Research *You Can Use*

Determining Golfer Exposure and Hazard to Pesticides

University of Massachusetts scientists investigate golfers' exposure and hazard to commonly used golf course pesticides.

BY J. MARSHALL CLARK, RAYMOND PUTNAM, AND JEFFERY DOHERTY

Pesticide exposure was measured by dosimetry and biomonitoring. The dosimetry group (on right) wore full-body cotton suits and personal air samplers. The biomonitoring group (on left) wore matching suits cut to simulate the body coverage of normal golfer attire.

Objectives:

1. Determine the level of hazard of volatile and foliar dislodgeable residues of the reduced-resk pesticides — carfentrazone (Quicksilver, Speed Zone, and Power Zone), halofenozide (Mach 2), and azoxystrobin (Heritage) — following full-course, full-rate applications.

2. Determine the effect of partial-course application strategies (e.g., tees and greens) and post-irrigation on volatile and foliar dislodgeable pesticide residues following full-rate applications of carfentrazone, halofenozide, and azoxystrobin.

3. Model the relationship of volatile and dislodgeable foliar residues vs. actual golfer exposure using urinary biological monitoring techniques or, for pesticides that are not amenable to biomonitoring, using dosimetry techniques.

Start Date: 2007

Project Duration: Three Years

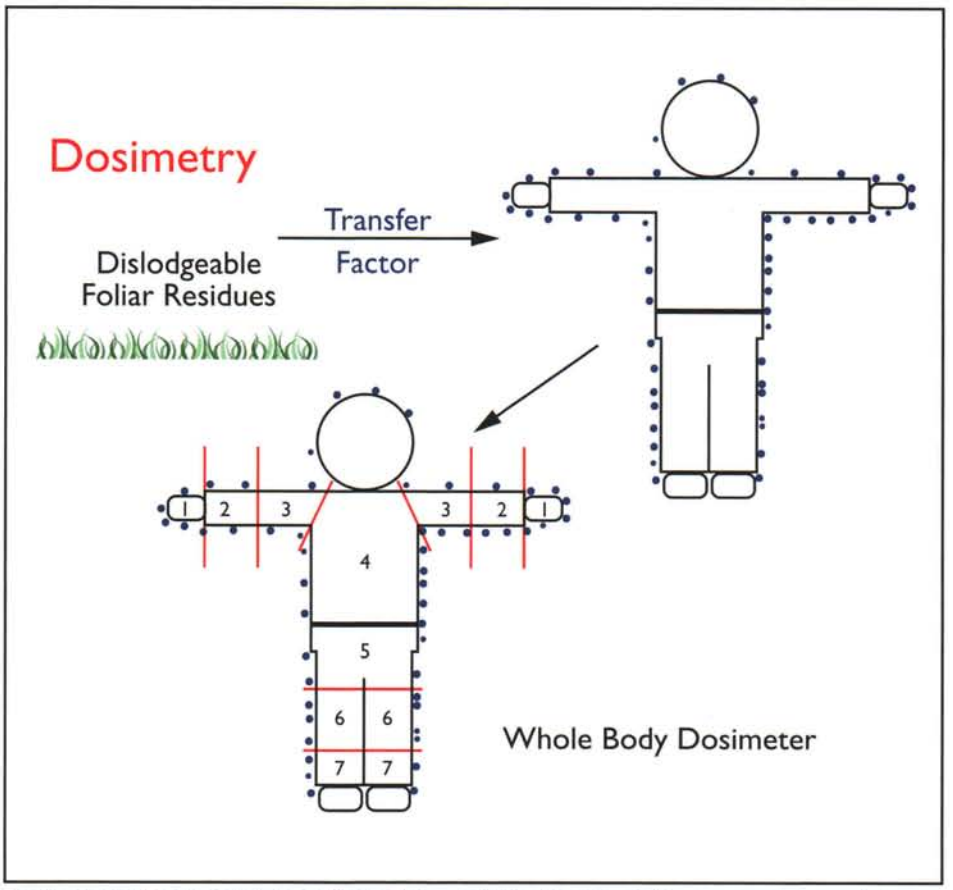
Total Funding: $90,000

This study seeks to determine actual levels of golfer exposure to reduced-risk pesticides following application to turfgrass. A major goal of this research is the development of a model for use by the turf industry and regulatory agencies that accurately predicts golfer exposure using easily collected environmental residue data. Dermal exposure (skin) and inhalation of pesticide residues are the primary routes by which golfers are exposed to turfgrass pesticides following application.

The fate of pesticides after application largely determines how much is available for potential human exposure. This process is influenced by many factors, including post-application irrigation, application rate, and integrated pest management (IPM) strategies such as partial course application, as well as the physiochemical properties such as water solubility and volatility of the pesticide itself. To understand these factors, we have analyzed pesticide residues in the air and on turfgrass leaves (dislodgeable foliar residues, DFR) in more than 40 pesticide applications using either chlorpyrifos (Lorsban), carbaryl (Sevin), cyfluthrin (Tempo), chlorothalonil (Daconil), 2,4-D, MCPP-p (mecoprop), dicamba (Banvel), and imidacloprid (Merit). In the 2007 season, two applications of the reduced-risk herbicide carfentrazone were made. Analysis of these samples is in progress.

