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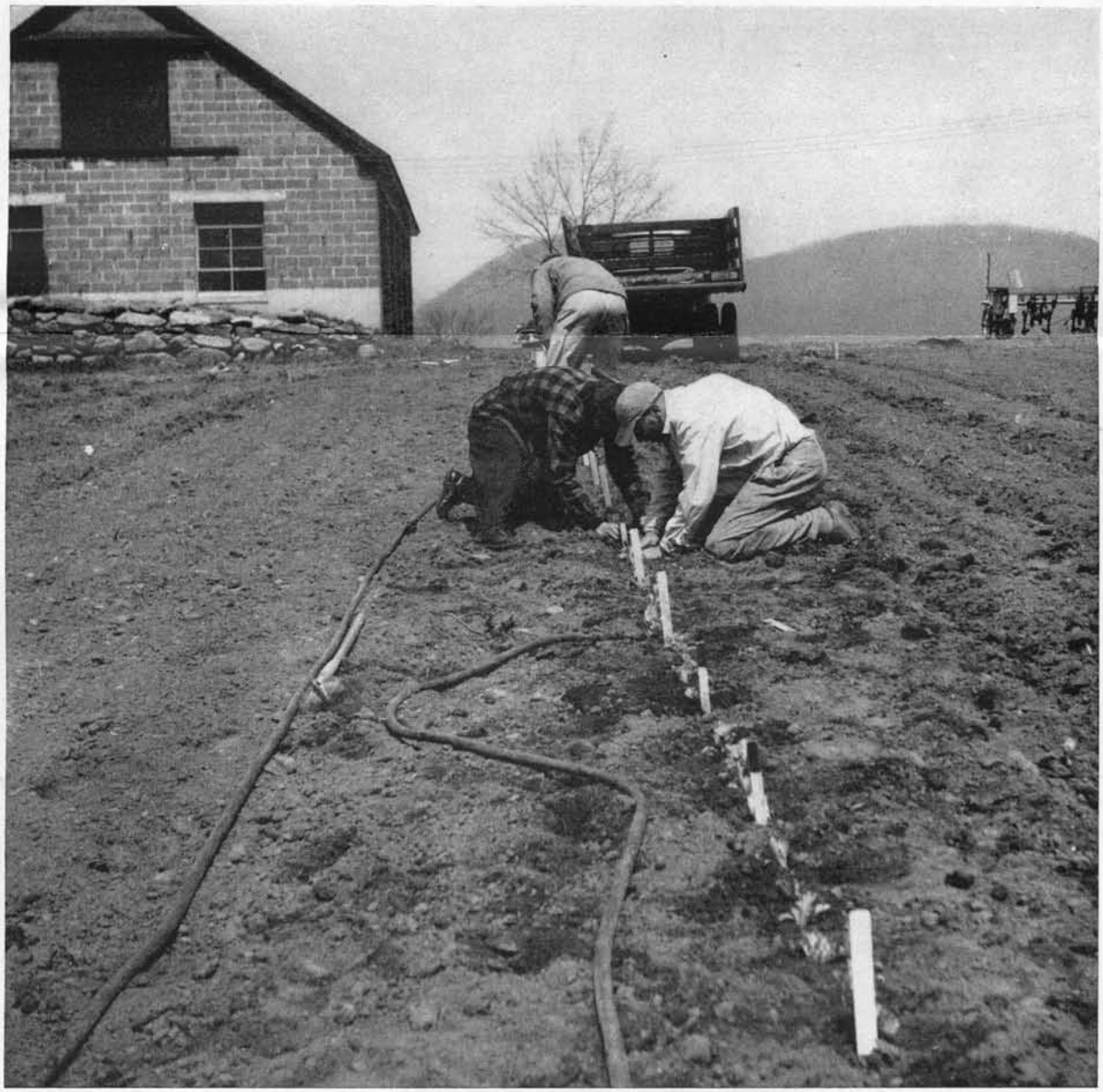
SPRING ISSUE

