Knotweed Treatment
In the Cedar River Municipal Watershed
2013 - 2015

Annual Report
Seattle Public Utilities and Neighborhoods Committee
Seattle City Council

Sally Nickelson
Major Watersheds Invasive Species Program Manager
Seattle Public Utilities, Watershed Management Division

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BACKGROUND

In August, 2010, Seattle City Council, recognizing both the extreme ecological threat posed by the highly invasive species Bohemian knotweed (*Polygonum* x *bohemicum)* and the limited options for treatment, passed an ordinance amending the Cedar River Municipal Watershed Secondary Use Policy Number 6-13 to allow limited application of the herbicide imazapyr to treat knotweed within the municipal watershed. This ordinance was effective through December 31, 2012. For a full report on the threat posed by knotweed, the background that lead to this decision, as well as treatment results 2010-2012, see the report, Knotweed Treatment 2010-2012, Annual Report to City Council, online at: http://www.seattle.gov/util/cs/groups/public/@spu/@ssw/documents/webcontent/01_026334.pdf

In early 2010, preliminary data had suggested that three consecutive years of herbicide treatment might be sufficient to kill some large patches of knotweed. Subsequent regional data, however, has indicated that it usually takes eight or more years of annual treatments to kill the large root masses and achieve greater than 98% mortality on large patches. Consequently, in May of 2013, Seattle Public Utilities (SPU) requested that the Seattle City Council pass a follow-up ordinance to allow continued treatment of knotweed with imazapyr for an additional three years (2013 – 2015). Ordinance 124191 (Council Bill 117765) was passed on May 28, 2013. It was virtually identical to the one passed in 2010, limiting herbicide treatment to only imazapyr used on knotweed, with water quality testing after each treatment, ongoing monitoring, and annual reports to City Council.

In 2013 SPU chose to request only three additional years in order to allow sufficient oversight and feedback from City Council on the knotweed program. However, at that time we informed City Council that because some patches of knotweed were more recently discovered and would have received insufficient treatments through 2015, SPU would need to seek another follow-up ordinance. A request for three additional years (2016-2018) was submitted in mid-2015. During this recent ordinance process, concern about the possible effect of imazapyr on European honey bees and other pollinators was raised. Consequently, Seattle City Council (in Council Bill 118481, passed on 9/8/2015) directed SPU to update and summarize the latest available science on the environmental and human health effect of imazapyr (most recently conducted in 2010 for the initial ordinance). See Appendix I for this updated literature review and risk assessment. In addition, City Council directed SPU to evaluate the long-term financial and environmental implications for knotweed control (see costs section, page 6, and long-term implications section, page 10). Ordinance 124852 was passed on September 8, 2015.

For more information about the watershed Invasive Species Program, see the Major Watersheds Invasive Species Management Plan, available online: http://www.seattle.gov/util/EnvironmentConservation/OurWatersheds/Habitat_Conservation_Plan/ManagingtheWatershed/ProtectWatershedHabitats/ProtectionEfforts/index.htm#invasiveSpecies .

Previous knotweed reports are available in the Project Plans and Reports section on: http://www.seattle.gov/util/EnvironmentConservation/OurWatersheds/Habitat_Conservation_Plan/ManagingtheWatershed/StreamRiparianHabitatRestoration/Metrics/index.htm .

SURVEYS FOR KNOTWEED

In 2013, based on recommendations from interested stakeholders, we identified over 1500 acres of off-road habitat that potentially could contain knotweed, based on location of known knotweed patches, streams and other water bodies, and extent of deciduous forest canopy. None of these sites had previously been surveyed for knotweed. These areas were sorted into high (1219 acres) and medium (388 acres) priority based on their proximity to existing knotweed and flowing water. These off-road surveys were successful in finding more knotweed patches. In 2013 we found a total of 2.15 additional acres of knotweed (most in the old Taylor Townsite), all of which were treated for the first time that year. By the end of 2015, only about 194 acres classified as high priority remain to be surveyed, and no further large knotweed patches have been found (Figure 1). We hope to survey the remaining high priority areas in 2016.

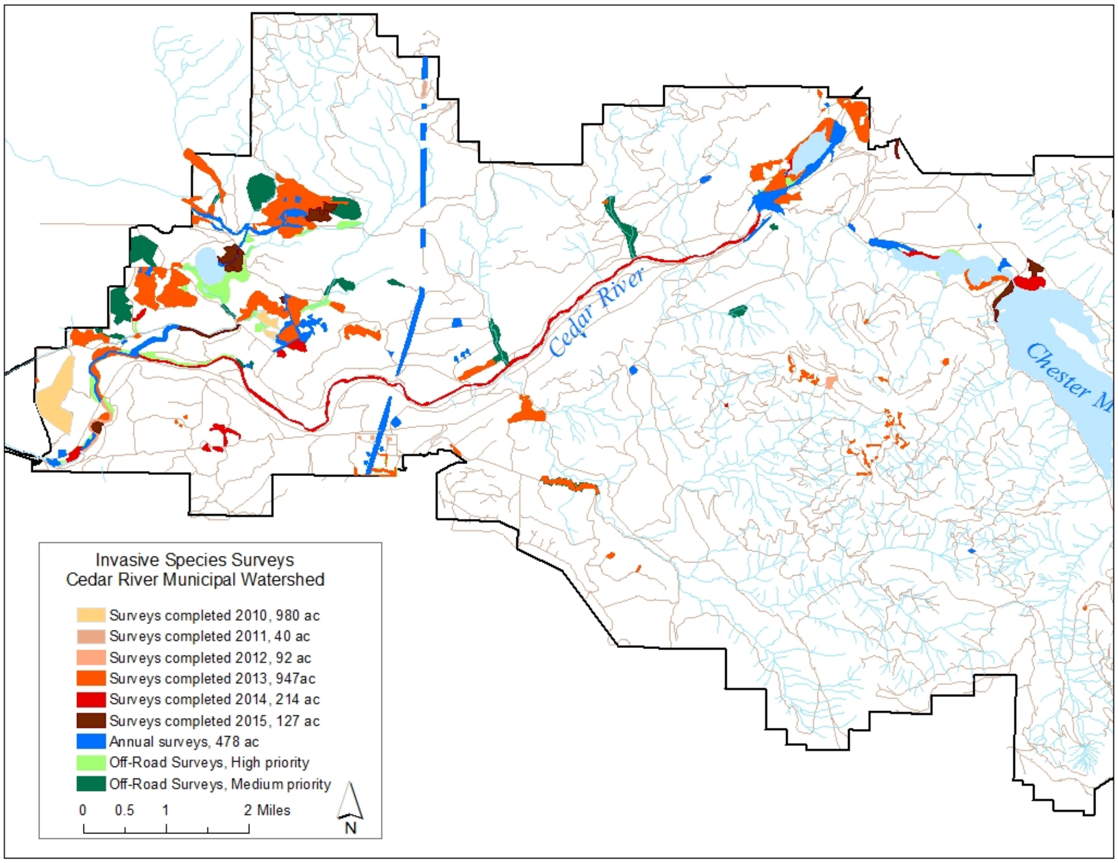

Figure 1. Off-road areas of high and medium priority to survey for knotweed, plus areas surveyed annually and areas surveyed by year, 2010–2015.

In addition, we survey approximately 475 acres of off-road habitat annually. This includes all known off-road knotweed patches plus areas routinely surveyed for other projects (e.g., wetlands surveyed for amphibian egg masses). We anticipate this level of survey to continue, and we will include additional priority acres as funding and staffing allow. In both 2014 and 2015 we also conducted comprehensive invasive species surveys of approximately 340 miles of road (334 miles of drivable roads and 6 miles of decommissioned roads) and 13 gravel pits (8 active) as part of the Early Detection/Rapid Response protocol used by the Major Watersheds Invasive Species Program. Only a single new knotweed plant (along an active road) was found during the 2014 road surveys and none were found along the roads in 2015. This level of road survey is also expected to continue. To date, dispersal appears to be by spread of plant fragments along travel corridors (streams, roads, wildlife paths). No new knotweed seedlings that appear to have been spread via seed have been found.

