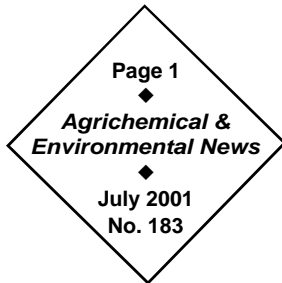


Agrichemical and Environmental News

A monthly report on pesticides and related environmental issues



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Pesticide Illness Data 1995-1999

Bill Mason and Jane Lee, Washington State Department of Health

For more than a decade, the Washington State Department of Health (DOH) has investigated suspected pesticide poisonings. Health care providers are required to report incidents of illness associated with pesticide exposure. The Pesticide Incident Reporting and Tracking (PIRT) Review panel created by the legislature coordinates state pesticide-related investigations.

From January 1, 1995, through December 31, 1999, DOH investigated 1,818 incidents of pesticide poisoning, involving 2,246 individuals (Table 1). An incident is a

pesticide exposure involving one or more individual cases. DOH categorizes the relationship between exposure and symptoms as follows:

TABLE 1			
Annual number of pesticide incidents investigated by DOH.			
Year	Number of investigations (incidents)	Number of persons affected (cases)	Number of definite, probable, or possible cases
1995	396	500	213
1996	398	500	233
1997	363	439	212
1998	390	475	213
1999	271	332	140
Total	1,818	2,246	1,011

Definite

cases: high degree of correlation between a pesticide exposure and resulting symptoms.

Probable cases: similar to definite cases, but lack conclusive objective evidence.

Possible cases: an exposure was present but ambiguity exists between exposure and reported symptoms. Symptoms may be non-specific and other possible etiologies (causes) may be present.

Unlikely cases: symptoms are not believed to be due to the reported exposure, but pesticide exposure cannot be ruled out.

Unrelated cases: either no pesticide exposure occurred (e.g., product was a fertilizer) or the health effects were determined to be caused by another agent.

Asymptomatic cases: exposure occurred but no symptoms resulted.

Unknown cases: insufficient information was available.

...continued on next page

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Of the 2,246 cases, 1,011 (45 percent) were definite, probable, or possible (Table 2), based on the likelihood that symptoms were related to pesticide exposure. This article summarizes pesticide cases investigated by DOH that occurred in non-agricultural settings. A future article will discuss agricultural cases.

TABLE 2			
Agricultural and non-agricultural definite, probable, or possible cases.			
Year	Agricultural	Non-Agricultural	Total Cases
1995	90	123	213
1996	97	136	233
1997	93	119	212
1998	102	111	213
1999	68	102	140
Total	450	561	1,011

Examples of “no application, indirect exposure” include waste collection workers and thrift shop workers exposed to pesticide spills, and a pesticide spill in a freight carrier. Only one incident occurred at a school, which involved an office worker using an insecticide on indoor plants.

Non-Agricultural Occupational

From 1995 through 1999, DOH received reports of 482 cases of suspected pesticide-related illness occurring in the non-agricultural occupational environment. DOH classified 291 of these as definite (40), probable (129), or possible (122). The cases included 150 males and 141 females. The majority of individuals received medical care for their pesticide illness: 141 (86 percent) at emergency rooms, 72 at physicians’ offices, and 38 at walk-in clinics. Two received advice from Washington Poison Center (WPC) and 37 did not seek medical care.

Where Did the Incidents Occur?

The 291 cases occurred in 30 of the 39 counties of Washington. Twice as many occurred in western Washington (197, or 68 percent) as in eastern Washington (93, or 32 percent). Forty-four percent occurred in the Puget Sound counties of King (69), Pierce(33), and Snohomish (25). In Eastern Washington, the counties with the most cases were Yakima(24), Spokane (20), Grant (14), and Benton (13).

The most common sites (45 percent) for non-agricultural occupational pesticide illness were office buildings; both commercial (69) and non-commercial (63) applicators were involved (Table 3). Homes were the second most common (20 percent) location. Exposures in homes resulted from both commercial (39) and non-commercial (19) applications.

Of the 132 cases occurring in offices, 59 percent involved exposure to pesticide residue (Table 4). These cases resulted from indirect exposure to pesticide residue from applications made hours before workers returned to the office. Twenty-three percent of cases in offices involved direct applications. Forty-eight percent of cases in offices involved non-commercial applications.

Thirty-five of the 58 occupational cases in homes or apartments occurred during the application (Table 4). Sixteen cases involved residue or drift exposure. Nineteen occupational cases also occurred in the home when a homeowner made the application and a worker, such as a plumber or builder, was exposed to pesticides at the residence.

TABLE 3				
Site of occupational cases* by commercial or non-commercial application.				
Location	Commercial Application	Non-Commercial Application	No Application (Indirect Exposure)	Total
Office Buildings	69	63		132
Homes/ Apartments	39	19		58
Industrial Sites		20		20
Parks/Golf Courses			7	7
Veterinary		4		4
Other			70	70
Total	108	106	77	291
*Limited to cases with illness classified by DOH as definitely, probably, or possibly due to pesticide exposure.				

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TABLE 4

Location of occupational cases* by type of pesticide exposure.

	Office		Home		Other	Total
	Commercial	Non-Commercial	Commercial	Non-Commercial		
Residue	46	32	5	4	45	132
Drift	11	5	6	1	5	28
Applications	9	22	24	11	23	89
Other	3	4	4	3	28	42
Total	69	63	39	19	101	291

**Limited to cases with illness classified by DOH as definitely, probably, or possibly due to pesticide exposure.*

Occupationally, men (60) were more likely to be involved in incidents from pesticide applications and women (66), from pesticide residue or drift. The routes of exposure in pesticide illnesses are inhalation, dermal, ocular, and ingestion. Seventy-one percent (207) reported one route of exposure and 29 percent (83) reported multiple routes of exposure. Inhalation was the most frequently reported route of exposure and occurred in 74 percent of cases (216).

How Serious Were These Cases?

The majority (81 percent) of cases were considered to have a mild medical outcome (Table 5). These cases frequently involved eye irritation, headache, shortness of breath, cough, and nausea.

Fifty-five cases had moderate symptoms, and one was severe. Twenty-seven of the "moderate severity" cases occurred in the office, 11 in homes, and six on industrial sites. Twelve locations were unknown. The type of activity included applications (22), cleaning/fixing (3), drift (6), residue (14), accident (8), and other (3). A severe case involved a licensed applicator who inadvertently allowed his gloves to become saturated with insecticide.

TABLE 5

Classification by severity.

Severity	Definite	Probable	Possible	Total
Mild	31	104	100	235
Moderate	9	24	22	55
Severe	0	1	0	1
Total	40	129	122	291

Non-Ag, Non-Occupational

From 1995 through 1999, 598 individuals were involved in pesticide-related non-agricultural and non-occupational incidents. Of these, a total of 270 cases were classified as definite (38), probable (84) or possible (148) (Table 6). More women (132, 65 percent) than men (71, 35 percent) over the age of 17 were involved in pesticide illness. Sixty-seven cases involved children less than 18 years of age. Among cases involving children ages 11-17, twice as many were males (9) as females (5). Gender was not a factor among children younger than age 11.

The counties reporting cases most frequently in western Washington were King (54), Pierce (31), Snohomish (22), and, in eastern Washington, were Spokane (25), Yakima (20), and Benton (15). The majority of non-occupational cases occurred in homes or apartments (83 percent), and involved non-licensed applicators (77 percent). See Table 6.

How Did the Exposure Occur?

Most of the non-agricultural and non-occupational pesticide cases were caused by the pesticide's user, through exposure during the application (40 percent) or to residues (25 percent) (Table 6). Inhalation was the most frequently reported route of exposure and occurred in 64 percent of cases; 118 reported inhalation exposure alone and an additional 55 reported inhalation in combination with other routes of exposure. Dermal exposure (35) or in combination with other routes of exposure (59), accounted for 35 percent of the cases. Ocular exposure occurred in 43 cases or 16 percent of the total.

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How Serious Were These Cases?

The majority (78 percent) of cases were considered to have mild medical outcomes (Table 7). The five definite or probable severe cases all occurred at home and involved three children and two adults. The activities associated with these exposures were applications, a spill, an accident, and ingestion by a toddler.

Conclusion

Illness due to exposure to pesticides is a serious public health issue.

Pesticide-related illnesses reported to and investigated by the Washington State Department of Health were reviewed to better understand the circumstances surrounding exposure and resulting health effects. From 1995 through 1999, DOH investigated 482 non-agricultural occupational cases. Sixty percent were confirmed definite, probable or possible cases. These incidents occurred primarily in offices (45 percent) and homes (20 percent) and resulted from exposure to applications or residues. Sixty-three

of the cases in offices involved non-commercial applications. Inhalation was the most common route of exposure (74 percent). Eighty-one percent of the cases had mild medical outcomes. One was severe.

In the non-agricultural non-occupational setting, DOH investigated 598 reported cases of pesticide illness. Forty-five percent (270) were confirmed definite, probable, or possible. The majority (83 percent) occurred in the home and involved non-licensed applicators. Inhalation was the most frequently reported route of exposure. The majority of cases (77 percent) were classified as mild, 20 percent were moderate, and 3 percent were severe. Sixty-seven cases involved children younger than 18 years old.

For more information, please contact Jane C. Lee, PIRT coordinator at (425) 453-1340 or jane.lee@doh.wa.gov.

Type of pesticide exposure by location of non-occupational cases.*						
		Residue	Drift	Applications	Other	Total
Home	Commercial	38	8	2	3	51
	Non-Commercial	20	11	101	40	172
Office	Commercial	2	1	2	0	5
	Non-Commercial	1	0	0	1	2
Industrial Site		0	7	2	0	9
Unknown/ Other		6	1	0	24	31
Total		67	28	107	68	270

***Limited to cases with illness classified by DOH as definitely, probably, or possibly due to pesticide exposure.**

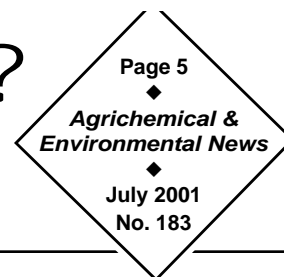
Non-occupational cases* by severity of symptoms.				
Severity	Definite	Probable	Possible	Total
Mild	27	64	119	210
Moderate	7	19	25	51
Severe 1	3	1	4	8
Severe 2	1	0	0	1
Total	44	86	148	270

CORRECTION

In last month's issue (*AENews* No. 181), we presented a recap of the Food and Environmental Quality Laboratory's April 17, 2001, advisory board meeting ("FEQL Advisors Meet," pp. 20-21, Issue No. 182). In listing the members of the board, we inadvertently omitted Ann George, who represents the Washington State Commission on Pesticide Registration on the FEQL board. George is Administrator of the Washington Hop Commission and is a valued advisor to the FEQL. The editorial staff of *AENews* apologizes for this omission.

