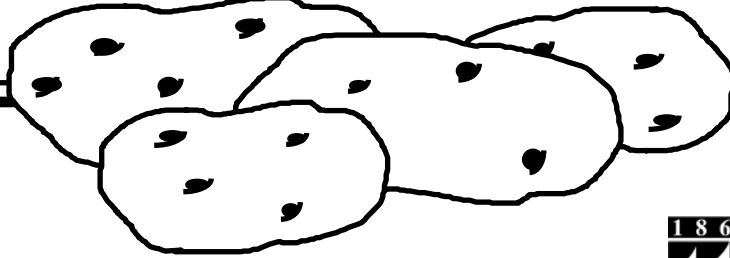


SPUDLINES



DECEMBER 2000
VOL. 38 NO. 3

CONFERENCE
ISSUE



Dear Potato Grower,

This first fall issue of SPUDLINES after the harvest of the 2000 crop. In this issue, we are enclosing the program for the Sixteenth Annual Maine Potato Conference and Trade Exhibit. The conference will be at the Caribou Inn and Convention Center on January 24 and 25, 2000. The trade show again promises to be interesting and informative. License recertification credits will be available for those holding a valid license from the Maine Board of Pesticides Control as will CCA credits for those so certified (call 760-9ipm for details). Be sure to attend and support the exhibitors at the booths.

The Maine Potato Board is holding their annual meeting the evening of January 26, 2000. We hope to see everyone there. Don't miss it.

This publication is in part supported by a grant from the Educational Committee of the Maine Potato Board. The potato growers, processors and brokers of Maine pay assessments. Portions of these assessments were approved for the educational purpose of keeping Maine potato growers and related Maine industry people informed.

Sincerely,

Steven B. Johnson, Ph.D.
Crops Specialist

January 9-11	Augusta Trade Show Civic Center, Augusta
January 24-25	Annual Extension Potato Conference Caribou Inn and Convention Center, Caribou
January 26	Maine Potato Board Annual Meeting Presque Isle Inn and Convention Center, Presque Isle
February 2	CCA Exam Cooperative Extension, Presque Isle

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Upcoming Programming of Interest

POTATO WART BASICS

Steven B. Johnson, Ph.D.

Crops Specialist

Potato wart or potato canker is a disease caused by the soil-borne fungus *Synchytrium endobioticum*. It is a serious disease that affects cultivated potatoes, tomatoes, and a number of wild *Solanum* species. Potato wart was once the most serious disease of potato, but now the spread of the pathogen and its subsequent disease have been controlled by quarantines. The wart disease problem is compounded by the

fact that the fungus has developed numerous strains, known as pathotypes. About 20 different pathotypes occur around the world, including four known in Newfoundland. Potatoes that are resistant to one pathotype may be susceptible to another. Although resistant potato plants do yield disease-free crops of tubers, the problem of contaminated soil remains. The disease still poses a serious threat to potato production because the spores of the fungus can remain viable in contaminated soil for many years.

History

Potato black wart was first described in Hungary in 1896, and was subsequently discovered in some Eastern European and most Western European countries except Portugal where it was eradicated. It is thought that the disease was first introduced to Europe with breeding material from the South American Andes in the aftermath of the 1840-50 potato blight disaster. Early in the 1900s the disease was reported in Newfoundland. Black wart has also been reported in Algeria, Bolivia, Bhutan, Chile, China, Falkland Islands, India, Lebanon, Mexico, Nepal, Peru, South Africa, Tunisia, Uruguay and New Zealand's South Island. Wart was reported in some Eastern states of the U.S. where it was contained and subsequently eradicated.

At the turn of the twentieth century, it was considered the most serious potato disease around. At that time the devastating impact of late blight, manifested as the Irish Potato Famine, was still a memory to some and the social impact was clearly understood.

Life Cycle

Synchytrium endobioticum is a soil-borne fungus that does not produce hyphae, but sporangia containing anywhere from 200-300 motile zoospores are produced. In the spring, at temperatures above 45 F and given sufficient moisture, the overwintering sporangium found in decaying warts in the soil germinate and release hundreds of swimming zoospores into soil water. Each zoospore possesses a single flagellum that enables it to move through soil water to reach the host. The swimming spore, which is about 1/50 the size of the resting spore, eventually reaches and penetrates susceptible potato tissue, thus perpetuating the disease cycle. Once a suitable host cell is encountered, the zoospore will shed its tail and penetrate the cell. The infected cell swells as the enclosed fungus forms a short-lived but quickly reproducing structure—the summer sporangium—from which numerous zoospores are released to infect neighboring cells.

