The X Disease of Peach and its Chemotherapy

Ernest M. Stoddard

CONNECTICUT AGRICULTURAL
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THE X DISEASE OF PEACH

AND

ITS CHEMOTHERAPY

E. M. STODDARD[1]

The X disease of peach is a major peach disease in many sections of the northeastern United States and is potentially serious in many other peach-growing sections in the northern half of the country. If one assumes that the apparently identical disease of peach found in western and far western states is the same as that occurring in the East, X disease has spread or its existence has been recognized from coast to coast in a little more than a decade. This bulletin will treat the X disease as it occurs in the eastern United States giving the observations made and results of experimental work done on the disease at this Station since its discovery.

The first known occurrence of this disease was in Connecticut in 1933, where it was determined to be a virus disease of peach and chokecherry and was described in 1938 (23) as a new disease under the name of X disease. (It is frankly admitted that this choice of name was not based on a highly scientific consideration of virus disease nomenclature but it does have the advantage of being easily spoken and remembered and probably is as valid a choice as phony disease of peach, Y disease of potatoes or several others that could be mentioned.) Other common names such as yellow-red virosis, yellow red-disease, Eastern X disease, X-virus disease and Eastern yellow-red virosis have been used synonomously (1,11,12,14,18,21). The descriptive scientific name of *Marmor lacerans* Holmes (16) has been given to X disease.

Later observations of tree condition following known occurrence of first symptoms led to the conclusion that this trouble was present in Connecticut for four or five years previous to its recognition in 1933. The records of spread subsequent to this date indicate that the original center of infection was probably located in Connecticut, although infected peach and chokecherry were quite general in southern Massachusetts as early as 1934. Be that as it may, X disease is now a peach disease of major importance in Connecticut, Massachusetts and New York. It occurs in Michigan (5), Pennsylvania and Ontario, Canada, but at this writing it is not considered of major importance in the latter locations. Infected chokecherry has been reported from Connecticut, Illinois (1), Indiana, Maine, Massachusetts, Michigan (5), New Hampshire, New York, Ontario, Canada, Pennsylvania (29), Rhode Island, Vermont and Wisconsin. The range on chokecherry is much wider than on peach because diseased chokecherry has been found in many sections where peaches are not grown.

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As far as is known, X disease occurs in nature only on peach, *Prunus Persica* Batsch.; nectarine, *Prunus Persica* var *nucipersica* Schneid., and chokecherry, *Prunus virginiana* L. All varieties and types of peaches tested or observed are susceptible, although there is some evidence of a variation in degree of susceptibility among the different types of peach.

X disease has been transmitted by budding from peach to the following hosts: western sand cherry, *Prunus Besseyi* (25) Bailey; wild goose plum, *Prunus Hortulana* Bailey; almond, *Prunus communis* Arcang., and Chinese bushcherry, *Prunus japonica* Thunb.[1] *Prunus Americana* Marsh., carries the virus without evident symptoms.[1] Wild black cherry, *Prunus serotina* Ehrh., and beach plum, *Prunus maritima* Marsh., are not found to be infected in nature, have not been infected by budding from peach or chokecherry and are considered immune.

DESCRIPTION OF SYMPTOMS

An outstanding characteristic of X disease is the delayed appearance of foliage symptoms after growth starts following the dormancy period. The foliage of a diseased tree is normal in size and color for seven or eight weeks after growth starts. The flowers are normal, with no deformation or breaking of color. Suddenly, a diffused and blended yellow discoloration appears on the foliage in scattered areas over the tree or perhaps localized on a single branch. Frequently, normal leaves are interspersed between diseased leaves on the affected twigs. The chlorotic leaves often show an upward longitudinal rolling but this characteristic is by no means constant. The discolored areas at first occur in a random pattern on the leaf but ultimately the entire leaf takes on a blended red and yellow color. At this point the leaves become stiff and brittle and the discolored areas usually fall out without necrosis, giving the leaf a characteristic tattered appearance. This character is most pronounced on field grown horticultural varieties of peach and nectarine. Even on these the tattering of the leaves may not always be observed, as the leaves frequently fall off before the abscission of the chlorotic areas. A diagnostic characteristic of X disease is the retention of the tip leaves on the twigs until the end of the growing season.

Trees grown in the greenhouse show marked longitudinal upward rolling of the leaves and a predominately yellow chlorosis with a minimum of tattering. Tree growth is stunted with some shortening of the internodes. These characters are found on seedlings grown either in the greenhouse or in the field.

The purple-leafed types of peach grown either in the field or greenhouse show a marked longitudinal upward rolling of the leaves with a bleaching of the purple color, finally exhibiting a yellow-green and purple coloration. There is very little leaf fall and no tattering of the foliage.

