THE EFFECT OF SODIUM CHLORIDE ON THE

THYROID AND KIDNEY FUNCTION

OF THE CHICKEN

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CHAPTER I

INTRODUCTION

Recently, investigators (2,9,10,12,13,14,23,27) have shown experimentally that both chronic and acute sodium chloride (NaCl) administration to mammals led to an increased renal excretion of iodide; this, in turn, caused a lowering of the circulating levels of iodide in the blood.

In the literature that deals with the effect of NaCl on the renal excretion of iodide by the kidney, mammals were used exclusively. It was the purpose of this study to obtain data, from a comparative standpoint, on the effect of NaCl on the chicken's thyroid and kidney. If the renal excretion of iodide in chickens is affected by NaCl concentrations in the diet, then this study may offer an explanation as to why some investigators (28) were able to produce thyroid enlargement in chickens by feeding a low iodine diet while other investigators (20) were not.

CHAPTER II

LITERATURE REVIEW

Low Dietary Iodine

Many investigators (2,17,18,19,21) have shown by experimental methods that a deficiency of dietary iodine was responsible for goiter. Levine, Remington and von Kolnitz (17) were probably the first to succeed in developing a diet that was suitable for goiter production in the rat. Their diet was low in iodine content (15 micrograms of iodine/kilo of feed). With this diet, the rats had a daily intake of 0.14 micrograms of iodine and goiters were produced in five weeks of feeding.

Money, Rall and Rawson (19) produced goiters in rats with a low iodine diet fed for 106 days. They suggested that rapid thyroid growth would not take place until the thyroidal levels of iodine were depleted to a critical level. They based their hypothesis on data collected from rats fed a diet which had an iodine content of 45 micrograms of iodine/kilo of feed. The control animals were fed Purina Chow and had a mean thyroid weight of 18.7 mg with the thyroids containing 26.7 micrograms of iodine. After 34 days of receiving the low iodine diet, the experimental rats had thyroids which had a mean weight of 17.5 mg and contained 12.7 micrograms of iodine. After 106 days of receiving the experimental diet, the rats had thyroids which had significantly increased in weight to 25.3 mg and contained only 5.3 micrograms of iodine. At the end of 206 days, the thyroids of the experimental animals had a mean weight of

55.5 mg and contained only 2.8 micrograms of iodine. The investigators concluded from those data that the thyroid was activated to enlarge only after the thyroid was depleted of iodine to about 50 percent of the control level. Their theory was supported by the observations of Astwood and Bissell (1), who found that rapid thyroid growth occured only after thiouracil had depleted the iodine concentrations of the thyroid to a low level.

Maloof, Dobyns and Vickery (18) used a diet that contained 90 micrograms of iodide/kilo of feed and found an increase in both thyroid size and radioiodine uptake in rats that had been fed the diet only 11 days; whereas, Money, Rall and Rawson (19) reported that thyroid enlargement was not apparent until after 106 days of feeding a low iodine diet.

Some investigators were not able to produce experimental goiters by the mere feeding of a low iodine diet. Van Middlesworth (26) fed a low iodine diet which contained 20-30 micrograms of iodine/kilo of feed to rats for 18 months and observed that the thyroids remained a normal size. When the rats received 0.5 mg thiouracil per day in the feed, the thyroid size doubled. When the rats received 0.5 mg of thiouracil and 100 micrograms of iodide per day the thyroids were normal in size. Van Middlesworth concluded from these data that it was difficult to produce symptoms of iodine deficiency by a low iodine diet unless a goitrogen was employed in the experiment and even then the goitrogen could be ineffective if the iodine content of the diet was too high.

Halmi (7) fed a low iodine diet for 19-20 days to rats and found that their thyroids failed to exhibit any morphological signs of stimulation. However, the thyroid:serum gradients of the radioiodide concentrations (T/S) were greatly increased by the low iodine treatment. The data indicated to Halmi that the thyroidal iodide concentrating mechanisms of the animals that were fed the low iodine diets were activated to an above-normal level even though there were no goiters produced.

The experimental observations that were reported above are but a small sampling of the actual work that dealt with goiter production by low levels of dietary iodine. The experiments that are reported are used as examples to show that it is now a generally accepted fact that the thyroid cannot function properly without adequate supplies of iodine, and they are also meant to illustrate that the results obtained by experimental methods are often quite variable. This variability is interpreted by some investigators to mean that there may be other factors involved in experimental goiter production besides a low iodine concentration of the diet. Some dietary factors that have been shown to produce goiters in experimental animals are: 1-5-vinyl-2thiooxazolidone found in the seeds of rape, cabbage, kale, and turnips; soybean oil meal; sulfonamides; thiourea; thiocyanate; and the iodide ion in large doses. The goitrogenic action of inorganic salts has been suspected and investigated (2,8,9,10,11,12,16,21,28).

Dietary Sodium Chloride

Remington (21) was one of the first to investigate the role of NaCl in thyroid physiology. He studied the effect of a varied NaCl concentration (either 0%, 1%, 2%, or 4% NaCl) in the diet of rats. He observed that the rats which were deprived of the salt had thyroids with a smaller absolute weight. But because these rats grew poorly, the differences due to a NaCl treatment disappeared when the thyroid weight was expressed as mg/100 gm body weight. Remington concluded that NaCl had no effect on the thyroid gland in spite of the fact that depletion of NaCl from the diet caused a threefold increase in the iodine content of the thyroid gland.

