

THE EFFECT OF VARIOUS ENVIRONMENTAL TEMPERATURES
ON THE THYROID SECRETION RATE
IN CHICKENS

By

RICHARD WILFORD HENINGER

Bachelor of Science

Brigham Young University

Provo, Utah

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Thesis Approved:

W. S. Newcomer

Thesis Adviser

L. E. McDonald

James Martin

Dean of the Graduate School

438638

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INTRODUCTION

Because of the important role which thyroid hormone plays in animal physiology, many studies have been directed toward determining the relation of changes in thyroxine secretion associated with changes in internal and external environment. The thyroid has been stressed as a regulator of energy metabolism, since the most notable effect of variations of thyroid function is the change in metabolic rate, which is a manifestation of changes in cellular oxidations in all cells of the body. Since so many tissues are dependent on normal thyroid activity for normal function, it is evident that thyroid function is one of the most important of the many regulatory mechanisms necessary for maintaining a constant internal environment in a constantly changing external environment.

Data on the rate of thyroxine secretion of birds and mammals in cold environments indicate that the thyroids of both classes of vertebrates produce an increase of thyroxine. Since the thermoregulatory mechanism operating to decrease heat loss under these conditions in both types of animal is virtually the same, a similar relation between external temperature and thyroid function would be expected. Ample evidence also is available on the effect of high ambient temperature (near body temperature) on the thyroxine

secretion rate in mammals; comparable data on birds are not available in the literature. Due to the fact that birds have no sweat glands and must lose heat from the body by panting and "wing-holding," and since their normal body temperature is 7 - 9° F. higher than mammals, there is reason to suspect that at high ambient temperatures the thyroxine secretion rate might not parallel that of mammals exposed to similar conditions as it did at cold environmental temperatures.

In view of the above consideration, this experiment was designed primarily to study the effect of high environmental temperature on the thyroid secretion rate of young chicks.

These results could have practical application. It is a well established fact that high external summer temperatures have an adverse effect on both growth rate and egg production of chickens in Oklahoma. Since research data indicate that thyroid function is associated closely with both of these phenomena (Winchester and Davis, 1952; Winchester and Scarborough, 1953), it is not unreasonable to suggest that the adverse effects on growth rate and egg production might be caused, in part, by changes in thyroid function. Thus, an approach to the problem might be to counteract these effects of high temperature by controlling thyroid function. Data on the rate of thyroxine secretion at various temperatures, along with investigations of the way in which thyroxine is metabolized and excreted under

these conditions, may very well pose a possible approach whereby thyroid function can be controlled and the adverse effects of high environmental temperature eliminated or controlled.

REVIEW OF THE LITERATURE

Seasonal Variation of Temperature and Thyroid Function

Some of the earliest observations correlating seasonal variations of temperature with thyroid function were those of Seidell and Fenger (1913) who found that the weight and iodine content of fresh thyroid glands of cattle and sheep generally showed a regular seasonal change. Low and high iodine contents were observed to correspond to winter and summer, respectively; the size of the glands was correlated more loosely with winter and summer, which was due perhaps to the unknown nature (age, size, source) of the animals supplying the thyroids. Using human patients, investigators in Switzerland noted similar results with respect to size of the thyroid at various seasons of the year (Herzfeld and Klinger, 1922).

Initial studies with the avian class of vertebrates were those of Riddle and Fisher (1925) who studied the seasonal variation in the size of thyroids of pigeons. The thyroids of three species of birds kept on the same diet throughout the year notably increased in size in autumn and winter and decreased in summer. The decrease in size began promptly with the appearance of warm external temperatures

in the early months of spring. They further stated that these data obtained on birds coincided closely with those already available for mammals. That temperature was an important factor in this variation was demonstrated by Cramer (1916) who subjected rats to cold for several days. Histological changes occurred which were similar to those observed in "winter" thyroids of other animals. Mills (1918) likewise found both directions of change in the thyroids of rabbits, cats, and guinea pigs subjected to heat and cold for one to four weeks. Finally, Adler (1920) observed that various subspecies of the grass frog (Rana temporaria) from higher and colder regions had larger thyroids than those from warmer regions; those from an intermediate altitude and climate had thyroids of intermediate size. In experimental cultures of tadpoles he also found atrophic thyroids in animals raised at high temperatures and hyperplastic thyroids in those raised at low temperatures.

These early data, though not exhaustive, led to several conclusions: the thyroids of higher vertebrates normally undergo enlargement in autumn and winter and become smaller during spring and summer; these size changes are responses, in large part, to the changes in external temperature; and the enlargement from this cause is accompanied by an increase of hormone production in this organ.

Confirmation of this thyroid-seasonal relationship was reported by later investigators. Cruickshank (1929), in

studying the iodine content of the thyroid and ovary in the fowl, presented data which revealed that the thyroid weight showed a marked seasonal variation. The average thyroid weight for the period from mid-March to mid-July indicated a loss of 35 percent of the total weight as against the control level established from January to March. The seasonal variation in iodine content followed the variation in thyroid weight very closely with regard to total iodine, i.e., low iodine content in summer and high in winter. Investigating the physiology of the sparrow thyroid, Miller (1939) reported that the greatest activity, as measured by histological evidence of high epithelium and small follicles, occurred in winter and was associated with a high basal metabolic rate. Galpin (1938) in his studies on the factors affecting the hatching weight of Brown Leghorn chickens, measured thyroid weight at various times of the year; his results were in complete agreement with the foregoing.

While a similar correlation between season and thyroid activity existed in both birds and mammals, the relation between season and thyroid iodine content presented an opposite correlation. In birds, the thyroid iodine content was greater in winter than in summer; mammals showed a decrease of thyroid iodine in winter as compared to summer (Winchester, 1940). Kendall and Simonsen (1928) earlier reported that a higher thyroxine-like fraction was contained in thyroids of mammals in mid-summer as compared to that in February. Unfortunately, similar determinations were not

done on chickens. This opposite relationship could be explained on the assumption that little storage of colloid takes place in the fowl during the summer as apparently occurs in the thyroids of mammals (Cruickshank, 1929).

While thyroid weight is a useful tool in studying thyroid function, it does not always represent the true functional activity of the gland. Keating, et al., (1945) found that in mammals, increase in thyroid weight was due to both colloid accumulation and increased epithelial constituents. Under conditions of colloid accumulation, however, the "hormonal" blood iodine and basal metabolic rate (B.M. R.) were decreased whereas increased epithelial constituents resulted in an increase of both of these criteria. Furthermore, thyroid weight changes may have been due to alterations in vascularity and stroma in addition to changes in the amount of colloid and size and number of acinar cells. In the fowl, thyroid weight changes were found to be a more useful index of thyroid activity for the following reasons: Cruickshank (1929), as previously mentioned, noted that iodine content of the chicken varied with season and was high in late winter and low in summer. The thyroid weights of the fowl (Galpin, 1938; Cruickshank, 1929) varied with season in the same manner, i.e., large thyroids in winter and small ones in summer. The iodine content, therefore, paralleled the thyroid weight. Since the greatest thyroid activity could be assumed to occur in the winter months, the thyroid weight in the fowl apparently indicated thyroid

activity to an approximate degree. Cruickshank (1929) and Galpin (1938) further concluded that, since iodine content paralleled thyroid weight, the fowl thyroid apparently stored only limited colloid in contrast to greater storage in the thyroids of mammals.

Seasonal variation in thyroid activity probably is in a large measure due to temperature differences, but this may not be the sole factor controlling thyroid secretion at various times of the year. Reineke and Turner (1945) found an apparent seasonal change in thyroid activity of White Leghorn chicks even when the temperature was controlled fairly well. They noted that, when thyroid secretion was measured in two-week-old chicks at various months of the year, there was a threefold variation from the maximum in October and November (2.45 micrograms per day) to the minimal value in July and August (0.75 micrograms per day). Salter (1940) and Curtis and Fertman (1943) believed that the preponderant influence of iodine-rich diets during the spring and summer was a factor to be considered in seasonal variation of thyroid iodine content and thyroid activity since thyroid iodine increased on feeding rats iodine-rich diets. A decrease followed iodine-deficient diets. An iodine-rich diet was not the sole factor because the rise in iodine in the thyroid began to appear in March when fresh, green, iodine-rich foodstuffs could not yet be had (Kendall and Simonsen, 1928). Elmer (1938), discussing seasonal variations of the thyroid, stated that ultra-violet rays

might be responsible partially for seasonal variation. It was known that the winter was a period characterized by a shortage of these rays in the environment and the spring and summer by an abundance of these rays. Furthermore, it long has been established that length of day, i.e., the period of light received per day, influenced gonadal function. If gonadal function affects thyroid function, part of the seasonal thyroid variation might have been due indirectly to variation in day length with season (Schultze and Turner, 1945).

Thyroid Secretion Rate

The thyroid secretion rate has been defined as the amount of thyroid hormone secreted into the general circulation by the thyroid gland in a given period of time under defined environmental conditions (Hurst and Turner, 1948). Although crystalline thyroxine may not be the true thyroid hormone (Harrington, 1933; Barker, 1955), values of the thyroid secretion rate were obtained that can be expressed in equivalent amounts of a definite standard, namely the free acid form or the sodium salt of d,l or l-thyroxine.

Investigating the daily thyroxine requirement in human beings, Boothby and Baldes (1925) calculated the thyroxine secretion rate to be somewhere near 0.4 milligrams per day. This calculation was based on the determination of the total thyroxine present in the body and the rate of loss of

thyroxine via the urine and feces per day. Thompson, et al., (1935) found that from 0.25 to 0.35 milligrams of thyroxine must be injected daily in order to maintain a normal basal metabolic rate in myxedematous patients at bed rest. Elmer (1938), reviewing the results by various authors, observed that, on the basis of the optimal iodine requirement on the one hand, and the minimal requirement of thyroid hormone in myxedema on the other, the thyroid of the human secreted about 0.33 milligrams in twenty-four hours. Salter and McKay (1944) determined the concentration of protein-bound iodine in blood serum and plasma and used this determination as an index of net thyroid function. Working with thyroidectomized rats, Evans, et al., (1939) found that injection of 0.005 milligrams of crystalline thyroxine resulted in a return to normal basal metabolic rate and also normal growth. To maintain normal heart rate in adult thyroidectomized rats, Fishburne and Cunningham (1938) found that 0.04 milligrams of thyroxine were required daily.

Although these various methods had definite value, the most elegant method devised for estimating the thyroid secretion rate was that developed by Dempsey and Astwood (1943) in rats. This process involved the administration of a goitrogen, thiourea, or thiouracil, which prevented the formation of thyroxine by the thyroid gland, and as a result the amount of circulating hormone was lowered. This decreased amount of circulating hormone resulted in a removal of the inhibition of TSH secretion from the anterior

pituitary. The response of the thyroid to this additional thyrotrophic hormone was one of hypertrophy and hyperplasia of the follicular epithelium and a simultaneous increase in weight. These goitrogens did not interfere with the effects of exogenous thyroxine injections (Whitehead, 1943) or with the effect of thyroxine on the pituitary cells in thyroidectomized rats (Griesbach and Purves, 1943). Therefore, by simultaneous injections of thyroxine into these goitrogen-fed animals the amount of thyrotrophic hormone (TSH) released from the pituitary and, consequently, the size and weight of the thyroid could be regulated. The amount of exogenous thyroxine required to maintain normal thyroid size when administered simultaneously with a goitrogen represented the amount of hormone that normally would be present to maintain a normal pituitary-thyroid balance. This required amount of thyroxine, then, represented the thyroid secretion rate of the animals under the stated conditions of the experiment.

