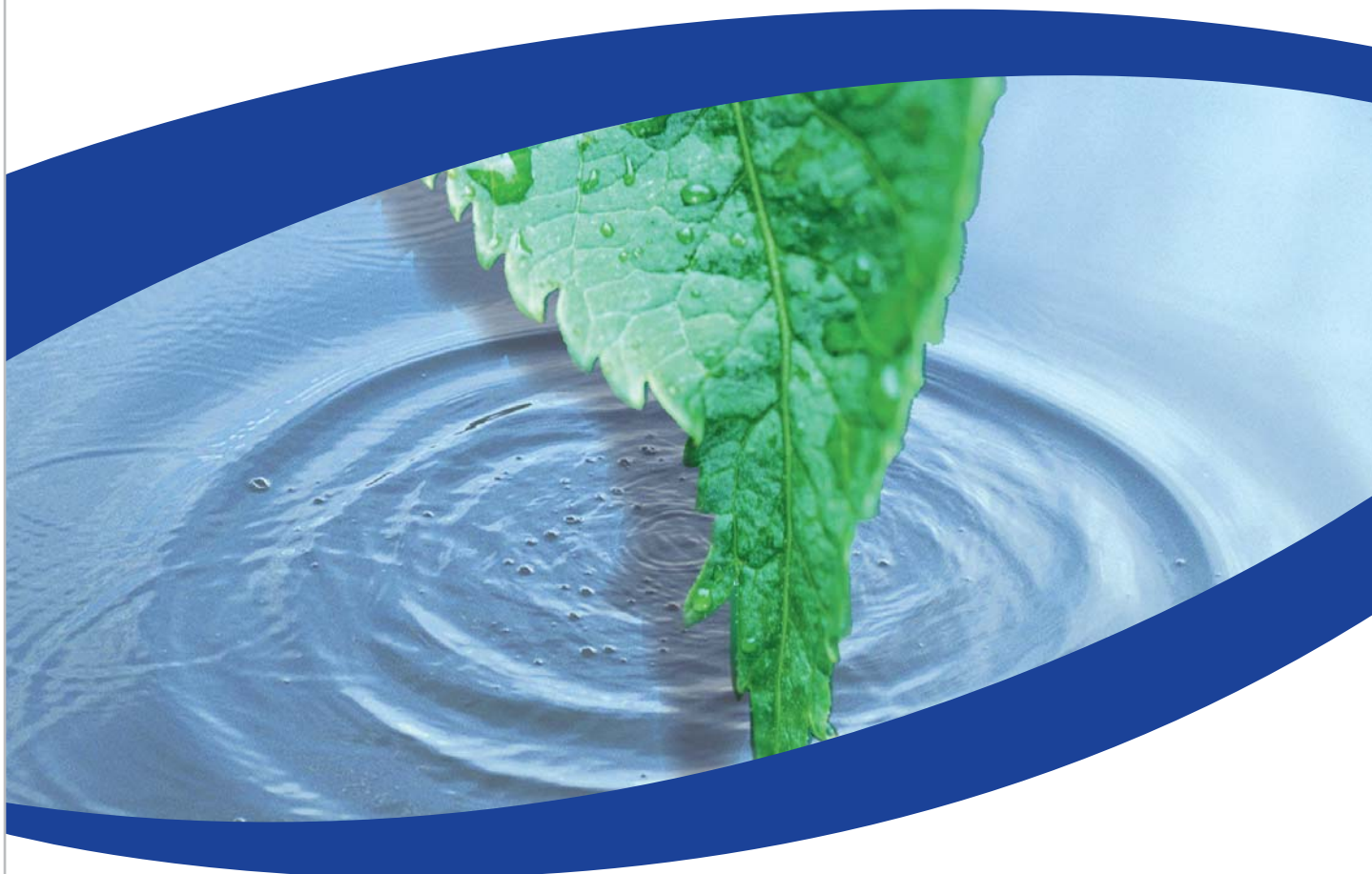


Vegetation Management in Electric Transmission Rights-of-Way and Potential Impacts on Groundwater



Vegetation Management in Electric Transmission Rights-of-Way and Potential Impacts on Groundwater

1020323

Final Report, November 2010

EPRI Project Manager
J. Goodrich-Mahoney

ELECTRIC POWER RESEARCH INSTITUTE
3420 Hillview Avenue, Palo Alto, California 94304-1338 • PO Box 10412, Palo Alto, California 94303-0813 • USA
800.313.3774 • 650.855.2121 • askepri@epri.com • www.epri.com

DISCLAIMER OF WARRANTIES AND LIMITATION OF LIABILITIES

THIS DOCUMENT WAS PREPARED BY THE ORGANIZATION(S) NAMED BELOW AS AN ACCOUNT OF WORK SPONSORED OR COSPONSORED BY THE ELECTRIC POWER RESEARCH INSTITUTE, INC. (EPRI). NEITHER EPRI, ANY MEMBER OF EPRI, ANY COSPONSOR, THE ORGANIZATION(S) BELOW, NOR ANY PERSON ACTING ON BEHALF OF ANY OF THEM:

(A) MAKES ANY WARRANTY OR REPRESENTATION WHATSOEVER, EXPRESS OR IMPLIED, (I) WITH RESPECT TO THE USE OF ANY INFORMATION, APPARATUS, METHOD, PROCESS, OR SIMILAR ITEM DISCLOSED IN THIS DOCUMENT, INCLUDING MERCHANTABILITY AND FITNESS FOR A PARTICULAR PURPOSE, OR (II) THAT SUCH USE DOES NOT INFRINGE ON OR INTERFERE WITH PRIVATELY OWNED RIGHTS, INCLUDING ANY PARTY'S INTELLECTUAL PROPERTY, OR (III) THAT THIS DOCUMENT IS SUITABLE TO ANY PARTICULAR USER'S CIRCUMSTANCE; OR

(B) ASSUMES RESPONSIBILITY FOR ANY DAMAGES OR OTHER LIABILITY WHATSOEVER (INCLUDING ANY CONSEQUENTIAL DAMAGES, EVEN IF EPRI OR ANY EPRI REPRESENTATIVE HAS BEEN ADVISED OF THE POSSIBILITY OF SUCH DAMAGES) RESULTING FROM YOUR SELECTION OR USE OF THIS DOCUMENT OR ANY INFORMATION, APPARATUS, METHOD, PROCESS, OR SIMILAR ITEM DISCLOSED IN THIS DOCUMENT.

THE FOLLOWING ORGANIZATIONS, UNDER CONTRACT TO EPRI, PREPARED THIS REPORT:

Cascade Environmental Consultants

NOTE

For further information about EPRI, call the EPRI Customer Assistance Center at 800.313.3774 or e-mail askepri@epri.com.

Electric Power Research Institute, EPRI, and TOGETHER...SHAPING THE FUTURE OF ELECTRICITY are registered service marks of the Electric Power Research Institute, Inc.

Copyright © 2010 Electric Power Research Institute, Inc. All rights reserved.

ACKNOWLEDGMENTS

The following organizations, under contract to the Electric Power Research Institute (EPRI), prepared this report:

Cascade Environmental Consultants
4178 NW Elmwood Dr
Corvallis, OR, 97330

Principal Investigator
J. Jenkins

This report describes research sponsored by EPRI.

This publication is a corporate document that should be cited in the literature in the following manner:

Vegetation Management in Electric Transmission Rights-of-Way and Potential Impacts on Groundwater. EPRI, Palo Alto, CA: 2010. 1020323.

PRODUCT DESCRIPTION

Chemical contamination of ground water is a growing utility concern. Pesticides and other potentially hazardous chemicals are used ubiquitously in everyday life. Pesticide use is widespread in urban, industrial, and agricultural settings, and in the case of utility rights-of-way (ROWs), is applied in a linear fashion across the landscape, often over long distances and intermingled with other uses. Thus, reliable assessment of risks arising from pesticide contamination of ground water and design of efficient and effective mitigation measures both require the capability to predict pesticide behavior. This report discusses the use of herbicides and tree growth regulators in electric utility ROWs integrated vegetation management. Use practices and mitigation measures are evaluated for the protection of ground water resources, with specific emphasis on human health risks associated with contaminated drinking water.

Results and Findings

This report provides information resources and an overview of the pesticide risk assessment process, focusing on drinking water. The scope of herbicide and tree growth regulator (TGR) use in electric utility ROW operations is evaluated. The report profiles 16 herbicides and two tree growth regulators, discussing current toxicology methods used to assess human health risks with specific emphasis on drinking water. In addition, the report examines chemical characteristics that influence environmental fate, factors that determine ground water vulnerability, and the potential for pesticide contamination of ground water resources. Information on pesticide toxicology and environmental fate is necessary to characterize risk to ground water resources. The report summarizes key data to facilitate comparison of chemical characteristics and toxicity of herbicide and TGR alternatives. Finally, the report presents resources and strategies for considering site-specific mitigation measures.

Challenges and Objectives

This report has the following objectives: 1) familiarize EPRI members with the basic concepts and methods used in a human health risk assessment currently employed by regulatory agencies, 2) summarize the plausible toxic effects of each chemical used in determining drinking water standards or guidelines, 3) discuss chemical behavior as it relates to the potential for ground water contamination and human exposure in drinking water, 4) examine the environmental factors influencing pesticide environmental fate and ground water vulnerability, and 5) identify mitigation measures and decision aids that may be applied to prevent ground water contamination.

Applications, Values, and Use

Herbicides and TGRs continue to be a valued component in integrated vegetation management along electric utility ROWs. However, with increased emphasis on sustainability and natural resource protection, vegetation managers need more robust information resources and decision aids in order to balance efficacious vegetation management with the protection of human and environmental health. When considering vegetation management practices—

including mitigation measures to reduce the potential for adverse impacts on natural resources—protecting ground water may well be the most challenging problem to date. The information in this report will help stakeholders, including vegetation managers, to 1) make more informed decisions regarding the potential for herbicides and TGRs to contaminate ground water and 2) evaluate the potential adverse impacts on human health associated with consumption of such water.

EPRI Perspective

Today's energy companies—under pressure to reduce O&M costs while increasing power line throughput—need information to effectively manage existing ROWs from an environmental perspective. This report evaluates the potential impacts and means of minimizing the effects of herbicides and TGRs on ground water resources. Utilities will find this information particularly valuable as they work to protect local ecology from the use of these substances. This report is the third in a series on environmental and human health risk from the use of herbicides and TGRs on ROWs. The two prior EPRI reports are *Human Health Risk Assessment of Chemicals Encountered in Vegetation Management on Electric Utility Rights-of-Way* (1005367, December 2003) and *Ecological and Wildlife Risk Assessment of Chemicals Encountered in Vegetation Management on Electric Utility Rights-of-Way* (1009445, December 2004).

Approach

This report follows the National Academy of Sciences risk assessment paradigm by identifying the hazards and potency of each chemical, evaluating each chemical's fate in the environment with emphasis on the potential for ground water contamination and human exposure in drinking water, and characterizing the human health risk by combining information on toxicity and exposure. Mitigation measures are discussed in this context, by identifying those chemicals that are the most potent (lowest drinking water standard or guideline) and those that are the most likely to contaminate ground water.

Keywords

Pesticides
Herbicides
Tree Growth Regulator
Ground Water
Drinking Water
Toxicity
Human Health
Vegetation Management
Utility Rights-of-Way (ROWs)

CONTENTS

1 INTRODUCTION AND PURPOSE	1-1
Introduction	1-1
Purpose of Risk Assessment and Mitigation Measures	1-2
Information Sources	1-5
Risk Assessment and Mitigation Organization	1-8
Overview of the Risk Assessment Process	1-9
Hazard and Potency Assessment	1-9
Endocrine Disruptors	1-14
Toxicology Testing for Pesticide Registration	1-15
Exposure Assessment	1-17
Variability and Uncertainty in Risk Assessment	1-19
Identity and Composition of Pesticide Products	1-19
Literature Review	1-22
 2 ELECTRIC UTILITY VEGETATION MANAGENENT PROGRAM.....	2-1
 3 HERBICIDE AND TREE GROWTH REGULATOR PROFILES.....	3-1
Introduction	3-1
2,4-D	3-2
Aminopyralid.....	3-4
Bromacil	3-6
Clopyralid	3-7
Diuron.....	3-8
Fosamine Ammonium	3-10
Flurprimadol	3-12
Glyphosate	3-14
Imazapic.....	3-16
Imazapyr	3-17

Isoxaben.....	3-19
Metsulfuron-methyl.....	3-20
Paclobutrazol	3-22
Pendimethalin	3-23
Picloram	3-26
Tebuthiruon	3-28
Triclopyr	3-30
Trifluralin	3-32
4 PESTICIDE FATE AND EXPOSURE ASSESSMENT	4-1
Pesticide Characteristics	4-1
Pesticide Mobility.....	4-3
Pesticide Persistence	4-4
Soil Conditions that Affect Ground Water Vulnerability and Surface Runoff	4-7
Soil as a Porous Media.....	4-7
Factors that Define Soils as a Porous Media	4-8
Influence of Soil Texture on Permeability and Sorption.....	4-11
Water Resource Characterization	4-12
Watersheds	4-12
Aquifers	4-14
The Hydrologic Cycle	4-15
Summary of Factors that Determine Pesticide Leaching	4-18
Pesticide Environmental Fate and Exposure Modeling.....	4-19
5 PESTICIDES AND GROUND WATER: RISK CHARACTERIZATION AND MANAGEMENT	5-1
Pesticide Risk Characterization.....	5-3
Pesticide Risk Reduction Strategies	5-7
Pesticide Risk Mitigation Measures.....	5-10
Tiered Approach to the Use of Pesticide Risk Mitigation Decision Aids	5-12
Use of the Soil and Water Assessment Tool in Evaluating Alternative IVM Practices.....	5-12
Challenges to Characterizing Pesticide Risks to Ground Water Resources	5-14
6 REFERENCES CITED.....	6-1
A GLOSSARY OF TERMS.....	A-1

LIST OF FIGURES

Figure 1-1 Cancer vs. Non-cancer Risk Assessment	1-10
Figure 4-1 Distribution Coefficient (K_d) and Henry's Law Constant (K_H).....	4-4
Figure 4-2 Pesticide Dissipation from a Surface Soil Compartment	4-5
Figure 4-3 Field Dissipation Half-life	4-6
Figure 4-4 Surface and Subsurface Water Zones	4-12
Figure 4-5 The Hydrologic Cycle	4-16
Figure 4-6 Factors Determining Pesticide Leaching	4-19
Figure 5-1 Continuum of Pesticide Risk Reduction Measures.....	5-8

LIST OF TABLES

Table 1-1 Open Literature Information Resources.....	1-5
Table 1-2 Herbicides and Tree Growth Regulators used on Electric Utility Rights-of-Way	1-8
Table 1-3 U.S. EPA Cancer Classification Scheme.....	1-14
Table 1-4 Battery of Tests for New Pesticide Chemicals (NRC 2006).....	1-16
Table 1-5 Chronic No Observable Adverse Effect Levels for Selected Pesticides	1-17
Table 2-1 Herbicide Treatments Commonly Used on Electric Transmission Rights-of-way in North America	2-3
Table 3-1 2,4-D acid chemical characteristics	3-4
Table 3-2 Aminopyralid chemical characteristics	3-5
Table 3-3 Bromocil chemical characteristics.....	3-7
Table 3-4 Clopyralid chemical characteristics.....	3-8
Table 3-5 Diuron chemical characteristics	3-10
Table 3-6 Fosamine Ammonium chemical characteristics.....	3-11
Table 3-7 Flurprimidol chemical characteristics	3-13
Table 3-8 Glyphosate chemical characteristics	3-15
Table 3-9 Imazapic chemical characteristics	3-16
Table 3-10 Imazapyr chemical characteristics	3-18
Table 3-11 Isoxaben chemical characteristics	3-20
Table 3-12 Metsulfuron methyl chemical characteristics	3-22
Table 3-13 Paclobutrazol chemical characteristics	3-23
Table 3-14 Pendimethalin chemical characteristics	3-26
Table 3-15 Picloram chemical characteristics.....	3-28
Table 3-16 Tebuthiuron chemical characteristics	3-29
Table 3-17 Triclopyr chemical characteristics	3-32
Table 3-18 Trifluralin chemical characteristics	3-34
Table 4-1 Water Resources of Concern	4-15
Table 5-1 Rating Criteria for Pesticide Characteristics	5-4
Table 5-2 Chemical Characteristics and Drinking Water Standards and Guidelines for Herbicides and TGRs.....	5-5
Table 5-3 WIN-PST Soil/Pesticide Interaction Leaching Potential for Three Massachusetts Soils	5-11

1

INTRODUCTION AND PURPOSE

Introduction

There is a growing concern for chemical contamination of ground water (NRC 1984, NRC 1993, USGS 2005). Pesticides, as well as other potentially hazardous chemicals, are used ubiquitously in everyday life. Pesticide use is widespread in urban, industrial, and agricultural settings, and in the case of rights-of-way, applied in a linear fashion across the landscape, often over long distances and intermingled with other uses.

Ground water is a valuable national resource, which can be vulnerable to contamination by pesticides from variety of uses, use as well as from leaks, spills and disposal. Although the full extent of the problem is not known, enough information has been reported to indicate that the problem is widespread in certain areas of the country. Most findings of pesticides in ground water have been at relatively low levels, although some significant levels have been reported in some areas, resulting in numerous well closings. Due to the lack of resources required for widespread monitoring, the full scope of pesticides in ground water remains unclear. However, there is concern that once widespread contamination of ground water by pesticides has occurred, it is often not economically or technically feasible to restore the resource. Even provisions of alternative drinking water or treatment to remove contamination from drinking water before it is used may be impracticable, if contamination is widespread. For these reasons, prevention of unacceptable contamination must be the primary focus of protection efforts.

The potential vulnerability of ground water to pesticide contamination is determined by a complex set of factors, which vary significantly from area to area. Furthermore, the use and value of ground waters vary considerably across the country. In some areas, ground water provides an irreplaceable source of drinking water for large populations, while in other areas, ground water is essentially unusable. These highly variable characteristics of the ground-water resource and the area-specific nature of the pesticide contamination concern suggest the need for a localized (site-specific) protection approach.

The reliable assessment of the risks arising from pesticide contamination of ground water, and the design of efficient and effective mitigation measures, require the capability to predict the behavior of pesticides from initial deposition and redistribution at the application site, movement with water percolating down through the aerated soil and unsaturated (vadose) zone, and in flowing ground water. Useful predictions of the potential for pesticides to move towards ground water require an understanding of the processes controlling fate and transport, including soil sorption, diffusion, hydrodynamic dispersion, and chemical and biological reactions that affect soluble concentrations in the ground. On the landscape surface, pesticide foliar or soil residues may volatilize to the atmosphere, be transformed by sunlight, or degrade chemically. However,

biologically-mediated processing by the soil microbial community (and to a lesser extent by plant uptake and metabolism) is often the predominant mechanism of pesticide degradation. Below the 2-3 ft soil root zone there is often a reduction in such biological processes, which slows the degradation of most pesticides. As biological degradation is significantly diminished, pesticides tend to persist longer once they reach ground water. In addition, when hydrogeologic conditions result in slow rates of ground water movement and natural flushing of aquifers, pesticides and other contaminants can persist for years (NAS, 1984).

Ground water vulnerability of pesticide contamination is highly variable and depends on many factors. These include landscape characteristics including topography and vegetation, soil physical and chemical properties, soil hydraulic properties, the presence of macropores, sinkholes, and other preferential flow paths, the depth of the ground water resource of concern, the presence of restrictive layers between the vadose zone and the ground water resource, and climate (precipitation). Ground water is the subsurface transporting agent for dissolved chemicals including pesticide contaminants. Once pesticides reach ground water they be transported from the application site by ground water flow; one potential result is the contamination of water drawn from wells down gradient. In addition, natural discharges of an aquifer, such as springs and seeps, can return pesticides to the surface.

Pesticides vary greatly in their hazard to humans: while we strive to keep all pesticides out of ground water at any concentration, there is greater concern for exposure to pesticides that are considered more hazardous to human health. Consequently, in evaluating the potential human health risks of electrical power transmission rights-of-way pesticide use practices that may result in ground water contamination, both pesticide toxicity and environmental fate will be considered.

Purpose of Risk Assessment and Mitigation Measures

The modern use of pesticides in the United States (U.S.) began after World War II. The early success of the insecticides parathion and DDT, as well as the herbicide 2,4 -D fostered a revolution in the use of chemicals as a primary means of pest control. However, it was not until the late 1970's that the potential for pesticides to contaminate ground water became a widespread concern. In 1979, two pesticides were discovered in ground water: dibromochloropropane (DBCP) in California and aldicarb in New York. Additional monitoring in other states shortly thereafter showed DBCP in ground water in Arizona, Hawaii, Maryland, and South Carolina; and aldicarb was found in Wisconsin in 1980.

Perhaps the most serious case of pesticide contamination of ground water was the discovery in 1982 of ethylene dibromide (EDB) in two California wells and in three wells in Georgia. By the end of 1983 EDB contamination of ground water had been discovered in 16 different counties in California, Florida, Georgia, and Hawaii. These findings caused United States Environmental Protection Agency (U.S. EPA) to issue an immediate suspension of all EDB soil uses in September 1983.

Until these discoveries of pesticides in ground water federal and state agencies did not monitor ground water for pesticides. There were several reasons: most ground-water monitoring until that time focused on urban rather than rural agricultural areas; analyses of water were usually for volatile organic contaminants, while most pesticides have low volatility; and reports of positive

findings of organic contaminants did not always distinguish between surface and ground-water systems.

The discovery of DBCP, aldicarb, and EDB in certain areas of the country stimulated a number of monitoring activities by federal and state agencies to investigate the extent of the problem. By 1986, a total of 19 different pesticides had been detected in ground water in 24 states where the source of the contaminant was most probably a result of agricultural application (non-point source) rather than from spills or other point sources of the pesticides (Garner, et al., 1986).

These finding suggested that ground water contamination resulting from normal pesticide use applications could occur over large geographical areas and result in exposure to large segments of the population.

Some of the more important findings from early state monitoring efforts are, as follows:

- California: Approximately 57 different pesticides were detected in California's ground waters.
- Hawaii: Thirteen public drinking water wells were found to be contaminated by EDB, DBCP, and/or trichloropropane.
- Florida: EDB was found in about 10% of public and private drinking water wells.
- New York: On Long Island, almost 2,000 wells were found to contain aldicarb; in about 50% of these wells the aldicarb concentration was above the New York State standard of 7 ppb.
- Minnesota: In 1986, one or more pesticides were detected in 23% of 225 private wells and 29% of 366 public wells sampled.
- Iowa: Nine herbicides and two insecticides were detected in monitoring studies conducted in Iowa; concentrations detected were generally less than one part per billion.

In the intervening years since the concern for pesticides in ground water was firmly established, monitoring has continued and educational programs have been developed, which promote pest management practices and mitigation measures designed to reduce the risk of ground water contamination. Monitoring at the federal level is largely the purview of the U.S. Geological Survey (USGS) within the Department of the Interior (DOI). In addition, state environmental agencies have ongoing monitoring programs, most notable is California's annual sampling for pesticides in well water, as mandated by the Pesticide Contamination Prevention Act of 1986.

In 2003, the California Department of Pesticide Regulation published a report of annual well water monitoring results from 1985-2003 (CDPR 2003). Eight herbicides that typically are used on electrical utility rights-of-way were included. In descending order of detections per number of samples analyzed, the results were: diuron 472/7,534, bromacil 256/8,996, 2,4-D 16/6,500, tebuthiuron 4/149, trifluralin 3/793, picloram 2/4,200, glyphosate 1/3,891, and pendimethalin 0/164.

As a part of a decade-long (1992–2001) national study by the USGS National Water-Quality Assessment (NAWQA) Program, the occurrence of pesticides in ground water was evaluated. Ground-water samples were collected from 5,047 wells. Most water samples were analyzed for

75 pesticides and 8 degradates, including 20 of the 25 most heavily used herbicides and 16 of the 25 most heavily used insecticides. The most frequently detected pesticides included 4 herbicides that typically are used on electrical utility rights-of-way: 2,4-D, bromacil, diuron, and trifluralin. However, these products are also used in other rights-of-way sites, agriculture, and urban sites. Modern analytical methods were employed lowering the detection limit and the limit of reliable measurement, thereby increasing the likelihood of detecting and quantifying pesticides present. Among the major findings are that pesticides are frequently present in ground water, but are seldom at concentrations likely to affect humans (USGS 2005).

As concentrations of pesticides in ground water have been found at low levels, most of the concern has been focused on the potential for chronic adverse health effects.

To understand the possible human health risks associated with pesticides in ground water, federal and state agencies use human health risk assessments as a fundamental tool to characterize the potential for harm to human health by chemical substances in the environment. These assessments form a key part of the basis upon which policy makers determine whether, and to what extent, measures to reduce risks are warranted.

United States Environmental Protection Agency (U.S. EPA) and state agencies use quantitative methods to estimate risks as a basis of most regulatory decisions. Risk assessment methods are based on a four-part process (NRC 1983), which includes: (1) hazard identification, (2) dose-response evaluation, (3) human exposure evaluation, and (4) risk characterization. The use of quantitative risk assessment facilitates discrimination between important and trivial risks, and a means to evaluate tradeoffs and set priorities, and allocate resources. Quantitative risk assessment methods often include “default options” to deal with uncertainty. Default options are used in the absence of convincing scientific knowledge where assumptions are required to assess exposure or risk. They are designed to be plausibly conservative, usually resulting in an overestimate of risk.

In characterizing the risks of pesticides in drinking water, U.S. EPA evaluates each pesticide’s human health hazards, potency (dose-response), and exposure potential. In evaluating the potential for exposure to pesticides in drinking water, U.S. EPA considers drinking water consumption patterns across the United States (U.S.) to discern regional differences. Differences may result from combination of factors including pesticide use patterns, the contribution and vulnerability of the drinking water source (surface or ground water) and drinking water treatment. The outcome of a quantitative risk assessment is a pesticide concentration in drinking water, resulting in an estimated daily exposure, above which, exceeds a level of concern. In addition, periodically U.S. EPA evaluates whether pesticide use practices are likely to result in pesticide drinking water concentrations that exceed a level of concern. This can result in cancellation of pesticide products or further restrictions on use.

A detailed understanding of pesticide properties (chemical characteristics and toxicity), and the relationship between pesticide use practices and the potential for ground water contamination, should allow for the development of integrated vegetation management practices and mitigation measures necessary to eliminate pesticide ground water contamination, or reduce contamination below a level of concern. Adopting practices that reduce or elimination pesticide ground water contamination may allow for the continued use of economical and efficacious pesticide products that would otherwise be lost through cancellation.

Information Sources

Information resources used for this report can be categorized as follows: 1) open literature, 2) information associated with pesticide product registration provided on U.S. EPA 's website, 3) information provided by the National Pesticide Information Center including EXTOWNET, 4) information from other credible sources, such as the US Geological Survey (USGS), the US Forest Service (USFS), the US Department of the Interior Bureau of Land Management (BLM), the World Health Organization (WHO), and the Centers for Disease Control (CDC) Agency for Toxic Substances and Disease Registry (ATSDR).

There are many commercial databases that can be used to search the published literature. Those used for this report are shown in Table 1-1.

Table 1-1
Open Literature Information Resources

Open Literature	Database Provider
TOXLINE	Cambridge Scientific Abstracts (CSA ProQuest)
PubMed (MEDLINE)	National Library of Medicine (NLM)
AGRICOLA	USDA National Agricultural Library
Web of Science	ISI Web of Science (Web of Knowledge)
SciFinder Scholar	Chemical Abstracts Service (CAS)
Environmental Sciences & Pollution Management (ESPM)	Cambridge Scientific Abstracts (CSA ProQuest)
Aquatic Sciences & Fisheries Abstract (ASFA)	Cambridge Scientific Abstracts (CSA ProQuest)
Orbis Academic Union Catalog	Orbis
WorldCat	FirstSearch
OSU Libraries Research Databases	Oregon State University Library
Electronic Journals	Oregon State University Library

Initial searches on-line searches are or TOXLINE, PubMed (MEDLINE), and AGRICOLA, which often identify most of the relevant published literature. TOXLINE (Toxicology Literature Online), a bibliographical database constructed by the U.S. National Library of Medicine (NLM), covers the pharmacological, biochemical, physiological, and toxicological effects of pesticides and agricultural and industrial chemicals that may included in the formulated product. TOXLINE is a collection of databases derived from BIOSIS (up to 2002), National Library of Medicine, American Society of Hospital Pharmacists, National Institute for Occupational Safety

and Health, Environmental Mutagen Information Center, Environmental Teratology Information Center, and U.S. Environmental Protection Agency. AGRICOLA, which was created and is maintained by the National Agricultural Library, is a bibliographical database of citations covering the agricultural and forestry literature. AGRICOLA is the most comprehensive database of bibliographical information available in agricultural research.

As required by the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), pesticides must be registered by the U.S. EPA prior to sale and use. For many pesticides, particularly those developed only in the past decade, the most relevant and critical information is found in unpublished studies submitted by the registrant of the pesticide to the U.S. EPA as part of the registration package. These studies are classified as “Confidential Business Information” and cannot be accessed without special clearance from the U.S. EPA. However, U.S. EPA publishes summaries of this information in Federal Register notices, Reregistration Eligibility Decision (RED) documents or other Agency publications such as Science Chapters prepared by Health Effects Division (HED) or the Environmental Fate and Effects Division (EFED) of the Office of Pesticide Programs.

A major advantage of the FIFRA studies submitted for pesticide registration is that they follow a relatively uniform set of guidelines or study protocols¹. These guidelines are intended to constitute a consistent set of study standards that are used by the Office of Pesticide Programs (OPP) as well as Office of Pollution Prevention and Toxics (OPPT). A very large number of guidelines are available in ten different areas:

The OPPTS harmonized guidelines are organized in the following 10 series:

- 810 - Product Performance Test Guidelines
- 830 - Product Properties Test Guidelines
- 835 - Fate, Transport and Transformation Test Guidelines
- 840 - Spray Drift Test Guidelines
- 850 - Ecological Effects Test Guidelines
- 860 - Residue Chemistry Test Guidelines
- 870 - Health Effects Test Guidelines
- 875 - Occupational and Residential Exposure Test Guidelines
- 880 - Biochemicals Test Guidelines
- 885 - Microbial Pesticide Test Guidelines

In addition, studies accepted by U.S. EPA for pesticide registration or reregistration purposes must comply with Good Laboratory Practices (GLPs). GLPs² are federal regulations promulgated by both the Food and Drug Administration (FDA) in 21 CFR Part 58 and the U.S. EPA. U.S.

¹ <http://www.epa.gov/opptsfrs/home/guidelin.htm>

² <http://www.ovpr.uga.edu/qau/resources/glps/>

EPA has separate GLPs for FIFRA in 40 CFR Part 160³ and for TSCA in 40 CFR Part 792. Under FIFRA, GLP regulations describe the minimal standards for conducting laboratory and field studies that support or are intended to support pesticide registration or reregistration. In addition, GLP compliant studies often require a greater degree of transparency - from study design, to sampling methods, analysis, and data interpretation. Lack of compliance with GLPs generally results in studies not being accepted by U.S. EPA.

In evaluating information in the open literature, and that provided to U.S. EPA as a part of the registration or reregistration process, greater weight is given to those studies that were specifically designed to be used for risk assessment purposes, and those conducted under GLPs.

For the most part, this report is based on primary literature, either from the open literature or U.S. EPA documents. In some cases, credible reviews were used directly as both a source of information and as the basis for risk assessment. In addition, some of the pesticides have been the subject of recent reviews and risk assessments by other agencies or organizations and it simply would not make sense to duplicate the effort. In general, credible reviews are limited to groups such as the U.S. EPA, the World Health Organization (WHO), the USGS, the USFS, and the Agency for Toxic Substances and Disease Registry (ATSDR). The U.S. EPA has conducted a large number of reviews on pesticides, and other chemicals that may be used in pesticide formulations. ATSDR, a part of the Centers for Disease Control (CDC), provides public health-related analyses specifically related to hazardous wastes and environmental spills of hazardous substances. These activities include the preparation of toxicological profiles for hazardous substances. While ATSDR reviews focus on chemicals other than pesticides, some of the chemicals reviewed are pesticide contaminants, such as hexachlorobenzene, which is a contaminant in two herbicides typically used in rights-of-way vegetation management; picloram and clopyralid.

The World Health Organization has conducted a large number of reviews on both pesticides and industrial chemicals, published in the Programme on Chemical Safety (IPCS) Environmental Health Monographs⁴. These reviews are particularly useful in that they often contain summaries of unpublished studies from Europe that were submitted to the WHO in support of the monograph preparation.

Google and Google Scholar searches were also conducted only when information was not available from the on-line resources given in Table 1-1. In addition, web sites of the chemical manufacturers and some environmental groups contain information pertinent to the risk assessment. These sites were used sparingly and with discretion in identifying reliable sources of information.

³ http://www.access.gpo.gov/nara/cfr/waisidx_06/40cfr160_06.html

⁴ <http://www.inchem.org/pages/ehc.html>

Risk Assessment and Mitigation Organization

Presented in this report are human health hazards and potency for electric utility rights-of-way herbicide products and tree growth regulators (TGRs) given in Table 1-2. In addition, to assess the potential for human exposure to the product active ingredients in drinking water, information used to evaluate their behavior in the environment is also provided. Included is information that will allow the evaluation of herbicide and TGR use practices, chemical characteristics, and site conditions in assessing chemical leaching potential and ground water vulnerability. Together this information can be used to evaluate electric utility herbicide and TGR use practices with regards to the potential for ground water contamination.

Table 1-2
Herbicides and Tree Growth Regulators used on Electric Utility Rights-of-Way

Trade Name	Active Ingredients	Formulation	Signal Word
Accord	Glyphosate	Liquid	Caution
Aqua Neat	Glyphosate IPA salt	Liquid	Caution
Arsenal	Imazapyr	Liquid/Granule	Caution
Chopper	Imazapyr	Ready to use	Caution
Escort	Metsulfuron Methyl	Dry flowable	Caution
Garlon 4	Triclopyr acid	Liquid	Caution
Garlon 3A	Triclopyr BEE	Liquid	Danger
Journey	Imazapic/glyphosate	Liquid	Caution
Karmex XP	Diuron	Dispersible granule	Caution
Krenite S	Fosamine Ammonium	Liquid	Caution
Krovar DF	Diuron/Bromacil	Dispersible granule	Caution
Milestone	Aminopyralid	Liquid	Caution
Pathfinder II	Triclopyr TEA	Ready to use	Caution
Pathway	Picloram, 2,4-D	Liquid	Caution
Pendulum Aqua Cap	Pendimethalin	Microencapsulated	Caution
Razor Pro	Glyphosate IPA salt	Liquid	Caution
Roundup	Glyphosate	Liquid	Caution
Roundup Pro	Glyphosate	Liquid	Caution
Snapshot	Trifluralin, Isoxaben	Granules	Caution
Spike 20P	Tebuthiuron	Wettable granule	Caution
Tahoe 4E	Triclopyr BEE	Emulsifiable concentrate	Caution
Transline	Clopyralid	Liquid	Caution
Tordon	Picloram	Liquid	Caution
Tree Growth Regulators (TGRs)			
Profile 2SC	Paclobutrazol	Liquid	Caution
Cutless	Flurprimidol	Water soluble powder	Caution

This is a technical support document, and it addresses some specialized technical areas. However, an effort was made to ensure that the document could be understood by individuals who do not have specialized training in the chemical and biological sciences. Some of the more complicated terms and concepts are defined, as necessary, in the text. In addition, in Appendix A contains a glossary of terms.

Overview of the Risk Assessment Process

According to the National Research Council of the National Academy of Sciences paradigm (NRC 1983, NRC 1994, NRC 2009), U.S. EPA assesses pesticide human and ecological risk employing a strategy composed of four broad but interrelated components; 1) hazard identification, 2) potency, i.e., dose-response relationship, 3) exposure assessment, and 4) risk characterization. Hazard identification is a qualitative evaluation of the evidence that suggest exposure to an environmental agent will result in adverse health effects in humans or wildlife. This may include evaluation of toxicity data that provide information on the nature and severity of health effects, or the use of structure activity relationships to infer the nature of toxicity for structurally related agents. Potency is a quantitative analysis of the relationship between dose and the incidence of adverse health effects (i.e., dose-response). Exposure assessment is the evaluation of all exposure pathways that may result in contact between the agent and humans or wildlife. Exposure assessment combines knowledge of use practices that determine an agent's initial distribution in the environment, environmental chemistry that determines fate and transport, and the behaviour of humans and wildlife that determines exposure to an environmental agent. Combining information on hazard, potency, and exposure, risk characterization estimates the likelihood of a specified detrimental health effect from exposure to an environmental agent in a defined population. Risk management is used to make policy decisions by considering risk characterization ("the science") along with social, political, and economic considerations.

