

Managing *Armillaria* root rot

Roland T. V. Fox

School of Plant Sciences, The University of Reading, 2 Earley Gate, Reading RG6 6AU.UK.

email:r.t.v.fox@reading.ac.uk

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Abstract

Controlling *Armillaria* infections by physical and chemical methods alone is at present inadequate, ineffective, or impractical. Effective biological control either alone or in integration with another control strategy appears necessary. Biological control agents of *Armillaria* function by the antagonists inhibiting or preventing its rhizomorphic and mycelial development, by limiting it to substrate already occupied, by actively pre-empting the substrate, or by eliminating the pathogen from substrate it has already occupied. Among the most thoroughly investigated antagonists of *Armillaria* are *Trichoderma* species. Depending on the particular isolate of a *Trichoderma* species, control may be achieved by competition, production of antibiotics, or by mycoparasitism. The level of control is also influenced by the growth and carrier substrate of the antagonist, time of application in relation to the occurrence of the disease, and several environmental conditions. Among a range of the other antagonists are several cord-forming fungi and an isolate of *Dactylium dendroides*. Integrating biological methods with an appropriate method of chemical could control the disease more effectively. However it is essential to determine whether the antagonist or the fungicide should be applied first, and the time interval between.

Key words: Disease, antagonist, fungi, substrate.

Steps Toward Managing *Armillaria*

1. First know the enemy: Until comparatively recently honey fungus root rot was considered to be caused by a single species, *A. mellea* with an extremely varied morphology and levels of virulence. Now thirty six species of *Armillaria* are known occur in suitable habitats worldwide¹. Of these only a few pathogenic species cause the destructive honey fungus root rot symptoms^{2,3,4,5}. All species of shrubs, vines, fruit, amenity and forest trees studied, as well as some herbaceous plants in temperate and tropical regions are affected³⁵. Many of the species that have been described are virtually harmless saprophytes. Others such as *A. mellea*, *A. ostoyae*, *A. novae-zelandiae* and *A. luteobubalina* are highly virulent pathogens and others, such as *A. lutea*, attack trees under stress as well as infecting some susceptible healthy plants like strawberry².

2. Detect the enemy: The effects of *Armillaria* the honey fungus on living hosts start with growth loss, leading to decay and death⁴. Other symptoms of *Armillaria* root diseases are rather more non-specific, including reduction of shoot growth, changes in foliage characteristics, crown dieback, stress induced reproduction, basal stem indicators, white rot decay and death. Areas of forest affected by *Armillaria* root disease can even be distinguished in aerial photographs⁶. Nearly all these symptoms are so non-specific that they could be induced by a number of biological and non-biological causes⁷. However some signs of infection are specific to *Armillaria* species like the distinctive mycelial fans, characteristic rhizomorphs (also evident with *in vitro* cultures) as well as the characteristic tawny toadstools. Occasionally the wood being actively decayed by some, but not all, *Armillaria* species may be bioluminescent³. To confirm *Armillaria* root disease, the root collar and lower bole of the tree must be examined for those signs specific to the fungus or by culturing it onto agar from the host. As with other diseases, early diagnosis is vital for successful control⁸. However, as with other soil diseases, it is frequently comparatively difficult to identify the presence of infection before

the appearance of above-ground symptoms, by which time it is extremely hard, if not impossible, to control the disease. Conventional identification techniques involve isolation of the fungus mycelium present and culturing on agar but this takes a longer time than for many other fungi hence the cultures are very prone to contamination. With the advent of the modern diagnostic techniques based on enzyme-linked immunosorbent assay (ELISA) and molecular methods based on nucleic acid, however, rapid identification of the disease has become possible⁹.