Later, when Axelrad, Leblond and Isler (2) were investigating the possibility of a positive goitrogenic factor in the low iodine diet that they were feeding to mice, they found that NaCl had an effect on the thyroid gland. They observed that omission of NaCl caused a decrease in the thyroid weight of mice fed a low iodine diet and also caused a decreased uptake of radioiodine by the thyroid. When they studied the relationship between the effects of dietary iodine and chloride on thyroid morphology, they found that activation of the thyroid (increased follicular cell size and decreased colloid content of the follicle) by NaCl decreased with an increased iodine intake. At the lowest level of iodine intake, the addition of NaCl to the diet caused an increase in thyroid weight. At the higher levels of dietary iodine, the addition of NaCl to the diet increased the radioiodine uptake by the thyroid but did not alter the thyroid weight.

Isler, Leblond and Axelrad (12) investigated the mechanism of thyroid stimulation by NaCl in the mouse. They found that an intraperitoneal injection of 30 mg NaCl increased the urinary loss of iodide by a factor of 6.5. This was in agreement with the findings of Riggs (23), Walser and Rahill (27), and Halmi _et al._ (8). Riggs found that administration of NaCl to dogs resulted in a prompt decrease in plasma iodide and an increase in the renal clearance of iodide. Halmi _et al._ found that the renal clearance of iodide in rats was greatly inhanced in rats by NaCl, and suggested that the tubular reabsorption of the iodide, in addition to being a passive process, was also an active transport which could be saturated by iodide and that chloride was possibly a competitor for loci on the iodide carrier. Walser and Rahill observed that infusion of saline in dogs resulted in a 40-fold increase in iodide clearance by the kidney. Isler (9) investigated the effect of several different ions on the urinary excretion of radioiodide in mice. He found that NaCl-treated mice excreted 22 times the amount of iodide excreted by the control animals. He noted that both sodium and chloride ions caused an increased excretion of iodide by the kidney. However, his data also indicated that a similar effect could be produced by salts other than NaCl. He observed that only the salts that were highly dissociated would increase the urinary excretion of radioiodide. Sodium, potassium, rubidium, chloride, bromide, and iodide ions would increase the excretion of radioiodide, while lithium, calcium, ammonium, carbonate, and acetate ions did not. He concluded that the renal excretion of iodide was increased whenever the ionic concentration of the plasma was increased.

Isler (10) investigated the effect of hormonal factors on ioduria and found that vasopressin and oxytocin increased the renal excretion of iodide in acute experiments, but vasopressin administered chronically did not stimulate the thyroid as did a chronic administration of NaCl. He also found that exogenous adrenal hormones had little effect on the renal clearance of iodide of intact mice. Experiments by Leveque (16) have shown that NaCl caused a release of vasopressin-containing neurosevretory material in the hypothalamus. Therefore, NaCl might have caused the increase in the iodide excretion by increasing the release of the posterior pituitary hormones. But Isler (11) perfused isolated rabbit kidneys with radioiodide-containing fluids with or without added NaCl and found that the NaCl did actually increase the iodide clearance in the isolated kidney. These data did not exclude an effect of NaCl mediated through the pituitary, but they do show that NaCl had an effect of its own on the kidney.

It seems fairly clear from the mammalian experimental data that NaCl directly modifies the clearance of iodide from the blood by the kidney, causes an abnormal loss of iodide into the urine, and restricts the amount of iodide available to the thyroid. A diminished rate of synthesis and release of the thyroid hormones causes the pituitary to respond by releasing increased amounts of thyrotropic hormone into the blood which stimulates the thyroid gland to increased growth and iodine uptake.

Kidney Function in the Chicken

Although no work was found in which the effect of NaCl on iodide excretion by the chicken kidney was studied, there has been some work reported on the effects of NaCl on the glomerular filtration rate (GFR) of the kidney of the chicken.

The capacity of the bird's kidney to excrete hypertonic urine was established by Korr (14). He found that after an injection of hypertonic NaCl solution the urine flow and osmotic pressure of the urine briefly increased and thereafter decreased to a low level. He concluded that the reduced filtration rate and reduced urine flow were due to a rapid dehydration and loss of body water which occured initially during the brief diuresis. His work also showed that the chicken has a maximum urine concentration capacity of about twice the osmolality of the blood. The chicken kidney is not as efficient in excreting salt and conserving water as the mammalian kidney because the dog can concentrate the urine eight times the plasma concentration (6); the rat can concentrate the urine ten times the concentration of the plasma (6).

Dantzler (3) has shown that a hypertonic NaCl infusion into chickens caused a decrease in GFR. He noted that when the chicken was subjected to a severe dehydration or a salt load, the rising plasma osmolality was inhibited. He stated that the chicken could accomplish the inhibition by the production of a hypertonic urine, but because the chicken cannot concentrate salt in the urine to more than twice that of the plasma (14), a hypertonic urine production is probably not the primary mechanism involved. Some birds (mainly marine birds) can inhibit the rising plasma osmolality by extrarenal excretion of salt via a nasal salt gland, but the chicken does not have a nasal salt gland. Dantzler has purposed that the chicken reduces what would otherwise be an increase in plasma osmolality following a salt loading or severe dehydration by actually decreasing the number of functional nephrons and thereby conserving water until more water could be ingested. He found that the GFR decreased as plasma osmolality increased. His data from studies of the tubular maxima for the reabsorption of glucose and secretion of para-aminohippurate support the concept that changes in the filtration rate in chickens, like those in amphibians (4) and reptiles (15) result from changes in the number of functional nephrons. He states that a decrease in GFR with the rising plasma osmolality probably resulted from a decrease in blood supply to the nephrons.

