國立臺灣大學生物資源暨農學院森林環境暨資源學系

碩士論文

School of Forestry and Resource Conservation
College of Bioresources and Agriculture
National Taiwan University

Master Thesis

以生態模式探討植物性狀與土壤微生物組成對植物土壤回饋之種間差異的影響

Plant Trait and Microbial Composition Interactively Determine Species Variation in Plant Soil Feedback —a Modeling Approach

柯柏如

Po-Ju Ke

指導教授：三木健 博士 Takeshi Miki, Ph. D

丁宗蘇 博士 Tzung-Su Ding, Ph. D

中華民國 102 年 6 月

June 2013

Acknowledgement

感謝我的指導老師，丁宗蘇老師與三木健老師。感謝兩位老師總是讓我自由地決定研究的方向，讓我有學習獨立研究的機會。特別感謝三木健老師在我大三的時候帶我進入理論生態的領域，以及你一路以來投注的時間與心力。感謝兩位老師總是不計回報地提供各種資源，讓一個碩士生有機會能出國那麼多次。感謝兩位老師在很多細微的地方給予提省與叮嚀，時常細微到讓我為自己的不獨立感到不好意思。兩位老師和善的待人處事、身為科學家的嚴謹態度、以及在研究與生活間的比例拿捏是我的學習典範。

感謝口試委員謝志豪老師、林宜靜老師、林雨德老師，感謝你們願意花時間閱讀論文，並從不同的面向提省我其中的不足。感謝關秉宗老師以及奧山利規老師在大學時教我統計跟R，讓我在研究所不再為此感到害怕。感謝久米朋宣老師、鍾國芳老師在各方面的協助與鼓勵。

感謝海洋所的大家。感謝這段時間一起討論的 403 學長姐們:貞儀、珮綺、蔡政翰以及其羽學長。感謝佑青、貓、Cot、Oscar、怡君、冠婷、煒忠、拉狗、梵絃、蘇民弦、Cirk、Eric、穎萱、恩諭、昭成、Carmen 及俊偉這段時間的照顧以及為生活帶來的歡樂，讓在海洋所短期居留的我不至於太過孤單。感謝謝志豪老師與王慧瑜老師，感謝你們願意讓一位到現在還是不太懂海洋的人參加meeting，並總是很和善地給予很好的意見。特別感謝 Take，總是私下教我很多小技巧並給予鼓勵。

感謝森林系的大家。感謝研究所這段時間一起努力的同學:許皓、舜雲、宗佑、樂寧、書亞、宜芳、瀚嶢。感謝所有因為森林系而結識的你們：小明、趙容、筠雯、阿嵐、王宇笙、阿京、小斑、老皮、阿德、忠慧、烜駿。特別感謝紅葳、小乃、小管以及 living，感謝你們總是讓我有抱怨和吐嘈的地方。雖然我從以前就一直在嗆你們以及森林系的總總，但森林系是家，就算我後來比較常待在海洋所，森林系仍是唯一一個能讓我感到安心與放鬆的地方。

感謝游旨价。要不是在北大武山上的你，以及我鳥隊羊頭山後在台北山水巧遇的你，我絕對不會走上理論生態這條路。感謝林瑋庭，和你的談話總是減緩我在海洋所的生活壓力。感謝小苗學姐，和你們的認識真的是緣分。

感謝 401 的大家，特別感謝田婕、Devil、Elaine、佩蓉、阿霞、正宇、妙蓉，感謝你們總是願意和我討論學術與八卦，抱歉我後來好少去 401，但我一直十分想念那風光明媚的地方。謝謝森多概的助教群與學生，雖然我偷懶只帶了一年，但因為你們，我才可以在生態模擬的同時，仍然不忘對大自然的熱情。在念研究所的這一兩年間，我發現這是一件無比重要的事。

感謝蔡昀真。謝謝你這幾年來的陪伴，感謝你總是用自己的包容換取我工作的時間，我很珍惜這得來不易的緣分。你總是樂觀地回應人生中的挫敗，並在極度繁忙的日子中享受生活，這樣的人生態度是我永遠的學習對象。

最後，感謝我的家人。讓我從小就可以選擇自己想念的科系。雖然我總是很任性地選了最讓人擔心，而且投資報酬率最低的選項，但感謝你們仍然一路支持到現在。

Abstract

Interaction of plants with the nearby soil environment, a process termed plant-soil feedback (PSF), is a structuring force for vegetation development. Understanding how plant functional traits control PSF strength variation among species is thus critical for plant community ecology. Studies have highlighted either nutrient cycling (litter-mediated PSF) or soil biota (microbial-mediated PSF) separately as two main drivers of PSF and thus focus on different sets of plant traits. However, the two PSF drivers are not independent and their way of interaction depends on the functional type of microbes (i.e. pathogens and mycorrhizas). An ecosystem model coupling indirect interaction between litter and microbial feedback is presented to identify which traits have strongest effect on PSF strength and, its dependence on soil microbial community composition. This model shows that the identity of the most influential plant functional traits alters when microbial-mediated PSF is considered along with litter-mediated PSF. The relative importance of traits depends on microbial composition. In particular, the importance of litter decomposability increases with the relative abundance of mycorrhizas due to its indirect positive effects on litter production. Plants with more easily-decomposable litter and with more beneficial plant-mycorrhiza associations are more advantageous than other plants species in pathogen-free soils. On the other hand, plants with better

defense traits are expected to be dominant in pathogen-rich soils. The results can provide useful insights into understanding the key determinants of successful plant invasion in different soil environments.

Keywords

functional trait-based ecology, litter decomposition, mycorrhiza, soil pathogen, indirect interaction, population stage structure, exotic plant invasion

中文摘要

植物與土壤間交互作用所產生的回饋現象(植物土壤回饋)會改變植群發展。因此，了解植物功能性狀如何調控植物土壤回饋強度之種間變異為植群研究的重要議題。過去研究只探討枯落物養分循環或土壤微生物之單一因子所造成之回饋現象。然而這兩項因子並非獨立，分開討論會忽略枯落物與微生物間的間接交互作用。此外，過去研究忽略了不同的微生物會透過不同的機制影響枯落物動態。本研究建立一個生態模式，同時納入枯落物循環與土壤微生物對植物土壤回饋的影響，以了解植物功能性狀如何調控植物土壤回饋強度，並探討土壤微生物群聚組成如何影響不同功能性狀的相對重要性。結果顯示當把土壤微生物的影響納入枯落物回饋的模式時，影響力較強的功能性狀差異相當大。此外，土壤微生物群聚組成會影響功能性狀的相對重要性，其中枯落物分解速率的重要性會隨著菌根菌的豐度增加而顯著上升。此模式預測在沒有土媒病害的土壤中，易分解及會與菌根菌形成高效益互利共生的植物佔有生長優勢；而當土媒病害豐度高時，具有較佳防禦策略的植物會佔優勢。本研究結果可應用於了解功能性狀如何在不同土壤環境中影響外來種的入侵。

關鍵字

功能性狀生態學、枯落物分解、菌根菌、土媒病害、間接交互作用、族群層級結構、外來植物入侵

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Introduction

Understanding the processes controlling community structure and the influence of functional traits of organisms on community characteristics through these processes is a central question for trait-based ecology (McGill *et al.*, 2006). While many studies have discussed the importance of plant functional traits (e.g. leaf economics, wood density) in determining aboveground species interaction and ecosystem processes in terrestrial ecosystems (Wright *et al.*, 2004; Chave *et al.*, 2009; de Bello *et al.*, 2010; Kunstler *et al.*, 2012), the plant traits that affect the interaction between plants and soil, a process termed plant-soil feedback (PSF), are also found important to plant community structure (Bever *et al.*, 2010; De Vries *et al.*, 2012). Plants with different traits are able to cultivate nearby soil environment differently, causing changes in the physical, chemical and biological properties of the soil, and eventually influence future performance of these same individuals or other individuals that grow nearby (Bever *et al.*, 1997; Ehrenfeld *et al.*, 2005). The direction and strength of such PSF can be determined by the relative growth rate of plants in soils with different cultivation history. When a plant changes the soil environment in a direction such that growth response of conspecific plants are larger (or smaller) compared to those individuals that were grown in heterospecific-cultured soils, the feedback is defined as positive (or negative) PSF (Bever *et al.*, 1997; Brinkman *et al.*, 2010).

Past empirical studies have revealed that PSF processes are ubiquitous in various ecosystems, ranging from temperate grassland (Klironomos, 2002), temperate forest (Packer and Clay, 2002), to tropical rainforest (Bell *et al.*, 2006; Mangan *et al.*, 2010). Empirical studies have also showed that species vary greatly in realized PSF strength, and suggested that such variation acts as a structuring force of plant community (Bever *et al.*, 2011; van der Putten *et al.*, 2013 and reference therein). PSF can change plant community composition by accelerating species replacement, which is the major driving force of succession in sand dune and grasslands (van der Putten and Peters, 1997; Kardol *et al.*, 2006; Kardol *et al.*, 2007). Other studies in temperate grasslands and tropical forests (Klironomos, 2002; Mangan *et al.*, 2010) showed that rare species suffer stronger negative PSF, implying that variation in PSF strength is a key to explain rarity of some species and thus have the power to shape community relative abundance. Moreover, there has been increasing recognition that PSF mediates the success of exotic plant invasion (Reinhart & Callaway, 2006). Invasive species often experience more positive PSF in their introduced range compared to that in their native range by leaving their belowground natural enemies behind (i.e. enemy escape hypothesis, Callaway *et al.*, 2004). Native species in the invaded area, however, often received stronger negative PSF after invasion processes due to altered nutrient cycling (Eppinga *et al.*, 2011) or accumulation of pathogens which have stronger negative

impact on natives (i.e. pathogen accumulation hypothesis, Eppinga *et al.*, 2006). These examples all show that PSF have the ability to shape vegetation properties such as community composition and resilience against perturbations (Miki & Kondoh, 2002; Miki *et al*. 2010). Given the importance of PSF in vegetation development, understanding what functional traits control species variation in PSF strength is a critical, but remain unsolved issue, for plant community ecology (van der Putten *et al.*, 2013).

While many mechanisms can generate PSF, the importance of nutrient cycling (i.e. litter-mediated PSF) and soil biota (i.e. microbial-mediated PSF) were most highlighted in past studies. Litter-mediated PSF considers the indirect interaction between plant and soil chemistry through litter dynamics, emphasizing the role of species-specific litter traits in controlling local nutrient cycling process through litter quantity (e.g. litter production rate) and quality (e.g. litter carbon: nitrogen (C:N) ratio, secondary compound concentration, Binley & Giardina, 1998). Litter feedback studies often suggest that the direction and strength of PSF depends on the litter decomposability of nearby trees (Berendse, 1994; Miki & Kondoh, 2002; Eppinga *et al.*, 2011). For example, species may create positive PSF by enhancing nutrient cycling through the production of quickly decomposing litter (Berendse, 1994; Miki & Kondoh, 2002), while negative PSF may be realized due to soil nutrient depletion

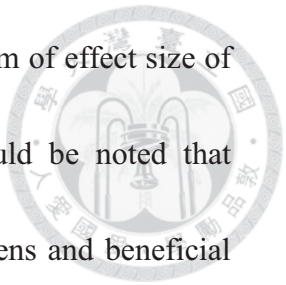
by other individuals or release of phytotoxic compounds during decomposition (Mazzoleni *et al.*, 2007).

The other well-documented mechanism is termed microbial-mediated PSF, which investigates the direct interactions between plants and soil organisms. Such studies emphasize the well-established observation that plants differ in their locally associated microbial community (e.g. due to difference in root exudation and architecture), and their response to individual microbial species (Bever *et al.*, 2011). Bever *et al* (1997) summarized past evidences and synthesized the microbial-mediated PSF model, which viewed the local soil community as a whole to have either net positive or negative effects on local plant performance (Bever *et al.*, 1997). Follwoing this framework, microbial-mediated PSF studies concluded that the sum of effect of each microbial group on plant performance is the most important determinant of PSF strength. Positive PSF is thus believed to occur when the plant facilitates population growth of beneficial microbes (e.g. mycorrhizal fungi and nitrifying bacteria) more than detrimental microbes (e.g. soil-borne pathogens and nematodes) during cultivation, while PSF will be negative if the impacts of detrimental microbes overwhelm that of beneficial organisms (Bever *et al.*, 1997; Kulmatiski *et al.*, 2011).

Although litter- and microbial-mediated PSF have been both widely documented,

some key properties of the PSF process are often neglected and thus hinder progress towards characterizing the functional traits that act as major determinants of interspecific variation in PSF strength. First, litter- and microbial-mediated PSF are not independent, but have strong indirect interaction between litter and soil microbes and thus affect plant performance simultaneously. Soil microbes can cause PSF directly by affecting plant population dynamics (e.g. influencing mortality and reproduction), and such process will also indirectly influence nutrient cycling through controlling litter input and nutrient uptake of plants (van der Heijden *et al.*, 2008). Litter dynamics, similarly, will influence microbial-mediated PSF since their effects on plant primary production will also influence plant-microbe interactions (Wardle, 2006). In addition, experimental studies often show that the direction and strength of PSF caused by soil microbes will change with soil nutrient conditions (de Dyne *et al.*, 2004; Manning *et al.*, 2008). This line of evidence also suggests that litter- and microbial-mediated PSF are not independent. A single plant trait can thus influence the direction and strength of PSF through both mechanisms simultaneously. Thus, the relative importance of traits in controlling PSF strength remains unclear unless effects of single trait on both litter- and microbial-mediated mechanisms are considered.

Moreover, viewing the soil community as a black box neglects the complex nature of indirect interaction between litter and microbes. While it is often assumed



that the net effect of local soil community on plant growth is the sum of effect size of each microbe when discussing plant-microbe interaction, it should be noted that microbes from different functional groups (e.g. detrimental pathogens and beneficial mycorrhizas) may indirectly influence litter dynamics through different processes. Pathogens interact with litter dynamics via inducing additional plant mortality since dead plant materials becomes litter, while mycorrhizal fungi operate through helping the plant to deplete soil nutrient, which can increase both plant productivity and litter production (Read & Perez-Moreno, 2003; Orwin *et al.*, 2011). Litter-mediated nutrient cycling also indirectly affects pathogens and mycorrhizas differently. An increase in soil nutrient content and plant biomass due to faster decomposition may support larger population size of soil-borne pathogens, but may alter plant-mycorrhiza interactions due to shifts in nutrient limiting status of the microbes (Wallander, 1995; Treseder, 2004; Johnson, 2009). With these lines of empirical evidence, studies need (1) to incorporate indirect interaction between litter and microbes via combining the two mechanisms, and (2) to separate microbial functional groups for better understanding of the relative importance of traits in determining PSF strength under different microbial composition.

When combining litter- and microbial-mediated PSF for different microbial groups, it is important to note that individuals of different stage classes within the

plant population interact with PSF drivers differently and thus their effects on soil properties are not identical. Seedlings and adults differ in their contribution to litter production and therefore may play different roles in litter feedback. In particular, adults can produce litter through tissue turnover (i.e. annual litter production) in addition to mortality (Clark *et al.*, 2001), while seedlings only contribute to the litter pool when individuals die. Seedlings are thus passively influenced by soil nutrient status which is mainly controlled by adults. Seedlings and adults also differ in their interactions with soil microbes. Seedlings are highly vulnerable to detrimental pathogens, while adults suffer less pathogen-induced mortality due to their better protected roots (Augspurger & Kelly, 1984; Alvarez-Loayza & Terborgh, 2011). In the case of beneficial mycorrhizal fungi, a larger percentage of fungal hyphae are associated with adults since they can provide more resources to the carbon limited microbes (Šmilauerová *et al*., 2012), while seedlings usually depend on mycorrhizal network which is mainly supported by adults (Dickie *et al.*, 2005).

Although empirical studies had detected large variation in PSF strength, it may be difficult to experimentally quantify the individual contribution of each trait since traits are often correlated and thus growth response measured in empirical studies cannot reveal their relative importance. Modeling is useful to approach this problem. Here, I present a stage-structured open ecosystem PSF model that couples both litter-

and microbial-mediated PSF. My main purpose is to identify which plant traits have the strongest effects on PSF strength when both litter- and microbial- mediated feedbacks are incorporated, and to elucidate how microbial community composition and stage structure of plants influence the relative importance of traits. In the model, I separate plant population as seedlings and adults, and model dynamics of litter and soil nitrogen to incorporate litter-mediated PSF. I consider two distinct groups of microbes: detrimental soil-borne pathogens and beneficial mycorrhizal fungi. I highlight these two groups of soil microbes because they have intense direct interactions with plant roots and may represent the positive and negative extremes of plant-microbe interaction, also because these two microbial groups are associated with most plant species and are abundant in soils of all ecosystems. I showed that the identity of the most influential plant functional traits alters when microbial-mediated PSF is considered along with litter-mediated PSF. The predicted relative importance of traits is different for different microbial functional groups. In particular, the importance of litter decomposability increases with the relative abundance of mycorrhizas in the whole microbial community. The results provide insights into understanding the key deternimant of success of exotic plant invasion and restoration, and can give better predictions of plant growth response in different soil environments.

Materials and Methods

Model Description

I developed a stage-structured model of PSF with six state variables in an open ecosystem to explore the effect of different plant traits on PSF strength via coupling litter and microbial dynamics. The model includes two stages of plants: seedlings and adults (with densities indicated by S and A, respectively). I included litter (L) and soil nitrogen content (R) for describing litter feedback mechanisms, while pathogen (P) and mycorrhiza nitrogen content (M) are included to represent two distinct functional groups for microbial feedback mechanisms. Major fluxes of the model are shown in Fig. 1, model equations are shown in Table 1 and 2.

Seedling and adult demographic dynamics

I divided plant individuals into seedlings and adults to emphasize the differences in litter contribution and interactions with microbes between the two stages. Density of seedlings increases with reproduction from adults. The rate of reproduction per adult is proportional to its soil nitrogen uptake rate rR (where r represents species-specific reproduction rate per nitrogen uptake). The density of adults increases with seedling maturation into the adult stage via nitrogen uptake for biomass growth (with biomass growth rate per nitrogen uptake g). I consider a negative shading

effect of adults on plant demography. Adult reproduction rate and seedling maturation rate decreases linearly with increasing adult density, represented by $rR \cdot (1 - \frac{A}{A_{\max}})$ and $gR \cdot (1 - \frac{A}{A_{\max}})$ respectively, where $A_{\max}$ represents the maximum adult density in the model ecosystem (Eqn.1 and 2 in Table 1). Plants are assumed to die with a density independent mortality rate (m_S and m_A for seedlings and adults, respectively) or due to pathogen-induced mortality rate $\alpha_S P$ and $\alpha_A P$ (with infection efficiency α_S and α_A for seedlings and adults, respectively).

Mycorrhiza and mycorrhizal-enhancement of plant growth

I assume mycorrhizal biomass is homogeneously distributed belowground and can thus be divided, proportionally to plant biomass, into those associated with seedlings or adults, $M_S = M \cdot \frac{S \cdot B_S}{S \cdot B_S + A \cdot B_A}$ and $M_A = M \cdot \frac{A \cdot B_A}{S \cdot B_S + A \cdot B_A}$, where B_S and B_A are the individual carbon biomass for seedlings and adults, respectively (Table 2). I assume interaction between mycorrhizas and plants is mainly based on negotiation for two currencies: plant photosynthetic carbon products and mycorrhizal nitrogen uptake. I assume mycorrhizas uptake soil nitrogen at a rate of uR (with uptake coefficient u), and transfers a minimum proportion $n_{\min}$ to associated host plant. Such a process is aimed to separate true mutualistic from parasitic relationships in the model. Mycorrhizas request for carbon from the plant in order to maintain its

C:N ratio (γ_M), resulting in carbon demand as $(1-n_{\min})uRM\cdot\gamma_M$ (Wallander, 1995; Johnson, 2009).

