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REVIEW

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Incorporating the disease triangle framework for testing the effect of soil-borne pathogens on tree species diversity

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Abstract

1. The enemy-induced Janzen–Connell (JC) effect, a classic model invoking conspecific negative density dependence (CNDD) and distance dependence, is a primary biodiversity maintenance hypothesis. Yet, conflicting evidence for the JC effect leads to disagreement about its role in maintaining forest diversity.
2. We focus this review on soil-borne pathogens, which are the primary agent inducing the JC effect in many forest ecosystems. Although the test of the pathogen-induced JC effect in ecology critically rests on the seedling mortality caused by soil pathogens, what has not been explicitly explored in the early literature but has increasingly received attention is the long-recognized fact that the environment can alter virulence of pathogens and host susceptibility (thus pathogen–host interactions), as predicted by the classic disease triangle framework enlightened by pathology research in agricultural systems.
3. Here, following the disease triangle framework we review evidence on how the pathogen-induced JC effect may be contingent on context (e.g. environmental conditions, pathogen inoculum load and genetic divergence in host and pathogen populations). The reviewed evidence reveals and clarifies the conditions where pathogens may or may not cause disease to hosts, thus contributing to reconciling the inconsistent results about the pathogen-induced JC effect in the literature. The context dependence of the disease triangle predicts that the pathogen-induced JC effect would change under global change.
4. Gaining insights from evidence that the pathogen-induced JC effect is context-dependent, we suggest that future tests on the JC hypothesis be conducted under the framework of disease triangle, and we stress the necessity by controlling the effect of context factors on plant–pathogen interactions when testing for the JC effect. We conclude the review by proposing three lines of future research for testing the importance of the JC effect in maintaining global forest tree species diversity, with a particular emphasis on testing the effect of global warming on the strength of pathogen–host interactions for better predicting changes of forest biodiversity under climate change.

KEYWORDS
conspecific negative density dependence (CNDD), disease triangle, forest diversity, global change, Janzen–Connell effect, pathogen virulence, soil pathogens, tree seedling survival

1 | INTRODUCTION

Among the many hypotheses proposed to explain the maintenance of tree species diversity in forest ecosystems (Wright, 2002), conspecific negative density dependence (CNDD) is a fundamental mechanism (Chesson, 2000; Comita, Muller-Landau, Aguilar, & Hubbell, 2010; Connell, Tracey, & Webb, 1984; Johnson, Beaulieu, Bever, & Clay, 2012; Mangan et al., 2010; Webb & Peart, 1999). Intraspecific competition and the enemy-regulated Janzen–Connell (JC) hypothesis (Connell, 1971; Janzen, 1970) are two key drivers underlying CNDD (Murphy, Wiegand, & Comita, 2017; Wright, 2002), but the latter is likely the primary one at the seedling stage because of weak direct seedling–seedling competition (Paine, Harms, Schnitzer, & Carson, 2008). The JC hypothesis stipulates that host-specific enemies near adult trees inhibit the establishment and later success of conspecific seedlings in the vicinity of adults (Connell, 1971; Janzen, 1970). The inhibition of conspecifics thus releases space close to the adult trees for heterospecifics to colonize. Despite the widespread appreciation of the JC effect in regulating tree species diversity (Bagchi et al., 2014; Liu, Yu, Xie, & Staehelin, 2012; Packer & Clay, 2000), inconsistent results for the JC effect have been obtained, and thus, its contribution to maintaining biodiversity remains controversial (Hyatt et al., 2003; McCarthy-Neumann & Kobe, 2010; Terborgh, 2012; Gripenberg et al., 2014; but see Comita et al., 2014), possibly arising from reasons including different experimental designs and contingent factors that may affect virulence of pathogens (Pfennigwerth, Van Nuland, Bailey, Schweitzer, & Farrer, 2018; Terborgh, 2012).

