Wheat Pasture Poisoning

in cooperation with the
TEXAS TECHNOLOGICAL COLLEGE
and the
U. S. DEPARTMENT OF AGRICULTURE

TEXAS AGRICULTURAL EXPERIMENT STATION
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SUMMARY

Experiments have been conducted since 1949 at PanTech Field Laboratory near Panhandle by personnel of the Texas Agricultural Experiment Station, Texas Technological College and the U. S. Department of Agriculture on wheat pasture poisoning.

The condition known as wheat pasture poisoning occurs primarily in sexually mature cows which are in the late stages of pregnancy or with a calf at side. Most cases developed sometime between 60 and 150 days on wheat, and in cows which had calves under 60 days of age.

When the level of several components of the blood serum of normal cows was compared with the serum of cows affected with wheat pasture poisoning, a decrease in inorganic phosphate, total and diffusible calcium, magnesium and the albumin-globulin ratio was found in the cases. The total serum protein, globulin and possibly the potassium levels were increased. The wide range of values observed in the cases suggests that the blood serum findings could be the result of the effect of wheat pasture poisoning rather than the cause.

Salt, cottonseed meal, mineral mixtures, silage and various dry feeds, either alone or in combination, gave little or no protection against wheat pasture poisoning. Presumably, acting as a diluent, they may lengthen the time necessary for the attack to occur. They do not, however, act as a preventive. Treating the wheat land with nitrogen or potassium fertilizers, or both, did not appreciably alter the composition of the wheat plant or the blood of cows and ewes grazing the fertilized wheat pastures.

The best treatment appears to be the injection of a calcium gluconate solution with or without fortification with magnesium and phosphorus. Recovery seems to be speeded by removing the cow from wheat pasture for a short time. No recurrence was observed in any animal which had recovered from the initial attack.
In the Winter Wheat Grazing Area, more particularly in the Texas and Oklahoma Panhandles, a condition develops which is known locally as wheat pasture poisoning when cattle are allowed to graze on the growing wheat plant. This condition is most pronounced during periods of lush growth with plentiful moisture.

**SYMPTOMS AND OCCURRENCE**

Wheat pasture poisoning symptoms begin with undue excitement, incoordination and loss of appetite. As the condition progresses, viciousness, staggering and falling develop. Nervousness becomes more apparent with muscular twitching, particularly of the extremities. The animal has an anxious expression and may grind its teeth and salivate profusely. The third eyelid protrudes or flickers as is seen in tetanus. General tetanic contractions of the muscles follow until the animal reaches a state of prostration; however, a sudden noise or merely touching the animal will cause a reflex response. Next to develop are labored breathing and a pounding heart followed by a comatose condition. If left untreated, convulsions with periods of relaxation will be seen which terminate in death. Six to 10 hours usually are required from the time the first symptoms develop until the animal passes into the comatose condition. If treatment is not begun before coma, there is little chance of recovery.

While wheat pasture poisoning has been reported in dry cows, heifers and sheep, we have observed this condition only in sexually mature cows which were pregnant or with calf at side, or both. The symptoms were observed in beef, dairy and cross-bred cows.

Case histories indicated that 80 percent occurred between 60 days and 150 days on wheat pasture. Thirty-three percent of the cases occurred after 60 to 90 days on wheat, 21 percent during the 90 to 120-day period and 26 percent during the 120 to 150-day period. The length of time on wheat pasture before the illness occurred varied from 8 days to 6 months.

The time the cow has the attack varied from the fifth month of pregnancy to 6 to 7 months post partum. Seventy-nine percent of the cases occurred in cows which had calves under 60 days of age. Seventeen percent of the calves were under 1 week, 24 percent were 1 to 2 weeks, 11 percent were 15 to 29 days and 27 percent were 30 to 60 days of age. Seven percent of the cases occurred in the last 3 months of pregnancy. The remaining 14 percent had calves over 60 days of age.

**POSSIBLE CAUSES**

Many theories have been advanced for the cause of wheat pasture poisoning, but the exact cause or the prevention is not known. Most of the early experiments were based on the theory of a disturbance in the acid-base ratio of the blood with particular reference to potassium. Among the possibilities suggested for this disturbance in acid-base ratio were:

1. The high potassium content of the wheat plant at the time of its rank growth.
2. Since the water in the Texas Panhandle is alkaline, it could augment the effect of the high potassium content of the wheat plant.
3. The wheat plant contains some substance or combination of substances which tend to make the potassium more readily available.
4. Interference with calcium absorption in the digestive tract because of the high potassium content of the wheat plant.