Plants use carbon from the atmosphere for primary production. The amount of carbon uptake when the plants are not associated with mycorrhizas is $(N-uptake_S+N-uptake_A)\cdot\gamma_P$, where γ_P is the plant tissue C:N ratio. $N-uptake_S$ and $N-uptake_A$ within the parentheses represents soil nitrogen uptake by seedlings and adults when unassociated with mycorrhizas, respectively (see *Soil nitrogen* section for detailed formulation). When plant-mycorrhiza associations are formed, plants may adjust their carbon uptake to $(N-uptake_S+N-uptake_A)\cdot\gamma_P\cdot(1+C_{max})$, where $C_{\max}$ represents the maximum proportion of primary production that plants will use as root exudation for benefit exchange (Cowden & Peterson, 2009). The maximum amount of carbon that the mycorrhiza can acquire from the plant after accounting for respiration loss is $(N-uptake_S+N-uptake_A)\cdot\gamma_P\cdot C_{max}\cdot e_M$, where e_M is the mycorrhiza carbon assimilation ratio (Bryla & Eissenstat, 2005).

I assume plants and mycorrhizas compare the carbon demand and supply at every time step in order to decide the amount of exchange between parties. If mycorrhizal carbon demand is larger than the maximum supply offered by plants, mycorrhizas are in a carbon-limited status. Under this situation, plants will transfer maximum amount of carbon, whereas mycorrhiza will transfer excess nitrogen to the

plant after meeting its own metabolic demands (Johnson, 2009). The total amount of transferred nitrogen is thus $uRM - \left[\left(N-uptake_S + N-uptake_A\right)\cdot\gamma_P \cdot C_{\max}\cdot e_M\right]\cdot\frac{1}{\gamma_M}$. On the other hand, if mycorrhizal nitrogen content is too low such that it is also limited by nitrogen (i.e. mycorrhizal carbon demand is less than plant supply), the microbe will keep nitrogen for its own metabolism (Johnson, 2009) and only transfer minimum amount of nitrogen, $n_{\min}uRM$. Plants adjust their carbon uptake and transfer the amount of carbon just enough to meet mycorrhizal demand.

I assume plants allocate their nitrogen, both from root uptake and mycorrhizal transfer, to reproduction, litter production, and growth. The quantity of mycorrhizal nitrogen content and mycorrhizal-enhancement of plant demography thus depends on the nutrient limitation status of mycorrhizas. I assume adults allocate nitrogen to reproduction and primary production with fixed proportion $l:r\cdot\frac{B_S}{\gamma_P}$, whereas seedlings allocate all their nitrogen for biomass growth. Seedling and adult dynamics are given by eqn. 1 and 2 in Table 1, whereas equations for mycorrhizal-enhancement of plant reproduction and growth is given as $r_{(S,A,M,R)}$ and $G_{(S,A,M,R)}$ in Table 2, respectively.

Mycorrhizal nitrogen content is released back to the soil following natural mortality (δ_M). Turnover of mycorrhiza hyphae may also result from the density-dependent negative effect of pathogens (β_{PM}) during competition for root

colonization sites (Sikes *et al.*, 2009). The dynamics of mycorrhiza is shown as eqn. 3 in Table 1, while its population growth under different nutrient limitation status is given as $\mu_{(S,A,M,R)}$ in Table 2.

Pathogens

I assume that pathogen infection causes the mortality of seedlings and adults with per plant infection rate $\alpha_S P$ and $\alpha_A P$, respectively (α_S and α_A represent infection efficiency). I assume that the infection efficiency of adults (α_A) is much smaller compared to that of seedlings (α_S) since adults have stronger physical protection (Augspurger & Kelly, 1984; Reinhart *et al.*, 2010). The nitrogen flux from plants to soil due to pathogen-induced mortality is the product of the number of dead individuals, their individual carbon biomass, and nitrogen:carbon ratio (γ_P^{-1}) ($\alpha_S SP \cdot \frac{B_S}{\gamma_P}$ and $\alpha_A AP \cdot \frac{B_A}{\gamma_P}$ for seedlings and adults, respectively). A fraction of this nitrogen flux will be incorporated into pathogen nitrogen content with assimilation ratio (b_P). Another fraction, f_P, will accumulate as litter and thus influencing litter dynamics. The remaining part, $1 - b_P - f_P$, is released back to the soil nitrogen pool. I assume that nitrogen in pathogens is released back to the soil due to natural mortality (δ_P) and density-dependent competition and inhibition by mycorrhizal fungi (β_{MP}) (Raaijmakers *et al.*, 2008). The dynamics of pathogens are shown in eqn.

4 in Table 1.

Litter

Nitrogen in plants enters the litter nitrogen pool due to mortality (natural or pathogen infection) and tissue turnover. The amount of litter caused by mortality is determined by the number of dead plants and their nitrogen content. Tissue turnover is assumed to be contributed by adults only. Litter production from seedlings is neglected due to their relatively small size. Adults uptake nitrogen for primary production at a rate of $lR \cdot (1 - \frac{A}{A_{\max}})$, and release it at the same rate into litter pool in order to remain fixed individual size. In addition, litter production can increase due to mycorrhizal-enhancement of nitrogen uptake, where the increment depends on the nutrient limitation status of mycorrhiza (indicated by $l_{(S,A,M,R)}$ in Table 2). For litter decomposition, I assume decomposition rate (dec) is mainly determined by litter quality (Berendse, 1994; Kurokawa & Nakashizuka, 2008). Nitrogen in litter is also lost from the system due to leaching (φ), which is an important characteristic of open ecosystems (Menge *et al.*, 2009). The equation for litter dynamics is given as eqn. 5 in Table 1.

Soil nitrogen

In this model, inorganic nitrogen is considered as the main limiting resource in the ecosystem. Nitrogen dynamics consists of external supply and loss, litter decomposition, release from dead material of plants and microbes, and biological uptake of plants and mycorrhizas (eqn. 6 in Table 1). Soil nitrogen pool is supplied by a constant deposition rate I and lost through leaching with the rate of Le. Nitrogen is released from biological components through litter decomposition, nitrogen released following pathogen-induced mortality of plants and those resulting from microbial turnover. Biological uptake includes both mycorrhizal and plant nitrogen uptake. The amount of plant nitrogen uptake (i.e. $N-uptake_S$ and $N-uptake_A$ for seedlings and adults, respectively) is determined by plant carbon allocation, biomass growth and plant tissue quality (Eppinga *et al.*, 2011). Seedlings accumulate photosynthetic carbon as its own biomass during maturation, the amount of carbon biomass yield is the product of the number of matured seedlings and the difference between carbon biomass of the two stage classes: $\left[gRA\cdot\left(B_A-B_S\right)\right]\cdot(1-\frac{A}{A_{\max}})$. Adults allocate primary production to reproduction and litter production and thus the amount of carbon uptake can be quantified by the sum of these two processes as $\left[rRA\cdot B_S+lRA\cdot\gamma_P\right]\cdot(1-\frac{A}{A_{\max}})$, where the first term within the bracket represents biomass of new recruits and the second term is the primary production that ended up as litter. The total amount of nitrogen uptake is determined via dividing primary

production by its C:N ratio.

Model Analysis and Simulation Experiments

In this study, I focused on the plant and microbial traits which are related to plant demography processes (r , g , m_A , m_S), litter dynamic processes (l , dec), plant-microbe interactions (γ_P , α_S , α_A , b_P , f_P , δ_P , u , $C_{\max}$, e_M , γ_M , δ_M), and microbial interaction (β_{MP} , β_{PM}) (Table 4). I examined the interactive effects of litter- and microbial-mediated PSF and the relative importance of traits via the following four scenarios: (1) a system only considering litter-mediated PSF without any direct-interacting microbes ($P = M = 0$ in Fig. 1), (2) a system considering litter-mediated PSF and pathogens as representative of microbial-mediated PSF ($P > 0, M = 0$ in Fig. 1), (3) a system with litter-mediated PSF and mycorrhizas ($P = 0, M > 0$ in Fig. 1) and, (4) a system with litter-mediated PSF and both pathogens and mycorrhizas ($P, M > 0$ in Fig. 1). In order to address the effect of microbial community composition on relative importance of traits, I set two interaction scenarios between pathogens and mycorrhizas for the system with both microbes: (4a) bidirectional interaction between the two microbes due to competition for root colonization site and (4b) unidirectional impact of mycorrhiza on pathogen

via producing antimicrobial metabolites. The effect of microbial community composition on PSF can thus be obtained via comparing the results of these two scenarios.

Empirical experiments typically quantify PSF strength via comparing biomass growth of the target species' seedlings in self-cultivated soil in greenhouses to those that grown in heterospecies-cultured soils. I perform a simulation experiment to quantify PSF strength following a similar framework. I consider two hypothetical plant species: a reference plant species (species *ref*) with trait values obtained from empirical studies (detailed parameter values shown in Table 4 and Appendix S4) and a target plant species (species *tar*). I also conducted basic mathematical analysis to check the positivity of microbe-free equilibrium and invasibility of microbes into the microbe-free equilibrium when choosing the plausible set of parameter values (Appendix S3). I set the target plant species to have only one trait value deviated from the reference plant species at a time in order to identify the relative importance of each trait on the direction and strength of PSF. Deviation of trait values was set to ±50% for scenarios without mycorrhizas. For the scenarios with mycorrhizas, deviation range was set to ±20% in order to remain a realistic range for e_M. I also calculate the effect size of larger deviation for other traits while still remaining in a realistic range (10% to 300% of the references plant species value, Appendix S1).

I first run the model numerically to equilibrium for each microbial composition scenario with both reference and target plant species parameter settings. This is a step for simulating plant trait-specific cultivation of nearby soil environment for a long period of time. Numerical simulation was carried out by C language using the fourth-order Runge-Kutta method with a fixed interval (see Appendix S5 for detailed numerical method). Equilibrium values of soil nitrogen (R_k^*) and microbes (P_k^* and M_k^*) are recorded to represent properties of the soil cultivated by the specific plant species *k* (*k* = *ref* or *tar*, stage 1 in Fig. 2). A sub-model was then prepared to simulate the dynamics of seedling growth of plant species *i* in the pots filled with cultivated soils (eqns. shown in Table 2 and 3). The sub-model is similar to seedling and adult dynamics in the full model in that it considers growth of seedlings, growth enhancement by mycorrhiza (represented as $g_{i(S^*,A^*,M^*,R^*)}$ in Table 2), seedling natural mortality and pathogen infection. However, the sub-model does not consider reproduction and shading effect of adults and the equilibrium values recorded from the full model are used to represent specific soil properties (i.e. R_k^*, P_k^* and M_k^*). These soil properties are assumed to be constant during the simulation of the sub-model following the recognition that seedling growth is determined by historical plant growth legacies (Kardol *et al.*, 2007; Kulmatiski & Beard, 2011). I run the sub-model to equilibrium (i.e. all seedlings are matured or dead) and denote the

equilibrium adult density as $A^{**}_{i,\,k}$, which represents the growth response of plant species *i* in soil cultivated by plant species *k* (*i* and *k* = *ref* or *tar*, stage 2 in Fig. 2). The PSF strength for the target plant is determined by comparing its biomass growth in the two different soils (i.e. $A^{**}_{tar,\,ref}$ and $A^{**}_{tar,\,tar}$) via the formulation following Petermann *et al*. (2008): $PSF_{tar} = \log\left(\frac{A^{**}_{tar,\,tar}}{A^{**}_{tar,\,ref}}\right)$. If the resulting PSF is positive, it means that the growth of the target plant species is benefitted in soils cultivated by conspecies due to deviation of that specific trait. However, if the PSF is negative, it means that such deviation of trait from the reference plant corresponds to a net disadvantage on home soils compared to the reference soils.

Results

Litter-mediated PSF without any direct-interacting microbes

Plant tissue and litter quality traits are most important traits in determining PSF strength compared to other plant functional traits under the scenario with litter-mediated PSF only (Fig. 3a, effect size of ±50% deviation from the reference value). An increase in litter decomposition rate (dec) and plant tissue C:N ratio (γ_P) resulted in strongest positive PSF as such increases produced larger soil nitrogen content in the target plant species cultivated soil compared to those of the reference plant species (i.e. $\Delta R^* > 0$, Fig. 3b). An increase in adult mortality rate (m_A) caused the target plant to realize negative PSF due to decreased soil nitrogen. I found qualitatively the same pattern for even larger deviation of the trait value (i.e. 10% - 300% of the reference plant species value, Appendix S1, Fig. S1). The identity of the most influential traits remains unchanged when other trait values, in addition to the target trait, were randomly assigned simultaneously (Appendix S2, Fig. S6). The direction of PSF resulting from positive deviation of traits is summarized in Table 5.

Litter-mediated PSF and pathogens

The traits related to plant defense against pathogens are most influential in determining PSF strength under the scenario with pathogens as the only representative

of microbial-mediated PSF (Fig. 4, effect size of ±50% deviation from the reference value). An increase in plant tissue C:N ratio (γ_P), pathogen mortality rate (δ_P) and seedling biomass growth rate per nitrogen (g) resulted in strongest positive PSF (Fig. 4a). This positive PSF is due to a combined effect of increased soil nitrogen (i.e. $\Delta R^* > 0$, Fig. 4b) and decreased pathogen nitrogen content (i.e. $\Delta P^* < 0$, Fig. 4c) of the target plant species cultivated soil. An increase in pathogen assimilation ratio (b_P) of plant tissue resulted in strongest negative PSF due to increased pathogen nitrogen content despite the accompanied increase in soil nitrogen. Increased plant reproduction (r) also generated negative PSF, which is due to synergic effects that are opposite to those of increased biomass growth rate. Relative importance of litter decomposability (dec) on PSF strength is low under this scenario because benefits of increased soil nitrogen are offset by the slightly increased pathogen nitrogen content. I found the same pattern for larger deviation of the trait value (Appendix S1, Fig. S2). The identity of the most influential traits remains unchanged when other trait values are simultaneously randomly assigned (Appendix S2, Fig. S7).

Litter-mediated PSF and mycorrhizas

The traits related to plant tissue and litter quality, as well as those related to mycorrhiza nutrient acquisition ability are most influential in determining PSF

strength under the scenario with beneficial mycorrhizal fungi as the only representative of microbial-mediated PSF (Fig. 5, effect size of ±20% deviation from the reference value). An increase in trait value such as plant tissue C:N ratio (γ_P), plant carbon transfer ratio ($C_{\max}$) and mycorrhiza carbon assimilation ratio (e_M) (i.e. traits related to plant-mycorrhiza carbon exchange) resulted in strong positive PSF (Fig. 5a). This positive PSF is due to significantly increased mycorrhiza nitrogen content (i.e. $\Delta M^* > 0$, Fig. 5c), despite soil nitrogen level was depleted (i.e. $\Delta R^* < 0$, Fig. 5b) to support growth of both plant and microbes. Relative importance of litter decomposability (dec) is high for this scenario as an increase of decomposition rate resulted in synergic increase of both soil nitrogen and mycorrhiza. Increase in other mycorrhiza traits such as mycorrhiza nitrogen uptake coefficient (u) and mycorrhiza C:N ratio (γ_M) resulted in large decrease of soil nitrogen and mycorrhiza nitrogen content, respectively, mainly due to carbon starvation of the microbe and are thus influential in determining negative PSF. The pattern remains qualitatively the same even for larger deviation of the trait value (Appendix S1, Fig. S3). The identity of the most influential traits remains unchanged when other trait values are simultaneously randomly assigned (Appendix S2, Fig. S8).

Litter-mediated PSF and both pathogens and mycorrhizas

The traits related to plant tissue quality and nutrient acquisition ability of microbes are most influential in determining PSF strength under the scenario with both pathogen and mycorrhiza (Fig. 6 and 7, effect size of ±20% deviation for bidirectional and unidirectional competition, respectively). An increase in plant tissue C:N ratio (γ_P) and traits related to plant-mycorrhiza carbon exchange (i.e. $C_{\max}$ and e_M) resulted in strong positive PSF for both competition scenarios (Fig. 6a and 7a). The positive PSF resulting from positive deviation of these traits was due to increased mycorrhiza relative abundance (i.e. $\Delta\left(M^*/M^*+P^*\right) > 0$, Fig. 6c and 7c), despite such increase is accompanied by decreased soil nitrogen (i.e. $\Delta R^* < 0$, Fig. 6b and 7b). An increase in mycorrhiza nitrogen uptake coefficient (u) and mycorrhiza C:N ratio (γ_M) resulted in negative PSF due to similar reasons in the case only with mycorrhizas.

The relative importance of some traits is different among the two competition scenario (bidirectional Fig. 6 vs. unidirectional Fig. 7). In particular, the effect size of litter decomposability (dec) is low when negative effects are bidirectional (i.e. $\beta_{MP}, \beta_{PM} > 0$, Fig. 6a), while it acts as an important determinant for PSF strength when only mycorrhizas have negative impact on pathogens (i.e. $\beta_{MP} > 0, \beta_{PM} = 0$, Fig. 7a). This pattern is confirmed when quantifying the effect size of litter decomposability under different competition scenarios via continuous changes in

β_{MP} and β_{PM} (Fig. 8a, effect size of ±20% deviation from the reference value), which can be explained by the difference in the relative abundance of microbes (Fig. 8b). When the competition tends to be symmetrical (i.e. $\beta_{MP} \approx \beta_{PM}$) or negative impact from pathogen on mycorrhiza is stronger (i.e. $\beta_{MP} < \beta_{PM}$), the relative importance of litter decomposability is low and is accompanied with lower relative abundance of mycorrhiza. This result corresponds to the case only with pathogens (Fig. 4a). On the other hand, effect size of litter decomposability is high when negative impact of mycorrhiza on pathogen is stronger (i.e. $\beta_{MP} > \beta_{PM}$), since under such asymmetric interaction scenario the relative abundance of mycorrhiza is higher (Fig. 8b). The results under these scenarios thus correspond to the case only with mycorrhizas (Fig. 5a). The same pattern was found for larger deviation of the trait value (Appendix S1, Fig. S4 and S5). The identity of the most influential traits remains unchanged when other traits, in addition to the target trait, are randomly assigned (Appendix S2, Fig. S9 and S10).

Discussion

The present study is a modeling attempt to identify the relative importance of traits on PSF strength when litter- and microbial-mediated PSF are coupled. The inclusion of microbial-mediated PSF alters the identity of the most influential traits predicted via litter-mediated PSF due to indirect interactions between litter and microbes. Pathogens and mycorrhizas interact with litter dynamics differently, and thus the relative importance of traits depends on the microbial community composition of cultivated soils.

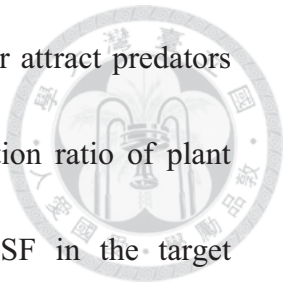
Effects of microbial community composition on relative importance of traits

While there has been increasing interest in understanding how plant functional traits may determine community properties and ecosystem processes (de Bello *et al.*, 2010), this study shows that the effect size of different functional traits on PSF direction and strength is context-dependent. In particular, identity of the most influential trait in determining PSF strength depends on the relative abundance of mycorrhiza and pathogen.

