COBALT DEFICIENCY IN NEW YORK STATE

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FROM time to time during the past several years, reports have filtered into the College of an unhealthy condition of cattle in the northern counties of New York State which clearly could not be diagnosed as deficiencies of phosphorus, calcium or a lack of total feed. As information concerning cobalt deficiency became more abundant, many points of similarity were recognized between the symptoms of these New York cattle and the described symptoms of cobalt deficiency. Chemical analysis of forage samples from so-called “trouble farms” by one of us (KCB) gave support to the cobalt deficiency hypothesis in that many forage samples ran critically low in this element. Several veterinarians in the northern areas, particularly Dr. Grace of Potsdam, cited remarkable responses in some unhealthy cattle when cobalt salts were administered as a drench.

In 1947 a study of cobalt metabolism in sheep was initiated having as its purpose the biological testing of forage crops from selected areas of the State for cobalt adequacy and furthermore to determine the metabolic role of cobalt in the animal body, particularly the ruminant. The present paper is a report from this study.

No attempt will be made here to review the literature extensively since several excellent reviews of cobalt nutrition are available. Among the more recent are those by Russell (1944) and Maynard and Smith (1947).

Methods and Materials

Lambs were chosen as the test animal in this study since the literature indicates that they are more sensitive to a lack of cobalt than cattle and because of their smaller size, larger numbers can be handled within a given budget. Western feeder lambs purchased from the stockyards were freed of parasites by dipping and drenching and carefully inspected for signs of other disease. They were then placed in pens for individual feeding of a cobalt-low ration.

Two experiments are here reported extending over a period of about 2 years. The general procedure in both of these studies was to feed all lambs within an experiment a low-cobalt ration until the majority gave evidence of cobalt deficiency. At this time the lambs were randomized into treatment groups and the study continued. The first experiment involved 16 lambs which were randomized into two groups—(A) continued on a cobalt-low
ration and (B) the same ration supplemented with orally administered cobalt salts. The second experiment involved 21 lambs randomized into three treatment groups: (1) cobalt-low ration; (2) cobalt supplemented orally and (3) cobalt supplemented by intramuscular injections.

The hay fed in both experiments was secured from New York farms that had past reports of trouble in cattle. The first experiment utilized hay from a farm in St. Lawrence County. It unofficially graded as a No. 3 mixed grass hay and averaged 0.04 ppm of cobalt. In the second experiment hay, unofficial grade No. 3 mixed grasses, was secured from a farm in Madison County. Cobalt analyses made at intervals during the trial ranged from 0.02 to 0.05 ppm. In both experiments the hay was supplemented with locally purchased shelled corn, which ranged in cobalt concentration from 0.01 ppm to 0.03 ppm and milk powder (Borden’s Klim) which by analysis contained 0.01 ppm of cobalt or less. Lambs which would take a full ration were fed at the rate of about 1 pound of hay, 1.25 pounds of shelled corn and 0.25 pound of milk powder per day. Half of the ration was fed in the morning and half in the afternoon. As appetites failed due to a deficiency of cobalt, much lower quantities of these feeds were consumed as is indicated later. Plain block salt and tap water were generally available to all lambs.

As appetites failed, more feed was presented to the lambs than they would clean up in 2-3 hours. This was purposely done to be certain that lambs were being fed all that they would take since feed intake is one of the important criteria of cobalt deficiency. Accurate records of the daily feed intake were kept for each lamb. Cobalt supplements were administered in the first experiment as the sulfate salt and in the second experiment as the chloride salt. In all cases, unless otherwise stated, cobalt was administered twice per week at the rate of 1 mg. of cobalt per lamb per day. Cobalt contamination was rigorously guarded against since lambs will respond to very minute amounts of this element.

The feed samples were analyzed for cobalt by the method of Ellis and Thompson (1945). Hemoglobin was determined on jugular blood samples by the oxyhemoglobin method (Sheard and Sanford, 1929). Blood serum calcium was determined by the Clark-Collip method and inorganic phosphorus by the Fiske-Subbarow technic. Blood serum proteins were determined with the gravity-gradient column (Lowry and Hunter, 1945) after the method had been satisfactorily checked with Kjeldahl-determined proteins (Chibnall et al., 1943) on large samples of sheep serum.