5. Malfunction of the parathyroid gland or hormone, or both, which would upset calcium metabolism.

Caldwell and Hughes (1) suggested the theory of the malfunctioning parathyroid gland. Previously, McMullen and Langham (2) had suggested wheat pasture poisoning to be a complex nervous and glandular disturbance rather than a simple mineral deficiency. The possibility of the high protein content preventing calcium absorption and the method of grazing also were considered.

**GRAZING STUDIES**

From 1949 through the spring of 1953, emphasis in the grazing studies with both cows and ewes was on the addition of mineral supplements correlated with changes in the composition of the blood. Objectives of this type of study were: 1. It was highly desirable to be able to produce or to increase the production of wheat pasture poisoning cases for laboratory study, and this was one possible procedure; 2. The possibility of the control of wheat pasture poisoning by mineral supplements presented a highly practical means of control which would be available to the ranchman and wheat farmer. Blood changes would serve as the diagnostic tool to determine when cases might occur.

The various supplements used were calcium in the form of calcium carbonate, calcium plus a urine acidifier-ammonium chloride, ammonium chloride alone, silage and calcium carbonate, silage and ammonium chloride, silage alone, calcium carbonate and magnesium oxide, and potassium bicarbonate. Drinking water was acidified with hydrochloric acid. Wheat was fertilized with nitrogen at 50 to 200 pounds per acre, with potash and with combinations of potash and nitrogen. In addition, one group of sheep received injections of the parathyroid hormone, parathormone. One study with ewes was made using a basic ration high in protein and low in minerals to which were added the following supplements: low potassium-high magnesium, high potassium-high magnesium and high potassium and low magnesium. Blood samples were taken every 28 days while the animals were on experiment.

During these experiments, none of the test or control animals developed wheat pasture poisoning, and no significant changes were observed in the composition of the blood from the control and test animals. These supplements had no effect in preventing or causing wheat pasture poisoning.

Barrentine (3) at Mississippi State College has been able to produce symptoms in lactating ewes similar to those of wheat pasture poisoning by controlling the method of grazing winter oats. The pasture was grazed or cut short, then a new group of ewes was placed on the pasture as the oats started a rapid growth. In this manner, he was able to produce symptoms in a very few days. When the Barrentine procedure was repeated at PanTech Field Laboratory with both wheat and oats, we were unable to obtain any symptoms or blood changes.

The effect of available stubble was studied during the 1953-54 grazing season. Two wheat pastures were used. Both had the same amount of wheat pasture available and one included a grain sorghum stubble field. Forty cows were placed in each field. Four cases of wheat pasture poisoning occurred in each wheat field. Three cases occurred in the pasture which had only wheat available before any developed in the combination pasture. Evidently the stubble field was effective only in increasing the time required for the onset, but was ineffective as a preventative agent.

Since salt, mineral mixes, cottonseed meal, dry feeds, silage or various combinations of these supplements had been suggested as a preventative measure, the histories of 41 cases of wheat pasture poisoning occurring during the 1953-54 winter wheat grazing season were analyzed for supplements (4). Table 1 gives the supplements fed during the 1953-54 winter wheat grazing season. Two wheat pastures were used. Both had the same amount of wheat pasture available and one included a grain sorghum stubble field. Forty cows were placed in each field. Four cases of wheat pasture poisoning occurred in each wheat field. Three cases occurred in the pasture which had only wheat available before any developed in the combination pasture. Evidently the stubble field was effective only in increasing the time required for the attack to occur, but they do not prevent it.

Because of the almost complete failure of winter wheat in this area, no grazing studies were conducted during the 1954-55 grazing season. A group of ewes were started in May 1955 on a small patch of irrigated volunteer wheat, then were placed on a simulated wheat ration until October 1955 when some wheat pasture became available. In December 1955, they were placed on dehydrated wheat pellets and finally back on green wheat from March until May 1956, at which time they had completed 1 year on green wheat or a simulated wheat ration. No symptoms were observed nor were there any significant blood changes.

### Table 1. Supplements Fed During Incidence of Wheat Poisoning

<table>
<thead>
<tr>
<th>None</th>
<th>Salt</th>
<th>Mineral mixtures</th>
<th>Cottonseed meal</th>
<th>Dry feed</th>
<th>Silage</th>
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<td>4</td>
<td>32</td>
<td>3</td>
<td>20</td>
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</tr>
</tbody>
</table>

1Salt alone. If salt is a part of a mineral mix, it is not included in this category, but under mineral mixture.
2Includes bundles, grain, hay or stalk field.
BLOOD STUDIES

Blood serum samples were obtained from September 1949 through May 1954, from 60 untreated cases of wheat pasture poisoning which were not complicated by other conditions. Forty-three of this number were seen during the 1953-54 winter wheat grazing season. For comparative purposes, 185 serum samples were obtained from Hereford cows grazing winter wheat at PanTech Farms. The cows were comparable in age, condition and length of time on wheat with those which had attacks of wheat pasture poisoning.