Orchard trees that become infected after three years of age seldom die as a direct result of X disease but trees under three years of age are frequently killed in a short time. However, the older trees soon become unproductive, because the fruit on infected branches dries up and falls off with the dropping of the leaves.

[1] E. M. Hildebrand.

When foliage symptoms appear on a branch late in the season, the fruit does not fall but develops an insipid, slightly bitter flavor as it ripens. The seed is nearly always abortive in such fruit. Healthy parts of an infected tree retain normal foliage and fruit throughout the season. Such healthy appearing branches are, in fact, healthy as the virus cannot be transmitted from them by budding or grafting. There is some die-back each year on infected trees, part of which can be ascribed to a direct effect of the virus and part to winter killing following a weakening of the diseased branches.

X disease affects chokecherry somewhat differently than peach, although there are some symptoms common to both, notably the red and yellow coloration, delayed symptom appearance and abortive fruit. Unlike the peach, the disease has successive symptom stages on the chokecherry and usually involves the entire plant through all the stages. The first evident symptom is a slight yellowing of the foliage with faint touches of red later in the season. The second season the foliage of an infected plant shows a gradually increasing intensity of color, culminating in vivid reds and yellows during August. In the third season the foliage has an intense dark green color with a distinct rosetting at the tips of the branches. These latter symptoms may be evident in the fourth season or a dull brown-red color may be shown with distinctly dwarfed foliage. Through all these stages there is no tattering or falling of the leaves and no blotching of the color as in the peach. Such fruit as may be borne is abortive, with very little flesh and no seed in the stone. Chokecherry usually does not survive beyond the fourth year after infection.

Inasmuch as this discussion is primarily concerned with peach and chokecherry, the symptoms on the other hosts mentioned will not be described in detail. The symptoms on nectarine are identical with those on peach and the other hosts all show some variation of red and yellow color accompanied by variable degrees of dwarfing. However, backbudding on peach from the various hosts produces the usual characteristic symptoms on the peach, and it is evident that the same disease is concerned in all cases.

In view of the variation in symptoms on different hosts, it might be postulated that a virus complex instead of a single virus is concerned and that the several hosts mask some component of the complex, thus causing the variation in symptom expression. The best supporting data for the hypothesis of a complex is the apparent failure to reinfect peach from herbaceous plants originally infected from peach by the use of dodder (17). This may be due to the fact that dodder transmits to the herbaceous plants only certain components of a virus complex which are symptomless on peach. Consequently, the transmission of the virus back to peach from the herbaceous plants by means of dodder results in no symptom expression on the peach and, hence, appears to be unsuccessful. It is conceivable also that the herbaceous host or the dodder might inactivate some component of a complex which is necessary for characteristic symptom expression in peach. This hypothesis should be checked by inoculating an herbaceous plant from a peach which had been back inoculated from an infected herbaceous plant.

TRANSMISSION

Like other stone fruit viruses, X disease can be transmitted artificially only by budding and grafting. It cannot be transmitted by the injection of expressed juice of the leaves or stems directly into the vascular system of the tree, by abrasion of the foliage or by cutting into the bark with a knife. It is presumed that the disease is transmitted in nature by an insect vector, although to date this has not been satisfactorily demonstrated. There is no evidence to show that X disease is ever transmitted from peach to peach under orchard conditions in Connecticut or in any of the areas in the northeastern United States where the disease occurs. It appears to be readily transmitted from chokecherry to peach and from chokecherry to chokecherry in these same areas by the assumed vector.

The virus is readily transmitted from peach to peach by budding or grafting, but transmission from peach to chokecherry or from chokecherry to green-leafed peach by budding is more difficult, and to date has been found impossible by grafting. It is assumed that this difficulty is due to the incompatibility of the two species and consequent poor union of stock and scion. Transmission from chokecherry to purple-leafed peach is readily effected by budding. This is doubtless due to the fact that the chokecherry buds make a more satisfactory union with this variety of peach, as there is no evidence to show that the purple-leafed peach is any more susceptible to X disease than are the green-leafed varieties.

The most successful transmission of the virus is obtained by using inoculum from material showing early stages of symptom expression. From this stage on, there is a gradual decline of virulence down to the point where only a small percentage or even no infection can be obtained. Another requisite for successful inoculation is freshness of the material used. The virus retains its initial virulence in cut twigs for about two days, after which it declines rapidly and at the end of a week it is completely inactive. This loss of virulence occurs long before the buds become incapable of uniting with the stock.

