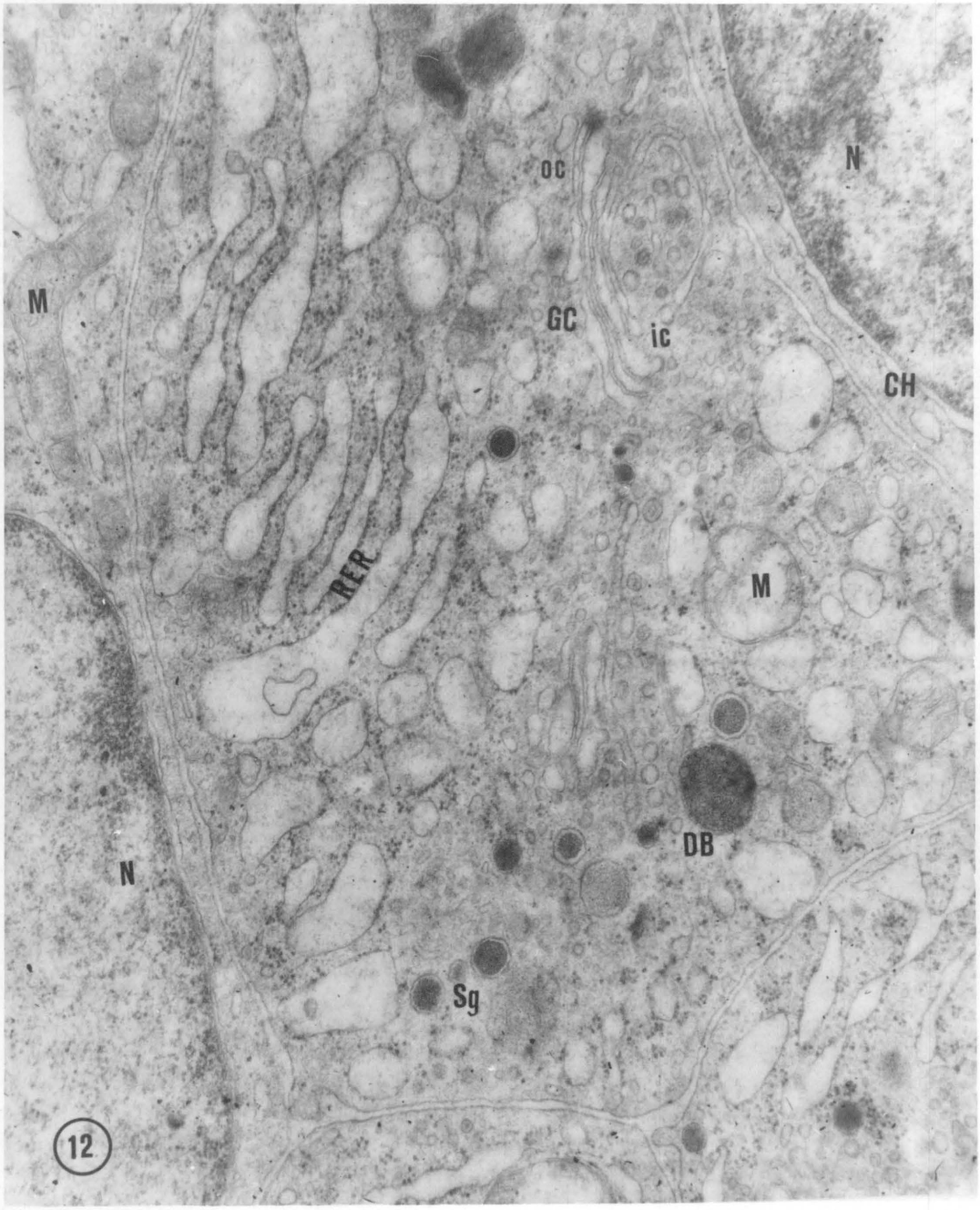
The rough endoplasmic reticulum (RER) cisternae are short and scattered. There are few smooth endoplasmic reticular vesicles (SER) interfacing with the RER. The Golgi complex (GC) is moderately developed with evidence of activity. Free ribosomes (r) are distributed in the cytoplasmic matrix. This cell is probably in a moderate stage of activity.



x24,000

Plate 12: A GT cell from LWS pullet A621F.

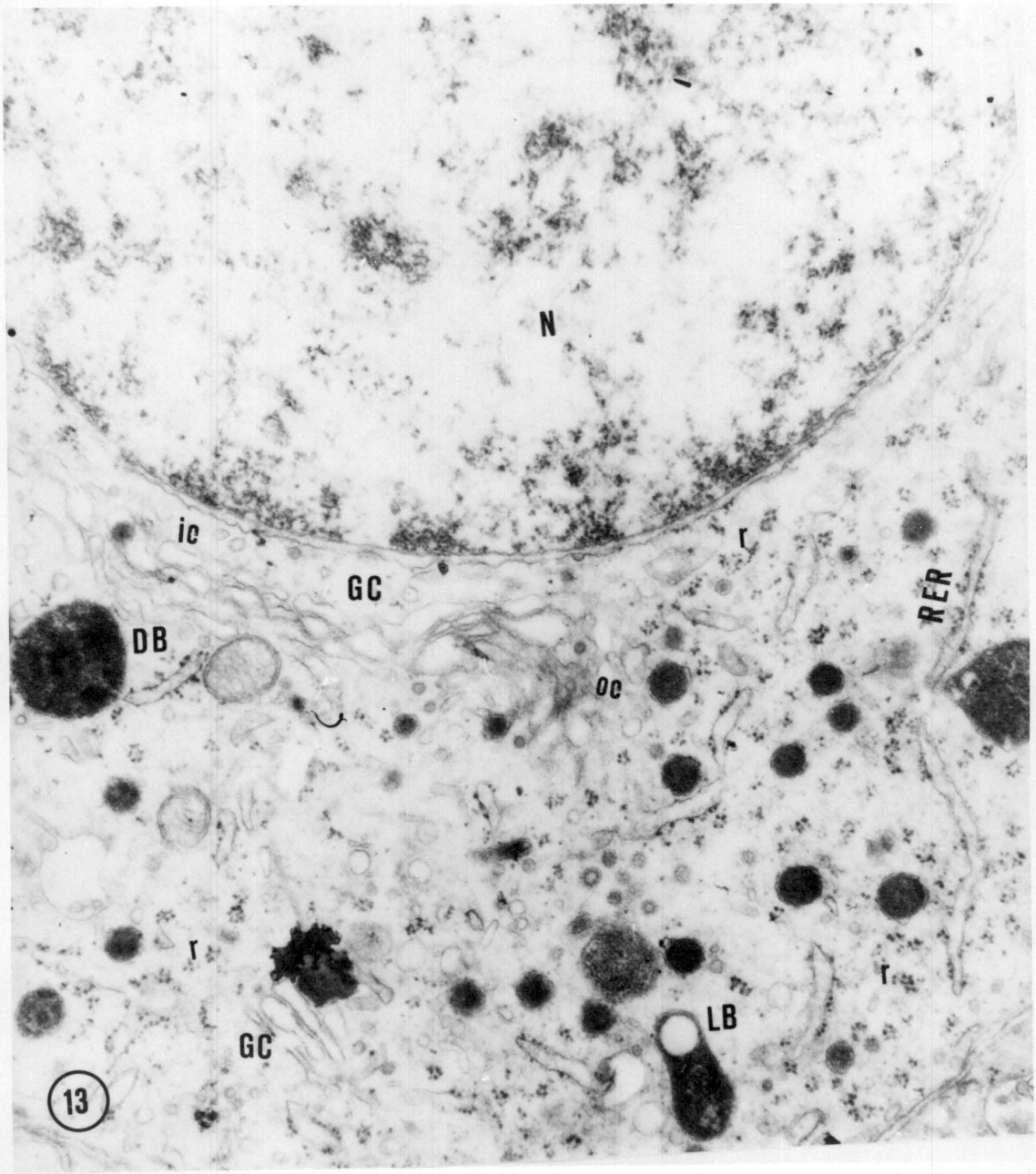
The rough endoplasmic reticulum (RER) cisternae are dilated and the Golgi complex (GC) is large and active. Several polymorphic condensing granules can be seen in the core of the cytoplasm circumscribed by the Golgi cisternae (OC) which are partially collapsed. The secretory granules (Sg) are enclosed in a conspicuous limiting membrane. Lysosomes are seen in the vicinitiy of the Golgi. Although a vacuolated dense body may also be seen near the nucleus, there is no recognizable evidence for crinophagy. The cell appears to be in a very active secretory state.



x50,000

Plate 13: A GT cell from LWS pullet A760J.

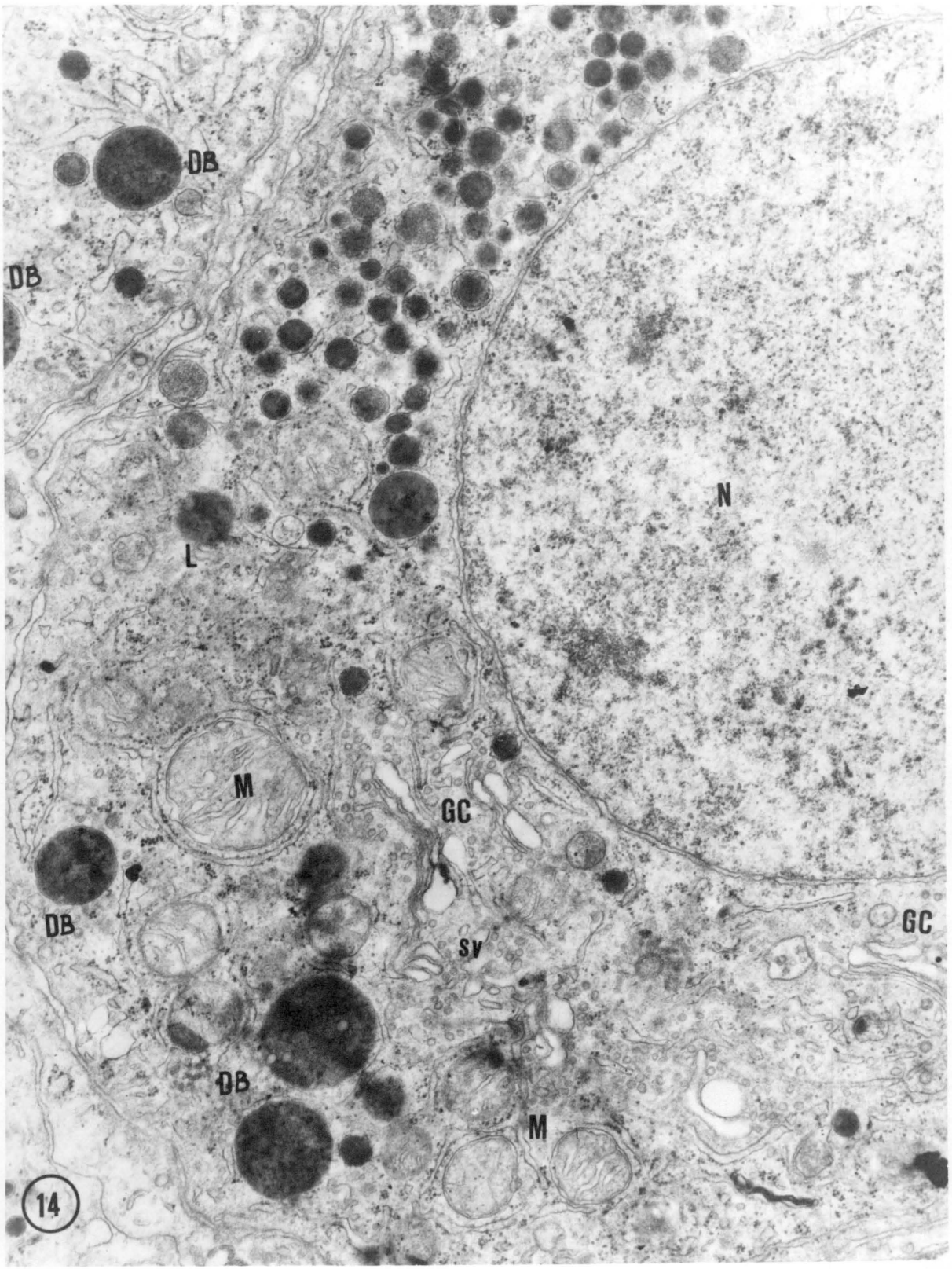
The rough endoplasmic reticulum (RER) cisternae are short and scattered. Ribosomes (r) are free in the cytoplasmic matrix. The Golgi complex (GC) is jaxtanuclear, elaborate and active. The presence of dense bodies (DB) and vacuolated lytic body (LB) indicate autodigestion of secretory product by lysosomal enzyme system.



x24,000

Plate 14: A GT cell from LWS pullet A660D.

