Volume 14 | Issue 3 Article 2

1952

Trichloroethylene Extracted Soybean Oil Meal Poisoning

J. C. Picken Jr.
Iowa State College

H. E. Biester
Iowa State College

C. H. Covault
Iowa State College

Trichloroethylene Extracted Soybean Oil Meal Poisoning

Dr. J. C. Picken Jr.
Dr. H. E. Biester
Dr. C. H. Covault

Editor's note: **This article elaborates on the Iowa State College information service news release which is printed below. It was prepared to give practitioners a better picture of the course of the disease and details of its diagnosis. Experimental work is still being carried on, and a complete report of research data will be printed in the professional literature in the near future. Dr. Picken, author of the article, is an associate professor at the Iowa State Veterinary Research Institute. He was assisted in its preparation by Dr. Biester, head of the Veterinary Research Institute, and by Dr. Covault, head of the Department of Veterinary Medicine at Iowa State College.**

Ames, Iowa, March 22—Studies conducted at Iowa State College during the past 10 months show that a fatal disease can be produced in cattle when fed certain batches of commercially prepared trichloroethylene-extracted soybean meal.

The results of experimental tests conducted by the Veterinary Research Institute and the Agricultural Experiment Station of the college were announced today by an all-college committee which has been studying the problem since June, 1951.

Cattle fed the toxic meals seem to thrive for varying periods of time. Symptoms are apparent only in the final stages of the disease. The animals become listless and go off feed. High body temperatures of 106 to 108 degrees Fahrenheit and bloody discharges in the nostrils and in the feces usually can be observed. The animals do not respond to medical treatment and die in relatively short periods of time.

The disease produced by controlled feeding experiments at Iowa State College is similar to a fatal hemorrhagic disease of cattle reported in Scotland in 1916 and later in Germany and surrounding countries in 1924 to 1926. Cattle losses with similar symptoms have been reported recently from Iowa, Minnesota, Kansas, Colorado, North Dakota, South Dakota and Japan. Field observations associated these outbreaks with the feeding of trichloroethylene-extracted soybean meal.

In the Iowa experiments both young calves and older cattle have been found susceptible to the toxic meals. The degree of toxicity has varied greatly even though tested meals have been commercially manufactured under similar plant conditions. While there are instances where trichoroethylene-extracted meals have been fed extensively to cattle without causing death, it is extremely difficult to determine whether any given meal will or will not have the fatal effects which have been observed many times. The factor or factors responsible have not been identified as yet. Until this is done, potentially toxic meals can be detected only after costly feeding tests with cattle.

Research workers at the college emphasize that feeding tests for many years have shown that expeller-soy bean meal and hexane-extracted meal are satisfactory and valuable protein supplements.

The trichloroethylene-extracted soybean meal production was less than 2 percent of the total production of the United States last year. However, production was expanded in the last few years, with distribution largely centralized in the Midwest.

One of the contributing factors to the expansion was the development of an extraction process by chemical engineers of the Engineering Experiment Station at Iowa State College. Certain

features of this process have been patented and licenses issued to an industrial concern to manufacture these parts. The development was the result of a demand for a plant that could be operated with non-inflammable solvents and at the same time be practical for small operations.

THE *"fatal hemorrhagic disease"* that is associated with the feeding of trichloroethylene extracted soybean oil meal to cattle is a specific condition brought about by the presence of a toxic entity or entities in the soybean oil meal. This disease condition manifests itself with a definite series of symptoms which, when coupled with a positive history of trichloroethylene extracted soybean oil meal consumption serve to clearly differentiate this hemorrhagc syndrome of cattle from hemorrhagic sweet clover disease, the *"hemorrhagic septicemia complex,"* bracken poisoning, and others. In trichloroethylene extracted soybean oil meal poisoning of cattle we are dealing with a specific disease produced by the consumption of this feedstuff.

The field outbreaks of trichloroethylene extracted soybean oil meal poisoning that have appeared with increasing frequency and severity during the last several years in the United States and in several foreign countries have been in all respects identical with the ones described and studied in Scotland and Germany in the early part of the century. That there is and has been a real correlation between the rather localized areas involved in these outbreaks and the presence of trichloroethylene soybean oil meal extraction plants selling their meal into these areas has been well established. The isolated outbreaks that have appeared in other locations, often far removed from the location of the major difficulties, have been traced in most cases to a shipment of meal into that area from one or more of the trichloroethylene soybean oil extraction plants.