When the soil lacks of both pathogenic and beneficial microbes, litter decomposability is most influential in determining PSF strength (Fig. 3a). Higher litter decomposition rate can release nitrogen stored in the organic litter to the soil at a

faster rate, resulting in nutrient rich environments and positive PSF (Fig. 3b). Other litter feedback models agree with the results by showing species that produce easily decomposing litter can accelerate nutrient cycling, and may gain growth advantage over other plants if it favors nutrient-rich environments (Berendse, 1994; Miki & Kondoh, 2002). In addition, the model shows that an increase in adult mortality rate resulted in strong negative PSF under this model setting. I speculate this is because higher adult mortality will produce more open canopy, which can enhance plant population growth but decrease soil nitrogen content due to larger uptake flux, and thus in turn has negative impact on future seedling growth.

When the soil biota is dominated by pathogens, the plant defense traits against pathogens are influential in determining PSF strength. Plant roots are able to exhibit a large variety of defense strategies, including physical defense via producing lignified roots, or chemical defense through excreting secondary metabolites (van Dam, 2009; Rasmann *et al.*, 2011). The model shows that an increase in plant tissue C:N ratio (e.g. increase in wood density) can result in strong positive PSF (Fig. 4a). Plants with such trait are low in nutrition quality and thus can significantly suppress pathogen level in the target plant-cultivated soils (Fig. 4c). This prediction is supported by Augspurger & Kelly (1984) as they show species with higher basic wood density suffer less disease mortality. Other defense traits that can increase pathogen mortality (e.g.



excrete secondary metabolites that directly act against pathogens or attract predators of the pathogen, van Dam, 2009), or decrease pathogen assimilation ratio of plant tissue (e.g. more lignified roots) can also generate positive PSF in the target plant-cultivated soils.

When mycorrhizas are included in the model, the traits that characterize plant-mycorrhiza interactions are influential in determining the strength of PSF. Moreover, when mycorrhizas are abundant, the model predicts that litter decomposability is also equally important as litter- and microbial-mediated PSFs interact strongly. Plant tissue C:N ratio remains an important determining factor for positive PSF. Plants with higher carbon transfer ratio and/or cooperating with mycorrhizas that have higher carbon assimilation ratio also realize strong positive PSF (Fig. 5a). In the model, carbon is assumed to be unlimited for plants. Plants with higher C:N ratio and higher carbon transfer ratio are able to supply more photosynthetic product to the microbe per unit nitrogen uptake. Since both plants and mycorrhizas rely on soil nitrogen for metabolism, species with such properties can form more beneficial plant-mycorrhiza associations and increase the mycorrhiza level in soils (Fig. 5c). In contrast, soils that are abundant in mycorrhizas with characteristics such as higher nitrogen uptake coefficient and/or higher C:N ratio will result in strong negative PSF. I speculate that this is due to stronger competition

between plants and mycorrhiza for soil nitrogen, and larger carbon demand of the microbe resulting from higher mycorrhizal C:N ratio, which may easily lead to microbial carbon starvation (Wallander, 1995; Johnson, 2009). Mycorrhiza abundance is expected to be lower for such plant-mycorrhiza associations and thus growth of seedlings is suppressed in self-cultivated soil.

Effects of microbial composition on the relative importance of litter-mediated PSF

When combining litter- and microbial-mediated PSF caused by different microbes, the importance of litter decomposability dramatically changes with microbial composition. Litter decomposition contributes largely to positive PSF when the soil contains no direct-interacting microbes (Fig. 3a). However, the importance of litter decomposability reduces when pathogens are included as the only representatives of microbial-mediated PSF (Fig. 4a). This is because although higher litter decomposability of the target plant can increase plant primary production due to enhanced nutrient cycling, increased plant population will also support more pathogens and thus cancelling out the beneficial effects of higher decomposability.

Compared to pathogen-dominated soils, litter decomposability has strong positive effect on PSF strength when mycorrhiza is abundant (Fig. 5a and 7a). Such difference is due to the different interaction of pathogens or mycorrhizas with litter

dynamics. The hyphae of mycorrhizas can grow into soil micropores to help the host plant obtain nutrients that would otherwise be unavailable (Veresoglou *et al.*, 2002). This mycorrhiza-enhanced nutrient uptake process can increase plant productivity and result in greater litter production (Orwin *et al.*, 2011). A higher decomposition rate of the target plant under such scenario will thus result in faster nutrient release of mycorrhizal-enhanced litter production back to the low-nitrogen soil, causing strong positive PSF due to its synergic positive effects on soil nitrogen content and mycorrhizal nitrogen content. This result is consistent with other studies demonstrating that mycorrhizas can increase plant litter input to the soil and thus increase soil carbon accumulation (Read & Perez-Moreno, 2003; Orwin *et al.*, 2011).

Effects of stage structure on PSF strength and relative importance of traits

Plant individuals from different stage classes within the population may experience different interaction strength with PSF drivers. Such differential interaction is especially important when discussing relative importance of traits if the soil biota mainly consists of pathogens. In particular, higher biomass growth of seedlings results in faster transition from the vulnerable seedling stage to a better-defended adult stage. Such characteristic can shorten the period of time when plants are most susceptible to disease and thus creating positive PSF via suppressing

pathogen population size in the target plant cultivated soils (Augspurger, 1990; Reinhart *et al.*, 2010). The pathogen level, however, will increase due to a larger supply of susceptible seedlings and thus create negative PSF if the target plant has higher reproduction rate per soil nitrogen. These results are consistent with empirical studies, which showed that plants in gap environments grow faster and thus suffer less pathogen-induced mortality (Augspurger, 1990; Reinhart *et al.*, 2010). Other experimental studies also revealed that negative density-dependent mortality is pervasive and severity of damping-off disease is higher when seedlings grow in high densities (Augspurger, 1983; Bell *et al.*, 2006, Bagchi *et al.*, 2010).

Insights for exotic plant invasion success

Interactions between plant and soil had long been recognized as an important factor determining the success of exotic plant invasion (Reinhart & Callaway, 2006). Past studies attribute the success of invasion to either modified nutrient cycling caused by the invader through altering litter dynamics (Miki & Kondoh, 2002; Eppinga *et al.*, 2011), or due to novel plant-microbial interactions (Callaway *et al.*, 2003; Mitchell & Power, 2003; Eppinga *et al.*, 2006) following the invasion process. Understanding the linkage between plant traits and PSF allows us to predict which

invading species is most likely to successfully invade due to its specific combination of functional traits.

One of the dominating hypotheses connecting plant invasion to local soil biota is the enemy release hypothesis, which states that the release from host-specific soil pathogens in their native range contributes to the success of plant invasion (Callaway *et al.*, 2003; Mitchell & Power, 2003). From the invader point of view, soils in the invaded area may be characterized as having few direct-interacting microbes (i.e. both pathogens and mycorrhizas are absent) or higher mycorrhiza relative abundance due to the absence of host-specific pathogens. My model indicates that under all soil environments without pathogens, decomposition rate is an important factor for the success of plant invasion. When the soil biota mainly consists of mycorrhizas, I suggest that traits of plant-mycorrhiza carbon exchange are also important factors in addition to litter decomposability. Invaders that produce easily decomposing litter are predicted to be most powerful in invading areas with little direct-interacting microbes, while invaders with easily decomposing litter and produces more root exudation as carbon supply to the mycorrhiza are best able to invade such enemy-released soils. Empirical studies support the predictions by showing that litter from the invader often decomposes at a faster rate (Allison & Vitousek, 2004), and litter decomposition rate is usually higher in invaded areas (Liao *et al.*, 2008). My model thus suggests that

indirect interaction between litter and mycorrhiza in enemy-released soils can explain the trend of higher litter decomposability of invaders.

Multiple possible outcomes have been documented when soils of the invaded area are also pathogen-rich for the invaders (e.g. pathogens are generalists or brought by the invader along invasion). One possible outcome is that the invasion may fail as the invader encounters pathogens in the invaded area which prevent the invasion, a situation related to the biotic resistance hypothesis (Mitchell & Power, 2003). Another possible scenario is termed pathogen accumulation hypothesis. Under this scenario invasion can still succeed if the invader promotes growth of pathogens that have a stronger negative effect on the surrounding native plants than on the invader itself (Eppinga *et al.*, 2006; Mangla *et al.*, 2008). My model indicates that when the invaded area contains high pathogen level, defense traits against pathogens plays a key role for successful invasion. In particular, the model predict that plants that produce higher wood density and/or exhibit other defense strategies are able to invade despite high level of pathogen in the invaded area. This may also answer why in some cases higher litter decomposition rate is not associated with invasion status (Drenovsky & Batten, 2007, Kurokawa *et al.*, 2010) since litter decomposability is not an important determinant of PSF in soils with high pathogen level. In conclusion, my model suggests that the contrasting trait characteristic of invaders at different sites can be

explained by the difference in soil microbial community composition of different invaded area.

Future work and Conclusion

To my best knowledge, this study is the first attempt to link plant functional traits with PSF strength through a modeling approach. The model presented is novel in its ability to combine litter-mediated and microbial-mediated PSF, also in its ability to give predictions by simulating field experimental settings. I highlighted the importance of indirect effects of microbes on litter dynamics and demonstrated that separation of plant stage classes and microbial functional groups is necessary for understanding the determinants of PSF strength. In particular, the effect of litter decomposability increases with the relative abundance of mycorrhizas. Past trait-based ecological studies tried to predict the shifts of ecological and ecosystem functions along abiotic gradients through focusing of the changes in values of specific traits (McGill *et al.*, 2006). My model results would challenge this traditional research framework by arguing that some traits may lose its impact on ecological and ecosystem processes along the abiotic gradient since its importance is also determined by indirect biotic interactions with other species. In the case of PSF, my results suggest a closer look in the microbial community composition is necessary for

development of trait-based approach. While many studies had revealed a systematic change of bacteria to fungi ratio across large geographic scale, I suggest that further studies targeted to reveal the ratio between detrimental and beneficial microbes across different ecosystems (e.g. from tropic to temperate regions) may provide knowledge for understanding the frequency distribution of functional traits (Manning *et al.*, 2008). Experimental studies often show that the response of species' PSF strength to nutrient enrichment is species-specific, suggesting that further studies focusing on more detailed characteristics of the plant, in addition to species identity, are needed. My trait-based approach can thus provide insights and pinpoint some potential traits which could be important PSF determinants.

Future works that extend the model to include multiple plant species interaction may reveal the importance of species-specific combination of litter quality and nutrient competition strategy (Berendse 1994, Miki & Kondoh 2002, Miki *et al*., 2010). Results from such models may give valuable predictions about the role of PSF in determining plant coexistence and the relative abundance patterns in plant community. I believe that integration of this new model framework with modeling aboveground and belowground interactions mediated through plant induced response (Wardle *et al.*, 2004; van der Putten *et al.*, 2009) can also contribute to a solid and

improved theoretical framework for understanding the role of functional traits in controlling plant community development.

References

Alvarez-Loayza P, Terborgh J. **2011**. Fates of seedling carpets in an Amazonian floodplain forest: intra-cohort competition or attack by enemies? *Journal of Ecology* **99**: 1045-1054.

Allison SD, Vitousek PM. **2004**. Rapid nutrient cycling in leaf litter from invasive plants in Hawai'i. *Oecologia* **141**: 612-619.

Augspurger CK. **1983**. Seed Dispersal of the Tropical Tree, *Platypodium Elegans*, and the Escape of its Seedlings from Fungal Pathogens. *The Journal of Ecology* **71**: 759-771.

Augspurger CK, Kelly CK. 1984. Pathogen mortality of tropical tree seedlings: experimental studies of the effects of dispersal distance, seedling density, and light conditions. *Oecologia* **61**: 211-217.

Augspurger CK. 1990. Spatial patterns of dampinf-off disease during seedling recruitment in tropical forests. In: Burdon JJ, Leather SR, eds. *Pests, Pathogens and Plant Communities*. Oxford, UK: Blackwell Scientific Publications, 131-144.

Bagchi R, Swinfield T, Gallery RE, Lewis OT, Gripenberg S, Narayan L, Freckleton RP. **2010**. Testing the Janzen-Connell mechanism: pathogens cause overcompensating density dependence in a tropical tree. *Ecology Letters* **13**: 1262-1269.

Bell T, Freckleton RP, Lewis OT. **2006**. Plant pathogens drive density-dependent seedling mortality in a tropical tree. *Ecology Letters* **9**: 569-574.

de Bello F, Lavorel S, Díaz S, Harrington R, Cornelissen JHC, Bardgett RD, Berg MP, Cipriotti P, Feld CK, Hering D, *et al.* 2010. Towards an assessment

of multiple ecosystem processes and services via functional traits. *Biodiversity and Conservation* **19**: 2873-2893.

Berendse F.1994. Litter decomposability - a neglected component of plant fitness. *Journal of Ecology* **82**: 187-190.

Bever JD, Westover KM, Antonovics J. **1997**. Incorporating the soil community into plant population dynamics: the utility of the feedback approach. *Journal of Ecology* **85**: 561-573.

Bever JD, Dickie IA, Facelli E, Facelli JM, Klironomos J, Moora M, Rillig MC, Stock WD, Tibbett M, Zobel M. **2010**. Rooting theories of plant community ecology in microbial interactions. *Trends in ecology & evolution* **25**: 468-478.

Binley D, Giardina C. 1998. Why do tree species affect soil? The warp and woof of tree-soil interaction. *Biogeochemistry* **42**: 89-106.

Brinkman EP, van der Putten WH, Bakker EJ, Verhoeven KJF. 2010. Plant-soil feedback: experiment approaches, statistical analysis and ecological interpretations. *Journal of Ecology* **98**: 1063-1073.

Callaway RM, Thelen GC, Rodriguez A, Holben WE. 2004. Soil biota and exotic plant invasion. *Nature* **427**: 731-733.

Clark DA, Brown S, Kicklighter DW, Chambers JQ, Thomlinson JR, Ni J, Holland EA. **2001**. Net primary production in tropical forests: an evaluation and synthesis of existing field data. *Ecological Applications* **11**: 371-384.

Chave J, Coomes D, Jansen S, Lewis SL, Swenson NG, Zanne AE. **2009**. Towards a worldwide wood economics spectrum. *Ecology Letters* **12**: 351–366.

Cowden CC, Peterson CJ. 2009. A multi-mutualist simulation: applying biological market models to diverse mycorrhizal communities. *Ecological Modelling* **220**: 1522-1533.

van Dam NM. **2009**. Belowground Herbivory and Plant Defenses. *Annual Review of Ecology, Evolution, and Systematics* **40**: 373-391.

De Deyn GB, Raaijmakers CE, van der Putten WH. **2004**. Plant community development is affected by nutrients and soil biota. *Journal of Ecology* **92**: 824-834.

De Vries FT, Manning P, Tallowin JRB, Mortimer SR, Pilgrim ES, Harrison K a, Hobbs PJ, Quirk H, Shipley B, Cornelissen JHC, *et al.* 2012. Abiotic drivers and plant traits explain landscape-scale patterns in soil microbial communities. *Ecology Letters* **15**: 1230-1239.

Dickie IA, Schnitzer SA, Reich PB, Hobbie SE. **2005**. Spatially disjunct effects of co-occurring competition and facilitation. *Ecology Letters* **8**: 1191-200.

Drenovsky RE, Batten KM. **2007**. Invasion by *Aegilops triuncialis* (barb goatgrass) slows carbon and nutrient cycling in a serpentine grassland. *Biological Invasion* **9**: 107–116.

Ehrenfeld JG, Ravit B, Elgersma K. **2005**. Feedback in the plant-soil system. *Annual Review of Environment and Resources* **30**: 75-115.

Eppinga MB, Rietkerk M, Dekker SC, De Ruiter PC. **2006**. accumulation of pathogens: a new hypothesis to explain exotic plant invasion. *Oikos* **114**: 168-176.

Eppinga MB, Kaproth MA, Collins AR, Molofsky J. 2011. Litter feedback, evolutionary change and exotic plant invasion. *Journal of Ecology* **99**: 503-514.

van der Heijden MGA, Bardgett RD, van Straalen NM. **2008**. The unseen majority: soil microbes as drivers of plant diversity and productivity in terrestrial ecosystems. *Ecology Letters* **11**: 296-310.

Johnson NC. 2009. Resource stoichiometry elucidates the structure and function of arbuscular mycorrhizas across scales. *New Phytologist* **185**: 631-647.

Kardol P, Bezemer TM, van der Putten WH. **2006**. Temporal variation in plant-soil feedback controls succession. *Ecology Letters* **9**: 1080-1088.

Kardol P, Corpins NJ, van Kempen MML, Bakx-Schotman JMT, van der Putten WH. 2007. Microbe-mediated plant-soil feedback causes historical contingency effects in plant community assembly. *Ecological Monographs* **72**: 147-162.

Klironomos JN. **2002**. Feedback with soil biota contributes to plant rarity and invasiveness in communities. *Nature* **417**: 67–70.

Kulmatiski A, Heavilin J, Beard KH. **2011**. Testing predictions of a three-species plant-soil feedback model. *Journal of Ecology* **99**: 542-550.

Kulmatiski A, Beard KH. 2011. Long-tern plant growth legacies overwhelm short-term plant growth effects on soil microbial community structure. *Soil Biology & Biochemistry* **43**: 823-830.

Kunstler G, Lavergne S, Courbaud B, Thuiller W, Vieilledent G, Zimmermann NE, Kattge J, Coomes D a. **2012**. Competitive interactions between forest trees are driven by species' trait hierarchy, not phylogenetic or functional similarity: implications for forest community assembly. *Ecology Letters* **15**: 831-840.

Kurokawa H, Nakashizuka T. 2008. Leaf herbivory and decomposability in a Malaysian tropical rain forest. *Ecology* **89**: 2645-2656.

Kurokawa H, Peltzer DA, Wardle DA. 2010. Plant traits, leaf palatability and litter decomposability for co-occurring woody species differing in invasion status and nitrogen fixation ability. *Functional Ecology* **24**: 513-523.

Liao C, Peng R, Luo Y, Zhou X, Wu X, Fang C, Chen J, Li B. **2008**. Altered ecosystem carbon and nitrogen cycles by plant invasion : a meta-analysis. *New Phytologist* **177**: 706-714.

Mangan SA, Schnitzer SA, Herre EA, Mack KML, Valencia MC, Sanchez EI, Bever JD. **2010**. Negative plant-soil feedback predicts tree-species relative

abundance in a tropical forest. *Nature* **466**: 752-755.

Mangla S, Inderjit, Callaway RM. **2008.** Exotic invasive plant accumulates native soil pathogens which inhibit native plants. *Journal of Ecology* **96**: 58-67.

Manning P, Morrison SA, Bonkowski M, Bardgett RD. **2008**. Nitrogen enrichment modifies plant community structure via changes to plant-soil feedback. *Oecologia* **157**: 661-673.

Mazzoleni S, Bonanomi G, Giannino F, Rietkerk M, Dekker SC, Zucconi F. **2007**. Is plant biodiversity driven by decomposition processes? An emerging new theory on plant diversity. *Community Ecology* **8**: 103–109.

McGill BJ, Enquist BJ, Weiher E, Westoby M. **2006**. Rebuilding community ecology from functional traits. *Trends in ecology & evolution* **21**: 178-185.

Menge DNL, Pacala SW, Hedin LO. 2009. Emergence and maintenance of nitrogen limitation over multiple timescales in terrestrial ecosystems. *The American Naturalis*t **173**: 164-175.

Miki T, Kondoh M. 2002. Feedbacks between nutrient cycling and vegetation predict plant species coexistence and invasion. *Ecology Letters* **5**: 624-633.

Miki T, Ushio M, Fukui S, Kondoh M. **2010**. Functional diversity of microbial decomposers facilitates plant coexistence in a plant-microbe-soil feedback model. *Proceedings of the National Academy of Sciences, USA* **107**: 14251-14256.

Mitchell CE, Power AG. 2003. Release of invasive plants from fungal and viral pathogens. *Nature* **421**: 625-627.

Packer A, Clay K. **2000**. Soil pathogens and spatial patterns of seedling mortality in a temperate tree. *Nature* **404**: 278-281.