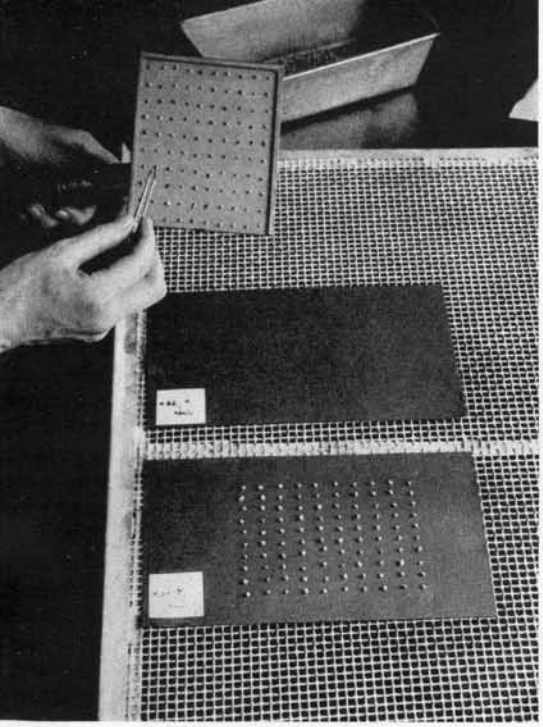


Spring preparations—planting is one of the many preliminary chores that must be done before the season's field research can begin at the Station's Mt. Carmel Experimental Farm.

THE CONNECTICUT AGRICULTURAL EXPERIMENT STATION, NEW HAVEN

THE SEED TESTING STORY

by Frances W. Meyer[1]



100 spinach seeds are counted on a plate by suction and turned onto a wet blotter for the germination test.

In the Station's Seed Laboratory the chief work is the testing of seed offered for sale in Connecticut stores, to determine if they meet quality claims made for them by their producers. Samples of such seeds are collected from both wholesale and retail dealers by the Department of Farms and Markets, which is responsible for the enforcement of the State seed law. Our work begins when the seed-filled envelopes arrive at the laboratory and is finished when the final report is sent to Hartford.

In general, samples going through our laboratory can be divided into three classes: vegetable seed, field seed and lawn mixtures.

Vegetable seed is tested for germination only. Our job is to determine whether the germination claim made by the seed producer is true or not. To do this, each lot of seed is counted into several 100-seed replicates. Corn, pea, and bean seeds are placed between wet paper towels. The pea test is rolled and stood upright in the germinator; the others lie flat on the cabinet's wire trays. Germinator walls and ceiling are sprayed with water, and a tray of standing water in the bottom keeps the humidity high. The cabbage family, tomatoes, peppers, and spinach are placed on top of wet blotters in the germinator. Lettuce and celery require light and are placed in Petri dishes in a daylite germinator. Grasses and endive likewise need light plus the additional stimulus of potassium nitrate.

Germination tests are read at intervals prescribed for each species, and two counts are usually made. The preliminary count is a good indication of the vitality of the seed, but the final count (generally two days to a week later) gives a more complete picture. The sum of these gives the germination percentage. This figure is then compared with the claim or the minimum standard requirement. An accepted tolerance is allowed.

Disease Hazard

Many diseases of vegetable and flower crops are seed-borne, and the analyst has to be alert for evidence of Helminthosporium blight of oats, black rot of crucifers, cucumber scab and many others. We use no seed treatments on the first germination test in this laboratory, because we want the test to be a true representation of what the seed will do in the field without treatment. However, if a sample proves very moldy and threatens to contaminate the germinator, a seed disinfectant is dusted onto the test, and the retest usually is treated before being put into the germinator.

This year "trueness-to-type" tests are being run at our Mount Carmel Experimental Farm on cabbage seed collected this spring. Three years ago similar tests were conducted at Windsor on beet and carrot seed as a supplement to the laboratory tests. The readings of these field trials are open to the general public, as well as seedsmen and vegetable growers, and show even more accurately than laboratory tests the value of a seed lot.

Field and lawn seed mixtures are analyzed for purity as well as germination. This entails taking a known weight of each sample and separating it into its component parts: i.e., pure seed, inert matter, other crop seed, and weed seed; these parts are weighed and then computed into percentages. Such figures are compared with the percentages on the tag which the law requires to be attached to each bag of field seed or box of lawn mixture offered for sale.

Weed seed is particularly examined for the species present. Any lot containing primary noxious weed seeds is prohibited from sale; this includes quackgrass, bindweed, Canada thistle, horse nettle and dodder. Secondary noxious weed seed includes plantains, mustard, dock, corn cockle, the catchflys, and hawkweed. These latter are described in the law as "very objectionable in fields, lawns, or gardens of this state," and their presence in any lot must be indicated on the tag as number of seed per ounce or per pound. Buyer beware!

