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Mandy Tu

Callie Hurd

John M. Randall

The Nature Conservancy

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Weed Control Methods Handbook: Tools & Techniques for Use in Natural Areas



Mandy Tu, Callie Hurd & John M. Randall
The Nature Conservancy
Wildland Invasive Species Team
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Mandy Tu, Callie Hurd, and John M. Randall

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Note: This manual is periodically revised, expanded, and improved. If you have any comments or questions please contact TNC's Wildland Invasive Species Team:

Barry Rice bamrice@ucdavis.edu or 530-754-8891

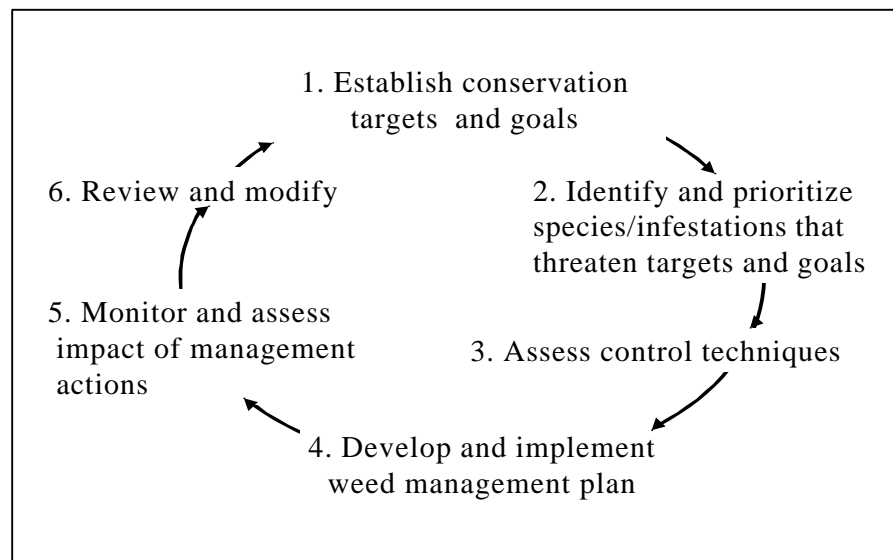
Mandy Tu imtu@tnc.org or 503-230-1221

INTRODUCTION

Invasive non-native plants are a serious threat to native species, communities, and ecosystems in many areas around the world. They can compete with and displace native plants, animals, and other organisms that depend on them, alter ecosystem functions and cycles significantly, hybridize with native species, and promote other invaders. The good news is that many plant invasions can be reversed, halted or slowed, and in certain situations, even badly infested areas can be restored to healthy systems dominated by native species. In most instances this requires taking action to control and manage those invasive plants. This handbook provides you with detailed information about the tools and techniques available for controlling invasive plants, or weeds, in natural areas. Whenever possible, language familiar to natural area managers is used, and unfamiliar terms and jargon borrowed from other fields are defined.

Before embarking on a weed management program, it is important to develop a straightforward rationale for the actions you plan to take. We believe this is best accomplished using an adaptive management approach as follows (see Figure 1): (1) establish management goals and objectives for the site; (2) determine which plant species or populations, if any, block or have potential to block attainment of the management goals and objectives; (3) determine which methods are available to control the weed(s); (4) develop and implement a management plan designed to move conditions toward management goals and objectives; (5) monitor and assess the impacts of management actions in terms of their effectiveness in moving conditions toward these goals and objectives; and (6) reevaluate, modify, and start the cycle again. Note that control activities are not begun until the first three steps have been taken. A weed control program is best viewed as part of an overall restoration program, so focus on what you want in place of the weed, rather than simply eliminating the weed. When selecting control methods, keep in mind that the ultimate purpose of the work is to preserve native species, communities, and/or functioning ecosystems.

Figure 1.
Adaptive Weed
Management
Approach



This Handbook is divided into eight chapters, covering a range of different control methods. More often than not, however, successful weed control requires the combination or sequential use of several methods (called integrated weed management). For example, cutting followed by herbicide applications has been used successfully in many programs, and prescribed fires followed by spot-applications of herbicides have been used well in others. Consider all available control options: manual, mechanical, promoting competition from native plants, grazing, biocontrol, herbicides, prescribed fire, solarization, flooding, and other, more novel, techniques. Each has advantages and disadvantages in terms of its effects against the target weed(s), impacts to untargeted plants and animals, risks to human health and safety, and costs. The chapters that follow discuss the advantages and disadvantages for each method and provide examples of their successful (and in some cases unsuccessful) use in natural areas.

Chapter 1 describes a variety of manual and mechanical techniques. Chapter 2 covers the use of grazing for weed control in natural areas including the types of animals that can be used and how to time grazing for best effect. Chapter 3 briefly discusses the use of prescribed fire to control invasive plants. TNC has specific guidelines and regulations for using prescribed fire that must be adhered to. See TNC's Fire Management Manual and contact TNC's Fire Initiative (<http://www.tncfire.org>) for details on the steps required to develop and implement a Site Fire Management Plan.

Chapter 4 covers biological control of invasive plants. Biocontrol agents typically have the capacity to persist, to spread to areas far from release sites, and may undergo genetic or behavioral changes that allow them to feed on new hosts. In spite of these risks, the use of biocontrol has the potential to be one of the most powerful tools available for invasive species control. TNC's policy is to not allow intentional releases of biocontrol agents on land it owns and manages, unless permission to do so has been granted by the Executive Director of TNC's Invasive Species Initiative. TNC's biocontrol release policy and standard operating procedures for requesting permission for releases are contained in this chapter.

Chapters 5 through 7 provide information on the use of herbicides to control invasive plants in natural areas. Chapter 5 discusses factors to consider when deciding whether to use herbicides or not, provides guidelines for herbicide use, and describes different application methods, who may apply herbicides and when they are most effectively applied. TNC staff should read the "Standard Operating Procedures & Guidelines" and "Herbicide Health & Safety Guidelines" in this chapter **PRIOR** to purchasing or using herbicides. Chapter 6 discusses general properties of herbicides, different types of herbicide formulations, their behavior in the environment, and human and environmental safety concerns. Chapter 7 provides detailed information for eleven herbicides that have been used in natural areas. It contains a table that summarizes important characteristics of each of the 11 herbicides, followed by detailed information about each one. Finally, Chapter 8 discusses the addition and use of adjuvants in herbicide tank mixes. Adjuvants are often added into a tank mix to improve herbicide penetration and/or to facilitate the mixing, application and effectiveness of that herbicide formulation.

Information on the biology and control of specific invasive plants are available from <http://tncweeds.ucdavis.edu> and other sites on the web. TNC staff that would like additional assistance are encouraged to contact TNC's Wildland Invasive Species Team. John Randall (530-754-8890 or jarandall@ucdavis.edu), Barry Rice (530-754-8891 or bamrice@ucdavis.edu) or Mandy Tu (503-230-1221 or imtu@tnc.org) are available to answer questions and provide advice, information and referrals regarding specific weed problems.

ACKNOWLEDGEMENTS

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Chapter 1 – MANUAL & MECHANICAL CONTROL TECHNIQUES

Manual and mechanical techniques such as pulling, cutting, and otherwise damaging plants, may be used to control some invasive plants, particularly if the population is relatively small. These techniques can be extremely specific, minimizing damage to desirable plants and animals, but they are generally labor and time intensive. Treatments must typically be administered several times to prevent the weed from re-establishing, and in the process, laborers and machines may severely trample vegetation and disturb soil, providing prime conditions for re-invasion by the same or other invasive species.

Manual and mechanical techniques are generally favored against small infestations and/or where a large pool of volunteer labor is available. They are often used in combination with other techniques, for example, when shrubs are pulled and cut, and re-sprouts and seedlings are treated with herbicides or fire several weeks or months later.

When using manual and mechanical methods, it is especially important to thoroughly clean and inspect all equipment and clothing before moving it off-site. This will lessen the probability of spreading the weed(s) to the next worksite.

In addition to the tools described here, the Wildland Invasive Species Team web page reviews other innovative tools. See <http://tncweeds.ucdavis.edu/tools.html>.

A. WEED PULLING

Pulling or uprooting plants can be effective against some shrubs, tree saplings, and herbaceous and floating weeds. Annuals and tap-rooted plants are particularly susceptible to control by hand-pulling. Weed wrenches and other tools are surprisingly powerful and can enable you to control large saplings and shrubs that are too big to be pulled by hand. It is not as effective against many perennial weeds with deep underground stems and roots that are often left behind to re-sprout.

How To: Minimize soil disturbance by pulling out weeds slowly and carefully, and replace soil to disturbed areas where possible. Trampled and disturbed areas can provide optimal germination sites for many weeds. Minimize trampling by limiting the number of people in the site and the amount of time spent there. Whenever a manual technique is used, it is wise to wear gloves, a long-sleeved shirt, and long pants. Some plants can cause moderate to severe skin irritation, especially when their stems and leaves are crushed and broken. Even the flimsiest weeds can leave hands raw and bleeding after several hours of pulling.

The advantages of pulling include its small ecological impact, minimal damage to neighboring plants, and low (or no) cost for equipment or supplies. Pulling is extremely labor intensive, however, and is effective only for relatively small areas, even when abundant volunteer labor is available.

1. Hand Pulling

Hand pulling is easy to plan and implement, and is often the best way to control small infestations, such as when a weed is first detected in an area. Hand pulling may be a good alternative in sites where herbicides or other methods cannot be used. The key to effective hand pulling is to remove as much of the root as possible while minimizing soil disturbance. For many species, any root fragments left behind have the potential to re-sprout, and pulling is not effective on plants with deep and/or easily broken roots.

Hand pulling has been effective against a variety of invaders in natural areas scattered across the U.S. For example, hand pulling by volunteers has successfully controlled *Centaurea diffusa* (diffuse knapweed) in the Tom McCall Preserve in northeast Oregon. Yellow bush lupine (*Lupinus arboreus*) was also controlled in coastal dunes in California by pulling small shrubs by hand. Larger shrubs were cut down with an ax, and re-sprouting was uncommon (Pickart and Sawyer 1998). Hand pulling has also been fairly successful in the control of small infestations of *Centaurea* spp. (thistles), *Melilotus officinalis* (white and yellow clover), and *Lythrum salicaria* (purple loosestrife) at TNC preserves scattered across the country.

2. Pulling Using Tools

Most weed-pulling tools are designed to grip the weed stem and provide the leverage necessary to pull its roots out. Tools vary in their size, weight, and the size of the weed they can extract. The Root Talon is inexpensive and lightweight, but may not be as durable or effective as the all-steel Weed Wrench, which is available in a variety of sizes. Both tools can be cumbersome and difficult to carry to remote sites. Both work best on firm ground as opposed to soft, sandy, or muddy substrates.

Root Talon

The Root Talon is an inexpensive and lightweight tool shaped something like a pick-ax with a plastic handle and metal head. It has a specialized claw and gripping device that allow the user to grab the plant stem and provide leverage to pull-up and remove the plants. It is best used for pulling shallow rooted plants such as sapling trees and herbs with sturdy stems. Plants that have been pulled using the Root Talon include young tree-of-heaven (*Ailanthus*), Scarlet wisteria (*Sesbania punicea*), and buckthorn (*Rhamnus* spp.). The Root Talon is not effective against deep-rooted plants, because it does not provide enough leverage. In addition, it is difficult to use the Root Talon to pull spiny plants because the plant stems (and spines) must be put into the gripping flange by hand. Advantages of the Root Talon are that it is lighter and less expensive than the Weed Wrench (see below), and provides easier and more effective control than hand pulling.

At the time of printing, the Root Talon retailed for \$47 plus \$5.25 shipping through Lampe Design, LLC, 262 South Griggs Street, St. Paul, MN 55105, (612) 699-4963, jklampe@worldnet.att.net or on the web at www.buckthorn.com.

Weed Wrench

The Weed Wrench provides more leverage than the Root Talon. Its all-steel frame is capable of withstanding more strain than the plastic handle of the Root Talon. It comes

in four sizes, from the “mini”, which weighs 2.4 kg (5.25 lbs) and is capable of pulling weeds with stems up to 2.5 cm (1.0 in) in diameter, to the “heavy”, which weighs 10.5 kg (24 lbs) and can handle weeds up to a diameter of 6.25 cm (2.5 in). Larger Weed Wrenches provide more leverage and pulling power. It is best to choose the smallest size needed, however, because larger Weed Wrenches are heavy and can be difficult to carry and use in remote sites.

Manufacturers of the Weed Wrench claim it is capable of handling any plant that can fit within the “jaws” of the wrench, as long as the plant stem is stronger than the anchoring strength of the roots. The Weed Wrench can be used on herbaceous plants that have a stem or bundle of stems strong enough to withstand the crush of the jaws. It has been used successfully to pull acacia (*Acacia melanoxylon*), buckthorn (*Rhamnus cathartica*), Russian olive (*Elaeagnus angustifolia*), multiflora rose (*Rosa multiflora*), willow (*Salix* spp.), tamarisk (*Tamarix* spp.), bush honeysuckles (*Lonicera* spp.), Scotch broom (*Cytisus scoparius*), French broom (*Genista monspessulanus*), and Brazilian pepper (*Schinus terebinthifolius*) at preserves across the mainland U.S. In Hawaii, the Weed Wrench has been used to pull Strawberry guava (*Psidium cattleianum*) and small saplings of Karaka nut (*Corynocarpus laevigatus*) from the Kamakou preserve on Molokai (Hawaii).

For more information, contact The Weed Wrench Company, at 2852 Willamette Street #403, Eugene, OR 97405, 1-877-484-4177, connect@weedwrench.com. You can also view their website at <http://www.weedwrench.com>.

B. MOWING, BRUSH-CUTTING, WEED EATING

Mowing and cutting can reduce seed production and restrict weed growth, especially in annuals cut before they flower and set seed (Hanson 1996). Some species however, re-sprout vigorously when cut, replacing one or a few stems with many that can quickly flower and set seed. For example, yellow starthistle (*Centaurea solstitialis*) can be controlled by mowing at the onset of flowering (when approximately 2 to 5% of the seed heads are flowering), but if mowed earlier, native species are negatively impacted and yellow starthistle is able to re-sprout (Benefield et al. 1999). Be sure to consider the biology of the weed before cutting.

How To: Mowing and cutting are often used as primary treatments to remove aboveground biomass, in combination with prescribed burning or herbicide treatments. It is important to collect the cut fragments of species capable of re-sprouting from stem or root segments to prevent them from washing or blowing into uninfested areas.

C. STABBING

Some plants can be killed by severing or injuring (stabbing) the carbohydrate storage structure at the base of the plant. Depending on the species, this structure may be a root corm, storage rhizome (tuber), or taproot. These organs are generally located at the base

of the stem and under the soil. Cutting off access to these storage structures can help “starve” or greatly weaken some species.

How To: To sever a taproot, place a flat-nosed spade, pruning saw, or knife at the base of the plant and push it as far below ground as possible. To prevent re-sprouting, the taproot should be severed below the caudex or root crown (where the stem becomes the root).

The stabbing technique has been used to control baby’s breath (*Gypsophila paniculata*) in Michigan (J. McGowan-Stinski, pers. comm.). The stabbing of root corms has also been an effective control technique for large (two yr old) plants of burdock (*Arctium lappa*) and wild parsnip (*Pastinaca sativa*) in Illinois and Wyoming (W. Kleiman, pers. comm.).

D. GIRDLING

Girdling is often used to control trees or shrubs that have a single trunk. It involves cutting away a strip of bark several centimeters wide all the way around the trunk. The removed strip must be cut deep enough into the trunk to remove the vascular cambium, or inner bark, the thin layer of living tissue that moves sugars and other carbohydrates between areas of production (leaves), storage (roots), and growing points. This inner cambium layer also produces all new wood and bark.

How To: To girdle a tree, cut parallel lines approximately three inches or more apart around the circumference of the tree. The cuts can be made using a knife, ax, or saw, and should be slightly deeper than the cambium. Strike the trunk sharply between the cuts using the back of an ax or other blunt object. The bark should come off in large pieces and prevent the tree from any further growth. It is important not to cut too deeply into the trunk because this could cause the tree to snap and fall in high winds. To determine the depth of the cambium, make two short test cuts and strike the bark between the cuts. After several strikes the bark should come off intact, exposing the cambium and wood (xylem) below.

Girdling is effective against pines, some oaks, and some maples. It typically requires less labor than cutting and removal, is inexpensive, and kills only the targeted plant. It also leaves no residue except the standing trunks. In addition, a dead standing tree (snag) can provide valuable wildlife habitat, and if left to decay, allows the nutrients of the tree to be returned to the system, rather than being removed and deposited elsewhere. A few species, notably black locust (*Robinia pseudoacacia*) and tree of heaven (*Ailanthus altissima*) should not be girdled because they respond by producing many fast growing root and stem sprouts. Therefore, before girdling, find out if the target species responds by re-sprouting. If so, use another control technique, such as hack and squirt herbicide applications or if you do girdle return at 1 to 4 month intervals to cut, burn, or herbicide all re-sprouts for at least 2 years.

Girdling has been used successfully on preserves in New York state to control quaking aspen (*Populus tremuloides*) and bigtooth aspen (*Populus grandidentata*). Girdling can also be used in combination with herbicides. Black locust (*Robinia pseudoacacia*) and

quaking aspen (*P. tremuloides*) in New York and Wisconsin, respectively, were controlled successfully using girdling with herbicide. This method, however, was not successful, in controlling tropical ash (*Fraxinus uhdei*) on the Kamakou preserve on Molokai, Hawaii.

E. MULCHING

Mulching can be used on relatively small areas, but will often stunt or stop growth of desirable native species. Mulching cannot control some perennial weeds because their extensive food reserves allow them to continue to grow up through the mulch.

How To: Cover the ground and/or seedlings with mulch (hay, grass clippings, wood chips, etc.) or other type of ground cover (newspaper clippings). This prevents weed seeds and seedlings from receiving sunlight necessary to survive and grow.

Hay mulch was used in Idaho with some success to control the spread of Canada thistle (*Cirsium arvense*). This hay mulch was applied several feet deep to established plants, and even though these plants were not completely eliminated, flowering rates were much suppressed by the end of the growing season.

F. TILLING

Tilling, or the turning-over of soil, is often used for weed control in agricultural crops. Its use in wildland management is largely limited, however, to restoration sites where soils are already badly disturbed. Tilling is effective against annuals and shallow-rooted perennials, but small fragments of some species, particularly those perennials with rhizomes, can often resprout following tillage. Tilling should be completed before seeds develop and are shed onto the soil. The best control is achieved when the soil remains dry, so that remaining plant fragments dry out. Moist soils help the fragments survive and re-grow.

How To: “Primary” tillage equipment is initially used to turn over soil and cuts roots at depths of six inches to two feet to prepare the soil for planting. “Secondary” tillage equipment, or equipment designed to work only the top six inches of soil, is used mainly to control weeds.

Many types of secondary tillage equipment are available. Equipment ranges from small hand-pushed models, to tractor mounted power-driven tillers. The appropriate model depends on the size and type of the habitat.

G. SOIL SOLARIZATION

Soil solarization is the technique of placing a cover (usually black or clear plastic) over the soil surface to trap solar radiation and cause an increase in soil temperatures to levels that kill plants, seeds, plant pathogens, and insects. In addition, when black plastic or other opaque materials are used, sunlight is blocked which can kill existing plants (Katan

et al. 1987). Soil solarization however, can cause significant biological, physical, and chemical changes in the soil that can last up to two years, and deter the growth of desirable native species.

Soil solarization is used in horticulture and for a few high value agriculture crops like strawberries. This method has not been used extensively for weed control in natural settings. The effectiveness of soil solarization depends, in part, on how susceptible weed seeds are to temperature increases. It is most effective against winter annual weeds that germinate under cool conditions (Elmore 1990). Summer annuals and other species adapted to higher temperatures, which germinate during warmer parts of the year, are less susceptible.

Soil solarization is most effective during the summer months, and may be less effective in cooler climates (DeVay 1990). The higher the temperature, the more quickly a kill is achieved. Solarization is effective only if done in wet soil. Where soils are typically dry, they must first be irrigated until soil from the surface to 50 to 60 cm deep is at field capacity (Grinstein & Hetzroni 1991).

How To: Polyethylene plastic film is the most useful for soil solarization (DeVay 1990). Less expensive thin films (1-1.5 mil) are more effective than thick films (2, 4, and 6 mil). Clear and black films both trap infrared radiation that is re-radiated from the soil surface, therefore keeping the soil hot. Transparent film allows more radiation to reach the soil than black films, as it lets visible light in, causing even greater temperature increases. Because black films exclude visible light however, they stop photosynthesis, which can be enough to kill some young annuals and perennials given sufficient time (Elmore 1990). Double layers of film have been found to increase soil temperatures by three to ten degrees over single layers (DeVay 1990).

Soil solarization is beneficial in that it releases nutrients that are tied up in the organic component of the soil, and that it can kill unwanted plants without the use of chemicals (Stapleton 1990). However, solarization leaves an open substrate that can be readily invaded by new organisms, both native and non-native once the plastic is removed (Stapleton 1990). The influx of nutrients that results from solarization can be advantageous to restoration efforts, but can promote aggressive, ruderal plants that typically thrive in nutrient-rich soils.

H. FLOODING

In situations where the water level of a wetland or riverine system can be manipulated, flooding can be used to control some plant species. Some species, however, have vegetative buds or underground storage organs that can survive several months or more under flooded conditions.

In Vermont, flooding was used successfully to kill seeds and seedlings of common buckthorn (*Rhamnus cathartica*). Flooding was also used in combination with herbicide to successfully control the spread of autumn olive (*Elaeagnus umbellata*) and reed

canarygrass (*Phalaris arundinacea*) in Ohio. At Wertheim NWR on Long Island, NY, *Phragmites australis* was controlled by burning and then flooding with several feet of water in impounded areas.

REFERENCES

- Benefield, C.B., DiTomaso, J.M., Kyser, G.B., Orloff, S.B., Churches, K.R., Marcum, D.B., and G.A. Nader. 1999. Success of mowing to control yellow starthistle depends on timing and plants branching form. *California Agriculture* 53(2): 17-21.
- DeVay, J.E. 1990. Historical review and principles of soil solarization. *In: DeVay, J.E., Stapleton, J.J., and C.L. Elmore (eds.), Soil Solarization. United Nations, Rome.*
- Elmore, C.L. 1990. Use of solarization for weed control. *In: DeVay, J.E., Stapleton, J.J., and C.L. Elmore (eds.), Soil Solarization. United Nations, Rome.*
- Grinstein, A. and A. Hetzroni. 1991. The technology of soil solarization. *In J. Katan and J.E. DeVay (eds.) Soil Solarization. CRC Publications, Boca Raton: 159-170.*
- Hanson, E. 1996. Tools and techniques. Chapter 3 *in Invasive plants. J. M. Randall and M. Marinelli, eds. Handbook #149. Brooklyn Botanical Garden, Inc., Brooklyn, New York. 111 pgs.*
- Katan, J., Grinstein, A., Greenberger, A., Yarden, O. and J.E. DeVay. 1987. First decade (1976-1986) of soil solarization (solar heating)-A chronological bibliography. *Phytoparasitica* 15:229-255.
- Pickart, A.J. and J.O. Sawyer. 1998. Ecology and restoration of Northern California coastal dunes. California Native Plant Society. Sacramento, CA. 152 pgs.
- Stapleton, J.J. 1990. Thermal inactivation of crop pests and pathogens and other soil changes caused by solarization. *In: DeVay, J.E., Stapleton, J.J., and C.L. Elmore (eds.), Soil Solarization. United Nations, Rome.*

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Chapter 2 – GRAZING

Grazing can either promote or reduce weed abundance at a particular site. By itself, grazing will rarely, if ever, completely eradicate invasive plants. However, when grazing treatments are combined with other control techniques, such as herbicides or biocontrol, severe infestations can be reduced and small infestations may be eliminated. Grazing animals may be particularly useful in areas where herbicides cannot be applied (e.g., near water) or are prohibitively expensive (e.g., large infestations). Animals can also be used as part of a restoration program by breaking up the soil and incorporating in seeds of desirable native plants.

When not properly controlled, however, grazing or other actions of grazing animals (wallowing, pawing up soil) can cause significant damage to a system, and promote the spread and survival of invasive weeds. Overgrazing can reduce native plant cover, disturb soils, weaken native communities, and allow exotic weeds to invade. In addition, animals that are moved from pasture to pasture can spread invasive plant seeds.

In general, the specific weed and desirable native plants will determine the number and species of animal grazers and the duration and frequency of grazing. A grazing plan should be developed in situations where prescribed grazing is desirable, and this plan must be tailored to fit the specifics of the site.

ANIMAL CHOICE

Cattle, goats, sheep, and even geese may be used to control weeds. Cattle will graze invasive grasses, can trample inedible weed species, and can incorporate native seeds into soil. Horses can also be used to control invasive grasses, but horses tend to be more selective than cattle. Geese are also useful for the control of invasive grasses, but are more subject to predation than other animals. Predation problems in many areas may dictate the type of grazing animals that can be used.

Sheep and goats prefer broadleaf herbs and have been used to control leafy spurge (*Euphorbia esula*), Russian knapweed (*Acroptilon repens*), and toadflax (*Linaria* spp.). These animals appear to be able to neutralize the phytochemicals toxic to other animals that are present in these and other forbs (Walker 1994). Goats can control woody species because they can climb and stand on their hind legs, and will browse on vegetation other animals cannot reach (Walker 1994). Goats additionally, tend to eat a greater variety of plants than sheep.

Sheep can be useful in the control of spotted knapweed (*Centaurea maculosa*), kudzu (*Pueraria lobata*), and oxeye daisy (*Chrysanthemum leucanthemum*) (Olson and Lacey 1994). Sheep are not recommended for the control of St. John's wort (*Hypericum perforatum*) or senecio (*Senecio* spp.) as these plants can be toxic.

Sheep do not graze an area uniformly. Consequently, a method (i.e: herding, fencing, or the placement of salt licks) should be employed to concentrate activities in an area (Olson and Lacey 1994). Sheep often need a period of adaptation before they will start to consume a new forage type. This process can be expedited by using herds as opposed to individual animals because sheep will follow the lead of their peers. Finally, leafy spurge seeds can remain viable after passing through the digestive tracts of sheep. Animals should therefore be kept out of uninfested areas until nine days after the last leafy spurge is consumed (Olson and Lacey 1994). Both sheep and goats are well adapted for grazing in steep or rocky terrain.

Plant availability, hunger, and previous experience can determine a grazer's selection of food plants (Walker 1994). Differences in vegetation quality may cause an animal to eat one species in one situation and to ignore the same species in another. A period of adjustment is generally required to get a grazing animal to eat a new type of forage (Walker 1994). It is therefore helpful to find animals previously experienced with the target weed.

Finding grazing animals to use for weed control is frequently a problem in the U.S., particularly when sheep or goats are needed. Land managers are sometimes forced to make use of the animals available in the immediate area, especially since transportation costs can be excessive. The following groups* lease-out goats specifically for weed control:

Southern Oregon Goat Producers
 HC 64 Box 77
 Lakeview, Oregon
 541-947-2691
 hbsb@ptinet.net

Ewe4ic Ecological Services
 Land Whisperer, LLC
 P.O. Box 3253
 Alpine, Wyoming 83128
 307-654-7866
 ewe4icbenz@aol.com

*Note: TNC does not endorse or necessarily use these listed services. The short list provided here is primarily for examples of grazing services, which may be available in your local area.

TIMING & DURATION OF GRAZING

Animals should be brought into an infested area at a time when they will be most likely to damage the invasive species without significantly impacting the desirable native species. Grazing during seed or flower production can be especially useful. On the other hand, some weeds are palatable only during part of the growing season. For example, cheatgrass (*Bromus tectorum*) is preferred in spring before seed heads develop, but avoided by cattle once it has begun to set seed because the seed heads have stiff awns that can puncture the mouth and throat tissue of livestock (Carpenter & Murray 1999).

Grazing will often result initially in an increase in stem density and root buds, but repeated grazing should lead to reduced stem densities in the longer term (Olson 1999).

Grazing should be closely monitored and the animals promptly removed when the proper amount of control has been achieved and/or before desirable native species are impacted. Consequently, land managers must be flexible and have control over herd movements. Lack of control can result in overgrazing of desirable species, which can enhance weed infestations or allow new weed species to become established. The necessary flexibility is not always possible with commercial herds.

In most cases, several years of intensive grazing followed by annual brief periods of grazing by the same grazing species is required to gain and maintain control of an infestation. However, gains achieved by grazing goats and sheep one year will not be maintained by cattle-only grazing in subsequent years because cattle tend to graze different types of plants.

ANIMAL FENCING & MOVEMENT

The containment and movement of grazers within and between infested areas is necessary for the successful implementation of an appropriate grazing plan. Temporary fencing erected to contain animals in a particular area may be suitable for goats and sheep, but is often inadequate for cows and horses. More stable and expensive barbed wire fencing may be required to contain these larger animals. Salt licks have been used successfully to concentrate animal impact in a particular area.

A herder is usually required to move goats and sheep between pastures or infestations and to ensure that the animals concentrate grazing on the appropriate species. Cattle must be moved periodically, but generally do not require a herder. Goats have been tied to stakes within infested areas to concentrate their activity and eliminate the need for full-time herders. "Open" herding is usually more beneficial than "close" herding, where animals are kept close together causing much of the forage to be trampled (Olson and Lacy 1994).

CONTROLLING SEED DISPERSAL

Seeds of leafy spurge (*Eurphobia esula*), spotted knapweed (*Centaurea maculosa*), and other species, can pass through the digestive tract of animals and remain viable. Animals that are removed from an infested area should not be transported to weed-free areas until all seeds have passed through their digestive tracts (five to nine days). Weed seeds can also be transported to new areas in animal hair. Care and precaution should be taken when moving animals from infested areas.

GRAZING CASE STUDIES

Patagonia/Sonoita Creek Preserve, Arizona – Cattle Grazing

Jeffrey Cooper and Ed Wilk have been using cattle and horses in Arizona to reduce the density of Johnson grass (*Sorghum halepense*) enough to allow native grasses to become re-established. They chose to use cattle and horses because they could be found locally and were not likely to suffer from predation.

Because they needed fenced pastures only while the grazers were using the land, they initially set up electric fences, which are cheaper and easier to install and remove than barbed wire fences. The electric fences, however, were inadequate for corralling these large animals and barbed wire fences are now being considered.

The animals were put on the land during the summer growing months. Ideally, Jeffrey and Ed would have liked the grass to be grazed repeatedly during a summer, moving animals on and off throughout the growing season. However, they did not have the personnel required to move the animals repeatedly. The animals were instead moved onto the pasture once, when the grass had achieved some significant growth, and were allowed to graze until the grass forage was essentially gone. The Johnson grass, however, did recover somewhat with the arrival of the rainy season.

After four years, stem density counts on established transects showed that stem densities decreased by 75%. Once the infestation was significantly reduced, the herbicide glyphosate (RoundUp[®]) was applied to control the remaining Johnson grass. Herbicide was applied in late spring to small plots from which cattle was excluded. One to two months following herbicide application, large native bunch grasses were planted on the herbicide-treated plots.

The results of this grazing-herbicide combination have been mixed. Although the combined grazing and herbicide treatments had reduced Johnson grass infestations significantly, this allowed other invasive broadleaf weeds to become established. In an effort to control these new weeds, Jeffrey and Ed cut down the invaders during flowering to reduce seed production and dispersal. Approximately five acres have been replanted with native grasses following the grazing and herbicide treatments. Some of the transplants, especially the more mature plants, are doing well. In other areas, the replanted natives were destroyed by gophers, and Johnson grass reinvaded. Jeffrey and Ed believe that if the exotic weeds can be kept down, the native grasses will eventually outcompete the exotics.

Marsh Creek, Idaho – Goat Grazing

Goats have been used to control leafy spurge (*Eurphobia esula*) on approximately 1,500 acres of mostly private land along Marsh Creek in South Central Idaho. Two trained herders spent five months on the site with approximately 600 goats. The goats were of mixed breed and age class. The area was stocked at about one goat per acre. Goats were herded on open range conditions. Trained herders were necessary to keep goats moving to new infestations and to prevent desirable native species such as willows (*Salix* spp.)

from being grazed. Because goats prefer to graze only two or three times in a particular area, herders were forced to continuously move the goats to new areas. Base camps were established and temporary fencing set up to corral the animals at night. These camps were moved on a regular basis according to the movements of the herd. By the end of the project, goats were traveling approximately four miles a day.

In addition to the cost of herders and temporary fencing, medical examination costs were also incurred. A few goats became ill due to the diet of almost exclusive leafy spurge. Supplemental feeding was required to maintain a balanced diet for the goats. An unexpected problem was that goats would not cross water, and makeshift bridges had to be erected for water crossings.

The land managers' long-term plan is to continue intensive goat grazing for five years until the abundance of leafy spurge is sufficiently reduced. Goats will be brought in for short periods once or twice a year thereafter, or small numbers of goats will be used along with grazing cattle to maintain leafy spurge control.

Sheep Grazing in Montana

Sheep have been used to control leafy spurge (*Eurphorbia esula*) in pastures and along rivers in Montana (Olson & Lacey 1994; Olson 1999). In some cases, continuous grazing by sheep resulted in significant reductions of leafy spurge stem density and viable seed bank (Olson & Lacey 1994; Olson & Wallander 1998). Leafy spurge is nutritious forage for sheep and can comprise up to 50% of their diet without ill effects. An added bonus is that sometimes the use of sheep for weed control does not cost anything to the landowner, because they provide free forage for the sheep (Olson 1999).

Red Canyon Ranch, Wyoming – Animal Impact

Bob Budd has used cattle to “beat down” infestations of Russian knapweed (*Acroptilon repens*) and leafy spurge (*Euphorbia esula*), and to prepare soil for native seeds on the Red Canyon Ranch in Wyoming. Although goats and sheep traditionally have been used to control these broadleaf species, these animals also eat some of the desirable native woody species on the Ranch. Consequently, Bob developed a plan to spray the infestations first with a 2,4-D herbicide, followed by a heavy dose of “animal impact”, or animal trampling, which breaks down any remaining weeds and turns up the soil in preparation for re-planting.

Bob uses approximately 800 head of cattle on three acres for one-half to one full day. Salt licks are placed within the infestations to help concentrate cattle in a specific area. After the animals are removed, native seeds are spread throughout the area.

Bob also uses animal impact without herbicides against infestations of dock (*Rumex* spp.) and kochia (*Kochia scoparia*) and believes that “animal impact” is the best use of animals for weed control. His advice to land managers is to not be afraid to hit the area hard with many animals. He states that one cannot gently “ease into” an animal impact strategy.

CONTACTS

Jeffrey Cooper or Ed Wilk
The Nature Conservancy
Patagonia/Sonoita Creek Preserve
P.O. Box 815
Patagonia, AZ 85624
Phone: (520) 394-2400

Bob Budd
The Nature Conservancy
Red Canyon Ranch
350 Red Canyon Rd.
Lander, WY 82520
Phone: (307) 332-3388

REFERENCES

- Carpenter, A.T. and T.A. Murray. 1999. Element Stewardship Abstract: *Bromus tectorum*. The Nature Conservancy's Wildland Invasive Species Program.
- Olson, B. E. 1999. Grazing and weeds. Chapter 8 in Biology and management of noxious rangeland weeds. R. L. Sheley and J. K. Petroff, eds. Oregon State Univ. Press. Corvallis, Oregon. 438 pgs.
- Olson, B. E., and J. R. Lacey. 1994. Sheep: a method for controlling rangeland weeds. Sheep Res. J. Special Issue 1994: 105-112.
- Olson, B.E. and R.T. Wallander. 1998. Effect of sheep grazing on a leafy spurge-infested Idaho fescue community. Journal of Range Management 51(2): 247-252.
- Walker, J. W. 1994. Multispecies grazing: The ecological advantage. Sheep Res. J. Special Issue: 1994: 52-64.

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Chapter 3 – PRESCRIBED FIRE

TNC and other agencies and organizations that manage land for biodiversity often use prescribed burns to promote desired vegetation and species. Fire is sometimes necessary to prompt the germination of some plants, including a number of rare and endangered species. On the other hand, fire can also sharply reduce the abundance of some species. The weather, topography, and available fuel will determine the temperature and intensity of the prescribed burn, and this along with the timing of the treatment, largely determine how the burn impacts the vegetation and the abundance of particular species.

The most effective fires for controlling invasive plant species are typically those administered just before flower or seed set, or at the young seedling/sapling stage. Sometimes prescribed burns that were not originally designed to suppress an invasive species have that happy side effect. But in some cases, prescribed burns can unexpectedly promote an invasive, such as when their seeds are specially adapted to fire, or when they resprout vigorously. These prescriptions must be modified or other management actions taken to undo or reverse the promotion of the invader.

Most successful weed control efforts that result from burning are due to the restoration of historical (natural) fire regimes, which had been disrupted by land use changes, urban development, fire breaks, or fire suppression practices. Many prescribed burn programs are, in fact, designed to reduce the abundance of certain **native** woody species that spread into unburned pinelands, savannas, bogs, prairies, and other grasslands. Repeated burns are sometimes necessary to effectively control weedy plants, and herbicide treatments may be required to kill the flush of seedlings that germinate following a burn.

When planning to implement a prescribed burn, be sure to that it fits within the context of an entire Site Conservation Plan. **TNC's Fire Initiative** can help you create a Site Fire Management Plan, and get necessary training and certification to conduct burns safely. Burns on TNC property can be conducted **ONLY** under the supervision of a TNC-designated Fire Leader ("burn boss"). The Fire Initiative has created a Fire Management Manual, which details TNC's Standard Operating Procedures for prescribed fires, information on how to start a burn program, writing a fire management plan, TNC requirements and guidelines for conducting burns, various administrative procedures, and fire management resources. The Manual can be downloaded from <http://www.tncfire.org>. The Fire Initiative can also be reached by phone at (850) 668-0827 and by e-mail at fire@tnc.org.

Spot-burning invasive weeds with a propane torch can be cheaper and easier than implementing a prescribed fire (permits are still required), but is only effective when the infestation is small. Spot-burning can be used to burn individual plants, groups of plants in a small area, or to ignite brush piles. Propane torches can be used in areas where there is little or no fine fuel to carry a prescribed burn, and can also be used to kill plants when

conditions are wet. See Appendix 2 for additional information on using a propane torch for spot-burning.

IMPLEMENTING A FIRE MANAGEMENT PROGRAM

Before implementing a fire management program to control the spread of invasive weeds, several steps must be taken. First and foremost, **contact the TNC Fire Initiative**. Any prescribed burn on any TNC property must be reviewed and approved by a Fire Manager trained and certified by TNC's Fire Initiative. Once site management goals and objectives have been compiled, and problem invasive plants and the methods that could be used against them have been identified, the following 4 steps should be completed:

1. Determine if fire management is needed.

It is important to determine, if the need to use prescribed fire to control weed invasions and meet other management goals, justifies the risks inherent in burning. Consider all available options for control of the weed; i.e., manual, mechanical, encouraging competition from native plants (restoration), herbicides, and biocontrol. Also consider the setting: is the weed in an old field, along roadsides, or in a pristine natural area with highly valued species and communities? Benefits from the chosen control option should always outweigh the overall risks and costs. In some cases the best option will be doing nothing to control the weed.

2. Develop a Site Fire Management Plan¹

The Site Fire Management Plan should be incorporated into the Site Conservation Plan, and designed to move conditions towards established conservation goals and objectives. TNC's Fire Initiative can assist in developing management plans for TNC preserves. A Site Fire Management Plan should include the following components:

- A. Site Background Information
- B. Fire Management Justification
- C. Fire Management Goals
- D. Fire Regime Proposal
- E. Site Specific Fire Operations
- F. Smoke Management Plan
- G. Neighbor and Community Factors
- H. Maps

3. Develop and implement a Prescribed Burn Plan¹

A Prescribed Burn Plan is a field document that includes specifics for conducting a particular burn treatment at a particular burn unit. It is also a legal document that details

¹ Modified from TNC's Fire Management Manual. Please refer to the Manual for specific details in developing and implementing each plan. See <http://www.tncfire.org>.

the professional standards and guidelines to be used when conducting the burn. A Prescribed Burn Plan includes the objectives to be accomplished by a particular burn, an acceptable range of environmental factors under which the burn can be carried out (such as wind speed and relative humidity), lists of equipment needed, sources of emergency assistance, maps, and a checklist for burn preparation and crew briefing. Before conducting any burn, be sure to get approvals for all parts of the management plan. Only qualified personnel² are allowed to conduct or work during a burn, and all burns must be supervised by a TNC-designated Fire Leader (“burn boss”).

4. Monitor and assess the impacts of management actions

Plan and implement a program to monitor the impacts of burning. The design and intensity of monitoring required will depend on the situation. John Randall at the Wildland Invasive Species Team is available to TNC staff for assistance with developing effective monitoring programs. Help is also available from TNC’s Fire Initiative as well as from Bob Unnasch, monitoring specialist and Senior Ecologist of TNC’s Aridlands Grazing Network. Analyzing monitoring data regularly will help determine whether management objectives are being met and if modifications are needed.

EXAMPLES OF PRESCRIBED FIRE TO CONTROL INVASIVE WEEDS

Spot-Burning

Spot-burning using a propane torch has been used successfully by Jack McGowan-Stinski in several Michigan preserves. Jack reported killing >90% of baby’s breath (*Gypsophila panicula*) seedlings with spot-burning. This method also kills most seedlings/saplings of buckthorn (*Rhamnus* spp.), where the adult plants have already been removed. In contrast, hand-pulling the seedlings requires more time and labor. Jack recommends burning buckthorn seedlings early in the first growing season after adult removal. Repeat burn treatments are necessary since seeds in the soil may germinate later and plants may resprout. These repeat treatments, however, are generally not labor intensive and is usually required only on a small patch basis.

Prescribed Burns

Prescribed burns are used to control a variety of weeds at sites scattered across North America. They are effective, especially in the short-term, for controlling the spread of Japanese honeysuckle (*Lonicera japonica*) in Alabama. Further north in southern New Jersey, where Japanese honeysuckle is semi-evergreen, winter burns were used to sharply reduce its abundance without any detectable impact on native species.

Carlen Emanuel of the Alabama Natural Heritage Program reports that prescribed burns are useful for controlling small seedlings and saplings of native loblolly pine (*Pinus taeda*), and that control rates are especially high when burning is combined with cutting.

² Training for burn crew personnel can be certified only through TNC’s Fire Initiative. Refer to the Manual for specific details regarding how to receive this training.

She also finds fire invaluable for preventing native sweetgum (*Liquidambar* spp.) from invading wetlands.

In California's Dye Creek and Vina Plains Preserves, prescribed burns help control the spread of invasive medusahead grass (*Taeniatherum caput-medusae*). California's Lassen Foothills Project also reported good success with >95% mortality of medusahead and yellow starthistle (*Centaurea solstitialis*) following prescribed burns.

Fire was used to kill small native Eastern redcedars (*Juniperus virginiana*) in Ohio, and to control alien tree-of-heaven (*Ailanthus altissima*) in Indiana's Blue River Project. Repeated burns were required, however, for full effectiveness. See Table 3.1 at the end of this chapter for more examples of the effects of burning on specific species.

Prescribed Burning and Herbicides

Some invasive species have underground storage organs that resprout vigorously after fire, and/or seeds whose germination is stimulated by fire. Some of these species may not be possible to control with fire, but some can be controlled with repeated burns and others may be especially vulnerable to herbicides after a burn. Resprouts or seedlings that are 1 to 3 months old are often especially sensitive to herbicides. Be sure to read the Guidelines for Herbicide Use and Developing a Rationale for Herbicide Use in this handbook, if you are considering the use of an herbicide.

In Illinois, reed canarygrass (*Phalaris arundinacea*) was controlled by a burning-herbicide combination treatment. Burning removed the surrounding thatch, and then glyphosate herbicide was applied. The spread of leafy spurge (*Euphorbia esula*) was halted, at least temporarily, with burning-herbicide treatments on preserve in Minnesota and Michigan. Burning initially reduces the litter layer, and also stimulates the seeds of leafy spurge to germinate, therefore reducing the seed bank.

Purple loosestrife (*Lythrum salicaria*) was successfully controlled in Michigan by burning, then applying glyphosate (Rodeo[®]).

Fire alone failed to control cogongrass (*Imperata cylindrica*) in Florida's Apalachicola Bluffs and Ravine Preserve, but good control was achieved when herbicide was applied following burns.

More examples of invasive weeds that have been controlled by prescribed fire, and the effects of burning on them, are presented in Table 3.1 at the end of this chapter.

OTHER CONSIDERATIONS

Timing of Burn

The timing of a burn can strongly affect the fire's impact on native and exotic plant populations. For example, in California's Carrizo Plain Natural Area, Meyer & Schiffman (1999) determined that warm-season prescribed burning (late-spring and fall) was most effective for reducing abundance of Mediterranean annual grasses. Native plant cover and diversity also increased significantly following warm-season prescribed burns. Winter burns, however, did not affect the abundance of native plants, and exotic plant cover was only moderately reduced.

Timing was also key in controlling smooth brome (*Bromus inermis*) and encouraging the growth of native grasses in Nebraska and Minnesota (Willson & Stubbendieck 2000). Timing prescribed burns so that they occurred at the time of tiller (aboveground lateral stem) elongation, yielded an immediate and persistent reduction in both tiller density and biomass of smooth brome.

Burning in Extensively Disturbed Areas

Not all burn treatments in wildlands are beneficial. When fires become too intense, crown-fires and death of native plants that typically survive fires can result. If temperatures are too hot, soil organisms and seeds, even those of species that require fire stratification for germination, may perish, and valuable soil nutrients may be volatilized or otherwise lost. In extensively disturbed areas of southwest Australia, fire actually enhanced the invasion of weeds along roadsides, and resulted in an overall decrease in the abundance of native species (Milberg & Lamont 1995). Schwartz & Heim (1996) reported that fire was at best moderately successful for garlic mustard (*Alliaria petiolata*) control in Illinois forests, and Luken & Shea (2000) determined that repeated prescribed burning had no significant effect on garlic mustard in Kentucky. In both cases, however, the burns were detrimental to native herbaceous species, reducing both density and richness. Even three years after the initial burns, native plant composition did not recover to pre-burn values.

Preventing Spread of Weeds

Keep all equipment, trucks, and engines clean of weed seeds. After each burn, and before moving to another site, be sure to clean (hose-off) all equipment, tools, and clothing used. This will minimize changes of carrying weed seeds directly to a new site where a fire might provide perfect conditions for their establishment.

Table 3.1. Examples of weeds that have been controlled by prescribed fire, and the effects of burning on these weeds.

Scientific Name	Common Name	Effects of Burning	Reference
<i>Bromus inermis</i>	Smooth brome	<ul style="list-style-type: none"> burning at time of tiller elongation, yields an instant and persistent reduction in tiller density and biomass 	Willson 1990 Willson & Stubbendieck 2000
<i>Bromus japonicus</i>	Japanese brome	<ul style="list-style-type: none"> litter accumulation aids in the growth of Japanese brome; burning once every 5 years will reduce litter and <i>B. japonicus</i> cover 	Whisenat 1990
<i>Centaurea maculosa</i>	Spotted knapweed	<ul style="list-style-type: none"> repeated burning will reduce spotted knapweed, but it is often difficult to get a burn to carry through dense knapweed patches burning is only effective where regrowth of native species is vigorous 	Mauer 1985 Watson & Renney 1974
<i>Cirsium arvense</i>	Canada thistle	<ul style="list-style-type: none"> fewer thistles were seen in years following a burn than before or year of the burn late spring burns (May-June) are most detrimental – thistles may increase the first year following a May burn, but will decline within 2 growing seasons; immediate reductions in thistles occur following a June burn early spring burns can increase sprouting and reproduction during first 3 years of control efforts, burning should be conducted annually 	Evans 1984 Hutchinson 1992 Sather 1988 Smith 1985
<i>Dipsacus sylvestris</i>	Teasel	<ul style="list-style-type: none"> in sparse stands, late spring burns are effective little control is provided by burning in dense stands, because fire will not carry through burning works best in conjunction with other means of control 	Glass 1991
<i>Euphorbia esula</i> <i>Euphorbia cyparissias</i>	Leafy spurge Cypress spurge	<ul style="list-style-type: none"> fire stimulates vegetative growth fire followed by herbicide treatment has been effective, because the regrowth is more vulnerable to herbicides late fall herbicide application of picloram and 2,4-D followed by a fall burn resulted in 100% control after 2 years of treatment 	Biersboer & Koukkari 1990 Cole 1991a
<i>Hypericum perforatum</i>	St. John's Wort	<ul style="list-style-type: none"> fire tends to increase stands 	Crompton et al. 1988

Scientific Name	Common Name	Effects of Burning	Reference
<i>Lysimachia nummularia</i>	Moneywort	<ul style="list-style-type: none"> ▪ best to burn in spring when moneywort is green and native vegetation is dormant ▪ regular burning regime for several years will be needed for control 	Kenney & Fell 1992a
<i>Melilotus alba</i> & <i>Melilotus officinalis</i>	White sweet clover & Yellow sweet clover	<ul style="list-style-type: none"> ▪ at least two burns are necessary for control ▪ increase in abundance in first year after burn ▪ burning in late spring of the second-year as the shoots elongate, results in a kill of second year plants prior to flowering and seed set ▪ mulching was found to be more effective than late spring burning ▪ dormant season burns stimulate germination and increase the chance that plants will survive to produce seeds ▪ dormant season burns can be used in conjunction with mowing or clipping in summer of the following year as plants flower 	Cole 1991b Eidson & Steigmann 1990 Kline 1983 Schwarzmeier 1984 Turkington et al. 1978
<i>Pastinaca sativa</i>	Wild parsnip	<ul style="list-style-type: none"> ▪ fire removes ground litter and standing litter, providing favorable conditions for the development of parsnip rosettes ▪ periodic burning may help maintain the vigor of native plants to allow them to better compete with parsnip 	Eckardt 1987 Kenney & Fell 1992b
<i>Phalaris arundinacea</i>	Reed canarygrass	<ul style="list-style-type: none"> ▪ growing season fires may reduce vigor and help control the spread ▪ growing season burns may give native species a competitive advantage 	Apfelbaum & Sams 1987 Henderson 1990
<i>Phragmites australis</i>	Phragmites	<ul style="list-style-type: none"> ▪ burning will not reduce growth unless the roots burn ▪ burning removes phragmites leaf litter, allowing seeds of other species to germinate ▪ burning in conjunction with chemical control has been found effective ▪ burn with caution, since spot fires can occur up to 100 feet from burning phragmites 	Beall 1984 Marks 1986
<i>Typha</i> spp.	Cattail	<ul style="list-style-type: none"> ▪ fire provides little or no control unless the roots are burned ▪ drawdown followed by burning and then flooding to a depth of 8 – 18” will provide control 	Apfelbaum 1985 Nelson & Dietz 1966

REFERENCES

- Apfelbaum, S. 1985. Cattail (*Typha* spp.) management. *Natural Areas Journal* 5(3): 9-17.
- Apfelbaum, S.I. and C.E. Sams. 1987. Ecology and control of reed canary grass (*Phalaris arundinacea* L.). *Natural Areas Journal* 7: 69-74.
- Beall, D.L. 1984. Brigantine Division – marsh vegetation rehabilitation. Chemical control of *Phragmites*. US Fish and Wildlife Service. 8pp.
- Biersboer, D.D. and W.L. Koukkari. 1990. Control of leafy spurge along rights-of-ways with burning and herbicides. Proceedings and Progress Reports of the Leafy Spurge Symposium, University of Wyoming, Laramie, WY.
- Cole, M.A.R. 1991a. Vegetation management guideline: leafy spurge (*Euphorbia esula* L.). *Natural Areas Journal* 11(3): 171-172.
- Cole, M.A.R. 1991b. Vegetation management guideline: white and yellow sweet clover (*Melilotus alba* Desr. and *Melilotus officinalis* (L) Lam.). *Natural Areas Journal* 11: 214-215.
- Crompton, C.W., Hall, I.V., Jensen, K.I.N. and P.D. Hildebrand. 1988. The biology of Canadian weeds. 83. *Hypericum perforatum* L. *Canadian Journal of Plant Science* 68: 149-162.
- Eckardt, N. 1987. Element Stewardship Abstract: *Pastinaca sativa*, wild parsnip. The Nature Conservancy, Arlington, VA. 4pp.
- Eidson, J. and K.L. Steigmann. 1990. Preliminary report on the response of yellow sweet clover (*Melilotus officinalis*) to late spring burning and mulch treatments. Proceedings of the 12th North American Prairie Conference. University of Northern Iowa, Cedar Falls, IA.
- Evans, J.E. 1984. Canada thistle (*Cirsium arvense*): a literature review of management practices. *Natural Areas Journal* 4: 11-21.
- Glass, W.D. 1991. Vegetation management guideline: cut-leaved teasel (*Dipsacus laciniatus* L.) and common teasel (*Dipsacus sylvestris* Huds.). *Natural Areas Journal* 11(4): 213-214.
- Henderson, R.A. 1990. Controlling reed canary grass in a degraded oak savanna (Wisconsin). *Restoration and Management Notes* 8(2): 123-124.
- Hutchinson, M. 1992. Vegetation management guideline: Canada thistle (*Cirsium arvense* (L) Scop.). *Natural Areas Journal* 12(3): 160-161.
- Kenney, J. and G. Fell. 1992a. Vegetation management guideline: moneywort (*Lysimachia nummularia* L.). *Natural Areas Journal* 12: 40.
- Kenney, J. and G. Fell. 1992b. Vegetation management guideline: wild parsnip (*Pastinaca sativa* L.). *Natural Areas Journal* 12: 42-43.
- Kline, V.M. 1983. Control of sweet clover in a restored prairie (Wisconsin). *Restoration and Management Notes* 1(4): 30-31.
- Luken, J.O. and M. Shea. 2000. Repeated prescribed burning at Dinsmore Woods State Nature Preserve (Kentucky, USA): Responses of the understory community. *Natural Areas Journal* 20(2): 150-158.
- Marks, M. 1986. Element Stewardship Abstract: *Phragmites* (*Phragmites australis*). The Nature Conservancy. 10pp.

- Mauer, T. 1985. Element Stewardship Abstract: spotted knapweed (*Centaurea maculosa*). The Nature Conservancy. 10pp.
- Meyer, M.D. and P.M. Schiffman. 1999. Fire season and mulch reduction in a California grassland: a comparison of restoration strategies. *Madrono* 46(1): 25-37.
- Milberg, P. and B.B. Lamont. 1995. Fire enhances weed invasion of roadside vegetation in southwestern Australia. *Biological Conservation* 73: 45-49.
- Nelson, J.F. and R.H. Dietz. 1966. Cattail control methods in Utah. Utah Department of Fish and Game Publication 66-2. 33pp.
- Sather, N. 1988. Element Stewardship Abstract: Canada thistle (*Cirsium arvense*). The Nature Conservancy. 15pp.
- Schwartz, M.W. and J.R. Helm. 1996. Effects of a prescribed fire on degraded forest vegetation. *Natural Areas Journal* 16(3): 184-190.
- Schwarzmeier, J.A. 1984. Sweet clover control in planted prairies: refined mow/burn prescription tested (Wisconsin). *Restoration and Management Notes* 2(1): 30-31.
- Smith, K.A. 1985. Canada thistle response to prescribed burning (North Dakota). *Restoration and Management Notes* 3: 87.
- Turkington, R.A., Cavers, P.B. and E. Rempel. 1978. The biology of Canadian weeds. 29. *Melilotus alba* Desr. and *M. officinalis* (L.) Lam. *Canadian Journal of Plant Science* 58: 523-537.
- Watson, A.K. and A.J. Renney. 1974. The biology of Canadian weeds. *Centaurea diffusa* and *C. maculosa*. *Canadian Journal of Plant Science* 54: 687-701.
- Whisenat, S.G. 1990. Post-fire population dynamics of *Bromus japonicus*. *American Midland Naturalist* 123: 301-308.
- Willson, G.D. 1990. Fire effects on three growth stages of smooth brome. *Proceedings 12th North American Prairie Conference, University of Northern Iowa, Cedar Falls, IA.*
- Willson, G.D. and J. Stubbendieck. 2000. A provisional model for smooth brome management in degraded tallgrass prairie. *Ecological Restoration* 18(1): 34-38.

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Chapter 4 – BIOLOGICAL CONTROL

John M. Randall and Mandy Tu

Biological control (biocontrol for short) is the use of animals, fungi, or other microbes to feed upon, parasitize or otherwise interfere with a targeted pest species. Successful biocontrol programs usually significantly reduce the abundance of the pest, but in some cases, they simply prevent the damage caused by the pest (e.g. by preventing it from feeding on valued crops) without reducing pest abundance (Lockwood 2000). Biocontrol is often viewed as a progressive and environmentally friendly way to control pest organisms because it leaves behind no chemical residues that might have harmful impacts on humans or other organisms, and when successful, it can provide essentially permanent, widespread control with a very favorable cost-benefit ratio. However, some biocontrol programs have resulted in significant, irreversible harm to untargeted (non-pest) organisms and to ecological processes. Of course, all pest control methods have the potential to harm non-target native species, and the pests themselves can cause harm to non-target species if they are left uncontrolled. Therefore, before releasing a biocontrol agent (or using other methods), it is important to balance its potential to benefit conservation targets and management goals against its potential to cause harm.

Organisms used to feed on, parasitize, or otherwise interfere with targeted pests are called biocontrol agents. There are several general approaches to using biocontrol agents: 1. 'Classical' biocontrol targets a non-native pest with one or more species of biocontrol agents from the pest's native range; 2. the 'New Association' or 'Neoclassical' approach targets *native* pests with non-native biological control agents; 3. 'Conservation', 'Augmentation' and 'Inundation' approaches maintain or increase the abundance and impact of biocontrol agents that are already present, and in many cases native to the area. Classical biocontrol is by far the most common approach for plant pests. Conservation and augmentation approaches show great promise on their own and especially for enhancing the impacts of classical biocontrol and other weed control measures as researchers and managers focus on managing to maximize native biological diversity in invaded ecosystems (Newman et al. 1998).

CLASSICAL BIOLOGICAL CONTROL OF WEEDS

It is hypothesized that some non-native plants become invasive, superabundant and damaging, at least in part because they have escaped the control of their 'natural enemies', the herbivores and pathogens that checked their abundance in their native ranges. Classical biocontrol addresses this by locating one or more herbivore and/or pathogen species from the weed's native range and introducing them so they can control the pest in its new range. These herbivores and pathogens are carefully selected and screened to determine if they will attack crops or other non-target plant species. Successful classical biocontrol programs result in permanent establishment of the control agent(s) and consequent permanent reduction in the abundance or at least the damaging impacts of the weed over all or in part of its introduced range. Classical biocontrol is not expected to eliminate the pest species completely and it often takes years or even decades after the initial release of control agents before their effects are obvious. Classical

biocontrol programs may fail for a variety of reasons. Some biocontrol agents never establish, or it may take repeated releases to establish viable populations. Some biocontrol agents may become established, but then have little or no detectable impact on the targeted pest (Greathead 1995).

Some of 'classical' biocontrol's greatest strengths are that once an agent is established, it will persist 'forever' and it may spread on its own to cover most or all of the area where the pest is present, generally with little or no additional cost. On the other hand, these strengths can become great liabilities if the agent also begins to attack desirable species (Pemberton 1985; Lockwood 1993, 2000; McEvoy and Coombs 2000). Because of this, weed biocontrol researchers take pains to locate and use agents that are specific to the targeted weed and will not attack other "important" plant species. This screening process contributes to the high cost and long time required for the discovery, testing, and approval of new biological control agents.

The selection and screening of candidate classical biocontrol agents

The first systematic biological control projects for weed species began over 100 years ago, and even at that time, potential control agents were tested to make sure that they did not harm agricultural crops. Scientific and public concern for native plant species with no known economic value has increased since then, particularly in the past few decades, and weed biocontrol programs administered by Agriculture Canada and the USDA expanded their host-specificity testing protocols to address these concerns. These programs now require checks for potential impacts on native plants, particularly rare species (DeLoach 1991; Harris 1988). This is in contrast to biocontrol programs that target insects and other arthropod pests, where even today, no host-specificity testing is legally required and few projects voluntarily screen potential control agents (Strong and Pemberton 2000). It has been suggested that this situation prevails because there is little public or professional outcry for the protection of insects, with the exception of non-native honeybees, other biocontrol agents, and possibly some native butterflies.

A key part of the screening process is host-testing, wherein potential control agents are given the opportunity to feed on a variety of crop species and native plants, including those most closely related to the targeted pest. No-choice tests isolate the potential control agent with one or more native species for feeding and/or egg-laying, so that if they do not use the native(s) they will die or fail to reproduce. Other tests give the proposed biocontrol agent a choice between feeding or reproducing on the targeted pest and non-target native species. Today, proposed biocontrol agents are screened for their ability to feed and reproduce on several to many native species, but it is still impossible to test all native species. For programs targeting species such as leafy spurge (*Euphorbia esula*) with many native congeners (over 100 native *Euphorbia* spp. in the U.S.), it is not even possible to test all the native species in the same genus. In addition, the tests cannot determine whether the control agents will adapt or evolve over time so that they will become more able or willing to feed on native species. For a more detailed description of the selection and host-testing processes, and suggestions for improving them, see McEvoy (1996).

McEvoy and Coombs (2000) argue that the potential effectiveness of candidate biocontrol agents has been given too little attention in the selection process. They note that ten or more species of biocontrol agents have been released against some weeds. Since there is some risk that each species will have unintended harmful impacts, the overall risk increases with the number of species released. In addition, some relatively ineffective species may actually interfere with and lessen the impacts of species that might be effective in their absence. Therefore, McEvoy and Coombs (2000) urge biocontrol practitioners to instead strive to release the minimum number of agents required to control the weed by first identifying and releasing only those species most likely to be effective. They advocate efforts to systematically identify traits common to successful control agents and the types of insects the target weed is most likely to be vulnerable to, based on its lifecycle and physiological attributes. Similarly, Louda et al. (1997) and Nechols (2000) advocate increased consideration of the interactions a candidate biocontrol agent is likely to have, with control agents and other organisms that are already present in the system.

Use of formal risk assessment procedures, efforts to minimize the number of agents released against a given target, and requiring follow-up studies designed to assess impacts on target and non-target species in order learn how to improve later programs would answer many of the concerns of conservation biologists (Miller and Aplet 1993; Simberloff and Stiling 1994; Strong and Pemberton 2000). The USDA has recently begun requiring post-release studies on the impacts of biocontrol agents for new releases in the U.S. (DeFosse personal communication), and is also considering the use of formal risk assessment procedures. Australia already has a legislative framework that requires a formal risk assessment before releases are granted which is designed to minimize nontarget impacts (McFayden 1998; Withers et al. 2000) and New Zealand is in the process of developing protocols for assessing and balancing risks and benefits of proposed introductions (Barratt et al. 2000)

Impacts of classical biocontrol on targeted weeds

Successful classical biocontrol projects reduce the abundance or impacts of the targeted pests to acceptable levels across large areas. There have been excellent post-release studies on Klamathweed (*Hypericum perforatum*) and tansy ragwort (*Senecio jacobaea*) biocontrol agents (Holloway and Huffaker 1951; Huffaker and Kennett 1959; McEvoy 1985; McEvoy and Rudd 1993; McEvoy et al. 1990; 1991; 1993), which provide quantitative information about reductions in the abundance of the target weeds. In each case significant reductions in the density of the targeted weeds were recorded after biocontrol agents were introduced.

Impacts of the four insects released to control purple loosestrife in the U.S. and Canada have also been monitored. The leaf feeding beetles *Galerucella pusilla* and *G. californiensis*, first introduced in 1992, have apparently reduced purple loosestrife stands at several sites already (Blossey et al., 1994; Scudder and Mayer, 1998). Results from release sites in Ontario, Michigan, and Minnesota indicate *Galerucella* beetles can significantly reduce above-ground abundance of purple loosestrife in as little as three years (Michigan State University, 1999). In southern Ontario, introductions of

Galerucella spp. reduced above ground purple loosestrife biomass from 2,000g/m² to less than 20g/m² in 4 years (The Ontario Biological Control Program, 1998). Additional studies found that at high *Galerucella* densities (200 larvae/plant), plants were entirely stripped of all green tissue and seed production was prevented (Butterfield et al., 1996). Even at lower beetle population densities, adult and early larval feeding destroyed meristematic regions thus, preventing normal growth. Nonetheless, it is not yet clear whether this feeding is significantly reducing the root biomass of established loosestrife stands.

Unfortunately, studies of the impacts of other biocontrol agents released against weeds have been extremely rare. For example, Lym and Nelson's recent (2000) paper on impacts of two flea beetle species released against leafy spurge is the only published study that quantifies population level impacts of any of the 13 insect biocontrol species released against this widespread pest in the U.S. and Canada. They found that both fleabeetles, *Aphthona lacertosa* and *A. czwallinae* reduced leafy spurge stem densities by about 65% up to 16 m from initial release sites within 3 to 5 years. A mixed population of both *Aphthona* species reduced stem densities by over 95% within 4 years after release. Establishment and rate of spread of these insects were similar regardless of the number of insects released initially. Unfortunately, qualitative before and after biocontrol release assessments of weed abundance are far more common.

Examples of weed biocontrol projects in North America that are regarded as having successfully reduced the abundance of the targeted species to acceptable levels include those to control Klamathweed (*Hypericum perforatum*), tansy ragwort (*Senecio jacobaea*), and alligatorweed (*Alternanthera philoxeroides*). Programs to control leafy spurge (*Euphorbia esula*) and purple loosestrife (*Lythrum salicaria*) appear to be on their way to at least partial success (Anderson et al. 2000). On the other hand, programs to control Canada thistle (*Cirsium arvense*), spotted and diffuse knapweed (*Centaurea maculosa* and *C. diffusa*) and yellow starthistle (*Centaurea solstitialis*) have not yet been successful, despite years of effort and releases of several insect species against each one.

Nontarget effects of classical biocontrol

Although biocontrol agents can be extremely selective against pest species, there is some risk that they may also attack desirable species. For example, the weevil *Rhinocyllus conicus* which was first introduced to North America to control non-native thistles in the 1960s has been documented attacking and significantly reducing seed set and reproduction of the untargeted native thistle species *Cirsium canescens* (Platte thistle) and *C. undulatum* (wavy-leaf thistle) (Louda et al. 1997; Louda 2000). Earlier studies determined that *R. conicus* feeds on several native *Cirsium* species, but they had not indicated whether or not this was causing population level impacts (Turner et al. 1987). Similarly, the cinnabar moth (*Tyria jacobaea*) that was introduced to control tansy ragwort (*Senecio jacobaea*), is known to attack native *Senecio triangularis* in Oregon (Diehl and McEvoy 1990).