Bushwhacked by Arsenic?

Part 2: Water, Water Everywhere, and a Drop of Arsenic, Too



Dr. Allan S. Felsot, Environmental Toxicologist, WSU

Amidst the flurry of the presidential pardons in the waning moments of the Clinton administration, the EPA had little generosity toward arsenic in drinking water. With publication of the January 22, 2001, Federal Register (the government's town crier for all rules and regulations), the longstanding 50 µg/L (ppb) maximum contaminant level (MCL) for arsenic was lowered to 10 ppb (11). Congress had mandated EPA to propose a new drinking water regulation for arsenic as part of the reauthorization of the Safe Drinking Water Act in 1996. Furthermore, EPA was to seek research that would help reduce the uncertainty in assessing health risks from exposure to low levels of this naturally occurring, ubiquitous element.

Although the stricter arsenic MCL would not be effective until January 2006, the Bush administration wasted no time in canceling the rule until further review, a move applicable to numerous last-minute regulations imposed by the preceding administration. This cancellation was perceived by some to represent a putative "poison policy" on the part of the new President. A litany of diatribes subsequently flooded the mass media. Experts on drinking water standards were called upon to investigate the matter.

Unfortunately, many of these experts seemed to have political agendas that left the listening public more informed about the divisive nature of partisan politics than about the rationale behind a fivefold drop in the maximum contaminant level for arsenic. To hear the advocates present their case, one would think that the issue was more about big, bad, greedy polluters than about a natural element that we are all exposed to, whether we like it or not.

The real story, which I reveal below, is about the basis for lowering the arsenic standard and the likelihood the new standard would actually protect our health. In this story, the public can learn the extent to which mathematical models are driving policy decisions and gain insight about how EPA (and presumably other government agencies) determines the costs and benefits of regulations.

Laced with Old Arsenic Standards

The political operatives were spinning the proposed hold on the old arsenic MCL as "kiddy poisoning," but the fact is that the drinking water standard had been 50 ppb since its inception by the Public Health Service in 1942 (12). EPA formalized the standard in 1975 under the National Interim Primary Drinking Water Regulation, which was mandated by the Safe Drinking Water Act (SWDA) (2, 12). The 50-ppb standard was based on the acute or short-term toxicity for possible high levels of exposure to arsenic in food and water.

Based on experiences with medicines containing arsenic and epidemiological reports from several countries (Taiwan, Chile, Argentina), the highly respected International Agency for Research on Cancer (IARC) declared arsenic a human carcinogen in 1980 (29). EPA followed suit in the mid-1980s, but the drinking water standard was not changed. Instead, EPA established a water quality criterion of 0.018 ppb under the aegis of the Clean Water Act. The water quality criterion was a guideline for discharges of point-source contaminants (e.g., from factories or mines) into all navigable (surface) waters, which may or may not be sources of drinking water. In contrast to the long-established drinking water standard, the water quality criterion was based on a formal risk assessment process emanating from a 1988 analysis of Taiwanese skin cancer data (12). The remarkably low criterion EPA adopted was pure risk management and designed to protect against one excess case of skin cancer per million people (a probability of 0.0001%).

The World Health Organization was the first agency to blink when, in 1993, it lowered its recommended drinking water standard from 50 ppb to 10 ppb (35). The European Union mandated its members to follow suit and now numerous countries have standards less than 50 ppb (37).

Realizing a serious discrepancy (and confusion) between the water quality criterion of 0.018 ppb and the MCL of 50 ppb, the EPA had been telegraphing

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for years that it desired to lower the MCL for arsenic (12). With the Congressional mandate to “fix” the drinking water standard, EPA commissioned the National Research Council (NRC), an independent research arm of the National Academy of Sciences, to advise them on the adequacy of the risk assessment models they planned to use to justify any actions to lower the standard.

Following the release (and blessing) of the NRC report in 1999 (27), EPA released its proposal to lower the arsenic drinking water standard (9). Under the SDWA, EPA must propose a maximum contaminant level goal (MCLG) for each primary drinking water contaminant (12). The MCLG is strictly health-based and should incorporate a safety factor. For carcinogens, EPA policy dictates an MCLG of zero. The MCL should be set as close as possible to the MCLG, but of course in reality zero doesn't exist. Thus, in releasing its proposal in 2000, EPA was asking for comments on MCLs of 3, 5, 10, and 20 ppb. The final rule, barely meeting the required Congressional deadline, was an MCL of 10 ppb. Even this proposed MCL did not meet EPA's desired goal of no more than a 1 in 10,000 chance of one excess cancer.

The Useless Rat

Normally, EPA decides a chemical causes cancer because rats develop tumors after being practically inundated with the stuff. Rodents, however, are not good models for testing arsenic. They are not nearly as sensitive as humans; in fact, it's downright difficult to give them enough arsenic to produce tumors without killing them first.

Adding insult to lack of rat injury, arsenic in its various forms seems not to be mutagenic (27). Rather, in some unknown way, arsenic interferes with cellular processes involved with repair of DNA damage. Depending on dose, arsenic in human lymphocyte cell cultures has differential effects: at low doses DNA synthesis can be stimulated, but at high doses it is inhibited (23). Interference with DNA synthesis can result in chromosomal breakage that may lead over long periods of time to loss of cell growth control and

eventually tumors. Stimulation of DNA synthesis can lead to proliferation of DNA carrying mutations. The implications of these effects are confusing, compounded by some authors' claims that arsenic may be essential in very low doses for animal health (2).

The recognized pathway for inorganic arsenic detoxification in animals occurs via enzymes known as methyl transferases that attach carbon- and hydrogen-containing methyl groups to arsenic. The transformation to the organic methylated form, which occurs most efficiently in the liver, facilitates the elimination of arsenic in the urine. Methylated forms of arsenic had been believed to be of low toxicity, but recent work with human cell cultures shows that even the methylated forms may induce DNA damage by some unknown mechanism (20, 22). Feeding rats at ridiculously high doses (1500 mg/kg) of methylated arsenic also resulted in lung-specific DNA damage (38).

Given that rats seem non-responsive to arsenic (unless you hit them on the head with a bottle of the stuff), we must turn to human exposure cases for assessing its health hazards. Much of what we know about human sensitivity to arsenic comes from observations of populations living in regions served by ground water having extremely high arsenic concentrations. In fact, the arsenic story is one of the very few where application of epidemiology to chemical exposures in the general population has actually helped definitively associate a wide variety of ailments with exposure to concentrations several fold above 50 ppb.

Smoking Epidemiological Guns

High levels of arsenic in drinking water had been known to cause skin lesions and a type of gangrene in the extremities known as blackfoot disease (BFD). Medical reports of skin cancer caused by ingestion of medicinal arsenic have been traced back to 1888, and breathing of arsenic-laced dust by copper smelter workers or agricultural workers using lead arsenate have been associated with cases of lung cancer (14, 25, 29).

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However, the epidemiological associations between cancer and environmental exposures to a population at large were weak until publication in the late 1960s of a study about southwestern Taiwanese populations with a high prevalence of BFD and non-melanoma skin cancers (i.e., both basal and squamous cell carcinomas) (31). Over 90% of the wells in the subject area had naturally occurring arsenic levels of 150 ppb or greater. The highest incidences of skin cancer were seen in villages using wells with over 600 ppb arsenic. Elevated levels of skin cancer had also been studied in regions of Argentina and Chile (31). As in Taiwan, drinking water was contaminated with levels substantially greater than the current 50 ppb MCL.

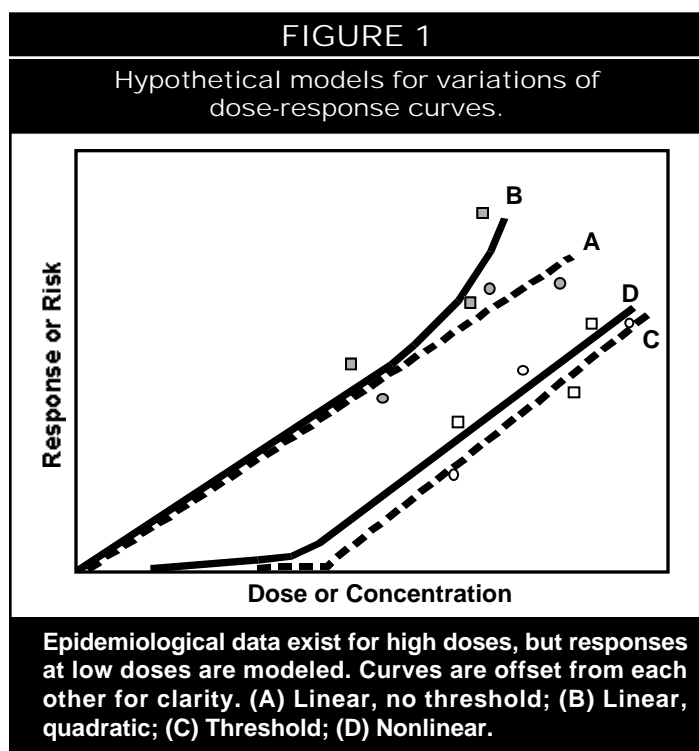
Toxicologists universally agree that arsenic at high levels in drinking water causes skin cancer. The elevated prevalence of skin cancer coincides with BFD or other skin abnormalities. But non-melanoma skin cancer is infrequently fatal, so an urgency to change the MCL was not pushed until reports of elevated incidences in internal cancers started flowing out of Taiwan in the mid 1980s (4, 6). By the late 1990s, Taiwanese researchers had published a host of landmark papers on the relationship between internal organ cancers and arsenic exposure in southwestern Taiwan, the endemic BFD region (3, 5, 7, 36). Bladder cancer was the pathology most strongly associated with high levels of arsenic intake, but lung and liver cancer incidences were also elevated. A specific type of bladder cancer pathology known as transitional cell carcinoma was recently found in another high arsenic-laced-water region in northeastern Taiwan (8). Although each of the Taiwanese reports essentially studied the same affected population from different angles, their conclusions have been bolstered by similar cancer incidence reports from regions in Argentina and Chile with high levels of arsenic in drinking water and elevated incidence of arsenic induced skin diseases (17, 30).