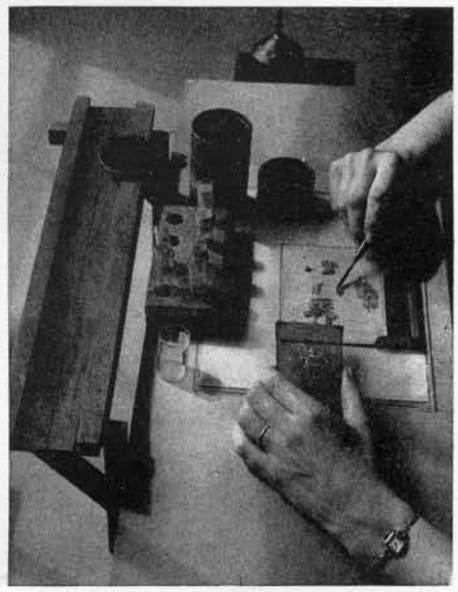
Lawn Mixtures

In general, field seed is a homogenous lot, while lawn seed is a mixture of many kinds of grass seed. We do not pass on the relative merits of the kinds of grass seed in the mixture, but only on whether the component parts are truly represented on the label. Contrary to some popular supposition, crab grass seed is rarely, if ever, found in lawn seed mixtures.

The seeds in a lawn mixture vary in size and weight and are separated differentially by air blast. The chaff is blown off first, then the light seeds, and so on. Sieves operated by hand and by a mechanical shaker prepare the seed for the final analysis by hand. The component parts of the mixture are then treated in the same manner as the field seed and the percentages compared with the claims. Germination tests of each kind of seed in the mixture then follow.

The Station does not test seed for seed producers. Samples of seed are tested by this laboratory only after they are labelled and put on the market. If seed is tested before labelling, the Station becomes a party to the claims made for it, although it cannot guarantee that contents of all packages meet these claims. Random sampling of packages already on the market makes it much more likely that the occasional cases of dishonest claims will be "found out." Results of our analyses are available to the public and this acts as a strong deterrent to a repetition of the offense.

[1] Mrs. Meyer is an assistant in the Plant Pathology Department.



Separation of large seeds in a lawn mixture is partly a hand operation. Smaller seeds are removed first by blowing and then run through coarse and fine sieves in a mechanical shaker before final hand separation.

The Chemistry of Tobacco Curing and Fermentation

by Hubert B. Vickery[1]

When the leaves of the tobacco plants that grow under the 6,000 or more acres of shade tents in the State of Connecticut are hung in the curing barns, an extremely complex series of chemical reactions and physical changes promptly begins. The two most obvious of these are the evaporation of water and the destruction of the green chlorophyll. After a few days the leaves pass through a brilliant yellow stage, but they finally become limp and brown.

Although the change in color is the most conspicuous event that occurs, very little is known about what happens to the pigments from the chemical point of view. When the chlorophyll is destroyed after the first few days, the yellow pigments which have been present from the beginning are revealed. As curing progresses, these substances are in turn largely destroyed and, in addition, brown colored substances are produced which give the leaves their characteristic final appearance. The quality of cured tobacco is to a considerable extent determined by the evenness of the coloration of the leaves, and careful control of the condition in the curing barn in necessary.

Typical curing shed where tobacco leaves undergo the chemical processes that will change them from a raw green product to the brown, fragrant leaf used for cigar manufacture.

Two Years of Research

A study of the chemical changes that occur during curing and fermentation of shade tobacco has been going on in the Biochemistry Department for the past two years. One of the most important of the reactions that take place during the first 10 days is the digestion of rather more than one-half of the protein of the leaves, the nitrogen being converted by an extremely complex series of reactions into asparagine which accumulates in an amount equal to more than 7 per cent of the organic substances present. At the same time, about 16 per cent of the organic solids disappear entirely, being completely oxidized by the process of respiration.

Another chemical reaction of great importance is the conversion of malic acid into citric acid. Green shade leaves contain about 15 per cent of their organic solids as malic acid and about 3 per cent as citric acid. After the leaves have been cured, they may contain as much as 11 per cent of citric acid and considerably more than one-half of the malic acid has disappeared. This reaction takes place during the first 10 days of the curing period before the leaves have become wholly brown and while the cells are still alive.

