



Crop Notes

May/June, 2007



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ENTOMOLOGY MESSAGE FROM COUNTY DIRECTOR SONYA HAMMOND

I am pleased to welcome Dr. Wai-Ki "Frankie" Lam, who has joined the Monterey County Cooperative Extension staff as a Staff Research Associate (SRA) in Entomology. In his capacity as SRA for Entomology, Dr. Lam will answer questions and conduct research on insects and pest management in Monterey, Santa Cruz, and San Benito Counties.

Frankie received his graduate training in Entomology and Integrated Pest Management. After graduation Frankie worked as a Research Associate in Iowa for USDA, and for the past six years he worked as an Extension Entomologist at Purdue University in Indiana. His research concentrated on the understanding of environmental factors, including biotic and abiotic, on the winter survival and population dynamics of insect pests. He also developed new sampling techniques for insects, studied the effects of chemicals on insect pests and biocontrol agents, and improved the IPM program for insect management on vegetables and melons in conventional and organic practices.

If you have questions on insects and their management methods, please contact Frankie at (831) 759-7359.

FOOD SAFETY AND SALINAS VALLEY CROPS: 1. CHARACTERIZING AND NAMING *E. COLI* 0157:H7

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University of California at Davis, Department of Plant Sciences

The purpose of this article is to help clarify and define terms used in discussions of pathogenic *E. coli*. Future articles will present research-based information on the biology, ecology, and management of pathogenic *E. coli*.

The Fall 2006 outbreak of *E. coli* O157:H7 associated with spinach grown in California will undoubtedly be long-remembered as the key event that triggered a dramatic change in the policy and practice of food safety for fresh leafy greens and other edible horticultural commodities. The extent of the outbreak (205 persons sickened in 26 states, 104 hospitalized, and 3 deaths), while not the largest produce-related outbreak, is tragic in the breadth of its impacts. The unprecedented response of the Food and Drug Administration (FDA), in announcing a widespread consumer advisory against eating spinach, garnered extensive consumer concern and media attention. The dialogue that has resulted among industry, government, non-government organizations, and research groups is also unprecedented for fresh produce. While details may not be fully resolved, all parties can agree that increased effort to reduce the potential for contamination and limit the impact of future outbreaks is



critical. Achieving this goal will depend, in large part, on increasing our understanding of this multi-faceted problem.

E. coli as common intestinal bacterium: *Escherichia coli* (*E. coli*, for short), is a complex bacterial organism that is the subject of extensive research and study. Because of this complexity, it is important to understand a few basics regarding the terminology and biology of this bacterium. *E. coli* is a prominent and essential resident of our gastrointestinal tract. The bacterium was studied and first characterized back in the 1880s by the scientist Theodor Escherich, after

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whom the bacterium would later be named. *E. coli* is but one of a number of bacterial species that naturally inhabit our intestinal systems and enable us to develop and grow normally. These intestinal bacteria are called “enteric bacteria” (“coliform bacteria” is sometimes used to designate the same organisms that share these features).

Escherichia coli belongs to the bacterial family Enterobacteriaceae, which also includes a number of other important human pathogens: *Salmonella*, *Serratia*, *Shigella*, *Yersinia*. Interestingly, this family also includes a few plant pathogens such as *Erwinia* species (causal agents of bacterial soft rot and fire blight diseases). *E. coli* strains from our gastrointestinal systems usually do not cause disease (nonpathogenic) and are therefore called “generic *E. coli*” or “commensal *E. coli*.” (Note, however, that under some circumstances, such as for an immunosuppressed individual, even these nonpathogenic strains can cause problems.) In addition to humans, *E. coli* is likewise a nonpathogenic, commensal intestinal bacterium for many mammals such as cows and pigs.

Isolates and strains of *E. coli*: When microbiologists refer to an “isolate,” they are talking about the growth of a single organism that was obtained by purifying (making sure the culture does not contain a mixture of bacteria) the culture in the lab. The isolate is derived from a single cell or small cluster of sibling bacteria and should not contain more than one type of microorganism. A “strain” refers to cells grown from a purified isolate that is distinct from other strains of the same bacterial species. With the help of a series of laboratory tests and molecular analyses, a particular bacterial strain can be characterized and separated from other strains in the same genus and species.

