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Lupins: A Remarkable Protein Crop Battling Anthracnose for a Greener Future

Lupins (*Lupinus* spp.), protein-rich grain-legumes that are beneficial for sustainable agriculture, face threats from anthracnose disease caused by *Colletotrichum lupini*. This case study focuses on pathogen diversity, evolution and lifecycle and discusses best practices to improve disease management and crop resilience.

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Abstract

Lupins (*Lupinus* spp.) are protein-rich legumes, serving as a sustainable alternative to soybeans and valued for both human and animal nutrition. Besides their high-yield potential, lupins enhance soil fertility and provide habitat for wild pollinators, boosting agrobiodiversity. However, lupin cultivation faces challenges from anthracnose disease, caused by the fungal pathogen *Colletotrichum lupini*. This presumed asexual pathogen consists of at least four clonal lineages, all originating from the Andes in South America. Lineage II has spread globally and drives the current anthracnose outbreak. Despite genetic uniformity, variations in morphology and virulence exist among and within lineages, likely due to a diverse transposable element (TE) landscape, mini-chromosome presence, and distinct effector repertoires. Infected seeds are the primary inoculum source and the main vehicle for global spread. Secondary inoculum in the form of conidia produced by necrotic lesions, becomes prevalent at flowering, causing rapid crop devastation. Resistance breeding has proven to be the most effective control strategy, with recently developed resistant varieties now available for the most widely cultivated lupin species. Combining resistance breeding with enhanced disease management and agronomic practices can support the expansion of lupins as a resilient protein crop, strengthening their role in sustainable agriculture.

Learning Outcomes

1. Explain the disease cycle and infection process of *Colletotrichum lupini*.
2. Discuss *C. lupini* diversity and evolution.
3. Understand lupin resistance breeding efforts.
4. List disease management strategies.
5. Recognise future prospects of lupin cultivation.

Introduction

Lupins (*Lupinus* spp.) are grain-legumes with a high-protein content comparable to that of soybeans, making them a valuable source for both animal and human nutrition, and attracting increasing attention for their use in cosmetics. Beyond their high-yield potential, lupins play a vital role in enriching soil health and fostering biodiversity. Through an exceptionally efficient nitrogen-fixing symbiosis with *Bradyrhizobium lupini*, they improve soil fertility and reduce the need for synthetic inputs. Additionally, lupins provide a rich pollen source for wild bees, providing habitats for pollinators and enhancing biodiversity across

agricultural landscapes. Four species, Andean (*L. mutabilis*), narrow-leaved or blue (*L. angustifolius*), yellow (*L. luteus*) and white (*L. albus*) lupin, are of agricultural importance. White, blue and yellow originate from the Mediterranean region, where white lupin was used already in ancient Greece and Egypt (3000 BP). Andean lupin originates from South America and has been domesticated in the Andes at least 1800 BP. Most lupin species are rich in alkaloids, making them toxic and bitter, and requiring extensive washing to remove these compounds. However, modern blue and white lupin varieties have been bred to be low in alkaloids. Notably, this reduction in alkaloid content has not been associated with increased disease susceptibility (Schwertfirm *et al*., 2024). Today, about 950,000 ha globally are dedicated to lupin cultivation, yielding roughly 1.6 million tons of grain annually (Fig. 1; FAOSTAT, 2021). Australia leads in production, accounting for an impressive 58% of the world's supply. Historically, lupins were widely cultivated across Europe in the early 20th century. However, with rising soybean imports, fluctuating yields, and disease outbreaks, European lupin production dwindled after the 1960s. Now, this versatile crop is making a comeback in Europe, which now contributes around 35% of global production. This resurgence in lupin production aligns with the European Union's push towards greater self-sufficiency in grain legume crops over the next decade. Recognized as an underutilized grain-legume with high potential, lupins are attracting new interest as a sustainable crop to meet the growing demand for plant-based proteins while supporting agroecosystem health. Yet, the full potential of lupins is threatened by the persistent challenge of anthracnose disease, caused by the fungal pathogen *Colletotrichum lupini*.