All lambs that died or were sacrificed were carefully autopsied and tissues taken for microscopic study. These tissues consisted of the liver, spleen, kidney, pituitary gland, thyroid gland and sections at various levels.  

1 Courtesy of Dr. P. Olafson of the New York State Veterinary College.
of the intestinal tract. The tissues were fixed in Susa's fixitive and stained with hematoxylin and eosin. In a few cases some tissues were also stained to demonstrate hemosiderin by the Prussian blue reaction.

Red blood cells were counted in the conventional way using a 1:400 dilution blood pipette. Centrifuge hematocrits were determined using the modified centrifuge tube previously described (Smith 1944).

Results

Experimental

The first gross symptom noted in the lambs as they became cobalt deficient was a waning appetite. This was followed by reduced rates of gain and then

![Graph showing daily consumption of concentrates](image)

**Figure 1.** The daily consumption of concentrates of cobalt-deficient and cobalt-fed lambs. The arrow indicates the start of cobalt supplementation.

weight losses. A progressive anemia occurred some time later. These symptoms have been described by others (Russell 1944).

In our studies a cobalt deficiency did not develop until after some months on the low-cobalt ration. In the first experiment cobalt deficiency was definitely recognized at about 5 months and in the second experiment at about 7 months and at these times treatments were begun.

The inappetence in cobalt deficiency and the rapid response of appetite following oral administration of cobalt salts is well illustrated in figure 1. The intake of concentrates (corn plus milk powder) only is shown in this figure. A similar response occurred in the intake of hay but to a lesser degree. It is noted that feed intake showed an increase over basal conditions at about
the sixth day after cobalt feeding. In the second experiment appetite responded definitely at about the eighth day after cobalt feeding. To all outward appearances a failing appetite is a dominant symptom of cobalt deficiency. Unfortunately this symptom is not specific for cobalt inadequacy. All other gross symptoms could well be secondary to the inappetence. There have been some reports of a depraved appetite in cobalt deficiency. This symptom has not been marked in our lambs. In the two experiments, only 2 lambs gave evidence of this by chewing the wool off of other lambs. While there was ample opportunity for the lambs to chew wood from the pens or to eat the sawdust bedding, no real evidence of this was observed.

Growth curves of the lambs in the first experiment are presented in figure 2. The progressive decline in body weight of those fed the cobalt-low ration only and the normal rates of growth for those lambs fed cobalt salts in addition is evident. The rate of gain for those fed cobalt supplements was 0.32 pound per lamb per day for the 13-week period. This rate of gain is considered good for commercially fattened lambs. It is noted that weight gains were evident at one week following cobalt therapy. Growth of similarly treated lambs in the second experiment followed a similar pattern.

Although an anemia has not consistently been reported for cobalt de-
Cobalt Deficiency in New York State

TABLE 1. TYPE OF ANEMIA IN COBALT-DEFICIENT LAMBS

<table>
<thead>
<tr>
<th>Treatment lambs</th>
<th>Weeks</th>
<th>Red cell count</th>
<th>Hemoglobin concentration</th>
<th>Mean cell volume</th>
<th>Mean cell hemoglobin</th>
<th>Mean cell hemoglobin concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td>First Experiment</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>+Co 8</td>
<td>13</td>
<td>9.87±0.234</td>
<td>11.5±0.31</td>
<td>37.9±1.28</td>
<td>38.5±1.09</td>
<td>30.4±0.61</td>
</tr>
<tr>
<td>-Co 5</td>
<td>13</td>
<td>6.60±2.888*</td>
<td>7.5±0.75**</td>
<td>37.9±1.99</td>
<td>11.5±0.45</td>
<td>30.4±1.20</td>
</tr>
<tr>
<td>Second Experiment</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>+Co 6</td>
<td>12</td>
<td>11.23±0.491</td>
<td>12.0±0.68</td>
<td>37.9±1.00</td>
<td>10.8±0.26</td>
<td>38.4±0.61</td>
</tr>
<tr>
<td>-Co 5</td>
<td>12</td>
<td>5.43±0.666**</td>
<td>5.1±0.79**</td>
<td>36.4±1.35</td>
<td>9.3±0.74</td>
<td>36.0±2.02</td>
</tr>
</tbody>
</table>

* Mean difference significant P = 0.05.
** Mean difference significant P = 0.01.

