Toxicity of Trichloroethylene-Extracted Soybean Oil Meal

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Trichloroethylene-extracted soybean oil meal has been the cause of several major outbreaks of a fatal hemorrhagic toxicity disease of cattle during the past 40 years. Trichloroethylene or its breakdown products may react directly with some component of the soybean during processing to form the toxic meal. Evidence supporting the possible autoxidative decomposition of trichloroethylene during the processing of soybeans has been presented. In order to evaluate this hypothesis, soybeans, defatted soybeans, soybean protein, and casein were treated with the products resulting from the autoxidation of trichloroethylene in the attempt to produce typically toxic preparations. None of these products, assayed in the young calf, appeared capable of producing the typical toxicity disease, though isolated manifestations of the disease syndrome developed. In further studies, the normal operation of a commercial trichloroethylene-soybean extraction plant was altered in the attempt to produce conditions both favorable and unfavorable to possible autoxidative decomposition of trichloroethylene. Assay indicated that the meal toxicity was neither enhanced nor decreased by the modifications.

That trichloroethylene - extracted soybean oil meal causes a refractory, hemorrhagic, aplastic anemia when fed to cattle has been demonstrated repeatedly by the many investigators who have studied in detail the several major outbreaks of this toxicity disease (*1*, *4*, *6*, *9–11*). These studies in the past have been directed toward the characterization of the disease and the demonstration of the primary cause, the trichloroethylene-extracted soybean oil meal. After the cause was established, production of the offending meal was stopped and the disease soon disappeared. Until recently no major effort has been directed toward discerning the nature of the toxic entity.

The studies described by McKinney and others (*5*) represent one approach. Their evidence suggested that, if such an autoxidation did occur under plant operating conditions, one or more of the autoxidation products might be associated with the toxicity either per se or by reaction with some component of the soybean during processing. The present report describes the studies undertaken to evaluate this hypothesis.

Two experimental approaches, directly related through the autoxidation hypothesis, were undertaken. The first investigated the possibility of altering the toxicity of trichloroethylene-extracted soybean oil meals by modifying the operating procedures of a commercial plant to produce conditions either more or less favorable to the autoxidation of trichloroethylene. The second examined the possibility of producing typically toxic preparations by reaction of products resulting from the autoxidation of trichloroethylene with soybeans, defatted soybeans, soybean protein, and casein. The individual samples prepared during these investigations are described in Table I.

Preparation of Samples

The modifications of the operating procedures and conditions of commercial processing of trichloroethylene-extracted soybean oil meal are represented by samples 1 and 2. In the preparation of sample 1, operating procedures were modified to allow recycling of the flakes two additional times through the extraction and desolventization phases. This recycling of the hot and dry flakes increased normal operating temperatures materially. In the preparation of sample 2, two changes were made in the normal operating conditions. Desolventization temperatures were reduced by decreasing the steam pressures below those normally maintained in the desolventizer jackets. In a further attempt to prevent autoxidation of the solvent, an oxidation inhibitor for trichloroethylene, triethylamine, was added to the working solvent of the plant at the level of 0.1% by weight. This concentration was maintained during the preparation of sample 2 by frequent additions.

In order to study directly the relationship of the autoxidation of trichloroethylene and meal toxicity, a series of samples was prepared in the laboratory in which autoxidation products of trichloroethylene reacted with soybean flakes and other protein sources. The initial sample of this series, No. 3, was prepared by treating undried commercial hexane-defatted soybean flakes with dichloroacetyl chloride, one of the main products resulting from the autoxidation of trichloroethylene. Analysis for total chloride on this preparation indicated that 95% of the dichloroacetyl chloride had remained with the flakes in one form or another. Analysis for total and Mohr chloride on the aqueous alcohol extract of the reacted flakes indicated that approximately 70% of the acid chloride used had reacted with the moisture in the flakes, while 25% had presumably reacted to acetylate components of the undried flakes. The pH of an aqueous slurry of this sample was 5.6. This preparation, possibly because of its lack of heat treatment, was comparatively unpalatable and seemed to be poorly digested. Pressure toasting, by increasing the moisture content to 20% and autoclaving for 15 minutes at 15 pounds per square inch gage, relieved these difficulties. As a precautionary measure, samples 4, 5, and 6 were pressure toasted by the same procedure prior to assay.

