Agrichemical and Environmental News

A monthly report on pesticides and related environmental issues

In This Issue

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Admiring Risk Reduction

Does Imidacloprid Have What It Takes?

Dr. Allan Felsot, **Environmental Toxicologist**, **WSU**

With all the scary headlines linking pollution and pesticides, you might think we were in the Pesticide Dark Ages. On the contrary, pesticide technology has been evolving just like cell phones and the Internet. Long before the Food Quality Protection Act of 1996 (FQPA), newer and safer compounds were being developed to replace the old pesticides. Today, the U.S. Environmental Protection Agency (EPA) uses a special euphemism for some of these new chemistries: "reduced-risk pesticides."

EPA considers a pesticide a candidate for reduced-risk status if the pesticide has one or more of the following characteristics in comparison to existing conventional products (39):

- low impact on human health
- low toxicity to non-target organism (birds, fish, and plants)
- low potential for groundwater contamination
- lower use rates (than conventional products)
- low potential for development of pest resistance
- compatibility with integrated pest management (i.e., low toxicity to parasitoids and predators)

Many of these criteria are very subjective, leaving a lot of room for interpretation. Such vague rites of passage for the exclusive "reduced-risk" club might lead a critic to complain that the only real criterion is "we'll know it's a reduced-risk pesticide when we see it." But attaining reduced-risk status is not a popularity contest. The manufacturer (or prospective registrant) of a product must nominate it, then the candidate compound must undergo EPA's rigorous risk assessment process. EPA is the sole judge of reduced-risk status so the onus is on the manufacturer to "deliver the goods," so to speak.

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Admiring Imidacloprid, cont.

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Once a pesticide is officially dubbed "reduced-risk," it is fast-tracked through the laborious registration process. This, of course, is a boon for the registrant.

While only a prospective registrant can nominate a product, sometimes a chemical's biochemical and environmental behavior can speak for itself. Thus, I present for your consideration the case of imidacloprid, an insecticide registered in the United States around 1994. We will look at imidacloprid from both a human health and an ecological perspective.

Identifying the Hazard

The first step in risk assessment is hazard identification. Essentially, this is where researchers expose test subjects to large (including fatally large) single doses of the substance under scrutiny to determine acute toxic effects, and also smaller, repeated doses to determine chronic toxic effects. (Give a rat enough of any substance, natural or synthetic, and it will react eventually.) The result of this step is a laundry list of the various adverse effects possible from exposure to that substance.

Imidacloprid is considered nicotine-like in its biochemical interactions with the nervous system, but it is far less toxic than nicotine (see box, "It's Not Your Granddaddy's Nicotine," opposite). Rats given an oral acute (single) lethal dose of imidacloprid show typical nervous system poisoning symptoms similar to those caused by overdoses of organophosphate insecticides: diarrhea, emaciation, lethargy, labored breathing, lack of coordination, staggering, trembling, and spasms (9) (Table 1). As for humans, the Material Safety Data Sheet (MSDS) for imidacloprid states, "no specific symptoms of acute overexposure are known to occur in humans" (4). Considering the horrors on the MSDS for naturally occurring substances like acetic acid (vinegar) and sodium chloride (table salt), that's a pretty mild statement.

TABLE 1

Hazard Identification and Dose-Response Relationships for Imidacloprid in Acute and Subchronic Toxicity Tests with Rodents (4, 40-43)

Toxicity Test & Exposure Route*	Number of Days Exposed	Doses Tested (mg/kg)†	LD50 or LOAEL (mg/kg)	NOAEL (mg/kg)	Notable Symptoms
Acute Oral	1	Technical formulation	LD50 = 454		Death; typical nervous system effects
		Flowable formulation	LD50 = 4067		
Acute Dermal	1		LD50 > 5000		No effects
Acute Inhalation‡	4 h		LD50 > 0.069 mg/L (aerosols); LD50 > 5.3 mg/L (dust)		No effects
Acute Neurotoxicity Oral	1	0, 42, 151, 307	42§	< 42	Death; decreased rearing behavior, grip strength, response to stimuli, motor activity; increased abnormalities in gait and righting reflex
Subchronic Dermal	21 days; 6 hours per day	1000		1000	No effects
Subchronic Inhalation	28 days; 6 hours per day	0, 0.005, 0.31, 0.191 mg/L as dust	0.31 mg/L	0.005 mg/L	Decreased body weight gain, thymus, and heart weight; increased liver weight; induction of liver detoxification enzymes
Subchronic Diet	90	0, 10, 66, 205	66	10	Decreased weight gain; decreased forelimb grip strength

***Oral exposure refers to a single dose given directly down the esophagus of the animal while diet exposure refers to mixing imidacloprid with the food and allowing the animal to eat freely (ad lib). Dermal exposure refers to shaving the animal's fur and placing the chemical directly in contact with the skin for six hours per day. For inhalation exposures, animals were placed in enclosed chambers and dusts containing imidacloprid were blown in.**

†Average of male and female dose.

‡The highest feasible dose of aerosols in air was 0.069 mg/L; a dust formulation is shown for comparison (from 27).

§Some observable effects on female motor activity but not statistically different than the 0 mg/kg dose level.

Bear in mind that the purpose of an acute oral toxicity study is to determine an LD50 (lethal dose to 50% of the tested rats) and to characterize the array of symptoms during intoxication. Such studies are not very informative about potential hazards following exposure to environmental residues (as the rates of exposure are much higher than would actually occur in a typical real-life situation), but they are useful for warning people who work with purified materials.

In the related acute neurotoxicity study, the objective is to determine whether high (but nonlethal) single doses to rats cause long term neurological impairment, including limb paralysis and/or behavioral impediments.

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The highest doses of imidacloprid (307 mg/kg) resulted in the death of some individuals; survivors had decreased motor skills and response to auditory stimuli. However, symptoms in surviving rats subsided five days after exposure (40). At the lowest dose (42 mg/kg), females but not males exhibited reduced locomotor activity (Table 1).

In other short-term toxicity tests, neither the technical nor the flowable formulation of imidacloprid caused skin or eye irritation or sensitization, whether rats were exposed to single doses or repeated doses (4, 41). Rats exposed to imidacloprid in air for four weeks reacted to the highest doses with decreases in body weight gain, increased liver weights, and induction of liver enzymes responsible for detoxification processes (4) (Table 1).

In chronic exposure tests over two years, dietary exposure resulted in no evidence of cancer (Table 2, page 4) (40-43). In eight of nine tests, imidacloprid caused no mutations or chromosomal breakage. The one test showing chromosomal aberrations was a test-tube-type study (in vitro) and the cells exhibited toxicity, which makes the outcome unreliable for judging gene damage (41).

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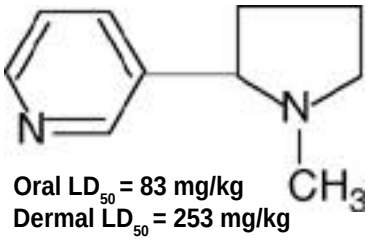
It's Not Your Granddaddy's Nicotine

Imidacloprid has been touted as the synthetic analog of the botanical product nicotine. Actually it is the first commercial pesticide in a family of chemicals originally known as nitromethylene heterocycles but now called neonicotinoids. The association of imidacloprid with nicotine sticks because they have similar biochemical interactions with the nervous system.

Nicotine

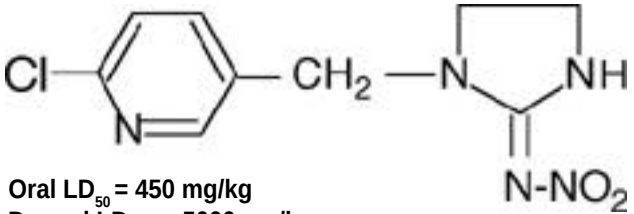


Oral LD_{50} = 83 mg/kg
Dermal LD_{50} = 253 mg/kg

Briefly, both imidacloprid and nicotine bind to nerve receptors called nicotinic acetylcholine receptors (nAChRs). One of several types of protein receptors, nAChRs are embedded in nerve endings in the brain and at the muscles. They bind acetylcholine, a neurotransmitter chemical that is released from adjacent nerve membranes. Acetylcholine crosses a microscopic space (the synapse) separating two nerve endings. When acetylcholine binds to the receptor, the membrane becomes permeable (i.e., more porous) to sodium and potassium ions, thereby kicking off a nervous impulse called the action potential. The action potential is like a wave of electricity that travels down the length of the nerve until it gets to the end where acetylcholine neurotransmitters are released, cross the synapse, and repeat the cycle of binding to the receptor and jumpstarting the action potential.

