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Heracleum mantegazzianum and *Toxicodendron succedaneum*: plants of human health significance in New Zealand and the National Pest Plant Accord

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Abstract

New Zealand's National Pest Plant Accord (NPPA) is a voluntary and cooperative agreement between industry, regional councils, and central government departments with biosecurity responsibilities (primarily the Ministry of Agriculture and Forestry and the Department of Conservation). Plant species included in the NPPA are declared unwanted organisms under the Biosecurity Act 1993, which prevents their sale, propagation, or distribution across the country.

Although MAF Biosecurity New Zealand (the lead agency in New Zealand's biosecurity system) has evaluated the potential human health impacts of 202 species considered for inclusion in the NPPA, two species were examined primarily due to their significance to human health: *Heracleum mantegazzianum* (giant hogweed, cow parsnip, wild parsnip) and *Toxicodendron succedaneum* (rhus tree, wax tree, Japanese wax tree). As a result of this process, *H. mantegazzianum* has been listed in the NPPA. In contrast, *T. succedaneum* was not included in the NPPA, as the latter was deemed to be an inappropriate mechanism for its control.

In this article the NPPA process is outlined, and the adverse impacts on human health of these two species are discussed—including symptoms, treatment, and possible management measures.

The National Pest Plant Accord

The National Pest Plant Accord (NPPA) is a voluntary and cooperative agreement between the Nursery and Garden Industry Association, regional councils, and government departments with biosecurity responsibilities (primarily the Ministry of Agriculture and Forestry [MAF] and the Department of Conservation [DOC]).

The NPPA seems to be the only agreement of its kind in existence (M Newfield, personal communication, 2006). All plant species listed under the NPPA are automatically declared unwanted organisms under the Biosecurity Act 1993, pursuant to section 2(1). This prevents their legal sale, propagation, or distribution within New Zealand.

When listing plants emphasis is given to species that are invasive, pose the greatest level of threat, are primarily spread by people, and have limited actual distribution relative to their potential distribution.[39]

The NPPA came into effect for the first time on 1 October 2001. It was revised in 2006, with signatory parties renewing their commitment to the Accord for another five years. Although the Accord is not a binding contract, it is intended to carry the same effect as a Memorandum of Understanding between the signatory parties. As a

result, these parties share responsibility to promote compliance with the rules of the Accord, and regional councils have specific responsibility for actively monitoring compliance (A Harrison, personal communication, 2006).

There were 202 species initially considered for inclusion in the NPPA during the 2006 revision of the Accord. The final approved list includes 109 individual plants species and all the species present in four genera.[40]

Following the creation of Biosecurity New Zealand within MAF in 2004, the Ministry has acquired greater biosecurity accountabilities including an oversight role for the biosecurity system and explicit accountability to protect the full range of societal values through its activities. As a result, MAF aims to ensure that human health values are adequately considered within its routine activities. Therefore, under this new role, apart from the potential impact on the environment and the economy, MAF Biosecurity New Zealand (MAFBNZ) was also accountable for evaluating the potential human health significance of all plants being assessed for inclusion in the NPPA.

Several introduced plants species in New Zealand can cause adverse reactions in humans via skin contact—e.g. *Urtica dioica* (perennial nettle) and *Ficus carica* (fig tree)—with a number of other species being poisonous if plant parts are ingested. However, two plant species in particular were evaluated for the NPPA as a result of their public health significance and the possible need to officially control them: *Heracleum mantegazzianum* Sommier et Levier (Apiaceae) (giant hogweed, cow parsnip, wild parsnip) and *Toxicodendron succedaneum* (L.) Kuntze (Anacardiaceae) (rhus tree, wax tree, Japanese wax tree).

Both species are introduced (i.e. not native) to New Zealand, and their adverse effects to human health have been widely documented. They are, for example, included in the list of plants in New Zealand that are poisonous to children compiled by Landcare Research.[37]

In this article, the relevance of these two species to human health are discussed, including symptoms and treatment, as well as the final decisions of the NPPA process and some possible management measures.

