

Biological Control of Sheath Blight in Rice Using Native Trichoderma Isolates

Philippa Bramwell¹ ●, Tiago Correia² ●, Solene Rousselot³ ●

¹ Senior Scientist, Department of Genetics and Plant Breeding, Wensleydale Institute of Livestock Science, Leyburn, United Kingdom. Email: p.bramwell@wils.ac.uk | ORCID: 0000-0001-2365-2755

² Research Associate, Department of Genetics and Plant Breeding, Alentejo Institute of Agrarian Sciences, Beja, Portugal. Email: t.correia@aias.pt | ORCID: 0000-0001-0816-0744

³ Professor, Department of Plant Pathology, Limousin Centre for Animal Health, Gueret, France. Email: s.rousselot@lcah-g.fr | ORCID: 0000-0001-2462-0165

ABSTRACT

*Sheath blight, caused by the soil-borne fungus *Rhizoctonia solani*, is among the most persistent diseases of irrigated rice, and *Trichoderma* is one of the most frequently proposed biological answers to it. Native isolates, collected from the soils and rice roots of the region where they will be used, are often preferred on the argument that they are already adapted to local conditions. This narrative review examines what published work says about that argument, drawing on the biology of the pathogen, the mechanisms by which *Trichoderma* suppresses it, the logic of isolate selection, formulation and delivery, and the design of the experiments that report control. It presents no new data. The evidence for antagonism is strong at the bench, where mycoparasitism, antibiotic metabolites and competition are readily seen, and the plant-mediated effects of *Trichoderma* make a field benefit plausible. The complication is that performance in a dish, a pot and a paddy are different questions. Results depend on isolate, formulation, timing, water management, canopy density and the host cultivar, and reports often omit the details needed to judge transfer. Integration with partially resistant cultivars and carefully timed fungicide use is the realistic setting for biocontrol, not a replacement for either. The review closes with the evidence a decisive multi-site trial would need to supply.*

Keywords: Sheath blight; *Rhizoctonia solani*; *Trichoderma*; Biological control; Rice; Native isolates; Integrated disease management; Narrative review

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1. Introduction

Rice feeds a large share of the world, and the diseases that attack it are among the quiet constraints on that task [1, 2]. Of these, sheath blight, caused by the soil-borne fungus *Rhizoctonia solani*, has long been counted among the injuries that matter most in intensive, irrigated production [3, 4]. It is the sort of disease a grower learns to expect: it comes with the dense, well-fertilised, humid canopy that high-yielding crops tend to produce, and it is hard to eradicate once the

inoculum is in the field.

Several features of the pathogen explain the difficulty. *Rhizoctonia solani* is not a single entity but a complex of anastomosis groups that differ in host range and behaviour, and the group that attacks rice is a particularly capable one [5]. Its genome points to a rich repertoire of enzymes that break down plant cell walls, in keeping with a pathogen that can infect many hosts [6]. Rice itself offers little help: resistance to sheath blight is partial and controlled by many genes, and no

cultivar is immune [7, 8]. Growers therefore lean on fungicides, with the cost, residue and resistance concerns that attend any single-site chemistry [9].

Against this background, biological control with *Trichoderma* is an obvious thing to try. These fungi are common in soil, they parasitise other fungi, they make antifungal metabolites, and they colonise roots in ways that can strengthen the plant [10, 11]. A further argument is often added: that isolates collected from the soils and rice roots of the region where they will be used, native isolates, should be better adapted to local temperature, water regime and soil than a strain imported from elsewhere.

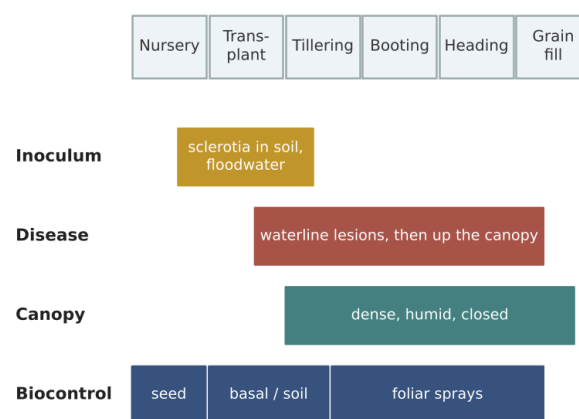
What is less clear is how far a result obtained with an isolate on an agar plate can be trusted to hold in a paddy. A claim that a native *Trichoderma* isolate controls sheath blight has two parts, a biological one and a practical one, and each can fail independently. An isolate may be a fine antagonist in a dish and a poor colonist of rice, or a good protector in a greenhouse pot and an unreliable one in a flooded field. This review sets out what is known about the pathogen, about how *Trichoderma* acts against it, and about how isolates are chosen, formulated and tested, and it asks how much weight the evidence can carry. The aim is to separate what has been shown from what has simply been repeated often.

The article is a narrative review. It draws on published work on *R. solani*, on *Trichoderma* biology and biocontrol, and on the design of disease experiments, and it deliberately reports no new data. Where the literature on rice is thin we say so, and we describe the evidence a convincing trial would need to supply, instead of quoting figures we have not been able to check against their sources.

2. Literature Review

2.1 Sheath Blight and Its Pathogen

The disease cycle explains much about where control might be placed (Figure 1). The fungus survives between seasons as sclerotia and as mycelium in plant debris, and when fields are flooded the sclerotia float and lodge on the sheaths of young plants at the waterline, where lesions first appear [4]. From there the fungus spreads upward through the canopy by contact between tillers and leaves, and the closed, humid microclimate of a dense crop favours it. Because the first infections occur at the water surface and low on the plant, a control agent has to be present in that same zone at the right time.



