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The Effects of Maternal Thiamine Nutrition on Thiamine Status of the Offspring in Broiler Chickens

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Abstract: The response of broiler chickens to a wide range of dietary supplementation of thiamine to broiler breeder diet was studied in order to understand the effects of maternal thiamine nutrition on the status of thiamine indices in the offspring. Thiamine, and thiamine pyrophosphate (TPP) content, and α-ketoglutarate dehydrogenase (KGDH) activity were measured in hearts from 20 day old chicken embryos and from chickens at 1, 7, 14, and 21 days of age and in blood at 21 days of age.

Total thiamine content in the heart of day old chicks was higher in comparison to 20 day old embryos. Maternal supplementation of thiamine increased heart thiamine in the offspring ($p < 0.001$), and increased the activity of KGDH in the hearts of day old chicks ($p < 0.001$), but not in the embryo. The TPP content in the heart increased in response to both maternal and offspring thiamine supplementation ($p < 0.001$), however the effect of broiler thiamine supplementation was largely independent from the maternal effect. The effect of maternal thiamine nutrition on the offspring's heart KGDH activity was apparent, but the responses to broiler supplementation were dependent largely on the maternal effect. Blood TPP content was not affected by maternal thiamine supplementation ($p = 0.39$), but thiamine supplementation in the offspring diets increased blood TPP ($p < 0.001$). Both maternal and offspring thiamine supplementation increased blood free base thiamine content (both $p < 0.001$). It is concluded from this study that maternal thiamine nutrition affects thiamine status indices and thiamine metabolism of the offspring.

Key words: Thiamine, maternal, transfer, nutrition, broiler

Introduction

Broiler chickens may have a higher requirement for thiamine because of the high carbohydrate content in a broiler ration, and the high metabolic demand for thiamine due to fast growth rate. Our previous study [1] has shown that prolonged and relatively high level of dietary thiamine supplementation were required to increase blood thiamine levels in broiler chickens. Further, in the absence of classical signs of thiamine deficiency, in chickens fed the basal diet (1.8 mg thiamine per kg), and even in those supplemented with 2 or 4 mg/kg, blood thiamine concentration declined over time. Since a decline in blood thiamine is the first sign observed in thiamine deprived animals, a pattern of declining blood thiamine in rapidly growing chickens indicates that the body demand for thiamine has not been at equilibrium with the dietary supply. This indicates that maternal transfer of thiamine may have been inadequate. In birds, the nutrients in the oocyte are influenced by the nutrition of the hen. All nutrients required for the development and growth of an avian embryo must be in the egg, and the egg yolk is the main source of nutrients

during the embryonic development. In the growing chick, the yolk sack is a significant contributor of nutrients for several days after hatching.

In broiler breeder, primary objectives of management are aimed at maximum egg production and fertility, and in order to achieve these goals considerable dietary restrictions are implemented. At present the effects of maternal nutrition on the performance and health of broilers are not well characterized.

In the present study we investigated the responses of the offspring to maternal nutrition of thiamine. Thiamine nutrition in broiler chickens is of interest because thiamine appears to have some protective effect on the heart that may be independent from its known metabolic function as a cofactor in enzymatic reactions [2, 3, 4, 5]. The incidence of heart diseases in broiler chickens is very high [6]. In broilers, a causal relationship between thiamine nutrition and sudden death can be inferred from the study of Classen *et al* [7] who observed that dietary thiamine supplementation decreased mortality due to Sudden Death Syndrome (SDS) in otherwise normal broiler flocks. Interestingly, both human and animal infants experience sudden death long before the appearance of fulminant thiamine deficiency signs in adults [8, 9].

The objective was to investigate the effects of dietary supplementation of thiamine in maternal diets on thiamine status indices in the heart tissue and blood of the offspring. Blood thiamine is a very sensitive measurement of dietary inadequacy. The heart was chosen as the test organ because: a) the broiler chicken heart appears to have much higher requirement for thiamine than liver or brain, b) thiamine levels in the heart increase at faster rate, and the effects of supplementation are more pronounced, c) almost all heart thiamine is in the form of TPP, and d) the heart does not store thiamine in the form of free base thiamine. For these reasons heart would be expected to be more sensitive to dietary thiamine manipulation.

Materials and Methods

Diets, Animals and Management

The broiler breeder diet was prepared according to the recommendation of the industry (Parent Breeder Management Guide; Hubbard Farms). The basal broiler diet was a practical wheat-soybean diet formulated according to current nutritional recommendations [10]. The mineral premix, thiamine free vitamin premix, and thiamine premix (wheat middlings containing 4 mg thiamine per gram) were prepared by Hoffmann-La Roche. The test diets were prepared by adding thiamine premix/wheat middlings to the basal diet at a rate to contain 0, 2, 8, and 32 mg supplementary thiamine/kg diet. Thiamine content in the basal breeder diets ranged from 2.9 to 3.6 mg/kg, and in the basal broiler diet 5.5 to 6.5 mg/kg.