Samples 4 through 6 were prepared from immature soybeans, because, in earlier studies, highly toxic meal had been produced with immature beans (*8*). Sample 4, prepared by treating dried flakes with dichloroacetyl chloride, did not contain appreciable aqueous alcohol-extractable acid, indicating that

essentially all of the dichloroacetyl chloride had reacted to acetylate components of the meal. The pH of an aqueous slurry was 6.1.

Samples 5 and 6 were prepared by reaction of the soybean flakes with a liquid oxidation mixture which contained about equal moles of dichloroacetyl chloride and trichloroethylene epoxide, along with about 1% of trichloroethylene polymer and traces of phosgene (*5*). Sample 5 produced an aqueous slurry of pH 4.6, indicating that much of the dichloroacetyl chloride and trichloroethylene epoxide components of the oxidation mixture had reacted with the moisture present in the flakes. By comparing Mohr chloride values with total acid values, 48% of the acidity could be accounted for as hydrogen chloride. This sample had a krautlike odor. Sample 6, prepared with dried flakes at 1° C., appeared, on Mohr chloride and total chlorine analyses, to have reacted directly with the dichloroacetyl chloride and the trichloroethylene epoxide in the oxidation mixture. The pH of an aqueous slurry was 5.4.

Samples 7 through 10 differed in several respects from those previously described. As indicated in Table I, the ratios of oxidized trichloroethylene to the flakes or protein material were much larger than with the previous preparations. Defatting, when necessary, was accomplished prior to the addition of oxidized trichloroethylene. The defatted flakes or protein materials were suspended in trichloroethylene rather than hexane. Immediately after the initial mixing of the reactants, the reaction mixture was subjected to heat until desolventization was complete. With this procedure any unreacted oxidized trichloroethylene products as well as the suspending solvent were driven off by direct heat, in contrast to draining and several washings with hexane prior to the application of heat.

The desolventization by heat was accomplished in all-glass equipment, which consisted of an indented 12-liter round-bottomed flask suspended horizontally in a heated oil bath. Suitable rotating and condensing facilities were provided. After being charged with the reactants, the flask was placed in the oil bath, the drive and condensing facilities were connected, and heat was applied. Desolventization of the charge, as represented by distillate, appeared to result in a small amount of low-boiling fraction, the trichloroethylene-water azeotrope, and finally the balance of the trichloroethylene. When the bath temperature reached 125° C. the run was stopped, the flask was disconnected, and the charge was emptied and allowed to cool.

It was apparent that samples 7 through 10 reflected to a marked degree the relatively large amounts of oxidized trichloroethylene used in their preparation. The pH's of the aqueous slurries were all acid and each sample had an acrid, offensive odor. Upon their addition to water or milk, the odor was even more evident and, as expected, this markedly affected the acceptability of the samples to the assay calves. Sample 9 had a definite krautlike odor.

A definite purpling of the casein preparation (sample 9) was evident each time a batch was prepared. Transitory purpling of the other samples of this series was noted occasionally during their preparation, but disappeared or was masked by browning of the products during the final stages of desolventization. Investigation of this purpling reaction revealed that oxidized trichloroethylene preparations could be substituted for Hopkins-Cole reagent (*2*) in the Hopkins-Cole test for protein (*3*), a fact which suggested the presence of glyoxylic acid or a similar aldehyde in the oxidation mixture capable of reacting with the indole nucleus of tryptophan. Upon further investigation glyoxylic acid was isolated from oxidized trichloroethylene preparations (*5*).