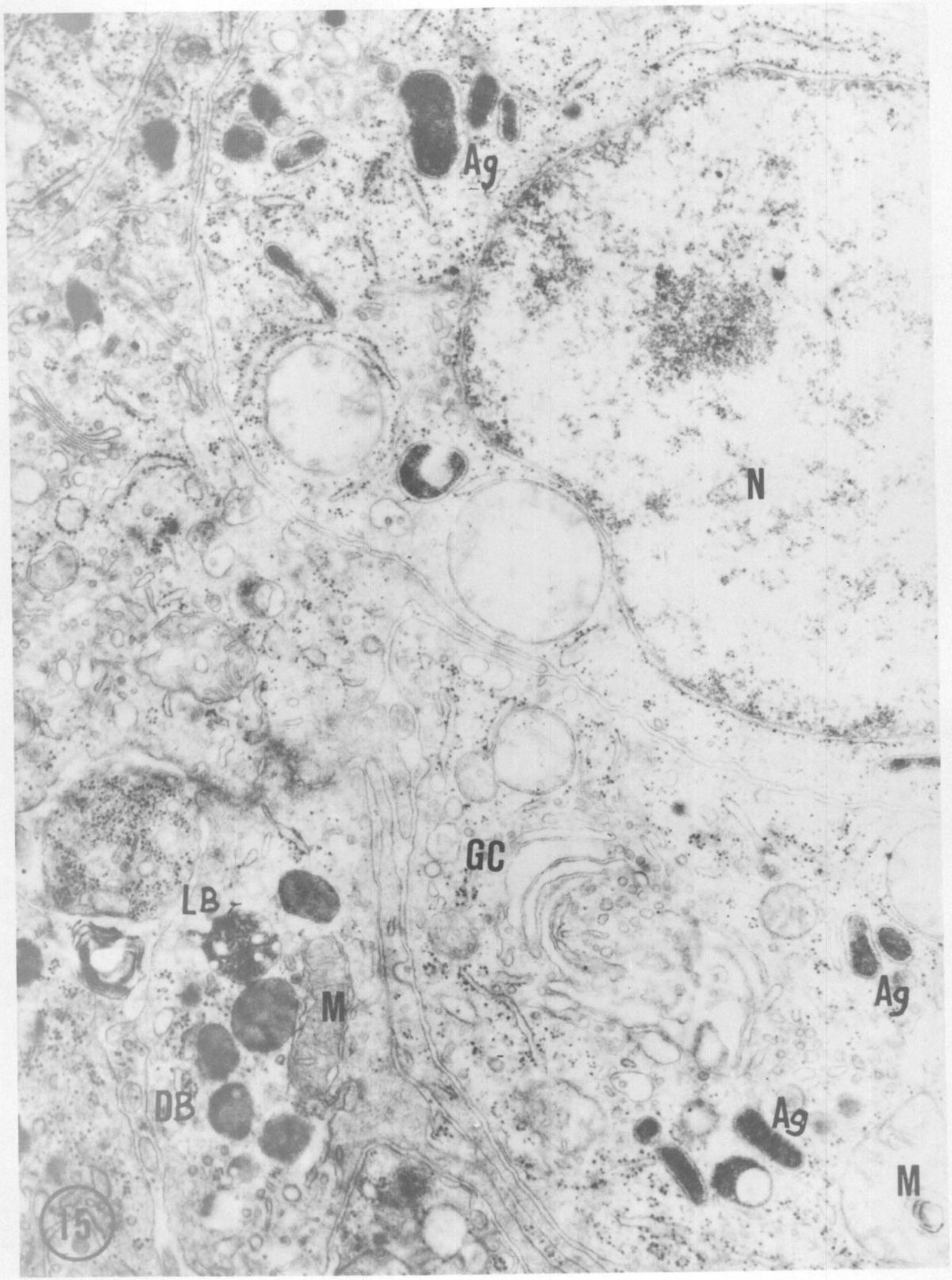
This cell shows evidence of aggregation of small secretory granules into larger units and a subsequent conversion to dense bodies (DB) and lytic bodies (LB). Lysosomes (L) are seen associated with secretory granules. The protein secreting apparatus is active.



x26,600

Plate 15: Two GT cells from LWS pullet A660D.

These cells present the evidence of active protein synthesis and simultaneous crinophagic condition. The aggregating granules, dense bodies (DB) and lytic bodies (LB) are seen along with an active Golgi complex (GC).



x21,000

EXPERIMENT V. FREQUENCY OF CHROMOSOMAL ABNORMALITIES IN EMBRYOS FROM LINES SELECTED FOR HIGH AND LOW JUVENILE BODY WEIGHT

Differences have been noted in the frequency of chromosomal aberrations among diverse populations of chickens. This experiment was designed to survey the incidence of chromosomal abnormalities in growth selected lines, known to have different reproductive parameters.

Materials and Methods

Genetic stocks and management: Data on the frequency of chromosomal aberrations were obtained during each of two years. Populations used the first year consisted of random samples of pullets from the S_{16} generation of the high weight (HWS) and low weight (LWS) lines, and second generation dwarf backcross to the HWS and LWS lines. The birds used the second year were the S_{17} generation parents selected to reproduce the S_{18} generation. The age of the pullets at the time eggs were gathered for karyological investigation were 36 and 51 weeks in the first and second years, respectively. Methods of rearing, management and photoperiod treatment during growing and laying periods were described in Experiment I and II.

Prior to artifical insemination, 187 eggs obtained from virgin females of these populations were incubated to determine the incidence of parthenogenesis. On the tenth day of incubation these eggs were broken and examined macroscopically for the development of membranes, blood or embryos (Olsen, 1966).

Four to five females were randomly assigned to each male within their respective population for artifical insemination. Pedigreed eggs were collected from the second to the ninth day post-insemination and stored at 10 to 15°C. Cold storage of eggs prior to incubation did not influence the frequency of chromosomal aberrations (Bloom, 1972).

Procedure: The techniques for chromosome preparations were similar to those described by Bloom et al. (1972). Briefly, eggs were incubated for 96 hours at 37.8°C in a Petersime model S-6 PH incubator. They were then removed from the incubator, a pinpoint hole made in the center of the broad end, and 0.2 ml of 0.05% aqueous colchicine solution injected. The eggs were then returned to the incubator for 45 to 60 minutes after which they were removed, broken and the contents poured into a petri dish. The embryos were carefully separated from the yolk sac, taking care that allantoic sac remained intact, and placed in a vial containing 2 to 3 ml of distilled water. After 15 minutes, the water was removed with a pasteur pipette and an approximately equal quanitity of 1:3 acetic ethanol was added to the vial. The vials were left at room temperature for 1 to 2 hours before being stored at 5 to

10°C for fixation. Satisfactory chromosome preparations could be made after a fixation period of 24 hours. For prolonged storage, the vials were transferred to the freezing chamber of a refrigerator.

Metaphase chromosome preparations were made by removing a small piece of the allantoic sac with a fine glass needle. The tissue was then placed in 1 to 2 drops of 45% acetic acid for about 5 to 10 minutes and squashed by placing a think cover slip over it by applying thumb pressure for about 15 seconds. Only unstained squashed preparations were examined by phase contrast microscopy, and a minimum of 10 metaphases were examined per embryo.

Phenotypic abnormalities were considered to be those with gross anatomical malformations, small embryos, and a poorly developed extraembryonic vascular system. Chromosomal aberrations represented variation in the chromosome complement from the standard diploid (2N=78).

Photomicrography: Unstained squashes were examined by phase contrast microscopy, and metaphase chromosomes were located using a Pv 25 objective. The clear preparations were photographed with a Lieca camera fitted to a Leitz microscope, using a Pv 90/1.15 apochromatic oil immersion objective lens and a green filter on Kodak high contrast copy film. Cells with clear chromosome spreads were selected on the basis of an apparent integrity of the cell. The film was developed in D-19 developer, printed on Kodak Kadabromide F-3 paper, and developed in Dektol developer. From the enlargements, individual chromosomes were cut and aligned on

the basis of relative length and centromere position. Karyotypes of birds reported in the Chromosome Atlas (Benirschke and Hsu, 1971) were used as a guide in the construction of idiograms.

Analysis: Pullets had been maintained under 2 lighting regimes--constant 14L:10D, and 14L:10D with a 3% weekly increase beginning at 19 weeks of age. Correlated chi-square analyses (Jensen et al., 1968) showed no significant differences between light treatments within lines (see Appendix Table 13). Accordingly, data from the 2 light treatments were pooled for subsequent analyses. Since 2 hypotheses were tested simultaneously on the same categorical data, the value of τ was set at 2 and tested at the 0.05 level of significance. For other comparisons, the Chi-square test of independence was used to test for differences among populations.

Results and Discussion

Preliminary examinations revealed no incidence of parthenogenetic development in these populations.

Intraline comparisons between dwarf and normal pullets: Phenotypic effects and chromosomal aberrations in 4-day embryos from S_{16} generation HWS and LWS lines and their dwarf backcrosses are presented in Table 37. There were no significant differences in phenotypic and chromosomal abnormalities between dwarf and normal pullets within either line. Since there was no significant difference between dwarf and normal pullets in the incidence of these abnormalities, data were pooled for further comparisons between lines.

Table 37. Phenotypic and chromosomal abnormalities from dwarf and normal pullets in lines selected for high and low body weight, S_{16} generation[1]

Line	Genotype	Total embryos	Phenotypic abnormal		Chromosomal aberrant	
			No.	%	No.	%
HWS	DW	46	8	17.4	5	10.9
	dw	30	4	13.3	4	13.3
LWS	DW	89	8	9.0	5	5.6
	dw	42	4	9.5	2	4.8

[1]Sires and dams maintained in a 14:10 LD photoperiod.

Interline comparisons--Chromosomal aberrations: Presented in Table 38 are data for the frequency of chromosomal aberrations in the S_{16} and S_{17} generations. Analyses revealed significantly higher aberration rates in the HWS than in the LWS lines. The results were consistent in both generations and the Chi-square value for data pooled over the two generations was also highly significant. The overall frequency of chromosomal aberrations of 14.5% in HWS line was similar to the 12.7% obtained by Miller et al. (1971) and the 11.0% reported by Fechheimer et al. (1972) for broiler populations.

Types and frequencies of chromosomal aberration: Summarized in Table 39 are reports on frequencies of chromosomal aberrations in different genetic stocks. The frequency of aberrations ranged from 1.4 to 14.5%. It is significant that rapid growing broiler stocks exhibited a higher incidence of chromosomal abnormalities than the unselected control lines or those selected for slow growth. Egg-type stocks, Jungle fowl, and Arancanas also had fewer aberrant embryos than the broiler populations.

Types and frequencies of chromosomal aberrations in the HWS and LWS lines are presented by generations in Table 40, with karyotypes of some of the normal and aberrant metaphase preparations shown in Plates 16 through 25. The frequency of euploid and aneuploid abnormalities were similar in both lines. Miller et al. (1971) reported signficantly more defects of the euploid series in the broiler stocks, while the defects of aneuploid series predominated in an egg-type line. Only one embryo with pure haploid cell

Table 38. Number and percentage of chromosomally normal and aberrant embryos in the high and low weight lines, S_{16} and S_{17} generations

Embryo karyotype		Line HWS	Line LWS	Total	X^2
		S_{16} generation			
Normal	N	116	194	310	
	%	85.9	94.2		
Aberrant	N	19	12	31	
	%	14.1	5.8		
Total	N	135	206	341	6.69**
		S_{17} generation			
Normal	N	37	48	85	
	%	84.1	92.3		
Aberrant	N	7	4		
	%	15.9	7.7	11	
Total	N	44	52	96	4.08*
		Generations pooled			
Normal	N	153	242	395	
	%	85.5	93.8		
Aberrant	N	26	16	42	
	%	14.5	6.2		
Total	N	179	258	437	8.43**

**$P \leq .01$

*$P \leq .05$

Table 39. Frequency of chromosomal abnormalities in different genetic stocks

Genetic stock	Frequency of Ch. aberrations %	Reference
Broiler (Selected for rapid growth)	12.7	Miller et al., 1971
Broiler (Selected for rapid growth)	11.9	Fechheimer et al., 1972
Broiler (Selected for rapid growth)	14.5	Reddy, Present study
Broiler (Unselected control)	3.2	Miller et al., 1971
Broiler strain	4.9	Duber et al., 1973
Broiler (Selected for slow growth)	6.2	Reddy, Present study
Leghorn (Commercial)	1.5	Fechheimer et al., 1972
Leghorn (Commercial)	2.8	Duber et al., 1973
Leghorn (Commercial)	1.4	Lodge et al., 1973
Leghorn (Commercial	3.4	Bloom, 1974
Inbred line (Egg type)	5.4	Bloom, 1974
Araucana	2.7	Bloom, 1972
Jungle Fowl	2.8	Bloom, 1972

Table 40. Types and frequencies of chromosomal abnormalities in HWS and LWS lines from S_{16} and S_{17} generations

Chromosome complement	Line					
	HWS			LWS		
	S_{16}	S_{17}	Total	S_{16}	S_{17}	Total
Euploid						
Haploid	1	-	1	-	-	-
Haploid mosaics						
A/2A	13	2	15	8	2	10
A/2A/3A	2	2	4	1	1	2
Diploid	115	37	152	195	48	243
% Euploid (other than diploid)/abnormal	84.2	57.1	76.9	75.0	75.0	75.0
Aneuploid						
Monosomics						
Ch. 1	1	1	2	1	-	1
3	1	-	1	1	-	1
4/6	-	1	1	-	-	-
Monosomic/trisomic mosiacs						
Ch. 2	-	1	1	1	-	1
Trisomics						
Ch. 3	1	-	1	1	-	1
Ch. 4	-	1	1	-	-	-
% aneuploid/abnormal	15.8	42.9	23.1	25.0	25.0	25.0
Total no. of embryos	135	44	179	206	52	258
No. with abnormal Chr.	19	7	26	12	4	16
Aberration rate	14.1	15.9	14.5	5.8	7.7	6.2

line (A-Z) was noticed in this study. The embryo was small at 4D.I with poorly developed extraembryonic vascular system.