CHAPTER III

MATERIALS AND METHODS

Diets

For Experiment I, a basal, low-iodine (6 micrograms of iodine/100 gm feed), low-chloride ration was prepared (Table I). The chloride content of the basal ration was not determined by analysis, but NaCl was not added and the diet was considered to be a low-chloride diet. Potassium iodide, sodium chloride and sodium carbonate were added to the basal ration in various combinations to produce four different diets (Table II). The four different diets when compared to a "normal" broiler ration were considered to be either a low-iodine, low-chloride; low-iodine, high-chloride; high-iodine, low-chloride; or a high-iodine, high-chloride diet.

In Experiments II and III, two rations were used. The control diet was a broiler ration; the experimental diet was the control diet with 5 lbs of NaCl added per 100 lbs of feed (Table III).

Analytical Procedures

Determination of the T/S. The T/S is a concentration factor which represents the equilibrium between the influx and efflux of iodide in the thyroid in which the organification of iodine has been inhibited. It is:

$$\frac{\text{concentration of iodide/mg thyroid tissue (wet weight)}}{\text{concentration of iodine/microliter of serum}}.$$

TABLE I

BASAL RATION* FOR EXPERIMENT I

Ingredient		Percent
Corn (Wisconsin, "low-iodine")		46.39
Wheat germ meal		13.53
Soybean oil meal (50%)		9.02
Gelatin		9.02
Fat		6.42
Dicalcium phosphate		4.64
Yeast (dried brewer's)		4.51
Filler		3.00
Calcium carbonate		2.14
Vitamin concentrate 2-59		0.89
d-l Methionine		0.28
Vitamin "A"		0.11
Vitamin "D"		0.04
$MnSO_4 \cdot H_2O$		0.02
	TOTAL	100.01

*This ration had an iodine concentration of 6.0 micrograms of iodine per 100 grams of ration (determined by A. L. Chaney Chemical Laboratory, Glendale, California).

TABLE II

PERCENTAGE COMPOSITION OF DIETS FOR EXPERIMENT I

Type of Diet	Basal Ration	$NaCO_3$	Corn*	NaCl**
Low-iodine, low-chloride	97.0	0.4033	2.55	----
Low-iodine, high-chloride	97.0	----	----	3.00
High-iodine, low-chloride	97.0	0.4033	2.55	----
High-iodine, high-chloride	97.0	----	----	3.00

*Wisconsin, "low-iodine".

**iodine-free (J. T. Baker Chemical Co. Analytical Reagent Grade).

A microliter of serum was used because its weight is roughly equal to 1 mg of tissue.

To determine the T/S, each bird was injected intraperitoneally with 5 mg 1-methyl-2-mercaptoimidazole (Lilly and Co.) per 100 gm of body weight to block the organic binding of iodide. After 30 minutes, each bird was given an intraperitoneal injection of 2.06 microcuries of radio-iodide (as $Na^{131}I$ from Iso/Serve Inc. and used without further purification). After an equilibration period of 2.5-3.0 hours, 8 ml of blood were withdrawn from each bird by intracardial puncture and placed in a test tube to clot overnight at room temperature. The birds were decapitated with a guillotine and the blood drained from their bodies. The thyroids were dissected out, trimmed of all excess tissue and weighed to

TABLE III

PERCENTAGE COMPOSITION OF DIETS FOR EXPERIMENTS II AND III

Control Ration	
Ingredient	Percent
Corn, ground	28.57
Soybean meal	17.20
Fat (tallow)	8.33
Milo, ground	7.89
Oatmeal feed	7.89
Meat and bone scraps	6.88
Fish meal	5.90
Wheat bran	3.95
Alfalfa meal	2.46
Wheat shorts	1.97
Dicalcium phosphate	1.94
Calcium carbonate	1.67
Yeast culture	0.99
Dried whey	0.99
Gelatin	0.74
Salt	0.50
VMC-60	0.50
Blood meal	0.49
d-l Methionine	0.15
TOTAL	100.00
High-chloride Diet	
Five pounds of sodium chloride were added to 100 pounds of the control ration.	

the nearest 0.01 mg on a precision torque balance. The thyroids were placed in a test tube that contained 1 ml of 0.04N NaOH. The tubes that contained the blood samples were centrifuged at 2000 RPM for 15 minutes; 0.1 ml of serum was removed from each tube and placed in a test tube that contained 1 ml of 0.04N NaOH. The radioactivities in the thyroids and serum samples were determined in a well-type, crystal scintillation detector (Baird Atomic, Model 810 with multiscaler II, Model 132). The data were corrected for background and expressed as T/S.

Determination of the 24-hour Uptake of ^{131}I by the Thyroid. The birds were injected intraperitoneally with 3.0 microcuries of radioiodide per 100 gm of body weight. Twenty-four hours later they were killed by decapitation and the blood was drained from their bodies. The thyroids were removed, trimmed, weighed and placed in a test tube that contained 1 ml of 0.04N NaOH. Two, 25-microliter aliquots of the ^{131}I-injection solution standards were each placed in a test tube that contained 1 ml of 0.04N NaOH. The radioactivities of the thyroids and standards were determined in a well-type, crystal scintillation detector. The data were corrected for background and expressed as the percentage uptake by the thyroids in 24-hours of the injected dose.