So, imidacloprid, like nicotine, is a nerve toxin that mimics the action of acetylcholine, and thereby heightens nerve firing with increasing doses. But, unlike nicotine which is extremely toxic in very small doses (smokers, beware!), imidacloprid toxicity to vertebrates is extremely low. Fortunately for mammals, birds, and fish, imidacloprid in contrast to nicotine hardly binds to their nAChRs. Insects, especially sucking bugs, however, are not so lucky. Their nervous systems are not only rich with nAChRs, but imidacloprid is particularly "sticky." The end result is essentially an insect nervous breakdown.

Imidacloprid



Oral LD_{50} = 450 mg/kg
Dermal LD_{50} = >5000 mg/kg

Admiring Imidacloprid, cont.

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Imidacloprid did not affect reproduction of rats in a two-generation study with constant exposure to high levels in the diet (Table 2). Lack of an effect in reproduction studies suggests that imidacloprid is not a hormonally active substance (i.e., an endocrine system disrupter). However, imidacloprid fed to pregnant rats and rabbits at high, maternally toxic doses (100 mg/kg or 72 mg/kg, respectively) caused skeletal malformations in a small percentage of fetuses (Table 2) (4, 40). The occurrence of maternal toxicity during pregnancy makes interpretatng the fetal effects difficult.

Dose-Response Relationships

Once the array of possible adverse effects is delineated during the hazard identification phase, the relationship between dose and effect is examined: How much is safe? First, we determine the harmless dose, the No Observable Adverse Effect Level (NOAEL). The lowest NOAEL among all of the acute and the chronic toxicity tests indicates the most sensitive toxicological effect; these, therefore, become the toxicological endpoints of concern.

For imidacloprid, the acute neurotoxicity test (Table 1) and the chronic dietary carcinogenicity test (Table 2) revealed the most sensitive toxicological endpoints. Although a NOAEL of 5.7 mg/kg/day was definitively established for the carcinogenicity study, the lowest dose tested in the acute neurotoxicity study (42 mg/kg/day) still caused symptomology, albeit statistically non-significant (43).

As toxicological endpoints of concern, NOAELs form the basis for estimating the safe exposure level where there is a reasonable certainty of no harm. This "safe" level is called the reference dose (RfD) and is calculated by dividing the NOAELs by 100 to hedge bets against humans being more susceptible to imidacloprid than rats and to account for the possibility of significant differences in susceptibility among different age groups. Thus, the RfD for acute and chronic toxicity is 0.42 mg/kg/day and 0.057 mg/kg/day, respectively.

The Food Quality Protection Act requires EPA to divide the RfD by an extra safety (or uncertainty) factor of up to tenfold if infants and children are more susceptible or react differently to a given dose than adults (based on developmental and reproductive toxicity studies). The FQPA safety factor may also be applied when neurotoxic symptoms do not subside

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TABLE 2

Hazard Identification and Dose-Response Relationships for Imidacloprid in Developmental, Reproductive, and Chronic Toxicity Tests with Rodents* (40-43)

Toxicity Test & Exposure Route†	Number of Days Exposed	Doses Tested (mg/kg/day)	LOAEL (mg/kg/day)	NOAEL (mg/kg/day)	Notable Symptoms at LOAEL Dose or Higher
Chronic Diet (dog)	365	0, 6, 15, 41/72‡	72	41	Increased oxidative enzyme activity
Chronic/ Carcinogenicity Diet	728	0, 5.7, 16.9, 51.3 (males) 0, 7.6, 24.9, 73 (females)	16.9	5.7	Decreased weight gain in females; increased thyroid lesions in males; no evidence of carcinogenicity
Carcinogenicity Diet (mouse)	730	0, 20, 66, 208, 414 (males); 0, 30, 104, 274, 424 (females)	414	208	Decreased body weight gain, food & water consumption; no evidence of carcinogenicity
Developmental Oral	10	0, 10, 30, 100 (pregnant rats)	10 (mother) 100 (fetus)	30 (fetus)	Mother: decreased body weight gain and food consumption; 5% of fetuses with wavy ribs
Developmental Oral (rabbit)	12	0, 8, 24, 72 (pregnant rabbits)	72	24	Decreased body weight gain and increased abortions; fetus with skeletal abnormalities
Reproductive Diet	Two generations	0, 7.3, 18.3, 52.0 (males) 0, 8.0, 20.5, 57.4 (females)	52	18.3	Decreases in body weight in both generations

***All tests with rats unless otherwise indicated.**

†Diet indicates imidacloprid was mixed into the food and animals ate freely (ad lib); oral indicates the imidacloprid was given in a single dose down the animal's esophagus.

‡After 119 days of exposure at 41 mg/kg without signs of toxicity, the dose was increased to 72 mg/kg.

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after dosing, or when the database is incomplete. The resulting new dose is called the population adjusted dose (PAD) (Table 3).

EPA determined that an extra 3X safety factor should be applied to imidacloprid for several reasons (40-43). First, they felt the database for the acute neurotoxicity study was incomplete, therefore no valid NOAEL had been determined. Second, imidacloprid caused neurotoxic symptoms (decreased motor activity in females) that lasted well beyond dosing. Third, EPA considered that imidacloprid and nicotine bind to the same nerve receptor, and nicotine can adversely affect brain development in fetal rats.

Although EPA claims to use a weight-of-evidence approach, the agency seemed to discount the fact that there was no statistically significant decrease in rat motor activity at the Lowest Observable Adverse Effects Level (LOAEL) of 42 mg/kg/day, and therefore this is likely the NOAEL. Furthermore, biochemical toxicity studies show that imidacloprid binds very poorly to rat acetylcholine nerve receptors, and therefore it is ***unlike*** nicotine in its ability to induce adverse effects. Nevertheless, the EPA has the last word, so the question at hand becomes how much exposure to expect.

TABLE 3

Acute Dietary Exposure and Human Health Risk Characterization for Imidacloprid (43)*

Population Subgroup	PAD† (mg/kg/day)	Dietary Exposure (mg/kg/day)	% of PAD	DWLOC‡ (ppb)
U.S. Population	0.14	0.0322	23	3900
Infants (< 1 y)	0.14	0.049	35	900
Children (1-6 y)	0.14	0.0644	46	760
Females (13-50 y)	0.14	0.0252	18	3600

***Exposure represents the 99th percentile; i.e., a person's diet resulting in the indicated dose is receiving exposures greater than 99% of the rest of the population.**

†Population Adjusted Dose, based on an RfD of 0.42 mg/kg and an FQPA 3X safety factor.

‡Drinking Water Level of Comparison, represents the level of imidacloprid in water that would cause the addition of dietary and drinking water exposure to exceed the PAD. The estimated environmental concentration was 17.4 ppb.

Worst-Case Exposure Scenarios

When calculating human risk, the FQPA mandates that EPA consider routes of exposure besides food. Therefore, possible exposures from drinking water and other uses around the home are added to the theoretical exposure a person might get through eating food containing residues of the subject chemical. Imidacloprid has not yet been routinely analyzed in public databases produced by the USDA, so we do not have actual residue data to work with. Acting conservatively, EPA defaulted to a calculation method known as Theoretical Maximum Residue Contribution (TMRC).

For acute dietary exposure (a one-day exposure), the agency

- assumed that all foods with registered use had residues at the level of the tolerance;
- assumed that all crop acres were treated;
- used the figures for the 99th percentile of consumption from the USDA food consumption database (known as the Continuing Survey of Food Intake by individuals, CSFII) for 1989-1992. The highest level of exposure was to one- to six-year-old children at 0.0644 mg/kg/day (Table 3).

Chronic (lifetime) dietary exposure was handled differently. The residue values were cut back using estimates as to how many acres of crops were actually treated. Figures for median (i.e., 50th percentile) exposures were used. The highest level of exposure was again in one- to six-year-olds, in this case, 0.0097 mg/kg/day (Table 4, page 6).

Imidacloprid analysis has not yet been included in the U.S. Geological Survey (USGS) National Water Quality Assessment program pesticides database yet, so actual residues in water, if any, are unknown. However, EPA always uses a combination of computer models to estimate residues, even when data

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are available. The agency estimated that surface water would have residues of 15.8-17.4 ppb, and ground water would have residues of 1.4 ppb. These are inordinately high levels given the low application rates of imidacloprid products (typically 0.1 to 0.5 lb per acre). However, imidacloprid does have a comparatively high water solubility (500 mg/L), persistence in soil, and mobility potential. (See related article, "Imidacloprid: Insecticide on the Move," p. 13).