Schematic of timing, not data; bar lengths carry no values

Figure 1. Where in the rice season sheath blight develops and where biocontrol can be placed (schematic, not data).

The pathogen is variable. Anastomosis groups differ in host range and in pathogenicity [5], and genome work shows a fungus well equipped to degrade plant tissue [6]. This matters for biocontrol studies because the choice of pathogen isolate used in a test is part of the experiment. An agent that suppresses one strain in a dish may behave differently against another, and a report that names only the species cannot be repeated with confidence. This is one reason the same isolate can look excellent in one report and unremarkable in the next.

Host resistance is the other half of the picture. Decades of work have identified cultivars with a degree of tolerance and a number of regions of the rice genome that contribute to it, but the resistance is quantitative and incomplete [7, 8]. For a biocontrol agent that is a mixed blessing. It means that no cultivar removes the need for further measures, but it also means that a biological agent has a realistic role as one component of a package, not as a lone answer.

2.2 *Trichoderma* as a Biocontrol Agent

Trichoderma is a genus of filamentous fungi that live in soil, on decaying wood and on roots, and its species are best known to plant pathologists as antagonists of other fungi [12, 13]. Comparative genome work suggests that attacking other fungi is the ancestral way of life of the genus, from which the ability to live with plants later developed [14]. The same work, and the broader genomic reviews that followed, describe a group that is metabolically versatile and competitive in complex microbial communities [15, 16].

This history matters for how *Trichoderma* should be described. It is better understood as an opportunistic, largely avirulent plant symbiont

than as a simple pesticide in a bag [10], and its effects on a crop come from several processes acting together. Some of those processes are direct, aimed at the pathogen. Others are indirect, mediated by the plant. A sheath blight trial that measures only final disease score cannot say which was at work, and the answer bears on whether the effect will transfer to another cultivar, soil or season. A fungus that has had time to establish behaves differently from one that arrives the same day as the pathogen.

Taxonomy is a quiet source of confusion. Species within the genus differ in behaviour, and older reports that name only a broad species concept, often *T. harzianum* or *T. viride*, may cover isolates that modern identification would separate [12, 15]. For native isolates, which are by definition collected rather than bought, identification to species level, ideally with sequence data and a deposited culture, is the minimum that lets a later reader know what was tested.

2.3 How *Trichoderma* Acts Against *Rhizoctonia solani*

The best-documented mechanism is mycoparasitism. Microscopy has shown *Trichoderma* hyphae coiling around those of *R. solani* and related soil-borne fungi, adhering to them and in places penetrating the wall [17]. The enzymes involved, chitinases and glucanases among them, attack the structural polymers of the target cell wall [18, 19], and genes for such enzymes have been used to improve resistance in transgenic plants, which indirectly shows how central they are to the antagonism [20]. These are laboratory observations of striking clarity.

Antibiosis is the second route. *Trichoderma* species produce a long list of antifungal metabolites, diffusible and volatile [21, 22], and some of these compounds also act on the plant, affecting growth and defence [23, 24]. Competition for nutrients and space is the third, and it is harder to demonstrate directly, although the speed with which the genus colonises substrates is a long-recognised part of its ecology [25, 26]. The relative weight of the three is not settled and probably differs between isolates, which is one reason a single mechanism assay seldom predicts how an isolate will behave on a crop. Genomic surveys make the same point, showing a large and varied set of genes behind these activities across the genus [15]. Table 1 summarises the mechanisms and sets beside each of them what it would need to work in a flooded field.

Table 1. Mechanisms by which *Trichoderma* acts against *Rhizoctonia solani*, and what each needs in the field.

Mechanism	Seen at the bench	Needed in the field
Mycoparasitism	Coiling around hyphae, wall penetration, lytic enzymes [17, 18, 20]	Contact with the pathogen at the sheath or in debris
Antibiosis	Diffusible and volatile metabolites that slow the pathogen [21, 23]	Metabolite production under flooded, low-oxygen conditions
Competition	Faster growth, use of nutrients and space [25, 26]	Establishment before the pathogen builds up
Induced resistance	Defence responses switched on in the host [27, 28, 29]	Root colonisation that persists through the season
Growth promotion	Larger seedlings, altered root growth [31, 33]	A response that is not masked by fertiliser

Note: Qualitative summary of the cited literature. A mechanism seen on an agar plate is a hypothesis for the paddy, not a result.

Nothing in the table is controversial. What it shows is the difference between a mechanism and a result. Each mechanism has conditions under which it is seen, and the conditions of a dual-culture plate, with a nutrient-rich medium, a short distance between the fungi and no competing microbes, are not those of the sheath zone of a rice plant standing in water. Small differences of this kind decide whether a fungus gets a foothold or not.

2.4 Plant-Mediated Effects: Resistance and Growth

A second line of argument for *Trichoderma* has little to do with the pathogen at all. Colonisation of roots by the fungus can switch on defence responses in the plant, shown in cucumber [27], and reviews describe systemic changes in defence signalling that follow colonisation by beneficial fungi [28, 29]. Colonisation itself seems to involve the fungus managing the plant's early defence response, so that it can establish without being rejected [30].

Growth effects are reported as well. In *Arabidopsis*, one species enhanced biomass and lateral root growth through an auxin-dependent route [31], and surveys of the plant-beneficial effects of the genus describe a range of responses that depend on the strain and the plant [32]. In rice, inoculation with *Trichoderma* has been reported to alter seedling growth and physiology [33]. Seed treatment with *T. harzianum* has been reported to reduce several kinds of stress in germinating seeds and seedlings [34].

The honest reading of this literature is that the plant-mediated route makes a field benefit plausible, but it also makes the result harder to predict. A response in which the plant plays a part will depend on the cultivar, the nutrition and the stage of growth, and it is exactly the kind of response that a pot trial with one cultivar cannot fully reveal. Reports that attribute a reduction in sheath blight to antagonism, when the plant may have contributed, should be read with that in mind.