In our studies, it has been a constant observation that cobalt deficiency in lambs is microcytic and hypochromic in type. In both of the studies here reported, the anemia has been simple, that is, normocytic and normochromic as the values in Table 1 clearly show.

A general summary of observations on the lambs in the first experiment is given in Table 2. Those lambs fed cobalt salts had normal hemoglobin levels at the end of the 13-week period and were sold as fat yearlings. Those lambs maintained on the low-cobalt ration were anemic and lost considerable weight.

Experiment 2 had as part of its design to test the effectiveness of cobalt salts variously administered, that is, feeding versus intramuscular injections. The amount of cobalt solution given at each injection was divided into three portions and injected into different sites. This was done to increase the amount of cobalt salts absorbed since some local irritation occurs at the site of injection. The effects of the various treatments—no cobalt, cobalt-fed
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TABLE 3. EFFECT OF COBALT SALTS WHEN FED OR INJECTED ON WEIGHT AND HEMOGLOBIN LEVELS OF COBALT-DEFICIENT LAMBS

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Number of lambs</th>
<th>Period of treatment</th>
<th>Initial weight</th>
<th>Final weight</th>
<th>Final hemoglobin level</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(wks.)</td>
<td>(lbs.)</td>
<td>(lbs.)</td>
<td>(lbs.)</td>
<td>(gms./100 ml.)</td>
</tr>
<tr>
<td>Co fed</td>
<td>7</td>
<td>7</td>
<td>60±3.7</td>
<td>71±2.6</td>
<td>11.6±0.18</td>
</tr>
<tr>
<td>Co injected</td>
<td>7</td>
<td>7</td>
<td>57±3.1</td>
<td>49±2.8**</td>
<td>7.1±0.64**</td>
</tr>
<tr>
<td>-Co</td>
<td>7</td>
<td>7</td>
<td>61±3.2</td>
<td>51±2.8**</td>
<td>7.1±0.82**</td>
</tr>
</tbody>
</table>

** Mean difference as compared to Co fed lambs significant P = 0.01.

and cobalt-injected are summarized in table 3. It is obvious from these data that cobalt salts injected had no effect on the course of cobalt deficiency as judged by growth and hemoglobin levels. In a previous paper (Gall, Smith, Becker, Stark and Loosli, 1949) it was shown that the rumen microflora in the cobalt-injected lambs was similar to the cobalt-deficient lambs and both unlike the cobalt-fed lambs.

In this study a preliminary attempt was made to detect a biochemical upset in cobalt deficiency. To this end blood plasma proteins, calcium, phosphorus and alkaline phosphatase activity were determined. The results of these observations are summarized in table 4.

It is noted that the cobalt-deficient lambs had slightly lower blood plasma protein levels as compared to the cobalt-fed lambs. This difference was significant (P = 0.01) only in the second experiment. The blood plasma calcium was slightly but significantly (P = 0.05) lower in the cobalt-deficient lambs. There was no significant difference between treatments so far as

TABLE 4. BLOOD PLASMA PROTEINS, CALCIUM, INORGANIC PHOSPHORUS CONCENTRATIONS AND ALKALINE PHOSPHATASE ACTIVITY OF COBALT-TREATED LAMBS