Imidacloprid has several registered residential uses. Because there is no evidence of imidacloprid causing toxicity via dermal and inhalational exposure (the usual routes of exposure following home and lawn use), EPA waived consideration of adult residential exposure (42). For children, there was a chance of hand-to-mouth soil and grass ingestion; EPA estimated a worst-case exposure of 0.072 mg/kg/day. If a household pet had been treated for fleas, the estimated exposure to a child engaging in hand-to-mouth behavior would be 0.058 mg/kg.

Risk Characterization

To determine the likelihood of harm following exposure by food, water, or residential use, EPA divides the estimated exposure by the PAD. The result for each exposure source and all sources added together should be less than 100%. For drinking water, however, EPA just estimates the residue level in water that should not be exceeded to maintain an exposure less than 100% of the PAD when all sources of exposure are aggregated.

Results of the various risk characterization calculations showed dietary exposure to imidacloprid was 50% or less of the PAD (Tables 3 and 4, "% of PAD"), indicating no cause for concern. The estimated water concentrations for imidacloprid were 10 to 100 times less than any level of concern (Tables 3 and 4, "DWLOC").

For children's residential exposure, EPA aggregated risk by using the chronic dietary exposure values. The resulting potential exposure to children was less than the PAD and no cause for concern.

In summary, imidacloprid risk to humans seems nil even when all exposure sources are considered. Since imidacloprid poses no hazard by dermal and inhalational exposure, workers should face minimal risk as well.

TABLE 4

Chronic Dietary Exposure and Human Health Risk Characterization for Imidacloprid (43)

Population Subgroup	PAD* (mg/kg/day)	Dietary Exposure (mg/kg/day)	% of PAD	DWLOC† (µg/L)
U.S. Population	0.019	0.0046	24	490
Infants (< 1 y)	0.019	0.0072	38	120
Children (1-6 y)	0.019	0.0097	51	92
Females (13-50 y)	0.019	0.0034	18	450

***Population Adjusted Dose, based on an RfD of 0.057 mg/kg and an FQPA 3X safety factor.**

†See Table 3 for explanation of DWLOC; the concentration was estimated as 15.8 ppb in surface water and 1.4 ppb in ground water.

Skeletons in the Closet? Degradation Products

Although imidacloprid seems pretty innocuous to mammals, largely because it does not bind nerve receptors sufficiently to trigger nervous activity, one of its known degradation products, desnitro imidacloprid (DNIMI) behaves like a mirror image. DNIMI binds very strongly to mammalian nerve receptors but not to insect nerve receptors. It is not toxic to insects, but it is about four to five times more toxic than imidacloprid to mice (6, 38). Such damning information could put the skids on imidacloprid's hopes for reduced-risk status, but digging into the details shows this concern may be a tempest in a teapot.

First, the ability to bind to nerve receptors tends to correlate with toxicity, but it is far from a perfect correlation. Absorption potential and metabolism rate will modify toxicity. When the toxicity of DNIMI was compared to imidacloprid, mice were injected directly with the substances. By this route of exposure, the LD50 of imidacloprid dropped to about 50 mg/kg, and that of DNIMI was about 10 mg/kg (38). Toxicity of

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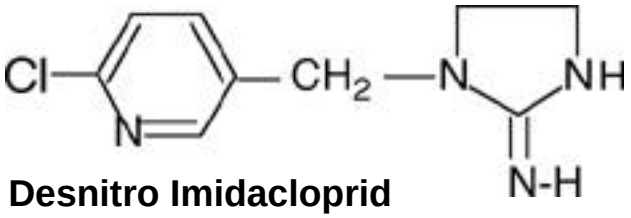

Desnitro Imidacloprid (DNIMI)

DNIMI was never measured by oral or dermal exposure, the most likely routes in the environment.

Second, if DNIMI were a significant product of metabolism, the rat would have been exposed to it during high-dose toxicity tests by oral exposure. Conceivably, the rats react at high doses because of DNIMI generation. However, rat metabolism studies submitted for EPA's risk assessment indicated very little, if any, DNIMI was generated (41).

Third, metabolism of imidacloprid is very quick. About 90% of the dose of imidacloprid is excreted within twenty-four hours, along with any possible metabolites. After forty-eight hours, residual material (less than 0.5% of the original dose) was found in the liver, as would be expected from the main organ of detoxification, but not in the brain (41). Furthermore, if DNIMI ever made it into the brain, biochemical studies show it is about as likely to bind to nerve receptors as nicotine, but it detaches (disassociates) from the receptors about eight times faster (10). Thus, imidacloprid interactions with the nerve endings are very transitory compared to nicotine.

Fourth, studies of imidacloprid metabolism in plants indicate approximately ten percent or less (depending on the crop) transforms into DNIMI (44). Thus, for all practical purposes human exposure to DNIMI is negligible.

Birds, Bees, and Waterfleas: Ecological Risks

Despite the rigors of high-dose testing and the assignment of an extra FQPA safety factor, imidacloprid smells like a rose, thanks in part to its low toxicity and low potential for human exposure. But EPA can still nix or severely restrict the use of a pesticide if residues exceed levels thought to harm nontarget organisms.

Perhaps the most studied aspect of ecological effects is aquatic toxicity. Just like mammals, fish and invertebrates seem pretty resistant to imidacloprid. For most of the aquatic species tested, imidacloprid falls into EPA's category of practically nontoxic (LC50 greater than 100,000 ppb) to slightly toxic (LC50 between 10,000 and 100,000 ppb) (Table 5). The one exception is the saltwater shrimp, ***Mysidopsis***, on which imidacloprid receives a very highly toxic rating: LC50 less than 100 ppb.

Of course, innate toxicity is only part of the ecological risk game, but published studies of imidacloprid water

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TABLE 5

Toxicity Parameters Risk Quotients for Exposure of Aquatic Organisms to Imidacloprid (based on 9, 27, 31, 36)

Test Organism*	Acute LC50 (µg/L)	NOEC† (µg/L)	Risk Quotient‡
AQUATIC INVERTEBRATES			
Water flea (Daphnia)	10,440 – 85,000		0.002-0.0002
Water flea-reproduction (21 day)		1800 – 3600	0.010-0.005
Brine shrimp (Artemia)	361,230		0.00005
Mysid shrimp (Mysidopsis)	37		0.47
Hyalella azteca (crustacean)	55		0.32
Mosquito (Aedes)	13		1.34
FISH			
Golden orfe	237,000		0.00007
Rainbow trout	211,000		0.00008
Carp	280,000		0.00006
Trout, 21 day		29,000 – 62,000	0.0006-0.0003

***Exposure durations for all tests, unless otherwise indicated, were between 48-96 hours.**

†No Observable Effect Concentrate.

‡Calculated as the estimated environmental concentration (17.4 ppb based on EPA modeling studies, reference 43) divided by the LC50 (for acute toxicity) or the NOEC (for chronic toxicity). Quotients below 0.5 and 0.05 pose no concerns for risk of acute toxicity for non-endangered and endangered species, respectively. Quotients below 1 pose no concerns for risk of chronic toxicity.

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residues for estimating exposure are rare. Using computer simulation models, EPA estimated surface water residues of 16 to 17 ppb (43). As usual, reality paints a different picture. For example, one Canadian government study reported recoveries of 0.1 to 4.4 ppb imidacloprid in streams near potato fields (19), far less than the EPA estimates. Even if imidacloprid makes it into water bodies, several published studies show that it is susceptible to degradation by sunlight (25). Residues steadily degrade even in the dark, where they have a half-life of about thirty-five to forty days (32). Nevertheless, comparison of imidacloprid toxicity to aquatic organisms with EPA's exaggerated surface water residues suggests that risks of adverse acute or chronic effects are extremely low, especially to endangered fish species (Table 5).

Toxicity, exposure, persistence, and target susceptibility are all parts of the risk picture.

Some concern has been expressed that imidacloprid may be toxic to birds, especially because it is commonly used as a seed treatment (9). While birds, on a body weight basis, seem more susceptible to imidacloprid toxicity than rodents when force-fed the pesticide, the LC50s from dietary exposure are quite high, suggesting a low susceptibility by normal routes of exposure (Table 6). Furthermore, studies of how birds handle seeds show that some or all of the outer husk, which would contain the greatest amount of imidacloprid from a seed treatment, is actually removed prior to ingestion (3). Imidacloprid-treated seed also seems repellent to several bird species (1, 2). Thus, the reality of exposure from the diet belies the comparatively low LD50s for birds.