It has not been possible to transmit X disease from peach to peach by budding after the trees have stopped growing in the fall or before foliage symptoms appear in the spring. Some successful inoculations by root grafts have been made on trees in the greenhouse during the winter, using root pieces taken from diseased trees growing outdoors. This method has not been uniformly successful, because of failure in many cases to get satisfactory graft unions. The fact that transmission cannot be effected from the above ground parts of the tree during the winter, and can be effected from the roots leads to the conclusion that the virus of X disease is absent or inactive in the top of the tree during that time. Inasmuch as low temperatures down to the death point of the plant tissue do not inactivate the virus, it would seem that the absence of the virus in the top of the tree may be explained by one of two hypotheses: either changes in the physiological processes of the tree inactivate the virus in all parts of the tree except the roots or all of the virus is translocated from the tops into the roots in late summer and autumn. It can be postulated that either the virus is reacti-

vated in the top by renewal of growth, or is translocated from the roots to the top when growth starts in the spring. Inasmuch as the same branch may not show symptoms in successive seasons, it would be more logical to assume that the virus is translocated from the roots to the top each season rather than that it is reactivated. These hypotheses could explain the fact that diseased trees make a normal healthy growth during the early part of the season and show symptoms after six or eight weeks of growth.

The matter of time between inoculation and symptom expression has not been studied particularly, although records of this interval have been kept in most of our experimental work. From these records it appears that symptoms may show any time from three weeks to two years after inoculation. It should be said that the two year period is a rarity with artificial inoculations. Rapidly growing trees, inoculated in early summer with fresh inoculum, will usually show definite symptoms in from three to six weeks. Cutting the top back to the bud within a week or 10 days after budding will sometimes hasten the appearance of symptoms. There is a considerable variation in the time interval between inoculation and symptom appearance among trees and, even under the above mentioned optimum conditions, it is not unusual to find some trees symptomless until they have gone through a period of dormancy. Budding in late summer, even though all other conditions are optimum, will usually result in delay of symptom expression until the following year.

In the orchard the time interval between inoculation and evident symptoms is more difficult to determine, because the time of inoculation is uncertain. Many individual tree records, covering a period of four years from time of setting, have been made on orchard trees adjacent to diseased chokecherry, and in these records there are no cases of trees showing X disease during the first year in the orchard. During the second year these same trees showed from 10 to 60 per cent infection, depending on the amount of diseased chokecherry and the distance of it from the peaches. The records of orchards from which diseased chokecherry had been eradicated show that when the chokecherry was eradicated before July 1 very little infection took place but, when the eradication was delayed until middle or late July, plenty of infection resulted. This suggests that the earliest symptoms of the disease do not show for a period of perhaps 11 months following natural inoculation, if we assume that inoculation takes place between June 15 and July 15.

Budding is the easiest and most satisfactory method of artificial inoculation and it has been used in all our experimental work, except in special experiments with root and top grafts. The effectiveness of this method of inoculation is doubtless due to the fact that the bud heals in very quickly, thus preventing inactivation of the virus before it can be transmitted to the inoculated plant. It is not necessary for the bud to grow to produce infection; in fact, it has been found that, if the bud remains alive for a week, it will usually make a successful inoculation. A piece of bark will transmit the virus as effectively as a bud, although the appearance of symptoms will generally be later than with a bud.

As a matter of some practical importance in the technique of budding, it may be said that the use of rubber budding strips for tying the buds will give a higher percentage of successful inoculations than will the use of raffia. Presumably, this is because the rubber strips hold the bark tighter around the bud, thus preventing exposure to the air and consequent inactivation of the virus.

It is assumed that an insect vector is responsible for the natural spread of X disease, but to date we have not been able to demonstrate this method of transmission. Approximately 1,000 attempts have been made to transmit the disease by insects of many species common on peach and chokecherry, all of which gave negative results. The insects used were various species of leaf hoppers, tree hoppers and aphids. Among the leaf hoppers was the peach leaf hopper, *Macropsis trimaculata* (Fitch), which has been shown to be a vector of peach yellows.

In this search for a vector the insects were handled in various ways. In one case the insects were allowed to feed several days on diseased plants, from which they were transferred to healthy plants. Another method allowed the insects a free choice of healthy and diseased plants in the same cage. Placing the individual insect in a small cage clamped to one leaf each from a healthy plant and a diseased plant which were still attached to the respective plants was the most satisfactory way of handling the insects. By this method the insect was compelled to feed on the particular leaves in the cage and the amount of feeding and choice of host plant, if any, could be observed. It was observed that most of the insects fed indiscriminately on both healthy and diseased peach and chokecherry leaves when caged in this manner.