Hazard and Potency Assessment

For the registration and reregistration of pesticides under FIFRA, hazard and potency are determined primarily from animal studies. The specifics of the animal studies can be found in EPRI report no. 1005367 "Human Health Risk Assessment of Chemicals Encountered in Vegetation Management on Electric Utility Rights-of-Way." Discussed here is how the results of the animal studies are used to assess hazard and potency.

Pesticide hazard is divided into two categories; those chemicals which are considered carcinogens and those that are not. For non-carcinogens it is assumed that there is a dose, below which there will be no observable adverse effects. In toxicity testing, the highest dose resulting in no observable adverse effects is called the NOAEL and the lowest dose with an observable adverse effect is called the LOAEL. This approach implies that for a specified adverse health effect, a series of increasing doses can discriminate between some effect and no effect; somewhere between the NOAEL and the LOAEL is the "threshold" of the quantal all-or-none-response. A threshold response is also characterized as non-linear, as plotting dose vs. response will result in a departure from linearity at or near the threshold (see Figure 1-1).

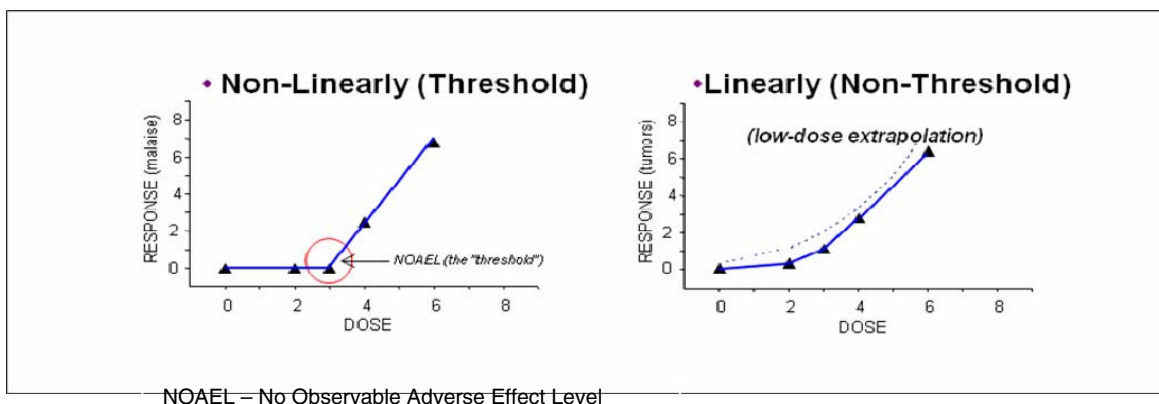


Figure 1-1
Cancer vs. Non-cancer Risk Assessment

Animal studies are commonly used to assess risks to humans. To establish a NOAEL from animal studies requires at least four doses, including a treatment control. Once the NOAEL is established the rest of the data are ignored. Despite preliminary range-finding studies, sometimes the NOAEL cannot be determined, and the LOAEL is used. An alternative approach called the

Benchmark Dose (BMD) is currently being used by some risk assessors, and is in many cases considered a superior method by U.S. EPA. The benchmark dose approach regresses all of the data to estimate a specified response level – the benchmark response – usually associated with a low level of risk such as 1-10%. The achievable low risk value and its lower confidence bound are usually determined the statistical significance of the data. Commonly used values are the BMD_{10} – the dose with where 10% of the animals were determined to have observable adverse effects – and the $BMDL_{10}$ – the 95% confidence bound on the BMD_{10} . The benchmark dose approach can be used for both threshold and non-threshold dose-response analysis. U.S. EPA has developed software to assist in curve fitting and statistical analysis⁵.

Pesticide threshold risk: the Reference dose (RfD) is “an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.”

The NOAEL, LOAEL, or BMDL determined from animal studies is used to determine a benchmark for acceptable intake in humans. Safety factors are added to account extrapolation from animals to humans, as well as variation in human susceptibility and other uncertainties. In addition, for exposure to pesticides in food (including drinking water), potential adverse health effects in sensitive subpopulations must also be considered, which may result in an additional safety factor. For pesticides, acceptable intake via oral exposure is determined on a daily basis; the resulting U.S. EPA benchmark is called the Reference Dose or RfD, expressed in mg/kg body wt/day. In the case where additional safety factors are used to address dietary risks to sensitive subpopulations, the benchmark is the PAD, or population adjusted dose. For varying

⁵ http://www.epa.gov/ncea/bmds/bmds_training/software/overp.htm

durations of exposure, the acute or chronic RfD or PAD may be used to characterize risk (referred to as aRfD, cRfD, aPAD, cPAD). These benchmarks are based on acute and chronic NOAELs, LOAELs, or BMDLs. For chronic inhalation exposure, the U.S. EPA benchmark is the reference concentration or RfC. Other agencies, such as ATSDR and WHO, use the NOAEL, LOAEL, or BMDL to calculate minimum risk levels (MRLs) and acceptable (or allowable) daily intakes (ADIs).

The **RfD** is determined by dividing the NOAEL by a uncertainty factor (UF), usually between 100 and 1000

- 10X – uncertainty in extrapolating from animal studies to humans (interspecies).
- 10X – to account for variation in human susceptibility (intraspecies).
- 2-10X – optional factor for inconsistent data
- 2-10X – to account for sensitive sub-populations¹ (e.g. children) used to determine Population Adjusted dose (PAD)

¹The Food Quality Protection Act of 1996 (FQPA) requires U.S. EPA to make a determination if an additional factor necessary.

For carcinogens the default assumption is that no threshold exists. A linear dose-response, for which there is no threshold, implies that exposure to a pesticide resulting in a single molecule reaching the target (i.e., nuclear DNA) may lead to cancer. Cancer risk associated with pesticide exposure is assessed in terms of probability; cancer resulting from exposure to a single molecule may be possible (i.e., biologically plausible), but not probable. Lifetime cancer risk in the US population (for all risk factors) is approximately 1 in 2 for men and 1 in 3 for women⁶. In regulating pesticides and other environmental agents, U.S. EPA's goal is to prevent excess cancers above these background rates. A common benchmark for acceptable excess lifetime cancer risk associated with pesticide exposure is the probability of less than 1 in a million or 10^{-6} . Why $< 10^{-6}$? At some point in the regulation of pesticides as food additives (NRC 1987), a probability of $< 10^{-6}$ excess cancer risk was equated with *de minimis*, or nearly zero risk. For some types of cancer there may be a threshold for effects. In regulating pesticides as carcinogens, the default assumption is that the dose-response is linear (no threshold). To demonstrate a threshold exists requires sufficient evidence describing a mode of action that supports a threshold response, and animal studies showing the dose-response for tumor incidence is non-linear.

For threshold carcinogens U.S. EPA, establishes a "Margin of Exposure" (MOE) between NOAEL, LOAEL, or BMDL and the benchmark for acceptable intake. A commonly used MOE is 100. Currently there are no pesticides classified as carcinogens for which U.S. EPA has sufficient data to establish an MOE.

⁶ <http://www.cancer.org/>

Animal studies are commonly used to assess pesticide cancer risk in humans. To estimate the probability of excess cancer in humans, animals (usually rats or mice) are fed high doses of a pesticide, up to a maximum tolerated dose (MTD). The MTD should produce limited toxicity when administered for the duration of the test period. It should not induce: overt toxicity, for example appreciable death of cells or organ dysfunction, or toxic manifestations that are predicted materially to reduce the life span of the animals except as the result of neoplastic development, or 10% or greater retardation of body weight gain as compared with control animals. In some studies, toxicity that could interfere with a carcinogenic effect is specifically excluded from consideration.

This conservative approach is necessary to allow for the detection of tumors even at low incidence. For example, a study with 60 animals per treatment would require at least 3 animals with tumors to be considered significantly different than the control. That translates to a cancer incidence of 5% or 1 in 20. To conduct a two-year study with 4 doses for both male and female rodents and 4 interim sacrifices would require approximately 3840 animals. Concomitantly, studies using low doses relevant to human intake to detect excess cancer risk at rate of 1 in a million could take millions of rodents. Therefore, to estimate excess cancer risk in humans, high dose animal studies, with a tumor incidence detection limit of approximately 1 in 20, are extrapolated to low doses associated with a 1 in a million increased probability of cancer in humans. Historically, a number of statistical methods have been used to extrapolate the high dose region used in animal studies to the low dose region relevant to human exposure. Currently, the method of choice is to draw a straight line between the point of departure (POD), i.e., the BMDL or the lowest dose for which the tumor rate exceeded background, and zero⁷. For linear cancer effects the carcinogenic potency is defined by the dose which results in an unacceptable probability of increased risk of cancer in a lifetime (i.e., $> 10^{-6}$ or 1 in a million); the lower the dose the more potent the carcinogen. Carcinogenic potency is expressed as Q_1^* , determined from the slope of the dose response curve in the low dose range (between the POD and zero). In addition, the upper bound of the 95% confidence limit is used to account for extrapolation from animals to humans, variation in human susceptibility, and other uncertainties. For non-linear cancer effects the toxicity is expressed as the point of departure (POD). The POD is determined from the NOAEL, LOAEL, or BMDL, and an MOE is used to determine acceptable intake.

<p>Pesticide cancer risk (non-threshold): The amount of a pesticide residue a person could consume daily for 70 years that would result in no more than 1-in-a-million (10^{-6}) increased chance of developing cancer as a direct result of consumption of (exposure to) that chemical.</p>

⁷ <http://www.epa.gov/opp00001/health/cancerfs.htm>

For both threshold and non-threshold effects, toxicity testing used to assess human health risks covers a broad range of adverse effects, to include cancer, effects on the nervous system, immune system, endocrine system, reproductive and teratogenic effects, irritation and sensitization of the skin and eyes. A variety of animals are used to assess pesticide toxicity, including rats, mice, rabbits, dogs, and hens. Tests are divided into acute, sub-chronic. Although human data would be preferred, rarely are pesticides tested on humans. Animal species responding most like humans are preferred. Likely route(s) of exposure (oral, dermal, inhalation) associated with anticipated pesticide use practices is also considered when designing animal studies.

For cancer risk, in addition to *in vitro* assays, such as the Ames test, and animal studies, human epidemiological data may also be considered. Risk assessors will assemble a data set comprised primary of studies required for registration, but also pertinent studies in the open literature. The quality of the data will be evaluated, and a weight-of-the-evidence approach will be used to determine where there is sufficient evidence to support a relationship between pesticide intake and an increased chance of developing cancer. The characteristics of a good data set include:

- Similar route as human exposure
- Similar pharmacokinetics⁸ and mechanism of action
- Dosing is at and below the maximum tolerated dose
- Dosing is chronic, preferably lifetime
- Large number of dose groups
- Large number of animals
- Proper controls
- Good survival
- Dose-response relationship evident
- Statistically significant increase or trend in tumor incidence
- Malignancy increases with dose and with time
- Latency decreases with dose

Using the weight-of-the-evidence approach, the summarized human and animal data are designated as sufficient, limited, inadequate, no data, or no evidence. Other evidence from short-term tests, pharmacokinetics, or structure-activity relationships is evaluated. Under the 1986 cancer guidelines, U.S. EPA would then classify overall weight-of-the-evidence using the above scheme shown in Table 1-3.

⁸ Pharmacokinetics is the study of the time course of adsorption into the bloodstream, distribution to organs, metabolism, and elimination from the body.

Table 1-3
U.S. EPA Cancer Classification Scheme

A	Human carcinogen	<i>Sufficient</i> human evidence
B ₁	Probable human carcinogen	<i>Limited</i> human evidence
B ₂	Probable human carcinogen	<i>Sufficient</i> animal evidence
C	Possible human carcinogen	<i>Limited</i> animal evidence
D	Not classifiable	<i>Inadequate</i> human and animal evidence
E	Evidence of noncarcinogenicity	Sufficient negative evidence

A similar scheme is used by the WHO International Agency for Research on Cancer (IARC). U.S. EPA's revised Guidelines for Carcinogenic Risk Assessment⁹, finalized in 2005, do not include the A-B-C-D-E classification scheme. Instead there is a provision for a weight-of-the-evidence narrative. However, many currently used pesticides are classified as carcinogens using the A-B-C-D-E classification, and IARC continues to use a similar classification.

Endocrine Disruptors

Beginning in the 1940's, scientists were aware that the organochlorine insecticide DDT could affect reproductive organ development in young roosters (Burlington and Lindeman 1950). In the ensuing decades, numerous studies suggested a link between chemicals in the environment and reproductive impacts in wild bird populations (Ames 1966, Keith 1966, Wurster and Wingate 1968). It was not until the early 1990s, however, that the term 'endocrine disruption' was conceived.

The concept of endocrine disruption pertains to multiple chemical classes, mechanistic pathways, target organisms, and biological endpoints. The term endocrine disruptor represents a class of mechanisms involving hormone signal disruption that may result in abnormalities in humans and animals that arise at varying stages of development in response to different levels of exposure to hormonally active chemicals (Krimsky 2001).

By U.S. EPA's working definition, endocrine disruptors "interfere with the synthesis, secretion, transport, binding, action, or elimination of natural hormones in the body that are responsible for the maintenance of homeostasis (normal cell metabolism), reproduction, development, and/or behavior." Many endocrine disruptors are thought to mimic hormones. They have chemical properties similar to hormones that allows binding to hormone specific receptors on the cells of target organs. However, endocrine disruptor chemistry varies greatly, as does potency – the effectiveness in binding and "turning on" the response. Most endocrine disruptors have very low

⁹ <http://www.epa.gov/cancerguidelines>

potency as their chemistry is significantly different from the hormones they mimic. Lower potency means that a greater amount of endocrine disruptor is required to elicit the same response of the hormone they mimic. In addition to potency, the potential for a hormone-like effect depends on dose. For all known endocrine disruptors there is some dose, below which there will be no effect. At doses slightly above this threshold some endocrine disruptors elicit a beneficial effect, whereas at higher doses the effect is adverse (harmful).

In response to concerns that chemicals have not been properly tested for endocrine disruption, new testing requirements were included in both the Clean Water Act and Food Quality Protection Act (FQPA). U.S. EPA established the Endocrine Disruptor Screening and Testing Advisory Committee (EDSTAC)¹⁰ to oversee investigations into endocrine disrupting chemicals (EDCs). Due to the wide-ranging chemical classes, mechanisms, and targets involved, the agency has initiated a system of high-throughput screening assays to initially identify EDCs. Subsequent, more in-depth tests in a variety of target organisms are designed to clarify mechanism. Over a decade later, the U.S. EPA is still considering appropriate testing strategies and endpoints. At issue are that current testing paradigms used for assessments of reproductive and developmental toxicity do not adequately address dose selection, age of animals at evaluation, and endpoints measured in proposed studies (Melnick et al. 2002). Additionally, studies have expanded from initial inquiries into reproductive system perturbation to thyroid hormone signaling disruption and disruption of bone mineral deposition. As many endocrine disruptors are thought to affect sex hormones, and therefore reproduction, the findings in multigeneration animal studies, currently required for pesticide registration by U.S. EPA, can provide strong evidence of the potential for endocrine disruption. Currently, U.S. EPA does not require additional testing for endocrine disruptor, but the Agency has recently informed registrants of 6 pesticides suspected to be endocrine disruptors that they must provide data necessary to evaluate risks.

Toxicology Testing for Pesticide Registration

As a part of the registration, pesticide products are subjected to a battery of tests designed to evaluate all plausible hazards (toxic endpoints) and assess potency (dose-response) in humans. These tests include those shown in Table 1-4. Pesticides are segregated into those that will be used on food crops and those that will not. On a case-by-case basis, some tests are required and others are conditional. For additional information see “Overview of Risk Assessment in the Pesticide Program¹¹” on U.S. EPA’s website, and “Toxicity Testing for Assessment of Environmental Agents: Interim Report” (NRC 2006). From the battery of tests listed in Table 1-4, U.S. EPA chooses the most sensitive tests to establish the acute NOAEL, LOAEL, or BMDL, the chronic NOAEL, LOAEL, or BMDL, and the cancer potency is expressed as Q_1^* . The agency assumes that this approach is protective of human health in that all plausible toxic effects have been evaluated and by choosing the most sensitive test to establish the risk number, it is protective of all other adverse outcomes. For example, Table 1-5 shows an example of the results of animal testing to determine the NOAEL for chronic effects. The lowest NOAEL would be used to determine the Reference dose (RfD).

¹⁰ [http://www.U.S. EPA .gov/endo/pubs/edspoverview/edstac.htm](http://www.U.S.EPA.gov/endo/pubs/edspoverview/edstac.htm)

¹¹ http://www.epa.gov/pesticides/about/overview_risk_assess.htm#health

Table 1-4
Battery of Tests for New Pesticide Chemicals (NRC 2006)

Acute tests
Acute oral toxicity—rat
Acute dermal toxicity
Acute inhalation toxicity—rat
Primary eye irritation—rabbit
Primary dermal irritation
Dermal sensitization
Delayed neurotoxicity—hen
Subchronic testing
90-day feeding studies—rodent and nonrodent
21-day dermal toxicity
90-day dermal toxicity
90-day inhalation—rat
90-day neurotoxicity—hen or mammal
Chronic tests
Chronic feeding of two species—rodent and nonrodent
Oncogenicity study of two species—rat and mouse preferred
Teratogenicity in two species
Reproduction—two-generation
Mutagenicity tests
Gene mutation
Structural chromosomal aberration
Other genotoxic effects
Special tests
General metabolism
Dermal penetration
Domestic animal safety

Table 1-5
Chronic No Observable Adverse Effect Levels for Selected Pesticides

Pesticide	90 day rat NOAEL (mg/kg/day)	90 day dog NOAEL (mg/kg/day)	1-year dog NOAEL (mg/kg/day)	2-year rat NOAEL (mg/kg/day)	Lowest NOAEL (mg/kg/day)
2,4 D	15	1	1	5	1
Acetochlor	80	10	2	10	2
Atrazine	1	6	5	3.5	1
Carbaryl	125	1	3.1	10	1

Exposure Assessment

For pesticide human health risk assessment, estimates of exposure results in a dose that is compared to an acceptable intake to determine risk. Exposure pathways include oral, inhalation, and dermal. Expected duration of exposure determines the need to assess acute, sub-chronic, and/or chronic risk. U.S. EPA uses pesticide use data to focus efforts in exposure assessment. The primary exposures of interest are pesticides in food, drinking water, the home, and the work place. What level of pesticide exposure is acceptable (i.e., does not exceed a “level of concern”) is ultimately determined by compliance with federal statutes and accompanying regulations. The Federal Insecticide Fungicide and Rotenticide Act (FIFRA), which authorizes the regulation of pesticides in the U.S., requires that pesticides registered by U.S. EPA do not pose “unreasonable adverse effects on the environment”¹². In addition, for those pesticides used on food, the Food Quality Protection Act of 1996, which amends both FIFRA and the Food Drug and Cosmetic Act (FDCA), requires “that there is a reasonable certainty that no harm will result from aggregate exposure to the pesticide chemical residue, including all anticipated dietary exposures and all other exposures for which there is reliable information.” In addition, 1996 Amendments to the Safe Drinking Water Act (SDWA) provide for source water assessment and protection programs to prevent drinking water contamination. States are required to develop comprehensive Source Water Assessment Programs (SWAPs) that will “identify the areas that supply public tap water; inventory contaminants and assess water system susceptibility to contamination and inform the public of the results.” U.S. EPA Office of Water is responsible for the review and approval of state SWAPs. Several programs specifically address ground water protection. For the purpose of evaluating herbicides used in electrical utility rights-of-way vegetation management, the risk assessment will characterize risks from an evaluation of the human health hazards and potency of these herbicides, as well as the relative potential to leach to ground water, which determines the likelihood of human exposure in drinking water. Drinking water standards, health advisories, and other human health benchmarks will be used to derive a level of concern. This will allow the

¹² FIFRA section 2(bb) defines “unreasonable adverse effects on the environment” to mean, in part, “any unreasonable risk to man or the environment, taking into account the economic, social, and environmental costs and benefits of the use of any pesticide....” (<http://www.epa.gov/pesticides/health/risk-benefit.htm>)

evaluation of the relative risk of herbicide use practices resulting in ground water concentrations that exceed a level of concern.

As required by FQPA, U.S. EPA has developed aggregate exposure and risk assessment methods¹³ that account for exposure to a pesticide by multiple routes and from multiple sources, including food, drinking water, residential, and other non-occupational sources. In developing aggregate risk assessment methods, U.S. EPA considered multiple exposures from each source via oral, dermal, and inhalation routes.

Each evaluation is highly specific to the source and considers almost all possible scenarios of exposure. For instance, U.S. EPA has evaluated approximately 30 different residential pesticide exposure scenarios, covering uses ranging from lawn and garden care and household insect control, to exposures to humans due to pet treatments and wearing clothes impregnated with insect repellents. Accurately assessing the complex pesticide risks from all these varied use patterns required the creation of new "aggregate exposure" procedures capable of combining exposures received by an individual from food, water, and residential uses, through oral, dermal, or inhalation routes.

In addition to the aggregate risk assessment tools, as mandated by FQPA, U.S. EPA has also developed procedures to perform cumulative risk assessments¹⁴, in which groups of pesticides that share a common mechanism of toxicity are evaluated together. This approach combines the estimates of aggregate exposure for individual chemicals with the same toxic effect and generates a cumulative risk assessment. U.S. EPA has conducted assessments for four pesticide groups:

- Organophosphate insecticides
- Carbamate insecticides
- Triazine herbicides
- Chloracetanilide herbicides

In developing aggregate and cumulative risk assessment procedures, U.S. EPA has developed new procedures to assist in the characterization of risk from the use of inert ingredients on food commodities.

For a detailed discussion on the risk assessment process see the 2004 EPRI report no. 1005367 "Human Health Risk Assessment of Chemicals Encountered in Vegetation Management on Electric Utility Rights-of-Way." The methods for assessing human health hazard (including potency) are generic to pesticide risk assessment, while the focus of the exposure assessment in this report was chemical exposure to workers rather than chemical exposure in drinking water.

¹³ <http://www.epa.gov/pesticides/trac/science/aggregate.pdf>

¹⁴ <http://www.epa.gov/pesticides/cumulative/>

Variability and Uncertainty in Risk Assessment

Most risk assessments use quantitative methods to estimate risks, i.e., risks are expressed with numbers; however, the numbers are far from exact. Variability and uncertainty may be dominant factors in any risk assessment, and these factors should be expressed. Within the context of a risk assessment, the terms variability and uncertainty signify different conditions.

Variability reflects knowledge or at least an explicit assumption about how things may change, while uncertainty reflects a lack of knowledge. For example, the focus of the human health dose-response assessment is an estimation of an “acceptable” or “no adverse effect” dose that will not be associated with adverse human health effects. For the herbicides and TGRs evaluated in this report, and for most other chemicals, however, this estimation regarding human health must be based on data from experimental animal studies, which cover only a limited number of effects. Similarly, the potential for human exposure to herbicides in drinking water is estimated from both laboratory and field studies conducted under conditions that may differ significantly from the actual use conditions. Therefore, judgment is often the basis for the methods used to make risk assessments. Although the judgments may reflect a consensus (i.e., be used by many groups in a reasonably consistent manner), the resulting estimations of risk cannot be proven analytically. In other words, the estimates regarding risk involve uncertainty. The primary functional distinction between variability and uncertainty is that variability is expressed quantitatively, while uncertainty is generally expressed qualitatively.

As stated previously, in characterizing the risks of pesticides in drinking water, U.S. EPA considers drinking water consumption patterns across the US to discern regional differences. Differences may result from combination of factors including pesticide use patterns, the contribution and vulnerability of the drinking water source (surface or ground water) and drinking water treatment. However, the outcome is often expressed numerically as a single number that represents a single estimate of the daily exposure, above which, exceeds a level of concern. Sensitive sub-populations may be considered (i.e., children), resulting in more than one level of concern. In the absence of a measure on variability (e.g., central estimate and range) to account for different forms of variability, the number chosen to represent the level of concern should be plausibly conservative.

Identity and Composition of Pesticide Products

Product chemistry testing is required for the registration and re-registration of pesticides under FIFRA¹⁵.

The term *pesticide product* is defined in the Code of Federal Regulations (40 CFR 152) as a pesticide in the particular form (including composition, packaging, and labeling) in which the pesticide is, or is intended to be, distributed or sold. The term includes any physical apparatus used to deliver or apply the pesticide if distributed or sold with the pesticide. All pesticide products can be classified into three categories:

¹⁵ http://www.epa.gov/opptsfrs/publications/OPPTS_Harmonized/830_Product_Properties_Test_Guidelines/Revised/830-1000.pdf

- Technical grade of the active ingredient
- Manufacturing-use products
- End-use products

Technical grade of the active ingredient,” which is used interchangeably with “*technical chemical,*” means a material containing an active ingredient which contains no ingredient, other than one used for manufacture or purification of the active ingredient and which is produced on a commercial or pilot-plant production scale (whether or not it is ever held for sale).

A “*manufacturing-use product*” is any pesticide product other than an end-use product. A manufacturing-use product may consist of the technical grade of the active ingredient only, or may contain inert ingredients, such as stabilizers or solvents. Manufacturing-use products are, as the term implies, used in the production of end-use products, primarily through reformulation, i.e., mixing the manufacturing-use product with different chemical substances such as solvents or diluents.

An “*end-use product*” is defined as a pesticide product whose labeling includes directions for use of the product (as distributed or sold, or after combination by the user with other substances) for controlling pests or defoliating, desiccating or regulating of plants and does not state that the product may be used to manufacture or formulate other pesticide products.

Formulation means the process of mixing, blending, or dilution of one or more active ingredients with one or more other active or inert ingredients, without an intended chemical reaction, to obtain a manufacturing use product or an end-use product. All pesticides are composed of one or more substances. For regulatory purposes the substances are classified either as active ingredients, intentionally-added inert ingredients, or impurities.

Since 1997, U.S. EPA has encouraged manufacturers, formulators, producers, and registrants of pesticide products to voluntarily substitute the term "other ingredients" for "inert ingredients" in the ingredient statement on the label of the pesticide product. Although the term inert is useful in describing ingredients in the formulation that are not the active ingredient(s), concern arose that the term "inert" is misleading to some, believing it to mean "harmless." Since neither federal law nor the regulations define the term "inert" on the basis of toxicity, hazard or risk to humans, non-target species, or the environment, it should not be assumed that all inert ingredients are non-toxic. In addition, pesticide products can contain more than one inert (other) ingredient, but federal law does not require that these ingredients be identified by name or percentage on the label. Only the total percentage of inert ingredients is required to be on the pesticide product label. As pesticide formulation is as much an art as a science, for each pesticide product the recipe developed by formulation chemists to meet all the requirements of storage, handling, application, effectiveness, and safety is considered a trade secret, and is protected by federal statute as "confidential business information". What is public knowledge is the list of U.S. EPA approved substances that can be used in pesticide products¹⁶.

¹⁶ <http://www.epa.gov/opprd001/inerts/lists.html>

The term “*active ingredient*” means any substance (or group of structurally similar substances, if specified by the Agency) that will prevent, destroy, repel or mitigate any pest, or that functions as a plant regulator, desiccant, or defoliant, within the meaning of FIFRA section 2(a).

In short, the active ingredients in pesticides are the substances which directly produce the intended pesticidal effect.

Pesticides contain “*impurities*,” which are defined as any substance (or group of structurally similar substances, if specified by U.S. EPA) in a pesticide product other than an active ingredient or an inert ingredient, including unreacted starting materials, side reaction products, contaminants, and degradation products and pesticide active ingredients other than those intended for that product.

There is increased interest by U.S. EPA regarding impurities in pesticides, particularly impurities associated with an active ingredient. The term “*impurity associated with an active ingredient*” means:

1. Any impurity present in the technical grade of the active ingredient (e.g., a substance carried over from a beginning material, or from an intermediate, and impurities formed through side reactions or by degradation of the active ingredient).
2. Any impurity which forms in a pesticide product through reactions between the active ingredient and any other component of the product or packaging of the product.

“*Inert ingredient*” means any substance (or group of structurally similar substances, if designated by U.S. EPA), other than an active ingredient, which is intentionally included in a pesticide product. Intentionally added inert ingredients include wetting agents, emulsifiers, surfactants, aerosol propellents, diluents, solvents, stabilizers.

Product identity and composition. U.S. EPA product chemistry guidelines for compliance with FIFRA are contained in OPPTS 830.1550 - Product identity and composition. FIFRA requires for each pesticide product a statement of formula identifying each **active ingredient**, each intentionally-added **inert ingredient**, and, in certain cases, **impurities** that may be present in the product while it is being distributed in commerce. As a part of the registration process a comprehensive listing of the ingredients that may be present in a product and the amounts of such ingredients and all of the major types of identifying information on a product and its ingredients is required. The composition information will be used primarily in subsequent evaluations of the safety of the product. The identifying information is used as an aid in locating data in the public literature concerning the human health and environmental properties of the product and/or its ingredients. Identification of an ingredient calls for a variety of information:

For all ingredients, the **chemical name** and **Chemical Abstracts Service (CAS) number** for active and intentionally-added inert ingredients, the purpose or function of the ingredient and for active ingredients, the **molecular, structural, and empirical formula**, the molecular weight (or range), as well as other means of identification.

In addition to identifying the ingredients in the product, the registrant must also provide certified limits for the ingredients listed in the statement of formula. Upper and lower certified limits must be established for each active ingredient and each intentionally-added inert ingredient. In

addition, for some types of products, an upper certified limit must be established for certain impurities. The upper certified limit is the maximum (and the lower certified limit is the minimum) amount of the ingredient that will be present in the product at any time while it is in commerce.

The registrant must also identify the product by the **product name** and **trade name(s)** (if different) and the company code number assigned to the product. The Statement of Formula is required as a means of identifying the ingredients in products. For end-use products, chemical names, and structural formulas and molecular weights for ingredients are given. For technical materials, impurities identified during development are given, as well as percent composition.

In addition to these requirements, pesticide active ingredients are also given a common name. Common names are usually selected by the appropriate professional society (entomology, weed science, plant pathology) and approved by the National Standards Institute and the International Organization of Standardization.

Literature Review

Following a review of the literature as described in “Information Resources” above, the primary sources of information for the active ingredients is as follows:

1. 2,4-D

EPRI Report No. 1005367. 2004. Human Health Risk Assessment of Chemicals Encountered in Vegetation Management on Electric Utility Rights-of-Way. 200 p.

USDA Forest Service. 2006. 2,4-D Human Health and Ecological Risk Assessment Final Report. 245 p.

U.S. Environmental Protection Agency. 2005. Reregistration Eligibility Decision for 2,4-D. 320 p.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/4.htm>), 2,4-D.

2. Aminopyralid

USDA Forest Service. 2007. Aminopyralid Human Health and Ecological Risk Assessment – FINAL REPORT. 231 p.

California Environmental Protection Agency Department of Pesticide Regulation Medical Toxicology Branch. 2005. Summary of Toxicology Data Aminopyralid. 11 p.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/29.htm>), aminopyralid.

3. Bromacil

U.S. Environmental Protection Agency. 1996. Reregistration Eligibility Decision for Bromocil. 320 p.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/88.htm>), bromocil.

4. Clopyralid

USDA Forest Service. 2004. Clopyralid - Human Health and Ecological Risk Assessment - Final Report. 154 p.

California Environmental Protection Agency Department of Pesticide Regulation Medical Toxicology Branch. 2008. Summary of Toxicology Data Clorpyralid. 9 p.

Footprint Pesticide Properties Database
(<http://sitem.herts.ac.uk/aeru/iupac/Reports/169.htm>), clorpyralid.

5. Diruon

U.S. Environmental Protection Agency. 2003. Reregistration Eligibility Decision (RED) for Diuron. 210 p.

Diruon, pesticide tolerance, Federal Register: June 13, 2007 (Volume 72, Number 113)

California Environmental Protection Agency Department of Pesticide Regulation Medical Toxicology Branch. 1997. Summary of Toxicology Data Diruon. 7 p.

Footprint Pesticide Properties Database
(<http://sitem.herts.ac.uk/aeru/iupac/Reports/260.htm>), diruon.

6. Flurprimidol

New York State Department of Environmental Conservation. 2007. Registration of a Major Change in Labeled (MCL) Use Pattern for the Active Ingredient Flurprimidol Contained in the Pesticide Product Topflor Ornamental Plant Growth Regulator (U.S. EPA Reg. No. 67690-20). 7 p.

U.S. Environmental Protection Agency. 2009. Problem Formulation for the Ecological Risk and Drinking Water Exposure Assessments in Support of the Registration Review of Flurprimidol. 26 p.

U.S. Environmental Protection Agency. 2009. Flurprimidol. Human Health Assessment Scoping Document in Support of Registration Review. 25 p.

U.S. Environmental Protection Agency. 1989. Flurprimidol (Cutless) U.S. EPA Pesticide Fact Sheet. 7 p. <http://pmep.cce.cornell.edu/profiles/index.html>

Footprint Pesticide Properties Database
(<http://sitem.herts.ac.uk/aeru/iupac/Reports/348.htm>), flurprimidol.

7. Fosamine Ammonium

EPRI Report No. 1005367. 2004. Human Health Risk Assessment of Chemicals Encountered in Vegetation Management on Electric Utility Rights-of-Way. 200 p.

USDA Forest Service. 1997. Final Environmental Impact Statement. Vegetation Management On Electric Utility Rights-Of-Way. May 1997. Allegheny National Forest. Department of Agriculture, Forest Service. Warren, PA.

U.S. Environmental Protection Agency. 1995. Reregistration Eligibility Decision (RED) Fosamine ammonium. 214 p.

California Environmental Protection Agency Department of Pesticide Regulation Medical Toxicology Branch. 1986. Summary of Toxicology Data Fosamine. 7 p.

Footprint Pesticide Properties Database
(<http://sitem.herts.ac.uk/aeru/iupac/Reports/362.htm>), Fosamine ammonium.

8. Glyphosate

EPRI Report No. 1005367. 2004. Human Health Risk Assessment of Chemicals Encountered in Vegetation Management on Electric Utility Rights-of-Way. 200 p.

USDA Forest Service. 2007. Allegheny National Forest Final Environmental Impact Statement Appendix G1 - Human Health Risk Assessment for Glyphosate and Sulfometuron Methyl. 334 p.

USDA Forest Service. 2003. Glyphosate - Human Health and Ecological Risk Assessment Final Report. 281 p.

U.S. Environmental Protection Agency . 1993. Reregistration Eligibility Decision (RED) Glyphosate. 281 p.

U.S. Environmental Protection Agency. 2006. Glyphosate; Pesticide Tolerance. Federal Register: December 20, 2006 (Volume 71, Number 244)

Footprint Pesticide Properties Database
(<http://sitem.herts.ac.uk/aeru/iupac/Reports/373.htm>), glyphosate.

9. Imazapic

USDA Forest Service. 2003. Imazapic - Human Health and Ecological Risk Assessment – Final Report. 110 p.

California Environmental Protection Agency Department of Pesticide Regulation Medical Toxicology Branch. 2005. Summary of Toxicology Data Imazapic. 7 p.

Footprint Pesticide Properties Database
(<http://sitem.herts.ac.uk/aeru/iupac/Reports/1152.htm>), imazapic.

10. Imazapyr

EPRI Report No. 1005367. 2004. Human Health Risk Assessment of Chemicals Encountered in Vegetation Management on Electric Utility Rights-of-Way. 200 p.

U.S. Environmental Protection Agency. 2006. Reregistration Eligibility Decision for Imazapyr. 107 p.

USDA Forest Service. 2004. Imazapyr - Human Health and Ecological Risk Assessment – Final Report. 149 p.

Footprint Pesticide Properties Database
(<http://sitem.herts.ac.uk/aeru/iupac/Reports/393.htm>), imazapyr.

11. Isoxaben

U.S. Environmental Protection Agency. 2007. Isoxaben Summary Document Registration Review Docket December 2007. www.regulations.gov Docket Number: EPA-HQ-OPP-2007-1038.

California Environmental Protection Agency Department of Pesticide Regulation Medical Toxicology Branch. 1991. Summary of Toxicology Data Ioxaben. 7 p.

Footprint Pesticide Properties Database
(<http://sitem.herts.ac.uk/aeru/iupac/Reports/411.htm>), isoxaben.

12. Metsulfuron methyl

EPRI Report No. 1005367. 2004. Human Health Risk Assessment of Chemicals Encountered in Vegetation Management on Electric Utility Rights-of-Way. 200 p.