When estimated residues caused by overspraying plant material (leaves, fruits, seeds) are compared to the dietary LC50s of several avian species, a low potential for acute and chronic adverse effects is indicated (Table 6). Similarly, the expected exposure of a very small twenty-gram bird to treated seeds planted as a solid set across a field with five percent of them left on the surface shows risks fall substantially below EPA's level of concern, even for endangered species.

EPA also considers risk to pollinators such as bees. Beekeepers in Canada and Europe have swarmed to complain about declining bee populations in recent years, pointing to imidacloprid as the culprit (5). According to one Internet document (8), imidacloprid-treated sunflower seeds result in mature plants with detectable levels of imidacloprid in nectar, and a metabolite of imidacloprid may be toxic to bees. Because the oral LD50 of imidacloprid lies somewhere between 4 and 41 nanograms per bee (33), EPA considers the compound to be highly toxic to bees.

Despite beekeeper protestations and a high bee toxicity rating, published experiments tend to lead to a conclusion of low hazard under actual environmental conditions. For example, the oral LD50 of imidacloprid translates to a nectar concentration of between 0.14 and 1.6 mg/kg (33). Yet nectar and pollen tested did not contain any imidacloprid above the analytical limit of detection, 0.0015 mg/kg (33). One of the insect-toxic metabolites, imidacloprid olefin, tested with an oral LD50 of >36 ng/bee (29), but its residues were not found in sunflower honey or nectar (33). Bumblebees (***Bombus terrestris***), which have a similar susceptibility to insecticides as honey bees, were not harmed by sunflowers grown from imidacloprid-treated seeds (37). Work at Washington State University showed that honey bees fed syrup with 2 mg/kg imidacloprid reduced their visits to the feeder by only 7% (24), hardly a significant impact considering the natural mortality factors in any colony. Based on the risk quotient calculated using published values for the dietary LC50 equivalent and the levels of residues in sunflower nectar reported by the French beekeeping industry, EPA is likely to also conclude low toxicity risk to honey bees (Table 6).

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TABLE 6

Toxicity Parameters and Risk Quotients for Exposure of Nontarget Terrestrial Organisms to Imidacloprid (toxicity and EEC data from 8, 9, 23, 27, 31, 33)

Test Organism & Exposure Method	Acute Oral LD50 (mg/kg body weight) or LC50 (mg/kg soil or feed)	NOEC*	EEC†	Risk Quotient‡
INVERTEBRATES				
Earthworm ***(Eisenia*** spp.), 14 days, soil	2.3 – 10.7		0.239	0.104-0.022
Earthworm ***(Eisenia spp.)*** sperm deformities, soil exposure, 10 days§		0.1	0.239	2.4
Honey bee (***Apis mellifera***), dietary**	0.14 – 1.57	0.02	0.005	0.25 – 0.036
MAMMALS				
Mouse (force-fed)	131 - 168		15.1	0.109-0.085
BIRDS				
Canary (force-fed)	25 - 50		0.054	0.109-0.054
House sparrow (force-fed)	41		0.054	0.066
Pigeon (force-fed)	25 - 50		0.054	0.109-0.054
Japanese quail (force-fed)	31		0.054	0.088
Bobwhite quail (force-fed)	152		0.054	0.018
Bobwhite quail (5-day dietary)	1420		26.8	0.019
Bobwhite quail (reproduction, dietary)		>243	26.8	0.107
Mallard duck (5-day dietary)	>5000		26.8	0.005
Mallard duck (reproduction, dietary)		125	26.8	0.208

***NOEC, No Observable Effects Concentration, mg/kg feed or soil; based on a chronic feeding study, usually observing effects of repeated daily exposure and/or reproductive potential.**

†EEC, estimated environmental concentration. Soil concentrations for the earthworm risk characerization reflect a maximum soil application rate of 0.312 kg/ha (0.278 lb/A) on potatoes. The EECs applied to the avian force-feeding studies represent milligrams imidacloprid on treated seeds per square foot assuming a seed application rate equivalent to 0.117 kg/ha (0.104 lbs/A) and solid seeding (based on 26). The EECs for the mouse study assumed a 0.125 kg/ha (0.112 lb/A) overspray, large insect/forage plant residue concentration of 15.1 mg/kg, based on EPA-recommended values published in (15), and a 15-gram mouse eating 95% of its body weight. The EECs for the avian dietary toxicity studies assume foliage is contaminated to a level of 26.8 ppm following a 0.125 kg/ha spray application to various vegetables (based on 27).

‡Risk Quotient, RQ; the estimated environmental concentration divided by the LD50, LC50, or the NOEC; value must be less than 0.5 or 0.1 to be of no concern for acute toxicity risk to non-endangered and endangered species, respectively. For chronic toxicity risk, values must be less than 1. Note that the RQ for the avian acute toxicity results (force fed studies) actually represents the number of LD50 equivalents per square foot.

§After 10 days of exposure, sperm deformities were 3.5% in soil with 0.2 mg/kg imidacloprid compared to 1.7% in soil without imidacloprid (23).

****The higher RQ for bees is based on the oral LC50, and the lower RQ is based on the NOEC.**

Compatible with IPM?

When imidacloprid was first introduced to the market, it was heavily touted as being soft on insect and mite predators and parasitoids (27). Pest management specialists are constantly searching for the holy grail of pesticides—something toxic to specific pests but harmless to biocontrol organisms known as pest natural enemies. Now that imidacloprid has been on the market for about eight years, and entomologists have had a greater opportunity to study it, compatibility with integrated pest management (IPM) is a "yes-and-no" story. For every paper suggesting little harm to natural enemies (7, 13, 14, 20, 21, 45), there seems to be another paper suggesting incompatibility (11, 16, 17, 18, 22, 35). To be sure, imidacloprid is much less toxic than the traditional organophosphorus, carbamate, and pyrethroid insecticides, but it is no magic bullet. Part of the problem in making a generalization about compatibility lies in the variation among pest control situations.

While we can't say definitively that imidacloprid is compatible with IPM systems, it is clear that the ability to use imidacloprid as a systemic soil or seed treatment should have definite benefits in protecting predators and parasitoids. Internal plant residues should not be accessible to insects probing along the leaf surface or scraping the epidermis. However, directly sprayed predators could be at risk (18). What remains to be seen is whether these predators would also become intoxicated after walking on dried deposits on leaf surfaces. Fortunately, imidacloprid seems to have a very short persistence on tomato leaf surfaces. Fifty percent of residues dissipate

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Admiring Imidacloprid, cont.

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within 1.4 days under cloudy conditions; dissipation is even quicker (fifty percent in 0.7 day) under sunny conditions (34).

Another aspect of compatibility with IPM systems is the rapidity with which a pest is likely to develop resistance and whether the new pesticide is likely to be compatible in chemical rotation schemes designed to delay resistance development. During the development of imidacloprid, numerous generations of the pest aphid ***Myzus persicae*** were repeatedly treated with different concentrations of imidacloprid (27). After ninety generations, resistance did not develop, giving hope that insects under field conditions would not develop resistance as readily as they did to the conventional pesticides.

Presently, full-blown resistance to imidacloprid does not seem to be a problem, but one study has indicated wide susceptibility differences among different populations of the Colorado potato beetle (CPB) (30). The comparatively more tolerant CPB populations existed before imidacloprid's widespread use on potatoes. However, the tolerant populations were also resistant to the carbamate insecticide carbofuran, which suggested the possibility of cross-resistance. Another study has shown that a field-collected strain of the tobacco aphid (***Myzus nicotianae***) exhibited a strong antifeeding response that made it tolerant in comparison to a known susceptible laboratory strain of the aphid (28). Whatever the mechanism of tolerance, the existence of variability in susceptibility among different populations rings the alarm for careful management to avoid resistance development (12).

"It WALKS Like a Duck..."

Imidacloprid has the appearance of a reduced-risk pesticide with its comparatively low hazards and low exposure potential for humans and nontarget organisms. Although the published experiments with bees downplay imidacloprid's hazards suggested by its very low LD50, more research is definitely needed to assuage beekeepers' fears that growers have not substituted yet another bee killer for the highly toxic organophosphorous insecticides. And the jury may still be out on imidacloprid's compatibility with IPM systems.

