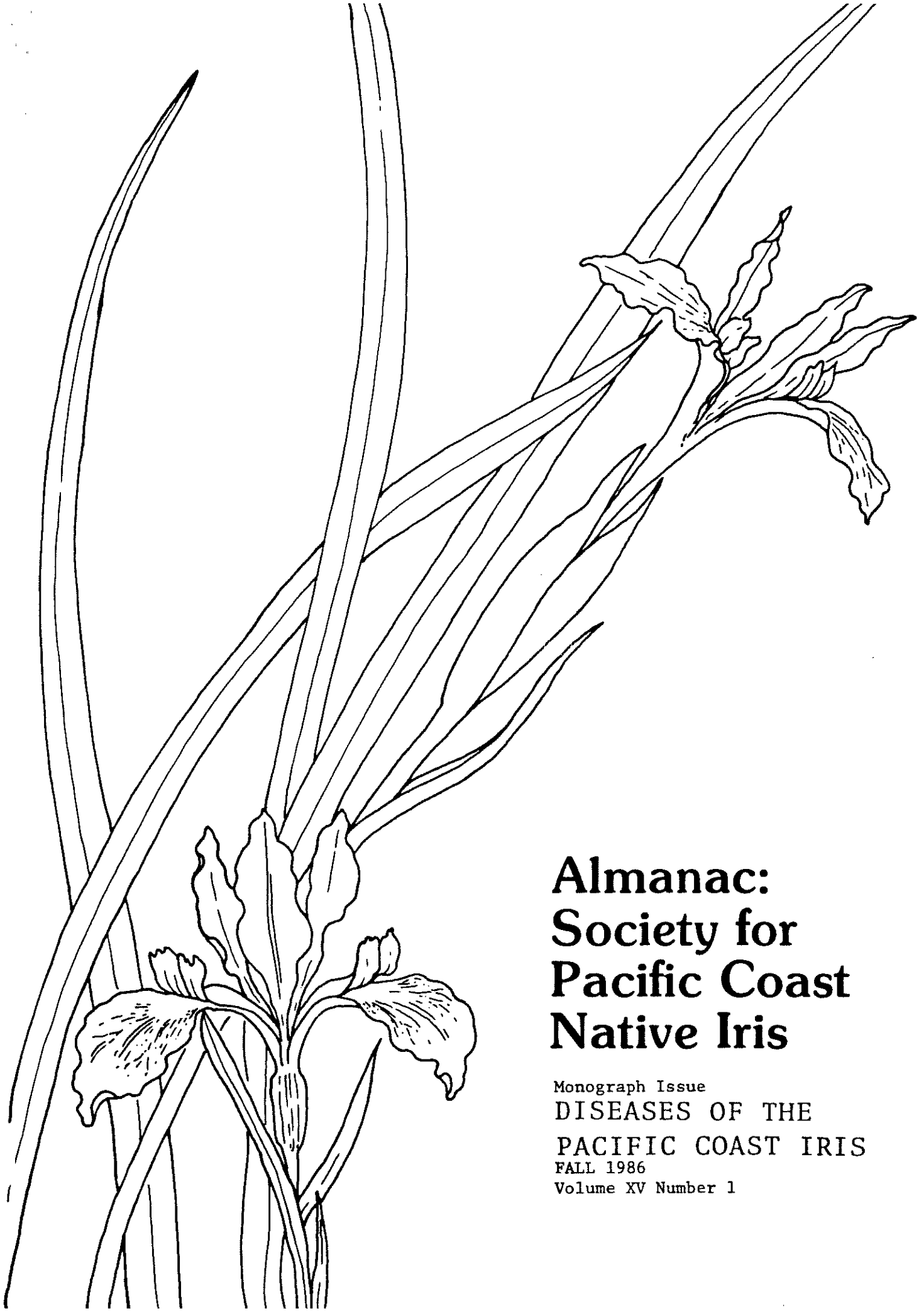

Almanac: Society for Pacific Coast Native Iris

Monograph Issue

DISEASES OF THE PACIFIC COAST IRIS

FALL 1986
Volume XV Number 1

Cover: Diana Gregory

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PUBLICATIONS AVAILABLE

Seed Planting

Almanac, Volume VII, Number 1 (Fall 1980) contains several valuable articles on raising Pacific Coast native Irises from seed. Copies are available from the Editor for $2.00 each, postage paid.

A Guide to Pacific Coast Irises, Victor A. Cohen; forward by E.B. Anderson. London: The British Iris Society, 1967. This 40-page booklet contains both colored and black-and-white photographs of selected species, line drawings and thumbnail descriptions of all species and major subspecies. There is general material on distribution and botanical affinities among the species, plus a map of western states showing distributions of the species in general. Copies are available from the Treasurer for $3.50 each, postage paid.

MEMBERSHIP SUBSCRIPTIONS

The *Almanac* is published in the spring and fall; copy deadlines are February 1 and August 1, respectively. For information about availability of back issues, please address the Editor.

Membership Rate	Individual	Family
Annual	$4.00	$5.00
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The Society for Pacific Coast Native Iris is a section of the American Iris Society; membership in the latter organization is a prerequisite for *membership* in the SPCNI. If you wish only to receive the *Almanac* (two issues per year), the annual subscription is $4.00.

PRESIDENT'S MESSAGE

Hi!

Baffled? Bewildered? Confused?

As you read earlier in a newsletter, we just don't seem to be able to make six and four equal seven.

Honestly though, we are trying, and if you get this Fall issue before you get the Spring issue, don't fret, there must be a reason behind it.

Polling the Board, we agreed you were entitled to every Almanac you'd paid for, and you'll get it even if I have to do it myself. (Heaven forbid!)

With the tremendous help of the Lawyers, we hopefully will get caught up to date, and you will eventually get the Spring 1986 issue, as it contains some informative articles you will appreciate.

As of this writing, I've been unable to get anyone to serve on a nominating committee and my term is up in July. Therefore, its going to be difficult getting a list of new officers published 30 days prior to that time. Will just keep trying and see what materializes.

'Bye, now! I can only hope you all enjoyed the happiest of holidays and that 1987 will be a banner year for you.

Help us make our society the best it can be, and thanks for your forebearance.

Duane Meek

FROM THE EDITORS

As in all of life, some effort must be expended to maintain its most fulfilling aspects; and this pertains to our efforts in the garden as well as in the pursuit of other forms of happiness.

When a disease threatens the survival of a garden treasure it stimulates a call to arms in defense of something we feel is worth fighting for.

Although, by and large, Pacific Coast Native Iris are a remarkably healthy lot, this issue of the Almanac is concerned with the dark side of growing PCNs, - the diseases which may plague them. And as with the federal budget for defense, (which some claim is never enough), research funds at universities are inadequate to finance a comprehensive research program. For that reason we have embarked on a study of the disease problem in our garden in the hope that, together with studies conducted by the University of California and input from other sources, conclusions beneficial to everyone growing PCNs will result.

In this issue we cover observations on the occurrence of two diseases in our garden, PCN Crown Rot, and rust. We cover our attempts to isolate and identify the causal pathogens in Crown Rot. Dr. Robert Raabe discusses the water molds which may be involved in Crown Rot and the fungicides available for their control. The final section deals with miscellaneous tests relating to PCN problems.

Although these studies are far from complete, we feel that some segments of the problems have been made clear and this groundwork will form a base for further studies.

HELP!

For the past three years we have been compiling data on the height of the bloomstalk and the number of blooms per stalk on the PCN cultivars which we grow. We have found that both measurements are quite consistent when comparing data taken on different years, or from different areas in the garden. So far, however, the "height" measurements we have obtained, don't compare very well with those included in the Check List. We are assuming that "height" means "height of bloomstalks", but maybe it doesn't. We have never seen information published relative to the number of blooms per stalk, but to us this is an interesting statistic

which could be included in the Check List.

We would greatly appreciate it if some of you readers could send us similar data from your own gardens so that we can determine the variability of such measurements. If this is done, however, we will all have to be consistent in the way we take the measurements. We take ours after the plant has finished blooming when we are cutting out the old stalks to prevent unwanted seed dispersal. Height is measured in inches from the ground to the tip of the highest withered bloom. We figure this would be about the center of the top flower if it were still in bloom. If there is only one bloomstalk, there is no problem. On old clumps with several stalks we average the tallest 3 to 5 stalks.