Heracleum mantegazzianum

Background—*Heracleum mantegazzianum* is a giant herb that varies in height from 2.0 to 5.0 m, and which may live for several years.[1] It is a popular plant in gardens due to its attractiveness (Figure 1).[2,3] *Heracleum mantegazzianum* is native to Asia, more specifically the western Caucasus but it is now widespread in Europe[1] and North America.[4]

In the United States this species escaped from cultivation and has become a public health hazard—being found in urban, suburban, and rural settings.[5] As a result, *H. mantegazzianum* is on the United States federal noxious weed list, which means that its importation into the country is illegal, as is the interstate and intrastate movement of this species.[6]

While *H. mantegazzianum* appears to be present in Dannevirke, Napier, Wellington City and in a few scattered localities in Marlborough, Canterbury, Otago, and Southland,[7] its actual current distribution in New Zealand is unknown.

Figure 1. *Heracleum mantegazzianum* with its large leaves and flowerheads (Photo courtesy of Rune Aanderaa, SABIMA)

Mode of action—The sap of *H. mantegazzianum* contains psoralens (furocoumarins) that lead to a relatively common type of dermatitis: phytophotodermatitis, which is produced by the interaction of such plant compounds with sunlight on human skin.[8–11] The psoralens are lipid-soluble and penetrate into the epidermis with ease.[12] The photochemical excitation of psoralens is induced by ultraviolet (UV) radiation, usually within the UVA wavelengths of 320–400 nm.[13] Note that the absorption of psoralens into the skin (and the consequent reaction) is enhanced by high humidity.[14]

A detailed study of the mechanisms of phytophotodermatitis has been provided by Pathak (1986).[14] In brief, two types of toxic reactions occur: one oxygen-independent where the ultraviolet-activated psoralens bind to RNA and nuclear DNA, and an oxygen-dependent reaction where the induced compounds cause cell membrane damage and oedema.[11,38]

These reactions consequently lead to cell death.[3,11,15,38] This is a phototoxic reaction, and not an allergic one so there is no immunological response. As a result, no prior sensitisation is necessary and anybody can be affected.[16] *Heracleum mantegazzianum* is one of the main causes of phytophotodermatitis in the United Kingdom and United States.[4]

The plant's clear watery sap is said to exude from all parts of the plant, so dermatitis is induced via contact with leaves, stem, seeds, and roots.[4] An analysis of

furocoumarin contents indicated that their concentration in *H. mantegazzianum* plants was highest in fruit, intermediate in leaves, and low in stems.[15]

Nonetheless, touching the plant or brushing against it appears to be enough to induce exposure to the sap, and all persons that come in contact with it seem to be affected to some extent. It should be noted that the content of furocoumarins in the sap of *H. mantegazzianum* varies with individual plants, season (being highest in spring), and probably also soil conditions and climate.[2,10,15]

Clinical features—The consequent symptoms of the induced phytophotodermatitis appear usually to have a benign character, but they can lead to severe blistering and painful burn-like lesions.[5,15] The diagnosis based on the symptoms displayed by the patient is difficult, and adequate diagnosis is clinically based on history and physical examination.[13,17] In some cases, the symptoms of phytophotodermatitis have been mistaken for child abuse.[4]

In addition, as a result of the aggressive progression of the symptoms, phytophotodermatitis induced by *H. mantegazzianum* is commonly mistaken for resistant staphylococcal infections or necrotising fasciitis.[5]

Contact with the plant sap and exposure to UV light leads to erythema, oedema, and burn-like lesions within 24 hours and possibly large, fluid-filled blisters within 48 hours.[3,8,10] The occurrence of pruritus is uncommon,[18] but secondary skin infection is a possible complication.[5]

The blisters can develop into purplish or blackened scars, with skin hyperpigmentation remaining visible for months or even years after exposure.[3,4,8,10,16]