E. coli as pathogenic bacterium: Some *E. coli* strains can cause serious infections in humans. The bacterium responsible for the recent outbreak involving spinach is one of these pathogenic strains. Such strains have acquired, through mutation or exchange of genetic material, genes that confer the ability to cause a number of diseases; such pieces of DNA are called “virulence genes.” The pathogenic *E. coli* strains that cause diseases in the gastrointestinal tract are called “diarrheagenic” *E. coli*. Researchers noted that there were different types of these pathogenic *E. coli* (called “pathotypes”) and therefore created six different categories of diarrheagenic *E. coli* based on the distinctive features of the various *E. coli* and the diseases they cause; acronyms for these categories are the following: EPEC, EHEC, ETEC, EAEC, EIEC, DAEC. The highly virulent *E. coli* O157:H7 belongs to the EHEC pathotype group (enterohaemorrhagic *E. coli*). The need to use such categories illustrates the diversity found in pathogenic *E. coli* (see Table 1).

Serotypes: Within the EHEC (and any) pathotype category, bacteriologists can distinguish strains based on analysis of two bacterial proteins, O and H. All strains that have the same O factor (lipopolysaccharide antigen) are placed in the same O group. Strains with a common H factor (flagellar antigen) are placed in the same H group. Each combination of O and H constitutes one “serotype.” If a pathogenic *E. coli* strain has the 157th identified O antigen and the 7th identified H antigen, that strain is characterized as serotype O157:H7. If another strain has the 111th identified O antigen, then that individual is placed in the O111 group. There are a great number of identified serotypes. O157:H7 is currently the most common and important EHEC serotype in North America, the United Kingdom, and Japan. Interestingly, different pathogenic serotype groups (O26 and O111, for example) are more important in other countries and regions.

Virulence genes and plasmids: Research reports will refer to virulence genes and plasmids; pathogenic strains need these genetic elements to survive and cause disease in their hosts. The presence or absence of these various elements allows researchers to identify and distinguish different *E. coli* strains. For example, *E. coli* O157:H7 causes disease by attaching to and colonizing host intestinal cells (called “attaching and effacing”). The *eae* gene codes for a protein that is essential for this attachment. Other genes (*stx1* and *stx2* for Shiga-toxin production; see below) enable *E. coli* O157:H7 to produce poisons that are absorbed by intestinal cells and cause severe cellular damage and even death. Most *E. coli* O157:H7 strains also have a “plasmid,” a circular piece of DNA that is separate from the main chromosome (hence it is called “extra-chromosomal”). In general, plasmids carry additional genes that provide some survival advantage to the bacterium, are readily traded and transported between bacteria, and are useful for identifying strains. The plasmid of interest for *E. coli* O157:H7 is well characterized and is named pO157.

Toxins: Toxins are proteins, produced by pathogens such as *E. coli*, which are absorbed by host cells and cause a wide range of damaging effects. Toxins play an important role in diseases caused by *E. coli* O157:H7 and are called “Shiga toxins” (Stx) or “Shiga-like toxins” (SLT) because these substances are related to toxins made by another pathogenic bacterium, *Shigella dysenteriae*. Some researchers call these same toxins “verocytotoxins (VT).” Depending on the type of toxin producing gene (*stx1* or *stx2*) present, EHEC strains can produce Shiga toxin 1 (Stx1), Shiga toxin 2 (Stx2), or both toxins.

E coli O157:H7 belongs to one of several different pathogen groups.

Researchers use “serotypes” to distinguish different pathogenic *E. coli*.

In order to cause disease in humans, the *E. coli* pathogen must contain several genes that make it virulent.

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Several DNA-based tests help identify pathogenic *E. coli*.

DNA technology: Molecular tests are useful in further identifying and differentiating *E. coli* O157:H7 strains. These tests “type” or subdivide strains into different genetic groups based on diversity that exists in virulence genes and other features of the strain’s DNA. Typing is a valuable tool in determining if different strains are related to one another, in seeing if various case patients are linked via a common pathogen, in recognizing an outbreak event, and in conducting trace-back studies that try to identify where contamination originated. Research studies and trace-back reports will refer to several molecular tests, with PFGE and MLST being two commonly used tests today. In the future, additional and perhaps more advanced methods will be used that will provide a better understanding of pathogenic *E. coli* and its detection.

PFGE (pulse-field gel electrophoresis) is a method that takes suspect *E. coli* bacteria, breaks them up to release their DNA, adds enzymes to slice up (“cleave”) the DNA, then processes the DNA pieces so that they cluster into a series of visible bands on a gel membrane. These clusters result in a “fingerprint” that characterizes that particular strain. Different isolates that have the same or similar fingerprint will likely be closely related. PFGE tests were used in

the recent spinach investigation. PFGE patterns from *E. coli* O157:H7 isolated from case patients and recovered bags of spinach were determined to be the same as patterns from *E. coli* O157:H7 found in diverse environmental and animal samples on an implicated ranch in San Benito County.