Leaps of Faith

If there is one point of agreement about arsenic, high levels of exposure in drinking water can cause cancer, and the bladder seems to be the most vulnerable

site of attack (27). Like the predictable results from high-dose rodent cancer tests, humans exposed involuntarily to arsenic at extremely high levels (relative to what is typical) also get cancer. Unlike the rat studies, however, the dose levels are not carefully controlled nor actually measured on an individual basis.

Because over 98% of the U.S. population drinks water with arsenic below 20 ppb (9, 13), EPA has the dilemma of translating the human epidemiology data from high arsenic concentrations to low (and more typical) concentrations. Here is where complex mathematics and statistical modeling come into play. When you don't have data to cover low doses, you "reason" these data into existence by assuming that the risk of cancer (i.e., incidence of cancers relative to the whole population) is linear from high doses to low doses. In other words, zero exposure to arsenic results in a zero probability (or zero incidence) of bladder cancer and any incremental exposure above zero results in a directly proportional increase in risk. This model, known as the linear dose-response,



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assumes there is no threshold for cancer induction, regardless of exposure (Figure 1). EPA defaults to the no-threshold assumption when the mechanism of toxicity is unknown (9), despite evidence there may be a threshold. (Another form of this model is the linear-quadratic response curve, where cancer risk begins to increase faster than the incremental increases in dose. Such an effect has been observed in many of the Taiwanese epidemiological studies.)

The no-threshold dose response has been widely criticized as being unrealistic (1, 15, 32). Some researchers maintain that at high concentrations of arsenic, the ability of the liver to detoxify it by methylation may be exceeded resulting in biochemical interactions not occurring at lower doses (1, 2). Other epidemiological studies do not support significantly increased risks for skin cancer or bladder cancer at doses less than several hundred ppb (15, 16). Studies of lower exposures to arsenic in the U.S. compared to southwestern Taiwan have failed to show elevated incidences of skin or bladder cancer (19, 21, 26, 33). Ten years ago, EPA's own Science Advisory Board recommended that the agency use a non-linear dose-response model to characterize risk (9). This model would allow minimal effects at very low doses, but for all practical purposes it is similar to the threshold model (Figure 1).

In its proposals for lowering the MCL, EPA seemed to dismiss any evidence that might support treating arsenic as if there were a threshold. Somehow this defies common sense in that we are continuously exposed to low levels of arsenic: our crops absorb arsenic from soils and drinking water always contains a greater-than-zero concentration of arsenic. To say there is no threshold is to say we are at increased risk for cancer when we eat fruit, vegetables, and cereal. I would hate to think Mom was wrong for telling me veggies were good for me.

In a follow-up discussion of its June 2000 proposal to change the MCL (10), the EPA highlighted a recently published paper (24) that tested several statistical models for re-analyzing the Taiwanese epidemiological data (6, 36) and computing the lifetime risk of dying from bladder cancer at different levels of arsenic exposure. The preferred model employed a technique known as Poisson regression and assumed no threshold in the dose-response curve. To make a long story short, EPA ran the models and presented its risk estimates for bladder and lung cancer at each of several proposed MCLs for arsenic in drinking water (Table 1).

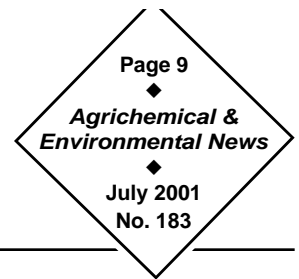
TABLE 1				
Probability (as % chance) of contracting bladder or lung cancer vs. not contracting these cancers at different levels of arsenic consumption in drinking water.^{1/}				
Proposed MCL	Average Chance of Contracting Cancer (%) ^{2/}	Average Chance of Not Contracting Cancer ^{3/}	90th Percentile Chance of Contracting Cancer (%)	90th Percentile Chance of Not Contracting Cancer
3	0.01	99.99	0.02	99.98
5	0.02	99.98	0.04	99.96
10	0.03	99.97	0.06	99.94
20	0.04	99.96	0.08	99.92
50	0.33	99.67	--	--

**1/ Assumes the incidence rate of cancer is the same as the death rate.
 2/ Probabilities for MCLs 3-20 ppb based on (11); 50 ppb based on (24).
 3/ Chances of not contracting cancer are calculated by subtracting the chance of contracting cancer from 100%.**

Probability Judo

At the current MCL there is a 0.33% chance of contracting bladder and lung cancer (Table 1). This chance can be thought of as the proportion of people in a specified population that might contract cancer or the chance an individual might contract it over their lifetime. If the standard were set to 10 ppb, the chance of contracting cancer would drop to 0.03%. While a tenfold decrease in the risk of cancer sounds very impressive, consider that the chance of not getting cancer is 99.67% and 99.97% at the 50 and

Bushwhacked, Part 2, cont.



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10 ppb MCL, respectively. In other words, the chances of not getting cancer from drinking arsenic-tainted water are pretty good even if the permissible level remains at 50 ppb.

How reliable is the estimate of a 0.33% combined chance of contracting bladder and/or lung cancer from drinking water with 50 ppb arsenic? Although the probability was estimated using a mathematical model because no reliable dose-response data exists in the United States, the question can be answered by examining current cancer rate statistics published by the National Cancer Institute. For example, in the database covering the years 1973-1996, the combined incidence/mortality of bladder and lung cancer was equivalent to a 0.126% chance of contracting those diseases (34). So, for the population covered by the database as a whole, the actual chance of contracting cancer was three times less than what EPA predicts would happen if they kept the current arsenic MCL at 50 ppb. Looking at risk more optimistically, a person has a 99.87% chance of not contracting lung or bladder cancer.

How do aging and cumulative exposure figure into the overall picture? If arsenic in water were contributing significantly to increases in bladder and lung cancer in the United States, then as the population aged and drank arsenic longer, it seems logical to predict increases in incidence of these cancers in older individuals. Indeed, the Taiwanese data for internal cancers shows big jumps in disease incidence in age groups 50 and above compared to younger age groups. While no comparable age-based studies have been conducted in the United States, it is important to note that the overall incidence of these cancers is decreasing, even as the median age of the population increases. In fact, the U.S. cancer statistics showed a 0.8% drop in incidence of bladder cancer and a 1.4% drop in incidence of lung cancer for the years 1990-1996. This is good news for all ages.

Finally, one advocacy organization, the Natural Resources Defense Council, stated erroneously that the NRC analysis indicated that one person out of

100 would get cancer from drinking arsenic in water at the current MCL (28). The NRC actually stated that a linear extrapolation of the dose-response curve for Taiwanese cancers would yield a combined bladder and lung cancer risk approaching one in 100. In other words, the risk could be 1%, unless of course you view the glass as half-full and see the chance of not getting cancer as 99%. Obviously, the NRDC has confused a risk estimate with real events. Of the total U.S. deaths, 540,000 were from all types of cancer, giving a probability of 0.2% (540,000 divided by 270 million U.S. residents), which is 2 deaths per 1000 people, clearly fivefold less than the very conservative risk estimate for arsenic-induced bladder and lung cancers. If a 0.2% chance of dying from cancer is scary, then consider that in the United States, in any given year, there is a 10% chance of dying from any cause (18).

The Bottom Line

How much will we pay to achieve a tenfold reduction in risk of contracting cancer (or a 0.3% improvement in the chance of not contracting cancer) by lowering the arsenic standard to 10 ppb? To estimate costs, EPA first examined the currently available feasible remediation technologies and assessed the expense of each (11). Next, they spread the cost around the country. The amount you are projected to pay depends on the size of your water system and where you live. Because the highest arsenic levels are clustered in the west and the northeast (9), residents of those states will probably pay higher bills than average, while those associated with water utilities already meeting the standard should not incur new costs. Finally, EPA calculated the benefits of saving one life from cancer. The agency then offset the arsenic treatment costs by the number of lives saved (i.e., cancers avoided) and calculated a benefit-cost ratio (Table 2).

The estimated total annual cost of implementing the 10 ppb MCL ranged from \$180-205 million (Table 2). On the other hand, EPA's modeling showed nineteen to thirty-six lives saved at an annual savings of \$3.7-5.5 million per cancer case. The benefit-cost ratio was

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estimated to be about 1, a break-even proposition. To reduce the standard any further below 10 ppb would provide an extra margin of safety, but the costs would then exceed the benefits, making any new regulation much less acceptable to cost-conscious legislators. No sense in rocking the boat too much when nearly 95% of community water supplies are already in compliance with the 10 ppb MCL.

In the end, politics rather than science are likely to determine the final MCL for arsenic. Numerous advocates will claim that scientific evidence dictates the standard be lowered to 10 ppb. Yet the chances of not getting cancer from water tainted with 10 or with 50 ppb of arsenic are hardly different. The real story, missed by the organizations supposed to inform the public, is that someone has to choose a mathematical model to estimate cancer risks, and that model is likely to be consistent with the chooser's preconceived notion of what happens at low doses. The outcome is a virtual reality, creating estimates of hazard where no data have gone before.

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TABLE 2				
Estimated annual costs and benefits of lowering the arsenic MCL (11)				
Arsenic Level (ppb)	3	5	10	20
Total Combined Cancer Cases Avoided	6-38	14-45	19-36	19-20
Total National Costs (\$ millions)	697-792	415-172	180-206	67-77
Total Combined Cancer Health Benefits (\$ millions)	214-491	191-356	140-198	66-75
Benefits-to-Cost Ratio	0.6-0.3	0.8-0.4	1.0-0.7	1.2-1.0
Annual Cost Per Cancer Case Avoided (\$ millions)	5.7-13.8	4.7-9.2	3.7-5.5	3.9-4.0

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IR-4 Projects

Input Needed Now for 2002

Dr. Douglas Walsh, State Liaison Representative, USDA/IR-4 Project

The Food Quality Protection Act (FQPA) of 1996 changed the landscape of food safety and pesticide use. We are now in year five of the FQPA era. Revised risk assessments of pesticides—for better or worse—are being ground through the regulatory system. In many cases, pesticide uses are being curtailed or dramatically restricted. As the U.S. Environmental Protection Agency restricts the use of key pesticides, registration of alternative products becomes even more important.