The onset of the brown coloration marks the point at which the death of the cells of the leaves occurs. Chemical changes that had been going on with remarkable speed up to this point either cease entirely or become so slow as to be scarcely detectable. Nevertheless, the changes that take place during the remainder of the two-month period that the leaves hang in the curing barn are of great technological importance. The color becomes a more even brown and the leaves gradually acquire the appearance of fully cured tobacco. Among the minor chemical changes that take place are the loss of a little nitrogen in the form of ammonia and the evaporation of a small proportion of the nicotine. The leaves finally reach a stage at which they are remarkably responsive to changes in the humidity of the atmosphere. On damp days, they become soft and pliable and can be handled without damage; on dry days they are crisp and brittle and would break if roughly treated.

The cured tobacco is next subjected to the process of fermentation, an operation carried out at the warehouse where several tons of leaves at a time are built into close-packed rectangular piles. Over a period of about a week, the tobacco heats up and, when a certain temperature is reached, the pile is rebuilt. This process of rebuilding is repeated five or more times until the tobacco has lost the sticky gum from the surface of the leaves and has acquired the characteristic odor and texture of cigar tobacco. The leaves are then examined one by one by expert graders and sorted into from 10 to 16 different grades of widely different value.

Chemical Changes Continue

A great many subtle chemical changes take place in the tobacco during these operations. The most obvious involve the loss of the gum and the change in odor. Unfortunately, the chemistry of these alterations is still unknown. However, a major chemical change that has been detected is the disappearance of all of the asparagine that was formed during the first stage of curing. In addition, a continuous, although small, loss of nitrogen as ammonia occurs, nicotine diminishes in amount and some 4 per cent of the organic solids are consumed in reactions of unknown nature.

The final result of this complex sequence of operations is Connecticut shade-grown wrapper tobacco, a product that provides the biochemist with some of the most intricate and difficult chemical problems he can hope to encounter. Many of these problems are, however, similar to the problems presented by other plant and animal tissues. The study of these matters is thus of importance not only for the tobacco industry but also to agriculture in general and so to all who depend on agriculture for their food.

[1] Dr. Vickery is head of the Station's Biochemistry Department.

Testing

Tests for poisonous metals such as lead, mercury and thallium are made with the spectrograph. Here Waddy Mathis is adjusting the power source, which will pass a high voltage current through carbon electrodes (like those in the tray at right) containing the samples.

A RECENT epidemic of dog deaths in Fairfield County has called attention again to the problem of testing animal remains for poisons. Such testing has been done by the Analytical Chemistry Department of the Station for many years.

A chemist was appointed by the State Board of Agriculture in the 1860's to analyze fertilizers for the protection of farmers of the State. His functions were transferred to the chemist of The Connecticut Agricultural Experiment Station, when it was established in 1875. Initially analyses were made of the fertilizers and then feeds, foods, and drugs sold in the State. The records do not show when the first tests for poisons were made, but the 1907 Station Report says "The station is constantly called upon by individuals to make examinations of . . . bodies of animals suspected of being poisoned," and there have been annual references to such examinations since 1911. In the early years the animals submitted were mostly cows, chickens, geese and ducks, with very few dogs; but in recent times the number of dogs has exceeded the cattle and poultry combined. This interesting change is no doubt due to the relative decline of the farm population as compared to the urban population in Connecticut. Records of some of the samples submitted in the earlier years give us a fair picture of the more common poison hazards of those days.

In 1912, two samples of wheat middlings contained Paris green; arsenate of lead was found in one cow, and arsenic but no lead in a group of four cows. In 1917, the first poisoned dog was reported; the poison was mercury. In 1923, there was one sample each of phosphorus, strychnine (in a dog) and lead arsenate, and a poisoned bait containing chromium. An unusual sample was one of wine made in a galvanized tub. The wine had caused illness and was found to contain 370 parts per million of zinc oxide. In 1933, the first case of nicotine poisoning (in a cow) was reported.

There have been some interesting examples of unusual poison cases in more recent times. In 1938, a dog food supplement supposed to be calcium phosphate was found to be 80 per cent sodium fluoride. In 1945, a dog's stomach contents were found to contain not only strychnine but also blue-dyed hemp seeds, thus proving conclusively that the source of the strychnine was a commercial mole killer. In 1947, a sample of salted hay submitted by a farmer because his cows refused to eat it was found to have been "salted" with arsenate of lead.

The newer organic insecticides began to appear as causes of poisoning in 1947, when DDT was found in a cow. Seven dogs died in 1949 from poisoning by a new rodenticide, alpha naphthyl thiourea ("ANTU").