AREA TREATED WITH HERBICIDE

In 2015 we re-treated all areas previously treated with herbicide in 2010-2014 (Figure 2). A total of 7.7 acres were treated for the sixth time, 7.9 acres for the fifth time, 0.3 acres for the fourth time, and 2.1 acres for the third time. The reason for the different number of treatments is that the ordinance was passed late in the year in 2010, so only about half of the known acres could logistically be treated that year (7.7 acres). The remainder of known acres were treated for the first time in 2011 (7.9 acres). Acres with fewer treatments include spots initially missed by the contractor and the off-road sites newly found in 2013. In summary, a total of 18 acres of knotweed was treated with herbicide in 2015, of which only 2.2 acres were within the hydrographic boundary of the Cedar River (Table 1).

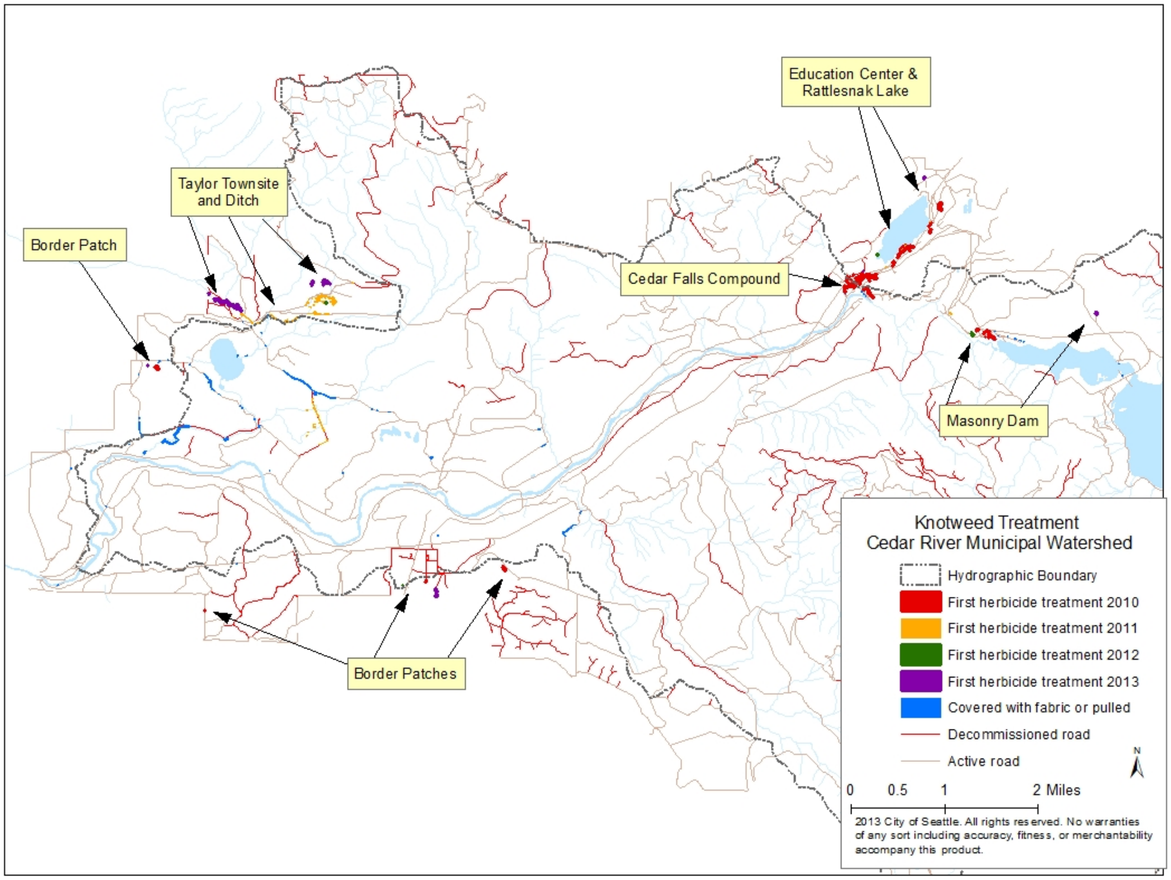

Figure 2. All known knotweed in the Cedar River Municipal Watershed symbolized by year first treated

Table 1. Number of knotweed-infested acres treated with imazapyr by site and year

Cedar River Hydrographic Boundary	Site	Number acres	Treatment Year						Total Treatments
			2010	2011	2012	2013	2014	2015	
Inside	Masonry Dam	0.31	X	X	X	X	X	X	6
		0.08		X	X	X	X	X	5
		0.19			X	X	X	X	4
		0.10				X	X	X	3
	Cedar Falls	1.55	X	X	X	X	X	X	6
	Total Inside	**2.23**							
Outside	Cedar Falls	1.71	X	X	X	X	X	X	6
		0.04		X	X	X	X	X	5
	Ed Center/ Rattlesnake Lake	3.04	X	X	X	X	X	X	6
		0.06		X	X	X	X	X	5
		0.08			X	X	X	X	4
		0.11				X	X	X	3
	Border	1.11	X	X	X	X	X	X	6
		0.02		X	X	X	X	X	5
		0.31				X	X	X	3
	Taylor	7.66		X	X	X	X	X	5
		0.01			X	X	X	X	4
		1.63				X	X	X	3
	Total Outside	**15.78**							

TREATMENT LOGISTICS

From 2013-2015 we used the same application method and herbicide concentration as in 2010 – 2012, i.e., a targeted backpack foliar spray of 1% aquatic formulation imazapyr mixed with 0.5-1% modified vegetable oil surfactant and a small amount of non-toxic blue dye in water. It was applied strictly according to label instructions, including restrictions such as not applying during rain, wind, or when there is a temperature inversion. The same safety procedures were followed, with certified herbicide applicators on site and doing all the mixing of the tank solutions. No spills, injuries, or any adverse effects were incurred by SPU staff or the contract crew members conducting any of the applications.

In both 2014 and 2015 knotweed plants were often quite small and difficult to see amongst the thick understory of shrubs and tall grass. Plus plants had a large variation in timing of growth, with small newly emerged growth found as early as May and as late as October. In order to get as much herbicide into the root system as possible, we attempt to time the herbicide application when the plants have put on maximum leaves, but before the leaves start to senesce. Application when the plant is actively growing and during the pre-bud stage, i.e., before the plant starts to flower, has been reported to be the most effective time to apply. This timing varies depending on elevation and site-specific conditions. For untreated knotweed at our elevation in the CRMW flowering generally occurs in September, so our target timing is mid to late August. However, because we bend the canes prior to the first application, and the vast majority of plants that have been treated at least once do not flower, the pre-bud issue was not applicable. See Appendix I for a complete discussion of knotweed treatment, flowering, and potential effect on pollinators. The other primary consideration on timing of application is the weather. August is generally the driest month, with rain in September quite variable. So for multiple reasons we target treatment during mid to late August whenever possible. In 2015, due to the drought, some small plants were starting to senesce as early as late July. So we started treating in late July, completed by mid-August.

In order to treat all aboveground biomass, we surveyed and treated each large site twice, four to six weeks apart. Plants treated with imazapyr showed signs of decline within that time, so were easily identifiable. During the second survey, we treated any newly emerged or previously missed plants. Other land managers in western Washington have also found this to be a useful technique. During 2015, the second survey and treatment occurred in mid to late September. As in previous years, no flowering plants were treated.

The vast majority of the herbicide was applied in terrestrial environments and did not require any specific permit. All treatment sites were more than 250 feet from the Cedar River and the nearest patch to the municipal water intake at Landsburg was over 10 miles. A small percentage of the application occurred in a riparian area in the Issaquah Creek watershed and was covered by an Aquatic Noxious Weed General Permit from the Washington State Department of Ecology under the Washington State Department of Agriculture National Pollutant Discharge Elimination System (NPDES) general permit. This area does not drain into water that reaches the Cedar River and the municipal water intake at Landsburg. None of the herbicide application occurred in water.

