Vitamin B₆ in Poultry Nutrition
A Review*

N. J. Daghir

Faculty of Agricultural Sciences,
American University of Beirut, Beirut, Lebanon.

I. INTRODUCTION

The role of vitamin B₆ in poultry nutrition is really as old as the discovery of this vitamin by Gyorgy (1934). Lepkovsky et al. (1936) were the first to demonstrate the need of chicks for “Factor I” and identify this factor with vitamin B₆. Carter and O’Brien (1936) made a similar observation with pigeons. Several observations on the necessity of the vitamin for chicks were reported soon after the isolation of the vitamin (Carter and O’Brien, 1939; Hegsted et al., 1939; Jukes, 1939). Harris and Folkers (1939) accomplished a complete synthesis of vitamin B₆ starting with ethoxycetacacetone and cyanocacetamide. They elucidated the structure as 2-methyl-3-hydroxy-4, 5-di-hydroxy-methylpyridine. In accordance with the chemical nature of this vitamin, Gyorgy and Eckardt (1939) suggested the use of the term pyridoxine.

Snell et al. (1942), as the result of the comparison of microbiologic assays on extracts of natural materials with the values based on chemical and animal assays, were the first to recognize the existence of other forms of vitamin B₆. In a later report Snell (1942) assigned the name pyridoxamine to the aminated product of pyridoxine and pyridoxal to the formyl derivative of pyridoxine. Luckey et al. (1945) conducted three experiments in which growth and survival data of chicks indicated that pyridoxal is slightly more than half and pyridoxamine is about three-fourths as active as pyridoxine. Sarma et al. (1946) compared the activity of these three forms for chicks and rats and found that when mixed in the ration, pyridoxamine and pyridoxal are less active than pyridoxine, but when given by dropper or injected intraperitoneally, they all possess equivalent activities. Davies et al. (1959) reported that when given separately from the diet, the three analogues were equally active for the chick. When included in the diet, pyridoxamine was in three tests with chicks about 80% as active as pyridoxine. Waibel et al. (1952) on the other hand reported that pyridoxal and pyridoxamine are as active as pyridoxine when autoclaved starch is the dietary carbo-

*Contribution from the Faculty of Agricultural Sciences, American University of Beirut, as Journal Number 471.
hydrate, but less active than pyridoxine when sucrose or glucose was the carbohydrate.

This review will attempt to cover all work that has been done on chickens, turkeys and ducks in relation to the vitamin B₆ nutrition of these species. It will cover functions of vitamin B₆ in biochemical systems that have been studied in birds, pathology of vitamin B₆ deficiency, dietary requirements and factors affecting those requirements. A small section is also included on vitamin B₆ content of feedstuffs and other practical considerations of the vitamin in poultry feeding.

II. BIOCHEMICAL FUNCTIONS OF VITAMIN B₆ STUDIED IN POULTRY

It is well recognized that pyridoxal phosphate is the coenzyme for a great variety of enzymes involved in catalyzing various metabolic transformations. It has been observed that nearly all of these pyridoxal phosphate-dependent reactions are those of amino acids. It is not the intent of this review however to discuss the role of vitamin B₆ in amino acid metabolism which could be very extensive, but simply to point out selective aspects of vitamin B₆ metabolism that have been studied with poultry.

A. Erythropoiesis

Anaemia has been described as one of the symptoms of vitamin B₆ deficiency in poultry by many workers, (Hogan et al., 1941; Hegsted and Rao, 1945; Ferguson et al., 1961). In an attempt to explain the relationship of B₆ to erythropoiesis, Schulman and Richert (1955) showed that heme is synthesized in the red cells of vitamin B₆-deficient ducklings from glycine 2-C¹⁴ at a rate which is half of that found with control ducklings. Addition of pyridoxal phosphate in vitro restored the ability of the deficient cells to synthesize heme at a normal rate. The same workers (1956) observed that heme synthesis from 6-aminolevulinic acid-2,3-C¹⁴ proceeded at the same rate in B₆-deficient and normal ducklings. They concluded that pyridoxine deficiency exerted its effect on heme synthesis in the utilization of glycine and succinate for the formation of 6-aminolevulinic acid. Richert and Schulman (1959) reported that the rates of incorporation of glycine 2-C¹⁴ and of 6-aminolevulinic acid-2, 2-C¹⁴ into heme in vitro by erythrocytes of young ducklings increased in a straight line relationship with reticulocyte count when the isotope incorporation was plotted against reticulocyte count. The incorporation of radioactive glycine and 6-aminolevulinic acid into heme was decreased in vitamin B₆ deficiency. Glycine uptake was decreased more than 6-aminolevulinic acid uptake.