The following determinations were made on the serum: alkaline phosphatase, inorganic phosphate, total calcium, diffusible calcium, magnesium, total proteins, albumin, globulin and the albumin-globulin ratio. Analytical results are detailed in Table 2 (5).

The data indicates a decrease in inorganic phosphate, total and diffusible calcium, magnesium and the albumin-globulin ratio, while the serum proteins, globulin and possibly the potassium levels increased in wheat pasture poisoning. Because of the extreme variation found in the alkaline phosphatase of the normal cows, it was not possible to make any comparison of this component. In addition to the values given in Table 2, a limited number of determinations were made of the serum sugar, carbon dioxide combining power, hematocrit, sedimentation rate and the red and white blood cell counts. No significant variation was found in any of these components. The wide range of values observed in the cases suggests that the blood serum findings could be the result of the effect of wheat pasture poisoning rather than the cause.

FORAGE ANALYSIS

Chemical analyses of the growing wheat plant were begun at the start of the 1953-54 winter wheat grazing season, and were continued through 1954-55. Analyses were made for ash, crude protein (nitrogen x 6.25), calcium, magnesium, phosphorus, potassium and sodium.

Samples of the growing wheat plant were taken from pastures where wheat pasture poisoning had occurred and from pastures where this condition had not been observed. No significant differences in composition were found in the wheat from the two types of pasture. There were certain differences in composition depending on the age of the plant. These differences are discussed in more detail later in this bulletin.

Since some of the theories as to the cause of wheat pasture poisoning were concerned with the high potassium and protein contents of the growing wheat plant, a series of plots were treated with nitrogen or potassium, or both, with one plot which was not fertilized serving as a control. The nitrogen treatment varied up to 200 pounds per acre, while all potassium treated plots received 50 pounds of potassium per acre. Samples of growing plants were taken at weekly intervals from the time the plants were tall enough to clip until they started to head. No differences in composition were found among the various test plots regardless of the fertilizer treatment. This was first observed on single samples taken from larger plots.

In another study, wheat was planted at intervals of 2 weeks and samples were taken as soon as the plants could be clipped. Forty days elapsed from the time the first sample from the first planting was taken until the first sample from the last planting was taken. Differences in the crude protein, phosphorus and potassium levels in samples taken the same day were noted. However, when samples were compared with regard to the time since planting, these differences almost completely disappeared. The younger plants had higher contents of crude protein, phosphorus and potassium. There was little variation in the calcium, magnesium and sodium levels. This same pattern was observed in oats and rye. Oats had four to five times the sodium content of either wheat or rye.

Blood composition studies were made on cattle and sheep grazing the experimental pastures. No changes from the normal blood serum levels were observed in any of the fertilized pasture grazing studies. Since no significant change in plant composition had been observed, this lack of change in the blood is not surprising.

Several varieties of winter wheat were analyzed to see if strain or variety differences could

| TABLE 2. BLOOD SERUM VALUES FOR NORMAL COWS AND COWS SUFFERING FROM WHEAT PASTURE POISONING |
|----------------------------------|---------------------------------|------------------------------|------------------------------|
| Item                             | Wheat pasture poisoning cases   | Normal Hereford cows        | Differences in means          |
|                                 | Range                           | Mean and std. dev.          | Range                        | Mean and std. dev. | means and probable errors |
|                                 | High   | Low   |                  | High   | Low   |                  |                             |
| Alkaline phosphatase, Bod. units | 11.5   | 0.6   | 4.2(2.11)        | 46.6  | 0.8   | 6.4(6.78)        | 2.17(0.40)                  |
| Inorganic phosphate, mg./100 ml.| 12.3   | 1.2   | 4.3(2.36)        | 8.9   | 1.5   | 5.3(1.56)        | 1.23(0.22)                  |
| Total calcium, mg./100 ml.      | 15.1   | 3.9   | 6.6(1.12)        | 12.4  | 9.4   | 11.0(0.67)       | 5.40(0.11)                  |
| Diffusible calcium, mg./100 ml. | 5.2    | 1.3   | 2.6(0.77)        | 5.8   | 3.9   | 4.9(0.45)        | 2.32(0.07)                  |
| Magnesium, mg./100 ml.          | 3.2    | 0.4   | 1.3(0.73)        | 2.8   | 1.0   | 2.0(0.25)        | 0.70(0.07)                  |
| Potassium, mg./100 ml.          | 47.0   | 12.3  | 23.4 (8.3)       | 28.5  | 14.1  | 18.7 (2.2)       | 3.70(0.92)                  |
| Total protein, gm./100 ml.      | 16.7   | 6.58  | 8.0(0.77)        | 9.10  | 6.44  | 7.3(0.45)        | 0.72(0.07)                  |
| Albumin, gm./100 ml.            | 5.38   | 2.35  | 4.0(0.54)        | 5.20  | 3.67  | 4.9(0.24)        | 0.38(0.05)                  |
| Globulin, gm./100 ml.           | 6.14   | 2.62  | 4.1(0.70)        | 5.20  | 1.95  | 2.9(0.53)        | 1.14(0.07)                  |
| Albumin/globulin ratio, globulin = 1: | 1.63   | 0.40  | 1.0(0.24)        | 2.44  | 0.71  | 1.6(0.29)        | 0.61(0.03)                  |
be observed. None was observed either in plant composition or in the blood levels of animals grazing the wheat. This was confirmed by the case histories of actual cases. Outbreaks occurred without regard to the strain of wheat or time of planting.