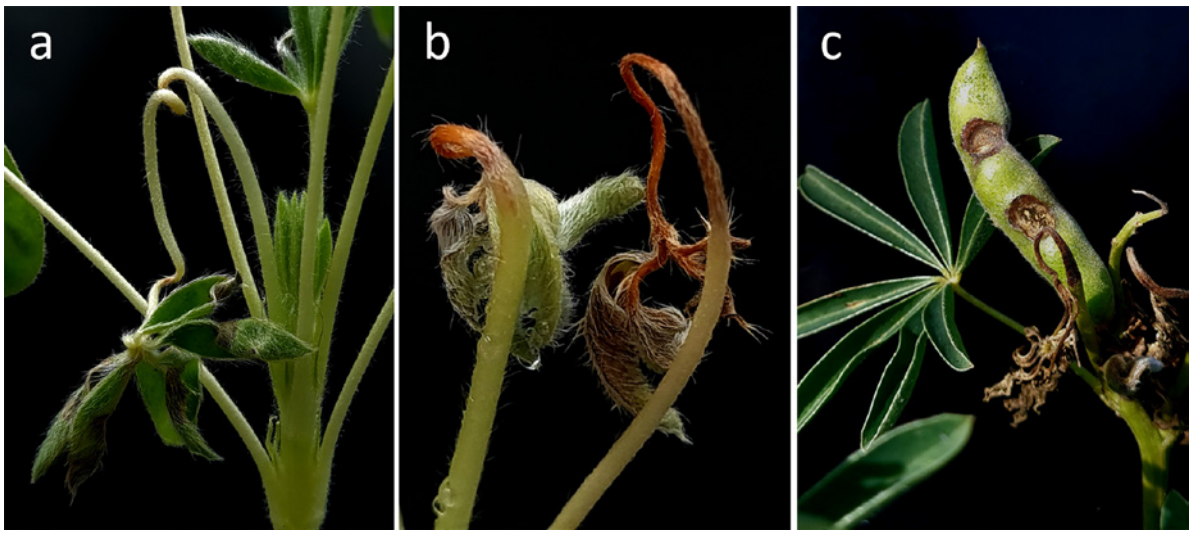

Fig. 1. Typical disease symptoms of anthracnose disease on white lupin. (a) typical early symptom (biotrophy) of petiole twisting (artificially inoculated plant 5 days post-inoculation), (b) typical late symptom (necrotrophy) of sporulating lesion and dead tissues (artificially inoculated plant 14 days post-inoculation), and (c) late symptom (necrotrophy) of sporulating lesion on a pod and dead tissues (natural infection in field conditions).

Current Status and Diversity of *C. lupini*

The fungal genus *Colletotrichum* comprises at least 15 species complexes and includes many important plant pathogens that cause anthracnose in a wide range of hosts. *Colletotrichum* is presumed to be mostly asexual and for only a few species sexual reproduction has been observed or suggested. Despite the rarity of sexual recombination, *Colletotrichum* species are highly diverse in both lifestyle and host-range. The *C. acutatum* species complex is particularly notorious as it contains many devastating plant pathogens. Within this species complex, *C. lupini* is grouped within clade 1 and is highly host-specific on lupins.

Lupin anthracnose was first reported in 1912 in Brazil, and the fungal pathogen was described as *C. gloeosporioides* in 1939, then as *C. acutatum* in 1994. In 2002, it was reclassified as a new species, *C. lupini*, and two different variants were described (groups I and II). A major outbreak occurred in the 1940s–1950s, primarily in North America, likely caused by group I strains. A second more severe epidemic emerged around the 1970s and persists until today (Talhinhas *et al*., 2016). At first, the disease mostly thrived in humid areas and coincided with a decline in lupin cultivation in Europe. However, it has since spread worldwide, from the United States to China and even impacting Australia's lupin crops. This rapid spread is largely attributed to the trade of symptomless, infected seeds, as even minimal levels of seed infection (0.01–0.1%) can cause substantial yield losses.

