It is thus a curious fact that most classes of antithyroid substances are either competitive substrates or inhibitors of peroxidase, the thiocarbonamides being in the former category (*7*) and sulfonamides (*10*), anilines, and polyphenols in the latter.

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Ascorbic Acid and Physiological Breakdown in the Fruits of the Pineapple (*Ananas comosus* L. Merr.)

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Many tropical fruits may be injured when they are stored too long at temperatures in the range of 0°–10° C. The injury is often called "chilling," and it is usually characterized by a darkening of the flesh or of the peel and by failures of the mature green fruits to ripen properly when they are subsequently stored at room temperature. Specific effects on flavor and chemical constituents may also appear, depending upon the particular kind of fruit involved.

The exact manner in which chilling modifies the normal metabolism of tropical fruits is not entirely understood, although several investigators have contributed valuable information to the subject. Jones (*1*), in his studies of the papaya, found that ripening changes (especially hydrolysis of sucrose) were retarded by low temperatures, and the effect on respiration was so marked as to suggest that the basic metabolism of the fruit was upset. Harris and Poland (*2*) reported a loss of ascorbic acid in chilled bananas, but the fruits had been severely injured by exposure to low temperatures, so that these particular changes may have been largely the result of autolysis of the cells. It was previously reported by the senior author (*3*) that two lots of imported pineapples showing physiological breakdown were characterized by a lower content of ascorbic acid than was true of normal fruits. These fruits showed advanced stages of physiological breakdown, and, as in the experiments of Harris and Poland, the destruction of ascorbic acid may have occurred during autolysis rather than in the earlier stages of exposure to low temperatures. The results that are now being reported were obtained by analyzing pineapples which had been stored at low temperature but which had not shown visible evidence of physiological breakdown when the fruits were sampled.

TABLE 1

EFFECT OF COLD STORAGE ON CERTAIN CONSTITUENTS OF THE JUICE OF THE ABACHI PINEAPPLE

Treatment	Bromelin activity ($1/t$)	Total soluble solids (%)	Total acid (g/100 ml)	Ascorbic acid (g/100 ml)
Stored at room temperature	0.377	12.9	0.98	42.4
Stored at 6° C for 1 wk, then at room temperature for 2 days	0.363	11.5	0.79	25.9

Abachi pineapples were grown in Florida, harvested in the "mature green" or "market ripe" stage of maturity and shipped to Pittsburgh, Pa., by railway express. Upon arrival at destination the fruits were divided into two lots, each comparable to the other in regard to stage of maturity of the individual fruits. One lot was held at room temperature (25°–30° C) for 2 days and then analyzed. The other lot was stored at 6° C for 1 week and then held at room temperature for 2 days before analyzing. Each fruit was sampled individually in order to facilitate statistical interpretation of the results. The expressed and filtered juice was analyzed for total soluble solids, total acids, ascorbic acid, and bromelin activity. Each lot consisted of 10 pineapples. The averaged results appear in Table 1.

The two lots did not differ significantly in regard to total soluble solids, total acids, and bromelin activity of the juice, but a highly significant difference in ascorbic acid was observed. The refrigerated lots of pineapples contained 25.9 mg ascorbic acid/100 ml juice, compared to 42.4 mg in the control fruits. This amounts to a reduction of 38.9%.

The pineapples showed no ill effects of the cold storage other than this loss of ascorbic acid. There was no discoloration of the flesh and no deleterious effect on flavor. In other words, the period of storage was interrupted before any visible symptoms of chilling had appeared, and it is concluded that destruction of ascorbic acid constitutes the first phase in the development of low-temperature injury.

This does not explain all that occurs when tropical fruits are injured by exposure to low temperatures. It *does* suggest that such a treatment interferes with a specific step in the respiratory processes of the plant cell. It is known, for example, that in one stage of respiration certain phenolic compounds are oxidized to quinones, the latter being black or brown in color. During the normal course of the process quinones are converted back to phenols by ascorbic acid. This reversible action continues as long as an adequate supply of ascorbic acid is present. Since physiological breakdown of pineapples is characterized by a darkening of the flesh, it seems logical to conclude that the discoloration in the affected fruits indicates that the

above-mentioned oxidation-reduction has been stopped in the quinoid stage. The same explanation may be applicable to low-temperature injuries of other tropical fruits, although our results have been obtained only with pineapples. The fact that immature fruits are more susceptible to low-temperature injuries than mature fruits tends to confirm the theory, because of the larger content of phenols in the form of soluble tannins in the immature fruits.

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Experimental Production of Hyperkeratosis ("X Disease") of Cattle with a Chlorinated Naphthalene[1]

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The baffling and costly malady known as X disease of cattle was first recognized in New York state and described by Olafson (*1*) in 1947. It has been recognized since that time in all states east of the Rocky Mountains, and the Southeastern states seem to have a higher incidence of diseased herds than any other section of the country. In Tennessee alone more than 2300 head of cattle have been affected since 1947. Valuable purebred herds have been affected, and the raising of calves in some herds became impossible over a three-year period. Such diseased herds were slaughtered.

Olson and Cook (*2*) produced the disease in cattle by using a commercially prepared feed that had been incriminated in an outbreak of the disease in a herd in Nebraska. Wagener (*3*) produced it in Germany by exposing cattle to a complex wood preservative used in the construction of a new barn. Olafson and McEntee (*4*) also produced the disease by feeding cattle a processed concentrate, and Bell (*5*) infected calves by feeding them a lubricant. None of these workers has identified the specific chemical compound or compounds that produced the disease.

This experiment was designed to use a known chemical compound, which may be used on many farms in many different ways, and the effect of which was unknown on the bovine. There is no reference in the literature to its having been administered to cattle. Thus the experiment, in the early stages, was one of trial and error to find a toxic dose which would not produce immediate death but which would make the animal ill.

[1] A contribution from the interregional project entitled "X Disease (Hyperkeratosis of Cattle), a Cooperative Study Participated in by the Agricultural Experiment Stations and the Bureau of Animal Industry, U. S. Department of Agriculture."

Two grade Hereford females, one year old and weighing about 500 pounds each, and one Jersey Hereford crossbred female, eight months old and weighing about 400 pounds, were used in the tests. Pentachloronaphthalene was used in the experiment and administered *per os* in capsules each day. The control animal of comparable size and age remained healthy throughout the experiment.

TABLE 1

No. days dosed	Dose/day (g)	Grams pentachloronaphthalene for entire period
10	2	20
10	4	40
10	6	60
10	8	80
Total 40		200

Hereford No. 748 was used as a pilot test to try to find the amount necessary to cause death when administered over a two-week period. This animal received 15 g/day for 13 days and was sacrificed on the 17th day because of its morbid condition. With this acquired knowledge of the toxicity and the amount necessary to cause death in 17 days, the other two animals were dosed according to Table 1.

Symptoms observed were identical with those seen in naturally occurring field cases. They included excessive lacrimation, diarrhea, polyuria, marked salivation, and a serous discharge from the nostrils. A chronic cough, poor appetite, and numerous red macules in the buccal cavity developed later. Some of the macules became 30 mm in diameter, with proliferations of the underlying tissues. By the 35th day hyperkeratosis of the skin had developed on the sides of the neck, across the withers, and around the mammary gland. The skin was dry, hard, stiff, and thrown up in rolls, which later developed fissures (Fig. 1).

FIG. 1. Note rolls of skin with fissures on side of neck of animal No. 766.