AMOUNT OF IMAZAPYR APPLIED

As in previous years, we averaged about 10 ounces imazapyr per acre on sites receiving a third treatment and two to six ounces per acre on sites receiving a fourth or fifth treatment. Total amount of imazapyr applied in 2015 was 61 ounces spread over 18 acres. Of this, a total of 13 ounces was applied inside the hydrographic boundary, spread over 2.2 acres. Total amount of herbicide applied has declined each year, from approximately 43 ounces per acre in 2010 and 2011, to an average of 3.4 ounces per acre in 2015 (Table 2). The legal maximum allowable application rate is 96 ounces per acre per year. The decline in application rate is due to the decreasing above-ground biomass of the knotweed resulting from the herbicide treatments (see following section).

Table 2. Total amount of imazapyr applied and application rate by year.

Year	Amount Imazapyr (oz)	Area Treated (ac)	Application Rate (oz/ac)
2010	334	7.7	43.4
2011	678	15.6	43.5
2012	241	15.9	15.2
2013	163	18.01	9.1
2014	120	18.01	6.7
2015	61	18.01	3.4

IMAZAPYR TREATMENT RESULTS

In 2015 most of the smaller knotweed sites, especially those along the watershed border, had either no or very few small stems. Above ground knotweed biomass (stems and leaves) declined by about half from 2014 levels, and by over 14 times from pre-treatment levels, indicated by the decline in application rate. Because we attempt to evenly coat every leaf on each plant, application rate is a good proxy for leaf biomass.

Most of the larger sites that had received four or five previous treatments still had numerous small to medium knotweed plants scattered throughout the site, indicating that the large root mass, although clearly damaged, was not yet dead. Experts hypothesize that the root system is able to compartmentalize, shutting off some sections from receiving an adequate herbicide dose. Because the rhizomes (roots that can sprout) can be up to 65 feet long and seven feet deep, that is potentially a large reservoir. We have to wait until all root segments send up shoots so that we can get sufficient amounts of herbicide into the plant system to kill it. Because roots can essentially hibernate for several years without sending up shoots, this process can take many years.

A visual record of knotweed response to treatment through the years is found in Appendix II.

WATER QUALITY TEST RESULTS

In all years, 2013-2015, water samples were taken both before (baseline) and after (post-treatment) the herbicide application. Samples were taken from two locations on the Cedar River (one at the point closest to a knotweed patch = 250 feet away, and the other at the Landsburg water supply intake facility), one location at Rattlesnake Lake, and one location on a small creek running through the Taylor Townsite. All water samples were analyzed for imazapyr at Pacific Agricultural Laboratory (PACLAB) in Portland, Oregon. PACLAB specializes in analysis of all types of pesticides and has an extremely low detection limit for imazapyr (0.02 ug/L, or 0.02 parts per billion). There were two samples (one in 2014 and one in 2015) that were inadvertently contaminated – one at the laboratory and one in the field (gloves stored adjacent to the chemical bottle were inadvertently used), but these problems were quickly detected and corrected. No imazapyr was detected in any of the municipal water samples in any of the three years.

COSTS

Cumulative total cost to treat 18 acres of knotweed with herbicide from 2010 through 2015 was approximately $104,000, or a cumulative average of $5,780 per acre for the six years. Annual cost per acre to treat the knotweed with imazapyr has declined from a high of $3,372 in 2010 to just $363 in 2015. This compares with a cumulative cost of approximately $200,000 ($44,000 per acre) to treat small scattered patches of knotweed by covering with geotextile fabric, a treatment we tried experimentally on a total of 4.5 acres from 2004 to 2012. Covering was only marginally successful on very small patches. The larger patches were still alive after more than eight years of continual covering. Fabric experimentally taken up along active roads was replaced and will be left down indefinitely. Isolated patches that were away from active roads and formerly covered were spot-treated with herbicide. Area treated and amount of herbicide used on these small patches was negligible.

Total annual cost to treat the knotweed with herbicide has decreased from a high of about $32,000 in 2011 to a low of about $6,500 in 2015. This annual cost is expected to continue to decline over the next three years. However, it will likely stabilize at around $3,500 because staff will need to continue to survey and monitor all the sites, which takes approximately 50 total person hours to thoroughly survey and treat the entire 18 acres. Contractor time and cost has declined each year, and we anticipate that by the end of 2017 staff alone will do all the survey and treatment. So the time and cost to continue to control knotweed after 2018 should be negligible and easily covered by the existing watershed Invasive Species Management Program budget and staff.

SITE RESTORATION

Ensuring knotweed treatment sites are repopulated with native plants following treatment is often the most effective method for preventing re-infestation of knotweed and other invasive plants. Our goal is to restore areas formerly occupied by knotweed to naturally functioning ecosystems dominated by a variety of native trees and shrubs. This will both increase resilience to future invasions by non-native species and provide high quality habitat for native wildlife, including birds, mammals, and insects. Many sites formerly occupied by knotweed will become infested with other non-native invasive species (Urgenson et al. 2014, Claeson and Bisson 2013). Consequently, many knotweed sites will need continued restoration work, including removal of other invasive species and planting native trees and shrubs.

In 2013 the Friends of the Cedar River Watershed (FCRW), in conjunction with SPU, received a 5-year King Conservation District grant (total of $46,000) to restore the formerly knotweed-infested area near the Education Center to native trees and shrubs. The grant funds a total of 12 volunteer events and four weeks of Washington Conservation Crew (WCC) time spread over the five years (through 2017). It also funds the purchase of approximately 2800 native plants. In 2015 FCRW dissolved and Forterra assumed management of the grant.

In 2013-2015 volunteers, SPU and FCRW staff, and WCC crews cleared the site of invasive blackberry (*Rubus armeniacus* and *Rubus laciniatus*), ivy (*Hedera helix*), locust (*Robinia pseudoacacia*), foxglove (*Digitalis purpurea*), mullein (*Verbascum thapsus*), Scots broom (*Cytisus scoparius*), and birdsfoot trefoil (*Lotus corniculaus*) that had invaded the area formerly dominated by knotweed. SPU staff partitioned the site into different planting zones, each with different long-term goals and specific planting plans (Figure 3). A total of 280 native trees (nine species) and 2580 shrubs (32 species) were planted from late 2013 through 2015 (Table 3). In addition, volunteers and contractors moved several hundred yards of mulch, surrounding each native planting with mulch to help suppress non-native weeds and provide more growing space for the plantings.

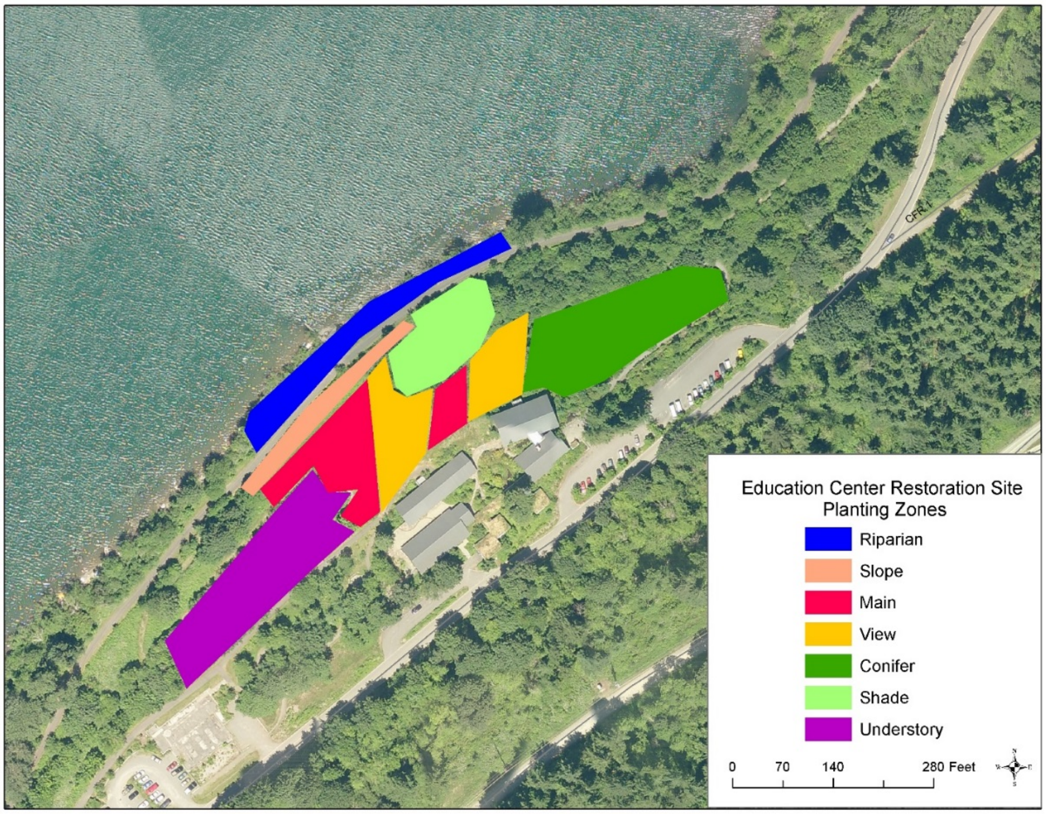

Figure 3. Location of the seven planting zones near the Education Center

Table 3. Species and number of native trees and shrubs planted near the Education Center, 2013-2015