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Plasma proteins %</th>
<th>Calcium mg./100 ml.</th>
<th>Phosphorus mg./100 ml.</th>
<th>Alkaline phosphatase activity mg.P/100 ml.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>First Experiment</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cobalt fed</td>
<td>7.10±0.278</td>
<td></td>
<td></td>
<td>12.8±1.61</td>
</tr>
<tr>
<td>-cobalt</td>
<td>6.95±0.159</td>
<td></td>
<td></td>
<td>5.5±1.33**</td>
</tr>
<tr>
<td>Second Experiment</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cobalt fed</td>
<td>6.33±0.040</td>
<td>12.4±0.37</td>
<td>8.1±0.32</td>
<td>14.1±1.87</td>
</tr>
<tr>
<td>-cobalt</td>
<td>6.03±0.081**</td>
<td>11.0±0.38*</td>
<td>7.1±0.45</td>
<td>6.5±1.35**</td>
</tr>
</tbody>
</table>

* Mean difference significant P = 0.05.
** Mean difference significant P = 0.01.
blood plasma phosphorus was concerned but the plasma alkaline phosphatase activity was appreciably less ($P = 0.01$) in the cobalt-deficient as compared to the cobalt-fed lambs in the first and second experiments. Bowstead et al. (1942) previously reported a slight reduction in blood proteins in cobalt deficiency.

The autopsy observations were generally negative. The only observation that showed some consistency was a fatty, degenerated liver that occurred in about 57 percent of the cobalt-deficient lambs. In two cases this was accompanied by petechial hemorrhages under the liver capsule. The microscopic study of several tissues likewise was largely negative except to confirm the occurrence of fatty livers and to observe a marked hemosiderosis of the spleens. These observations have previously been recorded by Filmer (1933) and Bowstead et al. (1942).

Careful observations were made of the wool of all lambs. No abnormality that might be considered specific for cobalt deficiency was noted. The wool of cobalt-deficient lambs was retarded in growth and very weak fibered—a result that is very likely secondary to the general inanition.

**Chemical Survey of Forages in New York**

The cobalt analyses here reported are observations made on samples collected at random initially, usually from farms where troubles of unknown etiology were reported in cattle. Later a more systematic study was made in St. Lawrence Country with the hope of establishing some correlation between the cobalt level of forages and soil types.

Each figure in table 5 is an average of from 1 to 3 samples taken from each field. The number of samples taken depended somewhat on the size of the field. Each sample was made up of about 50 small subsamples, thus it is felt that a good representative sample of each field was obtained. In interpreting these analyses, the reports of McNaught (1938) and Underwood and

<table>
<thead>
<tr>
<th>Kind of hay</th>
<th>No. of fields</th>
<th>No. of soil types</th>
<th>Total no. of samples</th>
<th>Distribution of fields according to the cobalt content of the forages</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.01-.03</td>
</tr>
<tr>
<td>Timothy</td>
<td>67</td>
<td>12</td>
<td>149</td>
<td>20</td>
</tr>
<tr>
<td>Legume</td>
<td>10</td>
<td>3</td>
<td>15</td>
<td>1</td>
</tr>
<tr>
<td>Mixed*</td>
<td>31</td>
<td>—</td>
<td>51</td>
<td>10</td>
</tr>
</tbody>
</table>

1 Hay with 10% or more of legumes.
Harvey (1938) were used. These workers agree that pasture forages containing less than 0.04 ppm of cobalt are potential deficiency areas. Cobalt values of 0.04-0.07 ppm are on the borderline of requirements. More recently Stewart et al. (1946) have reported data indicating that cobalt deficiency in lambs may develop on rations containing as much as 0.09 ppm cobalt. On the basis of such reports, nearly one-third of the timothy and the mixed-legume fields sampled were classified as cobalt deficient (0.01-0.03 ppm) and an additional 50 percent classified as borderline (0.04-0.07 ppm.). There is an indication from these data that legumes tend to run higher in cobalt than the grasses grown on the same fields. There was no consistent relationship of cobalt content of forages to soil type, partly because of the small number of samples collected from any one soil type. However, in St. Lawrence County where a more systematic study has been made, no fields on the Dunkirk, Vergennes and Parishville soils could be classified as deficient. On the other hand, in no case could all the fields on any one soil type be classified as deficient.