The Interregional Research Project Number 4 (IR-4) was established in 1963 to increase the availability of crop protection chemistries for minor crop producers. IR-4 is a federal/state/private cooperative that aspires to obtain clearances for pest control chemistries on minor crops. (For a complete description of IR-4's workings see "IR-4: Developing and Delivering Pest

Management Solutions for Minor Crop Producers," *AENews* No. 162, Oct. 1999, or log onto the IR-4 national website at <http://pestdata.ncsu.edu/ir-4/>).

Projects Currently Underway

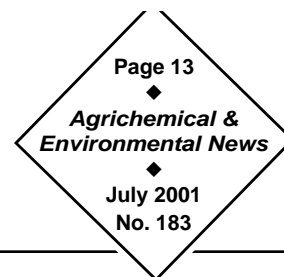
Each year, dozens of new projects are undertaken by IR-4. The new herbicide and insecticide projects initiated in 2001 are shown in the tables beginning below. (Fungicides will be listed in the August issue of *AENews*.) Past IR-4 projects, many of which are still in progress, can be found through the *AENews* website at <http://www2.tricity.wsu.edu/aenews/April00AENews/NewProducts.html>. Remember that crop registrations listed in the table below may not apply to Washington State; please consult the label.

Prioritization Workshop in September
 Each year, IR-4 receives a far greater number of

Herbicide	Trade Name	Crop/Registration
alpha-metolachlor	Dual Magnum	Registered on corn, beans, peas, potato, sorghum, onion, cabbage, and peach. Pending on tomato, grass seed, sugar beet, carrot, spinach, rhubarb, and asparagus. Potential use on garden beets, turnip greens, green onion, broccoli, melons, caneberry, blueberry, and pumpkin.
amicarbazone	Bay MKH	Pending registration on corn and sugarcane.
azafenidin	Milestone	Pending registration on various fruit and nut crops.
BAS 615 H		Registered on small grains.
beflubutamid	UBH-820	Potential use on wheat, barley, rye, and triticale.
bensulfuron methyl	Londax	Registered on rice.
bispyribac sodium	Regiment	Pending registration on rice.
carfentrazone-ethyl	Affinity, Aim	Registered on field corn and wheat. Pending use on sorghum, potato, barley, sweet corn, and oats. Potential use on caneberry.
cinidon-ethyl	Lotus	Registered on barley, wheat, and oats.
clefoxydim	Aura, Tetris	Registered on rice.
clethodim	Select, Prism	Registered on a wide variety of fruit, vegetable, and nut crops.
<i>Collectotrichum aloesporioides</i>	Mallet WP	

PLEASE NOTE: This is a six-column table to be read side-by-side with the following page.

IR-4 Projects, cont.



Dr. Douglas Walsh, State Liaison Representative, USDA/IR-4 Project

requests than the program can pursue, so projects are prioritized, and only the higher-priority projects are guaranteed investigation. The prioritization process takes place at an annual meeting. The IR-4 prioritization workshop for year 2002 projects will take place in Colorado, September 11 through 13, 2001.

Your Participation is Encouraged

As the Washington State Liaison to the IR-4 program and as a Commissioner on the Washington State Commission on Pesticide Registration, I need to know the pest control needs and concerns among the diverse agricultural producers of Washington State.

Submit a PCR Form

The first step toward making a pesticide need known is to submit a Pesticide Clearance Request form (PCR) to IR-4. Anyone can submit a PCR; parties in

Washington State can obtain them from me. I can assist interested parties in prompt submission of the form and I can help bring those needs to the attention of IR-4 at the September meeting.

Individuals or groups wishing to initiate review of a particular crop-chemistry combination should contact me right away. Washington State has a strong reputation for being proactive in pest control efforts. This is facilitated through communication between agricultural producers and university specialists. Please make your pest control needs and concerns known to me so that I can make your voice heard in Colorado.

Dr. Douglas B. Walsh is the Washington State Liaison Representative for IR-4. His office is located at WSU's IAREC facility in Prosser. He can be reached at dwalsh@tricity.wsu.edu or (509) 786-2226.

Registrant	Category	Comments
Syngenta	chloracetanilide	Same spectrum as metolachlor.
Bayer	trazolinone	Applied to the soil preplant or pre-emergence. It also has burndown activity. Soil and burndown activity are primarily on broadleaf weed species.
DuPont	pyridione (PPO inhibitor)	Broad spectrum pre-emergence residual herbicide.
BASF		Particularly active post-emergence on <i>Galium aparine</i> , among other broadleaf species, in small grains.
UBE Industries	phenoxy-butanamide	Post-emergence control of broadleaf weeds.
DuPont		Most broadleaf and sedge weeds.
Valent	sulfonylurea (ALS inhibitor)	Annual and perennial grasses and broadleaf weeds including large and/or herbicide-resistant barnyardgrass.
FMC	aryl triazolinone	Numerous broadleaf weeds, including cocklebur and water hemp.
BASF	isoindoldine (protox inhibitor)	Post-emergence control for broadleaf weeds.
BASF	cyclohexanone (ACCCase inhibitor)	Controls grass weeds.
Valent	cyclohexanone (ACCCase inhibitor)	Strictly a grass herbicide.
Encore Tech.	biopesticide	Naturally occurring fungus that is pathogenic to round-leaved mallow, small flowered mallow, common mallow, and velvetleaf.

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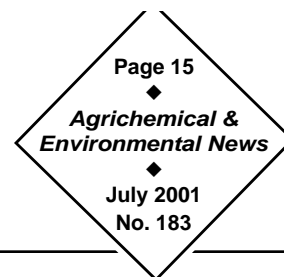
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Dr. Douglas Walsh, State Liaison Representative, USDA/IR-4 Project

Herbicide	Trade Name	Crop/Registration
clodinafop-propargyl	Discover	Pending registration on wheat.
cloransulam-methyl	Firstrate	Registered on soybean.
cyhalofop-butyl	Clincher	Registered on barley, oats, rice, and wheat.
diclosulam	Strongarm	Registered on peanut and soybean.
diflufenzopyr	Distinct	Registered on field and sweet corn and pasture grass.
dimethenamid	Frontier	Registered on dry beans, field corn, popcorn, seed corn, and grain sorghum. Pending use on dry bulb onion and garden beets.
dimethenamid-P	Frontier X-2	Pending use on corn, potato, seed grass, onion, peanut, and soybean.
<i>Drechslera monoceras</i>	MTB-951	Registered for use on rice.
flazasulfuron	Mission	Registered on grape and olive.
florasulam	DE-570	Unknown status on wheat, barley, and oats.
fluazolate	JV 485	Unknown status on wheat.
flucarbazone-sodium	Everest 70 WG	Pending use on wheat.
flufenacet	Axiom	Registered on corn, grass seed, potato, tomato, wheat, pepper, soybean, and onion.
flufenpyr-ethyl	S-3153	Pending registrations on corn. Potential use on snap bean, lima bean, and dry bean.
flumesulam	Broadstrike	Registered on corn. Pending registration on dry bean.
flumiclorac	Resource	Registered for use on corn and soybean.
flumioxazin	Valor 50 WD	Potential use on pome fruit, stone fruit, grape, carrot, and tomato.
fluroxypyr	Starane F	Registered on a wide variety of fruit and vegetable crops.
flurtamone		Unknown status on wheat, barley, oats, and peas.
fluthiacet	Action	Currently registered on soybean. Pending registration on corn and cotton.
foramsulfuron	AE F130360	Pending registration on corn and sugarcane.
glufosinate	Liberty, Rely	Registered on apple, grape, potato, and field corn. Pending use on sweet corn, canola, and sugar beet.
glyphosate	Roundup	Registered on a wide variety of commodities.
halosulfuron	Permit	Registered on field and sweet corn and grain sorghum. Pending use on cucurbits. Potential use on snap/dry beans, asparagus, and potato.
imazamox	Raptor	Pending use on edible legumes and canola.
isoxaflutole	Balance	Registered on field corn. Pending use on sweet corn, wheat, and barley.

PLEASE NOTE: This is a six-column table to be read side-by-side with the following page.

IR-4 Projects, cont.



New Herbicide Listing, pp. 12–17; New Insecticide Listing, pp. 18–21.

Registrant	Category	Comments
Syngenta	pyridylory-phenoxy propionate	Selective post-emergence control of wild oats, annual grasses, and other weeds.
Dow AgroSciences	sulfonamide (ALS inhibitor)	Pre-emergence or post-emergence control of broadleaf annual weeds.
Dow AgroSciences	phenoxy-propionate	Post-emergence graminicide. Reduced risk pesticide.
Dow AgroSciences	sulfonamide (ALS inhibitor)	Can be applied pre- or post-emergence for broadleaf weeds such as morningglory, cocklebur, velvetleaf, and nutsedge.
BASF	pyridine (auxin transport inhibitor)	Controls annual grasses and broadleaf weeds. Sold in a pre-mix with dicamba.
BASF	chloroamide	Annual grasses, broadleaf weeds, yellow nutsedge control.
BASF	chloroamide	Annual grasses, broadleaf weeds, yellow nutsedge control.
Mitsui Chemical	biopesticide carbohydrate	
Syngenta & ISK	sulfonylurea	Active against many grasses and broadleaf weeds with pre- and post-emergence activity.
Dow AgroSciences	triazolo-pyrimidine sulfonanilide	Provides post-emergence of broadleaf weeds, particularly <i>Galium aparine</i> .
Bayer and Monsanto		Pre-emergence control of broadleaf weeds and grasses.
Bayer	sulfonyl-aminocarbonyl-triazolinones	Manages wild oat and green foxtail and certain broadleaf weeds
Bayer	thiadizole or oxyacetamide	Soil applied for annual grasses and some broadleaf weeds.
Valent	PPO inhibitor	Excellent control of velvetleaf and morningglories.
Dow AgroSciences	sulfonamide (ALS inhibitor)	
Valent	N-phenyl-phthalimide derivative	Post-emergence control of velvetleaf.
Valent	N-phenyl-phthalimide derivative	Controls pre-emergence broadleaf weeds with contact activity and residual soil activity.
Dow AgroSciences	picolinic acid	Post-emergence control of annual and perennial broadleaf weeds including volunteer potato, kochia, and nightshade.
Aventis		Pre- and early post-emergence control of annual broadleaf weeds and some grasses.
Syngenta	protox inhibitor	Post-emergence control for velvetleaf, lambsquarter, and other broadleaf weeds. Also desiccant use.
Aventis	sulfonylurea (ALS inhibitor)	Post-emergence control of most annual and perennial grasses.
Aventis		Broad spectrum, non-selective.
Monsanto/Gowan	isopropylamine salt	Controls grasses and broadleaf weeds.
Monsanto/Gowan	sulfonylurea	Controls nutsedge, velvetleaf, cocklebur, and other broadleaf weeds.
American Cyanamid	imidazolinone	Pre- and post-emergence control of annual grasses and broadleaf weeds.
Aventis	isoxazole	Soil applied for many annual grasses and some broadleaf weeds.