Trees			
Big-leaf Maple	15	Sitka Spruce	40
Cherry, Bitter	50	Western Hemlock	30
Cottonwood, Black	15	Western Redcedar	50
Crabapple, Pacific	25	Western White Pine	25
Douglas-fir	30		
Total trees planted			**280**
Shrubs			
Cascara	75	Rhododendron, Pacific	50
Ceanothus, Redstem	50	Rose, Baldhip	75
Currant, Red-flowering	75	Rose, Clustered	50
Deer Fern	75	Rose, Nootka	75
Goatsbeard	115	Salmonberry	60
Hawthorn, Black	50	Serviceberry	75
Hazelnut, Beaked	70	Snowberry	75
Indian plum	80	Spirea	65
Kinnickinnick	55	Sweet Gale	20
Mock Orange	25	Sword Fern	115
Ninebark, Pacific	75	Thimbleberry	70
Oak Fern	75	Twinberry	150
Ocean Spray	120	Vine Maple	75
Oregon Grape, Tall	120	Willow, Hookers	100
Red Elderberry	60	Willow, Pacific	155
Red Osier Dogwood	150	Willow, Sitka	100
Total shrubs planted			**2580**

In 2014 and 2015 at the Taylor Townsite and overflow ditch, WCC and other contract crews cleared the original 6.67 knotweed-infested acres plus an adjacent 1.5-acre wetland of other invasive species, including invasive blackberry, foxglove, mullein, and non-native thistles. In the two years we planted a total of 1505 native conifer trees that should eventually help provide long-term shade that will help suppress invasive plants in the future. In addition, we planted 875 deciduous trees and 5075 native shrubs, to help restore the area to native habitat and ecological functioning (see Table 4 for number planted by species). We split the area into 12 different planting sites, and developed specific prescriptions and species mixes for each site, depending on the amount of soil moisture and sun exposure (Figure 4).

In both the Education Center and Taylor Townsite restoration projects, the variety of native trees and shrubs was designed not only to restore ecological functioning, but also to provide a diversity of native flowering plants to enhance native pollinator habitat. See Appendix I for a complete discussion of pollinators in relation to the knotweed project.

Table 4. Species and number of native trees and shrubs planted at Taylor Townsite, 2014-2015

Trees			
Cherry, Bitter	300	Sitka Spruce	475
Cottonwood, Black	275	Western Hemlock	220
Crabapple, Pacific	300	Western Redcedar	420
Noble Fir	135	Western White Pine	235
Shore Pine	25		
Total trees planted			**2,360**
Shrubs			
Cascara	350	Snowberry, Western	300
Ceanothus, Red-stem	300	Snowbrush	300
Current, Red-flowering	300	Spirea	50
Indian Plum	300	Sweet Gale	200
Mock-orange	300	Thimbleberry	200
Ninebark, Pacific	300	Twinberry	275
Red Osier Dogwood	250	Willow, Hooker	250
Rose, Nootka	300	Willow, Pacific	250
Sedge, Dewey's	200	Willow, Scoulers	250
Sedge, Slough	200	Willow, Sitka	250
Serviceberry, Western	300		
Total shrubs planted			**5,075**

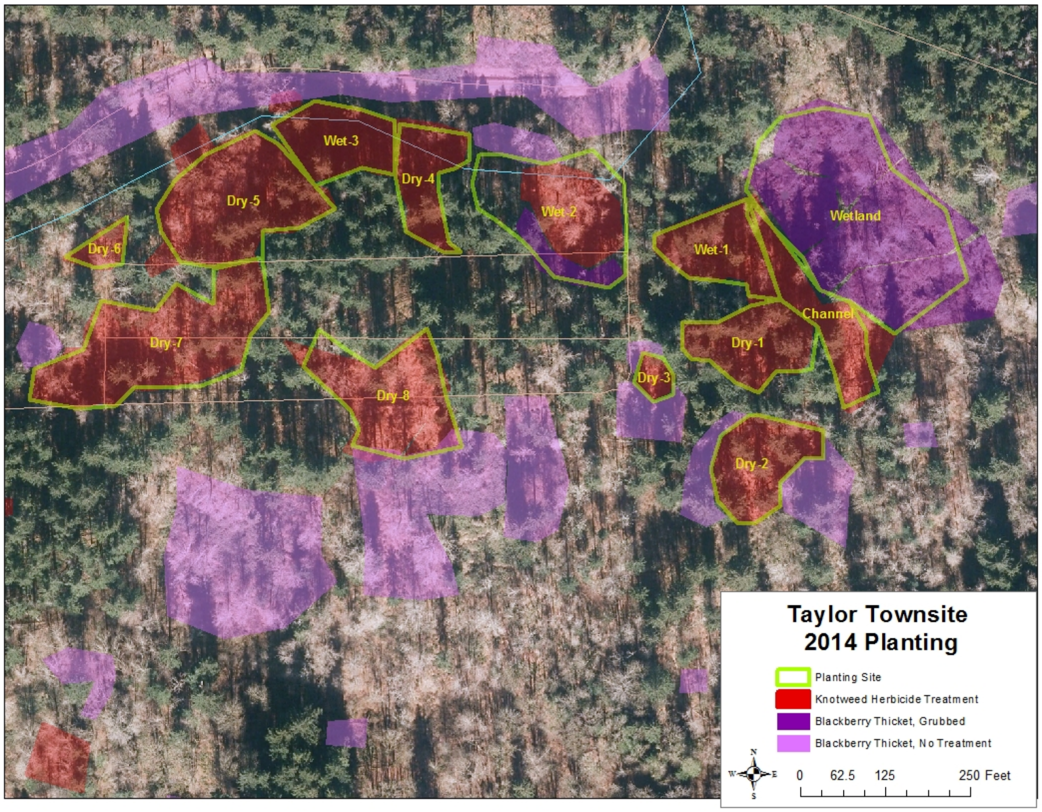

Figure 4. Twelve planting sites at the Taylor Townsite.

2016 PLANS AND MONITORING

We plan to monitor all the known knotweed patches and re-treat with imazapyr as needed in 2016. We anticipate that those sites that have received four or fewer treatments will require similar amounts of herbicide to that seen in the past three years, and those with more treatments will require even less. We will continue to monitor for knotweed patches during our annual road and gravel pit surveys and will conduct off-road surveys in high priority areas as funding and staffing allows. If we find any additional knotweed patches, we treat them in 2016 under Ordinance 124852. As in 2014 and 2015, we will re-check knotweed patches four to six weeks after treatment, and, weather-permitting, will treat any newly emerged or untreated plants at that time.

LONG-TERM IMPLICATIONS FOR KNOTWEED CONTROL

We are hopeful that by the end of 2018, the 15.6 acres that will have received eight or nine imazapyr treatments will have either been completely eradicated, or at such a low level that we can control any small growth by non-herbicide means (long-term covering). The 2.4 acres that will have received only six or seven treatments may or may not be reduced to this state by 2018, depending on site-specific conditions. By 2018 all large sites where natural regeneration of native trees and shrubs is insufficient will be planted to native species.

