REFERENCES

Interrelationship of Dietary Silver with Copper in the Chick

R. P. Peterson and L. S. Jensen
Department of Animal Sciences, Washington State University, Pullman, WA 99163
(Received for publication August 19, 1974)

ABSTRACT
Adding 900 p.p.m. silver (as silver nitrate) to a practical diet for chicks significantly depressed growth, increased wet and dry heart weight to body weight ratios and markedly increased mortality during a four-week experimental period. Blood packed cell volume was not affected. Supplementing the diet containing silver with 50 p.p.m. copper prevented cardiac enlargement and mortality, but only partially corrected the growth depression. Glycogen content of the heart was not affected, but aortic elastin content was significantly reduced by silver and restored to normal by supplemental copper. Dietary silver significantly reduced the copper content of blood, spleen, brain, liver, but except for the brain, the level of copper in these tissues was restored to normal by dietary copper supplementation. No significant differences in copper content of kidney tissue were observed among the treatments. Copper content of the excreta was not significantly increased by adding dietary silver, but was greatly increased by adding 50 p.p.m. copper to the diet containing silver.

INTRODUCTION
In experiments conducted to determine the effect of adding silver acetate or nitrate to practical diets on the performance of turkey poults, a marked deficiency of copper was observed (Jensen et al., 1974). Silver (900 p.p.m.) depressed growth rate, reduced packed cell volume, slightly reduced hemoglobin level, and caused cardiac enlargement. Adding 50 p.p.m. copper to the diet completely reversed the effects of silver. Hill and Matrone (1964) showed that adding silver to a purified diet deficient in copper accentuated all of the symptoms of copper deficiency. In the presence of copper, silver had no effect. Using rat ceruloplasmin levels as a criterion, Whanger and Weswig (1970) concluded that of the known copper antagonists they tested, dietary silver was the strongest with dietary cadmium, molybdenum, zinc and sulfate following in descending order.

The purpose of the present investigations was to determine the effect of adding a high level of silver to a practical diet on the performance of chicks and on the distribution of copper in the tissues, and to determine if added copper would prevent the effects of silver.
EXPERIMENTAL PROCEDURE

Two experiments were conducted using day-old Hubbard broiler chicks. Birds were maintained in multi-tiered Jamesway battery brooders with feed and water supplied ad libitum. Composition of the practical basal diet used in both experiments is presented in Table 1. The first experiment was conducted to determine if copper deficiency signs would be observed in chicks when fed a practical diet supplemented with silver, as had been previously observed with turkey poults (Jensen et al., 1974). Three groups of 20 chicks each were fed the following four treatments for a 28-day period: basal diet; basal + 50 p.p.m. copper (CuSO₄ · 5H₂O); basal + 900 p.p.m. silver (AgNO₃); and basal + 50 p.p.m. copper and 900 p.p.m. silver. At the end of the experiment the surviving birds were killed by electric shock. Heart weight (both wet and dry); body weight ratios and packed cell volume data were obtained. Dry heart weights were obtained by lyophilizing the hearts for 48 hours.

A second experiment was conducted to obtain further information on a silver induced copper deficiency in chicks. Three groups of 10 chicks each were fed each of the following diets: basal diet; basal + 900 p.p.m. silver; basal + 900 p.p.m. silver and 50 p.p.m. copper; and basal + 900 p.p.m. silver and 0.36% lysine. At 28 days of age the birds were killed and hearts taken from the first three treatments for determination of glycogen content. The glycogen was determined by the method of Kemp and van Heijningen (1954). Aortic segments of elastin determination were taken from all four groups and the aortic elastin was determined by the method of Starcher et al. (1964). Tissue samples of blood, brain, liver, spleen and kidney, as well as an excreta sample from the first three treatments were analyzed for copper content by the method of Kline et al. (1971). The copper content of both excreta and livers was determined on a dry-weight basis and that of the liver on samples before and after extracting with diethyl ether for 24 hours.

