THE UNIVERSITY OF MANITOBA

ELECTROLYTE AND WATER METABOLISM IN YOUNG TURKEY POULTS

by

CHO YAT PANG

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ABSTRACT

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The general consensus in published reports of the occurrence of oedema and ascites (waterbelly) in turkey poults was that high salt intake from feed and/or drinking water was probably responsible. Consequently, 4 experiments were conducted to study electrolyte and water metabolism in turkey poults.

Turkey poults, offered *ad lib.* tap water (125 ppm solids), could tolerate 1% salt diet with no harmful effects. Death with oedema and ascites occurred mainly between days 5 and 8 in poults offered *ad lib.* 3% or 6% salt diet and allowed free access to tap water. A similar mortality pattern was observed among poults offered *ad lib.* a 0.25% salt diet and tap water containing 90 mM or 105 mM added NaCl per litre. Mortality rate seemed to be related to levels of salt in the diet or drinking water. Positive salt and water imbalance was more severe in poults compared with that in similarly treated poults which survived to slaughter.

Data on mortality rate and plasma and body composition of poults offered *ad lib.* tap water containing 105 mM added NaCl per litre starting at different ages (4, 8 and 12 days) suggested that poults older than 8 days of age could tolerate high salt intake much better than could younger poults.

Renal clearance studies were conducted on control (offered *ad lib.*

a 0.25% salt diet and tap water) and treatment (offered *ad lib.* same diet and tap water containing 90 mM added NaCl per litre) poults. An increase in glomerular filtration rate (ml/min/kg B.W.) was observed in control poults between 4 and 12 days of age, but a marked increase in ability of tubular water reabsorption was not apparent until the poults were 12 days of age.

Compared to the control poults, increase in saline intake because of markedly higher urinary water loss due to notably lower renal water reabsorption ability during natriuresis was observed for treatment poults. Increased salt and water contents of extracellular fluid spaces without simultaneous compensation at glomerular level, was believed to have contributed to positive salt and water retention.

The mean values for GFR and tubular water reabsorption were markedly lower for oedematous than the rest of treatment poults of the same age. These findings lend support to the hypothesis that oedema and ascites formation in turkey poults on high salt intake is caused by renal insufficiency.

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INTRODUCTION

Sturkie (1953) remarked that, "knowledge in certain areas of avian physiology is limited, fragmentary, and often confused, and little or no new research is being conducted." Water and electrolyte metabolism in general and specially the renal aspects thereof is an area which has been much neglected as it has long been taken for granted that the avian kidney probably functions in principle like that of the mammals (Sykes 1971).

The present experiments were initiated as a result of consideration of an economically important pathological condition of young turkey poults manifested by oedema and ascites. It has been suggested that such "water-belly" is a result of excessive salt intake. Detailed investigation of water and electrolyte metabolism of young turkey poults has not been carried out heretofore.

The first experiment was conducted to study the plasma and body compositions of young turkey poults on normal and high salt intakes. Based on the information obtained in experiment 1, two subsequent experiments were carried out to further study the tolerance of young poults to salt intake with respect to age and dietary sources of salt. Finally, renal studies were performed to investigate salt and water excretion in young poults offered varying levels of salt intake. Emphases were made on the glomerular filtration rate and capability of tubular water reabsorption in the kidneys of young poults during chronic oral saline loading.

REVIEW OF LITERATURE

I. Historical review on the occurrence of oedema and ascites in baby chicks and turkey poults

Incidence of oedema and ascites in baby chicks and young turkey poults has been reported and opinions regarding the factors responsible for the occurrence of such disease have been expressed in the literature by many researchers. In general, one group of investigators believes that the occurrence of these conditions is the result of excessive dietary salt intake while the other group of investigators has the opinion that a toxic contaminant ("oedema factor") is directly responsible for the occurrence of the disease.

A. Salt toxicity hypothesis

The subject of salt toxicity in chickens appeared in the literature about half a century ago. Most of the earlier investigations concerning the salt tolerance of domestic birds were conducted with mature chickens (Sufferan 1908; Mitchell *et al.* 1929). Mitchell *et al.* (1929) reported that chickens, 9 to 21 weeks of age, could tolerate a diet containing as high as 8% salt.

