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Review: Physiological Approaches to the Improvement of Chemical Control of Japanese Knotweed (*Fallopia japonica*)

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Japanese knotweed is an aggressive alien species in Europe, North America, and Australia, causing a range of environmental problems. Eradication of Japanese knotweed is proving to be a difficult task, because the plant is able to propagate generatively by intra- and interspecific hybridization, and vegetatively from shoot and tiny rhizome pieces. Despite the economic consequences of Japanese knotweed on natural and built environments, its physiology is not yet fully understood; especially important are sink-source relations between old and young parts of the rhizome and growth of lateral and latent rhizome buds. Current methods of chemical control include three types of phloem-mobile herbicides, such as glyphosate, imazapyr, and synthetic auxins. These herbicides have limitations on their use, and all fail to eradicate the plant completely, for the reasons discussed in this review. Our aim is to suggest prospective approaches to enable chemical eradication: use of signals to induce controlled growth and development of quiescent rhizome buds; use of phytohormones, sugars, and light to increase allocation of phloem-mobile herbicides to the rhizome; use of xylem-mobile herbicides to exterminate the old rhizome parts; and use of different phloem-mobile herbicides at different growth stages.

Nomenclature: Glyphosate; imazapyr; synthetic auxins; Japanese knotweed, *Fallopia japonica* (Houtt.) Ronse Decraene POLCU.

Key words: *Fallopia*, *Polygonum cuspidatum*, *Reynoutria*, source-sink, phytohormones, photoregulation, glyphosate, imazapyr, 2,4-D.

Japanese knotweed is a perennial rhizomatous herb, native in Asia, where it can be found in a variety of habitats. The species belongs to the family Polygonaceae, but has been referred to three different genera because of the instability of taxonomy and nomenclature within the Polygonaceae (Beerling et al. 1994): *Fallopia japonica* (Houtt.) Ronse Decraene, *Reynoutria japonica* Houtt., and *Polygonum cuspidatum* Sieb. & Zucc. Other less-used synonyms are *Polygonum sieboldii*, *Polygonum japonicum*, *Polygonum zuccharini*, *Polygonum reynoutria*, and *Pleuropterus zuccarinii* (Child and Wade 2000).

In its native habitat Japanese knotweed reproduces by seed and by rhizome extension (Zhou et al. 2003). In its adventive range, Japanese knotweed spreads mainly vegetatively and is characteristically associated with sites subject to a degree of disturbance (Beerling et al. 1994; Bimova et al. 2003); it is also able to propagate sexually, producing a number of inter- and intraspecific hybrids (Grimsby et al. 2007). Where it is an invasive weed, the plant causes a range of environmental problems: the foliage forms a dense canopy, which restricts growth of existing vegetation (Beerling et al. 1994); the rhizome releases a series of secondary products with allelochemical properties, affecting the availability of mineral nutrients for native plants (Inderjit and Nishimura 1999; Vrchotova and Sera 2008; Weston et al. 2005); competition results in destruction of native flora, which affects faunal diversity (Lecerf et al. 2007; Maerz et al. 2005); the shoots push through asphalt, destroying pavements and car parks; and the rhizomes penetrate through foundations, walls, land drainage works, and flood defense structures (Beerling et al. 1994; Child and Wade 2000), damaging the built environment, which becomes a recurring expense if viable fragments of the rhizome remain. Taking into account the damage Japanese knotweed causes and its ability to spread (Bailey and Wisskirchen 2006), it is considered an aggressive alien species in many countries (e.g., Child and Wade 2000, USDA NRCS 2006).

There are few biological tools available to control Japanese knotweed. Where it has invaded, it has arrived without associated pests and pathogens. Only recently has the species been evaluated as a suitable target for biological control (Green 2003): Commonwealth Agricultural Bureau International (CABI) is currently conducting a 7-yr research program to identify in Asia arthropod and fungal agents that might be introduced in the European Union, and one psyllid, *Aphalara itadori*, has reached the point of official assessment for release (Shaw et al. 2009).

In the absence of biological control agents, a variety of eradication methods have been tried. Cutting and mowing seem the solution in conservation areas, but have proved to be insufficient and may even promote spread (Ahrens 1975; Beerling 1990; McHugh 2006; Seiger and Merchant 1997). Large-scale excavation combined with chemical control is the method of choice for building sites (Environment Agency 2006), but the cost of excavation is massive and its effectiveness questionable (Miller 2005; Soll 2004). Root barrier membranes can be effective in reducing the spread of Japanese knotweed (Environment Agency 2006). However, it is difficult to seal the soil containing rhizomes and shoots, because the sites are located in areas where development is to take place, so the membrane may be penetrated by service pipes, ducts, or drains or building foundations or piles.

Chemical control alone is used when digging is impossible or prohibitively expensive, and it takes a number of years to kill an extensive rhizome system (Soll 2004). The estimated cost of eradicating Japanese knotweed in the U.K. with current methods is approximately £1.56 billion (DEFRA 2003). In Washington State the state-wide Knotweed Control Program has been running since 2004, and $4.3 million has been already spent to control the weed (WSDA 2004, 2005, 2006, 2007, 2008).