Imidacloprid is already registered for a myriad of agricultural, urban, and veterinary uses. Achieving reduced-risk status would accelerate registration of new uses or formulations. It would also be an admission that innovation in pesticide technology was moving toward human and environmental safety long before the FQPA was conceived. If outstanding questions about bees and IPM compatibility are addressed in the near future, growers will be definitively reassured that they have another admirable tool that can be relied on for efficacy without harming their family, their workers, and their environment.

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Imidacloprid: Insecticide on the Move

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ED. NOTE: The preceding article deals with the chemistry of the insecticide imidacloprid, examining whether or not imidacloprid should be considered a "reduced-risk" pesticide under the Food Quality Protection Act. Solubility, persistence, and mobility of a compound must be considered when evaluating risk. This short essay examines these issues with respect to imidacloprid.

Imidacloprid was one of the first commercial insecticides to be registered in what has become a growing class of pesticides called neonicotinoids. It is manufactured and sold by the Bayer Company in several formulations, including those under the names of Admire, Provado, Gaucho, and Marathon. Imidacloprid has remarkably high insecticidal activity against aphids, white flies, and leafhoppers, tiny plant-sap-sucking insects in the order Homoptera. (See related article, "Homoptera and Neonicotinyls," p. 14.) Imidacloprid also has activity against fleas, the Colorado potato beetle, and termites.

Imidacloprid is one of the most versatile insecticides around. Its high biological activity is expressed whether it is sprayed directly on foliage, coated on seeds, or placed directly into the soil. Imidacloprid can be applied by diverse methods because it is highly systemic. The compound is easily absorbed by plant roots and transmitted through the xylem (vascular system) to all growing parts. Imidacloprid also has the ability to move from the treated side of a leaf to the untreated side, a property called translaminar movement. Applying imidacloprid to soil or seeds keeps residues inside the foliage, avoiding surface residue and airborne drift that can occur from spray application, greatly reducing the possibility of exposure to insect predators and parasitoids and human field workers.

Water solubility and vapor pressure are two of the most important properties driving environmental distribution of a compound and thus exposure potential. Exposure potential is also strongly influenced by biodegradation rate (speed of breakdown by soil bacteria, plants, and animals), which determines how long pesticide residues are likely to stick around. Imidacloprid has a comparatively high water solubility (510 mg/L) and very low vapor pressure (1.9×10^{-9} mm Hg), so it is unlikely to evaporate from soil and plant surfaces and become an air contaminant. On the other hand, its biodegradation rate in soil has been characterized as moderately slow, with about 50% of the applied residue dissipating in a range of 48-190 days.

Although imidacloprid has a comparatively low potential to cause adverse effects in mammals, birds, and fish, its high water solubility combined with its persistence in soil has raised a few concerns about groundwater contamination. Indeed, in early studies of imidacloprid's potential for sorption (a measure of its ability to adhere to soil particles), the compound looked like a leacher. Subsequent studies in the United States and France showed that sorption potential increased as imidacloprid concentration deceased and as its residues "aged" in soil.

When used as a systemic (applied to soil as opposed to sprayed on foliage), imidacloprid is applied at a maximum rate of only 1/3 pound (150 grams) per acre in comparison to the one to two pounds of the older organophosphate insecticides. Also, it is applied in the plant row or by the base of individual plants instead of over the whole field. Nevertheless, the EPA has reported that groundwater monitoring turned up residues of imidacloprid of 0.1-0.2 ppb in California and Michigan, and 1.9 ppb in Long Island, New York. While such levels indicate a need to better manage how imidacloprid is used, they are hundreds to thousands of times lower than levels that EPA said it would be concerned about.

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Homoptera and Neonicotinyls

Imidacloprid Aids GWSS Battle in California

Dr. Douglas B. Walsh, Entomologist, WSU

Aphids, cicadas, leafhoppers, planthoppers, treehoppers, psyllids, whiteflies, mealybugs, phylloxera, and scale insects are "homopterans." Homoptera are a particular suborder of insects that derive their name from the Greek "homo-" meaning uniform and "ptera" meaning wings. Most homoptera have wings with a uniform texture that fold tent-like over the body when the insect is at rest. They also have piercing/sucking mouthparts, enabling them to feed by withdrawing sap from vascular plants. This is where the trouble begins.

Homoptera Damage

Economic damage is manifested by homopterans in several different and specific ways.

- The mere presence of insects can be considered contamination. Industry and/or governments will often reject food products if insects or their parts surpass specific quantities in the food product. For example, Brussels sprouts are rejected when two percent or more are found to be infested at the packing shed.
- Direct feeding by homopterans can cause harm to infested plants if populations are high.
- Many species excrete honeydew, a sticky waste product that adheres to the plants upon which the insect feeds and lives.
    - Honeydew alone causes cosmetic injury to crop plants (not to mention creating an unsightly mess on cars and structures under heavily infested trees or shrubs).
    - Sooty molds will often grow on honeydew, making food products or ornamental plants look unappealing and reducing their cosmetic/economic value.
- Some homopterans have toxic saliva that is injected into plants while they are feeding. The saliva can cause plant damage through disfigurement and in some instances plant death.
- Finally, homopterans can vector disease-causing pathogens. Unlike direct damage, it does not take a large number of disease-vectoring insects to cause a problem.

Homoptera Control

Homoptera populations are typically regulated by natural enemies, with a wide range of arthropods acting as biological control agents. These beneficial insects include parasitic braconid and chacicoid wasps and generalist predators such as ladybird beetles, lacewings, and syrphid flies. As with many insect pests, population outbreaks of homoptera are often the result of disruption of the natural checks and balances within the agronomic, landscape, or forest system. Because natural biological control of homopterans is so successful, researchers have investigated use of biocontrol under outbreak conditions; indeed, classical biocontrol has been successful in suppressing pest homopteran populations to densities below economically damaging levels. Such interventions are most useful in situations where the homopterans are causing direct damage through their feeding or excrement of honeydew. Unfortunately, when homopterans vector disease, classical biological control may not provide sufficient population suppression. Under these circumstances, insecticidal control is common.

Chemical control efforts against homopterans focused primarily on the application of organophosphate nerve toxins in the years between World War II and the 1970s. Systemic organophosphate insecticides like demeton, disulfoton, and TEPP (among several others) were applied widely to a range of crops and provided good control of a number of pest homopterans. Most uses of these products have been restricted or limited due to risks associated with environmental contamination or human health.

Over the past fifteen years, chemical control of homopterans has shifted to emphasize chloronicotinyl (a.k.a. neonicotinyl) insecticides. (See related article on imidacloprid, "Admiring Risk Reduction," page 1.) Chloronicotinyls kill susceptible insects by binding to

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Homoptera and Neonicotinyls, cont.

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the receptor site for the neurotransmitter acetylcholine. Unlike organophosphate and carbamate insecticides that inhibit acetlycholinesterase (the enzyme that normally breaks down the neurotransmitter acetylcholine), neonicotinyls specifically bind to an insect's nicotinic receptor. This causes the exposed insect's nerves to fire uncontrollably, eventually leading to death.

Nicotine is a natural plant product that can be applied for insect pest control. However, natural nicotine is expensive to produce, is highly toxic, and is rapidly degraded and rendered ineffective by sunlight. Imidacloprid, thiocloprid, acetimiprid, are among these neonicotinyls that are less toxic to vertebrates but persist long enough under field conditions to control insects. (See "It's Not Your Granddaddy's Nicotine," page 3.)

Imidacloprid was the first of the neonicotinyls to gain widespread registration in the United States. It controls most sucking insects, including aphids and leafhoppers, but is generally less toxic to chewing insects and is ineffective against moth and butterfly caterpillar pests.

Enter GWSS

Homoptera control has been in the news recently because of a critical event in California: introduction and establishment of the glassy-winged sharpshooter (GWSS, ***Homalodisca coagulata***). GWSS is an efficient vector of Pierce's disease (***Xylella fastidiosa***), a lethal disease of grapevines. The half-inch-long GWSS feeds on plants infested with ***X. fastidiosa***, then transmits it to healthy vines. The ***Xylella*** bacterium attacks a plant's water-conducting tissues, resulting in infections that eventually cut off water and nutrient movement through the vine.