MLST (multilocus sequencing typing) is a molecular test that sequences fragments of suspect *E. coli* DNA, meaning that the exact composition and order of the DNA components are identified. These “nucleotide sequences” are then compared with known standards, allowing one to identify and compare various strains of *E. coli* O157:H7.

Application and impact: The accurate and precise identification of foodborne pathogens is of the utmost importance to both the consumer, who is possibly sickened by such organisms, and the farmer whose produce might be contaminated in either field or processing facility. Because *E. coli* as an organism is comprised of multiple layers of complex factors and genetics, it is imperative that reports, publications, and discussions precisely and accurately communicate which *E. coli* strains are being considered and identified. Lack of communication regarding such information can lead to misunderstandings and errors.

Familiarity with the terminology used for *E. coli* can help prevent misunderstandings when one reads and hears about *E. coli* research and reports.

Summary of identification factors used with *E. coli* strains (see also Figure 1):

- Within bacterial family Enterobacteriaceae: genera *Escherichia*, *Erwinia*, and others.
- Within genus *Escherichia*: species *E. coli* and others.
- Within species *E. coli*: **pathogenic** and nonpathogenic (=commensal or generic) strains.
- Within pathogenic *E. coli*: **EHEC**, EPEC, ETEC, and other pathotype categories.
- With EHEC: **O157:H7**, O111, O26, and other serotypes.

Characterizing O157:H7 strains:

- Type of Shiga toxin produced (Stx1, Stx2, or Stx1/Stx2)
- Presence of virulence genes (such as *eae*, *hlyA*, *stx1*, *stx2*)
- Presence of other markers unique to this pathogen
- PFGE patterns (analysis of DNA fragments)
- MLST sequences (analysis of DNA components)

Table 1. Various *E. coli* pathotype groups

Pathotype group	Features
EPEC = enteropathogenic <i>E. coli</i>	Mostly found in developing countries.
EHEC = enterohaemorrhagic <i>E. coli</i>	Causes distinct, severe illnesses (HC, HUS).*
ETEC = enterotoxigenic <i>E. coli</i>	Produces one or more enterotoxins (LT, ST).**
EAEC = enteroaggregative <i>E. coli</i>	Causes disease without making LT or ST toxins.
EIEC = enteroinvasive <i>E. coli</i>	Closely related to the pathogen <i>Shigella</i> .
DAEC = diffusely adherent <i>E. coli</i>	Usually affects only young children.