Petermann JS, Fergus AJF, Turnbull LA, Schmid B. 2008. Janzen-Connell effects are widespread and strong enough to maintain diversity in grassland. *Ecology* **89**: 2399-2406.

van der Putten WH, Peters BAM. **1997**. How soil-borne pathogens may affect plant competition. *Ecology* **78**: 1785-1795.

van der Putten WH, Bardgett RD, De Ruiter PC, Hol WHG, Meyer KM, Bezemer TM, Bradford MA, Christensen S, Eppinga MB, Fukami T, *et al.* 2009. Empirical and theoretical challenges in aboveground-belowground ecology. *Oecologia* **161**: 1-14.

van der Putten WH, Bardgett RD, Bever JD, Bezemer TM, Casper BB, Fukami T, Kardol P, Klironomos JN, Kulmatiski A, Schweitzer JA, *et al.* 2013. Plant-soil feedbacks : the past, the present and future challenges. *Journal of Ecology* **101**: 265-276.

Raaijmakers JM, Paylitz TC, Steinberg C, Alabouvette C, Moënne-Loccoz Y. 2008. The rhizosphere: a playground and battlefield for soilborne pathogens and beneficial microorganisms. *Plant and Soil* **321**: 341-361.

Rasmann S, Bauerle TL, Poveda K, Vannette R. **2011**. Predicting root defence against herbivores during succession. *Functional Ecology* **25**: 368-379.

Read DJ, Perez-Moreno J. **2003**. Mycorrhizas and nutrient cycling in ecosystems - a journey towards. *New Phytologist* **157**: 475-492.

Reinhart KO, Callaway RM. 2006. Soil biota and invasive plants. *New Phytologist* **170**: 445-457.

Reinhart KO, Royo AA, Kageyama SA, Clay K. 2010. Canopy gaps decrease microbial densities and disease risk for a shade-intolerant tree species. Acta *Oecologica* **36**: 530-536.

Šmilauerová M, Lokvencová M, Šmilauer P. **2011**. Fertilization and forb: graminoid ratio affect arbuscular mycorrhiza in seedlings but not adult plants of Plantago lanceolata. *Plant and Soil* **351**: 309-324.

Treseder K. 2004. A meta-analysis of mycorrhizal responses to nitrogen, phosphorus, and atmospheric CO2 in field studies. *New Phytologist* **164**: 347-355.

Veresoglou SD, Chen B, Rillig MC. **2012**. Arbuscular mycorrhiza and soil nitrogen cycling. *Soil Biology and Biochemistry* **46**: 53-62.

Wallander H. 1995. A new hypothesis to explain allocation of dry matter between mycorrhizal fungi and pine seedlings in relation to nutrient supply. *Plant and Soil* **168-169**: 243-248.

Wardle DA, Bardgett RD, Klironomos JN, Setälä H, van der Putten WH, Wall DH. **2004**. Ecological linkages between aboveground and belowground biota. *Science* **304**: 1629-33.

Wardle DA. 2006. The influence of biotic interactions on soil biodiversity. *Ecology Letter* **9**: 870-886.

Wright IJ, Reich PB, Westoby M, Ackerly DD, Baruch Z, Bongers F, Cavender-bares J, Chapin T, Cornelissen JHC, Diemer M, *et al.* 2004. The worldwide leaf economics spectrum. *Nature* **12**: 821-827.

Figures

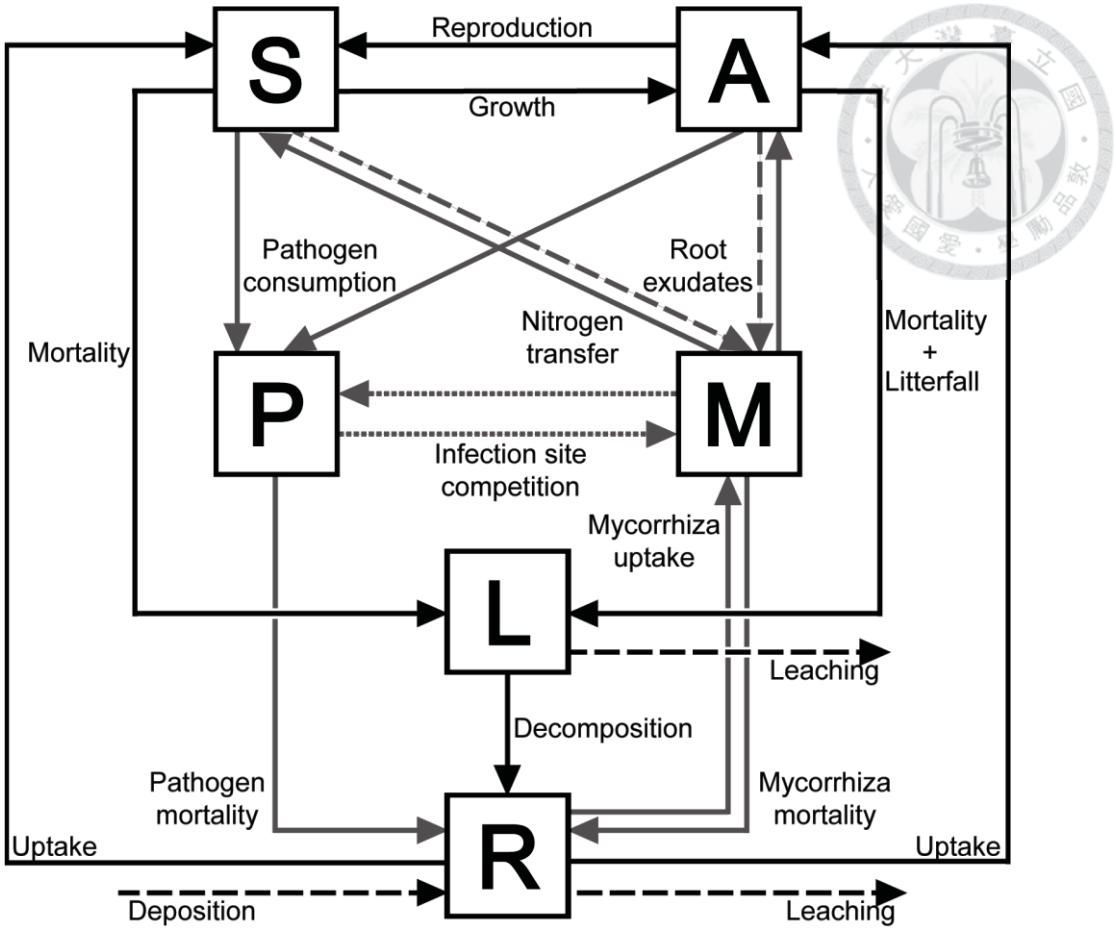


Figure. 1. Flow diagram of the opened-ecosystem stage-structured PSF model. Flows from the inorganic soil nitrogen pool (R) to the two plant stage classes (S and A) represent resource uptake and allocation of primary production. Flows from plants to litter pool (L) represent litter increase due to mortality and litter production, whereas flows from litter to nitrogen pool represent litter decomposition and mineralization. Grey solid lines from plants to pathogen (P) represent pathogen consumption, whereas solid and dashed lines between plants and mycorrhiza (M) represent nitrogen and carbon exchange between the two components, respectively. Dotted lines represent negative interaction between the microbes and solid lines from microbes to soil nitrogen represent nutrient release. Flows entering and leaving litter and soil nitrogen pool represent deposition and leaching, respectively.

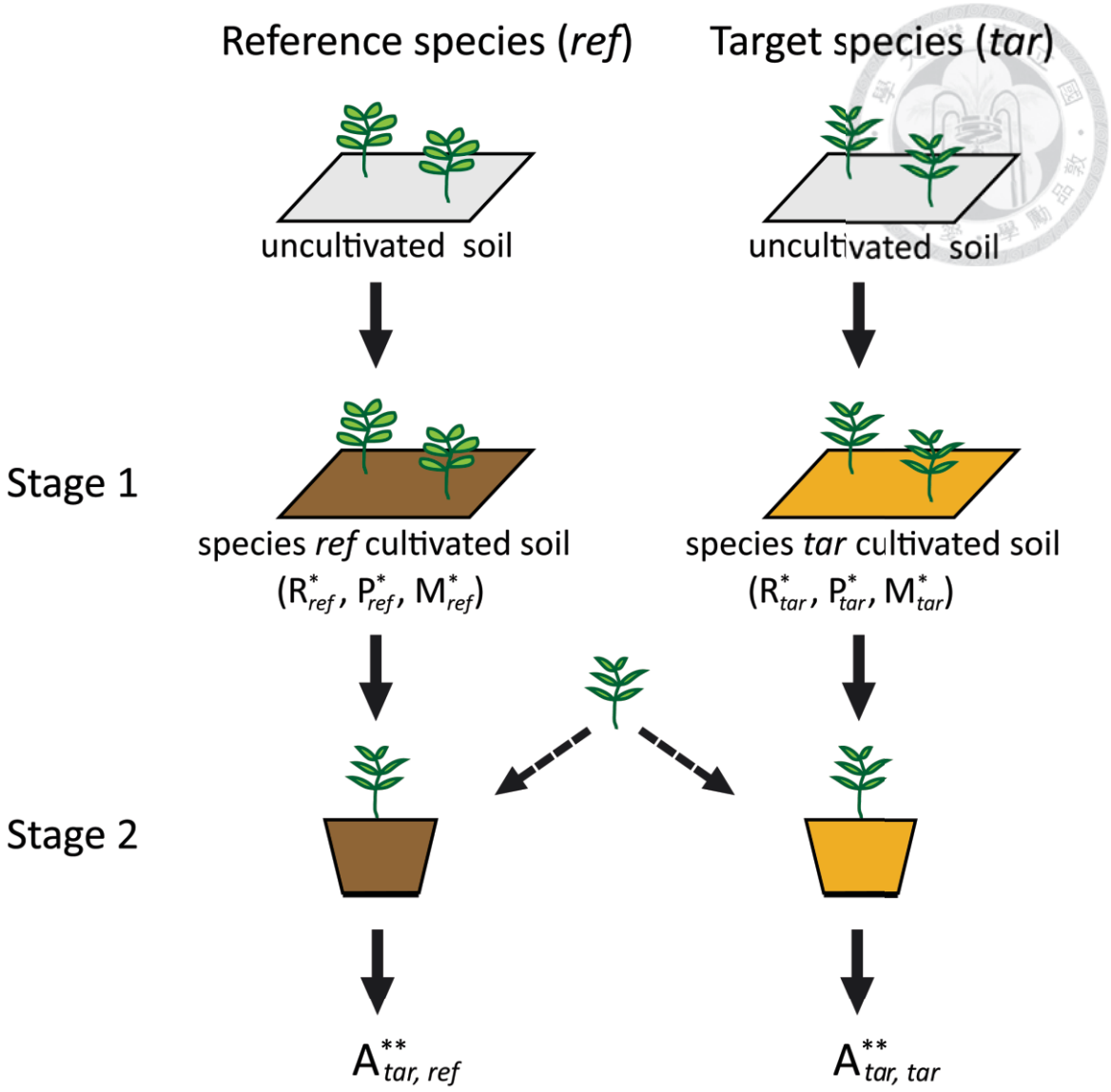


Figure. 2. Schematic diagram of model analysis. Stage 1 represents the stage when plants cultivate nearby soils for a long period of time, R_k^*, P_k^* and M_k^* represent extracted equilibrium values from the model using parameters for plant *k*. Stage 2 represents growth dynamics of seedlings in the greenhouse where $A_{tar,k}^{**}$ represents equilibrium adult density from the sub-model and is interpreted as growth response of the target plant species in soils cultivated by plant *k* (*k* = *ref* or *tar*).

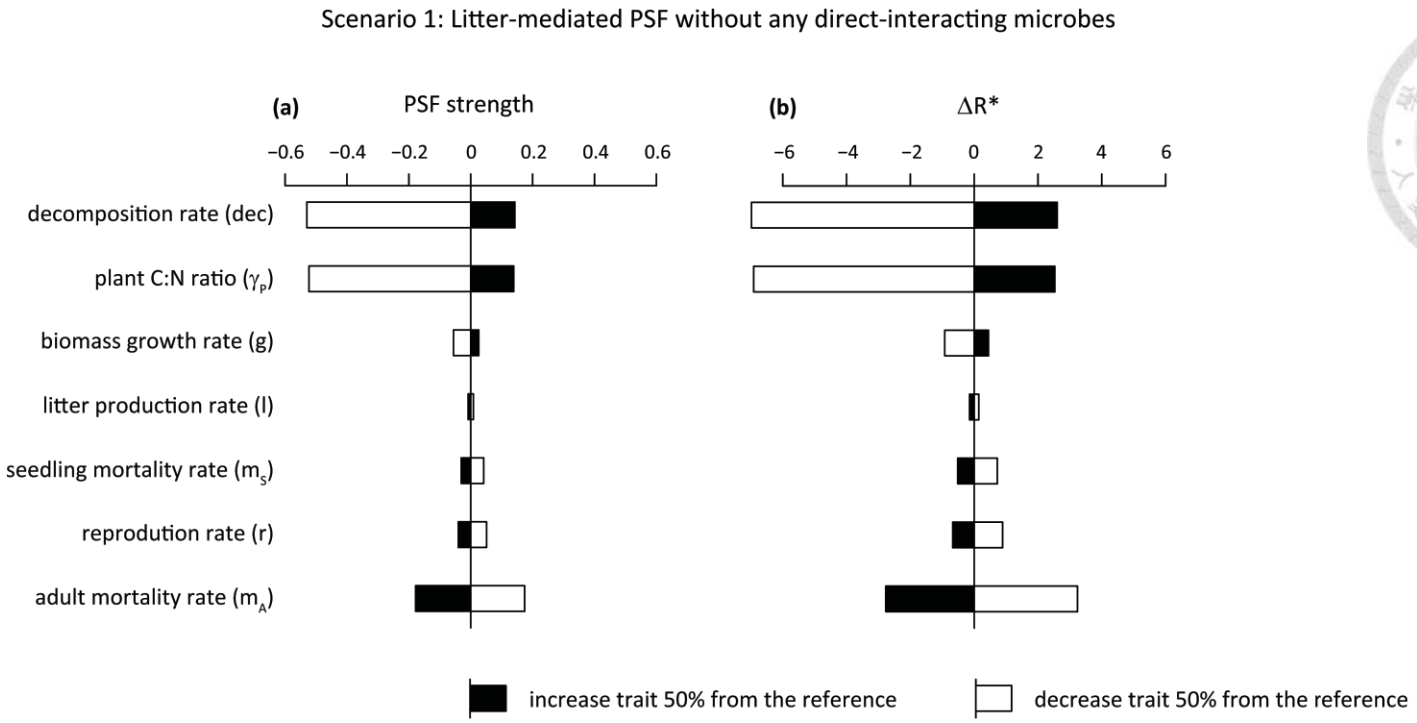


Figure. 3. Effects of ±50% deviation of each functional trait from the reference value for the scenario only considering litter-mediated PSF without any direct-interacting microbes. Closed bars represent (a) PSF strength and (b) change in equilibrium soil nitrogen level (represented as $R^*_{tar} - R^*_{ref}$) resulting from 50% increase of the trait value, whereas opened bars represent effect size of 50% decrease from the reference value. Traits are sorted from positive to negative PSF strength resulting from positive deviation of traits.

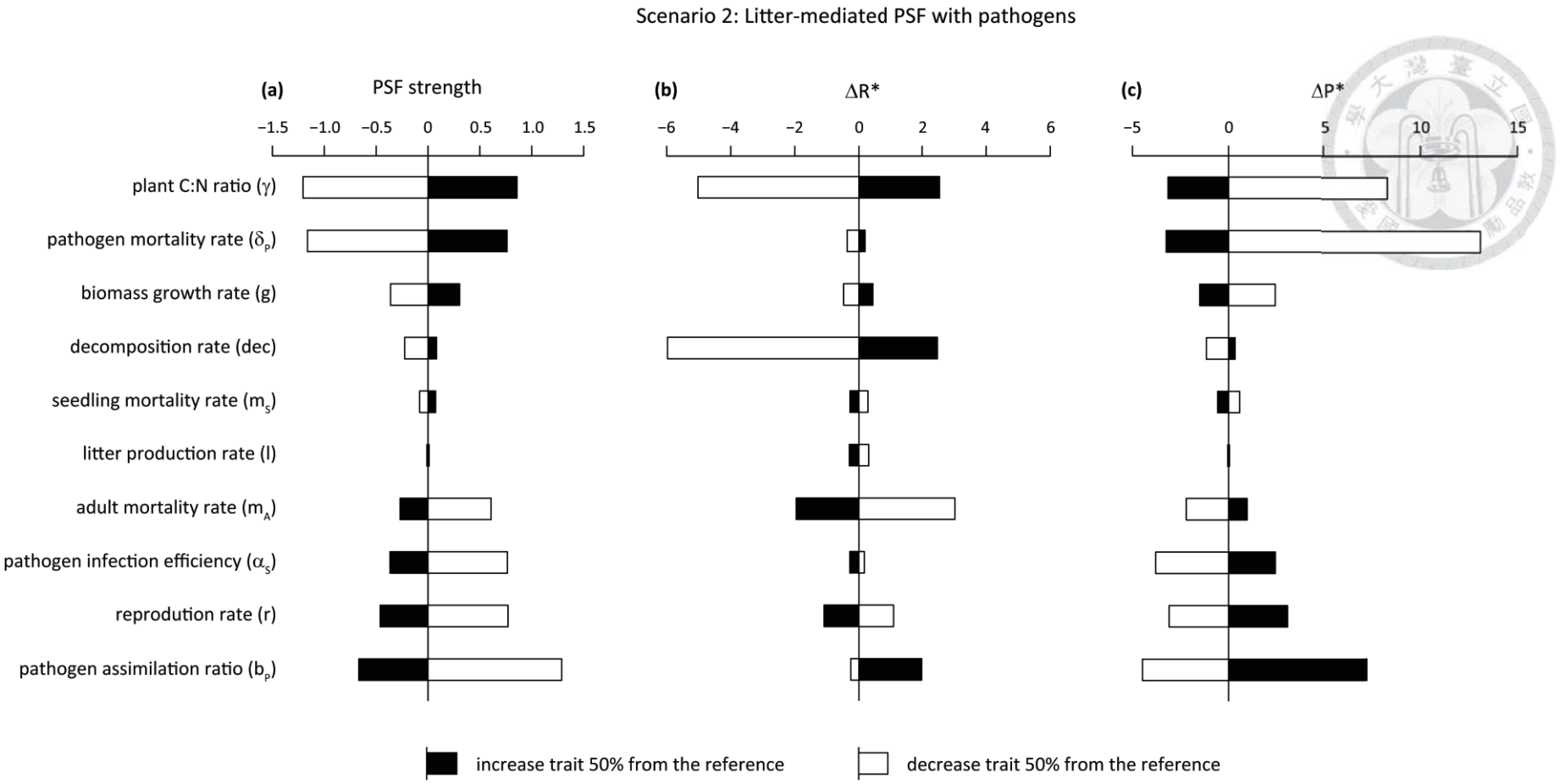


Figure. 4. Effects of ±50% deviation of each functional trait from the reference value for the scenario with litter-mediated PSF and pathogens. Closed bars represent (a) PSF strength, (b) change in equilibrium soil nitrogen level (represented as $R^*_{tar} - R^*_{ref}$), and (c) change in equilibrium pathogen level (represented as $P^*_{tar} - P^*_{ref}$) resulting from 50% increase of the trait value, whereas opened bars represent effect size of 50% decrease from the reference value. Traits are sorted from positive to negative PSF strength resulting from positive deviation of traits.