One of the earlier reports indicating that older chickens are less susceptible to the toxic effects of salt was made by Smith (1929) who observed 20% mortality in baby chicks placed on diets containing 3.5 and 4.5% of salt. Further experimentation on baby chicks offered diets varying from 0 to 10% salt was reported by Barlow *et al.* (1948) who observed that 1% salt in the chick diet produced optimum growth. Water

consumption increased linearly with the salt content in the diet. Mortality occurred in chicks fed diets containing more than 3% salt and mortality rate increased with increase in salt intake. The deaths, which were attributed to oedema, occurred mainly during the first four weeks of life. However, in a separate experiment, it was observed that a high salt diet (5% NcCl) did not result in oedema formation or death when offered to chicks starting at 3 and 6 weeks of age.

Studies on salt tolerance were extended to turkey poults by James (1946) who found that young turkey poults could tolerate a starter diet as high as 1% of salt but feeding a starter diet containing 2% of salt resulted in a mortality rate of 38.4% and the incidence of mortality occurred between 5 and 15 days of age. Postmortem findings on the deceased poults indicated the presence of generalized oedema and pulmonary congestion.

Field cases of oedema and ascites in baby chicks and turkey poults on high salt intakes were also reported by various investigators (Bullis and Van Roekel 1946; Doll *et al.* 1946; Scrivner 1948; Bigland 1950). In the poultry industry, this disease is commonly known as waterbelly. The patterns of mortality and the descriptions of pathological changes in the affected field birds, reported by these investigators, are in general similar. There was a high mortality rate of turkey poults about a week old but no mortality was observed after twenty days of age. The mortality pattern in baby chicks was less defined and death occurred in chicks up to the age of five weeks. The pathological changes in affected birds were characterized by severe oedema, ascites and subcutaneous oedema and in some cases hydropericardium, dilated right ventricle and oedematous kidneys were also observed. Except in a few affected birds in which *E.*

coli or *Salmonella* organisms were isolated, bacteriological examination on the oedematous birds yielded negative results. Infectious agents were considered unlikely to be the cause of oedema or death by all the above investigators. Bigland (1950) expressed the opinion that high salt intake as a result of "build-up" of sodium chloride in the feed troughs by gravitation could be responsible for the occurrence of oedema in young domestic birds.

The problem of salt toxicity in young domestic birds may be further compounded by the occurrence of high salinity in well water of some poultry farms. Water samples submitted to the [1]Alberta Department of Agriculture in 1966-1971 for analysis of salt content revealed that a majority of the well water contained 500-2000 ppm solids, while some contained as high as 6000-7000 ppm solids. The solids are mainly in the forms of sodium salts (Na_2SO_4, $NaHCO_3$ and NaCl). The natural occurrence of high salt content in well water was considered to be one of the main factors contributing to salt toxicity in baby chicks and young turkey poults.

In a review of literature, Robblee and Clandinin (1961) quoted experimental evidence to indicate that there is a lesser tolerance for salt in drinking water than in the diet for young chicks, and therefore experiments were conducted by these investigators to further study this problem in turkey poults. Field conditions were simulated by adding to a basal diet (0.4% NaCl) or drinking water (tap water containing 100 ppm

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[1] Report of the committee on water quality. The Alberta Dept. of Agriculture, Jan. 20, 1972.

solids), various levels of [2]mixed salts, the composition of which was similar to that found in the well water. These investigators found that the mortality rate and incidence of oedema and ascites were influenced by the amount of salt added to the feed as well as by the concentrations of salt in the drinking water. With high levels of salt in the feed, lower levels of salts in the water were toxic, while with lower levels of salts in the feed poults were able to tolerate higher levels of salt in the drinking water. Again, the mortality and incidence of oedema and ascites occurred starting at 3 days of age, reached a peak at 6 days of age and stopped after 12 days on treatment.

In the opinion of the present reviewer, addition of sodium salts of sulphate and bicarbonate in experiments of this nature did not contribute more clarification in the experimental results than simply using sodium chloride as the source of salt intake. Anions like sulphate and bicarbonate are very unlikely to be the cause of oedema. Bicarbonate ions, when absorbed, are either buffered in the body fluids or excreted and the renal reabsorption of which depends on the pH of the body fluids. Normally, the concentration of sulphate ions in the extracellular fluid is about 1 mEq/L and the renal reabsorption of this anion is limited by the very low transport maximum as in the case of mammals (Sykes 1971).