* HC = hemorrhagic colitis; HUS = hemolytic uremic syndrome

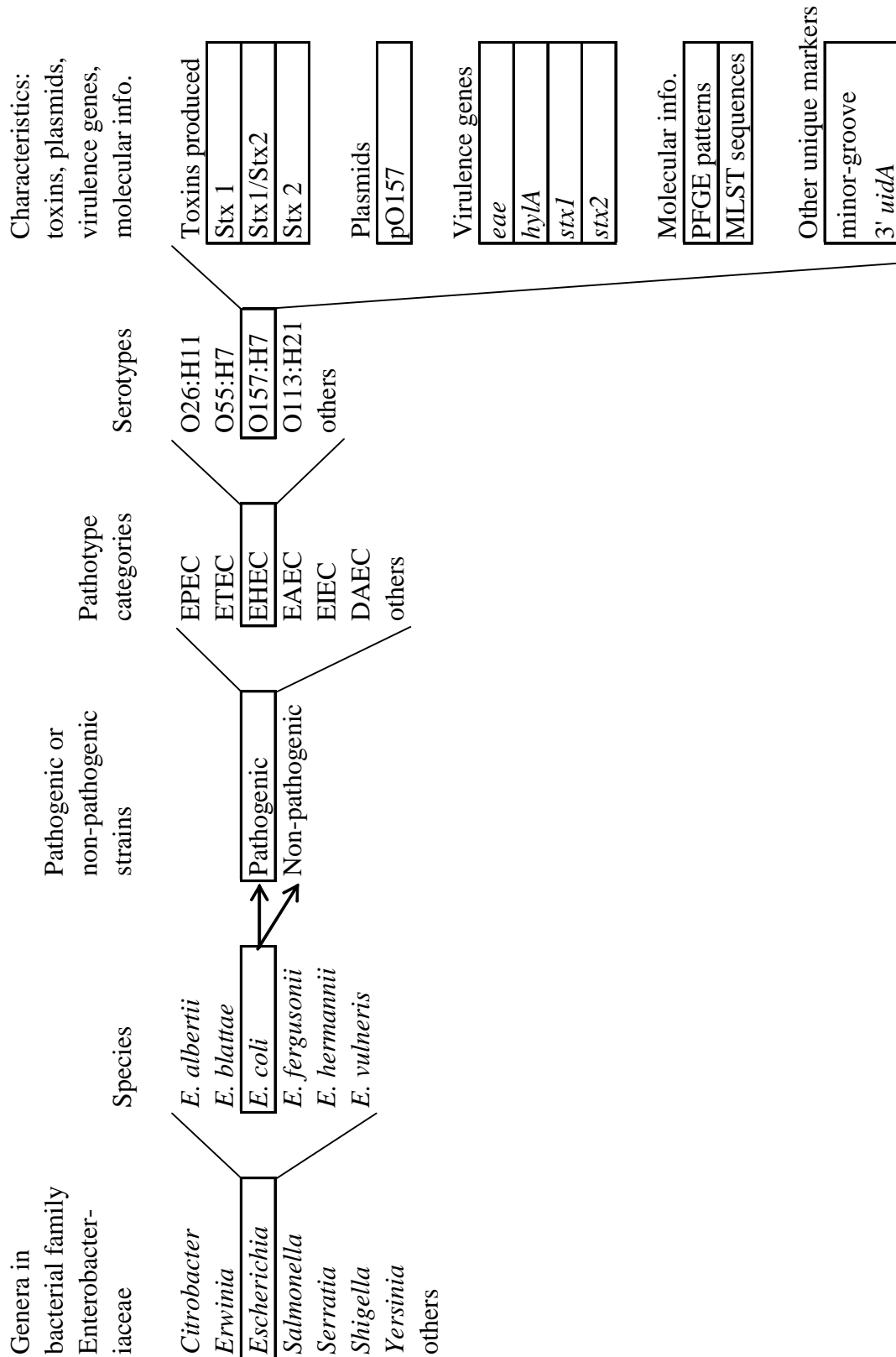
** LT = heat-labile enterotoxin; ST = heat-stable enterotoxin

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Figure 1. Organization of the different *E. coli* features used for identification, characterization, and naming.



The bacterium *E. coli* is a complex species, and pathogenic forms of *E. coli* fall into several categories and serotypes. *E. coli* O157:H7 is further characterized by its genes, toxins, and other features.



CAUSES, DIAGNOSIS AND MANAGEMENT OF “CRUMBLY FRUIT” IN CANEBERRIES

Mark Bolda, Strawberry and Caneberry Farm Advisor
UCCE, Santa Cruz County

Crumbly fruit is not an uncommon occurrence in caneberries in the growing region of the Central Coast.

There are several causes of crumbly fruit in caneberries.

Finally, growers and crop advisors should know that the pattern of crumbly fruit development in the individual caneberry plants as well as the field is very useful in indicating the cause.

Introduction: Crumbly fruit is not an uncommon occurrence in caneberries in the growing region of the Central Coast. The following article will outline the causes of crumbly fruit in caneberries, as well as explain steps that growers may take to diagnose and mitigate this problem. Crumbly fruit are caneberry fruit which do not stay together very easily, and crumble or fall apart when picked or handled. The cause of this unfortunate phenomenon is most often incomplete drupelet set, or drupelets on the fruit which have not filled (drupelets are the individual fruit of which in aggregate compose a raspberry or blackberry fruit). Unfilled and missing drupelets leave spaces in the fruit, causing a collapse of the surrounding drupelets into this space.

There are several causes of crumbly fruit in caneberries:

Poor pollination: Extreme weather conditions, either hot or cold, affect pollination and drupelet set. Moreover, honeybees do not move very well, if at all, in extremities of weather, and this can result in uneven pollination, resulting in a crumbly fruit. The current dearth of wild honeybees does not bode well for growers not employing professionally managed bee hives.

Vascular injury: Because of impeded water uptake in the caneberry plant, vascular injury from severe cold or physical damage can result, among other problems such as wilting and plant stunting, in crumbly fruit. Drupelets of the fruit need water to be filled, and interference with water transport into the fruit will result in unevenness in drupelet fill.

Viruses: Viruses are a common cause of crumbly fruit in caneberries. Raspberry leaf curl, tomato ringspot virus, raspberry bushy dwarf viruses are all causal agents of crumbly fruit in certain varieties. Plants can be infested with viruses at the propagation stage, so it is imperative that growers only work with and purchase plant stock which is certified.