Underpinning the JC effect is the critical prerequisite that natural enemies must have limited dispersal ability (so that they can only accumulate beneath their host trees to produce a distance-dependent effect; Janzen, 1970; Connell, 1971) or be host-specific (at minimum have substantially differentiated virulence across different tree species; Augspurger, 1990; Benítez, Hersh, Vilgalys, & Clark, 2013), in order to maintain tree species coexistence. Being dispersal-limited (Adler & Muller-Landau, 2005) or host-specific (Augspurger & Wilkinson, 2007; Gilbert, 2002; Sarmiento et al., 2017; Sedio & Ostling, 2013), soil pathogens meet the prerequisite originally assumed for the JC hypothesis and are the primary agent inducing the JC effect as shown *in situ* field manipulative experiments (e.g. Augspurger, 1983; Bell, Freckleton, & Lewis, 2006; Bagchi et al., 2010; Bagchi et al., 2014; Sarmiento et al., 2017) and greenhouse simulation experiments (Liu, Fang, Chesson, & He, 2015; Liu et al., 2012; McCarthy-Neumann & Ibáñez, 2013; Packer & Clay, 2000). Note that herbivores including seed predators were also proposed as an agent inducing the JC effect in Janzen's original paper (1970), but herbivore satiation after heavy seed rain (e.g. "mast seeding") and indiscriminate attack on seedlings of all species (Bagchi et al., 2014) contradict the prediction of the JC hypothesis (i.e. overcompensating CNDD in the original form of the JC hypothesis; Bagchi et al., 2010; Xiao et al., 2017; but see Swamy & Terborgh, 2010). Therefore, we focus this review exclusively on the pathogen-induced JC effect. In this review, host specificity of pathogens is defined as a degree of evolutionary association of pathogens with their host plants. Highly host-specific pathogens require (or establish an intimate relationship with) a single-host species (or a genotype) to complete their life cycle, while generalist pathogens can live with individual hosts of multiple species.

Nevertheless, evidence for the JC effect has not been unequivocal. For instance, some experimental studies fail to detect the JC effect in tropical forests (e.g. McCarthy-Neumann & Kobe, 2010; Gripenberg et al., 2014), while in the same systems others find evidence consistent with the prediction of the JC effect (e.g. Bell et al., 2006; Kobe & Vriesendorp, 2011; Bagchi et al., 2014). Enlightened by insights from the framework of classic disease triangle originally formulated from crop pathology research in agricultural systems, that is the concept of environment-dependent plant diseases (Agrios, 2005; Pangga, Hanan, & Chakraborty, 2011; Stevens, 1960), and also recognized in studies of forest tree diseases and their interactions with climate (Cobb & Metz, 2017; Sturrock et al., 2011), we propose to understand and reconcile the mixed results regarding the JC effect from the perspective of the disease triangle. It is a due fact that the importance of the environment on modulating plant–pathogen interactions (i.e. the context dependency) in ecology has been well noticed (Benítez et al., 2013; Bever, Mangan, & Alexander, 2015; van der Putten, Bradford, Brinkman, Voorde, & Veen, 2016) and several studies have also accounted for the abiotic context in which the JC effect operates (e.g. McCarthy-Neumann & Ibáñez, 2013; Pfennigwerth et al., 2018), but none of them draw on the disease triangle for their predictions.

The framework of the classic disease triangle (i.e. formed by pathogen, host and the environment as three corners) in agricultural pathology is supported by the interplay of two key processes required for a successful pathogen infection on host, namely favourable environmental conditions for infestation and contact between pathogens and host (Pangga et al., 2011; Stevens, 1960). The occurrence of a plant disease is subjected to effects of many environmental factors, with temperature, moisture and light being the key ones in many cases (see Figure 1a). For example, a fungal endophyte does not usually cause plant disease under a certain environmental condition but could be pathogenic under others (Moricca & Ragazzi, 2008). In testing the JC effect in ecology, environmental factors are often not considered as an inherent component of a study, but it is necessary to control the contingent effect of environment on the JC effect (Pfennigwerth et al., 2018) according to the disease triangle framework.