Measurement of the thyroid secretion rate by this method was dependent on the ability of the goitrogen to cause thyroid enlargement regardless of the amount of iodine in the circulation or gland itself. Schultze and Turner (1945), in reviewing the results of various investigators, reported that many agents had goitrogenic effects on the thyroid: raw meat diets; various toxemias; high fat diets; increased calcium in conjunction with low iodine diets; and soybean and cabbage diets. These effects could be reversed

by iodine administration. The first noteworthy reports of substances possessing goitrogenic properties that could not be reversed by iodine administration were those of MacKenzie, MacKenzie, and McCollum (1941) and MacKenzie and MacKenzie (1943). In the former study the object of the investigation was to determine the effectiveness of orally administered sulfaguanidine in combating intestinal infections in mice, but in the course of the experiment an effect on the thyroids was noticed. When administered at a 1 or 2 percent level to mice (21 - 23 days of age) which were sacrificed at periods varying from six to 16 weeks, it was found that sulfaguanidine produced glands three to eight times larger than those of control animals. MacKenzie and MacKenzie (1943) disclosed that the thyroid enlargement was not produced by sulfanilic acid or guanidine but by a series of sulfonamides and thioureas. Comparing the goitrogenic activity of thiourea and sulfaguanidine, they discovered that thiourea was eight times more effective than the same dietary level of sulfaguanidine during a two week testing period. The hyperplasia and hypertrophy were not reversed by adding iodide to the diet but were reversed by the administration of effective doses of thyroxine. The net effect of goitrogen administration was chemical thyroidectomy as indicated by the resultant decreased feed intake, decreased growth and development, and lowered basal oxygen consumption. The goitrogenic properties of various sulfonamides and thioureas decreased in the following order: thiourea,

sulfadiazine, sulfapyridine, sulfathiazole, sulfaguinidine, sulfanilyurea, sulfanilamide, and sulfasuxidine (Astwood, 1943; Astwood, Sullivan, Bissell, and Tyslowitz, 1943). They concluded that the hyperplasia which occurred under the influence of these drugs was compensatory to the failure of thyroid hormone synthesis.

Thiouracil appeared to have the highest goitrogenic activity of all the thiourea derivatives tested (Astwood, 1943; Astwood, Bissell, and Hughes, 1944). Thiobarbituric acid, diethylthiourea, and several derivatives of thiohydantoin were two to five times as active as thiourea but were somewhat more toxic, especially the thiohydantoins. Aniline derivatives were considerably less active than thiourea. Mixner, Reineke, and Turner (1944) fed thiouracil and thiourea in the standard ration to chicks for 14 days at the dosage level of 0.025, 0.05, 0.1 and 0.2 percent. There was progressive thyroid enlargement with both thiouracil and thiourea, and the maximal response was secured with both drugs at the 0.1 percent level. Similar results were obtained by Astwood, Bissell, and Hughes (1944). Mixner, Reineke, and Turner (1944) fed another group of chicks thiourea at levels of 0.4, 0.6, and 0.8 percent and found that all birds receiving 0.6 and 0.8 percent dosage levels of the drug died, indicating that it was toxic at these levels. The group receiving 0.4 percent reacted similarly to those receiving 0.2 percent; both of these groups had lower thyroid weights than the group receiving 0.1 percent.

Astwood, Bissell, and Hughes (1944) noted that 0.5 percent thiouracil in the feed produced marked retardation of growth and development of young chicks.

Mixner, Reineke, and Turner (1944) noticed a difference in the size of the goitre, induced by thiouracil feeding, depending on the sex of the birds. The females showed greater hypertrophy of the thyroid. They also observed that thyroidal response to goitrogens varied from one strain of chicken to another. This was in agreement with the reports of El-Ibiary and Shaffner (1950) and Shacklee and Shaffner (1952) who developed lines of New Hampshire chickens that varied widely in their response to thiouracil feeding.

With regard to the duration of effect of the goitrogenic agents, Pipes and Turner (1946) fasted three-week-old White Rock chicks for 12 hours and then allowed them access to feed containing 0.1 percent thiouracil. After two hours the feed was removed, and blood samples were taken at two-hour intervals. Thiouracil concentration showed a maximum (3.5 mg. per 100 cc.) two hours after feeding and a rapid decline until no traces were detected after 14 hours. Higher concentrations in the ration (0.2 percent and 0.6 percent) produced higher blood concentration, but these declined to zero levels after 18 and 20 hours, respectively. Mellen (1958) also administered thiouracil to chickens and found that it depressed the metabolic rate for approximately 18 hours after the drug was withheld. After this time the metabolic rate was the same as that of the controls.

Using this goitrogenic technique and assaying on the basis of equivalent amounts of d,l-thyroxine secreted per day, Schultze and Turner (1945) observed that the White Leghorn chick, four weeks of age, secreted 3.83 micrograms per day under "average environmental conditions." Hurst and Turner (1948) estimated the thyroid secretion rate of mature Schwing mice weighing 20 - 25 grams to be 0.5 micrograms per day under similar circumstances. Monroe and Turner (1946) reported a secretion rate of 3.1 micrograms of d,l-thyroxine per day in growing male rats weighing 50 - 100 grams. Thus, with the advent of this thyroxine-thiouracil technique, these and many more investigators collected data on thyroxine secretion rates of various animals under a wide variety of experimental and natural conditions.

Pipes, Premachandra, and Turner (1958) described a technique for the measurement of the thyroid hormone secretion rate of individual fowls. This procedure involved the daily measurement of thyroidal radio-iodine release in place of the measurement of blood I^{131} concentration. As thyroxine was discharged into the blood, the amount of thyroidal I^{131} gradually declined. When thiouracil was fed, the recycling of inorganic I^{131} formed as a product of thyroxine metabolism was prevented, and the apparent rate of thyroxine released from the thyroid gland was increased. When thyroxine was injected in increasing amounts and when it equalled the secretion rate, the exogenous thyroxine blocked the discharge of TSH from the pituitary. In the

absence of circulating thyrotrophin, the discharge of thyroidal I^{131} was prevented, and the decline in radioactivity of the thyroid was prevented. The point on the curve where the decline was stopped was considered to be the thyroid secretion rate.

Pipes, Blincoe, and Hsieh (1950), in an earlier report, employed a similar technique to estimate the thyroxine secretion rate without sacrifice of the animal. They proceeded on the assumption that the thyroxine secretion rate was governed by the thyroxine level of the blood; therefore, maintenance of the thyroxine level of the blood at or in excess of the normal secretion rate should result in minimal function of the thyroid. In their study they gave rats doses of thyroxine ranging from 0.5 to 10.0 micrograms per 100 grams of body weight and then injected them with 10 - 15 microcuries of I^{131} . In preliminary experiments, the rate of collection of radio-iodine by the thyroid and the formation of protein-bound iodine in the blood indicated that thyroid function reached a minimum as the thyroxine dosage approached the normal secretion rate.

Thyroxine Secretion Rate at Different Temperatures

Thyroid secretion studies as affected by temperature received considerable impetus when the new and sensitive goitrogenic technique of Dempsey and Astwood (1943) was developed. Theirs was the first attempt to quantify thyroid

secretion at fairly closely regulated temperatures. The experimental animals used were 80 - 100 gram rats. Thiouracil was fed as a 0.1 percent solution in the drinking water, and replacement therapy consisted of various doses of crystalline l-thyroxine. The various groups of rats were maintained at different temperatures. The cold environment was provided by placing the animals in a cold room at 1° C., while the hot temperatures were available by maintaining the rats in incubators regulated at 35° C. Control groups were kept in a regular animal room with the temperature varying between 20 - 26° C. A quantity of thyroid hormone equivalent to 5.2 micrograms of l-thyroxine daily was necessary to maintain a thyroid of normal weight at 25° C. A quantity of 9.5 micrograms was necessary at 1° C., and 1.7 micrograms were necessary at 35° C. These values were considered to be quantitatively equivalent to the amount of hormone produced by the normal thyroid gland under these conditions. Mixner, Reineke, and Turner (1944) used day-old chicks and an assay period of two weeks and found the secretion rate to be between 2.0 and 3.0 micrograms per day at a temperature near 80° F. Hoffman and Shaffner (1950) collected data on New Hampshire cockrels, from seven to eleven weeks of age, held in either a cool (45° F. with a relative humidity near 70 percent) or warm environment (74 - 80° F. with a relative humidity of 44 percent). Along with estimating the thyroxine secretion rate, oxygen consumption and histological studies were made. Utilizing the goitrogenic technique they

estimated the secretion rate to be 15.0 micrograms of d,l-thyroxine daily in the cool environment and 9.5 micrograms daily in the warm environment. The metabolic rate as determined by oxygen consumption showed a similar correlation, i.e., a greater oxygen consumption in those birds held at the lower temperature. This latter finding was in agreement with the work of Barott and Pringle (1946) who showed an inverse relationship between temperature and metabolic rate. Data on the thyroid weights of chick embryos by Hoffman and Shaffner (1950) revealed that the thyroids of chicks from eggs incubated at 98.6° F. weighed 7.38 milligrams as compared with 2.19 milligrams when the incubator temperature was 102.2° F. The latter glands showed histological evidence of reduced activity.

Rand, Riggs, and Talbot (1952), investigating the influence of environmental temperature on the metabolism of thyroid hormone in the rat, showed a statistically significant decrease in thyroid weight and serum protein-bound iodine in those rats held at 21 - 26° C. over those held at 1 - 7° C. Actual secretion rate values were not determined. Leblond, et al., (1944) used the fixation of radio-iodine as an index of thyroid activity and secretion. They found that rats subjected to a cold temperature (0 - 2° C.) for 26 days fixed an amount of radio-iodine 2.7 times the controls at room temperature. Exposure to temperatures between 32° C. and 34° C. resulted in a radio-iodine fixation of 0.33 times the controls at room temperature. At 80° F. mature Schwing

mice, averaging 22.5 grams in body weight, secreted the equivalent of 0.17 micrograms of d,l-thyroxine per day (Hurst and Turner, 1948). Pipes and Turner (1946), with the aid of radio-iodine techniques, held young female rats at temperatures of 90 - 92° F. and found they secreted the equivalent of 2.32 micrograms of d,l-thyroxine per 100 grams of body weight per day as compared to a secretion rate of 5.63 micrograms per 100 grams of body weight per day in similar rats held at a temperature of 70 - 75° F.

MATERIALS AND METHODS

Groups of young chicks were grown to an arbitrarily designated age of three weeks at environmental temperatures of 75° F., 95° F., and 105° F. Immediately following this growing period, and under the same environmental conditions, the chicks were subjected to a 10-day assay period in which the thyroid secretion rate was determined.

Experimental Chicks

The commercial strain of Arbor Acre White Rock male chicks used in this study were obtained from the Arbor Acre Farms in Springdale, Arkansas. They arrived in Stillwater two days after hatching.

The Growing Period

On arrival in the laboratory, each chick was weighed on an appropriate spring balance and the weight recorded. The chicks were tagged for identification and then randomly divided into four lots of 25 - 28 birds. Each lot was placed in a battery which measured 38 x 28 x 9 inches and was constructed of heavy wire mesh to enable adequate circulation of air and to permit equality of temperature with the

experimental environmental conditions. Each lot was confined to the same battery for the entire growing period.

A Buckeye, forced-draft, insulated incubator with a floor space of 270 square feet and a volume of 1,770 cubic feet was used as an animal chamber to maintain the temperature and relative humidity at constant levels. The arrangement of the batteries was such that all lots received approximately the same amount of light and were provided with as near equal conditions as possible. Circulation of air was accomplished by means of an 18-inch fan situated near the center of the incubator. Air movement such as this was found to have little or no effect on the thermoregulatory system of the fowl (Lee, et al., 1945). Artificial lights were kept on 24 hours a day.