Dryberry mite: Although not a problem on the Central Coast of California, dryberry mite is another cause of crumbly fruit in caneberries. Normally infesting the leaves of the developing primocane, this eriophyid mite can move to fruit during periods of high population. Damage to fruit is distinct from the crumbly fruit sources described above, since fruit infestation results in early ripening of some drupelets, leaving a badly misshapen fruit which is not necessarily crumbly.

Diagnosis of Crumbly Fruit: Finally, growers and crop advisors should know that the pattern of crumbly fruit development in the individual caneberry plants as well as the field is very useful in indicating the cause. Viruses infest the whole plant, and an infested plant would have crumbly fruit up and down the cane. Extremities in weather most often occur over a few days, so only those flowers and subsequent fruit exposed to these conditions will express crumbliness. Crumbly fruit then would be found through the whole field only at a certain height of cane in the case of exposure to poor weather. In the case of improper done plant propagation, problems can be transmitted from an initially low number of plants to large numbers of nursery stock. In the field this will manifest itself as being entire blocks expressing the problem evenly throughout.

Conclusion: The above has been a summary of the causes of crumbly fruit in raspberries and blackberries. Growers are encouraged to contact Mark Bolda with UC Cooperative Extension regarding the identification and management of this and other problems in strawberries and caneberries.



DIRECT SEEDED BROCCOLI POSTEMERGENCE WEED CONTROL TRIAL

Richard Smith, Farm Advisor

University of California Cooperative Extension, Monterey County

Weed control in direct seeded broccoli got a boost in late 2006 with the 24(c) registration of Goal Tender. The registration was for both broccoli and cauliflower, but the bigger use of this material will probably be for direct seeded broccoli. It was registered for use as a broadcast postemergence application at the rates of 4-6 ounce/A, but can be used as a directed spray up to 8 ounces/A. The label stipulates that the material can be applied when direct seeded broccoli has a minimum of four true leaves and that the material should not be mixed with adjuvants, fertilizers or other pesticides.

This registration was a new and welcome technology in the constant battle with weeds in direct seeded broccoli and many growers and PCAs began using it and have been learning the nuances of the material in a wide variety of environmental conditions and weed spectra. Goal tender is particularly effective as a burn down material when the weeds are small (i.e. <2 true leaves); however, once the weeds get past this stage, its effectiveness drops off. This is an important concept when considering the use of this material. One concern that was raised this spring was the lack of efficacy on shepherd's purse. As a result, we conducted a trial in the Greenfield area to examine some of the dynamics that may influence the efficacy of this material.

The trial was conducted in a broccoli field that was not treated with a preemergence herbicide and had a wide spectrum of weeds present. The soil type was Elder Loam and all materials were applied post planting when the broccoli plants had 1.5 to 2.0 leaves on April 3. Each plot was one 40-inch bed by 20 feet long. There were six replications arranged in a randomized complete block design. All materials were applied with a CO₂ backpack sprayer at 30 psi. The gallonage was varied by using one pass of an 8004 nozzle (23 GPA) or four passes of an 8008 nozzle (148 GPA).

This trial taught us a couple of key points about the use of Goal Tender for postemergence weed control on broccoli. Higher gallonage greatly improved the level of weed control over lower gallonage at the 4 or 6 ounce/A rate (Table 1). This was seen dramatically for weeds that are highly susceptible to Goal Tender such as Malva and Hairy Nightshade. It was also true for more difficult to control weeds such as Groundsel and Lambsquarter. In addition, it was true for weeds that Goal Tender only provides partial control such as Sow Thistle and Shepherds Purse. For instance, 6 ounces of Goal Tender only provided a weed control rating of 3.8 at 23 gallons/A spray volume, but received a rating of 7.0 at a spray volume of 148 gallons/A. The actual spray volume used in this trial may not be as important as the concept — **higher spray volume improved weed control**. In addition, notice that the weed control treatments were applied when the broccoli plants had 1.5 to 2.0 true leaves. This was about 30 days after planting and was early enough that the weeds were still small. For instance, Shepherds Purse plants were about the size of a nickel and only a few were the size of a quarter. The smaller nickel-sized Shepherds Purse plants were better controlled than the quarter sized plants which reinforces the other conclusion of this study — **the smaller the weeds the better control by Goal Tender**.

The level of safety provided to the crop in all treatments was acceptable and certainly no greater than AN20. Dow AgroSciences is interested in looking modifying the label to allow Goal Tender applications at the 2 true leaf stage of the broccoli and, if approved, this will greatly facilitate weed control because it will allow the Goal Tender to be applied when the weeds are smaller and more susceptible. There is a great deal of learning and experimentation occurring this year with postemergence applications of Goal Tender on broccoli. I am curious how it is going and please let me know if you have any particular issues with this new technology that we might be able to address with focused research.