This study also evaluates best management practices for reducing golfer exposure to reduced-risk turfgrass pesticides. This information is critical to reduce individual contributions of these pesticides to the USEPA/FQPA risk cup evaluation of agrochemicals, including turfgrass pesticides. While many standard pesticides have been removed from use, new reduced-risk pesticides have been added to the IPM practitioner's toolbox. To date, there is no dosimetry or biomonitoring data on these reduced-risk pesticides, which exhibit low mammalian and environmental toxicity, low potential for

The University of Massachusetts study investigated actual levels of golfer exposure to reduced-risk pesticides following application to turfgrass. Volunteers simulated a four-hour, 18-hole round of golf.



Dosimetry involves measuring pesticide residues on full-body cotton suits, gloves, and personal air samplers. A second method used was biomonitoring, which measured the metabolites excreted through urine. Together the results provide a unique database on golfer exposure.

groundwater contamination, low pest resistance potential, and are compatible with IPM. These comparative benefits are due to these compounds' novel physical and chemical properties.

To determine precisely how much of the environmental residues are actually transferred to and absorbed by golfers during a round of golf, we measure exposure to volunteer golfers using dosimetry (measuring pesticide residues on full-body cotton suits and personal air samplers) and biomonitoring (measuring urinary metabolites), respectively. This work is being done in cooperation with the New England Regional Turfgrass Foundation.

Dosimetry and biomonitoring, together with concurrently collected dislodgeable foliar and airborne residue data, provides a unique database on golfer exposure, and has allowed us to develop a golfer exposure model. The central predictor of exposure in the model is the transfer factor (TF), which is the ratio between the amount that actually ends up transferring to the golfer (as measured by dosimetry) versus the pesticide residues available in the environment (DFRs). We will compare the biomonitoring and dosimetry results for these reduced-risk compounds with those previously determined for chlorpyrifos, carbaryl, cyfluthrin, 2,4-D, MCPP, dicamba, chlorothalonil, and imidacloprid.

Regulators and health professionals now consider biomonitoring data the gold standard for measuring pesticide exposure, and we have used this to validate our TF model for chlorpyrifos, carbaryl, and cyfluthrin, chlorothalonil, MCPP, and dicamba. This season (2007) we determined exposure in 16 rounds of golf following application of carfentrazone without post-application irrigation. With the empirically derived TF model, pesticide exposure can be predicted solely using environmental residues (airborne and DFRs) and converted to dose:

Pesticide Dose (μg/Kg body weight) = DFRs (μg/m^2) × TF (cm^2/hr) × dermal penetration factor × 4 hr/70Kg + inhaled dose (μg)/70Kg).

The hazard associated with a given exposure is evaluated using the hazard quotient (HQ), which is determined by dividing the dose received by the USEPA reference dose (Rfd). HQs less than or equal to 1.0 indicate that the exposure resulted in a pesticide dose at which adverse effects are unlikely. A HQ greater than 1.0 does not necessarily infer the exposure will cause adverse effects, but rather that the absence of adverse effects is less certain.

HQ = Pesticide Dose (μg/Kg body weight/d) / EPA Rfd (μg/Kg body weight/d)

To date, all HQs determined (chlorpyrifos, carbaryl, cyfluthrin, 2,4-D, dicamba, chlorothalonil, MCPP, and imidacloprid) have been 20- to 300-fold below 1.0, indicating safe exposure levels using the EPA Hazard Quotient criteria.

Although biomonitoring is considered the gold standard, not all pesticides are amenable to this approach. Some pesticides do not possess a suitable urinary metabolite, or the pharmacokinetics (absorption, distribution, metabolism, and excretion) of the compound may not be available. In these cases, the TF model still allows us to calculate a hazard quotient in a meaningful fashion.

SUMMARY POINTS

- Researchers have evaluated exposure in 16 rounds of golf following the application of carfentrazone (Quicksilver, Speed Zone, and Power Zone) and will compare this and future results from halofenozide (Mach 2, 2008) and azoxystrobin (Heritage, 2009) with

CONNECTING THE DOTS

An interview with Dr. John Clark, University of Massachusetts, regarding the quantification of exposure and hazard to golf course pesticides.

Q: *Why did you initiate this research? Was there a perceived significant pesticide exposure hazard that golfers are exposed to as they play a round of golf?*

A: The *potential* for significant golfer exposure is quite substantial. There are many golf courses and many golfers. The frequency and level of pesticide use on golf courses is similar to that of many agricultural commodities. To date, there are no restrictions on "re-entry intervals" following pesticide applications to golf courses. The perceived exposure potential was therefore high in the eyes of many pesticide regulatory agencies.