Determination of the Renal Clearance of Iodide. The birds were anesthetized with sodium pentabarbitol (0.2-0.3 ml of 65 mg sodium pentabarbitol per ml) via an intravenous injection in the right, subcutaneous breast vein. Bilateral incisions were made into the peritoneal cavity 0.5 inch dorso-laterally from the cloacal opening. The ureters were hemisectioned by carefully cutting them with a sharp scapel. Polyethylene cannulas (Clay Adam's Intramedic, PE 90 or PE 190 polyethylene tubing cut diagonally at six inch intervals) were inserted proximally 1" inch through the incision in the ureters and tied into place with cotton sewing thread. The birds were injected with 1.0-3.0 microcuries of ^{131}I intravenously in the left subcutaneous breast vein. The urine was collected for thirty minutes in 15 ml graduated conical centrifuge tubes. During this period, the birds were kept lightly anesthetized with ether. At the end of the first 15 minute period of urine collection, a blood sample of 1.0-2.0 ml was taken by intracardial puncture and placed in a stoppered test tube to clot overnight at room temperature. The volume of urine excreted in 30 minutes was recorded and a measured aliquote of 0.18-0.5 ml was transferred to a test tube. After the radioactivities in the urine and serum samples were determined, they were corrected for background and physical decay and expressed as counts per minute per ml of serum. The renal clearance of iodide (ml of serum cleared of iodide per minute) was calculated by dividing the urine excretion rate (counts per minute per minute) by the serum concentration (counts per minute per ml). The urine and remaining serum samples were frozen and saved for the chloride determinations.

Determination of the Renal Clearance of Chloride. The chloride concentrations in the urine and sera were determined by a method based on the procedure of Schales and Schales (24). A chloride determination kit (Sigma Chemical Co., Kit 830-T) was used for the determinations. The chloride excretion rate was expressed as meq Cl^-/minute and the chloride concentration in the serum was expressed as meq Cl^-/ml of serum. The renal clearance of chloride (ml of serum cleared of chloride per minute) was calculated by dividing the excretion rate of chloride in the urine by the concentration of chloride in the serum.

Histological Studies. Thyroids, kidneys, and ureters were fixed in Bouin's solution, stained with Periodic Acid Schiff's stain plus hematoxylin and examined for morphological changes.

Experimental Procedures

Experiment I. Each of four diets (Table II) was fed 5-6 weeks to a group of 45 White Leghorn chicks. In each group, 10 birds were used to determine the T/S, 10 were used to determine the 24-hour uptake of radioiodine by the thyroid, 10 were used to determine the renal clearances of iodide and chloride, and 5 were used for histological studies.

Experiment II. In this experiment, the effects of chronic and acute NaCl treatment were studied. A broiler ration (Table III) and the experimental high-chloride diet (Table III) were fed to 85 White Leghorn chicks for 5-9 weeks. In each group, 10 birds were used to determine the 24-hour uptake of radioiodine by the thyroid, 10 were used to determine the renal clearances of iodide and chloride, and 5 were used to study the histology of the thyroids, kidneys and ureters.

The acute studies were done on 35 chicks from the group that received the broiler ration. The experimental chicks were injected intraveneously in the left wing vein with a NaCl solution (4.375% NaCl/100 gm of body weight) and the control chicks received an injection of distilled water (1 ml/100 gm of body weight). The renal clearances of iodide and chloride were determined by methods described in this chapter.

Experiment III. This experiment was a replication of Experiment II. The same diets and methods were used, except that there were no acute studies in this experiment.

CHAPTER IV

RESULTS

Experiment I

The effects of the addition of sodium chloride (3.0%) to either a low-iodine, low-chloride diet or to a high-iodine, low-chloride diet on the thyroid and kidney function were studied.

Thyroidal Effects of Sodium Chloride. From Table IV, it can be seen that when the dietary iodine was low, the T/S of the chicks that received the diet with the added NaCl was 345.4 ± 40.68 as compared to 351.4 ± 26.54 for the chicks that received the low-iodine, low-chloride diet. When the dietary iodine was high, the T/S of the chicks that were fed the high-iodine, high-chloride diet was 12.4 ± 1.69 as compared to 13.6 ± 2.22 for the T/S of the chicks that received the high-iodine, low-chloride diet. There was no difference in the T/S due to the addition of the NaCl but there was a big difference in the T/S of the chicks that received the low-iodine diets and the T/S of the chicks that received the high-iodine diets.

Table IV shows that NaCl caused a significant ($P < 0.005$) decrease in the 24-hour uptake of thyroidal radioiodine from $11.14 \pm 0.30\%$ in the chicks maintained on a low-iodine, low-chloride diet to $9.70 \pm 0.26\%$ in the chicks grown on the low-iodine, high-chloride diet. When the dietary iodine was high, the addition of NaCl to the high-iodine, low-chloride

TABLE IV

EXPERIMENT I: THE EFFECTS OF HIGH DIETARY SODIUM CHLORIDE ON THE RENAL CLEARANCES OF RADIOIODIDE AND CHLORIDE, T/S, 24-HOUR UPTAKE OF RADIOIODINE, AND THYROID WEIGHT OF CHICKS GROWN ON HIGH AND LOW LEVELS OF DIETARY IODINE

	Renal clearance of iodide (ml/min)	Renal clearance of chloride (ml/min)	T/S	24-hour uptake of radioiodine (percent of dose)	Thyroid weight (mg%)
Low-iodine, low-chloride diet	0.403±0.003[a]	0.01±0.01	351.4±26.54	11.14±0.30*	4.98±0.86
Low-iodine, high-chloride diet	0.67±0.12	0.04±0.02	345.3±40.68	9.70±0.26	5.45±0.92
High-iodine, low-chloride diet	0.50±0.003	0.02±0.03	13.6± 2.22	0.52±0.07	5.54±1.58
High-iodine, high-chloride diet	0.75±0.18	0.05±0.03	12.4± 1.69	0.37±0.10	6.45±1.70

[a]Mean ± the standard error of the mean.

*$P<0.005$, birds fed the low-iodine, low-chloride diet compared with the birds fed the low-iodine, high-chloride diet.

diet caused a non-significant decrease in the 24-hour uptake from 0.52± 0.07% to 0.37±0.10%.