Sibbald *et al.* (1961) conducted three separate experiments to obtain further information on the toxic effects of sodium chloride intake either solely from drinking water or diet upon the weight gain, feed efficiency and survival of chicks. When the drinking water contained no sodium

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[2] Mixed salt consisted of Na_2SO_4, NaCl, $NaHCO_3$ in the proportion of 3:2:1 by weight, respectively.

chloride, salt levels of 0.25 to 2.0% in the feed allowed satisfactory production. Mortality and oedema together with depressed weight gain occurred with a diet containing 4% salt. When salt intake was from drinking water only, chicks aged 0 to 4 weeks required 0.25 to 0.5% saline to perform satisfactorily and 0.2% of salt in the drinking water was sufficient for birds aged 5 to 7 weeks. Chicks from 0 to 4 weeks old could tolerate water containing as much as 0.5% of salt even though the diet also contained 0.5% salt.

The literature reviewed thus far does lend support to the hypothesis that the occurrence of oedema and ascites in baby chicks and young turkey poults in laboratory and field conditions is related to excessive salt intake. However, for all the aforementioned experiments, the investigators did not report feed or water consumptions when various levels of salt in the diet or drinking water were offered to young domestic birds and this makes it impossible for the readers to compare the relative quantitative toxic effects of salt in feed and drinking water.

B. Toxic contaminant hypothesis

During the autumn of 1957, a heavy incidence of waterbelly in broiler chicks appeared in several areas of central and eastern parts of the United States. One common factor was that the affected flocks consumed feed supplied by two particular feed mills and these companies were suspected to have furnished feed containing toxic substances which might be responsible for the outbreak of this disease. Laboratory tests of these feeds showed that oedema and ascites were also produced in chicks fed the same commercial feeds which had either been autoclaved or extracted with water (salt was added to the feeds after water extract

but the level was not reported). These experimental results were interpreted to suggest the presence of a heat-stable toxic substance in the fat of the feeds (Schmittle 1958). The [3]report issued by the Nutrition Council of the American Feed Manufacturers Association supported this view.

Since the release of the above publications, the speculation on the cause of waterbelly in young chicks has focused on a toxic contaminant in the fat of commercial feeds or possible interrelation with vitamin E or antioxidants. Friedman *et al.* (1959) produced waterbelly in chicks by adding to a basal diet, 6% of "toxic fat" obtained from a feed manufacturing plant but observed that addition of large doses of vitamin E in the feed diet not prevent the occurrence of waterbelly.

Brew *et al.* (1959), Friedman *et al.* (1959) and Wootton *et al.* (1959) believed that a toxic contaminant responsible for waterbelly formation was present in the unsaponifiable fraction of the "toxic fat". Chemical procedures for obtaining concentrated toxic materials from the "toxic fat" were reported and chemical structures of the toxic materials were suggested by these investigators. The addition of these toxic materials to the feed was successful in producing waterbelly in baby chicks (Brew *et al.* 1959; Friedman *et al.* 1959), the latter investigators also observed the occurrence of such disease in the control group. The diet used by Brew *et al.* (1959) contained 6% commercial mineral mix (composition of which was not mentioned) while that used by Friedman *et al.* (1959) contained 2% iodized salt.

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[3] "Feedstuff", Vol. 30(24), p. 1 and 99, 1958.

Douglass and Flick (1961) presented the results of a collaborative experiment on chick oedema. All seven collaborators reported death and presence of waterbelly in baby chicks fed different levels of "toxic fat". However, three of the seven collaborators also reported deaths of birds on the control diet and two of the control birds that died during the experiments displayed oedema and ascites.