Reproduction, Morphogenesis and Physiology of Japanese Knotweed

Reproduction and Propagation of Japanese Knotweed Sensu Lato. In its native habitat, Japanese knotweed is dioecious (gynodioecious, under discussion; Bailey 1994), and

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reproduces by seed and by rhizome extension (Adachi et al. 1996a; Marigo and Pautou 1998). In the U.K., genetic variability of *F. japonica* var. *japonica* was investigated, using molecular markers, such as random amplified polymorphic DNA (RAPD) and inter-simple sequence repeats (ISSRs) (Hollingsworth and Bailey 2000), and only a single genotype was detected all over the country, thus suggesting all individuals are ramets of an enormous single female clone (Bailey 1994). Consequently, rhizomes have been considered the sole means of reproduction (Zika and Jacobson 2003) and discussions of management have largely neglected the impact of seed dispersal (Child and Wade 2000; McHugh 2006; Soll 2004). But contrary to what has been reported in Britain, in the United States, *F. japonica* var. *japonica* is capable of sexual reproduction. It has been characterized as having a ''leaky'' dioecious or subdioecious system, with populations of both male and female plants that still maintain vestigial reproductive parts of the other sex. Seeds on female ramets are extremely abundant, with tens of thousands produced each year, and these can germinate under a variety of conditions (Bram and McNair 2004; Forman and Kesseli 2003). Some male ramets do set a few seeds (Forman and Kesseli 2003). In Massachusetts, simple sequence repeat markers were developed to determine the amount of sexual and clonal reproduction in three populations. Clonal growth was apparent, but the populations together contained 26 genotypes and had evidence of sexual reproduction (Grimsby et al. 2007).

In all countries, female plants *of F. japonica* var. *japonica* can also produce seeds by hybridization with related male-fertile taxa. They hybridize with a dwarf variety, *Fallopia japonica* var. *compacta*; with giant knotweed *Fallopia sachalinensis* (F. Schmidt) Ronse Decraene, and with Russian vine, *Fallopia baldschuanica* (Regel) Holub. Thus, plants called ''Japanese knotweed'' are often hybrids of different combinations with other knotweeds and their backcrosses with parental *F. japonica* (Bailey 1999). This was the reason for Bailey to refer to this mixture as ''Japanese knotweed sensu lato'' (Bailey 1999).

The hybrid between *F. japonica* and *F. sachalinensis* is called *Fallopia* × *bohemica* (Mandak et al. 2003). Reproduction and spread of this hybrid has been intensively studied in the Czech Republic and shown to be the most successful taxon in terms of regeneration and establishment of new shoots from rhizome and stem pieces (Bimova et al. 2003): the regeneration success was 61% for *Fallopia* × *bohemica*, compared to 52% for *F. japonica* var. *compacta*, 39% for *F. japonica* var. *japonica*, and 18% for *F. sachalinensis*. *Fallopia* × *bohemica* also gained the highest shoot biomass and biggest leaves. High invasiveness of *Fallopia* × *bohemica* was also confirmed when it was shown to spread approximately twice as fast as both parental species (Mandak et al. 2004). The additional danger of *Fallopia* × *bohemica*, compared to *F. japonica*, is that it produces flowers of either sex and viable seeds (Mandak et al. 2004). The hybrid between *F. japonica* var. *japonica* and *F. baldschuanica* is known as *Fallopia* × *conollyana*. Fortunately, this hybrid does not combine the vigor of its parents (Bailey 2001).

Soll (2004) noted, ''should extensive sexual reproduction be confirmed in the field, it would certainly alter the strategy for landscape level control projects.'' Thus we emphasize that with the new evidence from studies on sexual reproduction of *F. japonica* and genetic diversity of ''Japanese knotweed sensu lato,'' management for this weed should no longer ignore seed as a form of propagation.

Development of Patches. The development of a patch of Japanese knotweed starts from a seed or a vegetative fragment. Fragments as tiny as 1 cm can develop into a plant (Bimova et al. 2003). The growing plant develops several rhizomes, and the apical bud of each rhizome develops into a few aerial shoots. At the end of the growing season, the aerial shoot produces subterranean winter buds at its base and then withers. The following spring, the winter buds produce new aerial shoots, in almost the same position every year, forming a cluster of shoots called a ''shoot clump'' (Adachi et al. 1996a) (commonly referred to as a ''crown''). While the shoot clump keeps producing new shoots, the lateral buds on the rhizome remain dormant. When the shoot clump dies (their life span in native habitats is about 5 yr; Adachi et al. 1996a), a few dormant buds on the rhizome develop. The daughter rhizome branches from the mother rhizome with a regular angle and extends in a fairly straight line; apices of daughter rhizomes then develop new shoot clumps (Adachi et al. 1996a). While the patch develops by iterating these steps, after 6 yr the shoot density starts to decrease in the center, and finally a ring of shoots stands around an area of bare soil (Adachi et al. 1996a). This phenomenon is called ''central die-back.'' It is not clear yet if rhizomes remain alive in this central area, but if so, they do not produce new shoots. It has been shown that central die-back is not brought about by either interspecific or by intraspecific competition, but by the morphology of the rhizome (Adachi et al. 1996a), and a stochastic model of rhizome growth has been developed (Adachi et al. 1996b). Specific morphogenetic rules of Japanese knotweed development in native habitats were revealed by the modeling: the species has an intrinsic mechanism of programmed cell death, because the life span of shoot clumps is limited to 5 to 6 yr; the shoot apical dominance suppresses the rhizome branching; the branching of rhizome occurs when space for rhizome growth becomes limited, i.e., at an angles sharper than 40°.