Moreover, similar to the widely observed build-up of disease pressure over continuing cropping in agricultural cultivation (Schippers, Bakker, & Bakker, 1987; Zhou & Wu, 2012) as well as the attenuating advantage of invasive species over time (Dostál, Müllerová, Pyšek, Pergl, & Klinerová, 2013), disease severity of long-lived forest tree species at a site is expected to increase, resulting from pathogen accumulation over time (see Figure 2). This suggests that the colonization history of the host population or its population size could be an important disease factor, whereby the occurrence of a plant disease does not only require that pathogen encounter its host under favourable environment but also require a sufficient load

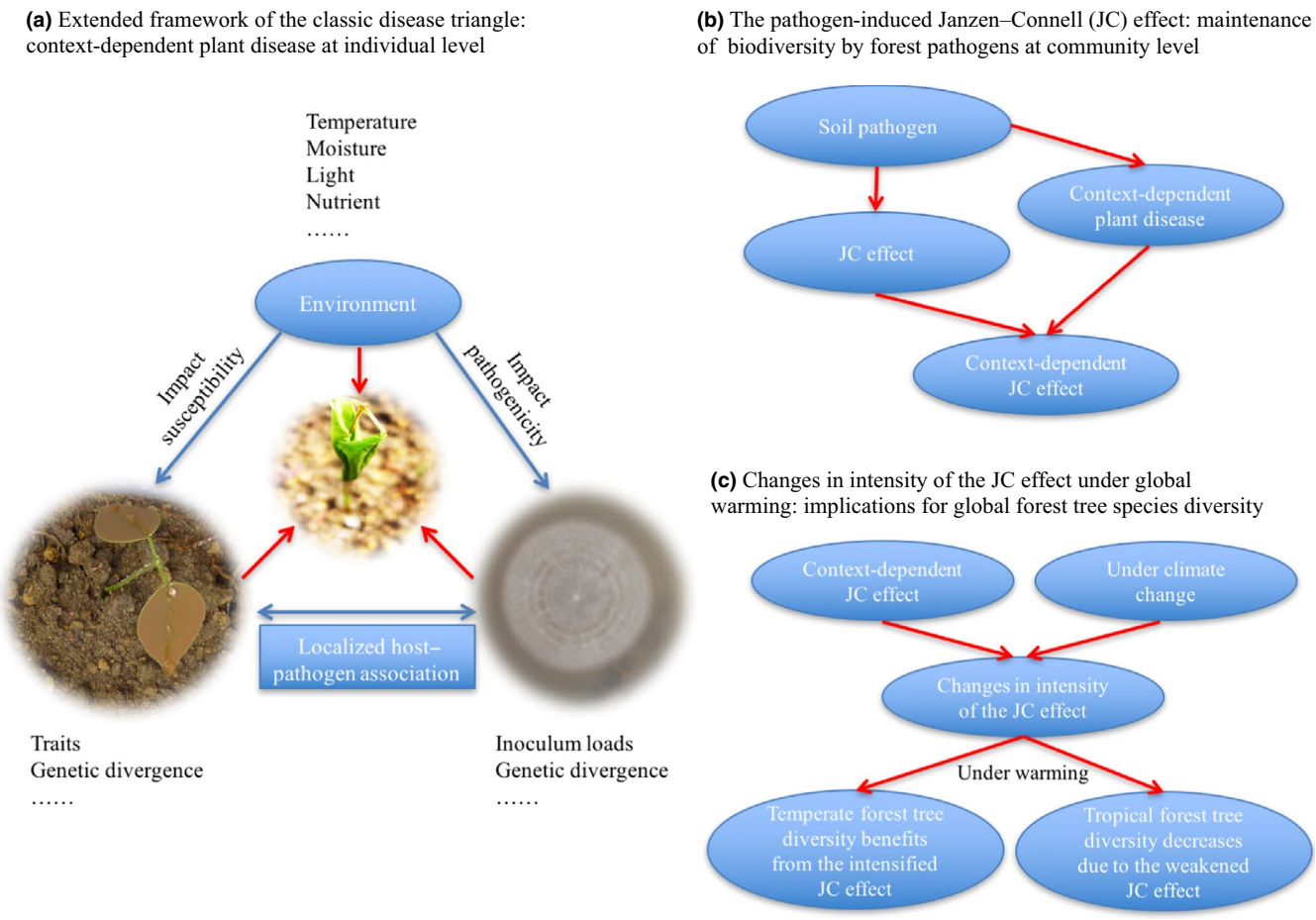

FIGURE 1 Framework of the disease triangle, and incorporation of the Janzen–Connell (JC) effect into the framework for projecting forest responses under global change. (a) Occurrence of a plant disease not only requires that virulent pathogens encounter the susceptible host plant under the environment conducive to disease development as originally proposed by Stevens (1960), but also depends on the amount of pathogen inoculum load (accumulating over time) and the localized pathogen–host association resulting from the genetic divergence in host as well as pathogens. (b) Following the framework of the disease triangle, test of the JC effect should consider the possible context-dependent effect. (c) The disease triangle framework predicts that changes in intensity of the JC effect under climate change can impact global forest tree diversity. In temperate forests, warming is expected to increase plant disease severity due to temperature lower than the optimal temperature of pathogen reproduction and growth, leading to intensified JC effect, while plant disease severity is expected to decrease with increase in temperature in the tropical forests, weakening the JC effect