Broiler ration EX-54 (Table I), as recommended by the Poultry Department of Oklahoma State University, was fed throughout the experimental period. Separate containers were used to store the ration for each lot, and a record of feed consumption by lots was kept for each group during the growing period only. Feed and water were available at all times. In calculating feed consumption, no correction was made for the slight unavoidable wastage of feed by the chicks themselves; however, the correction made for those birds that died during the experiment enabled a fairly accurate calculation of ration consumed by lots. These corrections for death were accomplished as follows: upon the death of one of the chicks, the total remaining feed was

weighed and a notation made of the amount of feed consumed up to that point by that particular number of birds in that lot. The feed consumption thereafter was based on one less than the original number of birds. As each subsequent bird died, appropriate notation was made and the remaining food weighed. No correction for ration consumed was made when deaths occurred during the first two to three days of the period, since in these first few days the amount eaten by each bird was exceedingly small.

TABLE I

OKLAHOMA STATE UNIVERSITY BROILER RATION, EX-54

	Percent
Marco B-75 (fat)	7
Ground Yellow Corn49
Pulverized Oats.	3
Alfalfa meal (17%)	2
Fish Meal (60%)12
Soybean Meal (44%)14.5
Meat and Bone Scraps (50%)	2
Dried Brewer's Yeast	2
Dried Whey	2
Dried Fish Solubles.	2
Dried Butyl Solubles	2
Di-calcium Phosphate (20% Phosphorus).	1.5
Trace Mineral Mix.	0.05
Salt	0.5
**VC-55 (Vitamin Mix Concentrate).	0.5
d,l Methionine	0.05

**VC-55 (per pound of ration)

Vitamin A	4,000 USP
Vitamin D ₃	2,000 USP
Riboflavin.	3 mg.
Pantothenic Acid.	4 mg.
Niacin.	20 mg.
Choline	300 mg.
Vitamin B ₁₂	3 ugm.
Procaine Penicillin	2 mg.
Menadione	3 mg.

To promote optimal conditions for growth during the first few days after hatching (Barott and Pringle, 1947; Randall, 1943), those groups subjected to 75° F. and 105° F. were maintained first at an environmental temperature of 90° F. for two days, after which the temperature was adjusted gradually to the desired level by the end of the fourth day. Those chicks subjected to the 95° F. temperature were maintained at this temperature beginning the day they were placed in the incubator.

Although the relative humidity was maintained at a constant level ($45 \pm 2\%$) for the duration of all treatment periods, it was impossible to standardize the barometric pressure. A continuous record of the temperature, relative humidity, and barometric pressure was kept by the use of a Bendix-Friez aerometerograph #712 geared to record for a seven day period without rewinding or changing the graph paper. From this record it was noticed that the magnitude of variation in barometric pressure was only 0.3 inches of mercury above and below the mean pressure of 29.0 inches, which would indicate little possibility of this parameter producing any appreciable effect on the results.

Assay Procedure

The method of determining the rate of thyroxine secretion used in this investigation was essentially the same as that employed by Dempsey and Astwood (1943) with rats and

later applied to chickens by Mixner, Reineke, and Turner (1944) and Schultze and Turner (1945). Prior to the time assay was to be made, all the chicks were removed from the batteries and thoroughly mixed. They were redivided then by random stratification into seven groups with 10 to 15 birds per group. Group I was used as the control group; group II was given thiouracil and no thyroxine injections; group III, thiouracil and 0.5 micrograms of l-thyroxine per day; group IV, thiouracil and 1.0 micrograms of l-thyroxine per day; group V, thiouracil and 2.0 micrograms of l-thyroxine per day; group VI, thiouracil and 4.0 micrograms of l-thyroxine per day; group VII, 6.0 micrograms of l-thyroxine per day and thiouracil; Group VIII (for the 95° F. temperature only) thiouracil and 8.0 micrograms of l-thyroxine per day.

Thiouracil, one of the potent goitrogenic thioureylenes (Astwood, 1943), was administered as a 0.1 percent solution in the drinking water. This method has a definite advantage over some of the other methods of administration by virtue of the ease with which it can be given. When given intravenously, Williams (1944) demonstrated 65 percent destruction of the thiouracil within three hours after injection. Evidence also has been presented by Schultze and Turner (1945) in studies on goats that subcutaneous injections produced considerably less goitrogenic effect than oral administration. Simultaneously with the thiouracil administration, replacement therapy was given in graded doses daily for 10 days by intraperitoneal injections of alkaline

solutions of crystalline l-thyroxine, prepared so that 0.1 ml. contained the daily dosage. At the end of this period of therapy, euthanasia was accomplished by placing the chicks in chloroform jars after which their weights were recorded and the thyroid glands removed. The glands then were weighed immediately to the nearest 0.1 mg. on a Roller-Smith torsion balance. The mean thyroid weight in milligrams percent plus or minus the standard error of the mean was plotted against dose of l-thyroxine. As thyroxine dosage increased, the thyroid weights decreased; the level of thyroxine necessary to produce a thyroid size equal to the mean size of the control animals was taken as the estimate of the normal thyroid secretion rate under the conditions of the experiment. This thyroid secretion rate was expressed as micrograms of l-thyroxine per bird per day and also as micrograms of l-thyroxine per 100 grams of body weight per day. Identical procedures were followed for each of the three different temperatures employed.

RESULTS AND DISCUSSION

Thyroxine Secretion Rates at Different Temperatures

Table II summarizes the results of the experiments performed to determine thyroxine secretion rates at different temperatures (see also Figures 1, 2, and 3 and Appendix A).

TABLE II
THE RELATION OF ENVIRONMENTAL TEMPERATURE TO
THYROXINE SECRETION RATE

Temp. °F.	Total Number of Birds	Body Weight (grams)	l-Thyroxine secreted per bird per day (micrograms)	l-Thyroxine secreted per 100 grams body weight per day (micrograms)
75	107	575	3.65	0.634
95	90	464	1.60	0.344
105	80	289	0.70	0.242

Chicks held at 75° F. secreted the equivalent of 3.65 micrograms of l-thyroxine per bird per day; the group held at 95° F. secreted 1.60 micrograms per bird per day; and those held at 105° F. secreted 0.70 micrograms per bird per day. There is the possibility that these values are an overestimation of the actual thyroxine output by the thyroid.

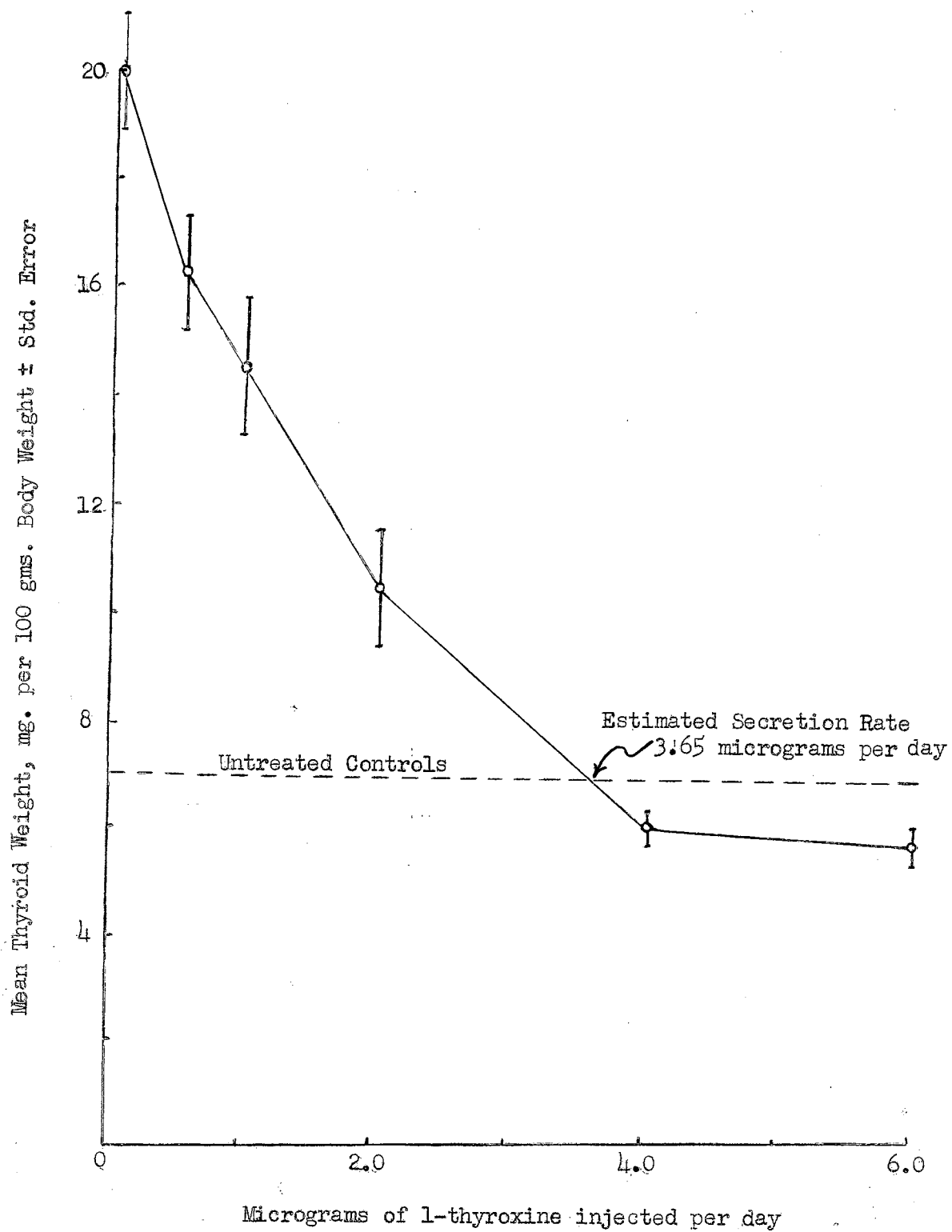


Figure 1. Thyroid Secretion Rate, Temperature - 75^oF.