Parental stock was from Hubbard Broiler Breeders (Hubbard Farms). A total of 144 females were randomly divided into groups of 36 (6 replications of 6 hens) and assigned to one of the four breeder thiamine treatment groups. The feeding regime of the breeder stock was according to the recommendation of the industry (Parent Breeder Management Guide; Hubbard Farms). The breeder hens were fed experimental diets from 15 weeks of age throughout the egg laying period. Collection of eggs started when the breeder hens were approximately 30 weeks of age. The eggs were collected for the period of 14 days, and stored at 12°C. The eggs were incubated at 37.2°C, and hatched at 36.1°C (Robinson, Incubator, Co. Denver CO).

For the measurement of thiamine status indices 96 day old chicks from each maternal group were randomly divided into 4 groups and assigned to one of the four broiler test diets. For the performance study, 96 day old chickens (48 per sex) from each of the four hen treatment groups were randomly divided into 4 groups of 24 (3 replications of 4 birds per sex) and assigned to one of four broiler dietary treatments with levels of supplemented thaimine at 0, 2, 8 and 32 mg/kg respectively. The chickens were housed in metal cages with raised wire floors in environmentally controlled rooms under a program of increasing lighting [11]. Temperature and light were controlled automatically. Feed and water were offered *ad libitum.* The birds were monitored daily for overt clinical abnormalities. The mortalities were collected every day and all dead birds were subjected to necropsy.

Sample and data collection

Hearts (12 individuals from each maternal group) were collected from embryos at day 20. After hatching, hearts were collected from 6 chickens from each of the four maternal groups on day 1, and thereafter from each treatment group at days 7, 14, and 21. Blood was collected from each treatment at day 21. For performance data live body weights were obtained on days 1, 7, 14, and 21. The feed consumption data were collected accordingly. Thiamine content and KGDH activity were measured in hearts from broiler chicken embryos (day 20), and from chickens at 1, 7, 14, and 21 days of age. Blood thiamine concentration was measured at day 21.

Analytical Methods

For the measurement of KGDH activity and thiamine concentration, heart tissue (approximately 150 to 300 mg) was homogenised in 3 ml of cold 50 mM phosphate buffer (pH 7.6) containing 2 mM mercaptoethanol and 1 mM EDTA.

Blood cells were lysed by freezing at –20°C. The activity of the enzyme was measured in crude heart homogenates diluted three times in working buffer. The working buffer (pH 7.6) was comprised at 63 mM Tris, 1.25 mM MgCl, 1.25 mM CaCl, 0.63 mM dithiothreitol, 0.63 mM 2-mercaptoethanol, 0.63 mM K-EDTA, 0.13% Triton X100. The α-KGDH activity assays were carried out on flat bottom 96-well microplates (Corning, NY). Three reaction mixtures: a) blank (3.1 mM NAD), b) assay (3.1 mM NAD, 0.13 mM coenzyme A, 2 mM α-ketoglutarate and c) TPP-assay (3.1 mM NAD, 0.13 mM coenzyme) A, 2 mM α-ketoglutarate, 0.3 mM TPP) were prepared using working buffer. The samples were distributed on the plate in aliquots of 10 µl per well, 4 wells per sample. Using a multichannel pipeter, 190 µl of either blank, assay or TPP-assay reaction mixtures were added to the appropriate wells using a predesigned pattern to allow a blank, assay and TPP-assay for each sample. The plate with prepared assays was placed in a temperature (37°C) controlled chamber of a microplate reader (Anthos Labtech Inst. Salzburg, Austria). The reaction was equilibrated for 2 min, and the measurements were performed at 1 min intervals for 5 min. The quantitation of the assay is based on the measurements of NADH elaborated during the reaction at 340 nm. The light path for the well containing 200 µl assay mixture was 6.1 mm, hence the value of 6.22 for 10 mm path of mM extinction coefficient for NADH at 340 nm was assumed as 3.7942 for 6.1 mm path. All reagents were supplied by Sigma. Protein content in the assays was measured using Sigma diagnostic Procedure 610-A (Sigma, St. Louis, MO). Thiamine was measured as described by Olkowski and Classen [1].

Statistical Analysis

Statistical analysis was carried out using ANOVA from the microcomputer package Number Cruncher Statistical System [12]. Three way analysis of variance was used to identify the main (maternal treatment, offspring treatment, age) effects and interactions. Fisher LSD test at $\alpha = 0.01$ [13] was used to determine statistically significant differences between the means.