B. Tissue Transaminase Activities

The importance of enzymatic transamination in the biological system and its implication in amino acid metabolism has been evident for many years. In comparison to mammalian tissue, little information
is relatively available on the relationship of vitamin B₆ to transaminase activity of avian tissue. Brin, et al. (1954) studied the effect of vitamin B₆ deficiency on transaminase activity of cardiac muscle in ducklings. It was found that the activity of the glutamic-aspartate system was depressed 30% and that of the glutamic-alanine system 50% below normal. Restoration of activity to near normal was achieved by parenteral treatment of the ducks 16 hours prior to sacrifice with 10 mg of pyridoxine, pyridoxamine, or pyridoxal or by supplementation of the homogenates in vitro with 10 mcg pyridoxal phosphate. Goswami and Robblee (1958) studied aspartate aminotransferase activity in total blood, heart and liver of SCWL chicks from 0–35 days of age. In the group receiving the pyridoxine-deficient ration, the levels of activity in heart, liver and blood at three weeks of age were approximately 40, 53 and 50% respectively of those observed in day-old chicks. In the groups fed supplemental pyridoxine, the enzyme activity in the heart and liver remained constant to 5 weeks of age. The enzyme concentration in the blood declined to a level approximately 2/3 of that found in day-old chicks. Feeding pyridoxine at the level in excess of the chick’s requirement did not result in any increase in enzyme activity over that of chicks receiving the requirement level. Daghir and Balloun (1963) reported that serum aspartate aminotransferase activity in Leghorns was significantly less affected by low dietary vitamin B₆ than in Rhode Island Red or Vantress x Arbor Acre chicks. Kirchgessner and Maier (1968) reported that biochemical criteria such as the activity of aspartate aminotransferase are the best methods for determining vitamin B₆ requirements. The same workers (Kirchgessner and Maier, 1968a) showed that levels of B₆ below 3 mg/kg of feed could occur in practical rations and these could be detected by reduced serum aspartate aminotransferase activity. Sifri et al. (1972) reported that varying the dietary levels of pyridoxine had no significant effect on serum alanine aminotransferase activity whereas serum aspartate aminotransferase activity decreased in chicks given diets containing less than 3 mg pyridoxine per kg diet. It is noteworthy to point out here that in this study, the pyridoxine requirement for maximum serum aspartate aminotransferase levels was observed to be higher than for maximum body weight, food consumption and food utilization. Shiflett and Haskell (1969) studied the effect of B₆ deficiency on leucine transaminase activity in chick tissue. They observed a decrease in the activity of this enzyme in kidney, liver, heart and brain tissue; kidney tissue being the most sensitive and brain the least. These workers concluded that leucine transaminase activity in kidney is a highly sensitive indicator of vitamin B₆ nutrition in the chick.

C. Vitamin B₆ and Lipid Interrelationships

Work on various species during the past two decades has presented evidence that vitamin B₆ may be involved in various aspects of lipid metabolism, particularly in the metabolism of cholesterol and the
essential fatty acids. Below is some of this work that has been done with
poultry. Dam et al. (1958) studied the effect of vitamin B₆ deficiency on
cholesterol contents of plasma, liver, heart and aorta of chicks fed fat-
free diets or diets containing 10% peanut oil, with or without 1% dietary
cholesterol. Plasma cholesterol was significantly higher in the chicks
fed B₆-deficient diets than in chicks receiving diets with added pyrido-
oxine. Deficient chicks had a somewhat higher cholesterol content in
the aorta than those receiving added pyridoxine. Liver cholesterol was
not affected by vitamin B₆ deficiency. With regard to heart cholesterol,
the diet without added pyridoxine caused lower values than did the
Corresponding diets with added pyridoxine. The biochemical nature of
this relationship between vitamin B₆ deficiency and cholesterol meta-
bolism is not understood. Gaylor et al. (1960) studied the effect of
niacinic acid and related compounds on sterol metabolism in the chick
and rat. Nicotinic acid did not alter the blood cholesterol level of rats
fed a hypercholesteremia-inducing diet. However, the blood cholesterol
level of chicks fed a diet containing 0.5% cholesterol was depressed by
nicotinic acid. On the basis of the above finding, it has been suggested
(Sakuragi, 1959) that vitamin B₆ may affect serum cholesterol levels by
stimulating in vivo conversion of tryptophan to niacinic acid which in
turn may depress serum cholesterol levels. To test this hypothesis,
Daghir and Balloun (1962) fed chicks diets adequate or deficient in
vitamin B₆, with or without 1% nicotinic acid and fat-free or with 4% soybean oil. Serum, aorta, heart and liver cholesterol were significantly
higher in chicks fed vitamin B₆-deficient diets than in chicks receiving
added pyridoxine. Significantly greater aorta weights, in relation to
body weight, were also observed in chicks fed vitamin B₆-deficient
diets than in control chicks. In this work, however, nicotinic acid had
no significant effect in counteracting the hypercholesterolemia induced
by vitamin B₆ deficiency.
Lupien and Migocovsky (1964), who also observed elevated serum
cholesterol levels in pyridoxine-deficient chicks, studied the effect of
pyridoxine deficiency on rates of incorporation of C¹⁴ acetate and
mevalonate 2-C¹⁴ into serum and liver cholesterol in chicks. Rates of
incorporation of C¹⁴ acetate into both liver and serum cholesterol were
significantly depressed in pyridoxine deficient chicks. Mevalonate
2-C¹⁴ incorporation into liver cholesterol was not significantly depressed
by the hypovitaminosis, while incorporation into serum cholesterol
was significantly lowered. The authors concluded that pyridoxine
deficiency behaves like many other conditions with respect to cholesterol
metabolism. The effect is at the premevalonate stage. Porooshani
(1965), in a study on cholesterol metabolism in the growing chick,
observed that dietary cholesterol caused a significant increase in serum
and liver cholesterol regardless of the absence or presence of vitamin
B₆ in the diet. Vitamin B₆ deficiency, on the other hand, irrespective of
the presence or absence of dietary cholesterol mildly increased serum
cholesterol. This worker concluded that the increased serum cholesterol
in vitamin B₆-deficient chicks was not apparently due to the vitamin itself but rather the result of conditions created by the deficiency. Lupien (1967) investigated the possibility that in pyridoxine deficiency of chicks both physiological and biochemical effects on cholesterol metabolism may result in part from reduced thyroid activity. He observed that protein-bound iodine levels were markedly decreased in the pyridoxine-deficient groups while the thyroxine treated deficient birds showed elevated PBI levels. Two micrograms of thyroxine per day alleviated partially or totally most of the symptoms observed in chick pyridoxine deficiency. This author believes that thyroxine and pyridoxine participate in the biosynthesis of cholesterol in addition to their well-established functions.

