Host Specificity of Plant Pathogens in Biological Weed Control

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Abstract

Host specificity is a major concern in biological weed control programs. Exhaustive studies are conducted to ensure that the biocontrol agent, if released, will not damage desirable plants. The value of some tests used to determine the host range of candidate agents has been questioned and perhaps many potentially valuable biocontrol agents have not been released due to aberrant feeding on non-host plants in those tests. Host range studies with plant pathogens in controlled environment conditions have often resulted in broader host ranges than reported or previously known. The host ranges of Puccinia species being considered for introduction into North America have been artificially expanded within controlled environment containment facilities. Perhaps these expanded host ranges should have been expected, however, additional tests must be developed to evaluate actual or field specificity. Concerns for host specificity are not limited to classical biocontrol candidates since facultative saprophytes are being evaluated as biological herbicides. These pathogens tend to have broader host ranges than obligate parasites, but variation in laboratory and field specificity are also to be expected. Strict host specialization is not a requirement for biological herbicides.

La Spécificité des Phytophatogènes en Lutte Biologique Contre les Plantes Nuisibles

La spécificité des organismes utilisés en lutte biologique est une préoccupation constante chez les chercheurs. Ils doivent mener des études approfondies afin de s’assurer que l’organisme, une fois libéré, n’endommagera que les plantes cibles. La valeur de certains tests utilisés pour déterminer les hôtes potentiels d’agents de lutte biologique est discutable; il se peut que dans certains cas, des agents de lutte prometteurs n’ont pas été libérés dû au fait qu’ils démontraient des comportements anormaux d’alimentation sur certains hôtes reconnus comme non-susceptibles à leurs attaques. Des tests de spécificité d’hôtes avec des phytopathogènes, menés en laboratoire sous conditions atmosphériques contrôlées, ont révélé un nombre plus élevé d’hôtes potentiels que ce qu’avait été décrit ou était connu jusqu’à présent. La liste d’hôtes potentiels de certaines espèces de Puccinia, présentement évaluées pour leur introduction en Amérique du Nord, a ainsi été élargie de façon artificielle du fait que ces tests ont été menés sous les conditions closes d’un phytootron. Quoique ces résultats auraient dû être prévisibles, il n’en demeure que des tests additionnels devront être développés afin d’évaluer la spécificité du phytopathogène telle qu’elle se manifeste en milieu naturel. Ces inquiétudes vis-à-vis la spécificité ne sont pas limitées aux candidats de l’approche classique de lutte biologique car certains saprophytes facultatifs sont évalués comme herbicide biologique. Ces phytopathogènes tendent à avoir une gamme d’hôtes plus considérable que dans le cas des parasites obligatoires. Neanmoins il faut prévoir des variations au niveau de leur spécificité tant en laboratoire qu’aux champs. Les herbicides biologiques ne requièrent pas une spécificité stricte du phytopathogène.

Introduction

Biological weed control is the deliberate use of natural enemies to reduce the population of a target weed. The classical and the inundative methods are the two main approaches, with classical biological control directed towards controlling naturalized weeds through the introduction of exotic natural enemies from the weed’s
native range and inundative biological control directed towards mass production and release of endemic natural enemies. Plant pathogens are being used in both classical and inundative biological weed control programs, and as suggested by many authors (Hasan 1983; Leonard 1982; Quimby and Walker 1982; Schroeder 1983; Templeton and Smith 1977; Wapshere 1982, 1983), host plant specificity is the most important factor in the selection of candidate pathogens as biocontrol agents.

**Specificity in Plant Pathogens**

The host range of plant pathogens may be narrow or broad, but most plant pathogens, whether facultative saprophytes or obligate parasites, generally have host ranges limited to one or a few species (Gäumann 1950; Holcomb 1982; Johnson 1976; Oku et al. 1979). Brian (1976) described the levels of specificity of fungal pathogens from non-pathogens to formae speciales and concluded that most fungal pathogens show a high degree of host specificity. However, little is known of the chemical basis of such specificity in pathogens (Oku et al. 1979).

Considerable variation occurs in the reported specificity of some pathogens (Brian 1976; Cother 1975; Eshed and Dinoor 1981). Often the procedures used to determine the host range and/or the taxonomy of the pathogen involved have been suggested as reasons for these discrepancies in the literature (Brian 1976; Eshed and Dinoor 1981). Another factor which compounds the difficulties in delimiting host ranges is the confusion concerning many terms used by various authors to discuss plant pathogen specificity. Merrill (1980) describes the major problem of concepts and terminology of such terms as resistant, susceptible, and immune. These terms, and others, have different usage from one report to another and consequently results of host specificity determinations can have many interpretations. A portion of Merrill's (1980) glossary is presented in Table 1 and it is suggested that this terminology be the basis of host specificity determination in biological weed control using plant pathogens.