Further collaborative investigations on oedema in chicks fed different levels of "toxic fat" were reported by Flick *et al.* (1961). The observations made by ten different laboratories were variable. Three out of ten laboratories reported occurrence of waterbelly and hydropericardium in the control group although the incidence was not significant compared to that found in the treatment group receiving "toxic fat" in the diet. Mortality due to intake of different levels of "toxic fat" was significant during the third week on the diet containing the highest level of "toxic fat". Even so, mortality due to oedema reported by the collaborators varied from none to 10 out of 12 in chicks fed the highest "toxic fat" in the diet. On the basis of the observation that the incidence of waterbelly was markedly higher in the treatment group than the control, these researchers supported the view that a toxic contaminant is the main factor in the causation of waterbelly in young chicks. The diet used in all these experiments contained 6% mineral mixture (composition of which was not specified) and 1.2% iodized salt.

Flick and O'Dell (1968) studied the effects of an oral diuretic in alleviation of chick oedema. Hydrochlorothiazide was fed at dietary levels of 0, 25, 50, 100, 500 and 1000 mg/kg feed along with a toxic fatty material known to have produced chick oedema. After a three week feeding regimen, it was observed that the higher dietary levels of the

diuretic were more effective in preventing the formation of oedema.

As in the case of the salt toxicity hypothesis, the toxic contaminant hypothesis failed to explain the observation that waterbelly only occurred in some but not all birds on the same treatment. The salt toxicity hypothesis attempts to establish the toxic effects of high salt intake with respect to dietary sources and age of bird but toxic contaminant hypothesis completely disregards the dietary salt and water intake although the retention of which, in the extracellular fluid compartments, is the pathological condition of oedema. In fact, the salt contents in the diets of the experiments reviewed for toxic contaminant hypothesis are at or well above the tolerance level for young domestic birds as mentioned before. Besides, the toxic contaminant hypothesis neither explains the reason why the toxic materials affect only young chicks nor does it offer any explanation as to how toxic materials can damage the body tissues to result in oedema.

Furthermore, the observation that an oral diuretic was effective in preventing oedema formation in chicks fed toxic materials may as well be interpreted as the therapeutic effects of the diuretic in decreasing salt and water reabsorption resulting in alleviation of oedema.

II. Present impact of waterbelly in poultry industry

In 1970, the Manitoba Department of Agriculture [4] reported that "A condition found in turkey poults, known as waterbelly, has caused losses in Manitoba turkeys . . . the losses usually begin after the first of

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[4] News Bulletin #4, August, 1970. M.D.A.

March each year and ranged from two to ten percent . . . It is obvious that a great deal of research is required in order to find the cause of this costly condition causing high mortality in young turkey poults."

Due to the occurrence of waterbelly in commercial turkey poults, the salt content of most turkey starter diets has been reduced to 0.25% in the last few years. Even with this low salt diet, the [1]Alberta Department of Agriculture suggested that turkey poults under the age of three weeks should receive drinking water containing less than 500 mg/L of salt. This recommendation does not solve the problem for existing poultry farms with well water containing salt content well above the recommended level. Indeed, several large local poultry producers are still confronted with sizeable economic losses due to the unsolved problem of waterbelly in young turkey poults (recent communication between L. D. Campbell of Department of Animal Science, the University of Manitoba and local turkey producers).

III. Disease in domestic fowls with pathological conditions similar to waterbelly

Round heart disease

The term "round heart disease" of turkey poults was coined by Luke in Ireland and this disease is also known as "Eierherzen" (egg heart) in Germany (Peckham 1972). Round heart disease of turkeys was first described in Canada by Magwood and Bray (1962) and at present the etiology of this disease is unknown.