Even though the true cause of these early outbreaks had been diagnosed in May 1949 by the Iowa State College Veterinary Clinic and several practicing veterinarians their diagnoses were questioned vigorously by the trichloroethylene extraction industry. The industry's argument was that their plants had incorporated a heat treatment step designed to destroy toxicity and therefore their plants did not produce toxic meals. It was not until additional outbreaks had appeared and had been studied that it became quite apparent on the basis of field evidence that the soybean oil meals produced in these commercial plants were toxic to cattle.

Since the major outbreaks of a year ago some of the plants involved have ceased to operate. Obviously the incidence of this disease has decreased markedly in these areas.

The question was raised repeatedly during the outbreaks of the last several years as to whether the other types of soybean oil meals, the expeller and hexane extracted soybean oil meals, currently on the market produced this same toxicity disease. Both field and experimental evidence of many years duration as well as recent experiments designed specifically to establish that point have demonstrated that neither the hexane extracted nor the expeller soybean oil meals are associated with this poisoning of cattle.

Experimental Studies

Experimental studies however, have shown that commercially produced trichloroethylene extracted soybean oil meals when fed to cattle under rigidly controlled experimental conditions produce a fatal hemorrhagic disease that is identical with that observed in the field. Complicating factors so often present under field conditions have been eliminated in the experimental studies. The experimental studies leave no doubt that the samples tested were toxic for cattle and that the toxic tricholroethylene extracted soybean oil meals were the direct cause of the syndrome observed.

Obviously the picture obtained by the controlled experimental studies unfolds in a much more logical and orderly fashion than the rather complex and often

confusing historical and clinical picture that confronts the veterinarian seeing a field outbreak for the first time. Several facts of importance gained from these studies will be of special interest to those who have had and who may have contact with this particular toxicity disease.

1. Commercially prepared trichlorothylene extracted soybean oil meals vary widely in their degree of toxicity to cattle under controlled experimental conditions, ranging from meals that are very toxic or moderately toxic, to those of a quite low degree of toxicity.
2. The toxicity associated with the consumption of these meals is cumulative. Meals of low levels of toxicity if fed at high levels to cattle for extended periods of time will have the same fatal outcome as highly toxic meals fed for relatively short periods of time.
3. Cattle under controlled experimental conditions are not uniformly susceptible to meals of known toxicity. While cattle will succumb rather uniformly when fed meals of a high toxicity, the individual animal variation appears when meals of lower toxicity are fed.
4. It has been observed that young cattle appear to be more susceptible to the toxic meals than older cattle.
5. Animals may manifest the typical terminal symptoms resulting in death at somewhat extended periods after ceasing to consume trichloroethylene extracted meals. For example cattle that have consumed appreciable amounts of the meal during the stabling period may appear quite normal when turned to pasture and eventually succumb to the disease up to several months later.

The rather widely different clinical pictures that have been reported for some of the natural outbreaks of this disease become more understandable when in addition to the above cited factors, one considers the rather wide variations that obviously occurred in the individual herds such as chance acquisition by the owner of either high or low toxicity meals, the amounts of this meal actually fed and consumed by the animals, the duration of feeding, the general nutritional status and management practices within the herd.

Diagnosis

With respect to diagnosis, trichlorethylene extracted soybean oil meal poisoning does not lend itself to early detection. It is rather remarkable that during the period of consumption of a toxic meal, until just prior to the appearance of the final terminal stages of the disease, there are no visible signs of the marked disintegration that is occurring within the animal. The animals will thrive, with excellent growth, performance, and general condition equal to that obtained with any good ration and management program.

The sudden onset of the clinical stages of this disease, usually in but one or two animals with the remainder in apparent excellent health, does not suggest to the owner or his veterinarian the true cause of the trouble, the toxic soybean component of the ration. The insidious nature of the toxicity, which may take from one to six months or even longer to manifest itself, often is not appreciated on the first appraisal.

The initial stages of disintegration within the animal may be detected by studies of the blood. There is a progressive development of leucopenia with a relative lymphocytosis. While the degree of leucopenia will often vary to some extent the relative lymphocytosis becomes progressively more absolute and immediately prior to the terminal stages differential white cell counts will invariably show 95 to 100 percent lymphocytes.

The onset of the clinical stages of the disease is rather sudden. There is indisposition and in milking animals a sudden drop in milk flow. About this time blood will usually be seen to trickle from one or both nostrils. The temperature is elevated to 105-109°F. There is shivering, staring haircoat, suspended rumination and decreased or lost appetite. Many of the affected animals show marked salivation. The feces usually appear to be normal at first but soon one detects admixtures of blood and, finally, little but blood clots are passed.

Abdominal pain is manifested by the paddling of the feet and kicking at the abdomen.