The growth pattern described above does not hold for many invaded countries (in the U.K., observations by P. Beckett, unpublished data), where stems may grow up to 3 m tall and form densely packed crowns; in Japan stems are short and sparse (Bailey 2003). This finding has led to the conclusion that Adachi's research (Adachi et al. 1996a,b) was performed on a native variety that resembles *F. japonica* var. *compacta*, rather than *F. japonica* var. *japonica* (Smith et al. 2007). Also, rhizome segments are not straight and the branching angle is much sharper, at 5° to 15°, compared to 30° to 50° in Japan (Smith et al. 2007). Thus, for invaded countries, a ''3-D correlated random walk model'' of the rhizome network of *F. japonica* var. *japonica* has been developed, which predicts the rhizome's ''clonal growth parameters'' (i.e., how fast the stand will grow, segment lengths, branching angles, number of daughters, and probability of forming a crown; Smith et al. 2007).

Sink-Source Relations between Above- and Belowground Organs. It is a popular view that phloem-mobile herbicides can be delivered to rhizomes of Japanese knotweed with the flux of photosynthetic assimilates from leaves. Thus, it is important to understand changes in sink-source relations between below- and aboveground organs of the plant during the year. Price et al. (2002) showed that in May and June (early summer in the Northern Hemisphere), all freshly synthesized carbohydrates (80 to 90%) were retained in shoots, but later in summer transport to belowground organs began: 35% of fresh photoassimilates were diverted toward rhizomes in August and 70% in September (Price et al. 2002). But before shoot senescence (sometime in October), transfer of carbon into rhizomes increased sharply: in September (before shoot senescence), only 15% of carbon fixed in May was recovered from the rhizome; however, in the following April (after the shoot withering and before the regrowth of new shoots), 90% of carbon fixed in May was recovered from the rhizome (Price et al. 2002). These data demonstrated clearly that the rhizome is the most effective sink in late autumn, during shoot senescence. Thus, earlier in the season, allocation of herbicides to the rhizome is lower than later in the season, so they are less likely able to kill the rhizome.

The ability to kill Japanese knotweed depends both on poisoning of storage parenchyma in the rhizome and, equally, on the ability of a herbicide to penetrate into rhizome buds. Unfortunately there are no direct studies concerning development, growth, and sink activity of the lateral buds (on rhizome surface) and latent buds (in rhizome cortex). What is known of the activity of rhizome buds during the year is summarized below.

Beerling (1990) showed that during the summer the growth of the rhizome buds was suppressed by shoot apical dominance and only cutting or poisoning of aerial shoots induced the lateral spread of clumps, so in summer the rhizome buds are likely to be dormant. During winter, rhizome biomass did not change and stored carbohydrates were not utilized (Price et al. 2002), so the buds on the rhizome are also likely to be dormant during winter. But in spring, active development of crown-based buds into aerial shoots was observed (Child and Wade 2000), so a simultaneous growth of the rhizome buds might be predicted. It has also been shown that during spring, growth of aerial shoots was strongly regulated by the mobilized rhizome resources (Price et al. 2002; Suzuki 1994): shoot mortality was found to be low, and shoots of smaller size grew faster than larger shoots, giving the clone an even appearance in height (Suzuki 1994). Thus, we suggest that the rhizome buds are weak sinks during winter dormancy and during summer, when shoot apical dominance is established. However, the season in which they are active remains unclear: Is it during autumn, when the rhizome is the active sink, or during spring, when the rhizome is the active source? When this question is answered it will be possible to adjust the application of herbicides and poison rhizome buds along with rhizome parenchyma.

Physiology of the Old Rhizomes. Everything described above concerning sink-source relations applies only to the part of the rhizome that produces aboveground shoots. Older parts of the rhizome, which already lack aboveground shoots, might be physically connected with younger parts, but are dead and not “dangerous.” As far as eradication of Japanese knotweed is concerned, it is important to know whether a patch is physiologically connected belowground. If there is still a physiological connection, then the old rhizome and its buds are alive, so potentially threatening.