Acknowledgements: We are grateful to the cooperation of Sergio Casillas and Armando Martinez for their cooperation in carrying out this study.

This registration was a new and welcome technology in the constant battle with weeds in direct seeded broccoli and many growers and PCAs began using it and have been learning the nuances of the material in a wide variety of environmental conditions and weed spectra.

The smaller nickel-sized Shepherds Purse plants were better controlled than the quarter sized plants which reinforces the other conclusion of this study — **the smaller the weeds the better control by Goal Tender**.

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Higher gallon Hage greatly improved the level of weed control over lower gallonage at the 4 or 6 ounce/A rate (Table 1).

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Table 1. Weed¹ and phytotoxicity² ratings 3 and 10 days after treatment (April 6 and April 13 respectively)

Material	Material /A	Application Volume Gallons/A	Malva		Lambs-quarter		Shepherds Pursue		Hairy Nightshade		Sow Thistle		Groundsel		Phytotoxicity	
			3	10	3	10	3	10	3	10	3	10	3	10	3	10
Goal Tender	4 oz	23	6.8	8.3	5.6	1.9	3.8	2.3	5.2	5.5	4.1	2.5	4.5	2.1	1.0	1.0
Goal Tender	6 oz	23	7.6	8.8	6.7	3.6	5.1	3.8	7.2	8.2	6.0	3.8	5.7	4.0	1.2	1.3
Goal Tender	4 oz	148	9.1	9.8	8.1	5.5	6.5	5.5	8.9	7.5	6.5	5.8	8.7	7.0	1.3	1.8
Goal Tender	6 oz	148	9.6	9.9	9.1	7.8	7.2	7.0	9.2	7.8	7.3	7.1	8.3	7.0	2.0	2.5
AN 20	35 gallons	35	6.2	9.1	1.8	1.1	5.1	2.8	6.2	2.5	3.7	1.3	2.7	0.9	1.8	2.9
Untreated	----	----	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
LSD (0.05)			2.9	0.9	3.8	n.s.	2.7	1.1	2.3	n.s.	1.6	1.6	1.5	2.7	0.7	0.9

1 – Scale: 0 = no weed control to 10 = weeds dead
 2 – Scale: 0 = no crop injury to 10 = crop dead.

Table 1. Weed¹ and phytotoxicity² ratings April 6 (3 DAT)

Material	Material /A	Water Volume Gallons/A	Malva		Lambs Quarter		Shepherds Pursue		Nightshade		Sow Thistle		Groundsel		Phytotoxicity	
			3	10	3	10	3	10	3	10	3	10	3	10	3	10
Goal Tender	4 oz	23	6.8	5.6	3.8	3.8	5.2	4.1	4.5	1.0						
Goal Tender	6 oz	23	7.6	6.7	5.1	7.2	6.0	5.7	1.2							
Goal Tender	4 oz	148	9.1	8.1	6.5	8.9	6.5	8.7	1.3							
Goal Tender	6 oz	148	9.6	9.1	7.2	9.2	7.3	8.3	2.0							
AN 20	35 gallons	35	6.2	1.8	5.1	6.2	3.7	2.7	1.8							
Untreated	----	----	0.0	0.0	0.0	0.0	0.0	0.0	0.0							
LSD (0.05)			2.9	3.8	2.7	2.3	1.6	1.5	0.7							

1 – Scale: 0 = no weed control to 10 = weeds dead; 2 – Scale: 0 = no crop injury to 10 = crop dead.

Table 2. Weed¹ and phytotoxicity² ratings April 13 (10 DAT)

Material	Material /A	Application Volume Gallons/A	Malva		Lambs Quarter		Shepherds Pursue		Nightshade		Sow Thistle		Groundsel		Phytotoxicity	
			3	10	3	10	3	10	3	10	3	10	3	10	3	10
Goal Tender	4 oz	23	8.3	1.9	2.3	2.3	5.5	2.1	1.0							
Goal Tender	6 oz	23	8.8	3.6	3.8	8.2	3.8	4.0	1.3							
Goal Tender	4 oz	148	9.8	5.5	5.5	7.5	5.8	7.0	1.8							
Goal Tender	6 oz	148	9.9	7.8	7.0	7.8	7.1	7.0	2.5							
AN 20	35 gallons	35	9.1	1.1	2.8	2.5	1.3	0.9	2.9							
Untreated	----	----	0.0	0.0	0.0	0.0	0.0	0.0	0.0							
LSD (0.05)			0.9	n.s.	1.1	n.s.	1.6	2.7	0.9							

1 – Scale: 0 = no weed control to 10 = weeds dead; 2 – Scale: 0 = no crop injury to 10 = crop dead.