USDA Forest Service. 2004. Metsulfuron Methyl - Human Health and Ecological Risk Assessment – Final Report. 152 p.

Footprint Pesticide Properties Database
(<http://sitem.herts.ac.uk/aeru/iupac/Reports/470.htm>), metsulfuron-methyl.

13. Paclobutrazol

U.S. Environmental Protection Agency. 2007. Paclobutrazol Summary Document Registration Review: Initial Docket March 2007 Case Number 7002. www.regulations.gov Docket Number EPA-HQ-EPA-2006-0109. 34 p.

U.S. Environmental Protection Agency. 1985. Paclobutrazol (Clipper 50 WP) Herbicide Profile. 3 p. <http://pmep.cce.cornell.edu/profiles/index.html>

U.S. Department of Energy Bonneville Power Administration. Paclobutrazol Herbicide Fact Sheet. 8 p.

Footprint Pesticide Properties Database
(<http://sitem.herts.ac.uk/aeru/iupac/Reports/333.htm>), paclobutrazol.

14. Pendimethalin

U.S. Environmental Protection Agency. 1997. Reregistration Eligibility Decision (RED) Pendimethalin. 239 p.

California Environmental Protection Agency Department of Pesticide Regulation Medical Toxicology Branch. 1997. Summary of Toxicology Data Pendimethalin. 17 p.

Footprint Pesticide Properties Database
(<http://sitem.herts.ac.uk/aeru/iupac/Reports/511.htm>), pendimethalin.

15. Picloram

EPRI Report No. 1005367. 2004. Human Health Risk Assessment of Chemicals Encountered in Vegetation Management on Electric Utility Rights-of-Way. 200 p.

USDA Forest Service 2003. Picloram - Revised Human Health and Ecological Risk Assessment – Final Report. 133 p.

U.S. Environmental Protection Agency. 1997. Reregistration Eligibility Decision (RED) Picloram. 301 p.

California Environmental Protection Agency Department of Pesticide Regulation Medical Toxicology Branch. 1999. Summary of Toxicology Data Picloram, Potassium Salt. 10 p.

Footprint Pesticide Properties Database
(<http://sitem.herts.ac.uk/aeru/iupac/Reports/525.htm>), picloram.

16. Tebuthiuron

U.S. Environmental Protection Agency. 1994. Reregistration Eligibility Decision (RED) Tebuthiuron. 232 p

California Environmental Protection Agency Department of Pesticide Regulation Medical Toxicology Branch. 2000. Summary of Toxicology Data Tebuthiuron. 9 p.

Footprint Pesticide Properties Database
(<http://sitem.herts.ac.uk/aeru/iupac/Reports/614.htm>), tebuthiuron.

17. Triclopyr

EPRI Report No. 1005367. 2004. Human Health Risk Assessment of Chemicals Encountered in Vegetation Management on Electric Utility Rights-of-Way. 200 p.

USDA Forest Service 2003. Triclopyr - Revised Human Health and Ecological Risk Assessment – Final Report. 133 p.

U.S. Environmental Protection Agency. 1994. Reregistration Eligibility Decision (RED) Triclopyr. 285 p.

California Environmental Protection Agency Department of Pesticide Regulation Medical Toxicology Branch. 2000. Summary of Toxicology Data Triclopyr. 12 p.

Footprint Pesticide Properties Database
(<http://sitem.herts.ac.uk/aeru/iupac/Reports/614.htm>), triclopyr.

18. Trifluralin

U.S. Environmental Protection Agency. 1996. Reregistration Eligibility Decision (RED) Trifluralin. 285 p.

California Environmental Protection Agency Department of Pesticide Regulation Medical Toxicology Branch. 1995. Summary of Toxicology Data Trifluralin. 17 p.

Footprint Pesticide Properties Database
(<http://sitem.herts.ac.uk/aeru/iupac/Reports/667.htm>), trifluralin.

2

ELECTRIC UTILITY VEGETATION MANAGEMENT PROGRAM

Sustainable and economically viable right-of-way vegetation management requires good planning, which incorporates stated goals and objectives into a rational, comprehensive and practical program. The planning process should recognize the environmental requirements of desirable plants, as well as the potential adverse impacts of altering the landscape. Consequently, mitigation of human and environmental adverse impacts often drives vegetation management decisions. The decision to use chemical control measures must balance mitigation of herbicide adverse impacts with sustainability and economic viability. A properly planned and executed vegetation management program will use a variety of vegetation control techniques and strategies in an integrated fashion, dictated by economics, terrain, vegetation type, and public relations.

Typically, electric utility rights-of-way integrated vegetation management (IVM) plans rely primarily of two types of control measures: mechanical and chemical.

Mechanical methods can be divided into two categories, depending on the dimensions of measurement: treated acreage and linear side trim acreage. Treated acreage is represented by two methods: mowed acres and hand-treated acres. The second type of mechanical control is a linear method known as a side trim. Side trim methods can be split into three categories: side trim with a bucket truck, side trim manual, and side trim with a helicopter.

Common chemical control methods for vegetation management along transmission rights-of-way are basal, cut surface, high-volume foliar application with a hand gun (HVF), low-volume foliar application with a backpack or hand sprayer (LVF), and low-volume foliar broadcast (LVFB) such as fixed boom and/or radiarc. The two general types of herbicide application techniques are selective and nonselective. Selective techniques, such as basal and low-volume foliar methods, target only the undesirable vegetation, leaving behind herbaceous and low-growing woody plants to establish shrub-dominated communities resistant to tree seedling invasion (Niering and Goodwin 1974; Bramble et al., 1991; personal communication, J. Goodrich-Mahoney, September 21, 2009). Cut-surface techniques can be either selective or nonselective, depending on the situation, because a mechanical cutting of brush is required. Nonselective techniques, such as high-volume foliar, are used to re-establish control of the right-of-way vegetation. These broad-sweeping techniques, used in areas containing over 2,000 stems per acre, generally kill all vegetation within a given right-of-way and create a "clean slate" where desirable vegetation can become established. In most cases, a period of intensive maintenance following nonselective techniques is required to achieve the desired results. If the maintenance is not performed, the ROW will revert to its original problem state.

In addition, Tree growth regulators (TGRs) can be used to slow the growth of trees, allowing more effective management of pruning cycles. TGRs have been shown to reduce the amount of biomass removed from trees and, in doing so, reduce the amount of time needed to prune and clip treated trees.

To assess vegetation management practices along rights-of-way, in 1995 a two-part study was developed in the Michigan State University Department of Forestry - one part focusing on transmission rights-of-way and the second focusing on distribution rights-of-way (Sulak and Kielbaso, 2000). The study was initiated following recommendations from the Vegetation Management Task Force in the Environmental Stewardship Strategy for Electric Utility Rights-of-Way. The primary purpose was to describe current vegetation control techniques used by utility companies, with a secondary objective of quantifying the active ingredient per acre applied for the herbicides reported.

The study used a survey sent to 220 Utility Arborist Association companies. The survey contained questions regarding right-of-way characteristics, control methods used, total dollars spent on vegetation management, and priorities of the vegetation management program. The ROW area reported represented over 48% of all the investor-owned ROWs over 39 Kv in service throughout the United States. More than 75% of the respondents reported using herbicides on their rights-of-way. However, acres treated mechanically outnumbered those treated chemically by a margin of 2.7:1. The most common herbicides reported in this survey were Garlon (triclopyr), Accord (glyphosate), Arsenal (imazapyr), and Krenite S (fosamine ammonium). Garlon 3A and Garlon 4 topped all herbicides, with a combined 220,574 projected gal (834,961 L) of the estimated 549,869 gal (2,081,474 L) of herbicide applied to transmission rights-of-way in 1995. The respondents reported the frequent use of application reduced rates as compared that recommended. Basal, high-volume foliar, and low-volume foliar with a backpack or handgun applications accounted for approximately 75% of the acres of transmission ROWs treated with herbicides. Table 2-1 compares the common application methods (percent of total reported), and the herbicides commonly applied for the individual application methods.

While herbicides were often the preferred control measure – 75% of those responding reported using herbicides on transmission ROW acres that required vegetation control – exclusive use of mechanical control measures on some acreage was also common.

Recently, four electric utility companies in New York reported using basal, cut surface, low volume foliar, low volume hydraulic foliar, and high-volume foliar treatment methods on 4, 35, 31, 17, and 13 percent of electric transmission right-of-way in 2008, respectively, (across a total of >13,000 acres) (personal communication, D. Morrell, New York State Public Service Commission, September 18, 2009).

In addition, U.S. EPA reports that 2,4-D and glyphosate are the most common herbicides used by industry (application including, but not exclusive to the electric industry) in the United States (2000-2001 Pesticide Market Estimates for the United States.¹⁷

¹⁷ www.epa.gov/oppbead1/pestsales/usage2001_3.htm, accessed 9/18/2009

Table 2-1
Herbicide Treatments Commonly Used on Electric Transmission Rights-of-way in North America

Application method*	Percent use in North America	Herbicides	
		Common names	Trade names
Basal	28	imazapyr; triclopyr	Garlon 4; Pathfinder II; Stalker
High-volume foliar	24	fosamine; glyphosate; imazapyr; metsulfuron methyl; picloram; triclopyr	Accord; Arsenal; Escort XP; Garlon 3A; Krenite S; Tordon K
Low-volume foliar with backpack or hand sprayer	23	fosamine; glyphosate; imazapyr; metsulfuron methyl	Accord; Arsenal; Escort XP; Krenite
Cut surface	19	2,4-D; imazapyr; glyphosate; picloram; triclopyr	Accord C; Arsenal; Garlon 4; Pathway RTU; Stalker
Aerial	4	fosamine; glyphosate; imazapyr; metsulfuron methyl	Accord; Arsenal; Escort XP; Krenite
Low-volume Broadcast	2	fosamine; glyphosate; imazapyr; metsulfuron methyl; picloram; triclopyr	Accord; Arsenal; Escort XP; Garlon 3A; Krenite S; Tordon K

* Results of 1995 survey (Sulak and Kielbaso, 2000).

3

HERBICIDE AND TREE GROWTH REGULATOR PROFILES

Introduction

Presented here are profiles for the active ingredients in the products listed in Table 1-2. These profiles summarize the information on active ingredient hazard, potency, and chemical characteristics used to evaluate environmental fate. In addition, when available, information on other ingredients in the product will be provided.

The profiles are organized as follows:

1. General use in electric utility rights-of-way vegetation management programs
2. Hazard assessment – how is the chemical toxic to humans
3. Dose-response assessment – what is the potency of the most toxic effect in humans
4. Chemical characteristics that affect fate in the environment and human exposure

The hazard and dose-response assessments will focus on the most sensitive toxic effect as determined from the animal studies, as it is this adverse effect that is used to establish the acute NOAEL, LOAEL, or BMDL, the chronic NOAEL, LOAEL, or BMDL, and the cancer potency is expressed as Q_1^* . Safety factors are applied to these risk numbers to determine the RfD. The RfD can be used to establish a level of concern for pesticides in drinking water. The derivation of pesticide drinking water standards, benchmarks, and guidelines, and their use in characterizing human health risk will be covered in detail in Chapter 5.

The Chapter on chemical characteristics will be used to evaluate fate in the environment that may lead to human exposure that exceeds a level of concern. For the purposes of this report environmental fate that may lead to ground water contamination will be emphasized. Based on the information provided in Chapter 4, the chemical characteristics for each herbicide or TGR will be used to evaluate the potential for the chemical to leach to ground water.

In addition, in assessing exposure to pesticides in drinking water, information that may be used to assess the cumulative risk of aggregate exposure will be included in the profile.

The profiles will also identify data gaps necessary to the evaluation of toxicity and exposure, as many of the products given in Table 1-2 were first registered many years ago, when required testing was less stringent. However, pesticide registration in the U.S. is a dynamic process, as new science and information on the impacts of current use becomes available a pesticide product's registration status may be changed. This is accomplished through the U.S. EPA

pesticide reregistration process. Periodic re-evaluation of pesticide registrations and tolerances to ensure that the scientific data supporting pesticide registrations will remain up to date in the future. The reregistration program (administered by U.S. EPA's Office of Pesticide Programs), is the critical mechanism used by U.S. EPA to implement tolerance reassessment. Whereby, reregistering food use pesticides means not only that U.S. EPA reassesses their tolerances, but also that U.S. EPA evaluates the safety of those pesticides for workers and the environment. The reregistration process involves evaluating all risks to human health and the environment based on current science and risk assessment procedures, as well as data on adverse effects resulting from product use¹⁸. The results are published in a Reregistration Eligibility Decision (RED)¹⁹, which describes the risk assessment process leading to a decision whether or not to reregister products containing a pesticide active ingredient. If the decision is to reregister, often there will be restrictions on how the pesticide can be used in order to mitigate risk. These restrictions are communicated to the user on the pesticide label. For example, if the concern is for worker exposure then the label may stipulate re-entry intervals, protective clothing requirements, or engineering controls (i.e., closed cab, closed mixing-loading systems). If the concern is food residues, then application rates and frequency, and a pre-harvest interval may be stipulated. If the concern is water quality, restrictions may include buffer zones²⁰. Mitigation measures under FIFRA usually apply nationally (with some exceptions). However, the consequences of each pesticide use is site-specific; ultimately, a pesticide's risk to human health and the environment is determined by the user.

When available, the profiles will use information derived from the RED. If no RED is available then the additional information resources given the Literature Review will be used. The information from these sources will be discussed without reference. The data on chemical characteristics were obtained from the Footprint Pesticide Properties Database.

2,4-D

2,4-D is an herbicide in the phenoxy or phenoxyacetic acid family that is used post-emergence for selective control of broadleaf weeds. 2,4-D is registered for use on a variety of food/feed sites including field, fruit, and vegetable crops. 2,4-D is also registered for use on turf, lawns, rights-of-way, aquatic sites, forestry applications, and is used as a plant growth regulator in citrus. Residents and professional applicators may use 2,4-D on home lawns. The electric utility right-of-way product Pathway[®] is a ready to use (RTU) liquid used in cut surface applications. Pathway contains both 2,4-D amine and picloram. Upon contact with water 2,4-D amine salt dissociates to the acid.

¹⁸ In addition to new information in the open literature, Section 6(a)(2) of FIFRA requires pesticide product registrants to submit adverse effects information about their products to the U.S. EPA

(<http://www.epa.gov/opppmsd1/fifra6a2/>).

¹⁹ <http://www.epa.gov/pesticides/regulating/registering>

²⁰ The RED may also require additional studies by the registrant in order to complete the risk assessment. This may result in a conditional registration and a timetable for completion of the required studies. A list of the status of pesticide REDs can be found at <http://www.epa.gov/pesticides/reregistration/status.htm>.

In acute studies, 2,4-D generally has low acute toxicity (Toxicity Category III or IV) via the oral, dermal and inhalation routes of exposure. 2,4-D is not a skin irritant (Toxicity Category III or IV), nor a skin sensitizer. Although the 2,4-D ester forms are not eye irritants (Toxicity Category III or IV), the acid and salt forms are considered to be severe eye irritants (Toxicity Category I).

In longer-term studies, at dose levels above the threshold of saturation for renal clearance, 2,4-D is toxic to the eye, thyroid, kidney, adrenals, and ovaries/testes. Rat lowest observed adverse effect levels (LOAELs) are based on gait abnormalities in a neurotoxicity study, skeletal abnormalities in pups in a developmental study, and decreased weight gain in a chronic toxicity study. Dogs show a LOAEL based on decreased body weight gain and decreased food consumption, and rabbits show a LOAEL based on ataxia, decreased motor activity, and abortions.

In 2005, U.S. EPA required the registrants to submit toxicity data, including a developmental neurotoxicity study in rat; subchronic (28-day) inhalation study, and repeat the two-generation reproduction study in rat using a more recent protocol to address concerns for endocrine disruption.

In addition, U.S. EPA has required that the registrants confirm that dioxin levels are within acceptable limits; all technical products should be analyzed for 2,3,7,8-TCDD, 2,3,7,8-TCDF, and their respective higher substituted chlorinated congeners using validated analytical methods.

U.S. EPA has established a 2,4-D acute RfD (Females 13-49 years of age; child bearing age) of 0.025 mg/kg/day based on the NOAEL of 25 mg/kg/day in the Rat Developmental Toxicity Study (LOAEL was 75 mg/kg/day based on skeletal abnormalities) and an uncertainty factor of 1000, 10x for interspecies variability, 10x for intraspecies variability, and 10x for uncertainty in the dietary exposure database.

The Acute Dietary (General population including infants and children) RfD is 0.067 mg/kg/day based on the NOAEL of 67 mg/kg/day in the Acute Neurotoxicity Study in Rats (LOAEL was 227 mg/kg/day based on gait abnormalities) and an uncertainty factor of 1000.

The chronic dietary RfD (all populations) is 0.005 mg/kg/day based on a NOAEL of 5 mg/kg/day in the Rat Chronic Toxicity Study (LOAEL was 75 mg/kg/day based on decreased body-weight gain (females) and food consumption (females), alterations in hematology, and clinical chemistry parameters, decreased T4 (both sexes), glucose (females), cholesterol (both sexes), and triglycerides (females)) and an uncertainty factor of 1000.

2,4-D has been classified as a Category D chemical, i.e., not classifiable as to human carcinogenicity, by the U.S. EPA Cancer Peer Review Committee in 1996.

U.S. EPA is required under the Federal Food, Drug, and Cosmetic Act (FDCA), as amended by the Food Quality Protection Act (FQPA), to develop a screening program to determine whether certain substances (including all pesticide active and other ingredients) "may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen, or other such endocrine effects as the Administrator may designate." When the appropriate screening and/or testing protocols being considered under the Agency's Endocrine Disruption Screening Program

(EDSP) have been developed, 2,4-D may be subject to additional screening and/or testing to better characterize effects related to endocrine disruption.

2,4-D acid chemical characteristics useful in evaluating environmental fate and human exposure are given below. A discussion of chemical characteristics and ratings is given in Chapter 4.

Table 3-1
2,4-D acid chemical characteristics

Chemical characteristic	Value (rating)
Solubility in water at 20 °C (mg/l)	23180 (high)
Octanol-water partition coefficient at pH 7, 20 °C	1.48×10^{-01} (low)
Vapor pressure at 25 °C (mPa)	0.0187 (volatile)
Henry's law constant at 20 °C (dimensionless)	1.40×10^{-09} (non-volatile)
Soil degradation $\frac{1}{2}$ life (days) (aerobic)	10 (non-persistent)
Koc - Organic-carbon sorption constant (ml/g)	56 (mobile)
GUS leaching potential index	2.25 (moderate)

Aminopyralid

The product Milestone® contains the triisopropanolamine (TIPA) salt of aminopyralid (40.6 % w a.i./v, equivalent to 21.1 % a.e. or 2 lbs a.e./gal). This formulation contains no inert ingredients other than water and triisopropanolamine.

Aminopyralid uses include forest and rangelands, and rights-of-way. Aminopyralid is typically applied by backpack (selective foliar) and high-volume foliar. The labeled application rates for aminopyralid are 0.03 to 0.11 lb a.i./acre for the control of rhizomatous weeds. For non-rhizomatous weeds, the application rate will generally be about 0.078 lb a.i./acre.

Because aminopyralid is a relatively new herbicide, no information is available in the published literature on the toxicity of aminopyralid to humans or other mammalian species.

U.S. EPA classifies Aminopyralid as toxicity category II (caution).

Aminopyralid is rapidly absorbed and excreted and is not substantially metabolized in mammals. The oral LD50 of aminopyralid has not been determined because aminopyralid does not cause any mortality at the dose limits set by the U.S. EPA for acute oral toxicity studies – i.e., up to 5,000 mg/kg bw.

Subchronic and chronic toxicity studies have failed to demonstrate any clear signs of systemic toxic effects.

Developmental studies involving gavage administration, however, have noted signs of incoordination in adult female rabbits. The incoordination was rapidly reversible and did not persist past the day of dosing.

Two chronic oral bioassays have been conducted; one in mice and the other in rats. A 1-year feeding study was conducted in dogs. Based on the results of the chronic bioassays as well as the lack of mutagenic activity in several mutagenicity screening assays, there is no basis for asserting that aminopyralid is a carcinogen.

Based on the chronic bioassays and several additional subchronic bioassays in mice, rats, dogs, and rabbits, there is no basis for asserting that aminopyralid will cause adverse effects on the immune system or endocrine function. The potential for effects on the nervous system is less clear.

Aminopyralid has also been subject to several bioassays for developmental toxicity and one multi-generation study for reproductive performance. No adverse effects on offspring have been noted in these studies other than decreased body weight in offspring that is associated with decreased food consumption and decreased body weight in adult females.

The chronic RfD for aminopyralid is 0.5 mg/kg/day. This RfD is based on a chronic rat NOAEL of 50 mg/kg/day and an uncertainty factor of 100. The acute RfD of 1 mg/kg/day is based on a NOAEL from a reproduction study of about 100 mg/kg/day. In deriving both of these RfD values, U.S. EPA used an uncertainty factor of 100, a factor of 10 for extrapolating from animals to humans and a factor of 10 for extrapolating to sensitive individuals within the human population. Both of these RfD values are based on NOAELs for the most sensitive endpoint in the most sensitive species and studies in which LOAEL values were identified. In addition, both of the NOAEL values are supported by other studies.

Aminopyralid chemical characteristics useful in evaluating environmental fate and human exposure are given below. A discussion of chemical characteristics and ratings is given in Chapter 4.

Table 3-2
Aminopyralid chemical characteristics

Chemical characteristic	Value (rating)
Solubility in water at 20 °C (mg/l)	2480 (high)
Octanol-water partition coefficient at pH 7, 20 °C	1.35×10^{-03} (low)
Vapor pressure at 25 °C (mPa)	2.59×10^{-09} (non-volatile)
Henry's law constant at 20 °C (dimensionless)	8.88×10^{-17} (non-volatile)
Soil degradation $\frac{1}{2}$ life (days) (aerobic)	35 (moderately persistent)
Koc - Organic-carbon sorption constant (ml/g)	8 (very mobile)
GUS leaching potential index	4.78 (high)

Bromacil

Bromacil is a broad spectrum herbicide used to control weeds in the agricultural food crops citrus and pineapple. In addition, both bromacil and its lithium salt are used to control weeds and brush in nonagricultural areas including utility right-of-ways, railroads, electrical switching stations, and industrial yards. Kroval[®] DF, a dispersible granular used in electric utility right-of-way applications, contains diuron and bromocil. Kroval DF can be applied as either high-volume or low-volume foliar.

In studies using laboratory animals, bromacil is slightly toxic by the oral, dermal, and inhalation routes and has been placed in Toxicity Category IV (the lowest of four categories) for these effects. Bromacil is mildly irritating to the eyes (Toxicity Category III). The lithium salt of bromacil, however, is moderately irritating to the eyes (Toxicity Category II).

In a chronic feeding study using beagle dogs, bromacil caused decreased body weight gain. In another chronic study using rats, effects in addition to reduced body weight gain include (1) increased incidence of thyroid cysts in the high dose males; (2) enlargement of the thymus in high dose females; and (3) dose-related incidence of thyroid tumors in the males.

Bromacil has been evaluated for potential carcinogenic activity in rats and mice. Bromacil is classified as a Group C possible human carcinogen based on increases in incidence of liver tumors in male mice, and positive trends in thyroid tumors in male rats, and, to a lesser extent, structural activity relationship to similar compounds.

Bromacil demonstrates some evidence of causing developmental toxicity effects in rats and rabbits. These effects are likely due to maternal toxicity from exposure to bromacil rather than from specific developmental toxicity of bromacil. U.S. EPA does not consider bromacil a developmental toxicant.

The chronic RfD for bromacil is 0.1 mg/kg/day, based on the NOAEL of 9.82 mg/kg/day in the chronic rat toxicity study. An uncertainty factor of 100 was used to account for the inter-species extrapolation and intra-species variability.

The FQPA requires U.S. EPA to apply an additional 10-fold uncertainty (safety) factor unless reliable data demonstrate that the additional factor is unnecessary to protect infants and children.

In determining the need for an additional safety factor U.S. EPA must consider available information on the aggregate exposures to the pesticide from dietary sources including drinking water as well as non-occupational exposures such as those derived from pesticides used in and around the home. U.S. EPA must also consider the potential cumulative effects of the pesticides or other substances that have a common mechanism of toxicity. Because bromacil has food uses, specific consideration of the risks to infants and children, as well as aggregate exposures and potential cumulative effects is warranted.

In determining whether a safety factor different than the additional 10-fold is or is not appropriate for assessing risks to infants and children, U.S. EPA evaluated two developmental and one reproduction study. Based on the evaluation of these studies U.S. EPA concluded that an additional uncertainty factor is not warranted for the bromacil acute or chronic risk assessments.

Bromocil chemical characteristics useful in evaluating environmental fate and human exposure are given below. A discussion of chemical characteristics and ratings is given in Chapter 4.

Table 3-3
Bromocil chemical characteristics

Chemical characteristic	Value (rating)
Solubility in water at 20 °C (mg/l)	815 (high)
Octanol-water partition coefficient at pH 7, 20 °C	7.59×10^{01} (low)
Vapor pressure at 25 °C (mPa)	4.10×10^{-02} (volatile)
Henry's law constant at 20 °C (dimensionless)	5.39×10^{-09} (non-volatile)
Soil degradation $\frac{1}{2}$ life (days) (aerobic)	60 (moderately persistent)
Koc - Organic-carbon sorption constant (ml/g)	32 (mobile)
GUS leaching potential index	4.44 (high)

Clopyralid

Clopyralid is a selective herbicide used primarily in the control of broadleaf weeds. Uses include forestry and rights-of-way. The commercial product commonly used on electric utility rights-of-way is Transline[®], a liquid formulation of clopyralid containing 40.9% clopyralid as the monoethanolamine salt and 59.1% inert ingredients. The identity of the inerts in Transline is proprietary with the exception of isopropyl alcohol and a polyglycol. Technical grade clopyralid contains hexachlorobenzene and pentachlorobenzene as contaminants. Nominal or average concentrations of hexachlorobenzene are less than 2.5 ppm. Nominal or average concentrations of pentachlorobenzene are less than 0.3 ppm. The most common Transline application methods are low-volume backpack (selective foliar) and high-volume boom spray (broadcast foliar).

U.S. EPA classifies Clopyralid as Toxicity Category II (caution).

The acute RfD for clopyralid is 0.75 mg/kg/day and a chronic RfD of 0.15 mg/kg/day. The acute

RfD is based on a short-term NOAEL of 75 mg/kg/day and an uncertainty factor of 100. The chronic RfD is based on a 2-year dietary NOAEL in rats of 15 mg/kg/day and an uncertainty factor of 100. Other studies in rats, mice, and dogs have noted general decreases in body weight, increases in liver and kidney weight, as well as a thickening in some epithelial tissue. Decreases in body weight and changes in organ weight are commonly observed in chronic toxicity studies and can indicate either an adaptive or toxic response. Changes in epithelial tissue are less commonly observed and the toxicologic significance of this effect is unclear.

The contamination of technical grade clopyralid with hexachlorobenzene and pentachlorobenzene can be quantitatively considered to a limited extent. The U.S. EPA has derived RfDs for both pentachlorobenzene and hexachlorobenzene and a cancer potency factor for hexachlorobenzene. Based on the levels of contamination of technical grade clopyralid with these compounds and the relative potencies of these compounds to clopyralid, this contamination is not significant in terms of potential systemic-toxic effects.

Even though hexachlorobenzene has shown carcinogenic activity in three mammalian species and has been classified as a possible human carcinogen, technical grade clopyralid (containing hexachlorobenzene at about 2.5 ppm or less) has been subject to several chronic bioassays for carcinogenicity and none of the bioassays have shown that clopyralid has carcinogenic potential.

In considering of chemicals in the Transline formulation, both monoethanolamine and isopropyl alcohol are approved food additives, and there is no evidence to assert that these compounds will materially impact the risks associated with the use of clopyralid. The other inert in Transline is Polyglycol 26-2. This compound is classified by the U.S. EPA (2003) as a List 3 inert. In other words, there is insufficient information to categorize this compound as either hazardous (Lists 1 or 2) or non-toxic (List 4).

Clopyralid chemical characteristics useful in evaluating environmental fate and human exposure are given below. A discussion of chemical characteristics and ratings is given in Chapter 4.

Table 3-4
Clopyralid chemical characteristics

Chemical characteristic	Value (rating)
Solubility in water at 20 °C (mg/l)	143000 (high)
Octanol-water partition coefficient at pH 7, 20 °C	2.34×10^{-03} (low)
Vapor pressure at 25 °C (mPa)	1.36 (volatile)
Henry's law constant at 20 °C (dimensionless)	1.46×10^{-08} (non-volatile)
Soil degradation $\frac{1}{2}$ life (days) (aerobic)	34 (moderately persistent)
Koc - Organic-carbon sorption constant (ml/g)	5 (very mobile)
GUS leaching potential index	5.06 (high)

Diuron

Diuron is a substituted urea herbicide for the pre-emergence control of a wide variety of annual grass and broadleaf weeds and some perennial broad leaved and grassy weeds on both crop and non-crop sites. The mechanism of herbicidal action is the inhibition of photosynthesis. Products containing diuron are intended for both occupational and residential uses. Occupational uses include agricultural food and non-food crops; ornamental trees, flowers, and shrubs; paints and coatings; ornamental fish ponds, and catfish production; rights-of-way and industrial sites. Residential uses include ponds, aquariums, and paints.

The diuron products Karmex[®] XP and Krovar[®] DF (which also contains bromocil) are dispersible granule formulations commonly used for electric utility right-of-way applications. These products are applied by both low-volume (backpack) and high-volume ground sprayers. For non-agricultural uses, diuron labeled rates range from 0.8 lbs to 12 lbs ai/acre.

Diuron has low acute toxicity (Toxicity Category III-IV) by the oral, dermal, or inhalation exposure routes. Diuron is not an eye or skin irritant, and not a skin sensitizer. The primary target organs are the hematopoietic system, the bladder, and renal pelvis. Erythrocyte damage resulted in hemolytic anemia and compensatory hematopoiesis, which were manifested as significantly decreased erythrocyte counts, hemoglobin levels, and hematocrit, and increased mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), abnormal erythrocyte forms, reticulocyte counts, and leukocyte count. Consistent observations of erythrocytic regeneration were seen in chronic toxicity studies in rats, mice and dogs. Gross pathology findings in chronic rat and mouse studies showed increased incidences of urinary bladder edema and wall thickening at high doses. Microscopic evaluation showed dose-related increases in the severity of epithelial focal hyperplasia of the urinary bladder and renal pelvis in both sexes. Diuron does not exhibit developmental or reproductive toxicity.

There are no adverse effects attributed to a single exposure identified in any available studies for diuron. In addition, diuron has low acute toxicity and no developmental or neurotoxic concerns. Therefore, no acute dietary endpoint was chosen and no acute dietary risk assessment was conducted. Also, no systemic toxicity was observed following repeated dermal dosing up to 1,200 mg/kg/day. Therefore, U.S. EPA has not established short- or intermediate-term dermal endpoints.

The short-term incidental oral and the inhalation endpoints are based on decreased maternal body weight and food consumption observed in a rabbit developmental toxicity study, NOAEL = 10 mg/kg/day. The intermediate-term incidental oral and intermediate-term inhalation endpoints are based on hematological effects observed at 10 mg/kg at 6 months in the chronic rat study. The NOAEL is 1 mg/kg/day. The chronic dietary, and long-term dermal and inhalation endpoints are based on hemolytic anemia and compensatory hematopoiesis, LOAEL = 1.0 mg/kg/day. Since the dose and endpoint for establishing the chronic dietary reference dose (RfD) is a LOAEL and a NOAEL was not established, a total uncertainty factor (UF) of 1,000 was applied (a UF of 100 to account for both interspecies extrapolation and intra-species variability and an UF of 10 since the 10X FQPA safety factor has been retained to protect infants and children).

Diuron is characterized as a "known/likely" human carcinogen based on urinary bladder carcinomas in both sexes of the Wistar rat, kidney carcinomas in the male rat, and mammary gland carcinomas in the female NMRI mouse. Diuron was not mutagenic in bacteria or in cultured mammalian cells and no indication of DNA damage in primary rat hepatocytes was observed. There were marginal statistically significant increases in cells with structural aberrations in a Sprague Dawley rat *in vivo* bone marrow chromosomal aberration assay. However, the levels of aberrations were within historical control range and assessed negative.

The Q_1^* for diuron is 1.91×10^{-2} (mg/kg/day)⁻¹, based on based on the urinary bladder carcinomas in the rat. In addition, a separate dietary cancer assessment was conducted for N'-(3-chlorophenyl)-N,N-dimethyl urea (MCPDMU), a potential residue of concern in drinking water,

but not found in food. The Q_1^* for MCPDMU is $1.52 \times 10^{-2} \text{ (mg/kg/day)}^{-1}$, based on male rat liver neoplastic nodule and/or carcinoma combined tumor rates.

Diuron chemical characteristics useful in evaluating environmental fate and human exposure are given below. A discussion of chemical characteristics and ratings is given in Chapter 4.

Table 3-5
Diuron chemical characteristics

Chemical characteristic	Value (rating)
Solubility in water at 20 0C (mg/l)	35.6 (low)
Octanol-water partition coefficient at pH 7, 20 0C	7.41×1002 (moderate)
Vapor pressure at 25 0C (mPa)	1.15×10^{-03} (volatile)
Henry's law constant at 20 0C (dimensionless)	2.06×10^{-08} (non-volatile)
Soil degradation $\frac{1}{2}$ life (days) (aerobic)	75.5 (moderately persistent)
Koc - Organic-carbon sorption constant (ml/g)	1067 (slightly mobile)
GUS leaching potential index	1.83 (moderate)

Fosamine Ammonium

Fosamine ammonium is an herbicide/plant growth regulator used to control brush and herbaceous plants on non-cropland. It is applied to nonagricultural rights-of-way (e.g., highways, railroads, and utilities), industrial sites, and fencerows.

Fosamine ammonium is formulated in end use products, such as Krenite® S, as a water soluble liquid. For electric utility right-of-way use, fosamine ammonium is applied once per year from spring to early fall as a low-volume foliar application (backpack). Use practice limitations prohibit fosamine ammonium from being used on croplands or in irrigation systems. It may not be applied directly to water or areas where surface water is present, including intertidal areas.

Fosamine ammonium is classified as Toxicity Category II by acute dermal studies in mammalian species. Fosamine ammonium is very mildly toxic for acute oral and acute inhalation (Toxicity Category IV), and is not a dermal sensitizer.

Because fosamine ammonium bears a chemical resemblance to organophosphate insecticides, it has been assayed for neurotoxicity. Oral administration of single doses up to 2000 mg/kg to rats in an acute neurotoxicity study caused diarrhea at 1000 mg/kg and “possible” neural effects (irregular respiration and slight closure of the eyelids) at 1000 and 2000 mg/kg. The NOAEL was 500 mg/kg. Fosamine ammonium does not produce delayed neurotoxicity, or inhibition of neurotoxic esterase or acetylcholine esterase after 21-day intoxication of hens.

A developmental toxicity assay in rats caused diarrhea, erratic weight gain and depressed feed consumption at the highest dose and marginal effects at 1000 mg/kg/day. At 3000 mg/kg/day, pregnancy rate, miscarriages, resorptions, litter size and fetal death all were not different from

control, even though the animals were affected. The NOAEL for maternal toxicity was 350 mg/kg/day; the fetal NOAEL was in excess of 3000 mg/kg/day.

Fosamine ammonium was not mutagenic in the Ames test, both with and without metabolic Activation. A forward mutation assay with Krenite formulation in Chinese hamster ovary cells (CHO) was negative with and without activation. An *in vivo* bone marrow assay for chromosomal aberrations in rats given single oral doses of Krenite was negative. An *in vitro* test for chromosome damage in CHO cells was positive; dose related increases in aberrations were seen in both activated and non-activated preparations. The finding triggered a requirement for a follow-up *in vivo* study with germ cells. The *in vivo* spermatid micronucleus assay carried out in response was negative.

Fosamine ammonium did not induce unscheduled DNA synthesis, although U.S. EPA considers the assay unacceptable because the maximum concentration of 0.88 mg/ml did not meet the requirements for a maximum tolerated dose (MTD). The U.S. EPA has not received chronic oral toxicity or carcinogenicity studies for fosamine ammonium. The anticipated data requirement for a developmental toxicity study will inform the U.S. EPA about the possible chronic toxic effects of fosamine ammonium. Further, because fosamine ammonium has no food or feed uses, dietary exposure and risk were not estimated in the 1994 human health risk assessment. Dietary analysis (from drinking water) has not been previously performed; however, a new drinking water exposure and risk assessment is anticipated for registration review.