of the pathogen inoculum (Figure 1a). A newly colonized host may not accumulate a sufficient load of pathogens to cause plant disease (for the JC effect to act), which is the basis for the natural enemy escape hypothesis widely used to predict the establishment of invasive species (Agrawal et al., 2005; Mitchell & Power, 2003). A corollary of this natural enemy escape hypothesis is that host-specific pathogens may not play a role in regulating low-density host populations because of low pathogen accumulation but are important in regulating high-density host populations (Liu et al., 2015; Zhu, Woodall, Monteiro, & Clark, 2015; Uriarte, Muscarella, & Zimmerman, 2018; but see Kempel, Eindisbacher, Fischer, & Allan, 2018, for opposite results). How the JC effect may regulate low versus abundant host populations is a critical but unanswered question in ecology (Chesson, 2000; Siepielski & McPeek, 2010; Wilson, 2011).

Another important aspect that is rarely considered in testing the JC effect but has significant implications to biodiversity maintenance according to the framework of the disease triangle is dispersal of soil pathogens (Figure 1a). Documented evidence shows that soil pathogens generally are poor dispersers (via run-off or mycelium spread), leading to local occurrence of pathogens (Gilbert, 2002). This, in combination with the sessile nature of trees, may form a "much more intimate (localized) pathogen-host interaction", leading to low pathogen-induced mortality of offspring beneath unrelated conspecifics compared to those beneath their parent trees (Augspurger & Kelly, 1984). The disease triangle further predicts that intraspecific genetic divergence among host provenances (or "genotypes" in Pérez-Izquierdo et al., 2017) and the genetic variation within pathogen populations (e.g. formae speciales in *Fusarium oxysporum*; Altinok, Can, & Altinok, 2017) could cause differential infestation, depending on the co-occurring genotypes of the host and pathogen. Therefore, the genetic divergence in host and pathogen should be taken into consideration for evaluating plant diseases (Figure 1a).