All the trees on which insects had been fed were held in field plots for three years, during which time none showed any symptoms of X disease. In view of the unsatisfactory results and the large amount of time involved in such investigations, this phase of the work has been discontinued for the time being, even though the discovery of the vector would be of considerable scientific interest.

CONTROL

The Effect of Distance on Spread

The fact that X disease does not spread from peach to peach in the East and does spread readily from chokecherry to peach, at once suggests eradication of chokecherry as a simple and logical method of control. It has been demonstrated experimentally and practically in a number of Connecticut orchards that this can be done.

The primary necessity in setting up such a control method is a determination of the effect of distance between chokecherry and peach trees on the chances of infection of the peach trees. The best data on this point were obtained in a nursery block 529 feet long by 403 feet wide with diseased chokecherry at longitudinally opposite ends. There were 193 diseased peach trees in this block in the second year from planting, of which 89.3 per cent were within 100 feet of the diseased chokecherry, 8.1 per cent between 100 and 150 feet and 2.6 per

cent between 150 and 225 feet from the chokecherry. Two hundred and twenty-five feet was the greatest distance from chokecherry that an infected peach tree was found, one tree being found at this point. From these data it is estimated that 250 feet is a reasonably safe distance separating chokecherry from a peach orchard.

It was realized that even a 250-foot strip around an orchard might not be practicable in all cases, so several newly set orchards in an area of heavy infection were selected to determine the amount of control obtained by eradicating the chokecherry only in the fence rows immediately adjacent to the orchard. At the end of four years these blocks showed a range of from 1.3 to 5.9 per cent of infected trees. Similar orchards in the same area with no eradication showed from 17.8 to 40 per cent infection during the same period. This is not perfect control, but it is considered sufficiently good to warrant limited eradication, if it is not possible to do otherwise. However, it is recommended to the grower that chokecherry be destroyed for as great a distance as possible around the orchard site before setting the trees, aiming at 500 feet as the optimum distance.

To be effective, the eradication of chokecherry must be thorough and complete. Cutting off the tops of the bushes or even grubbing them out is not effective, for the reason that even the most careful grubbing will not get all the root pieces and the sprouts from these will be diseased as were the original plants. The use of herbicides is the only sure and effective way to destroy chokecherry and even this method requires more than ordinary care in the application of the materials in order to get satisfactory results. If it is well done, there probably will be no necessity for repeating the operation during the life of the orchard.

Inactivation of the Virus by Heat

In 1941 a study of the effect of heat on the virus of X disease in living tissue showed that this virus could be inactivated in diseased peach buds by heat treatment, without appreciable injury to the buds. In the experiment here being discussed, random lots of 25 diseased peach buds with about an inch of twig were enclosed in cheesecloth bags and immersed in water heated to the desired temperature for the several periods of time. Immediately after treatment the buds were budded into healthy peach seedlings of uniform age and size. The number of buds subsequently failing to produce disease in the healthy seedlings was taken as the measure of inactivation of the virus. Twenty-five buds from the same source were similarly immersed in water at room temperature for the maximum period of time. Table 1 gives details of the heat experiments.

These treatments caused no consistent reduction in the viability of the buds up to and including 53°C for two minutes, but the percentage of transmission was reduced considerably at all temperatures with sufficient exposure.

Using the same technique and criterion of inactivation of the virus, a similar lot of diseased buds was subjected to low temperatures, ranging from 5°C to −10°C, for a varying number of hours. At these temperatures there was no inactivation of the virus except at the lowest temperature, which killed the buds also.

TABLE 1. EFFECT OF HEAT ON X DISEASE VIRUS

Time of treatment in minutes	Temperature in degrees Centigrade							
	45.5		46.5		48.5		53.0	
	No. live buds	Per cent diseased trees	No. live buds	Per cent diseased trees	No. live buds	Per cent diseased trees	No. live buds	Per cent diseased trees
2					19	100	15	0
4	21	100	22	100	14	78.6	8	0
8	19	100	24	96	21	33.3	0	0
16	23	65.5	18	33.3	15	0	0	0
32	18	50.0	16	6.25	13	0	0	0
64	11	18.2	19	5.27				

Checks 64 minutes room temperature.
No. live buds 21
Per cent diseased trees 95.2

Chemotherapy

Since early in our investigations of X disease, efforts have been directed toward inactivating the virus in the living plant tissue with chemicals. The virus of X disease seemed to offer good possibilities of chemical inactivation, as it appears to be unstable. Its instability is postulated on the fact that, unlike the tobacco mosaic virus, the X disease virus becomes inactive in expressed juice and decoctions of infected plant tissue and even in plant tissue itself, if such tissue be removed from the living plant for only a short time. It was assumed that chemical inactivation would result from the chemical acting either directly on the virus itself or on the system by which the virus is multiplied.