On many cultivars, we have found that the number of blooms per stalk is quite consistent, especially on cultivars having only 1 or 2 blooms per stalk. BANBURY TAPESTRY and FAIRY CHIMES, for example, have been consistently single-flowered. ENCIRCLE has been consistently double-flowered. CALIFORNIA NATIVE, which is usually triple-flowered, has occasionally branched and produced up to 7 flowers. When the bloom count is variable, we record 2,3 or 3,4. If it is widely variable, we would put 3-7; but were it mostly triple-flowered with an occasional branch that made it 6 or 7-flowered, we would record it as 3(7).

If you are able to send us some data of this sort, we would appreciate it if you would use a form similar to the one below.

CULTIVAR NAME	HEIGHT	# BLOOMS
Ami Royale	5	1
Augie	14	2(3)
El Centro	18	2-7
Pasatiempo	12	2,3

Lewis and Adele Lawyer

DISEASES OF THE PACIFIC COAST IRIS

PART 1, IN THE GARDEN

Lewis Lawyer

There are three diseases of the Pacific Coast Iris which occur in our garden. The first is caused by *Sclerotium rolfsii* which is of relatively little importance here, having been sighted only once, but which could be a major problem under the right conditions. The second, we are calling "PCN Crown Rot", which is more or less important wherever PCNs are grown. The third, iris rust, is important or not, depending largely on the climatic conditions where they are grown and the degree of resistance or susceptibility of the particular cultivars in which the grower is interested.

Few people who have grown Pacific Coast Irises for a period of years have escaped PCN Crown Rot and the sad experience of watching a well-established clump of some prized cultivar suddenly start to die. Then, as you watch helplessly, the disease spreads across the clump, fan by fan, until the entire plant has departed to some iris heaven in the sky.

Our first planting of natives was made on October 18, 1975 and, largely through the generous help of Joe Ghio, our garden contained over 200 established flowering plants by the time of the National Convention in 1978. True, we lost a few plants at planting time but, once established, they grew like mad.

Despite the fact that from that time on we grew between 150 and 200 established plants each year, we didn't experience any trouble or loss of an established clump until the spring of 1983 when a large plant of NATIVE MUSIC, planted in 1975, started to die. The disease spread rapidly through the main clump and by June it was too far gone to bloom. A division of the original clump which had been transplanted to another location a year earlier, also died.

The sudden death of established plants of the PCNs has been fairly well accepted as a natural phenomenon of the species. I remember discussing the problem with Lee Lenz during a telephone conversation

a couple of years ago. He confirmed that they had lost a few plants in the Botanic Garden at Claremont from time to time, but had not made any attempt to identify the cause since the occurrence was so sporadic. Then he went on to say that the Pacific Coast iris are such prodigious seed producers in their natural habitat that the loss of some of the older plants would be of minor consequence to their survival as a species. I remember thinking that this was an astute observation and suddenly realizing that it was only man's involvement with the plants that gave any importance to the disease. It is only after man has developed and selected a specific clone which he wants to perpetuate, that death becomes a serious problem. We will discuss two diseases which are involved: "Mustard Seed Disease" caused by the fungus *Sclerotium rolfsii*, and a more serious problem which, because of its nature, we are calling "PCN Crown Rot Disease", caused by a pathogen or pathogens not yet fully identified.

In the 12 years that the SPCNI Almanac has been published, there have been 3 references to this disease problem, two by Richard Richards of Southern California, and one by John Weiler of the San Joaquin Valley. In Volume 1, number 1, September 1973, Richards says, "Some clones of the *Californicae* appear prone to mysterious ailments not yet understood. They occasionally suffer from some sort of fungus..." Then, in the fall issue, 1981, in an article titled "Hot and Wet", Richards expands on the theory that "the problem" is intensified when you have to irrigate in hot climates such as in Corona, where he lives in Southern California. John Weiler, in the spring issue, 1984, in an article titled,"*Californicae* in the Central Valley", writes: "Still a third factor which may be the most important in success or failure is water in the garden." He goes on to say that despite the fact that these irises grow without apparent water in their native habitats, they will not survive without some water in his area. Nor will they stand excess water. He continues: "Such specific requirements for water during the summer months is known for many other plants.....In some cases, intolerance to summer water may be traced to one or more water molds which multiply rapidly in moist soil during warm weather. Particularly devastating is the mold *Phytophthora...*" He then points out that the fungicide, Subdue, is recommended for the control of such organisms.

Now to get on with our own experiences in our relatively cool-weather climate in the Oakland hills of the San Francisco Bay Area. In the fall of 1976 the cultivar. FICUS, which had been purchased from Cordon Bleu a few months earlier, suddenly turned brown. There were no visual symptoms except for a brown rot where the leaves joined the crown, but when we placed the plant in a moist chamber for a week, *Sclerotium rolfsii* (mustard seed fungus) developed. This is the only PCN plant in our garden which to my knowledge has ever developed *Sclerotium rolfsii*, but Joe Ghio and others have experienced some trouble with this fungus.

It is important to note that when we dug the diseased FICUS plant there were no visable symptoms of *Sclerotium rolfsii*. The typical symptoms of "mustard-seed"-like sclerotia which are so prominent on a diseased tall bearded iris plant were absent, and it was not until we had placed the plant in a moist chamber for several days that sclerotia developed. This was also true of a diseased plant which we received from Joe Ghio for diagnosis some years later. Adele was able to identify the fungus microscopically, but there were no sclerotia on the plant, even after several days in the moist chamber. We took the plant, moist chamber and all, to Dr. Raabe at the University at Berkeley for confirmation of Adele's microscopic diagnosis. Two days later Dr. Raabe called us to say that sclerotia were forming on the damp paper towel which we had placed under the plant as the moisture source in the chamber. We have been told by others that they have seen sclerotia forming on diseased PCN plants, but we can testify that they are not always apparent. This brings up the danger of treating for a disease before a reasonably positive identification has been made. The chemicals used in the treatment of *Sclerotium* and *Rhizoctonia* can be quite different from those

used for the control of the water molds, and if used incorrectly, may even intensify the problem.

I think that before we go any farther, I should acquaint you with the various locations in our garden where the PCNs are grown. All the beds in our garden have been given alphabet designations: "A," "B," "C," etc. Our discussion of PCN Crown Rot will start with its occurrence in Bed "Q," a relatively small bed in the west central area of our garden primarily devoted to bulb plants. There are, however, spaces for about 90 lined-out PCN seedlings at the west end of the bed.

We will continue the discussion with its appearance in bed P, about 20 feet down the hill from bed Q. Bed P is the location of our main planting of named varieties, and it was in this bed that the plant of FICUS, previously mentioned, died of *Sclerotium rolfsii*. Plants in bed P are spaced 18 inches by 18 inches, our normal spacing for ALL plantings other than lined-out seedling beds. Bed P is approximately 27 by 10 feet in size, and there are spaces for 120 plants. As with bed Q, above, bed P gets afternoon shade from a row of pine trees in the neighbor's yard to the west. We will be discussing the occurrence of the disease in bed P, how the plants were moved from this bed to a "standby bed" while bed P was being fumigated, and how they have now been moved back.

Just east of bed P, and across a narrow path, is bed "V." Bed V is one of the two beds where we plant our selected hybrids, and there are spaces for 58 such plants. Following our discussion of bed P, we will discuss the introduction and spread of Crown Disease in this bed. The other bed in which selected hybrids are planted is bed "S," immediately north of bed V. It is approximately 10 by 27 feet, and the area devoted to PCNs will accommodate 66 plants. We have had no problems in this area.

Lined-out seedlings are rotated to various garden beds. We will be discussing such plantings in beds "C" and "D". Both of these beds are located about 70 feet up the hill from beds P and V. Bed C is about 24 feet long and varies in width from 8 to 16 feet. Our lined-out seedlings are always spaced 6 inches apart in rows 12 inches apart, and at this spacing there is room for 342 plants in bed C. Bed D is just east of bed C, and at the time the disease was introduced into this bed, there were 205 seedlings lined-out in the west half of the bed.