3. Methods of control: Avoidance is often the best strategy^{10,11}. There are lists of plants of varying susceptibilities but these are a rough guide largely based on anecdotal evidence. For example some trees like holly and yew are much more tolerant than apple or lilac, they are not truly resistant like the grasses, some herbaceous plants¹² and some climbers like honeysuckle and ivy. So for complete confidence turf replant an infected area with grass, even though many herbaceous plants are also susceptible. Strawberry plants are killed within a few weeks enabling them to be used as "live bait" to reveal where *Armillaria* is present or absent in the soil. Although no woody plant has yet been demonstrated to be truly immune to infection by *Armillaria*, there are comparative differences in levels of tolerance between a variety of species that can be observed in the field inoculation experiments. In this way some rootstocks with useful resistance have been developed. While most *Prunus* species are severely damaged, plum (*Prunus domestica*) is generally more tolerant. These resistant rootstocks appear to be the only practicable control option in stone fruit orchards. Two rootstocks resulting from interspecific crosses between diploid plum and peach appear to satisfy both *Armillaria* resistance and other cultural demands. Exploitation of host resistance to *Armillaria* root rot requires knowledge of the genetics of both the host and the pathogen as well as environmental influences and managing mixtures of genotypes with varying levels of resistance. Discrepancies in resistance can be seen, and intraspecific or even individual variation in adaptation to sites. Considerable natural variation in interspecific and intraspecific

susceptibility may be observed even within a small locality, e.g. a hedge or an orchard². Managing *Armillaria* infections should start by evaluating the necessity for control, as the fungus may be present yet causes little damage¹³. Thus mere fungal presence may not provide an adequate basis to treat, especially as some poorly virulent species like *A. lutea* produce abundant rhizomorphs. Some well-adapted native forest trees may be sufficiently tolerant of *Armillaria* to act as a thinning agent in young stands and as a nutrient recycler in old stands. However following establishment control may be economically justified after the secondary spread of the disease^{14,5}.

Physical removal of inoculum by removing diseased trees and uprooting even neighbouring uninfected stumps has been recommended^{15, 17, 18,2}. In France trenches over a metre deep are dug to isolate the infected plants from healthy parts of a vineyard or a fruit orchard have been used to control spread of *Armillaria* root disease. This method has been adapted for cocoa and coffee plantations in Africa. Laying a plastic barrier in a trench and then backfilling it with the removed soil has been used for controlling the disease in kiwifruit orchards in New Zealand¹⁶, however these methods of physical control can be laborious and often impracticable for established forest trees¹⁹. Complete eradication of the fungus is also improbable and of doubtful value, and reinvasion by the fungus is possible. Soil disturbance may also stimulate fresh rhizomorph production, increasing the risk of disease to newly planted trees. Excessive removal of woody debris from a soil may be detrimental to antagonistic mycorrhizas²⁰. A major impediment in the chemical and biological control of *Armillaria* is the inability of the control agents to reach the site of inoculum inside wood in natural infections in sufficiently active state. The pathogen has also evolved highly sophisticated mechanisms of protection against outside deleterious effects. These include the production of antibiotics and the formation of pseudosclerotia. After becoming established in the roots, the vegetative mycelium of *Armillaria* develops a protective layer of thick-walled fungus cells, the pseudosclerotial envelope, about the body of the infected wood and the white mycelial fans in the cambium region. The rhizomorphs growing from the pseudosclerotium are also covered by this protective layer. Any chemical or biological control agent, would have to enter these resistant structures before it can eradicate the fungus²¹. Little experimental evidence exists to support the use of most commonly recommended fungicidal treatments for control of *Armillaria* species^{22,23,24} with the exception of soil fumigation with carbon disulphide, methyl bromide or chloropicrin after a susceptible crop. Although methyl bromide is due to be banned as it is a greenhouse gas, it is currently the most extensively used fumigant that is applied between crops because of its non-specific action and good penetrability in soil. Chloropicrin will destroy even the most resistant soil pathogens, but penetration through soil is much more difficult to achieve. Carbon disulphide can also be injected at regular intervals over an infected site after removing stumps, although it has a lower toxicity it is cheaper. It also has a high vapour pressure enabling it to penetrate deep into soil. This is a desirable attribute as *Armillaria* has been found viable at a depth of almost 3 m.