RESULTS AND DISCUSSION

A summary of the heart weight:body weight ratios, packed cell volume and mortality data is presented in Table 2. Addition of silver to the basal diet significantly depressed growth rate, but growth was markedly improved by adding 50 p.p.m. copper in the presence of the silver. The added copper failed to improve growth to a level comparable with the basal diet. This failure may have resulted from the induction of other mineral deficiencies. It is possible that the silver was also inducing a mild selenium deficiency as selenium deficiency signs were observed in turkey poults fed high levels of silver (Jensen et al., 1974). Adding silver significantly increased the wet weight:body rate ratio in comparison with the ratios obtained with the basal diet. Adding 50 p.p.m. copper in...
the presence of the silver prevented the cardiac hypertrophy. The effect on the heart was not as severe as previously observed with turkey poults fed similar diets (Jensen et al., 1974). The fact that the dry heart weight:body weight ratio was significantly increased in the deficient chicks demonstrated that the heart enlargement was not due simply to an increase in moisture content of the tissue, but was due to an increase in tissue mass. Adding silver to the diet had no significant effect on packed cell volume, but caused 35% of the birds to die during the experiment. No mortality was observed when the diet containing silver also was supplemented with 50 p.p.m. copper. The mortality was probably due to the failure of normal aortic elastin formation in the copper deficient chicks, as Hill and Matrone (1961) showed that this function of copper was more critical for the survival of chicks than was normal blood formation. Necropsy examination of the birds which died during the course of the experiment consistently revealed blood in the abdominal cavity, mouth, trachea, and esophagus. Almost all birds found dead had large blood clots in the oral cavity. The breast muscles of the dead chicks also were pale and watery. Leg weakness immediately prior to death was a common observation.

The effect of dietary silver and copper on glycogen content of the heart and aortic elastin content is presented in Table 3. No significant effect of the dietary treatments on cardiac glycogen content was observed, indicating that the severe copper deficiency did not apparently alter the metabolism of this energy source in the heart tissue. Aortic elastin content was significantly reduced when silver was added to the basal diet, but was returned to normal by the supplementation of the diet containing silver with 50 p.p.m. copper. Adding lysine to the diet containing silver did not significantly alter the elastin content. Copper is necessary for amine-oxidase activity of the aorta and a deficiency of this enzyme results in less lysine being converted to desmosine, which is necessary for normal elastin formation (Hill et al., 1967).

Adding dietary silver to the basal diet significantly reduced the copper content of the blood, spleen, brain and liver (Table 4). Adding copper to the diet containing silver returned tissue copper levels to control values except for the brain tissue where the value

---

**Table 2.—Effects of dietary silver alone and with supplementary copper on chicks to 28 days of age**

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Body weight (gm.)</th>
<th>Wet heart/body (%)</th>
<th>Dry heart/body (%)</th>
<th>Packed cell volume (%)</th>
<th>Mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diet I (control)</td>
<td>612</td>
<td>.61b</td>
<td>.16c</td>
<td>33a</td>
<td>0</td>
</tr>
<tr>
<td>Diet I + 50 p.p.m. Cu</td>
<td>588b</td>
<td>.65b</td>
<td>.18bc</td>
<td>34a</td>
<td>0</td>
</tr>
<tr>
<td>Diet I + 900 p.p.m. Ag</td>
<td>286c</td>
<td>.87a</td>
<td>.22b</td>
<td>32a</td>
<td>35</td>
</tr>
<tr>
<td>Diet I + 56 p.p.m. Cu</td>
<td>473b</td>
<td>.63b</td>
<td>.18bc</td>
<td>33a</td>
<td>0</td>
</tr>
</tbody>
</table>

1 Means within each column not having the same letter are significantly different at the 5% probability level.

**Table 3.—Effect of dietary silver and copper on cardiac glycogen and aortic elastin content**

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Glycogen (%)</th>
<th>Aortic elastin (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diet I (control)</td>
<td>0.25a</td>
<td>12.3a</td>
</tr>
<tr>
<td>Diet I + 900 p.p.m. Ag</td>
<td>0.27a</td>
<td>8.2b</td>
</tr>
<tr>
<td>Diet I + 900 p.p.m. Ag</td>
<td>0.24a</td>
<td>12.0a</td>
</tr>
<tr>
<td>+ 50 p.p.m. Cu</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diet I + 900 p.p.m. Ag</td>
<td>0.36% lysine</td>
<td></td>
</tr>
<tr>
<td>+ 0.36% lysine</td>
<td></td>
<td>8.1b</td>
</tr>
</tbody>
</table>

1 Means within each column not having the same letter are significantly different at the 5% probability level.
TABLE 4.—Effects of dietary silver on the distribution of copper in the bodies of 28-day-old broiler chicks

<table>
<thead>
<tr>
<th>Treatment</th>
<th>P.P.M. Cu on a dry weight basis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Blood</td>
</tr>
<tr>
<td>Diet A (control)</td>
<td>3.1*</td>
</tr>
<tr>
<td>Diet A + 900 p.p.m. Ag</td>
<td>1.5b</td>
</tr>
<tr>
<td>Diet A + 900 p.p.m. Ag +</td>
<td>3.3a</td>
</tr>
</tbody>
</table>