2.5 Native Isolates and the Problem of Colonisation

The case for native isolates rests on adaptation. An isolate taken from the soil or roots of local paddies has, in some sense, already survived the temperature range, water regime and microbial community of the place. That is a reasonable expectation, and it is the reason many programmes begin by sampling locally. It is not, however, a demonstrated rule. An isolate that survives in a soil may be a poor colonist of the rice root, and an isolate from a dry upland soil may behave very differently in a flooded one.

Rhizosphere competence, the ability to establish and persist on and around roots, is a property that has been emphasised for years as central to the success of a root-applied agent [35, 36]. It cannot be inferred from a plate assay. The same body of work argues that strains within a species differ in this respect and that the way an agent is applied can matter as much as the strain [35]. For sheath blight the question is sharper still, because the target zone is not only the root but also the base of the sheath at the water surface.

Native isolates raise one further question: what is the comparison? An isolate is said to be better adapted than an imported one, but trials that include both are scarce. Without that arm, local adaptation is an assumption. Without an arm for a standard fungicide, the practical scale of any effect cannot be judged. These are design questions, not reasons to doubt *Trichoderma*. Reviews of this kind are more useful when they say where the evidence stops than when they smooth it over.

2.6 Formulation, Delivery and the Road to a Product

Between an isolate and a product lies formulation. Spores or mycelium have to be produced, carried in a material that keeps them alive, stored, transported and applied by seed coating, soil application or spray. The review literature on microbial inoculants makes the general point that

a good organism is not enough: carrier, additives and storage conditions decide whether enough viable cells reach the field [37, 38]. Most of that work concerns bacteria, so the lessons are transferred by analogy, but the logic applies to fungal agents too.

Reviews of *Trichoderma*-based products describe a wide range of formulations on the market and the difficulty of moving a promising isolate through to a reliable product [11, 39]. Knowing how an agent works matters here beyond the question of efficacy, because mode of action informs what to select for, how to apply it and how to describe it to regulators and users [40]. A report that characterises its isolate only by plate inhibition leaves almost all of those questions open.

3. Materials and Methods

3.1 Scope and Question

The review asks one question in two parts: is there good reason to expect that native *Trichoderma* isolates can suppress sheath blight of rice under field conditions, and how consistent is the evidence that they do? The question was divided into sub-questions that any study would have to answer: how the isolate was obtained and identified, how the pathogen was chosen and applied, how disease was measured, how the design handled replication and variation between sites and seasons, and how the agent was formulated and applied.

3.2 Literature Assembly

The review draws on peer-reviewed work in four areas: the biology and genetics of *R. solani* and of sheath blight resistance in rice; the taxonomy, genomics and mechanisms of *Trichoderma*; formulation and the development of microbial products; and the methodology of experiments and their statistics. Sources were chosen for relevance, standing in the field and the clarity of their methods. Work on other crops, on other pathogens and on bacterial agents is used for what it teaches about mechanisms and limits, not as evidence of effect in rice.

We did not run a formal systematic search and we do not claim one. This is a narrative synthesis, and the choice of sources reflects the authors' judgement. The practical consequence is that the review is a better guide to what a sound study should contain than to the exact size of any reported effect, and it avoids quoting figures from individual field trials that could not be checked against the primary papers for this article.

3.3 Appraisal Framework

To judge how far a report can be trusted we asked a short set of questions, listed in Table 2. They concern the identity of the isolate and of the pathogen strain, the unit of replication, the way disease was scored and the presence of a standard comparator. Statistical questions were framed by the long-standing concern about pseudoreplication [41], by the evidence that published results in many fields are less reliable than they appear [42], and by the argument for reporting estimates with their uncertainty rather than only significance tests [43].

Table 2. Questions used to read a report of *Trichoderma* biocontrol of sheath blight.

Question	Why it matters	Weak answer looks like
How was the isolate identified?	Species differ in behaviour [12]	Genus only, no sequence or deposit
What was the pathogen source?	Strains of the pathogen differ in aggressiveness [5]	Single unstated strain
What was the unit of replication?	Overstated precision otherwise [41]	Plants as replicates in one tray or plot
How was disease scored?	Scales differ between studies	Method not described
Was a fungicide comparator included?	Gives the effect a practical scale	No standard treatment

Note: The questions are the authors' appraisal framework, not a scoring scheme.

4. Results

4.1 What the Bench Shows

At the bench the evidence is strong and consistent in kind. *Trichoderma* isolates inhibit *R. solani* on plates, coil around its hyphae and make enzymes and metabolites that damage it [17, 18, 21]. Early work on *R. solani* in a field crop, strawberry, reported suppression by *T. harzianum* [44], and bacterial agents have been characterised against the rice sheath blight pathogen in similar ways [45]. A native isolate that fails to inhibit the pathogen in a dish is unlikely to be of interest, so the plate is a reasonable first filter.

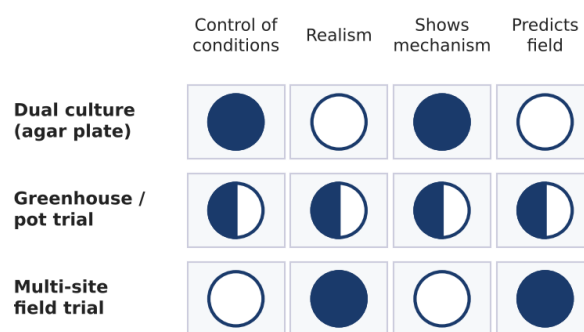
The limit of that filter is what it measures. It shows capacity for antagonism under convenient conditions. It does not show that the agent will reach the sheath, grow there, produce its metabolites at the right time, or survive the rest of the season. A plate result is best treated as a screen that removes poor candidates, not as a prediction of control, and the risk of mistaking one for the other is the main practical hazard in this

field. The point is not that a study is poor, only that its result travels less far than its abstract suggests.