If left untreated, there is evidence that the small amount of live knotweed present at treatment sites can return to the original infestation level in as little as three seasons, eventually surpassing the infestation level present prior to any investments in knotweed control. This would result in the loss of progress toward long-term knotweed control, increased future control costs, degradation of environmental quality, and the alteration of the sustainable ecological services of invaded sites (Washington Department of Agriculture 2013). In addition, it could jeopardize the extensive ongoing restoration projects along the Cedar River downstream of Landsburg. As mentioned above, long-term maintenance and control costs of knotweed in the CRMW should be minimal. But an ongoing monitoring program is essential to ensure that all known knotweed is eradicated and any newly discovered patches are treated before they have a chance to spread.

APPENDIX I.
RISK ASSESSMENT, LITERATURE REVIEW
Treating knotweed with imazapyr in the Cedar River Municipal Watershed

BACKGROUND

Seattle Public Utilities (SPU) focuses on being effective stewards of the municipal watershed lands and resources it owns or controls. Restoring and maintaining healthy forests, wetlands, streams, and lakes in the municipal watersheds that supply Seattle-area residents with drinking water is a priority for SPU. It is these healthy ecosystems that provide the abundant and high quality drinking water on which the citizens of this region depend. Protecting water quality for human use also protects resources used by other species. Lands of the Cedar River Municipal Watershed (CRMW) are managed under the 50-year Habitat Conservation Plan (HCP), which requires that SPU promote and protect native diversity of plants and animals.

SPU has a policy that was enacted in 1989 to not use herbicides (i.e., pesticides designed specifically to be toxic to plants) in the CRMW, as part of the Secondary Use Policies, adopted by Ordinance 114632. The intent was to stop broadcast spraying of herbicide to control vegetation along forest roads, a typical forest management technique at that time. This was prior to the widespread recognition of the damage that certain non-native invasive plants can do to ecosystems and water quality.

Non-native invasive species are organisms introduced deliberately or unintentionally outside their natural habitats, where they have the ability to establish, invade, and locally eliminate native species, and dominate their new environments. They pose serious challenges to the conservation and sustainable use of global, regional, and local native biodiversity, with significant undesirable impacts on the functions, goods, and services provided by ecosystems. Their management costs include not only costs of prevention, control, and mitigation, but also the direct and indirect costs associated with the adverse impacts on ecological services such as the production of clean, abundant water and the maintenance of habitat for salmon and other native fish and wildlife, including birds, mammals, amphibians, and insects.

RISKS POSED BY KNOTWEED

Among the numerous invasive plant species present in the CRMW, knotweed is considered to be one of the most threatening to native ecosystem functioning due to its rapid growth, ability to quickly displace native vegetation and alter soil and water chemistry. As is often the case with hybrids, the hybrid Bohemian knotweed, (*Polygonum* x *bohemicum*), has been found to be more competitive and invasive than either of the parent species (Japanese, *P. cuspidatum* and giant, *P.sachalinensis*) (Parepa et al. 2013). This hybrid is widespread throughout the Pacific Northwest, and is the species found in the municipal watershed.

Specifically, knotweed is known to:

- reduce the amount and diversity of native streamside vegetation both through competition for light and nutrients (Urgenson et al. 2012) and secreted chemicals that are toxic to other plants, i.e., allelopathy (Murrell et al. 2011);

- change the soil nutrients and alter soil nutrient cycling, affecting the growth and development of native plant species and insects living in the soil (Urgenson et al. 2009);
- decrease the abundance and richness of both native plants and native invertebrates (Gerber et al. 2008); and
- alter the quality, quantity, timing, and chemistry of leaf inputs into riparian areas and streams (Claeson et al. 2013, Urgenson et al. 2009, Urgenson et al. 2006).

Claeson et al. (2013) compared knotweed litter with native red alder (*Alnus rubra*) and black cottonwood (*Populus balsamifera*). They found that senesced knotweed leaves were lower in nitrogen and phosphorus, and higher in cellulose, fiber, and lignin content than alder leaves, but were more similar to cottonwood leaves. Fungal biomass differed among species and changed over time. Macroinvertebrate shredders collected from experimental leaf packs after 31 days were proportionately more abundant on alder leaves than knotweed and cottonwood. Decay rates were not significantly different among leaf species, but during the first 31 days alder broke down faster than knotweed. After 56 days, all of the leaf packs were mostly decomposed. Overall, the major discrepancies between leaf species were those related to initial litter structural and chemical quality. However, changes in the timing and quantity of litter inputs are important factors to be considered in understanding the impact of invasive knotweed on stream ecosystem processes. Bohemian knotweed drops all of its leaves in a three to four week period with the first hard frosts of late fall. In the Pacific Northwest, native deciduous shrubs and trees drop the majority of their leaves in the fall over a two to three month period, and coniferous trees shed litter over even longer time periods. Studies in England and France also found that aquatic hyphomycete and invertebrate assemblages differed between stream sites with and without knotweed (Lecerf et al. 2007).

Because knotweed inhibits native tree seedling establishment in riparian zones (Urgenson et al. 2012), it can also affect future large woody debris recruitment into streams, significantly affecting channel dynamics and fish habitat, potentially negatively affecting state and federally listed fish species (NMFS 2010).

Knotweed is also suspected to:

- destabilize stream banks, changing the patterns and amounts of streamside erosion and sediment input into streams, decreasing habitat quality for fish and other aquatic animals;
- provide little or no food or nesting habitat for native birds and mammals;
- modify the microclimate, making the area inhospitable to many native wildlife species, including reducing amphibian foraging success.

Once knotweed becomes established, it forms large monotypic stands that eliminate all native vegetation, are persistent, and are extremely difficult to eradicate. It can reproduce from tiny root or stem fragments, which are readily transported by water, wildlife, and humans. If unchecked, stands continue to expand and provide propagules that exacerbate infestations downstream and via other transportation routes.

Maerz et al. (2005) studied the effects of knotweed on green frogs (*Rana clamitans*) in terrestrial fields near wetlands. Frogs were allowed to forage in feeding buckets along transects that traversed ground from non-invaded to knotweed-dominated areas. They found that change in frog mass declined significantly along transects, with most frogs in non-invaded plots gaining body mass and no frogs in knotweed-invaded plots gaining mass. It was noted in the discussion that many factors would have been involved in the foraging activity of the frogs, but their results led them to hypothesize that knotweed invasions indirectly degrade terrestrial habitat quality for frogs by reducing arthropod abundance. Their study of vegetation structure and composition on the test sites showed that diverse assemblages of native plants that covered non-invaded plots were absent from areas invaded by knotweed.

KNOTWEED CONTROL OPTIONS

Since 2008 SPU, King County, and Forterra have received over $1,30,000 in grants for programs to control this destructive plant and restore riparian areas along the Cedar River below Landsburg. They have worked along a total of 19 river miles, using herbicides to treat knotweed scattered over 105 acres of riparian habitat. They have planted over 20,000 native plants, worked with 368 landowners and engaged over 900 volunteers (Stewardship-In-Action 2014). Continued upstream control in the CRMW is essential to the success of these extensive efforts to control knotweed downstream and restore critical habitat used by salmon and numerous other wildlife species.

No non-herbicide control methods for large knotweed patches has been found to be successful (USDA Forest Service 2010). SPU attempted to control a total of 4.5 acres in the CRMW by continual covering with geotextile fabric for eight years (2004-2012). This expensive (>$200,000) attempt was successful only on the smallest patches. Because of the scale of spread of knotweed and the extreme difficulty of control by physical means, The Nature Conservancy (2002) has recognized that herbicides will often need to be the primary means of control. Most cities and counties in western Washington are using herbicide to control knotweed, including both upland and riparian areas. The Washington Department of Ecology, in its Integrated Pest Management Profile for Knotweed (2007) states: “Except for small patches that might be controlled non-chemically, any management of the species will likely require some herbicide use.”

Scientists from the Washington State Extension Program and the King County Noxious Weed program have found that imazapyr is the safest and most effective herbicide for treating knotweed, resulting in the highest mortality and using the smallest amount of chemical (King County Noxious Weed Control Program 2015, Dr. T. Miller, pers. comm. 2014). Most land managers throughout western Washington are now using targeted foliar spray of 1% imazapyr on knotweed, as it is currently the least toxic and most effective option.