Suggestions that pyridoxine may function as a co-factor in the synthesis of arachidonic acid from linoleic acid have not been upheld by the results of Moore et al. (1968). Although they found increased linoleate and decreased arachidonate in the liver lipids of chicks on a low pyridoxine intake, the effect was associated with changes in the proportions of triglycerides and phospholipids and was apparently not due to a direct involvement of the vitamin in the biosynthesis of arachidonate from linoleate. Daghir and Porooshani (1968) were similarly unable to demonstrate involvement of pyridoxine in the interconversions of polyunsaturated fatty acids.

III. PATHOLOGY OF VITAMIN B₆ DEFICIENCY

A. Chickens

Symptoms of vitamin B₆ deficiency in chicks were first described by Jukes (1939) and consisted of slow growth, depressed appetite, and inefficient utilization of food, followed in some cases by spasmodic convulsions and death. These symptoms were later confirmed by several workers (Lepkovsky and Kratzer, 1942; Fuller and Kifer, 1959; Daghir and Balloun, 1963).

Haematology of vitamin B₆-deficient chickens has been studied by several workers. Jukes (1939) reported no departures from the normal range in differential blood cell counts of chicks fed either a vitamin B₆-deficient basal diet or the basal diet supplemented with pyridoxine hydrochloride. All counts were made on the seventeenth day of the experimental period. Luckey et al. (1945) observed that vitamin B₆ deficiency symptoms in the chick included a decreased clotting time, hyperprothrombinaemia, small spleens and anaemia. Hegsted and Rao (1945) reported microcytic anaemia as one of the symptoms of vitamin B₆ deficiency in chicks. Daghir and Balloun (1963) noted haemorrhages in various areas of the body of pyridoxine-deficient chicks most striking around follicles of wing feathers. Gehle and Balloun (1965) also reported that vitamin B₆ deficiency in chicks causes a significant depression in haemoglobin and packed cell volume values. Asmar et al. (1968) confirmed the above finding and reported a significant decrease in serum
albumin. These data indicate that vitamin B₆ deficiency in the chick depresses erythrocytopoiesis and haemoglobin synthesis.

Increased incidence of gizzard erosion in vitamin B₆-deficient chicks has been reported by Daghir and Balloun (1963). Erosions varied in severity and were in most cases accompanied by haemorrhages. Histopathological manifestations of eroded gizzards were extreme dilatation of the glands of the tunica propria with the cells lining these glands becoming almost squamous in nature. This report was the first definite implication of vitamin B₆ in gizzard erosion. Bird et al. (1936) observed characteristic lesions in the gizzard lining while studying a factor at that time called vitamin B₄. It is quite possible that vitamin B₆ could have been part of this so-called vitamin B₄ factor. In studying the essential nature of vitamin B₆ for chicks, Hegsted et al. (1940) reported severe gizzard erosions in chicks fed some of the basal diets used. No indication was given, however, as to which diet was actually responsible for the erosion. Daghir and Haddad (1975, unpublished data) were able to reduce the erosion resulting in the B₆ deficiency state by feeding deficient chicks 0.5–1.0% taurocholic acid. They also observed that the increased incidence of gizzard erosion in the B₆ deficiency state is not due to reduced feed intake since pair-fed controls did not exhibit the same level of erosion.

Daghir (1962) observed in a number of chicks fed vitamin B₆-deficient diets atonic crop or more commonly referred to as pendulous crop. Since this condition was not regularly observed in a large number of chicks, it was not classified by the author as a definite manifestation of the deficiency. Miller (1963) reported pendulous crop in chicks fed semipurified diets deficient in vitamin B₆ as early as one week of age. Peak incidence of the deficiency however occurred during the 2nd and 3rd weeks of age.

More recently Gries and Scott (1972) reported that one of the signs of a borderline deficiency of pyridoxine in chicks is severe perosis. Hogan et al. (1941) had previously stated that perosis was one of the symptoms characteristic of vitamin B₆ deficiency.