The only way of knowing for certain that an animal has been poisoned is to identify the poison in its body by chemical tests. Certain poisons produce characteristic symptoms — the convulsions of strychnine are a well-known example — but all of these symptoms, including rapidity of death, can also be produced by disease. The Fairfield County epidemic of dog deaths mentioned at the beginning of this article is a good example of this fact. When a number of dogs began to die suddenly in Stratford, without apparently having been ill previously, people were certain that the dogs were being deliberately poisoned, and some parents became panicky lest their young children pick up the poison and swallow it. Organs of the dead dogs were submitted to the Station; tests for all of the common poisons (including the originally suspected strychnine) proved negative, and a sample of what was stated to be the poisonous material—which a woman had snatched out of a dog's mouth—turned out to be a harmless cosmetic.

Disease, Not Poison

Poisoning seems to have been eliminated as the cause of the animals' deaths and the subsequent spread of the epidemic in a straight line from Stratford to Devon to Milford to West Haven shows the typical pattern of disease transmission. A veterinarian of long experience has informed us that he believes the deaths were due to "hard pad disease."

Contrary to popular opinion, chemical assay of poisons is not a rapid procedure. It may be necessary for even the most competent and experienced chemist to spend as much as two weeks in attempting to isolate and identify some of the organic poisons. It is true that new scientific instruments have greatly accelerated some analyses. The Station's spectrograph will test for metallic poisons such as lead, mercury and thallium very rapidly, but unfortunately there is no shortcut to the detection of such a relatively common poison as strychnine. For such reasons toxicological analysis is the most costly and time-consuming type of work carried out by the Station Laboratory; because of the expense many States have abandoned poison testing or limited such testing to farm animals alone, but so far this service is still supplied gratis to Connecticut citizens.

During 1952, specimens from 182 animals were examined in the labora-

r Poisons at the Experiment Station

by Harry J. Fisher[1]

tory; poisons were found in 78 of the animals. The most common poison found was lead, with 32 cases; zinc came next with 25 cases, followed by mercury, cadmium, DDT, "ANTU" and nitrophenide (a drug used to prevent coccidiosis in poultry) with four cases each. There were three cases each of arsenic and antimony poisoning; two each of poisoning by barium, cyanide and boric acid; and single cases of poisoning by a number of other materials, including strychnine, thallium, chromium, nitrate and kerosene.

In many cases the kind of poison found is a clue to where the animal could have picked it up. For instance, a finding of both lead and arsenic indicates that the animal ate arsenate of lead, which is used only as an insecticide. If lead and zinc and perhaps barium were found, but no arsenic, it would be almost certain evidence that the animal was poisoned by licking paint. Arsenic without lead could point to either an arsenical weed killer or an ant poison. Strychnine probably comes either from a poisoned bait or a rodent poison; thallium is found nowadays only in ant and rat poisons. The finding of barium in the absence of other heavy metals would suggest the animal ate a rodenticide whose active ingredient was barium carbonate. We have not yet found cases of poisoning from the more toxic of the newer insecticides, such as tetraëthyl pyrophosphate and parathion, possibly because the farmers who employ these insecticides have been fully informed by the manufacturers of the potential dangers in their use.

Rat Poisons

The poisons "1080" and "Warfarin" deserve special mention. The first of these, while it is probably the most effective rat poison known, is also extremely deadly to man and all other animals, and there is no known antidote. For this reason, its sale and use are restricted by law to experienced pest control operators. Unless a professional rat exterminator has been employed recently in the neighborhood, there is no possibility of animal poisoning from this cause.

"Warfarin" poisoning of pets is also extremely unlikely because this poison works by gradually lowering the coagulating power of the blood, as it is consumed over a period of time, until the animal eventually dies of internal hemorrhage. "Warfarin" rat poisons consist of grain containing a small quantity of the poison, which the rat must return to and eat for several days before it dies. Only if a dog kept coming back and eating a rat bait consisting mostly of corn could it conceivably become poisoned with "Warfarin." There is at the present time no chemical test for small quantities of "Warfarin" in biological tissues. "Ten Eighty" can be detected in animal remains by chemical means, but the test is complicated and time consuming.

Station Does No Autopsies

It should be emphasized that the Station is not equipped to do any pathological work or make bacteriological tests, and that it cannot accept whole animals and perform the autopsies required to separate the organs to be tested. If any Connecticut citizen has an animal that he suspects has died of poison, he should obtain the services of a veterinarian to remove the liver and stomach contents; these may then be submitted to the Station by either the veterinarian or the owner, and will then be tested. However, because it is never possible to decide from the manner of death alone whether the animal died of disease or poisoning, veterinarians will frequently first submit the organs to the Department of Animal Diseases of the University of Connecticut at Storrs for a pathological examination. If their examination raises any suspicion of poisoning, the Storrs authorities always forward the specimens to this Station.