This cycle of infection and release may be repeated for as long as conditions are suitable, resulting in the host tissue becoming thoroughly infected. The cells surrounding the infected ones also swell and the tissue proliferates, producing the characteristic cauliflower-like appearance.

Under certain conditions, the zoospores may fuse in pairs to form zygotes. A thick-walled structure called the winter sporangium is formed. These mature winter sporangium are resting spores and are released into the soil from rotting warts. These spores can survive between potato crop rotations for very long periods of time and remain viable for at least 40 years, their longevity contributing to the wart disease problem. The presence of abundant soil water following heavy rain is a key to the development of the disease. The disease is favored by cool, wet soils. The severity of the attack depends on soil conditions during tuber development and the variety of potato being grown.

The disease appears in new areas when the fungus is transported to new soil by infected tubers or by movement of contaminated materials: hence the severe restrictions placed on Newfoundland by the Canadian government.

The main means of spread is by infected seed tubers that may have incipient warts or infested soil attached to tubers. It can also be spread by movement of contaminated materials including the soil itself, implements used in potato cultivation, footwear worn when working potato plots and hooves of animals tethered on infested soil, or even by wind passing over dried infested soil. The sporangia can survive digestion by animals, and therefore can be spread by manure from animals fed on diseased potatoes. The movement of the disease to new sites in contaminated areas has been linked to foot traffic patterns as well as rail and automobile traffic patterns. The potato wart pathogen has been recovered from vehicles leaving contaminated areas.

Symptoms

The disease appears on all underground parts of the potato except the roots. Buds on stems, stolons and tubers are the centers of infection and abnormal growth activity leading to wart formation. A typical wart is white when under ground and black when decaying, roughly spherical, but it is usually not a solid structure. The warts may be as small as a pin and easily overlooked, or as large as a fist. They can range from small protuberances to large intricately branched systems. Warts are soft and pulpy and can be cut more easily than healthy tuber. Morphologically, a wart consists of distorted, proliferated branches and leaves grown together into a mass of tissue resembling the head of a cauliflower. A developing wart may become exposed at or above the soil line where it turns green. As the warts become older, whether above or below ground, they darken and decay. It is not uncommon for the entire tuber to be replaced by the warty proliferation. Warts that develop on potatoes in storage may be the same color as the tuber.

Above-ground symptoms are not usually apparent although there maybe a reduction in plant vigor. In a severe infestation, the tip of the stolon becomes infected and develops a gall

instead of a tuber. Tubers may bear more than one warty outgrowth and, in some cases, the whole tuber can be affected. When infected early, tubers can become so distorted and spongy that they are almost unrecognizable. Warts on stolons are similar to those on tubers and may be visible if the stolon is exposed above ground. Severe infestations destroy the potato crop by preventing tuber production. The yield of saleable potatoes from a severely affected crop can be less than the actual weight of seed potatoes planted.

Control

For practical purposes, potato wart has not been controlled, but rather contained. This disease is contained through quarantine measures. Long-distance spread has been contained through quarantine measures, whereas local spread has been contained through resistant varieties and local legislation. Laws identifying what specific potato varieties can be grown, and in some cases whether potato growing is even permitted, is an approach used in many parts of the world to contain the local spread of this pathogen and subsequent disease.

Quarantine Measures

Most, if not all countries in the world have quarantines to protect their agriculture where a similar form and inspection procedure takes place. Canada is no different in that they too have quarantines to protect agriculture in their country. Canada has a quarantine on potato wart with particular application to Newfoundland, restricting the movement of potato tubers as well as other materials. Potato wart falls under Canada's plant protection act restricting movement within Canada. The Canadian Food Inspection Agency's (CFIA) Policy Directive, *Import Requirements for seed potatoes and other potato propagative material*, regulates the importation of potato parts used for propagation, and addresses potato pests:

Potato wart (Synchytrium endobioticum (Schilb.) Perc.) occurs exclusively in Newfoundland. It is a serious potato pest which produces galls and outgrowths on tubers. The disease is subject to quarantine controls in many countries. Field grown potatoes and soil from Newfoundland are prohibited from being exported to other provinces of Canada.