As described earlier, specificity is a major concern in biological weed control and it is the responsibility of the biological control researcher to determine the host range of the candidate biocontrol agent. In plant pathology, disease or the host-parasite interaction is the product of the interaction between pathogen and a host in a given environment. These three factors (the pathogen, the host and the environment) must be considered in the development of appropriate or realistic host specificity testing procedures.

**Host Specificity Testing**

Because of the importance of safety in biological weed control programs, much has been written on host specificity screening and the limitations of these tests (Dunn 1978; Harris and Zwölfer 1968; Schroeder 1983; Wapshere 1974, 1982, 1983; Zwölfer and Harris 1971). Presently, most host specificity testing follows the centrifugal phylogenetic method proposed by Wapshere (1974) which delimits the extent of the biocontrol agent's host range and includes the testing of other plant species 'at risk'. Plants species 'at risk' tested should include those: (1) related to the target weed; (2) not previously exposed to the biocontrol agent; (3) having limited information on their natural enemies; (4) having similar secondary compounds and/or morphological similarities with the target weed; (5) attacked by related organisms; and (6) recorded as hosts of the candidate agent (Schroeder 1983; Wapshere 1982, 1983).

I concur with Schroeder's (1983) statement: 'Simple infectivity tests are made with pathogens ... phytophagous insects require more elaborate tests', but his conclusion, 'A
<table>
<thead>
<tr>
<th>Term</th>
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<tbody>
<tr>
<td>AGGRESSIVENESS</td>
<td>The rate at which virulence is expressed; i.e., if two organisms produce equal-sized lesions on a host, but one does it in 2 days whereas the other requires 4 days, the first is more aggressive (twice as aggressive) than the second.</td>
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<tr>
<td>ESCAPE</td>
<td>An inherently susceptible plant that does not become infected in the field due to chance, some quirk of nature, some property of the susceptible other than resistance (q.v.), or because environmental conditions were not suitable for infection.</td>
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<tr>
<td>FIELD RESISTANCE</td>
<td>The inherent ability of a plant to reduce the amount of rate of disease development caused by all races of the pathogen. The term is synonymous with horizontal resistance, nonspecific resistance, etc.</td>
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<tr>
<td>HYPERSENSITIVITY</td>
<td>Extreme sensitivity to the pathogen, so that there is death of host cells and temporary or permanent inactivation of the pathogen at the point of attack, thus limiting infection.</td>
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<tr>
<td>IMMUNE</td>
<td>Exempt from infection.</td>
</tr>
<tr>
<td>PATHOGENIC</td>
<td>Able to cause disease.</td>
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<tr>
<td>PATHOGENICITY</td>
<td>The capability of being pathogenic.</td>
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<td>PREDISPOSITION</td>
<td>The weakening of an organism by some factor(s) of either the physical or biotic environment so as to render the organism more susceptible to a pathogen.</td>
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<td>RESISTANCE</td>
<td>The inherent ability of an organism to overcome in any degree the effects of a pathogen. It implies that the pathogen is able to infect the organism, and thus to react with it. If no such reaction occurs, then the organism is not resistant but immune (q.v.). Thus immunity is not the highest form of resistance, but is distinct from it. Two types of resistance have been denoted: major gene resistance = vertical resistance = specific resistance = oligogenic resistance = resistance controlled by one or a few genes, and operative against only certain races of the pathogen. In contrast, minor gene resistance = nonspecific resistance = field resistance = polygenic resistance = horizontal resistance = resistance controlled by a few to several genes, each perhaps acting in an additive manner, and effective to some extent against all races of the pathogen.</td>
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<td>SUSCEPT</td>
<td>An organism that can be attacked by, or is non-immune to, a given pathogen.</td>
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<tr>
<td>TOLERANCE</td>
<td>The capacity of a plant to endure disease; i.e., the inherent ability to become severely diseased without significant reduction in yield. Severity of symptom expression is disproportionately greater than reduction in yield.</td>
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<tr>
<td>VIRULENCE</td>
<td>A measure of the degree of pathogenicity (q.v.)</td>
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pathogen is considered a safe biological control agent if it is unable to develop and produce disease symptoms on any of the test plants' requires further comment.