Table I: Cofactor requirements for KGDH activity study

Reaction Mixture	Heart KGDH (% activity)
Complete	100
Omitted: Coenzyme A	ND
Omitted: α-ketoglutarate	ND
Omitted: NAD^+	ND
Omitted: TPP	90–100

ND: not detectable

Results

Procedural

Thiamine analyses

The detection limit was < 1 ng thiamine/ml. The method was validated using reproducibility and recovery tests. The recovery of thiamine from samples spiked with thiamine ranged from 95–100% and the reproducibility test showed coefficients of variation of less then 5%. The extraction of thiamine from the samples was considered to be complete since subsequent extractions of the pellets remaining after the first extraction did not yield any detectable thiamine. Standard curves using data points from six concentrations ranging from 1.25 to 40 ng/ml routinely had correlation coefficients exceeding 0.999.

KGDH activity assays

Under the described assay conditions the enzyme activity was linear with respect to protein content and time for at least 10 min. The dependence of the assay on the presence of cofactors is shown in Table I. Optimal pH of the

Table II: Heart thiamine (total, TPP, and free base) content (µg/g tissue) and KGDH activities (nmol/mg prot/min) in 20 day old broiler chicken embryos and day old broiler chicks in response to various levels of thiamine supplementation in the maternal diet

Thiamine Supplement	Thiamine Status Indices in 20 Day Old Embryo				Thiamine Status Indices in Day Old Chicken			
(mg/kg)	Total B_1	TPP	Free Base	KGDH	Total B_1	TPP	Free Base	KGDH
0	†0.93^a	0.56	0.25^a	29.22^a	1.28^a	1.19^a	ND	30.75^a
2	1.02^a	0.50	0.39^b	29.00^a	1.66^b	1.47^b	ND	39.06^b
8	1.22^b	0.51	0.55^c	25.87^b	2.08^c	1.62^{bc}	ND	37.95^b
32	1.33^b	0.50	0.65^d	27.48^{ab}	2.38^d	1.81^c	0.03	33.81^a
				Statistical Analysis				
Pooled SE	0.043	0.026	0.028	0.69	0.077	0.065	NA	1.24
ANOVA (P)	< 0.001	= 0.291	< 0.001	< 0.01	< 0.001	< 0.001	NA	< 0.001

†-mean; ND: not detectable, NA: not analysed, different superscript letters indicate significant differences at $\alpha = 0.05$

assay was 7.5 to 7.6. Interestingly, the change of the medium pH does not appear to affect enzyme activity *per se,* but rather increases the requirement for coenzyme, as the losses in activity due to changes in pH can be restored by increasing the concentration of TPP in the medium.

Experimental

Heart thiamine status indices, the effects of age and maternal thiamine nutrition

Thiamine supplementation in the hen diet increased egg yolk thiamine ($p < 0.001$), with means of 0.93, 1.69, 2.54, and 5.97 (µg/g yolk) for the levels 0, 2, 8, and 32 mg of supplemented thiamine per kg of hen diet respectively. Almost all yolk thiamine was in the form of free base. Egg white was almost void of thiamine. The profile of thiamine status indices differed between 20 day old embryos and day old chickens (Table II). Heart total thiamine content of day old chicks was some 40 to 80% higher in comparison to 20 day old embryos. Dietary supplementation of thiamine in the hen diet increased heart thiamine in the offspring ($p < 0.001$). Of the embryo's heart thiamine, the increment was largely attributed to free base thiamine, which constituted 25 to 50% of the total thiamine. In the chicks, the increment in heart thiamine was attributed mainly to TPP, which constituted the bulk of heart thiamine. Free base thiamine was not detectable in the hearts of the majority of day old or older chickens, and was found in trace amounts only in a few individuals. Figure 1 shows the effects of age and maternal thiamine nutrition on heart TPP profile in the offspring not supplemented with thiamine. The content of TPP in the embryonic heart was largely not affected by maternal dietary thiamine ($p = 0.291$), but in day old chicks TPP level in the heart was correlated with the level of thiamine supplementation in the maternal diet ($p < 0.01$). The effect of age on heart thiamine content and the interaction between age and hen treatment were also significant (both $p < 0.01$). Overall the TPP content in the heart increased with age in the offspring of both thiamine supplemented and unsupplemented hens ($p < 0.001$), but the effect of maternal thiamine supplementation was apparent at 1, 7, and 14 days of age.