4.2 From Dish to Pot to Paddy

Each level of testing answers a different question (Figure 2). The plate has the best control of conditions and is the clearest about mechanism, but it is the furthest from the crop. A greenhouse or pot trial puts a living rice plant and a pathogen in the same pot, with temperature, humidity and inoculum under the experimenter's control. It is therefore the right place to ask whether an isolate protects rice at all. A field trial is the least controlled and the least informative about mechanism, but it is the only one that tests the claim a farmer cares about.

Moving from pot to field changes several things at once. The water is deeper and moves. The canopy is denser and carries its own microclimate. The soil holds a resident microbial community that the introduced fungus must live among [36]. The pathogen arrives as natural inoculum in sclerotia and debris and not as a measured dose applied at a chosen moment [4]. A result that holds when all of this is varied is worth far more than one obtained under a single set of conditions, and reports that include several sites or seasons are accordingly more informative than reports from one.



Filled = strong, half = partial, empty = weak (judgement, not data)

Figure 2. What each level of testing can tell the reader (qualitative judgement, not data).

Few reports provide that breadth. The published literature on *Trichoderma* biocontrol across crops offers many plate and greenhouse results and many field trials from single sites, and it has long been observed that field performance is more variable than laboratory performance [26, 35]. We cannot give a figure for that variability without quoting numbers from particular trials, which this review does not do. The qualitative pattern, however, is consistent across the sources we examined. Read this way, a failure to see an effect in one small trial is not proof that none exists.

4.3 Why Results Differ: Isolate, Formulation, Timing, Environment

The sources of inconsistency are listed in Table 3. The first is the isolate itself. Species and strains differ in the traits that matter [13], and genome comparisons show how diverse the genus is [15]. The second is formulation: the carrier and the storage conditions determine how many viable propagules reach the field, and the literature on inoculants documents the loss of viability as a major cause of failure [37, 38]. The third is timing. An agent has to be established before the pathogen builds up, which is the reason seed and seedling treatments feature in many programmes [34, 35].

The environment is the fourth source and probably the most important. Flooding changes the oxygen available to a fungus, and a dense canopy changes the humidity and temperature at the sheath, which also favour the pathogen [4, 36]. A *Trichoderma* isolate may do well in a season that is cool and open, and poorly in one that is hot and humid, and a trial in a single season cannot show which. The fifth is the cultivar. Partial resistance varies between varieties [7, 8], and the added effect of a biological agent on a tolerant cultivar may differ from its effect on a susceptible one.

Table 3. Sources of variation between bench, pot and field results.

Source of variation	Why it matters	What to report
Isolate and species	Antagonism differs between strains [13, 15]	Identity, origin, deposit
Formulation	Survival and shelf life depend on carrier [37, 38]	Carrier, viable count, storage
Timing and placement	Colonisation must precede infection [34, 35]	Seed, soil or foliar; dates
Water and canopy	Flooding and dense canopy shape both fungi [4, 36]	Water regime, spacing, fertiliser
Host cultivar	Partial resistance varies [7, 8]	Cultivar and its reaction
Chemical programme	Fungicides can harm the agent [9]	Product, dose, spacing from the agent

Note: Qualitative. The right-hand column states what a report should say so that a reader can judge transfer.

These factors interact, and that is what makes consistent field results hard to obtain. A good isolate in a poor formulation applied late in a dense canopy will do little, and the same isolate in a good formulation applied early may do a great deal. When a study reports only the final disease score, the reader cannot tell which combination

was tested. The three rows of Table 3 that matter most for the interpretation of a negative result are formulation, timing and water regime; a failure under one set does not condemn the isolate. A control that disappears when the season changes is not a control a farmer can plan around.

4.4 Integration with Host Resistance and Fungicides

Because partial resistance is the best that rice currently offers [7, 8], and because biological control rarely gives complete protection, the realistic role for *Trichoderma* is within an integrated programme. A partially resistant cultivar slows the epidemic; a well-timed fungicide can suppress it when pressure is high; a biological agent may reduce initial infection or add to the plant's own defences. The combination may be more consistent than any one measure, although that remains a proposition to be tested, not an established result.

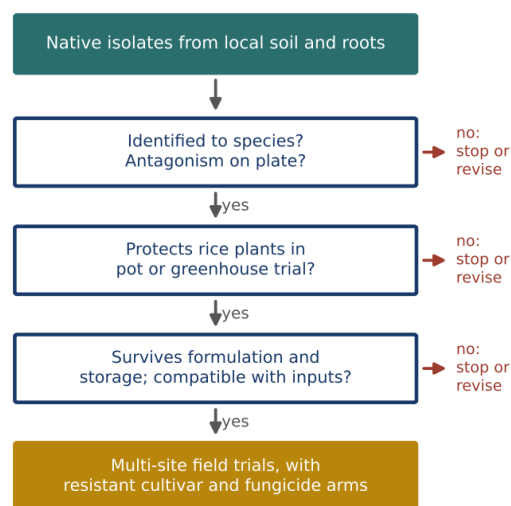
Compatibility is the practical difficulty. Fungicides are designed to inhibit fungi, and *Trichoderma* is a fungus. Single-site chemistries such as the strobilurins act on a specific target in fungal respiration [9], and whether a given product at a given dose harms the introduced agent is a matter for direct test. Spacing applications so that the agent is established before a fungicide is used, or choosing products with less activity against it, are possible solutions, but reports that examine such combinations in rice are not numerous enough for us to draw a general rule. Until such combinations are tested directly, any claim of an additive benefit is best treated as a hypothesis.