As a result of hemorrhage, swellings are sometimes observed in the subcutis one-half to several inches in diameter. A stiff gait or distinct lameness may be observed as a result of hemorrhage into the muscles.

When affected animals are examined carefully one will note small hemorrhagic areas in the mucosa of the sclera, nasal passages and vagina.

Few animals developing the above symptoms ever recover. Death usually takes place in four to five days after onset of the clinical symptoms. Some die suddenly, as early as the second day, while others linger for as long as fifteen days. Toward the end the heart-beat is tumultous, the mucous membranes anemic, and there is great general weakness. Toward the end of the terminal clinical stage the leucopenia rapidly becomes severe, white blood cell counts in the range of 1000 to 2000 are frequently observed. The relative lymphocytosis becomes essentially absolute with a complete absence of granulocytes. In cases where profuse intestinal bleeding has occurred marked anemia is common.

Post Mortem Findings

The basic changes found at necropsy of cattle that have died as the result of this malady consist of petechiae, ecchymoses, linear, or diffuse hemorrhages. Various types of hemorrhage and ulcers are noted in the gastro-intestinal tract. Hemorrhagic areas, sometimes progressed to ulcers, may be found on the mucous membrane of the nasal cavity including that of the septum and turbinates.

Hemorrhages are present in the subcutaneous tissues, muscles, tendons, and joints. The serous surfaces of the pleural and abdominal cavities are characterized by extensive hemorrhages. In some instances the muscles of the diaphragm are diffusely infiltrated with blood. Free blood may be found in the abdominal cavity.

The serous surfaces of the rumen, reticulum, omasum, abomasum and intestines present extensive hemorrhages. The folds or laminae of the omasum sometimes are severely affected, both the mucosa and underlying tissues being hemorrhagic. In the abomasum are found varying sized hemorrhages in the mucosa and submucosa that have developed into shallow ulcers. Some cases present ulcers, over which escaping blood has clotted and built up above the surface suggestive of cotyledons. Free blood may be present in the abomasum and in the intestines. In the terminal stage the cecum and large intestine, including the rectum, may be filled with blood containing feed residues.

The kidney often presents ecchymoses on the subcapsular surface and on the cut surface. The medulla may show large hemorrhages from ruptures of larger vessels. Subserous and submucous hemorrhages are usually present in the urinary bladder.

The liver also may contain hemorrhages ranging from the ecchymotic type to larger accumulations of blood. The entire organ may contain myriads of foci of necrosis. The gall bladder presents hemorrhagic suffusions under the serosa and smaller hemorrhages in the mucosa.

The pericardial sac may present hemorrhages, but subepicardial and subendocardial hemorrhages are nearly always present, either as discrete or diffuse hemorrhages one to two millimeters in thickness.

The spleen generally shows ecchymotic, diffuse and linear capsular hemorrhages.

When the brain is removed, varying amounts of clotted and free blood may be found in the cranial cavity.

Treatment

There is no effective treatment for this condition. Early diagnosis on the first or second animal, i.e., the most susceptible, with the immediate removal of the offending meal from the rations of all of the animals, may result in saving many or possibly all of the animals that have not shown clinical symptoms. Either expeller or hexane extracted soybean oil meal can be substituted for the trichloroethylene extracted meal that is removed.

Treatments used in hemorrhagic disturbances of cattle and other species have had no demonstrable effect on this disease. Symptomatic treatment possibly coupled with antibiotic therapy to assist the weakened animal in combating possible secondary invaders may be of some value.

Obviously a final diagnosis of trichloroethylene extracted soybean oil meal poisoning requires in addition to the typical clinical and pathological picture, evidence of an adequate consumption of trichloroethylene extracted soybean oil meal by the animals involved. A careful and thorough investigation must establish beyond any doubt whether trichloroethylene extracted soybean oil meal was fed. Remembering that this type of investigation will not be at all popular, the veterinarian should thoroughly establish his position with respect to a complete history, clinical and pathological findings, and the owner's source of the suspected soybean oil meal or commercial mixed feed before he or the owner embarks on the investigation.

Zinc is essential for growth and efficient operation of certain enzyme systems. It influences the rate of absorption of carbohydrates and proteins from the gastrointestinal tract.

Artificial insemination of cattle with gelatinized sperm in cellophane tubes is used by the majority of veterinarians in Denmark. Paraffined cellophane tubes are filled with gelatinized sperm and sealed with a paraffin plug. A special instrument is used for expelling the gelatinized sperm from the cellophane tube and depositing it into the cervical canal.

Trichocidin, an antibiotic produced by a mold belonging to the Penicillium group, prevents the growth of *Trichomonas fetus* and *Staphylococcus.*