A 90 day feeding study produced equivocal renal tubular effects in rats, with a NOAEL of 10 mg/kg/day. This study is the basis for the Reference Dose (RfD) of 0.01 mg/kg/day. There are no food crop uses for fosamine ammonium, however, the RfD can be used to assess the risk to humans from drinking water. An uncertainty factor of 1000 has been applied to the NOAEL of 10 mg/kg/day to accommodate certain data deficiencies, particularly the absence of the lifetime studies (2 year rodent study) that would have accompanied cancer assays.

Fosamine Ammonium chemical characteristics useful in evaluating environmental fate and human exposure are given below. A discussion of chemical characteristics and ratings is given in Chapter 4.

Table 3-6
Fosamine Ammonium chemical characteristics

Chemical characteristic	Value (rating)
Solubility in water at 20 °C (mg/l)	2500000 (high)
Octanol-water partition coefficient at pH 7, 20 °C	1.26×10^{-03} (low)
Vapor pressure at 25 °C (mPa)	0.53 (volatile)
Henry's law constant at 20 °C (dimensionless)	1.33×10^{-11} (non-volatile)
Soil degradation $\frac{1}{2}$ life (days) (aerobic)	8 (non-persistent)
Koc - Organic-carbon sorption constant (ml/g)	63 (mobile)
GUS leaching potential index	1.99 (moderate)

Flurprimadol

Flurprimidol is a turf grass and woody-plant growth regulator. Use sites for flurprimidol include golf courses, general turf, forest trees, various ornamentals, non-agricultural rights-of-way, greenhouses and shadehouses. Formulations for flurprimidol include water-soluble packets, liquid concentrate, granule, and soluble concentrate. The commercial product Cutlass® (water soluble powder) is commonly used in electric utility right-of-way applications. Cutlass is applied by several methods, including backpack sprayers and soil injection.

Flurprimidol is of low to moderate acute toxicity by all exposure routes. U.S. EPA classifies Flurprimidol as Toxicity Category III (slightly toxic) for acute dermal toxicity, acute inhalation toxicity and eye irritation potential. Flurprimidol is not a dermal sensitizer.

A 1-year dog study showed adrenal changes including decreased plasma cortisol response to adrenal cortice-tropine hormone (ACTH) stimulation (males), decreased relative and absolute adrenal weight (males) and degenerative changes of the adrenal cortex (males and females). This study satisfies the requirement for a chronic oral study in one species. The NOAEL was 1.5 mg/kg/day based on degenerative changes of the adrenal gland and decrease in adrenal weights.

Decreased body weight and food consumption were observed in the rabbit and rat teratology studies. In addition, the rat teratology study showed increased mortality, stained perigenital area and snout, chromodacryorrhea, decreased muscle tone, hypoactivity and alopecia.

A subchronic oral rat study showed an increased hepatic enzyme induction in males (significant and dose increases in p-nitroanisol o-demethylase activity).

A subchronic oral mouse study indicated an increased incidence of hepatocellular hypertrophy in the males.

A 21-day dermal toxicity study (rabbit) noted slight transient dermal irritation.

A 2-generation rat reproduction study showed parental systemic toxicity as an increased incidence of non-neoplastic hepatocellular alteration including fatty change and vacuolation (males) and increased susceptibility to stress factors. Decreased mating, fertility, fetal survival (stillbirths), neonatal survival and neonatal body weight in both sexes and in both generations were observed at the reproductive NOAEL of 1.8 mg/kg/day. Other parental signs of toxicity included increased susceptibility to stress (pregnant females) resulting in death, increased relative liver weight (males and females), depressed body weight, weight gain and food consumption (males and females).

Subchronic, oncogenicity and teratogenicity studies are not usually required for non-food use registrations. However, due to the structural similarity of flurprimidol to compounds of toxicological concern (fenarimol, triarimol, and nuarimol), these studies were required for registration.

Carcinogenicity studies were requested based on flurprimidol's similarity to an carcinogenic compound (triarimol) and teratogenicity studies were requested based on its similarity to compounds (triarimol and fenarimol) associated with developmental concerns.

Mutagenicity tests for gene mutation, chromosomal aberration and direct DNA damage were evaluated. Flurprimidol had no effect on induction of unscheduled DNA synthesis or chromosomal aberration. It was also negative for mutagenic activity.

Chronic feeding studies were conducted in both the rat and mouse. Hepatocellular changes in the males including enzyme induction, fatty change, hepatocellular eosinophilic change and focal atypia were observed in the rat study. An additional mouse study showed increased absolute and relative liver weight in females.

No evidence of carcinogenicity was observed at any dose level in either the rat or mouse studies.

U.S. EPA has established a RfD of 0.02 mg/kg/day for flurprimidol based on NOAEL of 1.8 mg/kg/day in a rat multigeneration reproduction study (liver and neuromuscular effects) and an uncertainty factor of 100.

Flurprimidol is classified as "not likely" to be a human carcinogen based on carcinogenicity studies in rats. This classification is supported by the lack of mutagenic activity.

Flurprimidol chemical characteristics useful in evaluating environmental fate and human exposure are given below. A discussion of chemical characteristics and ratings is given in Chapter 4.

Table 3-7
Flurprimidol chemical characteristics

Chemical characteristic	Value (rating)
Solubility in water at 20 °C (mg/l)	114 (moderate)
Octanol-water partition coefficient at pH 7, 20 °C	2.19×10^{03} (high)
Vapor pressure at 25 °C (mPa)	0.1 (volatile)
Henry's law constant at 20 °C (dimensionless)	1.12×10^{-07} (non-volatile)
Soil degradation $\frac{1}{2}$ life (days) (aerobic)	11 (non-persistent)
Koc - Organic-carbon sorption constant (ml/g)	185 (moderately mobile)
GUS leaching potential index	1.80 (moderate)

Glyphosate

Glyphosate is a non-selective herbicide registered for use on many food and non-food field crops as well as non-crop areas where total vegetation control is desired. When applied at lower rates, glyphosate also is a plant growth regulator. Glyphosate is among the most widely used pesticides by volume. For electric utility rights-of-way, the most commonly used products are Accord® and Aqua Neat®.

Glyphosate formulations are commonly applied as backpack-applied directed foliar sprays, or broadcast foliar ground applications by boom or hydraulic spray equipment. It is also possible to apply glyphosate in a cut-stem application. Application rates range from 1.4 to 6 kg a.e./ha (1.3 to 5.4 lb a.e./acre). A non-ionic surfactant is usually required for optimal efficacy. Most formulations of glyphosate that do not contain a surfactant indicate that a nonionic surfactant should be added to the spray mixture.

Glyphosate isopropylamine and sodium salts show relatively low oral and dermal acute toxicity. The surfactant commonly used in current glyphosate formulations is neutralized ethoxylated tallow amine. Most glyphosate formulations are toxicity category III. Glyphosate is considered non-volatile and inhalation studies with end-use products show low toxicity.

Glyphosate contains small amounts of N-nitrosoglyphosate (NNG) as an impurity from the manufacturing process. Some nitrosamines are carcinogenic; NNG has shown weak, if any activity. Levels of NNG in glyphosate are below one part per million. U.S. EPA considers these levels to be not toxicologically significant and therefore do not warrant carcinogenicity testing.

Aminomethyl phosphonic acid (AMPA) is a product of microbial metabolism of glyphosate, predominantly in soil. U.S. EPA has concluded that AMPA is not of toxicologic concern.

Glyphosate is not considered acutely toxic. In a 90-day sub-chronic rat study, based on the reduced body weight gains in both sexes, U.S. EPA has established a systemic toxicity NOEL= 500 mg/kg and a LOEL = 2500 mg/kg.

Based on a 2 year rat feeding study, U.S. EPA has set the chronic systemic toxicity NOEL at 362 mg/kg/day (male) and 457 mg/kg/day (female). The corresponding LOEL is 940 mg/kg/day (male) and 1183 mg/kg/day (female). Treatment-related effects observed only in the high-dose group included, female – decreased body weight gains; male – increased incidence of cataracts and lens abnormalities, decreased urinary pH, increased absolute liver weight and increased liver weight/brain weight ratio (relative liver weight). No significant systemic effects were observed in the low-dose and mid-dose male and female groups.

Acute and subchronic neurotoxicity studies in rats, as well as delayed neurotoxicity in hens, show glyphosate is not neurotoxic.

One reproductive toxicity study using rats showed kidney effects in the high dose male pups; another study showed digestive effects and decreased body weight gain.

The FQPA requires U.S. EPA to apply an additional 10-fold uncertainty (safety) factor unless reliable data demonstrate that the additional factor is unnecessary to protect infants and children.

In determining the need for an additional safety factor, U.S. EPA must consider available information on the aggregate exposures to the pesticide from dietary sources including drinking water as well as non-occupational exposures such as those derived from pesticides used in and around the home. U.S. EPA must also consider the potential cumulative effects of the pesticides or other substances that have a common mechanism of toxicity. Because glyphosate has food uses, specific consideration of the risks to infants and children, as well as aggregate exposures and potential cumulative effects is warranted.

U.S. EPA found no evidence of quantitative or qualitative increased susceptibility following *in utero* glyphosate exposure to rats and rabbits, or following prenatal/postnatal exposure in the 2-generation reproduction study in rats. A developmental neurotoxicity study was not required. U.S. EPA has determined that reliable data show that no additional FQPA safety factor, beyond the 100-fold uncertainty (safety) factor, is required to protect infants and children as a sensitive sub-population.

Glyphosate does not cause mutations.

In developmental toxicity studies using pregnant rats and rabbits, glyphosate caused treatment-related effects in the high dose groups including diarrhea, decreased body weight gain, nasal discharge and death. The rabbit developmental toxicity study LOAEL = 350 mg/kg/day, and the NOAEL = 175 mg/kg/day. The NOAEL and a UF = 100 were used to determine the chronic RFD = 1.75 mg/kg/day.

Several chronic toxicity/carcinogenicity studies using rats, mice and beagle dogs resulted in no effects based on the parameters examined, or resulted in findings that glyphosate was not carcinogenic in the study. U.S. EPA has classified glyphosate as a Group E carcinogen. Under the new cancer guidelines glyphosate is classified as a not likely human carcinogen.

Glyphosate chemical characteristics useful in evaluating environmental fate and human exposure are given below. A discussion of chemical characteristics and ratings is given in Chapter 4.

Table 3-8
Glyphosate chemical characteristics

Chemical characteristic	Value (rating)
Solubility in water at 20 °C (mg/l)	10500 (high)
Octanol-water partition coefficient at pH 7, 20 °C	6.31×10^{-04} (low)
Vapor pressure at 25 °C (mPa)	0.0131 (volatile)
Henry's law constant at 20 °C (dimensionless)	6.60×10^{-19} (non-volatile)
Soil degradation $\frac{1}{2}$ life (days) (aerobic)	12 (non-persistent)
Koc - Organic-carbon sorption constant (ml/g)	21699 (non-mobile)
GUS leaching potential index	-0.36 (low)

Imazapic

Imazapic is used in the control of grasses, broadleaves, and vines, and for turf height suppression in non-cropland areas. The commercial product Journey[®] (imazapic 0.75 lb a.e./gal + glyphosate 1.5 lb a.e./gal) commonly is used in electric utility right-of-way applications. For postemergence applications, a spray adjuvant may be used to aid in penetration. Imazapic may be applied by low-volume or high-volume foliar.

Imazapic is classified as toxicity category IV, very low toxicity.

Imazapic does not appear to be toxic to experimental rodents at relatively high concentrations in the diet but is toxic to dogs, causing adverse effects on muscle, blood, and liver. The NOAEL in rats is about 1625 mg/kg/day in the 13-week study or 1133 mg/kg/day in the 2-year study. Dogs, however, appear to be more sensitive than rodents, and the major signs of toxicity include adverse effects on the muscle, blood, and liver. Chronic exposure to imazapic at doses as low as 150 mg/kg/day have been associated with treatment-related effects on skeletal muscle.

U.S. EPA has established a chronic RfD of 0.5 mg/kg/day for imazapic, based on a chronic LOAEL in dogs of 137 mg/kg/day and an uncertainty factor of 300. The dog LOAEL is based on adverse effects on skeletal muscle.

In the test battery required for pesticide registration, imazapic has failed to show any indication of adverse effects on development or reproduction and no carcinogenic or mutagenic activity.

Imazapic chemical characteristics useful in evaluating environmental fate and human exposure are given below. A discussion of chemical characteristics and ratings is given in Chapter 4.

Table 3-9
Imazapic chemical characteristics

Chemical characteristic	Value (rating)
Solubility in water at 20 °C (mg/l)	2230 (high)
Octanol-water partition coefficient at pH 7, 20 °C	2.95×10^{02} (low)
Vapor pressure at 25 °C (mPa)	0.01 (volatile)
Henry's law constant at 20 °C (dimensionless)	5.07×10^{-10} (non-volatile)
Soil degradation $\frac{1}{2}$ life (days) (aerobic)	120 (persistent)
Koc - Organic-carbon sorption constant (ml/g)	137 (moderately mobile)
GUS leaching potential index	3.87 (high)

Imazapyr

Imazapyr is used for pre- and post-emergence control of a broad range of weeds, including terrestrial annual and perennial grasses, broad-leaved herbs, woody species, and riparian and emergent aquatic species. Agricultural uses of imazapyr include field corn and grass.

Imazapyr is also registered for use on a variety of commercial and residential use sites, including forestry sites, rights-of-way, fence rows, hedge rows, drainage systems, outdoor industrial areas, outdoor buildings and structures, domestic dwellings, paved areas, driveways, patios, parking areas, walkways, various water bodies (including ponds, lakes, streams, swamps, wetlands, stagnant water, and urban areas). Imazapyr may also be used as a spot treatment in recreation areas, athletic fields, and golf course roughs.

Commercial products used on electric utility right-of-way include Arsenal® (liquid or granule) and Chopper® (ready to use – RTU). Both products contain imazapyr as the propylamine salt.

While imazapyr formulations can be used in pre-emergence applications, the most common and effective applications are post-emergent when the vegetation to be controlled is growing vigorously. The most common methods of ground application for Arsenal formulations involve low-volume foliar (backpack) and high-volume foliar. Cut surface treatment methods may also be used with Chopper RTU.

Imazapyr has low acute toxicity via the oral (Toxicity Category IV) and dermal (Toxicity Category III) routes of exposure. Imazapyr has been placed in acute Toxicity Category II for the inhalation route of exposure. It is not irritating to the skin, and is negative for dermal sensitization; however, imazapyr results in irreversible eye damage (Toxicity Category I).

Most of the toxicity studies required for pesticide registration showed no effects to minimal effects, even at the highest dose tested.

There is no evidence of acute or chronic neurotoxicity resulting from exposure to imazapyr.

No developmental toxicity was observed in rabbits or rats up to the highest dose tested; however, maternal toxicity, based on salivation, was observed in rats at the mid-dose (300 mg/kg/day). Neither the rat nor the rabbit study showed an increased susceptibility of the fetus to imazapyr administered prenatally or post-natally. In addition, a 2-generation reproduction rat study did not show increased susceptibility to offspring. There were no compound-related effects in a one-year dietary toxicity study in beagle dogs.

A 1-year dog feeding study with a NOAEL of 250 mg/kg/day was selected for calculating the chronic RfD because it was the lowest NOAEL in the imazapyr database. The 250 mg/kg/day dose in the dog study was both the NOAEL and the highest dose tested for that study. Because there were no adverse effects seen in the dog study or in any of the imazapyr toxicity studies, U.S. EPA relied on a structural analog imazapic to choose an endpoint. Imazapic causes skeletal muscle effects in dogs at 137 mg/kg/day in males and 180 mg/kg/day in females. Despite imazapyr's structural similarity to imazapic, as well as its similarity to the pesticides,

imazethapyr and imazamethabenz-methyl, the available data do not support the conclusion that these pesticides share a common mechanism of toxicity such that combined exposure to them would result in cumulative effects. This finding is supported by toxicity data for imazapyr that show no adverse effects, including no skeletal muscle effects. Second, the toxic endpoints for the three structurally similar pesticides are quite varied: imazapic (skeletal muscle effects); imazethapyr (an increased incidence of clinical signs during gestation, ulcerations in the mucosal layer of the stomach and gall bladder, increased abortions, maternal deaths, decrements in body weight gain) and imazamethabenz-methyl (transient decreased body weight, mild liver effects, slight increase in a common kidney lesion).

The imazapyr chronic RfD of 2.5 mg/kg/day was derived from the 1-year dog feeding study NOAEL of 250 mg/kg/day and a UF of 100X (10X for interspecies extrapolation and 10X for intraspecies variation). Additional uncertainty or safety factors may also be applied. A FQPA Safety Factor has not been applied as there are no residual exposure uncertainties, no increased sensitivity to infants and children, and the toxicity database is essentially complete.

Imazapyr was classified by U.S. EPA in 1995 as “Group E”; no evidence of carcinogenicity in at least 2 adequate studies in the rat and mouse. Imazapyr is negative for mutagenic potential and a quantitative cancer risk assessment is not required.

Imazapyr chemical characteristics useful in evaluating environmental fate and human exposure are given below. A discussion of chemical characteristics and ratings is given in Chapter 4.

Table 3-10
Imazapyr chemical characteristics

Chemical characteristic	Value (rating)
Solubility in water at 20 °C (mg/l)	9740 (high)
Octanol-water partition coefficient at pH 7, 20 °C	1.29×10^{00} (low)
Vapor pressure at 25 °C (mPa)	0.013 (volatile)
Henry's law constant at 20 °C (dimensionless)	1.43×10^{-10} (non-volatile)
Soil degradation $\frac{1}{2}$ life (days) (aerobic)	11 (non-persistent)
Koc - Organic-carbon sorption constant (ml/g)	125 (moderately mobile)
GUS leaching potential index	1.98 (moderate)

Isoxaben

Isoxaben is a preemergent herbicide mainly used to control broadleaf weed species. Uses include Christmas trees, conifer trees, ornamentals (container grown, field grown, and landscape), ornamental bulbs, non-bearing fruit and nut trees, non-bearing vineyards, rights-of-way, and non-commercially grown turf.

Isoxaben is formulated as a granular, a water dispersible granule (also referred to as a dry flowable), and an impregnated material. The commercial product Snapshot® (a granular formulation containing trifluralin and isoxaben) is commonly used in electric utility right-of-way applications. Isoxaben may be applied by low-volume or high-volume foliar.

Isoxaben has low acute toxicity via the oral and dermal routes (category III and IV), and is slightly more toxic via the inhalation route (category II and III). It is not an acute eye or skin irritant and is not a dermal sensitizer.

Acute and chronic animal studies required for pesticide registration show the major target organ for isoxaben is the liver in rats, dogs and mice. In rats, liver effects include increased enzymes, and organ weight with concurrent liver hypertrophy. In dogs, increased liver weight and enzyme induction were noted. In the chronic mouse study, liver adenomas, hyperplasia, increased liver weight, hepatocytomegaly and hepatocellular vacuolation were observed at high doses following chronic exposure.

In the rat developmental study, fetal toxicity manifested as increased pre-implantation loss, increased resorptions, smaller litter size and increased numbers of runt fetuses and occurred in the presence of maternal toxicity (decreased body weight) at the limit dose of 1000 mg/kg.

There was no significant maternal or fetal toxicity in rabbits at the limit dose of 1000 mg/kg. In a three generation rat reproductive study, developmental toxicity (decreases in viable fetuses/litter, increased hydroureter, and microphthalmia) occurred only in the presence of maternal toxicity (decreased body weight/body weight gain, increased liver/body weight ratios).

Isoxaben toxicology data were last reviewed by U.S. EPA in 1987 and 1989. A reregistration eligibility decision is scheduled for 2013. The current RfD for isoxaben is 0.5 mg/kg/day, based on a NOAEL of 50 mg/kg/day and a UF of 100. The NOAEL was derived from the combined chronic toxicity and carcinogenicity study in rats which show increased blood urea nitrogen (BUN), decreased alkaline phosphatase and aspartate aminotransferase (AST), decreased food efficiency and increased heart/body weight in males.

U.S. EPA has classified isoxaben as a group C (possible human carcinogen), without a quantitative risk assessment based on a statistically significant increase in benign tumors in one species only (mouse). The tumors in the mouse (liver adenomas) were present in both sexes, however, tumor incidence was statistically increased at the high dose only, the tumors were of a common type, were predominantly benign, and there was no decrease in latency. The incidence was above historical controls, but a dose-response relationship was not found. The weight-of-evidence suggests there is not an overt mutagenicity concern for isoxaben. U.S. EPA has

estimated an oral cancer potency value $Q1^* = 2.3 \times 10^{-3} \text{ (mg/kg/day)}^{-1}$. Isoxaben currently has no established tolerances on food or animal commodities. Therefore isoxaben is currently considered to be a non-food use pesticide. Consequently, a dietary assessment has not been conducted and drinking water risks have not been assessed.

Isoxaben chemical characteristics useful in evaluating environmental fate and human exposure are given below. A discussion of chemical characteristics and ratings is given in Chapter 4.

Table 3-11
Isoxaben chemical characteristics

Chemical characteristic	Value (rating)
Solubility in water at 20 °C (mg/l)	0.93 (low)
Octanol-water partition coefficient at pH 7, 20 °C	8.71×10^{03} (high)
Vapor pressure at 25 °C (mPa)	5.50×10^{-04} (volatile)
Henry's law constant at 20 °C (dimensionless)	7.10×10^{-06} (moderately volatile)
Soil degradation $\frac{1}{2}$ life (days) (aerobic)	105 (persistent)
Koc - Organic-carbon sorption constant (ml/g)	601 (slightly mobile)
GUS leaching potential index	2.47 (moderate)

Metsulfuron-methyl

Metsulfuron methyl is a selective pre-emergence and post-emergence sulfonyl urea herbicide used primarily to control many annual and perennial weeds and woody plants. The commercial product containing metsulfuron methyl commonly used in electric utility rights-of-way is Escort® XP. This dry flowable granule formulation is 60% metsulfuron methyl and 40% inert ingredients. The most common methods of ground application for Escort XP involve low-volume foliar (backpack) and high-volume foliar operations.

Acute and chronic animal studies show metsulfuron to have low toxicity. The oral acute median lethal dose (LD50) in the rat is greater than 5000 mg/kg. Signs of toxicity after single oral doses of 500 mg/kg or greater are non-specific; they include lethargy, weight loss, and sensitivity to touch. Acute dermal toxicity is also low; the LD50 in the rabbit is greater than 2000 mg/kg. Higher doses were not practical to apply. Concentrated formulation (70%) was moderately irritating in rabbits. A repeat insult closed patch dermal sensitization assay in guinea pigs produced no dermal irritation, and no delayed hypersensitivity or allergic response at doses high enough to cause diarrhea.

Metsulfuron methyl is moderately to severely irritating to the eyes, if not washed. Effects that were seen were reversible, with no corneal injury.

In a 21-day subacute dermal study of rabbits, methsulfuron methyl administered up to 2000 mg/kg/day produced no effect on body or organ weight, hematology, blood chemistry or organ pathology. However, at the site of application there was reddening, thickening and grossly

evident dermatitis at 2000 mg/kg/day, and microscopically visible dermatitis at 500 mg/kg/day. The NOAEL was 125 mg/kg/day.

In a 90-day rat feeding study the metsulfuron methyl NOAEL for systemic toxicity 50 mg/kg/day. A 90-day dog feeding study was without effect at 125 mg/kg/day, the highest dose tested. However, a one-year feeding study of dogs produced an apparent decrease in serum lactate dehydrogenase at 12.5 mg/kg/day, with no effect at 1.25 mg/kg/day. This is a curious response because a change due to cellular injury should be an increase, occurring early in a course of treatment, and no change was seen in dogs at doses at least ten fold higher over 90 days. The finding has been disregarded by U.S. EPA in setting the NOAEL and reference dose.

Metsulfuron methyl is not a reproductive or developmental toxicant (up to the pregnant female maximum tolerated dose). A two-generation rat reproduction study at dose rates up to 250 mg/kg/day resulted in no reproductive or fetal toxicity at any dose. There was evidence of maternal toxicity, however, in the form of lower body weight gains at the highest dose rate.

The maternal NOAEL was 25 mg/kg/day.

Teratogenicity (birth defects) assays were negative in rats at doses up to 1000 mg/kg/day through the period of greatest sensitivity. In rabbits the NOAEL for teratogenicity and fetal toxicity was greater than 700 mg/kg/day, but for maternal toxicity the NOAEL was 25 mg/kg/day.

Following a weight-of-the-evidence evaluation of a battery of mutagenicity assays, U.S. EPA has determined that metsulfuron methyl is not mutagenic. Ames tests were negative; *in vitro* Chinese hamster ovary cell gene mutation assay, with and without activation, was negative; an assay of unscheduled DNA synthesis in primary rat liver cell culture was negative in two trials; an *in vivo* rat bone marrow cytogenetic assay was negative in both males and females; and a mouse bone marrow micronucleus assay was negative.

As a part of the battery of test for pesticide registration, 2-year chronic toxicity studies were conducted in rats and mice. The NOAEL for rats was 25 mg/kg/day, based on weight loss at a ten-fold higher dose. This study is the basis for the RfD of 0.3 mg/kg/day. (25mg/kg/day divided by an uncertainty factor of 100 and rounded to one significant digit.) The uncertainty factor includes a ten-fold allowance for species differences and ten-fold for differences within species. The systemic NOAEL for mice was greater than 750 mg/kg/day, which caused no evidence of toxicity.

The 18 month mouse and 24 month rat feeding studies showed no carcinogenic response.

Using the current cancer risk guidelines, in 2002 U.S. EPA described metsulfuron methyl as not likely to be carcinogenic. This finding supersedes the 1998 finding that metsulfuron methyl is a classified as group E. In 1998, U.S. EPA concluded that: “the weight-of-evidence indicates that metsulfuron methyl is neither genotoxic nor mutagenic and that “Metsulfuron methyl was not oncogenic in the chronic rat and mouse bioassays.”

Metsulfuron methyl chemical characteristics useful in evaluating environmental fate and human exposure are given below. A discussion of chemical characteristics and ratings is given in Chapter 4.

Table 3-12
Metsulfuron methyl chemical characteristics

Chemical characteristic	Value (rating)
Solubility in water at 20 °C (mg/l)	2790 (high)
Octanol-water partition coefficient at pH 7, 20 °C	2.00×10^{-02} (low)
Vapor pressure at 25 °C (mPa)	1.10×10^{-07} (non-volatile)
Henry's law constant at 20 °C (dimensionless)	6.17×10^{-15} (non-volatile)
Soil degradation $\frac{1}{2}$ life (days) (aerobic)	10 (non-persistent)
Koc - Organic-carbon sorption constant (ml/g)	39.5 (mobile)
GUS leaching potential index	2.40 (moderate)

Paclobutrazol

Paclobutrazol is a plant growth regulator that slows vegetative growth by inhibiting gibberilin biosynthesis creating more compact plants. Paclobutrazol can be used on ornamental plants (flowers, seedlings, etc.) grown in containers in nurseries, greenhouses, shade houses and interior landscapes. It can act as a nonselective, post-emergent herbicide for control of annual grasses and broadleaf weeds. It is used to reduce lawn mowing and to increase turf density.

Paclobutrazol is used on turf (e.g., residential, commercial, ornamental, and golf course applications). Paclobutrazol can be used as a tree injection, soil incorporation, and basal drench to reduce above ground vegetative growth (reduces terminal growth and pruning volume) of deciduous trees and pine trees for power line and utility rights of way. The commercial product commonly used in electric utility right-of-way applications is Profile[®] 2SC.

Paclobutrazol has no food uses. U.S. EPA has not performed a human health risk assessment for paclobutrazol. However, increased use on turf has increased interest in evaluating human health risk associated with exposure in drinking water.

In 1994 U.S. EPA convened an RfD Peer Review Committee to evaluate the existing toxicology data on Paclobutrazol. The report included the following information:

In a 21-Day Dermal- Rabbit Irritation the NOAEL was 10 mg/kg/day. The LOAEL was 100 mg/kg/day based on irritation appearing the second week; dose related effects included hyperkeratosis, acanthosis, and inflammation of superficial dermal.

In a 2-generation reproduction rat study the NOAEL was 2.5 mg/kg/day. The LOAEL was 12.5 mg/kg/day, based on increased liver weights and fatty changes in parental females; increases incidence of chromodacryorrhea and thickened eyelids, dental malocclusion, liver mottling or

accentuation of lobular structure, liver enlargement, pallor and discoloration in male and female pups.

A chronic RfD has not been established due to the lack of food uses with paclobutrazol.

U.S. EPA classifies Paclobutrazol as Class D, Not classifiable due to inadequate human and animal evidence. U.S. EPA may require new carcinogenicity studies if the current use pattern changes.

Paclobutrazol chemical characteristics useful in evaluating environmental fate and human exposure are given below. A discussion of chemical characteristics and ratings is given in Chapter 4.

Table 3-13
Paclobutrazol chemical characteristics

Chemical characteristic	Value (rating)
Solubility in water at 20 °C (mg/l)	0.248 (low)
Octanol-water partition coefficient at pH 7, 20 °C	1.29×10^{03} (high)
Vapor pressure at 25 °C (mPa)	0.0019 (volatile)
Henry's law constant at 20 °C (dimensionless)	9.24×10^{-07} (moderately volatile)
Soil degradation $\frac{1}{2}$ life (days) (aerobic)	112 (persistent)
Koc - Organic-carbon sorption constant (ml/g)	210 (moderately mobile)
GUS leaching potential index	3.44 (high)

Pendimethalin

Pendimethalin is a dinitroaniline herbicide used to control broadleaf weeds and grassy weed species in a number of crop and noncrop areas and on residential lawns and ornamentals. Formulations include liquid, solid, granular, emulsifiable concentrate, dry flowables, and microencapsulated. The commercial product Pendulum Aqua Cap® (a microencapsulated formulation containing 38.7% pendimethalin) is commonly used in electric utility rights-of-way for pre-emergent weed control. The most common methods of ground application for Pendulum Aqua Cap involve low-volume foliar (backpack) and high-volume foliar operations.

Pendimethalin is slightly acutely toxic by the oral and eye routes and has been placed in Toxicity Category III for these effects. It is practically non-toxic by the dermal and inhalation routes and has been placed in Toxicity Category IV.

As pendimethalin has low acute toxicity by the oral route ($LD_{50} = 1250$ mg/kg for male rats and $LD_{50} = 1050$ mg/kg for female rats), sub-chronic and chronic toxicity tests were used to establish the RfD. In addition, the results of the initial test battery for pesticide registration resulted in special testing requirements.

In a 30-day rat feeding study the LOAEL was 320 mg/kg/day based on increased liver weight, and the NOAEL was 160 mg/kg/day. In a 13-week feeding study in rats the LOAEL was 500 mg/kg/day based on decreased body weight gain and food consumption, decreased hematocrit and hemoglobin with an increase in platelets in males, red thyroids, increased liver weight, and hypertrophy of the liver. The NOAEL was 50 mg/kg/day. In a second 13-week feeding study in rats the LOAEL was not determined and the NOAEL was greater than 250 mg/kg/day.

In a special 92-day thyroid function rat feeding study at 100 ppm of pendimethalin in the diet there was decreased total T_4 , rT_3 , total free T_4 and increased percent T_3 , increased follicular cell height and decreased area occupied by colloid. At 5,000 ppm there were decreased body weight and food consumption compared to controls, increased thyroid weight, decreased total T_4 , total T_3 , rT_3 , total free T_4 and [125I]- T_4 to transthyretin bonding, increased percent free T_4 , percent free T_3 and [125I]- T_4 to albumin binding, increased follicular cell height and decreased area occupied by colloid and ultrastructural thyroid changes. Most parameters were reversible after treatment subsided except for decreased body weight. The NOAEL was established at 100 ppm (4.98 mg/kg/day). The LOAEL was determined to be 5000 ppm (249 mg/kg/day). In a special 56-day rat feeding study the LOAEL was 500 ppm (31 mg/kg/day) based on thyroid effects. The NOAEL could not be determined. In a special 14-day rat feeding study to determine thyroid function the LOAEL was 500 mg/kg/day based on thyroid effects. The NOAEL was 10 mg/kg/day. In a second special 14-day rat feeding study to determine biliary excretion and hepatic metabolism the LOAEL was 500 mg/kg/day based on thyroid effects. The NOAEL was 10 mg/kg/day.

In a 90-day dog feeding study the LOAEL is 250 mg/kg/day based on body weight loss. The NOAEL is 62.5 mg/kg/day.

In a 30-day feeding study in dogs, the LOEL was 125 mg/kg/day based on decreases in body weight and food consumption. The NOEL could not be determined.

In a 30-day feeding study in mice the LOAEL was not determined. The NOAEL was greater than 300 mg/kg/day.

In a 2-year feeding study in rats the LOAEL was 250 mg/kg/day based on decreased survival, body weight gain and food consumption, increased gamma glutamyl transferase and cholesterol, increase in absolute and/or relative liver weight, generalized icterus, dark adipose tissue in females, diffusely dark thyroids and follicular cell hyperplasia of the thyroid. The NOAEL was 25 mg/kg/day. There were thyroid follicular cell adenomas at 250 mg/kg/day.

In a 2-generation reproduction study in rats, the parental NOAEL could not be definitely determined. There were decreased pup weights during much of lactation at 5000 ppm pendimethalin in the diet. The LOAEL for reproductive effects is 5000 ppm (346 and 436 mg/kg/day in males and females, respectively). The NOAEL for reproductive effects is 2500 ppm (172 and 216 mg/kg/day, in males and females, respectively).

In a 3-generation reproduction study in rats the LOAEL for parental toxicity was 5000 ppm pendimethalin in the diet (250 mg/kg/day) based on decreased body weights. The NOAEL for parental toxicity was 500 ppm (25 mg/kg/day). Pup body weight gain was decreased during lactation. There were possible decreases in pups born alive and pup survival. The LOAEL for

reproductive toxicity was 5000 ppm (250 mg/kg/day) based on pup body weight gain and possible decreased pups born alive and pup survival. The NOAEL for reproductive toxicity was 500 ppm (25 mg/kg/day).

In a reverse gene mutation assay in bacteria the positive controls did induce the appropriate responses in the corresponding strains. This study was considered positive since at all doses tested there was evidence of a 2-fold dose-related increase in the number of induced mutant colonies over background. In a *Salmonella*/microsome plate incorporation assay and in an *Escherichia coli* reverse mutation assay there was no evidence of a mutagenic response (i.e., an increase number of mutant colonies over solvent control values) at any concentration of pendimethalin tested. In a forward mutation study locus in Chinese hamster ovary cells, there was no evidence of a mutagenic response (induced mutant colonies over background) at any concentration of pendimethalin tested. In a chromosomal aberration study in Chinese hamster ovary cells there was no induction of chromosomal aberrations at any concentration of pendimethalin tested. In a mouse micronucleus study, there was no evidence of a mutagenic response. In an alkaline elution assay in rats, there was no evidence of DNA single strand break induction or DNA/DNA or DNA/protein crosslink formation at any concentration of pendimethalin tested. A weight-of-the-evidence evaluation suggests that pendimethalin is not mutagenic.

Three rat studies were considered in choosing the NOAEL to be used in determining the RfD; the subchronic oral 92-day thyroid function study, the subchronic oral 56-day thyroid function study, and the 14-day intrathyroidal metabolism study.

The RfD for pendimethalin is 0.10 mg/kg/day based on the assumption that the 14-day NOAEL of 10 mg/kg/day accurately reflects a true NOAEL for thyroid effects since these effects have been demonstrated to have an early onset (before 14 days). In addition, U.S. EPA considered all three studies together and established the LOAEL at 31 mg/kg/day and the NOAEL at 10 mg/kg/day. An Uncertainty Factor of 100 was applied to account for interspecies extrapolation and intraspecies variability.

Because the effects of pendimethalin on thyroid function are considered endocrine disruption, further testing will be required in the future when endocrine disruption testing guidelines are implemented by U.S. EPA.

U.S. EPA classifies pendimethalin as a Group C (possible human) carcinogen, "based on statistically significant increased trend and pairwise comparison between the high dose group and controls for thyroid follicular cell adenomas in male and female rats." Based on language in the 1997 reregistration eligibility decision, U.S. EPA conditionally considers pendamethalin to be a threshold carcinogen and that the 0.10 mg/kg/day RfD is protective of both the chronic, non-carcinogenic effects as well as the carcinogenic effects.

Pendimethalin chemical characteristics useful in evaluating environmental fate and human exposure are given below. A discussion of chemical characteristics and ratings is given in Chapter 4.