The embryo's heart KGDH activity was largely not affected by maternal dietary thiamine with exception of embryos from hens fed 8 mg/kg of supplemented thiamine was lower (Table II). The reason for this discrepancy is uncertain. However, since the relative pattern of changes over time throughout the remainder of the experiment in this group was similar to other groups, we regard this as an experimental variation associated likely with sample collection or analytical variability rather than dietary effect *per se.* Overall supplementation of thiamine in the maternal diet increased the activity of KGDH in the hearts of day old chicks (Table II) with differences significant ($p < 0.001$) at the levels of supplementation of 2 and 8 mg/kg. There were significant effects of age and maternal thiamine on heart KGDH activity, and an interaction between age and hen treatment (all $p < 0.001$). Figure 2 shows the changes in heart KGDH activity with age and in response to maternal thiamine supplementation. Overall KGDH activity in the heart increased with age in the offspring of

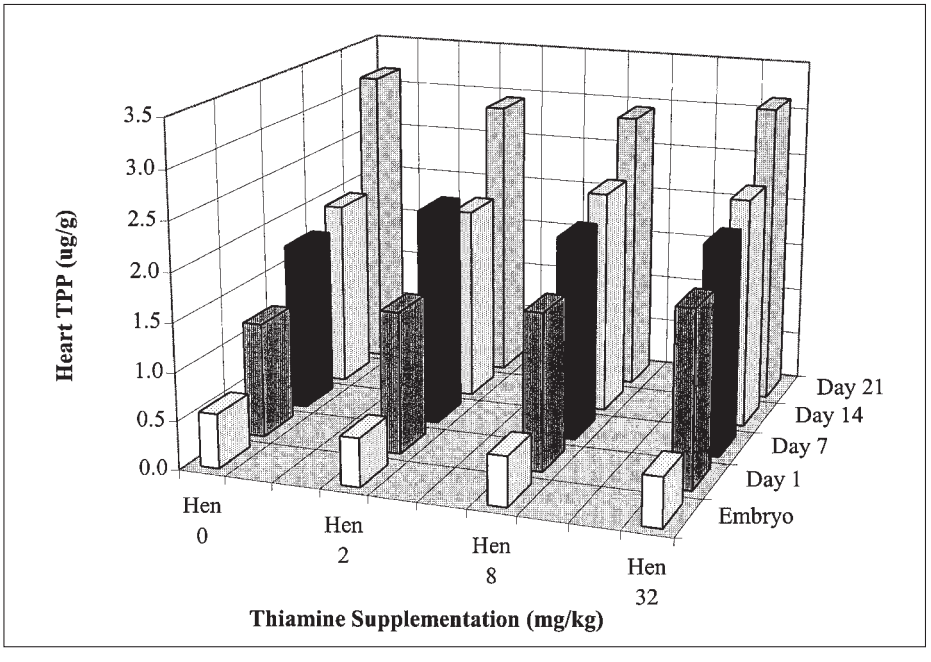

Figure 1: Changes in heart TPP content in the offspring of hens fed diets containing various levels of thiamine. To emphasize the effects of maternal thiamine nutrition the data represent heart TPP concentration of only those chicks that were fed broiler diet unsupplemented with thiamine.

both thiamine supplemented and unsupplemented hens ($p < 0.001$). However, unlike in the offspring of unsupplemented hens, the effect of maternal thiamine supplementation on KGDH activity was apparent at 1 and 7 days of age.

Heart and blood thiamine status indices, the effects of maternal and offspring thiamine nutrition

There were significant effects of maternal thiamine supplementation and offspring thiamine supplementation on heart TPP content (both $p < 0.001$), but there was no interaction between hen treatment and broiler treatment ($p = 0.72$). Figure 3 shows the effects of maternal and broiler dietary thiamine supplementation on heart TPP profile in the offspring. Overall the TPP content in the heart increased in response to both maternal supplementation and offspring thiamine supplementation, however the effect of broiler thiamine supplementation was largely independent from the maternal effect.

There were significant effects of maternal dietary thiamine supplementation and offspring dietary thiamine supplementation on heart KGDH activity (both $p < 0.001$), and a significant interaction between hen treatment and broiler treatment ($p < 0.02$). Figure 4 shows the effects of maternal and broiler dietary thiamine supplementation on heart KGDH activity profile in the offspring. The effect of maternal thiamine nutrition on the offspring's heart KGDH activity was apparent, however the responses to broiler thiamine supplementation were dependent largely on maternal effect.

Blood TPP content was not affected by maternal thiamine supplementation ($p = 0.39$), but broiler thiamine supplementation in broiler diets increased blood TPP ($p < 0.001$). There were significant effects of maternal thiamine supplementation and offspring thiamine supplementation on blood free base thiamine ($p < 0.001$), and significant interaction between hen treatment and broiler treatment ($p < 0.003$). Figure 5 shows the effects of maternal and broiler dietary thiamine supplementation on blood free thiamine profile in the offspring. The overall effects of thiamine supplementation in maternal diet or offspring diet were distinctly different (Figure 6). Maternal thiamine had considerably stronger impact on blood free thiamine a lower levels of supplementation.