The frequency of euploid mosaic embryos over two generations was 73.8% of which haploid/diploid mosaics predominated. This is consistent with the observations of Miller et al. (1971) and Fechheimer et al. (1972) who reported euploid mosaic frequencies of 76.3 and 77.4%, respectively among the aberrant embryos. Among the 25 haploid/diploid mosaics, 16 had a sex-chromosome constitution of Z/ZZ and 9 were Z/ZW. Although 6 cases of haploid/diploid/triploid mosaics were detected, no true triploid was observed. Miller et al. (1971) and Jaap and Fechheimer (1974) reported that the frequency of triploidy was highest in eggs from young dams early in the first laying cycle. They indicated that a hormonal imbalance might be implicated in the production of triploids. Hence, there may be an indirect relationship betweeh EODES and the frequency of triploidy.

Trisomy 3, 4, and monosomy 1, 3 noted here have also been reported by Fechheimer et al. (1968) and Bloom (1970 a,b). Trisomy 4 was a male and trisomy 3 was a female embryo.

Etiology and routes of chromosomal aberrations: The large number of euploid mosaic embryos which contributed to the high frequency of chromosomal aberrations may be unique to the growth characteristics of these populations. Lepore et al. (1963) provided evidence for greater utilization efficiency by HW embryos for certain amino acids. Further, Fofanova (1964) demonstrated the

presence of supernumerary nuclei and spermatozoa in meroblastic ova and showed that these nuclei were formed in eggs with a high yolk content. Presumably, these nuclei are concerned with yolk absorption. It may be hypothesized that greater need for nutrients by fast growing HW embryos may induce polyspermy and development of more supernumerary nuclei. Although supernumerary nuclei usually degenerate, one or more of them may undergo subsequent morphogenetic modification and contribute to the genome of the organism. A high concentration of spermatozoa at the site of fertilization was associated with high embryonic mortality (Lorenz et al., 1969).

Haploids: Although parthenogenesis may provide an explanation for the origin of haploid embryos, this is unlikely because the preliminary screening indicated no incidence of parthenogenesis in these stocks. Therefore, the haploid embryo observed in this study may be due either to gynogenesis or androgenesis. Moreover, parthenogenesis in turkeys and occasionally in meat-type chickens, give rise to diploid male embryos (Olsen, 1966). Fechheimer (1968), and Bloom (1969, 1974) observed that all pure haploids were of A-Z chromosome complement. Pure A-W haploids may have a lower viability and to date have not been reported. The haploid embryo noted here was from a mating of full sibs, however, we cannot at the present time establish any association between inbreeding and haploid development. Bloom (1972) reported relatively higher incidences of chromosomal abnormalities in inbreds than in randombred egg-type

chickens. Earlier he (Bloom, 1969) observed that two haploid embryos were from the same sire that was mated to different dams, and indicated that haploidy may be under genetic control.

Euploid mosaics: Bloom (1969, 1970) offered cytological evidence that haploid mosaics may be due to cell fusion. Such a phenomenon, however, would not explain the occurance of Z/ZW mosaics. Polyspermy has been demonstrated as a normal occurance in chickens (Fofanova 1964), since the structural state of embryonic disks for normal development depends directly on the presence in the disk of an optimum number of supernumerary sperm nuclei. Development of a haploid cell line may be due to morphogenetic changes and further proliferation of one or more of supernumerary sperm nuclei. A low frequency of triploid cells (8-10%) in haploid/diploid/triploid mosaics embryos suggests that these cells may be due to the nuclear fusions discussed by Bloom (1969), rather than fertilization by diploid ova. Pure triploid embryos would be primarily due to fertilization of diploid ovum by haploid sperm or vice versa.

Aneuploids: Pure trisomic or monosomic embryos result from meiotic non-disjunction or anaphase lag in either parent. Such an event would cause disomic and nullisomic gametes. At syngamy with a normal gamete either a trisomy or a monosomy would result. Double monosomy for 4/6 chromosomes is best explained by the simultaneous occurance of non-disjunction for two chromosomes. Since a diploid cell line also appeared in this embryo, aberrant mitosis must have occured in early cleavage divisions. The trisomy-monosomy mosaic

involving chromosome 2 had not only these 2 cell lines but also a diploid cell line. This could only be possible if a non-disjunctional event occured at mitosis during early cleavage. Had a non-disjunctional event occured at the first mitotic division we would have found only monosomic and trisomic cell lines. Similar explanations were provided by Fechheimer et al. (1968), Miller et al. (1971) and Bloom (1972, 1974).

Summary

The presentation discussed above provides a tentative evidence that selection for fast growth may result in high frequency of chromosomal defects, due to anomolies in meiosis, maturation of ova, ovulation, fertilization and early cleavage. The higher frequency of chromosomal defects in the HWS than in the LWS line is consistent with the suggested congruent association between growth rate and aberration rate reported by Miller et al. (1971) and Fechheimer et al. (1972). Additional studies on the types and frequencies of chromosomal aberrations in these lines should reveal interesting relationships between genetic and physiological influences.

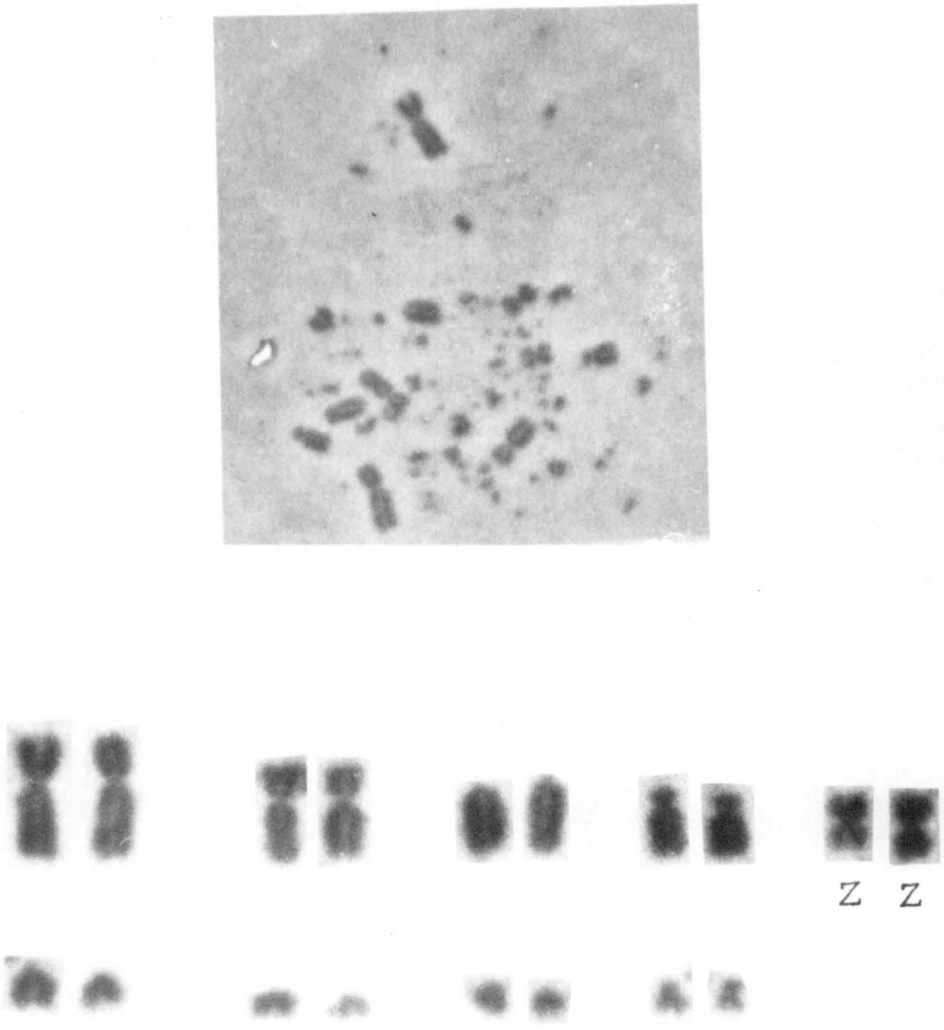


Plate 16. Karyotype of normal male (2A:ZZ)

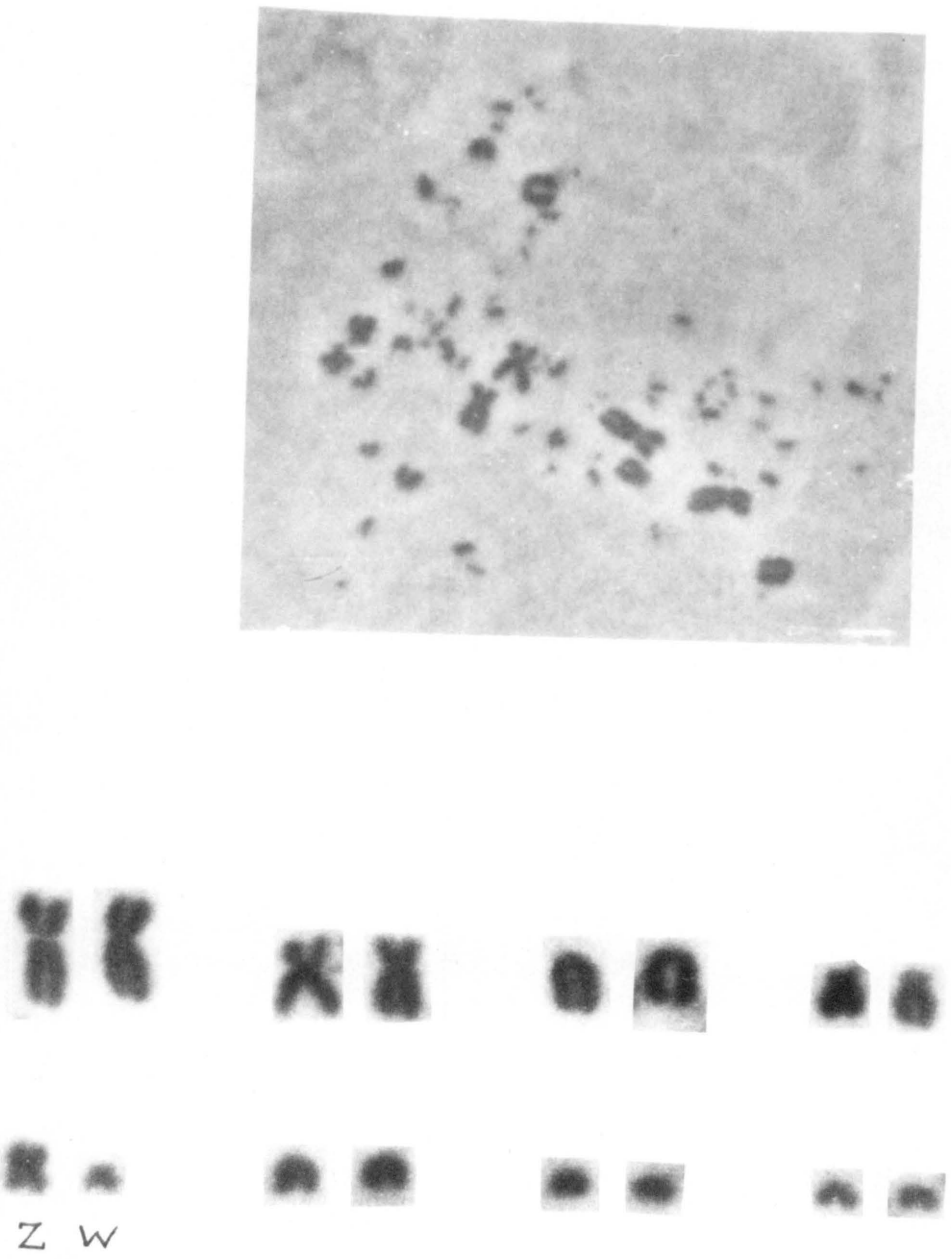


Plate 17. Karyotype of normal female (2A:ZW)