Worldwide sampling and genetic diversity analysis through single nucleotide polymorphisms (SNPs) showed that *C. lupini* consists of at least four clonal lineages (I–IV) and likely originates from South America (Fig. 1; Alkemade *et al*. (2023)). One lineage (II) has spread globally and is the cause of the current outbreak. Although genetic diversity within each lineage is very low, isolates display varied morphology and virulence. Whole genome sequencing (WGS) of a globally representative set of isolates uncovered a highly variable mini-chromosome present in lineages II, III, and IV, but absent in lineage I (Alkemade *et al*., 2024). Transposable elements (TE) were shown to be the main driver of genome expansion within the species, with each lineage displaying a distinct TE landscape. Genome size, gene content, TEs, and deletions revealed that lineage II could be split into two groups, II-A and II-B, further explaining observed diversity. The presence/absence variation of putative effectors appeared to be lineage-specific, suggesting that these genes play a crucial role in determining host range. Notably, no effectors were found on the mini chromosome. These findings suggest that, despite *C. lupini*'s largely asexual nature, it can still generate genetic diversity to adapt to changing environments and potentially overcome disease management strategies.

Disease Cycle and Infection Process of *C. lupini*

Infected seeds, which can appear healthy, are the main source of primary inoculum. Healthy looking seeds are generally less infected and have a slower disease development than severely infected seeds which suffer from bad germination and early more severe disease development. Upon seed germination, *C. lupini* remains dormant or grows endophytically within the lupin seedlings, not causing immediate harm. As the plant matures, typically around the flowering stage, the pathogen shifts to a biotrophic phase, during which it derives nutrients from living plant cells without killing them. This is quickly followed by a necrotrophic stage in which secondary infection hyphae invade and kill adjacent cells, leading to the classic symptoms of lupin anthracnose: twisting and bending of stems, along with necrotic lesions on stems and pods showing orange masses of conidia (Fig. 2). These conidia serve as the pathogen's secondary inoculum, rapidly spreading the disease within the crop. Dispersed by rain splash or wind, conidia landing on lupin tissue can germinate and produce appressoria, a specialized structure for host penetration, within 24 h (Figs. 3 and 4). Upon infection, *C. lupini* can switch from biotrophy to necrotrophy within 4 days post-inoculation, highlighting the speed of the infection.