Performance

Table III shows the effects of maternal and broiler dietary thiamine supplementation on body weight gain and gain: feed ratio. Feed efficiency was improved by the supplementation of maternal diet ($p < 0.024$), but was not significantly affected by thiamine supplementation in the broiler diet ($p = 0.18$). Maternal thiamine supplementation tended to increase body weight gain, but the differences were statistically not significant. Interestingly, broiler treatment of 32 mg/kg had detrimental effect on body weight gain. A total of 18 chickens died during the course of this study. Among 10 chickens that died of SDS, 6 were from unsupplemented hens, 3 and 1 SDS cases were from hens supplemented with thiamine at levels 2 and 32 mg/kg respectively.

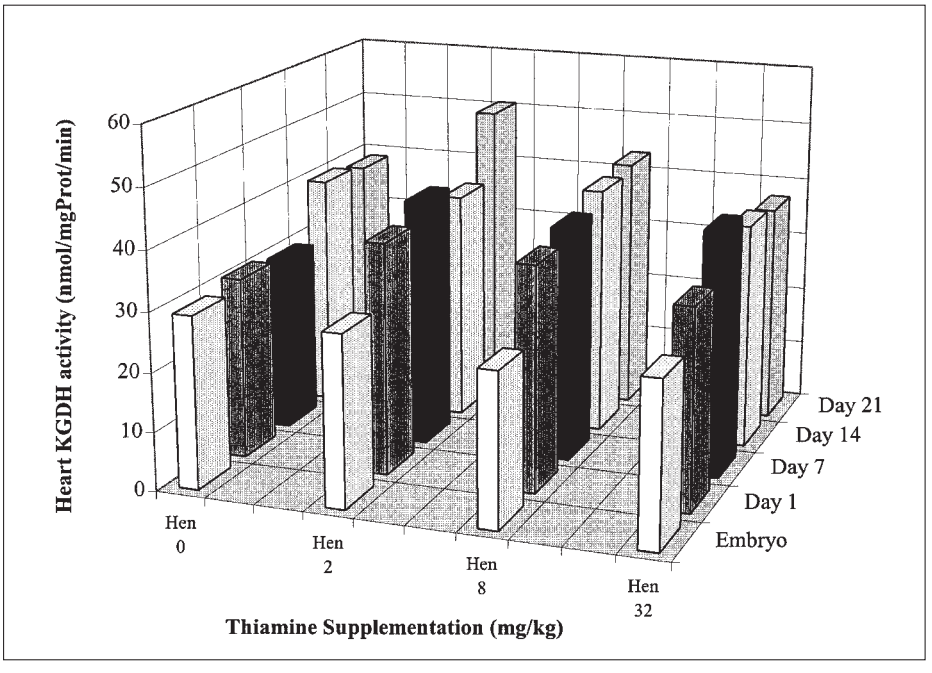

Figure 2: Changes in heart KGDH activity in the offspring of hens fed diets containing various levels of thiamine. To emphasize the effects of maternal thiamine nutrition the data represent KGDH activity only those chicks that were fed broiler diet unsupplemented with thiamine.

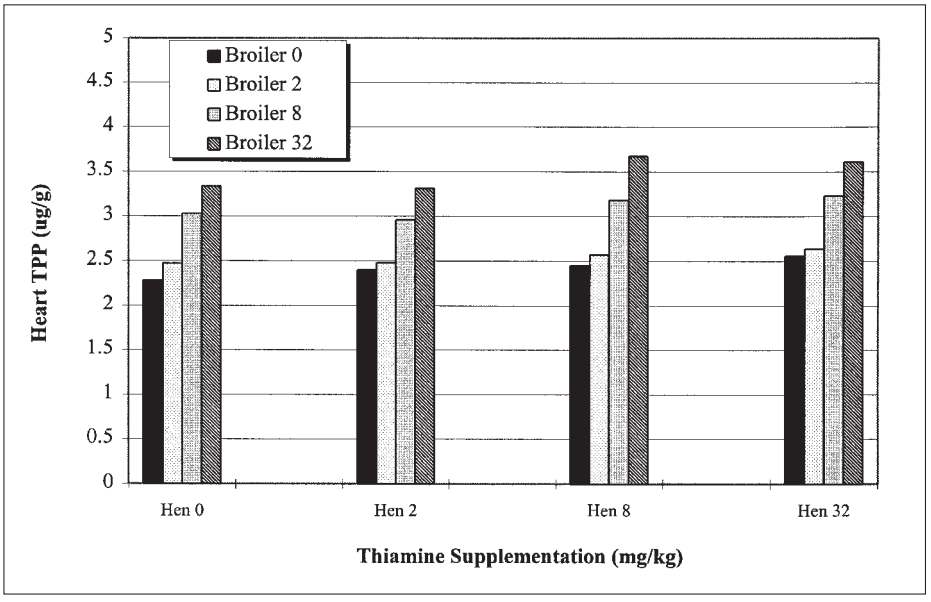

Figure 3: Changes in heart TPP content in the offspring in response to thiamine supplementation in maternal and offspring diets. The values are overall means of main effects of maternal and broiler dietary thiamine supplementation respectively.