Armillaria in ponderosa pine (*Pinus ponderosa*) stumps has been successfully controlled using methyl bromide, Vorlex, chloropicrin, carbon disulphide, and Vapam. Methyl bromide, Vorlex, and chloropicrin eliminated the fungus from the stumps. However Vorlex and chloropicrin, when applied to the root-collars of small-diameter ponderosa pines, failed to reduce mortality

caused by *A. ostoyae*. Copper sulphate, iron sulphate, and copper wire were also found ineffective. Carbon disulphide is only effective in drier soils and fumigants must be injected at least 60 cm deep in soil, and this, at best, only decreases the number of foci rather than totally eradicating the disease. Moreover, the use of fumigants can be prohibitively costly and their toxic effects on other soil flora and fauna may be environmentally unacceptable.

Armillatox, a phenolic emulsion containing 48% cresylic acid as the active ingredient, has been marketed for specific use against *Armillaria*. This formulation was developed following the successful suppression of *Armillaria* with creosote although the latter proved too phytotoxic. A similar mixture of cresylic acids (Bray's Emulsion) was also marketed but again there were some phytotoxicity problems, control is not sustained and it has been withdrawn. In an effort to find an alternative non-phytotoxic chemical to Bray's Emulsion, a new generation of fungicides have been investigated by *in vitro* and in field trials at Reading. The most effective chemicals against *A. mellea in vitro* on 3% Malt Extract Agar were hexaconazole and flutriafol (both triazoles), fenpropidin (a piperidine), guazatine (a guanide), and phenyl phenol (a phenolic compound) with Bray's Emulsion as a standard. The two triazoles and the piperidine are ergosterol biosynthesis inhibitors (EBIs). The degree of activity shown by the chemicals depends on the species of *Armillaria* treated. *A. mellea* was generally the most resistant species of *Armillaria* to the chemicals. In tests using wood blocks, the EBI fungicides were the most effective protectant treatments, but guazatine, phenyl phenol and cresylic acid were the most effective eradicator treatments. However, in a field trial in a pear orchard infected by *A. mellea*, no evidence of an effect of application of hexaconazole, flutriafol, fenpropidin, guazatine, or cresylic acid, applied as eradicants or protectants, was found^{25,26}. Similarly, on blackcurrant bushes, treatments of phenyl phenol and cresylic acid as protectants and cresylic acid as an eradicator drench showed no effect on infection. Protectant drench treatments with phenyl phenol and cresylic acid showed serious phytotoxic effects²⁷.

In further field experiments, the phenolic fungicides, cresylic acid and phenyl phenol did not reduce the incidence of infection of privet bushes or willow by *A. mellea* in the field, but fenpropidin was effective in reducing it in some experiments. Phytotoxicity was caused by cresylic acid and to a lesser extent by phenyl phenol. Both cresylic acid and phenyl phenol were found to be good eradicants of mycelium *in vitro* on 3% Malt Extract Agar, causing 100% eradication when applied at 2400 and 500 mg/l, respectively. Fenpropidin was found to be generally fungistatic but appeared to be fungicidal at 5000 mg/l. Additional experiments showed the chemicals failed to fully eradicate rhizomorphs in soil even with as high concentration as 10,000 mg/l, and a thin layer of bark prevented any of the chemicals from eradicating the subcortical mycelium. The phenolic fungicides were not effective as eradicants, whereas fenpropidin exhibited good protectant activity *in vitro* and slowed the rate of mortality of inoculated strawberry plants, as did fosetyl-AI (Aliette). Phosphonic acid, the active ingredient in fosetyl-AI, has been reported to control *Armillaria*.