In addition, the affected areas may remain hypersensitive to UV light for many years.[4,9,16] The occurrence of systemic manifestations is rare.[13] Children appear to be particularly attracted to playing with *H. mantegazzianum*'s large and hollow stems, which are used for play swords and telescopes.[5]

It is important to highlight that phytophotodermatitis may occur via indirect contact with *H. mantegazzianum* sap. For example, a woman developed phytophotodermatitis as a result of contact with her cat that had been playing with a specimen of the plant in the garden.[8] Dogs are also described as being frequent carriers of the plant's sap on their fur, which is then transferred to the owners' skin.[5] It seems that mammals other than humans may also be affected by phytophotodermatitis induced by this plant.[19]

Treatment—Immediately after exposure, the skin should be thoroughly washed with soap and cold water to remove plant sap.[5,9] The exposed skin should be protected from sunlight by covering and/or the application of sunscreen, until at least 48 hours post-exposure even if asymptomatic.[3] If sap enters the person's eyes, these should be thoroughly flushed with cold water or irrigation solution.[5] Although there is suggestion that exposure to furocoumarins can cause permanent blindness,[5] there appear to be no cases documented in the medical literature.

Since most people are unlikely to seek medical attention prior to the onset of symptoms, management is usually symptomatic and supportive.[3,5] Wet compresses, ice packs, and paraffin gauze dressings my assist to reduce swelling and inflammation.[3,5]

An effective treatment may consist of wound debridement and daily dressings with silver sulphadiazine, which seems to be effective and safe.[15] In any case, keeping blistered areas clean with the use of topical antiseptics is advisable to prevent the onset of secondary infection.[13]

In some cases, treatment with an oral or topical anti-inflammatory medication is advised,[5,15] with severe cases possibly requiring hospitalisation for analgesia and supportive care.[3,5] Where intense pruritus occurs, antihistamines may be used.[13]

The healing process can take up to 2 weeks,[15] but in some cases symptoms may last for over a month.[5] Moreover, it is necessary to monitor the patient for secondary infection, and educate the person to avoid future exposure.[18]

Although the subsequent hyperpigmentation requires no treatment, hydroquinones may be used.[18] Since affected areas may remain hypersensitive to sunlight for months or years, the continued use of sunscreen is advisable.[3,18]

***Heracleum mantegazzianum* in New Zealand and NPPA recommendations—** Unfortunately there is currently no information on the number of cases of phytophotodermatitis induced by *H. mantegazzianum* in New Zealand.

However, unlike other plants in New Zealand that cause adverse reactions in humans, *H. mantegazzianum* stands out as a particular threat for several reasons, including:

- Potentially severe symptoms caused by phytophotodermatitis;
- Its growth habit (giant herb) making it more likely for people to come into contact with the plant (compared with a tree or a low-growing herb);
- The fact that in other temperate countries it is a serious weed that invades high use areas likely to be used by people—such as river and stream banks, roadsides, and right-of-ways.

Although the distribution of *H. mantegazzianum* is currently limited in New Zealand, this species has the potential to become widespread along river and stream banks, especially in areas fenced off from grazing stock.

In addition, the use of *H. mantegazzianum* as a garden plant has been promoted in the past, even though current trade in this plant is very limited. Therefore, due to its significance to human health and its potential to become an invasive species in New Zealand, *Heracleum mantegazzianum* has been included in the list of plants covered by the Accord.

Toxicodendron succedaneum

Toxicodendron succedaneum is a relatively small deciduous tree, native to Eastern Asia, that grows to approximately 12 m.[20,21] It has attractive autumn foliage, which makes it sought after as an ornamental tree (Figures 2 and 3). However, *T. succedaneum* is as allergenic as poison ivy (*T. radicans*), although it seems to be less of a clinical problem than the latter since it grows as a tree rather than a creeper (M Rademaker, personal communication, 2005). Plants in the family Anacardiaceae are the main cause of allergic contact dermatitis (ACD) induced by plants worldwide,[16,22] with *Toxicodendron* spp. being by the far the most common cause.[23]