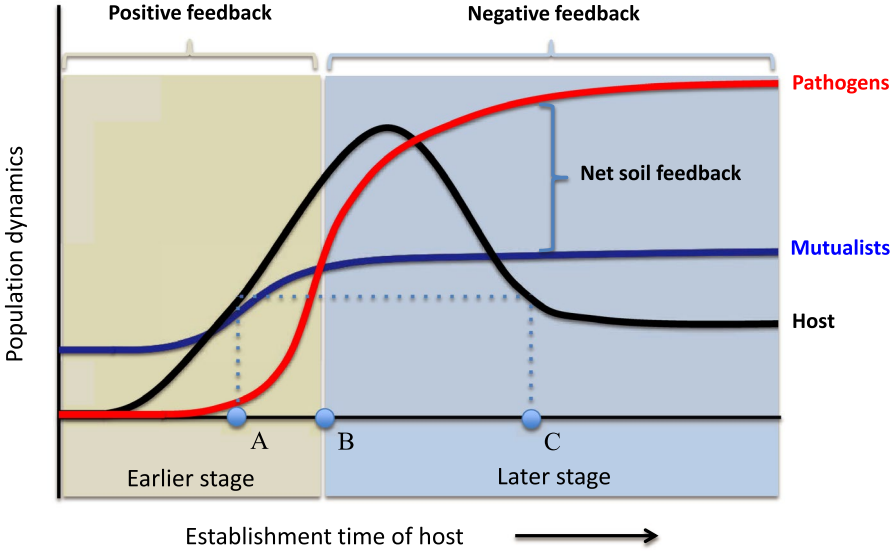

FIGURE 2 Hypothetical changes in plant population size (the black curve) along its establishment time in a site, the associated negative feedback (the red curve) caused by pathogens, and positive feedback (the blue curve) driven by mutualists such as soil mycorrhizae. Depending on the contrasting effects of accumulating pathogenic and mutualistic micro-organisms, the net effect (beneficial or inhibitory, i.e. the difference between the blue and red curves) of soil organisms on plants changes over time. At the early stage of plant establishment in a new site, the recruitment of the host species is enhanced due to the lack of natural enemies and existing generalist mutualists (e.g. the widely distributed mycorrhizal fungi) (the blue curve). As time increases (thus build-up of the host population size), the negative effect of soil pathogens on the host plants increases (the red curve), causing the net soil feedback shifting from positive to negative (after time point B). At time A, the host is regulated by positive plant–soil feedback by mutualists, while at time C, it is regulated by negative feedback by pathogens although the host population density at these two points is the same. The blue and red curves cross at time B where the importance of the effect of positive and negative feedback switches

In this review, we focus on the conceptual framework of disease triangle to explore potential factors and agents (i.e. environment, pathogen inoculum load and genetic divergence in host and pathogen populations) regulating the severity of plant disease while testing for the JC effect in a given system. We reviewed evidence that the JC effect can vary on circumstantial conditions (Figure 1b) as proposed by the disease triangle (Figure 1a). We concluded the review by proposing future foci of research for better understanding the importance of the JC effect in maintaining global forest biodiversity. We particularly emphasized the implication of potential changes in the intensity of JC effect under global change (Figure 1c).

2 | ENVIRONMENT-DEPENDENT PLANT DISEASE

Environmental conditions, particularly temperature, humidity/moisture and light intensity, can cause variation in virulence of pathogens as well as susceptibility of host plants (Roberts & Paul, 2006), thereby affecting disease incidence and severity of plants (Figure 1a). Increasing attention has recently been paid to environmental factors in forest pathology (Cobb & Metz, 2017; Desprez-Loustau, Marçais, Nageleisen, Piou, & Vannini, 2006; García-Guzmán, Trejo, & Sánchez-Coronado, 2016), and the importance of environmental regulation on the JC effect has been noted in ecology (Liu et al., 2012; McCarthy-Neumann & Ibáñez, 2013; Pfennigwerth et al., 2018; Rutten & Gómez-Aparicio, 2018). In this section, we will link the disease triangle with the JC effect to address several environmental factors which may obscure the detection of the JC effect.

2.1 | Ambient humidity/soil moisture

Many plant diseases require a certain level of ambient humidity to occur. For example, high humidity could turn *Discula quercina*, an endophytic fungus in *Quercus cerris* that does not cause symptoms at normal ambient humidity, into a pathogen (Moricca & Ragazzi, 2008). In another example, Swinfield, Lewis, Bagchi, and Freckleton (2012) demonstrated that seedlings of *Pleradenophora longicuspis* suffered much higher mortality from soil fungal pathogens if watered daily, which is consistent with the observation that CNDD is intensified in rainy years (Uriarte et al., 2018) and pathogen-infested seedling mortality is increased in wet sites (Brenes-Arguedas, Coley, & Kursar, 2009). In a meta-analysis, Comita et al. (2014) also found that there was a stronger CNDD in wetter sites even though the evidence is murky whether the CNDD was caused by pathogens or other processes. As for hosts, unfavourable humidity conditions can decrease plant defence and make them more susceptible (Atkinson & Urwin, 2012). For instance, under drought stress, disease severity of plant (e.g. dieback) was often increased (Desprez-Loustau et al., 2006). This increased plant mortality is due to the lowered defence of plants in stressful environments (Jactel et al., 2012), although drought can also weaken fungal pathogens. These together suggest