Should any of the readers of "Frontiers of Plant Science" lose a farm animal or a pet in the future under circumstances suggesting poisoning, he should first consult a veterinarian; if the veterinarian agrees that there is a possibility that poison and not disease caused the animal's death, and if he will remove the necessary organs for examination, the Station will gladly make the necessary tests for poison.

[1] Dr. Fisher is head of the Station's Analytical Chemistry Department.

Miss Janetha Shepard is conducting a test for "ANTU" (a rat poison) on a specimen from a dog suspected of having been poisoned.

From the Director

The costs of government are being reappraised. Many fear that taxation may kill the goose that lays the golden egg.

Since much scientific research is supported from taxation, we need to redetermine whether or not it contributes to garroting the golden goose. The Connecticut Agricultural Experiment Station costs many thousands of dollars annually. What does it return?

Among other things, it returns new wealth. Connecticut grows 39,000 acres of field corn. About 88 per cent is planted to hybrid corn, which was discovered at this Experiment Station, and which produces about 40 per cent more grain per acre than prehybrid varieties. This amounts to 682,000 bushels of extra corn in this little State. In 1951 corn sold for about $2.15 per bushel. This then means $1,450,000 of new wealth was produced that one year. This is more than *twice the entire* budget of the Station, and this is *renewable* wealth which is obtained again every year.

Chickens provide an even more spectacular case. In 1918 chickens could not be confined inside houses where they are mass-produced today. Dr. T. B. Osborne, of this Station, discovered the reason. Chickens in houses are starved for vitamin D which they make for themselves if allowed to "run on the range" in the sunlight. Osborne fortified the poultry diet with vitamin D and numerous chickens can now be produced where only one could be raised before. The poultry business of Connecticut has jumped from $5,000,000 in 1918 to $64,000,000 in 1951. About $48,000,000 of this is a new wealth that Connecticut would not have had without the vitamin researches of Dr. Osborne. This new wealth has been derived despite a *drop* in the cost of eggs. When chickens had to be grown on the range in 1918, eggs cost about $1.50 a dozen retail. Eggs cost about 75 cents now when chickens can be "grown in confinement." Without this development, the cost of eggs today could well be three or four dollars a dozen.

It seems evident that research creates golden geese where none were before and that wealth flows out of, not into, research laboratories.

James G. Horsfall

THE TOOLS OF SCIENCE:

Radioactive Tracers

by Albert E. Dimond[1]

Modern scientific instruments permit the scientist to answer questions that have remained unanswered for years. Some of these tools of science work with amazing speed and accuracy. For example, in research on plant diseases, radioactive tracers have been used in conjunction with an instrument known as a scaler which, with a Geiger counter, detects and counts very small amounts of radioactivity.

A question which had not previously been answered satisfactorily was recently examined at The Connecticut Agricultural Experiment Station. The question is a simple one: why do diseased plants wilt? Scientists have for some time puzzled over this in connection with diseases such as Dutch elm disease and the Fusarium wilt of tomato. Some have said that such diseased plants wilt because of poisons which are present in the sick but not in the healthy plant, poisons which make the leaves unable to absorb water from the rest of the plant. Others have thought that the wilt-diseased plant is suffering from an internal drought, caused by inability of the stem to conduct water to the leaves.

Measurement of Water Movement

With modern scientific instruments, it is far easier than it used to be to tell how fast water moves up a stem. If water moves up a sick plant much more slowly than it does in a healthy one, we suspect that the diseased plant is wilting because the leaves are not getting enough water from the stem.

Now it happens that fertilizer elements move through the stem with the water. Therefore, we can use radioactive phosphorus to tell the rate of water movement. The rate at which the phosphorus in very dilute solution moves through the stem also tells us how fast water moves. This experiment is a very simple one to perform.

A healthy tomato plant is cut off at the ground line and the cut end of the stem is dipped into a very dilute radiophosphate solution for a very short time, say 30 seconds. If, at the end of the 30-second exposure period, the stem is rapidly cut where each leaf leaves the stem and the leaves are also cut off, the radiophosphate is trapped in the segments of stem that remain.