Table 3-14
Pendimethalin chemical characteristics

Chemical characteristic	Value (rating)
Solubility in water at 20 °C (mg/l)	0.33 (low)
Octanol-water partition coefficient at pH 7, 20 °C	1.58×10^{05} (high)
Vapor pressure at 25 °C (mPa)	1.94 (volatile)
Henry's law constant at 20 °C (dimensionless)	1.50×10^{-03} (volatile)
Soil degradation $\frac{1}{2}$ life (days) (aerobic)	90 (moderately persistent)
Koc - Organic-carbon sorption constant (ml/g)	15744 (non-mobile)
GUS leaching potential index	-0.39 (low leachability)

Picloram

Picloram is a systemic herbicide used to control deeply rooted herbaceous weeds and woody plants in rights-of-way, forestry, rangelands, pastures, and small grain crops.

The commercial products Tordon[®] (a liquid formulation containing 24.4% picloram) and Pathway[®] (an RTU liquid containing 2,4-D and picloram) are commonly used in electric utility rights-of-way for pre-emergent weed control. The other ingredients in Tordon include polyglycol, which is included on the U.S. EPA List 3 for other ingredients that may be used in pesticide formulations. Very little additional information is available on these other ingredients. Pathway is used in cut surface applications, and Tordon is commonly applied as low-volume foliar (backpack) and high-volume foliar.

In evaluating the toxicity of picloram, U.S. EPA considered picloram acid and its three derivatives, triisopropanolamine picloram (TIPA-salt), isooctyl/ethylhexyl picloram (IOE), and potassium picloram (K-salt), collectively as "picloram". Tordon and Pathway contain picloram as the potassium (K) salt.

Hexachlorobenzene (HCB) is a contaminant in technical grade picloram. The average concentration of HCB in technical grade picloram is 8 ppm and the maximum concentration is 50 ppm. HCB is ubiquitous and persistent in the environment. The major sources of general exposure for the public to hexachlorobenzene involve industrial emissions, proximity to hazardous waste sites, and the consumption of contaminated food. Based on the amount of hexachlorobenzene in picloram and the amount of picloram used in electrical utility rights-of-way, this use of picloram will not substantially contribute to any wide-spread increase of ambient levels of HCB, and levels are not a threat to ground water.

Picloram and its derivatives are only slightly toxic by the oral and dermal routes and have been placed in Toxicity Categories III and IV (the lowest of four categories) for these effects. However, picloram acid is highly toxic and the three derivatives are moderately toxic by the inhalation route (Toxicity Categories I and II). Picloram and derivatives cause moderate eye

irritation (Toxicity Category III). Most are not skin irritants (Toxicity Category IV, except IOE in Category III). The three derivatives are skin sensitizers while picloram acid is not.

As picloram has very low acute mammalian toxicity, with acute oral LD₅₀ values in the range of 3000 to 5000 mg/kg body weight, an acute RfD has not been established.

Although technical grade picloram has been subject to several chronic bioassays for carcinogenicity and none of the bioassays have shown that picloram has carcinogenic potential, technical grade picloram does contain HCB, a compound that has shown carcinogenic activity in three mammalian species and has been classified as a potential human carcinogen by the U.S. EPA. Thus, this effect is considered both qualitatively and quantitatively in this risk assessment.

Picloram was not teratogenic in rats and rabbits, and no reproductive adverse effects were observed in rats. As a result of evaluation of the mutagenicity test battery required for pesticide registration, U.S. EPA has determined that picloram is not mutagen. A two-generation reproduction study of picloram (K salt) in rats reported no endocrine effects at doses as high as 1000 mg/kg/day. Endocrine effect endpoints included reproductive outcomes and histopathological examination of tissues. In this study, renal effects and increased body weight gain were observed at 1000 mg/kg/day (i.e., the maximum tolerated dose was tested).

In chronic toxicity studies, the most sensitive effect for picloram in mammals involves effects on the liver. In a 2-year rat feeding study the NOAEL was 20 mg/kg/day based on an increase in liver size and an alteration in the staining properties of centrilobular hepatocytes. Dogs appear to be somewhat more sensitive to picloram than rats. In a six month dog feeding study the two highest dose levels resulted in increase in absolute and relative liver weight in two males and changes in liver enzyme activity.

U.S. EP has established an RfD for picloram of 0.20 mg/kg/day based on a NOEL of 20 mg/kg/day for the 2-year rat chronic feeding study. An uncertainty factor of 100 was used to account for the inter-species extrapolation and intraspecies variability.

In 1995 U.S. EPA classified picloram as a Group E carcinogen (evidence of noncarcinogenicity for humans). Even though picloram was shown to be non-carcinogenic, a cancer risk assessment was performed on the maximum HCB concentration since U.S. EPA classifies HCB as a Group B carcinogen. Based on an analysis of HCB (as a picloram impurity) dietary exposure to the U.S. population, U.S. EPA estimates the excess cancer risk to be 7×10^{-7} , which is below a level of concern (generally set at 1.0×10^{-6} lifetime cancer risk).

Picloram chemical characteristics useful in evaluating environmental fate and human exposure are given below. A discussion of chemical characteristics and ratings is given in Chapter 4.

Table 3-15
Picloram chemical characteristics

Chemical characteristic	Value (rating)
Solubility in water at 20 °C (mg/l)	560 (high)
Octanol-water partition coefficient at pH 7, 20 °C	1.20×10^{-02} (low)
Vapor pressure at 25 °C (mPa)	8.0×10^{-05} (intermediate state)
Henry's law constant at 20 °C (dimensionless)	1.42×10^{-11} (non-volatile)
Soil degradation $\frac{1}{2}$ life (days) (aerobic)	90 (moderately persistent)
Koc - Organic-carbon sorption constant (ml/g)	35 (mobile)
GUS leaching potential index	4.80 (high leachability)

Tebuthiuron

Tebuthiuron is a relatively nonselective, soil activated herbicide. It is used to control broadleaf and woody weeds, grasses and brush on feed crop sites (pasture and rangeland) and a variety of non-food crop sites including airports/landing fields, outdoor industrial areas, non-agricultural rights-of-way, fencerows, hedgerows, uncultivated areas/soils, and under paved roads and sidewalks in areas where no future landscaping is planned. Primary uses include rangeland and near railroads and other industrial facilities. Single active ingredient formulations include granular, pelleted/tableted, wettable powder, water dispersible (wetable) granules, and technical grade/solid products. Three multiple active ingredient formulations (granulars) also are registered. The commercial product Spike® 20P is commonly used in electrical utility right-of-way applications. Spike® is applied to the soil surface as a low-volume (backpack) or high-volume broadcast. Application rates vary from 1.5 to 3.5 lb/acre – recommended rates increase with increasing soil organic matter content.

Tebuthiuron is moderately toxic by the oral route. The oral LD₅₀ values for technical tebuthiuron ranged from 387 to 477 mg/kg in rats and 528 to 620 mg/kg in mice. U.S. EPA classifies tebuthiuron as Toxicity Category II for this effect in rats, rabbits and cats, and in Category III for mice and dogs. Tebuthiuron is practically non-toxic by the dermal route (Toxicity Category IV), and only slightly toxic by the inhalation route (Toxicity Category III). Tebuthiuron is not a dermal irritant, causes only slight irritation to the eyes (Toxicity Category IV), and is not a dermal sensitizer.

In a 90-day rat feeding study, the NOAEL was 50 mg/kg/day and the LOAEL was 125 mg/kg/day. The toxic effects observed in both sexes were reduced body weight, increases in relative liver, kidney and gonad weights, and slight vacuolation of pancreatic acinar cells. In addition, males also had increased relative spleen and prostate gland weights.

In a 90-day dog study, the NOAEL was 12.5 mg/kg/day. The LOAEL 25 mg/kg/day, based upon findings of anorexia, weight loss, increases in blood urea nitrogen and alkaline phosphatase, and increases in spleen and thyroid gland weights.

Dermal application of 1000 mg/kg (only dose tested) of tebuthiuron to rabbits for 6 hours per day for 21 consecutive days resulted in slight erythema which cleared by 7 days, and increased blood glucose values. The NOAEL was less than 1000 mg/kg/day.

In a 1-year dog study, the NOAEL was 25 mg/kg/day. The LOAEL was 50 mg/kg/day, based on clinical signs of anorexia, diarrhea, and emesis and increases in thrombocyte count, alanine transferase and alkaline phosphatase levels, and weight of the liver, kidney and thyroid gland.

In studies in rats and rabbits, no compound-related developmental effects were observed.

In a two-generation study in rats, the NOAEL was 7 mg/kg/day based on reduced rate of body weight gain.

Results of in vitro mutagenicity studies indicate that tebuthiuron does not appear to be mutagenic.

The RfD for tebuthiuron is 0.07 mg/kg/day based on results of the 2-generation rat reproduction study. This value was determined from the NOAEL of 7 mg/kg/day and an uncertainty factor of 100 (factor of 10 for interspecies extrapolation and a factor of 10 for intraspecies variance).

In 2-year rat feeding studies (2 studies of identical design), the NOAEL was 40 mg/kg/day. The LOAEL was 80 mg/kg/day (highest dose tested), based upon a reduction in weight gain and elevated kidney weights. No compound related carcinogenic effects were observed.

In 2-year mice feeding studies (2 studies of identical design), the NOAEL was 228 mg/kg/day, the highest dose tested. No compound-related carcinogenic effects were observed.

Based on these studies, in which no compound-related carcinogenic effects were observed, in 1994 U.S. EPA classified tebuthiuron as a Group D (not classifiable as to human carcinogenicity).

Tebuthiuron chemical characteristics useful in evaluating environmental fate and human exposure are given below. A discussion of chemical characteristics and ratings is given in Chapter 4.

Table 3-16
Tebuthiuron chemical characteristics

Chemical characteristic	Value (rating)
Solubility in water at 20 °C (mg/l)	2500 (high)
Octanol-water partition coefficient at pH 7, 20 °C	6.17×10^{01} (low)
Vapor pressure at 25 °C (mPa)	0.27 (volatile)
Henry's law constant at 20 °C (dimensionless)	1.01×10^{-08} (non-volatile)
Soil degradation $\frac{1}{2}$ life (days) (aerobic)	400 (very persistent)
Koc - Organic-carbon sorption constant (ml/g)	80 (moderately mobile)
GUS leaching potential index	5.46 (high)

Triclopyr

Triclopyr is used as a selective herbicide to control broad leaf weeds and brush on a variety of sites including rights-of-way, pasture and rangelands, forests, rice, and turf, including home lawns. Triclopyr products are formulated as soluble concentrates, emulsifiable concentrates, liquids (pressurized and ready-to-use), granulars, wettable powders and pellets.

Two forms of triclopyr are used commercially as herbicides: the triethylamine salt and the butoxyethyl ester. Commercial products commonly used in electric utility right-of-way applications include Garlon® 3A, Garlon® 4, and Pathfinder II®. Garlon 3A contains the triethylamine salt of the triclopyr and inert ingredients and requires the use of a non-ionic surfactant. In addition to triethylamine, Garlon 3A contains EDTA, a common chelating agent, and ethanol. Garlon 4, Tahoe® 4e, and Pathfinder II RTU contain the butoxyethyl ester of triclopyr. Pathfinder II is a cut surface application. Garlon 4 and Tahoe 4e contain kerosene and surfactants. Exact concentrations of other ingredients in pesticide formulations are proprietary; however, kerosene is likely to be present at 1-6%. Pathfinder II is a cut surface application. Garlon products are commonly applied as low-volume foliar (backpack) and high-volume foliar.

In evaluating the toxicity of triclopyr, U.S. EPA considers triclopyr acid, triclopyr triethylamine salt (TEA) and triclopyr butoxyethyl ester (BEE) to be bioequivalent (similar potential for toxic effects, and similar adsorption, distribution, metabolism, and elimination from the body).

The major metabolite of triclopyr in both mammals and the environment is 3,5,6-trichloro-2-pyridinol (TCP). U.S. EPA considers TCP of toxicological concern. In addition, TCP is also a metabolite of the insecticide chlorpyrifos. In assessing the potential for triclopyr to contaminate ground water that may be used for drinking water, the aggregate exposure to TCP from the breakdown of both triclopyr and chlorpyrifos should be considered.

Technical triclopyr acid is slightly toxic by oral and dermal routes; U.S. EPA classifies triclopyr technical acid as Toxicity Category III for these effects. Triclopyr BEE and TEA are slightly toxic by oral (Toxicity Category III) and dermal (Toxicity Category III) routes of exposure, have very low toxicity by inhalation (Toxicity Category IV), and do not cause dermal irritation. In a primary eye irritation study, triclopyr TEA was found to be corrosive while BEE was found to be minimally irritating. Both TEA and BEE were found to cause dermal sensitization.

Animal testing for acute toxicity show LD₅₀ values for the 3 forms of triclopyr are similar; reported LD50 values for the free acid are 630 and 729 mg/kg, TEA as Garlon 3A are 828 mg/kg and 594 mg/kg, and triclopyr BEE is 803 mg/kg.

In a 13-week subchronic rat study, the NOAEL was 5 mg/kg/day, and the LOAEL was 20 mg/kg/day, based on histopathological changes in the kidneys.

In a 228-day study in dogs, the NOAEL was 10 mg/kg/day, and the LOAEL was 20 mg/kg/day, based on the decreased body weight gain in male dogs, decreased hematological parameters in male dogs, changes in clinical chemistry in male and female dogs, and liver histopathology in male and female dogs.

In a 22-month study in mice, the NOAEL was 28.6 mg/kg/day in male mice, and 26.5 mg/kg/day in female mice, and the LOAEL was 143 mg/kg/day in male mice and 135 mg/kg/day in female mice, based on the decreased body weight gain. There were no compound-related tumors observed in male mice. However, female mice had a significant increasing trend in mammary gland adenocarcinomas.

In a rabbit developmental toxicity study with triclopyr BEE, the NOAEL was 30 mg/kg. The LOAEL was 100 mg/kg, based on the cesarean section observations of decreased total live fetuses and increased total fetal deaths, as well as the observations of increased fetal and/or litter incidence of skeletal anomalies and variants observed at this dose.

In a rat developmental toxicity study with triclopyr TEA, the NOAEL was 100 mg/kg. The developmental LOAEL was 300 mg/kg based on decreased mean fetal weight, and increased fetal and litter incidence of skeletal anomalies, and increased fetal incidence of unossified sternebrae.

In a rabbit developmental toxicity study with triclopyr TEA, the NOAEL was 30 mg/kg. The LOAEL was 100 mg/kg, based on the decreased number of live implants, decreased live fetuses, and increased embryonic deaths.

In a rat 2 generation reproductive toxicity study with triclopyr acid, the parental NOAEL was 5 mg/kg/day (males and females), and the parental LOAEL was 25 mg/kg/day, based on increased incidence of proximal tubular degeneration in male and female P1 and P2 rats.

Based on U.S. EPA's evaluation of a battery of tests required for pesticide registration, triclopyr is not mutagenic.

In a 2-year rat study, the NOAEL was 12 mg/kg/day for males and 36 mg/kg/day for females. The LOAEL for males was 36 mg/kg/day based on marginal increases in proximal tubular degeneration at 6 months. There were no significant increasing trends in tumor incidence for male rats. There was a significant increase in the incidence of adrenal gland benign pheochromocytomas and benign and/or malignant pheochromocytomas combined, and in the incidence of skin fibromas. Female rats had significant increasing trends in mammary gland adenocarcinomas, and in adenomas and/or adenocarcinomas combined. There was a significant difference increase for mammary gland adenomas and/or adenocarcinomas.

In male rats and mice, no statistically significant dose-related trends in tumor incidence were apparent. Statistically significant increases were observed for some tumor types – benign and/or malignant pheochromocytomas combined as well as skin fibromas – in rats but not mice. In female rats and mice, there was a statistically significant dose-related increase in mammary gland adenocarcinomas. As a result of a weight-of-the-evidence evaluation, in 1998 U.S. EPA classified triclopyr as a Group D chemical (not classifiable as to human carcinogenicity). This decision was based on increases in mammary tumors in both the female rat and mouse, and adrenal pheochromocytomas in the male rat, which were considered to be only a marginal response, and the absence of additional support from structural analogs or genotoxicity.

The triclopyr chronic RfD is 0.05 mg/kg/day, based on the 2-generation reproduction toxicity study in rats with a NOEL of 5.0 mg/kg/day, and an uncertainty factor of 100 (10 for interspecies

differences in response, and 10 for intraspecies differences). U.S. EPA finds that an uncertainty factor of 100 is adequately protective of infants and children based on reliable pre- and post-natal data that indicate no special sensitivity of young animals to triclopyr.

The triclopyr acute RfD is 1.0 mg/kg/day for the general population. This is based on the NOAEL of 100 mg/kg/day from the rat developmental toxicity study. This acute RfD is not applicable to females between the ages of 13-50 years (females of child bearing age). For these individuals, the acute RfD is set at 0.05 mg/kg/day, equivalent to the chronic RfD.

Triclopyr chemical characteristics useful in evaluating environmental fate and human exposure are given below. A discussion of chemical characteristics and ratings is given in Chapter 4.

Table 3-17
Triclopyr chemical characteristics

Chemical characteristic	Value (rating)
Solubility in water at 20 °C (mg/l)	8100 (high)
Octanol-water partition coefficient at pH 7, 20 °C	4.17×10^4 (high)
Vapor pressure at 25 °C (mPa)	0.1 (volatile)
Henry's law constant at 20 °C (dimensionless)	4.00×10^{-8} (non-volatile)
Soil degradation $\frac{1}{2}$ life (days) (aerobic)	39 (moderately persistent)
Koc - Organic-carbon sorption constant (ml/g)	48 (mobile)
GUS leaching potential index	3.69 (high)

Trifluralin

Trifluralin is a preemergent herbicide used to control annual grasses and broadleaf weeds on a variety of food crops including soybeans and cotton, and non-food crop sites, including rights-of-way and residential use sites. The herbicide is formulated as a liquid, emulsifiable concentrate, granular, flowable concentrate, impregnated material, soluble concentrate/liquid, soluble concentrate/solid, and water dispersible granules (dry flowable). The commercial product Snapshot® (a granular formulation containing trifluralin and isoxaben) is commonly used in electric utility right-of-way applications. Trifluralin is applied to the soil surface as a low-volume (backpack) or high-volume broadcast.

U.S. EPA classifies trifluralin technical as Toxicity Category IV (very low toxicity) for acute oral toxicity and dermal irritation, and Toxicity Category III (slightly toxic) for acute dermal toxicity, acute inhalation toxicity and eye irritation potential. Trifluralin is also classified as a dermal sensitizer.

In a 90-Day rat feeding study, the NOAEL was 2.5 mg/kg/day. The LOAEL was 10 mg/kg/day, based on increased hyaline droplet formation in cortical cells, increased total urinary protein excretion, and changes in urine color and clarity.

In 31-Day rat dermal toxicity study, the NOAEL was 200 mg/kg/day based upon increased liver weight at the highest dose tested.

In a 1-year dog study, the NOAEL was 2.4 mg/kg/day and the LOAEL was 40 mg/kg/day based upon reduced body weight, decreased red cells and hemoglobin levels, increased thrombocyte, methemoglobin, cholesterol and triglyceride levels, and increased liver weight.

In a developmental toxicity rat study, the NOAEL was 100 mg/kg/day. The LOAEL was 500 mg/kg/day based on increased total litter resorptions.

In a developmental toxicity rabbit study, the maternal toxicity NOAEL was 100 mg/kg/day due to anorexia, cachexia and resulting abortion at higher dose levels. The developmental toxicity NOAEL was 225 mg/kg/day based on depressed fetal weight and an increased number of fetal runts at higher doses.

No teratogenic effects occurred in rats or rabbits.

In a 2-generation rat study, the NOAEL was 15 mg/kg/day. The LOAEL was 47mg/kg/day based on reduced body weights in parental animals.

In a second 2-generation rat study, the parental LOAEL 10 mg/kg/day. The reproductive and developmental toxicity NOAEL was 10 mg/kg/day, and the LOAEL was 32.5 mg/kg/day, based on reduced weanling body weights and reduced litter sizes.

Based on U.S. EPA's evaluation of a battery of tests required for pesticide registration, trifluralin is not mutagenic.

The trifluralin chronic RfD is 0.024 mg/kg/day, based on the 1-year dog study with a NOAEL of 2.4 mg/kg/day, and an uncertainty factor of 100 (10 for interspecies differences in response, and 10 for intraspecies differences). U.S. EPA finds that uncertainty factor of 100 is adequately protective of infants and children based on reliable pre- and post-natal data that indicate no special sensitivity of young animals to triclopyr.

The trifluralin acute RfD is 1.0 mg/kg/day for females between the ages of 13-50 years (females of child bearing age). This is based on the NOAEL of 100 mg/kg/day from the rat developmental toxicity study. U.S. EPA has not established an acute RfD for the general population, except females between the ages of 13-50 years.

Several rodent carcinogenicity studies have been evaluated by U.S. EPA.

In a 2-year rat study, (Fischer 344 rats) the highest dose (325 mg/kg/day) resulted in significant increases of combined malignant and benign urinary bladder tumors in females. An increase in the incidence of carcinomas of the renal pelvis was seen in all dose groups of males. In addition, an increase in the incidence of thyroid gland follicular cell tumors (adenomas plus carcinomas combined) in males was found. Other studies include a 2-year rat study (Sprague Dawley rats), a 78-week study (Osborne-Mendel rats) conducted by the National Cancer Institute, and a 2-year

study (Wistar rats). In Wistar rat study, the LOAEL was 40 mg/kg/day based on body weight changes.

Mouse carcinogenicity studies of trifluralin include a 2-year study in B6C3F₁ mice and a 2-year study in NMRI mice, which found no tumors due to the test compound.

U.S. EPA classifies trifluralin as Group C, possible human carcinogen, with a Q₁* of 5.8 x 10⁻³ (mg/kg/day)⁻¹.

Trifluralin chemical characteristics useful in evaluating environmental fate and human exposure are given below. A discussion of chemical characteristics and ratings is given in Chapter 4.

Table 3-18
Trifluralin chemical characteristics

Chemical characteristic	Value (rating)
Solubility in water at 20 °C (mg/l)	0.221 (low)
Octanol-water partition coefficient at pH 7, 20 °C	1.86 X 10 ⁰⁵ (high)
Vapor pressure at 25 °C (mPa)	9.5 (volatile)
Henry's law constant at 20 °C (dimensionless)	4.00 X 10 ⁻⁰² (volatile)
Soil degradation ½ life (days) (aerobic)	181 (persistent)
Koc - Organic-carbon sorption constant (ml/g)	8765 (non-mobile)
GUS leaching potential index	0.13 (low)

4

PESTICIDE FATE AND EXPOSURE ASSESSMENT

Pesticide Characteristics

For human and ecological risk assessments, pesticide physical and chemical characteristics are required as basic or supportive data for pesticide registration. Pesticide physical-chemical and degradation characteristics that influence environmental fate, including leaching potential, are described in more detail below.

Water solubility is defined as the solubility of a chemical in water at a specified temperature, usually expressed in mg/l (ppm), or mg/l (ppb). A useful axiom in understanding solubility is “like dissolves like – polar solvents will dissolve polar solutes and nonpolar solvents will dissolve nonpolar solutes”. Many pesticides are relatively nonpolar organic compounds and are not very soluble in water, a polar solvent. However, the water solubility can be used to differentiate pesticides with regards to their potential to distribute to aqueous compartments, and move with water, such as leaching or runoff. Solubility, like other physical-chemical characteristics, is most useful as a means of comparison, as the chemical composition of natural waters, such as pH, hardness, salinity, and dissolved organic matter (DOM), can influence how pesticides are distributed to in the environment.

The **pH** of water can have a dramatic effect on solubility for some pesticides. In understanding the influence of pH, pesticides can be divided in two groups, those that are in a charged state in the environment (i.e., not neutral) and those that are neutral. The charged state is highly variable from the strongly charged salts, such as sodium chloride to very weakly charged organic acids and bases. However, all of these compounds are considered polar. Neutral compounds (no charge) are considered nonpolar. Charged species are more soluble in water as compared to their neutral counterparts. It is therefore important to know whether or not, and to what extent, a pesticide forms ions in environmental systems. Polar pesticides are often weak acids or bases. Acids have a negative charge and bases have positive charge. For example, 2,4 D is an acid and atrazine is a base.

A special case is the herbicide glyphosate; glyphosate is a simple molecule, but it contains two acid functional groups and one basic group.

Volatilization is the conversion from a liquid (or solid) to a vapor. In an ideal system, **vapor pressure** is the partial pressure on the walls of a closed vessel when a chemical is at equilibrium between the gaseous and solid or condensed phases. The greater the amount of the chemical in the gaseous phase the greater the pressure. Vapor pressure is temperature dependent; the higher the temperature the greater the volatility. In natural settings, vapor pressure may be decreased over the ideal condition due to sorption to plant or soil surfaces. For pesticides in water,

exchange between water and the atmosphere is influenced by both vapor pressure and water solubility. This is represented by the **Henry's Law constant** (K_H).

The Henry's Law constant (K_H) represents the air-water distribution ratio for neutral compounds at dilute solute concentrations in pure water. In natural waters this air-water partition is approximated by K_H . Pesticides that have a high vapor pressure and low water solubility have a greater tendency to exchange with the atmosphere from water.

Octanol-water partition coefficient (K_{ow}): Around 1900, chemists began to use n-octanol as a surrogate for organisms in studying the uptake of non polar pharmaceuticals. Today the octanol-water partition coefficient (K_{ow}) is used to estimate equilibrium partitioning of non-polar organics between water and organisms. The K_{ow} is also directly proportional to partitioning into soil humus and other naturally occurring organic phases, such as plant epicuticular waxes. Experimentally, the K_{ow} measures the partitioning behavior of organic compounds between two immiscible liquids-- water and octanol. K_{ow} is a *dimensionless* equilibrium constant. K_{ow} is often represented by $\text{Log}_{10}(K_{ow})$, or simply $\text{Log } K_{ow}$, or $\text{Log } P$.

Pesticide degradation: When pesticides are transformed into other chemicals they often lose their efficacy, however, the transformation products may still be of concern. Consequently, pesticide registration under FIFRA requires that the major transformation products be identified and their toxicity evaluated. Pesticide transformation in the environment can be categorized into three processes, as follows:

- Photodegradation – transformation in the presence of sunlight
- Chemical degradation – abiotic chemical reactions
- Biological degradation – degradation by biota (living organisms)

The term degradation implies that the products are formed from a portion of the original chemical. However, pesticide “degradation” may result in products that are larger, such as dimmers, trimers, or polymers, or products that have the same molecular weight, but experience a conformational change – as from one isomer to another. However, the most common chemical reactions – oxidation and hydrolysis – usually result in products that are formed from a portion of the parent.

Degradation by living organisms, usually catalyzed by enzymes, is often called metabolism. Metabolism is the sum of all processes that assimilate and incorporate, or detoxify and eliminate pesticides. Detoxification usually results in metabolites that are smaller and more water soluble (more polar) than the parent pesticide, to facilitate elimination from the organism.

To what extent should a pesticide degrade before there is no longer a concern? Most organic pesticide must undergo many reactions before they are completely degraded. When does a pesticide truly “go away”? Soil microbial communities can be very efficient at utilizing organic compounds; the most complete degradation process is called mineralization – the products being water, carbon dioxide, ammonia, nitrate, and minerals (i.e., Cl, Na, F, Br). Pesticides that are not mineralized may be incorporated into organic matter. Plant uptake and metabolism may result in assimilation and incorporation into biochemicals – such as the incorporation of 2,4-D metabolites into amino acids – or detoxification and elimination.

The “complete” degradation pathway of pesticides – by photochemical, chemical, or biological pathways – is rarely known. However, FIFRA requires the identification of all degradation products of “toxicological concern”. This information is contained in research reports submitted to U.S. EPA as a part of product registration and often is not published in the open literature.

Pesticide Mobility

Pesticide **mobility** may result in redistribution within the application site or movement of some amount of pesticide off site. After application, a pesticide may:

- attach (sorb) to soil particles, vegetation, or other surfaces and remain near the site of deposition;
- attach (sorb) to soil particles and move with eroded soil in runoff or wind;
- dissolve in water and be taken up by plants, move in runoff, or leach; or
- volatilize or erode from foliage or soil with wind and become airborne.

Mobility is affected by the pesticide’s sorption, water solubility, and vapor pressure. Mobility is also influenced by environmental and site characteristics including weather, topography, canopy, and ground cover; and soil organic matter, texture, and structure.

Sorption describes the attraction between a chemical and soil, vegetation, or other surfaces. However, sorption most often refers to the binding of a chemical to soil particles. The term sorption is used to encompass both adsorption onto a two dimensional soil surface, and absorption into a three-dimensional soil matrix. Pesticides that are sorbed to soil particles are more likely to remain in the root zone where they may be available for plant uptake and microbial or chemical degradation. However, pesticides that are strongly sorbed to soil are usually less available for microbial degradation and plant uptake. Pesticides that sorb weakly to soil particles are more likely to move through the soil profile with infiltrating water.

Sorption is determined by the chemical characteristics of the pesticide. The specific mechanisms for the sorbing of a chemical to the soil are not easily defined. Numerous mechanisms may operate in a particular situation, including strong or weak ionic attraction, hydrophobic attraction, and hydrogen-bonding. For pesticides that are weak acids or bases sorption is influenced by the pH of the soil. Weak acid or base pesticides may carry a positive or negative charge, or no charge depending on pH.

Sorption is also influenced by soil moisture, organic matter content, and texture. Pesticides are more readily sorbed onto dry soil because water competes with pesticides for binding sites in moist soil. Organic matter and clay particles both have plenty of surface area and are chemically active. Soils high in clay or organic matter, or both, have a high potential to sorb pesticides. Clay content is also important for holding organic matter in the soil. Sand particles provide less surface area for sorption. Pesticides are more likely to move away from the point of application in sandy soils. Soils that have an organic layer, such as crop residues or thatch in turfgrass, may strongly sorb pesticides and reduce their mobility

The sorption of a particular pesticide to a soil is measured in a laboratory by mixing water, pesticide, and soil. After equilibrium has been reached, the amount of pesticide remaining in solution is measured. The concentration of pesticide sorbed to the soil in the mixture is divided by the pesticide concentration still in solution. This yields the **distribution coefficient, K_d** . A low distribution coefficient indicates that more of the pesticide is in solution; a higher value indicates that the pesticide is more strongly sorbed to soil.

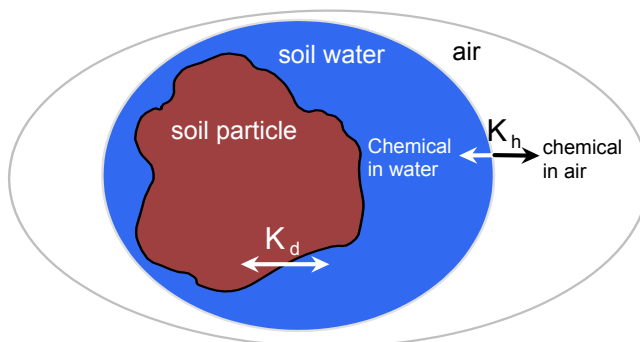


Figure 4-1
Distribution Coefficient (K_d) and Henry's Law Constant (K_h).

The distribution coefficient determined in the laboratory will vary depending on the ratio of soil to water and the chemical properties of both the pesticide and the soil. For this reason, a different number, the **sorption coefficient (K_{oc})**, is used to compare the relative sorption of pesticides. K_{oc} is the distribution coefficient divided by the amount of organic carbon in the soil (Soil organic carbon is directly proportional to soil organic matter, which is primarily responsible for a soil's sorption properties.). Pesticides with higher the K_{oc} values are more strongly sorbed, and therefore, less mobile.

Pesticide Persistence

A pesticide's persistence in the environment, sometimes called field dissipation, is the sum of the dissipation mechanisms as measured by the loss of a pesticide from a compartment (i.e., soil, air, water, biota). The measured compartment is usually more specific; for example pesticide dissipation may be determined in a specified soil profile, the harvested crop, the foliar surface, surface water, or ground water at a specified depth.

Processes that can contribute to field dissipation include all degradation pathways (i.e., photochemical, chemical, biological) and all mobility pathways (i.e., volatile loss, leaching, runoff, foliar wash-off, crop removal). The contribution of each process to the overall dissipation depends on the pesticide's physical-chemical properties, degradation kinetics, and the compartment measured. For example, photo-degradation and volatile loss may contribute more to dissipation from foliar surfaces as compared to subsurface soil. Microbial degradation may

play a larger roll in field dissipation in well aerated, nutrient rich, surface soils as compared to soils in lower horizons.

As with pesticide degradation, field dissipation is often estimated by assuming first order kinetics, which allows the use of half-life values to estimate persistence. The figure below (Nash, 1988) shows for a pesticide applied to soil, possible dissipation processes and their contribution to the dissipation curve. Although all processes may contribute simultaneously, volatile loss and photo-degradation may be primarily responsible for the initial loss, followed plant uptake, chemical and microbial degradation and leaching. The figure below is conceptual, as the processes that determine pesticide field dissipation and their contribution with time is highly variable, and will be unique to the pesticide, pesticide use practice and environmental conditions.

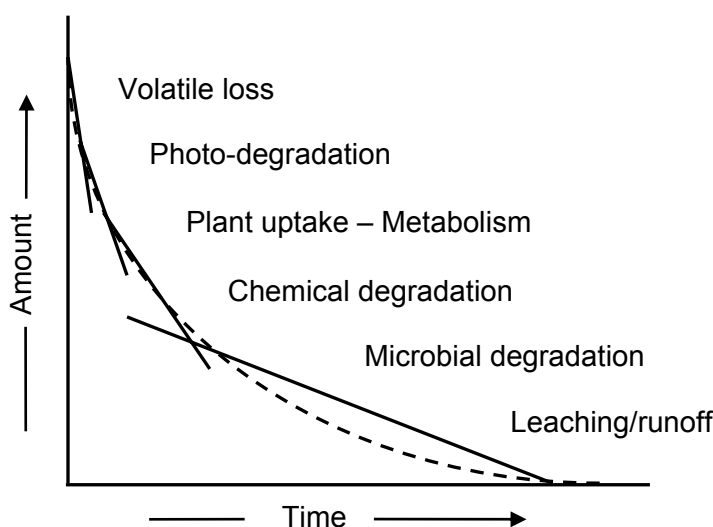


Figure 4-2
Pesticide Dissipation from a Surface Soil Compartment

Pesticide persistence is often expressed in terms of half-life. This is the length of time required for one-half of the original quantity to break down. Pesticides can be divided into three categories based on half-lives: *non persistent* pesticides with a typical soil half-life of less than 30 days, *moderately persistent* pesticides with a typical soil half-life of 30 to 100 days, or *persistent* pesticides with a typical soil half-life of more than 100 days.

The figure 4-3 shows field dissipation curves for 3 pesticides, applied at 1 kg/acre, with half-lives of 14, 20 and 90 days. Half-life is an estimate of how long it takes for half of the applied amount to dissipate. Two half-lives are one half of a half, or one quarter of the amount applied, and so on. The figure above shows the relationship between half-life and the percent of the amount applied remaining. After 3.3 half-lives there is 10% remaining, or 0.1 kg/Ha. Regardless of a pesticides characteristic half-life, the mass applied still has greatest effect on mass remaining with time after application.

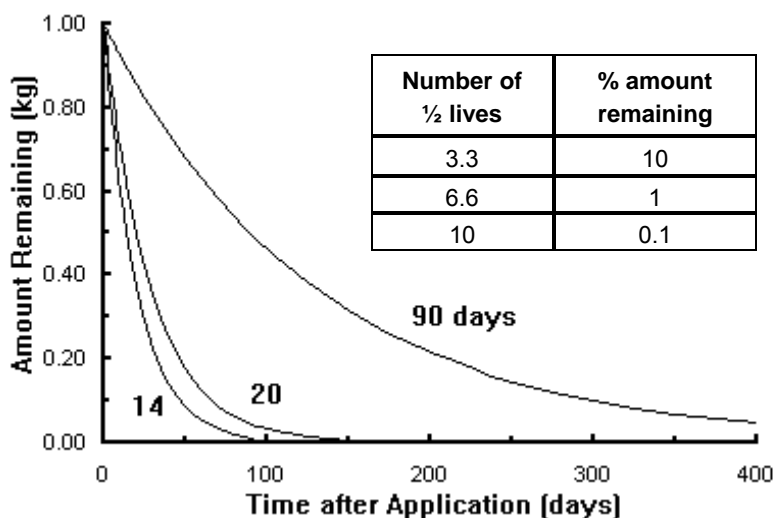


Figure 4-3
Field Dissipation Half-life

Diffusion vs. mass transfer: The pesticide physical-chemical constants K_d (K_{oc}), K_H , K_{ow} , and V_p describe the potential for the exchange of pesticides between soil, water, and air over short distances by diffusion. Transport over longer distances involves mass transfer with wind and water (runoff and leaching).