The North American Plant Protection Organization, a coordinating agency of Canada, Mexico and the U.S., has a quarantine on potato wart to prevent the spread of NAPPO A-2 quarantine pests within the NAPPO region.

On August 20, 1912 the USDA initiated a quarantine for white-pine blister rust, potato wart and the Mediterranean fruit fly. A portion of Section 160 reads as follows:

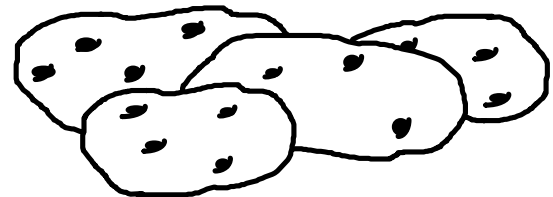
Whenever, in order to prevent the introduction into the United States of any tree, plant, or fruit disease or of any injurious insect, new to or not theretofore widely prevalent or distributed within and

throughout the United States, the Secretary of Agriculture shall determine that it is necessary to forbid the importation into the United States of any class of nursery stock or of any other class of plants, fruits, vegetables, roots, bulbs, seeds, or other plant products from a country or locality where such disease or insect infestation exists, he shall promulgate such determination, specifying the country and locality and the class of nursery stock or other class of plants, fruits, vegetables, roots, bulbs, seeds, or other plant products which, in his opinion, should be excluded.

The promulgation is in effect until withdrawn by the Secretary of Agriculture. This quarantine is still in effect and has been protecting U.S. agriculture from a myriad of pests, including the cause of potato wart. Anyone travelling back into the U.S. from overseas has passed through customs and had to fill out forms ensuring that produce is not being brought in. If you have been on a farm during an overseas visit, you may have to go through a separate inspection and have your footwear cleaned, as I have.

The U.S. to maintains quarantines to protect U.S. agriculture. Potato wart is a particular threat to the U.S. potato industry, as spores of the fungus can remain viable in contaminated soil for many years and can survive digestion by animals. For all practical purposes, viable spores have survived for the lifetime of the current scientists working on the pest. We should consider it a lifetime arrangement, which should not be something entered into lightly.

Introduction of potato wart into the U.S. would have far-reaching consequences. At this point, potato wart exclusion should be preferable to potato wart containment for all of the U.S. potato areas.



INSECTICIDAL MODES OF ACTION

James D. Dwyer
Crops Specialist

Science tells us that humanoids have existed on the planet for about three million years and that insects have existed on the planet for more than 250 million years. During their mutual time on Earth, these two life forms have in many instances been in conflict. This conflict has led to the development of pesticides and more specifically to insecticides.

The development of pesticides has been a long and complicated process. By definition, a pesticide is any substance that is used to control, prevent, destroy, or repel a pest. This would include everything from spreading mud on the body to prevent insect bites, to using modern chemicals.

As potato growers have struggled with the challenges of controlling the Colorado potato beetle, the tools available to the potato industry have continued to change. New chemicals and different methods of using chemicals are continuing to be introduced.

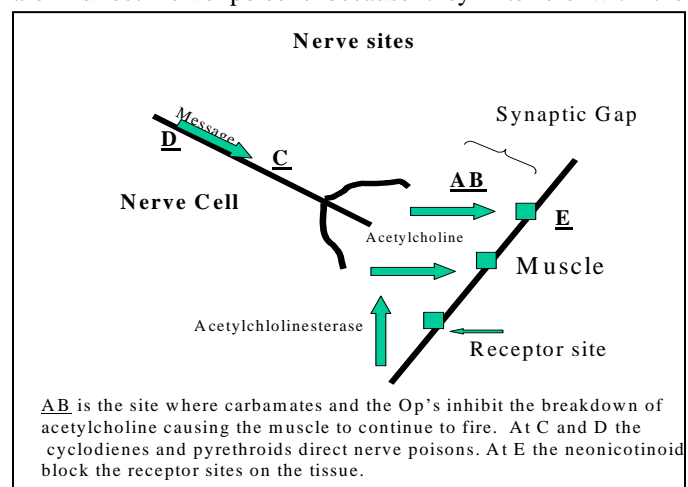
In order to use chemical tools effectively and enable the long-term use of these products, applicators must understand how these products work.