Another example of a biocontrol agent causing significant damage to native plants involves the cactus moth, *Cactoblastis cactorum*, which was used with spectacular

success to control several introduced species of *Opuntia* in Australia and several Caribbean islands, and then spread inadvertently to Florida where it is damaging native *Opuntia* species. It was first released in Australia in 1925, and later to South Africa and to the islands of Nevis (1957), Montserrat and Antigua (1962) and Grand Cayman (1970) in the Caribbean (Habeck and Bennett 1990). It dispersed, apparently on its own, to Puerto Rico, Haiti, the Dominican Republic, and the Bahamas. It either spread on its own or was unintentionally imported from Hispaniola on ornamental cactus pads (Pemberton 1995) to south Florida, where it was first detected in 1989 (Habeck and Bennett 1990). Two species it has already attacked in Florida are rare, and one of them, *O. spinosissima*, has just one known U.S. population containing a total of less than a dozen plants. By 1997, *C. cactorum* had spread north to Jacksonville, Florida, and there are concerns that it will spread further north and west across the Gulf coast into Texas, and beyond to the southwestern U.S. and northern Mexico, where there are numerous native *Opuntias*, some of which are rare or are of economic importance (Habeck and Bennett 1990; Johnson 1994; Stiling and Simberloff 2000). Ironically, one way to address the threat posed to North American *Opuntia* species may be by releasing biocontrol agent(s) to control *C. cactorum*.

Recent research indicates that biological control agents may also have undesirable indirect impacts on nontarget plants and animals. Callaway et al (1999) found that when biocontrol insects (knapweed root moth; *Agapeta zoegana*) fed on the roots of spotted knapweed (*Centaurea maculosa*), neighboring Idaho fescue (*Festuca idahoensis*) plants actually did more poorly than when grown with unattacked *C. maculosa*. They also found that knapweeds fed on by another non-native root feeder (*Trichoplusia ni*) had smaller root systems and exuded more total sugars than knapweeds protected from attack. The authors hypothesize that moderate herbivory stimulated compensatory growth and production of defense chemicals that had allelopathic effects or otherwise altered the competitive relationship between the invasive knapweed and the native bunchgrass. A different study in west-central Montana found that two spotted knapweed biocontrol agents, the gall flies *Urophora affinis* and *U. quadrifasciata*, were the primary food item for deer mice (*Peromyscus maniculatus*) for most of the year and made up 84-86% of their winter diet (Pearson et al 2000). These deer mice tended to select microhabitats with high or moderate densities of knapweed when the gall flies were in their larval phase, but switched to sites dominated by native prairie after the gall flies emerged and were unavailable. In turn, deer mouse predation on the gall flies was so strong that the authors speculate it may prevent the flies from controlling spotted knapweed populations.

Benefits and risks of using classical biocontrol in conservation areas

Many conservation biologists have what might be called a “green light - yellow light” attitude towards the use of classical biological control against natural area weeds. On the one hand, classical biological control gets a ‘green light’ or ‘go ahead’ since it has the potential to be one of the most selective, powerful and cost-efficient tools available for control of invasive plants. It is an attractive option in natural areas particularly because of its potential for specificity and its ability to act over huge areas for the long term with little or no cost after the initial research and release(s) of agents. In addition, many find biocontrol preferable to the use of herbicides because of the danger these compounds

may pose to other organisms, including humans, especially if they enter water supplies or otherwise move from sites of application. Biocontrol may be the only affordable option capable of bringing certain widespread natural area weeds like tamarisk (*Tamarix* spp.), melaleuca (*Melaleuca quinquenervia*) and purple loosestrife (*Lythrum salicaria*) under control over large areas. As a result, many land managers and researchers have urged that particular widespread and difficult to control pests, be targeted for classical biocontrol.

On the other hand, biocontrol gets a ‘yellow light’ (some might even say a ‘red light’) for caution largely due to concerns that biocontrol agents may attack and damage populations of non-target native species. Natural area managers are typically concerned with the health and growth of a wide variety of organisms, far more species than most farmers, ranchers or foresters. If a biocontrol agent does in fact attack any native non-target species, its persistence and ability to spread to areas far from release sites become serious liabilities. It is widely believed that the potential for harm to non-target organisms can be decreased with improved host-testing and risk reduction protocols for biocontrol. While biocontrol offers great promise, it will provide long-term benefits to natural areas and biodiversity preservation only if it is practiced carefully and its potential risks are fully recognized and addressed. In Australia, biological control programs for natural area and wildland pests are better supported and regulated, and as a result, are expected to be more successful (E. Delfosse pers. comm.; McFayden 1998; Withers et al. 2000).

There is also concern about releases of classical biocontrol agents among some conservationists precisely because the agents are themselves non-native introductions. In some cases the agents may carry additional non-native parasite and commensal species. There has been at least one case in the past decade in which a biocontrol release unintentionally included a second non-native look-alike species that has now become established. Intentional introductions of non-native classical biological control agents may, however, contribute to global biodiversity by significantly reducing large populations of targeted non-native organisms that would otherwise reduce or threaten populations of native species.

Of course, it must be recognized that all courses of action against pest organisms, including that of taking no action, carry some risk to valued, non-targeted organisms. If no action is taken, the pest may continue to spread and reduce or eliminate valued native species, and in the worst cases, drastically alter community and ecosystem functioning (Vitousek 1986; Vitousek et al. 1987; Whisenant 1990). Pesticide use may directly kill valued species or indirectly impact them by reducing food supplies, eliminating cover or otherwise altering the environment. Mechanical methods often disturb the soil and destroy vegetation enabling ruderal plants and “weedy” pioneer species to gain a foothold. With all control methods, there is also the risk that when one pest is eliminated another will merely take its place, and that the infestation is merely the symptom of a more fundamental problem. For example, in Douglas County, OR, Klamathweed populations were sharply reduced by biocontrol agents only to be replaced by tansy ragwort (*Senecio jacobaea*), which was in turn sharply reduced by biocontrol agents only to be replaced by Italian thistles (*Carduus pycnocephalus*; E. Coombs pers. comm.).

Coombs believes that while successful biocontrol agents will likely be found for these thistles, they will only be replaced by another pest and then another in an endless substitution series, unless cultural practices in the area are changed.

Wildland weed species targeted for classical biocontrol

Julien and Griffiths (1998) catalogued a world total of 1,120 intentional releases of 365 species of biocontrol agents released against a total of 133 weed species in 75 countries between the late 1800s and 1996. Until the 1980s most biocontrol programs directed against invasive weeds in North America were funded and initiated primarily because the targeted species were troublesome in rangelands, commercial forests, or in waterways used for navigation or irrigation. Many of these weeds also invade conservation areas and other wildlands. In the past decade or so, there has been greater focus on weeds that invade natural areas, but have little impact on agriculture or forestry.

At least one biocontrol agent has been released in the U.S. (including Hawaii) and/or Canada against each of the wildland invasive plants listed in Tables 1a and 1b. The invaders listed in Table 1c are currently the subject of research and testing as possible targets for future biocontrol releases.

Table 1a. List of weeds with released/available biocontrol agents.

Latin Name	Common Name	Where Available
<i>Acroptilon repens</i>	Russian knapweed	mainland US
<i>Ageratina adenophora</i>	crofton weed	HI
<i>Ageratina riparia</i>	Hamakua pamakani	HI
<i>Alternanthera philoxeroides</i>	alligatorweed	mainland US
<i>Calystegia sepium</i>	hedge bindweed	mainland US
<i>Carduus acanthoides</i>	plumeless thistle	mainland US
<i>Carduus nutans</i>	musk thistle	mainland US
<i>Carduus pycnocephalus</i>	Italian thistle	mainland US
<i>Carduus tenuiflorus</i>	slenderflower thistle	mainland US
<i>Centaurea cyanus</i>	bachelor's button	mainland US
<i>Centaurea diffusa</i>	diffuse knapweed	mainland US
<i>Centaurea maculosa</i>	spotted knapweed	mainland US
<i>Centaurea pratensis</i>	meadow knapweed	mainland US
<i>Centaurea solstitialis</i>	yellow starthistle	mainland US
<i>Centaurea virgata</i> ssp. <i>squarrosa</i>	squarrose knapweed	mainland US
<i>Chondrilla juncea</i>	rush skeleton	mainland US

Table 1a (cont.). List of weeds with released/available biocontrol agents.

<i>Cirsium arvense</i>	Canada thistle	mainland US
<i>Cirsium vulgare</i>	bull thistle	mainland US
<i>Clidemia hirta</i>	Kosters curse	HI
<i>Coccinia grandis</i>	ivy gourd	HI
<i>Conium maculatum</i>	poison hemlock	mainland US
<i>Convolvulus arvensis</i>	field bindweed	mainland US
<i>Cyperus esculentus</i>	yellow nutgrass	mainland US
<i>Cyperus rotundus</i>	nut grass	HI
<i>Cytisus scoparius</i>	Scotch broom	mainland US
<i>Eichhornia crassipes</i>	water hyacinth	mainland US
<i>Elephantopus mollis</i>	tobacco weed	HI
<i>Emex australis</i>	three cornered Jacks	HI
<i>Emex spinosa</i>	lesser Jacks	HI
<i>Euphorbia cyparissias</i>	cypress spurge	mainland US
<i>Euphorbia esula</i>	leafy spurge	mainland US
<i>Halogeton glomeratus</i>	halogeton	mainland US
<i>Hydrilla verticillata</i>	hydrilla	mainland US
<i>Hypericum perforatum</i>	St. Johnswort	mainland US
<i>Lantana camara</i>	lantana	HI
<i>Linaria genistifolia ssp. dalmatica</i>	Dalmatian toadflax	mainland US
<i>Linaria vulgaris</i>	yellow toadflax	mainland US
<i>Lythrum salicaria</i>	purple loosestrife	mainland US
<i>Melaleuca quinquenervia</i>	Melaleuca	mainland US
<i>Melastoma malabathricum</i>	Indian rhododendron	HI
<i>Myrica faya</i>	firebush	HI
<i>Opuntia cordobensis</i>	Opuntia	HI
<i>Opuntia ficus-indica</i>	mission prickly pear	HI
<i>Opuntia littoralis</i>	prickly pear	mainland US
<i>Opuntia oricola</i>	prickly pear	mainland US
<i>Passiflora tripartita</i>	banana poka	HI
<i>Pistia stratiotes</i>	water lettuce	mainland US
<i>Pluchea odorata</i>	sour bush	HI
<i>Rubus argutus</i>	prickly FL blackberry	HI
<i>Salsola australis = S. kali, S. iberica</i>	Russian thistle	mainland US
<i>Salvia aethiopsis</i>	Mediterranean sage	mainland US
<i>Schinus terebinthifolius</i>	Brazilian pepper tree	HI
<i>Senecio jacobaea</i>	tansy ragwort	mainland US
<i>Silybum marianum</i>	milk thistle	mainland US
<i>Sonchus arvensis</i>	perennial sow-thistle	Canada
<i>Tamarix chinensis</i>	tamarisk	mainland US
<i>Tamarix gallica</i>	tamarisk	mainland US
<i>Tamarix parviflora</i>	tamarisk	mainland US
<i>Tamarix ramosissima</i>	tamarisk	mainland US
<i>Tribulus cistoides</i>	puncturevine	HI
<i>Tribulus terrestris</i>	puncturevine	mainland US
<i>Ulex euronaeus</i>	gorse	mainland US

Table 1b. List of weeds with available native biocontrol agents.

<i>Cirsium arvense</i>	Canada thistle	mainland US
<i>Convolvulus arvensis</i>	field bindweed	Canada
<i>Cyperus rotundus</i>	nut grass	mainland US
<i>Diospyros virginiana</i>	persimmon	mainland US
<i>Eichhornia crassipes</i>	water hyacinth	mainland US
<i>Morrenia odorata</i>	milkweed vine	mainland US
<i>Myriophyllum spicatum</i>	Eurasian watermilfoil	mainland US
<i>Opuntia ficus-indica</i>	Indian fig	HI
<i>Opuntia littoralis</i>	prickly pear	mainland US
<i>Opuntia oricola</i>	prickly pear	mainland US
<i>Solanum elaeagnifolium</i>	silverleaf nightshade	mainland US

Table 1c. List of weeds with biocontrol agents currently being researched.

Latin Name	Common Name
<i>Abutilon theophrasti</i>	velvetleaf
<i>Acroptilon repens</i>	Russian knapweed
<i>Alliaria petiolata</i>	garlic mustard
<i>Amaranthus</i> spp.	pigweeds
<i>Crupina vulgaris</i>	common crupina
<i>Cynoglossum officinale</i>	houndstongue
<i>Cyperus rotundus</i>	nut grass
<i>Cytisus scoparius</i>	Scotch broom
<i>Eichhornia crassipes</i>	water hyacinth
<i>Euphorbia esula</i>	leafy spurge
<i>Hieracium aurantiacum</i>	orange hawkweed
<i>Hieracium pilosella</i>	mouse-ear hawkweed
<i>Hieracium pratense</i>	yellow hawkweed
<i>Lantana camara</i>	lantana weed
<i>Ligustrum</i> spp.	privets
<i>Linaria dalmatica</i>	Dalmatian toadflax
<i>Linaria vulgaris</i>	yellow toadflax
<i>Mikania micrantha</i>	mile-a-minute weed
<i>Onopordum acanthium</i>	Scotch thistle
<i>Phragmites australis</i> *	common reed
<i>Polygonum perfoliatum</i>	mile-a-minute plant
<i>Potentilla recta</i>	sulfur cinquefoil
<i>Pueraria montana</i> var. <i>lobata</i>	kudzu
<i>Rhamnus cathartica</i>	buckthorn
<i>Rhamnus frangula</i>	Buckthorn
<i>Schinus terebinthifolius</i>	Brazilian peppertree
<i>Tripleurospermum perforatum</i>	scentless chamomile

*Native to at least some areas where regarded as an invasive weed of conservation areas

Excellent updates on natural area weed biocontrol projects are available at:
<http://www.cabi.org/BIOSCIENCE/weeds.htm>
<http://www.nysaes.cornell.edu/ent/biocontrol/weedfeeders/wdfdrtoc.html>

Use of classical biocontrol in North American conservation areas.

Classical biocontrol agents targeting a wide variety of invasive weeds have been released in North American conservation areas or spread into them from other release sites. For example, since their approval as biocontrol agents for purple loosestrife in 1992, four species of beetles have been released at hundreds of sites across the northern half of the U.S. and southern Canada. At least two of these species, the leaf feeding beetles *G. californiensis* and *G. pusilla*, have been released into conservation areas and other wildlands managed by public agencies and private conservation organizations including National Wildlife Refuges, U.S. and Canadian National Parks, and at least 6 Nature Conservancy preserves in 5 states. Blossey (personal communication) reported that there was strong evidence that one or more of these biocontrol insects are reducing cover and/or numbers of purple loosestrife at a variety of sites across North America including:

- 1.) Tonawanda Wildlife Refuge, western NY, 25 acres of a 50-acre infestation defoliated;
- 2.) Circle Lake, MN (southwest of Minneapolis) 30 acres defoliated;
- 3.) Coulee Dam, WA, large stands being defoliated;
- 4.) Providence Zoo, Providence, RI, 10 acres defoliated.

Successes were also reported from the John Heinz National Wildlife Refuge (Tinicum Marsh), PA; a wetland near Logan, UT, and from a wetland along the Mississippi in IL.

Similarly, several of the 13 biocontrol agents introduced to control leafy spurge have been released on conservation areas across the northern U.S. and southern Canada. The flea beetles, *Aphthona nigriscutis*, *A. czwalinae*, and *A. lacertosa* have been more successful than most of the other agents, and have been released on BLM lands, National Forests and Grasslands, and National Parks and Monuments such as Theodore Roosevelt N.P. and Devil's Tower N.M. in the U.S., and Spruce Woods N.P. in Canada. These three species have also been released in over a dozen Nature Conservancy preserves in Montana, the Dakotas, Iowa, and Minnesota. Unfortunately, although the first of these releases on preserves was made nearly 7 years ago, leafy spurge cover has been reduced on only small portions of some of these preserves, so far.

Several biocontrol agents have been released recently against target weeds in the U.S., which are primarily natural area invaders. Since 1997, tens of thousands of adults and larvae of *Oxyops vitiosa*, a weevil that feeds on the meristems of flowering branches of *Melaleuca quinquenervia* (punk tree), have been released at sites in and around the everglades of south Florida, including Big Cypress National Preserve, Everglades National Park, and Loxahatchee National Wildlife Refuge (Center et al. 2000). *M. quinquenervia* has invaded large areas of Florida from the vicinity of Lake Okeechobee and south through the Everglades, but it is hoped that this agent and perhaps others that are still being tested, will bring it under control. The insects are establishing and reproducing well at most release sites and by the year 2000, there were 83,000 adults and 137,000 larvae at a site where just 3,300 larvae were released in 1997 (Center et al.

2000). The weevils are damaging melaleuca plants at the release sites, but it is not yet clear whether it will be enough to reduce melaleuca abundance.

In 1997 the fungal pathogen *Colletrichum gleosporioides* f. sp. *miconiae* was released in two test zones on Maui and the Big Island of Hawaii against *Miconia calvescens*, a tropical American tree that invades wet forests in Hawaii (Kilgore et al. 1999). This fungus can kill seedlings and young *M. calvescens* plants, but its impacts on adult trees are unknown. Nonetheless, by 1999 all plants inoculated at the two sites were defoliated and the fungus had spread to surrounding plants (Kilgore et al. 1999).

In 1999 the weevil *Diorhabda elongata* was released in field cages at 8 sites in 6 states against tamarisk. This is the first agent released against tamarisks in North America. Among the 8 release sites, are lands managed by the BLM, Wyoming Game & Fish and the U.S. National Park Service. Researchers now have permission to release the insects outside of the cages. Some conservation land managers in the southwestern U.S. sought to halt or delay releases of this species because they feared it might act so quickly that they would quickly kill and destroy tamarisk groves which are used as nesting habitat by the endangered southwestern willow flycatcher (*Empidonax traillii extimus*). Because of this, no releases will be made within 200 miles of sites where the flycatchers are known to nest until it can be determined whether the biocontrol agents will quickly destroy tamarisk stands, and if so, whether native woody species suitable for nesting will quickly re-establish.

The Oregon Department of Agriculture (ODA) began to emphasize the use of biological control against weeds in the mid-1970s in response to public pressure to reduce the use of pesticides (Coombs et al. 1992). ODA has now introduced 42 species against 20 target pest plants and has focused much of its efforts on infestations on federal lands, which comprise the majority of the state's wildlands (Coombs 1991). The California Department of Food & Agriculture also operates a large biological control program that has given some attention to wildland pest infestations, although it is concerned primarily with insect pests of agricultural and ornamental plants (Bezark 1994). Hawaii's biological control program on weeds of forest areas was initiated in 1983 with joint funding from the Hawaii Department of Land and Natural Resources, the U.S. National Park Service, and the USDA Forest Service (Markham et al. 1992). USDA's programs against leafy spurge and purple loosestrife have also directed much effort towards work in wildlands (Malecki et al. 1993; P.C. Quimby pers. comm.).

The Nature Conservancy requires careful review and formal internal approval of all intentional biocontrol releases on its preserves in order to ensure that potential non-target impacts are minimized. The following text box outlines TNC policies on biocontrol releases and on requesting permission to intentionally release biocontrol agents on lands owned and managed by the organization.

**TNC POLICIES & STANDARD OPERATING PROCEDURES REGARDING INTENTIONAL
RELEASE OF BIOCONTROL AGENTS ON TNC LAND:**

TNC policy prohibits intentional releases of non-indigenous biological control agents on conservation lands that we own or manage. However, exceptions allowing releases on individual preserves may be approved by the Executive Director of TNC's Invasive Species Initiative (Ann Bartuska). This policy is designed to ensure non-indigenous biocontrol agents are used only when the potential benefits clearly outweigh the risks that they may attack and damage non-target native species populations. The policy, from page 17 of TNC's Policies and Procedures Manual, is copied below.

The standard operating procedure for requesting permission to release biocontrol agents from pages 24 and 25 of the Manual is copied below. A formal proposal must be submitted first to the Director of TNC's Wildland Invasive Species Team (John Randall) who will evaluate it and make a recommendation to the executive director of ISI. The proposal must address questions about the benefits and risks of the release, including how the agent was tested for host-specificity, whether it has been shown to reduce populations of the target pest in the field and how impacts of the proposed release will be monitored. Contact John for more details on the scope of the proposal and assistance in preparing it (John may be reached at 530 754 8890 or jarandall@ucdavis.edu).

1.) Intentional Release of Non-Indigenous Biocontrol Agents

POLICY:

The irreversible introduction or intentional release of non-native biological agents, except where required by law, is prohibited on conservation lands owned and/or managed by The Nature Conservancy. Note that this policy does not apply to the release of organisms (such as cattle or angora goats to control vegetation) that 1) cannot persist on the site without human assistance and/or 2) can be maintained at desirable levels or removed entirely by managers.

Exceptions may be approved by the Executive Director of TNC's Invasive Species Initiative.

PURPOSE:

The release and establishment of non-native organisms has had devastating and unforeseen impacts on non-target organisms, contributing, in some cases to the alteration of ecosystems and the extinction of native species. Releases are typically irreversible action with substantial ecological risks. Failure to comply with this policy could result in permanent damage to the species, natural communities, and ecosystems The Nature Conservancy seeks to protect. Furthermore, commercial enterprises, such as forestry or agricultural operations, could suffer extraordinary economic losses.

ORIGIN:

Approved by the Board of Governors on March 15, 1996. This policy also reflects sections of the old Stewardship Manual.

REFERENCES, RESOURCES, and EXPLANATORY NOTES:

Conservancy land managers and scientists may request an exception to this policy (see standard operating procedure: Requesting Permission to Release Non-Indigenous Biocontrol Agents). Exceptions may only be approved by the Executive Director of TNC's Invasive Species Initiative.

2.) Requesting Permission to Release Non-Indigenous Biocontrol Agents

STANDARD OPERATING PROCEDURE:

Exceptions to The Nature Conservancy's policy prohibiting intentional, irreversible introductions of any non-native species, including biocontrol agents, to preserves under its management, may be granted by the Director, Conservation Science Division, when it is deemed that the benefits of doing so clearly outweigh the risks. Exceptions to this policy may be made on the basis of a written proposal (see Explanatory Notes, below)

TNC Policies & Standard Operating Procedures (cont.)**PURPOSE:**

The Nature Conservancy prohibits intentional releases of biocontrol agents because some have been known to attack and feed on species other than those they were targeted to control. In Hawaii and other Pacific islands, predatory snails (*Euglandina rosea*) introduced to control the giant African snail (*Achatina fulica*) were responsible for severe reductions and extinctions of native snail populations. The mongoose (*Herpestes auropunctatus*), introduced for rat control, was responsible for reductions and extinctions of populations of native reptiles and birds on Pacific and Caribbean islands. A moth (*Cactoblastis cactorum*) released to control prickly pear cacti (*Opuntia* spp.) in the Caribbean dispersed to south Florida where it now attacks native cacti, including the G1 species *Opuntia spinosissima*. These incidents point to the need for great caution in use of biocontrol agents. However, in some cases, use of biocontrol agents may be the only effective method to control pests, and Nature Conservancy land managers should be able to request permission to use biocontrol agents in appropriate situations.

ORIGIN:

This procedure was developed pursuant to Board-approved policy governing the intentional release of non-indigenous biocontrol agents and also reflects sections of the old Stewardship Manual.

REFERENCES, RESOURCES and EXPLANATORY NOTES:

To request permission to release a non-native biological control agent, a formal proposal must be submitted to The Nature Conservancy's Weed Specialist. Contact the Weed Specialist for more details on the scope of the proposal and assistance in preparing it. The Weed Specialist will evaluate the proposal and make a recommendation to the Director, Conservation Science Division, who has sole authority to grant exceptions to the policy prohibiting releases of biocontrol agents. Each introduction at each site will be considered separately and will require a separate proposal. At a minimum, each proposal must address the following points.

1. The target organism (plant or animal pest) is itself a non-native species and has been shown to be a serious threat to the ecosystem, natural communities, and/or species being protected.
2. Other measures (physical, chemical, or cultural), singly and in combination, have failed to adequately control the target organism or are judged to have potential to cause greater damage than the introduced biocontrol agent.
3. Research on diet and behavior of the biocontrol agent indicates it will not attack non-target native species.
4. Potential for the biocontrol agent to displace native species (e.g. other insects) through competition for food, nest sites, etc. has been considered and judged to be slight.
5. The biocontrol agent has been judged successful at reducing populations of the target species at other sites where it has been released.
6. The identity of the biocontrol agent can be verified, preferably by an independent laboratory or museum.
7. A monitoring program to assess effects of the biocontrol agent on populations of the target species and selected non-target species (especially the target's congeners and other closely related species) within the dispersal range of the biocontrol agent has been designed and will be implemented.
8. Observations made during the monitoring program will be fully documented in-house (within The Nature Conservancy). Reports may also be published in scientific and resource management journals. Because release of a biocontrol agent is intended to kill or reduce viability of other organisms, Nature Conservancy employees requesting permission to use biocontrol agents should be familiar with the policy: Removal of Plants and Animals and the standard operating procedure: Decision-Making Process for Removal of Plants and Animals.

ORIGINATING DIVISION:

Conservation Science

Obtaining and releasing Classical biocontrol agents

It is best to obtain biocontrol agents locally, if possible, as this will minimize losses in storage and transport and increase the likelihood that the agents can survive in the local environment. It is also wise to start lining up a supply of agents several months before you will need them. Most can be obtained from state or county noxious weed or biological control programs. They will often be free, but there may be a charge of \$0.25 to \$2 or more per insect for certain species that are difficult to breed or which were recently introduced and are not yet abundant. Another possible source of information on where to get insects is the USDA-APHIS Plant Protection and Quarantine office in your state (every state has one).

Contacts for several western states include:

Montana: USDA-APHIS 406 657 6282; Jerry Marks, Montana State Extension Service 406 721 4095, acxgm@montana.edu

North Dakota: USDA-APHIS 701 520 4473; Dave Nelson, North Dakota Dept. of Agriculture state entomologist, 701 328 4765, dnelson@state.nd.us

South Dakota: USDA-APHIS 605 224 1713; Ron Moehring, South Dakota Dept of Agriculture weed pest coordinator, 605 773 3796, ron.moehring@state.sd.us

Wyoming: USDA-APHIS 307 772 2323; Lars Baker, Fremont County Weed and Pest, 307 332 1052

There are also several websites with good information about specific weed biocontrol agents and how to obtain them:

<http://www.for.gov.bc.ca/hfp/pubs/interest/bioagent/bioagent.htm>

<http://www.nysaes.cornell.edu/ent/biocontrol/weedfeeders/wdfdrtoc.html> (particularly good for information on purple loosestrife control agents)

The Team Leafy Spurge homepage (<http://www.team.ars.usda.gov/>) has excellent information on biocontrol, including an excellent downloadable 24-page booklet titled ‘ Biological control of leafy spurge ’ with excellent advice on obtaining and releasing insects.

(Some weed biocontrol agents are also available from commercial suppliers. You can download a publication with a list of 143 suppliers of 130 organisms used for biocontrol of weeds, insects and other pests from the California EPA Department of Pesticide Regulation website at: <http://www.cdpr.ca.gov/docs/ipminov/bensuppl.htm>.)

Your choice of release sites may have a great deal of influence on the establishment and subsequent multiplication and spread of the agents. Optimum release sites differ for different agents and target species. The agency or company that supplies or gives you advice on how to collect biocontrol agents should be able to give you advice on selecting release sites. Important considerations include soil type and moisture, density of the target weed, exposure, aspect and shade.

The number of agents released at a particular site can also be of great importance in some species but for others any release of 50 to 100 individuals or more have the same chance of succeeding and

spreading rapidly. Once again the agency or company that supplies the agents should be able to advise you.

Finally, it is important to mark and map each release site so that impacts on the target weed can be monitored. The easiest way to mark sites is with metal or fiberglass fenceposts. At a minimum, before and after photographs should be taken at the same spot, time of day, and date and with the same (or similar) equipment.

OTHER BIOLOGICAL CONTROL APPROACHES

Conservation biocontrol

Conservation biocontrol is usually defined as actions that preserve, protect, or promote the abundance of organisms that may keep the abundance of another, pest organism in check (Ehler 1998). Usually this entails modifying the environment in ways that promote the abundance and/or impact of native or already established non-native organisms. To date, this approach has received relatively little attention for weed control. Studies to understand and enhance the impacts of two native insects species, and especially the weevil *Euhrychiopsis lecontei* on the non-native invasive Eurasian watermilfoil (*Myriophyllum spicatum*) are an exception to this (Creed and Sheldon 1995; Sheldon and Creed 1995). The weevil actually favors the non-native *M. spicatum* over its native host *M. sibiricum* but nonetheless it effectively controls *M. spicatum* only in some situations (Sheldon and Creed 1995; Solarz and Newman 1996). Researchers are currently studying the factors that limit the weevil's effectiveness in hopes of finding ways to enhance it. Competition from native aquatic plants, refugia from bluegill predation for the weevils in dense beds of native plants, and adequate shoreline overwintering habitat may all play a role in the success of the weevil (Newman et al 1998). These and other factors could be manipulated to enhance control of Eurasian watermilfoil.

There may be great gains to be made by focusing more attention and resources on conservation biocontrol approaches to management of weeds in conservation areas and other wildlands (Newman et al 1998). Native insects and pathogens will work only against some invasive plants and only in some situations but they are also less likely to have unintended harmful effects on nontarget species that exotic biocontrol agents, herbicides and other control methods have. In addition, the conservation approach can help enhance the impacts of non-native biocontrol agents that were intentionally or unintentionally introduced, perhaps in ways that will help reduce the necessity to use other, riskier control methods.

Inundative biocontrol

The "Inundative" or "augmentative" biocontrol approach uses mass releases of predators, herbivores, or pathogens, that are already present but whose effects on the target are normally limited by their ability to reproduce and spread. To date, this approach has been more commonly used against insect pests. "Inundative" biocontrol agents that are non-native and/or not target specific, such as the grass carp (*Ctenopharyngodon idella*) used to control aquatic vegetation, may be sterilized or otherwise rendered incapable of establishing permanent populations before they are released. Because they either fail to

establish or do not remain abundant enough to control the pest, they must be reared and released again each time the pest population erupts. There have, however, been instances in which mistakes or back-mutations allowed purportedly sterile control agents to establish permanent wild populations.

New Association (or Neoclassical) biocontrol

The “new association” technique in which non-indigenous control agents are introduced to control native pests, was first proposed by Pimentel in 1963. Later articles by Hokkanen and Pimentel provided more support for this technique (1984, 1989). This inspired programs to develop biocontrol for native organisms ranging from grasshoppers to mesquite (*Prosopis glandulosa* and *P. velutina*) and broom snakeweed (*Gutierrezia sarothrae*) (Carruthers and Onsager 1993; DeLoach 1981; 1985). Proponents of these programs seek agents that are host-specific and capable of reducing populations of species regarded as economic pests to acceptable levels. They point out that successful programs could result in great reductions in pesticide use and concomitant environmental damage. This technique has been tried in several instances in North America, most recently with the release of an Australian fungal pathogen to control native grasshoppers in North Dakota and Alaska (Carruthers and Onsager 1993). An earlier case, the highly successful program to control native prickly pear cacti *Opuntia littoralis* and *O. oricola* with the introduced cochineal insect *Dactylopius opuntiae*, is notable for two reasons: it was begun in 1939 long before Pimentel’s proposal and; it was carried out on Santa Cruz Island on land now managed as a preserve by The Nature Conservancy (Goeden and Ricker 1981). There is some evidence that *Pistia stratiotes*, or water lettuce, may be native to the southeastern U.S. and it has also been the target of several foreign biocontrol introductions (Julien 1992; D. Habeck personal communication). *Phragmites australis* is also native to North America and is currently the subject of research designed to identify and screen organisms from other continents where it is also native that might reduce its abundance here. It has been suggested that non-native biotypes of *Phragmites* have been introduced to North America and are behaving aggressively so a program to control this species does not necessarily fit neatly into the ‘classical biocontrol’ or ‘new association’ categories

Many native species targeted for control by exotic species, however, are ecological dominants in natural as well as in disturbed environments (Pemberton 1985). Examples include the mesquites (*Prosopis glandulosa* and *P. velutina*), creosote bush (*Larrea tridentata*), and big sagebrush (*Artemisia tridentata*). As dominants, these species are of critical importance in natural areas. Significant reduction in their populations would alter the communities they dominate, perhaps rendering the communities unrecognizable and useless as habitat for many other native species. Such damage has been caused by forest pests such as chestnut blight and Dutch elm disease, which were accidentally introduced to North America.

Other native species that have been targeted such as *Astragalus wootonii* are less conspicuous, but nonetheless important members of native communities (Pemberton 1985). Some of these plants may provide the main source of support for certain herbivores and pollinators. Lockwood (1993) noted that control agents considered for

release on pest grasshoppers would likely attack non-target native grasshoppers. He pointed out that one grasshopper species likely to be hit this way, *Hesperotettix viridis*, feeds on and may limit populations of native snakeweeds (*Gutierrezia* spp) that are considered range weeds. Thus, an inadvertent effect of this program could be to allow rangeweeds populations to expand. Lockwood's (1993) cost/benefit analysis of the grasshopper control program suggests that control agents will likely be greater liabilities than assets, even on rangelands. Their impacts on natural areas, where native insect diversity is valued, would likely be even more detrimental.

Pemberton (1985) and Lockwood (1993) both note that the ability of biocontrol agents to spread and perpetuate themselves becomes a clear liability when native species are targeted. Control techniques that are more confined in space and time should be used against native pests. This might include other biologically based techniques as well as pesticides, mechanical and cultural methods. Pemberton (1985) and Lockwood (1993) also note that when grazing and other harvest practices promote native pests, alteration of these practices may well be the best way to address the problem.

OTHER BIOLOGICALLY-BASED WEED CONTROL TECHNIQUES

Compounds derived from several pathogenic organisms have shown promise for use as bioherbicidal agents against wildland pests but development of delivery systems for some has proven difficult (Prasad 1992; Prasad 1994). For example, Gary Strobel of Montana State University and his students isolated a compound toxic to spotted knapweed (*Centaurea maculosa*) from cultures of *Alternaria alternata*, a fungal pathogen specific to it (Kenfield et al. 1988; Stierle et al. 1988). The compound, named maculosin, may be produced synthetically and may find use as a species-specific herbicide against *C. maculosa* which infests natural areas across much of the northern U.S. A few other mycoherbicides have been developed and some were marketed for short periods but only one, which controls a vine pest in Florida citrus orchards, was effective enough to be commercially successful. The best known biopesticides have been derived from various strains of *Bacillus thuringiensis* (B_t) and used against insect pests, particularly lepidoptera (moth and butterfly caterpillars). In the past few years plants that have been genetically manipulated to produce B_t on their own have been released for sale and the subject of intense controversy due to questions about the effects of such widespread presence of this compound in agroecosystems and in human food.

Mixing fungal bioherbicides (also called mycoherbicides) with pesticides can increase or decrease the severity of diseases they cause (Altman and Campbell 1977; Katan and Eshel 1973). Some adjuvants may sharply increase the severity of disease by allowing pathogens to penetrate plants where they otherwise would have difficulty (Wymore and Watson 1986). Certain growth-regulators have also been shown to enhance the effectiveness of bioherbicides (Wymore et al. 1987). In a few instances it has been found that sunscreens help extend shelf-life of bioherbicides presumably by protecting the active agents from harmful ultraviolet radiation (Morris 1983; Prasad 1994). Prasad (1994) also suggests that the addition of rainfastness agents may enhance the effectiveness of some bioherbicides. Different bioherbicides will probably require different mixtures of additives and different delivery systems to insure maximum effectiveness and these

will likely be discovered both by further research and by trial-and-error as more people attempt to use them.

INTEGRATION OF BIOCONTROL WITH OTHER CONTROL METHODS

Although biocontrol is often seen as an alternative to other methods, particularly herbicides, it can in fact be used in combination with them. Such combinations may interfere with or enhance each other. For example, prescribed fires could sharply reduce populations of biocontrol agents if lit when the agents are exposed and unable to flee, but the timing, frequency and spatial distribution of the burns might be adjusted so that they do not interfere or harm the agents, and may perhaps even enhance their impacts (Briese 1996). Likewise, mowing and other mechanical treatments can be timed or adjusted to enhance biocontrol. For example, mowing the thistle *Carduus thoermeri* at the bud or bloom stage significantly reduces populations of the biocontrol agent *Rhinocyllus conicus*, but mowing later in the season, after the primary inflorescences have senesced, actually enhances control by chopping lateral inflorescences usually missed by *R. conicus* (Tipping 1991).

Herbicide applications can also interfere with or enhance biocontrol. In most cases the interference is indirect and results from the reduction in food supply or other habitat changes caused by herbicide. Such indirect interference can sometimes be mitigated by leaving untreated areas where high populations of the control agent can survive and re-colonize treated areas if and when the weed re-appears there (Haag and Habeck 1991); of course these untreated sites may also provide the weed seed that re-colonizes the treated area! It has been hypothesized that sub-lethal doses of herbicide may make leafy spurge more attractive or nutritious to biocontrol agents and therefore enhance their impacts (Carrithers, personal communication). Similarly, application of plant growth retardants such as EL-509 and paclobutrazol can actually enhance the effectiveness of water hyacinth weevils by preventing the plants from outgrowing the damage inflicted by the weevil (Van and Center 1994; Newman et al 1998).

Addition of nutrients to an infested site may seem counterproductive, but in some cases it may help by making the weed nutritious enough to support rapid population increase of a biocontrol agent. For example, addition of nitrogen to nutrient poor waters infested by *Salvinia molesta* increased the weed's acceptability and nutritional quality for two biocontrol agents, and allowed one to increase to densities sufficient to effect control (Room et al. 1989; Room 1990; Room and Fernando 1992).

REFERENCES

- Altman, J. and C.L. Campbell. 1977. Effects of herbicides on plant diseases. Annual Review of Phytopathology 15: 361-365.
- Anderson, G.L., E.S. Delfosse, N.R. Spencer, C.W. Prosser and R.D. Richard. 2000. Ecologically-based integrated pest management of leafy spurge: an emerging success story. Pp 15-25. In Proceedings X International Biological Control Symposium. Bozeman, Montana 4-9 July 2000.
- Barratt, B.I.P., C.M. Ferguson, S.L. Golson, C.M. Phillips and D.J. Hannah. 2000. Predicting the risk from biological control agent introductions: a New Zealand

- approach. Pp. 59-75. In P.A. Follett and J.J. Duan (eds.) Nontarget effects of biological control. Kluwer Academic Publishers. Boston, Massachusetts.
- Bezark, L.G. (ed.) 1994. Biological control program annual summary, 1993. California Department of Food and Agriculture, Division of Plant Industry, Sacramento, CA.
- Blossey, B.D., Schroeder, D., Hight, S.D. and R.A. Malecki. 1994. Host specificity and environmental impact of two leaf beetles (*Galerucella californiensis* and *G. pusilla*) for biological control of purple loosestrife (*Lythrum salicaria*). *Weed Science* 42: 134-140.
- Briese, D.T. 1996. Biological control of weeds and fire management in protected natural areas: are they compatible strategies? *Biological Conservation* 77: 135-141.
- Butterfield, C., Stubbendieck, J., and J. Stumpf. 1996. Species abstracts of highly disruptive exotic plants. Jamestown, ND: Northern Prairie Wildlife Research Center Home Page. <http://www.npwrc.usgs.gov/resource/othrdata/exoticab/exoticab.htm> (Version 16JUL97).
- Callaway, R.M., T DeLuca and W.M. Belliveau. 1999. Biological control herbivores may increase competitive ability of the noxious weed *Centaurea maculosa*. *Ecology* 80(4): 1196-1201.
- Carrithers, V. Dow AgroSciences. Personal communication to John Randall, March 1997.
- Carruthers, R.I. and J.A. Onsager. 1993. Perspective on the use of exotic natural enemies for biological control of pest grasshoppers (Orthoptera: Acrididae). *Environmental Entomology* 22:885-903.
- Center, T. D., T.K. Van, M. Rayachhetry, G.R. Buckingham, F.A. Dray, S.A. Wineriter, M.F. Purcell and P.D. Pratt. 2000. Field colonization of the Melaleuca Snout Beetle (*Oxyops vitiosa*) in South Florida. *Biological Control* 19: 112-123.
- Coombs, E.M. 1991. Implementation of biological control on Federal lands in Oregon. In Proceedings of the First Oregon Interagency Noxious Weed Symposium. 3-4 December, 1991, Corvallis, OR. Oregon Department of Agriculture, Salem.
- Coombs, E.M., Biological Control Entomologist, Noxious Weed Control, Oregon Department Of Agriculture, Salem, OR, personal communication to J.M. Randall, November 1994.
- Coombs, E.M., D.L. Isaacson and R.B. Hawkes. 1992. The status of biological control of weeds in Oregon. In E.S. Delfosse and R.R. Scott (eds.) Proceedings of the Eighth International Symposium on Biological Control of Weeds. 2-7 February 1992, Canterbury, New Zealand. DSIR/CSIRO, Melbourne.
- Creed, R.P. and S.P. Sheldon. 1995. Weevils and watermilfoil: did a North American herbivore cause the decline of an exotic plant? *Ecological Applications* 5: 1113-1121.
- Delfosse, E.S., Director National Biological Control Institute, Animal and Plant Health Inspection Service, U.S. Department of Agriculture, Hyattsville, MD, personal communication to J.M. Randall, September 1994.
- DeLoach, C.J. 1981. Prognosis for biological control of weeds of southwestern U.S. rangelands. pp. 175-199 In E.S. Delfosse (ed.) Proceedings of the V International Symposium on the Biological Control of Weeds, 22-27 July 1980, Brisbane, Australia. CSIRO, Melbourne.
- DeLoach, C.J. 1985. Conflicts of interest over beneficial and undesirable aspects of mesquite (*Prosopis* spp.) in the United States as related to biological control. pp. 301-

- 340 In E.S. Delfosse (ed.) Proceedings of the VI International Symposium on the Biological Control of Weeds, 19-25 August 1984, Vancouver, Canada. Agriculture Canada.
- DeLoach, C.J. 1991. Past Successes and current prospects in biological control of weeds in the United States and Canada. *Natural Areas Journal* 11:129-142..
- Diehl, J. and P.B. McEvoy. 1990. Impact of the cinnabar moth (*Tyria jacobaea*) on *Senecio triangularis*, a nontarget native plant in Oregon. Pp. 119-126. In E.S. Delfosse (ed.) Proceedings of the VII International Symposium on Biological Control of Weeds, 6-11 March 1988. Ministero dell' Agricolturae delle Foreste, Rome, and CSIRO, Melbourne.
- Ehler, L.E. 1998. Conservation biological control: past, present and future. Pp. 1-8. In P. Barbosa (ed.) Conservation biological control. Academic Press, San Diego.
- Goeden, R.D. and D.W. Ricker. 1981. Santa Cruz Island - revisited. Sequential photography records the causation, rates of progress, and lasting benefits of successful biological weed control. pp. 355-365 In E.S. Delfosse (ed.) Proceedings of the V International Symposium on the Biocontrol of Weeds. July 22-27 1980, Brisbane, Australia. CSIRO, Melbourne.
- Greathead, D.J. 1995. Benefits and risks of classical biocontrol. Pp 53-63 In H.M. Hokkanen and J.M. Lynch (eds.) Biological control: benefits and risks. Cambridge University Press, Cambridge, U.K.
- Haag, K.H. and D.H. Habeck. 1991. Enhanced biological control of waterhyacinth following limited herbicide application. *Journal of Aquatic Plant Management* 29: 55-57.
- Habeck, D.H. Professor, University of Florida, Gainesville, Florida, personal communication to J.M. Randall, October, 1994.
- Habeck, D.H. and F.D. Bennett. 1990. *Cactoblastis cactorum* Berg (Lepidoptera: Pyralidae) a Phycitine new to Florida. Entomology Circular N. 333, August 1990, Florida Department of Agriculture and Consumer Services, Division of Plant Industry, Tallahassee
- Harris, P. 1988. Environmental impact of weed-control insects. *BioScience* 38: 542-548.
- Hokannan, H. and D. Pimentel. 1984. New approach for selecting biological control agents. *Canadian Entomologist* 116:1109-1121.
- Hokannan, H. and D. Pimentel. 1989. New associations in biological control: theory and practice. *Canadian Entomologist* 121:829-840
- Holloway, J.K. and C.B. Huffaker. 1951. The role of *Chrysolina gemellata* in the biological control of Klamath weed. *Journal of Economic Entomology* 44:244-247.
- Huffaker, C.B. and C.E. Kennett. 1959. a ten-year study of vegetational changes associated with biological control of Klamath weed. *Journal of Range Management* 12:69-82.
- Johnson, D.M. 1994. M.S. thesis, University of Central Florida.
- Julien, M.H. (ed.). 1992. Biological control of weeds: a world catalogue of agents and their target weeds, 3rd edition. Commonwealth Agricultural Bureaux, Wallingford, United Kingdom.
- Julien, M.H. and G.W. Griffiths (eds.) 1998. Biological control of weeds: a world catalogue of agents and their target weeds. CABI Publishing, CAB International, Wallingford, United Kingdom. 240 pp.

- Katan, J. and Y. Eshel. 1973. Interaction between herbicides and plant pathogens. *Residue Review* 45:145-177.
- Kenfield, D., G. Bunkers, G. A. Strobel and F. Sugawara. 1988. Potential new herbicides - phytotoxins from plant pathogens. *Weed Technology* 2:519-524.
- Kilgore, E.M. L.S. Sugiyama, R.W. Baretto and D.E. Gardner. 1999. Evaluation of *Colletrichum gleosporioides* for biological control of *Miconia calvescens* in Hawai'i. *Plant Disease* 83(10): 964
- Lockwood, J.A. 1993. Environmental issues involved in biological control of rangeland grasshoppers (Orthoptera: Acrididae) with exotic agents. *Environmental Entomology* 22:503-518.
- Lockwood, J.A. 2000. Nontarget effects of biological control: what are we trying to miss? Pp. 15-30 In P.A. Follett and J.J. Duan (eds.) Nontarget effects of biological control. Kluwer Academic Publishers. Boston, Massachusetts.
- Louda, S.M. 2000. Negative ecological effects of the musk thistle biological control agent, *Rhinocyllus conicus*. Pp. 215-243 In P.A. Follett and J.J. Duan (eds.) Nontarget effects of biological control. Kluwer Academic Publishers. Boston, Massachusetts.
- Louda, S.M., D. Simberloff, J. Conner, G. Boettner, D Kendall and A Arnett. 1997. Insights from data on the nontarget effects of the flowerhead weevil. *Biological Control News and Information*. 19:70-72.
- Lym, R.G. and J. A. Nelson. 2000. Biological control of leafy spurge (*Euphorbia esula*) with *Apthona* spp. along railroad right-of-ways. *Weed Technology*. 14: 642-646.
- Malecki, R.A., B. Blossey, S.D. Hight, D. Schroeder, L.T. Kok and J.R. Coulson. 1993. Biological control of purple loosestrife. *BioScience* 43(10):680-686.
- Markham, G.P., P-Y. Lai and G. Y. Funasaki. 1992. Status of biological control of weeds in Hawaii and implications for managing native ecosystems. pp. 466-482 In C.P. Stone, C.W. Smith and J.T. Tunison (eds.) Alien plant invasions in native ecosystems of Hawai'i: management and research. Cooperative National Park Resources Studies Unit, University of Hawaii, Manoa.
- McEvoy, P.B. 1985. Depression in ragwort (*Senecio jacobaea*) abundance following introduction of *Tyria jacobaeae* and *Longitarsus jacobaeae* on the central coast of Oregon. pp. 57-64 In E.S. Delfosse (ed.). Proceedings of the VI international symposium on biological control of weeds, 19-25 August 1984. Vancouver, Canada. Agriculture Canada, Ottawa.
- McEvoy, P.B. 1996. Host specificity and biological pest control. *BioScience* 46: 401-405.
- McEvoy, P.B. and E.M. Coombs. 2000. Why things bite back: unintended consequences of biological weed control. Pp. 167-194 In P.A. Follett and J.J. Duan (eds.) Nontarget effects of biological control. Kluwer Academic Publishers. Boston, Massachusetts.
- McEvoy, P.B., C.S. Cox and E. Coombs. 1991. Successful biological control of ragwort, *Senecio jacobaea*, by introduced insects in Oregon. *Ecological Applications* 1:430-442.
- McEvoy, P.B., C.S. Cox, R.R. James and N.T. Rudd. 1990. Ecological mechanisms underlying successful biological weed control: field experiments with ragwort *Senecio jacobaea*. pp. 55-66 In E.S. Delfosse (ed.). Proceedings of the VII international symposium on biological control of weeds, 6-11 March 1988, Rome, Italy. Ministero dell'Agricoltura e delle Foreste, Rome/CSIRO Melbourne, Australia.

- McEvoy, P.B. and N.T. Rudd. 1993. Effects of vegetation disturbances on insect biological of tansy ragwort, *Senecio jacobaea*. *Ecological Applications* 3:682-698.
- McEvoy, P.B., N.T. Rudd, C.S. Cox and M. Huso. 1993. Disturbance, competition and herbivory effects on ragwort *Senecio jacobaea* populations. *Ecological Monographs* 63:55-75.
- McFayden, R.E. 1998. Biological control of weeds. *Annual Review of Entomology*. 43: 369-393.
- Michigan State University, 1999. Michigan State University Purple Loosestrife Project Home Page. www.msue.msu.edu/seagrant/ppl/html/the_project.html
- Miller, M. and G. Aplet. 1993. Biological control: a little knowledge is a dangerous thing. *Rutgers law Review* 45:285-334.
- Morris, O.N. 1983. Protection of *Bacillus thuringiensis* from inactivation by sunlight. *Canadian Entomologist* 115:1215-1227.
- Nechols, J.R. 2000. Biological control of musk thistle: a reassessment. Pp 245-259 In P.A. Follett and J.J. Duan (eds.) *Nontarget effects of biological control*. Kluwer Academic Publishers. Boston, Massachusetts.
- Newman, R.M., D.C. Thompson and D.B. Richman. 1998. Conservation Strategies for the biological control of weeds. Pp. 371-396. In P. Barbosa (ed.) *Conservation biological control*. Academic Press, San Diego.
- Ontario Biological Control Program. 1998. Biocontrol Insects Feast on Purple Loosestrife. Manitoba Purple Loosestrife Project Home Page. www.ducks.ca/purple/biocontrol/biocon2.htm
- Pearson, D.E., K.S. McKelvey and L.F. Ruggiero. 2000. Non-target effects of an introduced biological control agent on deer mouse ecology. *Oecologia* 122: 121-128.
- Pemberton, R.W. 1985. Native weeds as candidates for biological control research. pp. 869-877 In E.S. Delfosse (ed.) *Proceedings of the VI International Symposium on the Biological Control of Weeds*. 19-25 August 1984, Vancouver, Canada. Agriculture Canada.
- Pemberton, R.W. 1995. *Cactoblastus cactorum* in the United States: an immigrant biological control agent or and introduction of the nursery industry. *American Entomologist* 41:230-232.
- Pimentel, D. 1963. Introducing parasites and predators to control native pests. *Canadian Entomologist* 95:785-782. In E. Delfosse (ed.) *Proceedings of the VI Symposium on Biological Control of Weeds*, August 1984, Vancouver. Agriculture Canada.
- Prasad, R. 1992. Some aspects of biological control of weeds in forestry. *Proceedings of the 1st International Congress on Weed Control* 2:398-402.
- Prasad, R. 1994. Influence of several pesticides and adjuvants on *Chondrostereum purpureum* - a bioherbicide agent for control of forest weeds. *Weed Technology* 8:445-449.
- Quimby, P.C., Research Leader, Biological Control of Weeds Research Unit, Rangelands Weed laboratory, Agricultural Research Service, U.S. Department of Agriculture, Bozeman, MT, personal communication to J.M. Randall, July 1994.
- Room, P.M. 1990. Ecology of a simple plant herbivore system: biological control of *Salvinia*. *Trends in Ecology and Evolution* 5: 74-79.
- Room, P.M. and I.V.S. Fernando. 1992. Weed invasions countered by biological control: *Salvinia molesta* and *Eichhornia crassipes* in Sri Lanka. *Aquatic Botany* 42:99-107.

- Room, P.M., M.H. Julien and I.W. Forno. 1989. Vigorous plants suffer most from herbivores: latitude, nitrogen and biological control of the weed *Salvinia molesta*. *Oikos* 54:92-100.
- Scudder, T., and M. Mayer. 1998. Release of *Galerucella californiensis* and *Galerucella pusilla* (Coleoptera: Chrysomelidae) to Control Purple Loosestrife, *Lythrum salicaria*. New Jersey Department of Agriculture Home Page. www.state.nj.us/agriculture/plant/pls98.pdf
- Sheldon, S.P. and R.P. Creed. 1995. Use of a native insect as a biological control for an introduced weed. *Ecological Applications* 5: 1122-1132.
- Simberloff, D. and P. Stiling. 1994. Risks of species introduced for biological control. in prep.
- Solarz, S.L. and R.M. Newman. 1996. Oviposition specificity and behavior of the watermilfoil specialist *Euhrychiopsis lecontei*. *Oecologia* 106: 337-344.
- Stierle, A.C. J.H. Cardellina II and G.A. Strobel. 1988. Maculosin, a host-specific phytotoxin for spotted knapweed from *Alternaria alternata*. *Proceedings of the National Academy of Sciences* 85:8008-8011.
- Stiling, P. and D. Simberloff. 2000. The frequency and strength of nontarget effects of invertebrate biological control agents on plant pests and weeds. Pp. 31-43 *In* P.A. Follett and J.J. Duan (eds.) *Nontarget effects of biological control*. Kluwer Academic Publishers. Boston, Massachusetts.
- Strong, D.R. and R.W. Pemberton. 2000. Biological control of invading species – risks and reform. *Science* 288: 1969-1970.
- Tipping, P.W. 1991. Effects of mowing or spraying *Carduus thoermeri* on *Rhinocyllus conicus*. *Weed Technology* 5: 628-631.
- Turner, C.E. R.W. Pemberton and S.S. Rosenthal. 1987. Host utilization of native *Cirsium* thistles (Asteraceae) by the introduced weevil *Rhinocyllus conicus* (Coleoptera: Curculionidae) in California. *Environmental Entomology* 16:111-115.
- Van, T.K. and T.D. Center. 1994. Effect of paclobutrazol and waterhyacinth weevil (*Neochetina eichhorniae*) on plant growth and leaf dynamics of waterhyacinth (*Eichhornia crassipes*). *Weed Science* 42: 665-672.
- Vitousek, P.M. 1986. Biological invasions and ecosystem properties: can species make a difference? pp. 163-176 *In* H.A. Mooney and J.A. Drake (eds.) *Ecology of biological invasions of North America and Hawaii*. Springer-Verlag, New York.
- Vitousek, P.M., L.R. Walker, L.D. Whiteaker, D. Mueller-Dombois and P.A. Matson. 1987. Biological invasion by *Myrica faya* alters ecosystem development in Hawaii. *Science* 238:802-804.
- Whisenant, S.G. 1990. Changing fire frequencies on Idaho's Snake River Plains: ecological and management implications. *Proceedings - Symposium on cheatgrass invasion, shrub die-off, and other aspects of shrub biology and management*. Las Vegas, NV 1989. USDA, Forest Service Intermountain Research Station General Technical report INT-276.
- Withers, T., R. McFayden and J. Marohasy. 2000. Importation protocols and risk assessment of weed biological control agents in Australia: the example of *Carmentis nr ithacae*. Pp 195-214. *In* P.A. Follett and J.J. Duan (eds.) *Nontarget effects of biological control*. Kluwer Academic Publishers. Boston, Massachusetts.

- Wymore, L.A. and A.K. Watson. 1986. An adjuvant increases survival and efficacy of *Colletotrichum coccodes*: a mycoherbicide for velvet leaf (*Abutilon theophrasti*). *Phytopathology* 76:1115-1116.
- Wymore, L.A., A.K. Watson and A.R. Gottlieb. 1987. Interaction between *Colletotrichum coccodes* and thidiazuron for control of velvetleaf (*Abutilon theophrasti*). *Weed Science* 35:377-383.

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Chapter 5 – GUIDELINES¹ FOR HERBICIDE USE

PURPOSE

These Guidelines are designed to ensure that you carefully consider the overall impacts of herbicide use on your conservation targets, other native species, and the ecological system. Base all decisions whether to control weeds, and whether to use herbicides instead of other methods, on the conservation targets and management goals for the site. In addition, the health and safety of applicators and others in the vicinity must be considered **BEFORE** pesticides are applied. Simply put, one should be confident that the proposed herbicide will do more conservation good than harm and not endanger the health of the applicators or others in the area.

TO SPRAY OR NOT TO SPRAY?

Determining the right course of action in weed management can be difficult. For many land managers, whether to apply herbicides is an ethical decision that is not taken lightly. Herbicides are often used as a last resort, when other attempts have failed, and action is imperative.

The following checklist summarizes the steps that need to be taken to ensure that proper consideration has been given to current weed problems, and that the use of herbicides is warranted for each individual case.

1. Determine whether invasive plants threaten conservation targets or management goals on the site. Use herbicides (versus other control methods) only if confident they can be used safely and will do more conservation good than harm. If you decide to use herbicides, be sure to record your reasons for doing so. TNC's Site Conservation Program (<http://www.consci.org/scp>) can help you identify targets and threats, and make a Site Conservation Plan. TNC's Site Weed Management Plan Template (<http://tncweeds.ucdavis.edu/products.html>) can help you set control priorities and develop a plan to implement them.
2. Develop safety protocols for **STORING, MIXING, TRANSPORTING, HANDLING SPILLS, and DISPOSING OF UNUSED HERBICIDES & CONTAINERS BEFORE** obtaining herbicides.

¹ These Guidelines and TNC's Standard Operating Procedures were designed to make TNC use of herbicides meet or exceed the Worker Protection Standard for Agricultural Pesticides enacted by the U.S. EPA January 1 1995. Although the Worker Protection Standard does not cover pesticide use in natural areas, except on sites leased for agricultural production, TNC's operations should at the very least measure up to this Standard.

It is **NOT** the purpose of TNC's Standard Operating Procedures nor of these Guidelines to require stewards to produce lengthy herbicide use plans.

3. Follow all federal, state and local regulations regarding herbicide use. You **MUST** read and follow product labels. It is a violation of federal law to use an herbicide in a manner inconsistent with its label.
4. Contact your State Department of Agriculture or County Agriculture Commissioner for information about state and local regulations regarding applicator permits and posting requirements. (See the list of state regulatory agencies in the Appendix.)
5. Check with the legal staff for your program (State or Regional Office) **BEFORE** obtaining herbicides if you have any questions about regulations or liability issues.
6. Herbicides may be applied only by TNC employees or contractors who have all certificates and licenses required by the state and/or county. Volunteers may NOT apply herbicides unless they are properly licensed AND have signed a consent & release form.
7. Applicators **MUST** wear all protective gear required on the label of the herbicide they are using. Provide all safety and protective gear requested by the employee(s) applying the herbicide. The health and safety of the applicator are of foremost concern.

SITE CONDITIONS

Site conditions to be considered include accessibility, proximity to open water, depth to groundwater, the presence of rare species and other conservation targets, and the site's sensitivity to trampling that could occur when the herbicide is being applied.

To prevent contamination of water bodies, management plans should carefully consider the hydrology of the system that is being treated. Hypothesize potential runoff scenarios and take appropriate measures (such as buffer zones) to prevent them. Underground aquifers and streams should be considered as well.

The herbicides covered in this Manual are regarded as posing relatively low risk for use in natural areas because they are not likely to contaminate groundwater, have limited persistence in the environment, and are of low toxicity to animals. Critical reviews of several common herbicides are available at a small charge from the Northwest Coalition for Alternatives to Pesticides (NCAP, P.O. Box 1393, Eugene, OR 97440, (503) 344-5044, <http://www.pesticide.org>). Information is also available from the National Coalition Against the Misuse of Pesticides (NCAMP, 701 E Street SE #200, Washington DC 20003, (202) 543-5450, www.ncamp.org).

In addition to federal pesticide registration, some states also have their own registration procedures and requirements and almost all states have their own pesticide applicator licensing, certification, or registration. To find out if a particular herbicide is registered

for use on wildlands in your state, call the state pesticide regulatory agency (see the Appendix for a list of state regulatory agencies).

ENDOCRINE DISRUPTING COMPOUNDS

The presence of synthetic chemicals in the environment, especially those designed to control unwanted species (insecticides and herbicides), and the acute and long-term effects of those chemicals on wildlife and humans have been of concern since the publication of Rachel Carson's book "Silent Spring" in 1962. New evidence indicates that the functioning of animals (including humans) endocrine systems can be severely altered by low-level cumulative exposure to some synthetic chemicals. Many different classes of industrial chemicals released into the environment exhibit potential endocrine-disrupting activities, such as mimicking or blocking the action of natural animal hormones. Exposure to these compounds during critical periods of development (in utero, or early postnatal) can result in irreversible damage to wildlife and to humans. In general, the compounds found in insecticides are usually more toxic than those in most herbicides, as most herbicides block or alter biochemical processes found exclusively in plants.

Numerous studies have reported that agricultural and industrial waste chemicals adversely effect wildlife populations. Endocrine-altering compounds, however, can also be found naturally (such as the phytoestrogen genistein, that is found in soy protein). Some studies suggest that the effects of synthetic chemicals are negligible relative to those of naturally occurring plant estrogens. Many synthetic compounds are known to bioaccumulate, which may greatly magnify their effects. It has also been suggested that combinations of synthetic compounds act synergistically with effects far greater than those of any one compound.

Some studies suggest that synthetic endocrine-disrupting chemicals alter growth, development, and reproduction rates, and can cause abnormal behavior in various wildlife species. Further, there is increasing concern regarding potential effects of synthetic endocrine disruptors on human reproduction and development, including, but not limited to, increased breast and ovarian cancers, infertility, increased testicular cancer, decreased semen quality, and increased spontaneous abortion rates.

A review by CAST (Council for Agricultural Science and Technology) published in 2000, concluded that current scientific evidence does not clearly link endocrine-disrupting chemicals with decreased male reproductive capacity or increased rates of breast cancer in women. However, this review did not completely dismiss the potential role that these chemicals may have as causative agents for adverse human health effects. Herbicides are only a small subset of all synthetic chemicals produced, and thus far, only 2,4-D has been implicated for possible endocrine-disrupting impacts. Some reproductive and developmental problems in wildlife populations have been attributed to endocrine-disrupting chemicals, but evidence of other effects are far from conclusive.

For more information:

Colborn, T., Dumanoski, D. and J.P. Myers. 1996. *Our Stolen Future: Are We Threatening Our Fertility, Intelligence and Survival. A Scientific Detective Story.* Penguin Books, New York.

Cornell University Program on Breast Cancer and Environmental Risk Factors in New York State. 2000. *Endocrine Disruption and Breast Cancer Risk.*
<http://envirocancer.cornell.edu/Bibliography/General/bib.endocrineDisruption.cfm>

Lyons, G. 1999. Endocrine disrupting pesticides. *Pesticides News* 46: 16-19. Pesticide Action Network UK.

Safe, S.H., Foster, W.G., Lamb, J.C., Newbold, R.R. and G. Van Der Kraak. 2000. Estrogenicity and endocrine disruption. Council for Agricultural Science and Technology (CAST), Issue Paper no. 16.

HERBICIDE PROPERTIES

Consider the following herbicide properties when deciding which compound to use:

1. Effectiveness against the target species.
2. Mechanisms of dissipation (persistence, degradation, and likelihood of movement via air or water to non-target organisms).
3. Behavior in the environment (in soils, water, and vegetation).
4. Toxicity to birds and mammals, aquatic species, and to other non-target organisms (including algae, fungi, and soil organisms).
5. Application considerations
6. Safety
7. Human toxicology

In general for work in natural areas, it is best to select compounds that are effective against the weed, not likely to drift, leach to groundwater or wash into streams, nontoxic to people and other organisms, not persistent in the environment, and is easy to apply. In some circumstances, a single application of a more toxic or persistent chemical that kills the weed, however, may be preferable to a less persistent, less toxic compound that must be applied repeatedly. Strive to do the job with the smallest **total** negative impact to the environment.

PROTECTIVE GEAR FOR APPLICATORS

The health and safety of the applicator are of foremost concern. Applicators **MUST** wear all protective gear required on the label of the herbicide they are using. Any additional safety and protective gear requested by TNC applicators must be provided. See the following textbox (page 5.6) for additional information regarding personal protection needs.

Even if not required, all TNC or volunteer applicators should wear the following when mixing or applying herbicides:

1. Rubber boots,
2. Protective aprons or suits (e.g., disposable tyvek suits) or sturdy overalls that are not used for other activities,
3. Rubber gloves (tyvek and nitrile gloves are recommended - one study indicated that neoprene can be penetrated by herbicides under field conditions),
4. Safety glasses or goggles.

Some applicators may even wish to wear respirators where not required. A dust mask may be worn when a respirator is not required, but pesticide safety officers point out that dust masks usually fit loosely and do not stop volatile compounds. Furthermore, they can indirectly increase chances of exposure if they cause heating, sweating, and irritation, which induce the wearer to repeatedly wipe or scratch their face.

Some companies that supply protective gear include:

A.M. Leonard, Inc.
241 Fox Drive
Piqua, Ohio 45356-0816
Phone: 1-800-543-8955
Web Address: <http://www.amleonard.com>

Ben Meadows Company
190 Etowah Industrial Court
Canton, GA 30114
Phone: 1-800-241-6401
Web Address: <http://www.benmeadows.com>

Forestry Suppliers, Inc.
P.O. Box 8397
Jackson, MS 39284-8397
Phone: 1-800-647-5368
Web Address: <http://www.forestry-suppliers.com>

Gempler's Inc.
P.O. Box 270
Belleville, WI 5350
Phone: 1-800-382-8473
Web Address: <http://www.gemplers.com>

Lab Safety Supply Inc.
P.O. Box 1368
Janesville, WI 53547-1368
Phone: 1-800-356-0783
Web Address: <http://www.labsafety.com>

Safety Solutions, Inc.
6161 Shamrock Ct.
P.O. Box 8100
Dublin, Ohio 43016-2110
Phone: 1-800-232-7463
Web Address: <http://www.safetysolutions.com>

PERSONAL PROTECTION IN HERBICIDE HANDLING

Adapted from Ohio State University's Extension Publication #825 "Applying Pesticides Correctly"
by Jennifer Hillmer, The Nature Conservancy-Ohio

PERSONAL PROTECTIVE EQUIPMENT

Herbicide labels indicate the minimum protective equipment required. This may vary by application technique. Cotton, leather, canvas, and other absorbent materials are not chemical resistant, even to dry formulations.