Evaluation of Toxicity

The samples were evaluated as to their ability to produce in the young calf the same disease produced by trichloroethylene-extracted soybean oil meals. Several recent detailed descriptions of this toxicity disease in older and mature cattle have been presented (*4, 7, 9*). The young calf fed a toxic trichloroethylene-extracted soybean oil meal exhibits essentially the same disease syndrome as older cattle (*8*). The syndrome is typical of aplastic anemia with characteristic clinical, hematologic, and necropsy manifestations. Clinically the young calf shows in the terminal acute stages a marked decline in condition, elevated body temperature, discharge of blood from one or more of the natural body openings, occasional visible subcutaneous hemorrhage, and finally collapse and death. Hematologic abnormalities of diagnostic significance include a gradual, and finally a precipitous, decline in total numbers of white blood cells, a relative lymphocytosis, and a moderate to severe decrease in the number of red blood cells. The lesions seen at necropsy examination are essentially those of hemorrhage throughout the entire body.

Both the size of the sample and duration of the assay influence the interpretation of the results obtained with the small calf. With meals of high potency, samples ranging from 20 to 40 pounds produce the typical disease in 30 to 50 days. With meals of very low potency, samples of from 0.5 to 1.5 tons are often required with assay periods from 1 to 2 years before the typical disease is produced (*8*). Therefore when the sample size is large, the assay is reliable for detecting the toxic component in meals of both high and low levels of toxicity. When the sample size is limited, positive assay results indicate the presence of the toxic entity in appreciable amounts, while negative assay results indicate only that the sample contains either none of the toxic component or not enough to produce symptoms of the disease during the necessarily limited assay period. With the exception of samples 1 and 2, the samples prepared for this investigation, because of their limited size, were subject to this restricted interpretation of toxicity.

The assay animals were young male dairy calves of various breeds, ranging in age from 11 to 94 days at the beginning of the assay. Where possible, calves less than 30 days of age were used. The older and larger calves were used only when the amount of sample permitted.

Samples were administered in the feed. The very young calves received their entire allotment as a finely ground product mixed in milk. Later, if quantity available permitted, the samples also were incorporated in the concentrate mixture and eventually when the calf was weaned from milk the entire allotment was fed in the concentrate. The sample consumption by each route was recorded.

The assay calves were observed daily for clinical signs of the disease. Hematologic examinations were made routinely at weekly intervals and more frequently as a disease syndrome developed. Red blood cell, white blood cell, differential white blood cell, hemoglobin, and hematocrit determinations were made on all blood samples. Detailed necropsy examinations were made on all calves that succumbed or exhibited any clinical or hematologic evidence of a diseased state.

Assay Results

The assays performed and the results are presented in Table I. The typical disease with its clinical, hematologic, and necropsy manifestations was produced with only two of the preparations, samples 1 and 2. None of the other preparations, with the amounts available, produced the typical disease, though several produced isolated manifestations of the disease syndrome.

Samples 1 and 2, when compared to results with other trichloroethylene-extracted soybean oil meals, assayed as moderately toxic preparations. The toxicity levels observed were in the normal range for meals produced by plants of this design during the late spring (*8*). The rather large variations within and between samples are not uncommon in the assay of meals of this