That a patch of Japanese knotweed is physiologically integrated was hypothesized following a study of the distribution of nitrogen in the patch (Adachi et al. 1996c). In the oligotrophic desert of Mount Fuji, nitrogen accumulated in the soil in the central part of the patch, whereas nitrogen levels at the periphery were one-fifth to one-tenth the levels found at the center. Such distribution happened because of mineralization of dead stalks in the patch center. However, nitrogen concentration in leaves showed almost homogenous distribution throughout the patch. When the old rhizome was severed from the younger rhizome, leaf nitrogen in severed young parts decreased. The consequences of nitrogen depletion were drastic: shoots stopped growing; their height, diameter, weight, number of leaves and branches, and total chlorophyll did not change, whereas in control patches the parameters were steadily increasing. The parameter most affected was flowering: 82% of the shoots in control patches bore flowers, but there was none in severed patches. When fertilizer was applied, growth of shoots recovered and flowering resumed up to the control value (Adachi et al. 1996c).

These data support the idea that old and young parts of Japanese knotweed rhizome are connected physiologically and strongly suggest the acropetal translocation of nitrogen and (presumably) other nutrients. It can be speculated that the old rhizome and its buds are alive, and that their livelihood depends on basipetal export of photoassimilates from shoots on younger parts of the rhizome. If so, this suggests that it would be even more difficult to deliver a lethal amount of phloem-mobile herbicide to the old rhizome than to younger parts, because the sink strength of the old rhizome is very low, as it was shown for quackgrass [*Elytrigia repens* (L.) Desv. ex Nevski] (Shieh et al. 1993). This suggests several crucial questions: How can efficacious levels of herbicide be delivered into the old rhizome and its buds? Could xylem-mobile herbicides be used to poison the old rhizome (instead of phloem-mobile herbicides used for the younger rhizome), as they effectively translocate mineral nutrients and water, rather than photoassimilates? At what time of the year is the old rhizome most metabolically active? Under what circumstances do buds on the old rhizome become active and grow?

Current Methods of Chemical Control: Three Types of Herbicide

Glyphosate. Glyphosate is the herbicide most frequently recommended to control Japanese knotweed (Child and Wade 2000), because it is nonpersistent and approved for use near water. Chemically it is a substituted glycine (N-[phosphomethyl] glycine) and in planta it inhibits the activity of enolpyruvyl shikimate phosphate synthase, which is involved in synthesis of phenolic amino acids (Amrhein et al. 1980). A reduction in concentration of phenolic amino acids reduces protein synthesis, eventually killing the plant (Geiger and Bestman 1990). It was also shown that glyphosate strongly inhibits induction of nitrite reductase (Cole et al. 1980), which could potentially result in inhibition of synthesis of all amino acids (Hoagland et al. 1979).

Glyphosate has been shown to control Japanese knotweed (see for example Child et al. 1998; Roblin 1988), although it often fails to kill (see Barney et al. 2006). During the last two decades, glyphosate has been widely used against other weeds, so some information is available to explain the limits of its efficacy.

Glyphosate was found only to be effective when a lethal dose was accumulated; for example, in high doses it eradicated volunteer potato in plantations of genetically modified glyphosate-tolerant sugar beet (*Beta vulgaris* L.) (Dewar et al. 2000). In sublethal doses it affected growth and the partitioning of carbon in many species, but did not kill them (Baur et al. 1977; Coupland and Casely 1975; Geiger et al. 1986; Scorza et al. 1984). Moreover, changes in aboveground organs had little effect on underground storage as shown for sugar beet and potato sprayed with low doses of glyphosate (Geiger et al. 1986; Haidar et al. 2005). From what is now known of the mode of action of glyphosate, we speculate for Japanese knotweed that the time when it is effectively transported into rhizomes and the time when it causes damage do not coincide. On the one hand, in autumn glyphosate is very effectively allocated to the rhizome, but fails to kill, because the plant is preparing for dormancy and there may be very low levels of protein synthesis in the rhizome (see above, "Sink-Source Relations between Above- and Below-ground Organs"). On the other hand, in summer, synthesis of proteins in the rhizome is maximal, due to buildup of its storage capacities, but the rate of glyphosate allocation to rhizomes in summer is insufficient to deliver a lethal dose (see above, "Sink-Source Relations between Above- and Below-ground Organs").

It has also been shown that glyphosate could be rendered less effective by the environment. For example, accumulation of roadway dust on the leaves of Japanese knotweed was shown to reduce the effectiveness of glyphosate by three times (Figueroa 1989); a high concentration of nitrogen in soil reduced the effect of glyphosate on quackgrass (Hunter et al. 1993); rainfall reduced glyphosate effect on tuberous purple nutsedge (*Cyperus rotundus* L.) and rhizomatous johnsongrass (*Sorghum halepense* Pers.) (Bariuan et al. 1999; Miller et al. 1998). Dilution via diversion of herbicide in planta can also occur. Glyphosate has both phloem and xylem mobility and so the presence of different sinks (e.g., a growing stem, leaf, or flower) could divert transport of the herbicide from the underground organs—as it was shown, for example, for sugar beet (Gougler and Geiger 1981)—away from the rhizomes. This would reduce the effectiveness of glyphosate because sublethal doses could be delivered in planta. Therefore, glyphosate may not be the most effective herbicide to control Japanese knotweed, and might be better used in combination with other herbicides and regulators of sink-source relations (see below).