In some experiments, the mycelium inside wood treated with the phenolic fungicides was eventually stimulated to grow into fungicide solution (*in vitro*), or treated soil (*in vivo*). This stimulation of growth was more pronounced after treatment with cresylic acid than with phenyl phenol, and an increase in fungicide solution eventually led to an increase in growth²³. Because the currently available chemicals for controlling *Armillaria* are either

ineffective or are phytotoxic or have other environmental consequences, there is a need for biological control^{28,29,30,31,32,33,34,35,36,37,38}. In this one or more antagonistic organisms reduces the inoculum density or disease-producing activities of the pathogen in either its active or dormant state³⁹. This may be accomplished naturally or by manipulating the host, antagonist or the environment⁴⁰. Inoculum of the pathogen can be reduced through decreased survival between crops; decreased production or release of viable propagules, decreased spread by mycelial growth, reduction of infection of the host by the pathogen or reduction of severity of attack by the pathogen. Antagonism includes several types of activity: Antibiosis is the inhibition of one organism by a metabolic product of another. Lysis is a general term for the destruction, disintegration, dissolution, or decomposition of biological materials. Competition: "the endeavour of two or more organisms to gain the measure each wants from the supply of a substrate, in the specific form and under the specific condition in which that substrate is presented when that supply is not sufficient for both". Parasitism and predation: The antagonist may operate by simply using the pathogen as a food source, for which it may use enzymes such as chitinases and cellulases to break down the walls of its host⁴¹. The most effective antagonists appear to possess a balance of all these properties⁴¹. An ideal antagonist should produce inoculum in excess (for ease of growth); resist, escape, or tolerate other antagonists; germinate and grow rapidly (if applied as spores); and invade and occupy organic substrates. That would otherwise be occupied by *A. mellea*. The features of a biological control agent to be useful in combination rather than in isolation are moderate production of antibiotics, the ability to invade the pathogen as judged from *in vitro* tests, and moderate growth rates.

To control *Armillaria*, a photosphere or wood-inhabiting organism might function by inhibiting or preventing rhizomorph and mycelial development, by limiting the pathogen to substrate already occupied, by actively pre-empting the substrate, or by eliminating *Armillaria* (perhaps through replacement) from substrate already occupied. Antagonistic organisms might not be able to prevent *Armillaria* from becoming established in stumps, but they may restrict further stump colonisation and thus limit the available food base. Among the most thoroughly studied fungal antagonists of *Armillaria* are *Trichoderma* species, common fungal hyperparasites in the majority of soils^{42,43}. Extensive studies around the world on their antagonistic activity against many plant pathogens has indicated that successful control of a number of diseases, both in glasshouse and field conditions, may be possible with several species and strains⁴⁴. Suitable isolates can be obtained from suppressive soils and from a great variety of screening and selection procedures⁴⁵. Depending on the particular isolate, control may be achieved by competition, production of antibiotics, or by mycoparasitism. *Trichoderma* can compete with other microorganisms as it can more readily tolerate changes in its environment^{46,47} and ability to degrade various organic substrates in soil, metabolic versatility, and resistance to microbial inhibitors such as trichodermin and other antibiotic compounds⁴⁸. Species of *Trichoderma* are not only sources of various toxic metabolites but also of various enzymes such as exo- and endoglucanases, cellobiases, chitinases, cellulases, and proteases⁴⁹. These enzymes permit *Trichoderma* to parasitise different structures of pathogenic fungi.

Commercial interest in the development of formulations based on *Trichoderma* has led to several that are produced for sale, however there is currently none in use for controlling *Armillaria* though this may reflect more on business rather than biological

constraints⁵⁰. Within segments of root *Armillaria* can remain viable within its pseudosclerotium for seven years despite the presence of *Trichoderma* on surface of the segments for the full period. However, towards the end of the period, *Armillaria* appeared to be losing its ability to prevent the invasion of *Trichoderma* through the pseudosclerotium. This weakening of viability accompanied the exhaustion of the food reserves and perhaps also the accumulation of toxic metabolic products. *Trichoderma*, however, showed no signs of weakening, perhaps due partly to its continuous sporulation. Artificially infected root segments still contained viable mycelium of *A. mellea* after 76 months after being buried in moist non-sterile soil containing *Trichoderma* and 82 months in moist non-sterile peat moss but in a further test, *Armillaria* was found viable after 107 months, with the pseudosclerotium still intact³². The established inoculum of *Armillaria* can remain viable for six years or more in moist non-sterile soil containing *Trichoderma*, and the period of viability is probably affected by the amount of food reserve within the pseudosclerotium. Though these studies were the first thorough investigation into the interaction between *Armillaria* and *Trichoderma*, and the conclusions drawn may still be valid, it is not clear whether other isolates of *A. mellea* will resist this or other isolates of *Trichoderma* in a similar way. Moreover, the populations of *Trichoderma* in the soil were probably low as no artificial inoculations of the antagonist were made, and while *Armillaria* had an adequate food base (root segments), the antagonist was not provided with organic substrates for growth and proliferation, and production of antibiotics.