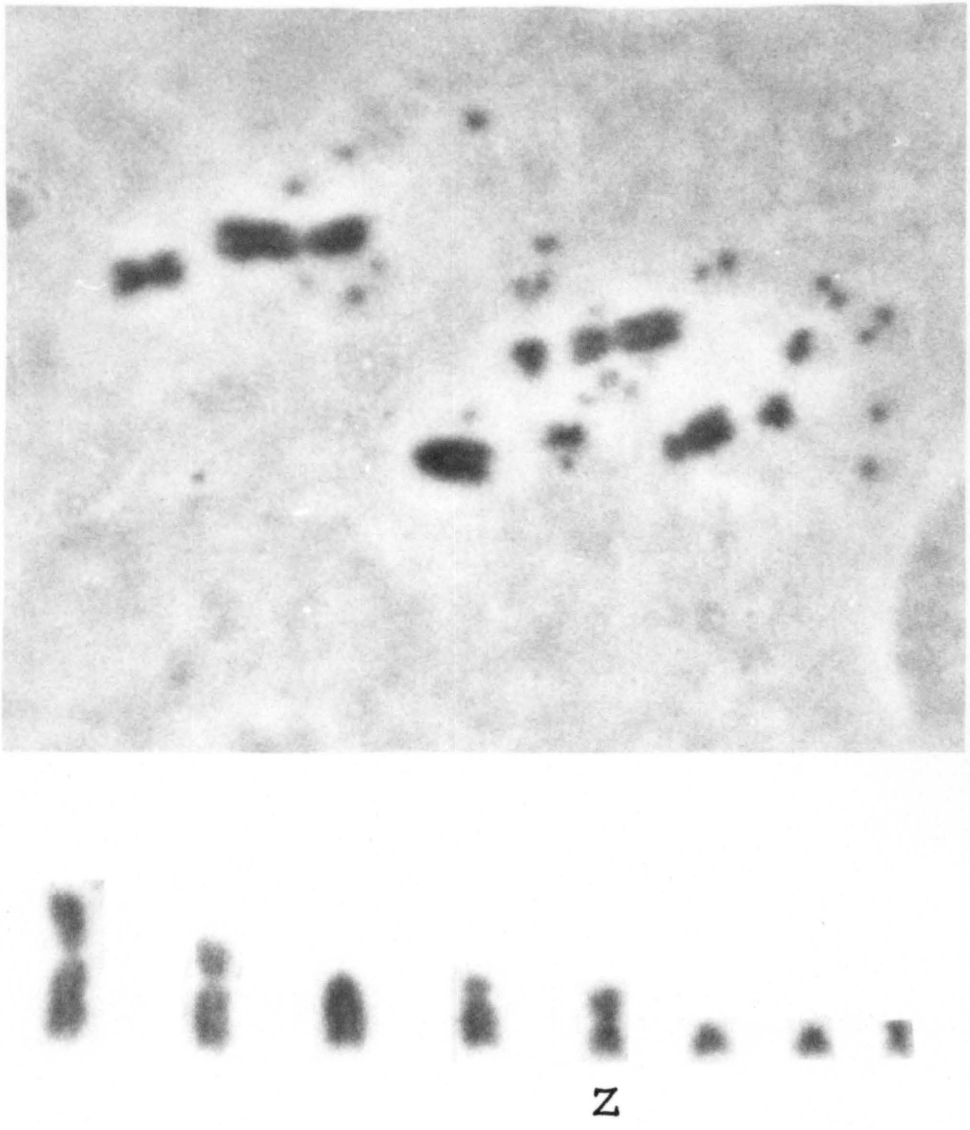


Plate 18. Karyotype of an haploid cell (A:Z)

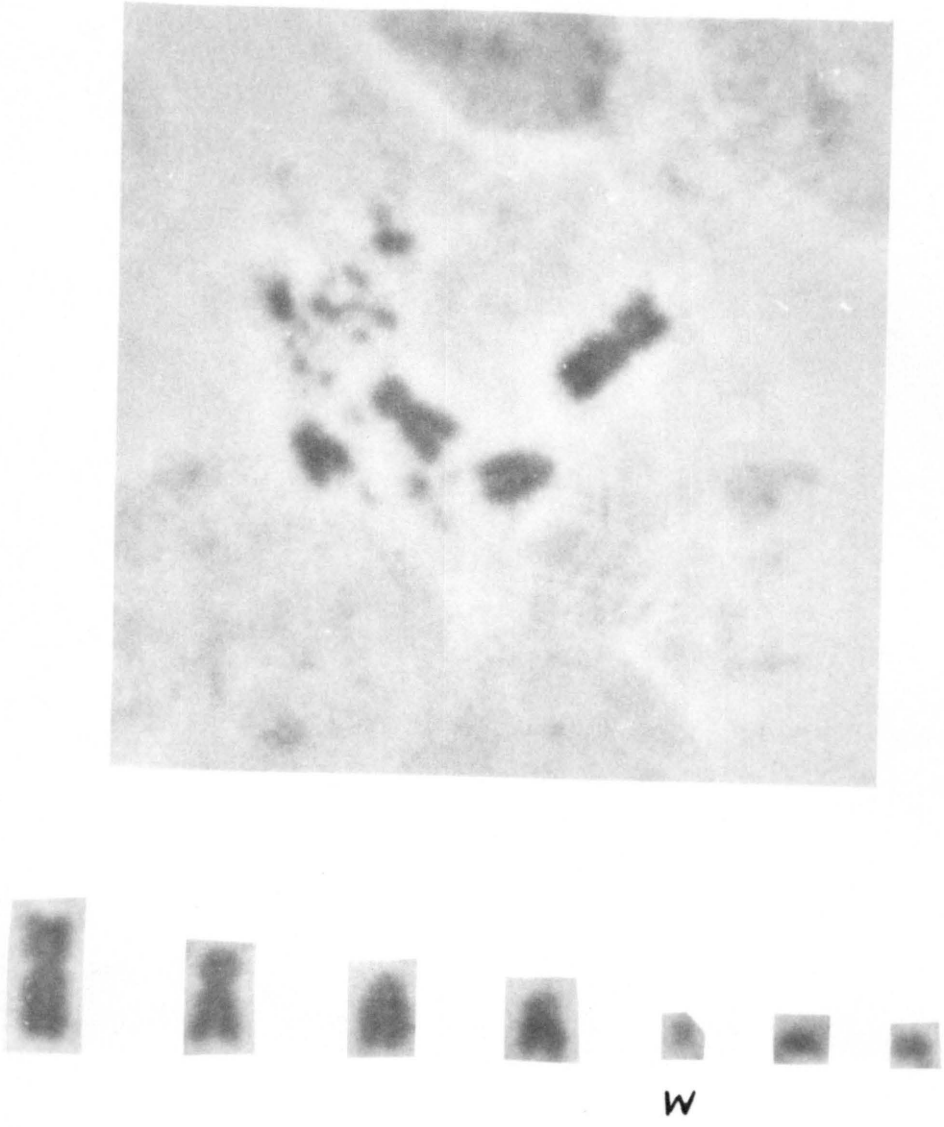


Plate 19. Karyotype of an haploid cell (A:W)

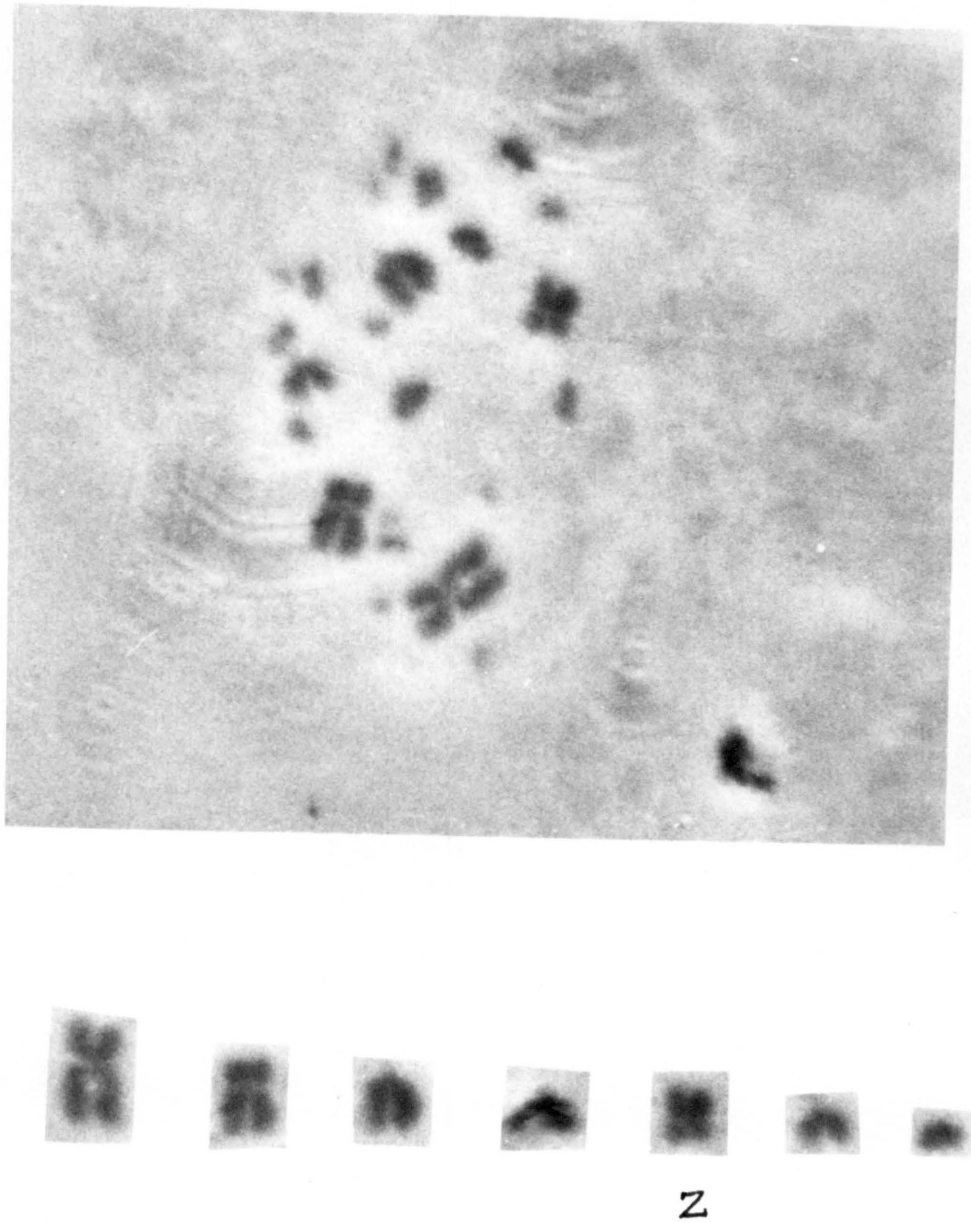


Plate 20. Karyotype of an haploid embryo (A:Z)

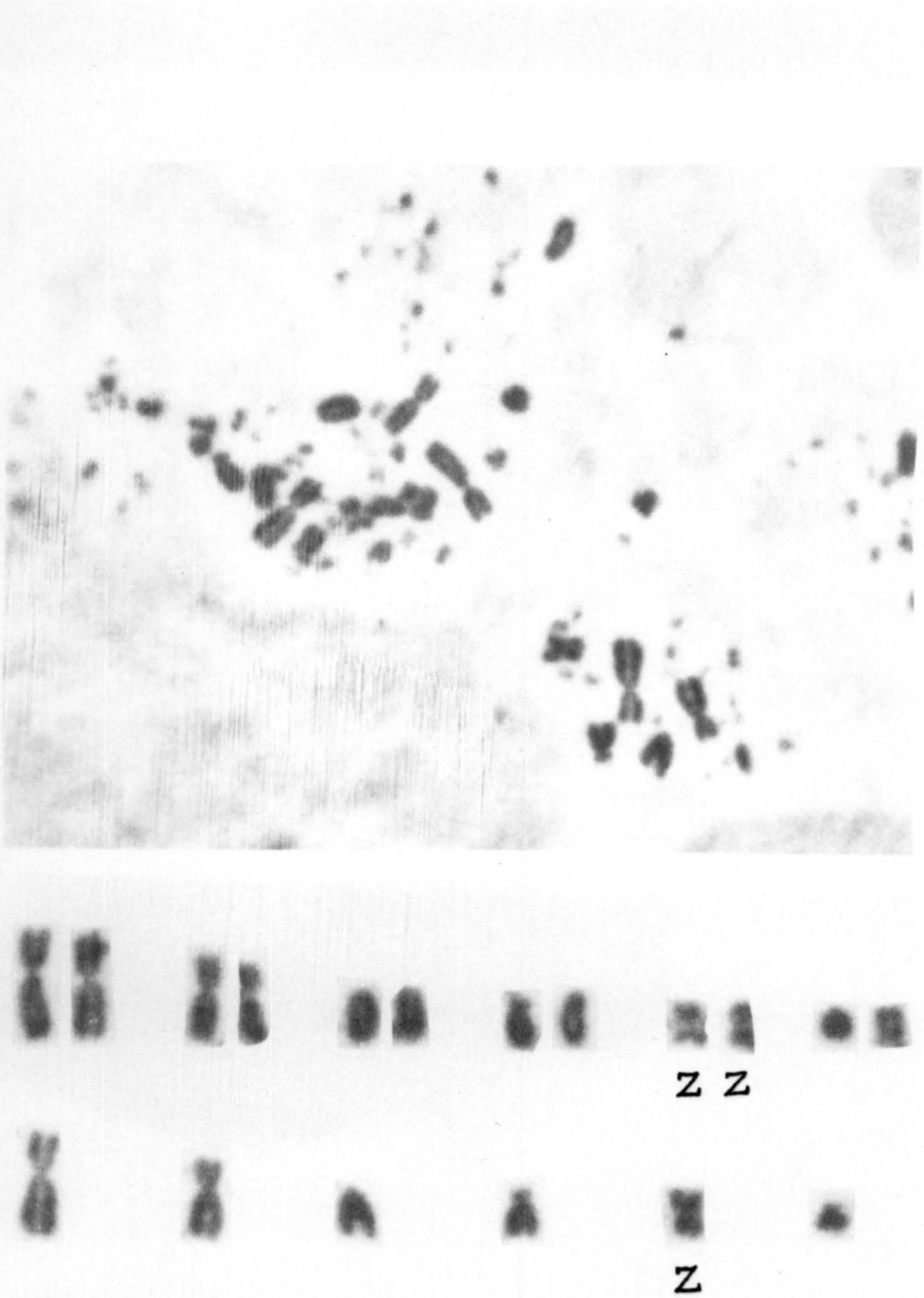
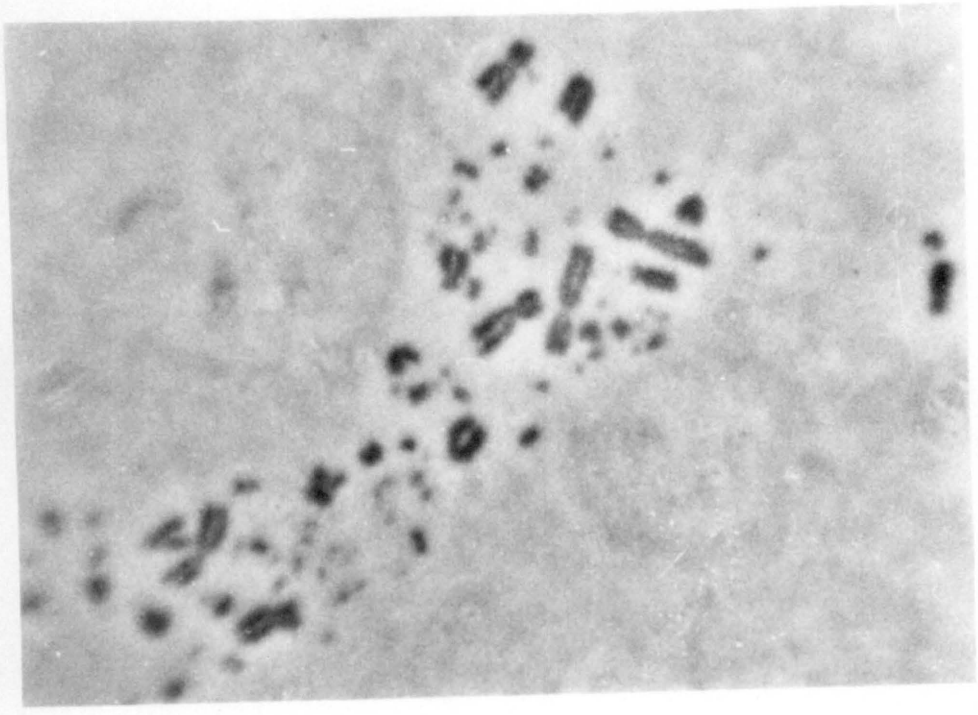