Imazapyr. Imazapyr belongs to the group of imidazolinones. Imidazolinones inhibit the activity of acetolactate synthase, also known as acetohydroxyacid synthase, which is involved in the synthesis of branched-chain amino acids (Shaner et al. 1984). A reduction in concentration of branched-chain amino acids reduces protein synthesis, eventually killing the plant. But additionally and crucially, imidazolinones somehow disrupt phloem transport: for example, imazamethabenz applied to the foliage of wild oat (*Avena fatua* L.) induced excessive accumulation of starch in chloroplasts and at the same time, the starch level in the main stem was reduced (i.e., phloem transport of assimilates was disrupted, and they were retained in leaves; Chao et al. 1994).

In a screening trial of different herbicides, imazapyr was found to be more effective than glyphosate in the control of Japanese knotweed (Figueroa 1989): 1 yr after application, the density of stems per square meter was reduced by 97% in the case of imazapyr, and for glyphosate, by just 56%. Figueroa (1989) also showed that the efficacy of imazapyr was less influenced by environmental conditions than was the efficacy of glyphosate (Figueroa 1989). However, imazapyr still can be diluted via diversion in planta in the same way as glyphosate. Imidazolinones have phloem and xylem mobility (transport of imazethapyr in both acropetal and basipetal directions has been demonstrated for common ragweed (*Ambrosia artemisifolia* L.) and giant ragweed (*A. trifida* L.); Ballard et al. 1995) and so the presence of another sink (e.g., a growing stem, leaf, or flower) can divert their transport from a rhizome to another organ.

Imazapyr and glyphosate were recently compared in pilot projects by Washington State Department of Agriculture. It was estimated that over 90% control could be achieved after just one application of imazapyr, and there was an improved control with foliar-applied Habitat^{R} (= imazapyr), compared to foliar-applied mixtures of Habitat^{R} + AquaMaster (= imazapyr + glyphosate), or injections of AquaMaster (= glyphosate). Thus a decision has been made to apply imazapyr and imazapyr–glyphosate mixtures, but not glyphosate alone (Miller 2005).

Thus, although the mode of imazapyr action resembles that of glyphosate, it is more effective, possibly because of (1) its ability to disrupt phloem transport and (2) its low rate of decomposition (persistency). We can speculate that in summer, when active development of the rhizome storage capacity occurs, imazapyr acts as an inhibitor of protein synthesis. In autumn it also acts as a disruptor of phloem transport and stops allocation of sugars to the rhizome. In the spring, following summer–autumn treatment, nondecomposed imazapyr poisons the growing buds.

Synthetic Auxins. Synthetic auxins are substances that mimic the activity of natural plant phytohormones—auxins. Five synthetic auxins have shown varied effectiveness in control of Japanese knotweed (Child and Wade 2000; Figueroa 1989; Remaley 1997). They are known under the common names of 2,4-D, dicamba, picloram, triclopyr, and clopyralid, but actually originate from two different classes of chemicals: 2,4-D and dicamba are substituted benzols, and picloram, triclopyr, and clopyralid are pyridines. Persistence of synthetic auxins differs between chemicals: 2,4-D amine is nonpersistent and approved for use near water (Child and Wade 2000); others are persistent, with various lifetimes in the soil.

The mode of action of synthetic auxins is believed to be a general disruption of the natural hormone balance in plants. Synthetic auxins cause cell elongation by acidification of the cell wall and increase of RNA, DNA, and protein synthesis, leading to disorganized growth of cells. Tissue proliferation results in epinasty, stem swelling, and disruption of the phloem, which prevents movement of photosynthate from the leaves to the root system (Cudney 1996).

The effectiveness of synthetic auxins to control Japanese knotweed varies substantially between chemicals and trials. In general, synthetic auxins were often more effective in low than high doses. For example, triclopyr at 5% concentration appeared to give good top-kill, but did not destroy the rhizome. However when used at a concentration of 0.75% applied in the same spray volume, triclopyr was found to be successful in the eradication of Japanese knotweed (Soll 2004). The suggested explanation was that high concentrations damage aboveground tissues before the herbicide is translocated to the rhizome. Comparison of different synthetic auxins showed that substituted benzols (2,4-D and dicamba) had the lowest efficacy on Japanese knotweed (Figueroa 1989), but pyridines (triclopyr, picloram, and clopyralid) were reported to be highly effective (Child and Wade 2000; Remaley 1997), although proper comparative trials need to be performed to confirm differences in effectiveness.

The efficacy of synthetic auxins was less affected by environmental conditions than that of glyphosate: clopyralid passed rapidly into leaves and roots of plants and was "rain-fast" within 2 h (Kloppenburg and Hall 1990). But, synthetic auxins can be diverted in planta in the same way as glyphosate and imazapyr: they have phloem and xylem mobility, so the presence of any other sink could divert their allocation to the rhizome.