In vitro interactions of *Trichoderma* and *Armillaria* suggest that *Trichoderma* must be considered a possible controlling factor in the spread of pathogenic fungi. The degree of parasitism of *Armillaria* by *Trichoderma* varied with the pH of the medium and with the position at which the respective hyphae came into contact with one another. Using scanning electron microscopy, *Trichoderma* species were seen to attack and penetrate the melanised outer tissue of the rhizomorphs and, once inside, they killed the *Armillaria* hyphae by coiling and direct penetration^{51,52}. After one week, the rhizomorphs infected with each of the three species of *Trichoderma* were devoid of hyphae. *T. harzianum* and *T. hamatum* have also been shown to digest the cell walls of *Sclerotium rolfsii* and *Rhizoctonia solani* enzymatically^{53,51}. This causes the leakage of the cytoplasm from the host cells resulting in their emptiness. The host cytoplasm is, apparently, utilised by the pathogen, which is capable of degrading proteins and lipids.

Several other fungi including several important pathogens of the commercial Paris mushroom have also been found antagonistic towards *Armillaria*. *Rhizoctonia lamellifera* prevented *Armillaria* from colonising tea roots⁵⁴. *Scytalidium lignicola* or scytalidin, the toxin it produces, halts the growth of *Armillaria* in culture. *Phlebiopsis (Peniophora) gigantea*, *Pleurotus ostreatus* and several other basidiomycetes are capable of excluding *Armillaria* from substrates that they occupy. In addition, *Phlebiopsis gigantea* and *Pleurotus ostreatus* also effectively prevent *Armillaria* growth in freshly cut stumps into which they had been inoculated.

Coriolus versicolor, *Stereum hirsutum*, and *Xylaria hypoxylon* inoculated into stumps simultaneously with *A. luteobubalina* each significantly reduced colonisation by *Armillaria*. A naturally occurring, cord-forming species of *Hypoholoma* proved to be even more competitive with *Armillaria*, as it occupies the same initial sites and behaves similarly except for pathogenicity⁵⁵.

Several other species of cord-forming basidiomycetes, particularly *Phanerochaete velutina*, *Hypoholoma. fasciculare*,

and *Steccherinum fimbriatum* have considerable potential to spread and colonise woody debris in field sites^{56,57}. Several of them produce networks of mycelial cords in soil and litter, which can infest additional woody substrates. The populations of some cord-formers can be manipulated by chemically treating stumps⁵⁸. Ammonium sulphamate increases the colonisation by cord-formers of belowground portions of treated stumps.

Mycophagous nematodes have also been implicated in the biological control of *Armillaria*⁵⁹. Mycophagous nematodes greatly reduced mortality of ponderosa pine seedlings inoculated with *Armillaria*⁶⁰. The nematodes affected the growth of the fungus adversely. *Aphelenchus avenae* destroyed the hyphae of *Armillaria in vitro* but grew well on *Trichoderma polysporum* without reducing its growth.

Studies *in vitro* have shown that ectomycorrhizal fungi can inhibit the growth of *Armillaria*. However, direct protection by mycorrhizas seems unlikely as the main infection sites for *Armillaria* are on far larger roots rather than the fine roots where mycorrhizas develop⁶¹.