- Always wear at least a long-sleeved shirt, long pants, sturdy shoes or boots, and socks. The more layers of fabric and air between you and the pesticide, the better the protection.
- A thick layer of spray starch on clothing will add some protection from pesticides.
- Hands and forearms usually receive the most pesticide exposure. Wear chemical-resistant gloves, and tuck shirt sleeves into gloves (gloves should reach up the forearm, with cuffs to catch runs and drips).
- Canvas, cloth, and leather shoes or boots are almost impossible to clean adequately. Wear chemical-resistant rubber boots that come up at least halfway to the knee if the lower legs and feet will be exposed to herbicides or residues.

AVOIDING CONTAMINATION

- Wear chemical-resistant gloves (rubber or plastic such as butyl, nitrile, or polyvinyl chloride are common types).
- Make sure gloves are clean, in good condition, and worn properly. Replace gloves often. Wash and dry hands before putting on gloves. Wash gloves before removing them.
- Wash hands thoroughly before eating, drinking, using tobacco products, or going to the bathroom.
- Cuff gloves if pesticide is expected to run down towards the sleeves. Tuck sleeves into gloves.

EYE AND RESPIRATORY PROTECTION

- PPE labeling might require goggles, face shields, or safety glasses with shields. Some formulas or handling activities pose more risks to eyes than others. Dusts, concentrates, and fine sprays have the highest risk of causing pesticide exposure.
- There are many types of dust-mist masks and respirators, all of which must fit and be used properly to be effective.
- Respiratory protection is most important in enclosed spaces or when the applicator will be exposed to pesticides for a long time.
- Pesticides that can volatilize require the use of respirators. Check label requirements.

PERSONAL CLEAN-UP AFTER HERBICIDE USE

- Wash gloves and footwear (if possible) with detergent and water before removing them.
- Change clothing and put clothes used during application in a plastic box or bag, and keep it away from children or pets. Use a mild liquid detergent and warm water to wash your hands, forearms, face, and any other body parts that may have been exposed to pesticides. Take a warm shower and wash your hair and body at the end of the work day.

LAUNDRY

- Do not wash work clothing and personal protective equipment in the same wash water with the family laundry. Handle with care and wash your hands after loading the machine.
- If you have chemical-resistant items, follow the manufacturer's washing instructions. Wash boots and gloves with hot water and liquid detergent. Wash twice, once outside and once inside. Air-dry boots and gloves.
- Rinse clothes in a machine or by hand.
- Wash in plenty of water for dilution and agitation.
- If using a washing machine, use heavy-duty liquid detergent in hot water for the wash cycles.
- After washing the clothes, run the washer through one complete cycle with detergent and hot water, but no clothing, to clean the machine.
- Hang items to dry if possible in plenty of fresh air. Do not hang in living areas.
- Using a clothes dryer is acceptable, but over time the machine may become contaminated with pesticide residues.

EMERGENCY PRECAUTIONS AND EQUIPMENT

Applicators must have easy access to emergency decontamination and first aid kits whenever they are applying herbicides, even if they are out in the field. All applicators should have access to an eyewash kit and at least 2 gallons of clean water.

Decontamination kits are available from many suppliers or can be assembled independently. Rubber buckets or tubs with tight sealing lids are convenient for homemade kits and should include:

1. Two (or more) 1 gallon containers filled with potable water,
2. Eyewash kits or eyewash bottles with buffered isotonic eyewash,
3. Hand or body soap (bring enough for all workers to thoroughly wash their hands when in the field),
4. Paper or other disposable towels,
5. A full tyvek coverall with foot covers,
6. A map and directions to the nearest medical facilities. Such maps should be posted in prominent locations at all preserve offices and work buildings. Include a copy as an Appendix to your weed control plan.

POSTING TREATED AREAS

Federal requirements for posting treated areas, if any, are listed on the herbicide label. Glyphosate, triclopyr and most other herbicides used in natural areas have no federal posting requirements. Some municipalities and counties have stricter requirements (e.g., Boulder, Colorado). Always keep treated areas off limits to the public at least until the herbicide dries. Treated areas may be kept off limits for longer periods if the herbicide is persistent in the environment.

When posting areas that are accessible to the public (trails, visitor centers etc.), place notices at the usual points of entry or along the perimeter of treated sites. The posting should include a notice that the area has or will be treated, the name of the herbicide used, the date of the treatment, appropriate precautions to be taken, the date when re-entry is judged to be safe, and a phone number for additional information. The notices should be removed after it is judged safe to re-enter the area.

STORING HERBICIDES

Store herbicides in a well ventilated, cool, dry area where food and drinks are never stored or prepared. Most pesticides should not be stored for any length of time below 40° F. The floor should be concrete or lined with plastic or other impermeable material to prevent leaks from reaching the soil.

The area should be inaccessible to the public and/or locked except when chemicals are being removed or returned. Containers should be labeled to indicate the following: contents (ratio of herbicide, surfactant, water, etc.), date mixed, and approximate volume remaining when placed in storage. The containers must be stored carefully and never stacked.

Heavy plastic garbage bags, a shovel, and a soil absorbent (e.g., cat litter) must be available for use in cleaning-up small leaks or spills. For more information on spills see below.

MIXING HERBICIDES

USE EXTREME CAUTION WHEN MIXING HERBICIDES! Dermal exposure to a small amount of a concentrated herbicide can be equivalent to the exposure received after a full day of working in a treated field (Libich et al. 1984). Before mixing any herbicide, **READ THE LABEL.** Herbicide labels are legal documents and users are obligated to read and obey them.

Establish a mixing area. Herbicides should be mixed only in pre-designated areas - preferably either in an industrial sink near the storage site or in an area near the treatment site(s) in which damage from small spills or other herbicide contamination would be minimal. Field mixing sites should have relatively few native or other desirable species, not be susceptible to erosion or runoff, and rarely, if ever, be visited by the public or preserve staff. In addition, mixing sites should provide easy access for containment and clean up of spills.

At the mixing site, assemble the appropriate equipment including safety and clean-up gear and measuring and mixing utensils. Heavy plastic garbage bags, a shovel, and an absorbent (e.g. cat litter) must be easily available at field mixing sites in case of a larger spill. Remember to wear all protective gear while handling and mixing herbicides. Avoid metal measuring utensils as some pesticides can react with metal. Clearly label herbicide-measuring equipment to avoid confusion with equipment used for measuring food. Wash all utensils before storage to prevent contamination of future mixes.

Prior to mixing, determine the order that chemicals will be added to the mix. Generally, adjuvants are added prior to the herbicide, but consult the label for specific instructions. When mixing, start by filling the spray tank or other mixing vessel half to three-quarters full with water. The water should be clean and clear to prevent contamination of the mixture or clogging of tank nozzles and hoses. The water should have a neutral or slightly acidic pH, as alkaline water can cause the pesticide to breakdown prior to application. Add a buffer or acidifier to the water if necessary.

Carefully measure the herbicide concentrate and add it to the tank water. Small measuring errors can lead to large errors in the amount of pesticide applied. Be aware of if you are using the active ingredient (a.i.) or acid equivalent (a.e.) of the herbicide (see sidebar below for more details). The measuring container should be rinsed and the rinsate added to the tank solution. The container of liquid herbicides should be triple rinsed with $\frac{1}{4}$ container volume of water. Add rinsate to the tank solution or store it in a separate container labeled "WATER AND RINSATE FOR HERBICIDE ONLY, NONPOTABLE"

ACTIVE INGREDIENT (A.I.) VS. ACID EQUIVALENT (A.E.)

Labels on herbicide containers and instructions for mixing herbicides sometimes use units of herbicide active ingredient (a.i.) or acid equivalent (a.e.). The herbicide may be sold in different concentrations, but units of a.i. or a.e. provide standard measures, so the mixing instructions can apply in all cases. In order to follow these instructions, you will need to determine how many a.i. or a.e. are in an ounce, or quart or liter, of the concentrate on hand.

The “active ingredient” (a.i.) of an herbicide formulation is responsible for its herbicidal activity or ability to kill or suppress plants. The a.i. is always identified on the herbicide label by either its common name or chemical name, or both. Herbicide formulations available for sale commonly contain other so-called “inert” compounds too.

The “acid equivalent” (a.e.) of an herbicide is just the acid portion of the a.i., and it is this acid portion that is responsible for herbicidal effects. The acid portion (or parent acid) is generally associated with other chemical compounds to form a salt or an ester, which is more stable and better able to move through a plant’s waxy cuticle, and into the plant. The salt or ester is the a.i.

Weak acid herbicides are formulated as salts or esters through the addition of a salt or ester molecular group to the parent acid molecule. This allows the herbicide acid to mix properly with adjuvants and enhances the compound’s ability to move into plant tissue. Once the herbicide enters the plant, the salt or ester group is cleaved off the parent molecule, allowing the acid to affect the plant.

Because the salt or ester molecular group can vary dramatically in size, a measure of the percent a.i., especially in the case of a weak acid herbicide, does not adequately reflect the percentage of acid in the formulation. Thus, the a.e. is used to determine the amount of the product to be applied.

Product labels for weak acid herbicides will list the product’s percentage of active ingredient, as well as other inert ingredients, at the top of the label. The percentage of acid equivalent in the formulation is usually listed below these percentages in a separate table or paragraph.

TRANSPORTING HERBICIDES

Herbicides should be transported in tightly sealed containers placed in a well-constructed and watertight carrying box or bucket, such as a Rubbermaid tub or cat litter bucket. A good container will prevent leaks in vehicles, onto applicators, or to the environment. Each program should develop techniques and use materials that will best serve the needs of a particular site or circumstance. In some cases, you may want to carry only a small amount of herbicide to treat weeds encountered while conducting daily activities in the field.

Jack McGowan-Stinski of TNC’s Michigan program uses large five-gallon buckets with tight lids to transport herbicides and application equipment into the field. The buckets are large enough to hold all the necessary equipment and can be carried by groups of volunteers. Jennifer Hillmer of TNC’s Ohio Program often treats weeds distributed over great distances while working in the field by herself. Jennifer keeps pesticides in a crook-necked squirt bottle for easy application and carries the squirt bottle and other application equipment in a four-liter, square, leak-proof, Nalgene bottle, which can be

carried in her backpack along with other field equipment. Jennifer recommends laboratory supply companies as a good place to find equipment for herbicide application and storage.

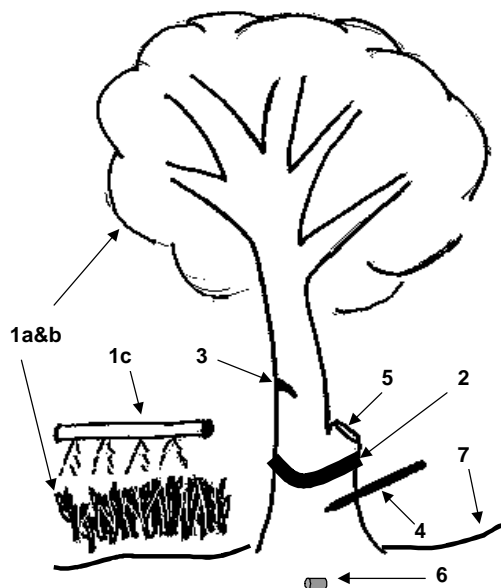
APPLICATION OF HERBICIDES

Application Methods

Herbicides can be applied in a variety of ways. The most appropriate application method is determined by the weed being treated, the herbicide being applied, the skills of the applicator, and the application site. Standard application techniques can sometimes be modified to better suit the needs of natural area management. A few land managers have come up with simple but ingenious techniques and tools that save money, are more effective and safer, and are easier to use than standard methods. We include some of these in the detailed descriptions of techniques below, and encourage you to innovate because there is still plenty of room for improvement.

Methods of application (diagrammed below) can be broadly classified as follows:

- 1) To intact, green leaves (foliar application)
 - a. Spot application (backpack applicator, spray bottle);
 - b. Wick application (wipe-on);
 - c. Boom application;
- 2) Around the circumference of the trunk on the intact bark (basal bark);
- 3) To cuts in the trunk/stem (frill; hack and squirt);
- 4) Injected into the inner bark;
- 5) To cut stems and stumps (cut stump);
- 6) In pellet form at the plant's base (rarely used in natural areas);
- 7) To the soil before the target species seeds germinate and emerge (rarely used in natural areas).



1. Foliar Applications

These methods apply herbicide directly to the leaves and stems of a plant. An adjuvant or surfactant is often needed to enable the herbicide to penetrate the plant cuticle, a thick, waxy layer present on leaves and stems of most plants. There are several types of foliar application tools available.

- A. Spot applicators – Spray herbicide directly onto target plants only, and avoid spraying other desirable plants. These applicators range from motorized rigs with spray hoses to backpack sprayers, to hand-pumped spray or squirt bottles, which can target very small plants or parts of plants. Crook-necked squirt bottles and similar equipment can be ordered from laboratory supply companies and are easy to carry over distances and through dense vegetation.
- B. Wick (wipe-on) applicators - Use a sponge or wick on a long handle to wipe herbicide onto foliage and stems. Use of a wick eliminates the possibility of spray drift or droplets falling on non-target plants. However, herbicide can drip or dribble from some wicks.
 - i. “Paint sticks” and “stain sticks” sold at local hardware stores have been used successfully for wick application. These sticks have a reservoir in the handle that can hold herbicide, which soaks a roller brush at the end of the handle. The brush is wiped or rolled across leaves and stems.
 - ii. The “glove of death” is a technique developed by TNC land stewards for applying herbicide in an otherwise high quality site. Herbicide is sprayed directly onto a heavy cotton glove worn over a thick rubber/latex (or nitrile) glove. The wearer of the glove can then apply the herbicide with total precision and little or no runoff.
- C. Boom applicator - A boom, a long horizontal tube with multiple spray heads, is mounted or attached to a tractor, ATV (or other four-wheel drive vehicle), helicopter, or small plane. The boom is then carried above the weeds while spraying herbicide, allowing large areas to be treated rapidly with each sweep of the boom. Offsite movement due to vaporization or drift and possible treatment of non-target plants can be of concern when using this method.

2. Basal Bark

This method applies a 6 to 12 inch band of herbicide around the circumference of the trunk of the target plant, approximately one foot above ground. The width of the sprayed band depends on the size of the plant and the species’ susceptibility to the herbicide. The herbicide can be applied with a backpack sprayer, hand-held bottle, or a wick. Ester formulations are usually best for basal bark treatments, as esters can pass most readily through the bark (as compared to salts). Esters can be highly volatile, however, so basal bark treatments should be performed only on calm, cool days. During summer, treatment is best carried out in the mornings, which tend to be cooler. The basal bark treatment works best on young trees with smooth bark. It is usually not effective against older plants with thick corky bark.

3. Frill or Hack & Squirt

The frill method, also called the “hack and squirt” treatment, is often used to treat woody species with large, thick trunks. The tree is cut using a sharp knife, saw, or ax, or drilled with a power drill or other device. Herbicide is then immediately applied to the cut with a backpack sprayer, squirt bottle, syringe, or similar equipment. Because the herbicide is placed directly onto the thin layer of growing tissue in the trunk (the cambium), an ester formulation is not required.

Jack McGowan-Stinski (TNC-Michigan) recommends using the drill treatment rather than cutting, for trees with dbh (diameter at breast height) greater than three inches. He has volunteers use “tree steps” to drill holes into trees. Tree steps are large metal screws that can be screwed into a tree trunk by hand to provide steps for tree climbing. When applying herbicide, tree steps are lightweight drilling tools that can be easily carried into the field and used by untrained volunteers. These tools are available at most hunting stores and cost only a few dollars each.

Jack recommends drilling one hole for each inch in dbh. (A ten-inch dbh tree would require at least ten holes.) Holes should be drilled at a slight downward angle to prevent the herbicide from running out, and should be deep enough to penetrate the inner bark or growing tissue.

Some added recommendations made by Jack for using the drill method include: 1) Spray-paint tree steps with a neon color to prevent them from being lost if dropped in dense vegetation. 2) Spray-paint circles directly onto the trees around the drilled holes. This will ensure that no holes are overlooked by the herbicide applicator. After the hole is filled with herbicide, the applicator can spray paint a line through the hole to indicate that it was treated.

4. Injection

Herbicide pellets can be injected into the trunk of a tree using a specialized tool such as the EZ-Ject Lance. The EZ-Ject lance’s five ft long, metal tube has “teeth” on one end that grip the trunk of the tree. A sharp push on the other end of the tube sends a brass capsule of herbicide into the tree trunk. It is a convenient way of applying herbicide and requires minimal preparation or clean up. In addition, it is an easy and safe way to apply herbicides with minimal exposure.

There are however, some serious drawbacks to this method. The lance and capsules are expensive (\$425 per lance; approximately \$500 per 4,800 capsules, depending on herbicide), and full-sized lances can be unwieldy, particularly in thickets. The lance furthermore, is difficult to thrust with enough power to drive the capsules far enough into thick barked trees to be effective. A large number of capsules placed close together are often necessary to kill large trees.

At the Albany Pine Bush Preserve in New York, glyphosate gel pellets were injected using an EZ-Ject Lance into trees with an average dbh of eight centimeters. In some

cases, crowns of treated trees later showed signs of stress, but most of these re-sprouted vigorously and none of the treated trees died (Hawver et al. 2000).

For information or to order an EZ-Ject Lance contact Odom Processing Engineering Consulting, Inc., 800 Odom Industries Road, Waynesboro, MS, 39367, (601) 735-2680, (888) 395-6732, www.ezject.com.

Herbicides can also be injected into herbaceous stems by using a needle and syringe. Jonathan Soll (TNC-Oregon) reports 100% control of small patches of Japanese knotweed (*Polygonum cuspidatum*) with no off-target effects, by injecting every single stem near the base with herbicide. He adds that this method may actually use more herbicide than foliar spraying (since you use high concentrations of the herbicide), and caution with the needle and syringe is necessary since you are carrying around a sharp object.

5. Cut-Stump

This method is often used on woody species that normally re-sprout after being cut. Cut down the tree or shrub, and immediately spray or squirt herbicide on the exposed cambium (living inner bark) of the stump. The herbicide must be applied to the entire inner bark (cambium) within minutes after the trunk is cut. The outer bark and heartwood do not need to be treated since these tissues are not alive, although they support and protect the tree's living tissues.

Herbicide can be applied to cut stumps in many ways, including spray and squirt bottles, or even paint brushes. Care must be taken to avoid applying too much herbicide, and allowing it to run-off the stump and onto the ground. Herbicide can also dribble from bottles or brushes and fall on desirable plants or the ground. To help avoid these problems, Jack McGowan-Stinski (TNC-Michigan) developed an inexpensive and easy to assemble application tool using PVC pipe and a sponge through which the herbicide can be applied. See the Appendix for a diagram and instructions on how to build one.

Sometimes even treated stumps will re-sprout, so it is important to check them at regular intervals (2 to 6 months) for at least a year. Depending on the vigor of the re-sprouts, these can be treated by cutting, basal bark applications, or foliar applications. Even when foliar applications are called for, treating re-sprouts is usually far easier and requires much less herbicide than treating the tree (before it was cut down) with a foliar application.

The cut stump treatment allows for a great deal of control over the site of herbicide application, and therefore, has a low probability of affecting non-target species or contaminating the environment. It also requires only a small amount of herbicide to be effective. Black locust (*Robinia pseudoacacia*) and buckthorns (*Rhamnus* spp.) have been successfully controlled using this method (Hawver et al. 2000; J. McGowan-Stinski, pers. comm.).

Selecting a Method

Minimize

Select a technique(s) that (1) minimizes risks of contact to the applicator and others that may be in the area during and after herbicide application, AND (2) minimizes release of herbicide to the environment, particularly if the herbicide could contact non-target species. Avoid using boom application where possible (1c above) because it can result in a relatively high amount of herbicide contacting non-target species and bare ground. Also, avoid using pellets and pre-emergence herbicides (6 & 7 above, respectively) because they are relatively persistent in the environment.

Use a dye

Mix a dye with the herbicide so applicators can see which plants have been treated and if they have gotten any herbicide on themselves or their equipment. Some pre-mixed herbicides include a dye (e.g., Pathfinder II[®] includes the active ingredient triclopyr, a surfactant, and a dye). Ester based herbicides like Garlon 4[®] require oil-soluble dyes like colorfast purple[®], colorfast red[®], and basoil red[®] (for use in basal bark treatments), which are sold by agricultural chemical and forestry supply companies. Clothing dyes like those produced by Rit[®] will work in water-soluble herbicides such as Garlon 3A[®]. These dyes are inexpensive and available at most supermarkets and drugstores.

Who May Apply Herbicides?

TNC employees or contractors who apply herbicides must have all certificates or licenses required by the state. Each state has its own requirements. Some require applicators working in natural areas to be certified and others do only if compounds designated "restricted-use" by the EPA or the state are to be used. Most states conduct applicator training programs and in many areas local Agricultural Extension Agents give workshops on proper herbicide use.

Volunteers may NOT apply herbicides unless they are properly licensed AND have signed a consent & release form. An example of such a form produced by the Illinois Field Office is provided as an Appendix. **Check with the legal staff for your program before drafting one of these forms or using volunteers to apply herbicides.** TNC staff who supervise volunteers should be properly licensed or certified.

Protection Against Herbicides

When using herbicides, the safety of the applicator, to others, and to the environment is of utmost importance. Be sure to read the earlier textbox (page 5.6) on "Personal Protection in Herbicide Handling" regarding specific equipment requirements, how to avoid contamination, eye and respiratory protection, how to clean-up after herbicide use, and how to launder clothes and equipment used during herbicide application.

When to Apply Herbicides

The best time to apply an herbicide is determined primarily by the herbicide's mode of action and the physiology of the target plants. In seasonal climates, it is often best to apply herbicides in autumn or prior to the dry season, 3 to 6 weeks before the target plant

goes dormant for the season. This is because many plants apparently transfer sugars and nutrients from their stems and leaves to belowground storage organs at this time and will carry herbicides along to these areas as well. Contrary to assumptions that plants will be most vulnerable when weak, herbicides are usually ineffective when applied during a drought or other stressful conditions. This is because most herbicides work by attacking growing tissue and metabolic processes, which plants 'shut down' when stressed. In fact, late winter or early spring are often good times to apply herbicide because this is when plants begin growing again, and can efficiently translocate the herbicide throughout their tissues. Fosamine ammonium, the dormancy enforcer, is best applied in the late fall just before leaf drop. The herbicidal effects of fosamine ammonium however, are not observed until the following spring when treated plants fail to re-foliate.

In some cases, the site of application may determine the best time to apply a herbicide. For example, buckthorns (*Rhamnus* spp.) growing in wet, boggy areas are easiest to treat during winter when the ground is frozen. Check the label or consult your distributor for the best application time under the conditions at your site.

Note that with some herbicides there is a long time lag between time of herbicide application and the first evidence that they are working. This is particularly true of herbicides that work by inhibiting amino acid or lipid synthesis, because the plant(s) can rely on stored supplies to continue growing.

Record Keeping

When using herbicides it is critical (and, in some cases, required by law) to keep records of all plants/areas treated, amounts and types of herbicide used, and dates of application. This information will be important in evaluating the project's success, improving methodology, and identifying mistakes. In addition, it documents the procedure for future site managers and biologists. Records of abundance/condition of the targeted weeds and nearby desirable plants before and after treatment will also be valuable in evaluating the effectiveness of the herbicide.

HERBICIDE DISPOSAL

Equipment cleanup

Following use, application equipment and empty containers should be triple rinsed with clean water using 10% of the container volume for each rinse. If possible, rinse equipment in the treatment area and apply the wastewater to weeds or store for future use as a dilutant. Left over herbicide mix that will not be used later should be treated as hazardous waste.

Container disposal

Use the state herbicide container recycling program where available. In Minnesota, herbicide dealers are required to collect empty containers from customers. If no specific agri-chemical container recycling program is available, puncture the empty container to prevent anyone from using it as a container again, and then dispose of or destroy it. In most areas, small numbers of empty, triple-rinsed containers can be disposed in the trash for pick-up or taken to the local dump, unless the label states otherwise. In parts of California and some other states you may be required to get written permission from your County Agriculture Commissioner to dispose of containers. Call your local Commissioner for details. Some jurisdictions require containers to be burned, while others prohibit burning pesticide containers. If the herbicide label states that the container may not be disposed of in regular sanitary landfills, call your county or municipal waste department for information on Hazardous Material Collection dates.

Equipment and applicator clean-up

After use, first clean and store application equipment and then thoroughly rinse personal protection gear (gloves, boots, etc.) with cold water from a hose or container that is hand-held (gloves off) and was not used during application work. All personal protection gear should then be washed in mild soap and water. Finally, applicators should wash their hands and any herbicide-exposed areas of their bodies. Applicators should shower and change clothing as soon as possible. Clothes used during the application must be washed and dried separately from other clothing before it is worn again, even if it appears uncontaminated.

Contaminated clothing

If herbicide concentrate spills on clothing, the clothing should be discarded or, where permitted, burned immediately. Wrap contaminated clothing and other materials in newspaper before placing in trash or landfill. Clothing and other items contaminated with certain commercial products, such as technical grade 2,4-D or formulations in which 2,4-D is the only active ingredient, are classed as hazardous waste. Call your local hazardous materials center for instructions on how to dispose of this material. In cases where small quantities are involved it may be possible to dispose of contaminated clothing in the trash.

RESPONDING TO SPILLS

Rules and regulations regarding pesticide spills vary between states and counties. Therefore, before obtaining herbicides, call the local fire department or county Hazardous Materials Office for information on local regulations. In most cases, the proper response to a spill depends on the volume and concentration of herbicide released, location of the spill, and the chemical(s) involved. If possible, inquire as to whether a report would be required in a hypothetical situation in which all the herbicide was spilled (1) on the soil in the interior of the preserve and (2) along a public road. A rule of thumb employed by some public land management agencies is not to call for help from the local Hazardous Materials Office for herbicide spills unless they contaminate too much soil to dig up and place in plastic garbage bags. However, since our goal is to protect biodiversity, land

managers are expected to minimize damage to native populations. Hazardous Materials officers we spoke to considered spills under 100 gallons to be “small”. Most emergency systems appear to be designed to deal with these larger volumes used in agriculture and industry, which are far larger than those typically used in natural areas.

Be sure to carry a “Pesticide Kit” for emergency spills (see the following Pesticide Spill Kit equipment list). If a spill occurs, keep people away from affected areas until the clean-up process is complete. When small volumes of dilute herbicide are spilled they may be treated by carefully digging up the affected soil and litter, and spreading this material at the legal rate or concentration. Small diesel (sometimes used as a crude surfactant) and gasoline spills may be treated by adding organic material (e.g., cow manure or compost) to the affected area and keeping it moist. It may take several years for the spilled material to degrade.

PESTICIDE KIT EQUIPMENT LISTS

adapted from work by Jack McGowan-Stinski and Jennifer Hillmer

PESTICIDE SPILL KITS

- Emergency phone numbers
- Labels and MSDSs of all pesticides on hand
- Personal Protective Equipment: gloves, footwear, apron, goggles, face shield, respirator
- Heavy plastic bags for material storage
- Containment “snakes” (chemsorb tubes or pads to contain & absorb spilled chemicals)
- Absorbent materials (cat litter, vermiculite, paper, etc.)
- Neutralizing agents (bleach and hydrated lime)
- Sweeping compound for dry spills
- Shovel, broom, dustpan
- Heavy duty detergent, chlorine bleach, and water
- Fire extinguisher certified for all types of fires
- Sturdy plastic container that closes tightly and will hold the largest quantity of pesticide on hand
- First aid supplies
- Fresh water (at least 3 gallons; bring extra for wash-up after application)
- Eyewash
- Soap (dish soap or hand soap)
- Towels
- Change of clothes
- Additional items required by labeling

ADDITIONAL HERBICIDE FIELD EQUIPMENT

- Extra application equipment (e.g., squeeze bottles, nalgene bottles, sponges)
- Funnel
- Herbicide dyes
- Herbicide in original containers
- Extra water, soap, towels, plastic bags

In any spill considered to be an emergency, call the local fire department. They may come to the site to help prevent further spread of the chemical but if the spill is large they will likely require a certified company to do the clean-up.

Companies often charge initial fees of roughly \$2,000 plus hourly fees of \$100/hour for the work to meet minimum legal clean-up requirements. If a spill occurs and there is uncertainty about legal requirements for reporting and clean-up, contact the program's legal staff immediately. They can ensure that all federal, state and local regulations are met.

REFERENCES

- Hawver, C. A., S. B. Gifford, and J. Hecht. 2000. Comparison of methods to control invasive plant species in the Albany Pine Bush Preserve. Albany Pine Bush Preserve Commission, Latham, New York. 50 pgs.
- Libich, S., J. C. To, R. Frank, and G. J. Sirons. 1984. Occupational exposure of herbicide applicators of herbicides used along electric power transmission line right-of-way. *Am. Ind. Hyg. Assoc. J.* 45(1):56-62.

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Chapter 6 – GENERAL PROPERTIES OF HERBICIDES

Herbicides belong to a group of chemicals known as pesticides, which prevent, destroy, repel, or mitigate any pest. Herbicides are any chemical substance that is used to specifically kill plants. Other familiar pesticides are insecticides, rodenticides, and fungicides.

MODE OF ACTION

An herbicide's mode of action is the biochemical or physical mechanism by which it kills plants. Most herbicides kill plants by disrupting or altering one or more of a their metabolic processes. Some disrupt the cellular membranes of plants, allowing cellular contents to leak out, but do not directly disrupt other metabolic processes. Some species or whole groups of plants are not susceptible to certain herbicides because they use different biochemical pathways or have slightly different enzymes. Animals typically suffer little or no effect from most herbicides sold today because these compounds principally affect processes exclusive to plants, like photosynthesis or production of aliphatic amino acids.

HERBICIDE FAMILIES VS. MODE OF ACTION

Herbicides that are chemically similar are said to belong to the same "herbicide family". The compounds in a given family typically exhibit similar characteristics and function, due to their chemical and structural similarities. For example, clopyralid, picloram, and triclopyr are all grouped in the pyridine family.

An herbicide's mode of action is the mechanism (biochemical or physical) by which it kills or suppresses plants. The mode of action is generally dictated by its chemical structure, and therefore, herbicides in the same family, tend to have the same Mode of Action. For instance, clopyralid, picloram, and triclopyr are all in the pyridine family and are all auxin mimic herbicides, while glyphosate is an amino acid inhibitor. Some herbicides from different families, however, can have the same mode of action. For example, the phenoxy 2,4-D is an auxin mimic, just like the pyridines picloram, clopyralid, and triclopyr.

The Herbicide Table in this handbook indicates the family and mode of action for each herbicide covered in this manual.

An herbicide is often chosen for use based on its mode of action. If one herbicide is ineffective, another herbicide with a different mode of action may provide better results. When and how an herbicide is applied may be determined by its mode of action.

"Pre-emergent" herbicides are those applied to the soil before the weed germinates, and either disrupt germination or kill the germinating seedling. "Post-emergent" herbicides are those that are applied directly to already established plants and/or soil. Some herbicides are effective both before ("pre-emergent") and after ("post-emergent") germination.

Some of the most common modes of action include:

- Auxin mimics (2,4-D, clopyralid, picloram, and triclopyr), which mimic the plant growth hormone auxin causing uncontrolled and disorganized growth in susceptible plant species;
- Mitosis inhibitors (fosamine), which prevent re-budding in spring and new growth in summer (also known as dormancy enforcers);
- Photosynthesis inhibitors (hexazinone), which block specific reactions in photosynthesis leading to cell breakdown;
- Amino acid synthesis inhibitors (glyphosate, imazapyr and imazapic), which prevent the synthesis of amino acids required for construction of proteins;
- Lipid biosynthesis inhibitors (fluazifop-p-butyl and sethoxydim), that prevent the synthesis of lipids required for growth and maintenance of cell membranes.

Auxin Mimics

Picloram, clopyralid, triclopyr, and 2,4-D are referred to as synthetic auxins. Auxin is a plant hormone that regulates growth in many plant tissues. Chemically, 2,4-D is classified as a phenoxy acetic acid, while picloram, clopyralid, and triclopyr are pyridine derivatives. When susceptible plants are treated with these herbicides, they exhibit symptoms that could be called 'auxin overdose', and eventually die as a result of increased rates of disorganized and uncontrolled growth.

In use since 1945, 2,4-D is one of the most studied herbicides in the world. It is known to affect many biochemical processes in plants, but it is still not clear which of the biochemical alterations 2,4-D and other auxin-mimic herbicides cause that is ultimately responsible for killing plants. It is possible that plants are weakened more or less equally by several of these disruptions with no one process being the most important.

The sequence of events following treatment with an auxin mimic herbicide differs from one species to another and depends on the age and physiological state of the individual plant. Marked changes in the permeability of the plant's cell wall or membrane can generally be detected within minutes of application. This change may lead to a rapid and sustained loss of H⁺ ions (protons) from the cell wall, which makes the wall more elastic, and often results in measurable cell growth within an hour. The loss of H⁺ ions may also lead to an accumulation of K⁺ ions in the stomatal guard cells, causing those cells to swell, increasing the size of the stomatal opening. The increased stomatal opening helps bring about a short-lived increase in photosynthesis, presumably because it allows higher concentrations of CO₂ to reach the photosynthesizing cells inside the leaf.

Other biochemical changes that occur within a day of treatment include a large increase in the concentration of soluble sugars and amino acids inside cells. This increase coincides with an increase in messenger RNA synthesis and a large increase in rates of protein synthesis. Treated plants also frequently produce ethylene, a gaseous plant hormone.

Grasses and other monocots are generally not susceptible to auxin-mimic herbicides. The reason for this selectivity is unclear because there are no apparent differences between the binding sites targeted by auxins in monocots and dicots. It may, however, be due to differences in vascular tissue structure or differences in ability to translocate or metabolize the herbicide (DiTomaso 1999).

Mitosis Inhibitors

Fosamine ammonium is another herbicide that acts as a plant growth regulator. It is sometimes referred to as a “dormancy enforcer,” but the specific mechanism of action has not been identified, even though there is evidence that fosamine ammonium inhibits mitosis in susceptible plants. When applied to deciduous plants up to two months before leaf drop, the compound is absorbed with little or no apparent effect. The following spring however, the plants fail to leaf-out because bud development is either prevented or limited to spindly, miniature leaves. Plants often die as the season progresses because they cannot produce enough photosynthate to sustain themselves. A distinctive feature of this mode of action is that treated plants do not go through a “brown-out” phase, as is often seen after the application of other herbicides. Susceptible non-deciduous plants such as pines, die soon after application because they simply do not produce enough photosynthate.

Photosynthesis Inhibitors

There are two types of photosynthesis inhibitors. Hexazinone is an example of the type that inhibits the transfer of electrons in photosystem II. It blocks electron transport from Q_A to Q_B in the chloroplast thylakoid membranes by binding to the D-1 protein at the Q_B -binding niche. The electrons blocked from passing through photosystem II are transferred through a series of reactions to other reactive toxic compounds. These compounds disrupt cell membranes and cause chloroplast swelling, membrane leakage, and ultimately cellular destruction.

Paraquat and diquat are examples of the second type of photosynthesis inhibitor. They accept electrons from Photosystem I, and after several cycles, generate hydroxyl radicals. These radicals are extremely reactive and readily destroy unsaturated lipids, including membrane fatty acids and chlorophyll. This destroys cell membrane integrity, so that cells and organelles “leak”, leading to rapid leaf wilting and desiccation, and eventually to plant death (WSSA 1994).

Amino Acid Synthesis Inhibitors

Glyphosate and imazapyr kill plants by preventing the synthesis of certain amino acids produced by plants but not animals. Glyphosate blocks the action of the enzyme 5-enolpyruvylshikimate-3-phosphate (EPSP) synthase, which inhibits the biosynthesis of certain aromatic amino acids such as phenylalanine, tyrosine, and tryptophan. These amino acids are required for protein synthesis, which, in turn, is necessary for plant growth and maintenance. Other biochemical processes such as carbohydrate translocation, can also be affected by these herbicides. Although these effects are considered secondary, they may be important in the total lethal action of glyphosate.

Imazapyr, another amino acid synthesis inhibitor, kills plants by inhibiting the production of the branched-chain aliphatic amino acids (valine, leucine, and isoleucine), which are required for DNA synthesis and cell growth. It does this by blocking acetohydroxy acid synthase (AHAS), also known as acetolactate synthase (ALS). Plants treated with imazapyr usually die slowly. The time it takes for a treated plant to die is most likely related to the amount of stored aliphatic amino acids available to the plant. AHAS (ALS) is widespread in plants but the biochemical pathway it catalyzes is not found in animals.

Lipid Biosynthesis Inhibitors

Fluazifop-p-butyl and sethoxydim are both grass specific herbicides that inhibit the synthesis of enzymes required for lipid synthesis. Both inhibit acetyl CoA carboxylase, the enzyme responsible for catalyzing an early step in fatty acid synthesis. Non-susceptible broadleaf species have a different binding site, rendering them immune. The inhibition of acetyl CoA carboxylase and the subsequent lack of lipid production leads to losses in cell membrane integrity, especially in regions of active growth such as meristems. Eventually shoot and rhizome growth ceases, and shoot meristems and rhizome buds begin to die back.

FORMULATIONS

A herbicide formulation is the total marketed product, and is typically available in forms that can be sprayed on as liquids or applied as dry solids. It includes the active ingredient(s), any additives that enhance herbicide effectiveness, stability, or ease of application such as surfactants and other adjuvants, and any other ingredients including solvents, carriers, or dyes. The application method and species to be treated will determine which formulation is best to use. In most cases, manufacturers produce formulations that make applications and handling simpler and safer. Some herbicides are available in forms that can reduce risk of exposure during mixing, such as pre-measured packets that dissolve in water, or as a liquid form already mixed with surfactant and dye (e.g., Pathfinder II[®]).

Sprayable/liquid formulations include:

1. Water-soluble formulations: soluble liquids (SL), soluble powders or packets (SP), and soluble granules (SG). Only a few herbicidal active ingredients readily dissolve in water. These products will not settle out or separate when mixed with water.
2. Emulsifiable formulations (oily liquids): emulsifiable concentrates (E or EC) and gels (GL). These products tend to be easy to handle and store, require little agitation, and will not settle out of solution. Disadvantages of these products are that most can be easily absorbed through the skin and the solvents they contain can cause the rubber and plastic parts of application equipment to deteriorate.
3. Liquid suspensions (L for liquid or F for flowable) that are dispersed in water include: suspension concentrates (SC), aqueous suspensions (AS), emulsions of water-dissolved herbicide in oil (EO), emulsions of an oil-dissolved herbicide in water (EW), micro-encapsulated formulations (ME), and capsule suspensions (CS). All these products consist of a particulate or liquid droplet active ingredient suspended in

a liquid. They are easy to handle and apply, and rarely clog nozzles. However, they can require agitation to keep the active ingredients from separating out.

4. Dry solids that are suspended in water: wettable powders (W or WP), water-dispersible granules (WDG, WG, DG), or dry flowables (DF). These formulations are some of the most widely used. The active ingredient is mixed with a fine particulate carrier, such as clay, to maintain suspension in water. These products tend to be inexpensive, easy to store, and are not as readily absorbed through the skin and eyes as ECs or other liquid formulations. These products, however, can be inhalation hazards during pouring and mixing. In addition, they require constant agitation to maintain suspension and they may be abrasive to application pumps and nozzles.

Dry formulations include:

1. Granules (G) – Granules consist of the active ingredient absorbed onto coarse particles of clay or other substance, and are most often used in soil applications. These formulations can persist for some time and may need to be incorporated into the soil.
2. Pellets (P) or tablets (TB) – Pellets are similar to granules but tend to be more uniform in size and shape.
3. Dusts (D) – A dust is a finely ground pesticide combined with an inert or inactive dry carrier. They can pose a drift or inhalation hazard.

Salts vs. Esters

Many herbicidally active compounds are acids that can be formulated as a salt or an ester for application. Once the compound enters the plant, the salt or ester cation is cleaved off allowing the parent acid (active ingredient) to be transported throughout the plant. When choosing between the salt or ester formulation, consider the following characteristics:

Salts

- Most salts are highly water soluble, which reduces the need for emulsifiers or agitation to keep the compound suspended.
- Salts are not soluble in oil.
- Salts generally require a surfactant to facilitate penetration through the plant cuticle (waxy covering of leaves and stems).
- Salts are less volatile than esters.
- Salts can dissociate in water. In hard water the parent acid (i.e. the active ingredient) may bind with calcium and magnesium in the water, precipitate out, and be inactivated.

Esters

- Esters can penetrate plant tissues more readily than salts, especially woody tissue
- Esters generally are more toxic to plants than salts
- Esters are not water soluble and require an emulsifying agent to remain suspended in water-based solvents
- Esters have varying degrees of volatility

Adjuvants (including surfactants)

An adjuvant is any material added to a pesticide mixture that facilitates mixing, application, or pesticide efficacy. An adjuvant enables an applicator to customize a formulation to be most effective in a particular situation. Adjuvants include surfactants, stickers, extenders, activators, compatibility agents, buffers and acidifiers, deposition aids, de-foaming agents, thickeners, and dyes. See the Adjuvant Chapter (Chapter 8) in this handbook for more details on adjuvants.

Surfactants

Surfactants are the most important adjuvants. They are chemical compounds that facilitate the movement of the active herbicide ingredient into the plant. They may contain varying amounts of fatty acids that are capable of binding to two types of surfaces, such as oil and water. Some herbicide formulations come with a surfactant already added, in others, surfactants can be added prior to application. Whether a surfactant should be added will be determined by the type of herbicide being applied and the target plant. Read the label for recommendations of appropriate surfactants.

MECHANISMS OF DISSIPATION

Dissipation refers to the movement, degradation, or immobilization of an herbicide in the environment.

Degradation

Degradation occurs when an herbicide is decomposed to smaller component compounds, and eventually to CO₂, water, and salts through photochemical, chemical, or biological (microbial metabolism) reactions (Freed and Chiou 1981). Biodegradation accounts for the greatest percentage of degradation for most herbicides (Freed and Chiou 1981). When a single herbicide degrades, it usually yields several compounds (“metabolites”), each of which has its own chemical properties including toxicity, adsorption capacity, and resistance to degradation. Some metabolites are more toxic and/or persistent than the parent compound. In most cases, the nature of the metabolites are largely unknown.

Photodegradation

Photodegradation refers to decomposition by sunlight. Sunlight intensity varies with numerous factors including latitude, season, time of day, weather, pollution, and shading by soil, plants, litter, etc. Studies of the photodegradation of herbicides are often conducted using UV light exclusively, but there is some debate as to whether most UV light actually reaches the surface of the earth. Therefore, photodegradation rates determined in the laboratory may over-estimate the importance of this process in the field (Helling et al. 1971).

Microbial Degradation

Microbial degradation is decomposition through microbial metabolism. Different microbes can degrade different herbicides, and consequently, the rate of microbial degradation depends on the microbial community present in a given situation (Voos and

Groffman 1997, McCall et al. 1981). Soil conditions that maximize microbial degradation include warmth, moisture, and high organic content.

Herbicides may be microbially degraded via one of two routes. They may be metabolized directly when they serve as a source of carbon and energy (i.e. food) for microorganisms (Hutzinger 1981), or they may be co-metabolized in conjunction with a naturally occurring food source that supports the microbes (Hutzinger 1981). Herbicides that are co-metabolized do not provide enough energy and/or carbon to support the full rate of microbial metabolism on their own.

There is sometimes a lag time before microbial degradation proceeds. This may be because the populations of appropriate microbes or their supplies of necessary enzymes start small, and take time to build-up (Farmer and Aochi 1987, Kearney and Karns 1960). If this lag time is long, other degradation processes may play more important roles in dissipation of the herbicide (Farmer and Aochi 1987). Degradation rates of co-metabolized herbicides tend to remain constant over time.

Chemical Decomposition

Chemical decomposition is degradation driven by chemical reactions, including hydrolyzation (reaction with hydrogen, usually in the form of water), oxidation (reaction with oxygen), and disassociation (loss of an ammonium or other chemical group from the parent molecule). The importance of these chemical reactions for herbicide degradation in the field is not clear (Helling et al. 1971).

Immobilization/Adsorption

Herbicides may be immobilized by adsorption to soil particles or uptake by non-susceptible plants. These processes isolate the herbicide and prevent it from moving in the environment, but both adsorption and uptake are reversible. In addition, adsorption can slow or prevent degradation mechanisms that permanently degrade the herbicide.

Adsorption refers to the binding of herbicide by soil particles, and rates are influenced by characteristics of the soil and of the herbicide. Adsorption is often dependent on the soil or water pH, which then determines the chemical structure of the herbicide in the environment. Adsorption generally increases with increasing soil organic content, clay content, and cation exchange capacity, and it decreases with increasing pH and temperature. Soil organic content is thought to be the best determinant of herbicide adsorption rates (Farmer and Aochi 1987, Que Hee and Sutherland 1981, Helling et al. 1971). Adsorption is also related to the water solubility of an herbicide, with less soluble herbicides being more strongly adsorbed to soil particles (Helling et al. 1971). Solubility of herbicides in water generally decreases from salt to acid to ester formulations, but there are some exceptions. For example, glyphosate is highly water-soluble and has a strong adsorption capacity.

The availability of an herbicide for transport through the environment or for degradation is determined primarily by the adsorption/desorption process (WHO 1984). Adsorption

to soil particles can stop or slow the rate of microbial metabolism significantly. In other cases, adsorption can facilitate chemical or biological degradation (Farmer and Aochi 1987). Adsorption can change with time and, in most cases, is reversible (i.e. the herbicide can desorb from the soil or sediments and return to the soil solution or water column).

Movement/Volatilization

Movement through the environment occurs when herbicides are suspended in surface or subsurface runoff, volatilized during or after application, evaporated from soil and plant surfaces, or leached down into the soil. Although generally studied and discussed separately, these processes actually occur simultaneously and continuously in the environment (Hutzinger 1981).

Volatilization occurs as the herbicide passes into the gaseous phase and moves about on the breeze. Volatilization most often occurs during application, but also can occur after the herbicide has been deposited on plants or the soil surface. The volatility of an herbicide is determined primarily by its molecular weight. Most highly volatile herbicides are no longer used.

Volatility generally increases with increasing temperature and soil moisture, and with decreasing clay and organic matter content (Helling et al. 1971). The use of a surfactant can change the volatility of a herbicide (Que Hee and Sutherland 1981). In extreme cases, losses due to volatilization can be up to 80 or 90% of the total herbicide applied (Taylor and Glotfelty 1988). Of the herbicides described in detail in this handbook, only 2,4-D and triclopyr can present significant volatilization problems in the field (T. Lanini, pers. comm.).

BEHAVIOR IN THE ENVIRONMENT

Perhaps the most important factor determining the fate of herbicide in the environment is its solubility in water (Hutzinger 1981). Water-soluble herbicides generally have low adsorption capacities, and are consequently more mobile in the environment and more available for microbial metabolism and other degradation processes. Esters, in general, are relatively insoluble in water, adsorb quickly to soils, penetrate plant tissues readily, and are more volatile than salt and acid formulations (Que Hee and Sutherland 1981).

Soils

An herbicide's persistence in soils is often described by its half-life (also known as the DT₅₀). The half-life is the time it takes for half of the herbicide applied to the soil to dissipate. The half-life gives only a rough estimate of the persistence of an herbicide since the half-life of a particular herbicide can vary significantly depending on soil characteristics, weather (especially temperature and soil moisture), and the vegetation at the site. Dissipation rates often change with time (Parker and Doxtader 1983). For example, McCall et al. (1981) found that the rate of dissipation increased until

approximately 20% of the applied herbicide remained, and then declines. Nonetheless, half-life values do provide a means of comparing the relative persistence of herbicides.

The distribution of an herbicide in the soil is determined primarily by the amount, type, and surface area of clays and organic matter in the soil, the amount and quality of soil moisture, and soil temperature and soil pH (Helling et al. 1971). Most natural soils have pH values between 5 and 8 (V. Claassen, pers. comm.). Rainfall and the amount of leaching that has occurred strongly influences these values. In wet areas and/or coarse soils, cations can be leached out, leaving the soil acidic. In arid and semi-arid regions, soils retain cations and are more alkaline. Acidic soils can also be found in bogs where organic acids lower the soil's pH.

Water

Water bodies can be contaminated by direct overspray, or when herbicides drift, volatilize, leach through soils to groundwater, or are carried in surface or subsurface runoff. Amounts of leaching and runoff are largely dependent on total rainfall the first few days after an application. Total losses to runoff generally do not exceed five to ten percent of the total applied, even following heavy rains (Taylor and Glotfelty 1988). High soil adsorption capacity, low rates of application, and low rainfall reduce total runoff and contamination of local waterways (Bovey et al. 1978).

The behavior of an herbicide in water is dictated by its solubility in water. Salts and acids tend to remain dissolved in water until degraded through photolysis or hydrolysis. Esters will often adsorb to the suspended matter in water, and precipitate to the sediments. Once in the sediments, esters can remain adsorbed to soil particles or be degraded through microbial metabolism. Highly acidic or alkaline waters can chemically alter an herbicide and change its behavior in water. The average pH of surface waters is between five and nine (Hutzinger 1981).

ENVIRONMENTAL TOXICITY

The toxicology information reported in this handbook is for the technical grade of the herbicide unless otherwise noted. In some cases, it is not the herbicide itself that is the most toxic component of the applied formula. Adjuvants, such as petroleum solvents (e.g. diesel fuel, deodorized kerosene, methanol), can be highly toxic (Ware 1991). In addition, impurities resulting from the manufacturing process can be more toxic than the active ingredient itself.

Birds and Mammals

A herbicide's toxicity is described by its LD50, which is the dose received either orally (taken through the mouth) or dermally (absorbed through the skin) that kills half the population of study animals. The oral LD50s reported here were determined for adult male rats. The dermal LD50s were determined for rabbits. The LD50 is typically reported in grams of herbicide per kilogram of animal body weight. LD50s are determined under varying circumstances so comparisons between different herbicides

may provide only a rough sense of their relative toxicities. Dermal LD50 values may be more meaningful to herbicide applicators because they are more likely to be exposed to herbicide through their skin rather than by oral ingestion. In any event, very few people, even among applicators, are exposed to herbicide doses as high as the LD50.

The LD50 does not provide any information about chronic, long-term toxic effects that may result from exposure to lesser doses. Sublethal doses can lead to skin or eye irritation, headache, nausea, and, in more extreme cases, birth defects, genetic disorders, paralysis, cancer, and even death. Impurities derived from the formulation of the herbicide and the adjuvants added to the formulation may be more toxic than the herbicide compound itself, making it difficult to attribute increased risks of cancer or other effects directly to a herbicide (Ibrahim et al. 1991).

The most dramatic effects of herbicides on non-target plants and animals often result from the habitat alterations they cause by killing the targeted weeds. For example, loss of invasive riparian plants can cause changes in water temperature and clarity that can potentially impact the entire aquatic community, and the physical structure of the system through bank erosion. Removing a shrubby understory can make a habitat unsuitable for certain bird species and expose small mammals to predation.

Aquatic Species

A herbicide's toxicity to aquatic organisms is quantified with the LC50, which is the concentration of herbicide in water required to kill half of the study animals. The LC50 is typically measured in micrograms of pesticide per liter of water.

In general, ester formulations are more dangerous for aquatic species than salt and acid formulations because ester formulations are lipophilic (fat-loving), and consequently, can pass through the skin and gills of aquatic species relatively easily. Ester formulations, additionally, are not water soluble, and are less likely to be diluted in aquatic systems.

Soil Microbes

Herbicides have varying effects on soil microbial populations depending on herbicide concentrations and the microbial species present. Low residue levels can enhance populations while higher levels can cause population declines. In many cases, studies are too short in duration to determine the true long-term impacts of herbicide use on soil microbes.

HUMAN TOXICOLOGY

When proper safety precautions are taken, human exposure to herbicides used in natural areas should be minimal. Properly fitted personal protection equipment and well-planned emergency response procedures will minimize exposure from normal use as well as emergency spill situations.

Exposure

Agricultural workers are often exposed to herbicides when they unintentionally re-enter a treated area too soon following treatment. People who mix and apply herbicides are at the greatest risk of exposure. The most common routes of exposure are through the skin (dermal) or by inhalation (to the lungs). Accidental spills or splashing into the eyes is also possible and with some compounds, can result in severe eye damage and even blindness.

Agricultural herbicide applicators are typically exposed to herbicide levels ranging from micrograms to milligrams per cubic meter of air through inhalation, but exposures through the skin are thought to be much greater (Spear 1991). Spilling concentrated herbicide on exposed skin can be the toxic equivalent of working all day in a treated field (Libich et al. 1984). Dermal exposure can occur to the hands (directly or through permeable gloves), splashes onto clothing or exposed skin, and anywhere you wipe your hands (e.g., thighs, brow). Some tests have found relatively high levels of dermal exposure to the crotch and seat of workers who got herbicide on their hands, and then touched or wiped the seat of their vehicles. Because adsorption through the skin is the most common route of exposure for applicators (Marer 1988), the dermal LD50 may provide more practical information on the relative toxicity of an herbicide rather than the oral LD50, which is based on oral ingestion.

Toxic Effects

A person's reaction to pesticide poisoning depends on the toxicity of the pesticide, the size of the dose, duration of exposure, route of absorption, and the efficiency with which the poison is metabolized and excreted by the person's body (Marer 1988, Ware 1991). Different individuals can have different reactions to the same dose of herbicide. Smaller people are, in general, more sensitive to a given dose than are larger people (Marer 1988).

Herbicides can poison the body by blocking biochemical processes or dissolving or disrupting cell membranes. Small doses may produce no response while large doses can cause severe illness or death. The effects may be localized, such as irritation to the eyes, nose, or throat, or generalized, such as occurs when the compound is distributed through the body via the blood stream. Symptoms can occur immediately after exposure or develop gradually. Injuries are usually reversible, but in extreme cases can be permanently debilitating (Marer 1988).

Common symptoms of low-level exposure (such as occurs when mixing or applying herbicides in water) to many herbicides include skin and eye irritation, headache, and nausea. Higher doses (which can occur when handling herbicide concentrates) can cause blurred vision, dizziness, heavy sweating, weakness, stomach pain, vomiting, diarrhea, extreme thirst, and blistered skin, as well as behavioral alterations such as apprehension, restlessness, and anxiety (Marer 1988). Extreme cases may result in convulsions, unconsciousness, paralysis, and death.

Impurities produced during the manufacturing process and adjuvants added to the formulation may be more toxic than the herbicide compound itself. Consequently,

LD50s determined for the technical grade of the herbicide may not be the same as that for the brand name formulation. Combinations of herbicides furthermore, can have additive and synergistic effects in which a formulation of two or more herbicides is two to 100 times as toxic as any one of the herbicides alone (Thompson 1996). Labels should be read carefully for manufacturer's warnings and safety precautions that may be required for a particular formulation.

NOTE: It is important to remember while interpreting study results discussed in this manual and elsewhere that changes in technology have lowered the detectable residue level 1,000-fold over the last twenty years. Herbicide residues that could only be detected to the parts per million (ppm) level (e.g. one microgram of pesticide per kilogram of soil) in the early 1970's can now be detected at the parts per billion (ppb) level (e.g., one microgram of pesticide per 1,000 kilograms of soil). When a study reports finding no residues it really means that no residues above the lowest detectable level were found. This can be an important difference in comparing the results of studies conducted in the 1960's and 70's to studies from the 1980's and 90's.

REFERENCES

- Bovey, R. W., C. Richardson, E. Burnett, M. G. Merkle, and R. E. Meyer. 1978. Loss of spray and pelleted picloram in surface runoff water. *J. Environ. Qual.* 7(2):178-180.
- DiTomaso, J. M. 1999. Growth regulators. Presented at the UC Davis Weed Science School, Feb. 24-26 and March 1-3, 1999. Davis, California.
- EXTOXNET. 1993. Manifestations of toxic effects. Extension Toxicology Network. Toxicology Information Briefs. <http://ace.orst.edu/info/extoxnet/tibs/manifest.htm>
- Farmer, W. J., and Y. Aochi. 1987. Chemical conversion of pesticides in the soil-water environment. Chapter 7 *in* Fate of pesticides in the environment. J. W. Biggar and J. N. Seiber, eds. University of California, Publication 3320.
- Freed, V. H., and C. T. Chiou. 1981. Physicochemical factors in routes and rates of human exposure to chemicals. Chapter 3 *in* Environmental health chemistry. J. D. McKinney, ed. Ann Arbor Science Publishers Inc., Ann Arbor, Michigan.
- Helling, C. S., P. C. Kearney, and M. Alexander. 1971. Behavior of pesticides in soil. *Adv. Agron.* 23:147-240.
- Hutzinger, O. 1981. Environmental and toxicological chemistry at the University of Amsterdam: Five years of philosophy and practice of environmental health chemistry. Chapter 2 *in* Environmental health chemistry. J. D. McKinney, ed. Ann Arbor Science Publishers Inc., Ann Arbor, Michigan.
- Kearny, P. C., and J. S. Karns. 1987. Microbial Metabolism. Chapter 10 *in* Fate of pesticides in the environment. J. W. Biggar and J. N. Seiber, eds. University of California, Publication 3320.
- Marer, P. J., M. L. Flint, and M. W. Stimmann. 1988. The safe and effective use of pesticides. University of California, Division of Agriculture and Natural Resources. Publication 3324. 387 pps.

- McCall, P. J., S. A. Vrona, and S. S. Kelley. 1981. Fate of uniformly carbon-14 ring labeled 2,4,5-Trichlorophenoxyacetic acid and 2,4-Dichlorophenoxyacetic acid. *J. Agric. Food Chem.* 29:100-107.
- Parker, L. W., and K. G. Doxtader. 1983. Kinetics of the microbial degradation of 2,4-D in soil: effects of temperature and moisture. *J. Environ. Qual.* 12(4):553-558.
- Que Hee, S. S., and R. G. Sutherland. The phenoxyalkanoic herbicides. Vol 1, CRC series in pesticide chemistry. CRC Press, Boca Raton, Fla.
- Spear, R. 1991. Recognized and possible exposure to pesticides. Chapter 6 *in* Handbook of pesticide toxicology, Vol. 1, General Principles. W.J. Hayes, Jr. and E. R. Laws, Jr. eds. Academic Press, Inc. San Diego, California. 1576 pages.
- Taylor, A. W., and D. E. Glotfelty. 1988. Evaporation from soils and crops. Chapter 4 *in* Environmental chemistry of herbicides, Vol I. R. Grover, ed. CRC Press, Boca Raton, Fla.
- Thompson, H. M. 1996. Interactions between pesticides; a review of reported effects and their implications for wildlife risk assessment. *Ecotoxicology* 5:59-81.
- Voos, G. and P. M. Groffman. 1997. Relationships between microbial biomass and dissipation of 2,4-D and dicamba in soil. *Biol. Fertil. Soils* 24:106-110.
- Ware, G. W.. 1991. Fundamentals of pesticides: A self-instruction guide. Thomson Publications. Fresno, California. 307 pgs.
- World Health Organization. 1984. 2,4-Dichlorophenoxyacetic acid (2,4-D), Environmental Health Criteria 29. United Nations Environment Programme, Geneva. 151 pgs.
- WSSA. 1994. Herbicide Handbook. Weed Science Society of America. Champaign, Illinois. 352pp.

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Herbicide	Brand Name Examples	Herbicide Properties				Y2000 prices for some trade products
		Chemical Name	Herbicide Family	Target Weed Sps.	Mode of Action	
2,4 D	Navigate [®] , Class [®] , Weed-Pro [®] , Justice [®]	(2,4-dichlorophenoxy) acetic acid	phenoxy	broadleaf weeds	Auxin mimic	\$35/gal WeedHo
Clopyralid	Reclaim [®] , Curtail [®] , Transline [®]	3,6-dichloro-2-pyridinecarboxylic acid	pyridine	annual and perennial broadleaf weeds	Auxin mimic	\$358/gal Transline
Fluazifop-p-Butyl	Fusilade DX [®] , Fusion [®] , Tornado [®]	(R)-2-[4-[[5-(trifluoromethyl)-2-pyridinyl]oxy]phenoxy]propanoic acid	aryloxyphenoxy-propionate	annual and perennial grasses	Inhibits acetyl-CoA carboxylase prohibiting fatty acid synthesis	\$65/qt Fusilade
Fosamine	Krenite [®]	ethyl hydrogen (aminocarbonyl) phosphonate	none generally recognized	trees and bushes	Mitotic inhibitor	\$55/gal Krenite
Glyphosate	RoundUp [®] , Rodeo [®] , Accord [®]	N-(phosphonomethyl)glycine	none generally recognized	annual and perennial weeds	Inhibits the shikimic acid pathway depleting aromatic amino acids	\$141 for 2 1/2 gal RoundUp; \$80/qt Rodeo
Hexazinone	Velpar [®] , Pronone [®]	3-cyclohexyl-6-(dimethylamino)-1-methyl-1,3,5-triazine-2,4(1H,3H)-dione	triazine	annual, biennials, perennial	Blocks Photosystem II	\$83/gal Velpar; \$469/lb Pronone
Imazapic	Plateau [®] , Plateau Eco-Pak [®] , Cadre [®]	(±)-2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-5-methyl-3-pyridinecarboxylic acid	imidazolinone	annual and perennial weeds	Inhibits AHAS synthesis, blocking amino acid synthesis	\$308/gal Plateau; \$72/Eco-Pak
Imazapyr	Arsenal [®]	(+)-2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-3-pyridinecarboxylic acid	imidazolidinone	perennial grasses, broadleaves, vines, brambles, brush,	Inhibits acetolactate synthase blocking amino acid synthesis	\$358/gal Arsenal
Picloram	TordonK [®]	4-amino-3,5,6-trichloro-2-pyridinecarboxylic acid	pyridine	perennial broadleaf weeds, vines, and woody	Auxin mimic	\$43/gal Tordon 101; \$107/gal Tordon K
Sethoxydim	Poast [®]	2-[1-(ethoxyimino)butyl]-5-[2-(ethylthio)propyl]-3-hydroxy-2-cyclohexen-1-one	cyclohexanedione	annual and perennial grasses	Lipid synthesis inhibitor	\$204/gal Poast
Triclopyr	Garlon [®] , Remedy [®]	[(3,5,6-trichloro-2-pyridinyl)oxy]acetic acid	pyridine	woody and annual broadleaf weeds	Auxin mimic	\$295 for 2 1/2 gal Garlon; \$155/gal Remedy

Weed Control Methods Handbook, The Nature Conservancy, Tu *et al.*

Herbicide	Behavior in Soils			Behavior in Water	
	Average Soil Half-life	Soil Sorption (Koc)	Mobility (based on Helling's classification system - Helling & Turner 1968)	Water Solubility	Average Half-life in Water
2,4 D	10 days	20 mL/g (acid/salt), 100 mL/g (ester)	moderate-high	900 mg/L (acid), 100 mg/L (ester), 796,000 mg/L (salt)	varies from hours to months
Clopyralid	40 days	avg 6 mL/g but ranges to 60 mL/g	moderate-high	1,000 mg/L (acid), 300,000 mg/L (salt)	8-40 days
Fluazifop-p-Butyl	15 days	5,700 mL/g	moderate*	1.1 mg/L	stable in water
Fosamine	8 days	150 mL/g	moderate*	1,790,000 mg/L	stable in water
Glyphosate	47 days	24,000 mL/g	low	15,700 mg/L (acid), 900,000 mg/L (IPA salt), 4,300,000 mg/L	12 days to 10 weeks
Hexazinone	90 days	poor, Koc unk.	moderate-high	33,000 mg/L	several days to > 9 months
Imazapic	120-140 days	206 mL/g	low?	36,000 mg/L (pH 7)	< 8 hours
Imazapyr	25-141 days	poor, Koc unk.	low-moderate	11,272 mg/L	2 days
Picloram	90 days	16 mg/L (can range -17-160 mL/g)	moderate-high	430 mg/L (acid), 200,000 (salts)	2-3 days
Sethoxydim	5 days	100 mL/g	high*	257 mg/L (pH 5), 4,390 mg/L (pH 7)	hours in sunlight
Triclopyr	30 days	20 mL/g (salt), 780 mL/g (ester)	moderate-high *unofficial estimate	430 mg/L (acid), 23 mg/L (ester), 2,100,000 mg/L (salt)	4 days

Herbicide	Degradation Mechanism			Toxicity & [EPA Toxicity Categories*]			
	Microbial Degradation	Chemical	Solar Degradation	Oral LD50 - Mammals (Rats)	LD50 - Birds (BW - bobwhite quail, M - mallards)	LC50 - Fish (bluegill sunfish)	Dermal LD50 - Rabbit
2,4 D	Primary mechanism	Minor mechanism	Low potential	764 mg/kg [low]	500 mg/kg (BW) [moderate]	263 mg/L [moderate]	NA
Clopyralid	Primary mechanism	Minor mechanism	Low potential	4,300 mg/kg [low]	1,465 mg/kg (M) [low]	125 mg/L [moderate]	>2000 mg/kg
Fluazifop-p-Butyl	Primary mechanism	Secondary mechanism	Low potential	4,096 mg/kg [low]	>3,528 mg/kg (M) [low]	0.53 mg/L [high]	>2420 mg/kg
Fosamine	Primary mechanism	Very minor mechanism	Very low potential	24,000 mg/kg [slight]	10,000 mg/kg (BW/M) [slight]	670 mg/L [low]	>1683 mg/kg
Glyphosate	Primary mechanism	Minor mechanism	Low potential	5,600 mg/kg [slight]	> 4,640 mg/kg (BW/M) [low]	120 mg/L [moderate]	>5000 mg/kg
Hexazinone	Primary mechanism	Minor mechanism	Moderate potential	1,690 mg/kg [low]	2,258 mg/kg (BW) [low]	370 mg/L [moderate]	>6000 mg/kg
Imazapic	Primary mechanism	Very minor mechanism?	Low?	> 5,000 mg/kg [slight]	> 2,150 mg/kg (BW) [low]	> 100 mg/L [moderate]	> 5000 mg/kg
Imazapyr	Primary mechanism	Minor mechanism	Moderate potential	> 5,000 mg/kg [slight]	> 2,150 mg/kg (BW/M) [low]	>100 mg/L [moderate]	>2000 mg/kg
Picloram	Primary mechanism	Primary mechanism	Moderate potential	> 5,000 mg/kg [slight]	> 2,510 mg/kg (M) [low]	>14.4 mg/L [high]	>2000 mg/kg
Sethoxydim	Primary mechanism	Minor mechanism	High potential	>2,676 mg/kg [low]	> 2,510 mg/kg (M) [low]	100 mg/L [moderate]	>5000 mg/kg
Triclopyr	Primary mechanism	Minor mechanism	Moderate potential	713 mg/kg [low]	1,698 mg/kg (M) [low]	148 mg/L [moderate]	>2000 mg/kg
				*based on EPA Toxicity Categories			

Herbicide	Notes
2,4 D	Inexpensive and common herbicide used for over 50 years.
Clopyralid	Highly selective herbicide developed as an alternative to picloram.
Fluazifop-p-Butyl	Toxic to most grasses except annual bluegrass and all fine fescues.
Fosamine	Not registered for use in California or Arizona.
Glyphosate	Little to no soil activity. Some formulations are highly toxic to aquatic organisms.
Hexazinone	Potential for ground water contamination. Toxic to algae.
Imazapic	Degree of control depends on selectivity of individual plants.
Imazapyr	Provides long-term total vegetation control.
Picloram	Environmental persistence can endanger non-target plants and animals.
Sethoxydim	Rapid degradation can limit effectiveness.
Triclopyr	Commonly used herbicide. The ester formulation is highly toxic to aquatic organisms.
	Date Authored: April 2001

2,4-D

Herbicide Basics

Chemical formula: (2,4-dichlorophenoxy) acetic acid

Herbicide Family: Phenoxy

Target weeds: broadleaves

Forms: salt & ester

Formulations: EC, WP, SL, GR, SP

Mode of Action: Auxin mimic

Water Solubility: 900 ppm

Adsorption potential: low-intermediate (higher for ester than salt)

Primary degradation mech: Microbial metabolism

Average Soil Half-life: 10 days

Mobility Potential: intermediate

Dermal LD50 for rabbits: unknown

Oral LD50 for rats: 764 mg/kg

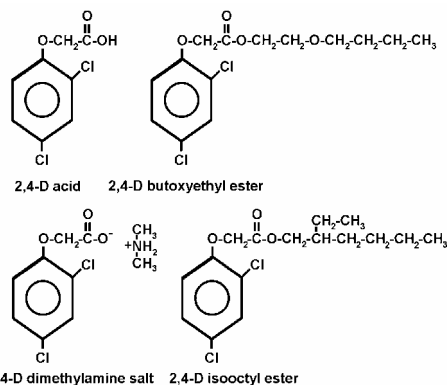
LC50 for bluegill sunfish: 263 mg/L

Trade Names: Aqua-Kleen[®], Barrage[®], and Weedone[®]

Manufacturers: Current manufacturers include Aventis, Dow AgroSciences, and Nufarm, U.S.A.

