Effect of sodium chloride deficiency on basal metabolism in broiler chickens

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1. Male broiler chickens were given a sodium chloride-deficient or NaCl-adequate diet from 7 to 21 d of age in Expt 1 and 28 to 56 d of age in Expt 2.

2. NaCl-deficient chickens had a markedly poorer growth and food conversion efficiency than those given the NaCl-adequate diet.

3. NaCl deficiency was associated with an increase in basal metabolic rate and increases in oxygen consumption, heat production and respiratory quotients were also noted. The glycogen content of chicken livers was also higher.

4. Measurements of acid–base balance were found to be changed in NaCl-deficient chickens. Values for pH and bicarbonate content in blood plasma were lowered.

5. NaCl deficiency increased the packed cell volume and thyroxine level in blood plasma. Sodium and chloride contents in blood were lowered.

6. Results are discussed in relation to the decreased food conversion in NaCl-deficient chickens.

A deficiency of dietary sodium chloride causes growth retardation accompanied by a reduction in food conversion in chickens (Burns *et al.* 1953; Summers *et al.* 1967; Nott & Combs, 1969; Dewar & Whitehead, 1973; Ryś *et al.* 1975; Ross, 1977). NaCl-deficient chickens had a lower plasma sodium content and hypertrophy of adrenal gland (Lumijarvi *et al.* 1966) and histological changes in kidneys (Siegel, 1961), while turkeys had kidney hypertrophy (Leeson *et al.* 1976*a*). A raised packed-cell volume in deficient chickens could suggest dehydration of the body (Lumijarvi *et al.* 1966). Considering the chicken's requirement for dietary chloride is nearly 50% lower than that for sodium, it could be suggested that the reduction in performance in chickens is attributable to Na rather than Cl^- deficiency (Burns *et al.* 1953; Summers *et al.* 1967). Hurwitz *et al.* (1973) found decrease in blood pH in Na-deficient chickens. In a previous study we showed that the modification of carbohydrate digestion produced by NaCl deficiency did not appear to be the main factor responsible for the decreased performance of NaCl-deficient chicks (Koreleski *et al.* 1976). The mechanism of the increased food expenditure for weight gain in Na-deficient chickens was not elucidated.

In the present study the effect of NaCl deficiency on the basal metabolic rate, plasma thyroxine level and measurements of acid–base balance in chickens were investigated.

EXPERIMENTAL

Two experiments, each using forty male broiler chickens (Cornish × White Rock) were carried out at from 7 to 21 d of age (Expt 1) and 28 to 56 d of age (Expt 2). Chickens were given standard starter mixture before the trial. During the experimental periods they were given a basal soya-bean diet (Table 1) either supplemented with 3 g NaCl/kg (control group) or unsupplemented (deficient group) when the diet contained only 0·18 g NaCl/kg.

In both experiments control or deficient diets were offered to groups of twenty individually fed chickens. Broilers were housed in separate cages with screen floors. Food and distilled

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Table 1. *Composition (g/kg) of the basal low-sodium chloride diet*

Component		
Soya-bean oil meal	36·0	
Maize meal	58·4	
Beef tallow	3·0	
Standard vitamin–mineral–antibiotic premix (free of NaCl)	1·0	
Dicalcium phosphate	1·0	
Limestone	0·5	
DL-methionine	0·1	
Analysis		
Crude protein (nitrogen × 6·25)	212·3	
Diethyl ether extract	64·6	
Crude fibre	40·0	
Metabolizable energy MJ (kcal)	12·8	(3060)

water were given *ad lib.* The weight gains and food conversion efficiency (g weight gain/g food intake) were recorded weekly.