Discussion

This study revealed several, previously not characterized features of thiamine metabolism in the chicken. Firstly, there seems to be a drastic increase in thiamine demand after hatching. Overall, the total thiamine content in the heart tissue increased in all groups of chickens. The TPP level in the heart after hatching increased more than 2 fold in comparison to the 20 day old embryo. This apparent increase in demand for TPP may be associated with changes in metabolic activity after hatching. TPP serves as a coenzyme for pyruvate dehydrogenase (PDH), α-ketoglutarate dehydrogenase (KGDH), and transketolase. PDH and KGDH are the two key rate limiting enzyme complexes involved in the synthesis of energy derived from carbohydrates.

Interestingly, free thiamine constituted a considerable share of the total thiamine in the heart of the embryo, but this data and our previous study [1] showed that in the heart of one day old or older chickens, free thiamine is virtually at not detectable levels. In the brain or liver tissue of the chicken free base thiamine constituted up to 20% of total thiamine [1]. It is intriguing that almost all thiamine in the chicken's heart is in the form of TPP. Trans-

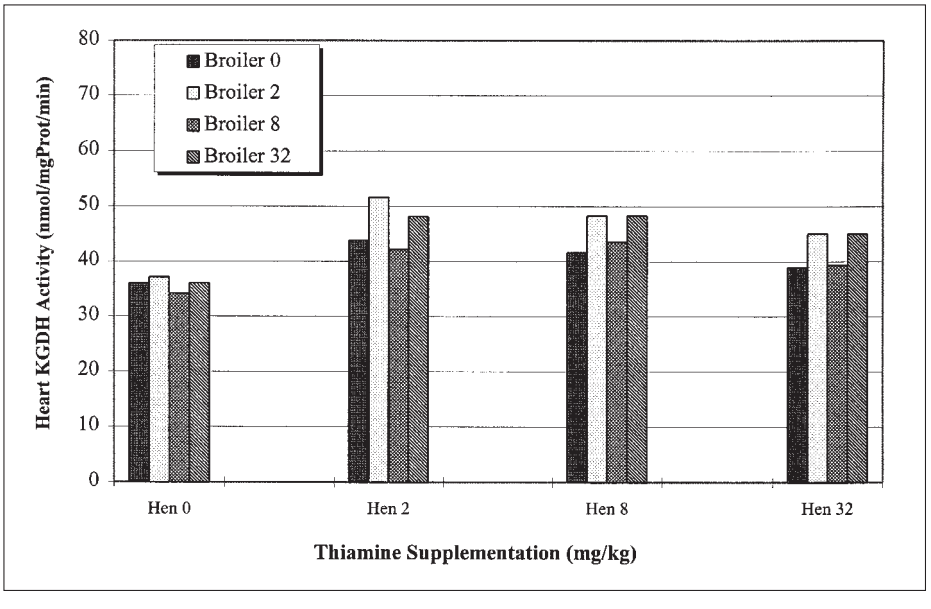

Figure 4: Changes in heart KGDH activity in the offspring in response to thiamine supplementation in maternal and offspring diets. The values are overall means of main effects of maternal and broiler dietary thiamine supplementation respectively.

Table III: Average body weight gain and gain: feed ratio in the offspring in response to thiamine supplementation in maternal or offspring diets. The values are means of main effects of maternal and broiler dietary thiamine supplementation for the period 1 to 21 days

Thiamine Supplement	Average Body Weight Gain (g) (main effects, days 1 to 21)		Average Gain: Feed Ratio (main effects, days 1 to 21)	
(mg/kg basal diet)	Maternal diet	Offspring diet	Maternal diet	Offspring diet
0	730.8†	751.3[a]	0.717[a]	0.727
2	745.0	765.9[a]	0.719[a]	0.729
8	753.1	749.4[a]	0.724[ab]	0.722
32	756.8	719.1[b]	0.735[b]	0.717
		Statistical Analysis		
Pooled SE	9.97	9.97	0.004	0.004
ANOVA (P)	= 0.27	< 0.013	< 0.024	= 0.18

†-mean; different superscript letters indicate significant differences at $\alpha = 0.05$

ketolase activity in the heart tissue of broiler chickens (if any in cardiomyocytes) is extremely low [1]. We did not examine PDH activity, but this study has shown that merely increasing the content of TPP in the heart does not result in increased activity of KGDH. Hence, there is a lingering question what is the metabolic significance of this apparent reserve of TPP in the heart.