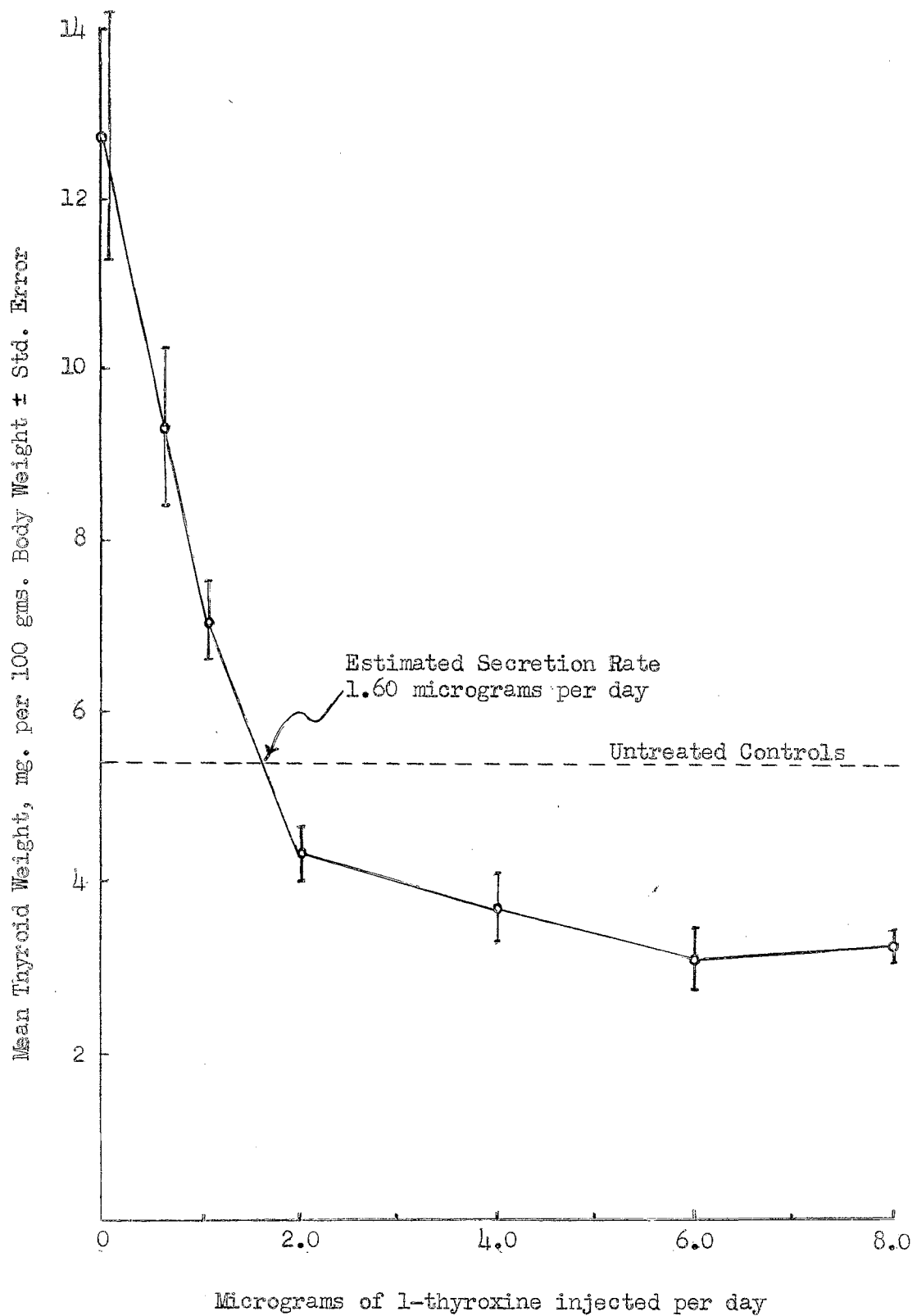


Figure 2. Thyroid Secretion Rate, Temperature - 95° F.

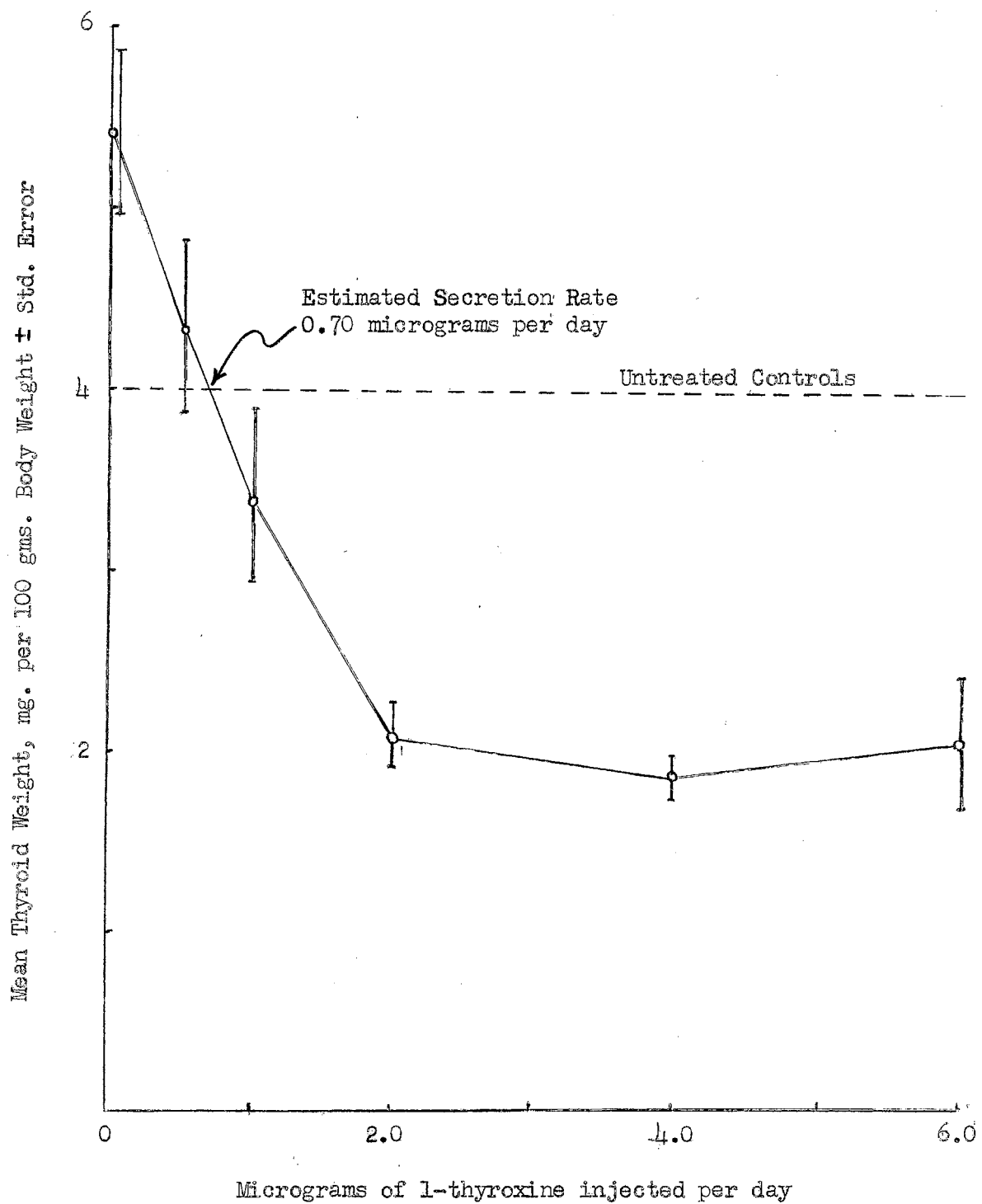


Figure 3. Thyroid Secretion Rate, Temperature - 105^o F.

Van Middlesworth, et al., (1959), using protein-bound iodine levels in the blood as an index of thyroid secretion, found: (1) that goitre prevention in propylthiouracil-treated rats was only achieved through daily injections of sodium l-thyroxine in amounts which elevated plasma concentrations of protein-bound iodine to values two to three times normal and (2) that goitre can occur despite some suppression in TSH production. This led them to suggest the possibility that the method of Dempsey and Astwood (1943) gave erroneously high values for the rate of thyroxine secretion by the thyroid. Newcomer (1957) showed that, in chickens, different doses of thyroxine were necessary to maintain control levels of various criteria (feather length, rectal temperature, thyroid weight, oxygen consumption, and suffocation time) in thiouracil-treated chicks. He concluded that these various criteria of thyroid hormone action were not equally sensitive measures in the chick for estimating thyroxine secretion, and the rate obtained depended on which criterion was used. The most sensitive criterion gave the lowest estimate of thyroxine secretion. This would lend support to the proposal of Van Middlesworth, et al., (1959).

From the results of this experiment summarized in Table II, it might appear that the reduction in thyroid secretion with increasing temperature was connected intimately with the decreased body weight associated with those birds subjected to higher temperatures, i.e., that the decreased secretion rate was a result of the decreased body weight.

This hypothesis can be disregarded due to the fact that Hurst and Turner (1948) estimated the secretion rate of mice at temperatures of 80° F. and 87° F. and found the lower secretion rate in those mice held at the higher temperature. The body weights of both groups were virtually identical. Furthermore, when the thyroxine secretion rates obtained in the present experiment were expressed on a body weight basis, the same relationship held true, i.e., those birds held at higher temperatures secreted less thyroxine per 100 grams of body weight than those at the lower temperatures. Thus the temperature itself must have been an important factor in causing the difference in secretion rate irrespective of the body weight changes.

A quantitative comparison of the results of this experiment with other investigations involving chickens cannot be made with any degree of accuracy since the conditions under which previous work was done were not adhered to in this study. Many variables must be controlled when thyroxine secretion rates are determined. There is ample evidence in the present study, and in the work of others heretofore mentioned, to indicate that environmental temperature is an important factor in regulating thyroid secretion. Another variable to consider is the age of the animal. Schultze and Turner (1945) determined the thyroid secretion rate of White Leghorn cockrels of increasing age groups from two to 27 weeks of age and found the average daily thyroxine secretion per chick increased with increasing age and body weight.

When expressed on a body weight basis, the thyroxine secretion rate increased up to four to seven weeks of age and then gradually decreased up to 27 weeks of age. Secretion rate values also vary between different strains of animals raised under identical conditions. Mature Schwing mice secreted the equivalent of 5.5 micrograms of d,l-thyroxine per 100 grams of body weight per day whereas mature Rockland mice of the same age secreted 2.1 micrograms per 100 grams of body weight per day (Hurst and Turner, 1948). These results were congruous with the results of Mixner, Reineke, and Turner (1944), Schultze and Turner (1945), and Mixner and Upp (1947), in which they observed strain differences in the thyroid secretion rate of chickens. It also has been observed that there are differences in secretion rate between sexes, as the females have the higher rate (Hurst and Turner, 1948; Monroe and Turner, 1946; Schultze and Turner, 1945).

Possible Mechanisms for the Control of Thyroid Secretion

Under normal circumstances the primary control operating to regulate thyroid function is probably the classical "feed-back" mechanism. D'Angelo (1955) recently has reviewed the development and present status of this concept, which emphasizes the level of circulating thyroxine as the primary cause of increased or decreased TSH secretion under average conditions. That this "feed-back" mechanism

involves the pituitary directly and not the hypothalamus has been demonstrated by Von Euler and Holmgren (1956) who injected thyroxine directly into the hypothalamus and obtained no decreased thyroxine secretion and presumably no decreased secretion of TSH. When injected directly into the pituitary, there was evidence of decreased TSH secretion as manifested by decreased thyroxine secretion.

The response of the thyroid of the chicken to various external temperatures may be explained partially on the fundamental basis of the "feed-back" mechanism. As external temperature increases, the need for increased heat production decreases with a consequent reduction in peripheral utilization of thyroxine. The result would be an increased blood thyroxine level which would inhibit further TSH secretion and decrease the output of thyroxine from the thyroid. Conversely, as external temperature decreases, the need for more thyroxine increases and a fall in blood thyroxine removes the inhibition from the pituitary, and more TSH and thyroxine are secreted. The studies of Rand, Riggs, and Talbot (1952) confirmed the increased rate of peripheral utilization of thyroxine in cold exposed rats by means of increased daily dosage of thyroxine necessary to maintain normal concentrations in the blood of thiouracil-treated animals in the cold. They also described a slight but "probably significant" decrease in the serum protein-bound iodine (PBI) of intact cold exposed rats at a time when thyroid activity was unequivocally increased. These results

confirmed the earlier results of Ershoff and Golub (1951) who found decreased PBI levels in cold exposed rats which led them to put forth the concept of increased peripheral utilization under these conditions as compared to similar rats exposed to warmer temperatures.

Variations in environmental temperature can alter thyroxine excretion in the urine and feces of rats and thus change the circulating thyroxine level in the blood, which could then act via the "feed-back" mechanism either to increase or decrease thyroxine secretion (Intoccia and Van Middlesworth, 1959; Leblond, et al., 1944).