Expt 1. For the determination of acid-base measurements on the 14th day of the experiment (21 d of age) blood samples were collected from eight randomly chosen cockerels per treatment. Blood was taken anaerobically from the wing vein into a heparinized glass capillary and maintained at 4–8°. Estimations of pH and partial pressure of carbon dioxide (pCO_2) were made using a Micro-Astrup AME-1 Radiometer. The bicarbonate content was calculated using the Hasselbach-Henderson equation, assuming a pK value of 6·09 and the solubility of carbon dioxide in water at 41·5° to be 0·0278 (Helbacka *et al.* 1964). A blood sample was also taken into microcapillaries and centrifuged for 10 min at 3000 *g* for determination of packed cell volume. At the same time basal metabolic rate was estimated in eight chickens from each group, which had live weights nearest to the average weight of the group. Chickens were fasted for 12–18 h with free access to distilled water, weighed and subjected to a period of adaptation, when they were maintained for 15 min in the dark at a neutral temperature (29°). The volume of oxygen consumed and CO_2 expired in a 30 min period were measured using a Kipp-Zonen MG-4 diaferometer. Respiratory quotient RQ and heat production were estimated. Heat production (not corrected for nitrogen catabolism) calculated for a 24 h period was expressed in J/kg body-weight or metabolic body-weight (body-weight $^{0.75}$).

On the day after basal metabolism estimation the chickens were killed and blood collected in heparinized test-tubes and centrifuged. Samples of plasma were stored in the cold (−20°) and then analysed for Na and Cl^- content.

Expt 2. At 56 d of age (after the 28 d of the experimental period) eight chickens were chosen from each group for basal metabolism estimations having live weights near to the average weight of the group as described for Expt 1. After estimations of basal metabolism chickens were killed and blood collected. Plasma was stored at −20° and then analysed for thyroxine content. Immediately after killing, the livers were removed, frozen in liquid N_2 and stored at −20° until analysed for glycogen content.

Analytical methods. Basal nutrient content of the diet was estimated as described by Skulmowski (1964). The levels of Na in the food and in blood plasma were analysed using a flame photometric method and Cl^- using the titration method with silver nitrate (Ostrowski, 1974). Thyroxine content in plasma was analysed using a competitive-protein-binding technique (Murphy, 1965). Glycogen in liver was estimated using a colorimetric method (Stefaniak, 1964).

Statistical analysis. The significance of differences between the two treatments was examined by Student's *t* test.

Table 2. *Expt* 1. *Effect of dietary sodium chloride on performance and basal metabolic rate in cockerels at* 3 *weeks of age*

Diet	Final body-wt at 28 d of age (g)	Wt gain from 7 to 28 d of age (g)	Food intake (g/chick)	Food conversion efficiency	Oxygen consumption (ml O_2/g per h)	Heat production (MJ/kg body-wt per 24 h)	Heat production (MJ/kg body-wt$^{0.75}$ per 24 h)	Respiratory quotient
Control	458	359	565	0·64	1·0203	0·475	0·364	0·6661
Low NaCl	316	215	493	0·43	1·4385	0·681	0·501	0·7295
SE of mean	36·0	30·0	40·0	0·05	0·0873	0·033	0·025	0·0558
df	18	18	18	18	6	6	6	6
Statistical significance of difference between diets	**	**	NS	**	**	**	**	NS

NS, not significant. ** $P = 0·01$.

Table 3. *Expt* 2. *Effect of dietary sodium chloride on performance and basal metabolic rate in cockerels at* 8 *weeks of age*

Diet	Final body-wt at 56 d of age (g)	Wt gain from 28 to 56 d of age (g)	Food intake (g/chick)	Food conversion efficiency	Oxygen consumption (ml O_2/g per h)	Heat production (MJ/kg body-wt per 24 h)	Heat production (MJ/kg body-wt$^{0.75}$ per 24 h)	Respiratory quotient
Control	2068	1320	3518	0·38	0·7332	0·240	0·409	0·6507
Low NaCl	1484	814	3179	0·25	0·9386	0·443	0·499	0·6999
SE of mean	62·3	64·3	448	0·005	0·0243	0·011	0·021	0·0367
df	18	181	18	18	6	6	6	6
Statistical significance of difference between diets	**	**	**	**	**	**	*	NS

NS, not significant. * $P = 0·05$, ** $P = 0·01$.

Table 4. *Expt* 1. *Effect of dietary sodium chloride on the blood acid–base measurements and packed cell volume and the level of sodium and chloride in the plasma of chickens*

Diet	pH	Partial pressure of carbon dioxide (mmHg)	Bicarbonate (mol/l)	Sodium (mol/l)	Chloride (mol/l)	Packed cell volume
Control	7·263	56·08	0·023	0·186	0·152	0·29
Low NaCl	7·193	54·60	0·019	0·149	0·138	0·40
SE of mean	0·024	3·06	0·001	0·013	0·002	0·01
df	14	14	14	14	14	14
Statistical significance of difference between diets	*	NS	**	*	**	**

NS, not significant.
* $P = 0{\cdot}05$, ** $P = 0{\cdot}01$.