Plate 21. Karyotype of an haploid/diploid mosaic embryo (2A:ZZ/A:Z)



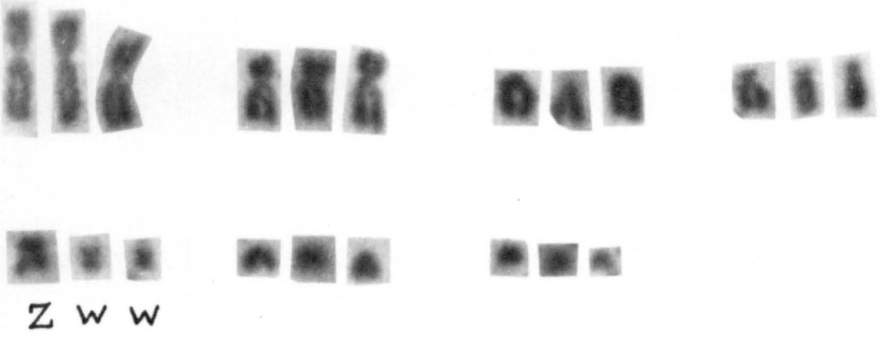


Plate 22. Karyotype of a triploid cell from an haploid/diploid/ triploid mosaic embryo.

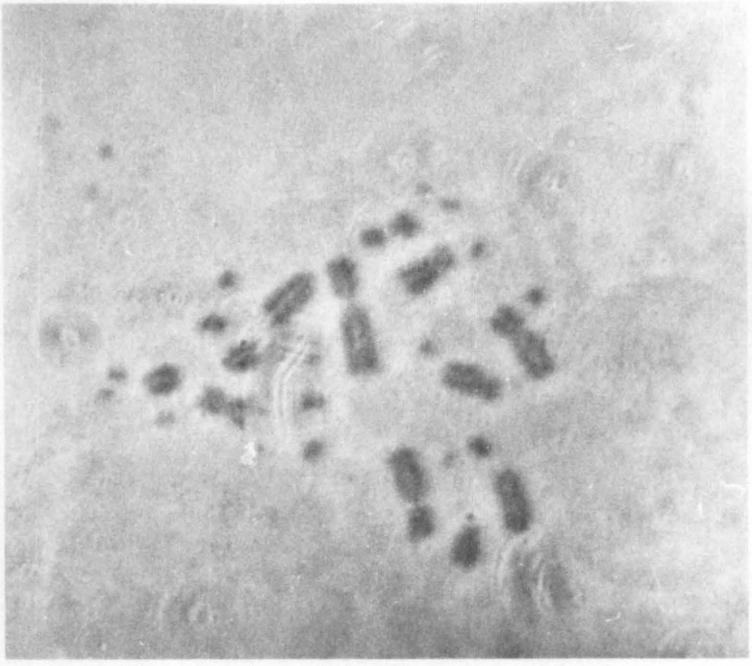


Plate 23. Karyotype of monosomic-1 embryo

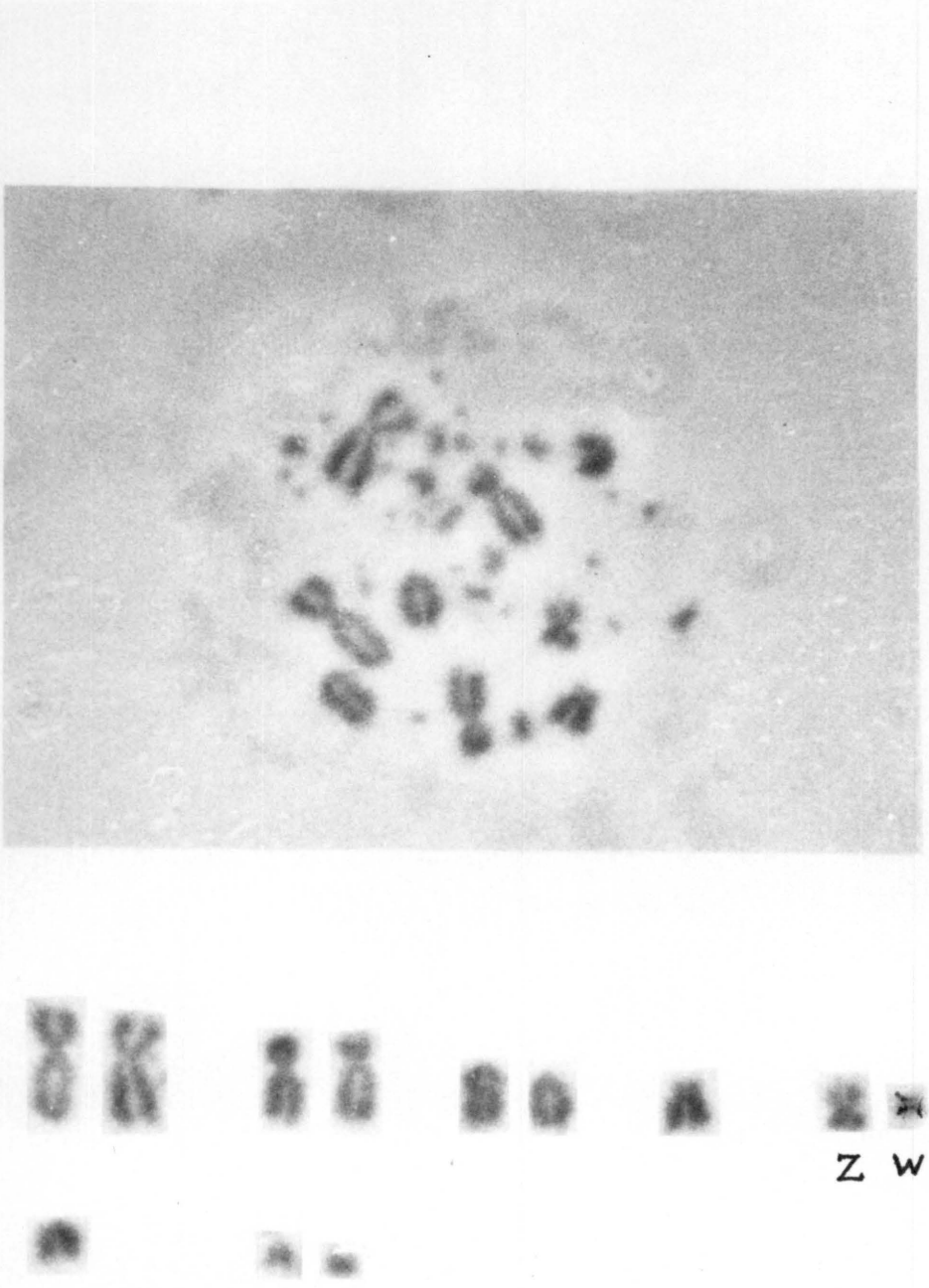


Plate 24. Karyotype of monosomic 4/6 embryo

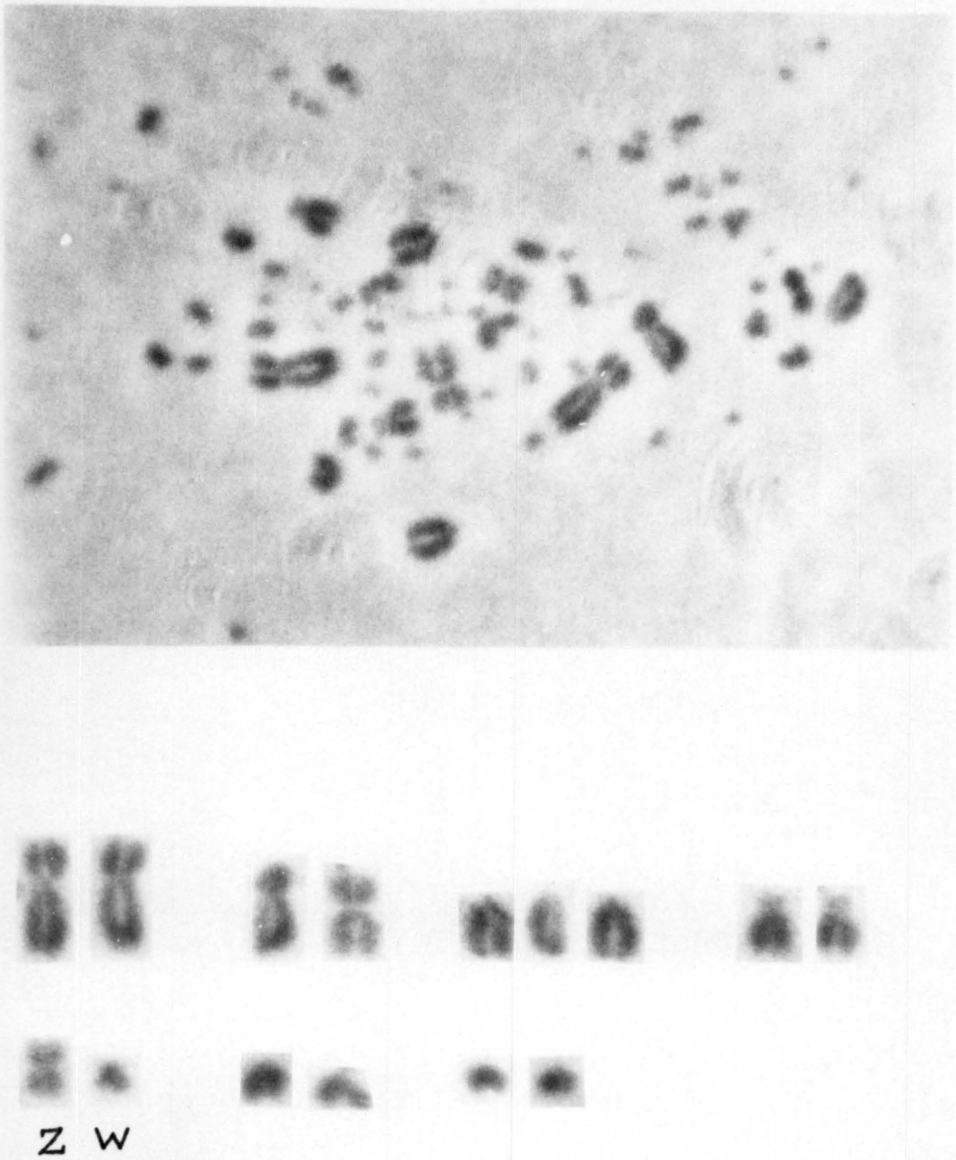


Plate 25. Karyotype of trisomic-3 embryo

GENERAL SYNTHESIS

This dissertation involved five interrelated experiments designed to investigate genetic and physiological aspects of reproduction in the lines of chickens that had undergone bidirectional selection for juvenile growth.