PLEASE NOTE: This is a six-column table to be read side-by-side with the preceding page.

IR-4 Projects, cont.

Dr. Douglas Walsh, State Liaison Representative, USDA/IR-4 Project

Herbicide	Trade Name	Crop/Registration
mesotrione		Pending use on field corn. Potential use on sweet corn.
oxadiargyl	Topstar 80 WP	Potential use on vegetables and tree crops.
oxasulfuron	Dynam, Expert	Pending registration for use on soybean.
pelargonic acid		Registered on all crops.
picolinafen	AC 00001	Pending registration for use on barley, rye, triticale, and wheat.
prosulfuron	Peak	Registered on various cereal crops. Pending registration on sugarcane.
propoxycarbazone	Olympus, Attribute	Pending registration for use on rye, triticale, and wheat.
pyraflufen-ethyl	Ecopart	Pending use on wheat and potato.
pyribenzoxim	Pyanchor	Pending registration for use on rice.
pyridate	Tough	Registered on various row crops. Pending registration on alfalfa.
pyrithiobac-sodium	Staple	Registered for use on cotton.
quinclorac	Facet, Paramount	Registered for use on rice, sorghum, and wheat.
quizalofop-ethyl	Assure	Registered and pending registration on a wide variety of crops.
rimsulfuron	Matrix	Registered on field corn, potato, and tomato.
sethoxydim	Poast	Registered on a wide variety of crops.
sulfentrazone	Authority	Registered on grain and row crops.
sulfosulfuron	Maverick	Registered on a variety of grain crops.
tepraloxydim	Equinox, Aramo	Pending registration on sugar beet, cotton, leek, onion, and soybean.
thiazopyr	Visor	Currently registered on several crops. Pending registration on a wide variety of fruit crops.
tralkoxydim	Achieve	Currently registered on wheat and barley.
triasulfuron	Amber	Registered on barley, pastures, rangeland, and wheat.
tribenuron-methyl	Upbeet	Currently registered on sugarbeet. Pending registration on chicory.
trifloxysulfuron	CGA-362622	Pending registration on cotton and sugarcane

PLEASE NOTE: This is a six-column table to be read side-by-side with the following page.

New Herbicide Listing, pp. 12–17; New Insecticide Listing, pp. 18–21.

Registrant	Category	Comments
Syngenta	cyclohezanedione	Pre- and post- emergence management of annual grasses and broadleaf weeds, including sulfonylurea-resistant weeds.
Aventis	oxadiazol	Broad-spectrum weed control, similar to oxidiazinon.
Syngenta	sulfonylurea (ALS inhibitor)	Post emergence use for cocklebur, ragweed, and other broadleaf weeds.
Dow AgroSciences	biopesticide	Contact, non-selective.
BASF	Aryloxpicolinamide (inhibits phytoene desaturase)	Post-emergence control of annual broadleaf weeds.
	sulfonylurea (ALS inhibitor)	Post-emergence control of cocklebur, kochia, lambsquarter, pigweed, ragweed, and velvetleaf.
Bayer	sulfonylaminocarbonyl trizolinone (ALS Inhibitor)	Post-emergence grass weed control and broadleaf weed control in the Cruciferae family.
Nihon Nohyaku	protox inhibitor	Post-emergence herbicide for general non-selective control of weeds or use as dessicant.
Rohm & Haas		Post-emergence material with broad-spectrum activity on annual and perennial weeds including grasses, broadleaves, and sedges.
Syngenta	phenylpyridazine	Controls broadleaf weeds.
DuPont	pyrimidinyl carboxy	Pre- and post-emergence control of a wide range of broadleaf weeds.
BASF	quinoline carboxylic acid	Post-emergence control of annual grasses and certain broadleaf weeds.
DuPont	phenoxy proprionic ester	Post-emergence grass herbicide
DuPont	sulfonylurea (ALS inhibitor)	Annual grass and broadleaf weed control.
BASF	cyclohexanedione (ACCCase inhibitor)	Post-emergence herbicide.
FMC	aryl trazolinone	Controls broadleaf and grass species.
Monsanto/ Gowan	sulfonylurea (ALS inhibitor)	Controls grasses/broadleaf weeds including quackgrass, bromes, and mustards.
BASF	cyclohexandione (ACCCase inhibitor)	Provides post-emergence grass weed control in broadleaf crops, at lower rates. At higher rates, it will control perennials such as johnsongrass and will suppress Bermuda grass.
Rohm & Haas	pyridine	Controls annual and broadleaf weeds, including crabgrass and nutsedge.
Syngenta	cyclohexanedione (ACCCase inhibitor)	Post-emergence control of grass weeds such as wild oats, green and yellow foxtail, and annual ryegrass.
Syngenta	sulfonylurea (ALS inhibitor)	Controls broadleaf weeds.
DuPont	sulfonylurea	Controls broadleaf weeds.
Syngenta	sulfonylurea (ALS inhibitor)	Controls broadleaf weeds.

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IR-4 Projects, cont.

Dr. Douglas Walsh, State Liaison Representative, USDA/IR-4 Project

Insecticide	Trade Name	Crop/Registration
acetamiprid	Assail 701	Pending use on pome fruit, grape, leafy vegetables, and fruiting vegetables.
bifenazate	Floramite, Acramite	Registered on ornamentals. Pending use on pome fruit, stone fruit, grape, and strawberry. Potential use on fruiting vegetables, cucurbits, caneberry, and mint.
buprofezin	Applaud	Pending use on cucurbits and lettuce. Potential use on grapes, stone fruit, pome fruit, and tomato.
<i>Carpocapsa</i> spp. granulosis virus	Pavois	Pending use on pome fruit, grape, leafy vegetables, and fruiting vegetables.
cinnamon oil	Valero	Potential use on grapes.
clothianidin	V-10066	Potential use on apple, pear, and turf/ornamentals.
<i>Cydia pomonella</i>	Virosoft CP4	Registered on various vegetables
cyfluthrin	Baythroid	Registered on potato, sweet and field corn, tomato, alfalfa, sorghum, and carrot. Pending use on dry pea.
cypermethrin	Ammo	Registered on various vegetables.
cyromazine	Trigard	Registered for use on various vegetable crops.
deltamethrin	Decis	Pending use on barley, broccoli, field corn, and popcorn.
diflubenzuron	Dimilin	Registered on mushrooms. Pending use on pear. Potential use on rhubarb and stone fruit.
emamectin benzoate	Proclaim, Strategy	Registered on leafy vegetables. Pending use on fruiting vegetables. Potential use on pome fruit and cranberry.
etoxazole	Baroque	Registered on various fruit crops.
fenoxycarb	Comply	Registered on various crops.
fenpropathrin	Danitol	Registered on a variety of fruit crops.
fenpyroximate	Akari	Pending registration on a variety of fruit crops.
fipronil	Regent	Pending registration on a variety of vegetable crops.
flufenzin		Registered on a variety of fruit and vegetable crops.
hexa-hydroxyl	Bioganic	
hexythiazox	Savey	Registered on a variety of fruit crops.
hydramethylnon	Amdro	
imidacloprid	Admire, Provado, Gaucho	Registered on many fruit and vegetable crops.
indoxacarb	Avaunt, Steward	Contact registrant for information.
iron phosphate	Sluggo	Registered on asparagus, caneberry, cantalope, cucumber, eggplant, and squash.
isomate BTW	Mating disruption Beet Armyworm	Registered on a variety of field crops.
jojoba oil	Detur, E-Rase	
kaolin	Surround	Registered on a wide variety of treefruit and row crops.
lambda-cyhalothrin	Karate, Warrior	Registered on a wide variety of treefruit and row crops.

PLEASE NOTE: This is a six-column table to be read side-by-side with the following page.

New Herbicide Listing, pp. 12–17; New Insecticide Listing, pp. 18–21.