Our studies relating to the cause, dissemination, and control of PCN Crown Rot Disease began in October, 1981 when 3 seedlings in Bed Q, which had been lined-out in May of that year, began to decline. The leaves turned progressively gray-green, and dark areas developed at the base of the leaves and on the upper crown. Adele, who was at the time Plant Pathologist in the Agricultural Research Department of the Del Monte Corporation and had access to their laboratory facilities at San Leandro, isolated a *Pythium sp.* from 2 of the 3 plants. The fungus, *Pythium*, is a common water mold, similar to the *Phytophthora* mentioned by John Weiler in his 1984 article cited above. *Pythiums* cause a sloughing-off of roots of many plants, and are generally responsible for the "damping-off" disease of very small seedlings. Such an organism could easily be the culprit in the problem. She also isolated *Rhizoctonia*, a fungus which causes a type of dry rot and death to many varieties of plants and which also could be involved.

The area was left untreated, and by the end of the following year 8 more plants had died. In the meantime 3 of the non-diseased seedlings were selected for bloom type. That fall, having still developed no disease symptoms, the 3 plants were transplanted to another area. Despite the fact that no chemical dips were used when transplanting, no problems ever developed with these plants. All the remaining plants were dug and discarded.

Without any intervening treatment, the area was replanted the following May, just to see what would happen. Of the 36 seedlings planted, 9 died the first year, all with the same symptoms as those in the original planting. Since this test, we have repeatedly shown that, once the disease has become established, you will experience nothing but trouble if you replant without treatment to reduce the population of the causal organism(s) in the soil.

I have often pondered about the origin of this particular infestation. As with all my seedling plantings, the plants were rather closely spaced: 6 inches

apart, in rows 12 inches apart. Yet, in the original planting, there was no evidence of spread from plant to plant. All the deaths were sporadic, as if each was a primary infection of its own. I have always thought that this infection came from some lily bulbs which had been purchased from a nursery in Oregon and planted in the area two years earlier. Of the 16 bulbs planted in that area, 11 had wilted and died of some mysterious ailment the first year after planting.

Actually, the real question is not the origin of that particular infestation, but why it has caused us so little trouble in view of the broad distribution of water molds. Water mold organisms are so widely spread throughout the world that it is a wonder we can plant anywhere without getting into trouble. Fortunately, most of the time they maintain a balance with other organisms in the soil and don't build up to the numbers required for invasion of our plants. As we shall see, however, once that critical balance has been surpassed, invasion is just a matter of course.

Progress of the disease in bed P, our main planting of named cultivars, was slow but relentless. Here, even though the plants are spaced much farther apart than they are in the seedling beds, the edges of old established clumps can be quite close and this could well be a factor in the spread of the disease to neighboring plants. In 1981, a plant of CALIFORNIAN which had been obtained the previous fall, died shortly after blooming. I have no other notes on this event and it would have gone unheeded except that the well-established clump of NATIVE MUSIC, mentioned earlier, which was planted adjacent to it, started dying a year later and was completely dead by bloom time, 1983. Both plant remains were then removed, but the 2 spaces were never replanted. There was no further spread from this center through 1985 when all the plants were moved out while the bed was being fumigated.

In April, 1982, a plant of CITIZEN, purchased the year before, started to die. It had not bloomed, but a picture of it taken at the time shows that it had developed 4 fans before it died. Two adjacent plants died the following year, two more a year later, and 3 more were dead or diseased at the time the bed was dug in 1985.

Dying plant in bed P

In 1983, plants of POGONIP and GO WILD, which had been purchased 6 months earlier, died shortly after blooming. Spread from these plants is also apparent, with a total of 7 surrounding deaths by the time the entire bed was dug in 1985.

We also have evidence in this bed, and in two other beds, of spread of the disease by washing rain water downhill from an original infestation. In this case 9 additional deaths resulted from this cause.

Thus there were 4 separate plants in the area on which the disease was primary. From these 4 plants, it had spread to 24 more during the 4-year elapsed time prior to digging the bed. During this 4-year period only 2 of the dead plants were removed, and no chemical treatment was given until the final year when 3 drenches of Subdue were applied at monthly intervals.

By the end of the 1984 bloom season, it had become apparent that we were going to be forced to move the plants out of bed P and fumigate the area. In mid-January, 1985, shortly before we began drenching the area with Subdue, we selected and cut

a start from each of the cultivars we wished to retain, a total of 70 plants. All the small, or dwarf types, 15 in all, were placed together in a permanent area in bed S, where PCNs had not been grown before. There were no deaths following this planting.

There was no large space available for a permanent planting of the larger-sized cultivars, so they were placed in a "standby" bed, where they were lined-out like seedlings, planted 6 inches apart in rows 12 inches apart. Some of the clumps from which the starts were obtained were partially diseased, so every effort was made to select starts which were completely disease free. Each start was thoroughly washed and given a 10 to 20 minute dip in a Subdue solution.

The most diseased clump from which we obtained a plant was that of SOQUEL COVE, in fact we obtained the last live fan on the clump. We carefully washed it, cut away all the diseased roots we could find, trimmed the rhizome, dipped it in a 10 percent chlorox solution for 5 minutes, and then soaked what was left in a Subdue solution for 6 hours. It was planted in an isolated spot in the standby bed, and then drenched again with Subdue. It grew beautifully, never showed a sign of the disease, and eventually furnished us with 3 transplants, all of which are growing normally today.

The clump of COUNCILMAN was also almost dead, but we were fairly certain that we had obtained a clean start. We were apparently wrong about this, however, because in late March the plant began to develop symptoms of the disease in the standby bed. It deteriorated rapidly, and on April 3 it was dug and removed. By chance, it had been planted at the end of a row, so we were able to shield the neighboring plants and treat the small area with Vapam. A couple of weeks later, 2 more plants located in discrete areas in the standby bed, also died, as did the original clumps in bed P from which they had been obtained. Both dead plants were surrounded closely by other plants, precluding the use of Vapam, so following their removal, the 2 areas were drenched with Subdue at 3 monthly intervals.

There were no further deaths in the standby planting, and no further spread from these 3 spots through October 24, 1986, when the entire planting was dug. Successful transplants were obtained from this standby planting in late January, 1986, when the plants were one year old, and again in late October, 1986. No disease has developed in the approximately 200 plants thus obtained.

On March 1, about two weeks after the above plants were taken from bed P, we applied the first of 3 monthly applications of Subdue to the diseased area of the planting. These applications were made solely for the purpose of learning a little more about the effectiveness of Subdue in an old established planting such as this. Applications were made in consultation with Dr. Raabe of the Department of Plant Pathology, U. C., Berkeley, and all the necessary materials were furnished by him. Concentration of the Subdue drench, as it is wherever mentioned was 0.3 ml. per gallon of water, (¼ teaspoon per 4 gallons). Application rate was 1 gallon for every 4 square feet of soil, an amount approximately equivalent to 0.4 inches of rainfall. This required some care to avoid excessive runoff even in our gravelly soil.

We have conflicting evidence regarding the effectiveness of the Subdue drench. On the positive side, there was no visible spread of the disease following the first of the 3 applications and up to the time when the bed was dug 5 months later. Furthermore, the large clump of COUNCILMAN, mentioned earlier, from which we had obtained the start which later died in the standby bed, showed a marked improvement during the course of the 3 applications. The large clump, which was 90 percent dead when the drenches began, showed an increase from 5 fans to double that number after the second drench one month later. Following the third drench, the 10 living fans appeared to be growing normally, and by the time the bed was dug on August 13, five of the fans appeared to be completely free of the disease. These 5 fans were thoroughly washed, given a 1-hour Subdue dip, and planted in 4-inch pots. Four of the 5 have survived a full year, and one, planted out in the garden last January, is growing vigorously today. These

February, 1985
Diseased COUNCILMAN clump in bed P before Subdue drenches

May, 1985
Picture taken from same location as the one above showing partial recovery of COUNCILMAN clump after 3 drenches with Subdue

August, 1985
Roots and crown from a section of the COUNCILMAN plant pictured above before dividing and replanting. Replants have remained healthy, indicating complete recovery. See text on page 8 for details.

successful transplants, made 7 months after the entire clump should have been dead, are certainly a big plus for Subdue.

On the negative side, soil from this area was placed in 4-inch pots into which young seedlings were transplanted. All the seedlings were dead within a short period of time, whereas seedlings transplanted into non-infested soil grew normally. This aspect will be covered in more detail later.