A staged route for deciding whether a native isolate deserves field testing is sketched in Figure 3. The sketch is not a protocol. It makes the point that each stage answers a different question and that an isolate should be dropped or revised when it fails one, not carried forward on the strength of an earlier success. The final stage is the one most often skipped: a multi-site trial with arms for cultivar resistance and a standard fungicide.

4.5 Reading a Claim of Control

A reader meeting a claim that a native isolate controls sheath blight can ask a short sequence of questions. How was the isolate identified, and is it deposited? Which pathogen strain was used, and how was it applied? What was the unit of replication, and how was uncertainty reported [41, 43]? Was a standard fungicide included as a comparator? Was the effect seen in more than one season or site? A report that answers all five is uncommon, and one that answers none should be

read as an observation, not as evidence of effect.



Schematic decision route; not a validated protocol

Figure 3. A staged route for advancing a native isolate (schematic decision tree, not data).

Context is the other half of a claim. A result from one paddy in one season says what happened there, and the reader needs the water regime, the planting density, the fertiliser programme and the weather to judge whether it applies elsewhere. Without them, even a sound experiment becomes an anecdote that is hard to compare with the next one [36, 40].

Small studies deserve particular caution. Estimates from small experiments are unstable, and those that reach significance tend to overstate the effect [42]. Where several plots are sampled within a single field the field is the replicate, not the plot, and treating plots as independent inflates apparent precision [41]. These problems are shared with much of applied biology and *Trichoderma* is not special in having them; they matter here because the case for biocontrol rests heavily on this kind of study.

5. Discussion

5.1 Interpretation

The central finding of this review is a conditional one. There is strong evidence that *Trichoderma* can antagonise *R. solani* and good reason to think it can help the rice plant, and there is much less evidence that any particular native isolate will deliver steady control in the field. The gap between those two statements is not a failing of the fungus. It is a consequence of how the question has been studied, with a heavy weight on mechanisms and a light one on multi-site, multi-season field performance. Nothing in this reading argues against using *Trichoderma*; it

argues for measuring what happens when it is used.

Native origin does not close that gap. Adaptation is an argument for sampling locally, and local isolates are a sensible place to begin, but nothing in the literature we reviewed shows that a native isolate outperforms a well-chosen non-native one where both have been compared in the same trial. The advantage of the native isolate may lie elsewhere, for instance in the speed with which a local programme can collect, characterise and deploy it, and in the ecological comfort of using an organism already present in the system.

The second conditional concerns formulation and timing. A promising isolate is wasted by a poor carrier or a late application. The literature on inoculants is clear that survival through production, storage and application is a problem to be solved deliberately [37, 38], and the commercial experience described for the genus suggests that this step takes as much effort as finding the isolate [11, 39]. These points apply with equal force to any microbial agent, not only to *Trichoderma*.

Three findings would change this reading. A multi-site, multi-season trial showing steady suppression by a named native isolate, relative to an untreated control and a standard fungicide, would settle the case for that isolate in those conditions. Evidence that suppression arises mainly through the plant rather than the pathogen would shift attention to cultivar interactions and colonisation. And a systematic comparison of native and non-native isolates in the same experiments would show whether local origin carries any measurable advantage.

5.2 Limitations

This is a narrative review and shares the limitations of that form. The selection of sources reflects judgement, no formal search or quality scoring was done, and the review does not quote numerical results from individual field trials, because these could not be checked against the primary reports for this article. A reader who needs an effect size should consult the original studies directly, applying the appraisal questions above, or wait for a formal systematic review with meta-analysis.

The review also draws on evidence from other crops, other pathogens and other microbial agents, which are imperfect proxies for rice and sheath blight. Mechanistic work was done on model plants and on dicot crops [27, 31], and formulation lessons come largely from bacterial

inoculants [37, 38]. We have used such evidence to frame hypotheses, and the conclusions should be read in that light.

5.3 Implications for Trial Design

A trial that could settle the question would identify the isolate to species and deposit it, and state the source and strain of the pathogen. It would include an untreated control, a standard fungicide comparator, a non-native reference isolate and, where possible, a partially resistant and a susceptible cultivar. It would record the formulation, the dose, the date and the placement of each application, and it would verify that the agent was present at the sheath and root, not only in the bag.

The statistical design would treat the field or block as the unit of replication, not the plot or the plant, with enough sites and seasons to see variation between them [41]. Disease would be scored by a described method at fixed stages, and results would be reported as estimates with intervals, together with yield where possible [43]. These requirements are demanding, but they are the ones that separate a result a grower can use from one that only supports a hunch.

5.4 Directions for Future Work

Three needs stand out. First, a formal systematic review of *Trichoderma* field trials against sheath blight, with a transparent search and quality appraisal, ideally with a synthesis that treats between-site variation as a finding to be explained. Second, side-by-side field comparisons of native and non-native isolates, in formulations that can be defended, with arms for resistance and fungicide. Third, work on how colonisation of rice roots and sheaths by *Trichoderma* actually proceeds in flooded fields, because the mechanisms best documented at the bench have not been demonstrated at the sheath [16, 40].

5.5 Practical Reading for Advisers and Growers

For an adviser, the sensible position is neither to dismiss *Trichoderma* nor to promise results. A grower who wishes to try a native isolate can do so as a small, recorded pilot: treated and untreated strips in the same field, on the same cultivar, with the same water and nutrient management, and with disease scored in the same way on both. Notes on the product used, the date and the weather are as valuable as the disease scores, because they let the result be interpreted later [41, 43].