The importance of rotating chemistry to prevent resistance is well known, but it is more important to rotate the *mode of action* of the chemistry being used to manage resistance. Many different chemicals use the same mechanism in order to kill insects. The mechanism or mode of killing is the most important consideration when planning a chemical rotation strategy.

Many insecticides have been developed from research that has focused on killing the insect by preventing nerves from functioning, thereby stopping muscle function.

A nerve cell, whether insect or human, sends an electrical impulse down the length of itself until it reaches the end where there is a physical gap. This gap is called the synapse. It literally is a gap or space between one nerve and the next nerve cell or the muscle that it stimulates. At this point, the transmitting nerve cell releases a chemical called acetylcholine into the synapse, where it diffuses across the gap to the receptor sites located on the receiving cell, thus stimulating the desired reaction. Once this is accomplished, an enzyme breaks the acetylcholine down and stops the stimulation, which in turn stops the next nerve from sending a message, or stops the muscle from moving.

Insecticides such as the carbamates and the organophosphates are indirect nerve poisons because they interfere with the



breakdown of acetylcholine. These products are called cholinesterase inhibitors, and prevent the message to the nerves and muscles from stopping, thus causing a spasm. Muscles and nerves no longer work and death results.

Material such as the cyclodiens and pyrethoids are direct nerve poisons and directly prevent the nerves from functioning.

Imidacloprid (*Admire*) and thiamethoxam (*Platinum*) are in the neonicotinoid class of insecticides, and prevent the message from one nerve to another nerve or muscle from being received by blocking the receptor sites on the receiving side of the cell. This is the only class of chemicals currently being used in the potato industry that impede nerve transmission in this manner.

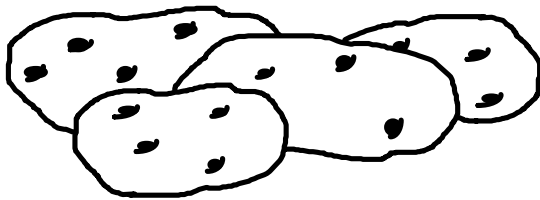
Insecticide Class	Mode of Action
Carbamates Vydate Sevin	Indirect Nerve Poison (cholinesterase inhibitor)
Organophosphates Guthion	Indirect Nerve Poison (cholinesterase inhibitor)
Cyclodiens Thiodan	Direct Nerve Poison (chloride channel interruptor)
Pyrethroids Ambush, Pounce Asana	Direct Nerve Poison (Sodium channel interruptor)
Natural Botanicals Rotenone	Cellular Respiratory Poison
Inorganics Kryocide	General Enzyme poison
Neonicotinoid Admire	Indirect Nerve Poison (Acetylcholine receptor inhibitor)

As growers deal with Colorado potato beetle resistance to and tolerance of currently used insecticides, it is important for growers to understand how the various insecticides work, especially with the introduction of new chemistry.

As the potato industry continues to use neonicotinoids, it will be very important to manage these products so as not to promote a buildup of resistance. Tolerance to neonicotinoids (imidacloprid) has been noted in some areas. It is up to *growers* to practice good stewardship of these products in order to maintain the current and new neonicotinoids.

Good stewardship of these products will mean following label instructions on rates and usage exactly. It will also mean not exposing any Colorado potato beetles to the same product or class of products more than once in a growing season.

Insecticide Class	Mode of Action	Products
Carbamates	Indirect Nerve Poison (cholinesterase inhibitor)	<i>Vydate</i> <i>Sevin</i>
Organophosphates	Indirect Nerve Poison (cholinesterase inhibitor)	<i>Guthion</i>
Cyclodienes	Direct Nerve Poison (chloride channel interruptor)	<i>Thiodan</i>
Pyrethroids	Direct Nerve Poison (sodium channel interruptor)	<i>Ambush, Pounce</i> <i>Asana</i>
Natural Botanicals	Cellular Respiratory Poison	<i>Rotenone</i>
Inorganics	General Enzyme Poison	<i>Kryocide</i>
Neonicotinoid	Indirect Nerve Poison (Acetylcholine receptor inhibitor)	<i>Admire</i> <i>Platinum</i>



THE 2000 ANTI-BRUISE CAMPAIGN

Steven B. Johnson, Ph.D.
Crops Specialist

The Maine Potato Board again financed the Anti-Bruise Campaign for the 2000 harvest. Four inspectors rated 653 tuber samples during September and October. Skinning injury and slight or serious bruise were evaluated by four inspectors working under the supervision of the Agricultural Bargaining Council. The Agricultural Bargaining Council also compiled data for the program.