Table I. Toxicity Studies of Autoxidation Products of Trichloroethylene

No.	Reaction mixtures	Preparation of Samples: Reaction temp., ° C.	Reaction times, hours	Extraction and desolventization	Assay of Samples: Calf No.	Age on expt., days	Assay period, days	Sample fed, lb.	Cond. end of assay	Typical disease[a]
1	Triple-processed meal[b] Commercial processing modified to allow recycling meal through extractor and desolventizer 3 times	Extraction. 60 1st, 78 3rd cycle. Desolventized meal emerged 130 1st, 160 3rd cycle	Recycling time 2 hr. 20 min.	Sample extracted and desolventized 3 times; desolventizer jacket temp. normal at 175–180° C.	3682 3707	73 11	47 150	74.0 255.0	Dead Fair	Yes Yes
2	Low temperature-inhibited meal[b] Commercial processing modified to reduce desolventizer temperatures; inhibitor concentration increased and maintained during extraction and desolventization[c]	Extraction at 60; meal from desolventizer at 120	Normal time 1 hr. 10 min.	Processing sequence normal; normal desolventizer jacket temp. 175–180° C. reduced to 155–160° C.	3690 3719	56 16	144 74	244.0 133.0	Good Dead	Yes Yes
3	Dichloroacetyl chloride–treated soybean flakes 100 lb. defatted soybean flakes[d] 1.1 lb. dichloroacetyl chloride Hexane to cover reaction mixture	Room temp. (25–30)	48	Reaction mixture drained from flakes; flakes desolventized at 60° C. in cyclone dryer	3745	27	143	86.0	Fair	No[e]
4	Dichloroacetyl chloride–treated soybean flakes 100 lb. partially defatted, dried immature soybean flakes[f] 0.84 lb. dichloroacetyl chloride (1 wt. % dry oil-free flakes) Hexane to cover reaction mixture	Room temp. (25–30)	96	Reaction mixture drained from flakes; flakes washed 4 times with hexane, desolventized at 140° C. in iron equipment	3805	24	64	87.0	Good	No[e]
5	Oxidized trichloroethylene-treated soybean flakes 100 lb. partially defatted immature soybean flakes[g] 3.7 lb. oxidized TCE[h] (5 wt. % dry oil-free flakes)	Room temp. (25–30)	96	Reaction mixture drained from flakes; flakes washed 4 times with hexane, desolventized at 140° C. in iron equipment	3837	13	57	26.1	Good	No[e]

[a] Production of clinical, hematologic, and necropsy manifestations of trichloroethylene-extracted soybean oil meal toxicity.
[b] Prepared May 1952 from 1951 beans in plant of design similar to that described by Sweeney and others (*12*).
[c] Triethylamine added to solvent and concentration maintained at 0.1 wt. % during extraction and desolventization.
[d] Commercial untoasted, hexane-extracted soybean flakes.
[e] During assay period calf gave no clinical or hematologic evidence of developing typical toxicity disease.
[f] Immature soybeans, flaked and dried to 0.1% moisture in vacuum; 27% of oil was removed by initial hexane extraction.
[g] Immature soybeans (12% moisture), flaked; approximately 27% of oil removed by initial hexane extraction.
[h] Oxidized trichloroethylene, prepared as described by McKinney and others (*5*).

level of potency. In interpreting the data presented in Table I, the apparently somewhat greater toxicity of sample 1 cannot be considered as significant.

Samples 3 through 6, in the amounts available, did not adversely affect the assay calves, nor was there any clinical or hematologic, indication of the typical toxicity disease.

Samples 7 through 10 appeared to exert a possible twofold effect upon the assay calves. These samples uniformly affected the growth and well-being of the calves adversely, even though relatively small amounts of the preparations were consumed. This effect apparently was nonspecific and probably was attributable to the corrosive nature of the preparations. Desquamation and ulceration of the mucosa of the abomasum and duodenum were observed in all the calves fed these preparations. Though of questionable significance because of this generalized nonspecific effect on the assay calves, additional symptoms and lesions observed during the assay of these preparations were remarkably similar to those seen in calves affected with the typical toxicity disease. The observations, associated with the respective samples, appear as footnotes to Table I.

Discussion

The limitations imposed by the assay for toxicity and the necessary criteria of toxicity under which the individual preparations were evaluated make it impossible to establish either positively or negatively the validity of the autoxidative decomposition hypothesis. However, if the data are considered collectively, a rational pattern emerges which may have a direct bearing upon the closely related fundamental question—the manner and conditions under which the toxic entity is formed.