In the laying hen, Cravens et al. (1943) observed that feeding a diet deficient in vitamin B₆ resulted in anorexia, rapid drop in body weight, and decreased egg production and hatchability. No convulsions were observed in these birds. Fuller et al. (1961) confirmed the previous findings and showed that severe pyridoxine deficiency resulted in complete cessation of laying. Attar et al. (1967) induced vitamin B₆ deficiency in laying hens by feeding them a pyridoxine-deficient diet for a period of 12 weeks. Deficiency symptoms observed were reduced feed intake, loss in body weight, cessation of egg production, and hyperexcitability alternating with droopiness. Reduction in feed consumption was observed at the end of the 2nd week of the experimental period, whereas hyperexcitability and droopiness appeared about the 5th–6th week.
B. Turkeys

Vitamin B₆ deficiency in Bronze turkey poults was studied by Bird et al. (1943) who reported that the deficiency in those birds was characterized by loss of appetite, poor growth, apathy, hyperexcitability when disturbed, convulsions, and death. Convulsions observed resembled those reported for the chick. Ferguson et al. (1961) in a study on the B-vitamin deficiency in mature turkey hens, observed that pyridoxine deficiency affected the appetite, decreased egg production to nearly zero after eight weeks of feeding, and depressed hatchability significantly. Anaemia and dermatitis of the legs and mouth were also observed in the deficient hens.

C. Ducks

Several studies on vitamin B₆ deficiency in the duck were conducted by Hegsted and Rao (1945). Severe acute deficiency in young ducklings was characterized by growth failure accompanied by severe anaemia. Neither convulsions nor paralysis were observed. Chronic vitamin B₆ deficiency in older ducklings caused lack of growth, paralysis, convulsions, severe microcytic anaemia, and poor feather development.

IV. DIETARY REQUIREMENTS FOR VITAMIN B₆

Early work on the vitamin B₆ requirements of chickens, turkeys and ducklings has been reviewed by Hogan (1950) and this was later updated by Fuller (1964). The following review therefore covers only work reported during the last decade. Table 1, however, summarizes all published reports available to the author on the vitamin B₆ requirements of poultry.

A. Chickens

With practical broiler rations, Maier and Kirchgessner (1967) reported that 3 mg of vitamin B₆ per kg of feed was adequate for maximum growth, feed efficiency, and deposition of dry matter, fat and nitrogen. The same workers (Kirchgessner and Maier, 1968) employing a semisynthetic diet found that the optimum requirement for broiler chickens up to 5 weeks of age was 5–6 mg per kg of diet. Besides using the above criteria for determining requirement, they added in this study serum aspartate aminotransferase and γ-globulin in total serum protein as indicators of adequacy.

Experiments with broiler chicks given purified diets containing levels of pyridoxine ranging from 1.7–7.1 mg/kg were conducted by Sifri et al. (1972). These workers concluded that the requirement was between 2–3 mg/kg and that the requirement for growth and feed utilization was less than for maximum serum aspartate aminotransferase levels. Gries and Scott (1972) reported that with a 22% dietary protein level, 1.4 mg B₆/kg diet was adequate for growth and prevention of all signs of deficiency.
TABLE 1: Requirements of Poultry for Vitamin B6 (mg/kg diet)

<table>
<thead>
<tr>
<th>Category</th>
<th>Requirement</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Growing chickens</td>
<td>2.5</td>
<td>Stockstad et al. (1940)</td>
</tr>
<tr>
<td></td>
<td>3.5</td>
<td>Hogan et al. (1941)</td>
</tr>
<tr>
<td></td>
<td>2.7-3.0</td>
<td>Briggs et al. (1942)</td>
</tr>
<tr>
<td></td>
<td>3.0</td>
<td>Lucas et al. (1946)</td>
</tr>
<tr>
<td></td>
<td>2.0</td>
<td>Kratzer et al. (1947)</td>
</tr>
<tr>
<td></td>
<td>3.3</td>
<td>Fuller and Kifer (1959)</td>
</tr>
<tr>
<td></td>
<td>3.5</td>
<td>ARC (1963)</td>
</tr>
<tr>
<td></td>
<td>5-10</td>
<td>Kirchgessner and Friesecke (1963)</td>
</tr>
<tr>
<td></td>
<td>2.6</td>
<td>Daghir and Balloun (1963)</td>
</tr>
<tr>
<td></td>
<td>3.0</td>
<td>Maier and Kirchgessner (1967)</td>
</tr>
<tr>
<td></td>
<td>6.7</td>
<td>Morimoto et al. (1967)</td>
</tr>
<tr>
<td></td>
<td>5-6</td>
<td>Kirchgessner and Maier (1968)</td>
</tr>
<tr>
<td></td>
<td>3.0</td>
<td>NRC (1971)</td>
</tr>
<tr>
<td></td>
<td>2.3</td>
<td>Sifri et al. (1972)</td>
</tr>
<tr>
<td></td>
<td>1.4</td>
<td>Gries and Scott (1972)</td>
</tr>
<tr>
<td>Laying chickens</td>
<td>2.0</td>
<td>Cravens et al. (1946)</td>
</tr>
<tr>
<td></td>
<td>2.0</td>
<td>ARC (1963)</td>
</tr>
<tr>
<td></td>
<td>3.0</td>
<td>NRC (1971)</td>
</tr>
<tr>
<td>Breeding chickens</td>
<td>2.0</td>
<td>Cravens et al. (1946)</td>
</tr>
<tr>
<td></td>
<td>4.4</td>
<td>Fuller et al. (1961)</td>
</tr>
<tr>
<td></td>
<td>4.0</td>
<td>ARC (1963)</td>
</tr>
<tr>
<td></td>
<td>4.5</td>
<td>NRC (1971)</td>
</tr>
<tr>
<td>Turkey poults</td>
<td>3.0</td>
<td>Kratzer et al. (1947)</td>
</tr>
<tr>
<td></td>
<td>3.0</td>
<td>ARC (1963)</td>
</tr>
<tr>
<td></td>
<td>3.9-4.4</td>
<td>Sullivan et al. (1967)</td>
</tr>
<tr>
<td></td>
<td>5.0</td>
<td>Jones et al. (1970)</td>
</tr>
<tr>
<td></td>
<td>4.0</td>
<td>NRC (1971)</td>
</tr>
<tr>
<td>Growing ducks</td>
<td>2.5</td>
<td>Hegsted and Roa (1945)</td>
</tr>
<tr>
<td></td>
<td>3.0</td>
<td>Hogan (1950)</td>
</tr>
<tr>
<td></td>
<td>2.5</td>
<td>ARC (1963)</td>
</tr>
<tr>
<td></td>
<td>2.6</td>
<td>NRC (1971)</td>
</tr>
</tbody>
</table>