Apart from the "feed-back" control of thyroid secretion, there are probably supplementary mechanisms involved. D'Angelo (1951, 1955) presented evidence to indicate that extra-thyroidal destruction of TSH takes place at varying rates under different circumstances. He showed that the disappearance rate of exogenous TSH from the circulation was much slower in hypophysectomized, thyroidectomized, or goitrous rats than in normal animals. While there is no supporting evidence, the possibility of external temperature modifying the extra-thyroidal destruction of TSH is not to be disregarded. An increased or decreased amount of TSH reaching the thyroid then would alter thyroxine secretion from the gland.

Considerable evidence indicates that the hypothalamus is involved in thyroid function since electrolytic lesions

in a certain area of the median eminence in rats abolished the thyroid hypertrophy which resulted from propylthiouracil feeding (Greer, 1952). Similar lesions were produced by Ganong, Fredrickson, and Hume (1955) in normal dogs with resultant thyroid hypofunction as evidenced by atrophy of the gland and a depression of thyroidal I^{131} uptake. On the other hand, stimulation of this area in the brain has on several occasions produced a sharp decrease in thyroid radioactivity which was interpreted to mean a greater discharge of thyroid hormone from the gland (Harris and Woods, 1957). It generally is agreed that the hypothalamus exerts its control on thyroid function by way of a neuro-humor liberated from the hypothalamus and carried to the pituitary through the hypophyseal portal system. Complete section of the pituitary stalk, which destroyed the portal system, led to reduced thyroid function (Barnett and Greep, 1951). It was inferred from this result that TSH production was under some measure of neural control from the hypothalamus and that severance of the pituitary stalk deprived the pituitary gland of this control. Florsheim (1959) recently has demonstrated a reduced secretion of TSH in rats with lesions in the area of the median eminence. It long has been established that the hypothalamus is connected intimately with the thermoregulatory processes of the body. As external temperature increases, the hypothalamus acts to increase heat loss and thus maintain normal body temperature. With evidence indicating that a hypothalamus-pituitary-thyroid

relationship exists, it is conceivable that external temperatures alter thyroid function by virtue of this relationship. While there is no evidence to support the concept, the theoretical implications suggest that alterations in thyroid secretion due to variations in temperature might be mediated through cutaneous temperature receptors to the hypothalamus and from there to the pituitary. Thus a control of TSH and thyroxine secretion might be maintained. A question that remains unanswered is how this control is exerted in terms of stimulatory or inhibitory impulses, i.e., does an increase in temperature send inhibitory impulses from the cutaneous receptors to the hypothalamus, or does a decrease in temperature stimulate the hypothalamus?

Another supplementary mechanism involves the adrenal gland and the adrenocorticotrophic hormone (ACTH). Brown-Grant (1956a) and Harris and Woods (1957) have presented evidence that ACTH and cortisone have an inhibitory effect on thyroid secretion by either affecting TSH secretion or the thyroid directly. Severe heat or cold could be classified as sufficient stressors to stimulate ACTH release and thus inhibit thyroxine secretion. This mechanism can explain why Dempsey and Searles (1943) found that even after the pituitary was isolated from any hypothalamic influence, by section of the stalk, thyroid atrophy still occurred when rats were exposed to very warm temperatures. No change in thyroid activity was observed under moderate temperature

variations. Furthermore, the inhibitory action of ACTH on thyroid activity can explain why Brown-Grant (1956, 1957) found that moderate degrees of cold were a more effective stimulus to thyroxine secretion than extreme cold. According to the "feed-back" mechanism, severe cold would produce greater stimulation than moderate degrees of cold if it is assumed that extreme cold would further increase peripheral utilization and excretion of thyroxine. Therefore, under extreme environmental temperatures there is the possibility of ACTH and the adrenal gland playing a role in regulating thyroxine secretion.

The results of the present experiment cannot be explained entirely on the basis of the foregoing mechanisms of thyroid control. To propose a further explanation for the mechanism of the effects of temperature on thyroid secretion, the work of Stetten (1956) offers an approach. He presented a method for showing the relationship between hormone dosage and response which was based on the fact that, whereas the physiological response to many endocrine products at low dosage levels is roughly proportional to dose, an apparent saturation often may be achieved at high dosage levels with the response becoming asymptotic with respect to dosage (Figures 1, 2, and 3). By analogy to the Michaelis-Menton concept that the abundance of enzyme-substrate complex limits the velocity of an enzyme-catalysed reaction, it was suggested that the magnitude of a hormone-provoked response was limited by the abundance of hormone

bound to appropriate sites on the target organ. He concluded that when dose/response was plotted against dose, a straight line should be secured with the slope and intercept (dose = 0) indicating the capacity of the target organ to bind the hormone and the affinity of the hormone for such binding sites, respectively. A decrease in response to a hormone on these coordinates may appear as an increase in the slope of the line reflecting a decrease in the total number of target organ sites. Alternatively such a decrease in response would appear as a line of unchanged slope but with a higher intercept, which indicates an altered affinity of the hormone to tissue-binding sites. Figure 4 shows the results of such a plot on the data collected in this investigation. Since goitre prevention is directly a measure of TSH secretion from the pituitary (Barker, 1955; Brown-Grant, 1957), the hormone referred to in Figure 4 is TSH. At temperatures of 75° F. and 95° F., there was little difference in the slope and intercept of the two lines indicating perhaps at these temperatures there was no, or very little, altered affinity of TSH for the sites on its target organ, the thyroid. However, at 105° F. the slope and particularly the intercept were markedly different from the lines plotted for the other temperatures. Using Stetten's hypothesis, this would indicate a considerable decrease in the affinity of TSH for its tissue binding sites at this temperature. This failure of TSH to be utilized by the thyroid effectively then would result in a decreased secretion of thyroxine.

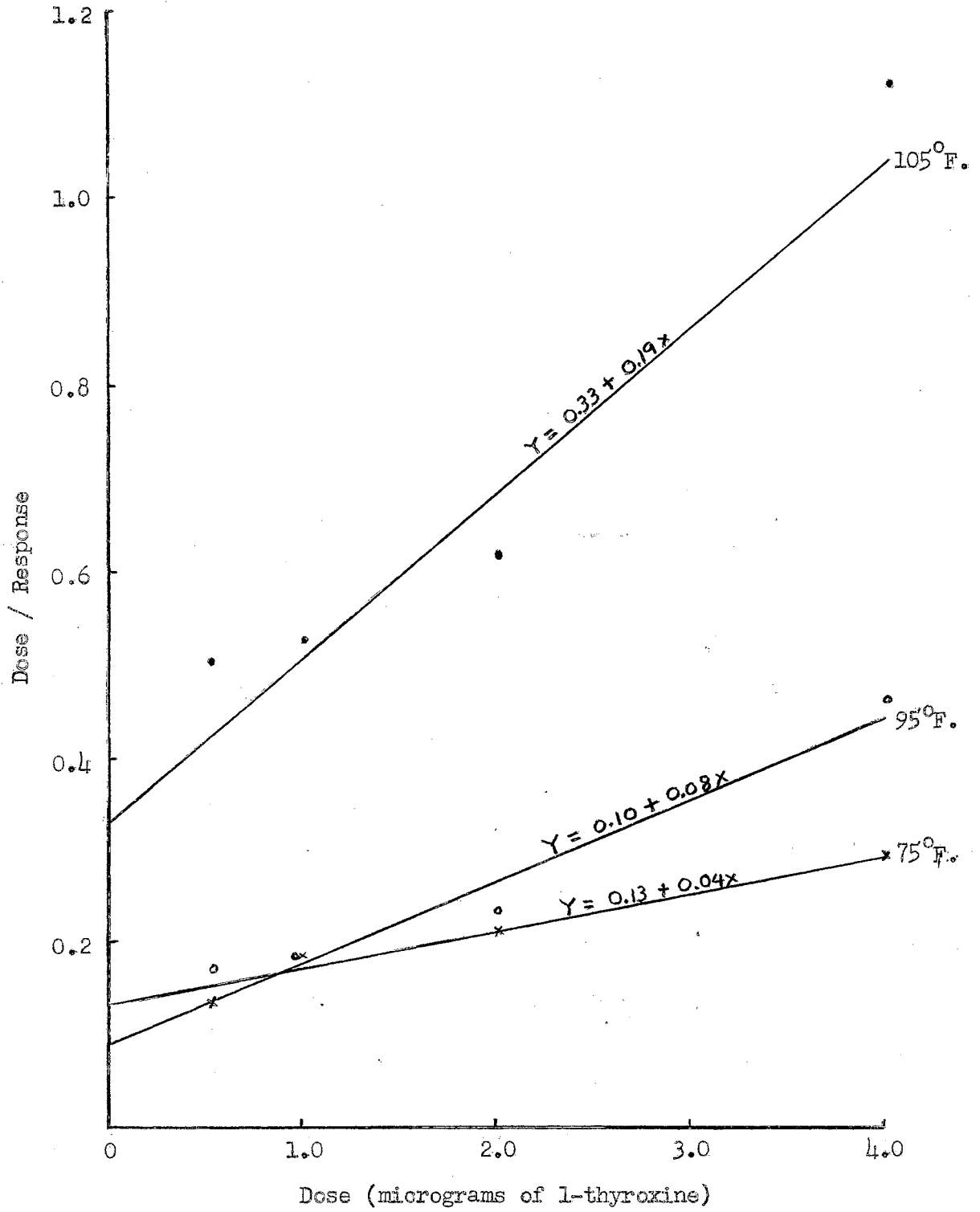


Figure 4. Relationship of Thyroxine Dosage to Decrease in Thyroid Weight Response

Since birds are homeothermic, and assuming the thermoregulatory mechanism was working normally, the body temperature at the environmental temperature of 75° F. or 95° F. would be the same. At temperatures higher than 95° F. this may not be the case, since Wilson (1948) reported that at 100° F. the body temperature of hens was 2° F. higher than at 70° F. It seems logical to assume that at an environmental temperature of 105° F., which is in the range of the normal body temperature of birds, the body temperature would rise even more than the two degrees reported by Wilson. This fact then could explain such a marked difference in effect of those birds held at 105° F. as revealed by the dose/response - dose plot as suggested by Stetten (1956). The conclusion follows that when body temperature rises there is a direct inhibitory effect of temperature on the response of the thyroid to TSH. Perhaps a heat sensitive enzyme system necessary for TSH activation is destroyed, thus decreasing thyroxine secretion. Bakke and Lawrence (1956) incubated beef thyroid slices with TSH at temperatures varying from 2° C. to 70° C. The I¹³¹ uptake by the slices increased up to 40° C. and then dropped sharply to near zero levels by 70° C. These data further support the concept of a direct inhibitory effect of high temperature on TSH activity.

Response of Thiouracil-treated Chicks to Various Temperatures

As shown in Figure 5, there was a difference in response of the thiouracil-treated chicks at the three temperatures; the lowest temperature produced the largest goitrogenic response. While thiouracil prevents the synthesis of thyroxine, it does not inhibit TSH secretion from the pituitary; therefore, the increased peripheral utilization and excretion of thyroxine at lower temperatures would lower blood thyroxine and stimulate more TSH secretion. The goitre under these circumstances would be larger than when TSH secretion is reduced by high temperatures as a result of a diminished utilization and excretion of thyroxine.

Hypothalamic influence also might be a factor to consider in the response of the thyroids of thiouracil-treated chicks to various temperatures, since Greer (1952) found that the normal response to chronic thiouracil feeding was diminished in rats with hypothalamic lesions. If, as has been assumed previously, the hypothalamus increased TSH secretion under conditions of reduced external temperature, the goitrogenic response under these conditions consequently would be greater than the response elicited at higher temperatures when hypothalamic stimulation to TSH secretion is decreased.