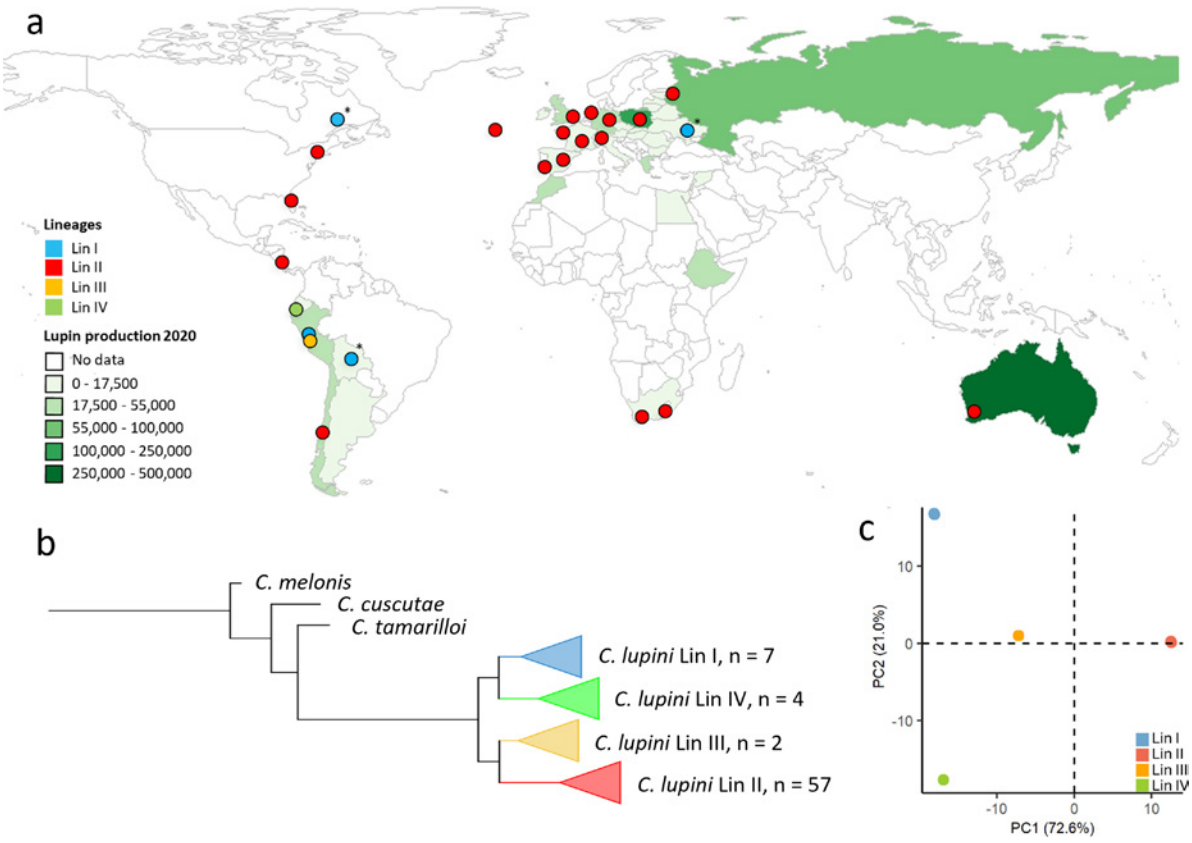

Fig. 2. Global sampling and diversity of *Colletotrichum lupini*. (a) sampling regions and lupin production in tonnes of product (FAOSTAT, 2021), asterisks indicate isolates from before 1990, (b) phylogeny based on 9923 SNPs, and (c) principal component analysis within *C. lupini* based on 1863 SNPs and 70 isolates, based on data from Alkemade *et al*. (2023).

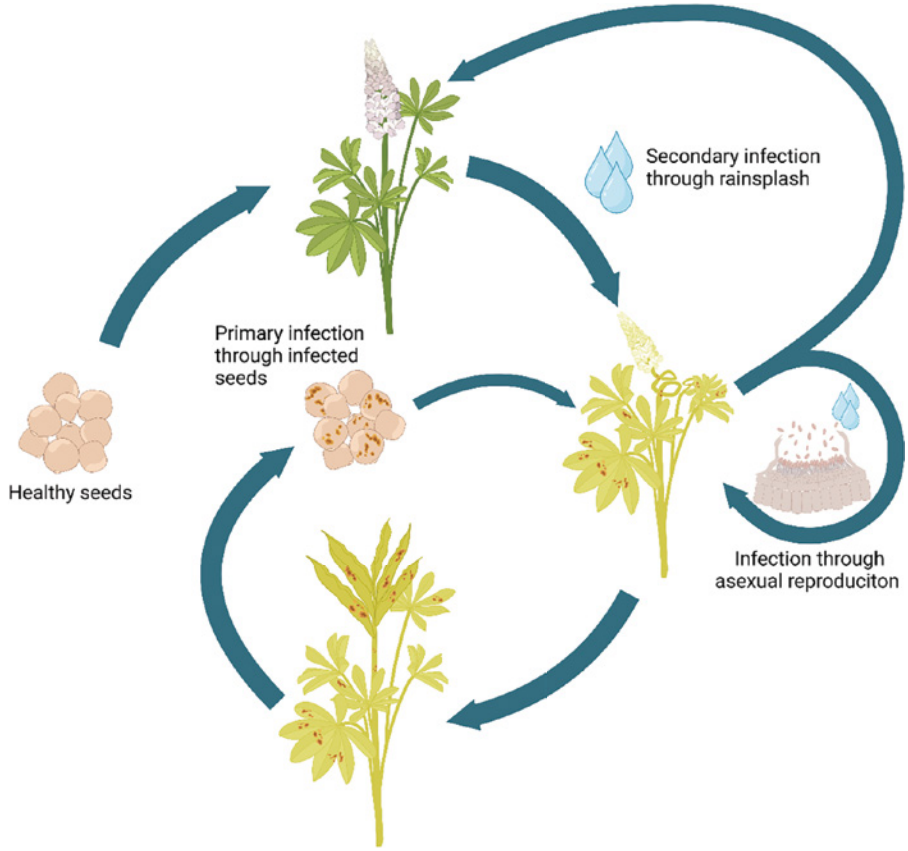

Fig. 3. Disease cycle of *Colletotrichum lupini*. Anthracnose inoculum originates from infected seeds (primary infection) or the introduction of spores through rain splash (secondary infection). Followed by a quick progression of biotrophy to necrotrophy. The pathogen develops acervuli containing asexually produced conidia which allows the spread of the infection throughout the whole growing season.