Another constraint to the use of synthetic auxins is that at certain times of the year they can promote development of storage organs, but not destroy them. It was shown that application of exogenous auxins increased cell division, cell enlargement, import of sucrose, and biosynthesis of starch at initial stage of potato tuber formation (Borzenkova et al. 1998). It is possible that the application of synthetic auxins to Japanese knotweed in spring and summer would induce enlargement of the rhizome, rather than its diminution. Therefore synthetic auxins (as disruptors of phloem flow) should be applied in the autumn only, when the intensive allocation of assimilates to the rhizome occurs and the disruption of phloem transport may stop supply of carbon to the rhizome.

Summary of Chemical Control Practices. Summarizing what is known of chemical control of Japanese knotweed, we can conclude that currently adopted practices often fail to eradicate the plant for reasons that include the following: application of less-effective herbicides; application of herbicides at an inappropriate stage of plant development; untimely damage to shoot apices; and diversion of herbicide in planta and in the environment. Other challenges, which have not yet been addressed, are to kill the old rhizome and the vast bank of quiescent rhizome buds, as almost nothing is known about their physiology, making it impossible to develop an appropriate right practice. We discuss several approaches below, which may increase the effectiveness of chemical eradication.

How to Increase Effectiveness of Chemical Eradication

"Wake-Up" Call for the Rhizome Buds. The ability to kill Japanese knotweed with a herbicide depends on its ability to translocate to the rhizome buds. However, most buds are dormant and thus are weak sinks. Therefore, an effective approach would be to provide a controllable "wake-up call" for the rhizome buds and to apply herbicides when they start to grow, i.e., when they become strong sinks.

There may be a range of treatments that could be used to break the dormancy of buds on underground organs. A possible approach is a controlled clipping of shoot apices to overcome apical dominance and thus induce growth of rhizome buds. It has been shown that while shoot clumps remained alive and produced new shoots, rhizome branching was suppressed due to apical dominance (Adachi et al. 1996a,b), but that when aerial shoots were cut or killed by a herbicide, lateral spread of clumps increased (Beerling 1990).

Another possibility is the application of low amounts of ethanol, which has been shown to result in growth of potato tuber buds (Claassens et al. 2005). Thus it might be useful to try ethanol injections into stems before or with application of a herbicide.

A further possible adjunct to herbicide treatment is the ethylene precursor ethephon, as it was shown to release the apical dormancy in quackgrass and stimulated growth of the lateral and basal buds on the rhizome (Chancelor 1970). Ethephon increased the accumulation of ^{14}C-labelled herbicide, fluazifop-butyl, in basal buds and proximal parts of the rhizome system; subsequently inhibition of the quackgrass growth by this herbicide was enhanced (Lawrie and Clay 1993). Other studies also showed that ethephon could initiate buds on a rhizome without growth of rhizome parenchyma (Fritz et al. 1991; Rylski et al. 1974). Recently, the effects of ethephon used in conjunction with kinetin (cytokinin) on herbicide transport were investigated for bracken [*Pteridium esculentum* (G. Forst.) Nakei] (Loresco et al. 2004). It was shown that application of both phytohormones enhanced translocation towards the rhizome of ^{14}C-desoxyglucose, which was used as a tracer of herbicide movement (Loresco et al. 2004). The authors speculated that kinetin and ethephon increased sink strength of the rhizome by induction of the rhizome buds, because parenchymal growth was not observed (the weight of rhizomes of treated plants did not differ from the untreated; Loresco et al. 2004). Other phytohormones as well as sugars and light might also induce the rhizome buds in Japanese knotweed, as they have been shown to change sink-source relations between below- and aboveground organs in other species (see below).

Use of Regulators of the Sink-Source Relations to Increase Import of Phloem-Mobile Herbicides into Rhizomes. It has been shown that translocation and allocation of substances into underground storage organs can be changed by application of phytohormones, sugars, and light. This approach might be used to manipulate sink forces in Japanese knotweed and hence to increase herbicide allocation to the rhizome. However, signals involved in switching to successive stages of morphogenesis have not been investigated for Japanese knotweed. At present, only speculations about possible involvement of phytohormones, photoreceptors, and sugars can be made, based on knowledge acquired from other plants.

Phytohormones. Elevated levels of endogenous cytokinin led to the formation of swollen starch-containing internodes in tobacco, so were able to induce tuberization even in a plant species that does not normally form tubers (Guivarc'h et al. 2002). Exogenous cytokinin and auxin also activated starch-synthesizing capacity in potato at the initial stage of tuber formation (Borzenkova et al. 1998). The authors hypothesized that exogenous cytokinins and auxins had increased the tuber sink strength even before it normally converts into a sink. We suggest that application of cytokinins and auxins (individually or in combination) in spring and summer may increase the sink strength of the Japanese knotweed rhizome much earlier than it normally converts into a sink. Such a shift should enable earlier spraying with herbicides.

Application of paclobutrazol (an inhibitor of gibberellin biosynthesis) promoted tuberization in potato (Simko 1994; Xu et al. 1998). Thus paclobutrazol may turn out to be useful as a false signal for inducing storage (similar to auxins and cytokinins).