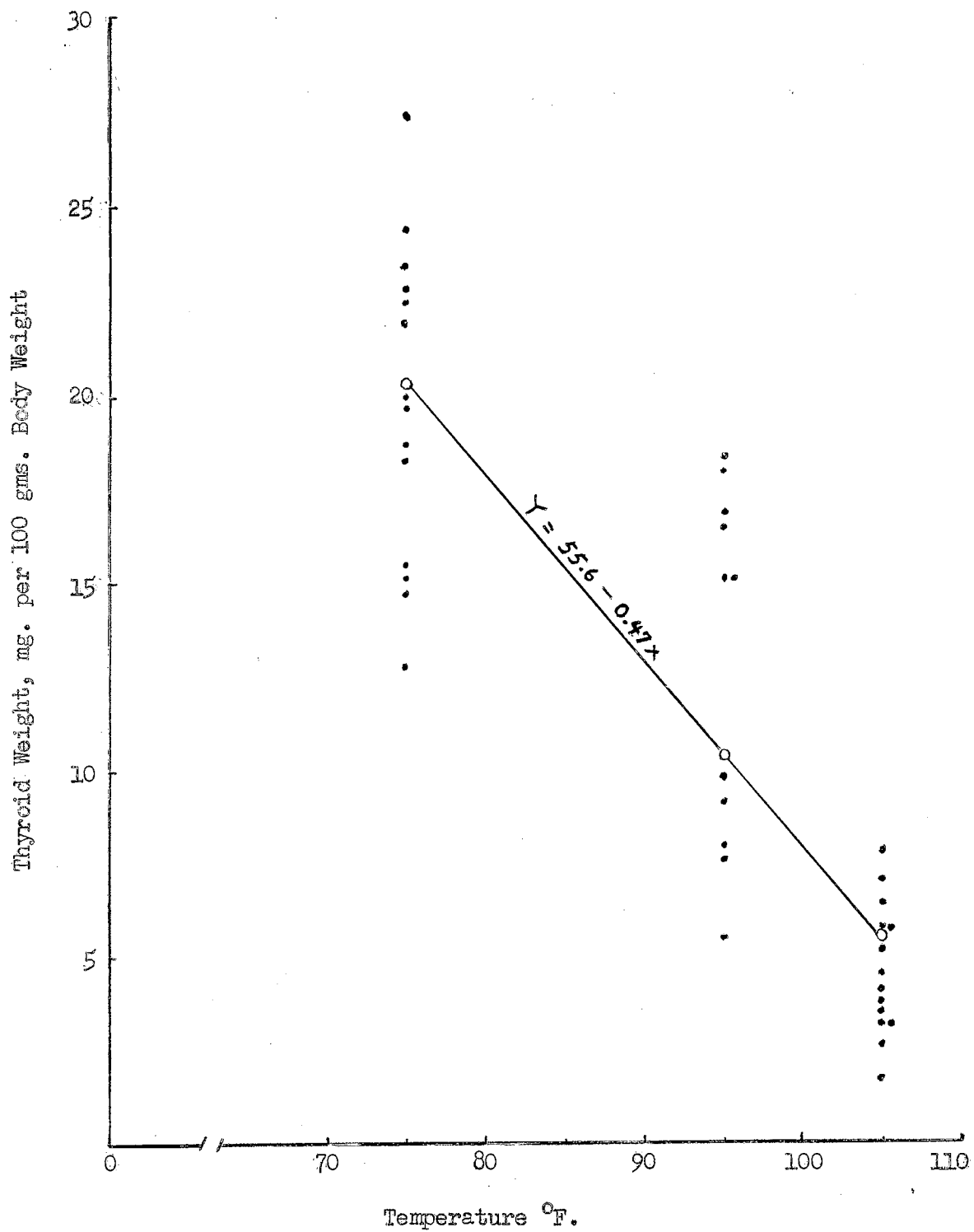


Figure 1. Effect of Temperature on the Thyroid Weight of Thicouracil-Treated Chicks

The Effect of Temperature on Body Weight and Feed Consumption

When chicks were subjected to increasing temperatures, there was a resultant decrease in final body weight, in body weight gains over the three week growing period, and in feed consumption. Hutson, Joiner, and Carmon (1957) exposed chickens to 90° F. and noticed both a decrease in weight and feed consumption of those birds as compared to ones held at room temperature. The findings of the present investigation support their results (Table III and Appendix B). Results of other investigators confirm the fact that high temperatures have adverse effects on body weight and feed consumption (Dempsey and Astwood, 1943; Barott and Pringle, 1947, 1949, 1950). It is a likely possibility that at high temperatures there is a decreased appetite of the animal; the consequent decrease in feed consumption might be the main factor in reduced body weight gain, since the feed efficiency did not change to any extent as temperature increased.

TABLE III
SUMMARY OF WEIGHT GAINS AND FEED CONSUMPTION
AT VARIOUS TEMPERATURES

Temp. °F.	Weight Gains in 3 Weeks (grams/chick)	Feed Cons. in 3 Weeks (grams/chick)	Feed Efficiency Feed Cons./ Wt. Gain
75	300.0	483.5	1.61
95	253.4	369.1	1.46
105	144.0	250.2	1.73

There was no apparent effect of the position of the batteries in the incubator on the weight gains of the chicks (Appendix B), since the variation between the mean weight gains of the various lots at one particular temperature was slight (15 - 20 grams). Furthermore, the lot that exhibited the greatest mean weight gain at one temperature did not show the greatest mean weight gain at another temperature.

SUMMARY AND CONCLUSIONS

Three groups of two-day-old chicks were grown to three weeks of age at environmental temperatures of 75° F., 95° F., and 105° F. and a relative humidity of 45 percent. Body weight gains and feed consumption were recorded for this same period. At the end of three weeks the thyroid secretion rate was determined using the goitrogenic technique of Dempsey and Astwood (1943).

The thyroid secretion rate at 75° F. was estimated to be equivalent to 3.65 micrograms of l-thyroxine per bird per day, or 0.634 micrograms per 100 grams of body weight per bird per day. At 95° F., the rate was estimated at 1.60 micrograms per bird per day, or 0.344 micrograms per 100 grams of body weight per day, while at 105° F., 0.70 micrograms per bird per day, or 0.242 micrograms per 100 grams of body weight per day, were secreted. Body weight gains at the end of three weeks showed an average increase of 300 grams in those birds raised at 75° F.; 253.4 grams at 95° F.; and 144 grams at 105° F. Feed consumption for the same three week period showed a similar trend. At 75° F. the average amount of ration consumed by each bird for the entire period was 483.5 grams; at 95° F., the average amount consumed was 369.1 grams; and at 105° F. it was 250.2 grams. The decrease in body weight was thought to be a result of

decreased appetite and consequent decreased feed consumption at the higher temperatures.

There are a large number of factors possibly involved in the control of thyroid secretion at various temperatures:

- (1) alterations in peripheral utilization of thyroxine,
- (2) increased or decreased excretion in the urine and feces,
- (3) fluctuations in the rate and extent of extra-thyroidal destruction of TSH, (4) hypothalamic control of TSH secretion by the mediation of cutaneous temperature receptors,
- (5) the inhibitory effect on thyroid activity of ACTH or cortical hormones under conditions where environmental temperature might act as a stimulus (stressor) for ACTH release, and (6) an additional factor, for which evidence is presented, is a direct effect of environmental temperature on the binding of TSH to the thyroid gland. When environmental temperature increases to the point where it produces a rise in the body temperature of the animal, there is a reduced affinity of TSH for its thyroid receptor site and a resultant decreased thyroid hormone secretion.

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APPENDICES

APPENDIX A

APPENDIX A

THYROID SECRETION RATE, TEMPERATURE - 75° F.

Bird No.	l-Thyroxine injected per bird per day (micrograms)	Thio-uracil in water (%)	Body Weight (gm.)	Thyroid Weight (mg.)	Thyroid Weight (mg.%)
<u>Group I</u>					
7403	--	--	594	36.8	5.1
7446	--	--	590	26.0	4.4
4894	--	--	566	37.0	9.4
7462	--	--	627	45.3	7.2
7448	--	--	622	47.2	7.6
7443	--	--	614	25.9	4.1
7492	--	--	673	42.3	6.2
7393	--	--	577	28.7	4.9
7405	--	--	512	32.1	6.2
7494	--	--	735	63.7	8.6
7440	--	--	680	48.0	7.0
7425	--	--	650	42.1	6.4
7434	--	--	750	43.2	5.7
7456	--	--	691	50.0	7.2
7418	--	--	532	30.0	5.6
7459	--	--	620	36.2	5.8
4895	--	--	550	44.4	8.0
7426	--	--	600	41.0	6.8
7488	--	--	661	33.4	5.0
4893	--	--	627	57.2	9.1
7400	--	--	623	60.2	9.6
7394	--	--	620	54.2	8.7
7480	--	--	606	50.0	8.2
Mean			623	42.4	6.8 ± 0.34*
<u>Group II</u>					
7496	--	0.1	648	153.6	23.7
7460	--	0.1	558	72.0	12.9
7483	--	0.1	615	135.1	21.9
7424	--	0.1	702	107.3	15.2
4898	--	0.1	550	109.7	19.9
7471	--	0.1	476	115.0	24.1
7399	--	0.1	571	104.6	18.3
7438	--	0.1	565	87.8	15.5
7485	--	0.1	601	138.3	23.0
7415	--	0.1	582	92.0	15.8

* Standard error of the mean

Bird No.	1-Thyroxine injected per bird per day (micrograms)	Thio-uracil in water (%)	Body Weight (gm.)	Thyroid Weight (mg.)	Thyroid Weight (mg.%)
4896	--	0.1	588	111.0	18.8
7422	--	0.1	507	101.4	20.0
7463	--	0.1	517	117.0	22.6
7430	--	0.1	594	126.0	28.0
Mean			577	112.2	20.0 ± 1.6
<u>Group III</u>					
7391	0.5	0.1	633	95.2	15.0
7441	0.5	0.1	432	74.0	17.1
7417	0.5	0.1	563	71.0	12.6
7420	0.5	0.1	631	85.1	13.4
4892	0.5	0.1	576	74.4	12.9
7377	0.5	0.1	517	59.6	11.5
7433	0.5	0.1	454	73.9	16.2
7486	0.5	0.1	578	96.5	16.6
7491	0.5	0.1	584	133.0	22.7
7392	0.5	0.1	551	126.2	22.9
7444	0.5	0.1	455	90.0	19.7
7479	0.5	0.1	595	130.8	21.9
7427	0.5	0.1	630	101.6	16.1
7410	0.5	0.1	591	57.8	9.7
Mean			556	90.6	16.3 ± 1.1
<u>Group IV</u>					
7442	1.0	0.1	543	113.3	20.8
7401	1.0	0.1	577	61.0	10.5
7474	1.0	0.1	531	68.0	12.8
7404	1.0	0.1	523	91.0	17.3
7454	1.0	0.1	594	94.5	15.9
7466	1.0	0.1	504	53.9	10.6
7423	1.0	0.1	468	64.0	13.6
7452	1.0	0.1	624	75.3	12.0
7429	1.0	0.1	523	78.0	15.2
7470	1.0	0.1	530	65.8	12.4
7493	1.0	0.1	620	86.0	13.8
7406	1.0	0.1	618	107.4	17.3
7402	1.0	0.1	647	87.4	13.5
7435	1.0	0.1	639	103.6	16.2
Mean			567	82.1	14.4 ± 0.80