Such a pilot is not a substitute for a trial and should not be reported as one. It can nonetheless tell a grower whether the practice is workable on the farm, whether the timing fits the routine, and whether any difference is large enough to notice against the ordinary variation between parts of a field. Its value lies in local decision-making, and the broader question of effect size will be settled by designed trials and by systematic synthesis [42].

6. Conclusion

Native *Trichoderma* isolates are a reasonable and attractive candidate for the biological control of rice sheath blight. The evidence for antagonism in the laboratory is strong, and the plant-mediated effects of the genus make a field benefit plausible. The evidence, as we can responsibly summarise it, does not yet establish that any particular native isolate gives consistent control in the paddy, and results depend on the isolate, the formulation, the timing, the water regime, the canopy and the cultivar. Readers should treat claims of reliable control with caution until they meet the standards set out here.

For the practitioner the message is practical rather than discouraging. A native isolate can be tried, provided it is identified, formulated and applied with care, and integrated with a partially resistant cultivar and sensible fungicide use. For the researcher, the opportunity is a set of multi-site trials that are large enough, long enough and well enough described to turn a plausible idea into a measured result on which rice growers can rely.

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9. Conflict of Interest

The authors declare no conflict of interest.

10. Author Contributions

P.B. conceived the review and wrote the sections on pathogen biology and host resistance. T.C. wrote the sections on *Trichoderma* mechanisms and formulation. S.R. wrote the sections on field evaluation and trial design. All authors revised the manuscript and approved the final version.

11. Ethics Statement

This is a review of published literature. No human participants or animals were involved and no ethical approval was required.

12. Data Availability

No new data were generated. All sources are cited in the reference list.

13. References

- [1] Savary, S., Ficke, A., Aubertot, J.-N., & Hollier, C. (2012). Crop losses due to diseases and their implications for global food production losses and food security. *Food Security*, 4(4), 519–537. DOI: [10.1007/s12571-012-0200-5](https://doi.org/10.1007/s12571-012-0200-5)
- [2] Savary, S., Willocquet, L., Pethybridge, S. J., Esker, P., McRoberts, N., & Nelson, A. (2019). The global burden of pathogens and pests on major food crops. *Nature Ecology & Evolution*, 3(3), 430–439. DOI: [10.1038/s41559-018-0793-y](https://doi.org/10.1038/s41559-018-0793-y)
- [3] Savary, S., Willocquet, L., Elazegui, F. A., Castilla, N. P., & Teng, P. S. (2000). Rice pest constraints in tropical Asia: Quantification of yield losses due to rice pests in a range of production situations. *Plant Disease*, 84(3), 357–369. DOI: [10.1094/PDIS.2000.84.3.357](https://doi.org/10.1094/PDIS.2000.84.3.357)
- [4] Lee, F. N., & Rush, M. C. (1983). Rice sheath blight: A major rice disease. *Plant Disease*, 67(8), 829–832. DOI: [10.1094/PD-67-829](https://doi.org/10.1094/PD-67-829)
- [5] Ogoshi, A. (1987). Ecology and pathogenicity of anastomosis and intraspecific groups of *Rhizoctonia solani* Kühn. *Annual Review of Phytopathology*, 25, 125–143. DOI: [10.1146/annurev.py.25.090187.001013](https://doi.org/10.1146/annurev.py.25.090187.001013)
- [6] Zheng, A., Lin, R., Zhang, D., Qin, P., Xu, L., Ai, P., ... Wang, P. (2013). The evolution and pathogenic mechanisms of the rice sheath blight pathogen. *Nature Communications*, 4, 1424. DOI: [10.1038/ncomms2427](https://doi.org/10.1038/ncomms2427)
- [7] Molla, K. A., Karmakar, S., Molla, J., Bajaj, P., Varshney, R. K., Datta, S. K., & Datta, K. (2020). Understanding sheath blight resistance in rice: The road behind and the road ahead. *Plant Biotechnology Journal*, 18(4), 895–915. DOI: [10.1111/pbi.13312](https://doi.org/10.1111/pbi.13312)