Since the review of Fuller (1964) no report has been published on the vitamin B6 requirement of the laying chicken with the exception of the NRC (1971) recommendation of 3 mg per kg diet.

B. Turkeys

Sullivan et al. (1967) in studies with young turkeys raised to four weeks of age, showed that these birds require between 3.9-4.4 ppm of dietary pyridoxine for maximum livability and body growth. In these studies, semipurified diets were used supplemented with levels of pyridoxine HCl ranging from 1.9-4.9 ppm. Jones et al. (1970) however studied the response of turkey poults fed practical diets to pyridoxine supplementation. These workers observed significant responses in rate of growth from the addition of 5 mg/kg of pyridoxine to the feed.

C. Ducks

The only recommendation available for the growing duck since the report of Fuller (1964) is that of the NRC (1971) which specifies a requirement of 2.6 mg per kg of feed.

V. FACTORS AFFECTING VITAMIN B6 REQUIREMENTS OF POULTRY

There seems to be several indications from the literature that a number of factors affect the vitamin B6 requirements of poultry and thus could increase the need for this vitamin.
A. Dietary Protein Level

The level of dietary protein has been reported to affect the vitamin B$_6$ requirement of a number of animals, and birds are no exception. Kirchgessner and Friesecke (1963) observed that the vitamin B$_6$ requirement in the growing cockerel is related to protein intake. White Leghorn chicks fed 18.7% protein and 2 mg of vitamin B$_6$ per kg diet were observed by these workers to develop deficiency symptoms, but no mortality. When 22 and 23% protein levels were fed, mortality was over 60% at the 2 mg per kg basal diet but not with 2.6 mg of the vitamin B$_6$ per kg. Gries and Scott (1972) reported that a level of pyridoxine of 1.4 mg/kg when fed with a 22% protein level produced growth which was as good as that obtained with higher levels of pyridoxine and prevented all lesions and signs of deficiency. When the protein level was raised to 31%, however, the minimum pyridoxine requirement was 3.4 mg per kg diet. Daghir and Shah (1973) confirmed the effect of dietary protein level on the vitamin B$_6$ requirement by showing that increasing the vitamin B$_6$ level from 3.1 to 6.1 mg/kg diet resulted in a significant increase in body weight gains, feed efficiency and serum aspartate aminotransferase activities at high dietary protein levels (25%) but not at low levels (15%).

B. Protein Quality

Just as the quantity of dietary protein has been shown to play an important role in determining the need of the animal for vitamin B$_6$ so has the quality of the protein used. Anderson et al. (1949) were among the first to observe that growth of chicks was depressed by the addition of methionine or cystine to a vitamin B$_6$-deficient diet and that in the presence of adequate levels of the vitamin the growth was increased by these same amino acid additions. Fuller (1964) confirmed the growth retardation caused by excess methionine in diets low in vitamin B$_6$. Furthermore, vitamin B$_6$ deficiency symptoms were accentuated by high levels of methionine. Increasing dietary levels of pyridoxine overcame this deleterious effect. Kazemi and Daghir (1971) conducted several experiments to determine whether vitamin B$_6$ additions could improve diets low or marginal in methionine. Vitamin B$_6$ in these studies could not spare methionine when this amino acid was severely deficient in a purified soybean-dextrose type ration. However, high vitamin B$_6$ could replace methionine in a partially methionine-deficient purified ration. Improvement of growth and feed efficiency by methionine supplementation of purified chick diets could be increased by adding levels of vitamin B$_6$ above 3 mg per kg of diet. This may account for some of the reports in which supplemental vitamin B$_6$ above the requirement has improved growth on practical type diets (Bird and Rubin, 1946; Dillon, 1962; Fuller and Kifer, 1959).

C. Effect of Vitamins and Minerals

Bird and Rubin (1946), in a study on the value of pyridoxine in chick diets free of animal protein, observed that additions of high levels
of pyridoxine were capable of exerting good effects on growth of chicks fed diets with high choline content. Saville et al. (1967) reported that excess choline was inducing a pyridoxine deficiency, which could be overcome by withdrawal of the choline or by giving extra pyridoxine.