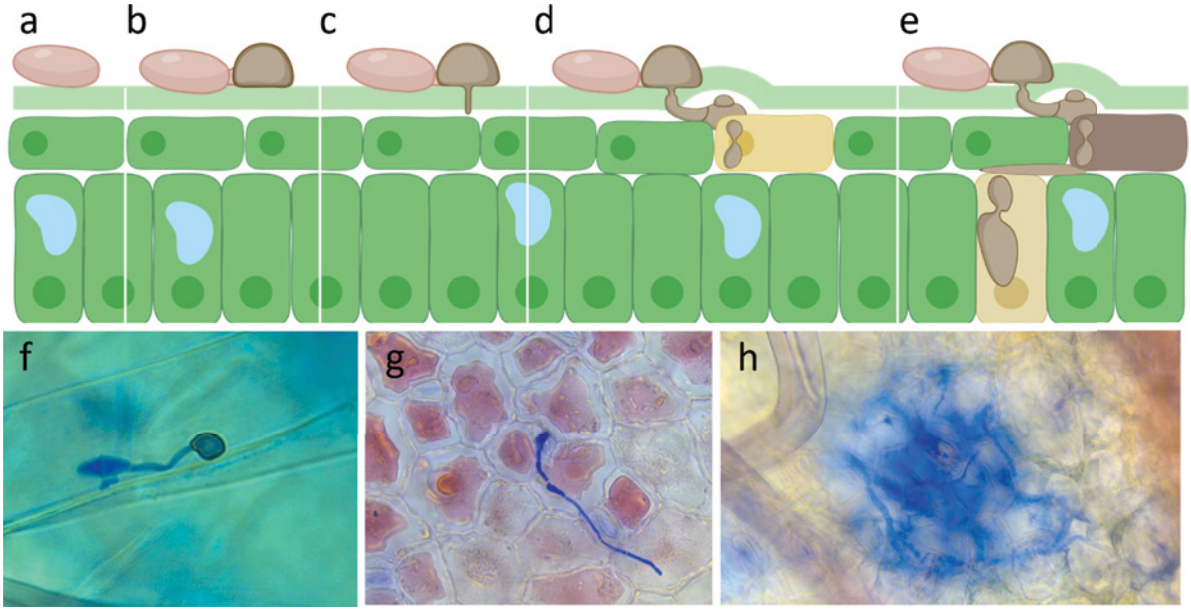

Fig. 4. Infection process of *Colletotrichum lupini*. After adhesion on any aerial part of the host plant (a), spores germinate quickly and produce melanized appressoria (b) which later form a penetration peg (1-day post-inoculation, c and f). After penetration (d and g), the fungus grows initially subcuticular intramural hyphae and later penetrates the epidermal cells entering the biotrophic phase, dissolving cell walls and cytoplasmic content with adjacent cells appearing intact (up to 4 days post-inoculation). This short biotrophic phase is followed by necrotrophy (e and h) with both intra- and extracellular hyphae, killing infected host cells (from 4 days post-inoculation). Finally, from 6 days post-inoculation acervuli are formed, disrupting the host cuticle and appear as dark sunken lesions (not shown in figure). Adapted from Guilengue *et al*. (2022).

Lupin Resistance Breeding Efforts

Significant progress has been made in breeding lupins for disease-resistant varieties over the past decades. Several single dominant resistance genes have been identified, particularly in narrow-leaved and yellow lupin. In narrow-leaved lupin, highly resistant varieties like 'Tanjil' (carrying the R gene *Lanr1*) and 'Mandelup' (with the R gene *AnMan*) are now available to farmers (Yang *et al*., 2008). Yellow lupin exhibits high genetic synteny with narrow-leaved lupin, and the *Lanr1* homologue in the yellow lupin cultivar 'Core 98' provides strong resistance (Lichtin *et al*., 2020). Recently, another resistance gene, *Llur*, was identified in the yellow lupin variety 'Taper'. In contrast, resistance in white lupin appears to be polygenic. Extensive screenings of genetic resources across different growing regions have identified only a few accessions with enhanced resistance, indicating a complex resistance mechanism. However, new cultivars like 'Frieda' and 'Celina,' which exhibit improved resistance to *C*. *lupini*, are now available for cultivation. Several new resistance loci have recently been identified, providing a foundation for marker-assisted selection, which could further accelerate breeding efforts (Książkiewicz *et al*., 2017, Alkemade *et al*., 2022b, Schwertfirm *et al*., 2024).