The effect of NaCl on the thyroids weights of birds grown on high and low-iodine diets are shown in Table IV. The thyroid weights of birds grown on a low-iodine diet that was also low in chloride content were not significantly changed by the addition of NaCl to the diet. The chicks that received the NaCl in their diet had thyroids that weighed 5.45±0.92 mg% as compared to 4.98±0.86 mg% for the thyroid weights of the chicks that received only the low-iodine, low-chloride diet. When NaCl was added to a high-iodine diet, the thyroid weights were again not effected significantly. The NaCl-treated chicks had thyroids that weighed 6.45±1.70 mg% and the chicks that received the high-iodine, low-chloride diet had thyroids that weighed 5.54±1.58 mg%.

Renal Effects of Sodium Chloride. In Table IV, it is shown that the renal clearance of iodide was increased (statistically non-significant) by the addition of NaCl to both the high and low-iodine diets. The NaCl caused an increased renal clearance of iodide from 0.403±0.003 ml/min in the low-iodine, low-chloride fed birds to 0.67±0.12 ml/min in the chicks that received the low-iodine, high-chloride diet. Sodium chloride also increased the renal clearance of iodide in the chicks that were fed the high-iodine, low-chloride diet from 0.50±0.003 ml/min to 0.75±0.18 ml/min for the renal clearance of the chicks that were fed the high-iodine, high-chloride diet.

The renal clearance of chloride was increased by the addition of NaCl to both the high and low-iodine diets. As indicated by Table IV, the renal clearance of chloride by the chicks fed the low-iodine diet was increased by the addition of NaCl from 0.01±0.01 ml/min to 0.04±0.02

ml/min. The renal clearance of chloride by the chicks that were receiving a high-iodine, low-chloride diet was increased by the addition of NaCl from 0.02±0.03 ml/min to 0.05±0.03 ml/min.

Histological Examination. Examination of the slides of the thyroid, kidney and ureter tissue revealed no detectable morphological change in any of the tissues in this or either of the two following experiments.

Experiment II

In this experiment, the effects of a high level (5.0%) of dietary sodium chloride and the effects of an acute injection of NaCl (4.375% NaCl/100 gm of body weight) on thyroid and kidney function were studied.

Thyroidal Effects of Dietary Sodium Chloride. As indicated in Table V, NaCl caused a significant decrease in the 24-hour uptake ($P<0.001$) of radioiodine by the thyroid. The percent uptake of the injected dose of radioiodide was decreased by NaCl from 8.88±2.24% in the control chicks to 3.93±1.01% in the NaCl-treated chicks.

Table V shows that the thyroid weight was significantly ($P<0.05$) reduced from 5.68±0.89 mg% in the controls to 4.05±0.52 mg% in the NaCl-treated chicks.

Renal Effects of Dietary and Acute Sodium Chloride. From Table V, it can be seen that the renal clearance of iodide was decreased in the chicks that received NaCl in their diet. The renal clearance of iodide in the control chicks was 0.70±0.26 ml/min as compared to 0.51±0.16 ml/min for the renal clearance of iodide in the NaCl-treated chicks. However, from Table VI it can be seen that injected NaCl did not have any effect on the renal clearance of iodide. The renal clearance of iodide

TABLE V

EXPERIMENT II: THE EFFECTS OF HIGH DIETARY SODIUM CHLORIDE ON THE RENAL CLEARANCES OF IODIDE AND CHLORIDE, 24-HOUR UPTAKE OF RADIOIODINE, AND THYROID WEIGHT

	Renal clearance of iodide (ml/min)	Renal clearance of chloride (ml/min)	24-hour uptake (% of dose)	Thyroid weight (mg%)
Control diet plus NaCl	0.51±0.16[a]	0.06±0.06	3.93±1.01*	4.05±0.52**
Control diet	0.70±0.26	0.06±0.03	8.88±2.24	5.68±0.89

[a]Mean ± the standard error of the mean.

*$P<0.001$, compared to the controls.

**$P<0.05$, compared to the controls.

TABLE VI

EXPERIMENT II: THE EFFECTS OF ACUTELY INJECTED SODIUM CHLORIDE ON THE RENAL CLEARANCES OF IODIDE AND CHLORIDE

	Renal clearance of iodide (ml/min)	Renal clearance of chloride (ml/min)
Injection of NaCl (first thirty min)	1.04±0.42[a]	0.12±0.07
Injection of dist. H_2O (first thirty min)	1.05±0.81	0.07±0.05
Injection of NaCl (second thirty min)	1.43±0.94	0.11±0.14
Injection of dist. H_2O (second thirty min)	1.12±0.71	0.02±0.02

[a]Mean ± the standard error of the mean.

in the acutely NaCl-treated chicks was 1.04±0.42 ml/min for the first 30 minute period of urine collection and 1.43±0.94 ml/min during the second 30 minute period of urine collection. The control values were 1.05±0.81 ml/min for the first period and 1.12±0.71 ml/min for the second period of collection.

The addition of NaCl to the diet did not change the renal clearance of chloride from the control value. The mean renal clearance of the chronically NaCl-treated chicks was 0.06±0.06 ml/min as compared to 0.06±0.03 ml/min in the control chicks. However, the acutely injected NaCl did cause an increase (statistically non-significant) in the renal clearance of chloride above the control values in both periods of collection. The NaCl treated chicks had a mean renal clearance of the chloride during the first period of collection that was 0.12±0.07 ml/min, while the mean renal clearance of the controls during the same collection period was 0.07±0.05 ml/min. During the second period of urine collection, the mean renal clearance of the NaCl-treated chicks was 0.11±0.14 ml/min as compared to the control value of 0.02±0.02 ml/min.

Experiment III

This experiment was designed to replicate Experiment II, except there were no acute studies associated with this experiment.