I. Arhythmic ovulation patterns and defective egg production were associated with growth characteristics. Pullets with high growth potential produced significantly more defective eggs at each of the physiological ages studied. The patterns of defective egg production, however, were different in lines selected for high and low body weight at eight weeks of age. Delaying sexual maturity by feed restriction did not appreciably change the pattern of defective egg production indicating that specific genetic factors are expressed at a particular physiological rather than chronological age. Selection for growth rate (protein anabolism) had a correlated effect on yolk synthesis as evidenced by the number of developing, ovulated, and ruptured follicles plus the incidence of internal laying. The higher ovulation rate in the HW lines may be attributed to a combined effect of greater yolk synthesis, higher levels of gonadotrophins, and lower neural thresholds for sensitivity to these hormones. Relaxing selection for growth rate had a correlated effect on yolk synthesis. The model hypothesizing the relationship between growth rate and yolk synthesis was in congruence with the egg production patterns observed in these lines. Although the frequency of broken eggs was significantly higher in

the HW lines, factors other than those associated with body size appear to be involved. Increasing the photoperiod beyond 14 hours did not have any consistent effect on either growth or reproductive traits.

II. The genetic parameters for egg production patterns and the relationships of such patterns with other reproductive and growth traits suggest approaches that may be used to increase hatching egg production in broiler mothers. The most pertinent of these was the heritability estimate of the composit trait, total defective eggs, which was moderate to high. The average heritability estimates for normal egg and yolk production were similar suggesting common genetic factors controlling these two traits. The frequency of defective eggs was largely independent of the environmental manipulations that modified age at sexual maturity and controlled growth rate reiterating that a genetic approach is essential in correcting the problem of defective egg production. There was evidence of genotype-environment interactions for the relationship between age at maturity and production of normal eggs. Knowledge of the negative association between normal and defective egg production should have practical application.

III. The effects of the dw gene on growth and reproduction were largely influenced by the background genotype of the testing populations. In general the depressive effect of the dwarfing gene on growth rate was more severe in the LW than in the HW line with the effect on

protein anabolism being larger than that for skeletal growth. Dwarfism consistently delayed sexual maturity in the LW line, whereas in the HW line the dwarfs matured earlier as the proportion of selected inheritence increased with repeated backcrossing. This experiment supported the hypothesis that the dw gene has a correlated depressive effect on lipoprotein anabolism which results in a reduction in yolk synthesis and number of growing follicles. These factors had a synchronizing effect on ovarian and oviducal functions. Thus, although the HW dwarfs produced fewer yolks, they were more efficient in incorporating them into normal eggs than were their normal sisters. Within the LW line, however, the dwarfs were reproductively inferior to their normal sisters. This differential response among the lines suggests that while the dw gene may be deleterious in some populations it may be beneficial in others where there is excessive yolk synthesis and arhythmic ovulation patterns. An optimum body weight seems to be a factor in the initiation and efficient production of normal eggs.

IV. Ultrastructural studies of adenohypophyseal gonadotrophs was undertaken to determine whether or not selection for growth rate influenced the synthetic and secretory potential of these cells. Qualitative analysis of gonadotrophic cell activity, as evidenced by the extent and complexity of protein synthetic apparatus and granule distribution, revealed higher synthetic activity in the HW than in the LW lines at various physiological ages. Although no significant differences were found among lines for the frequency of pullets

showing evidence of intracellular digestion of secretory products, the condition was more severe in pullets from the HW than LW lines. This suggested that selection for growth rate had a correlated effect on the protenaceous secretions from certain glands.

V. A preliminary survey of the incidence of chromosomal abnormalities in embryos from the HW and the LW lines revealed a significantly higher frequency of aberrations in the line with superior growth potential. This was primarily due to an increased frequency of euploid mosaics. It was hypothesized that the greater need for nutrients by fast growing embryos induce polyspermy and the development of supernumerary muclei which may induce mosaicism.

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APPENDIX

Experiment I	Tables 1 to 7
Experiment II	Table 8
Experiment III	Tables 9 to 12
Experiment IV	Cytological procedures
Experiment V	Table 13

Appendix Table 1. Means and standard errors of various measures of female reproduction by lines and photoperiods, for the laying of 40 days from age at first egg, S_{16} generation (Experiement I)

Trait	HWS		LWS	
	14L:10D	14L:10D(3%Δ)	14L:10D	14L:10D(3%Δ)
Age at 1st egg	195.3 ± 13.8	193.4 ± 11.8	202.8 ± 3.1	199.4 ± 10.2
Wt. at first egg	2987.5 ± 119.4	3026.0 ± 52.3	1504.0 ± 28.3	149.4 ± 74.1
Percent Hen-Day Production				
Normal	62.5 ± 6.7	62.5 ± 9.0	66.5 ± 9.9	58.0 ± 9.5
Defective	15.0 ± 8.6	27.0 ± 10.9	0	0.5 ± 0.5
Total	77.5 ± 8.7	88.8 ± 3.2	66.5 ± 9.9	58.5 ± 9.1
No. of eggs				
Normal	25.0 ± 2.7	25.0 ± 3.6	26.6 ± 4.0	23.2 ± 3.8
Defective	6.0 ± 3.4	10.8 ± 4.3	0	0.2 ± 0.2
Total	31.0 ± 3.5	35.8 ± 1.5	26.6 ± 4.0	23.4 ± 3.6
No. of follicles				
Ruptured	7.5 ± 0.3	6.4 ± 0.5	3.8 ± 0.4	4.2 ± 0.4
≥ 0.3 g	7.3 ± 0.3	8.0 ± 0.8	4.2 ± 0.6	4.4 ± 0.4
≥ 0.1 g	16.3 ± 1.6	17.0 ± 2.2	9.4 ± 1.9	8.8 ± 1.2
Wt. of				
Ovary (g)	48.7 ± 7.1	56.8 ± 5.3	21.7 ± 1.3	24.7 ± 2.8
Liver (g)	57.1 ± 3.4	70.3 ± 4.5	32.5 ± 1.4	38.6 ± 3.2
Thyroid (mg)	180.4 ± 12.2	161.1 ± 10.6	73.1 ± 6.4	59.2 ± 7.7
Pituitary (mg)	10.9 ± 1.5	9.5 ± 0.6	5.8 ± 0.6	6.1 ± 0.7
Adjusted for body wt.				
Ovary (g)	1.68± 0.21	1.86± 0.17	1.43± .06	1.51± .12
Liver (g)	1.99± 0.14	2.29± 0.12	2.16± .13	2.37± .11
Thyroid (mg)	6.38± 0.77	5.25± 0.27	4.84± .40	3.68± .48
Pituitary (mg)	0.39± 0.07	0.31± 0.02	0.39± .05	0.38± .06

Appendix Table 2. Means and standard errors of various measures of female reproduction by lines and photoperiods, for the laying period of 80 days from age at first egg, S_{16} generation (Experiment I)

Trait	HWS		LWS	
	14L:10D	14L:10D(3%Δ)	14L:10D	14L:10D(3%Δ)
Age at 1st egg	189.8 ± 9.8	195.4 ± 7.5	199.8 ± 8.9	212.4 ± 13.5
Wt. at first egg	2824.0 ± 85.0	3022.0 ± 123.9	1488 ± 36.1	1546.0 ± 40.1
Percent Hen-Day production				
Normal	72.8 ± 4.9	68.8 ± 5.4	71.0 ± 3.2	65.8 ± 6.7
Defective	10.0 ± 4.7	16.5 ± 5.8	0	0.3 ± 0.3
Total	82.8 ± 3.9	85.3 ± 3.1	71.0 ± 3.2	66.1 ± 6.6
No. of eggs				
Normal	58.2 ± 3.9	55.0 ± 4.3	56.8 ± 2.6	50.0 ± 7.8
Defective	8.0 ± 3.7	13.2 ± 4.6	0	0.2 ± 0.2
Total	66.2 ± 3.1	68.2 ± 2.5	56.8 ± 2.6	50.2 ± 7.8
No. of follicles				
Ruptured	6.4 ± 0.5	7.2 ± .6	3.8 ± 0.4	4.0 ± 0.3
≥ 0.3 g	6.8 ± 0.4	7.6 ± .5	4.4 ± 0.2	4.4 ± 0.2
≥ 0.1 g	17.4 ± 1.0	18.4 ± 2.2	10.4 ± 1.1	8.6 ± 0.7
Wt. of				
Ovary (g)	49.5 ± 4.8	61.7 ± 6.6	27.7 ± 2.5	27.7 ± 1.3
Liver (g)	61.3 ± 3.2	64.5 ± 6.5	35.7 ± 1.9	30.7 ± 2.5
Thyroid (mg)	128.3 ± 7.5	137.3 ± 16.7	72.8 ± 12.1	66.1 ± 12.6
Pituitary (mg)	10.5 ± 1.1	9.2 ± 0.9	5.8 ± 1.7	7.8 ± 0.6
Adjusted for body wt.				
Ovary (g)	1.53 ± .15	1.92 ± .16	1.55 ± .15	1.57 ± .08
Liver (g)	1.90 ± 0.14	2.01 ± .17	1.98 ± .07	1.75 ± .17
Thyroid (mg)	3.96 ± .18	4.30 ± .50	6.02 ± .58	3.69 ± .61
Pituitary (mg)	0.32 ± .03	0.29 ± .04	0.32 ± .09	0.45 ± .04

Appendix Table 3. Means and standard errors of various measures of female reproduction by lines and photoperiods, for the laying period of 120 days from the age of first egg, S_{16} generation (Experiment I)

Trait	HWS		LWS	
	14L:10D	14L:10D(3%Δ)	14L:10D	14L:10D(3%Δ)
Age at 1st egg	199.3 ± 9.9	194.2 ± 6.5	205.0 ± 4.5	210.8 ± 10.3
Wt. at 1st egg	2860.0 ± 205.5	2668.0 ± 214.6	1464.0 ± 43.5	1508.0 ± 42.9
Percentage Hen-day Production				
Normal	77.8 ± 6.5	68.0 ± 6.7	68.2 ± 3.0	65.8 ± 4.9
Defective	6.7 ± 5.9	1.3 ± .5	0.3 ± 0.3	0.5 ± .2
Total	84.4 ± 0.7	69.3 ± 6.8	68.5 ± 2.8	56.8 ±
No. of eggs				
Normal	93.3 ± 7.8	81.6 ± 8.1	81.8 ± 3.6	82.8 ± 4.3
Defective	8.0 ± 7.0	1.6 ± .6	0.4 ± 0.4	0.6 ± .2
Total	101.3 ± .9	83.2 ± 8.2	82.2 ± 3.3	83.4 ± 4.3
No. of follicles				
Ruptured	6.7 ± .3	7.4 ± .9	4.2 ± .2	4.6 ± .2
≥ 0.3 g	6.3 ± .3	6.0 ± .3	3.6 ± 0.4	4.0 ± .3
≥ 0.1 g	17.3 ± 2.6	14.8 ± 1.6	10.8 ± 1.5	8.6 ± 1.2
Wt. of				
Ovary (g)	51.9 ± 1.6	56.4 ± 4.8	25.6 ± 4.2	26.2 ± .8
Liver (g)	75.0 ± 2.6	74.2 ± 4.4	30.5 ± 1.7	30.5 ± 2.2
Thyroid (mg)	168.1 ± 14.6	132.5 ± 6.8	60.8 ± 7.8	64.8 ± 4.4
Pituitary (mg)	9.5 ± .7	8.4 ± .5	6.02 ± .5	6.5 ± .2
Adjusted for body wt.				
Ovary (g)	1.42 ± .08	1.64 ± .14	1.62 ± .19	1.52 ± .07
Liver (g)	2.05 ± .09	2.16 ± .15	1.71 ± .06	1.77 ± .12
Thyroid (mg)	4.62 ± .47	3.87 ± .27	3.6 ± .41	3.78 ± .35
Pituitary (mg)	0.26 ± .02	0.24 ± .01	0.34 ± .04	0.38 ± .02

Appendix Table 4, Means and standard errors of various measures of female reproduction by lines and photoperiods for the laying period of 160 days from age of first egg, S_{16} generation (Experiment I)

Trait	HWS		LWS	
	14L:10D	14L:10D(3%Δ)	14L:10D	14L:10D(3%Δ)
Age at 1st egg	181.7 ± 8.6	196.0 ± 3.8	223.6 ± 7.0	204.5 ± 6.8
Wt. at 1st egg	2623.3 ± 48.5	2942.0 ± 96.0	1672.0 ± 100.7	1625.0 ± 148.8
Percent Hen-Day Production				
Normal	75.4 ± 2.9	65.3 ± 3.6	49.3 ± 4.2	65.2 ± 4.6
Defective	2.5 ± 1.7	9.1 ± 2.5	0.3 ± .2	0.9 ± .5
Total	77.9 ± 4.0	74.4 ± 3.3	49.5 ± 4.1	66.1 ± 4.8
No. of eggs				
Normal	120.7 ± 4.7	104.4 ± 5.7	78.8 ± 6.7	104.3 ± 7.3
Defective	4.0 ± 2.7	14.6 ± 4.0	0.4 ± .2	1.5 ± .9
Total	124.7 ± 6.3	119.0 ± 5.3	79.2 ± 6.5	105.8 ± 7.6
No. of follicles				
Ruptured	7.7 ± .3	9.0 ± .7	4.8 ± .6	3.5 ± .3
≥ 0.3 g	5.3 ± .3	5.4 ± .5	3.6 ± .4	3.8 ± .6
≥ 0.1 g	16.7 ± 2.3	15.2 ± 1.7	9.4 ± .7	13.5 ± .9
Wt. of				
Ovary (g)	44.7 ± 2.2	51.9 ± 5.9	23.6 ± 3.3	26.2 ± 4.2
Liver (g)	71.5 ± 7.3	63.0 ± 6.0	29.2 ± .6	32.8 ± 2.0
Thyroid (mg)	129.0 ± 23.6	116.8 ± 7.9	64.5 ± 3.3	69.0 ± 5.0
Pituitary (mg)	7.4 ± .6	9.2 ± 0.8	6.6 ± 1.2	6.6 ± 2.9
Adjusted for body wt.				
Ovary (g)	1.30 ± .12	1.70 ± .29	1.20 ± .18	1.42 ± .22
Liver (g)	2.06 ± .11	2.01 ± .20	1.48 ± .07	1.79 ± .11
Thyroid (mg)	3.68 ± .54	3.72 ± .26	3.27 ± .27	3.77 ± .35
Pituitary (mg)	0.21 ± .01	.30 ± .03	0.34 ± .07	0.35 ± .03