Imazapyr is a non-selective herbicide used for the control of a broad range of invasive plants including terrestrial annual and perennial grasses, broadleaved herbs, woody species, and riparian and emergent aquatic species. It can only be applied as a foliar spray (not stem injection). Only glyphosate, which has higher toxicity than imazapyr, is certified for use with stem injection. Experience has shown that the stem injection method typically uses about five times more herbicide than foliar spraying, with no greater knotweed mortality rates. The advantage of using stem injection can be lower mortality to adjacent native plants. However, six years of experience in the CRMW has demonstrated that when foliar spray is correctly applied, there is no damage to adjacent plants in the conditions we have in the watershed.

IMAZAPYR CHEMISTRY AND MODE OF ACTION

Imazapyr is the common name for the chemical 2-[4,5-dihydro-4-methyl-4-(1-methylethyl)-5oxo-1*H*-31 imidazol-2-yl]-3-pyridinecarboxylic acid. It is sold under numerous trade names, and has both terrestrial and aquatic formulations. The aquatic formulation does not include surfactant. Several studies have found that the surfactant in terrestrial glyphosate formulations may be more toxic to amphibians than the main ingredient itself (King and Wagner 2010, Relyea and Jones 2009, Cauble and Wagner 2005). There is concern that this may also be true for imazapyr formulations. For this reason, we always use the aquatic formulation of imazapyr and mix with the least toxic surfactant available (see surfactant discussion below).

Imazapyr is absorbed quickly through plant foliage, and can also be taken up by roots. It is moved readily within the plant to the growing meristematic tissues, where it inhibits the enzyme acetohydroxy acid synthase (AHAS), also known as acetolactate synthase (ALS) (Tu et al. 2004). ALS is required for the synthesis of three essential amino acids required for protein synthesis and cell growth in the plant (valine, leucine, and isoleucine). Only plants have ALS and produce these three amino acids; animals must obtain them from their diet. Because animals do not synthesize these amino acids, imazapyr is specifically toxic to plants and has low toxicity to humans and other animals (including mammals, birds, fish, and insects) (Massachusetts Department of Environmental Protection 2012, Durkin 2011, Bautista 2005, Durkin and Follansbee 2004). 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.

IMAZAPYR TOXICITY – GENERAL INFORMATION

As noted above, because imazapyr inhibits an enzyme and amino acid synthesis found only in plants, it is classified as a Category III (low toxicity) herbicide by the US Environmental Protection Agency (EPA 2006). Imazapyr has relatively low toxicity to mammals, showing low toxicity if individuals get residues on their skin, and very low toxicity if it is eaten or inhaled. It is classified as “practically non-toxic” to “slightly toxic” to fish and “practically non-toxic” to birds.

Most of the toxicology studies are unpublished reports submitted to the EPA as part of the registration and re-registration process. This can potentially be a concern of bias. But, as stated in Durkin (2011), this concern is largely without foundation because there are strict guidelines developed by the EPA for conduct and reporting of studies. Plus all of these types of studies are conducted under Good Laboratory Practices, an elaborate set of procedures involving documentation and independent quality control and quality assurance that typically exceeds that seen in open literature peer-reviewed publications. Finally, the EPA reviews each study for adherence to their guidelines and practices.

IMAZAPYR RISK TO HUMAN HEALTH

Human health risk is evaluated in relation to toxicity testing on mammals. As reported by EPA (2006) and reviewed in Durkin (2011) and AMEC (2009), the acute oral LD_{50} (lethal dose at which 50% of the test subjects die) is greater than 5,000 mg/kg for mammals. This is the highest dose tested, but is a dose which still did not achieve a 50% mortality in laboratory animals. So a definitive mammal LD_{50} was not able to be determined. The chronic dietary No Observed Adverse Effect Level (NOAEL) is 10,000 ppm in dogs, rats, and mice.

An adequate number of multi-generation reproductive and developmental studies were conducted, and none indicated any adverse effects on reproductive capacity or normal development. Results of assays for carcinogenicity and mutagenicity are consistently negative, so the EPA categorizes imazapyr as Class E: evidence of non-carcinogenicity (EPA 2006). The EPA human health risk assessment for imazapyr finds no endpoints of concern associated with systemic toxic effects for either acute or chronic exposures (Durkin 2011). Available data indicate that orally administered imazapyr is well absorbed, and the majority of the dose is rapidly excreted unchanged in urine and feces (Durkin 2011). No endocrine or immune system effects were observed. Only one study of very high intravenous doses showed any signs of neurotoxicity (AMEC 2009). No other studies showed any neurotoxic effects.

Some clinical case reports of human intentional (attempted suicide) or accidental ingestion of large amounts of the formulation Arsenal are reported in the open literature. The reported signs and symptoms of imazapyr poisoning included vomiting, impaired consciousness, and respiratory distress requiring intubation (Lee at al. 1999). The respiratory distress was likely due to aspiration from vomiting and not from the imazapyr. There are no reports of human fatality due to large amounts of imazapyr ingestion. (Durkin 2011).

Studies on effects of acute dermal exposure, up to 2,000 mg/kg were not associated with any signs of systemic toxicity (AMEC 2009). When risk characterization for workers was computed, even at the highest application rate modeled, the upper range of hazard quotients was below the level of concern by a factor of 8.5 (AMEC 2009). Imazapyr is reported as a mild skin irritant and mild eye irritant. Two studies of 99.3% imazapyr powder (acid) installed directly into the eye not surprisingly found severe and irreversible eye damage (Durkin and Follansby 2004). Because only dilute liquid and not concentrated powder is used in general herbicide application, this finding was not considered relevant to the risk assessments (Durkin 2011).

Dr. Allan Felsot, a well-known and respected toxicologist and professor of environmental toxicology at Washington State University, prepared a worst case scenario for this project in which the entire maximum annual amount of herbicide used on all of the knotweed in the CRMW (not just that within the hydrographic boundary) was put directly into Lake Youngs, the municipal water storage lake. That would result in a concentration of 26.6 parts per trillion of imazapyr. He assumed no breakdown of the chemical and evaluated the human health risk of this concentration in the drinking water. His data showed that this concentration was at least 600,000 times lower than a benchmark derived from an exposure that is already 100 times less than a dose that was found to cause no adverse effects on a human child. Thus the risk from this concentration could not be distinguished from nil.

IMAZAPYR ENVIRONMENTAL RISK

In both 2005 and 2011 risk assessments, the US Forest Service found that no adverse effects are likely to occur for a variety of mammals and birds, with spraying at any typical application rate (Durkin 2011, Bautista 2005). Studies evaluated both acute (single) and chronic (extending over the average species lifetime) exposures. Test animals included small mammals such as mice, small insectivorous mammals, both large and small herbivorous mammals, medium carnivorous mammals, fish-eating birds, herbivorous birds, predatory birds, and insectivorous birds. Studies indicate that imazapyr is rapidly excreted in urine and feces by mammalian systems, with no bioaccumulation in the liver, kidney, muscle, fat, or blood (Soll 2004, Miller 1991). Although herbicides contain inert ingredients that are considered proprietary, these toxicity tests were performed on the entire formulation, not just the active ingredient, indicating that the inerts likely have low toxicity as well.

A peer-reviewed field study found that there were no adverse effects on benthic macroinvertebrates (including invertebrate biomass, community composition, and deformities) at rates as high as 100 times normal applications (Fowlkes et al. 2003). Another peer-reviewed study tested the embryos of zebra fish (*Danio rerio*), in an extremely sensitive in vivo test for the effects of endocrine system dysfunction (Stehr et al. 2009). They found an "absence of toxicity at relatively high exposure concentrations".

Trumble et al. (2009) in an acute toxicity study of bullfrog tadpoles (*Rana catesbeiana*), a surrogate for native amphibians, found the LC^{50} (lethal concentration in water in which 50% of the subjects die) for imazapyr was 1,739 mg/L. Any concentration over 100 mg/L is considered practically non-toxic. This extremely high concentration required to achieve 50% mortality indicates that imazapyr has very low toxicity to the tadpoles.