Bruise damage from the 2000 harvest was higher than last year. The serious bruise and especially the skinning injury are much higher than in 1999, and this is a concern. The low level of Ontario bruise-free potatoes is especially alarming considering the bruise-related damage that occurred last year with this variety. Table 1 lists bruise results from selected varieties. Samples from these eight varieties comprise 68 percent of the total samples. The complete report is available from both the Maine Potato Board and the Agricultural Bargaining Council.

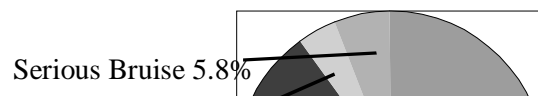
Table 1. Bruise Rating Selected Varieties.

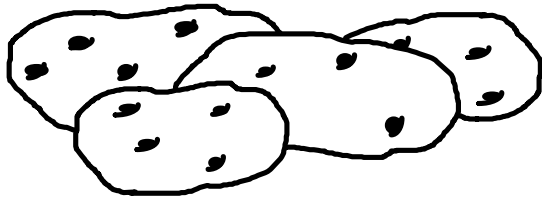
Variety	Percent Bruise-Free	
	1999	2000
Norkotah	97	93
Shepody	95	95
Russet Burbank	99	88
Superior	88	91
Atlantic	79	89
FL1625	97	70
FL1833	79	84
Ontario	91	72

Table 2. Bruise Ratings by Year.

Year	Percent Skinning	Percent Slight	Percent Serious	Sample Number
1994	7.6	5.2	8.4	606
1995	9.9	7.5	6.3	617
1996	2.2	4.9	4.8	519
1997	6.6	3.9	6.0	546
1998	3.5	3.8	2.2	569
1999	1.8	3.4	3.5	415
2000	10.7	4.3	5.8	653

Figure 1. 2000 Anti-Bruise Results





PLANT HORMONES AND PLANT GROWTH REGULATORS: AUXINS

Steven B. Johnson, Ph.D.
Crops Specialist

This is the first installment of a series of articles about plant hormones and plant growth regulators. Plant *hormones* are naturally produced substances that are produced in one part of a plant and transported to other parts, where they exert an effect that is disproportionate to their very small concentrations. Plant hormones exert an influence on plant growth, as do water and nutrients. Hormones exert an influence on plant growth when present in quantities of less than one part per million. *Plant growth regulators* (PGR) are substances synthesized outside of the plant but cause hormone-type activity when applied to plants.

Auxins are major plant hormones that regulate the amount, type, and direction of plant growth. The term “auxin” refers to both naturally occurring substances and related synthetic compounds that have similar effects. Auxins are found in all members of the plant kingdom. They are most abundantly produced in meristems, but are also produced elsewhere, such as in stems and leaves.

The name auxin comes from the Greek word *auxein*, meaning to increase or augment. The most commonly known naturally occurring auxin is indole-3-acetic acid or IAA. IAA is produced in the apical meristems of shoots and diffuses downward, suppressing the growth of lateral buds. IAA plays a major role in stem elongation, migrating from the illuminated portion of the stem to the dark portions and thus causing the stems to grow toward the light.

Auxins are widely used commercially to produce more vigorous growth, to promote flowering and fruiting as well as root formation in plants not easily propagated by stem cuttings, to retard fruit drop, and to produce seedless varieties of some plants by parthenogenetic fruiting. Auxins are defined more by their biological activity rather than by their structure.

History of Auxins

Experiments are routinely performed in high schools studying the process of phototropism or directional response of plant growth toward a light source. In the 1800s Charles Darwin studied the curvature of seedlings toward light. During his experiments he witnessed no bending when seedling tips were cut off, and none when tips were uncut but covered with foil; therefore he concluded that response to light originates in the tips of seedlings.