In table 6 there is presented the distribution of fields within each county as to the cobalt content of forage samples of timothy and mixed hays. Heavy legume mixtures were not included in this classification. The forage samples collected in St. Lawrence County are random samples whereas in the other counties the samples were selected in that they came from farms where troubles were reported in cattle. These troubles were not necessarily due to cobalt deficiency. Of the areas so far studied only eastern Essex County was found free of decidedly low-cobalt forages. It is of interest to note that the soils of this part of Essex County were developed from a very different material than were those of St. Lawrence and Franklin Counties.

Caution should be used in interpreting the cobalt analyses of forages for as McNaught (1938) and Underwood and Harvey (1938) have pointed out the correlation between cobalt content of forages and cobalt deficiency was
not perfect. Also the observation of low-cobalt forage in an area does not necessarily indicate that cattle and sheep in that area are lacking adequate cobalt for appreciable amounts of cobalt may be given in salt mixtures or in the concentrates fed. The important thing from the standpoint of an animal is the total intake and not the intake from any one feed. Obviously unsupplemented cattle and sheep on pasture in low-cobalt areas are most likely to be deficient in this element.

Discussion

Studies here reported as well as those reported by others have not as yet established symptoms which may be regarded as specific for cobalt deficiency. Such gross symptoms as inappetence, failure of growth and anemia are too general and may easily be confused with other nutritional diseases. Also, no biochemical or microscopic lesion has yet been detected which is specifically pathognomonic. This makes field diagnosis of the deficiency very difficult. In fact, the only method to date of diagnosing cobalt deficiency with some certainty is to feed cobalt salts under controlled conditions and to note a favorable response.

The complete failure of our lambs to respond to the injection of cobalt salts is in disagreement with the results of Ray et al. (1948) where a partial response was reported. A reconciliation of these different observations is not at the moment evident.

Since McCance and Widdowson (1944) first mentioned unpublished results showing that injected cobalt salts were ineffective, it has generally been theorized that this was evidence for a direct need by the ruminal bacteria for cobalt which was thus required by ruminants only indirectly. More direct evidence that cobalt affects the ruminal bacteria was given by Gall, Smith, Becker, Stark and Loosli (1949).

Studies by Becker et al. (1949) showed that cobalt-deficient lambs did not respond to the feeding or injection of vitamin B\textsubscript{12}. Apparently, then, cobalt in lambs has some major function(s) other than the formation of B\textsubscript{12}.

These studies showing that hay from two areas in New York State is deficient in cobalt for lambs combined with the studies of the cobalt content of New York forages leave little doubt that this state contains many cobalt-deficient areas. States which thus far have been demonstrated to have such deficiency areas are New Hampshire, Wisconsin, Michigan, Florida, North Carolina and New York. Undoubtedly as studies are extended, other states will be added to this list.

Summary and Conclusions

Feeding trials with lambs have clearly shown that New York State contains areas that are deficient in cobalt.
Lambs fed a low-cobalt ration developed a deficiency of this element in 4-7 months characterized by loss of appetite, loss of weight, a simple anemia and death. Cobalt salts given to cobalt-deficient lambs by mouth resulted in a return of appetite within about a week. This was quickly followed by increased weight gains. Hemoglobin levels started to rise sometime later, about six weeks.

Cobalt salts injected into cobalt-deficient lambs were completely ineffective in altering the course of the deficiency.

As compared to cobalt-fed lambs, cobalt-deficient lambs showed a slightly lower concentration of blood plasma proteins; a slightly lower blood plasma calcium concentration; a lower blood plasma alkaline phosphatase activity and about the same blood plasma inorganic phosphorus concentration.

Autopsy and microscopic tissue studies showed that cobalt-deficient lambs had fatty degeneration of the livers and a marked hemosiderosis of the spleens.

A chemical survey of forage plants in the State has given some estimate of the extent of cobalt-deficient areas.

**Literature Cited**