Many of the earlier experiments were unsuccessful, because of either faulty techniques or unfortunate choice of materials. However, these exploratory experiments served to eliminate chemicals that were toxic to the plant at any concentration, to discover usable concentrations of others, and perhaps most important of all, to establish the necessity of devising techniques by which complete distribution of the chemical in the plant tissue could be obtained.

The first successful experiment (Table 2), made in 1941 (26), was one in which diseased buds were soaked in aqueous solutions of chemicals and budded immediately into healthy peach seedlings. In this experiment randomized lots of ten buds from diseased twigs were soaked for one hour in a solution of the chemical being tested. A lot of 25 buds was soaked in tap water for one hour as checks. The buds were attached as they grew to a piece of twig about 1½ inches long, the entire piece with the attached bud being soaked. After treatment, each lot was rinsed in distilled water. The buds were then cut from the sticks in the usual manner for shield budding and set immediately into a random lot of healthy peach seedlings in the field.

The data show that some of the chemicals, notably quinhydrone, 8-hydroxyquinoline sulfate, hydroquinone, *p*-nitrophenol and calcium 8-hydroxyquinolate inactivated the virus in many of the diseased buds with the consequent failure to transmit the disease. The untreated buds and those treated with some of the chemicals failed to show inactivation of the virus as indicated by the ability of these buds to transmit the disease.

These data also show that the more effective chemicals, such as quinhydrone, the 8-hydroxyquinoline derivatives, resorcinol and *p*-nitrophenol all contain -OH groups. Benzoic acid contains -COOH. The significance of the -OH or -COOH is not known.

The action of 8-hydroxyquinoline sulfate is unique in that it produced a temporary inactivation of the virus. After the trees had gone through a period of winter dormancy, they all showed the symptoms of X disease on subsequent new growth. This temporary effect of 8-hydroxyquinoline sulfate has been noted when this chemical has been used as a chemotherapeutic agent for the control of Dutch elm disease and the Verticillium wilt of eggplant (28).

It seems logical to assume that the inactivation of the virus was due to the action of the chemical and not to a reduction of the concentration of the virus by the soaking, inasmuch as the untreated buds and the buds treated with ineffective chemicals were soaked for an equal length of time. It would further appear that the inactivation of the virus was due to the direct action of the chemical on the virus and not to any effect on the susceptibility of the budded plant, as the amount of chemical carried in the bud and adjacent tissue would seem inadequate to affect the entire plant.

TABLE 2. CHEMICAL TREATMENT OF DISEASED PEACH BUDS

Material	Per cent concentration	Per cent transmission[1]	Per cent living buds
Quinhydrone	.05	20	100
8-hydroxyquinoline sulfate	.1	40[2]	100
Hydroquinone	.05	50	90
p-nitrophenol	.05	50	100
Calcium 8-hydroxyquinolate	sat.	60	100
Urea	.1	100	100
Urea	.2	70	100
Urea	1.0	70	100
Thiourea	.05	70	100
Benzoic acid	.05	70	100
8-hydroxyquinoline benzoate	.1	75	80
Resorcinol	.2	77.7	90
Auramine	.05	80	100
Hg 8-hydroxyquinolate	sat.	80	100
Sodium thiosulfate	.1	88.8	90
Phenol salicylate	sat.	90	100
p-aminobenzene sulfonamide	.1	100	100
Cinchonine hydrochloride	.05	100	100
Quinine bisulfate	.1	100	100
Quinine salicylate	sat.	100	100
Tap water Check 25 buds	.....	96	100

Buds treated and trees budded August 1, 1941.
Data taken October 8, 1941[2]

[1] Per cent of live buds transmitting disease
[2] October, 1942. This lot showed 100 per cent infection. No change in any of the other lots.

The fact that the virus of X disease could be inactivated *in vivo* by chemicals, suggested that a satisfactory distribution of suitable chemicals in a tree would inactivate the virus in an infected tree as well as in an infected bud (26). Pre-

vious work had shown that injections of chemicals into the trunk or branches of peach trees gave a very poor distribution of the chemical in the tree. This defect in technique prompted the use of small seedlings and a method of injection by which chemicals in solution could be distributed through the entire plant.