Registrant	Category	Comments
Aventis	chloronicotinyl	Controls with contact and systemic activity via foliar applications. Excellent on sucking pests like aphids and whiteflies.
Uniroyal	carbazate	New mode of action with no cross resistance.
Aventis	thiadiazone	Unique mode of action, inhibits chitin synthesis.
	<i>Granulosis</i> virus	Good activity for nymphal stages of leafhoppers, plant hoppers, scales, and whiteflies
Mycotech	natural product	Controls mites and other insects.
Valent & Takela	neo-nicitinoid	Contact and stomach activity.
Biotepp	biopesticide	Controls codling moth.
Bayer	pyrethroid	Manages cabbage looper, leafhopper, Colorado potato beetle, European corn borer, flea beetle, and potato tuberworm.
FMC	pyrethroid	Activity on cutworms, thrips, leafhopper, weevils, armyworms, lygus bugs, corn earworm, aphids, and beetles.
Syngenta	triazine	Controls leaf miners and maggots.
Aventis	pyrethroid	Manages beetles, bugs, and Lepidoptera.
Uniroyal	IGR	Controls a wide range of leaf-feeding insects.
Syngenta	synthetic avermectin analogue	Effective on larval Lepidoptera.
Valent, Yashima	2,4-diphenyloxaline derivative	Insecticide /acaricide for control of <i>Panonychus</i> spp. and <i>Tetranychus</i> spp. including hexythiazox.
Syngenta	non-neurotoxic carbamate - IGR	Fire ants and a wide range of other insects.
Valent	pyrethroid	Controls aphids, whiteflies, various worms, mites.
Nohon Nohyaku	phenoxyprazole	Controls mites, including twospotted, European red, and citrus rust.
Aventis	phenylpyrazole - broad-spectrum neurotoxin, unique mode of action.	Controls Coleoptera, Lepidoptera, Diptera, Homoptera, Isoptera, and Thysanoptera. Systemic activity.
Chinoin	acaricide	
EcoSMART	plant oil	
Gowan	carboxamide	
BASF	amidinohydrazone	Slow-acting insecticide, formulated as a bait; effective on ants.
Bayer	chloronicotinyl	Primarily effective against sucking insects (aphid, whitefly, scale, etc.) as well as beetles and grubs.
Dupont	oxadiazine - unique mode of action	Controls most major Lepidopteran pest species. Possibly controls plant bugs. Soft on beneficials so it is a good fit with IPM.
W. Neudorff	iron salt	Biopesticide for use on slugs and snails.
Bio Control Ltd.	biopesticide - pheromone	Biopesticide for control of beet armyworm.
IJO Products	natural product	Controls whitefly and powdery mildew.
Engelhard Corp.	clay	Controls various insect and mite pests.
Syngenta	pyrethroid	OP alternative; controls a broad spectrum of insect pests.

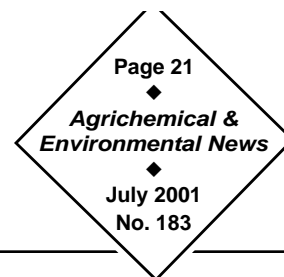
PLEASE NOTE: This is a six-column table to be read side-by-side with the preceding page.

Dr. Douglas Walsh, State Liaison Representative, USDA/IR-4 Project

Insecticide	Trade Name	Crop/Registration
lufenuron	Match	Pending registration on a wide variety of crops.
<i>Mamestra configurata</i>	Virosoft Viral Insecticide	
methoxyfenozide	Intrepid, Runner	Pending registration on a wide variety of crops.
milbemectin	Koromite, Milbeknock	Pending registration on a variety of fruit crops.
novaluron	Rimon	Pending registration on several fruit crops.
<i>Paecilomyces fumosoroseus</i>	PRF 97	
pirimicarb	Pirimor	Pending registration on alfalfa grown for seed, asparagus, celery, and lettuce.
pymetrozine	Fulfill	Pending registration on a wide variety of crops
pyridaben	Pyramite	Registered on apples and pending registration on a wide variety of fruit crops.
pyriproxyfen	Knack, Distance, Esteem	Registered on pome fruit, fruiting vegetables, and stone fruit. Potential use on blueberry.
S1812		Pending registration on cotton, eggplant, groundcherry, ornamentals, pepino, pepper, tomatillo, and tomato.
sodium tetrathiocarbonate	Enzone	Registered on grape.
spinosad	Success, Spintor	Registered on apple, fruiting and leafy vegetables, potato, sweet corn, legumes, wheat, cucurbits, stone fruit, and sorghum. Pending on barley, buckwheat, and turnip greens. Potential use on remaining vegetables, turnips, onion, blueberry, cranberry, grape, strawberry, asparagus, mint, and pear.
tebufenozide	Confirm, RH-5992	Registered on pome fruit, blueberry, caneberry, cranberry, mint, fruiting and leafy vegetables, turnips, and canola. Pending use on sugar beet and grass.
tebupirimphos	Aztec (combo w/cyfluthrin)	Registered on field, sweet, and popcorn. Potential use on lettuce.
tefluthrin	Force	Registered on field, sweet, and popcorn.
thiacloprid	Calypso	Pending registration on several pome fruit.
thiamethoxam	Actara, Platinum	OP alternative pending use on pome fruit, leafy and fruiting vegetables, barley, canola, sorghum, and wheat. Potential use on grape, strawberry, legumes, carrot, stone fruit, and cranberry.
TM 413	Kanremite	Pending registration on a wide variety of crops.
triazamate	Aphistar	Pending registration on a wide variety of crops.
zeta-cypermethrin	Fury, Mustang	Pending registration on alfalfa, sugar beet, sweet and field corn, green onion, popcorn, and sugarcane.

PLEASE NOTE: This is a six-column table to be read side-by-side with the following page.

IR-4 Projects, cont.



New Herbicide Listing, pp. 12–17; New Insecticide Listing, pp. 18–21.

Registrant	Category	Comments
Syngenta	benzoylurea (IGR - chitin inhibitor)	
Biotepp	<i>Granulosis</i> virus	Can be applied as a preventative treatment at planting or a curative foliar treatment for bertha armyworm.
Rohm & Haas	diacylhydrazine (molt accelerating compound)	Similar to tebufenozide in that it only controls Lepidoptera larvae. Better on budworm/bollworm, leafminer and diamondback moth. Excellent fit with IPM programs.
Sankyo, Gowan	complex fermentation product	Excellent miticide and also controls aphids, thrips, leafhoppers, and some Lepidoptera.
Makhteshim-Agan	benzoylphenyl urea (IGR)	Effective against larvae of Lepidoptera, Coleoptera, Homoptera, and Diptera.
Thermo Trilogy	biopesticide	Controls whitefly, aphids, thrips, and spider mites.
Syngenta	OP alternative	Effective IPM material for aphids.
Syngenta	pyridine azomethene	Acaricide.
BASF	pyridazinone	Activity on mites, whiteflies, aphids, mealybugs, leafhoppers, and thrips. A new class of insecticide offering long-term residual control. Good for IPM/resistance management programs.
Valent	pyridene (IGR - selective juvenile hormone analog)	Activity on mite, whiteflies, aphids, mealybugs, leafhoppers, and thrips. Good for IPM.
Valent		Controls scales, whiteflies, thrips, pear psylla, codling moth, and ants. Effective on eggs and immature stages. Excellent for IPM programs.
Entek Corp.	carbon disulfide generator	Controls cutworms, thrips, armyworms, etc.
Dow AgroSciences	Macrocyclic lactone	Controls Coleoptera, Isoptera, Lepidoptera, Thysanoptera, Siphonoptera, and mites. Has low environmental activity, good residual activity, and is safe to many beneficial insects making it ideal for use in IPM programs.
Rohm & Haas	diacylhydrazine	Molt accelerating compound.
Bayer	organophosphate	Controls only Lepidoptera larvae.
Syngenta	pyrethroid	Activity on a wide range of insects, including corn rootworm, wireworm, white grub, and seed corn maggot.
Bayer	second generation neonicotinoid	Broad-spectrum systemic control of sucking and chewing pests, specifically aphids, whiteflies, leafhoppers.
Syngenta	second generation neonicotinoid	Activity on a range of insects including plant bugs, pear psylla, weevils, fruit flies, oriental fruit moth, leafminers, and codling moth. Very safe to bees.
Tomen		Broad-spectrum mite control (no rust mite activity). Easy on beneficials with long residual activity.
Rohm & Haas	carbamate	
FMC	OP alternative	

PLEASE NOTE: This is a six-column table to be read side-by-side with the preceding page.

Foodborne Pathogens Is No One Safe?

Sally O'Neal Coates, Editor of Research Publications, WSU

I knew this year's conference would be different when the hotel hair dryer caught fire in my hand. This is not hyperbole. Day One of the two-day Food Safety Farm to Table Conference in Moscow, Idaho, dawned for me with a tiny orange flame shooting out of the wall-mounted hair dryer in my conference hotel bathroom, followed by a plume of acrid smoke. Was this going to set the tone for my conference experience? Was the Food Safety Conference a safe place to be?

The ninth annual Food Safety Farm to Table Conference was held May 30 and 31, 2001, at the Best Western University Inn Conference Center. A cooperative venture of Washington State University and the University of Idaho, this successful conference is always packed with information presented by a roster of experts in a wide array of fields relating to the safety of the food we eat—from safety on the farm through safe processing and handling to safe consumer practices.

This was my third year at the conference. Looking back, I remembered the 1999 conference, where the focus fell on meat ("Food Safety Conference Focuses on Pathogens," *AENews* No. 160, Aug. 1999). *E. coli* 0157:H7, *Salmonella*, and *Yersinia enterocolitica* were among the hot topics; the "culprits" were largely beef, pork, and poultry. As a vegetarian, I felt pretty safe. And pretty smug. Sure, there were sprouts to contend with, but from a layperson's perspective, it seemed my choices kept me safe. This year, I could run, but I couldn't hide. The bad news was that foodborne pathogens are everywhere and that my hair dryer was spewing flames. The good news is there's a lot we can do to minimize our exposure to foodborne pathogens. And the hotel had another hair dryer.

Pathogens du Jour

Dr. Alan McCurdy, Chair of the Food Science and Human Nutrition department at Washington State University (WSU) opened the conference by introducing Dr. Larry Branen, a food scientist and Dean of the College of Agriculture at the University of Idaho (UI). Dr. Branen shared with the group the history of the Food Safety Conference, which was born in the early

1990s out of a growing recognition of the need for food safety from the farm level right up to the consumer's table. He also praised the cooperative efforts of WSU and UI in co-sponsoring this consistently successful annual event.

The morning's session followed conference tradition by addressing "Pathogens du Jour," today's most talked-about foodborne pathogens and illness trends.

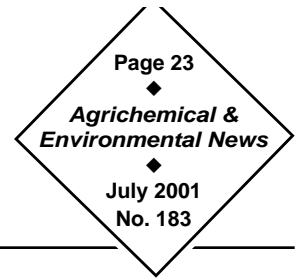
Washington: FBDO Leader

Dave Gifford with the Washington State Department of Health (DOH) addressed the past ten years of foodborne illness data in Washington State. Mr. Gifford pointed out that Washington is a leader in collecting and processing this kind of data and recognizing foodborne illness; in fact, Washington has reported over 20% of foodborne disease outbreaks (FBDOs) nationwide. Of the 906 foodborne disease outbreaks reported in Washington from 1990 to 1999, 55% of the outbreaks (18% of the cases) were of unknown etiology, due in large part from individuals' reluctance to provide stool samples. An overview of the decade showed a sharp rise in reported outbreaks beginning in 1993, the year of major *E. coli* outbreaks. Outbreaks peaked in 1994 and declined steadily through the remainder of the decade with the exception of another increase in 1999. Over these ten years, we saw foodborne illness associated with not only the "usual suspects" like meat and shellfish, but with foodstuffs ranging from rice to ice cream, from fruit juice to iced tea. Bacterial agents were the number one cause of outbreaks, followed by viral agents. Foodhandling factors most often associated with foodborne illness include (in descending order): temperature issues (inadequate hot holding, slow cooling, room temperature storage), inadequate hand washing (the factor most easily controlled and most emphasized by DOH), cross contamination, bare hand contact, ill or infected person, and unclean equipment.