LIGHT BROWN APPLE MOTH IN CALIFORNIA

Frankie Lam, Entomologist, UCCE, Monterey County

The U.S. Department of Agriculture (USDA) and the California Department of Food and Agriculture (CDFA) issued press release on March 22, 2007 announcing the confirmation of light brown apple moth (LBAM), *Epiphyas postvittana* (Walker) (Lepidoptera: Tortricidae), in California. On May 2, the USDA and the Animal and Plant Health Inspection Service (APHIS) implemented a Federal Order to restrict the interstate movement of certain regulated articles, including nursery stock, cut flowers and greenery, from several counties in California and the entire state of Hawaii to prevent the spreading of the LBAM. On May 3, a total of 1,767 LBAM were detected in Alameda, Contra Costa, Marin, San Francisco, San Mateo, Santa Clara, and Monterey Counties.

The LBAM is native to Australia and was established in Tasmania, New Zealand, England, and Hawaii. Its discovery in California is a new record in the mainland of North America. The larvae of the moth feed on more than 250 plants, including fruit crops, vegetables, ornamentals, and broad-leaf weeds. The LBAM belongs to the leaf-rolling family (Tortricidae) of moth. The greenish larvae construct silken shelters by rolling or webbing the leaves together. The fully-grown larva is 1/3 to 2/3 inch in length and will wriggle vigorously backwards when disturbed. The moth has three to five generations per year in Australia, depending on the Latitude.

For update information of the LBAM in California, the Federal Domestic Quarantine Order on May 2, the biology of the moth, the hosts of the pest, and photos of different life stages, please check the websites listed at the end of this article. If larvae of LBAM are suspected to be on your plants, please contact the CDFA, the local Agricultural Commissioner, the local University of California Cooperative Extension, or call 1-800-491-1899.

1. Light Brown Apple Moth by the California Department of Food and Agriculture
http://www.cdfa.ca.gov/phpps/pdep/lbam_main.htm
2. Light Brown Apple Moth Project by the California Department of Food and Agriculture
http://www.cdfa.ca.gov/phpps/pdep/lbam_profile.htm
3. Plant Health: Light Brown Apple Moth by the USDA, Animal and Plant Health Inspection Service
http://www.aphis.usda.gov/plant_health/plant_pest_info/lba_moth/index.shtml
4. Light Brown Apple Moth: Host List by the California Department of Food and Agriculture
http://www.cdfa.ca.gov/phpps/pdep/LBAM_HostList.pdf
5. Light Brown Apple Moth by Wikipedia, the free encyclopedia
http://en.wikipedia.org/wiki/Light_brown_apple_moth
6. Light Brown Apple Moth by the Zoological Museum, University of Amsterdam
<http://ip30.eti.uva.nl/bis/tortricidae.php?menuentry=soorten&id=197#>
7. Light Brown Apple Moth in Citrus by the New South Wales Department of Primary Industries
http://www.dpi.nsw.gov.au/_data/assets/pdf_file/76206/Light-brown-apple-moth-in-citrus-Primefact-216-final.pdf
8. Light Brown Apple Moth, *Epiphyas postvittana* by the UK moths
<http://ukmoths.org.uk/show.php?id=4388&detail=true>



LBAM adults:
Male (left) and
Female (right)



LBAM egg batch



A near-mature larva on a citrus leaf. Note the webbing on the leaf. Inset: Young LBAM larva on the stem end of a young fruit. Note the feeding damage around the stem end, which will later appear as 'halo' scar on the mature fruit.



Pupation takes place inside the silken feeding shelters. Pupae are red-brown and 10–12 mm long

Pictures courtesy of
www.dpi.nsw.gov.au
primefacts - June, 2006



LETTUCE DIEBACK DISEASE: REVIEW

Steven Koike and Bill Wintermantel

UC Cooperative Extension, Monterey County; USDA-ARS, Salinas

In Spring, 2007, early romaine lettuce plantings were affected by lettuce dieback disease. For the Salinas Valley, lettuce dieback usually occurs much later in the year, so these March and April cases appear somewhat unexpected. This article reviews the symptoms, cause, and control of lettuce dieback disease. This is a relatively recently described disease that has been documented only since the late 1990s.