The effect of varying doses of vitamin B$_6$ on the utilization of minerals was studied by Kirchgessner and Muller (1966). With a B$_6$-deficient diet, there was a significant reduction in the utilization of K and Mo. Increased vitamin B$_6$ supplements reduced the utilization of Mo as compared with the doses required to meet B$_6$ demands.

D. Naturally Occurring B$_6$ Antagonists

The presence in linseed meal of a factor antagonistic to vitamin B$_6$ was first reported by Kratzer and Williams (1948). Kratzer et al. (1954) found that this factor was water soluble. Mickelsen and Yang (1966) reported that this factor could be destroyed by autoclaving or by incubation of the meal either with water or 50% ethyl alcohol. The isolated antagonist was found to be as potent as 4-deoxypyridoxine and its concentration in linseed meal was reported to be 0.002–0.005%. The toxic principle was finally identified by Klosterman et al. (1967) to be 1-[(N-γ-L-glutamyl)-amino]-D-proline. Similar antagonists have not been reported in other feedstuffs, but their existence cannot be excluded. Slesinger and Otevrellova (1970) observed that the pyridoxine level in the serum of chicks fed a practical type of ration in which 70% of the protein had been replaced by toasted horsebeans was significantly lower than that of the controls. Whether this is an indication of the presence of a B$_6$ antagonist in horsebeans it is not known.

E. Effect of Drugs

The effects of supplementing poultry feeds with drugs on vitamin B$_6$ requirements have been the subject of several investigators. Fuller and Dunahoo (1959) tested the effect of various drugs used commercially in poultry rations. They found that furazolidone and arsanilic acid used in combination depressed the growth of chicks significantly only when the diet was deficient in vitamin B$_6$. The addition of pyridoxine HCl overcame most of this growth depression. Dillon (1962) confirmed the above growth depression of furazolidone and the effect of pyridoxine on this depression. Maier and Kirchgessner (1967b) on the other hand showed that supplementation of a practical ration with 80 mg DoT (3,5-Dinitro-O-toluamide) per kg does not influence the vitamin B$_6$ requirements of broilers. Kirchgessner et al. (1971) studied the effect of DL-penicillamine and chlortetracycline on carcass nitrogen content and N-retention of broiler chicks and the effects of high levels of vitamin B$_6$ on these criteria. Both DL-penicillamine and chlortetracycline enhanced carcass nitrogen particularly in groups receiving vitamin B$_6$. N-retention was improved by DL-penicillamine only when vitamin B$_6$ was added.
F. Effect of Breed

Several reports indicate that genetic differences between breeds can affect the vitamin B6 requirement. Lucas et al. (1946) suggested an exceptionally high B6 requirement for Rhode Island Red × Barred Rock chicks fed both simplified and purified diets. Daghir and Balloun (1963) evaluated the B6 requirement of Leghorns, RIR and Vantress × Arbor Acre chicks using several criteria. They observed that serum aspartate aminotransferase activity in Leghorns was significantly less affected by low dietary vitamin B6 than in the other two breeds.

VI. VITAMIN B6 CONTENT OF FEEDSTUFFS

The need for reliable data on the vitamin B6 content of feedstuffs to aid in the assessment of diets for poultry cannot be over emphasized. Generally, it has been assumed that the vitamin B6 content of feeds was adequate to meet the bird's requirements for this vitamin. However, we have seen in the previous section that a variety of conditions have been shown to increase the need for this vitamin.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood meal</td>
<td>0.99 (2)*</td>
<td>0.00 (2)</td>
<td>—</td>
</tr>
<tr>
<td>Fish meal</td>
<td>1.06 (9)</td>
<td>3.7 (9)</td>
<td>3.1 (14)</td>
</tr>
<tr>
<td>Fish solubles</td>
<td>—</td>
<td>—</td>
<td>2.7 (3)</td>
</tr>
<tr>
<td>Herring meal</td>
<td>1.30 (2)</td>
<td>3.7 (4)</td>
<td>4.4 (3)</td>
</tr>
<tr>
<td>Meat and bone meal</td>
<td>1.27 (7)</td>
<td>2.3 (7)</td>
<td>1.0 (2)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Alfalfa meal (20%)</td>
<td>—</td>
<td>—</td>
<td>8.4 (5)</td>
</tr>
<tr>
<td>Barley</td>
<td>1.86 (9)</td>
<td>2.7 (9)</td>
<td>4.5 (2)</td>
</tr>
<tr>
<td>Coconut meal</td>
<td>0.90 (2)</td>
<td>4.1 (4)</td>
<td>—</td>
</tr>
<tr>
<td>Corn</td>
<td>3.47 (4)</td>
<td>7.1 (5)</td>
<td>5.3 (19)</td>
</tr>
<tr>
<td>Corn gluten meal</td>
<td>—</td>
<td>—</td>
<td>7.4 (4)</td>
</tr>
<tr>
<td>Cottonseed meal</td>
<td>4.01 (4)</td>
<td>5.8 (6)</td>
<td>7.0 (2)</td>
</tr>
<tr>
<td>Linseed meal</td>
<td>3.27 (4)</td>
<td>16.4 (5)</td>
<td>8.8 (5)</td>
</tr>
<tr>
<td>Lucern green meal</td>
<td>4.97 (2)</td>
<td>8.2 (5)</td>
<td>—</td>
</tr>
<tr>
<td>Milo</td>
<td>2.70 (4)</td>
<td>4.1 (5)</td>
<td>3.5 (2)</td>
</tr>
<tr>
<td>Oats</td>
<td>2.02 (4)</td>
<td>1.3 (4)</td>
<td>2.5 (2)</td>
</tr>
<tr>
<td>Peanut meal</td>
<td>—</td>
<td>—</td>
<td>4.5 (1)</td>
</tr>
<tr>
<td>Rapeseed</td>
<td>—</td>
<td>12.8 (1)</td>
<td>—</td>
</tr>
<tr>
<td>Sesame meal</td>
<td>12.3 (1)</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Soybean meal</td>
<td>3.63 (3)</td>
<td>7.6 (5)</td>
<td>6.7 (17)</td>
</tr>
<tr>
<td>Sunflower meal</td>
<td>—</td>
<td>—</td>
<td>13.8 (1)</td>
</tr>
<tr>
<td>Wheat</td>
<td>—</td>
<td>—</td>
<td>4.1 (1)</td>
</tr>
<tr>
<td>Wheat bran</td>
<td>5.98 (3)</td>
<td>10.9 (4)</td>
<td>16.2 (1)</td>
</tr>
<tr>
<td>Wheat middlings</td>
<td>4.92 (4)</td>
<td>11.4 (4)</td>
<td>12.5 (1)</td>
</tr>
<tr>
<td>Dried Brewers yeast</td>
<td>—</td>
<td>29.9 (7)</td>
<td>—</td>
</tr>
</tbody>
</table>