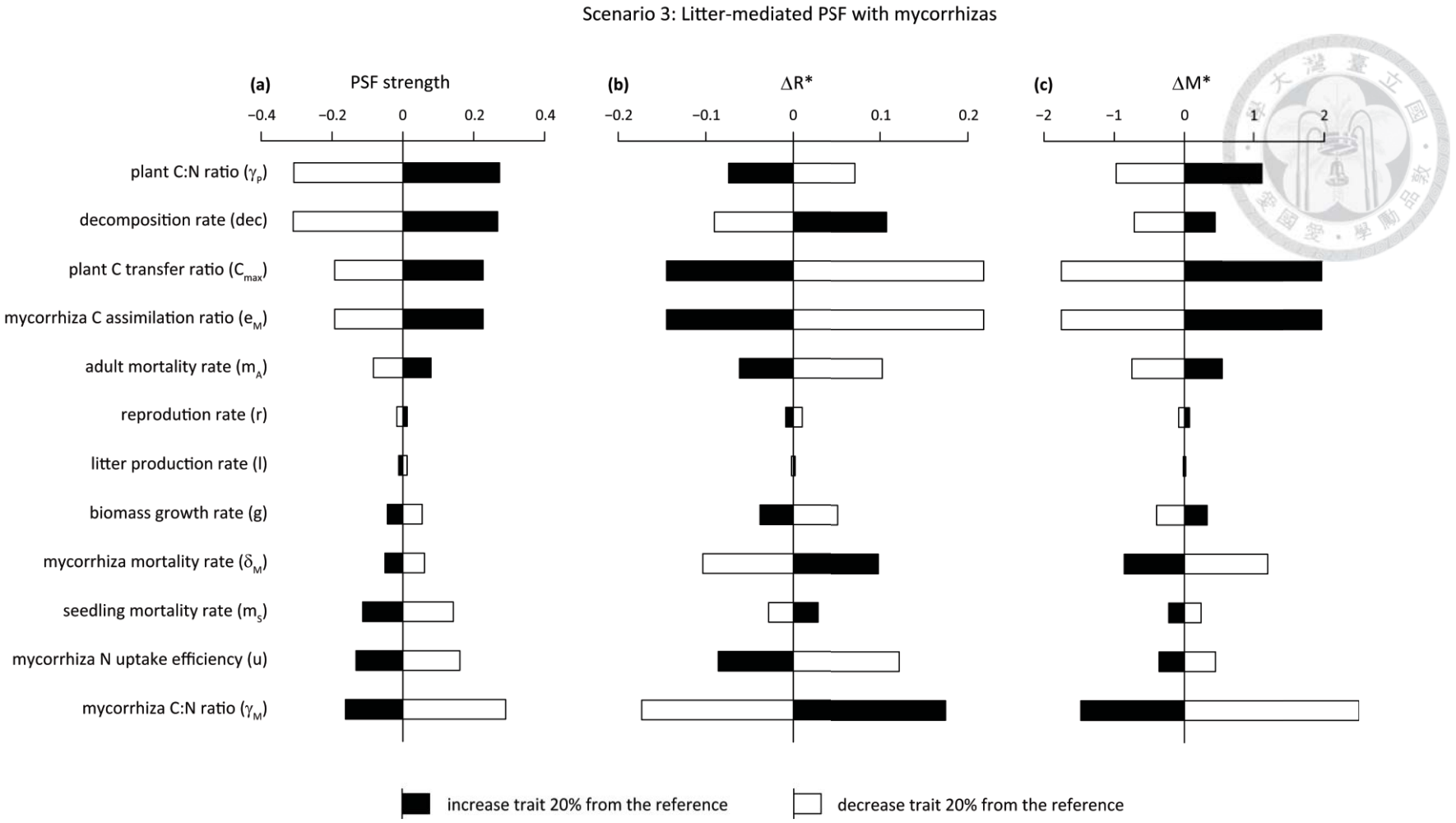


Figure. 5. Effects of ±20% deviation of each functional trait from the reference value for the scenario with litter-mediated PSF and mycorrhizas. Closed bars represent (a) PSF strength, (b) change in equilibrium soil nitrogen level (represented as $R^*_{tar} - R^*_{ref}$), and (c) change in equilibrium mycorrhiza level (represented as $M^*_{tar} - M^*_{ref}$) resulting from 20% increase of the trait value, whereas opened bars represent effect size of 20% decrease from the reference value. Traits are sorted from positive to negative PSF strength resulting from positive deviation of traits.

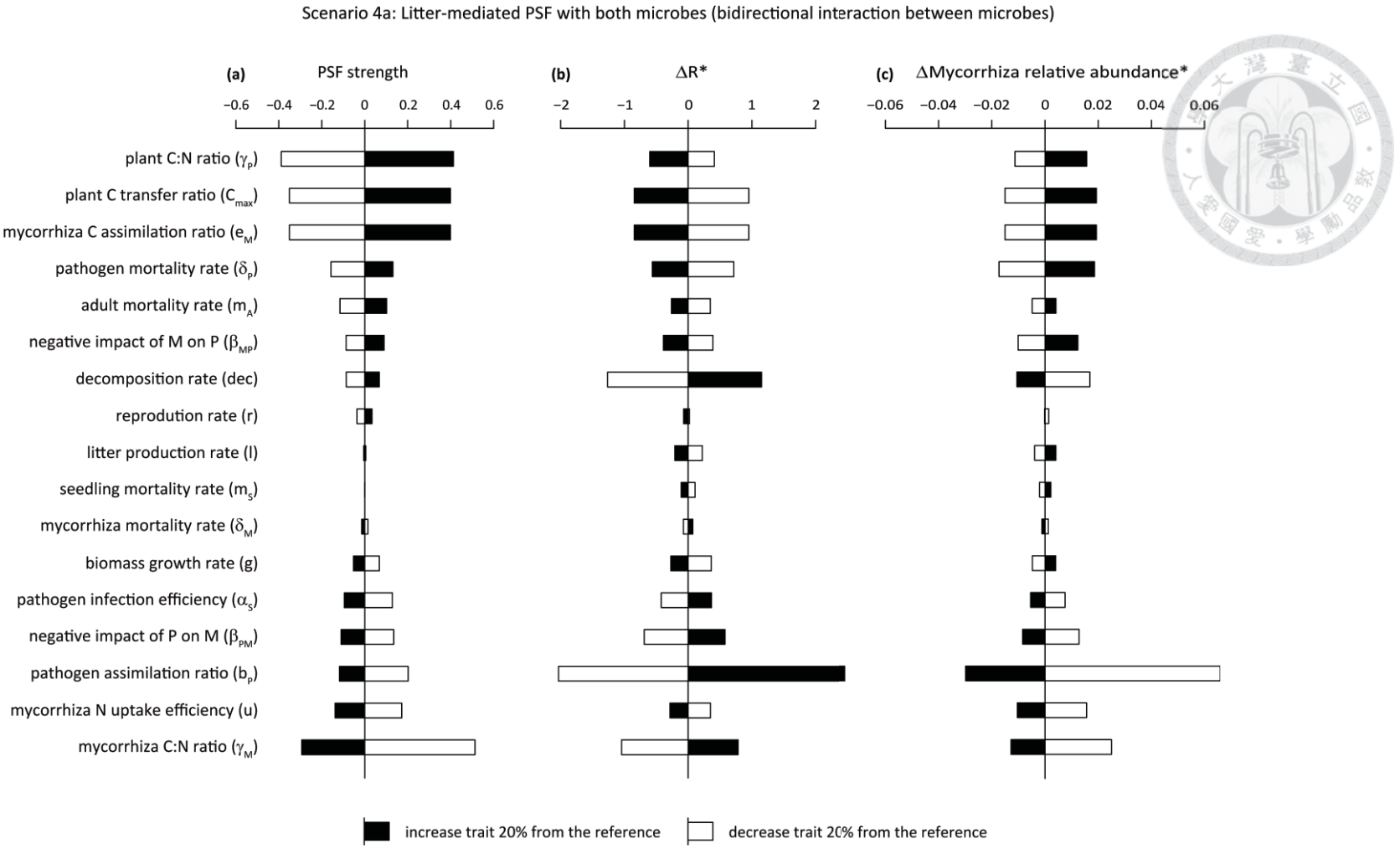


Figure. 6. Effects of ±20% deviation of each functional trait from the reference value for the scenario with litter-mediated PSF and both microbes, where negative effects is bidirectional between microbes. Closed bars represent (a) PSF strength, (b) change in equilibrium soil nitrogen level, and (c) change in equilibrium mycorrhiza relative abundance resulting from 20% increase of the trait value, whereas opened bars represent effect size of 20% decrease of trait value. Traits are sorted according to PSF strength resulting from positive deviation of traits.

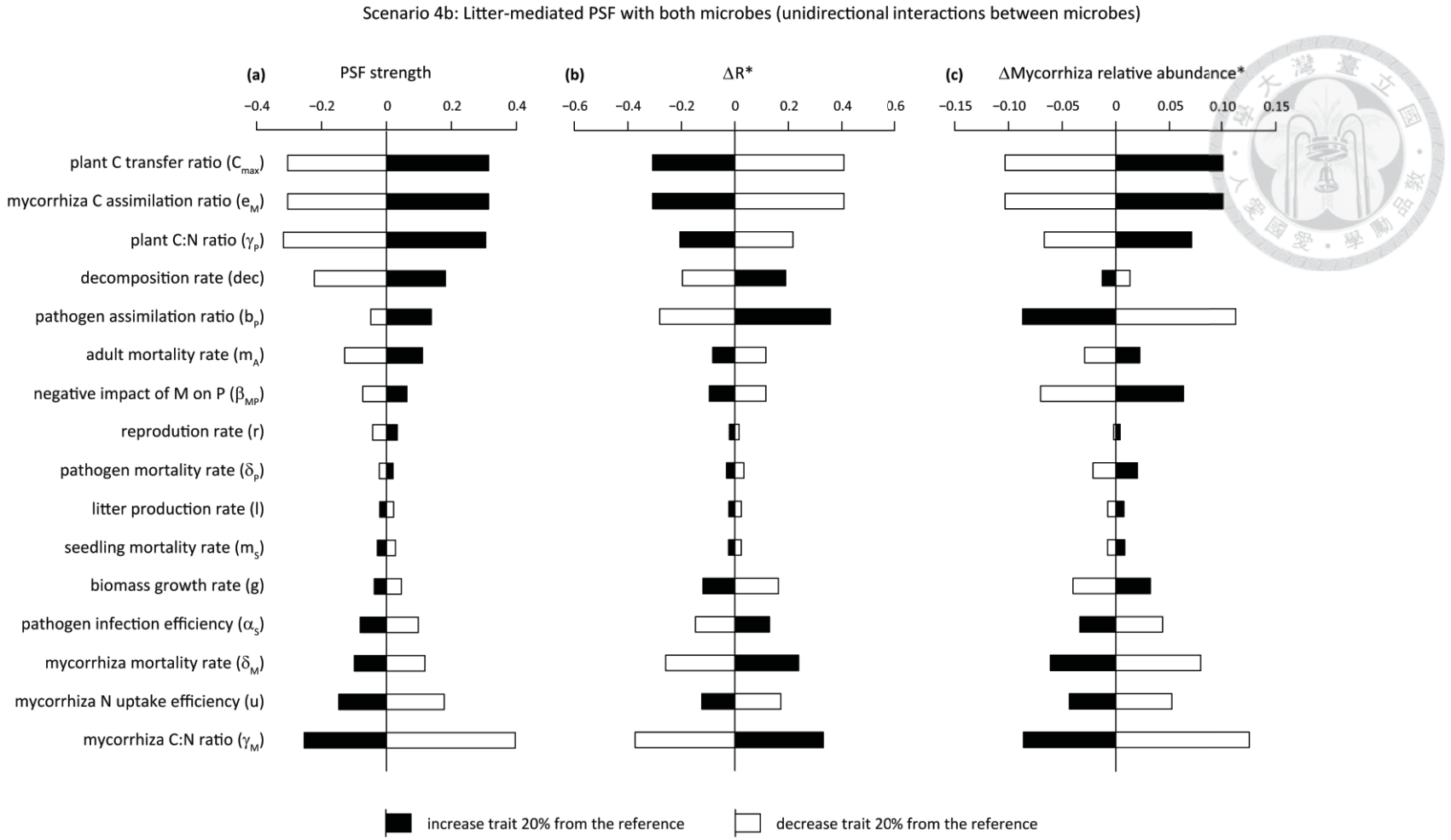


Figure. 7. Effects of ±20% deviation of each functional trait from the reference value for the scenario with litter-mediated PSF and both microbes, where negative effect is unidirectional from mycorrhizas on pathogens. Closed bars represent (a) PSF strength, (b) change in soil nitrogen level, and (c) change in equilibrium mycorrhiza relative abundance resulting from 20% increase of the trait value, whereas opened bars represent effect size of 20% decrease of trait value. Traits are sorted according to PSF strength resulting from positive deviation of traits.

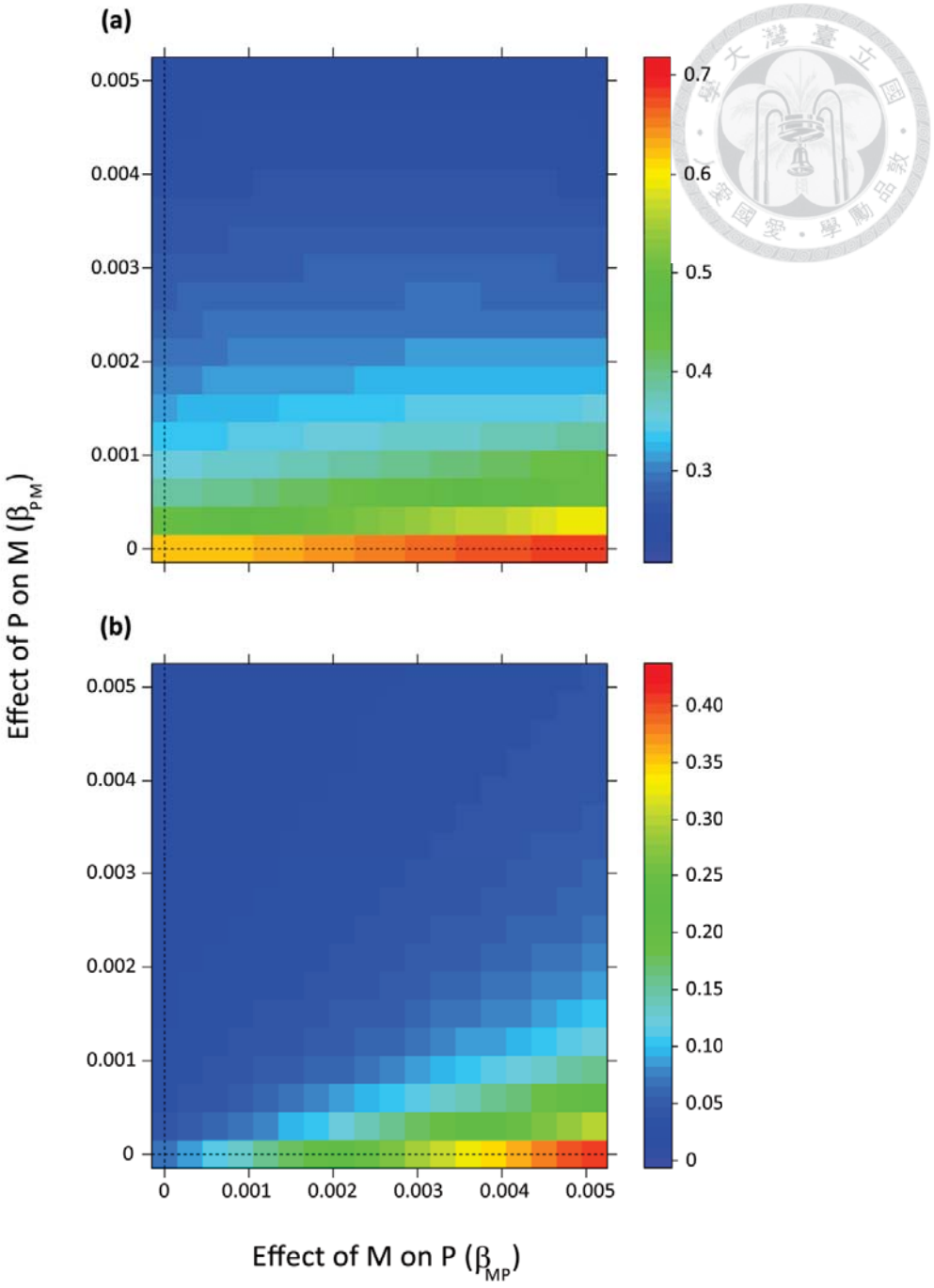


Fig. 8. Effects of doubling litter decomposability from the reference value under different combinations of interaction strength between pathogens and mycorrhizas. (a) PSF strength and (b) mycorrhiza relative abundance at equilibrium resulting from positively deviated litter decomposability

Tables

Table 1. Model equations

Seedling density (S)

$$\frac{dS}{dt} = rRA\left(1 - \frac{A}{A_{\max}}\right) - gRS\left(1 - \frac{A}{A_{\max}}\right) + r_{(S,A,M,R)} - G_{(S,A,M,R)} - m_S S - \alpha_S SP \quad (1)$$

= (reproduction) – (maturation and growth) + (mycorrhizal enhancement of reproduction) – (mycorrhizal enhancement of maturation and growth) – (natural mortality) – (pathogen-induced mortality)

Adult density (A)

$$\frac{dA}{dt} = gRS \cdot \left(1 - \frac{A}{A_{\max}}\right) + G_{(S,A,M,R)} - m_A A - \alpha_A AP \quad (2)$$

= (maturation and growth of seedlings to adults) + (mycorrhizal enhancement of maturation and growth) – (natural mortality) – (pathogen-induced mortality)

Mycorrhiza nitrogen content (M)

$$\frac{dM}{dt} = \mu_{(S,A,M,R)} - \delta_M M - \beta_{PM} MP \quad (3)$$

= (growth sustained by negotiation with plants) – (natural mortality and tissue turnover) – (negative impact of pathogens)

Pathogen nitrogen content (P)

$$\frac{dP}{dt} = b_P \cdot \left[\left(\alpha_S SP \cdot B_S + \alpha_A AP \cdot B_A\right) \cdot \frac{1}{\gamma_P}\right] - \delta_P P - \beta_{MP} PM \quad (4)$$

= (growth sustained by consumption of plants) – (natural mortality and tissue turnover) – (negative impact of mycorrhiza)

Table 1. *(Continued)*

Litter nitrogen content (L)

$$\frac{dL}{dt} = \left(m_S S \cdot B_S + m_A A \cdot B_A\right) \cdot \frac{1}{\gamma_P} + f_P \cdot \left[\left(\alpha_S SP \cdot B_S + \alpha_A AP \cdot B_A\right) \cdot \frac{1}{\gamma_P}\right] + lRA \cdot \left(1 - \frac{A}{A_{\max}}\right) + l_{(S,A,M,R)} - dec \cdot L - \varphi \cdot L \quad (5)$$

= (dead plant nitrogen due to natural mortality) + (dead plant nitrogen due to pathogen consumption) + (litter production from adults) + (mycorrhizal enhancement of litter production) – (decomposition) – (litter leaching)

Soil inorganic nitrogen content (R):

$$\frac{dR}{dt} = I - Le \cdot R + dec \cdot L + \left(1 - b_P - f_P\right) \cdot \left[\left(\alpha_S SP \cdot B_S + \alpha_A AP \cdot B_A\right) \cdot \frac{1}{\gamma_P}\right] + \left(\delta_P P + \delta_M M + \beta_{MP} PM + \beta_{PM} MP\right) - uRM - \left[\left(l + r \cdot \frac{B_S}{\gamma_P}\right) \cdot RA + gRS \cdot \left(\frac{B_A}{\gamma_P} - \frac{B_S}{\gamma_P}\right)\right] \cdot \left(1 - \frac{A}{A_{\max}}\right) \quad (6)$$