Wash-off fraction: Pesticides that are applied to foliar surfaces may be redistributed with rainfall or irrigation. This process is estimated by the wash-off fraction – the fraction of the pesticide on a foliar surface that may wash-off. The same processes that influence pesticide volatile loss from foliar surfaces affect the wash-off fraction. For pesticides applied in an aqueous carrier, before the spray has dried the potential for wash-off will be greatest, as the pesticide has little contact with the epicuticular surface; both the wax content and leaf morphology affect spray droplet dynamics, and in turn influence wash-off. As the spray deposit dries there is greater contact the epicuticular waxes and opportunity for sorption. With time the pesticide will become more tightly bound, on or within, the epicuticular waxes and less available for wash-off. In general, the greater the pesticide's water solubility the lower it's sorption to plant surfaces. Conversely, the greater a pesticide's K_{ow} , the greater the partitioning into plant epicuticular waxes and the lower the wash-off fraction.

Runoff is the movement of water over a sloping surface. Runoff can carry pesticides dissolved in water and pesticides sorbed to eroding soil. If irrigation or heavy rainfall shortly after application induces runoff, some pesticide may move off site. Heavy rainfall or overhead irrigation soon after application also may dislodge pesticide residues on foliage, creating loss with runoff. With time, residues on foliage are less likely to be washed off as they become incorporated in surface waxes. Runoff and erosion from agriculture have been identified as major contributors to water quality degradation, although runoff losses typically account for only 1–6% of applied

agrochemicals. However, runoff losses are dependent on the slope of the field, management practices (Jaynes et. al., 1999), presence or absence of sub-surface drains and the intensity and timing of rainfall after application (Southwick et. al., 1997). For example, sub-surface drains were found to decrease runoff volume and soil loss from the fields by increasing soil infiltration of rainwater. Pesticides with low water solubilities were typically found on the soil particles while those with higher solubilities were in the aqueous phase (Jury 2004).

Leaching is the removal of soluble materials by water passing through the soil. Ground water contamination occurs when pesticides move with infiltrating water through the soil profile to the water table. The closer the water table is to the surface, the greater the likelihood that it may become contaminated. Soils can also play a key role in determining the likelihood of a pesticide to leach into ground water. Pesticides that are highly water soluble, relatively persistent, and not readily sorbed to soil particles (low K_d , K_{oc}) have the greatest potential for movement.

Soil Conditions that Affect Ground Water Vulnerability and Surface Runoff

Four factors control the movement of pesticides through the soil, as follows:

1. Pesticide properties and use practices
2. Soil properties
3. Hydraulic loading
4. Vegetation management practices

Soils properties are a major determinant in the transmission of a pesticide through the soil profile. However, soil properties do not determine the risk to ground water. Proper water management, pesticide selection, low application rates, proper timing of applications, and careful handling of pesticides can reduce the risk of ground water contamination.

Soil as a Porous Media

For the purposes of describing pesticide movement, soil can be considered a porous media composed of minerals; sand, silt, clay and organic matter. Each soil has unique properties that influence the flux of air and water, pesticide sorption and pesticide transport in air or water. Pesticide solutes move with water bulk flow (convection), primarily influenced by gravitational forces. However, the porous soil creates a tortuous path resulting in hydrodynamic dispersion. Unique soils and conditions create unique patterns of pesticide convection-dispersion, making it difficult to predict water and solute flux. For non-sorbing solutes, such as bromide ion (Br^-) flux through a saturated soil can be predicted in terms of convection and dispersion. However, for most pesticides soil sorption must be considered. Sorption will determine the amount of pesticide in the soil water. As discussed previously the K_d for a given pesticide and soil estimates the amount of pesticide sorbed relative to the amount of pesticide in solution. Also important to understanding pesticide transport in soil water is pesticide desorption from soil. Once a pesticide is sorbed, the soil is equilibrated with successive clean water treatments to examine the movement of sorbed pesticide back in to the water phase. For most pesticides desorption is slower than sorption, i.e., the desorption isotherm has a shallower slope. This phenomenon is called hysteresis. The slower release of a pesticide into the soil water relative to sorption will

enhance retardation of pesticide movement in soil water by convection-dispersion under saturated conditions. Under non-saturated conditions, water moves through the soil profile by capillary flow, following the concentration gradient. Capillary flow can move water in all directions. For example, as water evaporates at the soil surface creating unsaturated conditions, water can move from within the soil profile back to the surface. Pesticide solutes in the soil water will move with the capillary flow. As with saturated flow, under non-saturated conditions, soil sorption and desorption will retard pesticide movement in the soil water.

Factors that Define Soils as a Porous Media

- Permeability
- Water table conditions
- Organic matter content
- Clay type and content

Permeability and water table conditions together define the rate at which water moves through the soil under both saturated and unsaturated conditions. Organic matter and clay type and content together characterize the *soil sorption potential*. Soil sorption potential determines the potential retardation of pesticide movement with soil water. Soils with a higher in clay content have a higher soil sorption potential.

Clay content refers to the percentage of microscopic plate-shaped grains in the soil. These tiny, flat particles have a tremendous amount of surface area per unit weight of soil, and their surfaces are chemically reactive. The higher the clay content, the greater the number of binding sites for pesticide retention. Clay content is particularly important in the subsoil, where the organic matter content is generally much lower than in the surface soil. Data on clay content are readily available in soil survey reports. For evaluation of sorption potential, it is sufficient to classify soils in generalized groups ranging from low sorption for the coarse-textured sands and gravels to high sorption for the fine-textured silty clays and clays.

The plate-shaped appearance of soil clays when viewed under a microscope is due to their crystalline structure, a lattice of silica and alumina layers. The clay lattice surface is densely negatively charged, which allows the exchange of cations (i.e., Na^+ , K^+ , Ca^{++}). Based on the nature of the lattice structure, clays can be divided into two types: expanding and non-expanding. Expanding clays, such as Illite and Montmorillonite, have a 2:1 ratio of silica to alumina layers and expand when hydrated giving access to internal surfaces. As expanding clays have a much greater (10-15X) surface area for sorption compared to the 1:1 non-expanding clays, such as Kaolinite, they have greater cation exchange capacity (CEC).

In natural soils the clay surface binding sites are often occupied with organic matter. Organic matter (OM) can be characterized as a chemically complex polymer. As demonstrated by Fulvic acid, the complex chemistry of soil OM associated with clay lattice surfaces provides a variety of binding sites for pesticides of varying chemistries and polarity.

Organic matter content is the most important variable affecting sorption of most pesticides.

Organic matter content in soil depends on climate, vegetation, position in the landscape, soil texture, and farming practices. Abundant rainfall, combined with lush natural vegetation, gives rise to soils with high organic matter contents.

Desert soils have very low organic matter contents. Grassland vegetation generally produces more organic matter deeper in the soil than forest vegetation. Organic matter decomposes more slowly in wet soils. As a result, poorly drained soils in low-lying areas tend to have more organic matter than better drained soils higher in the landscape. Sandy and gravelly soils tend to be droughty soils that support less vegetation. Under similar climatic conditions, these coarse-textured soils have less organic matter than medium- and fine-textured soils. The difference is particularly marked where rainfall is limiting for plant growth.

Management practices that return vegetation residues and animal wastes to soils help maintain soil organic matter content. Practices that harvest or destroy residues tend to reduce soil organic matters.

Soil permeability describes the porosity of soil as it influences the rate of water movement under both saturated and unsaturated conditions. Permeability is determined by the size and continuity of soil pores. Factors that influence permeability include the following:

- Texture
- Organic matter
- Structure
- Root and animal activity
- Density

Course-textured sandy and gravelly soils have the largest pores and the most rapid permeabilities. Fine-textured clayey soils have very fine pores and very slow permeability. Medium-textured loams, silt loams, and clay loams have intermediate rates of soil permeability.

Organic matter helps create and stabilize aggregates of the grains of sand, silt, and clay. These aggregates, or units of soil structure, have relatively large spaces between them, permitting more rapid water movement.

Although soil may be considered a porous media, it is rarely uniform. Extreme examples are cracking clay soils, and soils with preferential flow paths. These flow paths may be created by root channels, animal borrows, or course textured soils. Roots, burrowing insects and animals create large voids, or **macropores**, that can transmit water very rapidly under saturated conditions. Macropores are also common in very course-textured soils and in soils that crack extensively upon drying. Macropores are especially important where they are connected to the soil surface. Heavy rainfall or irrigation events may create temporarily saturated surface soil, which can lead to rapid flow through macropores. If pesticides are present, they can be carried deep into the soil in a short period of time. This will depend on the degree of sorption. For pesticides tightly bound to soil particles, macropore flow may reduce ground water vulnerability because water moving through macropores does not have a chance to extract pesticides from the

soil. Tillage generally reduces the number of macropores that are open to the soil surface. Dense, compact, or cemented soil layers have very slow rates of permeability.

Permeability of a soil in its natural setting is highly variable and extremely difficult to measure. Soil permeability for saturated conditions can be measured in the laboratory by measuring the rate of flow through a column of soil under a constant head of water. Permeability rates are given in inches per hour. Typical rates are 0.01 inches per hour for compact clay, 0.5 inches per hour for a loam with good structure, and 15 inches per hour for a loamy sand soil. The soil permeability rates published in the county soil survey reports are mostly estimates based upon soil properties, rather than results of actual measurements, but they are useful for comparing leaching potential of different soils.

Water table conditions refer to the height and duration of water tables in the soil. Shallow water tables that persist for long periods increase the risk of ground water contamination. Well-drained soils rarely have water tables that persist for long periods above a depth of 6 feet. They are much less sensitive than poorly drained soils, which may have water tables at or near the surface for several months. Two types of water tables occur in soils: perched and apparent. A perched water table is the top of a zone of saturation that is separated from permanent ground water by a soil layer of very slow permeability. An apparent water table is the top of a zone of saturation in a soil in which there are no dense or confining layers.

Perched water tables are less likely to increase the risk of ground water contamination to the same degree as apparent water tables. The soil layer that perches water acts as a barrier to prevent contaminants from moving to the permanent ground water supply. Perched water, however, is more likely to move into a surface water source, creating a concern for surface water quality. Soil survey reports contain information on water table conditions in soils. The depth to the water table, the months during which it persists, and whether it is perched or apparent all are given in tabular format. This information is very useful in assessing soil sensitivity.

Hydraulic loading refers to the total amount of water applied to the soil. No matter how permeable the soil, the leaching potential remains low if there is insufficient water to move completely through the soil. Where rainfall exceeds both plant consumptive use and the soil's ability to store water, leaching occurs. Water moving below the root zone ultimately reaches ground water, carrying with it soluble soil constituents. In these soils, the leaching potential is highly correlated with soil permeability. Irrigation compensates for water deficits in dry areas. Most irrigation water is taken up by plants, but some usually passes through the soil out of the root zone. Thus irrigation can increase ground water vulnerability. Careful management of the amount and timing of irrigation water applications can be very effective in reducing the risk of ground water contamination.

The position of a soil in the landscape also influences its hydraulic loading. Soils near a hilltop often shed water, either by runoff over the surface or by lateral flow within the soil. Soils lower on the hillside and where the slope begins to flatten out often receive excess water from the higher positions. These soils are more susceptible to leaching from the added hydraulic loading.

Influence of Soil Texture on Permeability and Sorption

As discussed previously, sorption involves a phase transfer process of vapor or dissolved molecules with adjacent solid phases (i.e., soil particles – sand, silt, and clay – and associated organic matter). Sorption retards movement of pesticides with soil water. For most pesticides, sorption potential is strongly influenced by clay and organic matter content. When pesticides are retained in the root zone due to soil sorption there is reduced risk of ground water contamination. Soil texture and organic matter content can influence permeability and pesticide sorption potential according to the following:

Fine-textured soils – silty clays and clays – have slow or very slow permeabilities and high sorption potentials. Macropore flow, however, in large cracks may be a problem.

Medium-textured soils – silt loams, silty clay loams, loams, and clay loams – have relatively slow permeabilities and relatively high sorption potentials, even in humid areas.

Coarse-textured soils – sands, loamy soils, and sandy loams – are more permeable and tend to have lower sorption potentials. Small differences in hydraulic loading and organic matter content in these soils affect pesticide movement toward ground water much more than in loamy and clayey soils.

Organic soils – those that consist almost entirely of decomposed plant material – have extremely high sorption potentials. Though these soils have naturally high water tables, cultivated organic soils have been artificially drained, which lowers the water table. Pesticide leaching potential in cultivated organic soils is low.

Soils are characterized by Natural Resources Conservation Service (NRCS) to a depth of 5-6 feet. Soils data for the U.S. is maintained in a geospatial format in the State Soil Geographic (STATSGO) and Soil Survey Geographic (SSURGO) Databases²¹. Figure 4-4 shows typical surface and subsurface water zones. Below the root zone, is an intermediate (vadose) zone that is transiently unsaturated (i.e., pores contain both air and water) and saturated depending on the recharge state of the unconfined aquifer below (saturated zone). Interflow from unconfined aquifers is often a primary source of surface water during periods when rainfall is insufficient to produce surface runoff. Deeper aquifers can be separated from shallow aquifers by layers that restrict water flow, sometimes called an aquitards. Pesticide degradation is greatest in the root zone where there is sunlight, higher temperatures, oxygen, and where the microbial community is most active. With depth the microbial community is diminished, and as pore air is replaced by water, aerobic degradation processes are greatly reduced. Most pesticides degrade much slower in the absence of oxygen (anaerobic conditions).

²¹ <http://soils.usda.gov/>

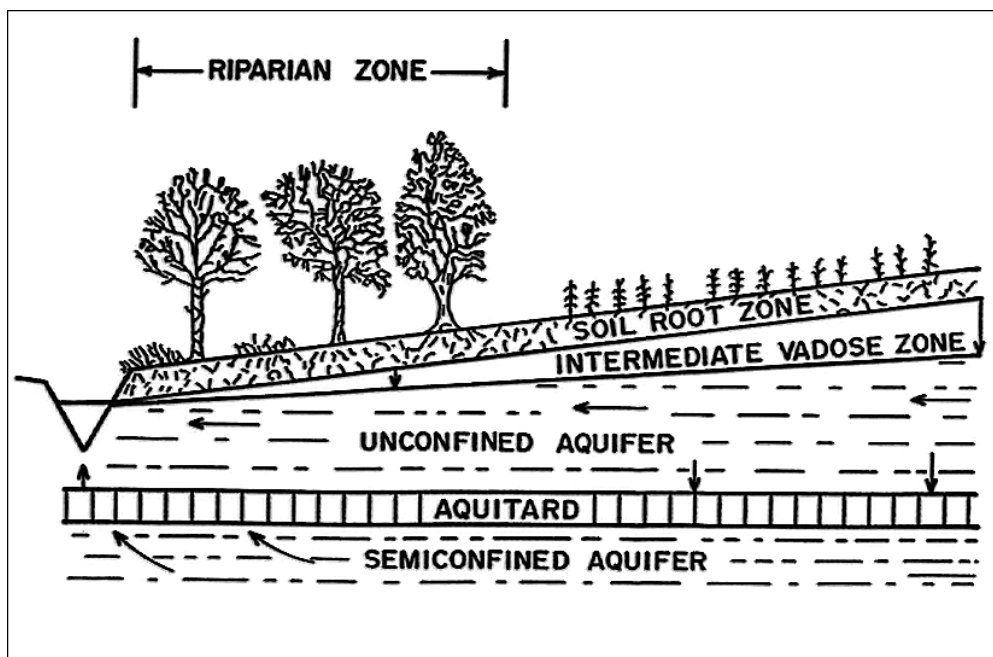


Figure 4-4
Surface and Subsurface Water Zones

Water Resource Characterization

Watersheds

The USGS has divided and sub-divided the United States into successively smaller hydrologic units²², which are classified into four levels: regions, sub-regions, accounting units, and cataloging units. The hydrologic units are arranged within each other, from the smallest (cataloging units) to the largest (regions). Each hydrologic unit is identified by a unique hydrologic unit code (HUC) consisting of two to eight digits based on the four levels of classification in the hydrologic unit system.

The first level of classification divides the Nation into 21 major geographic areas, or regions. These geographic areas contain either the drainage area of a major river, such as the Missouri region, or the combined drainage areas of a series of rivers, such as the Texas-Gulf region, which includes a number of rivers draining into the Gulf of Mexico. Eighteen of the regions occupy the land area of the conterminous United States. Alaska is region 19, the Hawaii Islands constitute region 20, and Puerto Rico and other outlying Caribbean areas are region 21. The second level of classification divides the 21 regions into 221 sub-regions. A sub-region includes the area drained by a river system, a reach of a river and its tributaries in that reach, a closed basin(s), or a group of streams forming a coastal drainage area. The third level of classification subdivides many of the sub-regions into accounting units. These 378 hydrologic accounting units nest within, or are equivalent to, the sub-regions. The fourth level of classification is the cataloging unit, the

²² <http://water.usgs.gov/GIS/huc.html>

smallest element in the hierarchy of hydrologic units. A cataloging unit is a geographic area representing part of all of a surface drainage basin, a combination of drainage basins, or a distinct hydrologic feature. These units subdivide the sub-regions and accounting units into smaller areas. There are 2264 Cataloging Units in the Nation. U.S. EPA calls these Cataloging Units watersheds.

To understand the potential impact of pesticide use practices within a watershed, climate, topography, land use, land cover, soils, pesticide use practices, and hydrologic information sufficient for routing surface and ground water are required. Each watershed may contain a number of management units. These units may result in edge-of-site pesticide losses, which become inputs to the watershed. These units are best managed through knowledge of their location within a watershed.

Runoff into surface water: An understanding of runoff types is necessary to understand the potential for pesticide loss in runoff in different climatic regions. Four types are distinguished: channel, surface, lateral subsurface flow, and base flow.

Channel runoff occurs when rain falls on a flowing stream. It appears in the hydrograph at the start of the storm and continues throughout the storm, varying with the rainfall intensity. This type of runoff is generally a negligible quantity in flood hydrographs and is ignored except in special studies.

Surface runoff or overland flow occurs when the rainfall rate is greater than the infiltration rate. The runoff equation was developed for this condition. The runoff flows on the surface of the watershed and through channels to the point of reference. This type of runoff appears in the hydrograph after the initial demands of interception, infiltration, and surface storage have been satisfied. It varies during the storm and ends during or soon after the storm. The volume of surface runoff flowing down dry channels of watersheds in arid, semiarid, or subhumid climates may be reduced by transmission losses, which could be large enough to eliminate the runoff.

Subsurface flow occurs when infiltrated rainfall meets a subsurface horizon of lower hydraulic conductivity, travels laterally above the interface, and reappears as a seep or spring. This type runoff is often called quick return flow because it contributes to the hydrograph during or soon after the storm.

Baseflow occurs when there is a fairly steady flow from natural storage. The flow comes from an aquifer that is replenished by infiltrated rainfall or surface runoff. Changes in this type of runoff seldom appear soon enough after a storm to have an influence on the hydrograph for that storm, but an increase in baseflow from a previous storm increases the streamflow rate.

All types of runoff do not regularly appear on all watersheds. Climate is one indicator of the probability of the types of runoff that will occur in a given watershed. In arid regions the flow on smaller watersheds is nearly always surface runoff. Subsurface flow is more likely in humid regions. A long succession of storms, however, may produce subsurface flow or changes in baseflow even in arid climates, although the probability of this occurring is less in arid than in humid climates. In flood hydrology baseflow is generally dealt with separately, and all other types are combined into *direct runoff*, which consists of channel runoff, surface runoff, and subsurface flow in unknown proportions.

Aquifers

A geologic formation from which significant amounts of ground water can be pumped for domestic, municipal, or agricultural uses is known as an *aquifer*. The term is relative: it means that a geologic unit yields water relative to surrounding materials, but does not indicate that a specific amount of ground water can be pumped. A small intermontane-valley aquifer yields significantly more water than its surrounding hard rock (bedrock) formations. Yet, wells within the intermontane valley may yield water at much lower rates than similar wells installed in a large alluvial river basin.

Aquifers sometimes are vertically separated by geologic formations that permit little or no water to flow. The formation that acts as water barrier is called an *aquitard* if it is much less permeable than a nearby aquifer but still permits flow (e.g., sandy clay). If the water barrier is almost impermeable (e.g., clay) and forms a more or less formidable flow barrier between multiple levels of aquifers, it is known as an *aquiclude*.

Aquifers can be of two major types: *unconfined* or *confined*. In an unconfined aquifer, there is no overlying aquitard or aquiclude. Where multiple levels of aquifers exist, the uppermost aquifer is typically unconfined. Vertical recharge by infiltration of rainwater or irrigation water downward to the unconfined aquifer is therefore not restricted. The water table at the top of the unconfined aquifer can migrate freely up and down depending on how much water is stored in the aquifer.

A confined aquifer, on the other hand, is “sandwiched” between an aquiclude above and an aquiclude or aquitard (e.g., bedrock) beneath. As a result of “backpressure”, water in the confined aquifer is pressurized. An *artesian well* occurs where the pressure is so large that the water level in a well drilled into the confined aquifer rises above the land surface—in other words, the well flows freely (if opened) without pumping. Note that a confined aquifer does not have a water table—it is completely filled with ground water.

An aquifer confined by an aquitard rather than an aquiclude is referred to as a *leaky aquifer*, or a *semiconfined aquifer*. In alluvial aquifers, the aquitard rarely is a contiguous layer of low-permeability clay, loamy clay, or sandy clay. Rather, it can be thought of as a local accumulation of multiple, horizontally discontinuous smaller clay “lenses” and other clay-rich or otherwise impermeable (or low permeability) layers. Though such lenses and layers are not contiguous, the overall effect on the regional aquifer below is identical to that of a solid, continuous aquitard. Similarly, alluvial aquifers typically are not contiguous, homogeneous layers of sand, clayey sand, and gravel, but, rather, a heterogeneous “sandwich package” with a significant amount of higher permeability material. Hence, the distinction between an aquifer and a nearby aquitard is sometimes based not on well-defined geologic boundaries, but on changes in the vertical frequency of the occurrence of (or lack of) less-permeable, horizontally-discontinuous layers of clay or other sediments that have a high percentage of fines (clay, silt).

Sometimes water collects above an impermeable layer or low-permeability layer within the unsaturated (aerated) zone, forming a “perched” water table. By definition, a perched water table is a saturated ground water zone separated from the aquifer below by a zone that is unsaturated or aerated. This should not be confused with an unconfined shallow aquifer that is separated from a deeper confined aquifer by thick, saturated layers of clay. Springs form where a water table intersects with the land surface. This may occur in a depression of the land surface,

particularly on hillsides. It can also occur where two geologic (rock) formations outcrop at the land surface, if the lower one is less permeable than the upper one. A spring also may form when back pressure forces water to the surface through a sinkhole, fracture, joint, or fault zone that acts as a conduit for water movement. Depending on the geologic history and on the surrounding geologic material, fault and fracture zones may sometimes represent a barrier to flow rather than conduit for flow, in which case water may be forced to the land surface along the top of the fault or fracture zone.

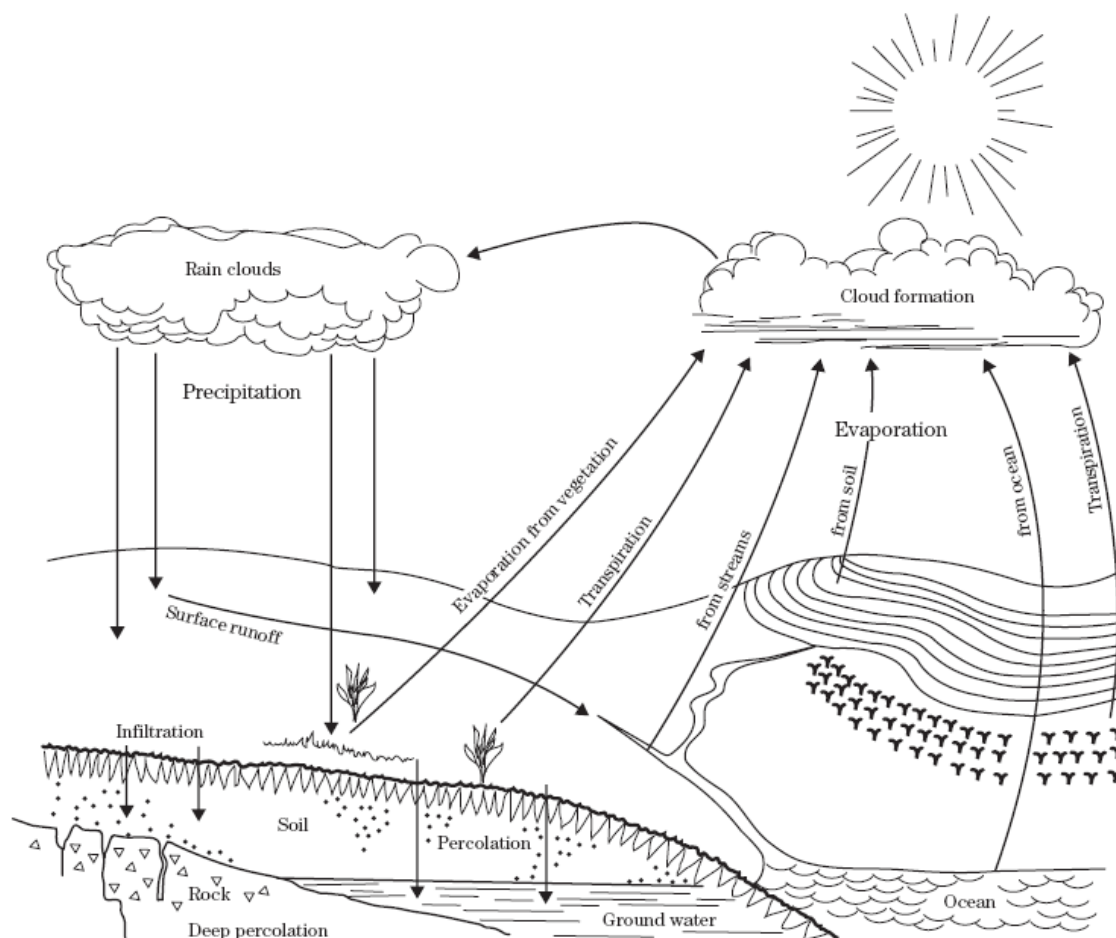
Table 4-1 demonstrates the importance of linking pesticide behavior information with awareness of water resources by region.

Table 4-1
Water Resources of Concern

Pesticide Use Location	Resource Use	Resource of Concern
Over a sole source aquifer	Drinking water	Ground water
Over a sensitive aquifer	Drinking water	Ground water
Over an aquifer recharge area	Drinking water	Ground water
Over ground water that discharges to surface water	Drinking water Aquatic habitat	Surface water
Where surface runoff goes to a water supply	Drinking water	Surface water
Where surface runoff goes to treams, ponds, rivers or lakes	Aquatic habitat	Surface water

The Hydrologic Cycle

The hydrologic cycle describes the continuous movement of water above, on, and below the surface of the Earth. Surface water occurs as streams, lakes, and wetlands, as well as bays and oceans. Surface water also includes snow and ice. The water below the surface primarily is ground water, but it also includes soil water. The hydrologic cycle commonly is portrayed by a very simplified diagram that shows only major transfers of water between continents and oceans (Figure 4-5). However, the hydrologic cycle is highly variable and can be viewed at a wide range of scales. For example, precipitation occurs nearly everywhere on earth, but its distribution is highly variable. Similarly, evaporation and transpiration return water to the atmosphere nearly everywhere, but evaporation and transpiration rates vary considerably according to climatic conditions. As a result, much of the precipitation moves back to the atmosphere and is returned to the oceans not as surface and subsurface runoff. The relative magnitudes of the individual components of the hydrologic cycle, such as evapotranspiration, may differ significantly even at small scales, as between an agricultural field or other application site and a nearby woodland.



USDA Natural Resources Conservation Service. 2004. National Engineering Handbook Part 630 Hydrology.

Figure 4-5
The Hydrologic Cycle

In the U.S. rainfall patterns differ significantly between the humid east and the arid west. In addition, in California alone there are many climatic zones depending primarily on latitude and proximity to the coastline.

Movement of ground water is difficult to visualize. Ground water moves along flow paths of varying lengths from areas of recharge to areas of discharge, at times it can be focused as an underground river, but often seepage patterns are more diffuse. The generalized flow paths start at the water table, continue through the ground-water system, and terminate at the stream or at the pumped well. The source of water to the water table (ground-water recharge) is infiltration of precipitation through the unsaturated zone. In the uppermost, unconfined aquifer, flow paths near the stream can be tens to hundreds of feet in length and have corresponding travel-times of days to a few years. The longest and deepest flow paths may be thousands of feet to tens of miles in length, and travel times may range from decades to millennia. In general, shallow ground water is more susceptible to contamination from human sources and activities because of its close

proximity to the land surface. Consequently, pesticide management practices focus primarily on the protection of shallow aquifers.

Similar to preferential flow paths (macropores) at the field scale, geologic features that provide direct conduits from the surface to ground water, bypassing the surface soils that can retard pesticide movement and provide an environment that facilitates degradation, are of concern regarding pesticide ground water contamination. Shallow soils, particularly those with a sandy soil texture, over fractured bedrock can result in contamination of deep aquifers. If the pesticide leaches beyond the surface soil layer, the preferential flow paths in the bedrock can allow rapid infiltration and lack sorptive surfaces to retard pesticide movement. Also of concern is karst topography. Karst topography is a landscape shaped by the dissolution of a layer or layers of soluble bedrock, usually carbonate rock, such as limestone or dolomite. Due to subterranean drainage, there may be very limited surface water, even to the absence of all rivers and lakes. Many karst regions display distinctive surface features, with sinkholes or dolines being the most common. Karst topography is also characterized by large springs, caves, and "losing" streams. Shallow ground water commonly flows into streams. In karst areas, however, surface water can flow into shallow ground water. In some landscapes distinctive karst surface features may be completely absent where the soluble rock is mantled, such as by glacial debris, or confined by superimposed non-soluble rock strata. Some karst regions include thousands of caves, even though evidence of caves that are big enough for human exploration is not a required characteristic of karst. Well known karst regions in the U.S. include southern half of Georgia and Florida, and Northeast Iowa. Both of these regions have in past experienced pesticide ground water contamination. In addition, Southeast Missouri, a large portion of West Texas and more than 50 percent of Kentucky have karst areas that are especially sensitive to ground water contamination because the sinkholes, caves, or other cracks in the bedrock may serve as a direct route from the surface to the ground water. Current efforts to reduce the potential for pesticide ground water contamination in these regions recognize the importance of understanding how geologic features such as karst topography contribute to ground water vulnerability in these regions.

Man-made conduits to ground water should also be considered, such as in-use or abandoned wells that can allow the surface water to reach ground water unimpeded. At pesticide application sites where runoff is expected, the common concern is the potential to contaminate surface water. However, nearby down slope or down gradient (direction of ground water flow) wells should be identified. Common well construction techniques include drilled with grout (neat cement, cement/bentonite, or bentonite), drilled with backfill (drill cuttings), driven-point (sand point), or dug. Construction usually includes a well casing sealed inside the drill hole (or driven-point) to prevent collapse, and when capped, protect the well opening at the surface. The casing should extend above the surface > 4 inches. Well casings sealed with cement or bentonite are generally the least likely to allow surface water to enter. Improperly sealed or cracked well casings, however, may provide a direct conduit from the surface to ground water. Surface water carrying chemicals can run down the outside of the well casing and contaminate ground water. In addition, a cracked casing may connect shallow ground water to deep ground water.

Summary of Factors that Determine Pesticide Leaching

Any pesticide will remain in the environment for some amount of time and move to some degree following application. To make sound vegetation management decisions, pesticide users, advisors, and resource managers should have an understanding of the fate of pesticides in the environment.

Pesticide fate in the environment depends on the rate, timing, and method of application, as well as a variety of dynamic and interrelated physical, chemical, and biological processes. These processes are influenced by environmental conditions that are often site-specific. Careful consideration of these fate processes and their interactions is necessary to evaluate the risk to ground water and surface water.

Soil and site conditions must also be considered. The most rudimentary evaluation should consider site topography and the proximity of water resources, as well as soil properties and water table conditions that influence leaching and runoff.

The properties and parameters introduced in this document are most useful as initial risk screening tools and can assist in developing relative rankings. They cannot be used to predict the absolute amount of pesticide that may enter ground water or surface water. More thorough evaluations require information on pesticide fate in conjunction with information on climate, specific soil and site characteristics, management practices, and toxicology.

Aquifers can span larger area across a number of states. Pesticide management at the watershed and regional scales should be considered in protecting these water resources.

Figure 4-6 shows the relationship between pesticide characteristics and use practices, soil properties, landscape features, and climatic factors that determine the potential for ground water contamination.

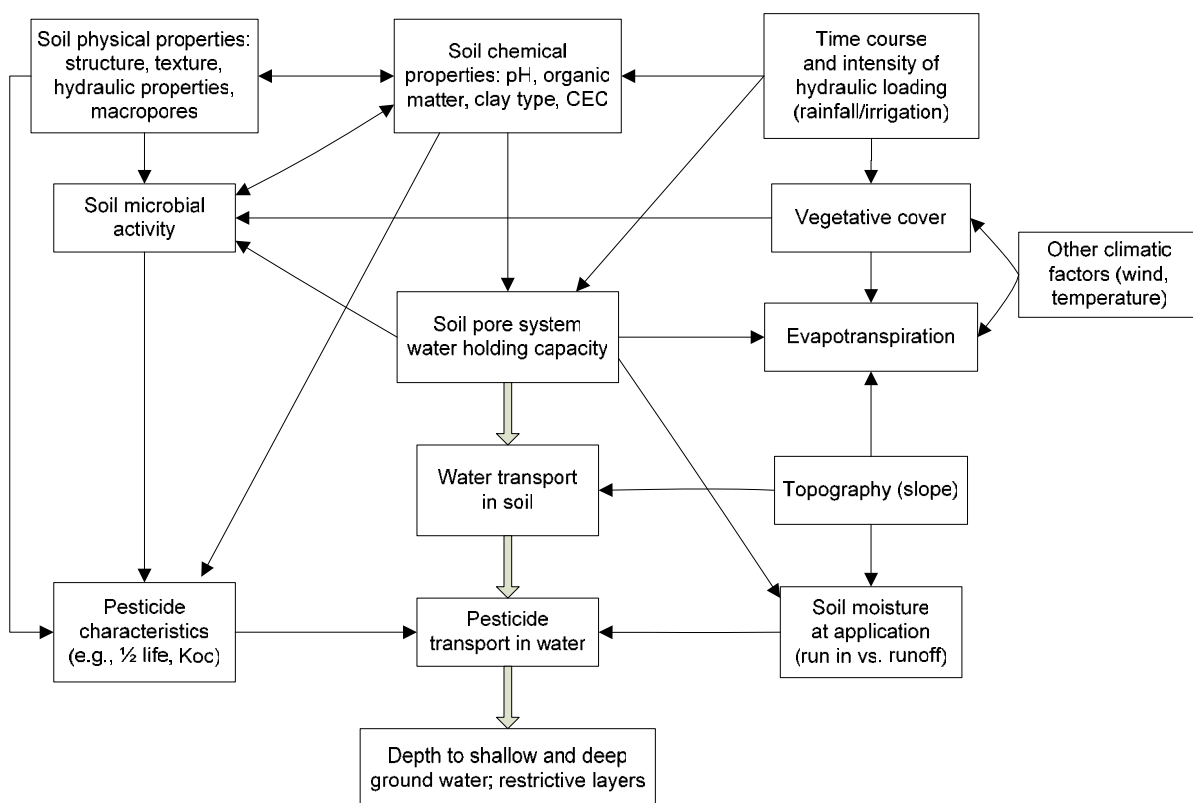


Figure 4-6
Factors Determining Pesticide Leaching

Pesticide Environmental Fate and Exposure Modeling

U.S. EPA relies on modeling, as well as measured pesticide quantities in the field, to establish the concentration levels which might be expected in surface and ground water. Relatively sophisticated environmental fate and transport models have been under development for use in risk assessment since the early 1980's. These include the Pesticide Root Zone Model (PRZM)²³ (Carsel et.al.,1984), Ground water Loading Effects of Agricultural Management Systems (GLEAMS)²⁴ (Leonard et al., 1989) and the EXposure Analysis Modeling System (EXAMS)²⁵ (Burns, 1997). Beginning in the early 1990's computer modeling of pesticide exposure began to play a larger role in risk assessment at U.S. EPA. However, environmental fate models such as PRZM, EXAMS, and GLEAMS are data and resource intensive, and not well suited to rapidly

²³ <http://www.epa.gov/ceampubl/gwater/przm3/index.htm>

²⁴ <http://www.tifton.uga.edu/sewrl/Gleams/tablcont.pdf>

²⁵ <http://www.epa.gov/ceampubl/swater/exams/index.htm>

screen pesticides for potential adverse impacts on surface or ground water resources (for a more detail see Water Models²⁶ on U.S. EPA's website).