that ambient humidity can change the strength of JC effect, depending on its respective effects on pathogens and host plants.

To further elaborate on the JC effect under the framework of the disease triangle, we extended an early study (Liu et al., 2015) to conduct a simple experiment to test for how the effect of pathogens on seedling mortality of a subtropical tree species was changed by ambient humidity and light intensity. The experiment was conducted in greenhouse, and soil inoculum was collected under the crown of *Ormosia glaberrima* adult trees where soil pathogens were found to infest this species (Liu et al., 2015). We compared the effects of two treatment combinations of humidity and light on pathogen-induced seedling mortality. The first treatment combination was high humidity (*ca*. 90%) and low light (photosynthetically active radiation ≈ 25 μE s − 1 m − 2; 12 hr per day), similar to under-canopy conditions in April when seeds of this tree species germinated in bulk and soil pathogen-induced mortality of seedlings was high (Liu et al., 2012). The second treatment was a combination of low humidity (65%) and high light (500 μE s − 1 m − 2; 12 hr per day). The experiment lasted for 16 weeks, and each treatment combination had 16 replicates. Results showed that seedling mortality was significantly increased under the treatment of high humidity and low light (Mann–Whitney test: $w = 1.5$, $P = 0.022$; Figure 3). The result is consistent with previous studies showing increased seedling mortality caused by pathogens under shaded conditions (Augspurger & Kelly, 1984; Roberts & Paul, 2006), while decreased soil moisture in forest gaps of high light intensity restrained pathogen activity (Augspurger & Kelly, 1984; García-Guzmán et al., 2016; Reinhart, Royo, Kageyama, & Clay, 2010).

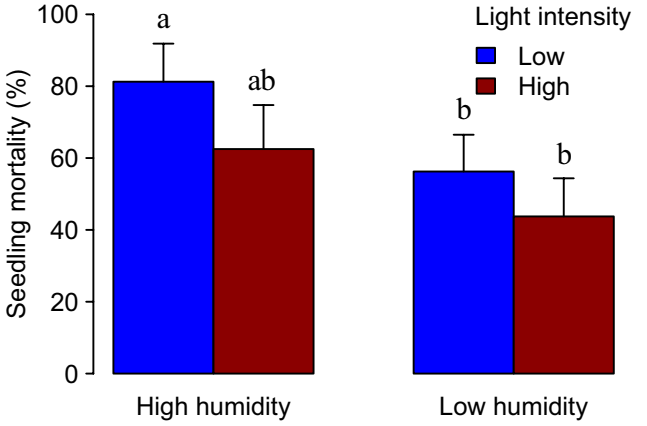

FIGURE 3 Effects of ambient conditions on seedling mortality of *Ormosia glaberrima*. Soil inoculum was collected under the crown of *O. glaberrima* adult trees where soil pathogens were found to infest *O. glaberrima*. Seedling mortality was significantly higher under the combination of high humidity and low light than the combination of low humidity and high light. Error bars in the panels are 95% bootstrapped confidence intervals. The letters indicate significant differences of seedling mortality among different treatment combinations by Mann–Whitney test