Thyroidal Effects of Sodium Chloride. From Table VII it can be seen that the addition of NaCl to the control diet caused a significant ($P<0.005$) decrease in the 24-hour uptake of radioiodine by the thyroid. The 24-hour uptake in the NaCl-treated chicks was 6.32±0.21% of the injected dose while the controls had a mean 24-hour uptake of 8.65±0.67% of the injected dose. The addition of NaCl to the control diet also

TABLE VII

THE EFFECTS OF HIGH DIETARY SODIUM CHLORIDE ON THE RENAL CLEARANCES OF IODIDE AND CHLORIDE, 24-HOUR UPTAKE OF RADIOIODINE, AND THYROID WEIGHT

	Renal clearance of iodide (ml/min)	Renal clearance of chloride (ml/min)	24-hour uptake (% of dose)	Thyroid weight (mg%)
Control diet plus NaCl	0.57±0.27[a*]	0.05±0.01	6.32±0.21[**]	5.88±1.16
Control diet	0.83±0.15	0.03±0.02	8.65±0.67	6.72±0.96

[a]Mean ± the standard error of the mean.

[*]$P<0.05$, compared to the controls.

[**]$P<0.005$, compared to the controls.

caused a decrease in the mean thyroid weight from 6.72±0.96% in the controls to 5.88±1.16 mg% in the NaCl-treated chicks.

Renal Effects of Sodium Chloride. The NaCl caused a significant (P 0.05) decrease in the renal clearance of iodide from 0.83±0.15 ml/min in the controls to 0.57±0.27 ml/min in the NaCl-treated chicks. It also caused an increase (statistically non-significant) in the renal clearance of chloride. The NaCl-treated chicks had a mean renal clearance for chloride of 0.05±0.01 ml/min as compared to 0.03±0.02 ml/min for the mean renal clearance of chloride in the control chicks.

CHAPTER V

DISCUSSION

The effect of NaCl on the renal excretion of iodide has been reported in several mammalian species (2,8,9,10,12,13,23), but no reports of iodide excretion in the chicken were found in the literature. The results reported by these workers indicate that the renal excretion of iodide in mammals was increased by either an acute or a chronic administration of NaCl. This increase, in turn, may have caused a change in thyroidal function as well as morphology. Investigators (2,9,11,12,29) have reported that when NaCl was administered to animals maintained on a low-iodine diet, the animals that received the NaCl had goiters which were larger than the goiters of the animals which were maintained on a low-iodine diet. The investigators concluded that NaCl increased the urinary loss of iodide which, in turn, lowered the plasma level of iodide. As a result, less iodide was available to the thyroid gland, and less thyroid hormone was secreted. The pituitary responded to the decrease in circulating levels of thyroid hormones by releasing more thyrotrophic hormone which stimulated the thyroid to grow and increase its activities. Other investigators (8,23) have reported that NaCl administration to animals maintained on diets which contained adequate amounts of iodine for normal thyroid function also caused an increased renal excretion of iodide.

Effect of Sodium Chloride on Renal Function

In the present study, the values obtained for the renal clearance of iodide in chicks indicate that it is decreased by chronic NaCl administration. This is an effect that is opposite from that reported in mammalian species.

When the ability of chickens to excrete a hyperosmotic urine is compared to that of mammals, it can be seen that the chicken is somewhat limited in its ability to excrete salt in the urine. The chicken can maximally concentrate the urine to about twice the osmolality of the blood (14) as compared to eight times in the dog (6) and ten times in the rat (6). When chickens were subjected to a salt load, the resulting increase in plasma osmoality must be prevented or inhibited. They could do this by the production of hyperosmotic urine, decreasing the glomerular filtration rate, or by extrarenal excretion of salt. However, chickens are not capable of extrarenal salt excretion as are some marine birds (3) and since they can only concentrate their urine to twice that of the plasma, they have to resort to decreasing the number of functioning nephrons and conserving water (3).

Because the chicken cannot concentrate NaCl as mammals do and because they react to salt loads by actually decreasing the number of functional nephrons, it follows that when the number of functional nephrons is decreased, the renal clearance of iodide may also be decreased. This may be a possible explanation for the decrease in the iodide excretion in chickens during NaCl administration and not an increase in iodide excretion as was reported in mammalian species. Chronic feeding of a high NaCl diet would result in a decrease in the number of functional nephrons until the plasma could be diluted by ingested water. The kidney would return toward normal until the plasma osmolality would begin to rise again due to the ingestion of more of the high NaCl diet.

When sodium chloride was injected acutely, no differences in renal clearance of iodide were found from control values. This was an unexpected result because there was a decrease in clearance in the chronic experiments (except for Experiment I in which there was no difference and was thought to be due to inexperience of the investigator). The fact that there were no differences in the acute study may be explained by the fact that the data were collected from 0-1 hour immediately following a single NaCl-injection. Perhaps the high concentration of NaCl must be maintained to the kidneys for a sustained period of time (by infusion or successive injections) before a difference in renal function can be detected.

Effect of Sodium Chloride on Thyroid Function

Since the effect of sodium chloride on the chicken kidney was opposite to that reported in mammals, one would expect that chronic sodium chloride administration might also have an opposite effect on the thyroid than that reported in mammals. The thyroidal uptake of radioiodine in the experimental chicks in this study was decreased, whereas, the thyroidal uptake in mammalian experiments was increased by a chronic NaCl-treatment (2,29).