For rapid screening of new pesticides or new pesticide uses, and in the reregistration process, U.S. EPA has developed a standard aquatic environment in which all chemicals can be assessed and compared on an equal footing; a "standard agricultural field-farm pond" scenario is used for all aquatic exposure assessments. This "standard pond" scenario assumes that rainfall onto a treated, 10 hectare agricultural field causes pesticide-laden runoff into a one hectare; 20,000 cubic meter volume; 2.00 meter deep water-body. Although this "standard scenario" was designed to predict pesticide concentrations in the standard farm pond, it has been shown to be a good predictor of upper level pesticide concentrations in small but ecologically important upland streams (Effland et. al., 1999).

The screening approach to exposure assessment is tiered, allowing the appropriate level of modeling needed to perform a risk assessment for each chemical. Each of the tiers is designed to screen out pesticides by requiring higher, more complex levels of investigation only for those that have not passed the previous tier. Each tier screens out a percentage of pesticides from having to undergo a more rigorous review prior to registration or reregistration. "Passing" a given assessment tier indicates that there is a low possibility of risk to the aquatic environment. "Failing" an assessment tier, however, does not mean the chemical is likely to cause environmental problems, but that the assessment should continue on to the next higher assessment tier. This approach should result in a more thorough analysis, focusing the greatest resources and efforts toward areas of greatest potential ecological threat.

Critical to the tiered approach is the use of sufficiently conservative procedures to minimize false negative outcomes – "passing" a given assessment tier when the outcome should have been "fail" (i.e., for every statistical comparison, there is always a defined probability that a difference will not be designated as significant, when in fact it is (Type I error or false negative when there is a null hypothesis of some adverse effect)).

Default assumptions which minimize Type I error should be chosen based on an understanding of variability and uncertainty. Variability reflects the knowledge of how things may change. Variability may take several forms. For example, three types of variability can be distinguished: statistical, situational, and arbitrary. Statistical variability reflects, at least, apparently random patterns in data. Situational variability describes variations depending on known circumstances. Arbitrary variability, as the name implies, represents an attempt to describe changes that cannot be characterized statistically or by a given set of conditions that cannot be well defined.

Variability reflects knowledge or at least an explicit assumption about how things may change, while uncertainty reflects a lack of knowledge. Variability is usually expressed quantitatively, while uncertainty is generally expressed qualitatively. For obvious reasons, when there is a paucity of data, characterizing uncertainty becomes increasingly important.

Variability may be in the form of a wide range in reported soil half-life values or soil sorption distribution coefficients (K_d) – key parameters for exposure models. Uncertainty may come from

²⁶ www.epa.gov/oppefed1/models/water/index.htm.

a lack of half-life or K_d data for a given soil type (i.e., sandy soils). Regarding uncertainty of pesticide toxic effects, current concern regarding pesticide toxicity related to effects on the endocrine system will soon require new studies (or new protocols for existing studies) to address uncertainty due to lack of data to evaluate this new toxic endpoint.

Modeling approach: For initial screening U.S. EPA uses GENEEC²⁷ (GENeric Estimated Environmental Concentration). GENEEC2 can mimic the much more sophisticated PRZM/EXAMS simulations, but requires far fewer inputs and much less time and effort to use. The model uses a chemical's label application information, its soil/water partition data and its degradation kinetics to estimate high level exposure values for the "standard" agricultural field/farm pond scenario described above. In addition, GENEEC is generic, as it does not consider differences in climate, soils, topography or crop in estimating potential pesticide exposure.

GENEEC is also simpler than the PRZM and EXAMS models in its treatment of hydrology. GENEEC is a single event model. It uses one single large rainfall/runoff event (6 inches of rain in 24 hours). Subsequent multiple-day average concentration values are calculated based on the peak day value considering degradation processes.

U.S. EPA believes that when using this tiered approach if a pesticide is not predicted to cause adverse effects on the environment (i.e., EECs do not exceed the LOC for the most sensitive species) that the possibility of harming the environment is low. Higher tier evaluations, PRZM/EXAMS simulations based on conditions more reflective of actual use site conditions, are used when LOCs are exceeded using EECs based on generic assumptions (non-use site specific).

Using linked PRZM and EXAMS models employing the "standard pond" scenario, and parameterized with local soil, weather, and farm management practices, pesticide applications to most US agricultural crops can be simulated. The PRZM/EXAMS modeling approach simulates the impact of daily weather on the treated agricultural field over a period of thirty-six years, resulting in 20-40 rainfall/runoff events per year. Pesticide enters the pond via runoff or drift where it begins degrading on the day it reaches the water. The output is daily pesticide concentrations in the pond over the thirty-six year period modeled.

The PRZM/EXAMS modeling approach assumes 5% and 1% spray drift for aerially and ground applied pesticides, respectively. The 5% assumption is based on a linear interpolation of spray drift data presented in Akesson (1990). AgDrift may also be used to estimate drift into surface water.

U.S. EPA also uses a screening model SCI-GROW²⁸ to estimate pesticide concentrations in vulnerable ground water. The model provides an exposure value, which is used to determine the potential risk to the environment and to human health from drinking water contaminated with the pesticide. The SCI-GROW estimate is based on pesticide chemical characteristics (soil aerobic half-life and the soil sorption coefficient K_{oc}), as well as the maximum application rate, and

²⁷ <http://www.epa.gov/oppefed1/models/water/index.htm#geneec2>

²⁸ http://www.epa.gov/oppefed1/models/water/scigrow_description.htm

existing data from small-scale prospective ground-water monitoring studies at sites with sandy soils and shallow ground water.

Pesticide concentrations estimated by SCI-GROW represent conservative or high-end exposure values because the model is based on ground-water monitoring studies, which were conducted by applying pesticides at maximum allowed rates and frequency to vulnerable sites (i.e., shallow aquifers, sandy, permeable soils, and substantial rainfall and/or irrigation to maximize leaching). In most cases, a large majority of the use areas will have ground water that is less vulnerable to contamination than the areas used to derive the SCI-GROW estimate. For this reason, it is not appropriate to use SCI-GROW concentrations for national or regional exposure estimates.

In addition to model estimates, monitoring data from U.S. EPA databases, U.S. Geological Survey, National Water-Quality Assessment Program, industry, states, and universities are also considered. These data are evaluated on a case-by-case basis to determine the likelihood, extent, and nature of pesticide concentration in water under current use practices and actual field conditions. When deemed reliable, U.S. EPA uses monitoring data to help characterize the levels of pesticide that are likely to be detected in surface or ground water associated with current pesticide use practices. Monitoring, however, may not target pesticide use areas or the time of year when pesticide concentrations may be at their peak, and therefore may not provide reliable estimates of acute exposure. When appropriate monitoring data shows higher confirmed detections than estimated by modeling, the higher monitoring values may be used in the risk assessment, and a re-evaluation of the model input parameters may be initiated to explore the impact of selected input values on the model output.

In situations where available toxicity data indicate that a pesticide formulation may be more toxic than the active ingredient, fate of other ingredients in the formulation may be considered. As the environmental fate of the individual components of pesticide formulations, with varying physical-chemical properties, is generally unknown, exposure modeling is limited to the instantaneous concentration in surface water following direct introduction of the formulation to surface waters via drift.

5

PESTICIDES AND GROUND WATER: RISK CHARACTERIZATION AND MANAGEMENT

As discussed in Chapter 1, to understand the possible human health risks associated with pesticides in ground water, human health risk assessments are used as a fundamental tool to characterize the potential for adverse health effects as a result of exposure to chemical substances in the environment. These assessments form a key part of the basis upon which policy makers determine whether, and to what extent, measures to reduce risks are warranted (i.e., risk management). This same process is key to pesticide users making informed decisions regarding pesticide use practices that are efficacious while meeting goals for the protection of human and environmental health.

Characterizing the risks of pesticides in drinking water requires the evaluation of each pesticide's human health hazards, potency (dose-response), and exposure potential. In evaluating the potential for exposure to pesticides in drinking water, U.S. EPA considers drinking water consumption patterns across the US to discern regional differences. Differences may result from combination of factors including pesticide use patterns, the contribution and vulnerability of the drinking water source (surface or ground water) and drinking water treatment. The outcome of a quantitative risk assessment is a pesticide concentration in drinking water, resulting in an estimated daily exposure, above which, exceeds a level of concern. In addition, periodically U.S. EPA evaluates whether pesticide use practices are likely to result in pesticide drinking water concentrations that exceed a level of concern. Once a pesticide is registered, the pesticide user is the primary determinate of the potential risk that the pesticide may pose to human and environmental health.

A detailed understanding of pesticide properties (chemical characteristics and toxicity), and the relationship between pesticide use practices and the potential for ground water contamination, should allow for the development of integrated vegetation management practices and mitigation measures necessary to eliminate pesticide ground water contamination, or reduce contamination below a level of concern. Adopting practices that reduce or eliminate pesticide ground water contamination may allow for the continued use of economical and efficacious pesticide products that would otherwise be lost through cancellation.

As stated in Chapter 1, The NAS risk paradigm of chemical hazard, potency, exposure assessment, synthesized through risk characterization, are the fundamental processes of risk assessment. Toxicity is a function of hazard and potency (dose-response) and risk is a function of toxicity and exposure. Expressed more concisely, as follows:

$$\text{Risk } f(\text{toxicity, exposure})$$

Profiles for herbicides and TGR commonly used in electric utility right-of-way operations are given in Chapter 3. These profiles include qualitative and quantitative information that can be used to evaluate human health risks associated with exposure in drinking water and the potential for ground water contamination; with the assumption that all ground water is a potential drinking water source. From these profiles, toxicity of each chemical will be evaluated based on two quantitative attributes, the reference dose (RfD) and the cancer slope factor (Q1*). In the absence of these “risk numbers” qualitative information will be used, such as the cancer classification. U.S. EPA also uses these risk numbers to develop standards and guidelines for pesticides in drinking water.

Pesticides in drinking water are regulated under the Safe Drinking Water Act²⁹ passed by Congress in 1974 focuses on setting national drinking water standards with U.S. EPA acting as the primary enforcement agency. Drinking water standards limit the population’s exposure to hazardous substances through drinking water. Prior to 1980, U.S. EPA had established standards for 23 contaminants. Regulations specified maximum contaminant levels (MCLs) and sampling frequencies for each contaminant. The U.S. EPA Office of Water has established Health Advisories (HAs), which are non-enforceable guidelines for chemical residues in drinking water, for approximately 200 chemicals, including about 50 pesticides. These include 1-day and 10-day HAs for children and adults and lifetime HAs for adults. The HA is the concentration of a chemical in drinking water that is not expected to cause adverse non-carcinogenic effects over the period of exposure. U.S. EPA has also established HAs for some pesticides classified as carcinogens. In addition, as a part of rulemaking under the Safe Drinking Water Act, the agency has established the Maximum Contaminant Level (MCL) and Maximum Contaminant Level Goal (MCLG) for about 25 pesticides in drinking water. The MCL, an enforceable standard, is the maximum allowable level of a contaminant in water delivered by a public water system. The MCLG is the concentration of a drinking water contaminant that is thought to be protective of adverse human health effects. In almost all cases, the MCL and MCLG are the same. In a few instances, the MCLG has been set at zero. In determining the lifetime HAs and MCLGs, the estimated dose is based on a 70 Kg person consuming 2 liters per day. For non-carcinogens, lifetime HAs and MCLs are derived from the drinking water equivalent to the Reference Dose (RfD). For carcinogens, the health advisory is based on excess cancer risk associated with lifetime ingestion of drinking water. The target for the lifetime HA or MCLG is a concentration in drinking water that results in less than 1 in 10,000 (1×10^{-4}) excess lifetime cancer risk. In determining the risk of consuming contaminated drinking water, other routes of exposure, such as diet, are considered so that the risk associated with the total daily exposure from all sources does not exceed the drinking water equivalent to the RfD for non-carcinogens or the 1 in 1,000,000 (1×10^{-6}) excess lifetime cancer risk for carcinogens. In the absence of actual data, it is generally assumed that drinking water comprises 20% of all routes of exposure (Toccalino 2003, 2007).

The World Health Organization (WHO) also publishes drinking water quality guidelines that are intended to be used as a basis for the development of national standards. See WHO Guidelines for Drinking Water Quality, Volume 2 (1996).

²⁹ <http://www.epa.gov/safewater/sdwa/sdwa.html>

In the absence of a standard or guideline the HA can be estimated from the DWEL (Drinking Water Equivalence Level, U.S. EPA, 1989). Estimated HAs in Table 5-2 are designated HA*. The DWEL is determined from the RfD assuming a 70 Kg person consumes 2 liters per day for a lifetime. To determine the HA, the DWEL is adjusted assuming that drinking water comprises 20% of the allowable daily intake of a given chemical. For carcinogens the HA is estimated based on U.S. EPA Cancer Class: if the U.S. EPA Cancer Class is C: $HA^* = RfD \times 700$, if the U.S. EPA Cancer Class is D, E, or unclassified: $HA^* = RfD \times 7000$.

Table 5-2 lists the quantitative information useful in characterizing the risk of pesticide ground water contamination extracted from the profiles. This information includes the chemicals solubility in water at 20 °C, the soil aerobic degradation half-life in days, the Koc, the GUS leaching index, as well as the drinking water standard or guideline; when MCLs and U.S. EPA HAs are not available, Health Advisories are estimated and designated as HA*.

The chemical characteristics in Table 5-2, as well as additional characteristics given in the profiles were obtained from the Footprint Pesticide Properties Database. The Pesticide Properties Database (PPDB) is a comprehensive relational database of pesticide physicochemical, toxicological, ecotoxicological and other related data. The database has been developed by the Agriculture & Environment Research Unit (AERU) based at the University of Hertfordshire, UK. It has evolved from a database that originally accompanied the EMA (Environmental Management for Agriculture) software (also developed by AERU), with additional input from the EU-funded FOOTPRINT project (<http://www.eu-footprint.org>).

The GUS, or ground water ubiquity score (Gustafson, 1989), is an empirically derived value that relates pesticide persistence (half-life) and sorption in soil (Koc). The GUS may be used to rank pesticides for potential to move toward ground water. The rating accompanying the GUS value is derived from the GUS. GUS ratings, as well ratings for the other pesticide characteristics, are given in Table 5-1.

Pesticide Risk Characterization

Risk characterization by the pesticide user involves an evaluation of alternatives based on information that allows discrimination between choices. All of the chemicals listed in Table 5-2 are considered in common use for electrical utility rights-of-way vegetation management operations, and are so labeled. This means that products containing the chemicals have been subjected to Risk characterization by U.S. EPA during the registration process and the ongoing reregistration process. The potential for these chemicals to contaminate ground water, however, is ultimately site-specific. Carefully considering the chemical characteristics, toxicity, use practices and site conditions (i.e., landscape, soils, climate, ground water vulnerability) may significantly reduce the likelihood of ground water contamination that exceeds a level of concern.

For example, the chemicals in Table 5-2 can be ranked based on a number of characteristics, such as lowest to highest drinking water standard or guideline. The lower the number the less that can be in drinking water before a level of concern is exceeded. Trifluralin has by far the lowest drinking water health advisory – 5 ug/l or 5 parts per billion (ppb). However this herbicide also has one of the lowest GUS index values indicating that it has low leaching

potential. By contrast, clorpyralid has one of the highest GUS index values – indicating high leaching potential, but a relatively high drinking water guideline. Both of these herbicides may be used without concern for ground water contamination at sites that contain soils high in clay and organic matter and where the underlying ground water is otherwise not vulnerable (deep water table and not compromised by preferential flow paths such as karst formations). Climate that drives the hydrologic cycle should also be considered, as well as topography and land cover. Foliar application to sites with dense canopy cover will initially distribute the herbicide primarily to the foliage. Only with rainfall soon after application will significant amounts wash off and reach the soil. Herbicide degradation and volatile loss from foliar surfaces usually results in greater field dissipation. In addition, with time herbicides become incorporated into the epicuticular waxes of foliage and other plant surfaces. If the herbicide degrades or volatilizes before it can move to the soil, then the potential for ground water contamination has been greatly reduced.

Table 5-1
Rating Criteria for Pesticide Characteristics

Chemical characteristic	Rating Criteria
Solubility in water at 20 °C (mg/l)	≤ 50 = Low 50 - 500 = Moderate > 500 = High
Octanol-water partition coefficient at pH 7, 20 °C	< 2.7 = Low bioaccumulation 2.7 – 3 = Moderate > 3.0 = High
Vapor pressure at 25 °C (mPa)	$< 1 \times 10^{-6}$ = Non-volatile $1 \times 10^{-6} - 1 \times 10^{-5}$ = Intermediate state $> 1 \times 10^{-5}$ = Volatile
Henry's law constant at 20 °C (dimensionless)	$> 2.5 \times 10^{-5}$ = Volatile $2.5 \times 10^{-7} - 2.5 \times 10^{-5}$ = Moderate volatility $< 2.5 \times 10^{-7}$ = Non volatile
Soil degradation $\frac{1}{2}$ life (days) (aerobic)	< 30 = Non-persistent 30 - 100 = Moderately persistent 100 - 365 = Persistent > 365 = Very persistent
Koc - Organic-carbon sorption constant (ml/g)	< 15 = Very mobile 15 - 75 = Mobile 75 - 500 = Moderately mobile 500 - 4000 = Slightly mobile > 4000 = Non-mobile
GUS leaching potential index	> 2.8 = High leachability 2.8 - 1.8 = Moderate < 1.8 = Low leachability

Table 5-2

Chemical Characteristics and Drinking Water Standards and Guidelines for Herbicides and TGRs

Active Ingredient	Solubility in water at 20 °C (mg/l) ¹	Solubility Rating	Soil aerobic degradation ½ life (days)	½ Life Rating (persistence)	Koc (ml/g)	Koc Rating	GUS leaching potential index	GUS Rating	Drinking Water Std/guideline (ug/l)
2,4-D	23180	High	10	Not	56	Mobile	2.25	Moderate	70 (MCL) ²
Aminopyralid	2480	High	35	Moderately	8	Very mobile	4.78	High	3500 (HA*) ³
Bromocil	815	High	60	Moderately	32	Mobile	4.44	High	70 (HA) ⁴
Clopyralid	143000	Very High	34	Moderately	5	Very mobile	5.06	High	3500 (HA*)
Diuron	35.6	Low	75.5	Moderately	1067	Slightly Mobile	1.83	Low	20 (HA)
Flurprimidol	114	Moderate	11	Not	185	Moderately mobile	1.80	Low	140 (HA*)
Fosamine Ammonium	2500000	Very High	8	Not	63	Mobile	1.99	Low	70 (HA*)
Glyphosate	10500	High	12	Not	21699	Non-mobile	-0.36	Low	700 (MCL)
Imazapic	2230	High	120	Persistent	137	Moderately mobile	3.87	High	3500 (HA)
Imazapyr	9740	High	11	Not	125	Moderately mobile	1.98	Low	17500 (HA*)
Isoxaben	0.93	Low	105	Persistent	601	Slightly mobile	2.47	Moderate	35 (HA)
Metsulfuron-methyl	2790	High	10	Not	39.5	Mobile	2.40	Moderate	1750 (HA)
Paclobutrazol	0.248	Low	112	Persistent	210	Moderately mobile	3.44	High	175 (HA)

Table 5-2 (continued)

Chemical Characteristics and Drinking Water Standards and Guidelines for Herbicides and TGRs

Active Ingredient	Solubility in water at 20 °C (mg/l) ¹	Solubility Rating	Soil aerobic degradation ½ life (days)	½ Life Rating (persistence)	Koc (ml/g)	Koc Rating	GUS leaching potential index	GUS Rating	Drinking Water Std/ guideline (ug/l)
Pendimethalin	0.33	Low	90	Moderately	15744	Non-mobile	-0.39	Low	70 (HA) ⁴
Picloram	560	High	90	Moderately	35	Mobile	4.80	High	500 (MCL)
Tebuthiuron	2500	High	400	Very	80	Moderately mobile	5.46	High	500 (HA)
Triclopyr	8100	High	39	Moderately	48	Mobile	3.69	High	350 (HA*)
Trifluralin	0.221	Low	181	Persistent	8765	Non-mobile	0.13	Low	10 (HA)

¹ Chemical characteristics are from the Footprint Pesticide Properties database.

² MCL: Maximum Contaminant Level; an enforceable drinking water standard.

³ HA* estimated from the DWEL: the Drinking Water Equivalent Level (DWEL) is based on the RfD and assumes a 70 Kg person drinks 2 liters per day over a lifetime. The HA* is determined from the DWEL by assuming that drinking water comprises 20% of the allowable daily intake. If the U.S. EPA Cancer Class is C: HA* = RfD x 700, if the U.S. EPA Cancer Class is D, E, or unclassified: HA* = RfD x 7000. ³HA: Health Advisory, a non-enforceable drinking water guideline for lifetime exposure.

⁴ World Health Organization drinking water guideline for pendimethalin is 20 ug/l.

These are just 2 examples of how the data in Table 5-2 can be used to aid in decision-making regarding vegetation control strategies that consider the potential affect on ground water resources. A broader approach is discussed under risk mitigation measures.

Pesticide Risk Reduction Strategies

Pesticide regulation in the U.S. is based on the premise that a registration process that includes extensive testing^{30,31} prior to approval can identify beneficial use practices that will not result in “unreasonable adverse effects on human health or the environment³²,” and consumption of food from crops treated with pesticides will result in “a reasonable certainty of no harm³³.” Tolerances are pesticide residue levels in food that are protective of human health. In addition to human toxicity, tolerances are based on the assumption that pesticides may be used on multiple crops and that there is an adequate understanding of dietary consumption patterns. Use practices that stipulate application rate, timing and frequency, as well as the number of crops that may be treated with a given pesticide, are designed to result in residue levels in each commodity, that when considering the total diet (including drinking water), are protective of human health. Pesticide product label language also addresses off-site movement (drift, airborne loss, runoff, leaching), as well as affects on wildlife, beneficial species, workers³⁴, and bystanders. To help insure that the provisions of the label are effectively administered, FIFRA and associated regulation requires that pesticide applicators receive training and continuing education. In addition, the use of certain pesticide products requires that the applicator be certified³⁵.

For an environmental protection strategy, that is largely invested in compliance with pesticide label instructions and use restrictions to be successful, it must also requires enforcement activities. In the U.S., this is largely delegated to the states with pass-through funds and oversight by U.S. EPA . State lead agencies (usually the state dU.S. EPA rtment of agriculture) determine which pesticide products that may be used within the state, oversee pesticide applicator certification and training, monitor pesticide product formulations, and investigate sales and use violations. Investigation of use violations, including off-site movement, may include monitoring (soil, water, air, biota sampling and analysis).

Over the last two decades, monitoring for pesticides in U.S. lakes, rivers, streams, and ground water has largely been the purview of the U.S. Geological Survey³⁶. The NAWQA Pesticide

³⁰ <http://www.epa.gov/opptsfrs/home/guidelin.htm>

³¹ http://www.access.gpo.gov/nara/cfr/waisidx_03/40cfrv21_03.html

³² FIFRA <http://www.epa.gov/oecaagct/lfra.html>

³³ FQPA <http://www.epa.gov/oecaagct/lqpa.html>

³⁴ <http://epa.gov/opp00001/health/worker.htm>

³⁵ <http://www.epa.gov/oppfead1/safety/applicators/applicators.htm>

³⁶ <http://water.usgs.gov/nawqa/pnsp/>

National Synthesis Project, which began in 1992, is a national-scale assessment of the occurrence and behavior of pesticides in streams and ground water of the U.S. and the potential for pesticides to adversely affect drinking-water supplies or aquatic ecosystems. Similar to the efforts of FDA and USDA related to food safety, USGS has invested significant resources in developing sampling and analysis methods for pesticides in ground and surface water. Because for many pesticides the benchmark concentrations in water protective of drinking water or aquatic life are extremely low, a major challenge has been the development of analytical methods with sufficient sensitivity to reliably detect pesticides and toxic degradates significantly below the benchmark concentrations. Screening a multitude of water samples for many pesticides also requires rapid multiresidue methods.

Monitoring pesticides in the food supply, off-site movement that may pose risks to humans and wildlife, and pesticide exposure in the workplace, aid in the ongoing evaluation of the risks associated with pesticide use practices. For optimal affect in societal risk decisions, monitoring data should be quality assured with transparent procedures that allow evaluation against predetermined data quality objectives.

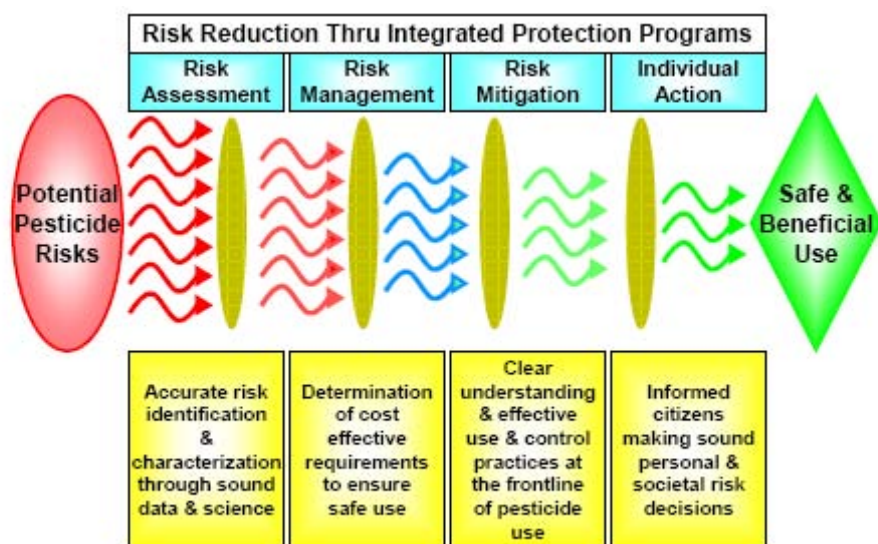


Figure 5-1
Continuum of Pesticide Risk Reduction Measures

Pesticide registration in the U.S. is a dynamic process, as new science and information on the affects of current use becomes available a pesticide product's registration status may be changed. This is accomplished through the U.S. EPA pesticide reregistration process. The FQPA requires periodic re-evaluation of pesticide registrations and tolerances to ensure that the scientific data supporting pesticide registrations will remain up to date in the future. The reregistration program (administered by U.S. EPA's Office of Pesticide Programs), while not a formal part of FQPA, is the critical mechanism used by U.S. EPA to implement tolerance reassessment. Whereby, reregistering food use pesticides means not only that U.S. EPA reassesses their tolerances but also that U.S. EPA evaluates the safety of those pesticides for workers and the environment. The reregistration process involves evaluating all risks to human health and the environment based on current science and risk assessment procedures, as well as data on adverse effects resulting from

product use³⁷. The results are published in a Reregistration Eligibility Decision (RED)³⁸, which describes the risk assessment process leading to a decision whether or not to reregister products containing a pesticide active ingredient. If the decision is to reregister, often there will be restrictions on how the pesticide can be used in order to mitigate risk. As described above, these restrictions are communicated to the user on the pesticide label. For example, if the concern is for worker exposure then the label may stipulate re-entry intervals, protective clothing requirements, or engineering controls (i.e., closed cab, closed mixing-loading systems). If the concern is food residues, then application rates and frequency, and a pre-harvest interval may be stipulated. If the concern is water quality restrictions may include buffer zones³⁹. Mitigation measures under FIFRA usually apply nationally (with some exceptions). However, the consequences of each pesticide use is site-specific; ultimately, a pesticide's risk to human health and the environment is determined by the user.

Although pesticide risk reduction in the U.S. is “front loaded”, i.e., relies heavily on label restrictions and the infrastructure at the federal, state and local levels (certification and training, education, enforcement, monitoring, and local assistance) to implement these restrictions, ultimately some pesticide risk management is “back loaded”, designed to manage or mitigate risk through efforts focused on individual actions at the frontline of pesticide use. Optimally, these approaches form the bookends of a series of integrated risk reduction programs (see Figure 5-1)⁴⁰.

Federal⁴¹ and state programs that assist pesticide applicators using pesticides in balancing agricultural production and environmental protection include the following:

- a. USDA Agricultural Research Service;
- b. USDA Natural Resources Conservation Service;
- c. USDA Soil and Water Conservation Districts;
- d. U.S. EPA Regional offices;
- e. State departments of agriculture and other state lead agencies;
- f. Land grant universities - Ag experiment stations, and Extension; and
- g. The National Pesticide Information Center (a cooperative agreement between Oregon State University and the U.S. EPA Office of Pesticide Programs).

³⁷ In addition to new information in the open literature, Section 6(a)(2) of FIFRA requires pesticide product registrants to submit adverse effects information about their products to the U.S. EPA

(<http://www.epa.gov/opppmsd1/fifra6a2/>).

³⁸ <http://www.epa.gov/pesticides/regulating/registering>

³⁹ The RED may also require additional studies by the registrant in order to complete the risk assessment. This may result in a conditional registration and a timetable for completion of the required studies. A list of the status of pesticide REDs can be found at <http://www.epa.gov/pesticides/reregistration/status.htm>.

⁴⁰ <http://www.epa.gov/pesticides/about/fieldprograms/fieldprograms.pdf>

⁴¹ <http://www.epa.gov/oecaagct/agctr.html>

Pesticide Risk Mitigation Measures

Risk mitigation begins with the detailed understanding of the application site, and as mentioned previously, some of the key information necessary to make an informed decision regarding the use of herbicide or TGR include the following

1. Herbicide or TGR chemical characteristics and drinking water level of concern as represented by the drinking water standard or guideline.
2. Soil characteristics and ground water vulnerability. Assistance in evaluating soils and ground water vulnerability is typically available from the Extension Service, NRCS, SWCD, and state agencies. Though the state NRCS offices assistance is available in identifying soils and also in using the Windows Pesticide Screening Tool (WIN-PST)⁴² which allows the determination of a leaching rating that considers both chemical characteristics and soil properties to give a leaching potential that is specific to that combination. WIN-PST also considers application method (soil surface, incorporated, or foliar), rainfall (humid vs arid regions), and site conditions, such as the presence of macropores or a perched water table. Table 5-3 shows an example WIN-PST outputs for 3 Massachusetts soils.
3. Consider application methods that reduce the potential for ground water contamination, to include the following:
 - a. Reduced rates.
 - b. Foliar applications.
 - c. Application timing that considers the rainfall forecast as to reduce the chance of rainfall soon after application and foliar wash-off.
 - d. Mixing and loading operations that contain all spillage and rinsate.
4. Develop an integrated vegetation management plan, which incorporates stated goals and objectives into a rational, comprehensive and practical program. The planning process should recognize the environmental requirements of desirable plants, as well as the potential adverse affects of altering the landscape. A properly planned and executed vegetation management program will use a variety of vegetation control techniques and strategies in an integrated fashion, dictated by economics, terrain, vegetation type, and mitigation of adverse affects on human and environmental health.
5. In developing vegetation management plans, consider a tiered approach to the use of decision aids in evaluating the potential for pesticide ground water contamination.

⁴² <http://www.wsi.nrcs.usda.gov/products/W2Q/pest/winpst.html>

Table 5-3
WIN-PST Soil/Pesticide Interaction Leaching Potential for Three Massachusetts Soils

Active Ingredient	Products	Application type	Soil degradation ½ life (days) ¹	Koc (ml/g)	Windsor Loamy Sand	Paxton Fine Sandy Loam	Canton Fine Sandy Loam
2,4-D	Pathway RTU	cut surface	10	20	Low	Very low	Very low
Aminopyralid	Milestone	low/high volume foliar	35	8	Low	Very low	Very low
Bromocil	Krovar DF	low/high volume foliar	60	32	High	Low	Intermediate
Clopyralid	Transline	low/high volume foliar	30	6	High	Low	Intermediate
Diuron	Karmex XP	low/high volume foliar	90	480	Intermediate	Low	Low
Fosamine Ammonium	Krenite S	low volume foliar	8	150	Low	Very low	Very low
Flurprimidol	Cutless	soil surface & incorporated	11	185	High	Intermediate	High
Glyphosate	Accord Aqua Neat Journey	low/high volume foliar	47	24000	Low	Very low	Very low
Imazapic	Journey	low/high volume foliar	232	137	High	Low	Intermediate
Imazapyr	Arsenal Chopper	low/high volume foliar basal/cut surface	90	100	High	Low	Intermediate
Isoxaben	Snapshot	low/high volume foliar	100	1400	Low	Very low	Very low
Metsulfuron-methyl	Escort	low/high volume foliar	120	35	High	Low	Intermediate
Paclobutrazol	Profile 2SC	basal/soil incorporated	200	400	Intermediate	Low	Low
Pendimethalin	Pendulum Aqua Cap	low/high volume foliar	90	5000	Low	Very low	Very low
Picloram	Tordon	low/high volume foliar	90	16	High	Low	Intermediate
Tebuthiuron	Spike 20P	high volume soil surface	360	80	High	Intermediate	High
Triclopyr BEE	Garlon 4 Tahoe 4e Pathfinder II	low/high volume foliar basal/cut surface	46	780	Intermediate	Low	Low
Triclopyr TEA	Garlon 3a	Low/high volume foliar	46	20	High	Low	Intermediate
Trifluralin	Snapshot	high volume soil surface	60	8000	Intermediate	Low	Low

¹Soil half-life and Koc values from the WIN-PST database; some values differ significantly from the Footprint Pesticide Properties database.

Tiered Approach to the Use of Pesticide Risk Mitigation Decision Aids

WIN-PST and the GUS index are examples of first tier screening tools. They require few inputs and therefore use conservative assumptions regarding leaching to ground water (i.e., error on the side of caution in assessing risks; see discussion in Exposure Models above). These tools, however, can be useful the initial evaluation of alternative control strategies. Often these tools are all that is needed in decision making.

Higher tier decision aids may include U.S. EPA models like GEENEC, SCI-GROW and AgDrift. These tools require more inputs, but still rely on conservative assumptions.

Third Tier models are those models that are run primarily in “research mode”. They require many inputs (often in the hundreds), can be site-specific, resource intensive, but provide more realistic output. U.S. EPA uses a combination of PRZM-EXAMS and AgDrift models to do site specific evaluation of pesticide ground and surface water contamination. The USDA Forest Service and the DOI Bureau of Land Management use GLEAMS and AgDrift to assess edge of field/site pesticide losses to surface and ground water. In the private sector, however, the use of these models as decision aids in vegetation management is not common, and requires significant in-house expertise or the services of consultants. In addition, there is increasing interest in watershed scale models that can interface with Geographic Information System (GIS) data sets.

Use of the Soil and Water Assessment Tool in Evaluating Alternative IVM Practices

Tier 3 Environmental fate models have become useful tools in evaluating both pesticide point and nonpoint source pollution at large scales. One such model is the Soil and Water Assessment Tool (SWAT)⁴³, developed by the USDA Agricultural Research Service. SWAT is based on over 30 years of modeling expertise. SWAT is a watershed scale hydrologic model that was developed to predict the affects of land management on water, sediment and chemical yields in large, variable basins. SWAT uses physical characteristics of the landscape including land use/land cover, soil types and topography along with weather data and physical chemical properties of compounds to perform mathematical simulations of the processes that dictate routing of water, chemicals and sediment. SWAT operates on a daily time step and can perform simulations over long periods of time (Arnold et al 1998, Neitsch et al. 2005).

SWAT has been utilized in a variety of applications worldwide as can be seen in the extensive review of model applications by Gassman et al. 2007. In the United States, SWAT has been accepted as a useful tool in assessing nonpoint source pollution. SWAT has been incorporated in the BASINS model developed by the US U.S. EPA as a tool to aide in the development of total maximum daily loads (Di Luzio 2002). SWAT is also one of two models that are utilized for modeling applications in the USDA Conservation Effects Assessment Program (CEAP). Within CEAP, SWAT is considered the preferred model due to its ability to provide more accurate simulations without any calibration of the model parameters (Heathman et al. 2008). The majority of the SWAT applications deal with hydrologic simulations as well as sediment and

⁴³ <http://www.brc.tamus.edu/swat/>

nutrient loadings, while other applications include the evaluation of land use/ land cover changes, evaluations of mitigation options and potential impacts of climate change. SWAT has also been utilized to evaluate the fate of pesticides at the basin scale. SWAT has been used in the St. Joseph River watershed and the Cedar Creek watershed within the St. Joseph River watershed to evaluate nonpoint source pollution due to the use of the herbicide atrazine (Larose et al. 2007 and Vazquez-Amabile 2006). Model outputs for the St. Joseph River watershed were also used to perform a risk analysis following the National Agricultural Pesticide Risk Analysis framework (Vazquez-Amabile et al. 2006). SWAT has also been utilized to evaluate nonpoint source pollution due to pesticide use in European watersheds (Holveot et al. 2008 and Gevaert et al. 2008). SWAT has also been used to evaluate the impact of implementation of beneficial management practices on water quality (Gassman et al. 2007, Bärlund et al. 2007, Gevaert et al. 2008, Bracmort et al. 2006, Santhi et al. 2006 and Holveot et al. 2007).