It is interesting to note that the 4 plants listed above as primary sources of the disease, multiplied and grew for a half year or so before the disease was detected. This "incubation period" ties in with a fairly well established principal of plant pathology, that the severity of many soil-borne diseases is directly proportional to the concentration of the causal organism in the surrounding soil. In this case, two explanations are possible: either there was a small amount of the disease present in the soil where the 4 plants were planted, or there was a small amount of the disease on the plants themselves when they were planted. In either case the disease builds up on the growing plant until enough inoculum is present to kill.

You are also reminded that this was before our use of Subdue. A 10-minute dip in a Subdue solution would have eliminated any disease clinging to the roots or crown. We also have substantial experimental evidence that the Subdue dip greatly reduces the chance of its contracting the disease even if it is present in fairly large amounts in the soil before planting.

Now to conclude our experiences with bed P, and to bring you up to date. In late summer, all the remaining plants were dug and the area cultivated. On August 22 the entire bed was drenched with Vapam, sprinkled for 10 minutes, and then immediately covered with a plastic tarp. The tarp was removed after 4 days and the area left untouched for a month. The surface was then raked to aid in the escape of the Vapam fumes. On November 22, that part of the bed where we intended to plant was drenched with Subdue. Because of a combination of weather conditions and our commitment to the Region 14 Bulletin, however, we were unable to start replanting until January 21, 1986. On that date, 40 new starts were taken from the standby bed and planted in bed P. To date there has been no recurrence of the disease in this planting. This fall, 16 more cultivars were added to the planting, and all are growing well. "Eradication" is a BIG word, however, and it is near-impossible that we can escape some future problems in bed P. For the present we will just have to wait and see.

We will now examine the next occurence of the disease which started in bed V, across a path from the diseased area in bed P. This is the bed in which we plant selected hybrid material, and this occurence was the first indication that, unless proper care is taken, the disease is likely to strike, preferentially, your best selections. In this particular case, the second plant to succumb was the plant we had selected the previous year as the "bluest flower we had ever seen". The first to die was the plant immediately in front of it where I had stood while gathering pollen from, or placing pollen on the beautiful flowers of this prize selection. I think this was a simple case of carrying the inoculum across the path on my shoes from the neighboring diseased area in bed P.

A third adjoining plant also died, and that fall (1983) the 3 dead plants and 3 adjoining healthy plants were dug and removed. Without any treatment, the 6 plants were replaced with 6 expendable seedlings to see what would happen. By the spring of 1984, all 3 plants which had been planted in the spots from which the dead plants had been removed, were dead,and by fall the other 3 replants also died. On October 22, 1984 the 6-plant area was treated with Vapam and drenched with Subdue. There have been no further occurrence of the disease in this bed, and pot tests using soil from the treated area have thus far failed to detect any residual disease.

There are four other areas in our yard where the disease was quite obviously spread by shoes to an area where a special seedling was being heavily used in crossing. All four were in closely-spaced seedling beds, one in bed C and 3 in bed D. Spread in these closely-spaced plantings was at a frightening rate, averaging about the same rate per month as the wider

spacing rate in bed P per year!

In bed C, we allowed the disease to spread at will for 4 months without any attempt to stop it by treatment. In this area, left untouched and with no removal of dead plants for the 4-month period, new deaths averaged 2.5 per month. After the 4-month trial, the diseased area plus one row of healthy plants around the area, was drenched with a Subdue solution without removing any of the dead and diseased plants. This treatment continued for 2 months, during which time the death rate was 4 plants per month. This may seem like a step in the wrong direction, and it certainly is no improvement, but you must remember that as the diseased area increases in size,there are many more plants on the periphery to be infected. After these 2 treatments, all dead and diseased plants were removed and the area drenched with a Benlate-Terrachlor-Subdue solution at the standard rate for each of the three fungicides. For the next 3 months following this treatment, and without any further treatment, only 3 new deaths occurred. Then we experienced a heavy rain which washed across the area. Downstream from the 3 dead plants, which had neither been removed nor treated, 12 plants died within a 3-month period.

On May 26, 1986, the entire diseased area was dug, the plants all removed from the premises, and the area was treated with one and a half times the standard dosage of Vapam. Pot tests were run, using soil taken from the area before and after the Vapam treatment. In the pre-treatment soil, all the plants were dead within a month after planting. In the post-treatment soil, none of the plants have died to date.

The rapid spread of the disease by rain washing lends support to our present belief that one or more water mold fungi are involved as pathogens. The spores of these fungi are produced by the millions and are motile in water. With the PCNs,therefore, we have an ideal condition: a host plant which is highly susceptible, the spores of the causal pathogen being produced in great numbers on a nearby diseased plant, and the water to spread them across the surface of the planting.

We want to emphasize that all the severe problems we have experienced with the disease have occurred when we were purposely doing something wrong in an attempt to obtain information about the problem. The fact that we purposely left dead plants in place as inoculum sources, and deliberately replanted in infested soils without taking any precaution, are not recommended agricultural practices. On the bright side, however, we have not yet experienced any lateral spread of the disease where we have immediately removed the plant and treated the area with either Vapam or Subdue.

Another factor contributing to the spread of the disease in our garden may be our automatic sprinkler system. Situated as we are, on a hill, there is no possibility of furrow irrigation. Nor has drip irrigation been satisfactory in our gravel; it just goes down and disappears somewhere. Depending on the amount of overlap of the large "rain bird"-type sprinklers, certain areas in the garden get as little as a half inch and others as much as a full inch of "rain" with each sprinkling. Left to themselves, the sprinklers are activated every 7 days, but unless the weather is extremely hot, we usually delay them manually for up to 10 days.

In no way are we going to give up the convenience of this system which allows us to be away from home for a month at a time, and go back to hand-watering our three-quarter acre property. Nor are we about to give up the other 90 percent of our garden plants which are non-iris and which require the water. In truth, I think the PCNs like the water, too. I don't think I have seen a planting anywhere that grows any better than ours.

Actually, we don't really know how much the sprinkling is effecting the disease; we can only surmise. Except for that one heavy rainfall, over which we had no control, we have seen no evidence of spread by water. We have good evidence that the disease spreads from plant to plant through root contact, however, and the presence of water around the roots should contribute to this type of spread. It is something like cigarette smoking: we haven't absolute proof, but it is reasonable to

think that drying out the plants more during the summer would be beneficial.

Now, to be fair, we will give equal time to the other side of the question. Plants growing wild along the coast and on the coastal side of the inland hills get unbelievable amounts of "drip irrigation" during the foggy summer mornings. I'm not sure how much dew forms on the plants in the Sierra foothills, but there you seldom, if ever, find them growing in the full sun. They are unknown, and evidently can't even survive in nature in the really dry interior valleys and hills. In my case, we are blessed with a second sprinkler irrigation system which covers the periphery of our yard where the azaleas and rhododendrons grow, and this system is set to deliver about a quarter inch of "rain" every single night. One sprinkler inadvertantly covers a small area of the large planting of named varieties in bed P, which we previously discussed. Just by chance, this small area of about 10 cultivars which had received about a quarter inch of "rain" every night for the past 10 years, was the only area in the planting where no disease occured.

Iris rust is the only other disease occurring on PCNs in our garden. Rust is a problem on several Iridaceae, and is caused by a fungus, *Puccinia*, usually *Puccinia iridis*. It is an important disease in many locations where PCNs are grown, especially along the coast and of no importance in most other areas. Degree of susceptibility is genetically controlled; therefore selection for resistance is the most sensible method of control. Natural resistance in PCN species and cultivars varies from plant to plant, ranging from highly susceptible to near-immune. Most plants of the coastal species, which have had centuries of natural selection pressure in an environment favorable to rust, are highly resistant. We have found most *Iris munzii* clones from the dry Sierras where there is no natural selection pressure, to be highly susceptible. We have evidence, however, that this is not always the case.

Our garden is an ideal environment for rust. We live in a relatively cool and moist area, and we overhead sprinkle. When we brought *munzii* pollen home from the Sierras and crossed it to some of our relatively resistant *munzii*-derived material, certain of the resulting lines were so infected with rust that every plant died back to the ground. Other lines, depending on the *munzii* pollen used, were relatively resistant, and the population included individual plants with no trace of rust. Dr. Lenz says he has never seen rust in his dry Rancho Santa Ana plantings, but some of his selections are quite susceptible, while others are resistant. Strangely enough, his SIERRA SAPPHIRE, a pure *munzii* selection, is quite resistant here. We saw rust in Thornton Abell's garden in coastal Santa Monica, yet, despite this selection pressure, some of his part-*munzii* clones which are growing in our garden are as susceptible as anything we have seen. Other Abell selections, however, are near-immune. Joe Ghio says that rust didn't occur in his PCNs the first several years he grew them, but then gradually increased. I have the feeling that this timing coincides too well with his introduction of *munzii* pollen into his breeding program to be ignored. Most of his introductions,however, involve primarilly coastal species, and are highly resistant in our garden.