Figure 2. Canopy of *Toxicodendron succedaneum* showing the characteristic bright reddish colours of its deciduous autumn leaves (Photo courtesy of DermNet)

Figure 3. A sapling of *Toxicodendron succedaneum* showing green leaves (Photo courtesy of Auckland Museum)

In the United States, ACD caused by *Toxicodendron* spp. is a significant occupational hazard for agriculture and forestry workers as well as recreational wilderness users.[24] There are substantial associated medical costs, and major economic losses as a result of the consequent morbidity in particular amongst forestry workers.[24]

Mode of action—The sap of *Toxicodendron* spp. contains urushiols that are extremely potent sensitisers, in particular the allergen catechols.[16,21,25,26] Urushiol is described as colourless or slightly yellow, but once exposed to air it oxidizes and polymerizes turning black.[24]

It is estimated that at least 50% (but possibly as much as 75%) of the adult population in the United States is hypersensitive to the urushiol of *Toxicodendron* spp. and would likely develop clinical symptoms.[12,23,24,27,28] Note that the expression of ACD is partially dependent on genetic factors.[12] ACD is a cell-mediated response to exposure to an antigen of a relatively low molecular weight that can penetrate the epidermis.[23] *Toxicodendron* induced dermatitis is more intense in adults, although many severe cases can be observed in children.[12]

The antigens of all *Toxicodendron* spp. are essentially the same, so cross-sensitivity between different species occurs.[26] Urushiol is found in all parts of the *Toxicodendron* plant including stems, leaves, roots, and fruit skin.[24] ACD is induced following exposure to a damaged part of the plant,[24,25] and the latter is necessary to allow the urushiols (oleoresins) to contact the skin, as the uninjured plant is innocuous.[24,26] In addition, allergic contact dermatitis can occur following inhalation of the smoke of burning plants, as the urushiols may be present in the particulate matter.[21,26,29]

Inhalation of these particles may therefore result in an allergic response affecting mucous membranes for example, and ACD may also occur on skin where particles may settle. In some cases contact with urushiols may occur via wind transmission.[21]

Urushiol is non-volatile and dries quickly on fomites—persisting on clothes and equipment indefinitely.[24] It seems that pets, particularly long-haired dogs, may be responsible for transmitting the oleoresin from *Toxicodendron* plants to children.[12] Aggravating the threat posed by *Toxicodendron* spp. is the fact that dermatitis can occur following contact with dead plant tissue, as urushiol retains its antigenic potential in the dry state indefinitely.[26]

Note that most of the literature available on *Toxicodendron* refers to North American species, mainly *T. radicans* (poison ivy), *T. vernix* (poison sumac), and *T. diversilobum* (poison oak). Nonetheless, since, as previously mentioned, the antigens of all *Toxicodendron* spp. are essentially the same,[26] symptoms of exposure, treatment, and management are applicable to all species.

Clinical features—Although most contact allergens require repeated exposures to trigger an immune response, the catechols of *Toxicodendron* spp. are potent, and susceptible people may become sensitised after only two exposures.[23] After a sensitised person comes into contact with urushiol, the symptoms usually appear within 2 days.[12] However, symptoms may appear as early as within 6 hours, and may be delayed for as many as 12 days after contact with the plant's urushiol.[30]

Erythematous papular lesions that itch intensely are usually the initial symptoms,[23] and these may be associated with an intense burning sensation, and often advance to

raised lesions.[30] Pruritus is intense in all stages of the lesions and is characteristic of *Toxicodendron* ACD.[23] The severe itching may lead to scratching with excoriation and secondary lesions, possibly leading to infection.