- [8] Srinivasachary, Willocquet, L., & Savary, S. (2011). Resistance to rice sheath blight (*Rhizoctonia solani* Kühn) disease: Current status and perspectives. *Euphytica*, 178(1), 1–22. DOI: [10.1007/s10681-010-0296-7](https://doi.org/10.1007/s10681-010-0296-7)
- [9] Bartlett, D. W., Clough, J. M., Godwin, J. R., Hall, A. A., Hamer, M., & Parr-Dobrzanski, B. (2002). The strobilurin fungicides. *Pest Management Science*, 58(7), 649–662. DOI: [10.1002/ps.520](https://doi.org/10.1002/ps.520)
- [10] Harman, G. E., Howell, C. R., Viterbo, A., Chet, I., & Lorito, M. (2004). *Trichoderma* species: Opportunistic, avirulent plant symbionts. *Nature Reviews Microbiology*, 2(1), 43–56. DOI: [10.1038/nrmicro797](https://doi.org/10.1038/nrmicro797)
- [11] Woo, S. L., Ruocco, M., Vinale, F., Nigro, M., Marra, R., Lombardi, N., ... Lorito, M. (2014). *Trichoderma*-based products and their widespread use in agriculture. *The Open Mycology Journal*, 8(1), 71–126. DOI: [10.2174/1874437001408010071](https://doi.org/10.2174/1874437001408010071)
- [12] Samuels, G. J. (2006). *Trichoderma*: Systematics, the sexual state, and ecology. *Phytopathology*, 96(2), 195–206. DOI: [10.1094/PHYTO-96-0195](https://doi.org/10.1094/PHYTO-96-0195)
- [13] Harman, G. E. (2006). Overview of mechanisms and uses of *Trichoderma* spp. *Phytopathology*, 96(2), 190–194. DOI: [10.1094/PHYTO-96-0190](https://doi.org/10.1094/PHYTO-96-0190)
- [14] Kubicek, C. P., Herrera-Estrella, A., Seidl-Seiboth, V., Martinez, D. A., Druzhinina, I. S., Thon, M., ... Grigoriev, I. V. (2011). Comparative genome sequence analysis underscores mycoparasitism as the ancestral life style of *Trichoderma*. *Genome Biology*, 12(4), R40. DOI: [10.1186/gb-2011-12-4-r40](https://doi.org/10.1186/gb-2011-12-4-r40)
- [15] Druzhinina, I. S., Seidl-Seiboth, V., Herrera-Estrella, A., Horwitz, B. A., Kenerley, C. M., Monte, E., ... Kubicek, C. P. (2011). *Trichoderma*: The genomics of opportunistic success. *Nature Reviews Microbiology*, 9(10), 749–759. DOI: [10.1038/nrmicro2637](https://doi.org/10.1038/nrmicro2637)
- [16] Mukherjee, P. K., Horwitz, B. A., Herrera-Estrella, A., Schmoll, M., & Kenerley, C. M. (2013). *Trichoderma* research in the genome era. *Annual Review of Phytopathology*, 51, 105–129. DOI: [10.1146/annurev-phyto-082712-102353](https://doi.org/10.1146/annurev-phyto-082712-102353)
- [17] Elad, Y., Chet, I., Boyle, P., & Henis, Y. (1983). Parasitism of *Trichoderma* spp. on *Rhizoctonia solani* and *Sclerotium rolfsii*: Scanning electron microscopy and fluorescence microscopy. *Phytopathology*, 73(1), 85–88. DOI: [10.1094/Phyto-73-85](https://doi.org/10.1094/Phyto-73-85)
- [18] Viterbo, A., Ramot, O., Chernin, L., & Chet, I. (2002). Significance of lytic enzymes from *Trichoderma* spp. in the biocontrol of fungal plant pathogens. *Antonie van Leeuwenhoek*, 81(1–4), 549–556. DOI: [10.1023/A:1020553421740](https://doi.org/10.1023/A:1020553421740)
- [19] Gruber, S., & Seidl-Seiboth, V. (2012). Self versus non-self: Fungal cell wall degradation in *Trichoderma*. *Microbiology*, 158(1), 26–34. DOI: [10.1099/mic.0.052613-0](https://doi.org/10.1099/mic.0.052613-0)
- [20] Lorito, M., Woo, S. L., Fernandez, I. G., Colucci, G., Harman, G. E., Pintor-Toro, J. A., ... Scala, F. (1998). Genes from mycoparasitic fungi as a source for improving plant resistance to fungal pathogens. *Proceedings of the National Academy of Sciences*, 95(14), 7860–7865. DOI: [10.1073/pnas.95.14.7860](https://doi.org/10.1073/pnas.95.14.7860)
- [21] Ghisalberti, E. L., & Sivasithamparam, K. (1991). Antifungal antibiotics produced by *Trichoderma* spp. *Soil Biology and Biochemistry*, 23(11), 1011–1020. DOI: [10.1016/0038-0717\(91\)90036-J](https://doi.org/10.1016/0038-0717(91)90036-J)
- [22] Mukherjee, P. K., Horwitz, B. A., & Kenerley, C. M. (2012). Secondary metabolism in *Trichoderma*: A genomic perspective. *Microbiology*, 158(1), 35–45. DOI: [10.1099/mic.0.053629-0](https://doi.org/10.1099/mic.0.053629-0)