Scientists in California have long known that ***X. fastidiosa*** is transmitted to grapevines by blue-green sharpshooters (***Graphocephala atropunctata***), a species in a subfamily of homopterans known as sharpshooter leafhoppers. Since blue-green sharpshooters are relatively weak fliers, they are not efficient ***X. fastidiosa*** vectors. The GWSS is a much stronger flier, making it a much more threatening, less controllable vector for Pierce's disease.

GWSS and the disease it vectors spread rapidly from Ventura, California, to the Mexican border, causing catastrophic economic losses to that region. Recently, the pest was found in California's Central Valley, posing a potentially greater threat to the $1 billion California grape industry, as well as other agricultural commodities in that productive growing region.

While most homopterans are host-specific to a single plant or related group of plants, GWSS thrives on a wide range of common plants. Adding insult to injury, there are many strains of ***Xylella***, too; the various strains have been known to infect crops from plums and berries to apples and citrus. The combination of vector mobility and multiple pathogen strains make this pest situation formidable indeed. Fortunately for those of us in the Pacific Northwest, ***Xylella*** does not tolerate cold temperatures well.

Imidacloprid's Role

The establishment of the GWSS in California has led to increased use of imidacloprid. It appears to be an effective chemical control, with the added benefit of being relatively "soft." (See related article, "Admiring Risk Reduction," p. 1.) So far, resistance development does not seem significant, but there are some indications that beneficial insect populations may be adversely affected by use of this chemical. For now, many uses for imidacloprid have been approved and other uses are pending. As for the other neonicotinyls, thiocloprid has been registered for use on several crops (including apples) and registrations for several other products in this class are pending.

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Pesticide Applicator Training Courses

Washington State University provides pre-license and recertification training for pesticide applicators.
Pre-license training provides information useful in taking the licensing exam.
Recertification (continuing education) is one of two methods to maintain licensing.
(The other is retesting every five years.)

Course registration (including study materials) is $35 per day if postmarked 14 days prior to the first day of the program you will be attending. Otherwise, registration is $50 per day. These fees do not include Washington State Department of Agriculture (WSDA) licence fees.
See WSDA Testing Sites and schedule below.

For more detailed information, visit the Pesticide Education Program website's training page at
http://pep.wsu.edu/education/educ.html
or call **(509) 335-2830**.

EASTERN WASHINGTON
PRE-LICENSE TRAINING

Date	City	Facility
Jan. 8, 9, 10	Pullman	University Inn (Moscow, ID)
Jan. 15, 16, 17	Pasco	Doubletree
Special **Aquatics** session in Pasco, Doubletree, Jan. 16 afternoon		
Jan. 22, 23, 24	Yakima	Convention Center
Feb. 12, 13, 14	Spokane	Valley Doubletree
Feb. 19, 20, 21	Moses Lake	Convention Center

WESTERN WASHINGTON
PRE-LICENSE TRAINING

Date	City	Facility
Jan. 15, 16, 17	Tacoma	Pacific Lutheran University
Feb. 12, 13, 14	Kirkland	Lake WA Technical College
Special **Pest Control Operator** (PCO) and **Wood Destroying Organism Inspector & Applicator** Courses Feb. 26, 27, 28 at WSU Puyallup		
Mar. 12, 13, 14	Puyallup	WSU Allmendinger Ctr
Mar. 26, 27, 28	Bellingham	Whatcom Community Coll
Apr. 16, 17, 18	Puyallup	WSU Allmendinger Ctr

WSDA Testing Sites

Location	Day	Time
Olympia	Every Tuesday	8:30 am and 1:00 pm
Yakima	Every Tuesday	8:30 am and 1:00 pm
Mt. Vernon	2nd Thurs. each month	8:00 am to 12:30 pm
Wenatchee	4th Tues. each month	12:30 pm to 4:30 pm
Moses Lake	3rd Tues. every other month	12:30 pm to 4:30 pm
Spokane	1st Weds. each month	12:30 pm to 4:30 pm

Reservations are required. To make a reservation for any of the above locations, call WSDA at 1-877-301-4555.

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EASTERN WASHINGTON RECERTIFICATION

Date	City	Facility
Nov. 6, 7 - 2001*	Pasco*	Doubletree
Nov. 7 SPANISH*		
Jan. 9, 10 - 2002	Pullman	University Inn (Moscow)
Jan. 16, 17	Pasco	Doubletree
Jan. 23, 24	Yakima	Convention Center
Jan. 30, 31	Wenatchee	Red Lion Hotel
Feb. 13, 14	Spokane	Valley Doubletree
Feb. 20, 21	Moses Lake	Convention Center

Special **Commercial Applicator Workshops** will be held Jan. 28 at WSU Tri-Cities Auditorium and Jan. 29 at Moses Lake Convention Center

WESTERN WASHINGTON RECERTIFICATION

Date	City	Facility
Nov. 19, 20*	Lynnwood*	Edmonds Comm Coll
Jan. 10, 11	Vancouver	WSU Vancouver
Jan. 16, 17	Tacoma	Pac Lutheran Univ
Jan. 24, 25	Lynnwood	Edmonds Comm Coll
Feb. 4, 5	Lacey	St. Martins Coll
Feb. 7, 8	Des Moines	Highline Comm Coll
Feb. 13, 14	Kirkland	Lake WA Tech Coll
Feb. 20, 21	Port Orchard	Givens Comm Center
Mar. 6, 7	Seattle	UW Ctr for Urban Hort
Mar. 26, 27	Bellingham	Whatcom Comm Coll

*LAST CHANCE 2001 CLASSES

The last opportunity to obtain recertification credits in 2001 will be Nov. 6-7 in Pasco (Spanish course available Nov. 7) and Nov. 19-20 in Lynnwood. See **http://pep.wsu.edu/education/educ.html** or call (509) 335-2830.

SPECIAL WORKSHOPS

Date	Topic/Emphasis	City	Facility
Oct. 17 - 2001	Weed Identification and Management	Seattle	UW Ctr for Urban Hort
Oct. 30 - 2001	Christmas Tree Problems	Puyallup	WSU Allmendinger Ctr
Jan. 10 - 2002		Vancouver	WSU Vancouver
Jan. 16 - 2002	Wood Treatment	Tacoma	Pac Lutheran Univ
Jan. 28 - 2002	Commercial Applicator	Richland	WSU Tri-Cities Auditorium
Jan. 29 - 2002		Moses Lake	Convention Center
Jan. 29, 30, 31 - 2002	Integrated Plant Health	Puyallup	WSU Allmendinger Ctr
Mar. 5 - 2002	Commercial Applicator	Puyallup	WSU Allmendinger Ctr

Private applicators must accumulate twenty recertification credits over a five-year period, with no more than eight credits taken in a single year. All other licensees must obtain forty credits over a five-year period, taking no more than fifteen per year. Credit statements are mailed to licensees in September each year. To obtain information on your current credits, you can contact the Washington State Department of Agriculture toll-free at (877) 301-4555.

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It's the Water
Legal Issues and Rural H_2O

Dr. Barbara Rasco, Food Scientist, WSU

Water usage in the West has been a critical issue for the past 250 years. In this regard, nothing has changed, but some of the current players are different. Today's concerns revolve around allocation, contaminants, and liability. These concerns are driven by natural and manmade forces including drought, the Clean Water Act, and the Endangered Species Act.

Drought Pressures

New water resource and allocation issues are emerging in our region as a result of recent shortages. The impacts of this year's drought conditions have been complicated by laws adopted in recent decades. When adopted, these laws were not intended to have the impact on agriculture they have had. The result has been that when we have a water crisis, the needs of agriculture take a backseat.

In May 2001, for example, a water allocation issue in Oregon's Klamath Basin was brought before a federal judge. Snow pack in this area was twenty-nine percent of normal—there was simply not enough water to go around. At issue were tribal water rights, the rights of certain fish species, and the rights of agricultural producers. The court upheld the Bureau of Reclamation's water allocation to support populations of sucker fish in Upper Klamath Lake and threatened coho salmon in the Klamath River under the Endangered Species Act. The court held that "it is clear that the farmers face severe economic hardship, [but] the threat to the survival of the fish is greater." In the end, water rights were denied to ninety percent of the 200,000 acres of irrigated land (about 1400 farms) in the Klamath Basin.

Whose Water Is It?

Water allocation has been touchy in Washington State as well. New regulations from the state Department of Ecology address both allocation and water quality issues.