Deijs and Wind (6) and Brouwer (7) from the Netherlands have suggested the possibility of the ratio of certain minerals in the plant as a possible cause of grass tetany. The minerals were potassium, sodium, magnesium and calcium. They observed changes in certain of these ratios in forage when cases of grass tetany developed in the Netherlands. However, when these same ratios were calculated for Oklahoma and Texas wheat, all of the samples, regardless of any incidence of wheat pasture poisoning, had ratios which were close to those found by Deijs and Wind at the time grass tetany was prevalent.

A few exploratory tests were made of the wheat plant for alkaloids and similar substances. Tests were made on the whole plant, the sap expressed by placing the plant under 8 tons of pressure, and on the residue from the pressing. None of the general screening tests used gave definite positive tests. It is felt, however, that further work of this nature might be of value.

TREATMENT OF CASES

During the period covered by these studies, the basic treatment for wheat pasture poisoning has been the intravenous and intraperitoneal injection of a calcium gluconate solution. For immediate response, the injection is given intravenously; for slower utilization and prolonged protection, the intraperitoneal route is preferred.

We have found that the need of a second treatment can be reduced if the preparation contains at least 17 percent calcium gluconate. Until 1953, treatment with calcium gluconate alone and leaving the cow on pasture was a satisfactory method. In the 1953 outbreak, best results were obtained with calcium gluconate preparations which were fortified with magnesium and phosphorus plus placing the cow on dry feed for a few days. Commercial preparations of the fortified and unfortified calcium gluconate are readily available.

Among other treatments tested were injections of thiamine hydrochloride and of dextrose, and drenches of sodium propionate and an organic calcium preparation—sodium and calcium salt of ethylenediamine tetraacetic acid. They either had no effect or were inferior to the calcium gluconate preparations.

The suggested method of treatment still is the injection of a calcium gluconate preparation. Our practice has been to give 500 ml, intravenously for immediate effect, followed by 500 ml, intraperitoneally for prolonged protection. The intravenous injection should be given slowly and carefully, since an injection made too fast may be fatal. Because of the effect on the heart, the intravenous injection should be given at such a rate so as to increase respiration only slightly. It also increases salivation. The time required for intravenous treatment may vary from 15 to 45 minutes, depending on the reaction of the cow.

No special precautions, other than to be sure the needle is in the peritoneal cavity, are necessary in intraperitoneal treatment. The calcium is absorbed slowly into the blood stream and does not produce the side effects noted when an intravenous treatment is used.

If a treatment was given during the first few hours of the symptoms, recovery usually was rapid and uneventful. If 8 to 12 hours elapsed before treatment, recovery generally was doubtful or slow, regardless of the type of treatment. In some cases, the animal would get to her feet after the first treatment but would go back down in a matter of hours. No recurrence of wheat pasture poisoning was observed in any animal which had recovered sufficiently from the initial attack to stay on her feet for 24 hours.

CONDITIONS CAUSING CONFUSION

Other maladies have been confused with wheat pasture poisoning and many investigations were made of what were thought to be cases in calves, steers and cows. The most common condition causing confusion has been spinose ear tick infestation with incoordination closely resembling that seen in wheat pasture poisoning. Black disease, or Clostridium novyi infection, supposedly confined to sheep, has accounted for many deaths of both yearling and mature cattle grazing wheat. This diagnosis was confirmed by bacteriological culture and animal inoculation. Wheat bloat, though a separate condition, has been observed.

Some other conditions which have been mistaken for wheat pasture poisoning are: acetonemia in mature cows, prussic acid poisoning from feeding bundles or grazing nearby stalk fields, pneumonia and shipping fever, milk fever in dairy breeds of cattle, "water belly" or urinary calculi in young steers and contagious abortion in cows—all of which were grazing wheat.
LITERATURE CITED


6 Personal communication 1952, Dejjs, W. B. and J. Wind.

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