One of the advantages of SWAT is the GIS interface that has been developed to manage required input data (Di Luzio et al. 2004, Srinivasan et al. 1998). As SWAT was developed to simulate large watersheds with variable features, geospatial methods are useful for gathering, managing and analyzing watershed data. The GIS interface populates the SWAT inputs that are required to describe the physical characteristics of the watershed. The GIS interface is also beneficial because it affords the user the opportunity to create a static spatial accounting of pesticide use. This static accounting of pesticide use can be used in relation to landscape characteristics to identify areas within a watershed that could contribute to the impairment of water quality due to pesticide use. This information can be used to focus modeling and monitoring efforts or to focus educational outreach efforts to pesticide users within the areas identified.

Environmental models such as SWAT go a step beyond the static account of landscape characteristics and chemical use to simulate the potential movement of chemicals within a watershed. SWAT couples the spatial and temporal chemical use data with the hydrology of the watershed to simulate chemical movement in the system. SWAT uses meteorologic data to drive the hydrologic cycle of the watershed. The movement of water in the system is the vehicle of chemical movement in the model. The primary routes for chemicals to enter water from the site of application within SWAT are through surface runoff and infiltration of applied chemicals into ground water that can reach surface waters through lateral flow and recharge. The model simulates mechanisms of chemical loss and degradation in the terrestrial environment as well as in the aquatic environment during the routing of the water through watershed. Coupling the movement of chemical via the routing of water through the watershed and simulating the routes of chemical loss, the model can provide estimated concentrations of chemicals in the waters of the system. This can be used to predict the concentrations of chemicals at certain points within the watershed. Another useful output of SWAT is the exceedance probability, or the probability that the concentration of a chemical in the surface waters will exceed a given level during a period of time. As SWAT simulations are based on decades of meteorological data, it takes into account weather patterns within the study area. Based on this data, the model calculates the probability that the chemical concentration at given points within the watershed will exceed a defined level. These calculations can be used to determine the potential for current chemical use practices to exceed levels defined by regulatory agencies. This information can be useful to producers and their advisors in developing monitoring and mitigation plans. This type of simulation can also be used to demonstrate to chemical users within the watershed the hydrologic connectivity of the system and how chemical use throughout the system can contribute to the chemical load.

SWAT can also be used to simulate how changes in the watershed could affect water quality. Changes in the landscape of the watershed, pest management strategies or the implementation of beneficial management practices can alter the chemical loading to the system. SWAT input data, model parameters and/or model processes can be changed in order to simulate changes in land cover/land use, changes in pest management or the implementation of beneficial management practices. This approach can be used to identify IPM and other beneficial management practices that have the potential to reduce pesticide loading. In addition, SWAT simulations can be used to identify areas within the watershed where the implementation of mitigation measures could have the greatest affect on the reduction of chemical loading. Simulations can be used to demonstrate to pesticide users potential practices or measures that can be implemented and aide chemical users in their decision making process. It can also be used to demonstrate to chemical users not only steps that can be taken by individual users but also how implementation throughout the entire watershed can reduce pesticide impacts on water quality. The exceedance probabilities calculated by the model can be useful in this type of exercise as well. In this case the exceedance probability can be used to demonstrate how changes in practices can reduce the probability that chemical concentrations can be reduced based on local weather patterns.

The use of models like SWAT requires significant resources and may only be useful to the evaluation of herbicide and TGR use along electric utility rights-of-way when there is concern for watershed scale impacts, or when it is known that the ground water the underlies that application site, or ground water under areas that may receive surface water from the application site, are particularly vulnerable. A resource intensive analysis using a model such as SWAT may also be warranted if the vulnerable ground water resource of concern is used for drinking water.

Challenges to Characterizing Pesticide Risks to Ground Water Resources

Today there is still much uncertainty regarding ground water vulnerability to chemical contamination. For example, while USDA's Natural Resources Conservation Services has evaluated and cataloged soil properties in the root zone, there is no comprehensive understanding of soils in the intermediate layers (vadose zone) between the root zone and ground water. In addition, while pesticide dissipation in the root zone has been characterized, there is still little information available on pesticide degradation processes (pathways and rates) in the vadose zone and ground water. This lack of information requires that decision aids remain overly conservative in their predictions.

Improvements in information and decision aids available to stakeholders, including vegetation managers, are needed in the following areas:

- Pesticide environmental fate models do not adequately consider water transport, and pesticide transport in water, in geologic zones below the root zone. There is a need to better understand the circumstances in which the properties of the intermediate vadose zone are critical to ground water vulnerability assessments and incorporate in to modeling efforts.
- Pesticide environmental fate models do not adequately consider the residence time of water along flow paths that describe ground water recharge and discharge.
- Current models do not adequately address soil macropores and other preferential flow pathways that can affect vulnerability.

- More comprehensive monitoring data for pesticides in ground water is needed to validate decision aids, and improve calibration and evaluation of environmental fate models used to assess ground water vulnerability.
- Pesticide characteristic databases are currently the source of much uncertainty in ground water vulnerability assessments, as available data are too general and often conflicting. For example, the current use of constants for physical-chemical properties should be replaced with functions that can respond to varying environmental conditions.
- USDA's Soil Conservation Service soil sampling scheme should include better methods for characterizing soil organic matter, a key soil component in estimating pesticide sorption and movement with soil water.
- There is need to develop methods for merging data obtained at different spatial and temporal scales into a common scale for vulnerability assessment, i.e., GIS-based models. This may be accomplished with improved analytical tools in GIS software to facilitate integration of assessment methods with spatial attribute databases and the computing environment.
- There is a need for decision aids that provide more meaningful categories of ground water vulnerability for assessment. This will only be accomplished when the most important processes are identified and incorporated into vulnerability assessments at different spatial scales across the landscape.
- Current drinking water standards and guidelines are inadequate for the proper evaluation of the potential for adverse impacts on human health of pesticides in ground water that may be used for drinking water.

6

REFERENCES CITED

Akesson, N.B. 1990. Drift evaluation field tests. In Evaluation of drift exposure and environmental fate of pesticide application, report prU.S. EPA red in compliance with drift contract WM175 between the Florida dU.S. EPA rtment of Environmental Regulation and the University of Florida, Gainesville.

Ames PL. 1996. DDT residues in the eggs of osprey in the northeastern United States and their relation to nesting success. *J Appl Ecol (Supp)*. 3:87-97.

Arnold, J.G.; Srinivasan, R.; Muttiah, R.S. and J.R. Williams. **1998**. Large Area Hydrologic Modeling and Assessment Part I: Model Development. *Journal of the American Water Resources Association*, **34** (1): 73-89.

Bärlund, I.; Krikkala, T.; Malve, O. and J. Kämäri. **2007**. Assessing SWAT Model Performance in the Evaluation of Management Actions for the Implementation of the Water Framework Directive in a Finnish Catchement. *Environmental Modelling & Software*, **22**: 719-724.

Bracmort, K.S.; Arabi, M.; Frankenberger, J.R.; Engel, B.A. and J.G. Arnold. **2006**. Modeling Long-Term Water Quality Impact of Structural BMPs. *Transactions of the ASABE*, **49**(2): 367-374.

Bramble, W.C., W.R. Byrnes, R.J. Hutnick, and S.A. Liscinsky. 1991. Prediction of cover type on rights-of-way after maintenance treatments. *J. Arboric*. 17:38-43.

Burlington H and Lindeman VF. 1950. Effect of DDT on testes and secondary sex characteristics of white leghorn cockerels. *Proc Soc for Exp Bio and Med*. 74: 48-51.

Burns, L.A. March 1997. Exposure Analysis Modeling System (EXAMSII) Users Guide for Version 2.97.5, Environmental Research Laboratory, Office of Research and Development, U.S. Environmental Protection Agency, Athens, GA.

California DU.S. EPA rtment of Pesticide Regulation. 2003. Sampling for Pesticide Residues in California Well Water, 2003 Well Inventory Database, Cumulative Report 1986-2003. Eighteenth Annual Report to the Legislature, DU.S. EPA rtment of Health Services, Office of Environmental Health Hazard Assessment, and State Water Resources Control Board. 168 p.

California Environmental Protection Agency DU.S. EPA rtment of Pesticide Regulation Medical Toxicology Branch. 1986. Summary of Toxicology Data Fosamine. 7 p.

References Cited

California Environmental Protection Agency DU.S. EPA rtment of Pesticide Regulation Medical Toxicology Branch. 1991. Summary of Toxicology Data areoxaben. 7 p.

California Environmental Protection Agency DU.S. EPA rtment of Pesticide Regulation Medical Toxicology Branch. 1995. Summary of Toxicology Data Trifluralin. 17 p.

California Environmental Protection Agency DU.S. EPA rtment of Pesticide Regulation Medical Toxicology Branch. 1997. Summary of Toxicology Data Diruon. 7 p.

California Environmental Protection Agency DU.S. EPA rtment of Pesticide Regulation Medical Toxicology Branch. 1997. Summary of Toxicology Data Pendimethalin. 17 p.

California Environmental Protection Agency DU.S. EPA rtment of Pesticide Regulation Medical Toxicology Branch. 1999. Summary of Toxicology Data Picloram, Potassium Salt. 10 p.

California Environmental Protection Agency DU.S. EPA rtment of Pesticide Regulation Medical Toxicology Branch. 2000. Summary of Toxicology Data Tebuthiuron. 9 p.

California Environmental Protection Agency DU.S. EPA rtment of Pesticide Regulation Medical Toxicology Branch. 2000. Summary of Toxicology Data Triclopyr. 12 p.

California Environmental Protection Agency DU.S. EPA rtment of Pesticide Regulation Medical Toxicology Branch. 2005. Summary of Toxicology Data Aminopyralid. 11 p.

California Environmental Protection Agency DU.S. EPA rtment of Pesticide Regulation Medical Toxicology Branch. 2005. Summary of Toxicology Data Imazapic. 7 p.

California Environmental Protection Agency DU.S. EPA rtment of Pesticide Regulation Medical Toxicology Branch. 2008. Summary of Toxicology Data Clorpyralid. 9 p.

Carsel, R.F., C.N. Smith, L.A. Mulkey, J.D. Dean and P. Jowise. 1984. Users manual for pesticide root zone model (PRZM): Release 1, Rep. U.S. EPA -600/3-84-109, 219 pp. Environmental Research Laboratory, Office of Research and Development, U.S. Environmental Protection Agency, Athens, GA.

Di Luzio, M.; Srinivasan, R. and J.G. Arnold. **2004**. A GIS-Coupled Hydrologic Model System for Watershed Assessment of Agricultural Nonpoint and Point Sources of Pollution. *Transactions in GIS*, **8(1)**: 113-136.

Di Luzio, M.; Srinivasan, R. and J.G. Arnold. **2002**. Integration of Watershed Tools and SWAT Model Into BASINS. *Journal of the American Water Resources Association*, **38(4)**: 1127-1141.

Diruon, pesticide tolerance, Federal Register: June 13, 2007 (Volume 72, Number 113)

Effland, W.R., Thurman, N.C., Kennedy, I. Proposed Methods For Determining Watershed-Derived Percent Cropped Areas and Considerations for Applying Crop Area Adjustments To

Surface Water Screening Models; USU.S. EPA Office of Pesticide Programs; Presentation To FIFRA Science Advisory Panel, May 27, 1999.

EPRI Report No. 1005367. 2004. Human Health Risk Assessment of Chemicals Encountered in Vegetation Management on Electric Utility Rights-of-Way. 200 p.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/4.htm>), 2,4-D.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/29.htm>), aminopyralid.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/88.htm>), bromocil.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/169.htm>), clorpyralid.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/260.htm>), diruon.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/348.htm>), flurprimidol.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/362.htm>), Fosamine ammonium.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/373.htm>), glyphosate.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/1152.htm>), imazapic.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/393.htm>), imazapyr.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/411.htm>), isoxaben.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/470.htm>), metsulfuron-methyl.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/333.htm>), paclobutrazol.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/511.htm>), pendimethalin.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/525.htm>), picloram.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/614.htm>), tebuthiuron.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/614.htm>), triclopyr.

Footprint Pesticide Properties Database (<http://sitem.herts.ac.uk/aeru/iupac/Reports/667.htm>), trifluralin.

Garner, W.Y., R.C. Honeycutt, H.N. Nigg. 1986. Evaluation of Pesticides in Ground water. American Chemical Society Symposium Series 315. 573 p.

Gassman, P.W.; Reyes, M.R.; Green, C.H. and J.G. Arnold. **2007**. The Soil and Water Assessment Tool: Historical Development, Applications, and Future Research Directions. *Transactions of the ASABE*, **50**(4): 1211-1250.

Gevaert, V.; van Griensven, A.; Holveot, K.; Seuntjens, P. and P.A. Vanrolleghem. **2008**. SWAT Developments and Recommendations for Modelling Agricultural Pesticide Mitigation Measures in River Basins. *Hydrologic Sciences*, **53**(5): 1075-1089.

Gustafson, D.I. (1989) Ground water Ubiquity Score: A Simple Method for Assessing Pesticide Leachability Environmental Toxicology and Chemistry, **8**, pp339-357).

Heathman, G.C.; Flanagan, D.C.; Larose, M. and B.W. Zuercher. **2008**. Application of the Soil and Water Assessment Tool and Annualized Agricultural Non-Point Source Models in the St. Joseph River Watershed. *Journal of Soil and Water Conservation*, **63**(6): 552-568.

Holveot, K.; Gavaert, V.; van Griensven, A.; Seuntjens, P. and P.A. Vanrolleghem. **2007**. Modelling the Effectiveness of Agricultural Measures to Reduce the Amount of Pesticides Entering Surface Waters. *Water Resource Management*, **21**: 2027-2035.

Holveot, K.; van Griensen, A.; Gevaert, V.; Seuntjens, P. and P.A. Vanrollechem. **2008**. Modifications to the SWAT Code for Modelling Direct Pesticide Losses. *Environmental Modelling & Software*, **23**: 72-81.

Jaynes DB, Hatfield JL and Meek DW, Water quality in Walnut Creek watershed: herbicides and nitrate in surface water. *J Environ Qual* **28**:45-59 (1999).

Jury, W. A., & Horton, R. (2004). *Soil physics* (p. 370). John Wiley and Sons.

Keith JA. 1966. Reproduction in a population of herring gulls (*Larus argentatus*) contaminated by DDT. *J Appl Ecol* (Supp). 3:57-70.

Krimsky S. 2001. An epistemological inquiry into the endocrine disruptor thesis. *Annals of the New York Academy of Sciences*. 948:130-42.

Larose, M.; Heathman, G.C.; Norton, L.D. and B. Engel. **2007**. Hydrologic and Atrazine Simulation of the Cedar Creek Watershed Using the SWAT Model. *Journal of Environmental Quality*, **36**: 521-531.

Leonard, R.A., W.G. Knisel and D.A. Still. 1989. GLEAMS : Ground water loading effects of agricultural management systems., *Transactions American Society of Agricultural Engineering*. 30: 1403-1418.

Melnick R, et al. 2002. Summary of the National Toxicology Program's report of the endocrine disruptors low dose peer review. *Envir Health Persp*. 110(4): 427-431.

Nash, R. G. 1988. Dissipation from soil. In: *Environmental Chemistry of Herbicides Volume 1*, R. Grover. Ed. CRC Press. P. 131-169.

National Research Council's *Risk Assessment in the Federal Government: Managing the Process*, 1983. National Academy Press 2101 Constitution Avenue, NW Washington, DC 20418.

National Research Council.1984. Geophysics Study Committee. II. Series. Ground water Contamination. National Academy Press 2101 Constitution Avenue, NW Washington, DC 20418. 192 p.

National Research Council. 1987. Regulating Pesticides in Food: The Delaney Paradox. National Academy Press 2101 Constitution Avenue, NW Washington, DC 20418. 272 p.

National Research Council. 1993. Ground Water Vulnerability Assessment: Predicting Relative Contamination Potential Under Conditions of Uncertainty Committee for Assessing Ground Water Vulnerability, National Academy Press 2101 Constitution Avenue, NW Washington, DC 20418.224 p.

National Research Council. 1994. Science and Judgment in Risk Assessment Committee on Risk Assessment of Hazardous Air Pollutants Board on Environmental Studies and Toxicology Commission on Life Science, National Academy Press 2101 Constitution Avenue, NW Washington, DC 20418.651 p. s

National Research Council. 2006. Toxicity Testing for Assessment of Environmental Agents: Interim Report Committee on Toxicity Testing and Assessment of Environmental Agents, National Academy Press 2101 Constitution Avenue, NW Washington, DC 20418. 270 p.

National Research Council. 2009. Science and Decisions: Advancing Risk Assessment Committee on Improving Risk Analysis Approaches Used by the U.S. U.S. EPA , National Academy Press 2101 Constitution Avenue, NW Washington, DC 20418. 424 p.

References Cited

National Marine Fisheries Service Endangered Species Act Chapter 7 Consultation Biological Opinion Environmental Protection Agency Registration of Pesticides Containing Chlorpyrifos, Diazinon, and Malathion. 2008. http://www.nmfs.noaa.gov/pr/pdfs/pesticide_biop.pdf

National Marine Fisheries Service Endangered Species Act Chapter 7 Consultation Biological Opinion Environmental Protection Agency Registration of Pesticides Containing Carbaryl, Carbofuran, and Methomyl. 2009. <http://www.nmfs.noaa.gov/pr/pdfs/carbamate.pdf>

Neitsch, S.L.; Arnold, J.G.; Kiniry, J.R. and J.R. Williams. **2005**. Soil and Water Assessment Tool Theoretical Documentation Version 2005.

Niering, W.A., and R. Goodwin. 1974. Creation of relatively stable shrublands with herbicides: Arresting "succession" on rights-of-way and pastureland. *Ecology* 55:784-795.

New York State D.U.S. EPA rtment of Environmental Conservation. 2007. Registration of a Major Change in Labeled (MCL) Use Pattern for the Active Ingredient Flurprimidol Contained in the Pesticide Product Topflor Ornamental Plant Growth Regulator (U.S. EPA Reg. No. 67690-20). 7 p.

Santhi, C.; Arnold, J.G.; Williams, J.R.; Hauck, L.M. and W.A. Dugas. **2001**. Application of a Watershed Model to Evaluate Management Effects on Point and Nonpoint Source Pollution. *Transactions of the ASAE*, **44**(6): 1559-1570.

Southwick LM, Willis GH, Mercado OA and Bengston RL, Effect of subsurface drains on runoff losses of metolachlor and trifluralin from the Mississippi River alluvial soil. *Arch Environ Contam Toxicol* **32**:106–109 (1997).

Srinivasan, R.; Ramanarayanan, T.S.; Arnold, J.G. and S.T. Bednarz. **1998**. Large Area Hydrologic Modeling and Assessment Part II: Model Application. *Journal of the American Water Resources Association*, **34**(1): 91-101.

Sulak, J.A. and J.J. Kielbaso. 2000. Vegetation Management along Transmission Utility Lines in the United States and Canada, *J Arboriculture* 26(4) 198-205.

Toccalino, P.L., Nowell, L.H., Wilber, W., Zogorski, J.S., Donohue, J., Eiden, C., Krietzman, S., and Post, G., 2003, Development of health-based screening levels for use in state- or local-scale water-quality assessments: U.S. Geological Survey Water-Resources Investigations Report 03-4054, 22 p.

Toccalino, P.L., 2007, Development and application of health-based screening levels for use in water-quality assessments: U.S. Geological Survey Scientific Investigations Report 2007-5106, 12 p.

Vazquez-Amabile, G.; B.A. Engel and D.C. Flanagan. **2006**. Modeling and Risk Analysis of Nonpoint-Source Pollution Caused by Atrazine Using SWAT. *Transactions of the ASABE*, **49**(3): 667-678.

USDA Forest Service. 1997. Final Environmental Impact Statement. Vegetation Management On Electric Utility Rights-Of-Way. May 1997. Allegheny National Forest. U.S. Department of Agriculture, Forest Service. Warren, PA.

USDA Forest Service. 2003. Imazapic - Human Health and Ecological Risk Assessment – Final Report. 110 p.

USDA Forest Service 2003. Picloram - Revised Human Health and Ecological Risk Assessment – Final Report. 133 p.

USDA Forest Service 2003. Triclopyr - Revised Human Health and Ecological Risk Assessment – Final Report. 133 p.

USDA Forest Service. 2004. Imazapyr - Human Health and Ecological Risk Assessment – Final Report. 149 p.

USDA Forest Service. 2004. Clopyralid - Human Health and Ecological Risk Assessment - Final Report. 154 p.

USDA Forest Service. 2004. Metsulfuron Methyl - Human Health and Ecological Risk Assessment – Final Report. 152 p.

U.S. Environmental Protection Agency. 2006. Glyphosate; Pesticide Tolerance. Federal Register: December 20, 2006 (Volume 71, Number 244)

USDA Forest Service. 2006. 2,4-D Human Health and Ecological Risk Assessment Final Report. 245 p.

USDA Forest Service. 2007. Aminopyralid Human Health and Ecological Risk Assessment – FINAL REPORT. 231 p.

USDA Forest Service. 2007. Allegheny National Forest Final Environmental Impact Statement Appendix G1 - Human Health Risk Assessment for Glyphosate and Sulfometuron Methyl. 334 p.

U.S. Forest Service. 2003. Glyphosate - Human Health and Ecological Risk Assessment Final Report. 281 p.

U.S. Department of Energy Bonneville Power Administration. Paclobutrazol Herbicide Fact Sheet. 8 p.

U.S. Environmental Protection Agency. 1985. Paclobutrazol (Clipper 50 WP) Herbicide Profile. 3 p. <http://pmep.cce.cornell.edu/profiles/index.html>

U.S. Environmental Protection Agency. 1989. Flurprimidol (Cutless) U.S. EPA Pesticide Fact Sheet. 7 p. <http://pmep.cce.cornell.edu/profiles/index.html>

References Cited

U.S. Environmental Protection Agency . 1993. Reregistration Eligibility Decision (RED) Glyphosate. 281 p.

U.S. Environmental Protection Agency. 1994. Reregistration Eligibility Decision (RED) Tebuthiuron. 232 p

U.S. Environmental Protection Agency. 1994. Reregistration Eligibility Decision (RED) Triclopyr. 285 p.

U.S. Environmental Protection Agency. 1995. Reregistration Eligibility Decision (RED) Fosamine ammonium. 214 p.

U.S. Environmental Protection Agency. 1996. Reregistration Eligibility Decision for Bromocil. 320 p.

U.S. Environmental Protection Agency. 1996. Reregistration Eligibility Decision (RED) Triclopyr. 285 p.

U.S. Environmental Protection Agency. 1997. Reregistration Eligibility Decision (RED) Pendimethalin. 239 p.

U.S. Environmental Protection Agency. 1997. Reregistration Eligibility Decision (RED) Picloram. 301 p.

U.S. Environmental Protection Agency. 2003. Reregistration Eligibility Decision (RED) for Diuron. 210 p.

U.S. Environmental Protection Agency. 2005. Reregistration Eligibility Decision for 2,4-D. 320 p.

U.S. Environmental Protection Agency. 2006. Reregistration Eligibility Decision for Imazapyr. 107 p.

U.S. Environmental Protection Agency. 2007. Isoxaben Summary Document Registration Review Docket December 2007. www.regulations.gov Docket Number: U.S. EPA -HQ-OPP-2007-1038.

U.S. Environmental Protection Agency. 2007. Paclobutazol Summary Document Registration Review: Initial Docket March 2007 Case Number 7002. www.regulations.gov Docket Number U.S. EPA -HQ-U.S. EPA -2006-0109. 34 p.

U.S. Environmental Protection Agency. 2009. Problem Formulation for the Ecological Risk and Drinking Water Exposure Assessments in Support of the Registration Review of Flurprimidol. 26 p.

U.S. Environmental Protection Agency. 2009. Flurprimidol. Human Health Assessment Scoping Document in Support of Registration Review. 25 p.

U. S. Geological Survey. 2005. Pesticides in the Nation's Streams and Ground Water, 1992–2001, <http://pubs.usgs.gov/circ/2005/1291/pdf/circ1291.pdf>

Wauchope RD, Pesticides in runoff: measurement, modeling, and mitigation. *J Environ Sci Health B31*:337–344 (1996).

Wurster CF and Wingate DB. 1968. DDT residues and declining reproduction in the Bermuda petrel. *Science* 159(3818): 979-981.

A

GLOSSARY OF TERMS

Acute toxicity – is the quality or potential of a substance to cause injury or illness from a single dose or short period of exposure. See subchronic and chronic.

Adjuvant –Any additive to a pesticide formulation that is not intended to be biologically active, but is intended to improve product performance or storage stability.

a.e. – Abbreviation for the acid equivalent of a pesticide active ingredient.

a.i. – Abbreviation for a pesticide active ingredient.

aRfD – acronym for the Reference Dose based on an acute NOAEL.

Cancer – A malignant growth of potentially unlimited size that invades local tissues, and MAY spread to other parts of the body.

Carcinogen – A chemical capable of inducing cancer.

Carcinogenic – Capable of causing cancer.

Chronic toxicity – (Long-term toxicity)-Chronic toxicity is the quality or potential of a substance to cause injury or illness after repeated exposure for a long period of time. Chronic toxicity tests run for a year or more; for rodents the period may extend through the entire life span. A chronic effect persists for months or years and may arise from acute or long-term exposure. See acute and subchronic.

Contaminant – In a formulation, usually residues or impurities from the manufacturing process present in small quantities. Pesticide registrants are required to identify contaminants that may be of toxicological concern to the regulatory agency.

cRfD – acronym for the Reference Dose based on a chronic NOAEL.

CFR – acronym for the Code of Federal Regulations

Degradation –Breakdown of a compound by physical, chemical or biochemical processes into basic components with properties different from those of the original compound.

Dose – is the amount of a chemical that enters the body (sometimes called the internal dose) to be distributed to all of the organs and cells. Distribution to tissues and cells is selective, and depends on the nature of the chemical and characteristics of each kind of cell.

Dose -response relationship – When evaluating the toxicity of a chemical or other stressor, the test animal is given increasing doses. If lethality is the toxic effect of interests, optimal doses will range from no effect to 100% lethality. In toxicity testing to determine dose-response, the default assumption is: as the dose (or concentration) of a chemical increases, the effect increases, and as the dose is lowered, the effect becomes less.

Enzymes – Complex proteins that catalyze (expedite) biochemical reactions. See Metabolism.

Epidemiology – is the scientific study of the cause, distribution, and control of epidemics or other disease in a region. In the context of these reports, epidemiology is the study of possible associations between environmental and occupational chemicals and occurrence of diseases. The term “associations” is used in its statistical sense, which means that the relationship cannot demonstrate cause and effect.

Evapotranspiration – is a term used to describe the sum of evaporation and plant transpiration of water from the Earth's land surface to atmosphere.

Exposure – is the amount of a chemical that reaches a surface from which it might be absorbed. The dose that is internalized is usually some fraction of the exposure. The exposed dose is only the material that reaches the skin (by contact), respiratory tract (by inhalation) or digestive tract (by ingestion).

FIFRA – is the acronym for the Federal Insecticide, Fungicide, and Rodenticide Act that regulates the sale and use of pesticides in the U.S.

Formulation – A complete pesticide prU.S. EPA ration as sold by a manufacturer for practical use. It includes the active ingredient and any necessary adjuvants and solvents. For use, it may or may not require further dilution or mixing with other substances. Formulation can also be defined as the process used by manufacturers in prU.S. EPA ring a pesticide for practical use.

FQPA – is the acronym for the Food Quality Protection Act of 1996 which amends FIFRA and **FFDCA**, the Federal Food Drug and Cosmetic Act. FQPA establishes a “reasonable certainty of no harm” for pesticides that have food uses, including drinking water. Sensitive subpopulations, such as infants and children, must be evaluated sU.S. EPA rately with assessing dietary risk.

GIS – Acronym for Geographic Information System.

Half-life –The length of time required for disappearance of half of the material present in an organism or in environmental media, such as soil or water. The term “half-life” was originally used to describe radioactive decay.

Hazard – in risk assessment represents the kind of an adverse effect that a chemical can cause. Hazard assessment may define toxic effects such as cancer, liver disease, skin irritation, reproductive problems, or whole organism effects, such as lack of weight gain.

Herbicide – A chemical substance or cultured biological organism, used to kill or suppress the growth of plants.

Hormone – A substance secreted by specialized endocrine cells and transported by the blood stream throughout the body to regulate biochemical activity of other cells. Insulin and testosterone are hormones.

Inert ingredient – Any component of a formulation that is purposely added and does not have pesticidal activity. This includes solvents and adjuvants, not manufacturing impurities. Inert ingredients are also called “other ingredients” to emphasize that ingredients other than the active ingredient in a pesticide formulation can be of toxicological concern.

Intermontane – is a feature that lies between mountains. The term refers to plateaus and basins formed by geologic processes.

Lethal – Causing death.

LD₅₀ – Acronym for Median lethal dose; for example, the dose that is lethal to half of the test animals.

LOAEL – Acronym for lowest-observed-adverse-effect level.

Lowest-observed-adverse-effect level (LOAEL) –The lowest dose of a chemical that produces significant increases in frequency or severity of adverse effects in exposed subjects.

Malignant – Deadly or very injurious. As applied to cancer, invasive of local tissues and metastatic (migration of cancer cells to other tissues).

Margin of Exposure – (MOE) The difference between the estimated dose of a pesticide and the NOAEL. A MOS of 100 (estimated dose 100 fold less than the NOAEL) is usually considered to assure that no adverse effects will occur.

Median effective dose (ED₅₀) – The dose or dose rate that causes 50% of subjects to respond. The nature of response must be specified, i.e., sedation, elevated blood pressure, death. The ED₁₀ is the dose effective in 10% of animals.

Median lethal dose (LD₅₀) –The dose of a chemical, biological agent, or other substances that is lethal (causes death) to half the test animals.

Metabolism – is the sum total of the biochemical reactions that a chemical undergoes in an organism. The processes include biochemical (enzymatic) reactions in the cells of the body that convert nutrients to energy and structural materials of the body; reactions that change wastes so

they can be removed; and reactions that convert foreign substances, such as some pesticides to forms that can be excreted.

MOE – Acronym for margin of exposure.

Mutagenic – Capable of producing genetic changes.

Mutagens – Chemicals that are able to induce gene or chromosome damage that is stable and survives cell division to reach the next generation of cells. See mutation.

Mutation – **is genetic** change in DNA of a cell that can be transmitted to the next generation of cells. If in sperm or egg cells, a mutation may be transmitted to offspring. If in somatic (body) cells such as liver, muscle or other organs, a mutation may pass to daughter cells in the organ. The change may have an effect on cell function; beneficial, benign, or harmful.

NOAEL –Acronym for no-observed-adverse-effect level.

No-observed-adverse-effect level (NOAEL) – The highest dose of a chemical that produces no significant increases in frequency or severity of adverse effects in exposed subjects.

Oncogenic – Able to cause cancer.

Persistence –The duration of measurable concentrations of a pesticide in soil, foliage or other media. (See Half-life).

Pesticide – Any chemical (or biological product) intended to control or kill pests. Herbicides, tree growth regulators, insecticides, and fungicides are classified (and regulated) as pesticides.

Pharmacokinetic –Relating to the rate and pattern of the absorption, distribution, metabolism and elimination of chemicals in an animal.

Potency –the dose-response determines a chemical’s potency for a specific toxic effect; the lower the dose that can elicit a toxic effect, the greater the potency of the pesticide.

Q₁^{*} – acronym for the cancer slope factor used in determining the cancer risk associated with exposure to a specific dose of a chemical. The cancer slope factor is an estimate of the increased cancer risk from a lifetime exposure to an agent. This estimate, usually expressed in units of proportion (of a population) affected per unit exposure (e.g., mg/kg-day or ug/m³), is generally reserved for use in the low-dose region of the dose-response relationship. It is often the statistical upper bound on the potency and therefore the risk. “Upper bound” in this context is a plausible upper limit to the true probability. For example, the excess cancer risk for the general population associated with exposure to a pesticide might be expressed as having a cancer slope factor of $1 \times 10^{-6} (\text{mg/kg/day})^{-1}$. Assuming an average body weight of 70 kg, for daily exposure over a lifetime (70 years) the probability of cancer would increase by an additional “1 in 1 million” for each 70 mg dose. This is sometimes called the “unit risk”.

RfD –Acronym for reference dose.

Reference dose (RfD) – is “an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.”

Registration – of pesticides is the process by which government (e.g., Canadian federal government) authorities determine that a pesticide is suitable for use. Standards of public and worker safety, environmental impact, and usefulness must all be met.

Risk –The probability (likelihood) that some adverse or undesirable effect will take place in the future, as a result of some specified activity. For pesticides, risk is the probability that use of the pesticide will result in some specified harm to humans or the environment.

Subchronic – For experimental studies, relatively long term, but not as long as a chronic study. Typically three to six months. See acute and chronic.

Teratogen –A chemical that can cause birth defects.

Teratogenic –Relating to or able to produce birth defects.

Threshold effect – a toxic effect for which the dose-response curve is non-linear; there is a break in the dose-response curve at the threshold, and at doses below the threshold adverse effects are not observed.

Toxicant –A toxic agent; a poison.

Toxicity –The whole pattern of harmful effects (injury, illness and other undesirable effects) that a chemical can cause. It is a property of the chemical.

Toxicology –The group of scientific disciplines that identifies and studies the adverse effects of chemicals on biological systems.

Toxin –A poisonous substance (poison) produced by a living organism.

Tumor – A new growth of cells multiplying progressively and without control.

Export Control Restrictions

Access to and use of EPRI Intellectual Property is granted with the specific understanding and requirement that responsibility for ensuring full compliance with all applicable U.S. and foreign export laws and regulations is being undertaken by you and your company. This includes an obligation to ensure that any individual receiving access hereunder who is not a U.S. citizen or permanent U.S. resident is permitted access under applicable U.S. and foreign export laws and regulations. In the event you are uncertain whether you or your company may lawfully obtain access to this EPRI Intellectual Property, you acknowledge that it is your obligation to consult with your company's legal counsel to determine whether this access is lawful. Although EPRI may make available on a case-by-case basis an informal assessment of the applicable U.S. export classification for specific EPRI Intellectual Property, you and your company acknowledge that this assessment is solely for informational purposes and not for reliance purposes. You and your company acknowledge that it is still the obligation of you and your company to make your own assessment of the applicable U.S. export classification and ensure compliance accordingly. You and your company understand and acknowledge your obligations to make a prompt report to EPRI and the appropriate authorities regarding any access to or use of EPRI Intellectual Property hereunder that may be in violation of applicable U.S. or foreign export laws or regulations.

The Electric Power Research Institute, Inc. (EPRI, www.epri.com) conducts research and development relating to the generation, delivery and use of electricity for the benefit of the public. An independent, nonprofit organization, EPRI brings together its scientists and engineers as well as experts from academia and industry to help address challenges in electricity, including reliability, efficiency, health, safety and the environment. EPRI also provides technology, policy and economic analyses to drive long-range research and development planning, and supports research in emerging technologies. EPRI's members represent more than 90 percent of the electricity generated and delivered in the United States, and international participation extends to 40 countries. EPRI's principal offices and laboratories are located in Palo Alto, Calif.; Charlotte, N.C.; Knoxville, Tenn.; and Lenox, Mass.

Together...Shaping the Future of Electricity

Program:

Rights-of-Way (ROW): Siting, Vegetation Management, and Avian Issues

© 2010 Electric Power Research Institute (EPRI), Inc. All rights reserved. Electric Power Research Institute, EPRI, and TOGETHER...SHAPING THE FUTURE OF ELECTRICITY are registered service marks of the Electric Power Research Institute, Inc.

1020323

Electric Power Research Institute

3420 Hillview Avenue, Palo Alto, California 94304-1338 • PO Box 10412, Palo Alto, California 94303-0813 USA
800.313.3774 • 650.855.2121 • askepri@epri.com • www.epri.com