Biosynthesis of Auxins

Auxins are synthesized by plants from the amino acid tryptophan. Plants can synthesize auxin, then store or transport it in an inactive form. IAA can be bound to other compounds such as sugars (IAA-glucose) or amino acids (IAA-aspartate). These are not biologically active until the IAA is cleaved off by enzymes. IAA is synthesized mainly in the apical meristems occurring at shoot tips; in young leaves; and in developing fruit.

Physiological Effect of Auxins

Auxin plays important roles in a number of plant processes: cell division and elongation, autumnal loss of leaves, the formation of buds and flowers, phototropism, gravitropism, apical dominance, fruit development, abscission and root initiation. Sensitive tissues can double cell elongation in as little as two hours of exposure to IAA.

IAA can move through the phloem from mature leaves, but its primary path of transport is through cells adjacent to the vascular bundles (parenchyma cells). This movement is directional or polar and occurs from apex to base in shoots, and toward tip in roots. The movement across membranes occurs when IAA is slightly chemically modified and then the procedure is reversed.

Auxin also increases the plasticity of the plant cell wall. A more plastic wall will stretch more during active cell growth while its protoplast is swelling. Since only very low concentrations of auxin are required to stimulate cell wall plasticity, the hormone must be broken down rapidly to prevent its accumulation. Plants do this by means of the enzyme indole acetic acid oxidase. By controlling the level of both IAA and IAA oxidase, plants can regulate their growth very precisely.

Auxin also promotes the growth of vascular tissue in stems, and the growth of the vascular cambium itself. It likewise increases fruit growth and acts in other ways to prevent leaves, fruits or flowers from falling off prematurely. In high concentrations, auxins can cause uncontrolled growth and plant death. The molecules of synthetic organic compounds that exhibit auxin activity have certain structural features in common. Two of these are alpha-naphthalene acetic acid (a-NAA); 2,4-dichlorophenoxyacetic acid (2,4-D).

Phototropism

Plant shoots display positive phototropism: when illuminated from one direction, the shoot grows in that direction. The direction of light is detected at the tip of the shoot. Auxin is

synthesized at the tip and translocated down along the shady side of the shoot. Auxin stimulates elongation of the cells on the shady side causing the shoot to bend toward the light.

Gravitropism

Gravitropism is a plant growth response to gravity. Plant shoots display negative gravitropism: a plant shoot will grow up when placed on its side. Roots display positive gravitropism: they grow down. When a root is placed on its side, amyloplasts (organelles containing starch grains) settle to the bottom of cells in the root tip. Auxin sent down from the shoot arrives in the central tissues of the root tip and is then translocated back along the under side of the root. This inhibits root cell elongation. (Roots and shoots differ in their sensitivity to auxin: concentrations of auxin that stimulate shoot growth inhibit root growth.). So the cells at the top surface of the root elongate, causing the root to grow down.

Apical dominance

Growth of the terminal shoot usually inhibits the development of the lateral buds on the stem beneath. This phenomenon is called apical dominance. If the terminal shoot of a plant is removed, the inhibition is lifted, and lateral buds begin growth. The release of apical dominance (pruning the terminal shoot) enables lateral branches to develop, and the plant becomes bushier. The process usually must be repeated because one or two laterals will eventually outstrip the others and reimpose apical dominance. Apical dominance seems to result from the downward transport of auxin produced in the apical meristem. If the apical meristem is removed and IAA applied to the stump, inhibition of the lateral buds is maintained.

Fruit development

Pollination of the flowers of angiosperms initiates the formation of seeds. As the seeds mature, they release auxin to the surrounding flower parts, which develop into the fruit that covers the seeds. Some commercial growers deliberately initiate fruit development by applying auxin to the flowers. Not only does this ensure that all the flowers will “set” fruit, but it also maximizes the likelihood that all the fruits will be ready for harvest at the same time.

Abscission

Auxin also plays a role in the abscission of leaves and fruits. As long as young leaves and fruits keep producing auxin, they remain attached to their stems. When the level of auxin declines, a special layer of cells (the abscission layer) forms at the base of the petiole or fruit stalk. Soon the petiole or fruit stalk breaks free at this point and the leaf or fruit falls to the ground. Fruit growers often apply auxin sprays to cut down the loss of fruit from premature dropping.