This was accomplished by using peach seedlings, ¼ inch in diameter, grown in sand culture in the greenhouse and injected by cutting off the top of the main stem and attaching a container of solution to the cut end with rubber tubing. The containers used were 10 inch lengths of one inch glass tubing, which were supported above the tree on plant stakes to which they were attached by two rubber bands for easy height adjustment. By this method it was found that solutions moved readily down the stem of the tree and were evenly and completely distributed in the stem and leaves. The distribution of the solutions could be quite accurately determined with most of the chemicals by characteristic plant reactions. For example, most of the sulfa compounds caused a yellow chlorosis of the immature foliage and forced the dormant buds into growth with dwarfed string-leaf foliage and maltose and dextrose caused white necrotic leaf margins. Besides determining the extent of distribution, it was possible to measure accurately the amount of material absorbed by the tree and the time required for absorption.

Peach trees take up solutions readily for a period of five or six days, after which the absorption rate falls off rapidly and ceases entirely in nine or 10 days. The trees do not regain the ability to absorb solutions for some time thereafter. Injury to the conductive tissue is the quick answer to this but it does not seem adequate as distilled water produces the same effect as does a chemical solution. This same curve of absorption rate has been noted in detached pieces of woody stems of various species with both chemical solutions and water and it would appear to be a function of woody tissue.

The trees took up much larger amounts of some chemicals than others and individual trees in the same lot varied somewhat in the amounts absorbed. There seemed to be no relation between either of these conditions and inactivation of the X disease virus.

Maltose and dextrose were taken up in the least amounts of any material and the addition of either of these to other chemicals reduced the intake of the combined solution. The addition of maltose or dextrose did not reduce the effectiveness of some of the chemicals, even though the total intake was reduced. Diseased trees showed a lesser intake than healthy trees, which is probably due to a lower transpiration rate of the diseased trees. The concentration of the solutes had no consistent effect on the amount of solution absorbed.

Injection of chemical solutions through the cut ends of trees is a valuable method of determining the action of the chemicals, but it is at once apparent that such a method could not be practicable for the treatment of larger trees. For this reason the injection method has been discontinued, except for the purpose of testing new materials. Currently the solutions are being put on the soil

TABLE 3. AMOUNTS OF CHEMICAL SOLUTIONS ABSORBED BY PEACH SEEDLINGS IN 10 DAYS BY TOP INJECTION

Chemical	Per cent concentration	Average amount in ml. per tree in 10 days	
		Trees healthy	Trees diseased
p-aminobenzene sulfonamide	.1	36.8	8.6
p-aminobenzene sulfonamide	.05	28.6	20.5
p-aminobenzene sulfonamide	.025	32.4	15.0
p-aminobenzene sulfonamide	.0125	34.2	21.3
p-aminobenzene sulfonamide plus maltose 2%	.1	17.9	
p-aminobenzene sulfonamide plus dextrose 2%		27.7	
Maltose	2.0	19.5	
Dextrose	2.0	16.3	
p-toluene sulfonamide	.1	33.2	
p-toluene sulfonamide	.05	33.0	
p-toluene sulfonamide	.025	42.0	
Sulfaguanidine	.05	33.4	
Sulfaguanidine	.025	42.0	
Sulfathiazole	.05	20.6	
Sulfasuxidine	.0166	88.4	
Sulfathalidine	.0166	94.8	
Sulfamerizine	.0166	79.6	
p-nitrophenol	.4	56.4	
Calcium chloride	.1	22.1	
Sodium chloride	.1	20.0	
Lauryl isoquinolinium bromide	.0166	41.4	
Lauryl isoquinolinium bromide plus dextrose 2%	.0166	33.8	
Hydroquinone	.00625	39.0	26.2
Hydroquinone plus dextrose 2%		25.0	12.6
Distilled water		31.2	

from which they are taken up by the trees through the roots. This method has promise of successful field use and with no apparent loss of effectiveness of materials.

The application of chemicals by both top injection and soil watering has been made before and after inoculation. The interval of time before inoculation has varied from five days to two weeks. The variation in time interval after inoculation has been much greater, extending from one week to 40 days, although most of the applications have been at one and two week intervals. As will be shown in Table 4, a few chemicals are more effective when applied before inoculation but most of them give better control when applied after inoculation. The effect of time of application in relation to time of inoculation has been investigated but at this writing the optimum interval has not been determined. It would appear that treatments after inoculation can be made any time before symptoms appear with a good probability of being successful. The time before inoculation probably cannot be extended beyond two weeks successfully.

The effect of single and multiple applications of chemicals to the soil on the inactivation of the virus is being studied and the results to date indicate that there is no very significant difference in effect between the two methods of application.