Synopsis

2,4-D is one of the oldest herbicides used in the United States. It was first developed during World War II and became famous as a component of the controversial Agent Orange used during the Vietnam War. Today, 2,4-D continues to be one of the most commonly used herbicides on the market. Because there is no longer a patent governing the manufacture and sale of 2,4-D, any company is free to produce it. Thus, a variety of inexpensive 2,4-D products are available from different manufacturers. Because it has been in use for so long, many of the studies regarding its behavior in the environment are old (e.g. pre-1980). 2,4-D is a selective herbicide that kills dicots (but not grasses) by mimicking the growth hormone auxin, which causes uncontrolled growth and eventually death in susceptible plants. The half-life of 2,4-D in the environment is relatively short, averaging 10 days in soils and less than ten days in water, but can be significantly longer in cold, dry soils, or where the appropriate microbial community is not present to facilitate degradation. In the environment, most formulations are degraded to the anionic form, which is water-soluble and has the potential to be highly mobile. Ester formulations are toxic to fish and aquatic invertebrates, but salt formulations are registered for use against aquatic weeds. 2,4-D is of relatively low toxicity to animals but some formulations can cause severe eye damage. Certain crops, such as grapes, are highly sensitive to 2,4-D and application of this herbicide should be avoided if they are nearby. Most formulations are highly volatile and should not be applied when conditions are windy or when temperatures are high.



Herbicide Details

Chemical Formula: (2,4-dichlorophenoxy) acetic acid

Trade Names: 2,4-D is sold as acid, salt (mostly amine), or ester formulations under many different trade names. Formulations include liquids, water-soluble powders, dusts, granules, or pellets of 2,4-D alone or in mixtures with other herbicides such as picloram and clopyralid. Some trade names include Aqua-Kleen[®], Barrage[®], Lawn-Keep[®], Malerbane[®], Planotox[®], Plantgard[®], Savage[®], Salvo[®], Weedone[®], Weedar[®] and Weedtrine-II[®].

Manufacturer: Current manufacturers include Aventis, Dow AgroSciences, Nufarm U.S.A., and many others.

History: 2,4-D is commonly known as a component of the controversial herbicide Agent Orange, which was extensively used by the U.K. in Malaysia and by the U.S. military during the Vietnam War to defoliate jungle regions. Pure 2,4-D and 2,4,5-T (the other component of Agent Orange) are relatively non-toxic. Agent Orange's infamy was primarily due to dioxin contamination of the 2,4-D and 2,4,5-T herbicides that it contained. 2,4-D is now manufactured with a process that produces no dioxin as a contaminant. It proved impossible to produce 2,4,5-T that was free of dioxin contamination, so its manufacture and sale have been prohibited in the U.S. since 1983. Small quantities of this dioxin is highly toxic, and has been linked with producing birth defects in mammals and increased rates of cancer.

Use Against Natural Area Weeds: 2,4-D controls many terrestrial and aquatic broadleaf weeds, but has little or no affect on grasses. Weeds that have been treated with 2,4-D in natural areas include: Canada thistle (*Cirsium arvense*), *Cardaria* spp., crown vetch (*Coronilla varia*), Russian knapweed (*Acroptilon repens*), water hyacinth (*Eichhornia crassipes*), and sulfur cinquefoil (*Potentilla recta*).

On some TNC preserves, 2,4-D has been used with moderate to high success against Canada thistle (*Cirsium arvense*). On TNC preserves in Oregon, hoary cress (*Cardaria draba*) was treated when in bud or flower for seven years, and although a few plants still appear every year, the weed has been nearly eliminated. Land stewards in Montana found that 2,4-D amine plus picloram is cheaper but less effective against leafy spurge than higher rates of picloram (Tordon[®]) alone. However, lower application rates may cause less environmental damage. Formulations that contain 2,4-D mixed other herbicides, such as Crossbow[®] (2,4-D and triclopyr), Curtail[®] (2,4-D and clopyralid), Pathway[®] (2,4-D and picloram), and Weedmaster[®] (2,4-D and dicamba), have been used on TNC preserves with varying degrees of success.

Mode of Action: 2,4-D is an "auxin mimic" or synthetic auxin. This type of herbicide kills the target weed by mimicking the plant growth hormone auxin (indole acetic acid), and when administered at effective doses, causes uncontrolled and disorganized plant growth that leads to plant death. The exact mode of action of 2,4-D is not fully understood, and it is possible that it causes a variety of effects which are fatal when combined. It is believed to acidify the cell walls which allows the cells to elongate in an uncontrolled manner. Low concentrations of 2,4-D can

also stimulate RNA, DNA, and protein synthesis leading to uncontrolled cell division and growth, and, ultimately, vascular tissue destruction. On the other hand, high concentrations of 2,4-D can inhibit cell division and growth. Plant death typically occurs within three to five weeks following application.

Dissipation Mechanisms:

Summary: In soils 2,4-D is degraded primarily by microbes. The fate of 2,4-D in the environment is largely dependent on the ambient pH (Aly & Faust 1964). At pH levels above 7, 2,4-D is converted rapidly to the anion (negatively charged) form, which is more susceptible to photodegradation and microbial metabolism, and less likely to adsorb to soil particles. At pH levels < 4, microbial degradation is inhibited, and 2,4-D retains its molecular form and resists degradation (Johnson et al. 1995a). Most formulations of 2,4-D are volatile (T. Lanini, pers. com.).

Volatilization

Most formulations of 2,4-D can be highly volatile and care should be used in their application. The most volatile of the 2,4-D esters, methyl and isopropyl, have been banned in the U.S. (Que Hee & Sutherland 1981), but some volatile ester formulations of 2,4-D remain available. Both localized and widespread damage from using 2,4-D have been reported (WHO 1984). To reduce the amount lost to vaporization, low-volatile (long-chain) ester formulations are available. In addition, the alkali and amine salt formulations are much less volatile and may be more appropriate for use where esters could volatilize and damage non-target plants (WHO 1984). Additionally, the potential for 2,4-D to volatilize increases with increasing temperature, increasing soil moisture, and decreasing clay and organic matter content in the soil (Helling et al. 1971).

Photodegradation

2,4-D degrades rapidly in sunlight under laboratory conditions, but photodegradation has not been demonstrated in the field (Halter 1980). Crosby and Tutass (1966) reported half-lives (the time it takes for half of the total amount of herbicide applied to be dissipated) of 50 minutes for 2,4-D salts and five minutes for 2,4-D esters under laboratory conditions. Aly and Faust (1964) obtained similar results in the lab but concluded that sufficient levels of ultraviolet radiation from sunlight are not likely in the field. In addition, Johnson et al. (1995 a & b) reported that 2,4-D degradation rates in soils remained relatively constant with and without sunlight, suggesting that photodegradation is not an important process in the field.

Microbial Degradation

In soils 2,4-D is degraded primarily by microbes. Hemmett and Faust (1969) concluded that the size of the microbial population, the concentration of 2,4-D, and the ratio of the two factors determine 2,4-D degradation rates. Soil conditions that enhance microbial populations (i.e. warm and moist) facilitate 2,4-D degradation (Foster & McKercher 1973). In addition, 2,4-D has been shown to dissipate more rapidly in soils that were previously treated with 2,4-D, presumably because there was an increase in 2,4-D degrading bacteria after the first application (Oh & Tuovinen 1991; Smith & Aubin 1994; Shaw & Burns 1998).

There are conflicting reports as to whether microbial degradation occurs in aquatic systems (Que Hee & Sutherland 1981; Wang et al. 1994a; EXTTOXNET 1996). Microbial degradation can take place in bottom sediments if the appropriate microbial population is present and the pH level is sufficiently high, but it is not likely to occur in the water column (Que Hee & Sutherland 1981). Under acidic conditions, microbial activity can be severely inhibited (Sandmann et al. 1991). Differences in reported half-lives may arise from differences in the microbial populations at the study sites (Shaw & Burns 1998). Some aquatic systems may have few of the microbes that readily degrade 2,4-D, while others may have many 2,4-D degrading microbes.

Adsorption

Salt formulations are water-soluble and do not bind strongly with soils. Ester formulations can adsorb more readily to soils. In the field, ester formulations tend to hydrolyze to the acid form, particularly in alkaline conditions, and, consequently, do not adsorb to soil particles in significant quantities (Aly & Faust 1964).

Johnson et al. (1995a) found that soil organic content and soil pH are the main determinants of 2,4-D adsorption in soils. Adsorption increases with increasing soil organic content and decreasing soil pH (Johnson et al. 1995a). Inorganic clays can also bind 2,4-D particles. A relatively high concentration of clay particles however, is required to bind small concentrations of 2,4-D (Aly & Faust 1964). Additionally, as the herbicide concentration increases, the percentage of herbicide adsorbed decreases, possibly because the number of binding sites on soil particles are finite and become filled (Johnson et al. 1995a).

Chemical Decomposition

Chemical decomposition is the degradation of an herbicide to one or more of its components via chemical reactions. 2,4-D is relatively persistent in the environment, and does not readily undergo chemical degradation, relative to other herbicides (Que Hee & Sutherland 1981). The hydrolysis of the ester formulations to its acid and alcohol compounds, however, can occur readily in alkaline waters (Que Hee & Sutherland 1981; Muir 1991). Additionally, the 2,4-D salt formulations dissociate to a salt and an acid in the environment (Smith 1988).

Behavior in the Environment

Summary: The World Health Organization (1984) concluded that 2,4-D does not accumulate or persist in the environment. The primary degradation mechanism is microbial metabolism, but mineralization and possibly photolysis may also play a role. The average half-life (the time it takes for the herbicide concentration to decline by 50%) is 10 days, but rates of degradation can vary from several hours to several months or longer. Degradation rates are determined by the microbial population, environmental pH, soil moisture, and temperature (Que Hee & Sutherland 1981; Sandmann et al. 1988; Wilson et al. 1997). The type of 2,4-D formulation applied does not significantly affect the rate of degradation (Wilson et al. 1997).

Soils

2,4-D may be applied in acid, salt, or ester formulations, but in most cases, each of these formulations are apparently converted rapidly to the acid form once it contacts soil (Foster & McKercher 1973; Smith 1988; Wilson et al. 1997). Consequently, the rate of dissipation from soils is often the same regardless of the formulation of 2,4-D that is applied (Wilson et al. 1997).

Half-lives are short, ranging from a few days to several months but detectable residues can persist for up to a year (McCall et al. 1981).

Degradation is almost entirely through microbial metabolism. Soil conditions that maximize microbial populations (i.e. warm and moist with a high organic content) maximize degradation rates (Foster & McKercher 1973; Ou 1984; Johnson & Lavy 1992; Han & New 1994; Johnson et al. 1995a; Veeh et al. 1996). Wilson et al. (1997) found that adequate soil moisture was the most influential parameter affecting degradation rates. Cold, dry soils can hold 2,4-D residues for significantly longer periods (Que Hee & Sutherland 1981). In at least one case, however, excessive soil moisture was shown to hinder 2,4-D degradation (Foster & McKercher 1973). In relatively dry soils with low bacterial counts, fungi play an increasingly important role in the degradation of 2,4-D (Ou 1984; Han & New 1996). Johnson et al. (1995b) found that dissipation rates did not differ significantly between rice field soils and bare ground, suggesting that plants do not play a significant role in eliminating 2,4-D from soils.

Lag times of up to eight weeks during which 2,4-D degradation is slow, have been reported following the first application of 2,4-D to soil (Audus 1960). These lags may indicate how long it takes for the abundance of 2,4-D degrading microbes to build up. Soils previously treated with 2,4-D do not exhibit a time lag and lose 2,4-D rapidly, presumably because of a pre-existing 2,4-D degrading microbial community (Sandmann et al. 1991).

Most formulations of 2,4-D do not bind tightly with soils and, therefore, have the potential to leach down into the soil column and to move off-site in surface or subsurface water flows. Leaching of 2,4-D to 30 cm has been reported (Johnson et al. 1995a). In many cases, extensive leaching does not occur, most likely because of the rapid degradation of the herbicide (Que Hee & Sutherland 1981). Where 2,4-D does leach, however, it will be more persistent because populations of microbes responsible for the degradation of 2,4-D tends to decrease with soil depth (Wilson et al. 1997).

2,4-D can also be lost from soils through volatilization. Volatilization rates are determined by the temperature and molecular form of the herbicide at the surface of the soil, which, in turn, is determined primarily by the soil's pH (McCall et al. 1981). In general, dry, alkaline soils with high organic content will be less likely to lose 2,4-D to volatilization (Que Hee & Sutherland 1981).

Water

2,4-D will change form and function with changes in water pH (Que Hee & Sutherland 1981). In alkaline (high pH; $\text{pH} > 7$) waters, 2,4-D takes an ionized (negatively charged) form that is water-soluble and remains in the water column. Theoretically, in water of a lower pH, 2,4-D will remain in a neutral molecular form, increasing its potential for adsorption to organic particles in water, and increasing its persistence (Wang et al. 1994a). 2,4-D is most likely to adsorb to suspended particles in muddy waters with a fine silt load (Que Hee & Sutherland 1981), but little adsorption has been observed in the field (Halter 1980).

Degradation mechanisms are difficult to isolate in the field and laboratory studies of microbial degradation and photolysis are conflicting. In sediments with sufficient microbial populations,

2,4-D can be degraded in a matter of hours (Aly & Faust 1964). When applied to eel grass along the coast of Prince Edward Island and New Brunswick, 2,4-D dissipated from the water within 20 days (Thomas & Duffy 1968). Wang et al. (1994b) however, found no significant degradation of 2,4-D in either sterilized or natural waters without sediments collected from four rivers in China. In this study, approximately 80% of the applied herbicide remained in the water after 56 days (Wang et al. 1994b). In other studies, 2,4-D was removed within hours by photodegradation (Aly & Faust 1964; Crosby & Tutass 1966). Aly and Faust (1964) concluded, however, that it was unlikely that a sufficient amount of ultraviolet radiation would reach the surface of natural waters to degrade 2,4-D.

Que Hee and Sutherland (1981) reported that concentrations of most 2,4-D residues found in lakes and streams are < 1 ppm, although concentrations of up to 61 ppm have been reported immediately following direct application to water bodies. These concentrations are well above the 0.1 ppm established as “permissible” levels for potable water by the U.S. E.P.A. (EPA 1998). Treated water should not be used for irrigation because concentrations as low as 0.22 ppm can damage soybeans and probably other crops (Que Hee & Sutherland 1981).

Vegetation

2,4-D residues taken up by plants remain intact in the foliage until it is lost as litter and degraded in soils (Newton et al. 1990). Fruits from treated trees have been found to retain 2,4-D residues for up to seven weeks (Love & Donnelly 1976, in Que Hee & Sutherland 1981).

Environmental Toxicity

Birds and Mammals

2,4-D is considered of moderate toxicity to animals, although LD50 levels vary significantly between formulations and animal species (Ibrahim et al. 1991). The majority of LD50 values range between 300-1,000 mg/kg. For example, the LD50 for 2,4-D acid in rats and bobwhite quail is 764 mg/kg and 500 mg/kg, respectively. Some animals such as dogs, however, are significantly more sensitive to 2,4-D organic acids than are rats and humans (Ibrahim et al. 1991). In 1991, Hayes et al. reported a significant increase in the occurrence of malignant lymphoma among dogs whose owners applied 2,4-D to their lawns.

2,4-D can bio-accumulate in animals. In Russia, residues of more than ten times the allowable level were found in eggs, milk, and meat products served by public caterers and one study reported residues in 46% of tested cattle (Que Hee & Sutherland 1981). Risk to browsing wildlife, however, is low, Newton et al (1990) analyzed 2,4-D residues in forest browse following aerial application to forests in Oregon and found them to be below the concentrations known to cause effects in mammals.

Aquatic Species

LC50 levels for bluegill sunfish and rainbow trout are 263 and 377 mg/L, respectively. Wang et al. (1994b) studied bioaccumulation of 2,4-D in carp and tilapia and found that accumulation of up to 18 times the ambient concentration occurred within two days of exposure. 2,4-D was found in oysters and clams in concentrations up to 3.8 ppm, and it persisted for up to two months (Thomas & Duffy 1968). The highest concentrations of 2,4-D were generally reached shortly after application, and dissipated within three weeks following exposure.

2,4-D can accumulate in fish exposed to concentrations as low as 0.05 ppm (Wang et al 1994b) and concentrations of 1.5 ppm can kill the eggs of fathead minnows in 48 hours (Thomas & Duffy 1968). After animals are removed from contaminated waters, they tend to excrete residues.

Other Non-Target Organisms

Moffett and Morton (1971) found that honey-bees directly sprayed with 2,4-D showed no injury and no residues were found in the bees or their honey (cited in Que Hee & Sutherland 1981). These results, however, are questionable as the LD50 for honey-bees is only 1 microgram/bee (WSSA 1994).

Application Considerations:

The most volatile of the 2,4-D esters, methyl and isopropyl, have been banned in the U.S. (Que Hee & Sutherland 1981), but some volatile ester formulations of 2,4-D remain available. Both localized damage from immediate drift, and widespread damage resulting from clouds of volatilized 2,4-D, have been reported (WHO 1984). To reduce the amount lost to vaporization, low-volatile (long-chain) esters are available. In addition, the alkali and amine salts are much less volatile and may be more appropriate for use where esters could volatilize and damage non-target plants (WHO 1984). Volatilization also can be reduced by using corn oil or cottonseed oil adjuvants (WHO 1984). Spray nozzles should deliver a coarse spray and 2,4-D should not be applied when wind speeds exceed five miles per hour (Hansen et al. 1984).

Safety Measures:

The acid and salt formulation can cause severe eye damage, while the ester formulation can cause moderate damage. Extra care should be taken to avoid splashing or other exposure of eyes to 2,4-D mixtures. The use of safety goggles is highly recommended.

When 2,4-D is used as an aquatic herbicide, do not treat the entire water body at one time. Treat only one-third to one-half of any water body at any one time, to prevent fish kills caused by dissolved oxygen depletion.

Human Toxicology:

2,4-D can be absorbed through the skin or through the lungs if inhaled. Applicators of 2,4-D, particularly those using back-pack sprayers, are at greatest risk of exposure (Ibrahim et al. 1991; Johnson & Wattenberg 1996). Libich et al. (1984) reported airborne residues of 1-35 micrograms/cubic meter of air when 2,4-D was applied using hand-held spray guns along power line right-of-ways. These workers later excreted <0.01-30 mg/kg of body weight in their urine. Absorption through the skin accounts for 90% of the 2,4-D absorbed by applicators (Ibrahim et al. 1991).

Once in the body, 2,4-D is distributed rapidly with the greatest concentrations appearing in the kidneys and liver (Johnson & Wattenberg 1996). The majority of the compound is excreted unmetabolized (Ibrahim et al. 1991). Due to its solubility in water, 2,4-D is not believed to accumulate in tissues, but is excreted in the urine in less than a week (Shearer 1980; Ibrahim et al. 1991; Johnson & Wattenberg 1996). Nevertheless, some agricultural workers and other

applicators have experienced long term complications including pain, paresthesias (tingling or numbness), and paralysis following exposure to 2,4-D (Shearer 1980).

Accidental inhalation resulted in one reported case of acute poisoning (Stevens & Sumner 1991). Symptoms included brief loss of consciousness, urinary incontinence, vomiting, muscular hypertonia (an abnormal increase in skeletal or smooth muscle tone), fever, headache, and constipation. Workers that entered an area shortly after treatment with 2,4-D experienced weakness, headache, dizziness, stomach pains, nausea, brief loss of consciousness, and moderate leukopenia (an abnormal reduction in the number of white blood cells, often reducing immune system function) (Stevens & Sumner 1991).

In 1991, a panel with expertise in epidemiology, toxicology, exposure assessment, and industrial hygiene convened to review the evidence available regarding the human carcinogenicity of 2,4-D (Ibrahim et al. 1991). The panel found that case-control studies showed evidence of a relationship between 2,4-D exposure and non-Hodgkins lymphoma in humans, with some studies showing an increased risk with increased exposure level (Ibrahim et al. 1991). Non-Hodgkins lymphoma is the human equivalent of the canine malignant lymphoma found to be associated with 2,4-D exposure in dogs (Hayes et al. 1991). When all evidence was evaluated, however, the panel could not find a cause-effect relationship between exposure to 2,4-D and human cancer (Ibrahim et al. 1991).

In another study of human exposure, female applicators were found to have a significant increase in cervical cancer associated with 2,4-D application. Due to the many confounding factors that make identification of cause and effect mechanisms difficult, other expert review panels including the U.S. EPA, Agriculture and Agri-food Canada, and the World Health Organization concluded that 2,4-D alone is not carcinogenic (Ibrahim et al. 1991; Mullison and Bond 1991).

References

- Aly, O. M., and S. D. Faust. 1964. Studies on the fate of 2,4-D and ester derivatives in natural surface waters. *Agric. Food Chem.* 12(6):541-546.
- Audus, L. J. 1960. Herbicide behaviour in the soil. Chapter 5 *in* Physiology and biochemistry of herbicides. L. J. Audus ed. Academic Press, New York, N.Y. 555 pgs.
- Crosby, D. G., and H. O. Tutass. 1966. Photodecomposition of 2,4-Dichlorophenoxyacetic acid. *J. Agr. Food Chem.* 14(6):596-599.
- Environmental Protection Agency. 1998. Tolerances and exemptions from tolerances for pesticide chemicals in food. Part 180 *in* Code of Federal Regulations Title 40, Volume 15 – Protection of the Environment.
- EXTOXNET. 1996. 2,4-D. Pesticide Information Profiles. Extention Toxicology Network. <http://ace.orst.edu/info/extoxnet/>.
- Foster, R. K. and R. B. McKercher. 1973. Laboratory incubation studies of chlorophenoxyacetic acids in chernozemic soils. *Soil Biol. Biochem.* 5:333-337.
- Halter, M. 1980. 2,4-D in the aquatic environment. Section II *in* Literature Reviews of Four Selected Herbicides: 2,4-D, dichlobenil, diquat & endothall. Shearer R., and M. Halter, eds.

- Han, S. O. and P. B. New. 1994. Effect of water availability on degradation of 2,4-dichlorophenoxyacetic acid (2,4-D) by soil microorganisms. *Soil Biol. Biochem.* 26(12):1689-1697.
- Hansen, G. W., F. E. Oliver, N. E. Otto. 1984. *Herbicide manual*. A Water Resources Technical Publication. U. S. D. I., Bureau of Reclamation, Denver, CO. 346 pgs.
- Hayes, H. M., R. E. Tarone, K. P. Cantor, C. R. Jessen, D. M. McCurnin, and R. C. Richardson. 1991. Case-control study of canine malignant lymphoma: positive association with dog owner's use of 2,4-dichlorophenoxyacetic acid herbicides. *J. Nation. Cancer Inst.* 83:1226-1231.
- Helling, C. S., P. C. Kearney, and M. Alexander. 1971. Behavior of pesticides in soil. *Adv. Agron.* 23:147-240.
- Hemmett, R. B. and S. D. Faust. 1969. Biodegradation kinetics of 2,4-dichlorophenoxyacetic acid by aquatic microorganisms. *Residue Rev.* 29:191-207.
- Ibrahim, M. A., G. G. Bond, T. A. Burke, P. Cole, F. N. Dost, P. E. Enterline, M. Gough, R. S. Greenberg, W. E. Halperin, E. McConnell, I. C. Munrun, J. A. Swendberg, S. H. Zahm, and J. D. Graham. 1991. Weight of the evidence on the human carcinogenicity of 2,4-D. *Environ. Health Perspect.* 96:213-222.
- Johnson, R. A., and E. V. Wattenberg. 1996. Risk assessment of phenoxy herbicides: an overview of the epidemiology and toxicology data. Chapter 3 in *Biological and Economic Assessment of Benefits from Use of Phenoxy Herbicides in the United States*. O. C. Burnside ed. U.S.D.A. National Impact Assessment Program. Special NAPIAP Report # 1-PA-96.
- Johnson, W. G., T. L. Lavy, and E. E. Gbur. 1995A. Sorption, mobility, and degradation of triclopyr and 2,4-D and four soils. *Weed Sci.* 43:678-684.
- Johnson, W. G., T. L. Lavy, and E. E. Gbur. 1995B. Persistence of triclopyr and 2,4-D in flooded and nonflooded soils. *J. Environ. Qual.* 24:493-497.
- Lavy, T. L., F. W. Roeth, and C. R. Fenster. 1973. Degradation of 2,4-D and atrazine at three soil depths in the field. *J. Environ. Quality* 2(1):132-137.
- McCall, P. J., S. A. Vrona, and S. S. Kelley. 1981. Fate of uniformly carbon-14 ring labeled 2,4,5-Trichlorophenoxyacetic acid and 2,4-Dichlorophenoxyacetic acid. *J. Agric. Food Chem.* 29:100-107.
- Mullison, W. R., and G. G. Bond. 1991. Epidemiology and toxicology of 2,4-D. *Weed Tech.* 5:898-906.
- Muir, D. C. G. 1991. Dissipation and transformations in water and sediment. Chapter 1 in *Environmental Chemistry of Herbicides, Volume II*. R. Grover and A. J. Cessna eds. CRC Press, Boca Raton, Florida. 302 pgs.
- Newton, M., F. Roberts, A. Allen, B. Kelpsas, D. White, and P. Boyd. 1990. Deposition and dissipation of three herbicides in foliage, litter, and soil of brushfields of Southwest Oregon. *J. Agric. Food Chem.* 38:574-583.
- Oh, K-H, and O. H. Tuovinen. 1991. Bacterial degradation of phenoxy herbicide mixtures 2,4-D and MCP. *Bull. Environ. Contam. Toxicol.* 47:222-229.
- Ou, L-T. 1984. 2,4-D degradation and 2,4-D degrading microorganisms in soils. *Soil Sci.* 137(2):100-107.
- Parker, L. W. and K. G. Doxtader. 1983. Kinetics of the microbial degradation of 2,4-D in soil: effects of temperature and moisture. *J. Environ. Qual.* 12(4):553-558.

- Que Hee, S. S., and R. G. Sutherland. 1981. The Phenoxyalkanoic Herbicides, Volume I: Chemistry, Analysis, and Environmental Pollution. CRC Press, Inc., Boca Raton, Florida. 319 pgs.
- Sandmann, E. R. I. C., M. A. Loos, and L. P. van Dyk. 1988. The microbial degradation of 2,4-Dichlorophenoxyacetic acid in soil. *Reviews Environ. Contam. Toxicol.* 101:1-53.
- Shaw, L. J., and R. G. Burns. 1998. Biodegradation of 2,4-D in a noncontaminated grassland soil profile. *J. Environ. Qual.* 27:1464-1471.
- Shearer, R., 1980. Public health effects of the aquatic use of herbicides – 2,4-D, dichlobenil, endothall and diquat. Section I *in* Literature Reviews of Four Selected Herbicides: 2,4-D, dichlobenil, diquat & endothall. Shearer R., and M. Halter, eds.
- Smith, A. E. 1988. Transformations in soil. Chapter 6 *in* Environmental Chemistry of Herbicides, Volume I. R. Groves ed. CRC Press, Boca Raton, Florida. 207 pgs.
- Smith, A. E., A. J. Aubin. 1994. Loss of enhanced biodegradation of 2,4-D and MCPA in a field soil following cessation of repeated herbicide applications. *Bull. Environ. Contam. Toxicol.* 53:7-11.
- Stevens, J. T., and D. D. Sumner. 1991. Herbicides. Chapter 20 *in* Handbook of pesticide toxicology, Vol. 3, Classes of Pesticides. W.J. Hayes, Jr. and E. R. Laws, Jr. eds. Academic Press, Inc. San Diego, California. 1576 pages.
- Thomas, M. L. H., and J. R. Duffy. 1968. Butoxyethanol ester of 2,4-D in the control of eelgrass (*Zostera marina* L.) and its effects on oysters (*Crassostrea virginica* Gmelin) and other benthos. *Proc. Northeast. Weed Control. Conf.* 22:186.
- Veeh, R. H., W. P. Inskeep, A. K. Camper. 1996. Soil depth and temperature effects on microbial degradation of 2,4-D. *J. Environ. Qual.* 25:5-12.
- Wang, Y-S., J-H. Yen, Y-N. Hsieh, and Y-L. Chen. 1994a. Dissipation of 2,4-D, glyphosate, and paraquat in river water. *Water Air Soil Pollut.* 72:1-7.
- Wang, Y-S., C-G. Jaw, and Y-L. Chen. 1994b. Accumulation of 2,4-D and glyphosate in fish and water hyacinth. *Water Air Soil Pollut.* 74:397-403.
- Wilson, R. D., J. Geronimo, and J. A. Armbruster. 1997. 2,4-D dissipation in field soils after applications of 2,4-D dimethylamine salt and 2,4-D 2-ethylhexyl ester.
- World Health Organization. 1984. 2,4-Dichlorophenoxyacetic acid (2,4-D), Environmental Health Criteria 29. United Nations Environment Programme, Geneva. 151 pgs.
- WSSA. 1994. Herbicide handbook. Weed Society of America. Champaign, Illinois. 352 pp.

Date Authored: April 2001

CLOPYRALID

M. Tu, C. Hurd, R. Robison & J.M. Randall

Herbicide Basics

Chemical formula: 3,6-dichloro-pyridinecarboxylic acid

Herbicide Family:

Pyridine (Picolinic Acid)

Target weeds: annual and perennial broadleaf weeds, esp. knapweeds, thistles, and other members of the sunflower, legume, and knotweed families

Forms: salt & ester

Formulations: SL, WG

Mode of Action: Auxin mimic

Water Solubility: 1,000 ppm

Adsorption potential: low

Primary degradation mech:
Slow microbial metabolism

Average Soil Half-life: 40 days

Mobility Potential: high

Dermal LD50 for rabbits:

>2,000 mg/kg

Oral LD50 for rats:

4,300 mg/kg

LC50 for bluegill sunfish:

125 mg/L

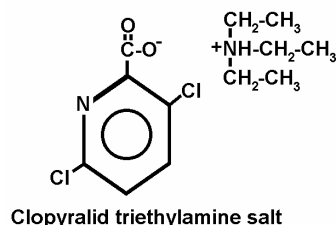
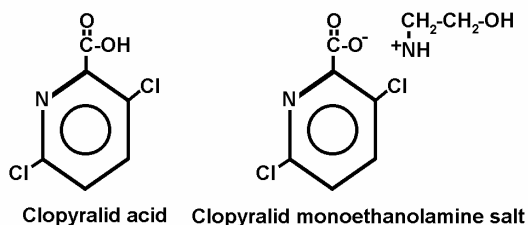
Trade Names: Transline[®], Stinger[®], Reclaim[®], Curtail[®], and Lontrel[®]

Manufacturers:

DowAgroSciences (formerly known as DowElanco)

Synopsis

Clopyralid is an auxin-mimic type herbicide. It is more selective (kills a more limited range of plants) than some other auxin-mimic herbicides like picloram, triclopyr, or 2,4-D. Like other auxin-mimics, it has little effect on grasses and other monocots, but also does little harm to members of the mustard family (Brassicaceae) and several other groups of broad-leaved plants. Clopyralid controls many annual and perennial broadleaf weeds, particularly of the Asteraceae (sunflower family), Fabaceae (legume family), Solanaceae (nightshade family), Polygonaceae (knotweed family), and Violaceae (violet family). It is chemically similar to picloram, but clopyralid has a shorter half-life, is more water-soluble, and has a lower adsorption capacity than picloram. Clopyralid's half-life in the environment averages one to two months and ranges up to one year. It is degraded almost entirely by microbial metabolism in soils and aquatic sediments. Clopyralid is not degraded by sunlight or hydrolysis. The inability of clopyralid to bind with soils and its persistence implies that clopyralid has the potential to be highly mobile and a contamination threat to water resources and non-target plant species, although no extensive offsite movement has been documented. Clopyralid can cause severe eye damage if splashed into the eyes during application, but otherwise is non-toxic to fish, birds, mammals, and other animals.



Herbicide Details:

Chemical Formula: 3,6-dichloro-2-pyridinecarboxylic acid

Trade Names: Clopyralid is sold as an acid, ester, or salt under the trade names Transline[®], Stinger[®], Reclaim[®], and Curtail[®]. Formulations labelled for non-cropland use include Transline[®] (clopyralid amine salt formulation) and Curtail[®] (clopyralid amine salt plus 2,4-D amine salt formulation).

Manufacturer: DowAgrosciences (formerly known as DowElanco)

Use Against Natural Area Weeds: Clopyralid is an auxin-mimic herbicide like picloram, triclopyr, or 2,4-D, but it is more selective than these compounds. Like other auxin-mimics, it has little effect on grasses and other monocots but also does little harm to members of the mustard family (Brassicaceae) and several other groups of broad-leaf plants. Clopyralid controls many annual and perennial broadleaf weeds, particularly of the Asteraceae (sunflower family), Fabaceae (legume family), Solanaceae (nightshade family), Polygonaceae (knotweed family), and Violaceae (violet family). The basis of this selectivity is not well understood.

On TNC preserves, clopyralid has been used against Canada thistle (*Cirsium arvense*), honey mesquite (*Prosopis glandulosa*), Russian knapweed (*Acroptilon repens*), yellow starthistle (*Centaurea solstitialis*), Chinese privet (*Ligustrum sinense*), bird's-foot trefoil (*Lotus corniculatus*), English ivy (*Hedera helix*), and Chinese wisteria (*Wisteria sinensis*). A mixture of clopyralid and 2,4-D (Curtail[®]) was used to control Canada thistle at the Silver Creek Preserve (Idaho) in 1991. In some cases, Canada thistle may remain dormant or inconspicuous for a season after being treated with these herbicides, but may recover two growing seasons after treatment. A follow-up treatment may be necessary.

DiTomaso et al. (1999) found clopyralid provided effective pre- and post-emergent control of yellow starthistle at very low application rates (e.g. 1 oz a.e./acre) in tests conducted at several sites in California. Season-long control was achieved with applications made in December or later. Earlier applications resulted in higher forage production than did later treatments (DiTomaso et al. 1999).

Mode of Action: Clopyralid is an “auxin mimic” or synthetic auxin. This type of herbicide kills the target weed by mimicking the plant growth hormone auxin (indole acetic acid), and when administered at effective doses, cause uncontrolled and disorganized plant growth that leads to plant death. The exact mode of action of clopyralid has not been fully described but it is believed to acidify the cell wall, which results in cell elongation. Low concentrations of clopyralid can stimulate RNA, DNA, and protein synthesis leading to uncontrolled cell division and disorganized growth, and ultimately, vascular tissue destruction. High concentrations of clopyralid can inhibit cell division and growth.

Degradation and Immobilization Mechanisms:

Summary: In soil and water, clopyralid is degraded primarily by microbial metabolism. It is resistant to degradation by sunlight, hydrolysis, or other chemical degradation. It is water-soluble, does not bind strongly with soils, and has the potential to be highly mobile in soils, especially sandy soils. Clopyralid is not highly volatile.

Volatilization

Clopyralid does not volatilize readily in the field (T. Lanini, pers. obs.). The potential to volatilize, however, increases with increasing temperature, increasing soil moisture, and decreasing clay and organic matter content (Helling et al. 1971).

Photodegradation

Clopyralid is not degraded significantly by sunlight (WSSA 1994; DowElanco 1997).

Microbial Degradation

Clopyralid is degraded primarily by microbes in soils and aquatic sediments (Pik et al. 1977). Rates of microbial metabolism increase with increasing soil moisture and temperature, and decrease with increasing amounts of organic matter. No metabolites accumulate during the degradation process, therefore, no additional contamination of the environment occurs (Pik et al. 1977).

Adsorption

Clopyralid is water-soluble and does not bind strongly to soils (Cox et al. 1996). During the first few months following application, clopyralid has a strong potential for leaching and possibly contaminating groundwater supplies. Adsorption has been shown to increase with time (Pik et al. 1977; DowElanco 1997), which can limit long term leaching. Pik et al. (1977) estimated that clopyralid was a class 5 (very mobile) herbicide using the Helling et al. (1971) scheme.

Chemical Decomposition

Clopyralid is not susceptible to hydrolysis or other types of chemical degradation (DowElanco 1977; Bergstrom et al. 1991).

Behavior in the Environment

Summary: Clopyralid is relatively persistent in soil, water, and vegetation. It is degraded almost entirely by soil microbes and is not susceptible to photo or chemical degradation. Once clopyralid is applied to soils, it rapidly disassociates (Shang and Arshad 1998), becoming extremely soluble in water, and does not bind strongly with soil particles. Lack of adsorption means that clopyralid has the potential to be mobile and could contaminate ground and surface water via leaching and surface and sub-surface water flows. However, no case of extensive off-site movement has been documented (J. DiTomaso, pers. comm.).

Soils

Clopyralid is moderately persistent in soils. Because it is degraded entirely by soil microbes, soil conditions that maximize microbial activity (warm and moist) will facilitate clopyralid degradation (Pik et al. 1977; DowElanco 1997). The average half-life of clopyralid in soils is one to two months but can range from one week to one year depending on the soil type, temperature, and rates of application (Pik et al. 1977; Smith and Aubin 1989; Bergstrom et al.

1991; Bovey and Richardson 1991; DowElanco 1997). Warm, moist soils treated at low rates will lose clopyralid in a comparatively short period, whereas when applied to cold, dry soils, or waterlogged soils, and at higher rates, clopyralid residues may persist for several years (Pik et al. 1977). Although some soils lose clopyralid quickly (3-4 weeks, Galoux et al. 1985), it generally takes a year or more for clopyralid to decrease to undetectable levels in treated soils (Pik et al. 1977; Smith and Aubin 1989).

When clopyralid enters the soil through direct spray, runoff from plant foliage, or translocation from the roots of treated plants, it rapidly disassociates to the anion form. The negatively charged anion form is highly water-soluble and has a very low capacity to adsorb to soil particles. Consequently, clopyralid has the potential to be highly mobile in the environment. Elliott et al. (1998) found clopyralid leached to depths as great as 180 cm within 20 days of application. Clopyralid's chemical characteristics suggest it has a high potential for movement, but most field studies found that it is not as mobile expected (Pik et al. 1977; Bergstrom et al. 1991; Bovey and Richardson 1991, DowElanco 1997). Where clopyralid leaches to lower soil depths, it persists longer than it does at the surface because the microbial populations generally decrease with soil depth (Pik et al. 1977).

Aquatic

Clopyralid is highly water-soluble and will not bind with suspended particles in the water column. Degradation is almost entirely through microbial metabolism in aquatic sediments, but because clopyralid does not bind with sediments readily, it can be persistent in an aquatic environment. The half-life of clopyralid in water ranges from 8 to 40 days (DowElanco 1977). Following aerial application to soils at the rate of 2.5 kg formulated product/ha (more than two times the label rate for non-cropland use in California), Leitch and Fagg (1985) recorded peak concentrations of 0.017 mg/L in a nearby stream that drained the area. They estimated that a total of 12 g of clopyralid (0.01% of that applied) leached into the stream during the first significant rainfall after the application that occurred three days later. Bergstrom et al. (1991) found a maximum of 0.02% of applied clopyralid was lost to runoff from clay soils in Sweden. Clopyralid is not registered for use in aquatic systems.

Vegetation

Clopyralid passes rapidly into leaves and roots of plants and is rain-fast within two hours (Devine et al. 1990; Kloppenburg and Hall 1990). Once inside the plant, clopyralid is converted to the anion form and transported throughout the plant. It is not readily degraded by the plant and can be persistent, even in non-susceptible species (DowElanco 1997).

Environmental Toxicity

Birds and Mammals

Clopyralid is practically non-toxic to birds and mammals. The LD50 for rats is 4,300 mg/kg. For mallards and bobwhite quail, the LD50s are 1,465 mg/kg and >4,640 mg/kg, respectively. The manufacturer reports that studies found that the majority of clopyralid ingested by mammals was excreted unmetabolized in their urine within 24 hours (DowElanco 1997). Some clopyralid, however, was retained in their livers and kidneys. Because clopyralid is not degraded rapidly in treated plants, wildlife could ingest clopyralid when feeding on treated browse. In a study of the effects of clopyralid on bobwhite quail egg hatchability and chick immunocompetence, Dabbert

et al. (1997) concluded that clopyralid did not cause significant effects to bobwhite quail embryos.

Aquatic Species

Clopyralid is of low toxicity to aquatic animals (DowElanco 1997). Its LC50s for bluegill sunfish and rainbow trout are 125 mg/L and 104 mg/L, respectively.

Other Non-Target Organisms

Clopyralid is of very low toxicity to most animals including soil invertebrates and microbes (DowElanco 1997).

Application Considerations:

Foliar application of clopyralid may provide more complete control than soil application (DowElanco 1997). In the case of Canada thistle, foliar application results in the death of both the roots and top-growth, while soil application will damage only the roots and may not kill the plants. Direct soil application, however, may prevent germinating seedlings from emerging (DowElanco 1997).

Safety Measures:

Clopyralid can cause severe eye damage. Care should be taken to prevent clopyralid from splashing or otherwise getting into anyone's eyes.

Human Toxicology:

Clopyralid is of relatively low toxicity to mammals but can cause severe eye damage including permanent loss of vision.

References

- Bergstrom, L. 1991. Leaching potential and decomposition of clopyralid in Swedish soils under field conditions. *Environ. Toxicol. Chem.*
- Bovey, R. W., and C. W. Richardson. 1991. Dissipation of clopyralid and picloram in soil and seep flow in the Blacklands of Texas. *J. Environ. Qual.* 20:528-531.
- Dabbert, C. B., R. B. Mitchell, and D. T. Oberheu. 1997. Northern bobwhite egg hatchability and chick immunocompetence following a field application of clopyralid. *Bull. Environ. Contam. Toxicol.* 58(5):801-806.
- DiTomaso, J. M., G. B. Kyser, S. B. Orloff, S. F. Enloe, and G. A. Nader. 1999. New growth regulator herbicide provides excellent control of yellow starthistle. *Calif. Agricult.* 53(2):12-16.
- DowElanco. 1997. Clopyralid, a North American technical profile. Indianapolis, IN.
- Elliott, J.A., A.J. Cessna, K.B. Best, W. Nicholaichuk, and L.C. Tollefson. 1998. Leaching and preferential flow of clopyralid under irrigation: Field observations and simulation modeling. *Journal of Environmental Quality* 27: 124-131.
- Galoux, M. P., A. C. Bernes, J-C. Van Damme. 1985. Gas chromatographic determination of 3,6-dichloropicolinic acid residues in soils and its application to the residue dissipation in a soil. *J. Agric. Food Chem.* 33:965-968.

- Helling, C. S., P. C. Kearney, and M. Alexander. 1971. Behavior of pesticides in soil. *Adv. Agron.* 23:147-240.
- Kloppenburg, D. J., and J. C. Hall. 1990. Penetration of clopyralid and related weak acid herbicides into and through isolated cuticular membranes of *Euonymus fortunei*. *Weed Res.* 30:431-438.
- Leitch, C., and P. Fagg. 1985. Clopyralid herbicide residues in streamwater after aerial spraying of a *Pinus radiata* plantation. *N.Z. J. For. Sci.* 15(2):195-206.
- Pik, A. J., E. Peake, M. T. Strosher, and G. W. Hodgson. 1977. Fate of 3,6-dichloropicolinic acid soils. *J. Agric. Food Chem.* 25(5):1054-1061.
- Schutz, S., B. Weibbecker, and H. E. Hummel. 1996. Gas chromatography-mass spectrometry analysis of the herbicide clopyralid in differentially cultivated soils.
- Shang, C., and M. A. Arshad. 1998. Sorption of clopyralid, dicamba and MCPA by two soils with conventional and no-till management. *Can. J. Soil Sci.* 78:181-186.
- Smith, A. E., and A. J. Aubin. 1989. Persistence studies with the herbicide clopyralid in prairie soils at different temperatures. *Bull. Environ. Contam. Toxicol.* 42:670-675.
- WSSA. 1994. *Herbicide handbook*. Weed Society of America. Champaign, Illinois. 352 pp.

Date Authored: April 2001

FLUAZIFOP-P-BUTYL

Herbicide Basics

Chemical formula: R-2-[4-[[5-(trifluoromethyl)-2-pyridinyl]oxy]phenoxy]propanate

Herbicide Family:

Aryloxyphenoxy-propionate

Target Species: annual and perennial grasses

Forms: butyl ester

Formulations: EC

Mode of Action: Lipid synthesis inhibitor

Water Solubility: 1.1 ppm

Adsorption potential: high

Primary degradation mech: microbial metabolism and hydrolysis

Average Soil Half-life:
15 days

Mobility Potential: low

Dermal LD50 for rabbits:
>2,420 mg/kg

Oral LD50 for rats:
4,096 mg/kg

LC50 for bluegill sunfish:
0.53 mg/L

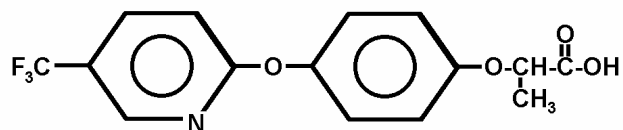
Trade Names: Fusilade[®], Horizon 2000[®], Ornamec[®], Fusion[®], Tornado[®]

Manufacturers:

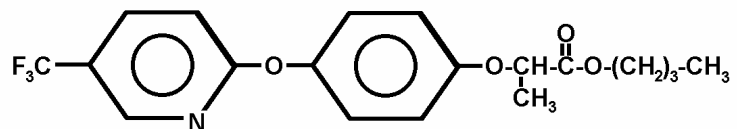
AgrEvo, PBI/Gordon, and Zeneca

Synopsis

Fluazifop-p-butyl kills annual and perennial grasses, but does little or no harm to broad-leaved plants (dicots). It kills by inhibiting lipid synthesis (lipids are necessary components of cell membranes), particularly at the sites of active growth. In the environment, fluazifop-p-butyl is degraded primarily through microbial metabolism and hydrolysis. It is not degraded readily by sunlight. The half-life of fluazifop-p-butyl in soils is one to two weeks. Because it binds strongly with soils, fluazifop-p-butyl is not highly mobile and is not likely to contaminate ground water or surface water through surface or sub-surface runoff. In water, fluazifop-p-butyl is rapidly hydrolyzed to fluazifop acid, which is stable in water. Fluazifop-p-butyl is of relatively low toxicity to birds and mammals, but can be highly toxic to fish and aquatic invertebrates.



Fluazifop-P acid



Fluazifop-P butyl ester

Herbicide Details

Chemical Formula: R-2-[4-[[5-(trifluoromethyl)-2-pyridinyl]oxy]phenoxy] propanate

NOTE: The fluazifop-butyl molecule can take two forms, the R- and S-isomers, but only the R-isomer is herbicidally active. A few years ago, formulations of fluazifop-butyl were changed so that they contain only the herbicidally active form (R-isomer), fluazifop-p-butyl. Some of the studies reported in this chapter were conducted using the mixed formulation of fluazifop-butyl, which contained both R- and S- isomers. There is some evidence that the two isomers behave differently in the environment. New formulations that contain only the R-isomer form may not behave in the environment as some older studies have predicted.

Trade Names: Fusilade 2000[®], Fusilade DX[®], Fusilade Turf and Ornamental[®], Fusilade Fiv[®], Fusilade Supe[®]r, Fusion[®], Horizon[®], Ornamec[®], and Tornado[®]

Manufacturer: Zeneca Agricultural Products, AgrEvo, PBI/Gordon

Use Against Natural Area Weeds: Both annual and perennial grasses can be controlled by fluazifop-p-butyl, including bromes (*Bromus* spp.), quackgrass (*Elytrigia repens*), johnsongrass (*Sorghum halepense*), and panic or witch-grasses (*Panicum* spp.).

Fluazifop-p-butyl has not been used extensively on TNC preserves, but small-scale trials have generated some promising results. In Ohio, smooth brome (*Bromus inermis*) was significantly weakened but not killed following applications of fluazifop-p-butyl. In Oregon on the Ewauna Flats Preserve, trials were conducted against quackgrass (*Elytrigia repens* var. *repens*) using glyphosate, sethoxydim, and fluazifop. Fluazifop had the greatest impact on the weed. After one application, stem density had not changed but quackgrass cover was reduced and the native plant targeted for conservation (*Astragalus applegatei*) increased in density. Darren Borgias and Molly Sullivan (TNC-SW Oregon) report that fluazifop must be applied repeatedly (late May, early July) to actively growing plants (> 6 in) for good control of quackgrass. A combination of burning followed by fluazifop also significantly lowered aboveground cover of quackgrass, but only by about 8%. They used a foliar spray at 0.125 kg/ai/ha of fluazifop with 0.25% Triton, a non-ionic surfactant. They hope that an intensive regime using controlled burns and multiple applications of fluazifop will provide complete control of quackgrass. Recent studies in California have also demonstrated effective control of jubatagrass (*Cortaderia jubata*) with fall applications of fluazifop-p-butyl (DiTomaso, pers. comm.; Drewitz 2000).

Mode of Action: Fluazifop-p-butyl is a post-emergence phenoxy herbicide. It is absorbed rapidly through leaf surfaces and quickly hydrolyzes to fluazifop acid. The acid is transported primarily in the phloem and accumulates in the meristems where it disrupts the synthesis of lipids in susceptible species (Urano 1982; Erlingson 1988). Fluazifop-p-butyl inhibits acetyl CoA carboxylase, an enzyme that catalyzes an early step in fatty acid synthesis. Lipids are important components of cellular membranes, and when they cannot be produced in sufficient quantities, cell membrane integrity fails, especially in regions of active growth such as meristems.

The cells then burst, or leak and die. Fluazifop-p-butyl affects susceptible grasses, but does not affect most other monocots or dicots.

Dissipation Mechanisms:

Summary: Fluazifop-p-butyl is degraded primarily by hydrolysis, and secondarily by microbial metabolism. It is not degraded by photolysis or other chemical means. It can bind strongly with soil particles and is not water-soluble. Fluazifop-p-butyl does not volatilize readily.

Volatilization

Fluazifop-p-butyl is non-volatile in the field (T. Lanini, pers. obs.). The potential to volatilize, however, may increase with increasing temperature, increasing soil moisture, and decreasing clay and organic matter content (Helling et al. 1971).

Photodegradation

The WSSA Herbicide Handbook (1994) reports negligible loss from photodegradation in laboratory studies. It is relatively stable to breakdown by UV or sunlight (EXTOXNET 1996). No published studies regarding photodegradation of fluazifop-p-butyl were found.

Microbial Degradation

Degradation of fluazifop-p-butyl in the environment can occur by either hydrolysis or by microbial degradation. Soils with conditions that favor microbial metabolism (i.e. warm and moist) will have the highest rates of degradation (Negre et al. 1993). Fluazifop's average field half-life is 15 days (WSSA 1994). Metabolism by soil microbes first converts the herbicide to its acid form (fluazifop acid), which is further degraded by microbes, and can have a half-life of less than 1 week (EXTOXNET 1996).

Adsorption

Fluazifop-p-butyl binds strongly with soils. Gessa et al. (1987) found that fluazifop-p-butyl can form irreversible bonds with certain clay soils by several different mechanisms. Despite its strong adsorption to soils, Kulshrestha et al. (1995) found that fluazifop-p-butyl leached to at least 15 cm deep in soybean fields in India. Fluazifop-p-butyl is reported to be of low mobility in soils and does not present an appreciable risk of groundwater contamination (EXTOXNET 1996; WSSA 1994).

Chemical Decomposition

Fluazifop-p-butyl readily degrades through hydrolysis to fluazifop acid in soils and water. Increased temperatures can increase the rate of hydrolysis (Balnova & Lalova 1992). No other mechanism of chemical degradation has been reported.

Behavior in the Environment

Summary: Fluazifop-p-butyl is rapidly hydrolyzed to fluazifop acid in vegetation, soils, and water. In plants, fluazifop acid is herbicidally active. In soils and water, both the ester and acid forms are metabolized by soil or sediment microbes, and broken-down to herbicidally inactive compounds. The average soil half-life of the ester form is one to two weeks. Fluazifop-p-butyl binds readily with soil particles, limiting leaching and soil runoff.

Soils

Fluazifop-p-butyl is rapidly hydrolyzed by microbes to fluazifop acid in soils (Smith 1987). The average half-life of fluazifop-p-butyl is one to two weeks (WSSA 1994). Conditions that promote microbial activity in soils, such as high moisture levels, favor degradation (Negre et al. 1988; Somich et al. 1988; Negre et al. 1993). Smith (1987) reported that in moist soils, only 8% of the fluazifop-butyl remained in the soil after 48 hours, whereas in dry soils, over 90% of the ester remained after 48 hours. One study showed that the S-isomer, which is no longer used in brand-name Fusilade[®] formulations, is more readily metabolized by microbes than the R-isomer (Negre et al. 1993). Complete degradation of formulations sold today (composed primarily of the R-isomer), therefore, may take longer than the S- and R-isomer fluazifop-butyl mixture.

Water

Fluazifop-p-butyl is not water-soluble. Because it binds strongly with soils, it is not highly mobile in soils and does not pose a significant risk of groundwater contamination (WSSA 1994). In water, fluazifop-p-butyl rapidly hydrolyzes to fluazifop acid, with the rate of hydrolysis increasing with increasing pH (Negre et al. 1988). Fluazifop acid is stable in water at all pHs tested.

Vegetation

Fluazifop-p-butyl is completely metabolized within the plant to fluazifop acid two to four weeks following application (Balinova & Lalova 1992; Kulshresha et al. 1995). The acid takes longer to degrade, with residues remaining in the plant up to 45 days after treatment (Balinova & Lalova 1992).

Environmental Toxicity

Birds and Mammals

Studies have shown fluazifop-p-butyl to be “slightly to practically nontoxic” to mammals and birds that ingest it and only “slightly” toxic to animal skin and eyes (EXTOXNET 1996). Oral LD50 levels of fluazifop-p-butyl were > 4,000 mg/kg for male rats, >3,500 mg/kg for mallard ducks, and >4,659 mg/kg for bobwhite quail.

Aquatic Species

Fluazifop-p-butyl can pass readily into fish tissue, and is highly toxic to fish and other aquatic species, including invertebrates (*Daphnia* 48 hr LC50 > 10 mg/L). Studies have shown “very high to high” toxicity in bluegill sunfish (96 hr LC50 = 0.53 mg/L) and rainbow trout (96 hr LC50 = 1.37 mg/L) (EXTOXNET 1996). Fluazifop-p-butyl is not registered for use in aquatic systems.

Other Non-Target Organisms

Fluazifop-p-butyl has been shown to inhibit fungal growth (Abdel-Mallek et al. 1996; Gorlach-Lira et al. 1997). Abdel-Mallek et al. (1996) found that fungal populations were temporarily (one to two weeks) decreased at rates above 3.0 ug/g and for longer periods of time (more than eight weeks) at rates above 6.0 ug/g. Fluazifop does not have a significant effect on fungal populations when applied at recommended field rates.

Application Considerations:

Fluazifop-p-butyl is ineffective under drought conditions. Growth regulating herbicides are only effective when plants are growing. Under drought conditions, no new plant growth occurs, and the herbicide is rendered ineffective. Some herbicides remain in the plant until new growth resumes, but fluazifop-p-butyl is metabolized rapidly by the plant and, consequently, is no longer present when growth resumes weeks or months later.

Synergistic Effects:

Synergism may occur when two or more herbicides are mixed and applied together and the impact of the mixture is greater than when the herbicides are applied separately. The effectiveness of the herbicide mixture, therefore, may be multiplied by using a lesser amount of total herbicide than if applied separately. Synergistic effects have the benefits of saving money (amount spent on herbicides) and reduce the total amount of herbicide applied, thereby minimizing potential for environmental contamination. A drawback of using more than one herbicide, however, is that adequate research (by the manufacturer and others) has not been conducted on the overall impacts and toxicity of mixtures of this sort.

Synergistic effects of fluazifop-p-butyl mixed with several herbicides have been noted. Harker and O'Sullivan (1991) found that a mixture of fluazifop-p-butyl and sethoxydim provided more control over grass species than the two herbicides applied separately. Additionally, because each herbicide provided better control over different set of grass species, the effects of mixing the herbicides were complimentary as well as synergistic. For example, fluazifop provided better control of wheat and barley, while sethoxydim provided better control of green foxtail (Harker & O'Sullivan 1991).

Antagonistic Effects

Antagonistic effects have been reported between fluazifop-p-butyl and auxin mimic herbicides such as 2,4-D. When applied together, the auxin mimic effectively controls broadleaf plants but the normal control of grasses provided by fluazifop-p-butyl is lost.

Safety Measures:

Fluazifop-p-butyl is irritating to skin, can cause eye damage, and is harmful if inhaled. Care should be taken to prevent accidental splashing or other exposure to the herbicide.

Human Toxicology:

Fluazifop-p-butyl is of relatively low toxicity to mammals, but can be an irritant (eye, skin, respiratory passages, and skin sensitizer), and is toxic if inhaled.

References

- Abdel-Mallek, A. Y., M. I. A. Abdel-Kader, and S. A. Omar. 1996. Effect of the herbicide fluazifop-butyl on fungal populations and activity in soil. *Water Air Soil Pollut.* 86:151-157.
- Balinova, A. M., and M. P. Lalova. 1992. Translocation, metabolism and residues of fluazifop-butyl in soybean plants. *Weed Res.* 32:143-147.
- Drewitz, J.J. 2000. Reproductive biology and control of jubatagrass (*Cortaderia jubata*). Master's Thesis, University of California, Davis.
- Erlingson, M. 1988. Fusilade - a strategy for long-term control of couch (*Elymus repens*). *Weeds and Weed Control.* 1:158-165.
- EXTOXNET. 1996. Fluazifop-p-butyl. Pesticide Information Profiles. Extension Toxicology Network. <http://ace.orst.edu/info/extoxnet/>.
- Gessa, C., A. Pusino, V. Solinas, and S. Petretoo. 1987. Interaction of fluazifop-butyl with homoionic clays. *Soil Sci.* 144:420-424.
- Gorlach-Lira, K., O. Stefaniak, W. Slizak, and I. Owedyk. 1997. The response of forest soil microflora to the herbicide formulations Fusilade and Roundup. *Microbiol. Res.* 152:319-329.
- Helling, C. S., P. C. Kearney, and M. Alexander. 1971. Behavior of pesticides in soil. *Adv. Agron.* 23:147-240.
- Negre, M., M. Gennari, V. Andreoni, R. Ambrosoli, and L. Celi. 1993. Microbial metabolism of fluazifop-butyl. *J. Environ. Sci. Health B28(5):545-576.*
- Negre, M., M. Gennari, A. Cignetti, and E. Zanini. 1988. Degradation of fluazifop-butyl in soil and aqueous systems. *J. Agric. Food Chem.* 36:1319-1322.
- Smith, A. E. 1987. Persistence studies with the herbicide fluazifop-butyl in Saskatchewan soils under laboratory and field conditions. *Bull. Environ. Contam. Toxicol.* 39:150-155.
- Urano, K. 1982. Onecide, a new herbicide fluazifop-butyl. *Jap. Pestic. Inf.* 41:28-31.
- WSSA. 1994. Herbicide handbook. Weed Society of America. Champaign, Illinois. 352 pp.

Date Authored: April 2001

FOSAMINE AMMONIUM

Herbicide Basics

Chemical formula: ethyl hydrogen (aminocarbonyl) phosphonate

Herbicide Family:
None generally recognized

Target weeds: woody and herbaceous plants

Forms: ammonium salt

Formulations: SL

Mode of Action: enzyme inhibitor

Water Solubility:
1,790,000 ppm

Adsorption potential:
medium-high

Primary degradation mech.:
microbial metabolism

Average Soil Half-life:
8 days

Mobility Potential: low

Dermal LD50 for rabbits:
>1,683 mg/kg

Oral LD50 for rats:
24,400 mg/kg

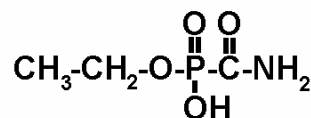
LC50 for bluegill sunfish:
670 mg/L

Trade Names: Krenite®

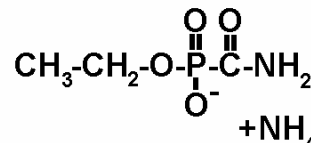
Manufacturer: Du Pont

Synopsis

Fosamine ammonium inhibits growth in woody plants and some herbs. It apparently prevents dormant tissues from becoming active and growing again, but its exact mode of action is not understood. When applied in late summer or early fall, effects are generally not visible until the following spring when treated vegetation fails to bud-out. Pine species can be treated during spring and summer, and their growth will be inhibited shortly thereafter. Few studies on the environmental fate and toxicity of fosamine ammonium have been conducted by independent researchers. This lack of research may be due, in part, to the relatively low toxicity and rapid microbial degradation of this herbicide. Fosamine ammonium is highly water soluble, but appears to bind readily with at least some soils. It is readily degraded by soil microbes and has a half-life in soils ranging from one to two weeks, which limits its movement. It is not readily degraded by abiotic chemical reactions or photolysis, but low pH and high temperatures have been shown to facilitate its breakdown. It is stable in water, but is generally degraded rapidly by microbes in aquatic sediments. Fosamine is only very slightly toxic to birds, mammals, fish, and aquatic invertebrates.



Fosamine acid



Fosamine ammonium salt

Herbicide Details

Chemical Formula: ethyl hydrogen (aminocarbonyl) phosphonate

Trade Names: Krenite S[®] and Krenite UT[®]

Manufacturer: Du Pont Agricultural Products

Use Against Natural Area Weeds: Fosamine is commonly used for brush control in rights-of-way, parklands, conifer plantations, and reforested areas. It is usually applied one to two months before autumn leaf-drop. On TNC preserves, fosamine has been used with varying levels of success to control leafy spurge (*Euphorbia esula*) and crown vetch (*Coronilla varia*). It was moderately effective against leafy spurge on the Paul Bunyan Savanna in Minnesota, at Pine Butte Preserve in Montana, and several preserves in South Dakota. On the Bluestem Prairie and Pembina Preserves in Minnesota, however, Brian Winter reports that fosamine provided only moderate to poor control of leafy spurge, and also caused severe damage to native grasses and forbs.

Mode of Action: Fosamine ammonium functions as a plant growth regulator. It is sometimes referred to as a “dormancy enforcer,” but its specific mechanism of action has not been identified. There is some evidence that it inhibits mitosis in susceptible plants. Deciduous plants treated with fosamine ammonium fail to re-leaf and die, without going through the “brown out” caused by many other herbicides. When applied to deciduous plants two months prior to leaf drop, the compound is absorbed with little or no apparent effect. In the following spring, buds either fail to open at all, or produce only spindly, miniature leaves. Evergreen plants such as pines show response soon after application.

Dissipation Mechanisms:

Summary: Fosamine ammonium is degraded primarily by microbial metabolism. It is not readily degraded by sunlight or un-catalyzed chemical processes. Fosamine ammonium’s average half-life in soils is one to two weeks. It is adsorbed by at least some soils, but has the potential to be mobile in the environment. It does not readily volatilize.

Volatilization

Fosamine is not highly volatile (T. Lanini, pers. obs.). The potential to volatilize, however, increases with increasing temperature, increasing soil moisture, and decreasing clay and organic matter content (Helling et al. 1971).

Photodegradation

Fosamine ammonium is not readily degraded by sunlight. However, increases in UV exposure, temperature, and a decline in pH can increase its photolytic degradation rates (Han 1979). Photosensitizers (photodegradation catalysts) did not enhance degradation rates.

Microbial Degradation

Fosamine ammonium is degraded readily by microbes in soils and aquatic sediments (Han & Krause 1979). Han (1979) found that 45-75% of fosamine ammonium in soils was microbially degraded within 90 days. Soil conditions that favor microbial metabolism, such as increased soil temperature, moisture, and organic content, will enhance degradation of fosamine ammonium.