We are in a quandry! Well over half the PCNs in our garden are *munzii*-derived. Last year, rust was responsible for the weakening and eventual death of one of the Thornton Abell clones in our garden. Other clones, desirable for our breeding, are highly susceptible and become weakened by the disease. Despite all this, we have been reluctant to spray, when one of the primary objectives in our breeding program is selecting for resistance. This year, for the first time, we are selectively spraying with Plantvax. Spraying has not eliminated the rust on highly susceptible plants, but none have died back to the ground the way they did last year. By this selective spraying, we enable the highly susceptible seedlings to grow normally until they flower. At that time we can discard them; but on the other hand we may discover some intensely blue, beautifully-formed flower that exists within the susceptible population. Subsequent crossing can reduce or eliminate the rust and, hopefully, retain the positive features of the plant.

PART 2, LABORATORY RESULTS

Adele Lawyer

As you have learned from the foregoing section, by 1981 we began to realize that growing and hybridizing Pacific Coast Native Iris would include solving a few transplanting problems, and dealing with a couple of diseases. We also knew, however, that PCNs were beautiful and fascinating enough to be worth the extra effort involved in confronting the problems. We therefore set out to improve our cultural practices as well as to clarify the disease problems. In the case of PCN Crown Rot Disease, this meant defining the typical symptoms, finding the causal pathogen or pathogens, and evaluating control methods.

We have been continuously involved in this study with variable intensity since 1981, and with increased intensity since 1984, when I retired from Del Monte Corporation's Agricultural Research Department and came home with two borrowed microscopes, an alchohol burner, and a load of test tubes and petri dishes. The research has been sporadic because it is not our only pursuit. Furthermore, space for pot tests has been limited because we do not wish to infest our garden soil with pathogens, and there is a limit to the space available indoors for tests of this kind. Dr. Robert Raabe of the Department of Plant Pathology, University of California, (U.C.), has been helpful in this regard, offering his time, greenhouse space, and materials to whatever extent necessary. Although, to date, the University tests have been less productive than we would have wished, we appreciate the personal interest Dr. Raabe has taken in our problems. PCNs, understandably, are not on as high an economic plane as are Easter lilies and poinsettias, but he has managed to find space for as many plants as we could spare for tests.

Addressing the first of our goals, that of defining the symptoms, we have found that, in our garden at least, the first indication of PCN Crown Rot Disease is a yellowing of the outer leaves. The color is somewhat yellow-orange, distinct from the tan-into-brown which is normal for maturing outer leaves of PCNs. At this stage and later, when the central leaves start to die and turn a grayish-green, the leaves can be pulled free from the crown at the ground line. They offer no resistance to even a light pull, in contrast to normal dried leaves which pull free with difficulty, if at all. Effected leaves are often black at the base. The surface of the crown may also be black and, when the plant is dug, a limited portion of the roots immediately adjoining the crown are also frequently black to tan. The disease seemingly affects the crown tissues at the surface of the soil preferentially, and separates both leaves and roots from their nutritional sources. The disease organism may enter the crown through a root or from surface contamination, but if the plant is dug and examined before general decay has occured, the bulk of the roots, and even the central tissues of the crown may still be white and turgid. If the plant is dug a little later it is nearly impossible to keep it in one piece, and the roots may fall away completely.

Plant with typical symptoms of PCN Crown Rot, showing how leaves pull away from the diseased crown and how most of the roots are still healthy.

Our first attempt to identify the causal organism(s) was in October, 1981, while I was still working at Del Monte.

As you have read, three seedlings in bed Q had developed symptoms of Crown Rot Disease. From two of these seedlings I isolated a *Pythium*, and from one of the two, a *Rhizoctonia*. The *Pythium* culture was taken to U.C. for identification as to species, but a complete identification was never made. Neither of these two cultures were ever tested for pathogenicity. These were the only cultures made before I retired, although some microscopic examinations disclosed the presence of water-mold organisms in a few other plants examined during that time.

Adele working in her makeshift, dining-room laboratory. Petri-dish cultures in the foreground.

A few months after my retirement, we established a "laboratory" at home, first in our dining room, and later in a spare bedroom where it still exists. In the latter area there is room to grow a few potted plants, set up the microscopes, and store cultures and equipment. Since 1984, many diseased PCNs from our garden have been examined here, and numerous cultures made. For microscopic examination, I take a tiny piece of discolored tissue from the edge of the advancing diseased area for viewing. In this area you can see the thread-like fungus structures called "mycelium", and also the sporophores and other organs which help in identification. To make cultures, I take apparently, still-healthy tissue just beyond the discolored area where the pathogen has not yet appreciably destroyed the cells. Here, the pathogen is more likely to be growing in advance of other contaminating microorganisms which quickly colonize diseased tissue.

The first isolations made in our home laboratory were taken from a couple of diseased plants given to us by Joe Ghio from his garden in Santa Cruz. Our observations of these plants coincide with those made on similar plants from our own garden. In a preliminary microscope examination, water-mold fungi, *Fusarium sp.*, and *Rhizoctonia* Type 1, were observed. All three of these fungi were also isolated. Among the water-molds, a *Phytophthora* and a *Pythium* were isolated.

In August, 1984, three of the above isolates, a *Pythium*, a *Phytophthora*, and the *Rhizoctonia* were taken to Dr. Raabe at the University, and a pathogenicity test, using these three cultures, was planned. At U.C., the three cultures were added to soil mixes in separate bags and left to incubate in the headhouse. In early September we dug and washed the 214 discarded seedlings remaining in our seedling bed, and met Dr. Raabe at the University greenhouses where the inoculated soil mixes were supposed to be waiting. All but one bag had mysteriously disappeared, and the identifying tag had been removed from that bag. There was sufficient soil mix in the remaining bag to fill 60 3-inch clay pots; so we revamped our plans to include the following variables, with 12 plants in each variable. There was one plant per pot, watered-in with tap water unless noted otherwise. All dips were 10 minutes in duration.

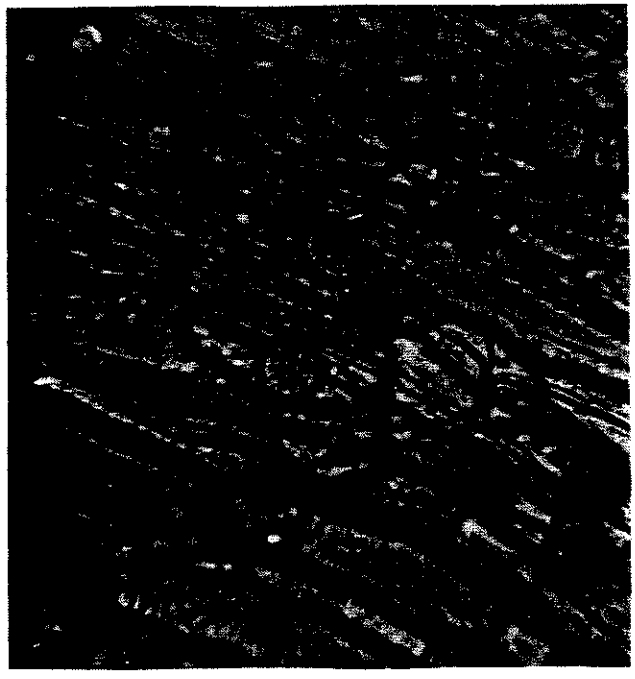
1. Dipped in Subdue before planting
2. Dipped in Subdue plus Benlate before planting
3. Dipped in Subdue plus Benlate and watered-in with Subdue-Benlate drench
4. Not dipped, but watered-in with Subdue-Benlate drench
5. Plants not dipped (untreated check)

The remaining 154 plants were planted without any treatment in separate pots in case the missing soils turned up later.