In a toxicity study directed at the Oregon spotted frog (*Rana pretiosa*), listed as federally threatened, Yahnke et al. (2013) exposed juvenile spotted frogs to tank mixes of imazapyr (aquatic formulation), surfactant (AgriDex), and dye for 96 hours at concentrations associated with an application rate of up to 96 oz/ac. Following exposure, the frogs were reared for two months. No mortalities or changes in feeding behavior, growth, or body and liver conditions were found. The tank mix used in the study (aquatic formulation and AgriDex) is the same one used in the CRMW, except that our application rate is far lower and we never apply to water.

In another amphibian toxicity study, Hurley and Shanaman (2007) conducted a risk assessment of imazapyr to the California red-legged frog (*Rana aurora draytonii*), also federally listed as threatened. They found that no direct adverse effect were expected for either the aquatic or terrestrial phase of the frog. They also found no indirect adverse effects through food sources were expected.

A recent study compared the relative sensitivity of amphibians and fish to over 50 different chemicals (Weltje et al. 2013). They found that for both acute and chronic sensitivity, amphibians and fish had very similar responses. So recent concern that amphibians may be more sensitive to various chemicals than fish may be unjustified.

IMAZAPYR RISK TO POLLINATORS

European honey bee (*Apis mellifera*) Colony Collapse Disorder (CCD) is a major concern in western Washington, as well as throughout the country and world. Since the disorder was first named in 2007, population declines in both the European honey bee and native bees and other pollinators have continued. Native bumble bees in particular have suffered significant range restrictions and reduced abundance (Hatfield et al. 2012). These pollinator declines have a significant negative effect not only on agricultural crop production, but also on native plant reproduction, and thus native biodiversity.

Recently, neonicotinoid insecticides (insecticides are pesticides specifically designed to be toxic to insects) have been identified as likely contributors to the population declines (Hopwood et al 2012). Unlike earlier insecticides, they are long-lasting compounds that can be systemic within the plant (including pollen and nectar), and are now extensively used both in agriculture and by homeowners. Several of these types of insecticides, including imidacloprid, the most widely used neonicotinoid product, are toxic at high doses to both honey bees and bumble bees (Schmuck et al. 2001). Data for chronic low dose exposures are less clear. It may or may not cause mortality, depending on specific factors and conditions. However, it still may cause sublethal alterations in navigation, learning, and foraging activity (Han et al. 2010, Decourtye et al. 2003).

Although no direct link has been demonstrated between neonicotinoids and CCD, it is likely one of several major contributors and stressors. Other contributors likely include disease, parasitic bee mites (including *Varrao* mite) and miticides used to control them in the hives, fungus and fungicides, nutrition, and synergistic effects between the stressors (Sanchez-Bayo and Goka 2014, Johnson et al. 2010). In their risk assessment of pesticide residues and bees, Sanchez-Bayo and Goka (2014) reported that a total of 161 pesticides have been found in bee hives, of which 83 were insecticides, 40 fungicides, 27 herbicides and 10 acaricides. Of the 49 most common compounds, six were herbicides, and none included imazapyr. Johnson et al. (2010) listed 121 pesticides found in apiary samples of wax, pollen, bee and honey, and imazapyr was not among them. Likewise, Wu et al. (2011) found 39 pesticides in brood combs, of which only two were herbicides and neither were imazapyr.

In a Pacific Northwest university extension pamphlet detailing how to reduce bee poisoning from pesticide, Hooven et al. (2013) listed causes of bee poisoning. The primary causes are highly toxic insecticides with residual toxicity longer than 8 hours. Their only reference to herbicides was in the quote: "The mode of action of herbicides affects plants, not insects, and herbicides are unlikely to cause bee poisoning incidents under field conditions." Imazapyr was not included in the 150 active ingredients most likely to cause bee toxicity (Hooven et al. 2013).

Imazapyr toxicity to humans and animals discussed above also applies to insects, i.e., because imazapyr inhibits enzymes found only in plants, it has very low bee toxicity. The honey bee was tested for toxicity during the initial toxicity studies (Atkins 1984, Atkins and Kellum 1983, cited in Durkin 2011), where the LD_{50} for both oral and contact toxicity studies was >0.1mg/bee (or >1,000 mg/kg of body weight). This is similar to the NOAEL values reported for mammals and birds. As with mammals and birds, they were unable to reach an LD_{50} level at the highest doses tested (i.e., less than 50% of the test subjects died).

Stark et al. (2012) conducted a study on the effects of three herbicides on Behr's metalmark butterfly (*Apodemia virgulti*), one of which was imazapyr. They used the terrestrial formulation (which includes surfactant) and the maximum legal allowable dose (96 oz/ac, compared to our 2015 application rate of 3.4 oz/ac in the CRMW) and sprayed butterfly instars while they were on buckwheat. In addition, they sprayed only the buckwheat, then fed it to the larvae. All three herbicides reduced the number of individuals reaching the pupal stage by 24-36%. Because each herbicide had a different mode of action, the authors stated that the effects were likely due either to 1) inert ingredients or 2) indirect effects on food plant quality, rather than direct toxicity from the herbicides. Stark (2015 pers. comm.) stated he knew of no ongoing or planned studies looking at the effects of imazapyr on bees or other pollinators. In 2015, the Pesticide Program Director for the Xerces Society for Invertebrate Conservation, (A. Code pers. comm.), also knew of no research looking at toxicity of imazapyr on pollinators. There are no published data to indicate that dermal contact or ingestion of imazapyr by bees or other pollinators causes any toxic effects, lethal or sublethal. Because past research has not found any significant toxicity of the herbicide imazapyr to bees, researchers are focusing on insecticides, many of which are highly toxic to pollinators, as discussed above.

Herbicides can indirectly affect pollinators if they remove a significant portion of their food sources. This can be a concern with knotweed, as large flowering patches can be used extensively by bees. In the municipal watershed, we bent the canes prior to the first herbicide treatment, then treated the regrowth which had no flowers. Our experience in the CRMW is that after the first herbicide treatment, the knotweed above-ground biomass is greatly reduced, and only an occasional isolated plant might produce a few flowers. But the vast majority of plants do not flower in subsequent treatment years. This has been confirmed by several other land managers in western Washington at knotweed working group meetings in 2015. In all years of treatment in the CRMW, 2010-2015, we have not observed any bees or other pollinators on the knotweed before, during, or after spraying.

In 2015 the King County Noxious Weed Control Program clarified its already existing practices with regard to treating knotweed with herbicide and potential effects on pollinators. In their updated Best Management Practices for Knotweeds brochure (King County 2015), they state that they avoid spraying knotweed when bees or other pollinators are present whenever feasible. Likewise, SPU also has always avoided spraying any plants when pollinators were present, but as stated above, this has not been an issue because the plants we have treated have not been in flower and no pollinators have ever been observed on the non-flowering plants.

SPU shares the concern about pollinator population declines. Consequently, we are planting a range of native flowering plants whenever appropriate during our restoration projects, including restoration of sites formerly dominated by knotweed. We choose a variety of native plants that have different flower colors and shapes, with flowering periods that vary throughout the growing season, providing nectar and pollen to a large number of pollinator species, focusing especially on our native bumble bees (Hatfield et al. 2012). This diversity of native species should provide better native pollinator habitat than the invasive knotweed, which flowers for a single short period during late summer or early fall, depending on weather, elevation, and site-specific factors such as soil type and moisture.

IMAZAPYR BREAKDOWN PROCESS AND PRODUCTS

Imazapyr is water soluble and is broken down by sunlight in water with a reported half-life in water as short as two days (Soll 2004), but no longer than five days (EPA 2006). A study of the persistence of imazapyr associated with smooth cordgrass control in an estuary in Willapa Bay, Washington State, found half-lives were <0.5 day in water and 1.6 days in sediment (Patten 2003).

The half-life of imazapyr in soils in the field have been has been reported to be as short as 10 days to as long as 17 months in humid temperate climates, depending on soil type and particle size, pH, temperature, moisture content, and organic material content. In soils imazapyr is degraded by microbial metabolism. Because imazapyr is water-soluble, it can move in soil and can potentially enter the ground water. However, amount of movement depends on soil pH. Below pH 5, adsorption capacity of imazapyr increases and its movement in soils is limited (Soll 2004). Most forest soils in western Washington are acidic, with soils under Douglas-fir generally under pH 6, and under red alder (common in riparian areas) under pH 5 (pers comm. Darlene Zabowski, soil science professor, University of Washington).