The radioactivity in these segments is then detected with a Geiger counter. If the height of each of the stem segments above the end dipped in radiophosphate is known, one can tell how fast the radiophosphate moved. The height to which the radiophosphate moved divided by the time for which the stem was exposed to radiophosphate gives the rate of movement of water and phosphate up the stem. For example, in a healthy plant, the Geiger counter detected radiophosphate as high as six inches above the cut end in a 30-second exposure period. Therefore, the radiophosphate and the water moved up the stem at the rate of 12 inches per minute.

Takes Longer for Sick Plants

But a diseased plant, showing early signs of Fusarium wilt, behaved very differently. It was found that a much longer exposure period had to be used because the rate of radiophosphate movement (and of water) was much less than in a healthy plant. Actually an 8-minute exposure period was necessary. Within this period, the radiophosphate moved a mere two inches, and the rate of movement proved to be only one-quarter of an inch per minute.

This study suggested that the diseased plant wilts because water cannot move through the stem as rapidly as it is lost by evaporation from leaves. Accordingly, the evaporation of water was measured, using leaves from diseased and healthy plants. This comparison indicated that the leaves on a diseased plant are subject to a severe drought, arising within the plant, and when this drought becomes severe enough, the plant wilts.

By using the same method, it is possible to find where the block to normal flow occurs, whether in the root, stem or petiole. Knowing where the blocking occurs makes easier the task of finding the cause of the trouble. In turn, this information is used by the scientist in finding better methods to control either Fusarium wilt of tomato or Dutch elm disease. Our modern tools of science make easy what used to be difficult.

[1] Dr. Dimond is head of the Station's Plant Pathology Department.

'BORER-RESISTANT' CORN

by Neely Turner[1]

On sweet corn the answer to the corn borer problem is the usual one for plant insect pests. Suitable insecticides have been found and treatment schedules devised which check damage by the borer on this crop.

Field and silage corn present a much more difficult problem. It is almost impossible to apply dusts or sprays to such tall plants growing close together. Furthermore, the cost of the treatment is high enough to make it of doubtful economic value. Control by other means, such as the development of corn resistant to borer attack, is thus highly desirable.

Corn breeders, working mostly in the middle western Corn Belt, sought and found types of corn that would resist the corn borer. Some of these were tried in Connecticut, but they proved to be just as susceptible as the varieties we were having trouble with here. This provided a first-class puzzle. Did growing in Connecticut change the resistant corn, or were Connecticut corn borers "different" from those in Ohio?

The first clue was found in the records of an experiment on effect of date of planting on infestation of sweet corn. This work showed that Spancross sweet corn planted April 20 might have 75 per cent of the ears infested, while the *same variety* planted June 1 was practically free from borers. Moreover, when early and late varieties of corn were planted so that the ears matured at the same time, they were equally infested. In other words, when the growth of the corn plant was synchronized properly with seasonal development of the corn borer, the plant was "susceptible." When the two were not synchronized, the plant was "resistant."

Effect of Growth Stage

This led to a thorough study of the effect of stage of growth of the corn plant as related to infestation and survival of the corn borer. Moths usually did not lay eggs on plants until a few days before the tassel formed. Even if eggs were laid on smaller plants, the hatching larvae did not live. (This proved to be of great importance in establishing schedules of treatment to control borers on sweet corn.) When all sizes of corn plants were growing when the eggs were laid, the moths selected the plants with green tassels or even older. In other words, the "resistance" or "susceptibility" depended on the stages of growth present and not on the inheritance of the corn.

This was proved in a striking manner by growing two hybrids, Lexington and Marcross, side by side as early corn. Each variety received the same number of eggs, but survival in Lexington was much lower than in Marcross. In growth Lexington reached the susceptible late whorl stage more slowly than Marcross, and then grew rapidly enough to be harvested at the same time as Marcross. Under these conditions Lexington was "resistant," because no eggs were laid after the late whorl stage. When the test was repeated with later plantings, the difference disappeared because eggs *were* laid after the late whorl stage.

'Different' Borers the Answer

With this information it became clear that the difference in behavior of "resistant" field corn in the Middle West and in Connecticut was probably a result of "different" corn borers rather than by a change in the corn. In the Middle West there is one generation of the corn borer, starting in June. In Connecticut, there are two generations of the corn borer, the first starting late in May and the second late in July. Field corn in the Middle West has not reached a highly susceptible stage of growth when eggs are laid. In Connecticut it could be in exactly the right stage for the second generation.