Adsorption

Fosamine ammonium is highly water-soluble but appears to bind readily with at least some soils. Han (1979) found adsorption rates of fosamine ammonium varied with soil type. Adsorption coefficient values, K, ranged from 0.7 in sandy loam to >20 in silt loam. Increasing organic matter or clay content generally increases adsorption values. Adsorption to soils and rapid microbial degradation are considered responsible for the poor uptake of fosamine ammonium by plant roots (Weigel et al. 1978).

Chemical Decomposition

Fosamine ammonium is relatively stable and not readily degraded in neutral to basic waters (pH 7 to 9). In acidic waters (pH 5 and less), small amounts of fosamine ammonium can be hydrolyzed to carbamoylphosphonic acid, but only at low concentrations (<5 ppm - Han 1979).

Behavior in the Environment

Summary: Fosamine ammonium is rapidly degraded by soil microbes. Treated soils are generally free of detectable residues within one year. Fosamine ammonium binds with some soils, preventing it from moving extensively in the environment. In water, fosamine ammonium is stable and can be persistent, and in plants, it is rapidly hydrolyzed to the acid form and does not readily degrade further.

Soils

Because fosamine ammonium is rapidly metabolized by soil microbes, it does not persist in soils. Reported half-lives in the field and laboratory range from one to six weeks, and some research suggests a half-life of only one to two weeks (Han 1979). Fosamine ammonium's metabolite carbamoylphosphonic acid (CPA) also has a short half-life. Han (1979) found that in field tests, CPA was completely eliminated from soils within three to six months. Radio-labeled carbon studies indicated that the herbicide was dissipated by natural degradation, not runoff in water (Han 1979).

Fosamine ammonium also does not appear to leach extensively. Han (1979) found that after one year and 165 cm of rainfall, 93% of the residual radio-labelled carbon from fosamine ammonium was recovered within the top 10 cm of the soil. Even in fine sand soils, 62% of the radio-labelled carbon was found in the top 10 cm after six months and 40 cm precipitation (Han 1979). Fosamine ammonium may also form insoluble salts or complexes with soil minerals, which prevent it from leaching (Han 1979).

Water

Adsorption to soil particles likely prevents significant amounts of fosamine ammonium from leaching or otherwise moving into nearby waterways, even though it is highly soluble in water. Once it enters an aquatic system, however, fosamine ammonium is stable and can be persistent.

It is readily degraded by microbial activity in aquatic sediments, however, which eventually eliminates it from natural water bodies. Han (1979) found the factors that can affect the degradation of fosamine in water, in order of importance, are low pH (< 5), high temperatures (25° C vs. 15° C), and UV exposure.

Vegetation

Fosamine ammonium is slowly absorbed through leaf tissue (WSSA 1994). On average, only 50% of the applied herbicide is absorbed and translocated throughout the plant. It is believed that tolerant species do not translocate the herbicide as well as susceptible species do (WSSA 1994). Once in the plant, the salt is rapidly hydrolyzed to the parent acid, which is further degraded to its metabolite, carbamoylphosphonic acid, within several weeks.

Environmental Toxicity

Birds and Mammals

Fosamine ammonium is only “very slightly toxic” to birds and mammals. The oral LD50 is 24,400 mg/kg for rats and 10,000 mg/kg for bobwhite quail and mallard ducks (Hernandez et al. 1974). No chronic toxic effects in adults or birth defects in offspring were reported (Chrzanowski et al. 1979). The dermal toxicity of fosamine, however, falls under the EPA Category II, indicating the second most severe level of acute toxicity for studies using laboratory animals. Fosamine is also an eye irritant.

Animals given the highest one subchronic oral dose in one study lost weight and exhibited some effects to the kidney and bladder (EPA 1995). Chrzanowski et al. (1979) reported that fosamine ammonium was eliminated from the rats’ bodies within 72-hours. An average of 79% of the herbicide administered was excreted unchanged, while 13% was excreted as a hydrolyzed metabolite.

Aquatic Species

The toxicity of fosamine ammonium to fish and aquatic invertebrates is low (EPA 1995). The LC50 (96-hour) is 1,000 ppm for rainbow trout and fathead minnow, and 670 ppm for bluegill sunfish (Hernandez et al. 1974). There is no evidence that fosamine bioaccumulates in fish (EPA 1995).

Other Non-Target Organisms

The presence of 10 ppm of fosamine ammonium in three soil types did not alter fungal or bacterial populations (Han & Krause 1979). In agar plates, however, fosamine ammonium concentrations above 100 ppm had detrimental impacts on some fungi.

Application Considerations:

Because fosamine ammonium is a salt formulation, it does not easily penetrate the leaves of mature plants, especially those with glossy or waxy leaves (Hernandez et al. 1974). It is best applied to the leaves of young plants, to cut stumps and new sprouts, and to notches made in the trunk (hack and squirt - Barring 1982; Hernandez et al. 1974). When used on more mature vegetation, it should be applied generously and evenly and allowed to set for at least 24 hours. Rainfall shortly after application will wash it off and minimize its impact (Hernandez et al. 1974).

According to Barring (1982), fosamine ammonium's impact can be unreliable, especially on pines. The manufacturers claim that plant response depends on the timing of application and the species being treated (Weigel et al. 1978). Herbaceous plants are said to "not respond significantly", perennials "may be repressed", and broadleaf evergreens are "usually not affected" (Hernandez, et al. 1974). Only the areas of susceptible plants that are directly treated exhibit a response, and for this reason, fosamine ammonium can be used as a trimming agent.

Semington (1977), a DuPont representative, made a number of application suggestions for best results:

1. Timing of application: For best results, apply fosamine ammonium in the late summer or early fall (within 2 months of leaf-drop for deciduous species).
2. Adequate use rates: The minimum recommended rates for dense stands of brush >6 feet high, is 8 lb (2 gal) / acre. A rate of 3 gal / acre may be best for taller brush and tough to control species.
3. Addition of a surfactant: Because fosamine ammonium does not penetrate leaves readily, addition of a surfactant can improve results. DuPont recommends Du Pont Surfactant WK, Tween 20 or Renex 30 by ICI, and Triton X-100 by Rohm and Haas at 0.25% by volume.
4. Spray concentrations: For ground applications, Du Pont recommends 1-1.5% concentration in water. More resistant species may not be killed but may be significantly repressed.
5. Complete coverage without drenching: To facilitate penetration to short brush, use smaller nozzles and higher pressures to disperse the spray.
6. Effects of hard water: "Hard" water (water high in calcium and magnesium) may bind fosamine ammonium molecules and lessen the effectiveness of the herbicide. To "soften" the water, sodium gluconate technical can be added and dissolved in water at 4 oz per 100 gals prior to adding the herbicide. Sodium gluconate can be purchased under the trade names Fisons and Premier.
7. Inclement weather: Because of the solubility of fosamine ammonium, best results are achieved when no rain occurs within one day of application. Foliage should be dry at time of application.

Safety Measures:

Fosamine can cause irritation to the skin and eyes, and inhalation can result in irritation of the upper respiratory passages. Ingestion of high doses may cause nausea, headache, or weakness. Care should be taken to avoid splashing or other exposure to skin and eyes.

Human Toxicology:

Fosamine is only slightly toxic to mammals, but excessive contact with the skin may initially cause skin irritation with discomfort or rash. The EPA classifies fosamine ammonium in Toxicity Category II for acute dermal exposure (second most severe), but regards it as mildly toxic for acute oral ingestion and acute inhalation. Inhalation and ingestion of high doses may result in nonspecific discomfort, nausea, headache, or weakness. Fosamine ammonium can irritate the eyes, causing discomfort, tearing, or blurring of vision.

References

- Barring, U. 1982. Results of country-wide experiments in forestry. *Weeds and Weed Control*, 23:270-276.
- Chrzanowski, R. L., J. C.-Y. Han, and C. L. McIntosh. 1979. Metabolism of [¹⁴C] fosamine ammonium in the rat. *J. Agric. Food Chem.* 27(3):550-554.
- DuPont Ag. Products. 1991. "Krenite" S Brush Control Agent. Material Safety Data Sheet.
- E.P.A. 1995. Fosamine ammonium. R.E.D. Facts. Prevention, Pesticides and Toxic Substances. EPA-738-F-95-005.
- Han, J. C. and R. L. Krause. 1979. microbial activity in soils treated with fosamine ammonium. *Soil Science* 128:23-27.
- Han, J. C. 1979. Stability of [C14] fosamine ammonium in water and soils. *J. Agric. Food Chem.* 27:564-571.
- Helling, C. S., P. C. Kearney, and M. Alexander. 1971. Behavior of pesticides in soil. *Adv. Agron.* 23:147-240.
- Hernandez, T. J., W. H. Hudson, and F. E. Gonzalez. 1978. A progress report on "Krenite" brush control agent. *Proc. South. Weed Sci. Soc.* 28:261-263.
- Weigel, R. C., Jr., E. M. Beyer, Jr., and J. D. Riggelman. 1978. Krenite (fosamine ammonium): A review of its biological properties. *Proc. Plant Growth Regul. Work Group Annu. Meet.* 5:250-251.
- WSSA. 1994. *Herbicide handbook*. Weed Society of America. Champaign, Illinois. 352 pp.

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GLYPHOSATE

M. Tu, C. Hurd, R. Robison & J.M. Randall

Herbicide Basics

Chemical formula: N-(phosphonomethyl) glycine

Herbicide Family:
None generally recognized

Target Species: most annual and perennial plants

Forms: salts

Formulations: SL, EC

Mode of Action: amino acid synthesis inhibitor

Water Solubility:
900,000 ppm

Adsorption potential: high

Primary degradation mech:
slow microbial metabolism

Average Soil Half-life:
47 days

Mobility Potential: low

Dermal LD50 for rabbits:
>5,000 mg/kg

Oral LD50 for rats:
5,600 mg/kg

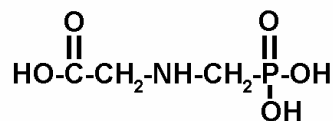
LC50 for bluegill sunfish:
120 mg/L

Trade Names: RoundUp[®], RoundUp-Pro[®], Rodeo[®], GlyPro[®], Accord[®], Glyphomax[®], Touchdown[®]

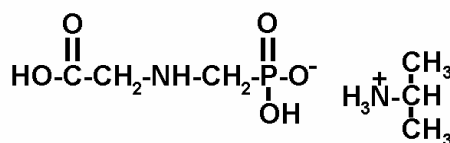
Manufacturers: Monsanto, Cenex/Land O'Lakes, Dow AgroSciences, Du Pont, Helena, and Platte.

Synopsis

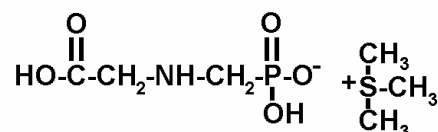
Glyphosate is a non-selective, systemic herbicide that can control most annual and perennial plants. It controls weeds by inhibiting the synthesis of aromatic amino acids necessary for protein formation in susceptible plants. Glyphosate is strongly adsorbed to soil particles, which prevents it from excessive leaching or from being taken-up from the soil by non-target plants. It is degraded primarily by microbial metabolism, but strong adsorption to soil can inhibit microbial metabolism and slow degradation. Photo- and chemical degradation are not significant in the dissipation of glyphosate from soils. The half-life of glyphosate ranges from several weeks to years, but averages two months. In water, glyphosate is rapidly dissipated through adsorption to suspended and bottom sediments, and has a half-life of 12 days to ten weeks. Glyphosate by itself is of relatively low toxicity to birds, mammals, and fish, and at least one formulation sold as Rodeo[®] is registered for aquatic use. Some surfactants that are included in some formulations of glyphosate, however, are highly toxic to aquatic organisms, and these formulations are not registered for aquatic use. Monsanto's patent for glyphosate expired in 2000, and other companies are already selling glyphosate formulations.



Glyphosate acid



Glyphosate isopropylamine salt



Glyphosate trimethylsulfonium salt

Herbicide Details

Chemical Formula: N-(phosphonomethyl) glycine

Trade Names: Monsanto discovered and held the patent for glyphosate, and was for many years, the only company that manufactured and sold this herbicide. The patent expired in 2000, however, and already several other companies are making and selling glyphosate formulations. Some of the current trade names include: Roundup Ultra[®], Roundup Pro[®], Accord[®], Honcho[®], Pondmaster[®], Protocol[®], Rascal[®], Expedite[®], Ranger[®], Bronco[®], Campain[®], Landmaster[®], and Fallow Master[®] by Monsanto; Glyphomax[®] and Glypro[®] by Dow AgroSciences; Glyphosate herbicide by Du Pont; Silhouette[®] by Cenex/Land O'Lakes; Rattler[®] by Helena; MirageR[®] by Platte; JuryR[®] by Riverside/Terra; and Touchdown[®] by Zeneca. As of November 2001, Rodeo[®] (previously manufactured by Monsanto) is now being manufactured by Dow AgroSciences and Monsanto is now producing Aquamaster[®].

Manufacturers: Current manufacturers include Monsanto, Cenex/Land O'Lakes, Helena, Platte, Riverside/Terra, Dow AgroSciences, and Zeneca.

Use Against Natural Area Weeds: Glyphosate is a broad-spectrum, nonselective systemic herbicide that kills or suppresses many grasses, forbs, vines, shrubs, and trees. Care should be taken, especially in natural areas, to prevent it from being applied to desirable, native plants, because it will likely kill them. In terrestrial systems, glyphosate can be applied to foliage, green stems, and cut-stems (cut-stumps), but cannot penetrate woody bark (Carlisle & Trevors 1988). Only certain formulations of glyphosate (e.g., Rodeo[®]) are registered for aquatic use, as glyphosate by itself is essentially non-toxic to submersed plants (Forney & Davis 1981), but the adjuvants often sold with glyphosate may be toxic to aquatic plants and animals.

Glyphosate is one of the most commonly used herbicides in natural areas, because it provides effective control of many species. Natural area weeds that have been controlled with glyphosate include: bush honeysuckle (*Lonicera maackii*), cogon grass (*Imperata cylindrica*), common buckthorn (*Rhamnus cathartica*), glossy buckthorn (*Frangula alnus*), Japanese honeysuckle (*Lonicera japonica*), and smooth brome (*Bromus inermis*). In TNC preserves, glyphosate has been used to control dewberries (*Rubus* spp.), bigtooth aspen (*Populus grandidentata*), and black cherry (*Prunus serotina*) at Kitty Todd preserve in Ohio, sweetclover (*Melilotus officinalis*) in Indiana preserves, leafy spurge (*Euphorbia esula*) and St. John's wort/Klamath weed (*Hypericum perforatum*) in Michigan preserves, and bindweed (*Convolvulus arvensis*) and velvetgrass (*Holcus lanatus*) in Oregon and Washington preserves.

In aquatic or wetland systems, glyphosate has successfully controlled common reed (*Phragmites australis*) in Delaware, Michigan, and Massachusetts preserves, purple loosestrife (*Lythrum salicaria*) in Indiana and Michigan preserves, reed canarygrass (*Phalaris arundinacea*) in Illinois preserves, and glossy buckthorn (*Frangula alnus*) and hybrid cattail (*Typha x glauca*) in Michigan preserves.

Mode of Action: Glyphosate kills plants by inhibiting the activity of the enzyme 5-enolpyruvylshikimic acid-3-phosphate synthase (EPSP), which is necessary for the formation of the aromatic amino acids tyrosine, tryptophan, and phenylalanine. These amino acids are important in the synthesis of proteins that link primary and secondary metabolism (Carlisle & Trevors 1988). EPSPs are present in the chloroplast of most plant species, but are not present in animals. Animals need these three amino acids, but obtain them by eating plants or other animals.

Glyphosate is therefore, relatively non-toxic to animals (Monsanto Company 1985). Certain surfactants or other ingredients that are added to some glyphosate formulations are toxic to fish and other aquatic species (EXTOXNET 1996).

Glyphosate can also act as a competitive inhibitor of phosphoenolpyruvate (PEP), which is one of the precursors to aromatic amino acid synthesis. It also affects other biochemical processes, and, although these effects are considered secondary, they may be important in the total lethal action of glyphosate.

Dissipation Mechanisms:

Summary: Glyphosate is degraded primarily by microbial metabolism. Glyphosate is believed to be susceptible to photodegradation (Lund-Hoie & Friestad 1986), but the extent to which this occurs is uncertain. Glyphosate is not significantly degraded by other chemical mechanisms in the field. Glyphosate is strongly adsorbed to soil, which can slow microbial metabolism but prevents excessive movement in the environment. Glyphosate is non-volatile (T. Lanini, pers. obs).

Volatilization

Glyphosate does not volatilize readily when applied in the field (T. Lanini, pers. obs.).

Photodegradation

Although originally thought to be unaffected by sunlight (Rueppel et al. 1977), later studies found glyphosate to be susceptible to photodegradation (Lund-Hoie & Friestad 1986; Carlisle & Trevors 1988). Lund-Hoie and Friestad (1986) reported a half-life of four days for glyphosate in deionized water under UV light.

Microbial Degradation

Glyphosate is degraded primarily by microbial metabolism. Two steady rates of degradation have been identified (Rueppel et al. 1977). It has been hypothesized that the more rapid rate of degradation represents the metabolism of unbound glyphosate molecules, while the slower rate represents the metabolism of glyphosate molecules bound to soil particles (Nomura & Hilton 1977; Rueppel et al. 1977). The degradation of glyphosate is slower in soils with a higher adsorption capacity. Degradation rate was also affected by the particular microbial community of each soil (Carlisle & Trevors 1988; Malik et al. 1989). The primary metabolite of glyphosate is aminomethylphosphonic acid, which is non-toxic and degraded microbially at a somewhat slower rate than the parent compound (Nomura & Hilton 1977; Rueppel et al. 1977;

Carlisle & Trevors 1988). A number of other minor, biodegradable metabolites have also been identified.

Adsorption

Glyphosate is water-soluble, but it has an extremely high ability to bind to soil particles. Adsorption of glyphosate increases with increasing clay content, cation exchange capacity, and decreasing soil pH and phosphorous content (Sprankle et al. 1975a,b; Hance 1976; Nomura & Hilton 1977; Rueppel et al. 1977; Glass 1987). Glyphosate is adsorbed to soil particles rapidly during the first hour following application and slowly thereafter (Sprankle et al. 1975b). Strong adsorption to soil particles slows microbial degradation, allowing glyphosate to persist in soils and aquatic environments. Because glyphosate rapidly binds to soils, it has little or no herbicidal activity (“killing power”) once it touches soil (Sprankle et al. 1975a; Hance 1976; Nomura & Hilton 1977). Glyphosate can also be inactivated by adsorption if mixed with muddy water.

Adsorption prevents glyphosate from being mobile in the environment except when the soil particles themselves are washed away (Sprankle et al. 1975b; Rueppel et al. 1977; Roy et al. 1989a). Comes et al. (1976) found that glyphosate sprayed directly into a dry irrigation canal was not detectable in the first irrigation waters flowing through the canal several months later, although glyphosate residues remained in the canal soils. In most cases, glyphosate is quickly adsorbed to suspended and bottom sediments (Feng et al. 1990).

Chemical Decomposition

Glyphosate is not readily hydrolyzed or oxidized in the field (Rueppel et al. 1977; Anton et al. 1993; Zaranyika & Nyandoro 1993).

Behavior in the Environment

Summary: Glyphosate binds readily with soil particles, which limits its movement in the environment. It is degraded through microbial metabolism with an average half-life of two months in soils and two to ten weeks in water. In plants, glyphosate is slowly metabolized.

Soils

Glyphosate is highly water soluble, but unlike most water-soluble herbicides, glyphosate has a very high adsorption capacity. Once glyphosate contacts soil it is rapidly bound to soil particles rendering it essentially immobile (Roy et al. 1989a; Feng & Thompson 1990). Unbound glyphosate molecules are degraded at a steady and relatively rapid rate by soil microbes (Nomura & Hilton 1977; Rueppel et al. 1977). Bound glyphosate molecules also are biologically degraded at a steady, but slower rate. The half-life of glyphosate in soil averages two months but can range from weeks to years (Nomura & Hilton 1977; Rueppel et al. 1977; Newton et al. 1984; Roy et al. 1989a; Feng & Thompson 1990; Anton et al. 1993). Although the strong adsorption of glyphosate allows residues to persist for over a year, these residues are largely immobile and do not leach significantly. Feng and Thompson (1990) found that >90% of glyphosate residues were present in the top 15 cm of soil and were present as low as 35 cm down the soil column in only one of 32 samples. Adsorption to soil particles prevents glyphosate from being taken-up by the roots of plants.

Water

Because glyphosate binds strongly to soils, it is unlikely to enter waters through surface or sub-surface runoff except when the soil itself is washed away by runoff, and even then, it remains bound to soil particles and unavailable to plants (Rueppel et al. 1977, Malik et al. 1989). Most glyphosate found in waters likely results from runoff from vegetation surfaces, spray drift, and intentional or unintentional direct overspray. In most cases, glyphosate will dissipate rapidly from natural water bodies through adsorption to organic substances and inorganic clays, degradation, and dilution (Folmar et al. 1979; Feng et al. 1990; Zaranyika & Nyandoro 1993; Paveglio et al. 1996). Residues adsorbed to suspended particles are precipitated into bottom sediments where they can persist until degraded microbially with a half-life that ranges from 12 days to 10 weeks (Goldsborough & Brown 1993; EXTOWNET 1996). At least one study found that >50% of the glyphosate added directly to the waters of an irrigation canal were still present 14.4 km downstream (Comes et al. 1976).

Vegetation

Glyphosate is metabolized by some, but not all plants (Carlisle & Trevors 1988). It is harmless to most plants once in the soil because it is quickly adsorbed to soil particles, and even when free, it is not readily absorbed by plant roots (Hance 1976). The half-life of glyphosate on foliage has been estimated at 10.4 to 26.6 days (Newton et al. 1984). Roy et al. (1989b) found 14% and 9% of applied glyphosate accumulated in the berries of treated blueberry and raspberry bushes, respectively. These residues dissipated from the fruit with a half-life of <20 days for blueberries and <13 days for raspberries (Roy et al. 1989b).

Environmental Toxicity

Birds and Mammals

Glyphosate is of relatively low toxicity to birds and mammals (Evans & Batty 1986). The LD50 of glyphosate for rats is 5,600 mg/kg and for bobwhite quail, >4,640 mg/kg. EPA's Re-registration Eligibility Decision states that blood and pancreatic effects and weight gain were noted during subchronic feeding studies with rats and mice (EPA 1993). Other studies show developmental and reproductive impacts to animals given the highest dose.

Newton et al. (1984) examined glyphosate residues in the viscera of herbivores following helicopter application of glyphosate to a forest in Oregon and found residue levels comparable to those found in litter and ground cover (<1.7 mg/kg). These residue levels declined over time and were undetectable after day 55 (Newton et al. 1984). Although carnivores and omnivores exhibited much higher viscera residue levels (5.08 mg/kg maximum), Newton et al. (1984) concluded that carnivores were at lower risk than herbivores due to the lower relative visceral weights and a proportionally lower level of food intake.

Batt et al. (1980) found no effect on chicken egg hatchability or time to hatch when an egg was submerged in a solution of 5% glyphosate. Sullivan and Sullivan (1979) found that black-tailed deer showed no aversion to treated foliage and consumption of contaminated forage did not reduce total food intake. Significant impacts to bird and mammal populations due to large-scale habitat alterations following treatment of forest clearcuts with glyphosate have been reported (Morrison & Meslow 1984; Santillo et al. 1989a,b; MacKinnon & Freedman 1993).

Aquatic Species

Glyphosate itself is of moderate toxicity to fish. The 96-hour LC50 of technical grade glyphosate for bluegill sunfish and rainbow trout are 120 mg/L and 86 mg/L, respectively. Fish exposed to 5 mg/L of glyphosate for two weeks were found to have gill damage and liver damage was observed at glyphosate concentrations of 10 mg/L (Neskovic et al. 1996). The technical grade of glyphosate is of moderate toxicity to aquatic species, and the toxicity of different glyphosate formulations can vary considerably. For example, Touchdown 4-LC[®] and Bronco[®] have low LC50s for aquatic species (<13 mg/L), and are not registered for aquatic use. On the other hand, Rodeo[®] has relatively high LC50s (>900 mg/L) for aquatic species and is permitted for use in aquatic systems. The surfactant in Roundup[®] formulations is toxic to fish, however, Rodeo[®] has no surfactant, and is registered for aquatic use.

The surfactant X-77 Spreader[®], which is often used in conjunction with Rodeo[®], is approximately 100 times more toxic to aquatic invertebrates than Rodeo[®] alone (Henry et al. 1994). The surfactant MONO818[®] is included in Roundup[®] formulations because it aids the break-down of surface tension on leaf surfaces, but it may also interfere with cutaneous respiration in frogs and gill respiration in tadpoles (Tyler 1997 a,b). In addition, MONO818[®] is highly toxic to fish (Folmar et al. 1979; Servizi et al. 1987). The LC50 of MONO818[®] is 2-3 mg/L for sockeye, rainbow, and coho fry (Folmar et al. 1979; Servizi et al. 1987; Tyler 1997 a,b). The LC50 of Roundup[®] for bluegill sunfish and rainbow trout is only slightly higher at 6-14 mg/L and 8-26 mg/L, respectively. Similarly for *Daphnia*, the 96-hour LC50 of glyphosate alone is 962 mg/L, but the LC50 of Roundup[®] drops to 25.5 mg/L (Servizi et al. 1987). Roundup[®] is therefore not registered for use in aquatic systems.

Despite these toxicity levels, Hildebrand et al. (1980) found that Roundup[®] treatments at concentrations up to 220 kg/ha did not significantly affect the survival of *Daphnia magna* or its food base of diatoms under laboratory conditions. In addition, Simenstad et al. (1996) found no significant differences between benthic communities of algae and invertebrates on untreated mudflats and mudflats treated with Rodeo[®] and X-77 Spreader[®]. It appears that under most conditions, rapid dissipation from aquatic environments of even the most toxic glyphosate formulations prevents build-up of herbicide concentrations that would be lethal to most aquatic species.

Other Non-Target Organisms

Roberts and Berk (1993) investigated the effects of Roundup[®] on chemoattraction of the protozoa *Tetrahymena pyriformis* and found that it significantly interfered with chemoreception but not motility. Doses of glyphosate <10 ppm were stimulatory to soil microflora including actinomycetes, bacteria, and fungi, while concentrations > 10 ppm had detrimental impacts on microflora populations in one study (Chakravarty & Sidhu 1987). While some short-term studies (< 30 days) found glyphosate caused significant impacts to microbial populations, Roslycky (1982) found that these populations rebound from any temporary increase or decrease within 214 days. Similarly, Tu (1994) found that microorganisms recovered rapidly from treatment with glyphosate and that the herbicide posed no long-term threat to microbial activities.

Application Considerations:

Glyphosate can be applied using conventional, recirculating, wet apron, hooded and hand-operated sprayers; controlled drop, rope-wick, roller, and carpet applicators; mistblowers; injectors; and wipe-on devices (Carlisle & Trevors 1988). Feng et al. (1990) found that 10 meter buffer zones limited unintentional effects through chemical drift and off-target deposits into streams during application, while Marrs et al. (1993) concluded that 20 meters was a safe buffer width. Liu et al. (1996) found that increasing the glyphosate concentration was more effective in controlling weeds than increasing the droplet size. Thielen et al. (1995) concluded that the cations of hard water, including Ca^{++} and Mg^{++} , can greatly reduce the efficacy of glyphosate when present in a spray solution. Addition of ammonium sulfate or other buffer can precipitate out heavy elements in “hard” water if added before the herbicide is mixed with water.

When glyphosate is used as an aquatic herbicide, do not treat the entire water body at one time. Treat only one-third to one-half of any water body at any one time, to prevent fish kills caused by dissolved oxygen depletion.

Safety Measures:

Some glyphosate formulations are in EPA toxicity categories I and II (the two highest categories) for eye and skin exposure. Care should be taken and protective clothing worn to prevent accidental contact of these formulations on skin or eyes.

Human Toxicology:

EPA classified glyphosate as a “Group E” carcinogen or a chemical that has not shown evidence of carcinogenicity in humans (EPA 1993).

References

- Anton, F.A., et al. 1993. Degradational behavior of the pesticides glyphosate and diflufenuron in water. *Bulletin of Environmental Contamination and Toxicology* 51:881-888.
- Batt, B.D., J.A. Black and W.F. Cowan. 1980. The effects of glyphosate herbicide on chicken egg hatchability. *Canadian Journal of Zoology* 58:1940-1942.
- Carlisle, S. M., and J. T. Trevors. 1988. Glyphosate in the environment. *Water Air Soil Pollut.* 39:409-420.
- Chakravarty, P., and S. S. Sidhu. 1987. Effect of glyphosate, hexazinone and triclopyr on in vitro growth of five species of ectomycorrhizal fungi. *Eur. J. For. Path.* 17:204-210.
- Comes, R.D., V.F. Bruns, and A.D. Kelley. 1976a. Residues and persistence of glyphosate in irrigation water. *Weed Science* 24(1):47-50.
- E.P.A. 1993. Glyphosate. R.E.D. Facts. Prevention, Pesticides and Toxic Substances. EPA-738-F-93-011.
- Evans, D.D. and M.J. Batty. 1986. Effects of high dietary concentrations of glyphosate on a species of bird, marsupial and rodent indigenous to Australia. *Environmental toxicology and chemistry* 5:399-401.
- EXTOXNET. 1996. Glyphosate. Pesticide Information Profiles. Extension Toxicology Network. <http://ace.orst.edu/info/extoxnet/>.
- Feng, J.C. and D.G. Thompson. 1990. Fate of glyphosate in a Canadian forest watershed: 2. Persistence in foliage and soils. *Journal of Agricultural Food Chemistry* 38:1118-1125.

- Feng, J.C., D.G. Thompson and P.E. Reynolds. 1990. Fate of glyphosate in a Canadian forest watershed: 1. Aquatic residues and off-target deposit assessment. *Journal of Agricultural Food Chemistry* 38:1110-1118.
- Folmar, L. C., H. O. Sanders, and A. M. Julin. 1979. Toxicity of the herbicide glyphosate and several of its formulations to fish and aquatic invertebrates. *Arch. Environ. Contam. Toxicol.* 8:269-278.
- Forney, D.R. and D.E. Davis. 1981. Effects of low concentrations of herbicides on submersed aquatic plants. *Weed Science* 29:677-685.
- Glass, R.L. 1987. Phosphate adsorption by soils and clay minerals. *Journal of Agricultural Food Chemistry* 35(4):497-500.
- Goldsborough, L.G. and D.J. Brown. 1993. Dissipation of glyphosate and aminomethylphosphonic acid in water and sediments of boreal forest ponds. *Environmental Toxicology and Chemistry* 12:1139-1147.
- Hance, R. J. 1976. Adsorption of glyphosate by soils. *Pestic. Sci.* 7:363-366.
- Helling, C. S., P. C. Kearney, and M. Alexander. 1971. Behavior of pesticides in soil. *Adv. Agron.* 23:147-240.
- Henry, C. J., K. F. Higgins, and K. J. Buhl. 1994. Acute toxicity and hazard assessment of RodeoR, X-77 SpreaderR, and Chem-TrolR to aquatic invertebrates. *Arch. Environ. Contam. Toxicol.* 27:392-399.
- Hildebrand, L. D., D. S. Sullivan, and T. P. Sullivan. 1980. Effects of RoundupR herbicide on populations of *Daphnia magna* in a forest pond. *Bull. Environ. Contam. Toxicol.* 25:353-357.
- Liu, S., R.A. Campbell, J.A. Studens, and R.G. Wagner. 1996. Absorption and translocation of glyphosate in Aspen (*Populus tremuloides*) as influenced by droplet size, droplet number, and herbicide concentration. *Weed Science* 44:482-488.
- Lund-Hoie, K, and H. O. Friestad. 1986. Photodegradation of the herbicide glyphosate in water. *Bull. Environ. Contam. Toxicol.* 36:723-729.
- MacKinnon, D.S. and B. Freedman. 1993. Effects of silvicultural use of the herbicide glyphosate on breeding birds of regenerating clearcuts in Nova Scotia, Canada. *Journal of Applied Ecology* 30:395-406.
- Malik, J., G. Barry and G. Kishore. 1989. A mini-review of "The herbicide glyphosate." *BioFactors* 2(1):17-25.
- Marrs, R.H., A. J. Frost, R. A. Plant, and P. Lunnis. 1993. Determination of buffer zones to protect seedlings of non-target plants from the effects of glyphosate spray drift. *Agriculture, Ecosystems and Environment* 45:283-293.
- Morrison, M.L. and E.C. Meslow. 1984. Effects of the herbicide glyphosate on bird community structure, western Oregon. *Forest Science* 30(1):95-106.
- Neskovic, N.K. et.al. 1996. Biochemical and histopathological effects of glyphosate on carp, *Cyprinus carpio*. *Bulletin of Environmental Contamination and Toxicology* 56:295-302.
- Newton, M. et.al. 1984. Fate of glyphosate in an Oregon forest ecosystem. 32:1144-1151.
- Nomura, N. S., and H. W. Hilton. 1977. The adsorption and degradation of glyphosate in five Hawaiian sugarcane soils. *Weed Research* 17:113-121.
- Paveglio, F.L. et.al. 1996. Use of Rodeo and X-77 spreader to control smooth cordgrass (*Spartina alterniflora*) in a southwestern Washington estuary: Environmental fate. *Environmental Toxicology and Chemistry* 15(6):961-968.

- Roberts, R.O. and S.G. Berk. 1993. Effect of copper, herbicides, and a mixed effluent on chemoattraction of *Tetrahymena pyriformis*. *Environmental Toxicology and Water Quality* 8:73-85.
- Roslycky, E. B. 1982. Glyphosate and the response of the soil microbiota. *Soil Biol. Biochem.* 14:87-92.
- Roy, D.N., S. K. Konar, S. Banerjee, D. A. Charles, D. G. Thompson, and R. Prasad. 1989b. Uptake and persistence of the herbicide glyphosate in fruit of wild blueberry and red raspberry. *Canadian Journal of Forest Research* 19:842-847.
- Roy, D.N., S. K. Konar, S. Banerjee, D. A. Charles, D. G. Thompson, and R. Prasad. 1989a. Persistence, movement and degradation of glyphosate in selected Canadian boreal forest soils. *Journal of Agricultural Food Chemistry* 37(2):437-440.
- Rueppel, M.L., B.B. Brightwell, J. Schaefer and J.T. Marvel. 1977. Metabolism and degradation of glyphosate in soil and water. *Journal of Agricultural and Food Chemistry* 25:517-528.
- Santillo, D.J., D. M. Leslie Jr., and P. W. Brown. 1989a. Response of small mammals and habitat to glyphosate application on clearcuts. *Journal of Wildlife Management* 53(1):164-172.
- Santillo, D.J., P. W. Brown, and D. M. Leslie, Jr.. 1989b. Response of songbirds to glyphosate-induced habitat changes on clearcuts. *Journal of Wildlife Management* 53(1):64-71.
- Servizi, J. A., R. W. Gordon, and D. W. Martens. 1987. Acute toxicity of Garlon 4 and Roundup herbicides to salmon, *Daphnia*, and trout. *Bull. Environ. Contam. Toxicol.* 39:15-22.
- Simenstad, C.A., et.al. 1996. Use of Rodeo and X-77 spreader to control smooth cordgrass (*Spartina alterniflora*) in a southwestern Washington estuary: 2. Effects on benthic microflora and invertebrates. *Environmental Toxicology and Chemistry* 15(6):969-978.
- Sprankle, P., W. F. Meggitt, and D. Penner. 1975a. Rapid inactivation of glyphosate in the soil. 1975a. *Weed Science.* 23(3):224-228.
- Sprankle, P. W. F. Meggitt, and D. Penner. 1975b. Adsorption, mobility, and microbial degradation of glyphosate in the soil. *Weed Science.* 23(3):229-234.
- Sullivan, T. P., and D. S. Sullivan. 1979. The effects of glyphosate herbicide on food preference and consumption in black-tailed deer. *Can. J. Zool.* 57:1406-1412.
- Thielen, K.D., E.P. Jackson and D. Penner. 1995a. The basis for the hard-water antagonism of glyphosate activity. *Weed Science* 43:541-548.
- Tu, C.M. 1994. Effects of herbicides and fumigants on microbial activities in soil. *Bulletin of Environmental Contamination and Toxicology* 53:12-17.
- Tyler, M.J. 1997a. Herbicides kill frogs. Newsletter of the declining amphibians population task force #21.
- Tyler, M. J. 1997b. Environmentally friendly: A false sense of security? *Species.* Newsletter of the Species Survival Commission, IUCN, The World Conservation Union. 29:20-21.
- WSSA. 1994. Herbicide handbook. Weed Society of America. Champaign, Illinois. 352 pp.
- Zaranyika, M.F. and M.G. Nydandoro. 1993. Degradation of glyphosate in the aquatic environment: An enzymatic kinetic model that takes into account microbial degradation of both free and colloidal (or sediment) particle adsorbed glyphosate. *Journal of Agricultural Food Chemistry* 41:838-842.

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HEXAZINONE

Herbicide Basics

Chemical formula: 3-cyclohexyl-6-(dimethylamino)-1-methyl-1,3,5-triazine-2,4(1H,3H)-dione

Target Species: annual, biennial, perennial, and woody weeds

Forms: not available as salt or ester

Formulations: SP, SC, WG, TB

Mode of Action:
Photosynthesis inhibitor

Water Solubility: 33,000 ppm

Adsorption potential: low

Primary degradation mech:
Slow microbial metabolism

Average Soil Half-life:
90 days

Mobility Potential: high

Dermal LD50 for rabbits:
>6,000 mg/kg

Oral LD50 for rats:
1,690 mg/kg

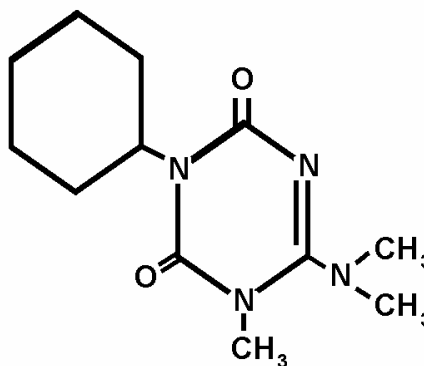
LC50 for bluegill sunfish:
370 mg/L

Trade Names: Pronone[®] and Velpar[®]

Manufacturers:
DuPont and Pro-Serve

Synopsis

Hexazinone controls some grasses, many annual and perennial broadleaf herbs, and some woody species, by inhibiting photosynthesis. It is water-soluble and does not bind strongly with soils, and so is of particular concern for groundwater contamination. Hexazinone can enter aquatic systems through surface and subsurface runoff following application and drift during application. It is degraded by microbial metabolism, but not readily decomposed chemically or by sunlight, and can therefore persist in aquatic systems. The average half-life of hexazinone in soils is 90 days, but it can sometimes be found in runoff up to six months after application. Although it is of relatively low toxicity to birds and mammals, legal application rates can leave residues that exceed EPA's Level of Concern for aquatic and terrestrial plants and small mammals. It is of relatively low toxicity to fish and aquatic invertebrates but can be highly toxic to some species of algae. Hexazinone contamination has been detected in small water-bodies in episodic, low-level pulses that were rapidly diluted in mainstream flows. High concentrations of hexazinone, however, could lead to significant losses of algae and macrophytic biomass, which could produce a ripple effect in the food chain that ultimately could impact fish and wildlife species. Although hexazinone can accumulate in treated crops, concentrations in vegetation are not likely to reach toxic levels for foraging animals when hexazinone is applied properly. Care should be taken in preparing and applying hexazinone as it can cause severe eye damage.



Herbicide Details

Chemical Formula: 3-cyclohexyl-6-(dimethylamino)-1-methyl-1,3,5-triazine-2,4 (1H,3H)-dione

Trade Names: Pronone[®] and Velpar[®]

Manufacturers: Du Pont and Pro-Serve

Use Against Natural Area Weeds: Hexazinone is a broad-spectrum herbicide that can control annual and perennial herbaceous broadleaf weeds, some grasses, and some woody species. It is often used to control brush in reforested areas, in tree plantations, and in rangeland and pasturelands.

Hexazinone is absorbed through the roots and foliage of plants, and best results are obtained for herbaceous species when applied in moist soil conditions, as either a foliage spray or basal soil treatment. Larger woody species are best controlled by injection or hack-and-squirt techniques. Species that have been controlled by hexazinone include: tansy-mustard (*Descurainia pinnata*), cheatgrass (*Bromus tectorum*), filaree (*Erodium* spp.), shepards-purse (*Capsella bursa-pastoris*), false dandelion (*Hypochaeris radicata*), privet (*Ligustrum* spp.), and Chinese tallowtree (*Sapium sebiferum*) (Du Pont 1993).

Hexazinone is water-soluble and does not bind strongly with soils, and so is of particular concern for groundwater contamination. It can persist in soils and aquatic systems for some time (average half-life in soil is 90 days), increasing the likelihood of contamination. No use of hexazinone was reported by TNC preserves in the 1998-99 TNC Weed Survey.

Mode of Action: Hexazinone is a systemic herbicide that inhibits photosynthesis in susceptible plants, diverting highly reactive molecules into a chain reaction that destroys chloroplast and cell membranes, and other vital compounds. It is usually applied as a pre-emergent herbicide, and soils must be moist (by rain or irrigation) to activate hexazinone. Hexazinone works by binding to a protein component of the photosystem II complex, which blocks electron transport. The result is a chain reaction in which triplet-state chlorophyll reacts with oxygen (O₂) to form singlet oxygen (O), and both the chlorophyll and singlet oxygen strip hydrogen ions (H⁺) from unsaturated lipids in cell and organelle membranes, producing lipid radicals. The lipid radicals in turn attack and oxidize other lipids and proteins, resulting in the loss of cell and organelle membrane integrity, loss of chlorophyll and carotenoids, leakage of cellular contents, cell death, and ultimately death of the plant (WSSA 1994).

Dissipation Mechanisms:

Summary: Hexazinone is primarily degraded through microbial metabolism in soils and sediments. It is not significantly affected by photo or chemical degradation. It is not readily adsorbed by sediments and can remain mobile in the environment until metabolized by microbes. Hexazinone is not highly volatile (T. Lanini, pers. obs.).

Volatilization

Hexazinone does not volatilize readily when applied in the field (T. Lanini, pers. obs). The potential to volatilize, however, increases with increasing temperature, increasing soil moisture, and decreasing clay and organic matter content (Helling et al. 1971).

Photodegradation

Hexazinone resists photodegradation (Neary et al. 1983). When exposed to artificial sunlight in distilled water, hexazinone degrades slowly (approx. 10% in five weeks) (Rhodes 1980b). Photodegradation can be three to seven times greater, however, in natural river water and/or in water containing a photoinitiator (a compound that catalyzes photodegradation) (Rhodes 1980b). Water pH and temperature do not affect hexazinone photodegradation rates significantly.

Microbial Degradation

Hexazinone is degraded primarily by microorganisms in soils (Rhodes 1980a, Jensen & Kimball 1987). Rhodes (1980a) found that no herbicidal degradation or loss occurred in soils kept in anaerobic conditions for 60 days. Conversely, in aerobic soils, 45-75% of the applied hexazinone was released as CO₂ within 80 days of application, likely as a result of microbial degradation (Rhodes 1980a).

Adsorption

Hexazinone has a comparatively low adsorption capacity. Adsorption of hexazinone to soil particles increases with increasing soil pH, organic content, and clay cation exchange capacity (Neary et al. 1983; Koskinen et al. 1996). Soil temperature does not alter the adsorption capacity significantly (Koskinen et al. 1996).

Chemical Decomposition

Hexazinone has been shown to degrade to eight or more different metabolites, identified as metabolites 'A' through 'H' (Rhodes 1980b). Only metabolite 'B' is believed to be toxic to plants, and even so, it has only 1% of the toxicity of hexazinone. The ratio metabolites formed during degradation varies with environmental conditions (i.e. climate and soil conditions control the predominance of a particular metabolites in soils) (Roy et al. 1989).

Jensen and Kimball (1987) found that under warm, moist conditions, hexazinone breaks down by chemical means to metabolite 'D'. In general, however, hexazinone has been found to be stable in aqueous solutions without the presence of sunlight or microbes (Rhodes 1980b), suggesting that independent of a catalyst, hexazinone does not readily degrade.

Behavior in the Environment

Summary: Hexazinone does not bind strongly with soils and can be highly mobile in the environment. It is degraded primarily through microbial metabolism with an average half-life of 90 days in soils and water. Its relative persistence and mobility make it a potential threat to off-site movement and contamination of non-target plants. As a result of its relative persistence and high mobility, it has a high potential to move off-site and contaminate water or kill desirable plants. Hexazinone residues can persist in leaf litter, releasing hexazinone into the environment long after application. Hexazinone metabolites are also persistent and mobile.

Soils

Hexazinone is relatively persistent in soils. Reported half-lives vary between one and six months with a mean of 90 days. Half-lives reported by Rhodes (1980) were one month in Delaware Keyport silt loam, two months in Illinois Flanagan silt loam, and six months in Mississippi Dundee silt loam. Neary et al. (1983) reported a half-life in mineral soils of NE Georgia of 10-30 days. In both clay and sand soils of a boreal forest in Ontario, Canada, the half-life of hexazinone was 43 days (Roy et al. 1989). Prasad and Feng (1990) found 1% of applied hexazinone and 0.4% of its metabolites remained in soils after one year.

Hexazinone does not bind strongly with soil particles and, theoretically, could be highly mobile (Rhodes 1980). Observations of vertical movement in soils, however, have been conflicting. Lavy et al. (1989) hypothesized that because adsorption increases with increasing organic content, adsorption should be highest in the soil's surface layers and decrease with soil depth. In support of this hypothesis, Roy et al. (1989) found that in sand and clay soils of boreal forests in Ontario, Canada, 98% of the hexazinone remained in the top 15 cm of soil, and leaching appeared to be retarded by mineral layers. Conversely, Zandvoort (1989) concluded that the slow degradation of hexazinone in soils could result in contamination of deep soil layers. In support of this conclusion, Feng et al. (1992) reported hexazinone and its metabolite residues leached to 130 cm and were still detectable two years after application in northern Alberta. Degradation is usually slowest in cold, dry climates, like that of northern Alberta.

The granular formulation of hexazinone has greater lateral mobility than the liquid formulation (Prasad & Feng 1990). Although the granular formulation initially provides spotty coverage, horizontal movement over time redistributes granular formulations to give coverage comparable to liquid formulations (Feng et al. 1992).

Because of the hexazinones' mobility in soils, it has the potential to move off-site and affect non-target species up to 100 meters away (Allender 1991). Sidhu and Feng (1993) reported that granular formulations applied during fall, contaminated nearby marsh reed grass in surface runoff during spring melt the following year. These residues persisted in the marsh reed grass up to two years later.

Water

Hexazinone is water-soluble and does not bind strongly with soils (EXTOXNET 1996). It can be highly mobile in surface and sub-surface water runoff and has the potential to contaminate groundwater and surface water bodies (Schneider et al. 1995; Peterson et al. 1997). Hexazinone has been found in groundwater in four states. The reported half-life of hexazinone in water varies between several days (Solomon et al. 1988) to more than nine months (Thompson et al. 1992). Thompson et al. (1992) reported that in treated enclosures on a boreal lake in Ontario, hexazinone concentrations of 1-10 mg/L persisted for 35-49 days. Neary et al. (1986) concluded that use of hexazinone would result in smaller water quality changes than do commonly used, intensive mechanical weed control techniques, particularly on steep forest slopes with fragile soils.

Mayack et al. (1982) and Neary et al. (1983; 1984) report that subsurface runoff of small amounts of hexazinone are episodic and unpredictable, and are diluted in the mainstream flow to very low concentrations. In a study funded by DuPont, Neary et al. (1986) found low concentrations of hexazinone in storm water runoff for seven months following application to the upper Piedmont in North Georgia, with a loss of 0.53% of the total volume applied. Lavy et al. (1989) evaluated the fate of hexazinone in a steep watershed in north-central West Virginia and found that 4.7% of the total hexazinone applied to the watershed leached into the local streams, but none of the compound was found in stream sediments.

Vegetation

Hexazinone is absorbed primarily through the root system, but also can be taken up from the foliage. In non-susceptible species, the herbicide is broken down into non-toxic or less toxic forms (e.g. metabolite 'A'), while in target species, the parent compound, hexazinone, and its phytotoxic metabolite 'B' persist and can inhibit photosynthesis and cause chloroplast damage (Sidhu & Feng 1993). Some studies have found that sublethal concentrations of hexazinone ($<10^{-6}$ M) can enhance photosynthesis rates in some species, but the mechanism for this is unknown (Sung et al. 1985; Johnson & Stelzer 1991).

A number of authors hypothesize that the leaves and other litter dropped from treated vegetation may serve as a reservoir for hexazinone, and can release it into the environment up to three years following application (Mayack et al. 1982; Neary et al. 1984; Lavy et al. 1989). This reservoir, however, may also function to prevent excessive runoff immediately following application in steep watersheds (Lavy et al. 1989).

Environmental Toxicity

Birds and Mammals

Hexazinone is of relatively low toxicity to birds and mammals. The oral LD50 for rats is 1,690 mg/kg and 2,258 mg/kg for bobwhite quail (WSSA 1994). Higher application rates can produce residues that exceed EPA's Level of Concern for small mammals. In a study of rat metabolism of hexazinone, at least 93.3% of the radio-labelled hexazinone was excreted by the animals and none of the compound was detected in the rats after 72 hours (Rhodes & Jewell 1980).

Treated plants can sequester herbicides in their foliage, which could be ingested by foraging wildlife. Sidhu and Feng (1993) found that at application rates of 4 kg a.i./ha, as much as 16 mg of hexazinone could be present in each kg of dry vegetation. They concluded, however, that even at this rate, the maximum level of hexazinone and its metabolites in foliage would be below the levels known to cause toxic effects in animals. Peterson et al. (1997) concluded that detrimental effects of hexazinone on wildlife would most likely be indirect, resulting from declines in food resources and habitat quality due to losses of primary productivity in treated areas.

Aquatic Species

Hexazinone is only slightly toxic to most aquatic animals, but can be extremely toxic to some algae and aquatic macrophytes (EPA 1994). Hexazinone may also cause indirect effects to aquatic communities through destruction of riparian vegetation (Mayack et al. 1982).

Fish

Hexazinone is only slightly toxic to fish (EXTOXNET 1996). The LC50 for rainbow trout and bluegill sunfish are 320 and 370 mg/L, respectively; well above the residue levels found in the streams of treated watersheds. When bluegill sunfish were exposed to concentrations ≤ 1 ppm for 28 days (more than twice the maximum runoff reported by Mayack et al. (1982) in Georgia), no mortalities or changes in behavior or physical condition were observed (Rhodes 1980b). Tissue residue levels were found to peak after one to two weeks of exposure and to be completely eliminated after two weeks of withdrawal.

Hexazinone has been found to be slightly toxic to juvenile Pacific salmonids, with LC50 (96-hour) values of 236-317 mg/L (Wan et al. 1988). The formulations of Pronone 10G[®] and Velpar[®] were found to be significantly less toxic, suggesting that the additives in these formulation were not only less toxic than hexazinone itself, but somehow reduced the toxic effect of hexazinone to salmonids (Wan et al. 1988). No hexazinone formulations are registered for aquatic use.

Phytoplankton

Thompson et al. (1992, 1993a) found that the biomass of phytoplankton in boreal forest lakes in Ontario, Canada was depressed at hexazinone concentrations as low as 0.01 mg/L, and that chronic exposure to concentrations > 0.1 mg/L caused irreversible damage to phytoplankton communities. A corresponding decrease in zooplankton populations occurred as well, likely as a response to food resources lost with the decline of phytoplankton. Field studies have reported temporary contamination levels as high as 0.04 mg/L (Lavy et al. 1982), but chronic exposure to higher rates following proper application of hexazinone is unlikely.

Aquatic Invertebrates

Kreutzweiser et al. (1992) and Schneider et al. (1995) both found that in simulated stream channels, the addition of hexazinone did not affect the survival of stream insects, and concluded that there was little risk of toxic effects to macroinvertebrates (Kreutzweiser et al. 1992, 1995; Schneider et al. 1995). Similarly, Mayack et al. (1982) found no differences in diversity or species composition of aquatic invertebrate communities between treated and untreated sites.

Periphyton

Several studies in labs, simulated streams, and lake enclosures have shown that hexazinone is toxic to algae and can slow growth rates after one day of exposure. Concentrations reported to cause detrimental effects (0.01-0.60 mg/L) are well above the monthly average levels (0.00025-0.0031 mg/L) reported by Lavy et al. (1989) in streams of West Virginia following proper application of hexazinone in the watershed. Although one-time concentrations following significant storm events may exceed the tolerance threshold for some alga species, chronic exposure to lethal doses does not seem likely.

If chronic exposure did occur, it could cause significant losses in biomass of some algae (About-Waly et al. 1991b; Kreutzweiser et al. 1995; Schneider et al. 1995). Schneider et al. (1995) hypothesized that exposure of algae to hexazinone for time periods equivalent to the algae population's doubling time could have significant consequences for the productivity and recovery of the community. A decline in green algae and diatoms following low-level contamination by hexazinone could ripple through the food chain and impact fish and wildlife productivity (Peterson et al. 1997). In addition, because impacts on cyanobacteria are relatively minimal, these organisms could proliferate where other algae are suppressed, altering the aquatic habitat and possibly contaminating drinking water supplies (Peterson et al. 1997).

Other Non-Target Organisms

Mayack et al. (1982) found that terrestrial macroinvertebrates accumulate hexazinone and its metabolites at levels one to two times the concentration in forest litter. It is not known whether hexazinone "magnifies" up the food chain, with organisms that feed on macroinvertebrates accumulating even higher concentrations. A study using rats suggests that mammals that ingest hexazinone can eliminate it from their systems (Rhodes & Jewell 1980).

Hexazinone has not been shown to be toxic to soil bacteria or fungi (Chakravarty & Chatarpaul 1990; Maynard 1993). Rhodes et al. (1980) found that slight increases in fungal and bacterial populations occurred with the addition of hexazinone. Fungal community structure was not altered, nor were populations of soil microbes reduced at hexazinone concentrations ≤ 10 ppm.

Soil Nutrient Cycling

Hexazinone has little if any effect on the cycling of nutrients in soils (Rhodes et al. 1980; Maynard 1993, 1997). Maynard (1993) found no effect on CO₂ respiration, ammonification, and nitrification or sulfur mineralization in incubated forest soils, and concluded that hexazinone would have little impact on nutrient-cycling processes when applied at the recommended field rates. Rhodes et al. (1980) additionally, found hexazinone had no effect on the soil-nitrifying process in three agricultural soils at hexazinone concentrations of five and 20 ppm over five weeks. Changes in vegetation coverage and the input of litter from plants killed due to the application, however, could lead to indirect effects on soil nutrient and carbon cycles (Maynard 1996). Nonetheless, Maynard (1996) found no changes in the total nutrient pool over six years in a treated boreal mixed-wood forest in Alberta.

Safety Measures:

Hexazinone can cause severe eye damage. Care should be taken to prevent accidental splashing or other exposure to eyes.

Application Considerations:

- Because hexazinone is absorbed by the roots, it is most effective in soils that do not readily bind it, such as those low in organic content, clay, silt, and cation exchange capacity, but high in sand (Minogue et al. 1988; Wilkins et al. 1993).
- Application of the liquid formulation reduces lateral movement of the herbicide, which may reduce impacts on non-target plants (Prasad & Feng 1990).

- Where granular formulations are applied in late autumn or early winter, hexazinone may be released during spring snow melt (Sidhu & Feng 1993).
- Most formulations require water to become activated, thus, best results occur when the soil is moist at the time of application and when 1/4-1/2 inch of rain falls within two weeks of application.

Human Toxicology:

Hexazinone is of relatively low toxicity to mammals, but can cause severe eye damage. The U.S. EPA classifies hexazinone as a "Group D" carcinogen, or a chemical that is not classifiable as a human carcinogen (EPA 1994).

References

- Abou-Waly, H., M. Abou-Setta, H. N. Nigg, and L. L. Mallory. 1991. Growth response of freshwater algae, *Anabaena flos-aquae* and *Selenastrum capricornutum* to atrazine and hexazinone herbicides. *Bull. Environ. Contam. Toxicol.* 46:223-229.
- Abou-Waly, H., M. M. Abou-Setta, H. N. Nigg, and L. L. Mallory. 1991. Dose-response relationship for *Anabaena flos-aquae* and *Selenastrum capricornutum* to atrazine and hexazinone using chlorophyll (a) content and ¹⁴C uptake. *Aquat. Toxicol.* 20:195-204.
- Allender, W. J. 1991. Movement of bromacil and hexazinone in a municipal site. *Bull. Environ. Contam. Toxicol.* 46:284-291.
- Chakravarty, P. and L. Chatarpaul. 1990. Non-target effect of herbicides: I. Effect of glyphosate and hexazinone on soil microbial activity. Microbial population, and in-vitro growth of ectomycorrhizal fungi. *Pestic. Sci.* 28:233-241.
- E.P.A. 1994. Hexazinone. R.E.D. Facts. Prevention, Pesticides and Toxic Substances. EPA-738-F-94-019.
- EXTOXNET. 1996. Hexazinone. Pesticide Information Profiles. Extension Toxicology Network. <http://ace.orst.edu/info/extoxnet/>.
- Feng, J. C., S. S. Sidhu, and C. C. Feng. 1992. Spatial distribution of hexazinone and metabolites in a luvisolic soil. *J. Environ. Sci. Health B27(6)*:639-654.
- Fowler, M. C. 1977. Laboratory trials of a new triazine herbicide (DPX 3674) on various aquatic species of macrophytes and algae. *Weed Res.* 17:191-195.
- Helling, C. S., P. C. Kearney, and M. Alexander. 1971. Behavior of pesticides in soil. *Adv. Agron.* 23:147-240.
- Jensen, K. I. N. and E. R. Kimball. 1987. Persistence and degradation of the herbicide hexazinone in soils of lowbush blueberry fields in Nova Scotia, Canada. *Bull. Environ. Contam. Toxicol.* 38:232-239.
- Johnson, J. D. and H. E. Stelzer. 1991. Loblolly pine photosynthesis is enhanced by sublethal hexazinone concentrations. *Tree Phys.* 8:371-379.
- Koskiene, W. C., D. M. Stone, and A. R. Harris. 1996. Sorption of hexazinone, sulfometuron methyl, and tebuthiuron on acid, low base saturated sands. *Chemosphere* 32(9):1681-1689.
- Kreutzweiser, D. P., S. B. Holmes, and D. J. Behmer. 1992. Effects of the herbicides hexazinone and triclopyr ester on aquatic insects. *Ecotoxicol. Environ. Saf.* 23:364-374.
- Kreutzweiser, D. P., S. S. Capell, and B. C. Sousa. 1995. Hexazinone effects on stream periphyton and invertebrate communities. *Env. Tox. Chem.* 14(9):1521-1527.

- Lavy, T. L., J. D. Mattice, and J. N. Kochenderfer. 1989. Hexazinone persistence and mobility of a steep forested watershed. *J. Environ. Qual.* 18:507-514.
- Mayack, D. T., P. B. Bush, D. G. Neary, and J. E. Douglass. 1982. Impact of hexazinone on invertebrates after application to forested watersheds. *Arch. Environm. Contam. Toxicol.* 11:209-217.
- Maynard, D. G. 1993. The influence of hexazinone on carbon dioxide evolution and mineralization of nitrogen, phosphorus and sulfur in a forest soil. *Can. J. Soil Sci.* 73:433-445.
- Maynard, D. G. 1997. Soil nutrient dynamics in a boreal mixedwood cutover following the application of hexazinone. *Ecol. Aps.* 7(2):416-430.
- Minogue, P. J., B. R. Zutter, and D. H. Gjerstad. 1988. Soil factors and efficacy of hexazinone formulations for loblolly pine (*Pinus taeda*) release. *Weed Sci.* 36:399-405.
- Neary, D. G., P. B. Bush, and M. A. Grant. 1986. Water quality of ephemeral forest streams after site preparation with the herbicide hexazinone. *For. Ecol. Manage.* 14:23-40.
- Neary, D. G., P. B. Bush, and J. E. Douglass. 1983. Off-site movement of hexazinone in stormflow and baseflow from forest watersheds. *Weed Sci.* 31:543-551.
- Neary, D. G., J. W. Taylor, Jr., and P. B. Bush. 1984. Fate of hexazinone in forest watersheds. U.S.D.A. Forest Service, Forest Pest Management Technology Update, Forestry Bulletin R8-FB/P 9.
- Privman, M., E. B. Rupp, and P. Zuman. 1994. Hexazinone: polarographic reduction and adsorption on lignin. *J. Agric. Food Chem.* 42:2946-2952.
- Prasad, R. and J. C. Feng. 1990. Spotgun-applied hexazinone: release of red pine (*Pinus resinosa*) from quaking aspen (*Populus tremuloides*) competition and residue persistence in soil. *Weed Tech.* 4:371-375.
- Rhodes, R. C. 1980a. Soil Studies with ¹⁴C-labeled hexazinone. *J. Agric. Food Chem.* 28:311-315.
- Rhodes, R. C. 1980b. Studies with ¹⁴C-labeled hexazinone in water and bluegill sunfish. *J. Agric. Food Chem.* 28:306-310.
- Rhodes, R. C. and R. A. Jewell. 1980. Metabolism of ¹⁴C-labeled hexazinone in the rat. *J. Agric. Food Chem.* 28:303-306.
- Rhodes, R. C., R. L. Krause, and M. H. Williams. 1980. Microbial activity in soils treated with hexazinone. *Soil Sci.* 129:311-314.
- Roy, D. N., S. K. Konar, D. A. Charles, J. C. Feng, R. Prasad, and R. A. Campbell. 1989. Determination of persistence, movement, and degradation of hexazinone in selected Canadian boreal forest soils. *J. Agric. Food Chem.* 37:443-447.
- Schneider, J., A. Morin, and F. R. Pick. 1995. The response of biota in experimental stream channels to a 24-hour exposure to the herbicide Velpar L^R. *Environ. Toxicol. Chem.* 14(9):1607-1613.
- Sidhu, S. S., and J. C. Feng. 1993. Hexazinone and its metabolites in boreal forest vegetation. *Weed Sci.* 41:281-287.
- Solomon, K. R., C. S. Bowhey, K. Liber, and G. R. Stephenson. 1988. Persistence of hexazinone (Velpar), triclopyr (Garlon), and 2,4-D in a Northern Ontario aquatic environment. *J. Agric. Food Chem.* 36:1314-1318.
- Sung, S. S., D. B. South, and D. H. Gjerstad. 1985. Bioassay indicates a metabolite of hexazinone affects photosynthesis of loblolly pine (*Pinus taeda*). *Weed Sci.* 33:440-442.

- Thompson, D. G., L. M. MacDonald, and B. Staznik. 1992. Persistence of hexazinone and metsulfuron-methyl in a mixed-wood/boreal forest lake. *J. Agric. Food Chem.* 40:1444-1449.
- Thompson, D. G., S. B. Homes, D. Thomas, L. MacDonald, and K. R. Solomon. 1993a. Impact of hexazinone and metsulfuron methyl on the phytoplankton community of a mixed-wood/boreal forest lake. *Environ. Toxicol. Chem.* 12:1695-1707.
- Thompson, D. G., S. B. Holmes, K. Wainio-Keizer, L. MacDonald, and K. R. Solomon. 1993b. Impact of hexazinone and metsulfuron methyl on the zooplankton community of a boreal forest lake. *Environ. Toxicol. Chem.* 12:1709-1717.
- Wan, M. T., R. G. Watts, and D. J. Moul. 1988. Evaluation of the acute toxicity to juvenile Pacific salmonids of hexazinone and its formulated products: Pronone 10G, Velpar^R L, and their carriers. *Bull. Environ. Contam. Toxicol.* 41:609-616.
- Wilkins, R. N., W. R. Marion, D. G. Neary, and G. W. Tanner. 1993. Vascular plant community dynamics following hexazinone site preparation in the lower Coastal Plain. *Can. J. For. Res.* 23:2216-2229.
- Zandvoort, R. 1989. Leaching of fluridone, hexazinone and simazine in sandy soils in the Netherlands. *Netherlands J. Agric. Sci.* 37:257-262.

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IMAZAPIC

Herbicide Basics

Chemical formula: (\pm)-2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-5-methyl-3-pyridinecarboxylic acid

Herbicide Family: Imidazolinone

Target Species: selected annual and perennial broadleaves and grasses

Forms: acid, ammonium salt

Formulations: SL, DG

Mode of Action: Inhibits the enzyme acetohydroxyacid synthase (AHAS), that is involved in the synthesis of aliphatic amino acids

Water Solubility: 2200 mg/L at 25° C

Adsorption potential: low

Primary degradation mech: microbial activity

Average Soil Half-life: 120 days

Mobility Potential: low

Dermal LD50 for rabbits:
>5,000 mg/kg

Oral LD50 for rats:
>5,000 mg/kg

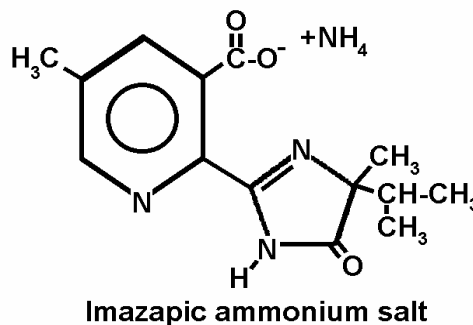
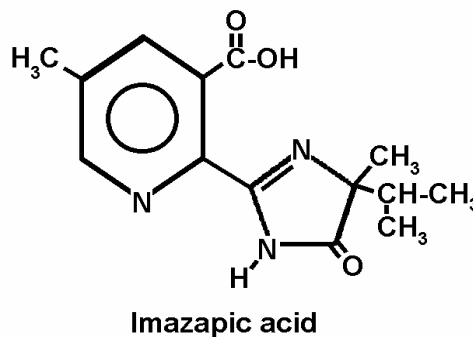
LC50 for bluegill sunfish:
>100 mg/L

Trade Names: Plateau[®], Cadre[®], Plateau Eco-Paks[®]

Manufacturer: BASF (previously American Cyanamid Company)

Synopsis

Imazapic is a selective herbicide for both the pre- and post-emergent control of some annual and perennial grasses and some broadleaf weeds. Imazapic kills plants by inhibiting the production of branched chain amino acids, which are necessary for protein synthesis and cell growth. It has been useful for weed control in natural areas, particularly in conjunction with the establishment of native warm-season prairie-grasses and certain legumes. Imazapic is relatively non-toxic to terrestrial and aquatic mammals, birds, and amphibians. Imazapic has an average half-life of 120 days in soil, is rapidly degraded by sunlight in aqueous solution, but is not registered for use in aquatic systems.