In order to present an over-all picture of the performance of the several chemicals which have in some degree successfully inactivated the virus of X disease, a summary of the results are given in the following tables. In these tables are summarized the results of the treatments with each chemical at all concentrations and all methods of application. The only distinction made is between treatments applied before and after inoculation as this was the only factor that gave consistently significant differences. All the treatments were made on seedling trees growing in sand in the greenhouse.

TABLE 4. INTERNAL CHEMICAL TREATMENT OF PEACH TREES FOR THE INACTIVATION OF THE X DISEASE VIRUS

Chemical	Time of treatment[1]	No. trees	Per cent control[2]	Reinoculated[3] No. trees	Reinoculated[3] Per cent control
p-aminobenzene sulfonamide[4]	Before	34	44	33	51.6
	After	25	96	....	....
p-toluene sulfonamide	Before	19	52.5	....	....
	After	16	37.4	....	....
Sulfadiazine	After	7	57	....	....
Sodium sulfadiazine	After	3	100	3	100
Sulfathalidine	After	4	100	4	50
Sulfamerizine	After	5	100	5	60
Sulfasuxidine	After	10	80	3	0
Sodium sulfamerizine	After	5	100	4	100
Sulfaguanidine	After	10	80	8	87.5
Zinc sulfate	Before	20	80		
	After	37	48	14	71.3
8-Hydroxyquinoline sulfate	Before	5	80	....	....
	After	22	73	....	....
8-Hydroxyquinoline benzoate	After	10	80	7	55
Disodium ethylene bisdithiocarbamate	Before	15	100	6	66.6
Maltose	Before	10	0	....	....
	After	15	66.6	7	29
Dextrose	Before	10	10	....	....
	After	15	40	....	....
Hydroquinone	Before	51	74.5	32	78
Lauryl isoquinolinium bromide	Before	16	56.2	17	58.8
	After	9	71.8	....	....
Check, average all checks for above treatments		100	7.5	25	2.5

[1] Time in relation to time of inoculation.
[2] Data taken 9-10 months after inoculation.
[3] Healthy trees after first inoculation and treatment reinoculated 10 months later with no further treatment.
[4] Treatment by injection only.

Concentrations of chemicals used in treatments shown in Table 4 follow:

p-aminobenzene sulfonamide	.1—.05—.025—.0125% optimum .05%
p-toluene sulfonamide	.1—.05—.025—.0125%
All other sulfa compounds	.0166%
Zinc sulfate	.1—.05—.025—.0125—.00625% mostly at .025%
8-hydroxyquinoline sulfate	.025—.0166%
8-hydroxyquinoline benzoate	.025%
Disodium ethylene bisdithiocarbamate	.05%
Maltose	2%
Dextrose	2%
Hydroquinone	.0125—.00625—.003125%
Lauryl isoquinolinium bromide	.0166%

Where there are data for comparison, it will be noted that most of the chemicals gave better control when applied after inoculation. The chemicals giving better control when applied before inoculation are zinc sulfate, *p*-toluene sulfonamide and disodium ethylene bisdithiocarbamate. The reason for this difference in action is not clear, but it may be postulated that, in the case of the chemicals that were effective after inoculation, the action was directly on the virus after it was present in the plant. In the case of the chemicals effective before inoculation, it would appear that the action may have been on the plant, thereby producing immunity to infection by the virus.

Buds from the trees not showing symptoms at 10 months after treatment were set in healthy seedlings and uniformly failed to produce infection. This would seem to preclude the possibility that the apparently healthy trees were carrying active virus without symptom expression.

The trees remaining healthy at the end of a 10-month period were budded with diseased buds without further treatment. The results of this reinoculation are given under the title "Reinoculation" in Tables 4 & 5. The trees treated with sodium sulfadiazine, sodium sulfamerizine, sulfaguanidine, zinc sulfate, hydroquinone and disodium ethylene bisdithiocarbamate showed considerable immunity 10 months after treatment. The remaining chemicals all increased resistance to infection in varying degrees. It is not known how much longer the trees will be immune to infection as the experiments to determine this point have not been completed.

In Table 5 are shown the results of some treatments of special interest which are not shown in Table 4. In this table are shown the effect of the addition of dextrose to 8-hydroxyquinoline sulfate, *p*-aminobenzoic acid to *p*-aminobenzene sulfonamide and of maltose and dextrose to *p*-aminobenzene sulfonamide. The increase in effectiveness of calcium chloride by increasing the concentration and the results of treatments with extracts of wild black cherry, *Prunus serotina,* are also shown.