It has been suggested by Hasan (1983), Schroeder (1983), and Templeton and Trujillo (1981) that host specificity tests using plant pathogens should be conducted under controlled conditions that are optimum for infection and disease development on the target weed. Unfortunately, results of host range or pathogenicity tests obtained from laboratory, growth chamber and greenhouse studies bear little, if any, similarity to what may occur in the field (Colhoun 1973, 1979; Cother 1975; Johnson and Taylor 1976; Yarwood 1959). Under artificial growth conditions the host ranges of many facultative saprophytes and obligate parasites have been artificially expanded (Table 2). There have been many cases of successful artificial inoculations of plants with pathogens which are not associated with these plants in the field.

Table 2. Some examples of pathogens which have had their field host range expanded in greenhouse or growth chamber conditions.

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>Reference</th>
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<tbody>
<tr>
<td><em>Alternaria cassiae</em> Jurair &amp; Khan</td>
<td>Walker (1982)</td>
</tr>
<tr>
<td><em>Cercospora rodmanii</em> Conway</td>
<td>Conway &amp; Freeman (1977)</td>
</tr>
<tr>
<td><em>Erysiphe graminis</em> DC.</td>
<td>Colhoun (1979)</td>
</tr>
<tr>
<td><em>Fusarium lateritium</em> (Nees) emend. Snyder &amp; Hansen</td>
<td>Walker (1981)</td>
</tr>
<tr>
<td><em>Phytophthora infestans</em> (Mont.) de Barry</td>
<td>Müller (1950)</td>
</tr>
<tr>
<td><em>Phytophthora palmivora</em> (Butl.) Butl.</td>
<td>Ridings <em>et al.</em> (1978)</td>
</tr>
<tr>
<td><em>Puccinia carduorum</em> Jacky</td>
<td>Politis <em>et al.</em> (1984)</td>
</tr>
<tr>
<td><em>P. jacea</em> Orth</td>
<td>Watson &amp; Alkhoury (1981); Mortenson (1985)</td>
</tr>
<tr>
<td><em>Puccinia centaurae</em> DC.</td>
<td>Clément &amp; Watson (1985)</td>
</tr>
<tr>
<td><em>Septoria avenae</em> Frank</td>
<td>Clark &amp; Zillinsky (1960)</td>
</tr>
</tbody>
</table>

The concept of predisposition (the tendency of nongenetic condition, acting before infection to affect the susceptibility of plants) and the results of studies on predisposition (Colhoun 1973, 1979; Schoeneweiss 1975; Yarwood 1959) can be used to explain the greenhouse phenomenon or growth chamber phenomenon that we have been encountering in the host specificity determination of candidate plant pathogens. Many factors are implicated in the phenomenon of induced susceptibility with reduced light intensity, increased temperature and water stress known to increase the susceptibility of plants to disease (Colhoun 1973, 1979; Schoeneweiss 1975; Yarwood 1959). Heat-induced susceptibility to nonpathogens is usually associated with a short exposure of a high temperature (50°C) (Chamberlain 1972). However, results of Baker and Larter (1963) demonstrated that a temperature of only 25°C was sufficient to disrupt the effectiveness of resistant genes in two barley cultivars to *Rhynchosporium secalis* (Oud.) Davis (Hyphomycetes). Environmental factors do affect the cuticle of plants (Schoeneweiss 1975) and secretions from the cuticle (Schönbeck 1976) which can affect infection by many plant pathogens.

It is obvious that environment has a profound effect on plant pathogen specificity and it appears relatively easy to expand the field host range of a plant pathogen (a candidate biocontrol agent) whether a facultative saprophyte or an obligate parasite, by testing in artificial greenhouse or growth chamber conditions. Therefore, in addition
to the need for more realistic inoculation or testing procedures (Cother 1975), we must be cautious in the interpretation of results obtained in our host specificity tests.

**Interpretation of Results**

The problem in accounting for the aberrant feeding of candidate biocontrol insects has received much attention in terms of host specificity testing procedures and conflict of interest discussions. The problems associated with interpretation of host range studies of plant pathogens conducted in greenhouses and controlled environment cabinets are probably more acute. Results of host range studies are questionable if the inoculation techniques and growth parameters are widely divergent from what naturally occurs in the field (Cother 1975). Interpretation of results obtained from inoculations under artificial conditions must be cautious and based on a sound, uniform understanding and interpretation of resistance and susceptibility in plant pathology.

Resistance and susceptibility are not absolute, but are different ends of the same scale (Gäumann 1950; Gracen 1982; Kuc 1979; Merrill 1980; Schoene Weiss 1975; Yarwood 1959). Plants which are not infected are not resistant, and should be termed immune. Resistance is thought to result from a specific interaction between the host and the pathogen, whereas susceptibility implies an absence of such an interaction (Johnson 1976). Daly (1976) suggested that "induced susceptibility" observed in artificial growth conditions was an impairment of the host's capacity to respond to signals of the infection. This suggests that resistance is an active process which represses or inhibits the invading microorganism.