Whatever the organism was in the bag,

the soil turned out to be non-pathogenic and the only thing learned from the test was that the best growth occured in the pots in treatment 3,the combined Subdue plus Benlate dip and drench.

All other cultures with which we have worked were isolated from diseased plants in our own garden, and I will begin my discussion of these with those from diseased plants in bed P. Although many such cultures were obtained, those of most interest were taken from the plant of COUNCILMAN which had been moved from bed P to the standby bed, where it later developed symptoms of the disease. As mentioned in the first part of this article, this plant of COUNCILMAN had been transplanted from bed P after being dipped in a Clorox solution and soaked in Subdue. In spite of this, however, it became diseased and on April 3, 1985, it was removed from the standby bed for examination. Two types of water molds were seen under the microscope, and an *Aphanomyces*, culture 85-6B, was isolated. ("85" is 1985, "6" is the sixth culture made that year, "B" is the second of two organisms growing out of this sixth piece of tissue.) *Rhizoctonia* type 1, *Fusarium*, *Pesticola*, and several saprophytes were also seen and cultured.



Aphanomyces, 85-6B, growing in the tissues of a PCN plant, 2064 times life size. Circular bodies are sporophores with thousands of motile spores in each.

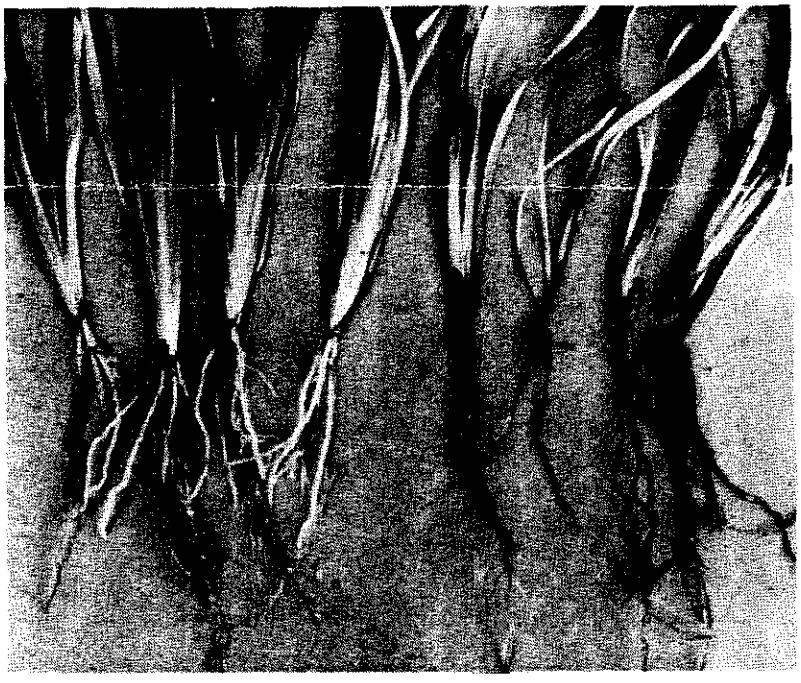
The *Aphanomyces* culture, 85-6B, was tested for pathogenicity in pot tests at our home in July, 1985, along with cultures of the *Fusarium* and *Pesticola*. Cultures were mixed into a clean soil mix (Rod McClellan steam-sterilized "Super Soil"), placed in 4-inch plastic pots, and planted with 4 seedlings in each pot. Plants in the pots containing *Aphanomyces* 85-6B were dead within four weeks, but *Pesticola* and the *Fusarium* were found to be non-pathogenic. Both of the latter fungi, however, have a possibility of being secondary organisms which could impart the often-seen black coloration by colonizing tissue previously invaded by the primary organism.

Our culture of *Aphanomyces*, isolate 85-6B, has been lost since the experiment just described was conducted. *Aphanomyces* is extremely difficult to isolate, notoriously fragile in culture, and difficult to maintain. The soil which we infested with 85-6B has been retained in pots, however, and PCN seedlings planted in this soil in August and November of 1985, and again in July of 1986, have all died. This *Aphanomyces* remains the only culture we have, thus far proved to be pathogenic.

I have observed *Aphanomyces* in the tissues of other diseased PCNs, but, to date, have failed to capture it in culture for a second time. Because of its difficulty, however, this is not surprising. On the other hand, I have not observed it often enough under the microscope to conclude that it is the only pathogen involved in PCN Crown Rot.

Observations have been made, and cultures obtained, from every area in our garden where Crown Rot has occurred, and on the whole, the results have been comparable. I generally find one or more of the water molds; but the fact that I have found at least 4 diverse species is puzzling unless the PCNs are susceptible to a wide array of water molds. I have found *Rhizoctonia* often enough to speculate that it, too is involved. Numerous *Fusaria* have been seen and isolated, but most have occurred after the plants were kept in a moist chamber for a few days. A *Fusarium* could be involved, since many in this group are pathogens, but we have seen no evidence to support their involvement.

Plants growing in pot test. On the left, those growing in non-diseased soil; on the right, in diseased soil from bed D.



Plants taken from a pot test. On the left, those grown in the clean-soil check; on the right, from soil infested with *Aphanomyces*, culture, 85-6B.

A summary of the organisms observed under the microscope shows that common soil inhabitants which are non-pathogenic, were most frequently seen, followed by water mold fungi and two types of *Rhizoctonia*. Water molds were also the most frequently recovered in cultures, with *Rhizoctonia* less frequently isolated. The table in the next column lists the frequency of each type seen or isolated. All figures are presented as a percentage of the total.

MICROSCOPE AND CULTURE SUMMARY
February 1984 through January 1987

	MICROSCOPE	CULTURES	
WATER MOLDS	31%	54%	33%
RHIZOCTONIA Type 1	20%	33%	14%
RHIZOCTONIA Type 2	8%	13%	9%
OTHER(SAPROPHYTES)	41%		44%

In the "OTHER" category were various bacteria, but they are not included in the count above. Included are the fungi *Alternaria, Cladosporium, Fusarium, Pen-*

icillium, *Pesticola*, and *Trichoderma*. Nematodes and soil mites were also found, and may be contributing to the spread of the disease by injury to the plant tissues. *Fusarium* species were the most frequent representatives of this group, both observed and isolated. *Fusaria* are common soil inhabitants and are easily isolated; but they can be pathogenic as well as saprophytic. For this reason, some of the *Fusarium* cultures have been tested for pathogenicity, but, thus far, with negative results. Some of the "other" fungi on the list, especially the last one, *Trichoderma*, are known to be antagonistic to certain pathogenic fungi.

The water mold fungi are in the class, *Phycomycetes*, a primitive class of fungi. Three of the water molds, *Aphanomyces*, *Phytophthora*, and *Pythium*, which I have isolated from PCN Crown Rot-infected plants, are all well known as plant pathogens, causing both seedling and major root diseases in many horticultural crops. They are called water molds because many of the species live directly in water, and all can survive, infect, and move to new infection sites in water with their motile, swimming spores. Without water they could not infect, but, unfortunately neither could the plant exist.

Rhizoctonia, on the other hand, thrives under hot, dry conditions and although moisture is no deterrent, it is not necessary for its survival. Primarily it causes a damping-off disease of seedlings, but it attacks older plants as well. I have isolated two types of *Rhizoctonia*, one with hyaline (transparent) mycelium, "type 1", and one with dark mycelium, "type 2". Although the dark one, which we are calling type 2, is widely prevalent in many crops, type one has been found associated with diseased PCNs twice as frequently as type two. Type 1 has also been isolated by Dr. Raabe from plants we brought to him.

To summarize, it should be emphasized that this section constitutes an update on the procedures followed in our efforts to identify the cause of PCN Crown Rot Disease. The study is not complete, and additional, more comprehensive pathogenicity tests are needed.

To date we would conclude that one of the water molds, an *Aphanomyces*, is pathogenic to PCNs. We strongly suspect that *Pythium* and *Phytophthora*, frequently seen in the tissues of diseased plants and often isolated during our studies, may be equally pathogenic; but this has not been proven in tests to date.

The hyaline *Rhizoctonia*, which we have arbitrarily called, type 1, may well be playing a role in combination with the water molds to produce the symptoms of this disease. Tests now in progress at our home and at U.C., in which this *Rhizoctonia* is being tested alone and in combination with a water mold, may confirm this assumption. Although we have loosely called this *Rhizoctonia*, type 1, the actual anastomosis type of this fungus will eventually have to be identified by experts at U.C.