"Just Part of Being a Chicken"

Campylobacter is the leading cause of gastrointestinal (GI) disease in the United States, causing as

Foodborne Pathogens, cont.



Sally O'Neal Coates, Editor of Research Publications, WSU

many as twice the number of diarrheal incidents than the next three leading causes. It may be less well known among the general public because it is seldom associated with outbreaks, but rather with individual cases. The morning's second presentation, by Michael Konkel of WSU's Department of Molecular Biosciences, focused on this organism and WSU's considerable current research into *C. jejuni*, the species responsible for 95% of cases of reported human distress (other important species include *C. coli*, responsible for 2 to 3% of human cases, and *C. fetus*, a strain causing abortion in livestock). *Campylobacter*, relatively unknown and very little studied a decade ago, has become a pathogen du jour because it can lead, in a small percentage of cases, to a type of arthritis and to an autoimmune disorder known as Guillain-Barré Syndrome. Of particular concern, *Campylobacter* has exhibited an alarming propensity for antibiotic resistance. Current research efforts are directed toward more rapid diagnosis and development of a vaccine. The vast preponderance of infections in humans are transmitted via contaminated poultry. As awareness of this organism increases, the battle against *Campylobacter* is being waged on the livestock and food processing fronts. On the consumer level, basic safety procedures including hand washing, prevention of cross-contamination, and thorough cooking help prevent infection from poultry sources. As outbreaks have been associated with raw milk and untreated water, consumers should avoid drinking these as well.

That Wacky Potato Salad

After a short break, during which no poultry or dairy products were served, Dr. Joseph Breese, with the Respiratory and Enteric Virus Branch of the Centers for Disease Control and Prevention (CDC), focused on "Norwalk-like virus" (NLV) as an "under-appreciated" or "emerging" cause of foodborne disease. NLV, which is spread by human fecal contamination, is the most common cause of non-bacterial acute gastroenteritis, accounting for as many as 90% of viral outbreaks. Unfortunately, NLV is very hard to study (notice it doesn't really even have a name of its own yet), because no animal model or cell culture system

exists. Formerly known as "small, round, structured virus" (SRSV), NLV is infrequently detected and reported because no simple detection assays are available for clinical labs. Unlike many other viral agents such as rotavirus, NLV affects all age groups, adults as well as children. Not surprisingly, restaurants and caterers are the most frequently implicated sources of NLV-contaminated food. Other "table" (consumer-level) examples have included deli meats at a university cafeteria, pastries in an Army mess hall, potato salad at a catered luncheon, and an unidentified source on a cruise ship. "Farm" (producer-level) examples have included contaminated oysters and raspberries (so much for vegetarian safety). Nineteen separate strains of NLV have been identified; molecular diagnostics are key for distinguishing among the various strains. As sophistication in detection has increased, it has become clear that "the more you look (for NLV), the more you find." CDC would like to implement an electronic system to identify and compare calcivirus strains in real time. To realize such a system, diagnostic tools would have to become simpler and more affordable.

Dr. Breese's deadpan, rapid-fire delivery caused a peculiar hilarity among conference participants. This conference is the only place I've ever heard a roomful of alleged adults break out in laughter at the mention of "ubiquitous potato salad" as a food pathogen culprit. Then again, this group has the singular ability to pose pertinent questions about molecular analytical techniques one minute and fall into giggling fits over fecal humor the next.

Is Fresher Better?

Dr. Steve Knabel from the Department of Food Science at Pennsylvania State University wrapped up the morning's session with a discussion of the importance and detection of injured cells of foodborne pathogens. "It's my job to restore your faith in LUNCH," he quipped as he began his pre-lunch presentation.

Pathogen cells must be healthy to cause disease. When we employ traditional food preparation pro-

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Sally O'Neal Coates, Editor of Research Publications, WSU

cesses such as heating, cooling, freezing, drying, pickling, and so forth, we injure pathogenic cells to the point where they cannot injure us. With today's trend toward eating fresher, less-processed foods, there is greater potential for cells to have minor injury. Cells with only minor injury may then recover and proceed to reproduce and cause illness. Therefore it becomes important for analysts to be able to detect injured cells as well as healthy ones. Dr. Knabel is developing techniques to detect injured cells.

From an analytical perspective, an injured pathogen is one that can reproduce in a non-selective medium, but not in a selective (salts, acids, metals, antibiotics, dyes, specific nutrients, oxygen) medium. Traditional research has relied on recovery and selected growth of injured pathogens in a matrix of background flora, which can cause confused results. Strategies being developed at Dr. Knabel's lab emphasize use of selective agents that inhibit background flora while allowing recovery of the injured cells targeted for study. Bear in mind that cell research is conducted in labs, while the natural pathogenic processes about which we are all concerned occur in foods and humans. Research is conducted under the assumption that injured cells can in fact repair themselves and proceed to reproduce on foods and in the human gastrointestinal tract.

In addition to detecting injured cells, the techniques under development at Penn State enhance the ability to detect non-injured cells in small numbers. Detecting low numbers of cells is becoming increasingly important as pathogens (including *E. coli* 0157:H7, *Listeria monocytogenes*, and *Campylobacter*) emerge that can cause infection at extremely low doses.

Virtual Val, Statistical Sandy

After lunch, Dr. Jeff Culbertson from the UI Department of Family and Consumer Sciences introduced the topic for the afternoon sessions: Consumer Food Safety and Concerns.

Dr. Val Hillers of WSU's Food Science and Human Nutrition Department and Dr. Sandra McCurdy of UI's

School of Family and Consumer Sciences gave the first two presentations on consumer education. They addressed the broad topics of

What should consumer food safety educators be teaching?

Do consumers practice what they learn from food safety educators?

"Virtual Val" addressed the group via videotape, as she was at home recuperating from hip replacement surgery. The audience missed Dr. Hillers' presence; she has been a key organizer and mainstay of the Food Safety Farm to Table Conference. But her video was informative, engaging, and well received. Dr. McCurdy picked up where the video left off, providing more details and real-life examples of some of the barriers consumers and educators face.

Food safety education has proven most successful when it focuses on factors under the consumer's control. These fall under five major categories: personal hygiene, adequate cooking, cross contamination, safe temperatures, and safe sources. In an effort to prioritize the many safe behaviors consumers can adopt, a formal workgroup of microbiologists, epidemiologists, educators, and policy makers produced a prioritized set of behaviors. This list, based on research literature and ranked and validated by food safety educators, is available in this month's electronic version of *AENews* (on the Internet at <http://www.tricity.wsu.edu/aenews/July01AENews/July01AENews.htm>) or contact me for a copy.

Research has shown that food safety education can result in subjects giving correct answers, but not applying them in full, in practice. (For example, about 95% of consumers claim to wash their hands after using a public restroom; in practice, only about 67% do. Similar disconnects have been shown in food preparation behaviors.) Research is currently underway to determine the success of food safety education at the in-home, consumer level. Because we know that knowledge does not necessarily equate to behavior, results are being sought in terms of verifiable behavior modification.

Sally O'Neal Coates, Editor of Research Publications, WSU

A series of fast-paced fifteen-minute "hot topic" consumer recommendation vignettes rounded out the afternoon.

Perils of Petting

Dr. John Grendon from the Washington State DOH discussed the consumer-level hazard presented by petting zoos and fairs. We don't allow animals in traditional foodservice venues such as restaurants, but many consumers think nothing of letting their children pet a calf, sheep, or llama, then buy an ice cream sandwich or cotton candy. Livestock present hazards including exposure to *E. coli* 0157:H7, *Salmonella*, *Cryptosporidium*, *Campylobacter*, and *Giardia*. Recommendations to reduce the risk of disease transmission include separating animal contact areas from foodservice areas and encouraging hand washing. Animal screening is not useful, as healthy animals can harbor enteric pathogens. Communicating safety information is very difficult, whether the target is operators or users of petting zoos. As for operators, simply finding all the petting zoos is difficult. There is no federal or state regulation of petting zoos, nor any central organization or coalition of petting zoos. Since no central authority is responsible for spreading the word about the potential food safety issues presented by petting zoos, everyone from public health agencies to departments of agriculture to cooperative extension must help spread the word.

Pregnancy Perspectives

WSU's Verna Bergmann explained food safety issues of special concern to pregnant women and explained some of the education efforts planned and currently underway in the Northwest. *Toxoplasma gondii* and *Listeria monocytogenes* are of special concern in pregnant women, as both can cause little or no symptoms in the mother, yet mean significant health risk for the baby. *T. gondii* is a parasite present in cats' feces and in the muscles of meat animals that have consumed food or water contaminated with cat feces. *L. monocytogenes* is a bacteria that has been found in soft cheeses and other raw foods and in refrigerated salads and processed meats such as hot dogs and cold cuts.

Unwanted Salad Dressing

If any self-righteous vegetarians were still in denial at this point in the conference, Dr. Dick Dougherty of WSU's Food Science and Human Nutrition department took care of that with the next presentation, in which he addressed the safety of fresh produce. Consumption of fresh fruits and vegetables has increased about 25% over the past thirty years and so, apparently, has incidence of foodborne illness associated with this consumption. A wide range of produce items, from sprouts to green onions to cantaloupe, have been implicated in outbreaks of a wide range of contaminants, from *Salmonella* to *E. coli* to *Shigella*. How does fresh produce become contaminated? Sources for contamination before and after harvest are many, from air, dust, and water to human, animal, and machine contact. At the consumer level, our best defense is washing (our own hands and our produce) and proper food handling (refrigeration, cooking, surface cleaning, etc.) All heads were nodding emphatically amongst this crowd of hand washing evangelists. But is it foolproof? To bring us back to reality, he showed us a series of close-up photos of various tough-to-wash fruits and vegetables: impossibly veiny cantaloupe rind, deeply textured broccoli florets, segmented and fragile raspberries.