Since 1917, Washington State has been issuing water rights permits allowing individuals and companies to install pipes or wells on surface waters or aquifers. Currently, the Department of Ecology grants these permits, called "water rights certificates." The department is now catching up on thirty years of legislation and case law in this area and is in the process of instituting changes affecting

- development of permits,
- evaluation of water rights,
- use of water,
- the permitting process,
- contamination of irrigation water, and
- surface water quality standards.

New Standards of Cleanliness

The new Washington State Department of Ecology surface water quality standards will go into effect in December 2001. The overarching policy will be that clean water is not to be polluted unless it is shown to be necessary or in the public interest. Specifically, the new standards include the following:

- Stricter dissolved oxygen standards to accommodate salmon and trout spawning and rearing.
- Tighter irrigation water standards to prevent buildup of suspended solids, bicarbonate, and salts that cause soil toxicity.
- Maintaining water temperatures critical to salmon and bull trout.
- Emphasizing enterococci instead of fecal coliforms as bacterial indicators. (This change took eight years to make, with discussions of it going back at least twenty years.)
- Prohibiting discharge of untreated fecal waste to surface waters.

In general, irrigation water standards are more permissive than standards for contact recreation water and shellfish rearing water. The specific clean water provisions outlined above do not apply to water utilized within a closed irrigation system, where drainage water from an individual agricultural operator is captured and reused.

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It's The Water, cont.

Dr. Barbara Rasco, Food Scientist, WSU

Under the new rules, the agency prohibits discharge of toxic, radioactive, pathogenic, or deleterious materials into irrigation and surface water. There are also prohibitions against uncontrolled discharge of livestock, pet, and human waste into irrigation waters as well as discharge of municipal wastewater into irrigation supplies. Control measures to maintain clean water include implementation of best management practices or waste treatment technologies as appropriate. However, practices chosen by the irrigator must be approved by the Department of Ecology.

Bacteria as a Pollutant

The Department of Ecology Working Group did not support establishing specific numerical criteria for bacteria in agricultural water supplies, but bacterial pollution is definitely being emphasized and scrutinized.

Government environmental agencies have recently targeted agriculture operations as non-point source polluters. The U.S. Environmental Protection Agency (EPA) claims to have reduced fecal coliform pollution by 12.2 million pounds over the past few years due to heightened enforcement activities. EPA has also formed partnerships with various states to deal with non-point source pollutant discharge.

A new bacterial pollution concern is that of antibiotic-resistant bacteria. Antibiotics are used as growth promoters in some animal feed. In a recent study, tetracycline-resistant genes were found in bacteria recovered from lagoons, wells, and in groundwater one-sixth of a mile downstream from two swine facilities which used antibiotics in this manner. Resistance genes were also found in the gut, feces, and commercial feed. Can these antibiotic-resistant genes find their way from farm animals into groundwater, then into the bodies of people and wildlife?

Liability Issues

Classifying bacteria as a "pollutant" (or not) has distinct legal implications. In recent litigation, a professional golfer sued a golf course after becoming sick from contacting water contaminated with harmful bacteria that was used at the course. The golf course's insurance company denied coverage based upon the pollution exclusion in its policy. The court held that bacteria do not fall "neatly" under the policy definition of pollutant: "To the extent that bacteria might be considered 'irritants' or 'contaminants,' they are living, organic irritants or contaminants which defy description under the policy as 'solid,' 'liquid,' 'gaseous,' or 'thermal' pollutants." Neither did bacteria fall under the definition of pollutants as "smoke, vapor, soot, fumes, acids, alkalis, chemicals, and waste," nor were they of a similar enough nature to conventional "industrial" pollutants to be included in that category's definition. The bacterial contamination was not covered by the pollution exclusion; so the golf course was liable and had to pay the claim.

Another recent case had a similar outcome, but with a far-reaching twist. An insurance carrier denied coverage to a farmer who inadvertently used well water contaminated with ***E. coli*** to make ice served in drinks at a county fair. The ice made a young child sick. The farmer's insurance carrier claimed that the pollution exclusion in the policy was ambiguous, but that it could be read to cover bacteria, providing them with a basis to deny the claim. The court held that the pollution exclusion was intended to cover only "industrial pollutants and waste." The court went on record, however, as noting that had the policy more clearly defined contaminant or waste to include biological or etiological agents or materials—as some insurers have already done—then coverage would have been justifiably denied. Clearly, there is a trend for insurance carriers to begin to include bacteria under pollution exclusions, increasing the likelihood that future incidents like this will not be covered.

More Legal Tangles

Numerous corporate and personal lawsuits have resulted in recent years from failure to handle water in such a way as to prevent pollution or illness. A particularly convoluted incident from 2001 involved Smithfield Foods. This company was held liable for criminal and civil violations of the Clean Water Act.

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It's The Water, cont.

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Smithfield, an animal feeding operation, was in the process of upgrading its waste management system, having paid $15 million for new technologies and $50 million for environmental enhancement programs. At one of its facilities, it had permission from the state (Virginia) to exceed discharge permit requirements while rerouting its new pipeline, but EPA claimed it was not bound by the company's agreement with state regulators and hit Smithfield with a $12 million fine for violating the Clean Water Act. In a bizarre twist of fate, the state of Virginia then turned around and sued Smithfield for violations of the same permit under a legal theory that the federally mandated National Pollution Discharge Elimination System (NPDES) permit can impose requirements from two separate sovereign governments.

Irrigation canal operators have also been sued under the Clean Water Act. In Jackson County, Oregon, dead fish were found downstream from a leaking canal waste gate. Environmental groups sued the Talent Irrigation District for failing to obtain an NPDES permit before applying acrolein to kill weeds in the subject canal. The district court held that the irrigation district did not need an NPDES permit since it could show that the herbicide was applied according to EPA requirements; the court ruled that further regulation under the Clean Water Act was unnecessary. However, on appeal, the court held that an NPDES permit was required. Now, irrigation districts can be required to obtain NPDES permits for aquatic herbicides even if they have complied with other EPA requirements for use of the herbicide. NPDES requirements are not based upon cost-benefit analyses, but only upon a determination that discharge of a pollutant satisfies EPA's effluent limitation imposed to improve water quality. (NPDES permits translate national effluent standards into site-specific limitations.) Getting an NPDES permit is not a trivial matter or cheap proposition; community opposition can cause the process to drag on for years.

NPDES, mandated by the Federal Environmental Protection Agency, requires that municipalities monitor and reduce the amount of non-point source pollutants that have the potential to be discharged into natural lakes and rivers. There is no requirement that a pollutant actually reach and pollute a navigable body of water, only that the subject canal or tributary flows into a navigable body of water and that the capability of spreading the pollutant and causing environmental damage exists.

Piling on the Paperwork

New pressures may also be applied to corporations regarding reporting environmental liabilities and risks. Current Security and Exchange Commission regulations require companies to file various statements with the government and to provide shareholders with information on financial risks or potential competitive impacts arising from their exposure to known environmental uncertainties. (These include the arcane 10K, 10Q, and 8K statements along with Item 303 of Regulation S-K which requires a management discussion and analysis disclosing known future uncertainties and trends that may materially affect a company's financial performance.) Enforcement of these requirements has been lax in the past, but there appears to be mounting pressure from environmental organizations and "socially responsible" investment firms that invest in public corporations to provide more complete and detailed information. Risks associated with water allocation, pollution, and the Endangered Species Act would fall under these disclosure requirements.

Dr. Barbara Rasco is with the Department of Food Science and Human Nutrition at Washington State University. Dr. Rasco is licensed to practice law in Washington State and in Federal court. She can be reached at (509) 335-1858 or rasco@wsu.edu.

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False Test Results Spell Trouble for Ohio Company

In the August 2001 issue of ***Agrichemical and Environmental News*** (Issue. No. 184) our cover story ("Is 'Good' Enough?") discussed the genesis of Good Laboratory Practices (GLP). In relating the history of GLP, author Dr. Vincent Hebert explained how a U.S. Food and Drug Administration audit in 1976 revealed that some unethical pesticide testing laboratories had been falsifying data. Falsifying test results can create a chemical exposure risk that may have adverse health impacts. Such discoveries hastened implementation of GLPs, a rigid set of protocols now required for pesticide studies submitted to the U.S. Environmental Protection Agency (EPA).

It seems not everyone got the message. On July 19, 2001, J.T. Eaton & Co. of Twinsburg, Ohio and two company executives, Stanley Z. Baker and Benjamin H. Baker, were indicted for allegedly submitting falsified test results to EPA pertaining to a product designed to repel squirrels and birds. The tests were allegedly run on samples of a chemical formulation of the product different from the one the company currently markets.