*Number in parenthesis refers to number of samples analysed.

Table 2 presents data on vitamin B6 content of some common feedstuffs used in poultry ration formulation. The three different reports are based on data obtained by microbiological assay. Friessecke and Kirchgessner (1961) used Neurospora sitophila as the assay organism.
Walter (1965) on the other hand used both *Saccharomyces carlsbergensis* and *Kloeckera brevis* as assay organisms. Scheiner and De Ritter (1968) chose *Saccharomyces carlsbergensis* since they felt that the method employing this organism was the most reliable according to both a careful review of the literature and their previous experience. The data shows considerable variation in values reported for the same ingredient. These variations are not only due to differences in assay methods and techniques, but can also be actual differences among samples due to factors such as climate and geographical location for plant products, and nutrition and environment for animal products. If we briefly survey these feedstuffs listed in Table 2, we find that in general the oil seed meals and cereal by-products are fairly rich sources of the vitamin, while the grains and the animal protein supplements are fair sources. This is bearing in mind that there are few exceptions to this general classification.

The above information on the variation in vitamin B₆ content of feedstuffs along with the various factors affecting the requirement for this vitamin may explain in part the differences in response obtained by various workers from supplementing practical poultry diets with pyridoxine HCl. It should be noted here that several reports published during the past decade showed favourable responses from supplementation of practical poultry diets with pyridoxine HCl. Friesecke and Kirchgessner (1963) reported that the vitamin B₆ contents of formula feeds for growing chicks and breeder hens in Germany do not meet the optimum requirements for this vitamin. Maier and Kirchgessner (1967a) also recommended supplementation with B₆ for certain combinations of feedstuffs because they observed that out of 50 broiler feeds assayed for vitamin B₆, several of these did not meet the requirement. Based on B₆ content of feedstuffs, Scheiner and De Ritter (1968) suggested the addition of vitamin B₆ to certain critical feeds particularly breeder diets. Jones *et al.* (1970) noted a significant response in rate of growth of turkey poults from the addition of 5 mg of pyridoxine per kg of practical type diet.

**Summary**

The importance of vitamin B₆ in the nutrition of poultry has been known ever since the discovery of the vitamin. Besides the well known functions of this vitamin in amino acid metabolism, several studies have been conducted on the role of this vitamin in amino acid metabolism, several studies have been conducted on the role of this vitamin in erythropoiesis and lipid metabolism in birds. The activities of several aminotransferases have been shown to be depressed in vitamin B₆ deficiency in poultry. Some workers have suggested the use of the levels of these enzymes in various tissues as possible indicators of vitamin B₆ nutriture in birds.

Several symptoms of vitamin B₆ deficiency have been described in the literature. Those shown to occur in chicks consist of slow growth, depressed appetite, poor feed utilization, convulsions, decreased clotting time, hyperprothrombinemia, decreased hemoglobin and packed cell volume, increased incidence of gizzard erosion, pendulous crop, and perosis. In the laying chicken, deficiency resulted in anorexia, rapid drop in body weight, hyperexcitability alternating with droopiness, and decreased egg production and hatchability. Deficiency symptoms in turkey poults were charac-
terized by loss of appetite, apathy, hyperexcitability when disturbed, and convulsions. In the turkey hen, the deficiency affected the appetite, decreased egg production and hatchability, and caused anaemia and dermatitis of legs and mouth. Severe acute deficiency in young ducklings was characterized by growth failure and severe anaemia. Chronic vitamin B6 deficiency in older ducklings caused lack of growth, paralysis, convulsions, severe microcytic anaemia and poor feather development.