Appendix Table 5. Means and standard errors[1] of egg production traits by lines, photoperiodic treatments and generations (Experiment I)

Trait	Line	S_{16} Photoperiod		S_{17} Photoperiod	
		14L:10D	14L:10D(3%Δ)	14L:10D	14L:10D(3%Δ)
%HDP of Normal eggs	HWS	67.92^{c}	70.41^{c}	66.34^{b}	68.68^{b}
	HWR	74.50^{d}	68.41^{c}	69.11^{b}	71.77^{b}
	LWS	64.67^{b}	69.79^{c}	64.22^{a}	63.56^{a}
	LWR	60.49^{a}	70.43^{c}	63.46^{a}	61.28^{a}
Double yolk eggs	HWS	0.75^{c}	1.08^{c}	1.22^{c}	1.57^{c}
	HWR	0.41^{b}	0.61^{b}	0.75^{b}	1.10^{b}
	LWS	0.02^{a}	0.07^{a}	0.02^{a}	0.02^{a}
	LWR	0.05^{a}	0.00^{a}	0.02^{a}	0.00^{a}
Broken eggs	HWS	1.32^{b}	1.37^{b}	1.78^{c}	1.73^{c}
	HWR	1.22^{b}	2.78^{c}	1.67^{c}	0.83^{b}
	LWS	0.12^{a}	0.17^{a}	0.11^{a}	0.22^{a}
	LWR	0.12^{a}	0.17^{a}	0.04^{a}	0.06^{a}
Total defective eggs	HWS	6.28^{c}	7.41^{c}	6.07^{d}	8.80^{e}
	HWR	3.75^{b}	7.83^{c}	4.67^{c}	5.24^{c}
	LWS	0.54^{a}	0.71^{a}	0.32^{a}	1.57^{b}
	LWR	0.61^{a}	0.24^{a}	0.41^{a}	0.34^{a}
Total yolks	HWS	74.96^{d}	78.89^{e}	73.63^{b}	79.05^{b}
	HWR	78.66^{e}	76.85^{e}	74.54^{b}	78.12^{b}
	LWS	65.23^{b}	70.56^{c}	64.56^{a}	65.15^{a}
	LWR	61.14^{a}	70.67^{c}	63.89^{a}	61.62^{a}

[1]Within a generation the means for a trait with the same superscript are not significantly different ($P \leq 0.05$).

Appendix Table 6. Means and standard errors[1] of body and egg weight traits by lines, photoperiodic treatments and generations (Experiment I)

Trait	Line	S_{16} 14L:10D	S_{16} 14L:10D(3%Δ)	S_{17} 14L:10D	S_{17} 14L:10D(3%Δ)
Body weight (g)					
8 weeks[1,2]	HWS	1027 ± 12[e]	1040 ± 14[e]	1029 ± 9[d]	1043 ± 7[d]
	HWR	955 ± 16[d]	890 ± 33[c]	908 ± 13[c]	936 ± 8[c]
	LWS	406 ± 7[a]	395 ± 8[a]	408 ± 8[a]	421 ± 7[a]
	LWR	459 ± 6[b]	480 ± 13[b]	485 ± 9[b]	480 ± 8[b]
24 weeks					
	HWS	2418 ± 29[d]	2386 ± 48[d]	2858 ± 29[d]	2988 ± 32[e]
	HWR	2238 ± 30[c]	2253 ± 70[c]	2584 ± 30[c]	2646 ± 28[c]
	LWS	1271 ± 18[a]	1296 ± 24[a]	1330 ± 20[a]	1374 ± 22[a]
	LWR	1422 ± 18[b]	1488 ± 34[b]	1427 ± 23[b]	1490 ± 34[b]
38 weeks					
	HWS	3231 ± 45[d]	3120 ± 71[d]	3417 ± 35[c]	3469 ± 41[c]
	HWR	2781 ± 132[c]	2917 ± 106[c]	3092 ± 59[b]	3135 ± 55[b]
	LWS	1677 ± 22[a]	1695 ± 19[a]	1743 ± 25[a]	1733 ± 27[a]
	LWR	1808 ± 43[b]	1797 ± 34[b]	1766 ± 33[a]	1757 ± 31[a]

Appendix Table 6 Continued

Trait	Line	S_{16} 14L:10D	S_{16} 14L:10D(3%Δ)	S_{17} 14L:10D	S_{17} 14L:10D(3%Δ)
Egg weight (g)					
Initial					
	HWS	42.4 ± 0.7[c]	44.4 ± 0.7[d]	40.8 ± 0.5[b]	39.7 ± 0.6[b]
	HWR	39.1 ± 0.7[b]	39.9 ± 0.8[b]	35.8 ± 0.6[a]	35.6 ± 0.6[a]
	LWS	37.0 ± 0.4[a]	37.3 ± 0.4[a]	34.7 ± 0.4[a]	35.3 ± 0.5[a]
	LWR	36.0 ± 0.5[a]	36.8 ± 0.7[a]	34.2 ± [a]	33.9 ± [a]
35 weeks					
	HWS	51.7 ± 0.8[c]	53.6 ± 0.8[c]	52.4 ± 0.5[c]	51.4 ± 0.5[c]
	HWR	49.5 ± 0.5[b]	48.1 ± 0.7[b]	47.9 ± 0.5[b]	48.6 ± 0.6[b]
	LWS	42.2 ± 0.4[a]	42.9 ± 0.4[a]	40.9 ± 0.4[a]	41.7 ± 0.4[a]
	LWR	42.7 ± 0.4[a]	41.9 ± 0.5[a]	41.9 ± 0.5[a]	41.3 ± 0.7[a]
Age at maturity					
	HWS	193.5 ± 2.0[b]	198.1 ± 1.7[b]	175.3 ± 1.6[b]	167.5 ± 1.5[a]
	HWR	186.9 ± 2.6[a]	195.4 ± 3.0[b]	170.3 ± 2.8[ab]	164.3 ± 1.8[a]
	LWS	203.8 ± 2.1[c]	200.6 ± 1.9[ac]	195.8 ± 2.1[c]	193.7 ± 1.9[c]
	LWR	190.8 ± 2.7[a]	195.2 ± 3.6[ab]	177.1 ± 2.5[b]	182.3 ± 2.8[b]

[1]Within a generation the means for a trait with the same superscript are not siginficantly different ($P \leq 0.05$).

[2]These weights were obtained before exposure to the different lighting regimes at 119 days of age, and are presented to demonstrate the similarity of samples.

Appendix Table 7. Least squares analysis of variance of reproductive and egg weight traits over two generations $S_{16}(R_3)$, $S_{17}(R_4)$ (Experiment I)

Source of variation	d.f.	Mean Squares					
		Age at maturity	Percent Hen-Day egg production			Egg weight	
			Normal	Defective	Yolks	Initial	35-wk
Generations (G)	1	35731**	401**	164*	550*	937**	52
Line (L)	3	14028**	703**	6128**	7379**	1435**	4729**
G X L	3	3759**	7	45	73	21	21
Treatment (T)	1	503	184*	141*	1178**	55	12
G X T	1	1854**	174*	93	336*	92	15
L X T	3	512	38	56	47	12	42
G X L X T	3	899*	95*	17	287	71*	100**
Error	761	240	33	30	84	21	18

**P $\leq$ 0.01

*P $\leq$ 0.05

Appendix Table 8. Heritability estimates and standard errors from paternal and maternal half-sib and full-sib correlations for various traits by lines and generations (Experiment II)

		S_{16}			S_{17}		
		4S/S+D+E	4D/S+D+E	2(S+D)/S+D+E	4S/S+D+E	4D/S+D+E	2(S+D)/S+D+E
Age at 1st egg	HW	.01 ± .24	.43 ± .40	.22 ± .18	.01 ± .23	.93 ± .40	.47 ± .17
	LW	.41 ± .25	.04 ± .21	.22 ± .14	.62 ± .38	.48 ± .32	.55 ± .21
	Combined	.29 ± .24	.11 ± .19	.20 ± .13	.35 ± .22	.68 ± .25	.52 ± .13
%HDP							
Normal	HW	.06 ± .18	.00 ± .32	.03 ± .15	.05 ± .18	.38 ± .33	.21 ± .14
	LW	.57 ± .31	.15 ± .22	.36 ± .17	.21 ± .26	.59 ± .36	.40 ± .17
	Combined	.34 ± .17	.03 ± .18	.19 ± .10	.12 ± .15	.48 ± .25	.30 ± .11
Total Defective							
	HW	.92 ± .44	.01 ± .27	.46 ± .24	.00 ± .13	.42 ± .34	.21 ± .13
	LW	.50 ± .27	.00 ± .20	.25 ± .15	.09 ± .24	.71 ± .39	.40 ± .17
	Combined	.84 ± .29	.11 ± .16	.48 ± .15	.00 ± .12	.51 ± .25	.26 ± .10
Ovulation defect							
	HW	.72 ± .38	.00 ± .28	.36 ± .22	.09 ± .19	.35 ± .33	.22 ± .14
	LW	.35 ± .23	.04 ± .22	.20 ± .13	.00 ± .19	.52 ± .37	.26 ± .15
	Combined	.67 ± .25	.10 ± .17	.39 ± .13	.06 ± .14	.40 ± .24	.23 ± .10
Yolks							
	HW	.06 ± .15	.00 ± .29	.03 ± .14	.02 ± .21	.72 ± .37	.37 ± .16
	LW	.57 ± .31	.14 ± .22	.35 ± .17	.61 ± .36	.32 ± .30	.47 ± .20
	Combined	.33 ± .16	.00 ± .17	.17 ± .10	.24 ± .19	.58 ± .25	.41 ± .12
Body weight at							
8 weeks	HW	.00 ± .38	1.67 ± .59	.84 ± .24	.21 ± .23	.41 ± .32	.31 ± .16
	LW	.44 ± .32	.66 ± .30	.55 ± .18	.20 ± .29	.84 ± .39	.52 ± .19
	Combined	.19 ± .35	1.35 ± .33	.77 ± .19	.21 ± .18	.59 ± .25	.40 ± .12
24 weeks							
	HW	.00 ± .30	1.25 ± .54	.62 ± .20	.40 ± .24	.00 ± .25	.20 ± .15
	LW	.27 ± .20	.09 ± .23	.18 ± .12	.37 ± .26	.10 ± .29	.24 ± .16
	Combined	.07 ± .18	.81 ± .27	.44 ± .12	.39 ± .18	.00 ± .19	.20 ± .11
Egg weight							
Initial	HW	.01 ± .29	.81 ± .46	.41 ± .19	.08 ± .20	.45 ± .34	.27 ± .15
	LW	.21 ± .19	.01 ± .24	.11 ± .12	.10 ± .22	.46 ± .36	.28 ± .16
	Combined	.14 ± .17	.48 ± .24	.31 ± .18	.09 ± .15	.45 ± .25	.27 ± .17
35 weeks							
	HW	.84 ± .47	.38 ± .33	.61 ± .26	.52 ± .36	.54 ± .35	.53 ± .21
	LW	.31 ± .29	.66 ± .33	.48 ± .18	.34 ± .36	.81 ± .44	.58 ± .22
	Combined	.63 ± .28	.51 ± .23	.57 ± .20	.45 ± .25	.64 ± .27	.54 ± .22

Appendix Table 9. Means and standard errors of various body size and reproductive traits by lines and genotypes in the B_1 generation (Experiment III)

Trait	HW			LW		
	Normal	Dwarf	Ratio[1]	Normal	Dwarf	Ratio[1]
Body weight (g)						
4 weeks	416 ± 6	378 ± 12	91	259 ± 6	222 ± 6	86
8 weeks	1091 ± 14	895 ± 36	82	698 ± 15	535 ± 17	77
12 weeks	1716 ± 22	1236 ± 32	72	1148 ± 19	744 ± 18	65
16 weeks	2237 ± 30	1565 ± 38	70	1471 ± 26	958 ± 31	65
20 weeks	2615 ± 53	1859 ± 47	71	1766 ± 29	1117 ± 30	63
24 weeks	3001 ± 59	2248 ± 66	75	2173 ± 40	1314 ± 40	61
32 weeks	3182 ± 82	2419 ± 123	76	2315 ± 47	1497 ± 47	65
40 weeks	3239 ± 78	2442 ± 82	75	2330 ± 39	1555 ± 49	67
Shank length (mm)						
12 weeks	113 ± 0.5	83 ± 1.1	76	100 ± 0.8	74 ± 1.3	75
16 weeks	117 ± 0.9	87 ± 0.9	74	105 ± 0.8	79 ± 1.7	75
40 weeks	117 ± 0.8	88 ± 1.9	75	106 ± 0.7	80 ± 1.8	75
Age at 1st egg	154 ± 9	170 ± 16	110	164 ± 19	166 ± 18	102
Percentage HDP						
Normal	43.1 ± 3.4	54.8 ± 4.7	127	58.7 ± 2.5	47.0 ± 2.7	80
Egg weight						
35 weeks	55.2 ± 0.7	54.8 ± 1.0	99	49.7 ± 0.6	45.0 ± 0.6	90