In mammals, it was thought that NaCl caused an increased excretion of iodide which lead to a decreased availability of iodide to the thyroid gland which over a period of time resulted in a stimulation of the thyroid. However, when radioiodide was injected into NaCl-treated chicks, a decreased uptake of the injected dose by the thyroids was observed. Because the kidneys may be clearing smaller quantities of iodide from the plasma due to the influence of NaCl, the plasma levels of iodide may be higher in the NaCl-treated chicks. When the radioiodide was injected into the NaCl-treated chicks, the specific activity of the radioiodide of the blood was actually much lower than the specific activity of the radioiodide in the blood of the control chicks. This resulted in data that could be interpreted in one of two ways. The most apparent interpretation would be that NaCl caused an inhibition of uptake of radioiodine by the thyroid but this is unlikely because these were chronic experiments and one might expect a change in thyroid morphology over a long period of time. A change in thyroid morphology was not indicated by the concurrent histological studies. The second and most likely interpretation was that the decrease in uptake of radioiodine was actually due to a difference of specific activities. This theory, unfortunately, cannot be proven without measuring the iodide concentrations of the blood and thyroid and will therefore remain a theory until further experimentation.

In the acute mammalian experiments (9,11,12), treatment with NaCl resulted in decreased uptake of radioiodine by the thyroid, whereas, the uptake of radioiodine was increased by a chronic NaCl-treatment (2,29). This decrease in uptake of radioiodine in the acute experiments and the increase in uptake of radioiodine in the chronic administration of NaCl to chicks caused a decreased uptake of the injected radioiodine. This indicates that their thyroids were not in a state of stimulation as were the thyroids of the mammals in the reported literature. Also the fact that there was no morphological change due to a NaCl-treatment indicates that the thyroids were not being stimulated by thyrotropin from the pituitary or by any other means. This also lends support to the renal clearance data.

Effect of Sodium Chloride on Thyroid Weight

The data from this study indicate that the chronic NaCl administration to chicks leads to a decrease in thyroid weight. The literature was searched for a similar observation by other investigators, but none could be found. A possible explanation for a decrease in thyroid weight due to a chronic NaCl-treatment cannot be presented at this time.

CHAPTER VI

SUMMARY AND CONCLUSIONS

White Leghorn chicks were fed diets with added NaCl or they were injected with a NaCl solution to determine if NaCl causes an increase in the renal clearance of iodide in chickens as it does in mammals.

The chronic feeding of NaCl caused a decrease in the renal clearance of radioiodide, a decrease in thyroidal uptake of radioiodine, and a decrease in thyroid weight. The acute injection of a NaCl solution had no apparent effect on iodide excretion in the chick but it was concluded that this might possibly be due to experimental procedure.

It was concluded that NaCl altered the iodide excretion by the chicken kidney in an opposite manner from that reported for mammalian species. It was also concluded that the decrease in the 24-hour uptake of radioiodine by the thyroid was due to a change in the specific activity of the injected radioiodide rather than a change in thyroid function.

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A P P E N D I X

BODY AND THYROID WEIGHTS: 24-HOUR UPTAKE; T/S; AND THE RENAL CLEARANCES OF IODIDE AND CHLORIDE IN EXPERIMENT I

Bird No.	Thyroid Wt. mg	Thyroid Wt. mg%	24-Hour ^{131}I Uptake	Renal clear. I^- ml/min	T/S	Renal clear. Cl^- ml/min	Body Wt. gm
			Low-chloride, low-iodide diet				
1	27.6	4.76	9.54	.029	515	.00275	580
5	37.6	5.77	12.29	.250	251	.00292	652
9	22.4	4.30	10.38	.422	364	.00484	520
13	29.1	4.73	10.92	.742	362	.00791	616
17	16.7	4.26	11.48	.108	252	.01072	392
21	31.3	6.14	11.39	.322	283	.02727	510
25	30.6	4.55	11.14	.517	365		672
29	30.0	6.52	12.77	.430	300		460
33	31.1	4.71	10.01	.976	443		660
37	17.9	4.08	11.45	.234	380		440
MEAN	27.4	4.98	11.14	.403	351	.0094	550
			Low-chloride, high-iodide diet				
2	28.7	5.52	.54	.159	12.8	.00792	520
6	29.5	5.22	.43	.632	11.6	.01027	564
10	25.7	5.14	.70	.185	31.6	.06888	500
14	16.7	4.26	.96	1.037	6.5	.02473	392
18	20.4	3.70	.56	.591	12.5	.03991	552
22	51.7	9.03	.48	.492	7.0	.00772	572
26	38.2	6.11	.43	.243	15.9	.00158	624
30	34.3	5.24	.03	.629	11.7		654
34	22.8	4.14	.44	.928	12.0		552
38	32.8	7.08	.66	.081	14.6		464
MEAN	30.1	5.54	.52	.498	13.6	.0230	539
			High-chloride, low-iodide diet				
3	28.1	4.92	8.74	1.28	341	.06890	570
7	32.2	5.44	10.80	.76	394	.04016	592
11	41.9	7.18	10.83	.64	172	.04950	584
15	30.8	6.16	9.01	.22	325	.02142	500
19	23.4	5.17	9.62	.83	323	.04166	452
23	36.4	6.59	8.38	.23	378	.00654	552
27	31.4	4.24	9.74	.35	203	.04008	740
31	31.1	4.78	9.97	1.27	237	.04840	650
35	33.1	5.42	10.52	.57	482	.02589	612
39	21.6	4.58	9.36	.53	598		472
MEAN	31.0	5.45	9.70	.666	345	.0381	572
			High-chloride, high-iodide diet				
4	30.4	5.62	.71	.191	9.6	.02996	540
8	34.6	6.26	.64	.221	9.7	.00928	552
12	39.9	6.92	.02	.628	13.4	.03729	576
16	67.4	9.63	.63	1.865	4.8	.10018	700
20	20.6	4.11	.76	.942	14.5	.04805	500
24	51.7	7.13	.03	1.351	8.7	.02147	724
28	32.7	4.19	.03	1.145	10.5	.06958	780
32	41.0	6.41	.10	.173	16.8		640
36	40.6	7.66	.39	.736	24.2		530
40	37.8	6.61	.43	.224	12.0		572
MEAN	39.7	6.45	.37	.747	12.4	.0451	611