Sugars. Changes in the concentration of sugars in plants depend on their rates of synthesis, transport (translocation), and storage (allocation). Translocation of sugars (for most species as sucrose) starts in leaves and is controlled by sucrose transporters: allocation to sink organs is controlled by several key enzymes, such as hexose transporters, invertases, and sucrose synthase (reviewed in Hellmann et al. 2000).

For underground organs, it was shown that application of exogenous sucrose to potato tubers induced tuberization and improved tuber weight. Sucrose promoted tuberization by increasing the levels of conjugated gibberellins at the expense of tuber-inhibiting free gibberellins (Simko 1994). Also, injection of exogenous sucrose into the top of shoots of sweet potato resulted in an increase of tuberous root production and in their weight. Moreover, the activity of adenosine 5′-diphosphate pyrophosphorylase (a key enzyme in starch biosynthesis and an important determinant of the sink activity of the sweet potato root) increased by 25% in plants that had been injected with sucrose (Tsubone et al. 2000). Thus, application of exogenous sucrose might change the sink strength of rhizome of Japanese knotweed and as such be used as a tool to increase herbicides allocation to the rhizome.

Light. Japanese knotweed grows best in full sunlight and the growth is reduced even in moderate shade. Analysis of stands along Welsh river banks showed that plants were shade intolerant when photon flux density was just 20% less than full daylight: at the end of August the biomass of shoots in shaded sites was three times lower than in full sunlight, despite a 100% increase in leaf area ratio (Beerling et al. 1994).

The effect of shading could be explained by important spectral difference between natural shading and full sunlight: in natural shade the ratio of red to far red light changes (Smith 1986). Indeed, light of different quality has been shown to affect morphogenesis in radish and change sink-source relations between the storage organ (soil-submerged hypocotyl) and aboveground shoots (Drozdova et al. 2001). It was found that development of the hypocotyl into a storage organ had already started on the 14th day if plants were grown under blue light but only on the 21st day if plants were grown under red light. Moreover, light quality changed the concentration of hormones in tissues: red light stimulated synthesis of gibberellins in petioles and stems, thus increasing their sink activity, whereas blue light increased the content of cytokinins and auxins in submerged hypocotyl and enhanced its development (Drozdova et al. 2001). Therefore, blue and far-red photoselective covers might increase sink strength of the rhizome of Japanese knotweed and so increase herbicide allocation.

Use of Xylem-Mobile Herbicides to Kill the Old Parts of Rhizome. As suggested above, the old parts of rhizomes of Japanese knotweed are alive and bear a bank of dormant buds, but they are very weak sinks because only roots are functional, supplying younger parts and shoots with water and nutrients. All herbicides used at present to control this species are phloem-mobile and thus very unlikely to be allocated to the old parts in lethal concentrations. Therefore, an alternative is to apply xylem-mobile herbicides, which are transported with water flow and able to poison the storage parenchyma.

There are xylem-mobile herbicides that damage chloroplasts and storage plastids: isoxaflutole (Pallett et al. 1998; Young and Hart 1998) and flurochloridone (Lay and Niland 1983). They inhibit phytoene desaturase (PDS) (isoxaflutole), which catalyses the initial steps in the biosynthesis of carotenoids, the desaturation of phytoene to lycopene, or they inhibit *p*-hydroxyphenylpyruvate dioxygenase (flurochloridone), which is essential in the biosynthesis of plastoquinone and which specifically regulates PDS (Boeger and Sandmann 1998). Application of such herbicides against nonphotosynthetic storage tissue resulted in a dramatic decrease in the number of stem amyloplasts, loss of amyloplast integrity, and loss of starch granules as shown for field dodder (*Cuscuta campestris* Yuncker) (Weinberg et al. 2003). The suggested explanation was that amyloplast membranes contain structural carotenoids and inhibition of their biosynthesis resulted in lesions and exposure of starch grains to hydrolytic enzymes in the cytoplasm (Weinberg et al. 2003). An additional advantage of these herbicides is that they do not damage shoot meristems (as shown for field dodder; Weinberg et al. 2003), and thus apical dominance will be preserved. The loss of plastid integrity should prevent Japanese knotweed from accumulating sufficient assimilates in the old rhizome, thus eventually exhausting and killing it. In sites near water, fluridone might be trailed to substitute flurochloridone and isoxaflutole, as fluridone has a similar mode of action (Popova 1996), is approved to use in the aquatic sites (McCowen et al. 1979), and in hydrilla was shown to inhibit tuber formation (MacDonald et al. 2008).

Integrated Program of Application of Various Herbicides at Different Growth Stages. As far as we can judge from present knowledge of modes of action of various herbicides, the best approach to eradicating this weed would be an integrated program that utilizes more than one herbicide during the growth season (see Figure 1). Possible schemes of application may involve the following:

Application of Contact Herbicide or Xylem-Mobile Herbicide in Spring to Early Summer. At this time of the year, resources stored in rhizomes are used for initial shoot growth and foliar expansion, so the rhizome functions as a source, rather than as a sink, and application of phloem-mobile herbicides is ineffective, as was recently demonstrated for glyphosate, dicamba, picloram, and clopyralid (Roadside Vegetation Management Research 2001). Top-kill would generate a need for the plant to repeat the production of leaves and bring about extra depletion of resources in rhizome. It is extremely important at this stage to select a herbicide that will not affect apical dominance, i.e., that will not harm shoot apical buds. Another possibility would be to apply a xylem-mobile herbicide to the soil before regrowth, so that it could not only kill the leaves, but also damage rhizome parenchyma.