Herbicide Details

Chemical Formula: (\pm) -2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-5-methyl-3-pyridinecarboxylic acid

Trade Names: Imazapic (formerly called imazameth or AC 263,222) is sold under the trade names Plateau[®] and Cadre[®]. Both brands are sold as soluble liquid (SL) or dispersible granule (DG) formulations, and are also sold in pre-measured Plateau[®] or Cadre Eco-Paks[®] (just mix into water). Cadre[®] is manufactured for application in peanut crops; Plateau[®] is registered for wildland, pasture, and rangeland use.

Manufacturer: Plateau[®] and Cadre[®] are exclusively manufactured by BASF (previously by American Cyanamid Company, which was purchased by BASF in 2000).

Use Against Natural Area Weeds: Imazapic selectively kills plants depending on the species and the rate of application. It can control some annual and perennial broadleaves and grasses, including cocklebur (*Xanthium strumarium*), buffalobur (*Solanum rostratum*), Johnsongrass (*Sorghum halepense*), cheatgrass or downy brome (*Bromus tectorum*), bermudagrass (*Cynodon dactylon*), bahiagrass (*Paspalum nutatum*), smartweed (*Polygonum persicaria*), and leafy spurge (*Euphorbia esula*). In some instances, non-native weeds are more susceptible than the desirable native species, and imazapic has been used in prairie renovation and restoration projects. Beran et al. (1999) demonstrated that by controlling invasive non-native weeds, imazapic helped encourage the growth of certain native legumes. Washburn et al. (1999, 2000) and Washburn & Barnes (2000) used imazapic to sharply reduce the exotic tall fescue (*Festuca arundinacea*) and allow native warm-season grasses to return to Kentucky grasslands. Native species productivity and diversity, especially bird diversity, was promoted when imazapic was applied pre- and post-emergence at 0.2 kg/ha.

Masters et al. (1998) reported that a single autumn application of imazapic at 140 g/ha effectively controlled leafy spurge (*Euphorbia esula*) in Nebraska. Further, imazapic promoted the establishment of native prairie wildflowers in areas with high weed interference (Beran et al. 1999). In Iowa, Joy Williams (IA Dept. of Transportation) reports that imazapic applied at 8 to 12 oz/ac with 2 oz/ac methylated seed soil suppressed non-native Kentucky bluegrass and tall fescue. The Iowa Dept. of Transportation is interseeding native grasses to enhance restoration.

Jeff Connor of Rocky Mountain National Park reported a short-term decrease in above-ground stem densities of leafy spurge (50 to 90% reductions) in areas treated with imazapic (applied 3 times at 4 to 8 oz/ac), but the roots were not impacted, even after 2 years of application. Thus, two years post-application, leafy spurge stem densities returned to nearly pre-treatment levels. Jeff recommends that imazapic be used with a biocontrol agent for good leafy spurge control (J. Connor, pers. comm.).

On TNC preserves, imazapic is being used for leafy spurge control at Big Bluestem Prairie in western Minnesota. Anton Benson and Pete Baumann report good control (over 90%) of leafy spurge by using imazapic. They initially used imazapic applied at 8oz/ac, but were concerned by some apparent stunting of some native wet prairie plant species the growing season post-

application. They have since halved their application rate (to 0.5oz/gallon in a backpack solution), and have not noticed any stunting of the native vegetation. Anton adds that there is a relatively narrow application timeframe for good results. He recommends applying imazapic to leafy spurge during green-up following summer senescence, but a few weeks prior to killing frost. Leafy spurge can regenerate from seeds in the soil seedbank, so repeat applications will be necessary for long term control. Anton also adds that the use of imazapic can be used to facilitate the establishment of the biocontrol agents (*Aphthona* beetles) on leafy spurge.

Use Against Cropland and Rangeland Weeds: Imazapic has been used successfully to control cropland and rangeland weeds. In Australian rangelands, Melland & McLaren (1998) reported promising results using imazapic (applied at 0.048 kg ai/ha) to control serrated tussock (*Nassella trichotoma*). In croplands, Wilcut et al. (1999) reported that imazapic applied at 72 g ai/ha controlled Johnsongrass, crabgrass (*Digitaria sanguinalis*), redroot pigweed (*Amaranthus retroflexus*), sicklepod (*Senna obtusifolia*), and morningglory (*Ipomoea* spp) in corn (*Zea mays*) without any noticeable damage to the crop. Monks et al. (1998) also found that imazapic (at 0.064 lb ai/acre) controlled Johnsongrass in West Virginia. Abayo et al. (1998) mixed imazapic with imazapyr (45 g ae/ha + 27 g ae/ha) and successfully delayed witchweed (*Striga* spp.) emergence in corn.

Post-emergence application of imazapic (at 50 to 70 g ai/ha), combined with crop rotations of corn, peanut (*Arachis hypogaea*), and cotton (*Gossypium hirsutum*), resulted in the successful control of purple nutsedge (*Cyperus rotundus*) (Warren & Coble 1999). Imazapic has also been reported to control quackgrass (*Elytrigia repens*) and Canada thistle (*Cirsium arvense*) in corn (Sprague et al. 1999), red rice (*Oryza sativa*) and *Echinochloa* spp. in soybean (*Glycine max*) (Askew et al. 1998; Noldin et al. 1998), and Palmer amaranth (*Amaranthus palmeri*), eclipta (*Eclipta prostrata*), and pitted morningglory (*Ipomoea lacunosa*) in peanut (Grichar 1997a,b; Grey et al. 2000). Additionally, imazapic suppressed seedhead production in bahiagrass (*Paspalum notatum*) (Baker et al. 1999).

Mode of Action: Imazapic kills plants by inhibiting the activity of the enzyme acetohydroxy acid synthase (AHAS or ALS). ALS catalyzes the production of three branched-chain aliphatic amino acids, valine, leucine, and isoleucine, required for protein synthesis and cell growth. The rate of plant death is usually slow (several weeks), and is likely related to the amount of stored amino acids available to the plant. Only plants have ALS and produce these three amino acids, therefore, imazapic is of low toxicity to insects, fish, and other animals. Animals need these three branched chain aliphatic amino acids, but obtain them by eating plants or other animals.

Dissipation Mechanisms:

Summary: Imazapic is degraded primarily by soil microbial metabolism. The extent to which imazapic is degraded by sunlight is believed to be minimal when applied to terrestrial plants or soil, but it is rapidly degraded by sunlight in aqueous solutions. Imazapic is not degraded by other uncatalyzed chemical reactions in the environment. It is moderately persistent in soils, and has not been found to move laterally with surface water. Imazapic does not volatilize when applied in the field.

Volatilization

Imazapic is not volatile, and binds weakly to moderately with most soil types. Adsorption increases with decreasing soil pH and increasing clay and organic matter content (American Cyanamid 2000).

Photodegradation

Imazapic's half-life on soils due to photolysis is 120 days. In aqueous solutions however, imazapic is rapidly broken down by photolysis with a half-life of just one or two days (American Cyanamid 2000).

Microbial Degradation

The primary mechanism of degradation is via microbial action. Imazapic's half-life in soil ranges from 31 to 233 days depending upon soil characteristics and environmental conditions (American Cyanamid 2000).

Adsorption

Imazapic is weakly adsorbed in high pH soil. Adsorption increases as the pH decreases and with increasing clay and organic matter content. There is little lateral movement of imazapic in soil (American Cyanamid 2000).

Chemical Decomposition

Imazapic is not degraded by other abiotic chemical reactions in the environment (American Cyanamid 2000).

Behavior in the Environment

Summary: Imazapic is moderately persistent in soil, but has only limited mobility. It is soluble, but not degraded, in water. Imazapic is however, rapidly photodegraded by sunlight in aqueous solution. "Leakage" of imazapic from plant roots is unlikely.

Soil

Based on field dissipation studies, imazapic is moderately persistent in soils with a DT50 (time required for concentration in soil to reach 50% of initial measured concentration) of 7 to 150 days depending upon soil type and climatic conditions.

Imazapic has limited horizontal mobility in soil, and generally moves just 6 to 12 inches, although it can leach to depths of 18 inches in sandy soils (R. Lym, pers. comm.). Soil binding is a complex function of soil pH, texture, and organic matter content. Imazapic adsorption to soil may increase with time. Imazapic does not volatilize from the soil surface and photolytic breakdown on soils is negligible. The major route of imazapic loss from soil is through microbial degradation (WSSA 1994; American Cyanamid 2000).

Water

Imazapic is soluble in water and is not degraded hydrolytically in aqueous solution. Imazapic in water is, however, rapidly photodegraded by sunlight with a half-life of one to two days. Field

studies do not indicate any potential for imazapic herbicide to move from soils with surface water (American Cyanamid 2000). Imazapic is not registered for aquatic use.

Vegetation

Imazapic is readily absorbed through leaves, stems, and roots, and is then translocated rapidly throughout the plant, and accumulates in the meristematic regions. “Leakage” of imazapic from the roots of a treated plant to other nearby plants is unlikely because imazapic has great difficulty crossing the Casparian strip in roots (J. Vollmer, pers. comm.). Treated plants stop growing soon afterwards. Chlorosis appears first in the newest leaves, and tissue death spreads from these points. In perennials, imazapic is translocated into, and kills, underground storage organs which prevents regrowth. Chlorosis and tissue necrosis may not be apparent in some plant species for several weeks after application. Complete kill of plants may not occur for weeks or even months after application (American Cyanamid 2000).

Environmental Toxicity

Birds and Mammals

Imazapic is of low toxicity to birds and mammals. According to the manufacturer, imazapic does not bioaccumulate in animals, as it is rapidly excreted in urine and feces. It is therefore, essentially non-toxic to a wide range of non-target organisms, including mammals, birds, fish, aquatic invertebrates, and insects. The oral LD 50 of imazapic is >5,000 mg/kg for rats and >2,150 mg/kg for bobwhite quail, indicating that imazapic is relatively non-toxic by ingestion in single doses. In the 2 ASU (2 lb. a.e./gal) liquid formulation, imazapic is nonirritating to skin and eyes in single doses. The acute dermal LD50 was > 5,000 mg/kg (body weight) for rabbits, and imazapic is not a skin sensitizer for guinea pigs. Even direct application of imazapic technical (100% active ingredient) causes only minimal, transient eye irritation, and complete recovery occurs within 72 hours. The inhalation toxicity of imazapic technical is also very low. Chronic consumption of imazapic technical in the diet of mice for 18 months, and by rats for 2 years elicited no adverse health effects at the highest doses tested. Chronic consumption by dogs in a one-year study caused minimal effects, which included a slight degeneration/necrosis of single muscle fibers and lymphocyte/macrophage infiltration in skeletal muscle in both males and females, and slightly decreased serum creatinine in females only. No clinical observations indicative of muscle dysfunction were noted in any animal in these studies, and microscopic analyses indicated that the impact to muscle cells, would not impair or adversely affect the functional capacity of the affected skeletal muscles (American Cyanamid 2000).

Aquatic Species

Imazapic itself is of moderate toxicity to fish. The LC50s for technical grade imazapic for both bluegill sunfish (*Lepomis macrochirus*) and rainbow trout (*Oncorhynchus mykiss*) are >100 mg/L. Water fleas (*Daphnia magna*) also had an LC50 of >100 mg/L. Imazapic, however, rapidly degrades in aqueous solution, rendering it relatively safe to aquatic animals (American Cyanamid 2000).

Other Non-Target Organisms

The LC50 for honey bees (*Apis mellifera*) is >100 mg/bee, indicating that imazapic is nontoxic to bees (American Cyanamid 2000).

Application Considerations:

Imazapic can be applied using conventional application methods (sprayers, controlled drop, injectors, wipe-on devices, etc.). The manufacturer suggests using either a broadcast sprayer or using a spot treatment, with a backpack or other ground equipment.

Post-emergent imazapic applications require the use of a spray adjuvant, such as methylated seed oil (MSO) or vegetable oil concentrate. Nonionic and silicone-based surfactants may also be used, but are generally less effective. Nitrogen-based liquid fertilizers may also be applied with imazapic, but may increase injury to desired species.

Imazapic may be mixed with other herbicides such as triclopyr (Garlon[®]), glyphosate (RoundUp[®]), picloram (Tordon[®]), imazapyr (Arsenal[®]), or other products to provide total vegetation control. Mixtures of imazapic with 2,4-D and other phenoxy-type herbicides, however, provided less control of perennial grass weeds than imazapic alone. Combining imazapic with other herbicides, according to the manufacturer, should not increase the toxicological risk over that of either herbicide when used alone.

Safety Measures: Provide adequate ventilation and wear a respirator, rubber gloves, goggles, and protective clothing when handling. Remove contaminated clothing and launder prior to reuse. Shower after completion of work shift. Wash hands with soap and water before eating, smoking, or using the toilet. Store in a secure, dry, well-ventilated, separate room, building or covered area.

Human Toxicology: Imazapic is not considered carcinogenic. The U.S. EPA has classified imazapic as a “Group E” compound, or one that has not shown evidence of carcinogenicity in humans, based on studies with rats and mice (American Cyanamid 2000).

References

- Abayo, G.O., English, T., Eplee, R.E., Kanampiu, F.K., Ransom, J.K. and J. Gressel. 1998. Control of parasitic witchweeds (*Striga* spp.) on corn (*Zea mays*) resistant to acetolactate synthase inhibitors. *Weed Science* 46: 459-466.
- American Cyanamid Company. 2000. Plateau herbicide, for weed control, native grass establishment and turf growth suppression on roadsides and other noncrop areas., PE-47015. Parsippany, NJ.
- Askew, S.D., Street, J.E. and D.R. Shaw. 1998. Herbicide programs for red rice (*Oryza sativa*) control in soybean (*Glycine max*). *Weed Technology* 12: 103-107.
- Baker, R.D., McCarty, L.B., Colvin, D.L., Higgins, J.M., Weinbrecht, J.S. and J.E. Moreno. 1999. Bahiagrass (*Paspalum notatum*) seedhead suppression following consecutive yearly applications of plant growth retardants. *Weed Technology* 13: 378-384.
- Beran, D.D., Gaussoin, R.E. and R.A. Masters. 1999. Native wildflower establishment with imidazolinone herbicides. *HortScience* 34(2): 283-286.

- Beran, D.D., Masters, R.A. and R.E. Gaussoin. 1999. Grassland legume establishment with imazethapyr and imazapic. *Agronomy Journal* 91: 592-596.
- Grey, T.L., Bridges, D.C. and B.J. Brecke. 2000. Response of seven peanut (*Arachis hypogaea*) cultivars to sulfentrazone. *Weed Technology* 14: 51-56.
- Grichar, W.J. 1997a. Control of Palmer amaranth (*Amaranthus palmeri*) in peanut (*Arachis hypogaea*) with postemergence herbicides. *Weed Technology* 11: 739-743.
- Grichar, W.J. 1997b. Influence of herbicides and timing of application on broadleaf weed control in peanut (*Arachis hypogaea*). *Weed Technology* 11: 708-713.
- Masters, R.A., Beran, D.D. and F. Rivas-Pantoja. 1998. Leafy spurge (*Euphorbia esula*) response to AC 263,222. *Weed Technology* 12: 602-609.
- Melland, A. and D. McLaren. 1998. Efficacy of herbicides against serrated tussock (*Nassella trichotoma*) in a pot trial. *Plant Protection Quarterly* 13(2): 102.
- Monks, C.D., Vencill, W.K., Hatton, J.P., McFarland, M.L. and D.P. Delaney. 1998. Johnsongrass response to postemergence herbicides applied the previous year. *Journal of Production Agriculture* 11(4): 507-509.
- Noldin, J.A., Chandler, J.M., McCauley, G.N. and J.W. Sij, Jr. 1998. Red rice (*Oryza sativa*) and Echinochloa spp. control in Texas Gulf coast soybean (*Glycine max*). *Weed Technology* 12: 677-683.
- Sprague, C.L., Frasier, A.L. and D. Penner. 1999. Identifying acetolactate synthase inhibitors for potential control of quackgrass (*Elytrigia repens*) and Canada thistle (*Cirsium arvense*) in corn (*Zea mays*). *Weed Technology* 13: 54-58.
- Warren, L.S., Jr. and H.D. Coble. 1999. Managing purple nutsedge (*Cyperus rotundus*) populations utilizing herbicide strategies and crop rotation sequences. *Weed Technology* 13: 494-503.
- Washburn, B.E. and T.G. Barnes. 2000. Postemergence tall fescue (*Festuca arundinacea*) control at different growth stages with glyphosate and AC 263,222. *Weed Technology* 14: 223-230.
- Washburn, B.E., Barnes, T.G. and J.D. Sole. 1999. No-till establishment of native warm-season grasses in tall fescue fields. *Ecological Restoration* 17(3): 144-149.
- Washburn, B.E., Barnes, T.G. and J.D. Sole. 2000. Improving northern bobwhite habitat by converting tall fescue fields to native warm-season grasses. *Wildlife Society Bulletin* 28(1): 97-104.
- Wilcut, J.W., Richburg, J.S. III and F.R. Walls, Jr. 1999. Response of Johnsongrass (*Sorghum halepense*) and imidazolinone-resistant corn (*Zea mays*) to AC 263,222. *Weed Technology* 13: 484-488.
- WSSA. 1994. AC 263,222. *In: Herbicide Handbook, 7th Edition.* Weed Science Society of America, Champaign, Illinois. 352 pp.

Date Authored: April 2001

Updated: January 2004

IMAZAPYR

Herbicide Basics

Chemical formula: (\pm) -2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1H-imidazol-2-yl]-3-pyridinecarboxylic acid

Herbicide Family:

Imidazolinone

Target Species: grasses, broadleaves, vines, brambles, shrubs and trees, riparian and emerged aquatics

Forms: acid & salt

Formulations: SL, GR

Mode of Action: Amino acid synthesis inhibitor

Water Solubility: 11,272 ppm

Sorption potential: low

Primary degradation mech: Slow microbial metabolism and photolysis

Average Soil Half-life: 25-141 days

Mobility Potential: high

Dermal LD50 for rabbits: >2,000 mg/kg

Oral LD50 for rats: >5,000 mg/kg

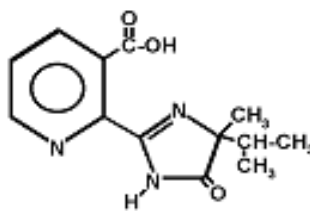
LC50 for bluegill sunfish: >100 mg/L

Trade Names: Arsenal[®], Habitat[®], Chopper[®], and Stalker[®]

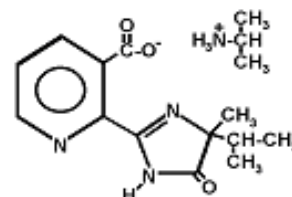
Manufacturer: BASF (previously American Cyanamid Company)

Synopsis

Imazapyr is a non-selective herbicide used for the control of a broad range of weeds including terrestrial annual and perennial grasses and broadleaved herbs, woody species, and riparian and emergent aquatic species. It controls plant growth by preventing the synthesis of branched-chain amino acids. Because imazapyr is a weak acid herbicide, environmental pH will determine its chemical structure, which in turn determines its environmental persistence and mobility. Below pH 5 the adsorption capacity of imazapyr increases and limits its movement in soil. Above pH 5, greater concentrations of imazapyr become negatively charged, fail to bind tightly with soils, and remain available (for plant uptake and/or microbial breakdown). In soils imazapyr is degraded primarily by microbial metabolism. It is not, however, degraded significantly by photolysis or other chemical reactions. The half-life of imazapyr in soil ranges from one to five months. In aqueous solutions, imazapyr may undergo photodegradation with a half-life of two days. Imazapyr is not highly toxic to birds and mammals, but some formulations (for instance, the inert ingredients in Chopper[®] and Stalker[®]) can cause severe, irreversible eye damage. Studies indicate imazapyr is excreted by mammalian systems rapidly with no bioaccumulation. It has a low toxicity to fish, and algae and submersed vegetation are not affected. Because imazapyr can affect a wide range of plants and can remain available, care must be taken during application to prevent accidental contact with non-target species. Further, a few studies have reported that imazapyr may be actively exuded from the roots of legumes (such as mesquite), likely as a defense mechanism by those plants. This exudate and the ability of imazapyr to move via intertwined root grafts may therefore adversely affect the surrounding desirable vegetation with little to no control of the target species.



Imazapyr acid



Imazapyr isopropylamine salt

Herbicide Details

Chemical Formula: (\pm) -2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5-oxo-1*H*-imidazol-2-yl]-3-pyridinecarboxylic acid

Trade Names: Arsenal[®], Chopper[®], and Stalker[®]. As of September 2003, imazapyr has received an EPA aquatic registration for Habitat[®].

Manufacturer: BASF (previously by American Cyanamid Company, which was purchased by BASF in 2000)

Use Against Natural Area Weeds: Imazapyr is a broad-spectrum herbicide that controls terrestrial annual and perennial grasses and broadleaved herbs, woody species, and riparian and emergent aquatic species. It can be used where total vegetation control is desired or in spot applications. Imazapyr is relatively slow acting, does not readily break down in the plant, and is therefore particularly good at killing large woody species. Imazapyr can control saltcedar (*Tamarix ramossissima*), privet (*Ligustrum vulgare*), blackberries (*Rubus* spp.), field bindweed (*Convolvulus arvensis*), bahiagrass (*Paspalum notatum*), and downy brome (*Bromus tectorum*) (American Cyanamid 1986). Caution should be used when applying imazapyr, as a few reports to TNC from the field indicate that imazapyr might be exuded from the roots of target species. Some legume species, such as mesquite, may actively exude imazapyr (J. Vollmer pers. comm.). Imazapyr herbicide can be mobile within roots and transferred between intertwined root systems (root grafts) of many different plants and/or to several species. Movement of imazapyr via root grafts or by exudates (which is a defense mechanism of those plants) may therefore adversely affect the surrounding vegetation. This movement of herbicide may also be compounded when imazapyr is incorrectly overapplied. Movement of soil particles that contains imazapyr can also potentially cause unintended damage to desirable species.

Imazapyr is effective for creating openings for wildlife use. It can be applied pre-emergent, but is most effective when applied as a post-emergent herbicide. Care should be taken in applying it around non-target species, as it is readily adsorbed through foliage and roots, and therefore, could be injurious by drift, runoff, or leaching from the roots of treated plants. To avoid injury to desirable trees, do not apply imazapyr within twice the drip line (tree canopy).

On TNC preserves in Texas, imazapyr provided good control of saltcedar (*Tamarix* spp.) and Chinese tallow tree (*Sapium sebiferum*). In North Carolina preserves, it was effective against oriental bittersweet (*Celastrus orbiculata*), cut-stumps of Chinese privet (*Ligustrum sinense*), and tree-of-heaven (*Ailanthus altissima*). Recent work in California demonstrated that foliar applications of imazapyr effectively controlled jubatagrass and pampasgrass (*Cortaderia jubata* and *C. selloana*) (DiTomaso et al. 1999; Drewitz 2000), and experimental studies in Washington showed that imazapyr provided excellent control of smooth cordgrass (*Spartina alterniflora*) in tidal estuarine habitats (Patten 2002).

Mode of Action: Imazapyr is absorbed quickly through plant tissue and can be taken up by roots. It is translocated in the xylem and phloem to the meristematic tissues, where it inhibits the enzyme

acetohydroxy acid synthase (AHAS), also known as acetolactate synthase (ALS). ALS catalyzes the production of three branched-chain aliphatic amino acids, valine, leucine, and isoleucine, required for protein synthesis and cell growth. The rate of plant death usually is slow (several weeks) and is likely related to the amount of stored amino acids available to the plant. Only plants have ALS and produce these three amino acids, and therefore, imazapyr is of low toxicity to animals (including fish and insects). Animals need these three branched chain aliphatic amino acids, but obtain them by eating plants or other animals.

Dissipation Mechanisms:

Summary: Imazapyr is degraded in soils primarily by microbial metabolism. It will quickly undergo photodegradation in aqueous solutions (photohydrolysis), but there is little to no photodegradation of imazapyr in soil, and it is not readily degraded by other chemical processes. Imazapyr does not bind strongly with soil particles, and depending on soil pH, can be neutral or negatively charged. When negatively charged, imazapyr remains available in the environment.

Volatilization

Imazapyr does not volatilize readily when applied in the field (T. Lanini, pers. obs.). The potential to volatilize, however, increases with increasing temperature, increasing soil moisture, and decreasing clay and organic matter content (Helling et al. 1971).

Photodegradation

Imazapyr is rapidly degraded by sunlight in aquatic solutions. In soils, however, there is little or no photodegradation of imazapyr (WSSA 1994). The half-life of imazapyr due to photodegradation in aqueous solution is approximately two days, and decreases with increasing pH (Mallipudi et al. 1991, Mangels 1991a).

Microbial Degradation

Microbial degradation is the primary mechanism of imazapyr degradation in soils (WSSA 1994). American Cyanamid (1986) reported that the half-life of imazapyr in soils typically ranged from one to seven months, depending on soil type, temperature, and soil moisture (Mangels 1991b). The half-life of imazapyr is shorter at cooler soil temperatures (25° C versus 35° C) and in sandier soils (sandy loam versus clay loam) (American Cyanamid 1986). Degradation rates are decreased in anaerobic soil conditions (WSSA 1994).

In studies of the related compound imazaquin, microbial degradation rates increased with increasing soil moisture content (between 5-75% of field capacity) and increasing soil temperatures (from 15° C to 30° C) (Mangels 1991b). Microbial degradation additionally, was more rapid in soils that did not bind the herbicide strongly. Imazapyr that is bound strongly to soil particles may be unavailable for microbial degradation.

Adsorption

The adsorption of imazapyr to soil particles is generally weak, but can vary depending on soil properties (Mangels 1991b). Adsorption is reversible, and desorption occurs readily (WSSA 1994). Because the exact chemical form of the herbicide is determined by environmental pH, the adsorption capacity of imazapyr changes with soil pH. A decline in pH below 5 increases

adsorption of imazapyr to soil particles. Above pH 5, imazapyr becomes ionized, increasing its negative charge, and limiting its ability to bind with soils (Mangels 1991b). Vizantinopoulos and Lolos (1994) found that adsorption decreased with increasing soil temperature, and Dickens and Wehtje (1986) found that adsorption increased with time and decreased soil moisture. In general, imidazolinone herbicides show an increase in soil adsorption capacity with an increase in soil clay content and organic matter, but studies of imazapyr have been conflicting (Dickens and Wehtje 1986, Wehtje et al. 1987, Mangels 1991b, McDowell et al. 1997, Pusino et al. 1997, El Azzouzi et al. 1998).

Chemical Decomposition

Imazapyr changes form readily with changes in pH, but is not necessarily degraded in this process. It does not readily undergo hydrolysis (Mangels 1991a), and no other chemical degradation mechanisms have been reported.

Behavior in the Environment

Summary: Imazapyr is slowly degraded by microbial metabolism and can be relatively persistent in soils. It has an average half-life in soils that range from one to five months. At pH above 5, it does not bind strongly with soil particles and can remain available (for plant uptake) in the environment. In water, imazapyr can be rapidly degraded by photolysis with a half-life averaging two days. There have been a few reports from the field of unintended damage to desirable, native plants when imazapyr has either exuded out of the roots of treated plants into the surrounding soil, or when intertwined roots transfer the herbicide to non-target plants. Make sure to not overapply imazapyr, and also confirm that soil particles with imazapyr are not moved in-contact with desirable species.

Soils

Depending on environmental conditions, imazapyr has an average half-life in soils of several months (Vizantinopoulos and Lolos 1994, El Azzouzi et al. 1998). El Azzouzi et al. (1998) reported half-lives between > 58 to 25 days in two Moroccan soils. In a laboratory study, the half-life of imazapyr ranged from 69-155 days, but factors affecting degradation rates were difficult to identify because the pH varied with temperature and organic content (McDowell et al. 1997). In a more extreme example, Vizantinopoulos and Lolos (1994) found that in loam and clay loam soils with pH 7-8, half-lives ranged up to 50 months. The manufacturer reports that persistence in soils is influenced by soil moisture, and that in drought conditions, imazapyr could persist for more than one year (Peoples 1984).

Lee et al. (1991) reported that imazapyr residues in soil following postemergent application increased eight days after initial application and continued to increase until a peak of 0.23 ppm at day 231 post-treatment. The authors attributed these increases to runoff of residues from plant surfaces following rainfall and to the release of residues from decaying plant matter.

Under most field conditions imazapyr does not bind strongly to soils and can be highly available in the environment. Above pH 5, the herbicide will take on an ionized form, increasing the risk of herbicide runoff. McDowell et al. (1997) found that heavy rainfall caused significant movement

of the herbicide (or more likely, moved the soil particles that the imazapyr was adsorbed to), and leaching up to 50 cm deep in soils have been reported (WSSA 1994).

Water

Despite its potential mobility, imazapyr has not been reported in water runoff, and we found no reports of imazapyr contamination in water. If it enters the water column, imazapyr can be photodegraded by sunlight with an average half-life of two days (Mallipudi et al. 1991).

Vegetation

Because imazapyr kills a wide variety of plants and can be relatively persistent and remain available in soils, damage to desirable non-target plants is possible. When imazapyr is applied in high rates, directly to soil, it can result in season-long soil activity. Plant species that are resistant to imazapyr apparently metabolize it to an immobile form that cannot be translocated to the meristematic tissues (Shaner & Mallipudi 1991).

Environmental Toxicity

Birds and Mammals

Imazapyr is of relatively low toxicity to birds and mammals. The LD50 for rats is > 5,000 mg/kg, and for bobwhite quail and mallard ducks is >2,150 mg/kg (WSSA 1994). American Cyanamid reports that studies with rats indicate that imazapyr was excreted rapidly in the urine and feces with no residues accumulating in the liver, kidney, muscle, fat, or blood (Miller et al. 1991). Imazapyr has not been found to cause mutations or birth defects in animals, and is classified by the U.S. EPA as a Group E compound, indicating that imazapyr shows no evidence of carcinogenicity.

Aquatic Species

Imazapyr is of low toxicity to fish and invertebrates. The LC50s for rainbow trout, bluegill sunfish, channel catfish, and the water flea (*Daphnia magna*) are all >100 mg/L (WSSA 1994). As of September 2003, imazapyr (tradename Habitat[®]) is registered for use in aquatic areas, including brackish and coastal waters, to control emerged, floating, and riparian/wetland species. A recent study from a tidal estuary in Washington showed that imazapyr, even when supplied at concentrations up to 1600 mg/L, did not affect the osmoregulatory capacity of Chinook salmon smolts (Patten 2003). Similarly, the Washington State Department of Agriculture reported that the 96-hour LC50 for rainbow trout fry to be 77,716 mg/L (ppm) -22,305 ppm of the active ingredient- which represents a greater concentration of imazapyr than found in commercially-sold containers (J. Vollmer, pers. comm.).

Other Non-Target Organisms

Limited information was found on the effects of imazapyr on other non-target organisms such as soil bacteria and fungi. The manufacturers report that Arsenal[®] is non-mutagenic to bacteria (Peoples 1984).

Application Considerations:

Imazapyr is a slow acting herbicide that is not readily metabolized in plants. It can be very effective against woody species. Due to its persistence in the environment, it may be preferable to

apply imazapyr directly to vegetation (using a low-volume backpack, cut-stump, or basal bark application) instead of using a broadcast spray method. When using a cut-stump application, be careful to avoid overapplication of imazapyr on the stump, as this may lead to excess imazapyr to be transferred between root grafts or movement by soil particles. When completing a cut-stump treatment, apply imazapyr only to the outer cambium layer of the stump (versus applying herbicide to the entire cut-stump), and this should sufficiently kill the tree (J. Vollmer, pers. comm.).

A study of wipe-on applications to the reed *Phragmites australis*, however, found that this method provided some suppression of reeds in the short-term, but failed to control them in the long term (Kay 1995). Malefy and Quakenbush (1991) reported better results when imazapyr was applied at 21° C rather than 32° C. Rainfall is considered important for good activity following soil application (Malefy and Quakenbush 1991) but can increase movement of imazapyr in the soil column. A non-ionic surfactant can improve the efficacy of imazapyr.

Safety Measures:

Some formulations of imazapyr can cause severe irreversible eye damage. Care should be taken to prevent accidental splashing or other exposure of eyes to the herbicide.

Human Toxicology

Imazapyr is of relatively low toxicity to mammals, and shows no mutagenic or teratogenic potential. It can be an eye and skin irritant, but is not a dermal sensitizer (American Cyanamid 1986; Cyanamid Ltd. 1997).

References

- American Cyanamid. 1986. Arsenal herbicide: technical report. American Cyanamid Agricultural Division.
- Cyanamid, Ltd. 1997. Summary of toxicity studies on imazapyr. *Journal of Pesticide Science* 22: 360-364.
- Dickens, R. and G. Wehtje. 1986. Mobility and soil solution characteristics of imazapyr (Arsenal) and sulfometuron methyl (Oust) in Alabama soils. *Proc. South. Weed Sci. Soc.* 39:368.
- DiTomaso, J., E. Healy, C.E. Bell, J. Drewitz, and A. Tschohl. 1999. Pampasgrass and jubatagrass threaten California coastal habitats. CalEPPC-UC WeedRIC leaflet #99-1.
- Drewitz, J.J. 2000. Reproductive biology and control of jubatagrass (*Cortaderia jubata*). Master's Thesis, University of California, Davis.
- El Azzouzi, M., A. Dahchour, A. Bouhaouss, and M. Ferhat. 1998. Study on the behavior of imazapyr in two Moroccan soils. *Weed Res.* 38:217-220.
- Helling, C. S., P. C. Kearney, and M. Alexander. 1971. Behavior of pesticides in soil. *Adv. Agron.* 23:147-240.
- Lanini, T. 2001. Personal Communication. Department of Vegetable Crops & Weed Sciences, University of California at Davis.
- Lee, A., P. E. Gatterdam, T. Y. Chiu, N. M. Mallipudi, and R. Fiala. 1991. Plant metabolism. Chpt 11 in *The Imidazolinone Herbicides*, D. L. Shaner and S. L. O'Connor, eds. CRC Press. Boca Raton, FL. 290 pgs.

- Malefy, T. and L. S. Quakenbush. 1991. Influences of environmental factors on the biological activity of the imidazolinone herbicides. Chpt. 8 in *The Imidazolinone Herbicides*, D. L. Shaner and S. L. O'Connor, eds. CRC Press. Boca Raton, FL. 290 pgs.
- Mallipudi, N. M., S. J. Stout, A. R. daCunha, and A. Lee. 1991. Photolysis of imazapyr (AC 243997) herbicide in aqueous media. *J. Agric. Food Chem.* 39(2):412-417.
- Mangels, G. 1991a. Behavior of the imidazolinone herbicides in the aquatic environment. Chpt 15 in *The Imidazolinone Herbicides*, D. L. Shaner and S. L. O'Connor, eds. CRC Press. Boca Raton, FL. 290 pgs.
- Mangels, G. 1991b. Behavior of the imidazolinone herbicides in soil – a review of the literature. Chpt 16 in *The Imidazolinone Herbicides*, D.L. Shaner and S. L. O'Connor, eds. CRC Press. Boca Raton, FL. 290 pgs.
- McDowell, R. W., L. M. Condron, B. E. Main, and F. Dastgheib. 1997. Dissipation of imazapyr, flumetsulam and thifensulfuron in soil. *Weed Res.* 37:381-389.
- Miller, P., C. H. Fung, and B. Gingher. 1991. Animal metabolism. Chpt 12 in *The Imidazolinone Herbicides*, D.L. Shaner and S. L. O'Connor, eds. CRC Press. Boca Raton, FL. 290 pgs.
- Patten, K. 2002. Smooth cordgrass (*Spartina alterniflora*) control with imazapyr. *Weed Technology* 16: 826-832.
- Patten, K. 2003. Persistence and non-target impact of imazapyr associated with smooth cordgrass control in an estuary. *Journal of Aquatic Plant Management* 41: 1-6.
- Peoples, T. R. 1984. Arsenal herbicide (AC 252,925): a development overview. *Proc. South. Weed Sci. Soc.* 37:378-387.
- Pusino, A., S. Petretto, and C. Gessa. 1997. Adsorption and desorption of imazapyr by soil. *J. Agric. Food Chem.* 45:1012-1018.
- Shaner, D. L. and N. M. Mallipudi. 1991. Mechanisms of selectivity of the imidazolinone herbicides. Chpt 7 in *The Imidazolinone Herbicides*, D.L. Shaner and S. L. O'Connor, eds. CRC Press. Boca Raton, FL. 290 pgs.
- Vizantinopoulos, S., and P. Lolos. 1994. Persistence and leaching of the herbicide imazapyr in soil. *Bull. Environ. Contam. Toxicol.* 52:404-410.
- Vollmer, J. 2003. Personal Communication. BASF Ecological Restoration Specialist.
- WSDA. No date. Washington State Department of Agriculture Pesticide Fact Sheet, available at: <http://agr.wa.gov/PlantsInsects/Weeds/Imazapyr/docs/ImazapyrFactSheet.pdf>. Accessed April 14, 2004.
- WSSA. 1994. *Herbicide handbook*. Weed Society of America. Champaign, Illinois. 352 pp.

Date Authored: April 2001

Updated: June 2004

PICLORAM

Herbicide Basics

Chemical formula: 4-amino-3,5,6-trichloro-2-pyridinecarboxylic acid

Herbicide Family:

Pyridine (Picolinic acid)

Target Species: broadleaf herbs, vines, and woody plants, esp. leafy spurge

Forms: salt, & ester

Formulations: SL, EC

Mode of Action: Auxin mimic

Water Solubility: 430 ppm

Sorption potential: low

Primary degradation mech:

Microbial and chemical degradation

Average Soil Half-life:

90 days

Mobility Potential: high

Dermal LD50 for rabbits:

>2,000 mg/kg

Oral LD50 for rats:

>5,000 mg/kg

LC50 for bluegill sunfish:

>14.5 mg/L

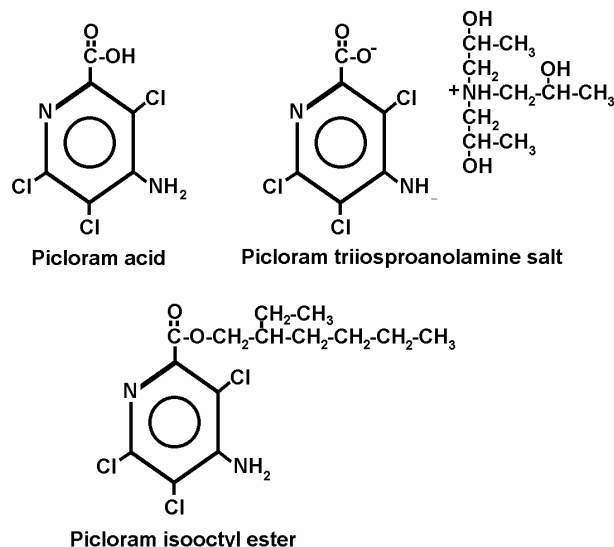
Trade Names: Grazon[®], Tordon[®], Access[®], and Pathway[®]

Manufacturer:

Dow AgroSciences

Synopsis

Picloram kills or damages annual and perennial broadleaf herbs and woody plants. It acts as an “auxin mimic” or synthetic growth hormone that causes uncontrolled and disorganized growth in susceptible plants. Picloram does not bind strongly with soil particles and is not degraded rapidly in the environment, allowing it to be highly mobile and persistent (half-life of picloram in soils can range from one month to several years). In soils, picloram is degraded primarily by microbial metabolism, but it can be degraded by sunlight when directly exposed in water or on the surface of plants or soil. Picloram can move off-site through surface or subsurface runoff and has been found in the groundwater of 11 states. Picloram may also “leak” out of the roots of treated plants, and be taken up by nearby, desirable species. Picloram is not highly toxic to birds, mammals, and aquatic species. Some formulations are highly toxic if inhaled, while other formulations can cause severe eye damage if splashed into the eyes. Because of the persistence of picloram in the environment, chronic exposure to wildlife is a concern, and studies have found weight loss and liver damage in mammals following long term exposure to high concentrations. Concentrations in runoff reported by researchers are often adequate to prevent the growth of non-target terrestrial and aquatic plants, and therefore, picloram should not be applied near waters used for irrigation.



Herbicide Details

Chemical Formula: 4-amino-3,5,6-trichloro-2-pyridinecarboxylic acid

Trade Names: Grazon PC[®], Tordon K[®], and Tordon 22K[®] are picloram salt formulations. Access[®] is mix of triclopyr and picloram esters. Grazon P+D[®], Tordon RTU[®], and Pathway[®] are mixtures of picloram and 2,4-D salts.

Manufacturer: Dow AgroSciences is the primary manufacturer of picloram.

Use Against Natural Area Weeds: Picloram is a dicot-selective, persistent herbicide used to control a variety of annual and perennial broadleaved herbs and woody species. It can persist in an active form in the soil from several months to years, and can also be released from the roots of treated plants into the soil, where other non-target species may take it up and be injured or killed (Hickman et al. 1989). The cut-stump treatment is typically used to control woody species. Examples of weeds successfully controlled using picloram include: leafy spurge (*Euphorbia esula*), knapweeds (*Centaurea* spp.), toadflax (*Linaria vulgaris*), buckthorns (*Rhamnus* spp.), and Russian olive (*Elaeagnus angustifolia*).

On TNC preserves in Idaho, Minnesota, Montana, and Oregon, picloram has been used successfully against cinquefoil (*Potentilla recta*), several knapweed and thistle species (*Acroptilon repens*, *Centaurea maculosa*, *C. diffusa*, *C. solstitialis*), toadflax (*Linaria vulgaris*), birdsfoot trefoil (*Lotus corniculatus*) and hoary cress (*Cardaria draba*). In each case, control results were reported as good to excellent. Dave Carr and Dave Hannah (unpublished data) found that native forb densities on plots treated with picloram up to five years earlier at TNC's Pine Butte Swamp preserve in Montana, were significantly lower than forb densities on untreated plots. In general, picloram does not harm grasses, but repetitive treatments and higher application rates can negatively damage native grasses (Clint Miller, personal observation).

Because of its picloram's persistence in the environment, it is often the herbicide of choice for controlling leafy spurge (*Euphorbia esula*). Leafy spurge is not effectively controlled by other less persistent herbicides. Natural area managers and researchers have found picloram to be more effective against leafy spurge than glyphosate, 2,4-D or triclopyr. It is usually regarded, however, as adequate only to contain or reduce spurge infestations, and generally cannot eliminate them. It effectively top-kills leafy spurge but often fails to adequately damage the deep, extensive root systems. Imazapic (sold as Plateau[®]), a relatively new herbicide, is being tested against leafy spurge and may prove to be more effective than picloram in some circumstances.

Picloram is often sold mixed with 2,4-D, and this formulation has also been used in natural areas against herbaceous species including leafy spurge and spotted knapweed (*Centaurea maculosa*), and in cut-stump treatments against a variety of woody species, particularly in prairie preserves. It was reportedly effective when immediately applied to cut stumps of glossy and common buckthorn (*Rhamnus frangula*, *R. cathartica*), Russian olive (*Elaeagnus angustifolia*) and Siberian elm (*Ulmus pumila*) on Tallgrass Prairie Preserves in

western Minnesota. It was ineffective and allowed resprouting when used on the native redbud (*Cercis canadensis*).

Mode of Action: Picloram is an “auxin mimic” or synthetic auxin. This type of herbicide kills susceptible plants by mimicking the plant growth hormone auxin (indole acetic acid), and when administered at effective doses, causes uncontrolled and disorganized plant growth that leads to plant death. The exact mode of action of picloram and other auxin-mimic herbicides have not been fully described, but it is believed to acidify the cell wall, which loosens the cell wall and allows cell elongation. Low concentrations of picloram can stimulate RNA, DNA, and protein synthesis leading to uncontrolled cell division and growth, and, ultimately, vascular tissue destruction. High concentrations of picloram can inhibit cell division and growth.

Dissipation Mechanisms:

Summary: Picloram is metabolized slowly by microbes and can be degraded through photolysis when directly exposed to sunlight. The half-life of picloram in soils can vary from one month to three years depending on soil and climate conditions. Other methods of chemical degradation do not occur readily. Picloram does not bind strongly with soils and can be highly mobile, moving to soil depths of two meters and laterally to one km. It is not highly volatile.

Volatilization

Picloram does not volatilize readily when applied in the field (T. Lanini, pers. obs.). The potential to volatilize, however, increases with increasing temperature, increasing soil moisture, and decreasing clay and organic matter content (Helling et al. 1971).

Photodegradation

Picloram is readily degraded when exposed to sunlight in water or on the surface of plant foliage and soils (Merkle et al. 1967; Johnsen & Martin 1983; Cessna et al. 1989; Woodburn et al. 1989). Photodegradation will occur most rapidly in clear, moving water (WSSA 1994), and slowly when exposed on the soil surface. Merkle et al. (1967) reported 15% degradation of picloram after one-week exposure on soil, compared to 65% from exposure in an aqueous solution. There is some evidence that photodegradation occurs more rapidly at higher elevations (Johnsen & Martin 1983) possibly due to increased UV radiation (Merkle et al. 1967). Photolysis of picloram results in the generation of at least two organic acid photoproducts (Woodburn et al. 1989).

Microbial Degradation

Although microbial degradation of picloram is generally slow, it is believed to be the major pathway of picloram degradation in soils (Spiridonov et al. 1987; WSSA 1994). The primary metabolites produced during microbial degradation are degraded through microbial metabolism more rapidly than the parent compound (WSSA 1994). Conditions that favor microbial activity such as high soil moisture and temperature can increase the rate of microbial degradation of picloram (Merkle et al. 1967; Phillips & Feltner 1972, Michael et al. 1989; Watson et al. 1989).

Adsorption

Picloram has a very low adsorption capacity in most soil types ($K_{oc}=16$ mL/g). High organic content, heavy soil texture, low pH, high hydrated iron and aluminum oxide contents, and low soil temperature can increase the adsorption capacity (Merkle et al. 1967; Farmer & Aochi 1974; Neary et al. 1985; Liu et al. 1997). Rates of adsorption also increased with time (McCall & Agin 1985). Unlike many other herbicides, however, clay content does not affect the adsorption capacity of picloram (Grover 1971; Farmer & Aochi 1974).

Chemical Decomposition

Hance (1967) determined that the half-life of picloram due to chemical degradation alone is between 9 and 116 years. Non-biological chemical degradation is therefore not regarded as an important process in the dissipation of picloram from soil (Hance 1967).

Behavior in the Environment

Summary: Picloram is water-soluble, does not bind strongly to soil particles, and can be persistent and mobile in the environment. In plants, picloram is either metabolized (in non-susceptible species) or can remain intact for some time (in susceptible species). Unabsorbed picloram may photodegrade or be washed-off by rainfall. Absorbed picloram may be released into soil by passive transport in plant roots, and can then be taken up by nearby plants.

Soils

Picloram is not readily degraded in soils and can be persistent and mobile. Estimates of the persistence of potentially toxic concentrations vary from a few months to three years, depending on soil and environmental conditions (Scrifres et al. 1972; Fryer et al. 1979; Johnsen 1980; Norris et al. 1982; Neary et al. 1985; Smith et al. 1988; Bovey & Richardson 1991; Close et al. 1998). In soils where picloram persists for long periods of time, it has high potential to move vertically and horizontally, which can lead to contamination of water sources and non-target (terrestrial and aquatic) sites. Smith et al. (1988) reported that one and two years after treating a site with 3.38 kg/ha of picloram, residues were found in the soils and groundwater of an untreated site one km away.

Differences in the half-life of picloram between soil types are difficult to compare because the rate of degradation of picloram varies with time (Fryer et al. 1979). A half-life calculated from residues measured shortly after application will tend to be significantly shorter than those calculated from data collected several months after application (Deubert & Corte-Real 1986). In general, reports of the half-life of picloram vary from less than a month to more than three years (Deubert & Corte-Real 1986; WSSA 1994; Close et al. 1998). In addition, degradation rates can vary with soil depth. Soils where picloram can leach to deep layers may retain picloram for significantly longer periods, probably because significantly smaller microbial populations reside at lower soil depths. Close et al. (1998) used an herbicide movement and persistence model to estimate a half-life of 203 days for the top 30 cm and 986 days for the 30-70 cm layer of silt loam soils in New Zealand.

The mobility of picloram in soils is determined by the adsorption capacity of the soil, soil moisture, and post-application rainfall (Smith et al. 1988). In heavy textured soils with a high organic content that can bind the herbicide, picloram tends to remain in the top 30 cm (Merkle et

al. 1967; Jotcham et al. 1989). In sandy soils or soils with cracks and fissures that allow it to flow to lower depths, picloram has been found more than two meters deep (Phillips & Feltner 1972). Rainfall following application will aid horizontal and vertical movement of picloram in the soil (Merkle et al. 1967; Smith et al. 1988).

Water

Because picloram is water-soluble and does not bind strongly to soil, it is capable of moving into local waterways through surface and subsurface runoff (Michael et al. 1989). The extent to which picloram enters a waterway depends largely on the type of soil, rates of application, rainfall received post-application, and distance from point of application to nearest water body or groundwater (Trichell et al. 1968; Baur et al. 1972; Mayeux et al. 1984). In general, the larger the buffer between treated sites and surface water bodies or groundwater, the smaller the potential for water contamination. Picloram runoff from sites treated with aqueous spray and those treated with pellets (pellets are no longer available in the U.S.) at the same rate does not differ significantly (Bovey et al. 1978). Once in a waterway, picloram may be degraded through photolysis, especially in clear and moving water. Woodburn et al. (1989) found the half-life of picloram in water was 2 to 3 days.

Maximum herbicide runoff generally occurs following the first significant rainfall, after which runoff concentrations drop to lower levels that can persist for up to two years post-application (Scrifres et al. 1971; Johnsen 1980; Mayeux et al. 1984; Michael et al. 1989). Concentrations of 50 ppb are enough to prevent the growth of leafy spurge and concentrations of <1 ppb will inhibit the growth of many common food crops. Runoff concentrations of >1 ppb are common following the application of picloram at recommended rates even under low-runoff conditions (Baur et al. 1972; Bovey et al. 1978; Mayeux et al. 1984; Neary et al. 1985; Michael et al. 1989; Bovey & Richardson 1991). Groundwater concentrations of 0.01-49 micrograms/L have been reported in 11 states (EXTOXNET 1996), and Smith et al. (1988) found picloram in groundwater one km off-site and 35 months following application of 3.38 kg/ha of picloram.

Most researches have concluded that picloram runoff concentrations are not great enough to be a hazard to aquatic species. These concentrations however, could damage crops if used for irrigation, and have been shown to cause damage to the submersed macrophytes *Potamogeton pectinatus* L. and *Myriophyllum sibiricum* Komarov (Forsyth et al. 1997). Because many studies were conducted under laboratory conditions, it is difficult to draw conclusions regarding the impact of picloram contamination in wildland aquatic systems.

Vegetation

In non-susceptible species such as grasses, picloram is metabolized rapidly, while in susceptible species, picloram can remain intact for extended periods (WSSA 1994). When applied to soil, picloram is readily absorbed by plant roots. When applied to foliage, the majority of picloram (70-90%) remains in the leaves and only a small percentage is conducted to stems and roots (Meikle et al. 1966; Cessna et al. 1989; Hickman et al. 1990). Unabsorbed picloram remaining on leaf surfaces may photodegrade in sunlight or be washed off with rainfall or irrigation. Picloram absorbed by plants can be released into the soil by passive transport through the roots and then taken up by roots of other nearby plants (Hickman et al. 1990). Therefore, even

selective application of picloram to specific target plants could potentially harm nearby desirable plants.

Environmental Toxicity

Birds and Mammals

Picloram is “slightly to practically nontoxic” to birds and mammals. The LD50 for rats is >5,000 mg/kg (WSSA 1994). For bobwhite quail and mallard duck the LD50s are >5,000 and >2,510 mg/kg, respectively. However, because of the long-term persistence of picloram in the environment, chronic exposure of wildlife to picloram is of concern. John-Greene et al. (1985) evaluated the potential effects of chronic picloram exposure in New Zealand white rabbits and concluded that there was weight loss in rabbits receiving 200-400 mg/kg/day, but no embryotoxic or teratogenic responses occurred. Liver damage has also been associated with chronic exposure, but only at very high doses that would not be expected from normal pesticide application (EXTOXNET 1996).

Aquatic Species

Picloram is “slightly to moderately toxic” to aquatic species (EXTOXNET 1996). The LC50 (96 hours) for rainbow trout, bluegill sunfish, and fathead minnow are 19.3 mg/L, 14.5 mg/L, and 55 mg/L, respectively (EXTOXNET 1996). These values are above the peak runoff concentrations reported by researchers under various environmental conditions (Baur et al. 1972; Bovey et al. 1978; Neary et al. 1979; Johnsen 1980; Mayeux et al. 1984; Lym & Messersmith 1988; Smith et al. 1988; Michael et al. 1989; Bovey & Richardson 1991). Mayes et al. (1987) evaluated the toxicity of picloram to rainbow trout life stages and concluded that picloram is not an acute or chronic hazard to aquatic species when used as directed. Gersch et al. (1985) evaluated the acute and chronic toxicity of picloram to the aquatic invertebrate *Daphnia magna*, and found an LC50 (48 hours) of 68.3 mg/L. The authors concluded that these findings corroborated the “low toxicity” rating of picloram to wildlife and aquatic species (Gersch et al. 1985).

Other Non-Target Organisms

Breazeale and Camper (1972) evaluated the effects of picloram on three soil microbes. Picloram had no effect on two species, *Erwinia carotovora* and *Bacillus* sp., but inhibited growth in *Pseudomonas fluorescens* by 28.8%. Experiments by Dow AgroSciences concluded that picloram does not bioaccumulate in organisms, reducing the potential that it could be passed through the food chain to various animals including humans (Mullison 1985).

Application Considerations:

Brian Winter, TNC land steward in western Minnesota, recommends mixing a dye (e.g., Highlighter[®]) with the picloram formulation for use against leafy spurge, so treated and missing areas are easy to spot. Treated areas should be checked for regrowth in June and individual spots sprayed with a backpack sprayer. An additional benefit of using a dye is that the chance of overspray is minimized.

Safety Measures:

Picloram acid and its derivatives can be highly toxic if inhaled. Severe eye damage can also be caused by picloram. Product labels and Material Safety Data Sheets should be thoroughly reviewed prior to use and all precautionary measures followed to prevent dangerous exposure.

Human Toxicology:

As with all herbicides, applicators are at greatest risk of exposure to potential toxicants. Libich et al. (1984) reported that workers applying picloram on electric right-of-ways with hand-held spray guns were exposed to airborne residues of <0.2-10.5 micrograms per cubic meter of air. These workers later excreted <0.01-1.30 mg of picloram for every kilogram of body weight through their urine (Libich et al. 1984).

In a study of the fate of picloram in humans, six volunteers were given picloram either orally or dermally at 0.5 or 5.0 mg/kg of body weight. Study results found that picloram was absorbed rapidly through the gastrointestinal tract when ingested but passed through skin slowly with dermal exposure (Stevens & Sumner 1991). After 72 hours over 90% of the ingested picloram was passed through unchanged in the urine. The volunteers reported no adverse effects (Stevens & Sumner 1991).

The Suggested No-Adverse-Response Level for picloram recommended by the National Research Council's Safe Drinking Water Committee is 1.05 ppm (Mullison 1985). At least one study found picloram runoff concentrations in excess of this amount (2.3-3.3 ppm) when applied as pellets at the rate of 2.24 kg/ha to soils of the Blackland Prairie in Texas (Bovey et al. 1978). The pellet formulation of picloram, however, is no longer available in the U.S. Picloram and its derivatives can be highly toxic when inhaled and can cause severe eye damage. EPA classified picloram as a "Group E" compound, or a chemical that has not shown evidence of carcinogenicity in humans (EPA 1995).

References

- Baur, J. R., R. W. Bovey, and M. G. Merkle. 1972. Concentration of picloram in runoff water. *Weed Sci.* 20(4):309-313.
- Bovey, R. W., and C. W. Richardson. 1991. Organic chemicals in the environment. *J. Environ. Qual.* 20:528-531.
- Bovey, R. W., C. Richardson, E. Burnett, M. G. Merkle, and R. E. Meyer. 1978. Loss of spray and pelleted picloram in surface runoff water. *J. Environ. Qual.* 7(2):178-180.
- Breazeale, F. W., and N. D. Camper. 1972. Effect of selected herbicides on bacterial growth rates. *Appl. Microbiol.* 23(2):431-432.
- Cessna, A. J., and J. Waddington, and S. Bittman. 1989. Residues of 2,4-D and picloram in aspen poplar and soil after application with a roller. *Can. J. Plant Sci.* 69:205-212.
- Close, M. E., L. Pang, J. P. C. Watt, and K. W. Vincent. 1998. Leaching of picloram, atrazine and simazine through two New Zealand soils. *Geoderma* 84:45-63.
- Deubert, K. H., and I. Corte-Real. 1986. Soil residues of picloram and triclopyr after selective foliar application on utility rights-of-way. *J. Arbori.* 12(11):269-272.

- E.P.A. 1995. Picloram. R.E.D. Facts. Prevention, Pesticides and Toxic Substances. EPA-738-F-95-018.
- EXTOXNET. 1996. Picloram. Pesticide Information Profiles. Extension Toxicology Network. <http://ace.orst.edu/info/extoxnet/>.
- Farmer, W. J., and Y. Aochi. 1974. Picloram sorption by soils. *Proc. Soil Sci. Soc. Amer.* 38:418-423.
- Forsyth, D. J., P. A. Martin, and G. G. Shaw. 1997. Effects of herbicides on two submersed aquatic macrophytes, *Potamogeton pectinatus* L. and *Myriophyllum sibiricum* Komarov, in a prairie wetland. *Environ. Pollut.* 95:259-268.
- Fryer, J. D., P. D. Smith, and J. W. Ludwig. 1979. Long-term persistence of picloram in a sandy loam soil. *J. Environ. Qual.* 8(1):83-86.
- Gersich, F. M., D. L. Hopkins, and D. P. Milazzo. 1985. Acute and chronic toxicity of technical picloram (4-amino-3,5,6-trichloropicolinic acid) to *Daphnia magna* Straus. *Bull. Environ. Contam. Toxicol.* 35:121-126.
- Grover, R., and G. G. Bowes. 1981. Picloram residue levels for the control of leafy spurge regrowth. *Can. J. Plant. Sci.* 61:661-664.
- Hance, R. J. 1967. Decomposition of herbicides in the soil by non-biological chemical processes. *J. Sci. Food Agric.* 18:544-547.
- Helling, C. S., P. C. Kearney, and M. Alexander. 1971. Behavior of pesticides in soil. *Adv. Agron.* 23:147-240.
- Hickman, M. V., C. G. Messersmith, and R. G. Lym. 1990. Picloram release from leafy spurge roots. *J. Range Manage.* 43(5):442-445.
- Johnsen, T. N., Jr. 1980. Picloram in water and soil from a semiarid pinyon-juniper watershed. *J. Environ. Qual.* 9(4):601-605.
- Johnsen, T. N., Jr., and R. D. Martin. 1983. Altitude effects on picloram disappearance in sunlight. *Weed Sci.* 31:315-317.
- John-Greene, J. A., J. H. Ouellette, T. K. Jeffries, K. A. Johnson, and K. S. Rao. 1985. Teratological evaluation of picloram potassium salt in rabbits. *Food Chem. Toxic.* 23(8):753-756.
- Jotcham, J. R., D. W. Smith, and G. R. Stephenson. 1989. Comparative persistence and mobility of pyridine and phenoxy herbicides in soil. *Weed Tech.* 3:155-161.
- Libich, S., J. C. To, R. Frank, G. J. Sirons. 1984. Occupational exposure of herbicide applicators to herbicides used along electric power transmission line right-of-way. *Am. Ind. Hyg. Assoc. J.* 45(1):56-62.
- Liu, L., J. A. Dumas, and C. L. Cacho. 1997. Picloram groundwater contamination from pasture use. *J. Agric. Univ. P. R.* 81(3-4):211-218.
- Lym, R. G., and C. G. Messersmith. 1988. Survey for picloram in North Dakota groundwater. *Weed Tech.* 2:217-222.
- Mayeux, H. S., Jr., C. W. Richardson, R. W. Bovey, E. Burnett, M. G. Merkle, and R. E. Meyer. 1984. Dissipation of picloram in storm runoff. *J. Environ. Qual.* 13(1):44-49.
- Mayes, M. A., D. L. Hopkins, and D. C. Dill. 1987. Toxicity of picloram (4-amino-3,5,6-trichloropicolinic acid) to life stage of the rainbow trout. *Bull. Environ. Contam. Toxicol.* 38:653-660.
- McCall, P. J., and G. L. Agin. 1985. Desorption kinetics of picloram as affected by residence time in the soil. *Environ. Toxicol. Chemistry* 4:37-44.

- Meikle, R. W., E. A. Williams, and C. T. Redemann. 1966. Metabolism of Tordon herbicide (4-amino-3,5,6-trichloropicolinic acid) in cotton and decomposition in soil. *J. Agr. Food Chem.* 14(4):384-387.
- Merkle, M. G., R. W. Bovey, and F. S. Davis. 1967. Factors affecting the persistence of picloram in soil. *Agron. J.* 59:413-415.
- Michael, J.L., D. G. Neary, and M. J. M. Wells. 1989. Picloram movement in soil solution and streamflow from a coastal plain forest. *J. Environ. Qual.* 18:89-95.
- Mullison, W. R. 1985. A toxicological and environmental review of picloram. *Proc. West Soc. Weed Sci.* 38:21-92.
- Neary, D. G., P. B. Bush, J. E. Douglass, and R. L. Todd. 1985. Picloram movement in an Appalachian hardwood forest watershed. *J. Environ. Qual.* 14(4):585-592.
- Neary, D. G., J. E. Douglass, and W. Fox. 1979. Low picloram concentrations in streamflow resulting from forest application of Tordon-10K. *Proc. South. Weed Sci. Soc.* 32:182-197.
- Norris, L. A. 1986. Accuracy and precision of analyses for 2,4-D and picloram in streamwater by ten contract laboratories. *Weed Sci.* 34:485-489.
- Phillips, W. M., and K. C. Feltner. 1972. Persistence and movement of picloram in two Kansas soils. *Weed Sci.* 20(1):110-116.
- Scifres, C. J., R. R. Hahn, J. Diaz-Colon, and M. G. Merkle. 1971. Picloram persistence in semiarid rangeland soils and water. *Weed Sci.* 19(4):381-384.
- Smith, A. E., D. Aite, R. Grover, L. A. Kerr, L. J. Milward, and H. Sommerstad. 1988. Persistence and movement of picloram in a northern Saskatchewan watershed. *J. Environ. Qual.* 17(2):262-268.
- Spiridonov, Y. Y., V. G. Shestakov, V. S. Bonadarev, N. S. Trunovaskaya, and A. V. Varovin. 1987. Contributions of the principal biological and physicochemical processes to the detoxification of picloram in soil. *Soviet Soil Sci.* 19(3):41-45.
- Stevens, J. T., and D. D. Sumner. 1991. Herbicides. Chapter 20 *in* Handbook of pesticide toxicology, Vol. 3, Classes of herbicides. W.J. Hayes, Jr. and E. R. Laws, Jr. eds. Academic Press, Inc. San Diego, California. 1576 pages.
- Trichell, D. W., H. L. Morton, and M. G. Merkle. 1968. Loss of herbicides in runoff water. *Weed Sci.* 16:447-449.
- WSSA. 1994. Herbicide handbook. Weed Society of America. Champaign, Illinois. 352 pp.
- Watson, V. J., P. M. Rice, and E. C. Monnig. 1989. Environmental fate of picloram used for roadside weed control. *J. Environ. Qual.* 18:198-205.
- Woodburn, K. B., D. D. Fontaine, E. L. Bjerke, and G. J. Kallos. 1989. Photolysis of picloram in dilute aqueous solution. *Environ. Toxicol. Chem.* 8:769-775.

Date Authored: April 2001

SETHOXYDIM

Herbicide Basics

Chemical formula: 2-[1-(ethoxyimino)butyl]-5-[2-(ethylthio)propyl]-3-hydroxy-2-cyclohexen-1-one

Herbicide Family:

Cyclohexanedione

Target Species: annual and perennial grasses

Forms: not available as a salt or ester

Formulations: EC, SL

Mode of Action: Lipid synthesis inhibitor

Water Solubility: 4,000 ppm

Adsorption potential: low

Primary degradation mech: Microbial metabolism and photolysis

Average Soil Half-life: 5 days

Mobility Potential: high

Dermal LD50 for rabbits: >5,000 mg/kg

Oral LD50 for rats: >2,676 mg/kg

LC50 for bluegill sunfish: 100 mg/L

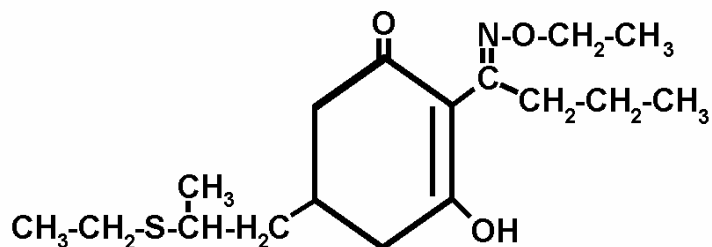
Trade Names: Poast[®], Torpedo[®], Ultima[®], Vantage[®], Conclude[®], and Result[®]

Manufacturer:

BASF, TopPro, Monterey

Synopsis

Sethoxydim kills grasses by preventing the synthesis of lipids, but it has little or no impact on broadleaf herbs or woody plants. Sethoxydim is readily degraded through microbial metabolism and photolysis, and possibly by hydrolysis. Numerous degradation products have been identified, some of which are also toxic to plants. The average half-life of sethoxydim in soils is four to five days, but half-lives can range from a few hours to 25 days. Because sethoxydim is water-soluble and does not bind strongly with soils, it can be highly mobile. No reports, however, were found referring to water contamination or off-site movement by sethoxydim. Sethoxydim is of relatively low toxicity to birds, mammals, and aquatic animals, and has little noticeable impact on soil microbe populations. An oil adjuvant or non-ionic surfactant should be used to facilitate absorption of sethoxydim by plants.