Heart KGDH activity appears to be sensitive to changes in pH. Our *in vitro* observations indicate that even small changes in pH may result in a relatively large decrease in activity of this enzyme. Interestingly, however, the effect of pH does not appear to affect the apoenzyme, but rather the requirement for coenzyme, as loss of the activity can be restored by increasing the concentration of TPP in the medium. Could the tendency of the heart to store most of its thiamine reserves in the form of TPP be a form of protective mechanism to maintain its energy metabolism functional in a situation of local or systemic acidosis or alkalosis states? Larrieu *et al* [2] reported beneficial effects of a cocarboxylase (TPP) solution in the treatment of an experimentally created acute myocardial infarction in dogs.

This study showed that thiamine supplementation in the offspring diet increased heart and blood thiamine content, which was an entirely expected outcome [1]. However, the present study showed also a highly significant effect of maternal thiamine nutrition on heart and blood thiamine status in the offspring. Based on the effects on blood thiamine, it can be inferred that the maternal nutrition of thiamine appears to have a long term effect on overall thi-

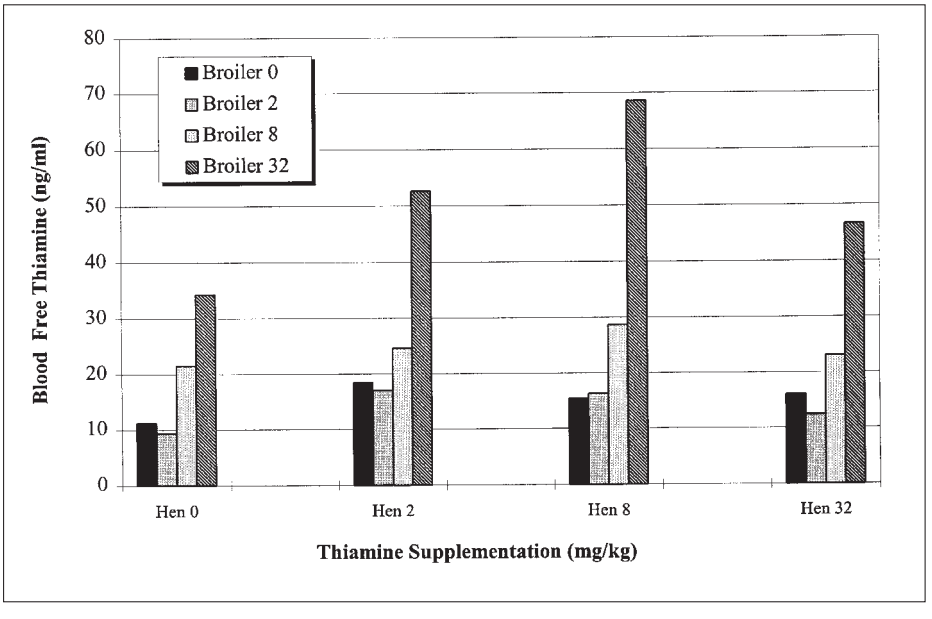

Figure 5: Changes in blood free thiamine content in the offspring in response to thiamine supplementation in maternal and offspring diets. The values represent effects for maternal and broiler diet respectively at the age of 21 days.

amine status in the offspring. Notably, blood TPP level was not affected by maternal thiamine nutrition. The most prominent impact of maternal effect was on the pool of free thiamine in the blood. From a thiamine metabolism standpoint this may be of significance since free base thiamine in the blood is likely the source of thiamine distributed to the organs. A strong interaction of maternal and offspring effects (Fig. 5) indicates that maternal thiamine supplementation resulted in metabolic changes enabling the offspring to utilize dietary thiamine more efficiently. Our previous study [1] showed that relatively high level of supplementation in broiler diet was required to increase blood thiamine. A similar trend was seen in the present study, but interestingly a strong effect of maternal thiamine nutrition was apparent at lower levels of supplementation (Fig. 6).

This study demonstrates that maternal nutrition may affect the metabolism of the offspring. To a certain extent some of these effects could be associated with an increased load of thiamine in the egg from supplemented hens. For instance, the increases in tissue or blood thiamine content can be explained by higher thiamine load, but the distribution of thiamine vitamers in blood or tissue, interaction of the effect of maternal and offspring nutrition, and the profile of KGDH activity indicate that the mechanism by which maternal nutrition affects offspring metabolism is more complex.