Bird No.	l-Thyroxine injected per bird per day (micrograms)	Thio-uracil in water (%)	Body Weight (gm.)	Thyroid Weight (mg.)	Thyroid Weight (mg.%)
<u>Group V</u>					
7408	2.0	0.1	590	65.0	11.0
7411	2.0	0.1	613	76.9	12.5
7484	2.0	0.1	660	40.5	6.1
7428	2.0	0.1	508	47.0	9.2
7437	2.0	0.1	561	114.4	20.3
7436	2.0	0.1	672	95.7	14.2
7413	2.0	0.1	616	67.2	10.9
7481	2.0	0.1	590	46.0	7.8
7467	2.0	0.1	497	34.5	6.9
7451	2.0	0.1	525	37.8	7.2
7407	2.0	0.1	522	48.0	9.1
7472	2.0	0.1	442	30.0	6.7
7468	2.0	0.1	591	82.6	13.9
7432	2.0	0.1	500	52.5	10.5
Mean			563	59.8	10.5 \pm 1.02
<u>Group VI</u>					
7416	4.0	0.1	511	43.1	8.4
7376	4.0	0.1	594	42.6	7.1
7482	4.0	0.1	610	43.5	7.1
7396	4.0	0.1	481	27.2	5.6
7449	4.0	0.1	557	31.1	5.5
7439	4.0	0.1	600	38.3	6.3
7431	4.0	0.1	479	22.0	4.5
7487	4.0	0.1	605	34.6	5.7
7447	4.0	0.1	482	32.4	6.8
7498	4.0	0.1	515	34.4	6.6
7414	4.0	0.1	627	45.4	7.2
7378	4.0	0.1	308	15.6	5.1
7445	4.0	0.1	572	30.0	5.2
7398	4.0	0.1	608	25.8	4.2
Mean			539	33.3	6.1 \pm 0.31
<u>Group VII</u>					
7495	6.0	0.1	584	68.2	11.6
7489	6.0	0.1	472	22.4	4.7
7397	6.0	0.1	619	46.2	7.4
7453	6.0	0.1	533	39.0	7.3
7395	6.0	0.1	646	32.4	5.0
7455	6.0	0.1	598	29.6	4.9

Bird No.	l-Thyroxine injected per bird per day (micrograms)	Thio-uracil in water (%)	Body Weight (gm.)	Thyroid Weight (mg.)	Thyroid Weight (mg.%)
7497	6.0	0.1	483	23.5	4.8
7469	6.0	0.1	659	25.2	3.8
7490	6.0	0.1	587	26.7	4.5
7450	6.0	0.1	625	26.2	4.1
7477	6.0	0.1	555	31.0	5.5
7458	6.0	0.1	520	38.0	7.3
7465	6.0	0.1	633	50.8	8.0
7478	6.0	0.1	532	31.0	5.8
Mean			574	35.0	6.0 \pm 0.28

THYROID SECRETION RATE, TEMPERATURE - 95° F.

Bird No.	l-Thyroxine injected per bird per day (micrograms)	Thio-uracil in water (%)	Body Weight (gm.)	Thyroid Weight (mg.)	Thyroid Weight (mg.%)
<u>Group I</u>					
6158	--	--	484	25.0	5.1
6129	--	--	420	16.0	3.8
6109	--	--	543	34.0	6.2
6126	--	--	544	32.3	5.9
4876	--	--	418	25.0	5.9
4877	--	--	415	19.8	4.7
4878	--	--	504	24.9	4.9
6107	--	--	385	22.7	5.8
6150	--	--	305	25.4	8.3
6128	--	--	378	19.7	5.2
6117	--	--	468	26.3	5.6
47	--	--	418	16.6	3.9
6143	--	--	412	22.2	5.3
4879	--	--	476	30.0	6.3
Mean			440	24.3	5.4 ± 0.13
<u>Group II</u>					
46	--	0.1	478	78.6	16.4
6127	--	0.1	478	40.0	8.3
6134	--	0.1	463	83.2	17.9
6147	--	0.1	535	81.2	15.2
45	--	0.1	493	75.0	15.2
6135	--	0.1	458	83.8	18.2
6132	--	0.1	547	34.0	6.2
6142	--	0.1	445	36.2	8.1
6139	--	0.1	418	71.2	17.0
6131	--	0.1	439	44.0	10.0
6137	--	0.1	372	35.2	9.4
Mean			466	51.1	12.7 ± 1.4
<u>Group III</u>					
4880	0.5	0.1	471	35.6	7.5
6123	0.5	0.1	531	64.8	12.2
6106	0.5	0.1	548	42.6	7.7
6111	0.5	0.1	530	56.0	10.2
6105	0.5	0.1	478	43.0	9.0
6103	0.5	0.1	494	32.0	6.4
6125	0.5	0.1	568	52.4	9.2
6122	0.5	0.1	518	78.5	15.1

Bird No.	1-Thyroxine injected per bird per day (micrograms)	Thio-uracil in water (%)	Body Weight (gm.)	Thyroid Weight (mg.)	Thyroid Weight (mg.%)
6116	0.5	0.1	478	50.5	10.5
6112	0.5	0.1	405	32.8	8.1
6114	0.5	0.1	425	40.0	9.4
Mean			495	48.0	9.5 \pm 0.82
<u>Group IV</u>					
6167	1.0	0.1	551	34.0	6.1
6173	1.0	0.1	539	40.0	7.4
4886	1.0	0.1	590	43.2	7.3
6169	1.0	0.1	510	45.1	8.8
6165	1.0	0.1	440	34.3	7.7
4881	1.0	0.1	429	25.2	5.8
6171	1.0	0.1	522	45.0	8.6
6166	1.0	0.1	550	44.4	8.0
6157	1.0	0.1	532	40.6	7.6
6154	1.0	0.1	361	23.8	6.5
4883	1.0	0.1	422	27.6	6.5
Mean			495	36.6	7.0 \pm 0.30
<u>Group V</u>					
6183	2.0	0.1	612	28.0	4.5
6193	2.0	0.1	509	27.8	5.4
4891	2.0	0.1	471	18.0	3.8
6189	2.0	0.1	491	14.6	3.0
6184	2.0	0.1	472	22.6	4.8
6180	2.0	0.1	514	12.7	2.5
4889	2.0	0.1	345	17.6	5.1
6198	2.0	0.1	378	20.8	5.5
6194	2.0	0.1	311	13.3	4.2
6181	2.0	0.1	377	16.2	4.3
6188	2.0	0.1	457	23.0	5.0
Mean			449	19.5	4.4 \pm 0.29
<u>Group VI</u>					
6153	4.0	0.1	453	10.2	2.2
4887	4.0	0.1	580	13.0	2.2
6196	4.0	0.1	456	12.0	2.6
6190	4.0	0.1	470	13.0	2.7
6176	4.0	0.1	472	19.0	4.0
6195	4.0	0.1	452	16.8	3.7
4888	4.0	0.1	439	22.6	5.1

Bird No.	l-Thyroxine injected per bird per day (micrograms)	Thio-uracil in water (%)	Body Weight (gm.)	Thyroid Weight (mg.)	Thyroid Weight (mg.%)
6200	4.0	0.1	374	20.5	5.4
4890	4.0	0.1	378	19.8	5.2
6130	4.0	0.1	496	19.6	3.9
6178	4.0	0.1	389	10.0	2.5
Mean			451	16.0	3.7 ± 0.37
<u>Group VII</u>					
6175	6.0	0.1	580	13.0	2.0
6151	6.0	0.1	350	13.1	3.7
4884	6.0	0.1	524	23.4	4.2
6155	6.0	0.1	430	21.2	4.9
6159	6.0	0.1	527	9.4	1.7
4882	6.0	0.1	487	14.0	2.8
4885	6.0	0.1	570	11.8	2.0
6168	6.0	0.1	403	17.2	4.2
6162	6.0	0.1	441	11.3	2.5
4897	6.0	0.1	420	14.0	3.3
6170	6.0	0.1	456	18.0	3.9
Mean			472	15.1	3.1 ± 0.31
<u>Group VIII</u>					
6108	8.0	0.1	613	20.2	3.2
6138	8.0	0.1	529	16.4	3.2
6164	8.0	0.1	483	16.7	3.4
6113	8.0	0.1	428	16.4	3.8
6144	8.0	0.1	404	14.4	3.5
6115	8.0	0.1	446	15.6	3.4
6110	8.0	0.1	372	14.9	4.0
6124	8.0	0.1	434	14.3	3.2
6118	8.0	0.1	396	10.0	2.5
6140	8.0	0.1	378	13.0	3.4
Mean			448	15.2	3.35 ± 0.13

THYROID SECRETION RATE, TEMPERATURE - 105° F.

Bird No.	l-Thyroxine injected per bird per day (micrograms)	Thio-uracil in water (%)	Body Weight (gm.)	Thyroid Weight (mg.)	Thyroid Weight (mg.%)
<u>Group I</u>					
4391	--	--	297	23.1	7.7
4375	--	--	299	9.7	3.2
4313	--	--	337	8.2	2.4
7390	--	--	283	7.4	2.6
7387	--	--	401	17.5	4.3
4341	--	--	300	14.2	4.7
7353	--	--	337	8.1	2.4
4324	--	--	289	15.2	5.2
4305	--	--	305	13.6	4.4
4385	--	--	356	13.9	3.9
4303	--	--	310	12.0	3.8
4328	--	--	356	21.1	5.9
4381	--	--	284	10.3	3.6
4395	--	--	308	7.8	2.5
Mean			319	12.3	4.0 ± 0.27
<u>Group II</u>					
4355	--	0.1	261	14.0	5.3
7354	--	0.1	283	11.5	4.0
4340	--	0.1	410	11.0	2.6
7351	--	0.1	274	12.0	4.3
4397	--	0.1	371	22.0	5.9
7389	--	0.1	327	26.3	8.0
4367	--	0.1	257	18.9	7.3
4330	--	0.1	369	22.8	6.1
4351	--	0.1	314	11.0	3.5
4312	--	0.1	224	15.4	6.8
4348	--	0.1	338	15.9	4.7
4383	--	0.1	238	14.0	5.8
4356	--	0.1	317	12.8	4.0
4347	--	0.1	254	15.2	5.9
Mean			303	15.9	5.3 ± 0.40
<u>Group III</u>					
4398	0.5	0.1	225	3.6	1.6
4333	0.5	0.1	348	8.0	2.2
7372	0.5	0.1	310	12.1	3.9
4308	0.5	0.1	275	10.4	3.7
4342	0.5	0.1	277	13.0	4.6

Bird No.	l-Thyroxine injected per bird per day (micrograms)	Thio-uracil in water (%)	Body Weight (gm.)	Thyroid Weight (mg.)	Thyroid Weight (mg.%)
4396	0.5	0.1	298	20.3	6.8
7371	0.5	0.1	278	11.2	4.0
4387	0.5	0.1	237	13.8	5.8
7352	0.5	0.1	228	12.7	5.5
4315	0.5	0.1	314	14.0	4.4
4307	0.5	0.1	252	15.0	5.9
4349	0.5	0.1	371	8.0	2.1
4377	0.5	0.1	300	20.8	6.9
4373	0.5	0.1	250	9.2	3.6
Mean			283	12.3	4.3 \pm .45
<u>Group IV</u>					
7375	1.0	0.1	237	5.1	2.1
4379	1.0	0.1	280	8.2	2.9
4353	1.0	0.1	256	7.0	2.7
4366	1.0	0.1	288	25.7	8.9
4394	1.0	0.1	238	8.0	3.3
7379	1.0	0.1	333	16.3	4.8
7374	1.0	0.1	320	5.8	1.8
7381	1.0	0.1	215	5.1	2.3
4384	1.0	0.1	222	12.4	5.5
4301	1.0	0.1	199	7.9	3.9
7383	1.0	0.1	335	8.2	2.4
4329	1.0	0.1	312	11.4	3.6
4370	1.0	0.1	270	9.6	3.5
4374	1.0	0.1	312	6.6	2.1
4362	1.0	0.1	226	4.8	2.1
Mean			270	9.5	3.4 \pm 0.47
<u>Group V</u>					
4392	2.0	0.1	310	4.5	1.4
4369	2.0	0.1	361	6.3	1.7
4360	2.0	0.1	269	4.7	1.7
4389	2.0	0.1	316	6.0	1.8
7366	2.0	0.1	286	4.1	1.4
7382	2.0	0.1	270	10.3	3.8
4326	2.0	0.1	242	5.1	2.1
Mean			293	5.9	2.0 \pm 0.10