Herbicide Details

Chemical Formula: 2-[1-(ethoxyimino)butyl]-5-[2-(ethylthio)propyl]-3-hydroxy-2-cyclohexen-1-one

Trade Names: Poast[®], Torpedo[®], Ultima[®], Vantage[®], Conclude[®], and Rezult[®]

Manufacturers: BASF, TopPro, and Monterey

Use Against Natural Area Weeds: Sethoxydim is a selective herbicide used to kill and suppress annual and perennial grasses. It is applied as a post-emergent herbicide, and requires the addition of an oil adjuvant or nonionic surfactant for maximum effectiveness (WSSA 1994). According to manufacturers, sethoxydim can be used to control bahiagrass (*Paspalum notatum*), crabgrass (*Digitaria sanguinalis*), downy brome (*Bromus tectorum*), quackgrass (*Elytrigia repens*), annual ryegrass (*Lolium multiflorum*), wild oats (*Avena* spp.), and witchgrasses (*Panicum* spp.) (BASF 2000; TopPro 2000).

Sethoxydim can be highly mobile in the environment and has not been used extensively on TNC preserves. In Illinois, sethoxydim was applied against reed canarygrass (*Phalaris arundinacea*), and initial results indicate that significant control was achieved. In southwest Oregon, however, sethoxydim was applied against quackgrass (*Elytrigia repens*), but was less effective than fluazifop-p-butyl (Fusilade[®]) and is therefore not highly recommended for quackgrass control (Borgias et al. 2000).

Mode of Action: Sethoxydim is absorbed rapidly through leaf surfaces, transported in the xylem and phloem, and accumulated in meristematic tissues. Sethoxydim inhibits acetyl CoA carboxylase (much like the herbicide fluazifop-p-butyl), the enzyme responsible for catalyzing an early step in fatty acid synthesis. Non-susceptible broadleaf species have a different acetyl CoA carboxylase binding site, rendering them immune to the effects of sethoxydim. The inhibition of acetyl CoA carboxylase prevents fatty acid production, which leads to failure of cell membrane integrity, especially in regions of active growth. This results in a cessation of shoot and rhizome growth, leading to necrosis and death of shoot meristems and rhizome buds, and ultimately plant death.

Dissipation Mechanisms:

Summary: Sethoxydim is degraded readily by light and microbial metabolism. It is unclear what role hydrolysis plays in the removal of sethoxydim from the environment. Sethoxydim is water-soluble and does not bind readily with soils, and therefore has the potential to be mobile. Rapid degradation, however, generally limits extensive movement of sethoxydim in the environment. Sethoxydim is not highly volatile.

Volatilization

Sethoxydim does not readily volatilize when applied in the field (T. Lanini, pers. obs.). The potential to volatilize, however, increases with increasing temperature, increasing soil moisture, and decreasing clay and organic matter content (Helling et al. 1971).

Photodegradation

Sethoxydim is readily degraded by light (Shoaf & Carlson 1992). Photodegradation occurs in less than one hour in water and in less than four hours on soil (WSSA 1994; EXTOKNET 1996).

Microbial Degradation

Microbial metabolism is the primary means of degradation of sethoxydim in soils. Roslycky (1986) reports a half-life of sethoxydim in soils, due to microbial metabolism, that averages 25 days.

Adsorption

Sethoxydim is water-soluble and does not bind strongly with soil particles. Adsorption of sethoxydim to soil particles increases with increasing soil organic content (WSSA 1994).

Chemical Decomposition

Reports of sethoxydim's susceptibility to hydrolysis are conflicting (Campbell & Penner 1985; EXTOKNET 1996). EXTOKNET (1996) reports that the product Poast® is fairly stable in water. Campbell and Penner (1985), however, report that sethoxydim degrades rapidly in water at room temperature.

Behavior in the Environment

Summary: Sethoxydim is degraded rapidly by microbial degradation and photolysis. The half-life of sethoxydim in the field ranges from 5 to 25 days. Because it is water-soluble and does not bind strongly with soils, it can be highly mobile in the environment. In water, sethoxydim can be degraded by sunlight within several hours.

Soils

Sethoxydim is of low soil persistence. It is degraded in soils rapidly by microbial metabolism and photolysis. The half-life of sethoxydim in soils ranges from a few hours to 25 days (Roslycky 1986; Shoaf & Carlson 1992; Koskinen et al. 1993). Roslycky (1986) also found that degradation rates are rapid during the first few weeks following application, but then decrease, taking 50 days to reach 80% and 100 days to reach 90% degradation. Similarly, Koskinen et al. (1993) detected residues 38 days following application, while half-lives averaged less than one week.

Although sethoxydim is water-soluble and does not bind strongly with soils, significant movement of sethoxydim has not been documented, possibly because it degrades rapidly, before it can travel any noticeable distance. Koskinen et al. (1993) reported that in three Minnesota soils, sethoxydim leached only minimal amounts with residues occasionally reaching 45 cm.

Water

In water, sethoxydim can be rapidly degraded by light in less than one hour (EXTOXNET 1996). Sethoxydim, because it binds weakly with soil particles, has the potential to move off-site and contaminate local waterways. No reports of water contamination, however, have been documented. If sethoxydim does enter an open water body, it can be degraded by sunlight in a matter of hours.

Vegetation

Sethoxydim is readily absorbed by plant roots and foliage and transported through the xylem and phloem to the meristematic tissues of the plant (WSSA 1994). It is hypothesized that in non-susceptible species, sethoxydim is metabolized rapidly to non-phytotoxic metabolites (WSSA 1994).

Environmental Toxicity

Birds and Mammals

Sethoxydim is slightly toxic to birds and mammals (EXTOXNET 1996). The LD50 for rats is 2,600-3,100 mg/kg. For bobwhite quail and mallard duck the LD50s are >5,620 and >2,510, respectively. Effects of chronic ingestion include anemia, and reproductive and teratogenic effects (EXTOXNET 1996).

Aquatic Species

Sethoxydim is moderately to slightly toxic to aquatic species (EXTOXNET 1996). The LC50 for bluegill sunfish and rainbow trout are 100 mg/L and 32 mg/L, respectively (EXTOXNET 1996). The LC50 for *Daphnia* is 1.5 mg/L (EXTOXNET 1996).

Other Non-Target Organisms

Roslycky (1986) investigated the effects of sethoxydim on populations of soil microbes. At sethoxydim concentrations <50 ppm, negligible response was noted in microbial populations. At higher concentrations (1000 ppm), soil actinomycetes and bacteria populations were stimulated, but fungal populations changed little (Roslycky 1986).

Application Considerations:

Because sethoxydim degrades rapidly, it should be used solely as a post-emergent herbicide. Sethoxydim is most effective when mixed with an oil adjuvant or nonionic surfactant (WSSA 1994). Marked increases in sethoxydim effectiveness have been reported with increases in humidity and temperature (Rhodes & Coble 1983; Coupland 1987). On the other hand, reduced soil moisture significantly reduced sethoxydim's effectiveness (Coupland 1987). Sethoxydim is rainfast (will not wash-off plants) within 10-15 minutes of application (Rhodes & Coble 1983; Coupland 1987).

Safety Measures:

Sethoxydim is slightly toxic if ingested, can cause skin and eye irritation, and inhalation of dusts or vapors can cause irritation of the throat and nose. Care should be taken to avoid splashing or other exposure of skin and eyes to the herbicide.

Human Toxicology:

Although some of the effects of chronic exposure to sethoxydim have been identified in rabbits and dogs, EXTOXNET (1996) concluded that chronic effects in humans from expected exposure levels were unlikely. Sethoxydim is not mutagenic or carcinogenic in humans. The U.S. EPA reports that the level of toxicity of sethoxydim to mammals is low, and that sethoxydim is practically nontoxic if absorbed through the skin. It can however, cause skin and eye irritation. Sethoxydim is slightly toxic by ingestion, and inhalation can cause irritation to the throat, nose, and upper respiratory system. Symptoms of sethoxydim poisoning include loss of coordination, sedation, tears, salivation, tremors, blood in the urine, and diarrhea.

References

- BASF. 2000. Poast Herbicide Label. C & P Press.
- Borgias, D., R. Dovel, R. Huddleston, N. Rudd, and D. Salzer. 2000. Management and monitoring of *Elytrigia repens* (L.) Nevski [quackgrass], invading the habitat of *Astragalus applegatei* [Applegate's milk-vetch], a listed endangered species, on Ewauna Flat Preserve, Klamath County, Oregon. The Nature Conservancy of Oregon, ELRE Final Report.
- Campbell, J. R., and D. Penner. 1985. Abiotic transformations of sethoxydim. *Weed Sci.* 33:435-439.
- Coupland, D. 1987. Influence of environmental factors on the performance of sethoxydim against. *Weed Res.* 27:329-336.
- EXTOXNET. 1996. Sethoxydim. Pesticide Information Profiles. Extension Toxicology Network. <http://ace.orst.edu/info/extoxnet/>.
- Helling, C. S., P. C. Kearney, and M. Alexander. 1971. Behavior of pesticides in soil. *Adv. Agron.* 23:147-240.
- Rhodes, G. N., and H. D. Coble. 1983. Environmental factors affecting the performance of sethoxydim. *Proc. S. Weed Sci. Soc.* pg. 154-155.
- Roslycky, E. B. 1986. Microbial response to sethoxydim and its degradation in soil. *Can. J. Soil Sci.* 66:411-419.
- Shoaf, A. R., and W. C. Carlson. 1992. Stability of sethoxydim and its degradation products in solution, in soil, and on surfaces. *Weed Sci.* 40:384-389.
- TopPro. 2000. Vantage Herbicide Label. C & P Press.
- WSSA. 1994. Herbicide handbook. Weed Society of America. Champaign, Illinois. 352 pp.

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TRICLOPYR

M. Tu, C. Hurd, R. Robison & J.M. Randall

Herbicide Basics

Chemical formula: [(3,5,6-trichloro-2-pyridinyl)oxy] acetic acid

Herbicide Family:
Pyridine (Picolinic acid)

Target Species: Broadleaf herbs and woody species

Forms: salt & ester

Formulations: EC, SL

Mode of Action: Auxin mimic

Water solubility: 430 ppm (acid), 23 mg/L (ester), 2,100,000 mg/L (salt)

Adsorption potential:
Intermediate (higher for ester than salt)

Primary degradation mech:
Microbial metabolism, photolysis, and hydrolysis

Average Soil Half-life: 30 days

Mobility Potential: Intermediate

Dermal LD50 for rabbits:
>2,000 mg/kg

Oral LD50 for rats:
713 mg/kg

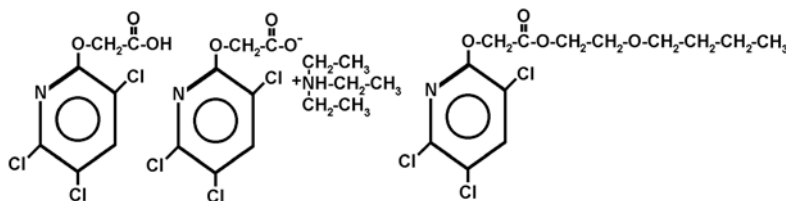
LC50 for bluegill sunfish:
148 mg/L

Trade Names: Garlon® and Access®

Manufacturers: Dow Agro-Sciences and Platte

Synopsis

Triclopyr is a selective systemic herbicide used to control woody and herbaceous broadleaf plants along right-of-ways, in forests, and in grasslands and parklands. It has little or no impact on grasses. Triclopyr controls target weeds by mimicking the plant hormone auxin, causing uncontrolled plant growth. There are two basic formulations of triclopyr - a triethylamine salt, and a butoxyethyl ester. In soils, both formulations degrade to the parent compound, triclopyr acid. Degradation occurs primarily through microbial metabolism, but photolysis and hydrolysis can be important as well. The average half-life of triclopyr acid in soils is 30 days. Offsite movement through surface or sub-surface runoff is a possibility with triclopyr acid, as it is relatively persistent and has only moderate rates of adsorption to soil particles. In water, the salt formulation is soluble, and with adequate sunlight, may degrade in several hours. The ester is not water-soluble and can take significantly longer to degrade. It can bind with the organic fraction of the water column and be transported to the sediments. Both the salt and ester formulations are relatively non-toxic to terrestrial vertebrates and invertebrates. The ester formulation, however, can be extremely toxic to fish and aquatic invertebrates. Because the salt cannot readily penetrate plant cuticles, it is best used as part of a cut-stump treatment or with an effective surfactant. The ester can be highly volatile and is best applied at cool temperatures on days with no wind. The salt formulation (Garlon 3A®) can cause severe eye damage.



Triclopyr acid

Triethylamine salt

Butoxyethyl ester

Herbicide Details

Chemical Formula: [(3,5,6-trichloro-2-pyridinyl)oxy]acetic acid

Trade Names: There are two basic formulations of triclopyr: a triethylamine salt (triclopyr amine or salt), and a butoxyethyl ester (triclopyr ester). The amine formulation is sold under the trade name Garlon 3A[®] and is marketed in garden shops and hardware stores as Turflon Amine[®] or as Brush-B-Gone[®]. The ester formulation is sold under the trade name Garlon 4[®] and is marketed in garden shops and hardware stores as Turflon Ester[®]. Other trade names include Access[®], Crossbow[®], ET[®], PathFinder II[®], Redeem[®], and Remedy[®]. These products also may be mixed with picloram or 2,4-D to increase their versatility.

Manufacturers: Dow Agrosiences (formerly known as DowElanco or Dow Chemical), Platte

Use Against Natural Area Weeds: Triclopyr is used to control broadleaf herbs and woody species (WSSA 1994). It is particularly effective at controlling woody species with cut-stump or basal bark treatments. Susceptible species include the brooms (*Cytisus* spp., *Genista* spp., and *Spartium* spp.), the gorses (*Ulex* spp.), and fennel (*Foeniculum vulgare*). Triclopyr ester formulations are especially effective against root- or stem-sprouting species such as buckthorns (*Rhamnus* spp.), ash (*Fraxinus* spp.), and black locust (*Robinia pseudoacacia*), because triclopyr remains persistent in plants until they die.

Even though offsite movement of triclopyr acid through surface or sub-surface runoff is a possibility, triclopyr is one of the most commonly used herbicides against woody species in natural areas. Bill Neil, who has worked extensively on tamarisk/saltcedar (*Tamarix* spp.) control, concluded that Pathfinder II[®], a triclopyr ester formulation by DowElanco, is the most cost effective herbicide for combating saltcedar. On preserves across the U.S., triclopyr has provided good control of tree-of-heaven (*Ailanthus altissima*), salt cedar (*Tamarix* spp.), glossy buckthorn (*Frangula alnus*), common buckthorn (*Rhamnus cathartica*), sweet fennel (*Foeniculum vulgare*), Brazilian peppertree (*Schinus terebinthifolius*), and Chinese tallow tree (*Sapium sebiferum*). TNC preserves in Hawaii have successfully used triclopyr to control blackwood acacia (*Acacia melanoxylon*), bush honeysuckle (*Lonicera maackii*), Chinese banyan (*Ficus microcarpa*), corkystem passionflower (*Passiflora suberosa*), eucalyptus (*Eucalyptus globulus*), Florida prickly blackberry (*Rubus argutus*), Mexican weeping pine (*Pinus patula*), Monterey pine (*Pinus radiata*), strawberry guava (*Psidium cattleianum*), tropical ash (*Fraxinus uhdei*), and velvet leaf (*Miconia calvescens*). Triclopyr can also be used in forest plantations to control brush without significant impacts to conifers (Kelpsas & White). Spruces (*Picea* spp.) can tolerate triclopyr, but some species of pine (*Pinus* spp.) however, can only tolerate triclopyr during the dormant fall and winter months (Jotcham et al. 1989).

Mode of Action: Triclopyr is an auxin mimic or synthetic auxin. This type of herbicide kills the target weed by mimicking the plant growth hormone auxin (indole acetic acid), and when administered at effective doses, causes uncontrolled and disorganized plant growth that leads to plant death. The exact mode of action of triclopyr has not been fully described, but it is believed to acidify and “loosen” cell walls, allowing cells to expand without normal control and

coordination. Low concentrations of triclopyr can stimulate RNA, DNA, and protein synthesis leading to uncontrolled cell division and growth, and, ultimately, vascular tissue destruction. Conversely, high concentrations of triclopyr can inhibit cell division and growth.

Dissipation Mechanisms:

Summary: Both the ester and amine formulations are degraded by sunlight, microbial metabolism, and hydrolysis. In soils, both the ester and amine formulations will degrade rapidly to the parent compound, triclopyr acid. The acid and ester formulations bind well with soils, and therefore, are not likely to be mobile in the environment. The salt however, does not readily adsorb and can be mobile. The ester can be highly volatile (T. Lanini, pers. com.).

Volatilization

Ester formulations of triclopyr can be highly volatile, and care should be taken in their application. The potential to volatilize increases with increasing temperature, increasing soil moisture, and decreasing clay and organic matter content (Helling et al. 1971).

Photodegradation

Both the ester and salt formulations are degraded readily in sunlight to the parent compound, triclopyr acid, which is also photodegradable. A study of photolysis found the half-life of triclopyr acid on soil under midsummer sun was two hours (McCall & Gavit 1986). Photodegradation can be particularly important in water. Johnson et al. (1995) found triclopyr acid dissolved in water had a half-life due to photolysis of one to 12 hours.

Microbial Degradation

Microbial metabolism accounts for a significant percentage of triclopyr degradation in soils. In general, warm, moist soils with a high organic content will support the largest microbial populations and the highest rates of herbicide metabolism (Newton et al. 1990). Johnson et al. (1995a) found that microbial degradation of triclopyr was significantly higher in moist versus dry soils, and higher at 30° C than at 15° C (DT50 is 46 days versus 98 days in dry soils, and 57 days versus 199 days in moist soils, respectively). Additionally, the presence of sunlight plays a role in the rates of microbial metabolism of triclopyr. Johnson et al. (1995a) found that microbial metabolism was slowed when soil was deprived of light.

Chemical Decomposition

Hydrolysis of both the salt and ester to the acid form occurs readily in the environment and within plants (Smith 1976). McCall and Gavit (1986) reported that the ester was converted to an acid with a half-life of three hours, and that the rate of hydrolysis in water increased with an increase in pH.

Adsorption

Adsorption temporarily or permanently immobilizes triclopyr, but adsorption is not degradation. Adsorption is more important for the immobilization of the ester than of the salt formulation. The ester binds readily with the organic component of the soil, with adsorption rates increasing as organic content increases and soil pH decreases (Pusino et al. 1994; Johnson et al. 1995a). The salt form is soluble in water and binds only weakly with soil (McCall & Gavit 1986). The

strong bond between the ester and soils accounts for the relatively low mobility of the ester in soils, whereas the salt form is much more mobile (McCall & Gavit 1986). In practice, however, both compounds are degraded rapidly to triclopyr acid, which has an intermediate adsorption capacity.

Behavior in the Environment

Summary: In soils, both formulations are degraded by photolysis, microbial metabolism, and hydrolysis to the parent compound, triclopyr acid. Triclopyr acid has an intermediate adsorption potential, limiting movement of the acid in the environment. The acid degrades with an average half-life of 30 days. In water, the salt will remain in the water column until it is degraded, which can occur in as little as a few hours under favorable conditions. The ester formulation, however, is not water-soluble and can take significantly longer to degrade in water. Within plants, both the salt and ester formulations are hydrolyzed to the acid form, and transported through the plant. Residues can persist in the plant until the tissues are degraded in the environment.

Soils

Both the ester and salt formulations degrade rapidly in soils to triclopyr acid, and thereafter, behave similarly in soils. Adsorption, photodegradation, microbial metabolism, and volatility, can all play a role in the dissipation of triclopyr from soils. The reported half-life of triclopyr in soils varies from 3.7 to 314 days, but averages 30 days, depending on the formulation applied and the specific soil and environmental conditions. If soil conditions are warm and moist, microbial metabolism can be the primary means of degradation (Newton et al. 1990).

Johnson et al. (1995a) reported an average half-life of triclopyr acid in four laboratory soils of 138 days, but this time varied significantly with soil temperature. At 15°C half-lives ranged from 64-314 days, while at 30°C half-lives were 9-135 days (Johnson et al. 1995). In Southwest Oregon, Newton et al. (1990) found 24-51% of triclopyr residues remained after 37 days in a dry and cool climate. Following an increase in warmth and moisture, however, dissipation increased dramatically and triclopyr residues exhibited a half-life of 11-25 days. In a study of triclopyr persistence in soil and water associated with rice production, triclopyr had a half-life of less than ten days in the three soil types tested (Johnson et al. 1995b). In a pasture near Corvallis, Oregon, the half-life of triclopyr acid was estimated to be 3.7 days (Norris et al. 1987).

Because of the importance of photodegradation and a decrease in the size of microbial populations with soil depth, triclopyr located deeper in the soil column (>15 cm) degrades more slowly than residues near the surface (Johnson et al. 1995a). Traces of triclopyr residues have been found at soil depths of 45 cm as late as 477 days after application (Newton et al. 1990). Sandy soils that are highly permeable may therefore, retain triclopyr longer. Most studies, however, found that triclopyr generally does not tend to move in significant quantities below the top 15 cm of soil (Norris et al. 1987; Newton et al. 1990; Stephenson 1990; Johnson et al. 1995a).

Water

In water, the two formulations can behave very differently. The water-soluble salt is degraded in the water column through photolysis and hydrolysis (McCall & Gavit 1985). The ester, however, is not water-soluble and can be persistent in aquatic environments. The ester binds to organic particles in the water column and precipitates to the sediment layers (McCall & Gavit 1986). Bound ester molecules will degrade through hydrolysis or photolysis to triclopyr acid (Smith 1976), which will move back into the water column and continue to degrade. The rate of degradation is dependent on the water temperature, pH, and sediment content.

Triclopyr acid has an intermediate soil adsorption capacity. Thus, movement of small amounts of triclopyr residues following the first significant rainfall are likely (McCall & Gavit 1986), but further leaching is believed to be minor (Newton et al. 1990; Stephenson et al. 1990; Thompson et al. 1991). Movement of triclopyr through surface and subsurface runoff in areas with minimal rainfall is believed to be negligible (Newton et al. 1990; Stephenson et al. 1990). In southwest Oregon, Norris et al. (1987) found that neither leaching nor long-distance overland water flow contributed significant amounts of the herbicide into a nearby stream, and concluded that the use of triclopyr posed little risk for non-target organisms or downstream water users. Triclopyr can, however, enter waterways via aerial drift and inadvertent overspray. When the acid was applied to rice paddy fields, residues remained in the water column and were not found in significant amounts in the soil (Johnson et al. 1995b). Degradation in water was rapid and showed a half-life of four days.

Vegetation

Both the ester and salt formulations are hydrolyzed to the acid after entering plant tissue. The acid tends to remain in plants until they die or drop leaves and begin to decay (Newton et al. 1990). Newton et al. (1990) reported that triclopyr in evergreen foliage and twigs showed remarkable persistence. Although concentrations of triclopyr in the soil will decrease quickly and remain low through the winter, levels can rise again in the spring if a new supply of contaminated foliage falls from defoliating crowns (Newton et al. 1990). The residues of some herbicides in fruit have been shown to persist up to one month (Holmes et al. 1994). There is therefore a potential for long-term exposure of triclopyr to animal species that eat wild fruit. In non-target plants, triclopyr soil residues can cause damage via root uptake (Newton et al. 1990).

Environmental Toxicity

Birds and Mammals

Triclopyr is regarded as only slightly toxic to birds and mammals. The oral LD50 for rats is 630-729 mg/kg. The LD50s for mallard ducks and bobwhite quail are 1,698 mg/kg and 2,935 mg/kg, respectively. Newton et al. (1990) predicted that triclopyr would not be present in animal forage in doses large enough to cause either acute or chronic effects to wildlife, and concluded that the tendency for triclopyr to dissipate quickly in the environment would preclude any problems with bioaccumulation in the food chain. Garlon 3A[®] can cause severe eye damage to both humans and wildlife, due to the high pH of its water-soluble amine salt base. Care must be taken during mixing and application to prevent accidental splashing into eyes.

In a study of the potential effects of herbicide residues on forest songbirds, sub-lethal doses of triclopyr ester (500 mg/kg in the diet for 29 days) were found to cause weight loss and behavior alterations in zebra finches (Holmes et al. 1994). In a 1987 study of triclopyr metabolism using one cow, all traces of triclopyr were eliminated from the cow's urine within 24 hours, and no residues were detected in its milk or feces. This study, however, did not track whether any triclopyr was absorbed into the cow's tissues, or whether the triclopyr recovered in the urine was still active (Eckerlin 1987).

Aquatic Species

Triclopyr acid and the salt formulation are slightly toxic to fish and aquatic invertebrates. The LC50 of the acid and the salt formulation for rainbow trout are 117 mg/L and 552 mg/L, respectively, and for bluegill sunfish 148 mg/L and 891 mg/L, respectively. The ester formulation is highly toxic to fish and aquatic invertebrates, with an LC50 (96-hour) of 0.74 mg/L in rainbow trout and 0.87 mg/L in bluegill sunfish (WSSA 1994; EPA 1998). The hydrophobic nature of the ester allows it to be readily absorbed through fish tissues where it is rapidly converted to triclopyr acid. The acid can be accumulated to a toxic level when fish are exposed to sufficient concentrations or for sufficient durations.

The extent to which the toxic effects of the ester are reduced by degradation is poorly understood. Studies have shown that the ester formulation degrades rapidly to less toxic forms (Thompson et al. 1991). Kreutzweiser et al. (1994) however, has shown that there is a significant chance of acute lethal effects to fish exposed to low level residues for more than six hours. In addition, delayed lethal effects were seen in fish exposed to high concentrations for a short duration. Considering that Thompson et al. (1991) concluded that organisms subjected to direct overspray were exposed to a high level of herbicide for short periods of time while organisms downstream were exposed to low levels for longer periods, the findings of Kreutzweiser et al. (1994) are of concern.

Nevertheless, most authors including the authors of the fish mortality study have concluded that if applied properly, triclopyr would not be found in concentrations adequate to kill aquatic organisms. As a measure of precaution, however, Kreutzweiser et al. (1991) suggest that some water bodies remain at risk of lethal contamination levels including shallow and slow moving water bodies where dissipation is slow, and heavily shaded streams that experience reduced photodegradation.

Other Non-Target Organisms

Triclopyr inhibited growth of four types of ectomycorrhizal fungi associated with conifer roots at concentrations of 1,000 parts per million (ppm) and higher (Estok et al. 1989). Some evidence of inhibition of fungal growth was detected in bioassays with as little as 100 ppm triclopyr. Typical usage in forest plantations, however, results in triclopyr residues of only four to 18 ppm on the forest floor (Estok et al. 1989).

Application Considerations:**Application Under Unusual Conditions:**

Several natural area managers have found that Garlon 4[®] and 3A[®] are effective when applied in mid-winter as a cut-stump treatment against buckthorns (*Rhamnus cathartica* and *R. frangula*). It is often easier to get to these plants when boggy soils around them are frozen. Randy Heidorn, Deputy Director for Stewardship of the Illinois Nature Preserve Commission (INPC), recommends three protocols to increase the safety of triclopyr ester application in winter:

- (1) use a mineral oil based carrier;
- (2) make sure that at the time of application, no water is at or above the ground surface, and no snow or ice is present that might serve as a route to spread the herbicide following a thaw, and;
- (3) initiate a monitoring program to assess ambient water concentrations of triclopyr ester in communities that seasonally have water at or above the ground surface with little or no discharge (i.e. bogs).

Safety Measures

The salt formulation in Garlon 3A[®] can cause severe eye damage because of the high pH of its water-soluble amine salt base. Care should be taken to prevent splashing or other accident contact with eyes.

Human Toxicology

Because studies into the carcinogenicity of triclopyr have produced conflicting results, EPA has categorized triclopyr as a “Group D” compound, or a chemical that is not classifiable as to human carcinogenicity. The salt formulation in Garlon 3A[®] can cause severe eye damage.

References

- Eckerlin, R.H., J. E. Ebel, Jr., G. A. Maylin, T. V. Muscato, W. H. Gutenmann, C. A. Bache, and D. J. Lisk. 1987. Excretion of triclopyr herbicide in the bovine. *Bull. Environ. Contam. Toxicol* 39:443-447.
- Estok, D., B. Freedman, and D. Boyle. 1989. Effects of the herbicides 2,4-D, glyphosate, hexazinone, and triclopyr on the growth of three species of ectomycorrhizal fungi. *Bull. Environ. Contam. and Toxic.*, 42:835-839.
- Helling, C. S., P. C. Kearney, and M. Alexander. 1971. Behavior of pesticides in soil. *Adv. Agron.* 23:147-240.
- Holmes, S. B., D. G. Thompson, K. L. Wainio-Deizer, S. S. Capell, and B. Staznik. 1994. Effects of lethal and sublethal concentrations of the herbicide triclopyr butoxyethyl ester in the diet of Zebra finches. *J. Wildlife Dis.* 30(3):319-327.
- Johnson, W. G., T. L. Lavy, and E. E. Gbur. 1995a. Persistence of triclopyr and 2,4-D in flooded and non-flooded soils. *J. Environ. Qual.*, 24:493-497.
- Johnson, W. G., T. L. Lavy, and E. E. Gbur. 1995b. Sorption, mobility, and degradation of triclopyr and 2,4-D on four soils. *Weed Sci.* 43:678-684.
- Jotcham, J. R., D.E.W. Smith, and G.R. Stephenson. 1989. Comparative persistence and mobility of pyridine and phenoxy herbicides in soil. *Weed Tech.* 3:155-161.

- Kelpsas, B.R. and D.E. White. no date. Conifer tolerance and shrub response to triclopyr, 2,4-D and clopyralid. Northwest Chemical Company, Salem, Oregon.
- Kreutzweiser, D. P., S. B. Holmes, and D. C. Eichenberg. 1994. Influence of exposure duration on the toxicity of triclopyr ester to fish and aquatic insects. *Archives of Environ. Contam. Toxic.* 26:124-129.
- McCall, P. J. and P. D. Gavit. 1986. Aqueous photolysis of triclopyr and its butoxyethyl ester and calculated environmental photodecomposition rates. *Environ. Toxic. Chem.* 5:879-885.
- Newton, M., F. Roberts, A. Allen, B. Kelpsas, D. White, and P. Boyd. 1990. Deposition and dissipation of three herbicides in foliage, litter, and soil of brushfields of southwest Oregon. *J. Agric. Food Chem.* 38:574-583.
- Norris, L., M. L. Montgomery, and L. E. Warren. 1987. Triclopyr persistence in western Oregon hill pastures. *Bull. Environ. Contam. Toxic.* 39:134-141.
- Pusino, A. W. Liu, and C. Gessa. 1994. Adsorption of triclopyr on soil and some of its components. *J. Agric. Food Chem* 42:1026-1029.
- Smith, A. E. 1976. The hydrolysis of herbicidal phenoxyalkanoic esters of phenoxyalkanoic acids in Saskatchewan soils. *Weed Res.* 16:19-22.
- Stephenson, G. R., K. R. Solomon, C. S. Bowhey, and K. Liber. 1990. Persistence, leachability, and lateral movement of triclopyr (Garlon) in selected Canadian forestry soils. *J. Agric. Food Chem.* 38:584-588.
- Thompson, D. G., B. Staznik, D. D. Fontaine, T. Mackay, G. R. Oliver, and J. L. Troth. 1991. Fate of triclopyr ester (Release[®]) in a boreal forest stream. *Environ. Toxic. Chem.* 10:619-632.
- WSSA. 1994. *Herbicide Handbook*. Weed Society of America. Champaign, Illinois, 352 pp.

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Chapter 8 - ADJUVANTS

M. Tu & J.M. Randall

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Synopsis

An adjuvant is any compound that is added to a herbicide formulation or tank mix to facilitate the mixing, application, or effectiveness of that herbicide. Adjuvants are already included in the formulations of some herbicides available for sale (e.g. RoundUp[®]), or they may be purchased separately and added into a tank mix prior to use. Adjuvants are chemically and biologically active compounds, and they may improve the effectiveness of the herbicide they are added to, either increasing its desired impact and/or decreasing the total amount of formulation needed to achieve the desired impact. Some herbicides require the addition of an adjuvant to be effective. Some adjuvants enhance the penetration of herbicide into plants by ensuring adequate spray coverage and keeping the herbicide in contact with plant tissues, or by increasing rates of foliar and/or stomatal penetration.

The U.S. Environmental Protection Agency (EPA) regulates the inclusion of certain ingredients in adjuvant formulations, but it does not stringently test and regulate the manufacture and use of adjuvant products (as they do for herbicides and other pesticides). As such, there is little information on the effects of these different adjuvants, other than that provided by the manufacturer. A herbicide label may specify what types of adjuvant are appropriate or advisable to use with that herbicide, but it will not suggest specific brands. Therefore, there is no good single resource or system to help you determine which specific adjuvant product (if any) to use for each application situation. However, it is worth checking the label of any adjuvant you are considering to see if it is registered in certain states, such as California or Washington. These states regulate adjuvants and require the disclosure of their ingredients, results from efficacy trials, and data from environmental and toxicological studies. The best source of information for which adjuvant to use (if any) in each situation is usually your local agriculture or university extension agent, county weed coordinator, or herbicide company representative.

INTRODUCTION TO ADJUVANTS

An adjuvant is any compound that can be added to a herbicide formulation to facilitate the mixing, application, or effectiveness of that herbicide. Adjuvants are already included in the formulations of some herbicides available for sale (e.g. RoundUp[®]), or they may be purchased separately and added into a tank mix prior to use (Pringnitz 1998). Herbicides must overcome a variety of barriers to their entry into plants in order to be effective. For example, herbicides applied to foliage must remain on the leaf instead of beading up and rolling off, then get past the leaf hairs and waxes on the leaf surface, then finally penetrate through the cell walls and cell membranes (DiTomaso 1999; Hull et al. 1982). Some adjuvants alter the formulation so that they more completely and evenly cover plant surfaces thereby keeping the herbicide in contact with plant tissues rather than beading up and rolling off. Others increase the formulation's penetration through the cuticular wax, cell walls and/or stomatal openings. In some situations, an adjuvant may enhance the formulation's ability to kill the targeted species without harming other plants (i.e. enhance its selectivity; Hess & Foy 2000). Adjuvants may also improve a herbicide's efficacy so that the concentration or total amount of herbicide required to achieve a given effect is reduced, sometimes as much as five- or ten-fold (WSSA 1982). In this way adding an appropriate adjuvant can decrease the amount of herbicide applied and lower total costs for weed control (Green 2001, 1992).

Adjuvants are chemically and biologically active (NOT chemically inert) compounds. They produce pronounced effects in plants and animals, and some adjuvants have the potential to be mobile and pollute surface or groundwater sources. Be especially aware of the use of adjuvants near water, as adverse effects may occur in some aquatic species (Parr 1982). The Material Safety Data Sheets (MSDSs) of most adjuvants will list materials that are incompatible with the adjuvant, conditions in which they should not be used, and some toxicological information (LC50 or LD50s), but this information is usually not as complete as that found on herbicide labels and MSDSs.

Unfortunately, there is no good system available to help you assess which types of adjuvants (if any) to select for different situations, much less which brand will best meet your needs. Most herbicide labels specify the *type* of adjuvant to use for best control (see Box 1), but there are many different brands of most types of adjuvants to select from and few sources of good information regarding their relative performance under different conditions. The best source of information is most likely your local agriculture or university extension agent, local county weed coordinator or herbicide company representative. Local herbicide dealers may also offer suggestions, but be sure that the dealer is qualified to make recommendations (Carroll 2001).

Adjuvants may be classified in a variety of ways, such as by their function (activator or utility), chemistry (such as organosilicones), or source (vegetable or petroleum oils) (Penner 2000b). This adds to confusion about which adjuvant to select in different situations. In this chapter, we group adjuvants by their function, as either activator adjuvants or utility adjuvants (see Box 1). Activator adjuvants work to enhance the activity of the herbicide, often by increasing rates of absorption of the herbicide into the target plant(s). Utility adjuvants, sometimes called spray modifiers, work by altering the physical or chemical characteristics of the spray mixture to improve its ease of application, its ability to remain on the plant surface rather than rolling off, or its persistence in the environment (McWhorter 1982). There is much disagreement regarding

how certain adjuvants should be categorized, and to complicate matters further some adjuvants perform more than one function and thus really do fit in more than one category.

Box 1: Adjuvant Types*

Activator Adjuvants

- Surfactants
 - Nonionic (incl. organosilicones)
 - Ionic
 - Amphoteric
- Oil adjuvants (incl. crop oil concentrates)
 - Petroleum oil concentrates
 - Vegetable oils
- Ammonium (nitrogen) fertilizers

Utility Adjuvants (including Spray Modifiers)

- Wetting agents (spreaders)
- Dyes
- Drift control & foaming agents
- Thickening agents
- Deposition agents (stickers)
- Water conditioners
- Compatibility agents
- pH buffers
- Humectants
- Defoaming & antifoam agents
- UV absorbents

*There are many ways to classify adjuvants. In this chapter, we divide adjuvants into two primary types (activator or utility), based on their functions. For a more complete listing of available adjuvants in the U.S., see the Compendium of Herbicide Adjuvants, available at <http://www.herbicide-adjuvants.com/index.html>.

Adding adjuvants allows the applicator to customize the tank formulation for each particular situation (Green 2001). In many situations, adding an adjuvant can significantly enhance a herbicide's effect (Green & Hazen 1998). However, it is important to note that in some circumstances, adding adjuvants will not significantly improve control. For example, there is no benefit to be gained from adding an activator adjuvant when applying a herbicide to broadleaf weeds with thin cuticles that are growing in high humidity and shade (Kudsk & Streibig 1993). Sometimes adjuvants can have negative effects, such as actually decreasing the killing power of the herbicide (antagonistic effects), increasing the formulation's ability to spread or persist in the environment where it is not wanted, or otherwise increasing harmful affects to non-target plants and animals (see Environmental Fates and Toxicity sections). There is no universal adjuvant that can improve the performance for all herbicides, against all weeds, or under all environmental conditions. The herbicide and adjuvant selected and the relative amounts used must be tailored to the specific conditions of each application.

SELECTING AN ADJUVANT

Choosing an appropriate and effective adjuvant can be daunting. To begin with, it is sometimes difficult to determine which adjuvants actually meet the recommendations on the herbicide label. There are hundreds of adjuvants available, and choosing the best one(s) will depend on the plant species targeted, its phenological stage, site conditions, current environmental conditions, and the method of application, etc. (see Box 2). Herbicide labels and MSDS sheets often list the *types* of adjuvants recommended (see Box 4) (e.g., a nonionic surfactant), but they cannot recommend specific adjuvant brands. A further complication is that the US EPA does not stringently regulate the manufacture and marketing of adjuvants since many compounds in adjuvants are classed as ‘inert’ compounds. Compounds in a herbicide are often referred to as ‘inert’ if they do not kill plants or regulate their growth directly (i.e. they are not the active ingredients such as glyphosate or triclopyr). These compounds however, may be (and usually are) chemically and biologically active. In contrast, adjuvant formulations that do not cause any significant biological or chemical effects, are often referred to as ‘inert’; this is closer to the meaning of the word ‘inert’ as it is used in the study of chemistry, but still not entirely the same. Another source of confusion is that adjuvant manufacturers sometimes change the chemical formulation of an adjuvant formulation from one year to the next, even though it is marketed under the same name. A factor to consider is that certain adjuvants or adjuvant mixes may sometimes be more toxic to certain non-target organisms than the herbicide itself. For example, the surfactant included in RoundUp[®] is more toxic to fish than the active ingredient glyphosate. For this reason it is not legal to use RoundUp[®] over water bodies, but glyphosate formulations sold without a surfactant (e.g. Rodeo[®]) are legal in aquatic situations.

Box 2: Some factors to consider when choosing an adjuvant

Environment

Site conditions (Aquatic or terrestrial? In sensitive areas?)

Current conditions (Air temperature? Windy?)

Water chemistry (Hard or soft water? Low or high pH?)

Target(s)

Species and growth form

Phenological stage

Dense or sparse growth? (Will it warrant high volumes of spray?)

Barriers to penetration (Waxy, hairy or thick leaves?)

Method of application (foliar spray, boom spray, stump paint, hack & squirt)

Other

Product interactions or compatibility issues

Order of mixing into the tank mix

How to choose an adjuvant

We recommend that once you decide which herbicide to use, you should contact your local (county or otherwise) weed coordinator, agriculture commissioner, and/or your local university weed extension agent for suggestions. Some herbicide companies, such as DuPont (2000, 2001), produce lists of brand-name adjuvants that are approved for use with their herbicides, but most companies do not. Local representatives from the herbicide companies (or their technical help phone lines), as well as your local chemical supplier or dealer, may also suggest adjuvants that enhance the effects you want from an herbicide.

California and Washington have some of the strictest state regulations for herbicides and adjuvants. In order for an adjuvant to be registered in these two states, the adjuvant company must divulge all product ingredients, list all registered components on the label, and submit efficacy data to prove the product will do what the company says it will. To see if the adjuvant that you are interested in using is labeled in these states, see:

<http://www.cdpr.ca.gov/docs/label/labelque.htm#regprods>
<http://picol.cahe.wsu.edu/LabelTolerance.html>

Box 3. Tips for adjuvant selection (from Brian Carroll of the Helena Chemical Company 2001)

- Read labels, but remember that adjuvants are not regulated on the federal-level by the U.S. EPA, and are therefore not held to any strict standards.
- Always consult your local agriculture extension agent, local weed coordinator, or local chemical dealer
- Calculate the cost of adjuvant based on % active ingredient
- Be familiar with the adjuvant company and salesperson—are they reputable?
- Look for a California or Washington registration number on the label – these states require all adjuvant products sold within the state to be registered with their state’s equivalent to the U.S. EPA. To be registered, the company must divulge all product ingredients, list all registered components on the label and submit efficacy data to prove the product will do what the company says it will do
- Buy high-quality adjuvants
- It is not always necessary or desirable to add an adjuvant
- Use good application techniques and calibrate equipment often.

A complete and up-to-date listing of all currently available adjuvants, listed by name and by type, is available in the Compendium of Herbicide Adjuvants, prepared by Bryan Young (2000) of Southern Illinois University. This compendium is available hardcopy for \$3.00, or can be viewed online at <http://www.herbicide-adjuvants.com/index.html>.

Cautions about adjuvant use

Always follow the herbicide label for mixing instructions and proportions to use. U.S. EPA-approved label directions have the full force of federal law behind them, and must be followed for lawful herbicide application. Consider how the resulting formulation (herbicide plus adjuvants) will affect populations of desirable native plants and other organisms. Will the adjuvant increase damage to the desirable plants to unacceptable levels? If the formulation is likely to do more harm than good overall, do not use it. Also consider the timing of your

application. For example, plants under stress (drought, etc.) do not translocate herbicides as well as fast-growing, healthy plants and are therefore *more* difficult to kill with herbicide (Pringnitz 1998).

Adding more than one adjuvant to a tank mix adds complexity, because different products may interact and interfere with one another, and/or it may be illegal to combine them (Pringnitz 1998). Read labels of each product you intend to add to the mix to determine if there are any restrictions regarding their use. Remember that in most states, adjuvants are not regulated, so you may need to take extra care to determine whether they will perform as advertised. Compare the quantity of active ingredients in similar types of additives to help determine value. Be wary of any product that makes exaggerated claims.

Herbicide (Examples of common brands)	Box 4. Recommended* adjuvant types, for the herbicides listed in this handbook
2,4-D (many brands)	Most brands recommend adding a nonionic surfactant; may be mixed with a nitrogen fertilizer or crop oil concentrate
Clopyralid (Transline [®] , Stinger [®])	Nonionic surfactant
Fluazifop-p-butyl (Fusilade DX [®])	Nonionic surfactant or crop oil concentrate
Fosamine ammonium (Krenite S [®])	Oil-based surfactant suggested
Glyphosate (RoundUp Original [®])	Adjuvants already added; nonionic surfactant or ammonium sulfate may also be added
Glyphosate (RoundUp Ultra [®])	Adjuvants already added; ammonium sulfate may also be added
Glyphosate (Rodeo [®] , Aquamaster [®] , Glypro [®])	Nonionic surfactant
Hexazinone (Velpar L [®])	No recommendations on label
Imazapic (Plateau [®])	Methylated seed oil or crop oil concentrate; nonionic surfactant; silicone-based surfactant; fertilizer-surfactant blends
Imazapyr (Arsenal [®])	Methylated seed oil or crop oil concentrate; nonionic surfactant; silicone-based surfactant; fertilizer-surfactant blends
Picloram (Tordon K [®] , Tordon 22K [®])	Nonionic surfactant
Sethoxydim (Poast [®] , PoastPlus [®])	Methylated seed oil or crop oil concentrate; urea ammonium nitrate or ammonium sulfate (not recommended in Pacific Northwest, not allowed in California)
Sethoxydim (Vantage [®])	Adjuvants already added, none needed
Triclopyr (Garlon 3A [®] , Garlon 4 [®])	Nonionic surfactant

*Recommended from herbicide labels. Be sure to always follow the label instructions for specifics on choosing and mixing herbicides and adjuvants.

REGULATION OF ADJUVANTS

As of 2003, regulatory agencies in the U.S. still pay relatively little attention to the regulation of adjuvants for herbicides. In the absence of consistent labeling laws, thousands of different formulations and brands of adjuvants are marketed and sold without consistent ingredient lists or proper scientific trials (Swisher 1982). This lack of regulation is probably at least partially the result of the role of adjuvants in reducing overall rates of pesticide use in the U.S. and because adjuvants have historically been perceived as ‘inert’ or GRAS (generally regarded as safe) compounds.

The U.S. EPA has exempted about 2,500 chemical compounds from restrictions for use as adjuvants, and therefore, does not require that they be tested and registered. The 1996 Food Quality Protection Act (FPQA), however, is eliminating the ‘inert’ classification and changing how adjuvants will be regulated. FPQA requires that the U.S. EPA review all adjuvant exemptions to ensure “reasonable certainty of no harm.” As of early 2003, there has been little action and resources from the U.S. EPA to begin this testing or to regulate adjuvants. A few states however, have their own laws regarding adjuvants. For instance, California and Washington both regulate adjuvants and require the disclosure of their ingredients, data from efficacy trials, and environmental and toxicological studies.

Prompted by regulations at the state-level, concern regarding product quality, and government proposals to require full disclosure of ingredients, the adjuvant industry has started to self-regulate (Green 2001; Underwood 2000). There is currently a movement by the Chemical Producers and Distributor Association (CPDA) to require a certification process based on 17 standards for labeling and manufacturing, but this has not yet been fully implemented. These adjuvant producers also state that they intend to hold properly designed experimental trials and to make these results available to the herbicide companies as well as to consumers (Green 2001).

ENVIRONMENTAL FATES OF ADJUVANTS

The long-term fates of most adjuvants in soils and elsewhere in the environment are largely unknown, partially because of the lack of long-term monitoring data, but also because the ingredients in most adjuvants are not disclosed. Most adjuvant labels or Material Safety Data Sheets (MSDSs) include information on the adjuvant’s physical properties (boiling and freezing points, specific gravity, evaporation point, etc.), fire and explosion hazard data, reactivity data, and health hazard data. Unlike herbicide labels however, most adjuvant labels or MSDSs do not include information of the compound’s behavior or fates in the environment (in plants and soil). Most adjuvant labels and MSDSs also do not describe the adjuvant’s mechanism of action, rates of metabolism within plants, rates of photodegradation or microbial degradation, persistence (half-life) in the environment, potential for volatilization, or potential mobility in soil or water.

It is known that many surfactants adsorb to soil particles (Bayer & Foy 1982). Because of this, surfactants tend to be less toxic to plants in soil than to plants growing in water or other aqueous solutions or in nutrient culture alone (Bayer & Foy 1982). Adjuvants from different chemical groups have different effects and toxicities in different soil types. For instance, when applied directly to soils (as in pre-emergence herbicide formulations), ester adjuvants tend to have greater impacts in sandy soils while ether adjuvants are most effective on clay soils. Adjuvants that are alcohols are most effective in soils with high levels of organic matter.

TOXICITY OF ADJUVANTS

Although adjuvants are typically categorized as “inert” or “essentially non-phytotoxic” (i.e. not toxic to plants) compounds, many can produce wide ranging effects on physiological and metabolic processes within plants, animals, and/or microorganisms (Norris 1982; Parr & Norman 1965). Almost all of these effects can occur at low concentrations or doses. Some adjuvants can have pronounced effects on biochemical processes, including enzyme activity. Some can disrupt or otherwise alter biological membranes, which affect the quantity and rate of uptake and movement of nutrients and other materials within plants. Some adjuvants may create changes in cell membrane permeability or enzyme activity. Some can deter seedling germination, but the level of impact varies among plant species. Adjuvants can enter plants through their leaves, stems or roots. Plant roots tend to be extremely sensitive to adjuvants in nutrient solutions since their fine roots have no waxy cuticle layer to prevent absorption, unlike leaves and stems.

The effects of some adjuvants are subtle and transitory, but the impacts of others can be long lasting (Parr & Norman 1965). Parr (1982) reports that some surfactants produce either stimulatory or inhibitory effects on the growth and metabolic processes of biological systems, depending on the plant species, and the chemistry, concentration, and dose of the surfactant. There is typically a dose response when adjuvants are used, meaning that an adjuvant may have no effect at relatively low concentrations, be stimulating at intermediate concentrations, and toxic at high levels (Norris 1982). For instance, most surfactants work to decrease surface tension at the spray droplet-leaf cuticle interface, and this reduction is typically maximized at concentrations ranging from 0.01 to 0.1%. However, pronounced toxic effects in plants can be found once surfactant concentrations become greater than 0.1%. Some plants are stimulated to grow by nonionic surfactants when applied up to 0.001%, but these same compounds have phytotoxic effects at a mere 0.005%. Cationic surfactants repressed algal growth at concentrations of only 0.0005%, but after 2 weeks, growth was stimulated, indicating that these effects can be transitory depending on the surfactant used (Parr 1982).

Some adjuvants can have adverse effects on aquatic species, and certain types can be extremely toxic to fish and shellfish. Some adjuvants (such as the surfactant MONO818[®] in RoundUp[®]) are toxic to fish and also interfere with cutaneous respiration in frogs and gill respiration in tadpoles (Tyler 1997 a,b; Folmar et al. 1979). Parr (1982) reports that some adjuvants caused noticeable alterations in fish gill tissue, and that the toxicity of these adjuvants increased as exposure time increased. Other adjuvants can inhibit bacteria by disrupting their cell membranes (Norris 1982). Earthworms incubated in soil with a cationic surfactant, however, showed no detrimental effects even after a 90-day exposure (Bayer & Foy 1982). “Normal” environmental exposure levels of surfactants and emulsifiers to humans, however, would appear to be negligible based on the extremely high dosages that are typically necessary to cause toxic responses in mammals (Parr 1982).

TYPES OF ADJUVANTS

There are many ways to classify adjuvants, and there is currently no standard system used by all adjuvant or herbicide manufacturers. A good review of different adjuvant terms and definitions can be found in Hazen (2000) or in Van Valkenburg (1982). In this chapter, we divide adjuvants into two primary types based on their functions: activator adjuvants and utility adjuvants (Hess

1999; Kirkwood 1994). Activator adjuvants enhance the activity of the herbicide, often by increasing rates of absorption of the herbicide into the target plant(s). Utility adjuvants, which are sometimes called spray modifiers, alter the physical or chemical characteristics of the spray mixture making it easier to apply, increasing its adherence to plant surface so that it is less likely to roll off, or increasing its persistence in the environment.

When deciding which type of adjuvant to use, remember to always read and follow the directions on the herbicide label.

Activator Adjuvants

Activator adjuvants are compounds that when added to the spray tank, enhance herbicide activity (Penner 2000a). Activator adjuvants include surfactants, oil carriers such as phyto-bland (not harmful to plants) oils, crop oils, crop oil concentrates (COCs), vegetable oils, methylated seed oils (MSOs), petroleum oils, and silicone derivatives, as well as nitrogen fertilizers. Some brands of herbicide formulations already include activator adjuvants (e.g. RoundUp Ultra[®] contains the herbicide glyphosate and a surfactant, and Pathfinder II[®] which contains the herbicide triclopyr, an oil carrier which is an activator, and a dye which is a utility adjuvant).

Oils are sometimes used alone as contact herbicides and in other situations as adjuvant carriers for synthetic herbicides. Salts may also be used as activator adjuvants, often to fertilize and enhance the growth of the target plant in the short-term, which can increase the uptake and effect of the herbicide in the slightly longer term. Salt adjuvants of this type are used extensively in crop agriculture and in some rangelands, but are rarely appropriate in wildlands.

Surfactants

Surfactants are the most widely used and probably the most important of all adjuvants (Miller & Westra 1998). The name is derived from **surface active agents** and these compounds facilitate or enhance the emulsifying, dispersing, spreading, sticking or wetting properties of the herbicide tank mix (includes spray modifiers). Surfactants reduce surface tension (see Box 5) in the spray droplet, which ensures that the formulation spreads out and covers plants with a thin film rather than beading up. This facilitates herbicide absorption into the plant. Surfactants can also directly influence the absorption of herbicides by changing the viscosity and crystalline structure of waxes on leaf and stem surfaces, so that they are more easily penetrated by the herbicide (Kirkwood 1999; Coret et al. 1993).

Some herbicide formulations come with a surfactant already added, but most require the addition of a surfactant for good control results. Surfactants are generally not added to pre-emergent herbicides that are applied directly to soil (Miller & Westra 1998).

Box 5: Surface Tension

All fluid surfaces exhibit a phenomenon called “surface tension.” Surface tension results because molecules in a pure fluid are attracted strongly to each other, but molecules on the surface of a fluid are not completely surrounded by other fluid molecules and so have unmatched forces. These unmatched forces contain potential energy. Nature strives to disperse energies, and in this case works by minimizing the surface area of the fluid surface, which in turn minimizes the number of unmatched molecules and therefore minimizes potential energy. The unmatched molecular forces on the surface of the liquid also tend to form a barrier between the volume of fluid and its surroundings, much like an elastic skin.

It is surface tension that makes droplets become spherical in shape, and makes water bead up on glass. A sphere has the minimum possible surface area for a given volume of liquid. Surface tension influences the sizes of droplets in a spray, rates of evaporation, the likelihood that droplets will roll off leaves, etc. Static or equilibrium surface tension (EST) is the surface tension strength of well-established surfaces, while dynamic surface tension (DST) is the surface tension strength of new or highly disturbed surfaces, such as the surface of a newly formed spray droplet, or the surface of a droplet striking a leaf surface.

Increasing the concentration of a surfactant in a tank mix generally decreases DST, which in turn increases the probability that a droplet will adhere to a leaf and spread onto its surface, thus improving penetration of the herbicide through leaf cuticle. Adding too much surfactant, however, can sometimes negatively affect this wetting and spreading ability. For instance, some surfactants work by increasing droplet size to decrease DST, and are thus less prone to drift. If too much surfactant is added however, these larger spray droplets may roll or fall off, therefore being less likely to adhere to a leaf surface. Hence, more adjuvant does *not* necessarily translate into better control results.

Surfactants work by improving contact between spray droplets and plant surfaces, and enhance absorption by:

1. Making the spray solution spread more uniformly on the plant
2. Increasing retention (or ‘sticking’) of spray droplets on the plant
3. Increasing penetration through hairs, scales, or other leaf surface structures
4. Preventing crystallization of spray deposits
5. Slowing drying and increasing water retention in the spray droplets

The effectiveness of a surfactant is determined by environmental conditions, characteristics of the target plant, and by interactions between the surfactant and the herbicide. Surfactants contain varying amounts of fatty acids, which are compounds capable of binding to two types of surfaces, such as oil and water. It is important that the degree of solubility of the surfactant in oil or water match the solubility of the herbicide. The Hydrophilic –Lipophilic Balance (HLB) is a measure of the balance between hydrophilic (water-soluble) and lipophilic (oil-soluble) components in fatty acids (see Box 6). A surfactant’s HLB can therefore indicate the conditions under which the surfactant will perform best.

Box 6: Hydrophilic-Lipophilic Balance (HLB)

Surfactants contain both hydrophilic and lipophilic components (this is called amphiphatic). The hydrophilic-lipophilic balance (HLB) is a measure of the molecular balance of the hydrophilic and lipophilic portions of the compound. Many herbicides have an optimum surfactant HLB, and surfactants that most closely match a particular herbicide's optimum HLB will optimize the formulation's spread on and penetration into plants. Unfortunately, information about the HLB of most surfactant products is not available or hard to find, and so matching them appropriately is difficult (Green 2001).

For nonionic surfactants, the optimum surfactant HLB for a herbicide can be predicted based on the solubility of the herbicide in water (Griffen 1954 *in* Green 2001). For ionic surfactants, the HLB can be estimated by observing their dispersability in water (with no dispersion = 1 to 3; poor dispersion = 3 to 6; unstable milky dispersion = 6 to 8; stable milky dispersion = 8 to 10; translucent to clear dispersion = 10 to 13; and clear solution = 13+).

Typically, low HLB surfactants work best with water insoluble herbicides, while high (>12) HLB surfactants work best for water-soluble herbicides. For example, surfactants with a high HLB are more active with the hydrophilic herbicide glyphosate, while more lipophilic, low HLB surfactants are more active with the lipophilic quizalofop-P ester. Surfactants with intermediate HLB values are the most active with intermediately soluble nicosulfuron. Additionally, low HLB surfactants permit the formation of invert emulsions (water-in-oil). Mid- and upper-range HLB may be wetting agents or for oil-in-water emulsions, and high HLB surfactants are often used as detergents or solubilizers. On the other hand, a surfactant that is incorrectly matched may even deactivate a herbicide (Gaskin & Holloway 1997)

Nonionic Surfactants

Nonionic surfactants are the most commonly recommended and used adjuvants. Labels for most post-emergent herbicides used in wildlands that do not already contain a non-ionic surfactant often recommend the addition of one. Nonionic surfactants have no ionic charge and are hydrophilic (water-loving). They are generally biodegradable and are compatible with many fertilizer solutions. Some nonionic surfactants are waxy solids and require the addition of a cosolvent (such as alcohol or glycol) to solubilize into liquids. Glycol cosolvents are generally preferred over alcohols, as the latter are flammable, evaporate quickly, and may increase the number of fine spray droplets (making the formulation likely to drift when sprayed). The adjuvant label or MSDS should specify the active ingredient (alcohol, glycol, ether, etc.) of the adjuvant product.

Organosilicone and silicone surfactants are two types of nonionic surfactants. Organosilicone surfactants drastically reduce surface tension to the point where the herbicide droplets thin and coalesce to form a thin layer on the leaf surface (known as "superspreading"). They can even reduce surface tension to the point that some of the formulation may be able to slide through the microscopic stomatal openings on leaf surfaces. Once through the stomates however, the herbicide formulation must still penetrate the thin cuticle and cell membranes of the cells that line the cavity below the stomates.

Silicone surfactants also decrease surface tension and may allow spray solutions to penetrate the stomates. They can also make the formulation nearly impossible to wash off (rainfast) even if it rains shortly after they are applied (Green 2001; Roggenbuck et al. 1993). Silicone surfactants can also influence the amount/rate of herbicide that is absorbed through the cuticle.

Ionic Surfactants

Ionic surfactants possess either a positive (cation) or a negative (anion) charge, and can pair readily with oppositely charged herbicides, increasing the solubility of polar herbicides in water. Ionic surfactants may complex with other compounds in the mix (including contaminants in the water) in unexpected ways, and this can interfere with their function. For this reason, nonionic surfactants are more commonly recommended.

Ionic surfactants are not often used in wildland settings, but are frequently used in agriculture. The most common cationic surfactants used in agriculture may be the tallow amine ethoxylates, which are often used with glyphosate. The most common anionic surfactants are sulfates, carboxylates, and phosphates attached to lipophilic hydrocarbons.

Amphoteric Surfactants

Amphoteric surfactants contain both a positive and negative charge and typically function similarly to nonionic surfactants. A commonly used amphoteric surfactant is lecithin (phosphatidylcholine), which is derived from soybeans. There is little published research on the use and efficacy of amphoteric surfactants.

Oil Adjuvants

Oil adjuvants can increase the penetration of oil-soluble herbicides into plants, and are commonly used when conditions are hot and dry, and/or when leaf cuticles are thick. They are derived from either refined petroleum (mineral) oils or from vegetable oils (including seed oils), and do not readily mix with water. Therefore, when an oil adjuvant is combined with water in a spray tank, a surfactant emulsifier must also be added, which distributes the oil droplets (micelles) uniformly throughout the mix. These “emulsifiable oil” adjuvant combinations typically contain both a non-phytotoxic oil (typically ranging 80 to 99%) and a surfactant (1 to 20%), and are added to the spray tank usually as just 1% of the total spray volume (Hess 1999).

Emulsifiable oil adjuvant blends can enhance the absorption of an oil-soluble herbicide into the plant more than an oil adjuvant by itself. Adding a surfactant to the mixture not only emulsifies the oil in the water-based spray solution, but also lowers the surface tension of the spray solution. These adjuvants can also increase herbicide absorption through the plant cuticle, increase spray retention on leaf surfaces, and reduce the time needed for the herbicide formulation to become rainfast (Pringnitz 1998; Miller & Westra 1996). The exact mode of action of these oils is unknown, but they enhance the spread of droplets on plant surfaces (Gauvit and Cabanne 1993 *in Green* 2001). They may also split open the cuticle and increase both the fluidity of cuticular components and herbicide diffusion rates (Santier & Chamel 1996 *in Green* 2001).

Two types of emulsifiable oil adjuvants are “crop oils” and “crop oil concentrates” (COC). Crop oils contain up to 5% surfactant and COCs may contain up to 20% surfactant (Hess 1999). COCs enhance spreading and penetration and are used primarily with grass-specific herbicides (Miller & Westra 1996). Crop oils and COCs do not necessarily contain oil derived from crop plants (although some do), but are so named because they are intended for application to crops (Pringnitz 1998).

Petroleum oils

Petroleum oils or petroleum oil concentrates are highly refined oils, which are often used as carriers of oil-soluble herbicides. They are typically used in low quantities (generally 0.25 to 1 gallon/acre), and when used as carriers, can reduce surface tension, increase wetting and spreading, give quicker absorption, improve rainfastness, and reduce loss of carrier during and after application (Bohannan & Jordan 1995 *in Green* 2001).

Petroleum oil concentrates may include paraffinic and naphthalenic oils. Paraffinic oil can smooth epicuticular wax, or cause cracks in the cuticle, allowing increased herbicide penetration (Foy & Smith 1969 *in Green* 2001). Paraffinic oils are sometimes referred to as dissolving waxes, but in fact, paraffinic oils are poor solvents and only soften wax.

Vegetable oils

Vegetable-derived oils (from soybeans, cottonseeds, etc.) also decrease surface tension, but they are not as effective as other surfactants at increasing spreading, sticking, or penetration (Miller & Westra 1996). Vegetable oils are generally of two types: triglycerides or methylated oils. Triglycerides are essentially oil-surfactant hybrids, and are generally called “seed oils.” These seed oils are extracted from plants by pressing or solvent extraction, and tend to have higher viscosities than methylated oils. Triglyceride oils usually contain only 5 to 7% surfactant emulsifier, while methylated seed oils contain 10 to 20% surfactant.

Methylated seed oils (MSO) are better solvents than petroleum-based oils, but their role as a solvent of cuticular waxes is controversial. The composition of these oils varies depending on the seed source and can influence efficacy (Nalewaja 1994). Esterified seed oils are vegetable-seed oils with a surfactant or an emulsifier already added. They have good spreading and penetration properties, but tend to be more expensive than other oil adjuvants.

Ammonium (Nitrogen) Fertilizers

Ammonium, or nitrogen, fertilizers are often added to herbicide mixes in range and row-crop agriculture situations, where the addition of fertilizer works to both enhance herbicidal effects as well as to stimulate the growth of desirable crop or forage plants. Ammonium fertilizers can function as utility adjuvants, because they help prevent the formation of precipitates in the tank mix or on the leaf surface. They also decrease surface tension, increase spreading of the herbicide on the leaf surface, neutralize ionic charges, and increase herbicide penetration into the leaf (Nalewaja & Matysiak 2000). Ammonium fertilizers are used primarily with broadleaf-specific herbicides (Miller & Westra 1996; Wanamarta et al. 1993).

Ammonium fertilizers used as adjuvants include urea-ammonium nitrates (UAN), ammonium sulfates, ammonium nitrates and ammonium polyphosphates. Although their exact mode of action in herbicide control is unknown, they are often used to enhance the postemergence activity of weakly acidic herbicides, primarily by increasing herbicide absorption. The activity of ammonium fertilizers is strongly herbicide- and species-specific, and is probably dependent on several mechanisms.

Ammonium sulfates are also used to reduce antagonism by hard water ions in spray solutions. Iron, zinc, magnesium, sodium, potassium and calcium ions can react with certain herbicides (such as 2,4-D and glyphosate) to form precipitates or herbicide salts, decreasing the efficacy of those herbicides (Nalewaja and Matysiak 1993). Ammonium sulfate prevents the formation of the calcium salt of glyphosate (Thelen et al. 1995), and is recommended in most areas with hard water (Hartzler 2001).

Utility Adjuvants (including Spray Modifiers)

Utility adjuvants are added to improve the application of the formulation to the target plants. By themselves, they do not directly enhance herbicidal activity (McMullan 2000). Instead, they change the physical or chemical properties of the tank mix in ways that make it easier to apply to the target plant(s), minimize unwanted effects, and broaden the range of conditions under which a given herbicide formulation can be effective.

Most utility adjuvants are typically not used in wildland situations, since herbicides applied in wildlands are generally not applied aerially, with large booms, or in tank mixtures with several herbicides and other additives. Examples of the different types of functions that different utility adjuvants have are listed below. There is some overlap of these functional categories. Some activator adjuvants are also utility adjuvants and some even have herbicidal effects of their own.

Wetting or Spreading Agents

Wetting agents or spreading agents lower surface tension in the spray droplet, and allow the herbicide formulation to form a large, thin layer on the leaves and stems of the target plant. Since these agents are typically nonionic surfactants diluted with water, alcohol, or glycols (Hazen 2000), they may also function as activator adjuvants (surfactants). However, some wetting or spreading agents affect only the physical properties of the spray droplets, and do not affect the behavior of the formulation once it is in contact with plants.

Dyes

Dyes are commonly used for spot or boom spraying. We recommend the use of a dye for most herbicide treatments in wildlands even if applied with small handheld sprayers or wicks because the presence of a dye makes it far easier to see where the herbicide has been applied and where it has dripped, spilled or leaked. Dyes make it easier to detect missed spots and to avoid spraying a plant or area twice. It is never appropriate to use food coloring or any other substances that have not been approved or labeled by the U.S. EPA for use as herbicide adjuvants.