Of particular interest here are the responses of enzyme activity in relation to responses in total thiamine and coenzyme levels. Notably, the KGDH activity was mainly influenced by maternal dietary thiamine supplementation, but the effect of maternal nutrition of thiamine on the activity of the offspring's heart KGDH was not associated with the presence of coenzyme in the oocyte, since virtually all thiamine in the egg was in the form of free base thiamine. Further, there was a drastic increase in TPP level in the heart after hatching, which was likely associated with the overall change in metabolic profile. It is also important to stress that both TPP level and KGDH activity continued to increase with age, and that maternal thiamine supplementation greatly increased the response of these variables during the early growth stages of the chickens. Notably, although the offspring thiamine nutrition resulted in a similar effect on heart TPP status (Fig. 3), the KGDH activity was very little affected by offspring thiamine nutrition, but increased by maternal dietary thiamine supplementation (Fig. 4). Therefore the activity of this enzyme was not induced merely by increased levels of TPP in the tissue, but by a more complex mechanism. Hence, it is likely that an enhanced expression of this biochemical marker has occurred in the offspring, and was influenced by maternal nutrition.

The observation from our study generally support the metabolic programming hypothesis, whereby manipulation of the maternal diet may lead to a complex alteration in metabolic and humoral homeostasis of the offspring [14–16]. Barker *et al* [17] proposed that maternal undernutrition may program permanent physiological and biochemical changes in the conceptus that may cause diseases in later life. Based on extensive epidemiological observation, an inadequate intrauterine nutrition has been associated with an increase in the risk of heart disease later in life in humans [15, 17, 18]. It is notable that primary objectives of management of broiler breeders are aimed at

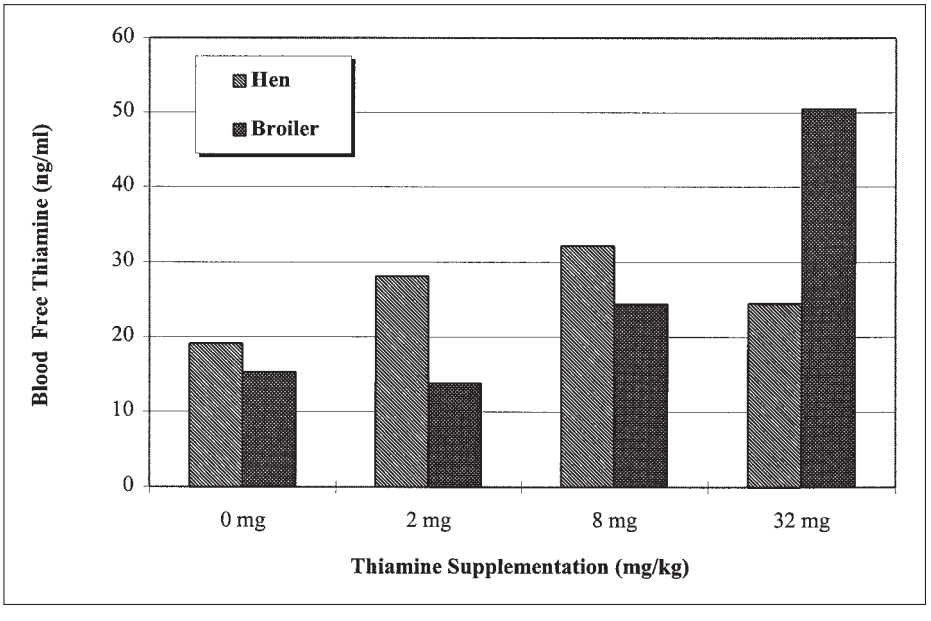

Figure 6: Main effects of thiamine supplementation in maternal or offspring diets on blood free thiamine content in the offspring at the age of 21 days.

maximum egg production and fertility, and in order to achieve these goals considerable dietary restriction are implemented. In this situation, the nutritional status of broiler breeders may have characteristics of marginal sufficiency. The issue of nutrient adequacy in broiler breeder nutrition in the context of the potential effects on health and performance of broilers warrants further investigation.

It is noteworthy that higher levels of thiamine in the diet had a detrimental effect on the host, which was evidenced by lowered thiamine status indices and performance (see Fig. 2, 4, 6, Hen32 data sets). The issue of over-supplementation deserves a comment. We observed a similar effect in our work on riboflavin [19], where the excess of dietary riboflavin tended to reduce tissue FAD, FMN, and riboflavin content. Hence, it appears that the level of dietary vitamins may determine how the host manages the available resources of these nutrients. It can be speculated that the feed-back mechanism regulates both absorption and tissue storage of vitamins, and in the situation of dietary abundance the efficiency of absorption, retention, and storage may be down-regulated by the host.

The present study brought about some new interesting data from an applied nutrition standpoint. Notably, overall feed efficiency was not affected by thiamine supplementation in the broiler diet, but was significantly improved by the thiamine supplementation in the maternal diet. Also maternal thiamine supplementation tended to have a beneficial effect on body weight gain, but the differences were not statistically significant. Nevertheless, the trends in body weight gain responses to maternal or broiler diet supplementation observed in this study warrant testing using larger populations. Also, there appears to be some correlation between maternal thiamine nutrition and the distribution of mortality due to SDS, and this also warrants further examination using larger population.

Acknowledgments

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