TABLE 5. ADDITIONAL CHEMICAL TREATMENTS

Chemical	Time of treatment	No. trees	Per cent control	Reinoculated	
				No. trees	Per cent control
8-hydroxyquinoline sulfate .025%	Before	9	89	....	....
8-hydroxyquinoline sulfate .025% plus dextrose 2%	Before	8	14.3	....	....
Prunus serotina					
Water extract	Before	14	43	11	72.7
Alcohol extract	Before	9	67	....	....
p-aminobenzoic acid .1%	After	7	14.3	....	....
p-aminobenzoic acid .1% plus *p*-aminobenzene sulfonamide .1%	After	10	20	....	....
p-aminobenzene sulfonamide .1%	After	10	100	....	....
p-aminobenzene sulfonamide .05%	After	15	80	21	57
plus maltose 2%	Before	19	74	....	....
p-aminobenzene sulfonamide .05%	Before	10	40	....	....
plus dextrose 2%	After	15	80	....	....
Calcium chloride .1%	After	20	25	....	....
Calcium chloride .2%	After	10	100	....	....
Checks, average all checks for above treatment		25	10	25	2.5

The addition of dextrose to 8-hydroxyquinoline sulfate caused a very considerable reduction in control as compared to treatments of 8-hydroxyquinoline sulfate without dextrose. This action appears to be similar to the action of *p*-aminobenzoic acid on *p*-aminobenzene sulfonamide. The effect is not due to a decreasing of the intake of the solution.

Wild black cherry, *Prunus serotina,* does not in nature or by artificial inoculation become infected with X disease and is considered immune. It was thought that this plant might contain some substance which induced this immunity and, accordingly, peach trees were treated with extracts of the leaves and twigs with the results shown in Table 5. The extracts were made by grinding 25 grams of leaves and stems in a mortar with 100 ml. of distilled water or 95 per cent alcohol. The water extract was injected into the trees without dilution. The alcohol extract was diluted 1 to 500 with distilled water and injected in like manner.

The results of the treatments with the extracts suggest that there is an extractable fraction in wild black cherry which will inactivate the virus of X disease.

It is known that *p*-aminobenzoic acid antidotes the effect of *p*-aminobenzene sulfonamide on bacteria in animals and it was found that it had the same effect on the action of *p*-aminobenzene sulfonamide on the virus of X disease. In Table 5 it will be seen that the addition of *p*-aminobenzoic acid reduces the effectiveness of *p*-aminobenzene sulfonamide very considerably besides having very little effect itself.

p-Aminobenzene sulfonamide causes a chlorotic string-leaf growth of peach leaves. It was found that the addition of maltose or dextrose, particularly maltose, reduced this effect to a considerable degree without antidoting the function of inactivating the virus. In fact with the addition of maltose there was not the expected difference between the "before" and "after" treatments. It was at first thought that the maltose or dextrose was an antidote for the toxic action of the sulfa compound. However, in the light of more complete data it would appear that the addition of these materials may have acted by reducing the amount of *p*-aminobenzene sulfonamide taken up by the tree (see Table 3) and, by their own action on the virus, prevented a reduction of control, which might have been expected from the lesser amount of the sulfa compound taken up by the tree.

The most striking instance of the effect of different concentrations of a chemical is found with calcium chloride. It will be seen in Table 5 that increasing the concentration from .1 to .2 per cent increased the control from 25 to 100 per cent.

SUMMARY

1. X disease was first recognized on peach in Connecticut in 1933 and is now a major peach disease in the northeastern United States. The same or very similar disease is recognized on peaches and chokecherry throughout the northern half of the country to the Pacific coast.

2. It has been shown to be a virus disease of peach and chokecherry, *P. virginiana,* spreading in nature from the chokecherry to peach and chokecherry but not from peach to peach in the northeastern United States. Also, there is no evidence that X disease spreads naturally from peach to chokecherry.

3. The disease can be transmitted artificially by budding or grafting but not by mechanical introduction of expressed juice or decoctions of diseased tissue. It is transmitted readily from peach to peach and from chokecherry to purple leaf peach by budding or grafting. Transmission from chokecherry to green leaf peach or from green leaf peach to chokecherry is more difficult by these methods.

4. An insect vector is postulated but, to date, all attempts to determine such a vector in Connecticut have failed.

5. Peach orchards can be protected from incidence of X disease by the simple expedient of eradicating all chokecherry growing in the vicinity of the orchard.

6. The peach virus causing X disease can be inactivated in diseased buds by heat without injury to the plant tissue.

7. Inactivation of the virus in diseased living peach buds has been accomplished by soaking such buds in aqueous solutions of various chemicals.

8. Living peach trees can be immunized against artificial inoculations of X disease by injections of or watering with aqueous solutions of various chemicals. The results on this work have been obtained on potted trees in the greenhouse. Field trials are in progress but no data are yet available.

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