The apparent inability of the recycling under commercial operating conditions (sample 1) to enhance appreciably the toxicity of a trichloroethylene-extracted soybean oil meal may be significant. If the assumption is made that recycling of a meal will not materially increase its

Table I. Toxicity Studies of Autoxidation Products of Trichloroethylene (Continued)

	Preparation of Samples				Assay of Samples					
No.	Reaction mixtures	Reaction temp., ° C.	Reaction times, hours	Extraction and desolventization	Calf No.	Age on expt., days	Assay period, days	Sample fed, lb.	Cond. end of assay	Typical disease[a]
	Hexane to cover reaction mixture									
6	Oxidized trichloroethylene-treated soybean flakes	1	168	Reaction mixture drained from flakes; flakes washed 4 times with hexane, desolventized at 140° C. in iron equipment	3836	11	47	21.5	Good	No[e]
	100 lb. partially defatted, dried, immature soybean flakes[f]									
	2.5 lb. oxidized TCE[h] (3 wt. % dry oil-free flakes)									
	Hexane to cover reaction mixture									
7	Oxidized trichloroethylene-treated soybean flakes	Initial, 25–30; final, 125	Approx. 2 for desol.	Reaction mixture desolventized by direct heat in all-glass equipment	286	18	8	0.93	Moribund	No[k]
	4.2 lb. defatted soybean flakes[j]				3823	15	69	10.1	Fair	No[m]
	1.6 lb. oxidized TCE[h]									
	6.4 lb. trichloroethylene									
8	Oxidized trichloroethylene-treated soybean flakes	Initial, 25–30; final, 125	Approx. 2 for desol.	Reaction mixture desolventized by direct heat in all-glass equipment	3784	94	68	9.9	Fair	No[n]
	4.4 lb. defatted soybean flakes[d]									
	1.6 lb. oxidized TCE[h]									
	6.4 lb. trichloroethylene									
9	Oxidized trichloroethylene-treated casein	Initial, 25–30; final, 125	Approx. 2 for desol.	Reaction mixture desolventized by direct heat in all-glass equipment	287	18	26	2.9	Moribund	No[p]
	5.0 lb. casein[n]									
	1.3 lb. oxidized TCE[h]									
	8.3 lb. trichloroethylene									
10	Oxidized trichloroethylene-treated soybean protein	Initial, 25–30; final, 125	Approx. 2 for desol.	Reaction mixture desolventized by direct heat in all-glass equipment	290	18	67	7.7	Good	No[e]
	5.0 lb. purified soybean protein[q]									
	1.3 lb. oxidized TCE[h]									
	0.75 lb. water									
	9.0 lb. trichloroethylene									

[i] Defatted in laboratory with trichloroethylene at room temperature.
[k] Calf exhibited elevated terminal temperatures and hemorrhagic lesions at necropsy; no hematologic evidence of disease observed.
[m] Calf exhibited a few hemorrhages at necropsy; no clinical or hematologic evidence of disease observed.
[n] Casein, plain, untreated obtained from General Biochemicals, Inc., Chagrin Falls, Ohio.
[p] Calf exhibited severe rather typical leucopenia; a few hemorrhagic lesions observed at necropsy.
[q] Drackett protein, specification 220 (assay type), obtained from Drackett Products Co., Cincinnati, Ohio.

toxicity, certain hypotheses may be suggested. It is possible that the meal has accepted or produced within itself during the first cycle the maximum amount of toxicity it is capable of accepting or producing. Also, the conditions favorable to the formation of toxicity during the initial cycle may be absent during the second and subsequent cycles. Further exploration in these areas conceivably could add much to the knowledge of the mode of formation of the toxic entity.

If it may be assumed that the isolated manifestations of the typical toxicity disease observed with samples 7 through 9 were not secondary and were, therefore, manifestations in the assay calf of a specific toxic entity, the data as a whole suggest that the direct application of heat may be an important factor in the production of the toxic entity. On this basis the autoxidative decomposition hypothesis might have merit and the relationship of heat to the formation of the toxic entity deserves further consideration. The apparent nonconformity of sample 10 with the other preparations of this latter series may have been due to the difference in its preparation—i.e., the addition of water to the reaction mixture.