Romaine cultivars show the most pronounced and serious symptoms of lettuce dieback, though several leaf and butterhead cultivars are also susceptible. Most modern crisphead cultivars are resistant. Symptoms rarely develop until after lettuce is thinned and plants reach rosette stage, but can develop at any developmental stage. It is not uncommon to find symptomatic plants of vastly different sizes in affected areas. Infected lettuce can be severely stunted; mature, diseased plants may fail to develop beyond the 8 to 10 leaf stage (Picture 1). Extensive yellowing occurs on the outermost leaves, with small areas of brown leaf tissue (necrosis) occurring in and between veins, eventually expanding into extensive areas of dead tissue (Picture 2). The younger, inner leaves remain dark green in color, but can be rough and leathery. In some lettuces, particularly romaine, star shaped flecks can be observed within the vein area of the inner leaves when affected leaves are held up to a light source (Picture 3). Because affected plants have such distinctive symptoms, the plants will not meet quality standards and will not be harvested.

The causal agent is Lettuce necrotic stunt virus (LNSV), a recently characterized member of the virus genus Tombusvirus. LNSV is closely related to, but distinct from, another tombusvirus, *Tomato bushy stunt virus* (TBSV), and was identified by researchers at the USDA-ARS in Salinas while studying lettuce dieback. Either LNSV or TBSV can induce symptoms on lettuce. Although rapid diagnostic tests for LNSV and TBSV in lettuce have been developed, consistent detection and confirmation of pathogenic

tombusviruses in lettuce needs to be improved. As a result, pathologists confirm LNSV by using a combination of molecular and traditional approaches that can be time consuming. Lettuce plants are tested initially using molecular methods that analyze for the genetic material of the virus. In addition, indicator plants are rub-inoculated with sap from symptomatic lettuce, allowing tombusviruses to infect these test plants; the indicator species are then tested using serological or molecular methods.

Both LNSV and TBSV are unusual in that they have no known insect, nematode, or fungus vectors. The viruses persist in plant debris, soil, and water and are spread in river and irrigation water, floods, and infested soil and mud. Infection is believed to occur directly through the root. Lettuce dieback symptoms are often severe in tombusvirus-infested fields that have poor leaching and saturated soils.

Both LNSV and TBSV have relatively large host ranges, including tomato, beet, spinach and pepper, but are rarely considered serious problems on non-lettuce hosts. However, both LNSV and TBSV have been known to cause serious economic losses for greenhouse tomato grown hydroponically.

To manage lettuce dieback disease, keep good records of disease occurrence and field conditions, and do not plant romaine in fields having a history of the problem. If it is necessary to plant romaine in such locations, use resistant romaine cultivars; a number of these are now commercially available. Iceberg lettuce can be planted in infested fields because all major commercial cultivars are resistant to lettuce dieback. Preliminary experiments indicated that the disease was not controlled by the application of soil fumigants, which is consistent with lack of a fungal vector. Avoid spreading infested soil and mud, via tractor and other equipment, to clean fields. Do not over-irrigate poorly draining soils, and be aware that flooding can introduce the virus to fields that were previously uninfested.

Picture 1: Lettuce Dieback disease resulting in stunted, yellowed romaine.



Picture 2: Older leaf symptom: brown, necrotic areas on leaves.



Picture 3: Younger leaf symptom: yellow veins and flecks (“star-shaped”).



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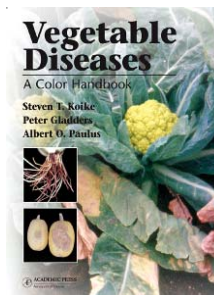
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Two plant pathologists with the University of California, Steven Koike (UC Cooperative Extension, Monterey County) and Albert Paulus (UC River side), have teamed up with a researcher from the United Kingdom to write a new reference book entitled Vegetable Diseases: A Color Handbook. The book emphasizes disease diagnosis, field biology, and other practical aspects of the diseases that affect vegetables. The book cites current research that has been published for each disease. This 448 page book includes over 600 high quality color photographs and should be useful to researchers, technicians, extension personnel, growers, pest control advisors, and students interested in agriculture and plant pathology. Coastal crops are thoroughly covered: lettuce, celery, broccoli, cauliflower, spinach, asparagus, and onion. Because of the interest that Koike has in minor vegetable crops, the book includes disease descriptions for crops such as artichoke, arugula, basil, cilantro, endive, fennel, leafy mustards, Swiss chard, and others. Such specialty crops are rarely covered in other publications. The book is available in North America from Academic Press/Elsevier Books (<http://books.Elsevier.com>).



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MONTEREY COUNTY

Crop Notes



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