Approximately 48 to 72 hours after exposure, vesicular lesions develop and erupt, releasing plasma that forms a crust.[23] Vesicles are often numerous and small but bullae can occur in severe reactions.[23] Facial oedema with marked periorbital swelling are particularly common in children.[21]

In moderately severe cases, oedematous swelling of various parts of the body may occur, while in severe cases *Toxicodendron* ACD is characterised by widespread symptoms and marked oedema of the extremeties and face.[26]

The fluid that exudes from vesicles and bullae do not contain the allergen and therefore the patient cannot spread the dermatitis to other persons or other parts of the body.[24,26,27,31] However, the rash may grow in size and new vesicles may develop during the first 2 weeks without further exposure to urushiol, which leads to the common belief that the serum from the vesicles contains the antigen.[23]

The severity of the response will also vary between individuals, some experiencing mild reddening while other patients may become temporarily disabled.[31] The extent and severity of the lesions will vary due to a number of factors, especially level of exposure (area of contact and amount of urushiol involved), patient's sensitivity to the allergen, site of contact, and skin thickness.[23,30,31] In addition, lesions may develop in areas not directly exposed to the plants due to secondary exposure (via contaminated hands, clothing, tools, etc.), and also due to the non-specific effect of the cell-mediated response.[23]

The ACD induced by *Toxicodendron* spp. usually resolves within 3 to 4 weeks,[23] but it may last for 6 weeks in more susceptible individuals.[24] Hyperpigmentation may occur in darkly pigmented individuals, which may last for months.[23,24] Although complications and systemic effects may occur, these appear to be uncommon.[24] Secondary infection, however, appears to be more common.

Ingestion of *Toxicodendron* plant material leads to symptoms that occur mostly within 1 day.[32] Chewing or ingestion of the leaves is likely to result in inflammation of the oral mucous membranes, and may cause severe gastroenteritis—with nausea, vomiting, diarrhoea, abdominal pain, and proctitis.[12] Other systemic symptoms may include fever, chills, headache, and fatigue—and, in very serious cases, hypotensive shock may occur.[32]

Ingestion of *Toxicodendron* plant material can also lead to systemic contact dermatitis, with symptoms as those resulting from direct skin contact.[32]

Treatment or management—Following contact with the sap of *Toxicodendron* plants, it is important to wash the affected area immediately with warm soapy water.[25] All clothing, tools, or other objects or pets that have been exposed to the urushiol should be adequately washed with common soap or detergent, which renders the urushiol-contaminated areas or fomites harmless.[26]

Urishiol may remain under a person's fingernails, which must be washed to prevent further self-exposure or contamination of other individuals.[26] Note, however, that the urushiol binds to skin proteins within a few minutes after exposure, and thorough washing would only remove the remaining oleoresin yet to bind.[26,30]

After approximately 30 minutes post-exposure, all urushiol is likely to have been absorbed into the skin.[24] However, there are some specific detergents that seem to optimise the removal of urushiol from human skin, which may be applied a couple of hours after exposure.[23,24,30]

Eruptions may be treated with topical corticosteroids, although severe cases may require hospitalisation and systemic administration.[25,28] In New Zealand, three-quarters of the patients confirmed to be affected by *Toxicodendron* dermatitis were treated with a reducing dose of prednisone, and the remaining with potent topical corticosteroids and systemic antihistamines.[33]

Note that such corticosteroids may alleviate but not prevent the development of symptoms.[30] Detailed discussions on the treatment of *Toxicodendron* ACD have been provided by other authors.[12,23,30]

***Toxicodendron succedaneum* in New Zealand and NPPA recommendations—** *Toxicodendron succedaneum* is without doubt the most allergenic plant species in New Zealand causing contact dermatitis, and one that certainly causes public harm (M Rademaker, Waikato Hospital, personal communication, 2006). In 1993 alone there were at least 20 cases of allergic contact dermatitis due to *T. succedaneum* recorded in the Waikato Hospital.[34] There were at least 92 cases of contact dermatitis due to *T. succedaneum* in the Waikato region between 1982 and 1994.[33]

At least 55 cases involved young people (0–20 years) who were affected during outside play, most of which involved lesions to the face.[21] In contrast, almost all cases involving those aged 21 or older occurred while gardening.[21]

Toxicodendron succedaneum is not yet officially controlled in New Zealand, but it is classified as a noxious weed in the Australian states of South Australia[35] and New South Wales,[36] where all specimens of this plant must be destroyed.