The interpretation of susceptibility data is also often confounded by the absence of quantitative data. Results of host range studies with plant pathogens for biocontrol are presented in different ways from the infection type scale (0–4) for rust pathogens (Hasan 1972; Politis et al. 1984), disease ratings scale (0–5) (Walker and Sciumbato 1981) to a simple disease reaction with $S =$ susceptible and $R =$ resistant (Walker 1981, 1982).

It is not my intention to suggest the most appropriate rating system for our biocontrol studies since each case is different and one scale may be more appropriate depending on the characteristics of the host-pathogen and the response range. However, it is imperative that we recognize the importance of using standardized terminology and that we use disease rating systems that are as descriptive as possible. For example, Daniel et al.'s (1973) use of the term tolerant in their Table 2 should have been replaced by resistant or perhaps immune.

Results of host range studies obtained in artificial growth conditions give an indication of what may happen in the field, but these results do not provide indisputable evidence as to the susceptibility of a plant to a certain pathogen (Cother 1975). Possible explanations for the extended or broader host ranges of pathogens tested under growth chamber and greenhouse conditions are: (1) the pathogen and host have not come into effective contact in nature; (2) field observations have not been sufficiently intense; or (3) the results are artifacts of our testing program.

**Specificity and Classical Biological Control**

In classical biological control, it is difficult to overemphasize the importance of specificity. Rigorous host range testing is essential to ensure that a prospective exotic pathogen will not damage beneficial plants in the area or country of proposed introduction (Hasan 1983; Leonard 1982; Wapshere 1982). Early work with the heterococous *Rumex* rust, *Uromyces rumicis* (Schum.) Wint. (Uredinales), found all test
plants, except *Rumex crispus* L. and *R. maritimus* L. (Polygonaceae), were immune to infection with urediniospores (Inman 1971). However, Inman (1971) was unable to infect the known alternate host, *Ranunculus ficaria* L. (Ranunculaceae), with teliospores and the host range of this spore stage could not be tested. Absence of host range data on the teliospore stage prevented possible consideration for introduction of *U. rumicis* into North America. Recent work in Switzerland has produced techniques for obtaining teliospore germination and infection of the alternate host, thereby enabling the host range tests to be completed (Schubiger *et al.* 1985). Both spore stages of this rust appear to be narrowly restricted in their host range.

Elaborate host range tests with the autoecious rust *Puccinia chondrillina* Bubak & Syd. (Uredinales), confirmed its strict specialization to one host plant, *Chondrilla juncea* L. (Compositae) (Hasan 1972). Specificity in this pathogen is so strict that certain strains are virulent on only one form of *C. juncea*. The original virulent strain imported from Vieste, Italy, into Australia for the common form of *C. juncea* was capable of infecting the other two Australian forms, although less vigorously. However, after prolonged rearing and introduction into Australia it was unable to infect the other two forms (Cullen 1974).

The interpretation of the host range results of the strictly specialized *P. chondrillina* was relatively straightforward and the success of the *Chondrilla* rust program provided encouragement for research on rust pathogens of *Carduus*, *Centaurea* (both Compositae) and other weedy genera. Autoecious rusts of *Carduus* and *Centaurea* are reported to have narrowly restricted host ranges (Gäumann 1959; Guyot 1967; Ialongo and Boldt 1977; Savile 1970a, b, 1973). Host range testing of some of these *Puccinia* species have been conducted in containment facilities in the United States and in Canada. In all studies conducted to date, the laboratory (greenhouse or growth chamber) host range is broader than the reported field host range of these rusts. The controlled environment host range of *P. jacea* Otth. includes safflower (*Carthamus tinctorius* L.; Compositae) (Mortensen 1985; Watson and Alkhoury 1981); *P. centaurea* DC. includes safflower (Clément and Watson 1985), and *P. carduorum* Jacq. includes globe artichoke (*Cynara scolymus* L.; Compositae) (Politis *et al.* 1984). These crop plants have not previously been reported as hosts of these rusts. Details of these host range studies are published elsewhere or are in preparation. It is of interest to note that *P. carduorum* is already present in North America (Watson and Brunetti 1984), as is *P. centaureae* (Savile 1970b) and perhaps so is *P. jacea*. Interpretation of these results for the *Carduus* and *Centaurea* rusts are difficult and further studies are required before valid decisions on whether or not to introduce them can be made.