Bird No.	l-Thyroxine injected per bird per day (micrograms)	Thio-uracil in water (%)	Body Weight (gm.)	Thyroid Weight (mg.)	Thyroid Weight (mg.%)
<u>Group VI</u>					
4346	4.0	0.1	318	6.1	1.9
4390	4.0	0.1	353	5.6	1.5
4393	4.0	0.1	267	3.8	1.4
7363	4.0	0.1	272	5.1	1.8
4352	4.0	0.1	276	6.1	2.2
4309	4.0	0.1	166	4.0	2.4
7364	4.0	0.1	236	4.4	1.8
7362	4.0	0.1	309	5.0	1.6
Mean			275	5.0	1.8 ± 0.12
<u>Group VII</u>					
4380	6.0	0.1	291	5.0	1.7
7368	6.0	0.1	191	6.1	3.1
7388	6.0	0.1	346	6.8	1.9
7367	6.0	0.1	241	9.2	3.8
7386	6.0	0.1	304	5.0	1.6
4386	6.0	0.1	253	4.0	1.5
4350	6.0	0.1	285	5.6	1.9
4365	6.0	0.1	257	7.6	2.9
Mean			271	6.2	2.3 ± 0.33

APPENDIX B

APPENDIX B

WEIGHT GAINS, TEMPERATURE - 75° F.

Lot #1				Lot #2			
Bird No.	Weight on Arrival	Weight at 3 Weeks	Gain	Bird No.	Weight on Arrival	Weight at 3 Weeks	Gain
4894	37	312	274	4893	42	346	304
7399	37	346	309	7418	42	270	228
4898	40	317	277	7420	40	352	312
7400	45	352	307	7421	39	367	328
7398	39	377	338	7422	44	315	271
7397	43	356	313	7423	42	312	270
7396	42	326	284	7424	48	402	354
7394	50	340	290	7425	47	351	304
7395	42	367	325	7426	39	357	318
7393	42	332	290	7427	47	345	298
7392	42	308	260	7428	45	287	242
7391	42	363	321	7429	40	310	270
7401	40	316	321	7430	43	352	309
7402	41	355	314	7431	42	255	213
7403	47	333	286	7432	37	300	267
7404	38	303	265	7433	42	325	283
7405	40	303	263	7434	47	422	375
7406	40	332	292	7435	45	362	317
7407	44	363	321	7436	45	387	342
7408	41	357	316	7437	42	338	296
7410	47	367	320	7438	42	306	264
7411	41	341	300	7439	43	366	323
7413	42	367	325	7440	53	385	332
7414	41	376	335	7441	43	247	204
7415	42	336	294	7442	54	334	280
7416	40	329	289	7443	45	343	298
7417	41	351	310	7444	40	267	227
Mean	41.7	343.3	301.4	Mean	43.6	333.4	290

Lot #3				Lot #4			
Bird No.	Weight on Arrival	Weight at 3 Weeks	Gain	Bird No.	Weight on Arrival	Weight at 3 Weeks	Gain
4895	39	297	258	4892	37	368	331
7445	43	348	305	7472	39	312	273
7446	38	332	294	7474	34	295	251
7447	37	285	248	7476	42	240	198
7448	42	348	306	7477	42	335	293
7449	37	322	285	7478	38	338	300
7450	42	393	351	7479	43	358	325
7451	41	300	259	7480	45	351	306
7452	40	391	351	7481	42	354	312
7453	40	343	303	7482	44	365	321
7454	42	314	272	7482	44	365	321
7455	40	365	325	7483	41	370	329
7456	37	395	358	7483	41	370	329
7377	34	317	283	7484	47	373	327
7458	46	333	287	7484	47	373	327
7459	42	346	304	7485	42	333	291
7460	42	336	294	7485	42	333	291
7376	37	348	311	7486	42	366	322
7462	41	360	319	7486	42	366	322
7463	42	310	268	7487	42	351	309
7465	42	364	322	7487	42	351	309
7466	45	307	262	7488	43	372	329
7467	38	318	280	7488	43	372	329
7468	44	351	307	7489	40	277	237
7469	42	402	360	7489	40	277	237
7470	45	340	295	7490	47	360	313
7471	45	303	258	7490	47	360	313
	Mean 41.4	340	302.4	7491	41	340	299
	Mean of all lots 42	340.7	300	7491	41	340	299
				7492	42	383	341
				7492	42	383	341
				7493	42	365	323
				7493	42	365	323
				7494	40	398	358
				7494	40	398	358
				7495	37	350	313
				7495	37	350	313
				7496	42	373	331
				7496	42	373	331
				7497	42	358	316
				7497	42	358	316
				7498	43	315	272
				7498	43	315	272
				Mean 41.5	346	304.6	

WEIGHT GAINS, TEMPERATURE - 95° F.

Lot #1				Lot #2			
Bird No.	Weight on Arrival	Weight at 3 Weeks	Gain	Bird No.	Weight on Arrival	Weight at 3 Weeks	Gain
4876	35	233	198	6126	32	319	287
4877	34	297	263	6127	38	276	238
6103	36	275	239	6128	40	262	222
4878	38	334	296	6129	36	266	330
6105	36	290	254	6130	32	334	302
6106	32	315	283	6131	45	291	246
6107	37	287	250	6132	37	302	261
6108	35	327	292	47	34	299	265
6109	40	318	278	6134	37	252	215
6110	35	277	242	6135	37	263	216
6111	34	295	261	6137	36	253	217
6112	37	303	266	6138	42	297	255
6113	32	295	263	6139	37	307	270
6114	35	290	255	6140	43	287	244
6115	37	318	281	6142	39	262	233
6116	35	312	277	6143	35	295	260
6117	37	321	284	6144	37	288	251
6118	36	285	249	46	41	279	238
4879	37	331	294	6146	39	298	259
4880	34	275	241	6147	35	292	257
6122	34	275	241	45	37	278	241
6123	39	302	263	6150	36	231	195
6124	38	322	284				
6125	41	298	254				
Mean	36.1	299	262.8	Mean	37.7	284	245

Lot #3				Lot #4			
Bird No.	Weight on Arrival	Weight at 3 Weeks	Gain	Bird No.	Weight on Arrival	Weight at 3 Weeks	Gain
6151	35	230	195	6176	32	269	237
4881	40	237	197	6178	34	266	232
6153	34	285	251	6180	34	298	264
6154	39	268	229	6181	32	263	231
6155	34	262	228	4880	33	302	269
4882	39	273	234	6183	34	340	306
6157	31	280	249	6184	37	277	240
6159	41	313	272	4889	33	260	227
6158	35	301	266	4890	36	266	230
4897	42	299	251	6188	37	321	284
4883	32	292	260	6189	33	287	254
6162	35	303	268	6190	34	272	238
4884	34	291	257	4891	31	277	246
4885	40	325	285	6193	34	287	253
6165	38	275	237	6194	32	261	229
6166	34	320	286	6195	35	312	277
6167	32	307	275	6196	35	257	222
6168	35	280	245	6198	35	269	234
6169	32	296	264	6200	35	269	234
6170	38	327	289				
6171	35	295	260				
4886	41	333	292				
6173	35	309	274				
4887	33	339	306				
6175	34	317	283				
Mean	36.1	294	258.3	Mean	34.6	282	247.7
Mean of all lots	36.1	290	253.4				

WEIGHT GAINS, TEMPERATURE - 105° F.

Lot #1				Lot #2			
Bird No.	Weight on Arrival	Weight at 3 Weeks	Gain	Bird No.	Weight on Arrival	Weight at 3 Weeks	Gain
7363	42	185	143	7362	33	200	167
7379	33	203	170	4326	38	152	114
4303	35	297	172	4328	39	224	185
4305	32	200	168	4329	31	193	162
4307	37	174	137	4330	37	226	189
4308	37	177	140	4331	33	247	214
4309	32	125	93	7386	32	189	157
4312	37	134	97	4333	45	226	181
4313	34	194	160	7388	32	117	85
4315	37	197	160	7387	40	251	211
7381	35	134	96	4337	35	147	112
7382	34	164	130	7389	34	190	156
7383	32	197	145	4340	40	262	222
4322	41	145	104	4341	33	198	165
4324	34	180	146	4342	32	167	135
7366	38	194	156	4346	32	214	182
7364	37	158	121	4347	40	162	122
				4348	39	202	163
				4349	33	241	208
				4350	30	112	82
				7368	35	135	100
				7367	34	169	135
Mean	35.9	174.5	137.5	Mean	35.3	192	156.7

Lot #3				Lot #4			
Bird No.	Weight on Arrival	Weight at 3 Weeks	Gain	Bird No.	Weight on Arrival	Weight at 3 Weeks	Gain
4351	41	190	149	7390	32	172	140
4352	34	176	142	4377	36	184	148
4353	34	161	127	4379	33	178	145
4354	35	147	112	4380	34	187	153
4355	38	156	118	4381	37	165	128
4356	37	184	147	4382	39	203	164
7351	32	173	141	4383	35	165	130
4358	37	139	102	4384	32	127	95
4360	34	172	138	4385	38	223	185
4362	33	162	129	4386	40	152	112
4364	32	152	120	4387	32	138	106
4365	35	172	137	4389	37	208	171
4366	35	180	145	4390	38	241	203
4367	32	162	130	4391	31	181	150
7352	37	132	95	4392	40	188	148
4369	39	220	181	4393	42	196	154
4370	39	164	125	4394	32	142	110
7371	37	166	129	4395	34	185	151
7353	40	209	169	4396	33	171	138
4373	38	145	107	4397	37	235	198
4374	41	186	145	4398	37	185	148
4375	42	182	140	4400	37	196	159
7372	42	214	172	7375	37	151	114
7354	40	177	137	7374	35	201	166
7370	38	182	144				
Mean	36.7	172	135.5	Mean	36	182.2	146.5
Mean of all Lots	36	180	144				

VITA

Richard Wilford Heninger

Candidate for the Degree of

Master of Science

Thesis: THE EFFECT OF VARIOUS ENVIRONMENTAL TEMPERATURES ON
THE THYROID SECRETION RATE IN CHICKENS

Major Field: Physiology

Biographical Sketch:

Personal Data: Born September 28, 1931, at Raymond,
Alberta, Canada, the son of Wilford A. and Mabel
K. Heninger.

Education: Attended grade and high school at Raymond,
Alberta, Canada, and graduated from Raymond High
School in 1949; received the Bachelor of Science
degree from Brigham Young University in June,
1957; completed the requirements for the degree of
Master of Science at Oklahoma State University in
May, 1959.

Professional experience: Employed as a laboratory
instructor in Zoology at Brigham Young University
during 1955 and 1957; in 1957 appointed assistant
in the Department of Physiology and Pharmacology
at Oklahoma State University, Stillwater,
Oklahoma.

Honorary Societies: Associate member of Sigma Xi.