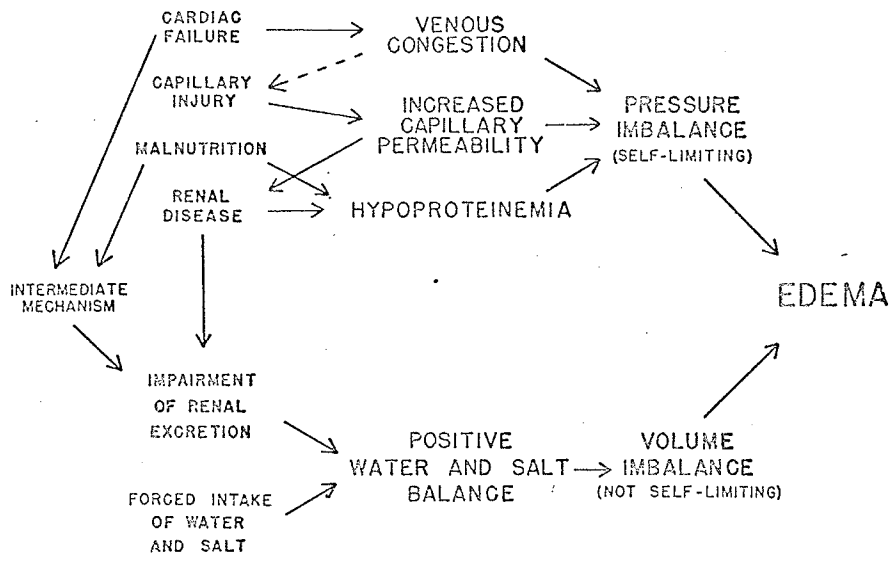
Peckham (1972) has given a detailed description of this disease. Briefly summarized, the majority of the adult birds that die with round heart disease do not manifest any premonitary signs. Death occurs suddenly. This disease has been observed in adult turkeys. The pathological conditions are; distortion of the right ventricle, the wall of which is flaccid; the lungs are frequently oedematous and the kidneys enlarged. Excess pericardial and peritoneal fluids are observed. This disease affects primarily male birds but occasionally such conditions are found in females. The young poults usually have ruffled feathers, drooping wings and an unthrifty appearance. This disease is also found in chickens of any age. All attempts to isolate or demonstrate an infectious agent and to transmit this disease with tissue from affected birds have been unsuccessful.

Hunsaker (1969) reported that the heart rate, blood pressure and cardiac output of round heart poults are respectively 7, 4 and 50% lower than those of the normal poults. The growth rate of the round heart poults was depressed but some of them could grow to maturity and were able to reproduce. Mating affected males with affected females increased the incidence from less than 5% to 40%. It was stated that this increased incidence suggested some degree of heritability of round heart disease in turkeys.

Hypoproteinemia and alpha globulin deficiency have recently been observed in round heart turkeys. The implication of these findings with regard to the etiology of round heart disease awaits further investigation (Meirom *et al.* 1974).

IV. General etiology of oedema formation

The following scheme relating to factors leading to oedema formation was suggested by Wolf (1960).



Pressure imbalance and volume imbalance are believed to be the two main factors responsible for the formation of oedema although the intermediate mechanisms are still conjectural. Pressure imbalance involves changes in effective osmotic pressure, hydrostatic pressure, tissue pressure, capillary permeability or lymphatic drainage. Oedema caused by pressure imbalance is self-limiting as it is unlikely to produce a volume of oedema fluid exceeding that of plasma.

However, volume imbalance brought about by positive salt and water balance, through either impairment of renal excretion and/or through excessive intake of salt and water, can cause a relatively unlimited type of oedema. In this process, the pressure change may be so small as to defy measurement, yet unlimited oedema has been observed. In most

cases, pressure and volume imbalance do co-exist (Wolf 1960).

V. Excretion of solutes and water in domestic birds in relation to regulation of body fluid volume

A. Excretory systems of salt, water and nitrogenous waste

In most birds, the urine at most is twice as concentrated as the plasma. Extrarenal secretory organs are found in aquatic birds (e.g. sea gulls and Peking ducks) and desert birds (e.g. ostrich and partridge). These extrarenal secretory organs (generally known as salt glands) can actively secrete electrolytes. By virtue of these organs, the marine and desert birds can tolerate either high salt intake or high environmental temperature and water deprivation in spite of the inability of the kidneys to produce highly concentrated urine (Holmes *et al.* 1968; Schmidt-Nielsen 1973).

However, in the domestic hen, the nasal glands (which are the homologues of the salt glands of aquatic birds) are small and non-functional (McLelland *et al.* 1968) and consequently the kidneys of the domestic hen are the main guardians of the milieu intérieur.

It is generally held that excretion of nitrogenous end products mainly as uric acid (uricotelism) enables birds to conserve water. Uric acid has a very low solubility in water and exerts a low common ion effect and hence low osmotic pressure when excreted in the semi-solid form (Nason 1965; Schmidt-Nielsen 1973). In domestic birds, about 84% of the urinary nitrogen is in the form of uric acid (Sykes 1971).