*Means within each column not having the same letter are significantly different at the 5% probability level.

was significantly lower than the control, but significantly higher than the diet containing silver alone. There were no significant differences in the copper content of the kidney of the birds fed the three different diets. This suggests that the silver was causing a greater loss of copper through the kidneys. There was little difference in the amount of copper present in the excreta from the control birds and those fed silver, but there was significantly more copper in the excreta of birds fed the silver diet supplemented with copper, as would be expected since tissue levels of these birds were not elevated in copper content over the control birds. The failure to demonstrate a significant increase in copper excreted by birds fed silver alone is puzzling because these birds retained less copper in their tissues. The excreta sample used by analysis was collected for one day only near the end of the experiment. Perhaps a sample representing the total excreta voided during the experiment would have shown a significantly higher copper content than that in a similar sample from chicks fed no silver.

The results of these experiments do not determine whether the silver is interfering with copper absorption or with metabolism within the animal. Silver does result in less copper uptake by various tissues, however, and it is likely that the silver ion is competing with copper ion for copper binding sites in enzymes and other proteins in the tissues. Hill et al. (1964) suggested that the antagonism between silver and copper in the diet of the chicks can be explained on a basis of similar chemical parameters of the copper ion with the divalent silver ion (Ag⁺⁺). In an experiment to determine the effect of silver on the absorption and distribution of radioactive copper, Van Campen (1966) observed that silver had little effect on copper uptake, but a significantly greater proportion of the absorbed isotope was deposited in the liver and significantly less was retained by the blood of the silver treated rats. The experiment by Van Campen (1966), however, was of an acute nature and the tissues were sampled after only three hours from the time of dosage. The long-term effects of dietary silver on copper metabolism, therefore, could not be revealed by such an experiment.

REFERENCES

DIETARY Ag AND Cu INTERRELATIONSHIP

Importance of dietary copper in the formation of
Van Campen, D. R., 1966. Effects of zinc, cadmium,
silver and mercury on the absorption and distribution
Whanger, P. D., and P. H. Weswig, 1970. Effect of

Effects of Drinking Water Temperature on Broiler
Performance

G. C. Harris, Jr., G. S. Nelson, R. L. Seay and W. H. Dodgen
Departments of Animal Sciences and Agricultural Engineering, University of Arkansas, Fayetteville, Arkansas 72701
(Received for publication August 20, 1974)

ABSTRACT In the first experiment, higher body weight gain and feed consumption were attained with water at a temperature of 23.9° C. as compared with 35.0° C. No significant differences in feed efficiency due to water temperature were evident. Livability was significantly reduced during brooding for the birds given warm water (35.0° C). A significant interaction for body weight gain was observed between the initial ambient air brooding temperatures and water temperatures during the growing period.

In the second experiment six water temperatures during brooding to three weeks of age were studied. Body weight gain and feed consumption were significantly depressed at a drinking water temperature of 40.6° C. as compared with drinking water temperatures between 17.8° C. and 35.0° C. No differences in livability were noted which was in contrast to the results of the first experiment.

A drinking water temperature below ambient air temperature is apparently beneficial to the growth of the broiler chick. The placement of the waterers in relation to the brooder stoves could influence water temperature which would affect the growth of broilers.

INTRODUCTION

WATER is the nutrient required in largest quantity and should be considered the most important essential nutrient. Adolph (1933) in a review of water metabolism pointed out that water ranks above every other substance in the body in regard to rate of turnover. However, very few studies have been conducted concerning the temperature of water and its effects on the growing chick.

The chicken is acutely sensitive to the temperature of water when it is adjusted above or below ambient air temperature (Gates and Kare, 1961; Prince and Kare, 1962). When given a choice the chicken rejected water barely warm to humans but in a no-choice situation it would drink the water to a limited degree (Gates and Kare, 1961). The water consumption of broilers increases with elevated ambient air temperatures (Barrott and Pringle, 1947, 1949, 1950; Joiner and Hustin, 1957), even though the water temperature increases in direct relationship to room temperature. Milligan et al. (1957) studied the effects of three water temperatures (10°, 21.1° and 32.3° C.) during the daytime in hot weather on the growth of the chick to 6 weeks of age. Warm water (32.3° C.) depressed both growth and feed efficiency but the results were nearly the same for the chicks given 10° C. water as those receiving 21.1° C. water.

The present study was conducted to determine the influence of water temperature as