[1](Dwarf ÷ Normal) x 100

Appendix Table 10. Means and standard errors of various body size and reproductive traits by lines and genotypes in the B_2 generation (Experiment III)

Trait	HW			LW		
	Normal	Dwarf	Ratio[1]	Normal	Dwarf	Ratio[1]
Body weight (g)						
4 weeks	403 ± 7	344 ± 9	85	212 ± 5	181 ± 5	85
8 weeks	1068 ± 13	821 ± 15	77	554 ± 17	433 ± 18	78
12 weeks	1882 ± 28	1270 ± 31	68	986 ± 20	627 ± 14	64
16 weeks	2172 ± 34	1479 ± 31	68	1223 ± 21	743 ± 17	61
20 weeks	2286 ± 45	1590 ± 30	70	1445 ± 27	862 ± 19	60
24 weeks	2535 ± 46	1764 ± 31	70	1710 ± 26	940 ± 22	55
32 weeks	3136 ± 47	2101 ± 41	67	2023 ± 33	1064 ± 28	53
40 weeks	3394 ± 49	2290 ± 45	68	2224 ± 52	1143 ± 34	51
Shank length (mm)						
12 weeks	112 ± 0.8	87 ± 0.8	78	96 ± 0.5	70 ± 0.6	73
16 weeks	115 ± 0.7	88 ± 0.8	76	103 ± 0.6	70 ± 0.6	68
40 weeks	117 ± 0.8	92 ± 0.8	78	106 ± 0.5	72 ± 0.6	68
Age at 1st egg (Days)	189 ± 4	192 ± 4	102	190 ± 4	201 ± 5	106
Percentage HDP						
Normal	67.5 ± 3.2	71.8 ± 2.1	106	64.7 ± 2.1	36.8 ± 2.3	57
Defective	10.9 ± 2.1	2.3 ± 0.7	21	1.0 ± 0.3	0.6 ± 0.2	60
Yolks	80.1 ± 2.2	74.1 ± 2.1	93	66.1 ± 2.0	37.4 ± 2.3	57
Egg weight (g)						
Initial	42.0 ± 1.2	40.4 ± 0.8	96	39.0 ± 0.7	34.6 ± 0.8	89
35 weeks	52.7 ± 0.9	49.8 ± 0.8	95	46.5 ± 0.6	40.7 ± 0.8	88
Shell thickness (2.24^{-2mm})						
Initial	10.8 ± 0.2	10.7 ± 0.2	99	11.0 ± 0.2	9.8 ± 0.2	89
35 weeks	12.2 ± 0.3	12.4 ± 0.3	102	11.9 ± 0.2	11.2 ± 0.4	99

[1](Dwarf ÷ Normal) x 100

Appendix Table 11. Means and standard errors of various body size and reproductive traits by lines and genotypes in the B_3 generation (Experiment III)

Trait	HW			LW		
	Normal	Dwarf	Ratio[1]	Normal	Dwarf	Ratio[1]
Body weight (g)						
4 weeks	353 ± 5	310 ± 5	88	181 ± 4	160 ± 4	88
8 weeks	1026 ± 19	810 ± 16	79	492 ± 10	368 ± 9	75
12 weeks	1781 ± 20	1213 ± 16	68	832 ± 14	525 ± 11	63
16 weeks	2306 ± 49	1478 ± 26	64	1109 ± 22	650 ± 15	59
20 weeks	2488 ± 31	1802 ± 28	72	1389 ± 21	763 ± 18	55
24 weeks	2830 ± 32	2067 ± 30	73	1617 ± 21	884 ± 23	55
32 weeks	3019 ± 45	2248 ± 37	74	1776 ± 24	885 ± 31	50
40 weeks	3372 ± 50	2384 ± 39	71	1947 ± 29	1033 ± 33	53
Shank length (mm)						
12 weeks	112 ± 0.6	86 ± 0.5	77	90 ± 0.6	66 ± 0.7	73
16 weeks	115 ± 0.6	87 ± 0.5	76	97 ± 0.6	67 ± 0.7	69
40 weeks	115 ± 0.5	90 ± 0.5	78	99 ± 0.6	70 ± 1.0	71
Age at 1st egg (days)	171 ± 2	165 ± 2	97	172 ± 3	175 ± 2	102
Percentage HDP						
Normal	67.3 ± 2.2	70.7 ± 1.6	105	64.9 ± 2.8	32.0 ± 2.5	49
Defective	8.6 ± 1.6	1.2 ± 0.3	14	1.7 ± 0.4	0.6 ± 0.2	35
Yolks	74.8 ± 1.8	72.1 ± 1.6	96	66.8 ± 2.7	32.6 ± 2.5	49
Egg weight (g)						
Initial	39.7 ± 0.7	35.3 ± 0.8	89	32.5 ± 0.7	29.3 ± 0.6	90
35 weeks	51.2 ± 0.6	48.8 ± 0.5	95	42.4 ± 0.5	36.1 ± 1.0	85
Shell thickness (2.24-2mm)						
Initial	11.6 ± 0.1	10.8 ± 0.2	93	10.7 ± 0.1	9.4 ± 0.1	88
35 weeks	12.0 ± 0.2	12.3 ± 0.1	103	11.4 ± 0.2	10.2 ± 0.3	89
Specific gravity	1.074 ± .001	1.075 ± .001	100	1.077 ± .001	1.075 ± .001	100
Haugh units	87.4 ± 0.9	87.7 ± 0.8	100	85.8 ± 0.8	90.35 ± 1.2	105

[1](Dwarf ÷ Normal) x 100

Appendix Table 12. Means and standard errors of various body size and reproductive traits by lines and genotypes in the B_4 generation (Experiment III)

Trait	HW			LW		
	Normal	Dwarf	Ratio[1]	Normal	Dwarf	Ratio[1]
Body weight (g)						
4 weeks	382 ± 5	331 ± 5	87	171 ± 3	152 ± 3	89
8 weeks	1120 ± 10	856 ± 8	76	465 ± 7	354 ± 8	76
12 weeks	1614 ± 23	1145 ± 11	71	729 ± 14	464 ± 12	64
16 weeks	2090 ± 19	1446 ± 14	69	868 ± 27	567 ± 18	65
20 weeks	2266 ± 26	1584 ± 25	70	1078 ± 28	635 ± 30	59
24 weeks	2671 ± 29	1873 ± 20	70	1329 ± 29	726 ± 35	55
32 weeks	2943 ± 40	2165 ± 23	74	1706 ± 53	980 ± 57	57
40 weeks	3149 ± 38	2206 ± 41	70	1903 ± 42	1023 ± 64	54
Shank length (mm)						
12 weeks	112 ± 0.5	85 ± 0.5	76	92 ± 0.8	68 ± 1.3	74
16 weeks	113 ± 0.5	87 ± 0.4	77	95 ± 1.0	67 ± 1.6	71
40 weeks	115 ± 0.4	91 ± 0.3	79	99 ± 0.8	71 ± 2.2	72
Age at 1st egg (days)	193 ± 1	188 ± 2	97	201 ± 3	209 ± 3	104
Percentage HDP						
Normal	68.9 ± 1.3	74.3 ± 1.4	108	60.5 ± 3.8	28.7 ± 3.5	47
Defective	10.1 ± 1.1	1.6 ± 0.3	16	3.5 ± 1.1	3.3 ± 1.0	94
Yolks	80.2 ± 1.2	76.1 ± 1.4	95	64.2 ± 3.3	32.0 ± 3.3	50
Specific gravity	1.081 ± .001	1.082 ± .001	100	1.085 ± .001	1.081 ± .002	100

[1](Dwarf ÷ Normal) x 100

Appendix (Experiment IV)

Cytological Techniques

1. Composition of fixative:

Stock Solutions		
Solution A:	$NaH_2PO_4H_2.2H_2O$	27.6 g
	Distilled water	1000 ml
Solution B:	Na_2HPO_4	28.4 g
	Distilled water	1000 ml

2. Fixatives

A. Glutaraldehyde (3% solution) pH 7.4	
Glutaraldehyde (70%)	2 ml
Phosphate buffer (0.1 M)	45 ml
B. Osmium tetroxide (O_5O_4) pH 7.4	
Crystalline O_5O_4	0.25 g
Distilled water	12.5 ml
Buffer stock (0.1 M)	12.5 ml
Sucrose	1.125 g
Dissolve overnight in dark	

3. Fixation, Dehydration and Embedding Schedule

1. Phosphate buffered glutaraldehyde at 5°C	60 minutes
2. 0.1 M phosphate buffer stock (wash)	Overnight
3. Phosphate buffered O_5O_4	60 minutes
4. 30% ethanol	3 minutes
5. 50% ethanol	3 minutes
6. 70% ethanol	3 minutes
7. 95% ethanol	3@3 minutes
8. Propylene oxide	2@3 minutes
9. 1:1 Epon-propylene oxide at 60°C	30 minutes
10. Epon 812 (50:50 hard) at 60°C	36-48 hours

4. Epon 812 (50:50 hardness)

Dodecenyl succinic Anhydride (DDSA)	18.6 g
Epoxy Resin (Epon 812)	36.0 g
Nadie Methyl Anhydride (NMA) hardness	15.6 g
Tri (dimethyl Aminomethyl) Phenol (DMP-30) Accelerator	0.98 ml

Mix well with a magnetic stirrer

Appendix Table 13. Effect of photoperiod on the frequency of phenotypic and chromosomal abnormalities in the embryos from HWS and LWS pullets, of S_{16} generation

	Photoperiod			
	14L:10D		14L:10D(3%Δ)	
	HWS	LWS	HWS	LWS
Total embryos	76	131	59	75
No. phenotypically abnormal	12	12	16	5
Percentage abnormal	15.8	9.2	27.1	6.7
No. chromosomal aberrant	9	7	10	5
Percentage aberrant	11.8	5.3	17.0	6.7

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A GENETIC ANALYSIS OF REPRODUCTION IN GROWTH SELECTED LINES OF CHICKENS

by

Pashuvula Ramakrishna Reddy

(ABSTRACT)

Genetic and physiological aspects of egg production patterns and their relationships with growth parameters were studied utilizing S_{15}, $S_{16}(R_3)$ and $S_{17}(R_4)$ generation pullets from lines selected for high (HW) and low (LW) 8-week body weight. HW pullets laid significantly more defective eggs than LW pullets at each of the physiological ages measured from the date of first egg to 160 days of lay. Also, the pattern of egg production differed among lines. Arhythmic ovulation patterns were associated with physiological age, and delaying sexual maturity by feed restriction did not alter the patterns. Relaxing selection had a correlated effect on yolk synthesis and production of certain egg types. Increasing photoperiods beyond 14 hours in a 24-hour cycle did not have a consistent effect on growth reproduction.

The average heritability estimates for percentage hen-day production of normal eggs and yolks were similar and ranged from 0.19 to 0.41 indicating common genetic factors controlling these traits. Heritabilities of the composit trait, total defective eggs, ranged from 0.26 to 0.48. Genetic correlations were generally higher than the phenotypic correlations and genotype-environment interactions

were evident in the relationships between age at maturity and egg production traits. Environmental forces that delayed sexual maturity had no effect on the production of defective eggs. The genetic correlations between normal eggs and total yolks ranged from 0.88 to 0.93, while the environmental correlations between normal and defective eggs ranged from -0.34 to -0.46, suggesting that a genetic approach to the problem of defective eggs is realistic.

The dw gene was introduced into HW and LW lines and the specific selected inheritence was increased by repeated backcrossing. Data from the B_3 and B_4 generations enabled comparisons among Dw and dw alleles in specific genetic backgrounds. The depressive effect of the dwarfism was more severe in the LW than HW line and for body weight than for skeletal growth. In the B_3 and B_4 generations, the HW dwarfs matured 5 days earlier, while the LW dwarfs matured 6 days later than their normal sibs. On a percentage hen-day basis, HW dwarfs produced fewer yolks but more normal eggs than their nondwarf sibs. This supports the hypothesis that dw has a synchronizing effect on ovarian and oviducal functions in populations with excess yolk synthesis and arhythmic ovulation patterns.

A qualitative analysis, based on ultrastructural studies of adenohypophyseal gonadotrophs revealed higher synthetic activity and autodigestion of secretory products in the HW than in the LW pullets.

The frequencies of chromosomal abnormalities in random samples of embryos were 14.5% and 6.2% in the HW and LW lines, respectively. The primary cause of this difference was the higher incidence of euploid mosaics in the HW line. It was hypothesized that greater need for nutrients by fast growing embryos may induce mosaicism through polyspermy and development of supernumerary nuclei.