The production of the severe leucopenia in the assay calf receiving the treated casein preparation (sample 9) is of significance for several reasons. It was without doubt the most substantial segment of evidence obtained indicating that a specific chemical entity, possibly related to that in the trichloroethylene-extracted soybean oil meal, had been produced, either in the oxidized trichloroethylene preparation or by the interaction of some component of the oxidation mixture with casein. It permits also the speculation that the protein component of the soybean may be the one associated with the toxicity of the soybean meal. Finally, the results with the casein preparation suggest that the soybean per se may not be essential to the formation of the toxic entity and that under suitable processing conditions other oil seeds, meat products, and proteinaceous materials might produce toxic products.

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HIGH-FREQUENCY COOKING

Browning Methods in Microwave Cooking

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Microwave cooking, because of its penetrating nature and minimum generation of environmental heat, yields a product that may differ in appearance, surface texture, and eating characteristics from a product cooked by conventional methods. One such important characteristic, desirable in many cooked foods, is a certain amount of browning of the food surface. This paper describes a method which utilizes the addition of materials normally found or used in foods to give microwave-cooked foods the characteristic browning of conventional cooked foods with a minimum of cooking time.

In conventional ovens the process of cooking to the center of food masses by conduction requires a temperature gradient, in which surface temperatures are usually much higher than the final cooked temperature of the food. For example, a gas-fired oven may be maintained at 300° to 400° F. to produce a meat roast with an internal temperature of 140° F. Under these conditions, browning of the surface is readily obtained. In microwave cooking, which is the phrase used to describe cooking in equipment of the Radarange type (Raytheon Manufacturing Co.), the ambient temperature in the cooking space is approximately room temperature. Any rise in temperature in this space is due to heat loss from the food to its environment. In these electronic ranges alternating electric fields are generated by a magnetron oscillating at a frequency of 2450 megacycles. The energy of electromagnetic waves, which are called microwaves at this frequency, is immediately absorbed within the food mass. The resulting increase in thermal energy in the food depends on an interaction between the microwave energy and the particles of which the food is composed, such as electrons, atoms, molecules, and charge carriers. This interaction occurs throughout the food mass, instantly produces heat, and results in rapid cooking.

Normally, food is cooked in utensils which are not receptive to the microwave energy. The food is the absorber of energy, and it shows a definite pattern of absorption, with the lowest temperatures initially at the surface and at a point deep within a large food mass. Economy of energy is inherent in the microwave method compared with the conventional situation, in which the temperature of the environment is raised, in order to heat and cook through the mass of food by conduction. However, this economy of energy has important consequences which affect the acceptance of foods cooked by the microwave method. The food habits of consumers require that the food surface have certain familiar characteristics. The normal charred appearance of steak, the deep brown color of roast beef, and the golden brown color of pie crust are notable examples. A substantial percentage of the flavor and odor is also determined by the reactions causing these color changes. Proper use of microwave cooking requires that these "cooked" colors be obtained, so that the resulting food product will not demand major adjustments in food habits of consumers.

The normal cooked color of foods is a function of temperature, time, and composition. In many foods cooked by microwaves, natural browning occurs to an acceptable degree. Generally the long-time cooking foods show considerable color. Meat roasts and large chickens are in this group. Users of Radarange equipment have employed various substances such as sauces or salts to obtain a desirable color—e.g., a chestnut flour and chicken fat sauce have been used in poultry cooking, and salt pack for meat roasting. In this laboratory, dough made of either regular cake flour or chestnut flour did not brown in microwave heating. However, good results were obtained with commercially available powdered gravy mixes sprinkled on the surface of chopped meat patties and chops. Although a desirable color and