Imazapyr is degraded slowly in soils primarily by microbial metabolism. It will undergo rapid photodegradation (breakdown by sunlight) in water, 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 for continued uptake by the target species until it is degraded by soil microbes.

In water imazapyr initially photodegrades rapidly to two primary products, “CL 119060”, and “CL9140” (7-hydorxyfurol[3,4-b]pyridine-5(7H) and 2,3-pyridinedicarboxylic acid). According to the manufacturers, CL119060 is biologically oxidized to CL 9140, and eventually mineralizes to carbon dioxide (CO_2) following the cleavage of the pyridine ring structure. Both imazapyr degradation products rapidly degrade, with half lives of two to five days (Mangels and Ritter 2000).

Dr. Felsot, referenced above in the Risk to Human Health section, was asked about the potential toxicity of breakdown products. He said that all of these compounds are biodegradable. When the formulation is given to test animals in high doses, they result in similar breakdown products within the animals as would occur in the environment. Indeed, these breakdown products are even more bioavailable than any that would occur in the environment because they are already in systemic circulation within the animal. In the environment, bioavailability is limited by interactions with solid surfaces, such as soil, sediment, plant waxes, etc. Thus, these breakdown products, if toxic in and of themselves, would have affected the physiology of the test animals. Yet, all of the listed compounds do not cause acute toxicity at environmental levels of exposure. In fact, none of the compounds even cause chronic or sub-chronic toxicity at levels of environmental exposure.

ADJUVANTS

Adjuvants are compounds added to the formulation or the spray tank to improve its performance. They can enhance the activity of an herbicide’s active ingredient (activator adjuvant, including surfactants) or offset any problems associated with its application (special purpose or utility modifiers such as defoamers). On the label these compounds are often called inert or other ingredients. Surfactants are one type of adjuvant that makes the herbicide more effective by increasing absorption into the plant by lowering the surface tension between the liquid herbicide formulation and the solid leaf surface. Adjuvants can make a significant difference in how well the herbicide treatment works. Adjuvants present in terrestrial formulations generally include both inert ingredients and surfactants (discussed separately below). Those in aquatic formulations include inert ingredients, but not surfactants.

INERT (OTHER) INGREDIENTS

Formulations of herbicides often contain proprietary carriers and other so-called “inert” ingredients that are usually not identified on herbicide labels. The EPA now uses the term “other ingredient” rather than “inert” to describe these compounds that are intentionally added to a formulation, but have no inherent herbicidal activity. Inerts are most often added to the formulation to facilitate its handling, stability, or mixing.

Inerts and surfactants are not under the same registration guidelines as are the active ingredients in pesticides. The EPA classifies these compounds into four lists based on the available toxicity information. List 1 contains “inerts of toxicological concern”; List 2 contains “potentially toxic inerts, high priority for testing”; List 3 contains “inerts of unknown toxicity”; and List 4 contains “minimal risk inerts” or “inerts for which EPA has sufficient information to conclude that their current use patterns will not adversely affect public health or the environment.” If the compounds are not classified as toxic, then all information on them is considered proprietary and the manufacturer need not disclose their identity.

The identity of inert compounds used in imazapyr formulations is generally confidential, but Syracuse Environmental Research Associates (SERA) reviewed them, using the Freedom of Information Act, for preparation of risk assessments conducted for the US Forest Service (Durkin 2011, Bautista 2005, Durkin and Follansbee 2004). They conducted very comprehensive searches of the literature and used peer-reviewed articles from public scientific literature, current U.S. Environmental Protection Agency documents available to the public, and Confidential Business Information to evaluate toxicity and risk from the herbicides analyzed. No apparently hazardous materials were identified in the review of the inerts used in either the terrestrial or aquatic formulations of imazapyr.

The Northwest Coalition for Alternative to Pesticides obtained information on inert ingredients in the formulation Arsenal (aquatic formulation) under the Freedom of Information Act, and posted it on their website. The only inert listed other than water is glacial acetic acid (defined as anhydrous or water-free acetic acid, i.e., undiluted). Dilute acetic acid, the major component in vinegar, is an approved food additive and is classified as a Generally Regarded as Safe compound (AMEC 2009).

SURFACTANTS

There are several types of surfactants, including non-ionic which form stable emulsions, oil-based or methylated seed oil concentrates, organosilicone, and nitrogen containing compounds. They are usually proprietary blends of heavy-range paraffin-based petroleum oil, polyol fatty acid esters, and/or polyethoxylated derivatives thereof. They improve pesticide application by modifying the wetting and deposition characteristics of the spray solution, resulting in a more even and uniform spray deposit on the leaves of the target species.

There is generally scant information on the human health and environmental effects of surfactants. However, most studies have found the least toxic surfactant to be Agri-Dex®, a crop oil concentrate containing a proprietary blend of heavy range paraffin base petroleum oil and polyoxyethylate derivatives of polyol fatty acid esters. Acute toxicity studies by the Washington State University and the DFG-Aquatic Toxicology Lab have indicated that Agri-Dex is “practically non-toxic” and is less toxic to fish and aquatic invertebrates than R-11®, a very commonly used surfactant (Anderson unpublished report).

In toxicity tests on rainbow trout performed by the Washington Cooperative Fish and Wildlife Unit at the University of Washington, Agri-Dex was found to be by far the least toxic surfactant tested (Smith et al. 2004). In their laboratory tests it took 271 parts per million (ppm), or a concentration of >1000 mg/L, for an LC50 dose (the concentration at which 50% of the test subjects died). This compares to only 6 ppm for R-11, 17 ppm for LI700, and 74 ppm for Hasten. They also studied the relative concentrations of the surfactants in relation to water depths expected in the field. Even at the maximum allowed concentration of Agri-Dex of 5% (>5 times that used in knotweed control), a trout stream would have to be sprayed directly and be less than 5 mm (or about ¼ inch) deep in order to reach the LC50 concentration for trout. Clearly trout could never survive in such shallow water, so in practice no mortality would occur.

The 2008 Material Safety Data Sheet (MSDS) for Agri-Dex reports that it is expected to be adsorbed to soil and should be biodegradable. Bioaccumulation is unlikely due to the low water solubility of the product. Animal toxicity data for similar products required very large doses (>2,000 mg/kg) to cause mortality, showed low inhalation toxicity, and were practically non-irritating to skin and eye in tests on rabbits.

The Washington State Department of Agriculture requires aquatic toxicity tests if a surfactant is labeled for aquatic use in that state. In 2012 they summarized the aquatic acute toxicity data for adjuvants allowed for use on aquatic sites (WSDA 2012). Of the 25 products reviewed, Agri-Dex had by far the least toxicity to rainbow trout and daphnids (LC50 of >1000 mg/L).

Consequently, SPU uses Agri-Dex (0.5 – 1%) as the surfactant mixed with the aquatic formulation of imazapyr to treat knotweed in the Cedar River Municipal Watershed. All available data continues to indicate that this combination is the least toxic option.

APPENDIX II

Photographic record of results of knotweed treatment with imazapyr at the Education Center, 2010 – 2015. This site has had the most re-growth of any of the large treatment sites, so represents the worst case scenario during this time period.

Knotweed before initial 2010 treatment. 12-foot tall knotweed covered the entire site.

May 2011. Spring after the first treatment, showing the dead canes from the first treatment. Canes had been bent prior to treatment to facilitate access for the applicators.

August 2011. One year after first treatment, showing dead canes, knotweed regrowth, and initial invasion by Himalayan blackberry.

August 2011. Large patch of Himalayan blackberry encroaching one year after first treatment.

September 2012. One year after 2nd treatment, showing scattered medium sized knotweed plants. Dead canes had been hand-cleared from the site to make finding re-growth easier.

September 2012. Invasive black locust take over a portion of the site one year after 2nd treatment. Mullein, foxglove, and other non-native plants are also starting to invade.

September 2013. One year after 3rd treatment and initial KCD grant restoration work (blackberry, locust, other invasive species removal, planting native trees and shrubs).

October 2014. One year after 4th treatment, with continued KCD grant restoration work (spreading mulch, planting).

August 2015. One year after 5th treatment, showing small scattered knotweed plants amongst the planted native trees and shrubs.

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