PART 3, WATER MOLD ROOT ROTS

Robert D. Raabe
Department of Plant Pathology, University of California, Berkeley

The water molds are the most important of the root rotting fungi. Although there are a number of fungi which are called the water molds, the most common are those in the genera *Pythium* and *Phytophthora*. There are a large number of species of each and they vary in the amount of damage they do and in their host ranges. Some attack many different types of plants whereas others may be limited to a few plants. The water molds are common in soils where plants are found; some even have been found on the roots of desert plants. Many of the water molds infect the smaller roots with little noticeable damage. Others may cause extensive root rot and even move up into the lower portions of the main stems, killing the tissues and eventually causing the death of the plants. Many of the water molds are opportunists; they are presnt in the roots doing a little damage, but as long as the plant has good growing con-

ditions the amount of damage is not noticeable. However, if such plants are put under stress of any kind, they quickly take advantage of the situation, invade more of the plant and cause more damage. They are called water molds because they are favored by wet conditions; they produce motile spores that swim in free water and they are capable of growing under reduced oxygen levels. At the same time, such wet conditions are unfavorable for the growth of many plants, thus weakening them and allowing the fungi to do more damage.

Symptoms of infection are not usually diagnostic. Plants may not show any obvious symptoms, they may be stunted or in advanced stages, the plants may wilt or may die. If roots are examined,varying amounts of disease will be seen. These symptoms are not always due to water molds because other fungi also may produce similar symptoms. In many plants, particularly those that are woody, if the water molds move up into the lower portions of the stems, they turn the tissues under the bark dark brown. This, if observed as the plant is dying, is a good diagnostic symptom.

Because the water molds are so common, because so many different kinds of plants are infected, and because they are in the soil where it is difficult to get to them, control is difficult. Providing as good growing conditions as possible for any type of plant is important. This is especially true for plants which require conditions such as good drainage and excellent aeration or limited amounts of water during any part of the growing season. Giving plants conditions as similar to those they have in their natural habitats frequently will prevent root rot problems.

In addition, there are some fungicides that have proven to be effective in giving control to this group of organisms. these include the following:

Fenaminosulf, which was sold under the trade name of Dexon* and later as Lesan*. It is no longer available commercially though there still may be some around. This material is effective principally against *Pythium* species, though it may give some control against *Phytophthora* species. When wet, the fungicide breaks down rapidly in the light. It also loses its effectiveness in the soil in as short a time as two weeks. It is relatively non-toxic to plants but care must be taken because it is toxic to animal systrms if not used correstly.

Etridiazole, sold under the name of Truban* also is principally effective against *Pythium* species. It tends to be more toxic to plants so it is important to follow directions given on the packages.

Fosetyl-al, sold under the name of Aliette* is effective principally against some of the *Phytophthora* species. One of its advantages is that it is water soluble, thus allowing for better soil penetration than those previously mentioned.

Propamocarb, sold under the name of Banol* is effective against both *Pythium* and *Phytophthora* species. Relatively higher concentrations of this material are needed than with other fungicides effective against this group of fungi.

Metalaxyl, sold under the name of Subdue* (also under the names of Ridomil* or Apron*) is effective in controlling both *Pythium* and *Phytophthora* species. It is effective in very low concentrations and tends to move readily through soils. In experiments in containers, three applications at monthly intervals have been shown to kill all the water molds. This has not been found for other materials.

Although much information is available about the effectiveness of these fungicides, it should be noted that much of the research has been done in container-grown plants. Not as much information is available regarding plants in open ground. More research needs to be done under such conditions.

One important concern is that other fungi than the water molds also cause root rots. Using chemicals which are effective against water molds only may create a serious problem if other root rotting fungi are present. This is because control of one group of fungi will leave less competition and the other organisms will be given an advantage they normally would not have. This suggests that perhaps another fungicide such as benomyl or thiophanate methyl should be used in connection with the fungicides effective against water molds. Another approach is to obtain help from a plant pathologist who can culture and identify

the organisms causing problems in your plants so it is known what organisms are present and which fungicides should be used for control.

* Registered Trade Mark

PART 4, MISCELLANEOUS TESTS

Lewis and Adele Lawyer

EVALUATION OF CONTROL MEASURES

To test the effectiveness of control measures which we had employed in our garden, replicated pot tests were conducted. The first of these was in September, 1985, when disease-infested soil was taken from bed C and from the area in bed P where the diseased plant of COUNCILMAN had been growing. Both of these areas had been drenched three times with Subdue. A third sample was obtained from the same area in bed C, but after it had been treated with Vapam. The three soil samples were potted in 4-inch plastic pots and PCN seedlings planted. A month after planting, all the plants in the pre-Vapam soil were diseased or dead whereas those in the Vapamed soil were alive and healthy.

A second test, started in November, included the three variables above, but with the addition of soil taken from the diseased area in bed V after treatment with Vapam, and a "clean-soil check", using garden soil taken from an area in our garden where no disease had ever occurred. As in the first test, the growth in the Vapamed soil was excellent. In fact it was even better than the growth in the clean-soil check. Plants in the non-Vapamed soils were all dead or diseased.

Starting in August, 1986, a third, more comprehensive test was run, this time for a 6-month period. Soil from five Vapam-treated beds, beds C, D, P (2 different areas), Q, and V were included, along with soil which had been gathered from the same areas before they were Vapamed. Also included were clean-soil checks, as in the second test above. In all the areas which had been Vapamed, separate samples were obtained from the surface, and from a depth of 6 to 8 inches. No disease has occured in the seedlings planted in the clean-soil or in any of the Vapamed soils. First symptoms occurred in the non-vapamed variables 18 days after the seedlings were planted, and all were dead or diseased at the conclusion of the test 6 months later.

In this third experiment, an additional variable was added to each treatment. Four plants in each treatment were dipped in Subdue before planting, and 4 plants were not dipped. There was a marked difference in favor of the Subdue-dipped plants. At the end of the first 3 months, 78 percent of the treatments showed superior growth and vigor for the Subdue-dipped plants, and in the disease-infested soils, all but one of the undipped plants had died, whereas the dipped plants were becoming infected at a much slower rate. Some of the dipped plants were still alive in the bed C and in the *Aphanomyces*, culture 85-6B, variables after the 6-month test period. The Subdue dip was ineffective in the bed D variable where we had observed *Rhizoctonia* to a great extent.

It is difficult to explain why the Subdue drench has been so ineffective in reducing or eliminating the disease in our garden soil when, as you have learned from Dr. Raabe's discussion in the previous section, it has done so in pots. The probability is that in drenching such a large, uninclosed volume of soil, there are pockets which, for some reason, escape the drench. Of course another valid explanation is that we are not dealing solely with a water mold.

TRANSPLANT PROBLEMS

Death of transplants which are being taken or cut from an established clump and moved to a new position, seems to be an almost universal problem. Everyone who has grown PCNs for a year or two knows that there is a right and a wrong time to try to move them; but even if you move them when the roots are white and beautiful, you are seldom 100 percent successful.

The first year I set out PCN plants, almost the only thing I knew about them for certain was that they would die if they so much as heard the splash of water. Of course this isn't true, and because of over-cautious watering, I lost 25 of the 77 plants set out that year, or 32 percent. Subsequently we saw to it that all transplants were kept wet until they were well established.

Even after that, however, we lost from 7 to 12 percent each year. For example, in 1976 there were 8 deaths out of 64 transplants, or 12.5 percent. In 1981 there were 6 deaths out of 86 transplants, or 7 percent. Compare this to our transplanting deaths for the past 3 years, where every plant was dipped in a Subdue solution for 10 or more minutes. In 1984 we set out 159 transplants, in 1985, 67, and in 1986, 82, for a total of 308 transplants, of which 1 died.

The same is true for our seedling transplants, the little, 4-month-old seedlings which we transplant from the seedling bed to the line-out bed. We never have lost many of them, even before Subdue. For example, from 1979 through 1982 we lined-out 819 seedlings, 22 of which died. This is a 3.3 percent death rate. For the last two years we have dipped all the seedlings in Subdue. All other procedures have remained the same as in previous years. Of the 611 transplants, none have died.