Blaming Bossie

Revisiting *Campylobacter*, Cheryle Becker from the South Central Health District of Idaho summarized a study conducted of 45 cases of *Campylobacter* in seven counties throughout 2000. To oversimplify, "all roads led to cows." The counties studied have extremely high cattle populations; indeed, cow-to-person ratios range from 1:1 to 3:1. The risk factors identified included individuals who worked with cows or other animals or had family members who did, especially those who were relatively new to the cattle/animal trade. Factors that were screened but did not seem to contribute to the problem included travel, water source, day care, poultry (handling or consumption), and raw milk consumption. The major recommendations resulting from the study were increased education regarding hygiene.

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Foodborne Pathogens, cont.

Sally O'Neal Coates, Editor of Research Publications, WSU

Down Home on the Farm

Dan Rice, with WSU's Veterinary Clinical Sciences, finished out the day by discussing the issues faced by farm families with respect to household contamination with human pathogens of livestock origin. Rice's PowerPoint presentation demonstrated graphically the near-impossibility of keeping clean when one works in a farm environment. He shared a recent set of studies involving samples taken from vacuum cleaner bags and electrostatic dust mops used in homes of individuals with and without exposure to livestock operations. Household contamination with *Salmonella* was more common in households involved with livestock. Recommendations for farm families who wish to reduce contamination include leaving work boots outside the home, washing boots with disinfectant, installing hardwood or laminate (as opposed to carpet) in home entryways, and continu-

ing to educate farm workers as to the dangers of contamination.

After a lively discussion of fresh produce and boot washing techniques, Day One of the two-day conference ended and participants adjourned. I presume most (67%?) washed their hands prior to consuming canapés at the ensuing reception, or at least made an effort to consume beverages with preservative or disinfectant qualities. In next month's *AENews*, I will present a summary of the proceedings of Day Two, during which I was somewhat reassured that the world, despite defective hair dryers and vigorous viruses, is still a safe place.

Sally O'Neal Coates, Editor of AENews, has an avid interest in food and fire safety. She can be reached at scoates@tricity.wsu.edu or (509) 372-7378.

Pesticide Container Recycling

DATE	TIME	LOCATION	SPONSOR	CONTACT	PHONE (509)
7/3	8a-10a	Palouse	McGregor Company	Dale Deerkop	878-1321
	1p-3p	Garfield	McGregor Company	Ted Deerkop	635-1591
7/5	8a-10a	Palouse	Dale's Flying Service	Dale Schoefflin	878-1531
	1p-3p	Garfield	Cascade Flying Service	Doran Rogers	635-1212
7/6	8a-11a	Rosalia	Western Farm Service	John Hartley	523-6811
7/9	1p-3p	Pasco	Air Trac	Gerald Titus	547-5301
7/10	8a-10a	Eltopia	Wilbur Ellis	Vern Records	297-4291
	1p-3p	Eltopia	Eastern Wa Spray Serv.	Willis Maxon	297-4387
7/11	8a-10a	Pasco	Pfister Crop Care	Steve Pfister	297-4304
	1p-3p	Connell	B&R Crop Care	Chris Eskildsen	234-7791
7/12	8a-10a	Othello Airport	Conner Flying Inc	Mark Conner	488-2921
	1p-3p	Connell	L&L Farms	Dean Cockran	521-2728
7/13	8a-10a	Bruce	Cenex	Lori Anderson	488-5261
	1p-3p	Bruce	Simplot	Chuck Spytex	488-2132
7/17	8a-10a	Tonasket	Wilbur Ellis	Mel Schertenleib	486-2244
7/18	8a-11a	Brewster	Wilbur Ellis	Brian Hendricks	682-5315
7/19	8a-11a	Chelan	Wilbur Ellis	Brian Hendricks	682-5315
7/20	8a-11a	Cashmere	Wilbur Ellis	Ron Johnson	782-2301
	1p-3p	Wenatchee	Wilbur Ellis	George Craig	663-8753
7/24	8a-11a	Yakima	Wilbur Ellis	Doug Whitner	248-6171
7/25	8a-10a	Granger	Ag Air	Lenard Beierle	865-1970

Washington Pest Consultants Association (WaPCA) contracts with Northwest Ag Plastics to collect and recycle plastic pesticide containers. Containers should be clean and dry, with lids removed. For more information on the program, contact Clarke Brown at (509) 965-6809, Dave Brown at (509) 961-8524, or NW Ag Plastics at (509) 457-3850. A complete schedule through October is on-line at <http://pep.wsu.edu/waste/wapca.html>. For information on a specific collection date or site, call the contact number listed in this table. **THERE IS NO FEE FOR THIS SERVICE.**

Pesticides as "Fertility Drugs" for Mites

Dr. David G. James, Entomologist, WSU

The obvious aim of insecticides and miticides is to kill offending bugs as quickly as possible; formulations and application rates are developed with this aim in mind. However, as we develop and use pesticides that are more targeted to particular pests (as opposed to the broad-spectrum, "annihilate everything" approach), many non-target insects and mites are exposed to less-than-deadly ("sublethal") concentrations of chemicals.

Studies on the sublethal effects of pesticides on insects and mites often show measurable impacts on longevity and reproduction. Length of bug life can be shortened by exposure to sublethal doses of chemicals, perhaps by subtly interfering with normal body maintenance. A shorter bug life may mean a shorter period of reproduction and ultimately a population decline. Some fungicides, while not killing predatory mites, act as sterilants, thus suppressing biological control almost as effectively as predator-toxic insecticides.

Viagra for Mites?

However, not all sublethal effects of pesticides on insects and mites are necessarily detrimental to the species involved or to overall pest management. In 1997, I showed that an Australian predatory mite important in biological control programs increased egg production by 25-54% when exposed to the aphicide imidacloprid (Provado, Admire). Predatory mite populations in orchards sprayed with this aphicide were larger than in orchards where this aphicide was not used.

Is it possible that other species of mites, including pest mites, could respond in a similar way to imidacloprid?

Spider Mite Speculation

In the Yakima Valley, twospotted spider mite (TSM) is an extremely serious pest of hops, producing enormous populations during the summer months.

Imidacloprid, used for control of hop aphids, is usually the first insecticide applied to hops each season. As reported in the February 2001 *AENews* (Issue No. 178, "Which Pesticides Are Safe to Beneficial Insects and Mites?") imidacloprid is harmful to a number of beneficial arthropods important in hops and its use therefore may exacerbate the mite problem by interfering with biological control. But is another, more direct effect also occurring? Does imidacloprid increase egg production in TSM?



Twospotted
Spider Mite

To answer this question, we are conducting a series of laboratory experiments comparing the egg-laying potential of TSM exposed to imidacloprid with non-exposed individuals. Although our experiments are by no means complete, we do have enough information to suggest that there is a "fertility drug" effect of imidacloprid on TSM. In some tests, imidacloprid-sprayed mites produced 36% more eggs during their lifetime than non-treated mites. This translates into twenty to fifty extra eggs per female. Given that females usually represent 60-70% of spider mite populations and egg-to-adult development can take as little as eight to ten days, it is easy to see

how this increased reproductive potential could hasten the buildup of TSM populations. The effect was also apparent when imidacloprid was applied to plants as a systemic application and mites absorbed the compound through feeding on the leaves. Initially, we used the registered field rate of imidacloprid on hops in these tests but are now exploring the effects of lower and higher rates. A full report on this work will appear in these pages in the future.

Et Tu, Fungicides?

TSM on hops also have to endure multiple applications of fungicide, primarily for control of powdery mildew. Do these have any impact on mite populations? Results from a field trial conducted at Prosser in 2000 seem to indicate that some types of fungicides may also have a "fertility drug" effect on mites. A season-long assessment of mite populations in

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Dr. David G. James, Entomologist, WSU

various fungicide-treated plots showed that mite numbers were two to three times larger in plots treated with myclobutanil or trifloxystrobin compared to those in untreated plots or plots treated with spiroxamine or quinoxyfen.* Numbers of mite predators were similar in all plots.

Another Layer

The sublethal effects of pesticides on pests and their natural enemies adds another layer of complexity to understanding population dynamics in crop ecosystems. But it is a layer that we need to understand if we are to gain the most out of "intelligence-based" pest management. There will undoubtedly be opportunities where we can exploit sublethal effects of pesti-

cides on insects and mites in our management programs. Optimal use of chemicals with biologicals is after all, one of the foundation stones of Integrated Pest Management.

David G. James is an Entomologist with WSU's Irrigated Agriculture Research and Extension Center (IAREC) in Prosser. He can be reached at djames@tricity.wsu.edu or (509) 786-2226.

REFERENCE

James, D.G. (1997). Imidacloprid increases egg production in *Amblyseius victoriensis* (Acari: Phytoseiidae). *Experimental and Applied Acarology* 21: 75-82.

*EDITOR'S NOTE: Myclobutanil is registered under a Section 18 exemption in Washington State. Trifloxystrobin was registered under a Section 18 at the time these studies were conducted. Spiroxamine, and quinoxyfen are not registered for use on hops. These particular chemicals were chosen for experimental purposes to represent a range of fungicides with varying mechanisms of action.

Editorial Changes for AENews Federal Register and Tolerance Information to Be Discontinued

Agrichemical and Environmental News (AENews) has served the agricultural producers and other citizens of Washington State and the Pacific Northwest for thirty years. Over the years, editorial coverage has changed to reflect the needs and priorities of our readers. We strive to provide the most meaningful content for our readers, emphasizing original analysis of issues and publication of information to which our readers may not otherwise have access. With this in mind, we have decided to discontinue publishing the Federal Register Summary/Excerpts and the Tolerance Information table that have been regular monthly features in recent years. This information is available from other sources and we would be happy to assist readers in finding those sources. Should you require this assistance, please contact AENews Managing Editor Catherine Daniels at (509) 372-7495 or cdaniels@tricity.wsu.edu or Editor Sally O'Neal Coates at (509) 372-7378 or scoates@tricity.wsu.edu.