The vitamin B6 requirements of chickens, turkeys and ducks have been summarized. These requirements have been shown to be affected by the dietary protein level, the quality of the protein used particularly the level of methionine, the level of choline in the diet, the presence of B6 antagonists in the feed, the use of drugs such as furazolidone and arsanilic acid, and the genetic make up of the bird.

The vitamin B6 content of feedstuffs have been shown to vary from one laboratory to another, and this variation has been shown to be not only due to differences in assay methods and techniques but also actual differences for the same ingredient. This along with the various factors that have been shown to affect vitamin B6 requirement, have prompted several authors to recommend supplementation of vitamin B6 to certain poultry feeds such as starter and breeder rations.

Resume

L’importance de la vitamine B6 dans la nutrition des volailles est connue depuis la découverte de cette vitamine. Outre des fonctions connues dans le métabolisme des acides aminés, plusieurs études ont été conduites sur son rôle dans l’érythropoïèse et le métabolisme des lipides des vaisseaux. On a montré que les activités des plusieurs aminotransférase étaient déprimées lors d’une déficience en vitamine B6 chez les volailles. Certains chercheurs ont suggéré l’utilisation du taux de ces enzymes dans divers tissus comme indicateur possible du niveau alimentaire de la vitamine B6 chez les oiseaux.

Plusieurs symptômes de déficience en vitamine B6 ont été décrits dans la littérature. Ceux observés chez le poulet sont une croissance lente, une baisse de l’appétit, un mauvais taux de conversion, des convulsions, une diminution du temps de coagulation, une hyperprothrombinémie, un abaissement du taux d’hémoglobine et du volume des cellules sanguines, une fréquence plus grande de l’érosion du gésier, un jabot pendant, et du perosis. Chez la poule pondeuse, une carence a pour effet l’anorexie, une chute rapide du poids corporel, des alternances d’hyperexcitabilité et de somnolence, une diminution de la ponte et du taux d’éclosion. Les symptômes de carence chez les jeunes din des sont caractérisés par une perte d’appétit, de l’apathie, une hyperexcitabilité en réponse à un dérangement, et des convulsions. Chez la din de adulte, la carence affecte l’appétit, abaisse la production d’œufs et l’éclosion, cause une anémie et une dermatite des pattes et de la bouche. Une carence aiguë chez les caneton s’est caractérisée par une faible croissance et une sévère anémie. Une déficience chronique de vitamine B6 chez des caneton plus âgés produit un retard de croissance, de la paralysie, des convulsions, une anémie microcytique sévère et un mauvais développement des plumes.

Les besoins en vitamine B6 des poulets, din dons et canards sont résumés. Il est montré que ces besoins sont affectés par le taux protéique de l’aliment, la qualité de ces protéines et en particulier le taux de méthionine, de choline, la présence d’antagonistes de la vitamine B6 dans le ration, l’emploi de médicaments comme la furazolidone et l’acide arsanilique, enfin la constitution génétique de l’oiseau.

Latenteur des aliments en vitamine B6 donne lieu à des estimations variables d’un laboratoire à l’autre, et il a été démontré que cette variation est due, non seulement aux différences de méthodes et de techniques d’analyse, mais aussi à des différences mé elles de teneur. Ceci, associé aux divers facteurs que l’on sait affecter le besoin en vitamine B6, a poussé plusieurs auteurs à recommander la supplémentation en cette vitamine de certains aliments pour volailles, comme les rations di démarrage ou pour reproducteurs.

Zusammenfassung

VITAMIN B6 IN DER GEFLÜGELERNÄHRUNG EINE ÜBERSICHT

Neben der seit langem bekannten Bedeutung des Vitamins B6 für den Aminosäure-Stoffwechsel beschäftigen sich zahlreiche Untersuchungen mit der Rolle dieses Vitamins für die Bildung von Erythrozyten und den Fettstoffwechsel

Die in der Literatur bekannten Symptome, mit denen ein Vitamin B6-Mangel bei Junghennen und Legehennen, bei wachsenden und ausgewachsenen Puten und bei Enten unterschiedlichen Alters in Erscheinung tritt, wurden eingehend beschrieben.

Der Vitamin B6-Bedarf von Hühnern, Puten und Enten wurde zusammenge stellt. Es wurde gezeigt, daß der jeweilige Bedarf abhängig ist vom Eiweißgehalt in der Ration, von der Qualität und besonders vom Gehalt an Methionin im Eiweiß, vom Choleingehalt der Ration, dem Anteil an Vitamin B6-Antagonisten im Futter, der Anwendung von Chemikalien wie Furazolidon und Arsenilsäure, sowie von der genetischen Veranlagung der Tiere.

Es hat sich gezeigt, daß die Untersuchung der Vitamin B6-Gehalte des Futters in verschiedenen Laboratorien zu unterschiedlichen Ergebnissen führen kann und daß diese Schwankungen nicht nur durch Unterschiede in der Untersuchungsmethode, sondern auch durch echte Unterschiede gleichartiger Futtermittelbestände bedingt sind. Diese Tatsache und die Erkenntnis, daß bestimmte Faktoren den Vitamin B6-Bedarf verändern können, hat zahlreiche Autoren veranlaßt, eine Supplimentierung bestimmter Geflügelfutter mit Vitamin B6 zu empfehlen.

References