Most breeding efforts took place in Europe and Australia where only *C*. *lupini* lineage II is present. As *C*. *lupini* diversity in South America remains mostly uncharted, there is a high chance that strains from this region could break recently developed resistance. For example, a lineage II strain from Chile was shown to be significantly more virulent on white lupin than other aggressive lineage II strains. Andean lupin has likely co-evolved with *C*. *lupini* and shows a great variety in disease phenotypes against the various South American strains. This diversity in Andean lupins remains underexplored and holds considerable potential for discovering new resistance genes, offering valuable resources for future breeding programs aimed at more robust and durable resistance.

Disease Management Strategies

Starting with clean seeds is crucial to managing anthracnose disease. However, because infected seeds often appear healthy externally and low concentrations (0.01%) of infection can already cause disease, systematic seed testing is essential to effectively control the spread and intensity of the disease. Seed testing can be done through seed germination assays but more recently a qPCR method has been developed to detect *C*. *lupini* in seed lots (Kamber *et al*., 2021). Although these testing methods provide a solid foundation for seed certification programs, no such system is currently in place. Seed disinfection, while challenging, has shown some promise as well. For instance, field trials demonstrated that treating seeds with vinegar significantly reduced disease incidence (Alkemade *et al*., 2022a), though further validation is needed before use at field scale. Seed dressing with the fungicide thiram has proven effective as well, but its active compound is no longer authorized within the European Union. For foliar applications, which are most effective during flowering, broad-spectrum fungicides with metconazole were shown to reduce the disease. These fungicides, however, are considered problematic due to their environmental impact and are unsuitable for organic cultivation systems. Given the patchy spread of lupin anthracnose, row-cropping or other intercropping strategies could potentially be highly effective to control the disease. Especially white lupin would be well-suited for this strategy due to its cluster roots, which actively improve phosphorus availability in the soil. However, mixed cropping with oats did not show disease reduction in field trials in Switzerland, and there are currently no documented cases of intercropping cases in which lupin anthracnose was successfully controlled. Due to the limited success and disadvantages of fungicide treatments and agronomic practices, resistance breeding remains the most promising approach to control lupin anthracnose. In a 4-year trial at two high disease pressure sites in Switzerland, varieties with enhanced resistance consistently yielded nearly twice as much as susceptible varieties, underscoring the potential of breeding efforts to improve disease management and ensure stable yields.

Future Prospect of Lupin Cultivation

As a high-quality protein crop, lupins have great potential to outperform soybeans in import regions such as Europe while enhancing resilience and agrobiodiversity. With their efficient nitrogen fixation, lupins also make an excellent fallow crop, enriching soil fertility and reducing the need for synthetic fertilizers. Advances in breeding and disease management over the recent decades indicate that achieving sustainable lupin cultivation is within our reach. By incorporating unexplored resistance sources and deepening our

understanding of *C. lupini* evolution and population dynamics, anthracnose disease could be controlled in the near future. Besides anthracnose, breeding should focus on (i) low alkaloid content, (ii) resistance to other fungal pathogens such as *Pleiochaeta setosa* (Brown spot), *Septoria glycines* (Septoria brown spot), *Fusarium oxysporum* (Fusarium wilt) and *Diaporthe toxica* (Phomopsis stem and pod blight), and (iii) resistance to key viral diseases such as bean yellow mosaic virus (BYMV) and cucumber mosaic virus (CMV; Wolko *et al*., 2011). Especially the fungal pathogen *D. toxica* is notorious as it produces mycotoxins affecting the liver when consumed. Overcoming these challenges would further accelerate global lupin production and disease control. Thereby adding a sustainable, yet underutilized crop to our agricultural systems, contributing to a more resilient and diverse food supply.

Discussion Points

1. How can better knowledge of the *C. lupini* disease cycle and infection process help resistance breeding efforts?
2. How to monitor *C. lupini* diversity and virulence to minimise the risk of lupin resistance breaking?
3. How can breeding efforts be best complemented with other treatments and agronomic practices to minimise anthracnose disease?
4. How to combine resistance breeding with selection for other key traits, e.g. early ripening time, low alkaloid (bitterness) content and other nutritional traits?

Conflict of interest

The authors have no conflicts of interest to declare.

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Further Reading

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