- [23] Vinale, F., Sivasithamparam, K., Ghisalberti, E. L., Marra, R., Woo, S. L., & Lorito, M. (2008). *Trichoderma*-plant-pathogen interactions. *Soil Biology and Biochemistry*, 40(1), 1-10. DOI: [10.1016/j.soilbio.2007.07.002](https://doi.org/10.1016/j.soilbio.2007.07.002)
- [24] Vinale, F., Sivasithamparam, K., Ghisalberti, E. L., Marra, R., Barbetti, M. J., Li, H., ... Lorito, M. (2008). A novel role for *Trichoderma* secondary metabolites in the interactions with plants. *Physiological and Molecular Plant Pathology*, 72(1-3), 80-86. DOI: [10.1016/j.pmpp.2008.05.005](https://doi.org/10.1016/j.pmpp.2008.05.005)
- [25] Howell, C. R. (2003). Mechanisms employed by *Trichoderma* species in the biological control of plant diseases: The history and evolution of current concepts. *Plant Disease*, 87(1), 4-10. DOI: [10.1094/PDIS.2003.87.1.4](https://doi.org/10.1094/PDIS.2003.87.1.4)
- [26] Papavizas, G. C. (1985). *Trichoderma* and *Gliocladium*: Biology, ecology, and potential for biocontrol. *Annual Review of Phytopathology*, 23, 23-54. DOI: [10.1146/annurev.py.23.090185.000323](https://doi.org/10.1146/annurev.py.23.090185.000323)
- [27] Yedidia, I., Benhamou, N., & Chet, I. (1999). Induction of defense responses in cucumber plants (*Cucumis sativus* L.) by the biocontrol agent *Trichoderma harzianum*. *Applied and Environmental Microbiology*, 65(3), 1061-1070. DOI: [10.1128/AEM.65.3.1061-1070.1999](https://doi.org/10.1128/AEM.65.3.1061-1070.1999)
- [28] Shores, M., Harman, G. E., & Mastouri, F. (2010). Induced systemic resistance and plant responses to fungal biocontrol agents. *Annual Review of Phytopathology*, 48, 21-43. DOI: [10.1146/annurev-phyto-073009-114450](https://doi.org/10.1146/annurev-phyto-073009-114450)
- [29] Pieterse, C. M. J., Zamioudis, C., Berendsen, R. L., Weller, D. M., Van Wees, S. C. M., & Bakker, P. A. H. M. (2014). Induced systemic resistance by beneficial microbes. *Annual Review of Phytopathology*, 52, 347-375. DOI: [10.1146/annurev-phyto-082712-102340](https://doi.org/10.1146/annurev-phyto-082712-102340)
- [30] Brotman, Y., Landau, U., Cuadros-Inostroza, A., ... Willmitzer, L. (2013). *Trichoderma*-plant root colonization: Escaping early plant defense responses and activation of the antioxidant machinery for saline stress tolerance. *PLoS Pathogens*, 9(3), e1003221. DOI: [10.1371/journal.ppat.1003221](https://doi.org/10.1371/journal.ppat.1003221)
- [31] Contreras-Cornejo, H. A., Macías-Rodríguez, L., Cortés-Penagos, C., & López-Bucio, J. (2009). *Trichoderma virens*, a plant beneficial fungus, enhances biomass production and promotes lateral root growth through an auxin-dependent mechanism in Arabidopsis. *Plant Physiology*, 149(3), 1579-1592. DOI: [10.1104/pp.108.130369](https://doi.org/10.1104/pp.108.130369)
- [32] Hermosa, R., Viterbo, A., Chet, I., & Monte, E. (2012). Plant-beneficial effects of *Trichoderma* and of its genes. *Microbiology*, 158(1), 17-25. DOI: [10.1099/mic.0.052274-0](https://doi.org/10.1099/mic.0.052274-0)
- [33] Doni, F., Isahak, A., Zain, C. R. C. M., & Yusoff, W. M. W. (2014). Physiological and growth response of rice plants (*Oryza sativa* L.) to *Trichoderma* spp. inoculants. *AMB Express*, 4, 45. DOI: [10.1186/s13568-014-0045-8](https://doi.org/10.1186/s13568-014-0045-8)
- [34] Mastouri, F., Björkman, T., & Harman, G. E. (2010). Seed treatment with *Trichoderma harzianum* alleviates biotic, abiotic, and physiological stresses in germinating seeds and seedlings. *Phytopathology*, 100(11), 1213-1221. DOI: [10.1094/PHYTO-03-10-0091](https://doi.org/10.1094/PHYTO-03-10-0091)
- [35] Harman, G. E. (2000). Myths and dogmas of biocontrol: Changes in perceptions derived from research on *Trichoderma harzianum* T-22. *Plant Disease*, 84(4), 377-393. DOI: [10.1094/PDIS.2000.84.4.377](https://doi.org/10.1094/PDIS.2000.84.4.377)
- [36] Whipps, J. M. (2001). Microbial interactions and biocontrol in the rhizosphere. *Journal of Experimental Botany*, 52(suppl_1), 487-511. DOI: [10.1093/jexbot/52.suppl_1.487](https://doi.org/10.1093/jexbot/52.suppl_1.487)
- [37] Bashan, Y., de-Bashan, L. E., Prabhu, S. R., & Hernandez, J.-P. (2014). Advances in plant growth-promoting bacterial inoculant technology: Formulations and practical perspectives (1998-2013). *Plant and Soil*, 378(1-2), 1-33. DOI: [10.1007/s11104-013-1956-x](https://doi.org/10.1007/s11104-013-1956-x)
- [38] Schisler, D. A., Slininger, P. J., Behle, R. W., & Jackson, M. A. (2004). Formulation of *Bacillus* spp. for biological control of plant diseases. *Phytopathology*, 94(11), 1267-1271. DOI: [10.1094/PHYTO.2004.94.11.1267](https://doi.org/10.1094/PHYTO.2004.94.11.1267)
- [39] Lorito, M., Woo, S. L., Harman, G. E., & Monte, E. (2010). Translational research on *Trichoderma*: From 'omics to the field. *Annual Review of Phytopathology*, 48, 395-417. DOI: [10.1146/annurev-phyto-073009-114314](https://doi.org/10.1146/annurev-phyto-073009-114314)
- [40] Köhl, J., Kolnaar, R., & Ravensberg, W. J. (2019). Mode of action of microbial biological control agents against plant diseases: Relevance beyond efficacy. *Frontiers in Plant Science*, 10, 845. DOI: [10.3389/fpls.2019.00845](https://doi.org/10.3389/fpls.2019.00845)
- [41] Hurlbert, S. H. (1984). Pseudoreplication and the design of ecological field experiments. *Ecological Monographs*, 54(2), 187-211. DOI: [10.2307/1942661](https://doi.org/10.2307/1942661)
- [42] Ioannidis, J. P. A. (2005). Why most published research findings are false. *PLoS Medicine*, 2(8), e124. DOI: [10.1371/journal.pmed.0020124](https://doi.org/10.1371/journal.pmed.0020124)
- [43] Cumming, G. (2014). The new statistics: Why and how. *Psychological Science*, 25(1), 7-29. DOI: [10.1177/0956797613504966](https://doi.org/10.1177/0956797613504966)
- [44] Elad, Y., Chet, I., & Henis, Y. (1981). Biological control of *Rhizoctonia solani* in strawberry fields by *Trichoderma harzianum*. *Plant and Soil*, 60(2), 245-254. DOI: [10.1007/BF02374109](https://doi.org/10.1007/BF02374109)
- [45] Nagarajkumar, M., Bhaskaran, R., & Velazhahan, R. (2004). Involvement of secondary metabolites and extracellular lytic enzymes produced by *Pseudomonas fluorescens* in inhibition of *Rhizoctonia solani*, the rice sheath blight pathogen. *Microbiological Research*, 159(1), 73-81. DOI: [10.1016/j.micres.2004.01.005](https://doi.org/10.1016/j.micres.2004.01.005)