Integrated Control

Several examples of integrated control of *Armillaria* have been reported. *Trichoderma viride* can replace *Armillaria* in roots after fumigation between tree crops. Unfortunately such biocidal fumigants are unsuitable for use on established trees as they are too phytotoxic⁶². When root pieces infected with *A. mellea* were fumigated with carbon disulphide and incubated either without soil or in soil that was previously sterilised, *Armillaria* survived but when the infected root pieces were buried after fumigation in unsterilised soil or in soil amended with *Trichoderma*, *A. mellea* was killed and replaced by the antagonist. As pure soil cultures of *Trichoderma* were also able to kill *Armillaria* in unfumigated root inocula, *Armillaria* was not killed by direct fungicidal action of carbon disulphide but by the increase in the populations of *Trichoderma* spp. which overcome the protective pseudosclerotium then reaches every part of the fungus body and kills the mycelium by antibiotic action⁶³.

Sublethal methyl bromide fumigation prevents the production of antibiotics by *Armillaria*^{62,64}. A similar effect may be caused by heating or drying⁶⁵. These stress factors may be very critical and may concurrently stimulate antagonistic organisms resulting in further damage to the already weakened *Armillaria*, whose metabolism is affected, hindering the repair of any ruptures in the pseudosclerotial walls, breaching the defence mechanisms and ending antibiotic production. So the simultaneous increase in the growth of *Trichoderma* results in the death of *Armillaria*. Damaged mycelium of *Armillaria* leaks substances that could be attractive or stimulatory to *Trichoderma* spp. and other antagonists of *A. mellea*. *Trichoderma* spp. are several times more resistant to methyl bromide than *A. mellea*. Thus *Trichoderma* spp. able to survive much higher concentrations of methyl bromide than *A. mellea*, and populations are presumably unaffected or even increased by field fumigations with methyl bromide that are lethal to *A. mellea*^{66,67}. *Trichoderma* spp. are much more tolerant of environmental conditions than *A. mellea*. The responses of the two fungi to temperatures of 30 to 39 °C are particularly noteworthy, as the growth of *Trichoderma* increased as temperatures increased, whereas *A. mellea* was severely affected and its growth ceased. Hence soil heating might enhance biological control by *Trichoderma*. In experiments at Reading when the eradicator and protectant capability of a number of isolates of *Trichoderma* spp. and some other potential fungal antagonists

isolated from mushroom were tested against two isolates of *A. mellea in vitro*, several showed promising activity alone and in combination. Sterile spent mushroom compost was a suitable carrier substrate for the delivery of the fungal antagonists to soil.

The objectives of this work were to test the biocontrol efficacy of the antagonists in the glasshouse and field conditions both alone and as mixtures of antagonists, to integrate the biocontrol potential of selected antagonists with fungicide chemicals, to test different carrier substrate inocula at different dose rates, to find the best time for introducing the antagonists in relation to the occurrence of the disease and to study the effect of media type and concentration on antagonism. The results, which are due to be published shortly, warrant further evaluation.

Competitive Antagonists

The cord-forming saprotrophs *Hypholoma australe* and *Phanerochaete filamentosa* reduced the colonisation of karri stumps by *A. luteobubalina* significantly but only when the stumps were treated with ammonium sulphamate (AMD)^{68,55,69}. Although *P. filamentosa* all but eliminated *Armillaria*, AMS alone significantly enhanced colonisation by *Armillaria* below ground level, whereas at ground level or above ground level, colonisation by the pathogen was reduced. Stump decay was increased, and coppice occurrence reduced, with AMS treatment. The naturally occurring fungi *Stereum hirsutum* and *Trametes versicolor* fruited only on the AMS-treated stumps, whereas *Chondrostereum purpureum* fruited only on those not treated with the chemical.

Bliss⁶² suggests a possible role in limiting the saprophytic spread of *Armillaria* to new host plants by occupying the available substrates and taking hold of the niches that would otherwise be colonised by the pathogen. Preventing or eradicating infections in living host plants with these fungi would, however, not be advisable as they themselves cause decay. Control with *Trichoderma* is, therefore, desirable as it can not only work as an antagonist towards the pathogen, but is also safe to the host plant and can even stimulate growth responses in it.

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