**Specificity and Biological Herbicides**

Since native plant pathogens are being evaluated as biological herbicides, strict host specialization is not required. Some authors have indicated that bioherbicide pathogens must be host specific (Auld *et al.* 1983; Hasan 1983). However, most fungi that are being evaluated as potential bioherbicides are facultative saprophytes with relatively wide host ranges, including some host-limited strains (restricted to one or a few plant species) (Daniel *et al.* 1973; Ridings *et al.* 1978; Walker 1981, 1982). Wapshere (1982) used the phrase 'sufficiently safe' which aptly describes the host specificity requirements of bioherbicides.

One of the first organisms to be evaluated as a biological herbicide, *Colletotrichum gloeosporioides* (Penz.) Sacc. f. sp. *aeschnomone* (Melanconiales), was thought to be narrowly specialized (Daniel *et al.* 1973), but recent tests involving more species has
revealed a larger host range than originally described (TeBeest 1984). Greenhouse and
growth chamber host range studies with other native facultative saprophytes considered
for the bioherbicide approach have not demonstrated strict host specialization (Conway
and Freeman 1977; Ridings et al. 1978; Walker 1981, 1982; Walker and Sciumbato
1979). When tested in field conditions, the host range is usually more restricted (Conway
and Freeman 1977; Walker 1982).

In some respects, the host range evaluations of candidate bioherbicides are analagous
to crop safety and efficacy testing for chemical herbicides. Obviously there must be a
margin of safety required for crops in which the target weed occurs and this safety
margin is less important for crops in which the target weed is not a problem. For
example, atrazine [2-chloro-4-(ethylamino)-6-(isopropylamino)-s-triazine] provides
effective weed control in corn, but is not recommended for use in cereal crops because
of its phytotoxicity to these crops.

Potential Risks and Suggested Approaches

No technology is without risk and the use of plant pathogens as biological control
agents of weeds is no exception. As discussed in this paper, the major concern of
biological weed control with plant pathogens is host specificity and the possible risk
of damage to desirable plant species. Leonard (1982) has reviewed the hazards involved
and suggested approaches to reduce these risks.

Host range changes could occur through mutation, adaptation, or hybridization or
result from predisposition of the pathogen during host range studies. The number of
mutations is a function of the number of spores produced. However, all mutations would
not result in host range expansion, most mutations would be deleterious to the pathogen
and some mutations may even be beneficial to the biocontrol program. The genetics
of host specificity in most plant pathogens is complex and a single mutation is not
sufficient to extend the host range of rust fungi (Leonard 1982). Is it reasonable to
assume mutations occurring with a pathogen used in a biological weed control program
to be different from those occurring or which have already occurred in the natural
habitat?

Adaptation of a pathogen to become more virulent on a host plant is possible and
has been demonstrated by repeated inoculation of some facultative saprophytes
(Gäumann 1950; Johnson 1976; Leonard 1982). However, repeated passage through
resistant or noncongenial host, especially for an obligate pathogen, reduces virulence
and results in progressive reduction in inoculum quantity until the 'inoculum threshold'
is reached, whereby there is insufficient inoculum for infection to occur (Gäumann
1950).

The possibility of hybridization occurring between two pathogens does exist, but as
Leonard (1982) stated, inheritance at the host species level is very complex and hybrids
usually have much reduced virulence on one or both parents. The prospects of virulent,
aggressive hybrids developing from crosses between P. jaceae and P. carthami Corda
or P. centaureae and P. carthami are remote. However, these crosses should be
attempted within containment facilities.

Although predisposition is not associated with an actual change or alteration in the
pathogen, it does have an effect on the expression of host range. Host range testing in
controlled environment conditions tends to predispose plants to infection which results
in artificial expansion of the host range. The possibility of predisposing normally
susceptible plants to become resistant in these tests is less likely (Colhoun 1973, 1979;
Yarwood 1959).
Concerns of risk and conflicts of interest with the use of plant pathogens as biocontrol agents for weeds will likely intensify. Questions of host specificity testing methods and data interpretation have been discussed and the potential for use of plant pathogens in biological weed control programs remains outstanding. Host range studies conducted solely in controlled environment conditions are not adequate for accurate host range determination of candidate biocontrol pathogens. I support the suggestions of Conway and Freeman (1977), Cother (1975), and others and strongly recommend: (1) Host range studies be conducted under realistic conditions that closely approximate the natural situation of the plant, the pathogen and the environment; and (2) Host range testing be two-phased with the first phase conducted within controlled environment conditions followed by a second phase of field evaluation. The field studies should be appropriately designed and replicated trials which are repeated each growing season for at least two or more (preferably three) growing seasons. Field evaluation of native pathogens requires fewer precautions and depending on the level of risk, exotic pathogen field testing may be restricted initially to the country of origin.

References