If convicted, Stanley and Benjamin Baker each face maximum sentences of up to five years in prison and/or a fine of up to $250,000. The company faces a maximum fine of up to $500,000. The case was investigated by EPA's Criminal Investigation Division, the FBI, the Defense Criminal Investigative Service Office of Inspector General and the Ohio Environmental Protection with the assistance of EPA's National Enforcement Investigations Center. It is being prosecuted by the U.S. Attorney's Office in Cleveland. An indictment is an accusation and all defendants are presumed innocent unless or until proven guilty in a court of law.

Pesticide Container Recycling Schedule

Washington Pest Consultants Association

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. The schedule is also 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.

DATE	TIME	LOCATION	SPONSOR	CONTACT	PHONE
Oct. 2	9a-11a	Ephrata	The Crop Duster	Martin Shaw	(509) 754-3461
Oct. 3	9a-11a	Quincy	Wilbur Ellis	Dale Martin	(509) 787-4433
Oct. 4	9a-11a	Waterville	Western Farm Service	Dale Gromley	(509) 745-8857
	2p-4p	Coulee City		Pete Thiry	(509) 632-5697
Oct. 9	8a-11a	Mount Vernon	Wilbur Ellis	Marty Coble	(360) 466-3138
Oct. 10	8a-11a	Conway	Cenex Farm Supply	Will Cox	(360) 445-5015
			Tronsdal Air Service	Kevin Belisle	(360) 661-0422
	12p-2p	Seattle	WA Tree Service	Ron Angle	(206) 362-9100
Oct. 11	8a-11a	Puyallup	WSU Research Station	Roy Jensen	(253) 445-4517
	8a-10a	Tacoma	Wilbur Ellis and DOT	Randy Knutsen	(253) 351-6591
				Dave Patterson	(253) 589-7255
Oct. 12	8a-10a	Centralia	Lewis Cty Public Works	John Prigmore	(360) 740-1193
	8a-10a	Vancouver	WSU Research Station	Martin Nicholson	(360) 576-6030
	1p-3p	Chehalis	Farm & Forest Helo	Dan Foster	(360) 262-3197
	3p-4p	Morton	DOT	Craig Robbins	(360) 496-5516
Oct. 15	9a-Noon	Ellensburg	DOT	Susanne Tarr	(509) 962-7577
	2p-4p		Rumble Spray Inc.	John Rumble	(509) 968-3001
Oct. 16	8a-10a	Sunnyside	Simplot	John Cullen	(509) 837-6261
	1p-3p		Olsen Brothers Farms	Keith Oliver	(509) 781-1106
Oct. 22	8a-10a	Spokane	WSDA	Tim Schultz	(509) 533-2686
			WSU Coop Ext	Jim Lindstrom	(509) 533-2690
	1p-3p	Mead	Cenex	Todd Race	(509) 466-5192
Oct. 23	8a-10a	Deer Park	Inland Agronomy	Jim McAdam	(509) 276-2611
	1p-3p	Fairfield	Wilbur Ellis	Ric Murison	(509) 283-2411
Oct. 24	8a-10a	Oakesdale	Wilbur Ellis	Jerry Jeske	(509) 285-4511
	1p-3p	Rosalia	Western Farm Service	John Hartley	(509) 523-6811
Oct. 25	8a-11a	Rosalia	Reed Aviation	Pete Reed	(509) 245-3248
	2p-4p	St. John	Gossard Aviation Inc.	Wesley Gossard	(509) 648-3722
Oct. 26	9a-11a	Tekoa	McGregor Company	Charles Wedin	(509) 284-5391
Oct. 29	9a-3p	Outlook	Snipes Mtn. Trans. Stn.	Mark Nedrow	(509) 574-2472
Oct. 30	9a-3p	Terrace Heights	Terrace Hts. Landfill		

"Our industry does not want pesticide containers to become a waste issue. If we take the time to clean and recycle these products, we can save money, show that the industry is responsible in its use of pesticides, and reduce inputs to the waste stream."

Bug of the Month

Big-Eyed Bug

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Dr. W. E. Snyder and Amanda K. Fallahi, WSU; Dr. M. D. Eubanks, Auburn University

Big-eyed bugs, ***Geocoris*** spp. (Heteroptera: Lygaeidae), are voracious predators of pest insects. In Washington, they are often the most abundant predators in crop fields. They are known to eat huge numbers of pests, but because they are small size and skittish (with their big eyes and excellent vision, they can see you coming and run away), they are difficult to count and easy to overlook.

Description

Big-eyed bugs are one of the most distinctive-looking predators in the agricultural fields of Washington. They are small (1/4-inch or less in length), oval-shaped insects with large, protruding eyes on the sides of their heads. The adults have wings and are able to fly, while the juveniles look like smaller, wingless versions of the adults.

Habits and Life Cycle

Big-eyed bugs are common in a wide variety of crops throughout Washington, from irrigated fields in the Columbia Basin to the dryland farms of the Palouse. They are active throughout the growing season, from mid-May until early October, and go through several generations per year. Active big-eyed bugs can be seen on cold, windy spring days with temperatures below 50°F, and on hot, dry summer days with temperatures over 100°F.

Unlike specialized biocontrol agents such as parasitic wasps, which often only attack a single prey species, big-eyed bugs are generalists. They feed on a wide variety of pests, including aphids, spider mites, caterpillars, insect eggs, and beetle larvae. Big-eyed bugs feed using what entomologists call "piercing-sucking mouthparts." In other words, their mandibles form what is essentially a long, sharp straw. They attack prey by spearing it on the end of their mouthparts, then sucking its body fluids through the straw. For this reason, big-eyed bugs can only attack prey with exoskeletons soft enough to puncture.

Big-eyed bugs are somewhat unusual among predatory insects because they also feed on plants to a small degree, piercing the plant tissue and feeding like aphids. Such feeding is too infrequent to result in any noticeable plant damage, but it allows big-eyed bugs to stay alive in agricultural fields when pest insects are not abundant. It also allows big-eyed bugs to form a first line of defense when pests begin to colonize fields.

Tiny Bugs, Big Appetites

Despite their small size, big-eyed bugs are voracious predators. We have been studying their feeding rates on two common potato pests, the green peach aphid and the Colorado potato beetle. We have found that a single big-eyed bug can eat more than twenty aphids per day. They can also eat up to ten Colorado beetle eggs or five small larvae over the course of a few days. In some potato fields we have found big-eyed bug densities as high as two per plant. With about 17,000 plants per acre and two big-eyed bugs per plant, the big-eyed bugs in a 120-acre circle have the potential to eat over eighty million aphids PER DAY – quite a lot of pests for such a small predator!

Think IPM, Save the Beneficials

Like many other beneficial insects, big-eyed bugs are very susceptible to broad-spectrum pesticides. Working in potato fields in Washington, we have found that big-eyed bugs are six times more abundant in fields sprayed with selective pesticides (e.g., Fulfill and Success) than in fields treated with broad-spectrum pesticides (e.g., Monitor). The new, "softer" (more selective) chemicals might allow growers in Washington to take advantage of the pest control that big-eyed bugs gladly provide for free. Big-eyed bugs and other predators can slow the rate of pest resurgence following application of softer pesticides, making fewer treatments necessary than would be required if broad-spectrum pesticides were used.

Bill Snyder and Amanda Fallahi are with the Department of Entomology at Washington State University in Pullman. Micky Eubanks is with the Department of Entomology and Plant Pathology at Auburn University in Alabama. Bill Snyder can be reached at wesnyder@wsu.edu or (509) 335-3724.

Washington Pest Consultants Association

Annual Meeting

November 15 and 16, 2001
Yakima Convention Center

Tree Fruit Sessions

cherry virus management
fruit lenticel disorders

Row Crop Sessions

micronutrients in row crop production
onion neck rot
plant physiology during drought conditions

Dryland Cropping System Sessions

broadleaf weeds in cereal production
when and how to use surfactants
nitrogen management in hard red spring wheat

General Interest Sessions

airborne herbicide residues on wine grapes

riparian buffers and pending regulations

household pest control

systemic acquired resistance strategies

fine-tuning irrigation scheduling

improving water efficiency with organic amendments, polymers and calcium

precision agriculture in Washington State

State representative Gary Chandler will be the featured luncheon speaker. For further information, contact Ellen Bentley at ellen_bentley@wsu.edu or (509) 786-9271.