Drift Control & Foaming Agents

Drift control agents are designed to reduce spray drift, which most often results when fine (< 150 µm diameter) spray droplets are carried away from the target area by breezes, including those caused by the aircraft or vehicle carrying the spray equipment (Downer et al. 1998). Drift control agents alter the viscoelastic properties of the spray solution, yielding a coarser spray with greater mean droplet sizes and weights, and minimizing the number of small, easily-windborne droplets (Hewitt 1998). These agents are typically composed of large polymers such as polyacrylamides, and polysaccharides, and certain types of gums.

Foaming agents also act as drift control agents. When used with specialized nozzles, these agents create foams with different degrees of stability (Witt 2001). These foams can be placed more precisely than standard liquid sprays, and are sometimes used to mark the edge of spray applications. Foams ensure complete coverage without over-spraying. Foaming agents are usually added in quantities of 0.1 to 4.0% of the entire spray mixture (McWhorter 1982).

Thickening Agents

Thickening agents can modify the viscosity of spray solutions and are used to reduce drift, particularly for aerial applications (Witt 2001). They are used primarily in agriculture. Thickening agents may include water swellable polymers that can produce a “particulated solution,” hydroxyethyl celluloses, and/or polysaccharide gums. Viscosity can also be increased by making invert emulsions (follow directions on individual herbicide labels) of the spray solution. The compatibility of the thickening agent with the tank mix can be influenced by the order of mixing, pH, temperature, and/or the salt content of the tank solution. Thickening agents are typically used in areas where sensitive populations or crops are growing close to treated areas (McWhorter 1982).

Deposition Agents (Stickers)

Deposition agents, or stickers, are used to reduce losses of formulation from the target plants due to the droplets evaporating from the target surface, or beading-up and falling off. Spray retention on difficult-to-wet leaf surfaces is regulated by the degree of surface tension and energy dissipation during the spray process. Deposition agents such as guar gum can reduce surface tension while increasing the viscoelasticity of the droplets (Bergeron et al. 2000). Stickers keep the herbicide in contact with plant tissues by remaining viscous, and therefore resist being washed-off by rain or knocked off by physical contact. Stickers are generally the most useful with dry wettable powder and granule formulations (Hazen 2000).

Film-forming vegetable gels, emulsifiable resins, emulsifiable mineral oils, vegetable oils, waxes, and water-soluble polymers can all be used as stickers (Witt 2001). Fatty acids (technically anionic surfactants) are frequently used as stickers, and although they are “naturally derived” and are typically considered safe, they may have considerable contact activity. Certain oils may also function as stickers, but only if they have a low degree of volatility (Hazen 2000).

Water Conditioners

Water conditioners are frequently added when the water used in the formulation is high in salts in order to minimize or prevent reactions between ions in the spray solution and the herbicide, which would result in the formation of precipitates or salts. When there are many cations present, as in hard water, they can react with the herbicide, decreasing the uptake and effect of the herbicide. For instance, high levels of calcium in water (hard water) reduce the control efficacy of glyphosate (Nalewaja & Matysiak 1993). Similarly, sodium bicarbonate reduces the efficacy of sethoxydim (Matysiak & Nalewaja 1999). A water conditioner, such as ammonium sulfate (which also happens to be a nitrogen fertilizer), can negate this effect for both glyphosate and sethoxydim (McMullan 2000).

Compatibility Agents

Compatibility agents prevent chemical and/or physical interactions between different herbicides and fertilizers that could lead to non-homogeneous or unsprayable mixtures when these compounds are combined. For instance, if the herbicides bentazon and sethoxydim are mixed, they may react to form precipitates, resulting in reduced rates of sethoxydim penetration (Wanamarta et al. 1993). In most cases, the herbicide label will state which herbicides may or may not be mixed together.

Some herbicides are applied with fertilizers or fertilizer solutions, especially in agricultural settings. Compatibility agents are used to keep these herbicides in suspension, and are generally added with a liquid fertilizer (Witt 2001). Most herbicides can be applied in nitrogen solutions without any compatibility problems, but compatibility may be poor when the water contains high levels of various salts (hard water), or when the water is unusually cool. When 2,4-D is applied with liquid-nitrogen fertilizers the solution may separate even if mixed vigorously unless a compatibility agent is added to the mix.

pH Buffers

pH plays a large role in herbicide efficacy. The pH of the tank mix affects the half-life solubility and efficacy of the herbicide, and may determine whether or not precipitates form (McMullan 2000). Being able to buffer or otherwise control changes of pH in the tank mix can be important in preventing herbicides from being degraded by acid or base hydrolysis in aqueous solutions. Some herbicides are sold with a pH buffer already included. Adjuvants that adjust or buffer pH can also improve the herbicide's dispersion or solubilization in the mix, control its ionic state, and increase tank-mixture compatibility. pH buffers are most beneficial when used in extremely alkaline or acid water, which could otherwise have detrimental effects on the herbicide's performance (McWhorter 1982).

Humectants

Humectants, like stickers, increase the amount of time that the herbicide is on the leaf, in a form available for uptake (Hazen 2000). When water evaporates from the spray droplet and the herbicide becomes a crystalline residue, it is no longer available for uptake into the leaf. Humectants keep the spray deposit moist and in true solution, and therefore extend the time that it is available for absorption (Hess 1999). They are generally water-soluble and increase the water content of spray deposits by slowing the drying time or by drawing moisture from the environment. Commonly used humectants include glycerol, propylene glycol, diethylene glycol,

polyethylene glycol, urea, and ammonium sulfate. Even glucose and molasses were used as humectants in the past, but they are not labeled for such use and should not be added to any herbicide formulation.

Defoaming and Antifoam Agents

Defoaming and antifoam agents reduce or suppress the formation of foam in spray tanks (Witt 2001). Many spray mixtures have a tendency to foam excessively, especially when mixed with soft water, which can cause problems during mixing (foam overflow) or when rinsing the sprayer (McMullan 2000). Most defoamer agents are dimethopolysiloxane-based, but silica, alcohol, and oils have also been used for this purpose. Defoaming agents can reduce surface tension, physically burst the air bubbles, and/or otherwise weaken the foam structure. In general, it is easier to prevent foam formation than to eliminate foam after it forms (Green 2001). Antifoam agents are usually dispensed from aerosol cans or plastic-squeeze bottles, and are added directly to the mix at the onset of foam formation. The highest concentration needed for eliminating foam is typically about 0.1% of the entire tank. Some applicators in agricultural settings even use kerosene or diesel fuel at about 0.1% for eliminating foam in spray tanks, but this is not recommended in natural areas.

UV Absorbents

Natural sunlight, especially ultraviolet light, may degrade some herbicides (Green 2001). A few adjuvants that protect herbicides from the deleterious effect(s) of sunlight are available. They may do this by either physical or chemical processes, such as by increasing the rate of herbicide uptake into the cuticle, or by absorbing the UV-light themselves.

A FEW EXAMPLES OF COMMONLY USED HERBICIDES AND ADJUVANTS IN NATURAL AREAS

The choice of herbicide and adjuvant to be used will depend on the target weed, site and environmental conditions, cost of chemicals, and in some cases, on state regulations. The herbicides and adjuvants listed below are not necessarily examples of the best combinations to use, but these mixes have been used in a few natural areas with some success. Examples are given only for glyphosate and triclopyr, and contact information for the mentioned land managers follow these examples.

GLYPHOSATE

RoundUp Pro[®]

Andropogon virginicus (broomsedge), *Paspalum conjugatum* (buffalograss), *Melinis minutiflora* (molasses grass) and *Setaria palmifolia* (palmgrass)

Pat Bily (TNC-Hawaii) used a 2% solution of RoundUp Pro[®] with water-soluble packets of blue Turfmark[®] dye for foliar applications in Hawaii. A surfactant was already included in the RoundUp Pro[®] formulation so there was no need to add any other adjuvants.

Panicum repens (torpedo grass) and *Urochloa distichya* (Tropical signalgrass)

Mike Renda and Jovan Dodson (TNC-Florida) used a 2% solution of RoundUp Pro[®] with SunEnergy[®] surfactant (applied at 1 oz/gallon) for foliar applications.

Rodeo[®]

Phragmites australis (common reed) and *Rosa multiflora* (multiflora rose)

Curtis Hutto (Virginia Dept. of Cons. & Rec.) reports a 90% kill rate for common reed and multiflora rose using a 2% solution of Rodeo[®] with 0.5% TL-90[®] non-ionic surfactant, applied with a backpack or ATV-mounted sprayer. Curtis adds that it will take 2 successive fall applications to multiflora rose to achieve a 90% mortality rate. It takes 2 or 3 applications to get a 90% kill rate on common reed.

Mimosa pigra (catclaw mimosa), *Lygodium japonicum* (Japanese climbing fern), *Panicum repens* (torpedo grass), *Paederia foetida* (skunkvine), *Lantana camara* (lantana), *Solanum viarum* (tropical soda apple) and *Imperata cylindrica* (cogon grass)

Michael Jenkins (Florida Park Service) reports excellent control (>95% kill) results with a 4% solution of Rodeo[®] plus a 0.3% solution of either Silken[®] or Kinetic[®] organosilicone surfactant to catclaw mimosa foliage. He also reports excellent control on Japanese climbing fern, torpedo grass, skunkvine, lantana, and tropical soda apple with a 2.5% solution of Rodeo[®] plus a 0.3% solution of Silken[®] or Kinetic[®]. He has also controlled cogon grass using a 1% solution of Rodeo[®] with 0.3% solution of Silken[®] or Kinetic[®], applying it on foliage in late fall.

Phalaris arundinacea (reed canarygrass)

Mandy Tu (TNC-Oregon) reports good control of reed canarygrass by first mowing in late spring-early summer at the onset of flowering, then applying a foliar spray of Rodeo[®] in a 2% solution with either 0.5% Bio-88[®] or R-11[®] nonionic surfactant in fall, before the first frost. The formulation can be applied with a backpack sprayer or an ATV with a boom attachment.

Typha spp. (cattails)

Russ McClain (TNC-West Virginia) reports near 100% kill of cattails in West Virginia by combining 2.5 gallons Rodeo[®], 1 quart Surf-Ac 820[®] nonionic surfactant plus Blazon[®] blue turf dye and 7.25 gallons of water to make 10 gallons of tank mix. Since cattails often grow in sensitive wetland areas, Russ recommends applying the formulation using the “bloody glove” or “glove of death” (herbicide soaked cotton gloves worn over rubber or nitrile gloves, and stroked over the target weed leaf surfaces) technique for minimal off-target effect.

Accord[®]

Hypericum perforatum (St. Johnswort), *Lythrum salicaria* (purple loosestrife), and *Phalaris arundinacea* (reed canarygrass)

Jack McGowan-Stinski (TNC-Michigan) uses Accord[®] herbicide in a 2.5% a.i. with Hi-Light Dye[®] tablets (1 tablet per gallon mix) for the control of St. Johnswort. He applies the formulation to St. Johnswort foliage by either wicking using a modified exterior sponge PVC adapted to a Solo[®] backpack sprayer, or by using a backpack sprayer. For purple loosestrife and reed canarygrass, he first cuts the stems then applies Accord[®] in a 5% a.i. solution with the Hi-Light Dye[®], and applies the mix using either a backpack sprayer or a sponge wicking applicator to the stem and cut surface. Jack adds that the sponge wicking applicator gives extremely targeted applications with minimal off-target effects (see Appendix 1 for details on how to construct one of these applicators).

Rhamnus frangula (glossy buckthorn)

Jack McGowan-Stinski (TNC-Michigan) controls buckthorn shrubs using a cut-stump herbicide treatment. He first cuts each stem 6 inches above the ground surface, and within at most 5 minutes, applies Accord[®] in a 14% a.i. mix directly to that cut surface using a sponge-tipped applicator (see Appendix 1 for more details). He has also controlled buckthorn by wicking a 5% a.i. Accord[®] mix to the foliage with a specially made PVC tube tipped with a sponge applicator and connected to a Solo[®] backpack sprayer. Accord[®] can also be sprayed onto foliage using a 2% a.i. mix.

TRICLOPYR

Garlon 3A[®]

Polygonum cuspidatum (Japanese knotweed)

Jonathan Soll (TNC-Oregon) reports near 100% kill of knotweed using a 3 to 5% solution of Garlon 3A[®] with 1 oz/gallon Hasten[®] ethylated seed oil. For treatments near water, he uses a 3-5% solution of Garlon 3A[®] with 1 oz/gallon of R-11[®] nonionic surfactant. Jonathan recommends first cutting the stems in spring, then foliar spray the regrowth with a backpack sprayer in fall.

Foeniculum vulgare (fennel)

Bob Brenton and Rob Klinger (UC Davis) report near 95% kill of fennel in California by using 1 lb a.i./acre of Garlon 3A[®] with a 0.25% solution of Pro-Spreader[®] activator nonionic surfactant. They recommend using a backpack sprayer to apply to foliage in early spring.

Dioscorea bulbifera (air potato)

Michael Jenkins (Florida Park Service) reports good control of air potato with a 2.5% solution of Garlon 3A[®] plus a 0.3% solution of either Kinetic[®] or Silken[®] surfactant, applied as a foliar spray onto leaves.

Rosa multiflora (multiflora rose), *Elaeagnus umbellata* (autumn olive) and *Ailanthus altissima* (tree of heaven) Curtis Hutto (Virginia Dept. of Cons. & Rec.) applied undiluted Garlon 3A[®] with no additional adjuvant, to cut stems of multiflora rose and autumn olive and achieved 100% mortality for those species. He found that the season of application did not matter for these species. He has also used undiluted Garlon 3A[®] with no adjuvant on tree of heaven, using a girdle and squirt (cut into bark with a girdling knife, squirt in herbicide using a spray bottle) technique which caused about 95% mortality.

Wedelia trilobata (trailing daisy)

Mike Renda and Jovan Dodson (TNC-Florida) report moderate control of trailing daisy using repeated treatments of a 2% solution of Garlon 3A[®] with 1 oz/gallon CideKick II[®] surfactant. They also add TurfMark[®] dye (1 to 2 oz/gallon) for these foliar treatments.

Tibouchina herbacea (glorybush) and *Ulex europaea* (gorse)

Pat Bily (TNC-Hawaii) controls these two invasive species in Hawaii using a 2% solution of Garlon 3A[®] combined with a 0.2% solution of Breakthru[®] organosilicone surfactant as a foliar spray. Pat adds that he obtains similar success by using either Sylwet L-77[®] or Sylgard[®] surfactants, applied using the same concentrations.

Garlon 3A[®] or Garlon 4[®]

Senna pendula (climbing cassia), *Colubrina asiatica* (Asiatic colubrina), *Schinus terebinthifolius* (Brazilian peppertree), *Casuarina equisetifolia* (Australian pine), and *Cupaniopsis anacardioides* (Carrotwood)

Mike Renda and Jovan Dodson (TNC-Florida) have also had excellent control of these woody invaders by using either a cut-stump treatment with a 50% solution of Garlon 3A[®] (in water), or a basal bark treatment with 10% Garlon 4[®] mixed with 90% JLB[®] oil solution. For both types of treatments, no other surfactants were used, but Turfmark[®] dye was added at a rate of 1 to 2 oz/gallon tank mix.

Garlon 4[®]

Rhamnus cathartica (common buckthorn)

Bill Kleiman (TNC-Illinois) reports good control results on common buckthorn with a solution of 20% Garlon 4[®] and 80% mineral oil using the basal bark application technique. He also adds Basal Red[®] dye at 3 oz/15 gallons to the tank mix.

Garth Fuller & Colin McGuigan (TNC-Minnesota) also report good control of buckthorn, but they use a cut-stump treatment using a solution of 25% Garlon 4[®] with 75% Diluent Blue[®].

Tamarisk spp. (salt cedar, tamarisk)

Ian Torrence (National Park Service- Utah) reports good kill rates for salt cedar by using two different treatments and concentrations of Garlon 4[®]. He reports a 90 to 95% kill rate for a basal bark spray of 20% Garlon 4 in 80% JLB Oil Improved Plus[®] applied with a low-volume backpack sprayer. He reports a 80 to 85% kill rate using a cut-stump treatment with 25% Garlon 4[®] to 75% JLB Oil Improved Plus[®]. Ian reports good control with trees up to 6 inches in diameter using the basal bark method. For larger trees with thicker bark, Ian recommends the cut-stump method, where the tree is first cut at its base and herbicide immediately applied to the cut surface (using squirt bottles or brushes), especially to the outer cambium layer. Ian adds that JLB Oil Improved Plus[®] oil comes with a red dye already mixed in.

CONTACTS

Pat Bily, Invasive Plant Specialist
The Nature Conservancy-Maui Project, Hawaii
808-572-7849
pbily@tnc.org

Bob Brenton
brenvms@sbcglobal.net

Curtis Hutto, State Natural Area Steward
Virginia Department of Conservation and Recreation
804-692-0479
cjhutto@dcr.state.va.us

Michael Jenkins, OPS Exotic Removal
Florida Park Service
Michael.R.Jenkins@dep.state.fl.us

Bill Kleiman, Nachusa Restoration Ecologist
The Nature Conservancy-Nachusa Grasslands Preserve, Illinois
815-456-2340
bkleiman@tnc.org

Russ McClain, Conservation Ecologist
The Nature Conservancy- West Virginia Chapter
304-637-0160
rmclain@tnc.org

Jack McGowan-Stinski, West Michigan Land Steward
The Nature Conservancy-West Michigan Program Office
616-776-0230
jmcgowan-st@tnc.org

Colin McGuigan, Central Minnesota Land Steward
The Nature Conservancy-Minnesota Field Office
612-868-5038
cmcguigan@tnc.org

Mike Renda and Jovan Dodson, Restoration Coordinator and Restoration Assistant
The Nature Conservancy-Blowing Rocks Preserve, Florida
561-744-6668
mrenda@tnc.org or jdodson@tnc.org

Jonathan Soll, Portland Area Stewardship Ecologist
The Nature Conservancy-Oregon Field Office
503-230-1221
jsoll@tnc.org

Ian Torrence, Vegetation Manager
National Park Service, Southeast Utah Group
435-719-2137
Ian_Torrence@nps.gov

ADJUVANT FAQs and TIPS:

Q: Are adjuvants necessary for good control results?

Adjuvants are necessary for best control results in most herbicide applications. Some brands of herbicide already include adjuvants and no others are needed.

Q: If adding adjuvants or surfactants at labeled rates can lead to increased rates of control efficacy...should I add more to get even better performance?

No! Do not add any more adjuvant than amounts specified on the label. Adding more adjuvant may lead to antagonistic effects between the adjuvant and the herbicide, rendering the mix useless. Using adjuvants above label rates may also cause unwanted damage to non-target plants, soils, and to surface or groundwater sources.

Q: Where do I find relevant information about herbicide and adjuvant compatibility?

The herbicide label or MSDS will specify the best type of adjuvant to use with that herbicide. It will also specify whether that herbicide can be mixed with any other herbicides and which ones.

Q: Are surfactants ok to use in wetland or aquatic situations?

Some surfactants (such as those included in RoundUp[®]) are toxic to fish, shellfish, and/or other aquatic invertebrates. When applying herbicides to areas over or adjacent to water (including wetlands), be sure to use only those herbicides and surfactants (and other adjuvants) specifically approved for aquatic use. In general, adjuvants (particularly surfactants) will not improve herbicide effectiveness against submerged aquatic weeds, but they may be important for use on emergent aquatic and riparian plants.

Q: Are surfactants necessary in cut-stump applications?

It is probably not necessary to use a surfactant in most cut-stump applications. This may be, in part, because there is no waxy cuticle layer on a cut stump.. Jack McGowan-Stinski (TNC-Michigan) has had success using herbicides without surfactant (e.g., Rodeo[®] instead of RoundUp[®]) and stresses the importance of applying the herbicide to the stump a short time after it is cut; best if no more than 5 minutes. Jonathan Soll (TNC-Oregon) notes that whether you need to add a surfactant depends on what you are trying to kill. In most cases, a general nonionic surfactant will suffice if the herbicide beads-up on the surface of the stem. If the cut stumps of the plant you are treating exude an oily substance, use an oil-type of surfactant for good control.

Q: Is it OK to add impure water into the tank mix? Can I use pond water, salt water, or water from a well for making the tank mix?

Wherever possible, use pure, clean, moderate-temperature water in your tank mix. Pond water may contain soil particles that may adsorb to and render some herbicides or adjuvants useless, and water that is too cold may cause the herbicide to precipitate out of solution. Good quality well water may be used, but if it contains high concentrations of ions (hard water - calcium, magnesium, etc.) or salts, try to find purer water (unless a buffering adjuvant is also used). Well water can be tested locally for impurities. Do not use salt water because the salts and ions it contains may create antagonistic effects with the herbicide, the adjuvants, or both, rendering the mix worthless.

Q: Can I use food coloring instead of a registered dye?

No! Food colorings are not registered for use with herbicides, and therefore should not be used as a dye in herbicide mixes.

Q: Will the adjuvant decrease in effectiveness if I don't use it up right away?

In general, if adjuvants (as well as most herbicides) are stored under appropriate conditions (as specified on the label), they are relatively stable compounds and can be stored and used successfully for some time. For instance, the herbicide hexazinone is stable for at least two years, and glyphosate can be stored for at least five years. Most adjuvants do not include shelf-life information on the label, but may have use-by dates on the container.

REFERENCES

- Bayer, D.E. and C.L. Foy. 1982. Action and fate of adjuvants in soils. *In: Adjuvants for Herbicides*, WSSA, Champaign, IL. Pp. 84-92.
- Bergeron, V., Bonn, D., Martin, J.-Y. and L. Vovelle. 2000. Controlling droplet deposition with polymer additives. *Nature* 405: 772-775.
- Carroll, B. 2001. Selecting the right adjuvant. Helena Chemical Company's Guide to Adjuvants. <http://www.helenachemical.com/proprietary/products/adjuvants/introduction.htm#>
- Coret, J., Bambonnet, B., Brabet, F. and A. Chamel. 1993. Diffusion of three ethoxylated octylphenols across isolated plant cuticles. *Pesticide Science* 38: 201-209.
- DiTomaso, J.M. 1999. Barriers to foliar penetration and uptake of herbicides. *Proceedings of the California Weed Science Society* 51: 150-155.
- Downer, R.A., Mack, R.E., Hall, R.F. and A.K. Underwood. 1998. RoundUp Ultra with drift management adjuvants. *In: McMullan, P.M. (ed.) Adjuvants for Agrochemicals: Challenges and Opportunities*. Proceedings of the Fifth International Symposium on Adjuvants for Agrochemicals, Chemical Producers Distributors Association, Memphis, TN. Pp. 468-474.
- DuPont. 2000. Guidelines to qualify adjuvant for use with DuPont row crop and cereal herbicides. DuPont Agricultural Bulletin H-87285.
- DuPont. 2001. 2001 Approved adjuvant list for use with DuPont row crop and cereal herbicides. DuPont Agricultural Bulletin H-91222.
- Folmar, L.C., H.O. Sanders, and A.M. Julin. 1979. Toxicity of the herbicide glyphosate and several of its formulations to fish and aquatic invertebrates. *Arch. Environ. Contam. Toxicol.* 8:269-278.
- Gaskin, R.E. and P. Holloway. 1992. Some physicochemical factors influencing foliar uptake enhancement of glyphosate-mono(isopropylammonium) by polyoxyethylene surfactants. *Pesticide Science* 34: 195-206.
- Gaskin, R.E. and P.J.G. Stevens. 1993. Antagonism of the foliar uptake of glyphosate into grasses by organosilicone surfactants. *Pesticide Science* 38: 185-200.
- Green, J.M. 2001. Herbicide adjuvants. *In: UC Davis WRIC Weed Science School*, September 26-28, 2001, Woodland, CA.
- Green, J.M. 1992. Increasing efficiency with adjuvants and herbicide mixtures. *Proceedings of the First International Weed Control Congress*, Melbourne, AU. Pp. 187-192.
- Green, J.M. 2000. Adjuvant outlook for pesticides. *Pesticide Outlook*. October: 196-199
- Green, J.M. and J.L. Hazen. 1998. Understanding and using adjuvants properties to enhance pesticide activity. *In: McMullan, P.M. (ed.) Adjuvants for Agrochemicals: Challenges and*

- Opportunities. Proceedings of the Fifth International Symposium on Adjuvants for Agrochemicals, Chemical Producers Distributors Association, Memphis, TN. Pp. 25-36.
- Hartzler, B. 2001. Role of AMS with glyphosate products. Iowa State University Extension Agronomy. <http://www.weeds.iastate.edu/mgmt/2001/ams.htm>
- Hazen, J.L. 2000. Adjuvants - Terminology, classification, and chemistry. *Weed Technology* 14: 773-784.
- Hess, F.D. 1999. Surfactants and additives. Proceedings of the California Weed Science Society 51: 156-172.
- Hess, F.D. and C.L. Foy. 2000. Interaction of surfactants with plant cuticles. *Weed Technology* 14: 807-813.
- Hewitt, A.J. 1998. The effect of tank mix and adjuvants on spray drift. *In: McMullan, P.M. (ed.) Adjuvants for Agrochemicals: Challenges and Opportunities. Proceedings of the Fifth International Symposium on Adjuvants for Agrochemicals, Chemical Producers Distributors Association, Memphis, TN. Pp. 451-462.*
- Hull, H.M., Davis, D.G. and G.E. Stolzenberg. 1982. Action of adjuvants on plant surfaces. *In: Adjuvants for Herbicides, WSSA, Champaign, IL. Pgs. 26-67.*
- Kirkwood, R.C. 1999. Recent developments in our understanding of the plant cuticle as a barrier to the foliar uptake of pesticides. *Pesticide Science* 55: 69-77.
- Kudsk, P. and J.C. Streibig. 1993. Formulation and adjuvants. *In: Strigib, J.C. and P. Kudsk (eds.) Herbicide Bioassays. CRC Press, Boca Raton, FL. Pp. 99-116.*
- Matysiak, R. and J.D. Nalewaja. 1999. Temperature, adjuvants, and UV light affect sethoxydim phytotoxicity. *Weed Technology* 13: 94-99.
- McMullan, P.M. 2000. Utility adjuvants. *Weed Technology* 14: 792-797.
- McWhorter, C.G. 1982. The use of adjuvants. *In: Adjuvants for Herbicides, WSSA, Champaign, IL. Pgs. 10-25.*
- Miller, P. and P. Westra. 1996. Herbicide surfactants and adjuvants, no. 0.559. Colorado State University Cooperative Extension, Production Crop Series.
- Miller, P. and P. Westra. 1998. How surfactants work, no. 0.564. Colorado State University Cooperative Extension, Crop Fact Sheet. <http://www.ext.colostate.edu/pubs/crops/00564.html>
- Nalewaja, J.D. 1994. Esterified seed oil adjuvants. North Central Weed Science Society Proceedings 49: 149-156.
- Nalewaja, J.D. and R. Matysiak. 1993. Optimizing adjuvants to overcome glyphosate antagonistic salts. *Weed Technology* 7: 337-342.
- Nalewaja, J.D. and R. Matysiak. 2000. Spray deposits from nicosulfuron with salts that affect efficacy. *Weed Technology* 14: 740-749.
- Norris, R.F. 1982. Action and fate of adjuvants in plants. *In: Adjuvants for Herbicides, WSSA, Champaign, IL. Pgs. 68-83.*
- Parr, J.F. 1982. Toxicology of adjuvants. *In: Adjuvants for Herbicides, WSSA, Champaign, IL. Pgs. 93-114.*
- Parr, J.F. and A.G. Norman. 1965. Considerations in the use of surfactants in plant systems: A review. *Botanical Gazette* 126(2): 86-96.
- Penner, D. 2000a. Activator adjuvants. *Weed Technology* 14: 785-791.
- Penner, D. 2000b. Introductory statement on adjuvants. *In: Young, B. Compendium of Herbicide Adjuvants, 5th edition. Southern Illinois University, Carbondale.*
- Pringnitz, B. 1998. Clearing up confusion on adjuvants and additives. Iowa State University Extension Agronomy. <http://www.weeds.iastate.edu/mgmt/qtr98-2/cropoils.htm>

- Roggenbuck, F.S., Penner, D., Burow, R.F. and B. Thomas. 1993. Study of the enhancement of herbicide activity and rainfastness by an organosilicone adjuvant utilizing radiolabelled herbicide and adjuvant. *Pesticide Science* 37: 121-125.
- Swisher, E.M. 1982. Adjuvant regulation and registration. *In: Adjuvants for Herbicides*, WSSA, Champaign, IL. Pgs. 115-118.
- Thelen, K.D., Jackson, E.P. and D. Penner. 1995. The basis for the hard-water antagonism of glyphosate activity. *Weed Science* 43: 541-548.
- Tyler, M.J. 1997a. Herbicides kill frogs. Newsletter of the declining amphibians population task force #21.
- Tyler, M.J. 1997b. Environmentally friendly: A false sense of security? *Species*. Newsletter of the Species Survival Commission, IUCN, The World Conservation Union. 29:20-21.
- Underwood, A.K. 2000. Adjuvant trends for the new millennium. *Weed Technology* 14: 765-772.
- Van Valkenburg, J.W. 1982. Terminology, classification, and chemistry. *In: Adjuvants for Herbicides*, WSSA, Champaign. Pgs. 1-9.
- Wanamarta, G., Kells, J.J. and D. Penner. 1993. Overcoming antagonistic effects of sodium bentazon on sethoxydim absorption. *Weed Technology* 7: 322-325.
- Witt, W.W. 2001. Adjuvants. University of Kentucky College of Agriculture, Agripedia. <http://www.ca.uky.edu/agripedia/pls404/adjuvant.htm>
- WSSA. 1982. *Adjuvants for Herbicides*. Weed Science Society of America, Champaign. 144 pgs.
- Young, B. 2000. Compendium of herbicide adjuvants. Southern Illinois University, Carbondale. 66 pgs.
- Zollinger, R.K. 2000. Extension perspective on grower confusion in adjuvant selection. *Weed Technology* 14: 814-818.

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APPENDIX 1: CUT-STUMP HERBICIDE APPLICATOR

Designed by Jack McGowan-Stinski, The Nature Conservancy – Michigan Chapter

PARTS

- 1 – 1 inch diameter PVC threaded male cap
 - 1 – 1 inch diameter PVC threaded female cap
 - 1 – ¾ inch diameter PVC cap, unthreaded
 - 1 – 1 inch diameter PVC threaded female coupling
 - 3 – 1 inch diameter PVC threaded male coupling
 - 1 – 1 inch diameter PVC 45° elbow coupling, unthreaded
 - 1 – 1 inch diameter PVC threaded ball valve
 - 1 – 1 inch diameter PVC pipe (12 to 15 inches)
 - 2 – 1 inch diameter PVC pipe pieces, approximately 1 inch long
 - 4 – 1 ¼ inch diameter rubber lavatory gaskets
- heavy duty sponge (2 x 4 x 1 ½ inches)
PVC cement
PVC pipe cutters or hacksaw
Drill, 1/16 inch bit, ¾ inch bit
Ruler
Scissors

ASSEMBLY INSTRUCTIONS

Cement threaded male coupling onto one end of a length of PVC pipe (12 to 15 inch length suggested). Cement the threaded female coupling onto the other end of the pipe (reservoir). Additional PVC sections can be thread together to make a longer handle or reservoir when needed. Slip one rubber gasket over a threaded male cap and attach it to the threaded female end of reservoir. Slip one rubber gasket over threaded male end of reservoir, and attach one end of a threaded ball valve. The rubber gaskets will allow the sections of applicator to be tightened together snugly so that no herbicide will leak out around coarse PVC threads.

To make the “drip holes” for herbicide, cut off the bottom of the ¾ inch diameter PVC cap so that a flat disk remains. File disk until it fits snugly into the unthreaded 1 inch diameter PVC 45° elbow coupling. A ridge inside the elbow will keep the disk centered. Use a 1/16 inch drill bit to make two holes near the center of the disk. Cement the disk inside one end of the elbow coupling.

Using the 1 inch diameter PVC pipe pieces (1 inch length or less), cement 1 inch diameter threaded male couplings onto each end of the elbow. Slip rubber gaskets over each threaded male coupling. The end of the completed elbow without the drip holes disk attaches to the other end of the ball valve.

Drill a ¾ inch hole into the end of the 1 inch diameter PVC threaded female cap. The sponge tip twists into this ¾ inch hole, and this cap is then threaded onto the end of the elbow with the drip holes disk.

The sponge tip, which is roughly 1 inch diameter by 1 ½ inch length, can be cut with scissors, or a 1 inch diameter metal pipe section that is sharpened on one end can be used to rapidly cut out numerous sponge tips. Wet the sponge tip before twisting it into threaded female cap with the ¾ inch hole. Allow ¼ to ½ inch of sponge to extend out of tube to treat stump tops.

TO USE

With ball valve in the “OFF” or “CLOSED” position, pour the herbicide mix into the reservoir and close it with the threaded male cap (the top of applicator). Open the ball valve then slightly open the threaded male cap to allow air into the reservoir. Once the sponge tip begins to saturate, tighten the threaded male cap and close the ball valve. When the sponge is saturated, only a light touch to a cut-stump is needed. Open the ball valve when more herbicide is needed in the sponge tip.

HELPFUL HINTS

- During colder weather the ball valve may have to be left open to allow enough herbicide to saturate the sponge. Drip holes also can be made larger if faster herbicide flow is desired.
- Do not allow left-over herbicide mix to remain in the reservoir in extreme temperatures.
- Always clear drip holes of any residue before using the applicator again. A paper clip works well for cleaning out residues.
- When the sponge becomes worn, replace it (recommended after every work day at a minimum).
- When using the applicator during freezing conditions, duct tape a disposable chemical hand warmer around the section with the drip hole disk to reduce the chance of drip holes freezing shut.
- Use an herbicide dye to check for leaks, monitor applications, and identify any exposure to the person using the applicator.

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APPENDIX 2: SPOT-BURNING USING PROPANE TORCHES

Adapted from Jack McGowan-Stinski, Land Steward
The Nature Conservancy, Michigan Chapter

Spot-burning means burning individual plants or groups of plants (or a small area) using a propane torch or similar device. These torches can also be used to ignite brush piles. Two advantages to spot-burning are: 1) the torches can be used in areas where there is little or no fine fuel to carry a prescribed burn; the primary fuel source is propane, and 2) these torches can be used during wet conditions to kill invasives.

EQUIPMENT SET-UP:

The torch we use in Michigan is made by Flame Engineering, Inc.(1-800-255-2469), and is the "VT 3-30C Red Dragon Vapor Torch Kit". This torch has a maximum output of 500,000 BTU/hr, with maximum flame temperature of 2,050 - 2,075 °F. Under normal operating pressure about 10 lbs. propane/hr will be used, with maximum consumption at maximum operation of about 23 lbs./hr. Burn time when using a full 20 lb. propane tank is about 2 to 2.5 hours for spot-burning; burn time is 1 hour with 10 lb. tank. This VT 3-30C Red Dragon Vapor Torch has a 3 inch diameter bell (torch tip) and is used for burning large areas; we use a VT 2 ½-2C with a 2 inch diameter bell for more precise burning (burning individual invasive plants next to rarities).

Propane tanks (10 or 20 lb.) are carried on exterior-frame aluminum backpacks (shoulder straps plus waist belt). The packs are modified by attaching a base that the tanks fit into; the base is made out of 1/2 inch angle aluminum, which is bolted to backpack, and the bottom of the tank is bolted to this aluminum base with wingnuts, which provides a stable support and ease in changing tanks. A torch kit includes a 10 foot gas line; we shorten to 5 feet to allow freedom of movement but reduce snagging on brush while torching.

Use a carrying case/safety case for torch and gas line when transporting to site or into preserve. The case is made of a 4.5 foot 4 inch diameter section of PVC (schedule 3 plastic "sewer-and-drain" pipe) with a cap cemented on bottom and a cap (uncemented) on top. Extra o-rings for the torch-to-tank coupling are duct-taped inside top cap. Bags strapped on to case include wrench, flint lighter, soapy water (in saline solution squeeze bottle; to test for gas leaks at connections and supply hose). The case is "strapped" onto backpack so you are "hands-free" when walking into site. Approximate backpack weight of torch, carrying case, and propane tank (using a full 20 lb. tank) is 48 lbs.

Spot-burning Equipment:***Communication:***

- Burn prescription copy
- Radios with chest holders, and ear phones
- Checklists, emergency numbers
- Cellular phone
- Weather kit(s)

Ignition:

- Propane tanks, 10 and 20 lb.
- modified exterior-frame aluminum backpacks, straps and/or bunji cords
- Propane torches in PVC carrying cases, each with soap mix, wrench, flint lighter, extra o-rings

PPE:

- Nomex firesuits
- Leather gloves
- Leather boots, 8 inches high, Vibram sole, leather laces
- Hard hats with Nomex ear/neck protectors, face shields, chin straps
- First aid kit
- Water cooler with 1 liter water bottles, minimum 2 per crew member
- Belt and pouches to carry water bottles

Water-related and Holding:

- Backpack pumps
- Replacement parts for backpack pumps
- “Slip-on” pump unit includes: 110 gallon water tank cabled onto wooden cradle and bolted to truck frame, Honda water pump, manifold valving system with pressure gauge and recirculation hose, garden hose with combination adjustable nozzle and short section 1 ½ inch hose with combination adjustable nozzle, laminated directions for “Opening/Closing” valves
- Portable water pumps (Honda)
- 1 gal fuel can w/premium unleaded, 10W30 oil, drafting bottle for pumps
- Garden hose
- Intake or suction hose with foot valves
- Combination Nozzles
- Grass/thatch Rakes

Some safety tips:

- Crew is outfitted in fire safety gear (nomex, hard hat with face shield and nomex ear/neck protector, leather gloves, leather boots).
- When attaching tank to backpack make sure pressure relief valve on top is pointing AWAY from person carrying pack. This valve is designed to release pressure/vent gas if tank heats up too much, and this vented gas can ignite, forming a "torch" of its own. I suggest that anybody using these torches “talk safety” with a local propane dealer.
- The torch tip will heat up very quickly, and retain heat, so always be cautious where you set torch down; I recommend cooling down with water where possible.
- These torches produce lots of noise. For spot-burning we wear ear plugs; for prescribed burning the igniter uses an ear phone attached to fire radio, PLUS the igniter should always keep an eye out for visual signs.
- These torches should only be used by somebody who has both some fire training AND common sense, or you will end up with injuries, damage rarities, and/or start a

wildfire (and if you do not believe these torches produce a lot of heat, try melting a golf ball – it does not take long!).

- Do not turn your back and backpack towards open flame (remember that you are carrying an explosive on your back...), and do not set tank down near fire or in recently burned area.

Removal Method for Seedling Buckthorn (*Rhamnus* spp.)

Once adult buckthorn have been removed from an area it is likely that large numbers of seedlings will germinate in the area during the next growing season. There also will likely be saplings that were not herbicided, and/or some resprouts from cut-stumps that missed treatment.

Hand-pulling of seedlings is labor- and time-intensive and not always effective. For example, on one work day in June 1996 it took eight volunteers two hours to remove the 125,000+ seedlings from an area approximately 5m x 10m, and this area was soon revegetated with *Rhamnus* seedlings from the disturbed seed bank and missed seedlings.

A more efficient and effective method to kill buckthorn seedlings is to burn them in the first growing season after (non-growing season) removal.

Before work day: Obtain a burn permit from local fire department. Recruit burn crew. Other law enforcement and neighbor notifications occur as necessary. Equipment is checked, propane tanks filled.

During work day: Weather is taken on-site before and during operation. Area to be cleared is defined and checked for fire hazards (wildfire potential, poison sumac/ivy). In fen areas a portable water pump with hose is usually positioned at a seep near work area and used to wetline if necessary, or more frequently used during mop-up. In uplands a slip-on pumper is used. Water backpacks are also used as needed.

A work crew consists of some individuals operating torches (torchers) while others (spotters) monitor progress, wildfire potential, and safety hazards. The crew rotates duties frequently. In large dense seedling patches torchers position themselves in a parallel line and walk slowly while burning in an overlapping pattern; usually only one spotter is needed with this procedure. It is also easier to be non-selective and burn everything except rarities, and let area seed in naturally, or plant with native seed. In areas with scattered buckthorn seedling patches the crew works in teams of torcher and spotter (usually with water backpack.). A “heat shield” (section of metal ductwork) can be used to separate target from non-target species if needed.

The maximum flame temperature occurs 6 to 12 inches from torch bell tip. It will take some practice to learn the most efficient distance to hold torch tip from target. It is also more efficient to use torch with wind direction to reduce torch blow-out or flame blowing back toward igniters. Torching seedlings until wilting occurs is usually sufficient to kill; it is not usually necessary to torch seedlings to ash (although this is more satisfying). If

possible it is more efficient to torch seedlings and saplings at stem base rather than the entire plant.

Mop-up an area completely after torching. Allow torches to cool down (or cool with water) before disconnecting from propane tanks and putting them back into PVC safety cases. Follow disconnecting, storage, and maintenance suggestions in Red Dragon Torches Operating Instructions and Parts Manual.

Usually one treatment removes most seedlings/saplings, but repeat treatment in same growing season or next growing season may be necessary due to seed bank input, or some sapling re-sprouts. Seedlings usually are not capable of resprouting if torched in first growing season (before August), although I have had good success in removal when spot-burning in September. Repeat treatments are usually on an individual or small patch basis.

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APPENDIX 3: HOW TO READ A PESTICIDE LABEL

All pesticides registered for use in the U.S. must have a label that has been approved by the federal Environmental Protection Agency (EPA). The label contains information about the product, including its relative toxicity, potential hazard to humans and the environment, directions for use, storage and disposal, and first aid treatment in case of exposure. Product labels are legal documents whose language is determined and approved by the EPA during the pesticide registration process. Any use of a pesticide inconsistent with the label requirements is prohibited by law.

Labels contain very specific information in language that is tightly regulated by the US EPA. The word “must” is used for actions that are required by law, while the word “should” is used for actions that are recommended but not required. One of the “Signal words” (caution, warning, danger, and poison) used by the EPA to indicate relative toxicity to humans, must appear on each label (see below).

Material Safety Data Sheets (MSDSs) are similar to product labels but need not contain the same information. While product labels are regulated and required by the EPA, MSDSs are required by the U.S. Occupational Safety and Health Administration (OSHA) for the protection of employees using pesticides or other hazardous chemicals. All chemical manufacturers must provide a MSDS to employers purchasing the chemicals. The product label and MSDS should both be included with any product. Both documents contain important and reliable information that should be thoroughly reviewed before the product is used.

Label Contents

1. **Precautionary Statements** – Pesticide labels highlight three types of hazards associated with use of the product. The “hazards to people and domestic animals” section explains if and why a pesticide is hazardous, its potential adverse effects, and safety gear that applicators are required to wear. The “environmental hazards” section discusses potential environmental damage including impacts to non-target organisms, such as fish and wildlife, and provides measures that can minimize ecological impacts. The “physical and chemical hazards” section outlines potential hazards due to the chemical and physical nature of the product, such as flammability and explosiveness.
2. **Directions for Use** – The directions outline where, when, and how much of a pesticide may be used and any special restrictions. For herbicides, it lists all plants or types of plants that the formulation in question is registered to control. The law requires compliance with these directions. An herbicide may not be used to control a species or type of plant that is not listed on its label.

Sample Product Label

<p>1 PRECAUTIONARY STATEMENTS</p> <p>HAZARD TO HUMANS AND DOMESTIC ANIMALS (Signal Word) _____</p> <p>_____</p> <p>_____</p> <p>ENVIRONMENTAL HAZARDS</p> <p>_____</p> <p>_____</p> <p>PHYSICAL OR CHEMICAL HAZARDS</p> <p>_____</p> <p>_____</p> <p>DIRECTIONS FOR USE: It is a violation of Federal law to use this product in a manner inconsistent with its labeling.</p> <p>2</p> <p>RESTRICTED USE PESTICIDE</p> <p>3 RE-ENTRY STATEMENT (if applicable)</p> <p>4 STORAGE AND DISPOSAL</p> <p>STORAGE _____</p> <p>_____</p> <p>DISPOSAL _____</p> <p>_____</p>	<p>5 RESTRICTED USE PESTICIDE</p> <p>Due to: [insert reason]</p> <p>For retail sale to and use only by Certified Applicators or persons under their direct supervision and only for those uses covered by the Certified Applicator's certification.</p> <p>PRODUCT NAME</p> <p>6 ACTIVE INGREDIENT(S): XX.00%</p> <p>INERT INGREDIENTS: XX.00%</p> <p>TOTAL: 100.00%</p> <p>This product contains ___ lbs of ___ per gallon.</p> <p>KEEP OUT OF REACH OF CHILDREN</p> <p>7 Signal Word [Poison]</p> <p>[Skull & Crossbones]</p> <p>First Aid</p> <p>8 If Swallowed _____</p> <p>If Inhaled _____</p> <p>If on Skin _____</p> <p>If in Eyes _____</p> <p>SEE SIDE PANEL FOR ADDITIONAL PRECAUTIONARY STATEMENTS</p> <p>EPA Registration No. _____ [Registrant Name]</p> <p>EPA Establishment No. _____ [Address, City, State, zip code]</p> <p>9 Net Contents _____</p>	<p>2 Directions for Use (continued)</p> <p>CROP/SITE _____</p> <p>_____</p> <p>CROP/SITE _____</p> <p>_____</p> <p>CROP/SITE _____</p> <p>_____</p> <p>CROP/SITE _____</p> <p>_____</p> <p>CROP/SITE _____</p> <p>_____</p> <p>CROP/SITE _____</p> <p>_____</p> <p>CROP/SITE _____</p> <p>_____</p> <p>CROP/SITE _____</p> <p>_____</p> <p>10 WARRANTY STATEMENT</p> <p>_____</p> <p>_____</p> <p>_____</p>
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3. Reentry Statement – This section identifies the period of time following treatment when re-entry to the treated area is prohibited. If no statement is given, re-entry should not be attempted until the spray dries or the dust settles. Check with the county agricultural commissioner for local restrictions.
4. Storage and Disposal Directions – This section outlines appropriate storage and disposal procedures for unused portions of the pesticide and of the pesticide container.
5. Statement of Use Classification – Each pesticide is designated and prominently labeled as “General Use” or “Restricted Use”. “Restricted use” pesticides are those that would pose a significant threat to the applicator or the environment without further regulatory restrictions. Only certified pesticide applicators may apply “restricted use” pesticides, and additional safety precautions may be required. The status of each pesticide can be found in the U.S. EPA’s Restricted Use Products list (<http://www.epa.gov/RestProd/ropoct00.htm>). Of the herbicides

listed in this handbook, only picloram is of “restricted use.” Be sure to check for additional state restrictions (for example, certain formulations of 2,4-D are of “restricted use” in California).

6. Brand Name, Chemical or Trade Name, Common Name, Formulation, Ingredients, & Contents – The *brand name* is the name chosen by the manufacturer for marketing purposes. Often the same herbicide formulation is marketed for different uses under different brand names. For example, triclopyr amine is sold as Garlon 3A[®] for commercial use, but a slightly different formulation is sold as Turflon Ester[®] for residential use. The *chemical name* describes the molecular formula of the active ingredient. Examples of chemical names include: 3,6-dichloro-pyridinecarboxylic acid for clopyralid, or N-(phosphonomethyl) glycine for glyphosate. The *common chemical name* is for the active ingredient itself - it is not specific to the formulation. Examples of common chemical names include glyphosate and triclopyr.

Pesticides are marketed in a variety of *formulations* including emulsifiable concentrates, wettable powders, and soluble powders. Often the brand name indicates the formulation type. For example, Garlon 3A[®] is the amine formulation of triclopyr.

The product *ingredients* are listed as the percentage of active and “inert” ingredients in the product. The active ingredient is the pesticidally active chemical. Unlike most commonly accepted definitions of “inert”, the inert ingredients in a pesticide product include all ingredients that are not pesticidally active. This does not necessarily imply that these ingredients are non-toxic, non-flammable, or otherwise non-reactive. The *contents* describe the total product weight or liquid volume in the package.

Study	Category I	Category II	Category III	Category IV
Acute Oral	≥ 50 mg/kg	>50-500 mg/kg	>500-5000 mg/kg	>5000 mg/kg
Acute Dermal	≥ 200 mg/kg	>200-2000 mg/kg	>2000-5000 mg/kg	>5000 mg/kg
Acute Inhalation	≥ 0.05 mg/liter	>200-2000 mg/liter	>2000-5000 mg/liter	>5000 mg/liter
Eye Irritation	Corrosive or corneal involvement or irritation persisting >20 days	Corneal involvement or irritation clearing in 8-20 days	Corneal involvement or irritation clearing in < 7 days	Minimal effects clearing < 24 hrs
Skin Irritation	Corrosive	Severe irritation > 72 hrs	Moderate irritation > 72 hrs	Mild or slight irritation
Signal Word	DANGER	WARNING	CAUTION	CAUTION

7. Signal Word – The signal word indicates how dangerous or toxic a product can be. The signal words “danger”, “warning”, or “caution” is determined by a combination of acute toxicity studies, and the toxicity of each of the product components. Each toxicity study is assigned a toxicity category, and the highest category determines the signal word that appears on the label. Additionally, “poison” and the skull-crossbones symbol are required for products in toxicity category I for acute oral, dermal, or inhalation exposure, or for products that contain certain “inerts”.
8. Statement of Practical Treatment – This section highlights important first aid information for treating people exposed to the product.
9. Manufacturer, Registration and Establishment Numbers – The name and address on the label should be used for contacting the product manufacturer. The Registration number is the EPA number that identifies the registered product. The Establishment number identifies where the product was produced.
10. Warranty – The warranty statement is not required but often is provided by the manufacturer.

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APPENDIX 4: HOW PESTICIDES ARE REGULATED IN THE U.S.

Three federal laws regulate pesticide use in the United States. Herbicides are the subgroup of pesticides that kill plants. Other types of pesticides include insecticides, fungicides, rodenticides, etc. The Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) is the primary law governing the registration, sale, and use of pesticides nationwide. The Federal Food, Drug and Cosmetic Act (FFDCA), administered by the EPA and the Food and Drug Administration, establishes the maximum pesticide residue levels allowable in food and other commodities. The Food Quality Protection Act (FQPA) of 1996 modified and strengthened both FIFRA and FFDCA.

FIFRA requires that all pesticides (imported or domestic) sold or distributed in the U.S. be registered with the EPA. Several types of pesticide registration are available. When the EPA registers a pesticide for either general or restricted use, it is called a Federal Registration Action. Experimental Use Permits are granted to allow manufacturers and researchers to test new pesticides in the field prior to registration. Emergency Exemptions or Special Local Needs permits are granted for unregistered pesticides or for new uses of currently registered pesticides in emergency situations for which no other registered pesticide or control measure is effective.

A Federal Registration Action occurs only after a thorough investigation of the pesticide's ingredients, intended uses, toxicity, and related characteristics. The manufacturer is required to provide data sufficient to determine the pesticide's potential to damage the environment or cause injury to humans, wildlife, fish, crops, or livestock. In addition, use of the pesticide must not result in illegal residue levels in food or feed. In some cases, the EPA will issue conditional registrations under which use of the pesticide is permitted until further testing shows whether or not the pesticide is problematic. As part of the registration process, the EPA determines what language should appear on the product label. Use of a product inconsistent with the information and instructions on its label is illegal.

The FIFRA amendments of 1988 established a re-registration procedure for pesticides that were first registered prior to 1984. The purpose of the re-registration process was to ensure that older pesticides conform to modern health and safety requirements. When necessary, manufacturers must provide more information on the toxicity and other properties of the pesticide. The pesticide is eligible for re-registration once the EPA has determined that enough information has been presented to demonstrate that no unreasonable risks to human health or to the environment will be incurred when the pesticide is used properly.

FQPA was passed by congress in 1996, amending both FIFRA and FFDCA, changing the way EPA regulates pesticides. FQPA required new safety standards that must be applied to all pesticides used on foods. It stated that the registration of all pesticides should take into account the possible lifetime cumulative exposure, potential synergies with other compounds, and had stricter allowable exposure rates for children.

Although the EPA oversees pesticide registration, individual states now have the primary enforcement responsibility, termed “state primacy”. Each state must demonstrate that their regulatory mechanisms equal or exceed those of the EPA. States that do not properly enforce federal requirements in a timely manner can lose their enforcement authority. States may also require that pesticides be registered under their own systems, before the pesticide may be used in that state (for example, California requires this).

Date Authored: April 2001

APPENDIX 5: PERSONAL CONTACTS

(for sources listed in this Handbook)

Bily, Pat
Invasive Plant Specialist
The Nature Conservancy, Hawaii
808-572-7849 ext. 28
pbily@tnc.org

Budd, Bob
Director of Stewardship
Red Canyon Ranch, Wyoming
The Nature Conservancy
307-332-3388
bbudd@tnc.org
bbudd@wyoming.com

Cooper, Jeffrey
Preserve Manager
Patagonia/Sonoita Creek, Arizona
The Nature Conservancy
520-394-2400
vulture@dakotacom.net

DiTomaso, Joe
Extension Non-Crop Weed Ecologist
University of California, Davis
530-754-8715
ditomaso@vegmail.ucdavis.edu

Hillmer, Jennifer
Northeast Ohio Land Steward
Ohio Chapter
The Nature Conservancy
440-285-8622
jhillmer@tnc.org

Kleiman, Bill
Preserve Manager
Nachusa Grasslands, Illinois
The Nature Conservancy
(815) 456-2340
bkleiman@tnc.org

Lanini, Tom
Extension Weed Ecologist
University of California, Davis
530-752-4476
wtlanini@ucdavis.edu

McGowan-Stinski, Jack
Land Steward
Michigan Chapter
The Nature Conservancy
517-332-1741
jmcgowan-st@tnc.org

Miller, Clint
Northern Tallgrass Prairie Ecoregion
The Nature Conservancy
605-874-8517
cmiller@tnc.org

Randall, John
Director
Wildland Invasive Species Team
The Nature Conservancy
530-754-8890
jarandall@ucdavis.edu

Rice, Barry
Associate Scientist
Wildland Invasive Species Team
The Nature Conservancy
530-754-8891
bamrice@ucdavis.edu

Wilk, Ed
Preserve Assistant
Patagonia/Sonoita Creek, Arizona
The Nature Conservancy
520-394-2400
edwilk@dakotacom.net

Appendix 6: State Pesticide Regulatory Agencies

Updated: June 2003 from <http://ace.orst.edu/info/npic/state1.htm>

Compiled by: Julia McGonigle (TNC-Oregon volunteer)

Alabama

Alabama Department of Agriculture
Division of Plant Protection and Pesticides
1445 Federal Way
Montgomery, AL 36107
334-240-7171
800-642-7761
<http://www.agi.state.al.us/pppm.htm>

Alaska

Alaska Department of Environmental Health
Pesticide Services
500 S Alaska St
Palmer, AK 99645-6340
907-745-3236
800-478-2577 (in state only)
<http://www.state.ak.us/dec/deh/pesticides/home.htm>

Arizona

Arizona Department of Agriculture
Environmental Services Division
1688 W Adams
Phoenix, AZ 85007
602-542-3578
800-423-8876
<http://www.agriculture.state.az.us/ESD/esd.htm>

Arkansas

Arkansas State Plant Board
Pesticide Division
#1 Natural Resource Dr.
Little Rock, AR 72205
501-225-1598
http://www.plantboard.org/pesticides_about.html

California

CA Department of Pesticide Regulation
1001 I Street
P.O. Box 4015
Sacramento, CA 95812-4015
(916) 445-4300
<http://www.cdpr.ca.gov/>

Colorado

CO Department of Agriculture
Division of Plant Industry
700 Kipling St Suite 4000
Lakewood, CO 80215-8000
303-239-4140
<http://www.ag.state.co.us/DPI/home.html>

Connecticut

CT Department Environmental Protection
Pesticide Division
79 Elm St
Hartford, CT 06106
860-424-3369
<http://www.state.ct.us/doag/>

Delaware

DE Department of Agriculture
Pesticides Section
2320 South Dupont Hwy
Dover, DE 19901
302-698-4500
<http://www.state.de.us/deptagri/pesticides/index.htm>

Florida

FL Department of Agriculture & Consumer Services
Bureau of Pesticides
3125 Conner Blvd.
Building #6, Mail Stop L29
Tallahassee, FL 32399-1650
850-487-0532
<http://doacs.state.fl.us/~aes/pesticides/>

Georgia

GA Department Agriculture
Pesticide Division
19 Martin Luther King Dr
Atlanta, GA 30334
404-656-4958
http://www.agr.state.ga.us/html/pesticide_division.html

Hawaii

Hawaii Department of Agriculture
Plant Industry Division of Pesticides
1428 S King St (PO BOX 22159)
Honolulu, HI 96823-2159
808-973-9401
http://www.hawaiiag.org/hdoa/pi_pest.htm

Idaho

ID Department of Agriculture
Division of Agricultural Resources
PO BOX 7723, Boise, ID 83701
2270 Old Penitentiary Rd.
Boise, ID 83712
208-332-8605
<http://www.agri.state.id.us/agresource/pesttoc.htm>

Illinois

IL Department of Agriculture
Bureau of Environmental Programs
PO BOX 19281
State Fairgrounds
Springfield, IL 62794-9281
217-782-2172
800-273-4763 (in state only)
<http://www.agr.state.il.us/Environment/>

Indiana

Office of Indiana State Chemist
Pesticide Section
Purdue University
1154 Biochemistry Bldg
W Lafayette, IN 47907-1154
765-494-1585
http://www.isco.purdue.edu/index_pest1.htm

Iowa

Iowa Department of Agriculture
Pesticide Bureau
Wallace Bldg
Des Moines, IA 50319
515-281-5321
<http://www.agriculture.state.ia.us/pesticidebureau.htm>

Kansas

KS State Board of Agriculture
Pesticide & Fertilizer Program
109 SW 9th Street, 3rd Floor
Topeka, KS 66612-1281
785-296-3786
<http://www.accesskansas.org/kda/Pest&Fert/Pest-mainpage.htm>

Kentucky

KY Department of Agriculture
Division of Pesticide Regulation
100 Fair Oaks Ln 5th Fl
Frankfort, KY 40601
502-564-7274
http://www.kyagr.com/enviro_out/pesticide/index.htm

Louisiana

LA Department of Agriculture & Forestry
Pesticide & Environmental Programs
PO BOX 3596
Baton Rouge, LA 70821-3596
225-925-3796
<http://www.ldaf.state.la.us/divisions/aes/pesticide&ep/>

Maine

Maine Department of Agriculture
Board of Pesticides Control
State House Station 28
Augusta, ME 04333
207-287-2731
<http://www.state.me.us/agriculture/pesticides/>

Maryland

MD Department of Agriculture
Pesticide Regulation Section
50 Harry S Truman Parkway
Annapolis, MD 21401
410-841-5710
<http://www.mda.state.md.us/geninfo/genera10.htm>

Massachusetts

Mass Department of Agriculture
Pesticide Bureau
251 Causeway Street, Suite 500
Boston, MA 02114
617-626-1700
<http://www.state.ma.us/dfa/pesticides/>

Michigan

Michigan Department of Agriculture
Pesticide and Plant Pest Management Division
611 W. Ottawa, 4th Floor
PO BOX 30017
Lansing, MI 48909
1-800-292-3939
http://www.michigan.gov/mda/0,1607,7-125-1572_2875-8324--,00.html

Minnesota

MN Department of Agriculture
Agronomy and Plant Protection Division
90W Plato Blvd
St. Paul, MN 55107
800-967-2474
651-296-5639
<http://www.mda.state.mn.us/APPD/default.htm>

Mississippi

MS Department of Agriculture
Bureau of Plant Industry
PO BOX 5207
MS State, MS 39762
662-325-7765
<http://www.mdac.state.ms.us/Library/BBC/PlantIndustry/PesticidePrograms/PesticidePrograms.html>

Missouri

Web site Missouri Department of Agriculture
Bureau of Pesticide Control
PO BOX 630 - 1616 Missouri Blvd.
Jefferson City, MO 65102
573-751-4211
<http://www.mda.state.mo.us/Pest/d7.htm>

Montana

MT Department of Agriculture
Pesticide Programs
PO BOX 200201
Helena, MT 59620-0201
406-444-2944
<http://agr.state.mt.us/programs/asd/pesticide.shtml>

Nebraska

NE Department of Agriculture
Bureau of Plant Industry
301 Centennial Mall South
P.O. Box 94756
Lincoln, NE 68509-4756
402-471-2394
800-831-0550
<http://www.agr.state.ne.us/division/bpi/bpi.htm>

Nevada

NV Department of Business and Industry
Department of Agriculture
350 Capitol Hill Ave
Reno, NV 89502
775-688-1180
<http://agri.state.nv.us/>

New Hampshire

NH Department of Agriculture
Division of Pesticide Control
PO BOX 2042
Concord, NH 03302-2042
603-271-3550
<http://www.state.nh.us/agric/peco.html>

New Jersey

NJ Department of Environmental Protection
Pesticide Control and Local Programs
22 S Clinton Ave.
4 Station Plaza, 3rd Fl
PO BOX 411
Trenton, NJ 08625-0411
609-530-4070
<http://www.state.nj.us/dep/enforcement/pcp/index.html>

New Mexico

NM Department of Agriculture
Bureau of Pesticide Management
MSC 3189, Corner of Gregg and Espina
PO BOX 30005
Las Cruces, NM 88003-8005
505-646-2133
<http://nmdaweb.nmsu.edu/DIVISIONS/AES/pest.html>

New York

NY Department of Environmental Conservation
Solids and Hazardous Materials
Pesticides Management Program
50 Wolf Rd Rm 498
Albany, NY 12233-7254
518-457-6934
<http://www.dec.state.ny.us/website/dshm/pesticid/pesticid.htm>

North Carolina

NC Department of Agriculture and Consumer Services
Food & Drug Protection Division
Pesticide Section
PO BOX 27647
Raleigh, NC 27611
919-733-3556
<http://www.ncagr.com/fooddrug/pesticid/>

North Dakota

ND Department of Agriculture
Pesticide Programs
State Capitol, 600 E Blvd Ave, Dept 602
Bismark, ND 58505-0020
701-328-2231
800-242-7535
<http://www.agdepartment.com/Programs/Plant/Pesticides.html>

Ohio

Ohio Department of Agriculture
Division of Plant Industry
8995 E Main St
Reynoldsburg, OH 43068
800-282-1955 (in state only)
614-728-6200
http://www.state.oh.us/agr/PRS/index_1.htm

Oklahoma

OK Department of Agriculture
Division Plant Industry
2800 N Lincoln Blvd
PO Box 528804
Oklahoma City, OK 73105-4298
405-521-3864
<http://www.oda.state.ok.us/pics.htm>

Oregon

OR Department of Agriculture
Pesticides Division
635 Capitol St NE
Salem, OR 97301-2532
503-986-4635
<http://oda.state.or.us/pesticide/>

Pennsylvania

PA Department of Agriculture
Bureau of Plant Industry
2301 N Cameron St
Harrisburg, PA 17110-9408
717-787-4843
<http://www.agriculture.state.pa.us/plantindustry/site/>

Rhode Island

RI Department of Environmental Mgmt
Division of Agriculture
235 Promenade St.
Providence, RI 02908-5767
401-222-2781
<http://www.state.ri.us/dem/programs/bnatres/agricult/index.htm>

South Carolina

South Carolina Department of Agriculture
Pesticide Regulation
Clemson University
511 Westinghouse Rd.
Pendleton, SC 29670
864-646-2150
http://dpr.clemson.edu/index_flash.html

South Dakota

SD Department of Agriculture
Pesticide Program, Foss Bldg
523 E Capitol
Pierre, SD 57501-3188
800-228-5254 (in state only)
605-773-3724
<http://www.state.sd.us/doa/das/hp-pest.htm>

Tennessee

TN Department of Agriculture
Ag Inputs & Pesticides
Ellington Agricultural Center
PO BOX 40627
Nashville, TN 37204
615-837-5150
<http://www.state.tn.us/agriculture/regulate/aip/>

Texas

TX Department of Agriculture
Pesticide Division
PO BOX 12847
Austin, TX 78711
1-800-835-5832
512-463-7476
<http://www.agr.state.tx.us/pesticide/>

Utah

UT Department of Agriculture
Division of Plant Industry
350 N Redwood Rd
PO BOX 146500
Salt Lake City, UT 84114-
801-538-7180
http://ag.utah.gov/plantind/plant_ind.html

Vermont

Vermont Department of Agriculture
Plant Industry Division
116 State St
Montpelier, VT 05602
802-828-2431
<http://www.vermontagriculture.com/pid.htm>

Virginia

VA Department of Agriculture
Office of Pesticide Services
PO BOX 1163
Richmond, VA 23218
804-371-6558
800-552-9963 (in state only)
<http://www.vdacs.state.va.us/pesticides/>

Washington

WA State Department of Agriculture
Pesticide Management Division
1111 Washington St. SE
PO Box 42589
Olympia, WA 98504-2589
877-301-4555 (in state only)
360-902-2010
<http://www.wa.gov/agr/PestFert/Pesticides/>

Washington D.C.

Department of Health
Environmental Health Administration
Bureau of Hazardous Materials
Toxic Substance Division
51 N Street NE
Washington, DC 20002
202-535-2500
<http://www.dhra.dc.gov/main.shtm>

West Virginia

WV Department of Agriculture
Pesticide Regulatory Program
1900 Kanawha Blvd E
Charleston, WV 25305-0190
304-558-2209
http://www.state.wv.us/agriculture/divisions/plant_industries.html#pesticide

Wisconsin

WI Department of Agriculture
Agricultural Resources Mgmt Division
PO BOX 8911 2811 Agric. Dr
Madison, WI 53708-8911
608-224-4500
<http://datcp.state.wi.us/core/agriculture/pest-fert/index.html>

Wyoming

WY Department of Agriculture
Technical Services Division
2219 Carey Ave
Cheyenne, WY 82002
307-777-7324
<http://wyagric.state.wy.us/techserv/tspest.htm>

Puerto Rico

Puerto Rico Department of Agriculture
Agrological Laboratory
PO Box 10163
Santurce, PR 00908
787-796-1650, 1835, 0138
http://www.nass.usda.gov/pr/de_ag_PR.htm

Virgin Islands

Department of Agriculture
Estate Lower Love
Kingshill
St. Croix, US VI 00850
340-778-0997
340-774-5182
<http://www.usvi.org/agriculture/>

Guam

Guam Department of Agriculture
192 Dairy Road
Mangialo, GU 96923
671-734-3942, 3943

American Samoa

Department of Agriculture
American Samoa Government
Executive Office Building, Utulei
Territory of American Samoa Pago Pago
American Samoa 96799
684-699-1497
<http://www.asg.gov.com/departments/doa.asg.htm>

Common Wealth of the Northern Mariana Islands

Division of Environmental Quality, CNMI
PO Box 1304
Saipan, Mariana Islands 96950
670-664-8500
<http://www.deq.gov.mp/>