Table 5. *Expt* 2. *Effect of sodium chloride deficiency on the thyroxine level in blood plasma and glycogen content in fresh liver of chickens*

Diet	Thyroxine (μg/100 ml plasma)	Glycogen (mg/100 g liver)
Control	1·70	7·27
Low NaCl	2·43	14·47
SE of mean	0·1157	1·57
df	18	18
Statistical significance of difference between diets	**	**

** $P = 0{\cdot}01$.

RESULTS

Deficiency of NaCl caused significant growth retardation and decreased food intake and food conversion in chickens of both ages (Tables 2 and 3).

Basal metabolic rate was increased in NaCl-deficient chickens and this effect was not dependent on age. There was a statistically significant increase ($P = 0{\cdot}01$) in O_2 consumption and heat production. Differences in RQ were not statistically significant.

NaCl deficiency in chickens significantly changed acid–base measurements (Table 4). Bicarbonate content and pH values in the blood were lowered ($P = 0{\cdot}05$), as were Na and Cl^- levels in blood plasma ($P = 0{\cdot}05$ and $P = 0{\cdot}01$ respectively). Packed cell volume in NaCl-deficient chickens was significantly increased ($P = 0{\cdot}01$). Deficiency caused a 100% increase in the glycogen content of the liver (Table 5). The level of thyroxine in blood plasma was also increased ($P = 0{\cdot}01$).

DISCUSSION

Results of both experiments were similar to published results and showed depressed performance in chickens given the NaCl-deficient diet. It seems that some of the effects recorded for NaCl-deficient birds resulted from reduced food intake, which was of the order of 12%. Lower food intake, however, could not be the main reason for the marked food conversion and growth depression (by about 33·5 and 39%, respectively) as it was found in the experiments. Apart from the latter finding, dietary NaCl deficiency affected the body water metabolism. As the result of an imbalance in osmotically active Na and Cl^- ions it

was suggested that there was movement of water from extra- to intracellular fluids. The translocation of water could have caused body dehydration in chickens, as suggested by Tosteson & Hoffman (1960). These changes resulted in increased packed cell volume and an imbalance in the acid–base equilibrium.

A similar result was reported by Lumijarvi *et al.* (1966), who found increased values for packed cell volume and a decreased plasma Na content in NaCl-deficient chickens. Dehydration was noted in spite of increased water intake in NaCl-deficient chickens. These findings suggest that the level of body water may have been one of the reasons for reduced growth in NaCl-deficient chickens. Under conditions of dehydration there is stimulation of some processes for endogenous water synthesis in the body (Leeson *et al.* 1976*b*), e.g. lipid metabolism with coupled oxidative phosphorylation in the respiratory chain. Increased lipid catabolism is accompanied by enhanced heat production and O_2 consumption, and symptoms of increased heat production were noted in our experiment in the instance of NaCl-deficient chickens. Values for RQ and increased glycogen accumulation in liver also support this suggestion.

It is known that there is functional interaction between thyroid gland activity and Na circulation in the body of rats (Fregly & Taylor, 1964; Taylor & Fregly, 1964). The Na absorption process in renal tubules requires energy and is related to energy-releasing metabolic reaction; Matty & Green (1962) observed that in the absence of Na and presence of thyroxine there is an increased transport of Na through the cell membrane, accompanied by increased O_2 and water consumption. It may be suggested that energy requirements are increased in Na-deficiency states and these are accompanied by an increased metabolic rate and a stimulation of thyroid activity. The latter could be the reason for the increased levels of thyroxine observed in the second experiment. It is also possible that higher levels of thyroxine could decouple to some extent oxidative phosphorylation (Slater, 1953; Karlson, 1970). From our results it is difficult to ascertain whether the higher levels of thyroxine in blood are the result of increased hormone synthesis or a consequence of blood dehydration. The question could also be raised concerning the extent of decoupling of oxidative phosphorylation under the influence of total thyroxine activity in the body or thyroxine level in blood.

It would seem likely that our hypothesis could explain the decreased food conversion observed in NaCl-deficient chickens.

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