Root initiation

Auxins stimulate the formation of adventitious roots in many species. Adventitious roots grow from stems or leaves rather than from the regular root system of the plant. The development of adventitious roots from a cutting can often be hastened by treating the cuttings with auxin.

Effects of auxin on gene expression

Many auxin effects are mediated by changes in the transcription of genes. Auxin enters the cell by active transport through special auxin transporter molecules in the plasma membrane; it binds to molecules; it enters the nucleus and binds to the DNA sequence. The action of auxin on gene transcription is quite similar to the action of steroid hormones in animals.

Synthetic auxins as weed killers

Compounds with the biological activity of auxins can be chemically synthesized: 2,4-dichlorophenoxy acetic acid, or 2,4-*D*, and 2,4,5-trichlorophenoxy acetic acid, or 2,4,5-*T*, are synthetic auxins and are widely used as weed killers. 2,4,5-*T* is 2,4-*D* with a third chlorine atom instead of a hydrogen atom. 2,4-*D* and its many variants are popular because they are selective herbicides, killing broad-leaved plants but not grasses. Normally IAA is imported and exported from plant cells. 2,4-*D* is imported by the same importer, but the exporter is unable to export 2,4-*D* from the cell. This leaves a buildup of toxic levels. A mixture of 2,4-*D* and 2,4,5-*T* was the “agent orange” used during the Vietnam War to defoliate the forest in parts of Southeast Asia. 2,4,5-*T* is no longer used in the U.S.

Auxin-Imitating PGR-Type Products Available for Potato Production

	Cytokinin	GA	IAA
	%	%	%
<i>Trigger</i>	0.012	0	0
<i>Early Harvest PGR</i>	0.09	0.03	0.045
<i>Stimulate</i>	0.009	0.005	0.005

Other PGR-Type Materials Available for Potato Production:

	GABBA	l-glutamicAcid
	%	%
<i>Auxigrow</i>	29.2	29.2

Fertilizers:

<i>ACA Plus (7-0-0)</i>	<i>Crop Set (0-0-0)</i>	
acetic acid	S	1.5%
	Cu	0.2
	Fe	0.5
	Mn	1.5

Spudlines is published by the University of Maine Cooperative Extension to provide information for the Maine Potato Industry. The annual subscription rate is \$5.00. The Educational Committee of the Maine Potato Board provides sponsorship of growers they represent and the allied industry needed to support their growers. For further

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electronically at *sjohnson@umext.maine.edu*

HANNAH HEADS SOUTH

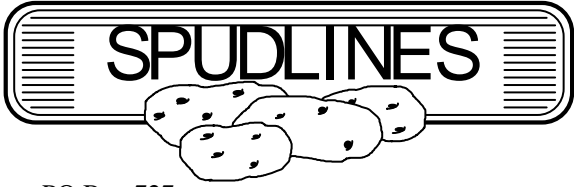
Hannah Carter has left her position as Potato Pest Management Professional with the University of Maine Cooperative Extension to start her doctorate work at the University of Florida. Hannah was a very familiar face at the Presque Isle Extension office; she began her work at the Extension office in 1992 as an Integrated Pest Management field scout. She continued working in the office part-time while completing her bachelor's degree in biology at the University of Maine at Presque Isle. Upon graduation, she accepted a full-time position in the office, and in the summer of 1997 she left to attend the University of Florida to work toward her master's degree. After receiving her master's degree in Agricultural Education and Communication, she found her way back north and accepted a position as Potato Pest Management Professional. In this capacity she provided support to the Crops Specialists in the Presque Isle Office, oversaw the Integrated Pest Management Program for Aroostook County, and assisted in the educational programming efforts of the Presque Isle Extension office.

Hannah's involvement with agriculture has not ended with her job at Extension: her talents are now being used by Florida agriculture. While she is pursuing her doctorate in agricultural leadership, she is working as Program Coordinator for the Florida Leadership Program for Agriculture and Natural Resources. This is an intensive program that develops the leadership capabilities of adults who have been targeted as the future leaders of Florida agriculture.

Hannah commented, "I thoroughly enjoyed my work with Extension, the IPM Program, and with the potato growers in our area. The decision to leave my job and Maine again was not an easy one to make, but I hope to one day return and use my education and experience to benefit the potato industry in Maine."

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