We have conducted no tests in which the concentration of the Subdue soution has been varied, although others have told us of using it both as a dip and a drench at much higher concentrations without injury to PCN plants. We see no reason for this, however, since the recommended dosage of 0.3 ml. per gallon, (¼ teaspoon per 4 gallons) of water, seems adequate. We have, however varied the time in the dip solution because it is often inconvenient to remove every plant in exactly 10 minutes. We have found no added benefit or harm from dips up to one hour over the recommended 10-minute time, and have left one plant in the solution for 24 hours with no detrimental effect.

We are certain that the use of a Subdue dip before planting is beneficial, but why it is, is not clear. The Ciba Geigy Technical Release on Subdue states that it has provided excellent control of many of the diseases caused by the *Phycomycetes*, including *Pythium* and *Phytophthora*, but that it has little or no activity against the *Ascomycetes*, *Basidiomycetes*, or the *Fungi Imperfecti*. Those last three classes include all the other plant pathogens, so the benefit must have something to do with a fungus or fungi in the *Phycomycetes*, such as *Pythium*. Perhaps root injury at the time of digging makes the plant especially vulnerable to one of these fungi, and the Subdue keeps it away long enough for the tissue to heal.

PHYTOTOXICITY OF DIPS AND DRENCHES

In an experiment to determine if injury would result from dipping PCN seedlings in solutions of various common fungicides, the following materials were tested: Subdue, Benlate, and Terraclor solutions were tested at concentrations recommended on the labels. The fourth material, Clorox, was diluted to make a 10 percent solution. A tap water dip was also used as a check. Dip duration was 10 minutes in all cases. The plants were planted in 4-inch pots, but before planting, the dipped plants were divided into two groups, one being watered in with the dip solution and the other with tap water. Both the Clorox-dipped groups were watered in with tap water only. The only injury was to the Clorox dipped plants which were severely burned and stunted. Three months later these plants are starting to grow, and there is no indication they will not survive. Otherwise there were no significant differences between treatments. It is our belief that, depending

on the problem encountered at transplant time, any of these dips but Clorox could be used safely.

BIOLOGICAL CONTROL

One more concept which we are testing should be mentioned, that of biological control. Fast-growing, non-pathogenic, soil-inhabiting organisms can compete and replace pathogenic fungi when growth conditions are favorable. Freshly fumigated soil certainly constitutes a very favorable condition for such growth, since most organisms, to the depth fumigated, have been greatly reduced in population. If competitive organisms are introduced shortly after the toxic fumigant dissipates from the soil, they have a good chance of becoming the dominant soil inhabitants and reducing or preventing pathogens from reinfesting the soil.

Bacteria, *Trichoderma*, and *Penicillium* have been employed as biological control agents with limited success in other crops, both as a seed treatment and as a soil additive. One experiment of this sort is now in progress in cooperation with Dr. Raabe at the University. A second biological control experiment is being tried in freshly fumigated soil in our garden. Here, a microbial inoculent, MATSCI MS-5, prepared by the Materials Science Company of Santa Barbara, California, has been dissolved in water and used as a drench following Vapam fumigation in three areas in our garden. These applications should tell us whether soil so treated will in any way influence the growth of plants or the occurrence of disease. MATSCI MS-5 was recommended to us by Dara Emery of the Santa Barbara Botanic Garden, during our visit with him last December. He says it has resulted in improved growth in problem areas in the Botanic Garden. It is a standardized mixture of bacteria and fungi which the formulators say will allow the plants to absorb nutrients more efficiently. To our knowledge, no university has recommended this material and, to date, we have no conclusions.

PART 5, GENETIC RESISTANCE

Lewis Lawyer

It's the only way to fly! Well, it may not be the only way, but it certainly is the best. Let's call it a genetic adaptability to specific conditions, which may be achieved through breeding and selection.

With rust, we already have immunity or very high resistance in most cultivars, and there is no reason to look further until the rust fungus changes and these cultivars become susceptible. Rusts are notoriously prone to change readily through mutation and our resistant cultivars could suddenly become susceptible to new races of rust.

We do have quite a way to go with our *munzii* lines, but we have already seen rust resistant plants showing up bright green and glowing in the midst of their browned and pock-marked siblings. As long as we remain constantly aware that there is a problem, we will succeed.

With *Sclerotium rolfsii* (mustard seed fungus), however, we have a different story. This fungus attacks a very wide range of hosts, and its mode of attack is such that prevention seems to be the only solution. It has very aggressive mycelium which under favorable conditions of moisture and temperature, grows rapidly on almost anything, living or dead, producing a cotton-like mass of threads. The fungus then exudes an acid which kills living tissue on which it is growing, then it colonizes the dead tissue. Such a mode of entry seems difficult to stop genetically, and to our knowledge has not been accomplished with other plants. Fortunately, we have not heard many complaints from PCN growers, and it must not presently be a major problem.

Genetic resistance to PCN Crown Rot also seems difficult, in this case because there is apparently more than one pathogen involved. There are consistent reports, however, that some clones are more tolerant than others to conditions existing in the grower's garden. We

feel that this is also true in our garden where we have experienced very poor growth,or even complete failure with certain cultivars, some of which, we are told, do well elsewhere. Such differences could conceivably be attributed to a tolerance to some element of the crown rot complex which occurrs in a particular garden. On our property, however, where we had the disease, it seemed to attack whatever cultivars happened to be growing there at the time. Nonetheless, we should be searching for resistance, and this is where members of SPCNI can be of help. For our part, we will try to build up a Crown Rot infested soil in which to plant seeds to test for resistance. For your part, you can send us the seeds.

We don't want any seeds unless you have some reason to believe that they may have some possibility for resistance. Especially valuable would be species which are found growing in locations wetter than normal for PCNs. For those of you living in hot areas, the seed could be from a clone which you believe to be particularly tolerant to "hot and wet" conditions. Those of you living in areas where PCNs are particularly difficult might be able to spot something which we can not. There is a possibility that one or more of the Cal-Sibes could be tolerant to part of the complex. If there is anything else you can think of, send it in. We will try to test it and start a breeding program for resistance.

If you can send seed, please tell us everything you know about it, including why you thought it might be tolerant. If seed is found in the wild, unless you think that one of the plants is superior to all the others, it might be best to get a few seeds from each of several plants. Maybe if we all work at this we will eventually get PCNs which will succeed everywhere. Surely this is a goal worthwhile!

THE RAABE-LAWYER CONNECTION

The professional careers of Bob Raabe and ourselves has been remarkably parallel in many respects, although Bob Raabe comes from Wisconsin, whereas Lewis is a native of Alhambra in southern California, and Adele of San Francisco

We first became acquainted when Bob was doing graduate work at the University of Wisconsin in the late 1940s. His research project on white rust disease of spinach was of interest to us because Del Monte Corporation was growing the bulk of their spinach near Crystal City,Texas ("the Spinach Capitol of the World") where this disease was a serious economic problem. Bob subsequently conducted his study on the site at Crystal City, and his definitive data on the life cycle and environmental requirements of the causal organism was an aid to us at that time and much later in the 1970s and 80s when developing tolerance to white rust of spinach was one of Adele's major occupations.

Back in California, we found that, after receiving his doctorate, Bob had joined the staff of the Plant Pathology Department at the University of California at Berkeley, our home stamping ground. And he was working on *Armillaria* root rot, which had been Lewis' major focus of study in graduate school. In fact, it was while working together on *Armillaria* at U.C. that Lewis and Adele first met and worked together. They continued with *Armillaria* research with Del Monte, where hundreds of acres of peach orchard were fumigated under Lewis' direction in order to control the disease.

In the intervening years, our mutual interest in flowers has been the basis of our continuing association with Dr. Raabe. At Phytopathological Conventions we have managed to take off during breaks for hurried visits to botanic gardens in whatever part of the country the annual meeting was being held.

And now it is iris diseases that has us working together. What better basis for a friendship!

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FEBRUARY 1, 1987

CASH ON HAND APRIL 23, 1986		$ 231.58
DUES AND RECEIPTS:		
Dues Collected	$ 537.00	
Dues Collected by AIS	125.00	
Sales of Cohens	74.00	
Sales of Almanacs	4.00	
Sales of Check Lists	139.00	879.00
		$ 1,110.58
DISBURSEMENTS:		
Stamps	$ 28.00	
Form Letter	17.23	
Postage "	56.98	102.21
BALANCE ON HAND FEBRUARY 1, 1987		$ 1,008.37

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