In Australia, *T. succedaneum* was sold for many years as a garden plant, but since its declaration as a noxious weed it can no longer be offered for sale;[36] a similar situation to Japan.[33,34] In addition, in New South Wales for instance, public education has assisted in leading to a considerable reduction in the number of trees.[36]

While the potential environmental impact of *T. succedaneum* in New Zealand is uncertain, there seems to be no naturalised population of this plant. However, in Sydney (Australia) *T. succedaneum* is considered to be a serious weed problem where birds spread the seeds in their droppings—and many thousands of seedlings were flourishing in home gardens, in public areas, and in urban bushland.[36] *T. succedaneum* can also be spread by movement of garden soil containing seed, which remains viable for many years.[36]

The Steering Group overseeing the NPPA process decided that there was no justification for including *T. succedaneum* in the NPPA list. According to the horticultural industry, "this plant is not a species that is currently being sold in New Zealand."

The NPPA's Technical Advisory Group also concluded that there is no evidence that this species is invasive in New Zealand or is spread by humans. Therefore, although *T. succedaneum* warrants some management due to its potentially serious effects on human health, it did not meet the criteria for inclusion in the NPPA, and this was

consequently deemed not to be the appropriate mechanism to address the risks to human health associated with this plant.

Hazard management and conclusion

As a result of the inclusion of *H. mantegazzianum* in the National Pest Plant Accord, this plant is now an unwanted organism. As a result, its sale, propagation, and distribution across the country are illegal. There is however, no requirement for existing plants to be destroyed.

Due to *H. mantegazzianum*'s threat to public health and its potential invasiveness, MAFBNZ encourages the general public, regional, and local authorities to destroy this plant. Nonetheless, extreme care should be exercised when removing these plants, and it must be stressed that contact with dead plant parts and with inanimate objects or pets that have been in contact with such plants is dangerous.

The use of protective water-resistant clothing and protective goggles is advisable when dealing with *H. mantegazzianum*, as is the simultaneous avoidance of exposure to sunlight.[1,15]

A detailed management plan for *H. mantegazzianum* was produced by Nielsen et al. (2005) who assessed various control methods.[1] The authors stated that: "currently used control methods comprise a variety of manual and mechanical methods, grazing and herbicide application", and that "rather than recommending a single control method, a control programme based on an integrated weed management strategy (IWMS) is preferred" (p30).

In regards to *T. succedaneum*, since it has not been listed in the NPPA, no official measures have been imposed on its sale or propagation in New Zealand. However, MAFBNZ encourages local and regional authorities to consider taking action against this species in the interest of public safety.

Specifically, MAFBNZ recommends that councils promote or carry out active removal of *T. succedaneum* (and also *H. mantegazzianum*) from public places—including schools, parks, reserves, and other high public use areas. It seems that the Hamilton City Council, for example, no longer plants *T. succedaneum* and has removed many such trees from public places or other areas on medical request.[34]

Regarding the removal of *T. succedaneum*, like *H. mantegazzianum*, dead plant parts or anything that has been in contact with the plant poses a risk, as ACD can be developed by contacting tools, pets, or clothing that have been in direct contact with the urushiol previously.

The removal of *Toxicodendron* plants consequently has to be done with care, and as much of the skin area as possible should be adequately covered. It seems that it is necessary to use heavy-duty vinyl gloves, as rubber gloves are not very protective as the catechols in urushiol can penetrate most, if not all, types.[12] The plants removed should be buried or burnt.[26] However, as previously pointed out, burning *T. succedaneum* may also lead to exposure, and any person in the vicinity should maintain a safe distance to avoid exposure to urushiol that may be carried in the ashes or smoke.

Finally, medical practitioners that come across cases of dermatitis as a result of contact with these plant species in private properties should recommend the removal

of the specimen(s). Moreover, when the particular plant is located on public land, the medical practitioner should inform the local authorities, as consideration should be given on whether the plant needs to be removed in the interest of public health.

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