= (external supply) – (soil nitrogen leaching) + (litter decomposition) + (free nitrogen due to pathogen-induced mortality of plants) + (free nitrogen due to microbial turnover) – (mycorrhizal nitrogen uptake) – (plant nitrogen uptake)

Table 2. Model equations of mycorrhizal-enhancement of plant reproduction, plant maturation, mycorrhiza growth and litter production under different mycorrhiza nutrient limitation status (i.e. carbon-limited or nitrogen-limited)

	Mycorrhiza nutrient limitation status	
	carbon-limited	nitrogen-limited
reproduction: $r_{(S,A,M,R)}$	$\left[r\left(l+r\cdot\frac{B_S}{\gamma_P}\right)^{-1}\cdot uRM_A - rRA\left(1-\frac{A}{A_{\max}}\right)\cdot C_{\max}e_M\frac{\gamma_P}{\gamma_M}\right]$	$\left[r\left(l+r\cdot\frac{B_S}{\gamma_P}\right)^{-1}\cdot n_{\min}uRM_A\right]$
maturation: $G_{(S,A,M,R)}$	$\left[\left(\frac{B_A}{\gamma_P}-\frac{B_S}{\gamma_P}\right)^{-1}\cdot uRM_S - gRS\left(1-\frac{A}{A_{\max}}\right)\cdot C_{\max}e_M\frac{\gamma_P}{\gamma_M}\right]$	$\left[\left(\frac{B_A}{\gamma_P}-\frac{B_S}{\gamma_P}\right)^{-1}\cdot n_{\min}uRM_S\right]$
mycorrhizal growth: $\mu_{(S,A,M,R)}$	$\left[\left(l+r\cdot\frac{B_S}{\gamma_P}\right)\cdot RA+gRS\cdot\left(\frac{B_A}{\gamma_P}-\frac{B_S}{\gamma_P}\right)\right]\cdot\left(1-\frac{A}{A_{\max}}\right)\cdot\left(C_{\max}e_M\cdot\frac{\gamma_P}{\gamma_M}\right)$	$(1-n_{\min})\cdot uRM$
litter production: $l_{(S,A,M,R)}$	$\left[l\left(l+r\cdot\frac{B_S}{\gamma_P}\right)^{-1}\cdot uRM_A - lRA\left(1-\frac{A}{A_{\max}}\right)\cdot C_{\max}e_M\frac{\gamma_P}{\gamma_M}\right]$	$\left[l\left(l+r\cdot\frac{B_S}{\gamma_P}\right)^{-1}\cdot n_{\min}uRM_A\right]$
maturation in sub-model: $g_{i(S_k^*,A_k^*,M_k^*,R_k^*)}$ †	$\left[\left(\frac{B_{A,i}}{\gamma_{P,i}}-\frac{B_{S,i}}{\gamma_{P,i}}\right)^{-1}\cdot u_iR_k^*M_{S,k}^* - g_iR_k^*S_i\cdot C_{\max,i}e_{M,i}\cdot\frac{\gamma_{P,i}}{\gamma_{M,i}}\right]$	$\left[\left(\frac{B_{A,i}}{\gamma_{P,i}}-\frac{B_{S,i}}{\gamma_{P,i}}\right)^{-1}\cdot n_{\min,i}u_iR_k^*M_{S,k}^*\right]$

† equilibrium values extracted from the model under different scenarios for plant *k* (*k* = *ref* or *tar*) are used to represent soil properties (i.e. R_k^*, P_k^* and M_k^*)

Table 3. Sub-model equations used for model analysis via simulating field experiments (growth of plant *i* in soil cultivated by plant *k* †)

Seedling dynamics of plant *i* in soil *k*

$$\frac{dS_i}{dt} = -g_i R_k^* S_i - g_{i(S_k^*, A_k^*, M_k^*, R_k^*)} - m_{S,i} S_i - \alpha_{S,i} P_k^* S_i \quad (7)$$

= (maturation and growth) – (mycorrhizal enhancement of maturation and growth) – (natural mortality) – (pathogen-induced mortality)

Adult dynamics (growth response) of plant *i* in soil *k*

$$\frac{dA_i}{dt} = g_i R_k^* S_i + g_{i(S_k^*, A_k^*, M_k^*, R_k^*)} \quad (8)$$

= (maturation and growth) + (mycorrhizal enhancement of maturation and growth)

† equilibrium values extracted from the model under different scenarios for plant *k* (*k* = *ref* or *tar*) are used to represent soil properties (i.e. R_k^*, P_k^* and M_k^*)

Table 4. Model parameters and state variables. Parameters explained in boldface indicate that they are the traits examined.

Symbol	Interpretation	Units	Default value	References **
S	Seedling density	#ind. m^{-2}	—	—
A	Adult density	#ind. m^{-2}	—	—
M	Mycorrhiza nitrogen content	g N m^{-2}	—	—
P	Pathogen nitrogen content	g N m^{-2}	—	—
L	Litter nitrogen content	g N m^{-2}	—	—
R	Soil nitrogen content	g N m^{-2}	—	—
r	**Plant reproduction rate per unit nitrogen**	g N^{-1} m^2 day^{-1}	0.3	1,2
g	**Seedling biomass growth rate per unit nitrogen**	g N^{-1} m^2 day^{-1}	9×10^{-7}	3
m_A	**Adult mortality rate**	day^{-1}	0.00005	5,6
m_S	**Seedling mortality rate**	day^{-1}	0.008	1,2
l	**Litter production rate of adults per unit nitrogen**	g C g N^{-1} day^{-1} $ind.^{-1}$	0.008	6,7
dec	**Litter decomposition rate**	day^{-1}	0.006	8
γ_P	**Plant tissue carbon:nitrogen ratio**	—	40	4
α_S	**Pathogens infection efficiency of seedling**	g C^{-1} m^2 day^{-1}	0.008	1,2
α_A	**Pathogens infection efficiency of adult**	g C^{-1} m^2 day^{-1}	$\alpha_S \times 10^{-4}$	—
b_P	**Pathogen assimilation ratio of plant nitrogen**	—	0.6	—
δ_P	**Pathogen mortality rate**	day^{-1}	0.01	9
f_P	**Litter return ratio flowing pathogen infection**	—	0.35	—
u	**Mycorrhiza nitrogen uptake coefficient**	g N^{-1} m^2 day^{-1}	0.06	10
C_{max}	**Maximum carbon transfer ratio from plant**	—	0.2	10

Table 4. *(Continued)*

e_M	**Mycorrhiza carbon assimilation ratio**	—	0.6	10
γ_M	**Mycorrhiza carbon:nitrogen ratio**	—	8	11
δ_M	**Mycorrhiza mortality rate**	day^{-1}	0.01	12
β_{MP}	**Negative impact coefficient of mycorrhiza on pathogen**	$g\ N^{-1}\ m^2\ day^{-1}$	0.005	13
β_{PM}	**Negative impact coefficient of pathogen on mycorrhiza**	$g\ N^{-1}\ m^2\ day^{-1}$	0.005	13
n_{min}	Minimum transfer ratio of mycorrhiza uptake nitrogen	—	0.2	—
A_{max}	Maximum adult density the system can support	$ind.\ m^{-2}$	0.5	3
B_A	Carbon biomass of individual adult	g C	130000	3,14
B_S	Carbon biomass of individual seedling	g C	10	15
I	Deposition input to the ecosystem	$g\ N\ m^{-2}\ day^{-1}$	0.005	7,16
Le	Soil nitrogen leaching rate	day^{-1}	0.0002	7,16
φ	Litter leaching rate	day^{-1}	0.00008	16

**References (see Appendix S4 for detailed citation):

(1) Augspurger (1983) (2) Bell *et al*. (2006) (3) Pyle *et al*. (2008) (4) Cernusak *et al*. (2010) (5) King *et al*. (2006) (6) Malhi *et al*. (2009) (7) Vitousek & Sanford (1986) (8) Kurokawa & Nakashizuka (2008) (9) Hancock (1981) (10) Bryla & Eissenstat (2005) (11) Hodge & Fitter (2010) (12) staddon *et al*. (2003) (13) Sikes *et al*. (2009) (14) Clark *et al*. (2001) (15) Markesteijn & Poorter (2009) (16) Menge *et al*. (2009)

Table 5. Summary of PSF directions resulting from positive deviation of each trait under different microbial composition scenarios. Directions in boldface indicates that the resulting PSF strength is significant based on the analysis in Appendix S2. NA represents that the trait is not considered under the specific microbial composition.

	r	g	m_A	m_S	l	dec	γ_P	α_S	b_P	δ_P	u	$C_{\max}$	e_M	γ_M	δ_M	β_{MP}	β_{PM}
scenario 1	-	+	**-**	-	-	**+**	**+**	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
scenario 2	-	+	-	+	-	+	**+**	-	**-**	**+**	NA	NA	NA	NA	NA	NA	NA
scenario 3	+	-	+	**-**	-	**+**	**+**	NA	NA	NA	**-**	**+**	**+**	**-**	-	NA	NA
scenario 4a	+	-	+	+	+	+	**+**	-	-	+	-	**+**	**+**	**-**	-	+	NA
scenario 4b	+	-	+	-	-	**+**	**+**	-	+	+	-	**+**	**+**	**-**	-	+	-

Appendix S1: PSF strength using trait values with larger deviation from the reference plant

In this section I examine the effects of larger positive (to maximum 300%) and negative (to minimum 10%) deviation of trait values from the reference plant on PSF strength, in order to check the robustness of results based on smaller deviation range (Fig. 3-7). Each line in Fig.S1-S5 represents the PSF strength due to the corresponding deviation of one specific trait (0% represents that the target plant is identical to the reference plant and thus PSF strength is 0). Larger deviation of the trait values showed qualitatively the same pattern as the ranking of functional traits based on their resulting PSF strength is almost identical to that in Fig. 3-7. Note that effect sizes of some traits were not simulated to largest range in order to remain a realistic range for the parameter.

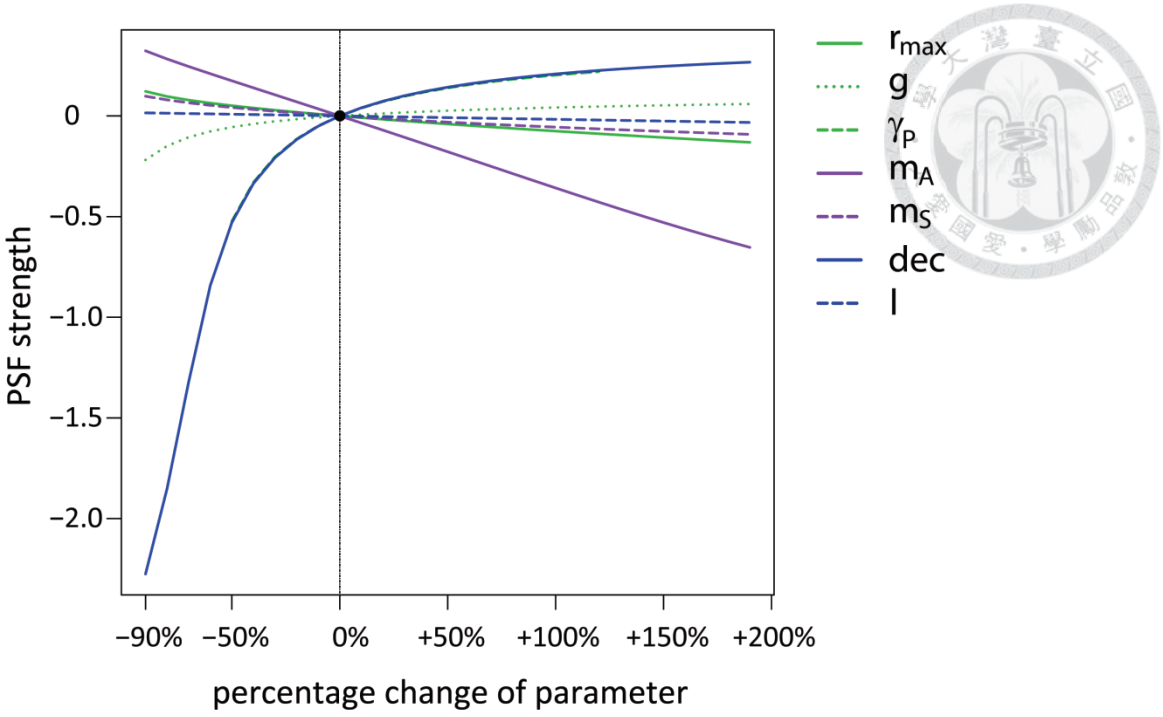


Figure. S1. Effect sizes of larger deviation of functional traits on PSF strength for the scenario only considering litter-mediated PSF without any direct-interacting microbes. The x-axis represents percentage change of trait value from the reference plant (i.e. 10% - 300% of the original value). Each line represents the effect size of one specific functional trait.

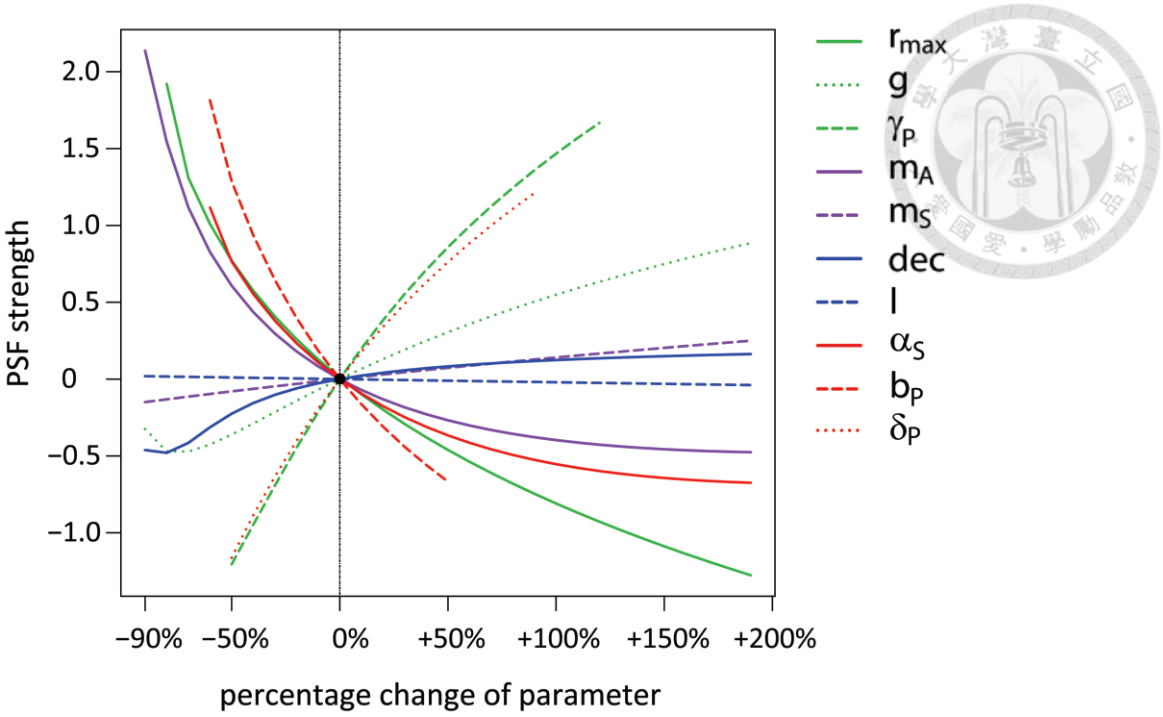


Figure. S2. Effect sizes of larger deviation of functional traits on PSF strength for the scenario with litter-mediated PSF and pathogens. The x-axis represents percentage change of trait value from the reference plant (i.e. 10% - 300% of the original value). Each line represents the effect size of one specific functional trait.

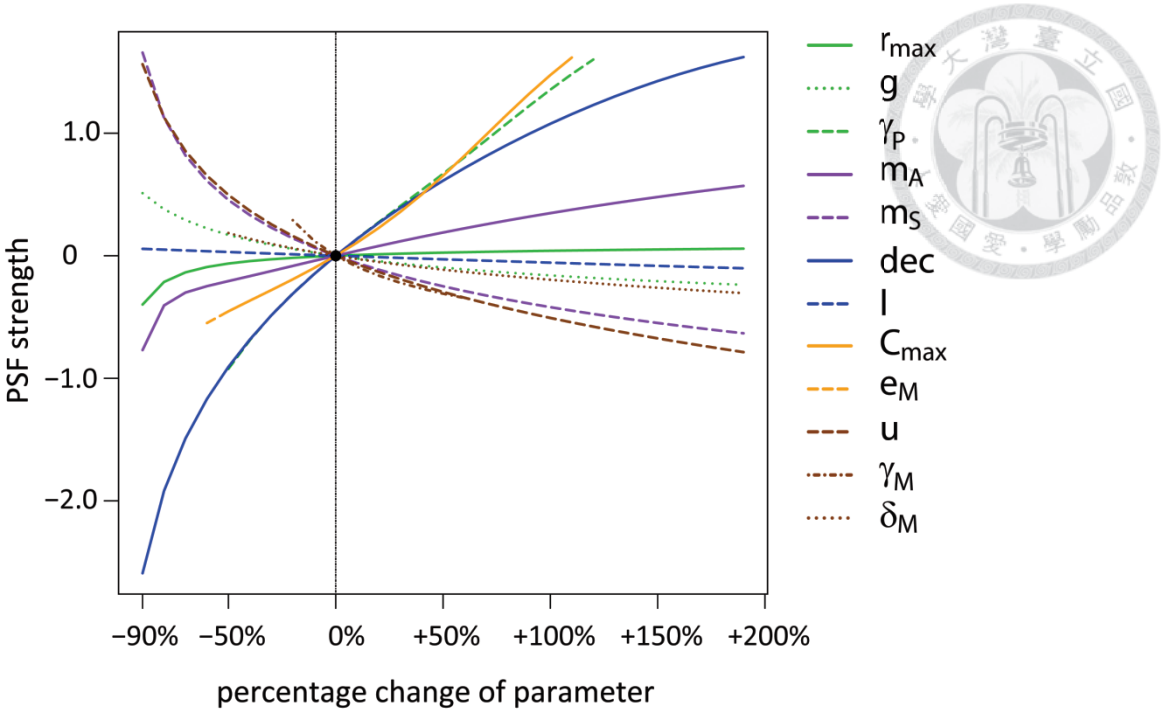


Figure. S3. Effect sizes of larger deviation of functional traits on PSF strength for the scenario with litter-mediated PSF and mycorrhizas. The x-axis represents percentage change of trait value from the reference plant (i.e. 10% - 300% of the original value). Each line represents the effect size of one specific functional trait.