Q: *What specific requirements categorize a pesticide as* reduced risk?

A: Reduced-risk pesticides elicit low mammalian and environmental toxicity (i.e., they are selectively toxic to pest organisms), low potential for groundwater contamination, low pest resistance potential, and are compatible with IPM, due to their novel physical and chemical properties.

Q: *Did your previous work with chlorpyrifos, carbaryl, cyfluthrin, chlorothalonil, 2,4-D, MCPP-p, dicamba, and imidacloprid raise any red flags regarding the hazard to golfers playing a typical 18-hole round of golf?*

A: No, actually quite the contrary. All resulted in Hazard Quotients less than 1.0, indicating safe exposures. Because the reference dose used to determine hazard quotients is based on the No Observable Effect Level (NOEL) that has been further corrected to be more safe by inclusion of uncertainty factors (e.g., incomplete toxicity data) and modifying factors (e.g., children safety factor), this hazard assessment is considered to be quite conservative.

Q: *You refer to the* risk cup *as denoted from the USEPA and the Food Quality Protection Act. Explain what this concept is regarding pesticide exposure.*

A: In 1996, the Food Quality Protection Act required that the U.S. Environmental Protection Agency consider the cumulative effects of exposure to pesticides that have a common mechanism of toxicity. Thus, the toxicity of individual pesticides that belonged to large classes of pesticides that share a common mechanism of toxicity, such as the organophosphorous insecticides, are now added together as a class in any risk assessment and are no longer considered independent of each other. The idea of the *risk cup* is that all the individual risks associated with pesticides that share common mechanisms of toxicity are summed (poured) into a *risk cup*. When the cup overflows (exceeds the critical value of risk), the group of commonly acting pesticides is restricted or removed from use.

Q: *You also refer to* transfer factor *as the ratio of the amount that actually ends up transferring to the golfer versus the pesticides measured in the environment. From your previous studies, what parts of the golfer's body are most prone to pesticide exposure, and what common-sense lessons can we learn?*

A: Our initial assumption (and that of many others) was that the hands of golfers were the most likely route of exposure to pesticides. What we have found by our dosimetry research is that legs are the primary route of exposure, particularly for golfers wearing shorts. This type of transfer is particularly available when pesticides are applied early in the morning when there is still substantial dew on the turfgrass. Once the sun dries the turf, pesticide transfer is greatly reduced. Additionally, post-irrigation of applied pesticides substantially reduced the level of transferable residues from the turf to the golfer. Without post-application irrigation, hands become the primary route of exposure.

Q: *By comparing dosimetry and biomonitoring data, it is possible to calculate a dermal penetration factor (percent of pesticide on the skin that gets absorbed). What are some of these values for different turfgrass pesticides? Do you use these calculated values when you calculate pesticide dose from exposure to specific chemicals, or do you use some other value for the sake of a conservative estimate of hazard?*

A: Dermal penetration factors for most pesticides can range dramatically depending on how the measurements are carried out (0 to ~70%). The degree of skin hydration, skin moisture, and occlusion all affect penetration. Different parts of the body also affect penetration. The palms of the hands and the soles of the feet are usually less susceptible to penetration than, say, the back of the ear or in the bend of the arm. Also, the use of sunscreens and moisturizers affects penetration, as does the concentration of the pesticide, the presence of carriers and formulations, and the ambient temperature. The use of a dermal penetration factor is necessary to estimate the absorbed dose following a skin exposure event. For our penetration estimates, we have usually chosen values in the higher percentage range to model worst-case scenarios.

Q: *All of us face multiple risks every day — driving our cars, playing sports, air travel — you name it. Please put into perspective the typical golfer's pesticide exposure on golf courses. Is there a reason to be concerned by those who love the game?*

A: I personally am not concerned, given our research findings. Nevertheless, there can be many compounding factors (e.g., other non-golf-related exposures, specific health concerns, and health history of families) that make this choice complex and individual. If this is the case, there are a number of safety precautions that one can take: play ~12-24 hours following applications, wear long pants and socks, periodically wipe or wash your exposed skin, play only after the sun has dried the turfgrass, leaving no dew, etc.

Jeff Nus, Ph.D., *manager, Green Section Research.*

those results of previous experiments on chlorpyrifos, cyfluthrin, carbaryl, chlorothalonil, 2,4-D, MCPP-p, dicamba, and imidacloprid.

- Determination of golfer exposure to "reduced-risk" pesticides will provide a novel dataset for these IPM-compatible compounds.

RELATED INFORMATION

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J. Marshall Clark, Ph.D., *director and professor of Environmental Toxicology and Chemistry;* Raymond A. Putnam, *laboratory manager; and* Jeffery Doherty, *graduate student; Department of Veterinary & Animal Science, Massachusetts Pesticide Analysis Laboratory, University of Massachusetts, Amherst, Mass.*