To test the hypothesis, two inbreds susceptible to the corn borer and two resistant in the Middle West were planted in Connecticut. The most highly resistant inbred in the Middle West was most susceptible in Connecticut.

Thus, the "resistance" recorded in the Middle West was a result of the occurrence of stages of growth unfavorable to the corn borer when the plants were being infested.

The seasonal history of the corn borer is such in Connecticut that it may be difficult to breed field corn having this sort of "resistance." However, another sort of "resistance" has been found by our corn breeders. Much of the loss of field and silage corn has been caused by breakage of infested stalks. Field corn types having rigid stalks highly resistant to breakage under infestation by borers have been selected. These have solved the problem at least temporarily, and Connecticut farmers have been able to maintain and increase field and silage corn production in spite of the corn borer.

[1] Mr. Turner is head of the Station's Entomology Department.

Field corn too young to support European corn borer. Any eggs laid on plant or one younger would probably survive.

The youngest stage of corn suscept to high survival of the corn borer. If and later stages of growth occur when e are laid, the plant can be heavily infes

Corn in the most attractive stage fo festation by the corn borer.

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List of New Station Publications

BULLETINS

561. Fertilizer Placement of Connecticut Tobacco.
562. The Toxicology of *Habrobracon* Venom: A Study of a Natural Insecticide.
564. Growing Tobacco in Connecticut.
565. Boxwood Insects and Their Control.
566. The Formation and Development of the Norway Spruce Gall Caused by *Adelges abietis* L.
567. A Study of Spray Machines in Connecticut Orchards.
568. Control of the Andromeda Lace Bug and the Holly Leaf Miner.

CIRCULARS

184. Control of the Japanese Beetle and the Asiatic Garden Beetle.

SPECIAL PUBLICATIONS

Combating the Dutch Elm Disease (revision).

Laws and Regulations Concerning the Inspection of Nurseries in Connecticut and Transportation of Nursery Stock.

CONTROL OF TOBACCO SUCKERING

Removing the lateral shoots or "suckers" from tobacco plants hastens maturity, increases size of the leaves, and results in better leaf texture. At present, suckers are pulled individually by hand—a laborious, time-consuming operation. Chemical control looks promising, although it is still in the experimental stage. Reports on research with two materials are contained in the paragraphs below.

With Maleic Hydrazide

by E. L. Petersen[1]

The practice of suckering tobacco may be eliminated from the chores of stalk-cut tobacco growers in the future by the use of chemical compounds. Maleic hydrazide (M.H.) is one of the chemical compounds which prevents the development of sucker shoots after the tobacco plant has been "topped."

Spraying the plants with 1 per cent M.H. at the rate of 50 gallons per acre has held sucker growth to negligible amounts in tests covering the past four years. Yields and grading have been as high or higher than for untreated tobacco. In three years out of the four, no detrimental effects were observed. However, in 1951 some of the treated tobacco was damaged at stripping time because of an excessive moisture content. This condition did not recur in 1952 yet further tests must be run to determine if such a condition is a result of the treatment.

Although some cigars have been made with the treated tobacco with no evident detrimental effects, further tests are being conducted to determine if the quality of cigar leaf is injured by the M.H. treatment.

Until the curing problem has been resolved and quality tests have proved that M.H. can be used without detrimental effects, its general use by growers is not feasible.

[1] Dr. Petersen was formerly a geneticist at the Station's Tobacco Laboratory at Windsor.

With Mineral Oil

by A. B. Pack[1]

Preparations of white mineral oil and oil-water emulsions have been applied to open-field tobaccos for the control of sucker growth. One to two teaspoonfuls of the undiluted oil or emulsion are applied to the apex of the plant immediately after topping. The oil runs down the stalk and apparently forms a smothering coat over the sucker buds to cause death or suppressed growth.

In three years of experiments good control of suckering has been achieved for two to three weeks with certain heavy oils and 1:1 emulsions. Control is practically complete on the upper half of the plant but occasionally suckers near the base are not effectively suppressed. Cured tobacco from oil-treated plants has been of equal or better quality on the sorting bench than leaves from plants suckered normally. It has been observed that a soft rot of the stalk or leaf axil sometimes occurs on oil-treated plants. The oil injury has varied from moderate amounts on some plants to practically none on others. This rot seems to be associated with seasonal or growing conditions and is encouraged to some extent by the oil treatments.

Until further study of the soft rot and of the effect of oil treatments on leaf chemical composition can be made, this method of sucker control is uncertain.

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