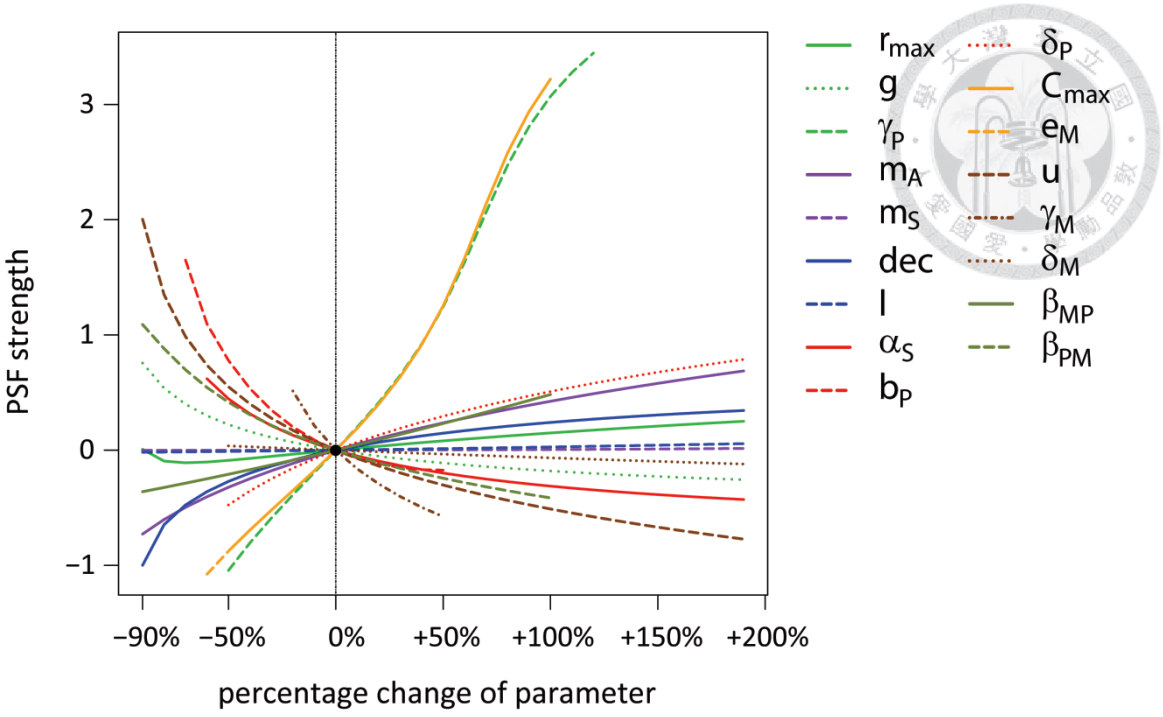


Figure. S4. Effect sizes of larger deviation of functional traits on PSF strength for the scenario with litter-mediated PSF and both microbes, where negative effects is set as bidirectional between pathogens and mycorrhizas. The x-axis represents percentage change of trait value from the reference plant (i.e. 10% - 300% of the original value). Each line represents the effect size of one specific functional trait.

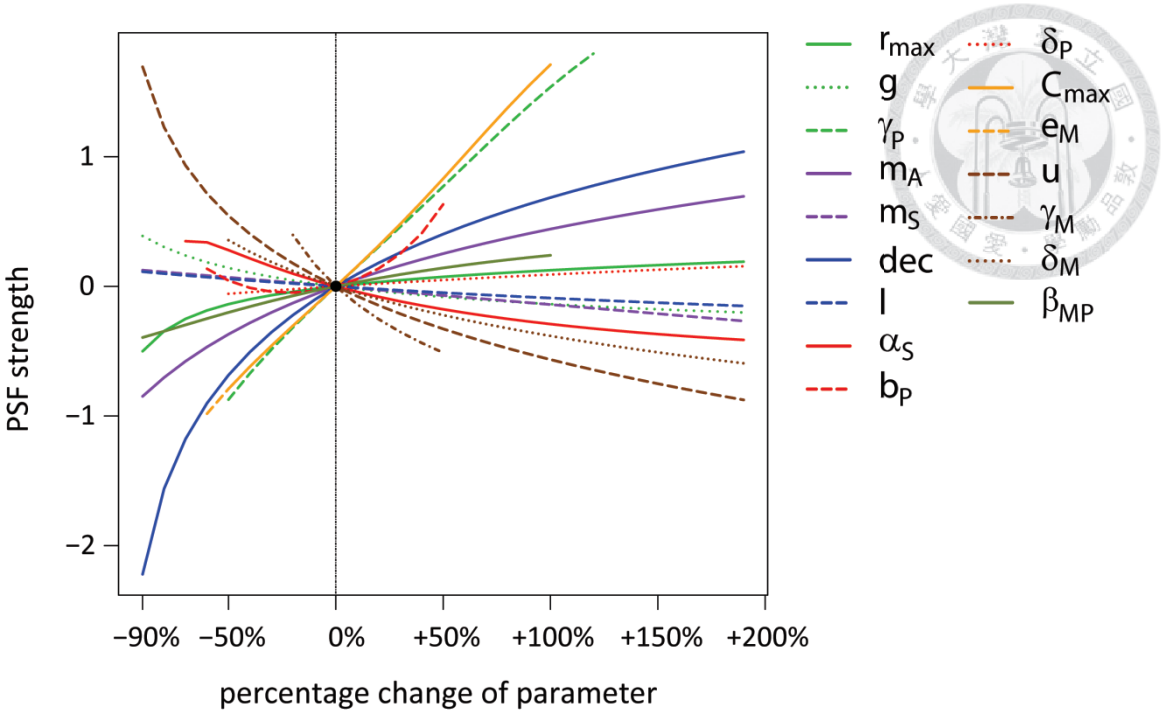


Figure. S5. Effect sizes of larger deviation of functional traits on PSF strength for the scenario with litter-mediated PSF and both microbes, where negative effects is set to be unidirectional as only mycorrhizas have negative impact on pathogens. The x-axis represents percentage change of trait value from the reference plant (i.e. 10% - 300% of the original value). Each line represents the effect size of one specific functional trait.

Appendix S2: Robustness of results based on randomly assembled target plants

In this section the robustness of the results is examined by allowing the target plant to have more than one trait deviated from the reference plant simultaneously. In this analysis, I assemble a target plant with random trait combination. When quantifying the effect size of a specific trait, I fix the deviation of that specific target trait at k % ($k > 0$) from the reference plant value, while randomly assigning trait deviation from a uniform distribution in between $\pm s$% for other traits (k and s depends on the specific microbial composition scenario in order to remain a realistic range for the parameter). I run the simulation 200 times for each scenario and calculate the PSF strength for each target plant by comparing its growth in conspecific cultivated soil to reference plant cultivated soil as described in the main text. Results of this analysis showed that the most influential traits remain unchanged even for a broad spectrum of randomly assigned target plants which have other traits also slightly different from the reference plant.

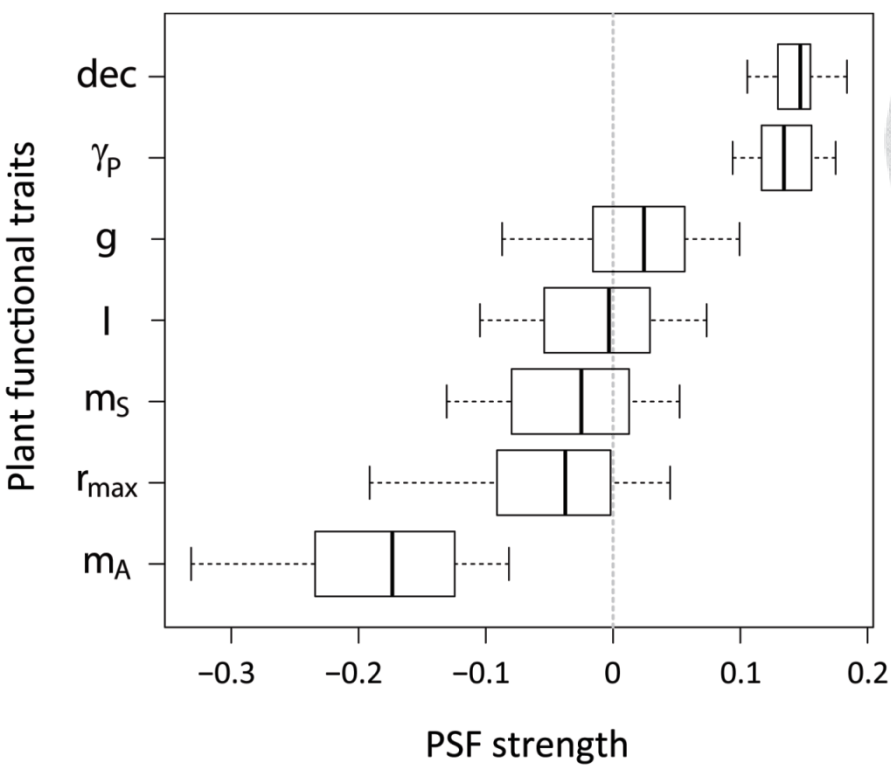


Figure. S6. PSF strength for target plants with random trait combinations for the scenario only considering litter-mediated PSF without any direct-interacting microbes. The target functional trait is set to deviate 50% from the reference plant while other traits were randomly assigned to deviate between ±20%. Traits are sorted from positive to negative according to the PSF strength resulting from positive deviation of traits in Fig. 3. Thick bars within boxes represent median values. Boxes enclose data points between the upper and lower quartiles, while whiskers extend from the quartiles to maximum 1.5 times of the data range.

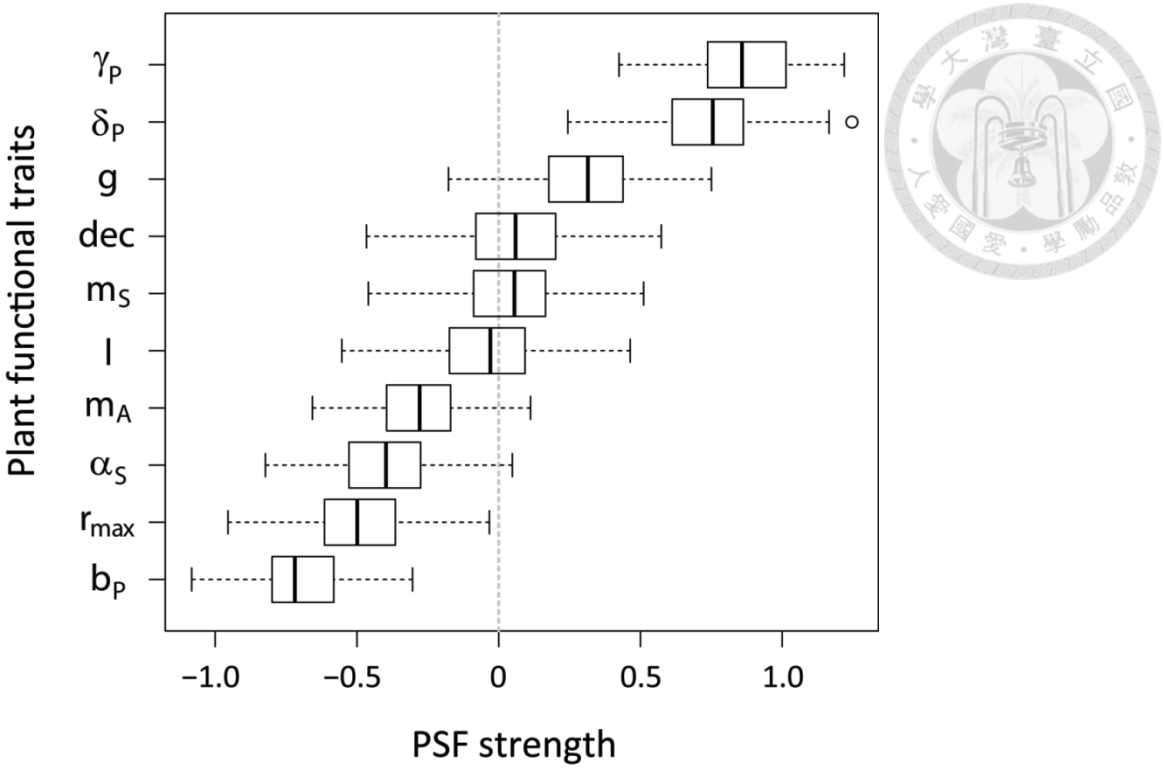


Figure. S7. PSF strength for target plants with random trait combinations for the scenario with litter-mediated PSF and pathogens. The target functional trait is set to deviate 50% from the reference plant while other traits were randomly assigned to deviate between ±20%. Traits are sorted from positive to negative according to the PSF strength resulting from positive deviation of traits in Fig. 4. Figure captions are as in Figure. S6. Open circles represent outliers exceeding 1.5 times of the data range from the quartiles.

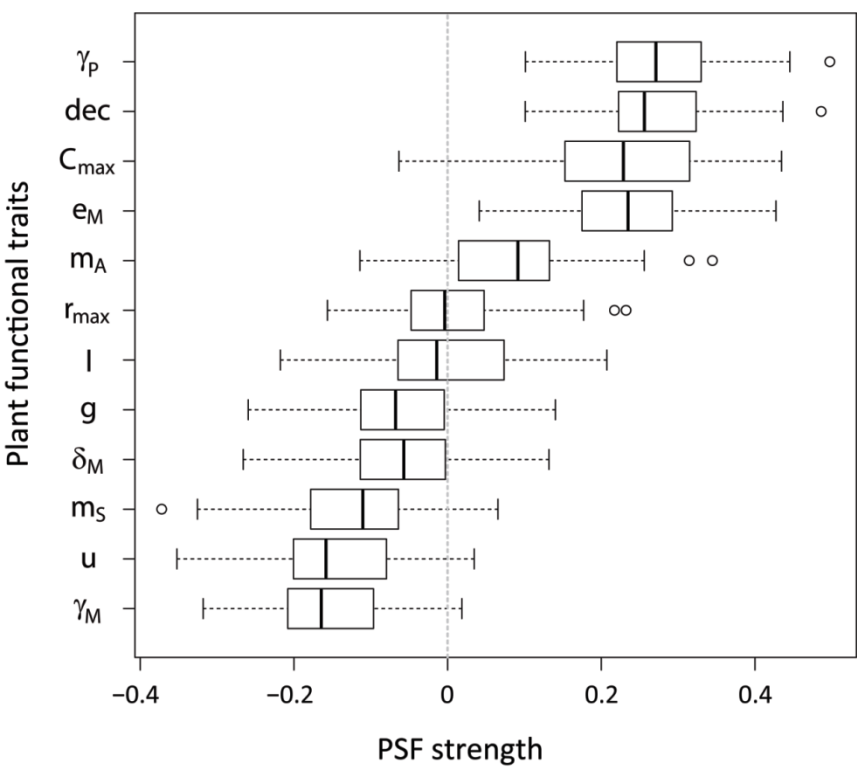


Figure. S8. PSF strength for target plants with random trait combinations for the scenario with litter-mediated PSF and mycorrhizas. The target functional trait is set to deviate 20% from the reference plant while other traits were randomly assigned to deviate between ±5%. Traits are sorted from positive to negative according to the PSF strength resulting from positive deviation of traits in Fig. 5. Figure captions are as in Figure. S6. Open circles represent outliers exceeding 1.5 times of the data range from the quartiles.

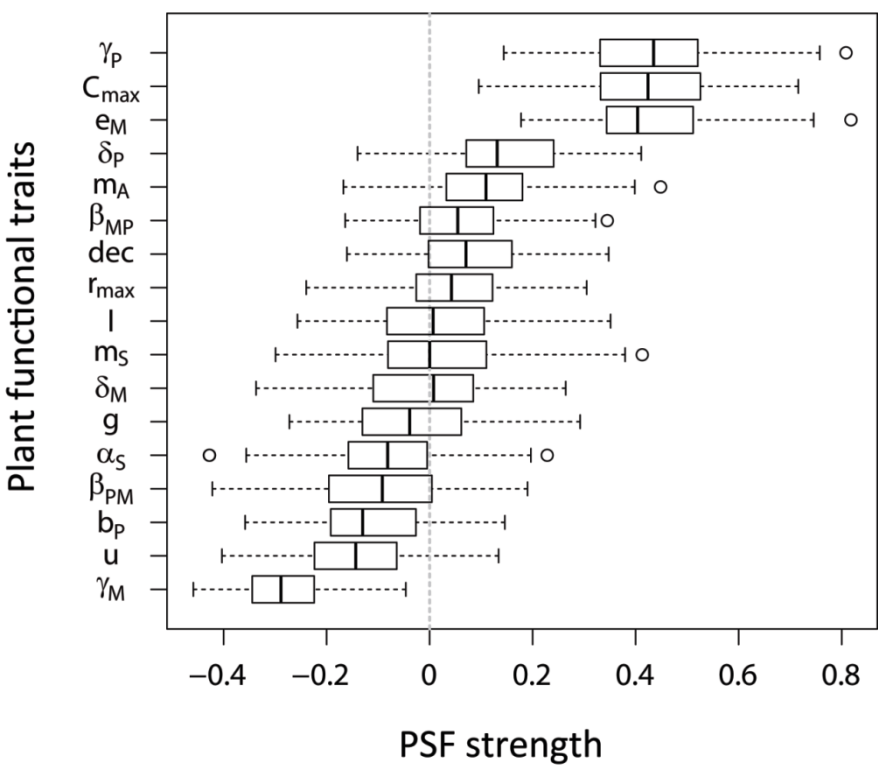


Figure. S9. PSF strength for target plants with random trait combinations for the scenario with litter-mediated PSF and both microbes, where negative effects are bidirectional between pathogens and mycorrhizas. The target functional trait is set to deviate 20% from the reference plant while other traits were randomly assigned to deviate between ±5%. Traits are sorted from positive to negative according to the PSF strength resulting from positive deviation of traits in Fig. 6. Figure captions are as in Figure. S6. Open circles represent outliers exceeding 1.5 times of the data range from the quartiles.

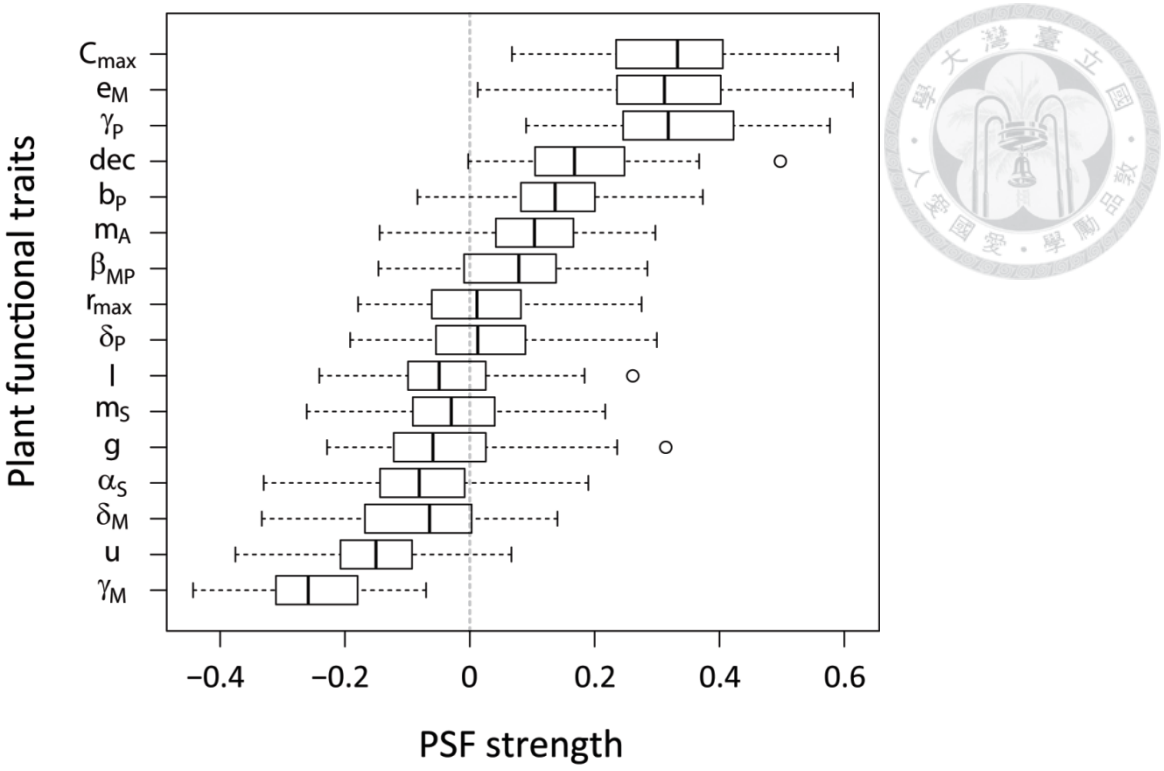


Figure. S10. PSF strength for target plants with random trait combinations for the scenario with litter-mediated PSF and both microbes, where negative effect is unidirectional between pathogens and mycorrhizas. The target functional trait is set to deviate 20% from the reference plant while other traits were randomly assigned to deviate between ±5%. Traits are sorted from positive to negative according to the PSF strength resulting from positive deviation of traits in Fig. 7. Figure captions are as in Figure. S6. Open circles represent outliers exceeding 1.5 times of the data range from the quartiles.