Application of Imazapyr, Glyphosate, or Both from Midsummer Onward. At this stage the formation of new storage capacities in rhizome parenchyma starts, so protein synthesis is intensive and application of herbicides that interrupt this process is justified. The efficiency of such herbicides can be enhanced by use of regulators of sink-source relations and initiators of growth of lateral buds (see above).

Additional Application of Imazapyr or Synthetic Auxins in Autumn. At this stage withdrawal of carbon and nitrogen

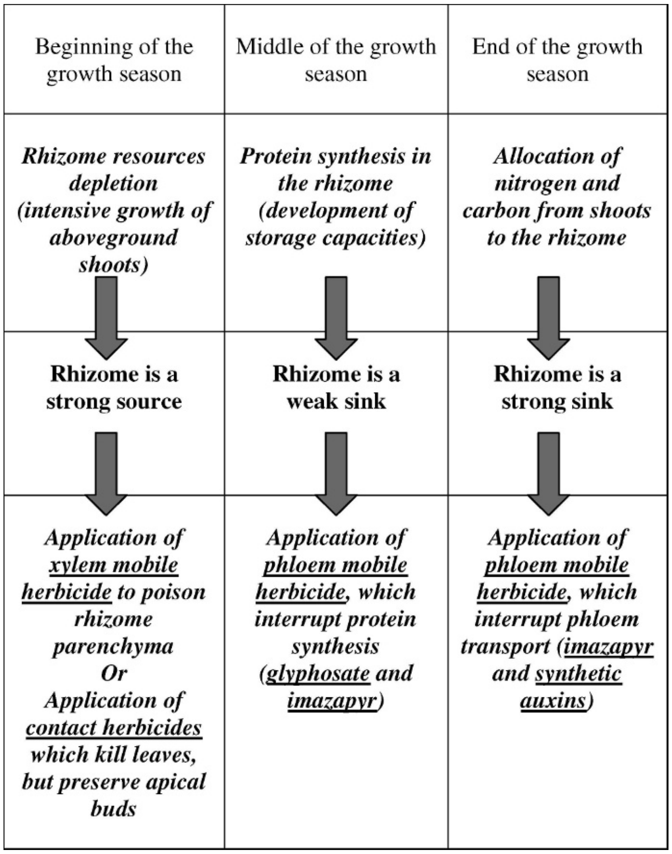

Figure 1. Diagram of an integrated program, which utilizes several herbicides to control Japanese knotweed. The program matches phenological events with timing and types of herbicides. Full explanations are given in the text.

from shoots into rhizomes increases dramatically before shoot senescence. Thus it is timely to spray with herbicides that inhibit phloem transport, such as synthetic auxins or imazapyr. Another advantage of these herbicides is that they are relatively persistent and might poison new growth the next spring. The efficiency of such herbicides can be enhanced by usage of regulators of sink-source relations (see above).

In this review we suggest prospective approaches to enable chemical eradication of Japanese knotweed. We propose the use of different signals to induce controlled growth of quiescent rhizome buds; the use of phytohormones, sugars, and light to increase allocation of phloem-mobile herbicides to the rhizome; the use of xylem-mobile herbicides to exterminate the old rhizome parts; and the use of various phloem-mobile herbicides at different growth stages to enable complete eradication of this weed. But knowledge of the physiology of Japanese knotweed is scanty and several important facts are missing from our understanding, thus making it difficult to be certain of the effectiveness of the practices we have suggested.

In order to rectify these omissions, it is very important, first, to identify stages of rhizome development when buds break dormancy and when rhizome branching occurs during the growth season. At present, it is difficult to judge when the best time might be for a herbicide to kill rhizome buds. Second, no research has been performed on the physiological state of buds on the old rhizome and very little is known about the old rhizome itself, thus restricting our ability to develop practices for eradication of large well-established patches. Third, it would be an advantage to map stages of development of underground organs (rhizome and buds) against stages of development of aboveground organs (shoots and flowers). At present, the development of underground organs are mapped against month of the year (as given in the review), but Japanese knotweed grows in several climatic zones at different latitudes, where the length of the growing season and the weather differ considerably; consequently, data from different countries are difficult to match. As shown in the review, because it is crucial to apply certain herbicides at particular growth stages, ontogenetic mapping is better than mapping against time of the year. Fourth, environmental and internal signals switching successive stages of morphogenesis have yet to be identified for Japanese knotweed. At present, we can only speculate on any involvement of phytohormones, photoreceptors, and sugars, but it is important to be certain of the triggers that enable shifts of the developmental stages.

Although lack of knowledge makes it difficult to revise completely the procedures for eradication of Japanese knotweed, the review has highlighted areas where new research is required in order that this goal be achieved.

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