BODY AND THYROID WEIGHTS; 24-HOUR UPTAKE
IN EXPERIMENT II

Bird No.	Body Wt. gm	Thyroid Wt. mg	Thyroid Wt. mg%	24-Hour ^{131}I uptake %
		Control diet plus 5.0% NaCl		
1	348	15.54	4.47	5.185
2	260	11.46	4.41	3.128
3	344	13.23	3.85	3.364
4	280	9.39	3.35	3.183
5	252	12.31	4.88	4.055
6	308	14.49	4.71	5.071
7	296	10.79	3.65	2.434
8	354	13.24	3.74	3.561
9	332	12.31	3.71	3.809
10	240	9.00	3.75	5.467
MEAN	301	12.18	4.05	3.926
		Control diet		
11	444	21.60	4.87	3.986
12	364	25.00	6.87	8.592
13	432	24.23	5.61	8.063
14	436	26.50	6.08	9.800
15	384	25.67	6.69	10.864
16	312	15.09	4.84	7.029
17	352	17.00	4.83	8.233
18	388	18.52	4.77	9.944
19	384	20.60	5.37	11.630
20	352	24.20	6.88	10.664
MEAN	385	21.73	5.68	8.881

RENAL CLEARANCE OF IODIDE AND CHLORIDE IN THE CHRONIC SALT-TREATED BIRDS OF EXPERIMENT II

Bird No.	Renal Clear. of I^- ml/min.	Renal Clear. of Cl^- ml/min.
	Control diet plus 5.0% NaCl	
1	.3458	.0227
2	.4836	.0313
3	.3748	.0335
4	.3550	.0228
5	.8444	.1188
6	.6263	.0133
7	.3966	.2123
8	.6053	.0540
9	.5438	.0795
10	.4976	.0159
MEAN	.5073	.0604
	Control diet	
11	1.0835	.0588
12	.9380	.0371
13	.5458	.0514
14	.6286	.0211
15	.6316	.0670
16	.2675	.0974
17	.5970	.1103
18	.9327	.0556
19	.9314	.0646
20	.4117	.0158
MEAN	.6968	.0579

RENAL CLEARANCE OF IODIDE AND CHLORIDE IN THE ACUTE SALT-TREATED BIRDS OF EXPERIMENT II

Bird No.	Renal Clear. of I⁻ ml/min		Renal Clear. of Cl⁻ ml/min	
	30 min	60 min	30 min	60 min
	NaCl-injected birds			
1	1.432	1.322	.16598	.09371
2	1.475	1.826	.19713	.30404
3	1.117	1.379	.08823	.02535
4	1.476	2.974	.20192	.43035
5	1.425	1.402	.04415	.03421
6	.817	1.168	.12516	.09628
7	.660	.423	.20104	.01095
8	.269	.148	.02628	.05667
9	.741	.734	.16318	.02717
10	.996	2.878	.02226	.06251
MEAN	1.041	1.425	.1235	.1141
	Dist. H_2O-injected birds			
11	1.165	1.376	.02606	.00534
12	2.999	2.677	.18071	.01390
13	1.563	1.300	.05704	.05286
14	.576	1.428	.12784	.01847
15	.726	.849	.06365	.00525
16	.736	1.059	.04939	.03467
17	.608	.546	.06021	.00471
18	.500	.541	.01955	.00107
19	.535	.295	.01884	.00642
MEAN	1.045	1.117	.0670	.0173

BODY AND THYROID WEIGHTS; 24-HOUR; AND THE RENAL CLEARANCE OF IODIDE AND CHLORIDE IN THE CHRONIC SALT-TREATED BIRDS OF EXPERIMENT III

Bird No.	Thyroid Wt. mg	Thyroid Wt. mg%	24-Hour ^{131}I uptake % of dose	Renal Clear. I^- ml/min	Renal Clear. Cl^- ml/min	Body Wt. gm
			Control diet plus 5.0% NaCl			
1	41.20	4.90	4.11	.8557	.09929	840
2	37.95	6.08	4.46	.3737	.00076	624
3	41.46	6.28	5.34	.3352	.09363	660
4	46.37	6.07	4.94	.4597	.00087	764
5	40.41	5.71	6.07	.6429	.06249	708
6	36.45	5.09	5.55	.1463	.00875	716
7	24.80	3.63	5.21	.8159	.09982	684
8	44.60	8.08	10.35	.9983	.07551	552
9	41.96	7.04	8.13	.7001	.00120	596
10	30.80	5.92	8.99	.4007	.05650	520
MEAN	38.62	5.88	6.32	.5729	.0499	666
			Control diet			
11	60.77	7.48	8.27	.9174	.01410	812
12	62.45	7.15	9.20	.9937	.03163	816
13	44.97	6.21	8.80	.9193	.04110	724
14	41.86	5.66	8.80	1.0643	.00428	740
15	54.47	7.52	8.38	.6497	.01036	724
16	39.47	5.25	7.43	.7904	.03962	752
17	65.47	7.98	9.56	.7044	.00894	820
18	40.24	5.82	8.78	.8426	.03193	692
19	54.81	7.21	7.89	.6124	.02376	760
20	43.90	6.38	9.37	.7679	0.4738	688
MEAN	50.83	6.72	8.65	.8262	.0253	754

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