Appendix S3: Positivity of the microbe-free equilibrium and invasibility analysis for the microbes

In this section I derive the analytical solution of the microbe-free equilibrium E_{MF} $\left(S^*_{MF}, A^*_{MF}, L^*_{MF}, R^*_{MF} > 0\right)$. I perform mathematical analysis for the positivity of the equilibrium and invasibility analysis for the microbes to invade the microbe-free equilibrium in order to derive inequalities for choosing suitable parameter values.

The microbe-free equilibrium

I derive the microbe-free equilibrium via setting $P = M = 0$ and solve the following subset of our full model with only four state variables:

$$\frac{dS}{dt} = \left(rR^*_{MF}A^*_{MF} - gR^*_{MF}S^*_{MF}\right)\cdot\left(1-\frac{A^*_{MF}}{A_{\max}}\right) - m_S S^*_{MF} = 0 ,$$

$$\frac{dA}{dt} = gR^*_{MF}S^*_{MF}\cdot\left(1-\frac{A^*_{MF}}{A_{\max}}\right) - m_A A^*_{MF} = 0 ,$$

$$\frac{dL}{dt} = \left(m_S S^*_{MF}\cdot B_S + m_A A^*_{MF}\cdot B_A\right)\cdot\frac{1}{\gamma_P} + lR^*_{MF}A^*_{MF}\cdot\left(1-\frac{A^*_{MF}}{A_{\max}}\right) - dec\cdot L^*_{MF} - \phi\cdot L^*_{MF} = 0 ,$$

$$\frac{dR}{dt} = \left(I - Le\cdot R^*_{MF}\right) + dec\cdot L^*_{MF} - \left[\left(l + r\cdot\frac{B_S}{\gamma_P}\right)\cdot R^*_{MF}A^*_{MF} + gR^*_{MF}S^*_{MF}\cdot\left(\frac{B_A}{\gamma_P} - \frac{B_S}{\gamma_P}\right)\right]\cdot\left(1-\frac{A^*_{MF}}{A_{\max}}\right) = 0 .$$

The microbe-free equilibrium is solved as:

$$A^*_{MF} = \frac{A_{\max}}{-2a'}\cdot\left[a' - c' + \sqrt{\left(a'+c'\right)^2 - d'}\right] ,$$

$$S^*_{MF} = \eta A^*_{MF} ,$$

$$R^*_{MF} = \frac{IA_{\max} + \left(\frac{dec}{dec+\phi}\right)\cdot\left(m_S n_S \eta + m_A n_A\right)A_{\max}A^*_{MF}}{LeA_{\max} + A^*_{MF}\cdot\left(A_{\max} - A^*_{MF}\right)\cdot\left[rn_S + g\eta\cdot\left(n_A - n_S\right) + l\cdot\left(\frac{\phi}{dec+\phi}\right)\right]} = f_{\left(A^*_{MF}\right)} ,$$

$$L^*_{MF} = A^*_{MF}\cdot\left[m_S n_S \eta + m_A n_A + l\cdot f_{\left(A^*_{MF}\right)}\cdot\left(1-\frac{A^*_{MF}}{A_{\max}}\right)\right]\cdot\left(\frac{A^*_{MF}}{dec+\phi}\right) .$$

where,

$$\eta = \frac{-gm_A + \sqrt{g^2 m_A^2 + 4grm_S m_A}}{2gm_S},\ n_S = \frac{B_S}{\gamma_P},\ n_A = \frac{B_A}{\gamma_P},$$

$$a' = \left[g\eta^2 \left(\frac{dec}{dec+\phi} \right) m_S n_S - g\eta \left(\frac{\phi}{dec+\phi} \right) m_A n_A + (g\eta - r) m_A n_S - \left(\frac{\phi}{dec+\phi} \right) l m_A \right] A_{\max},$$

$$c' = g\eta I,\ d' = 4a' m_A Le.$$

Positivity analysis of the microbe-free equilibrium

In order to select parameter values that are able to obtain positive, and thus biologically reasonable, microbe-free equilibriums, I perform positivity analysis to derive the criteria of parameters that can insure $S_{MF}^*, A_{MF}^*, L_{MF}^*, R_{MF}^* > 0$. From the analytical form of E_{MF}, one can observe that the equilibrium will be positive as long as $A_{MF}^* > 0$, which gives the inequality:

$$\frac{m_A}{g\eta} < \frac{I}{Le}\ .$$

This inequality can be rewrite to give the following criteria for choosing parameters:

$$gr > \left(\frac{I}{Le} \right)^2 \cdot m_A m_S + \left(\frac{I}{Le} \right) \cdot g m_A\ .$$

Invasibility of pathogens into the microbe-free equilibrium

I perform invisibility analysis for the pathogen to invade into the microbe-free equilibrium. This is necessary for selecting suitable parameter values from empirical studies, which ensures the persistence of the pathogen with the plant population. I

evaluate the per capita growth rate of pathogens under the microbe-free equilibrium when its population size is low. Pathogens are able to invade the microbe-free equilibrium and then can persist when

$$\lim_{P\to 0}\frac{dP}{dt}\cdot\frac{1}{P}\bigg|_{E^*_{MF}} = b_P\cdot\left[\left(\alpha_S S^*_{MF}\cdot B_S+\alpha_A A^*_{MF}\cdot B_A\right)\cdot\frac{1}{\gamma_P}\right]-\delta_P>0\ ,$$

which is only possible when the following inequality is fulfilled:

$$\frac{1}{\alpha_S\eta n_S+\alpha_A n_A}\cdot\frac{\delta_P}{b_P}<A^*_{MF}\ .$$

Invasibility of mycorrhizas into the microbe-free equilibrium

I perform invisibility analysis for the mycorrhiza via evaluating the per capita growth rate of mycorrhiza under the microbe-free equilibrium when its population size is low. I perform the analysis for both carbon and nitrogen limiting situation. Mycorrhizas are always able to invade the microbe-free equilibrium when it is carbon limited. This is because population growth rate under such scenario is independent to mycorrhizal abundance but depends on plant population growth. Mycorrhizas which are nitrogen limited can invade and persist only when

$$\lim_{M\to 0}\frac{dM}{dt}\cdot\frac{1}{M}\bigg|_{E^*_{MF}} = \left(1-n_{\min}\right)uR^*_{MF}-\delta_M>0\ ,$$

which is only possible when the following inequality is fulfilled:

$$\frac{\delta_M}{\left(1-n_{\min}\right)u}>R^*_{MF}\ .$$

Appendix S4: Sources of parameter values used for the reference plant

In this Appendix I explain how the model parameters for the reference plant were derived from empirical studies. I considered a hypothetical tree species in the tropics as the reference plant species. Plant trait and environmental parameter values were thus obtained mainly from literature data reported from the tropical forest ecosystem.

Plant Reproduction rate (r) and seedling mortality rate (m_S) was derived from the study of Augspurger (1983) and Bell *et al.* (2006). Both studies focused on one dominant tree species, *Platypodium elegans* and *Sebastiana longicuspis*, respectively, and provided data for germinating seedling density and natural mortality. I assumed the reference plant species to have parameter values between the two plant species, so that trait values can fall within a realistic range when conducting simulations.

The living plant biomass was reported to be 133 Mg C ha^{-1} for an Amazonian forest (Pyle *et al.*, 2008). Under the assumption that at steady state one hectare can support the living of 1000 adult individual, the biomass of adults (B_A) was set as 130000 g C for one adult. The biomass of one seedling individual (B_S) was assumed to be 10 g C as Markesteijn & Poorter (2009) reported that seedling biomass after one growing season is between 1 to 35 g for 62 tree species in an Amazonian forest.

The value of plant C:N ratio is adapted from the study of Cernusak *et al.* (2010).

The study showed that leaf N:C ratio lies between 20 to 60 (mg N g^{-1} C) for 13 plant species. I assumed a C:N ratio (γ_P) value of 40 (g C g^{-1} N), which corresponds to a value within their reported range.

Seedling biomass growth rate was estimated from aboveground wood productivity, which is reported to lie between 2.11 to 4.33 Mg C ha^{-1} $year^{-1}$ for tropical forests (Pyle *et al.*, 2008; Malhi *et al.*, 2009). Under the assumed adult biomass and plant C:N ratio, I used 9×10^{-7} g N^{-1} m^2 day^{-1} as the growth parameter. For the adult mortality rate, it was estimated that 0.02 adult individuals dies per year (King *et al.*, 2006). Therefore, I assumed an adult mortality rate (m_A) of 5×10^{-5} day^{-1}.

The value of litter decomposition rate is based on the study by Kurokawa & Nakashizuka (2008), which reported that the litter decomposition rate for 40 tree species in Malaysian tropical forest lie between 0.67 to 4.85 $year^{-1}$. I thus assumed a decomposition rate (dec) of 0.006 day^{-1} for the reference species. Litter production rate of adults per unit N uptake (l) was estimated from litterfall data from tropical forests (Clark *et al.*, 2001; Malhi *et al.*, 2009), which estimated that the magnitude of this flux is between 3.4 to 7.3 Mg C ha^{-1} $year^{-1}$. I set the trait value as 0.008 g C g N^{-1} day^{-1} $ind.^{-1}$ such that the modeled flux has the same order of magnitude.

I adapted the value of pathogen infection efficiency (α_S) from Augspurger (1983) and Bell *et al.* (2006). It was documented that 58.25% of *Platypodium elegans*

seedlings died due to pathogen infection within three months, while 50% of *Sebastiana longicuspis* seedlings died across five weeks. Similar to previous traits, I assumed the reference plant species to have trait values between the two plant species. The pathogens infection efficiency of adult (α_A) is assumed to be much smaller compared to those of seedlings (i.e. $\alpha_S \times 10^{-4}$). I assumed the pathogen assimilation ratio (b_P) of the reference plant as 0.6 following other theoretical studies (Miki *et al.*, 2010), which is a reasonable value since my model considered nitrogen flux instead of carbon. The value of litter return ratio (f_P) is set to 0.35 as I assumed 5% of plant nitrogen will directly return to the soil N pool after pathogen infection.

For the pathogen mortality rate (δ_P), Hancock (1981) observed that the decline of *Pythium ultimum* in the field follows exponential decay with a half-life of approximately 30 days, yielding a decay rate around 0.02 day^{-1}. For the case of mycorrhiza, an experiment by Staddon *et al.* (2003) showed that some fraction of mycorrhizal hyphae have a turnover time up to 30 days (i.e. mortality rate equal to 0.03 day^{-1}). Here, I assumed a slightly smaller pathogen mortality rate with the value 0.01 day^{-1} to fulfill mathematical conditions from Appendix S3. I assumed the same value for mycorrhiza mortality rate (δ_M).

For the parameter value of carbon transfer ratio ($C_{\max}$), Bryla and Eissenstat (2005) suggested that the total cost of plant-mycorrhiza associations ranges from 3 to

36% of the carbon fixed by photosynthesis. Whereas among the carbon allocated to mycorrhizal associations, more than 40% is consumed by respiration (Bryla & Eissenstat, 2005). Therefore, I set the value of carbon transfer ratio as 0.2 and the mycorrhiza carbon assimilation ratio (e_M) as 0.6.

The value of negative impact coefficient between microbes is calculated from a study by Sikes *et al*. (2009), which showed that the percentage colonization of root by pathogens declined 20 to 40% within five months when growing with mycorrhizas. I set the negative impact of mycorrhizas on pathogens (β_{PM}) as 0.005 g N^{-1} m^2 day^{-1} so that a similar decrease can be realized in the model system. I assumed that the negative impact of pathogens on mycorrhizas (β_{MP}) have the same magnitude.

For the environmental parameters, Menge *et al*. (2009) reported that the atmospheric N deposition flux is within the order of 10-100 kg N ha^{-1} $year^{-1}$ in polluted ecosystems, while the leaching flux of plant-available inorganic N and plant-unavailable inorganic N lies between 0.1-10 and 0.2-70 kg N ha^{-1} $year^{-1}$, respectively. I assumed a deposition input (I) equal to 0.005 g N m^{-2} day^{-1}, which is adapted from Vitousek & Sanford (1986) from tropical forest studies. This value corresponds to a flux of 18.25 kg N ha^{-1} $year^{-1}$, which is reasonable if I assumed an ecosystem with low anthropogenic impact. I assumed the leaching rate of inorganic N (Le) and organic N (φ) is 0.0002 day^{-1} and 0.00008 day^{-1}, respectively. The values of

these parameters are chosen in a way that the modeled N flux was within the range reported by Menge *et al*. (2009).

References

Augspurger CK. 1983. Seed Dispersal of the Tropical Tree, *Platypodium Elegans*, and the Escape of its Seedlings from Fungal Pathogens. *Journal of Ecology* **71**: 759-771.

Bell T, Freckleton RP, Lewis OT. 2006. Plant pathogens drive density-dependent seedling mortality in a tropical tree. *Ecology letters* **9**: 569–574.

Pyle EH, Santoni GW, Nascimento HEM, Hutyra LR, Vieira S, Curran DJ, van Haren J, Saleska, SR, Chow VY, Carmago PB *et al*. 2008. Dynamics of carbon, biomass, and structure in two Amazonian forests. *Journal of Geophysical Research* **113**: 1–20.

Cernusak LA, Winter K, Turner BL. 2010. Leaf nitrogen to phosphorus ratios of tropical trees: experimental assessment of physiological and environmental controls. *The New phytologist* **185**: 770–779.

King DA, Davies SJ, Noor NSM. 2006. Growth and mortality are related to adult tree size in a Malaysian mixed dipterocarp forest. *Forest Ecology and Management* **223**: 152–158.

Malhi Y, Aragão LEOC, Metacalfe DB, Paiva R, Quesada CA, Almeida S, Anderson L, Brando P, Chambers JQ, da Costa ACL *et al*. 2009. Comprehensive assessment of carbon productivity , allocation and storage in three Amazonian forests. *Global Change Biology* **15**: 1255–1274.

Vitousek PM, Denslow JS. 1986. Nitrogen and phosphorus availability in treefall gaps of a lowland tropical rainforest. *Journal of Ecology* **74**: 1167–1178.

Kurokawa H, Nakashizuka T. 2008. Leaf herbivory and decomposability in a Malaysian tropical rain forest. *Ecology* **89**: 2645–2656.

Hancock JG. 1981. longevity of pythium ultimum in moist soils.pdf. *Phytopathology* **71**: 1033-1037.

Bryla DR, Eissenstat DM. 2005. Respiratory costs of mycorrhizal associations. In: Lambers H, Ribas-Carbo M, eds. *Plant Respiration: From Cell to Ecosystem.* New York, USA: Springer-Verlag, 207-224

Hodge A, Fitter AH. 2010. Substantial nitrogen acquisition by arbuscular mycorrhizal fungi from organic material has implications for N cycling. *Proceedings of the National Academy of Sciences, USA* **107**: 13754-13759.

Staddon PL, Ramsey CB, Ostle N, Ineson P, Fitter AH. 2003. Rapid turnover of hyphae of mycorrhizal fungi determined by AMS microanalysis of ^{14}C. *Science* **300**: 1138–1140.

Sikes BA, Cottenie K, Klironomos JN. 2009. Plant and fungal identity determines pathogen protection of plant roots by arbuscular mycorrhizas. *Journal of Ecology* **97**: 1274–1280.

Clark DA, Brown S, Kicklighter DW, Chambers JQ, Thomlinson JR, Ni J, Holland EA. 2001. Net Primary Production in Tropical Forests: An Evaluation and Synthesis of Existing Field Data. *Ecological Applications* **11**: 371-384.

Markesteijn L, Poorter L. 2009. Seedling root morphology and biomass allocation of 62 tropical tree species in relation to drought- and shade-tolerance. *Journal of Ecology* **97**: 311–325.

Menge DNL, Pacala SW, Hedin LO. 2009. Emergence and maintenance of nutrient limitation over multiple timescales in terrestrial ecosystems. *The American naturalist* **173**: 164–175.

Miki T, Ushio M, Fukui S, Kondoh M. **2010**. Functional diversity of microbial decomposers facilitates plant coexistence in a plant-microbe-soil feedback model. *Proceedings of the National Academy of Sciences, USA* **107**: 14251-14256.

Appendix S5: Processes of numerical simulation

In this section, I provide the pseudocode for the numerical simulation processes. Simulations are programmed by C language, using fourth-order Runge-Kutta method with a fixed interval (i.e. 0.01 day step length) as the numerical integration method. Simulations for all microbial compositions follow these steps:

1. Simulate the time evolution of the system governed by Eqns. 1-6 (Table 1) using the reference plant species parameter setting (Table 4). For the scenarios with mycorrhizas, evaluate the quantity of carbon demand and supply at every step in order to select the proper equations from Table 2. Run the simulation from $t = 0$ to $t = 2{,}555{,}000$ days. This simulation time is long enough for all microbial composition scenarios to reach equilibrium.
2. Save the equilibrium value for pathogen, mycorrhiza, and soil nitrogen level when using the reference plant species' parameters as P^*_{ref}, M^*_{ref}, and R^*_{ref}, respectively.
3. Select a trait which is concerned under the specific microbial composition.
4. Assign a new deviation width (Δ) subsequently within the range -0.9 to 2.0. Multiply the reference value of the selected trait by $(1+\Delta)$ and save it as the trait value for the target plant species. The range of Δ corresponds to a

minimum 10% and maximum 300% of trait value deviation from the reference plant (Appendix S1).

5. Run the model system governed by Eqns. 1-6 for 2,555,000 days using the new-assigned target plant species' parameters.

6. Save the equilibrium value for pathogen, mycorrhiza, and soil nitrogen level as P^*_{tar}, M^*_{tar}, and R^*_{tar}, respectively.

7. Simulate the time evolution of the sub-model governed by Eqns. 7 and 8 (Table 3). In order to compare the growth of target plant species in two different soils, substitute plant trait parameters in the sub-model by the target plant species' parameters (i.e. *i* = *tar*). Simulate the sub-model twice with two different sets of soil properties obtained from previous steps (i.e. substitute (P^*_k, M^*_k, R^*_k) by either (P^*_{ref}, M^*_{ref}, R^*_{ref}) or (P^*_{tar}, M^*_{tar}, R^*_{tar})). Run the simulation from $t = 0$ to t =365,000 days, which is long enough for all sub-models to reach equilibrium.

8. Save the equilibrium adult density for the two simulations as $A^{**}_{tar,\,ref}$ (growth response in reference plant-cultivated soil) and $A^{**}_{tar,\,tar}$ (growth response in target plant-cultivated soil). Calculate the PSF strength resulting from the deviation of the specific trait as $PSF_{tar} = \log\left(\frac{A^{**}_{tar,\,tar}}{A^{**}_{tar,\,ref}}\right)$.

9. Repeat step 4 to 8 until the deviation range for that specific trait is completed. Note that the range of deviation is slightly different for different traits depending

on both realistic range reported in literature and mathematical analysis (Appendix S3).

10. Select another trait of interest for the specific microbial combination and repeat the analysis starting from step 3.