2.2 | Ambient temperature

Temperature is perhaps the most important factor second to moisture in regulating pathogen–host interaction (Dorrance, Kleinhenz, McClure, & Tuttle, 2003). It has been shown that increased temperature often favours growth and infection of pathogens in warm seasons (Weed, Ayres, & Hicke, 2013) and also increases their overwintering survival in temperate regions (Penczykowski, Walker, Soubeyrand, & Laine, 2015; Pfender & Vollmer, 1999). However, it is widely known that there is an optimal temperature for a pathogenic fungus to reproduce within its host (e.g. 20°C for some fungi; Scherm & van Bruggen, 1994) and cause the most severe disease in its host (Roger, Tivoli, & Huber, 1999). Therefore, severity of plant disease is predicted to increase with temperature up to the optimal temperature and then decrease with further increase in temperature (Roger et al., 1999). The implication of temperature dependence of pathogen infection is that pathogen–host interaction is expected to change with global warming (Chakraborty, 2013), although the response to temperature rising may vary across different climate regions.

2.3 | Soil nutrient levels

The effect of soil nutrients on pathogen–host interaction is not typically considered in the disease triangle, even though severity of plant disease usually tends to increase under fertilization (e.g. nitrogen fertilization; Veresoglou, Barto, Menexes, & Rillig, 2013). At the same site where we did humidity and light intensity treatment for *O. glaberrima* (see Figure 3), Luo, De Deyn, Jiang, and Yu (2017) found that the negative effect of soil biota was increased under nutrient enrichment. At low nutrients, however, the negative effect of soil pathogens on host plants can be counteracted by the positive effect of soil mutualists such as mycorrhizal fungi (van der Putten et al., 2016). We expect that the effect of soil pathogen would become intensified under anthropogenic nitrogen deposition (Galloway et al., 2004).

The evidence reviewed in this section makes it clear that the environment should be taken into account for evaluating the pathogen–host interaction (Hersh, Vilgalys, & Clark, 2012). Without fully considering the variation in environmental variables, the JC effect could be obscured. Such environment-dependent JC effect is likely attributed to the variation of pathogens (pathogenicity and/or density of pathogens) across sites (e.g. higher seedling mortality caused by pathogens in the wet sites versus in the dry ones; Brenes-Arguedas et al., 2009).

3 | CHANGE IN PATHOGEN INOCULUM LOAD OVER TIME (OR ACROSS SITES)

The impacts of pathogens on tree species change over life history by which the newly germinated seedlings are found to suffer the largest effect (Harms, Wright, Calderón, Hernández, & Herre, 2000), while effects are negligible at later stages (Zhu, Comita, Hubbell, Ma, & Shefferson, 2015). Similarly, for a single host population, the local pathogen inoculum load is expected to increase as the host population

size increases (i.e. the "cumulative effect") (Terborgh, 2012). This is similar to the effect of increasing negative plant–soil feedback (PSF) for invasive species as the invasion history prolongs (and the invaded population size expands) (Dostál et al., 2013), or the aggravating "soil sickness" after continuing cropping in agricultural cultivation (Zhou & Wu, 2012). Understanding how pathogen load accumulates over time or varies across host populations of different size is important for answering questions about maintenance of rare species (or low-density populations) versus abundant species (or high-density populations) (Chesson, 2000; Liu et al., 2015) and is the focus of this section.

3.1 | Pathogen accumulation over time

Packer and Clay (2004) tended to consider the concept of disease triangle (Figure 1a) in testing for possible shifts in the net PSF effect under the successively planting rotations of *Prunus serotina* seedlings even though the disease triangle was not explicitly mentioned in their work. Their study uncovered that seedling survival benefited from mutualistic microbes and experienced a positive (net) PSF at the initial cropping, but the direction of the net PSF shifted quickly from positive to negative after a few rotations (Packer & Clay, 2004), as predicted by the conceptual model shown in Figure 2. It shows the net PSF is determined by the sum of positive PSF driven by mutualists such as soil mycorrhizae and negative PSF caused by pathogens (Mariotte et al., 2018), yet the degree of host specificity is different between mutualists and pathogens.

Relative to soil mutualists (e.g. mycorrhizal fungi and nitrogen-fixing bacteria), soil pathogens generally have high specificity with host plants or have substantially differentiated virulence on host plants to overcome host defence (Augspurger, 1990; van der Putten, Dijk, & Peters, 1993; Sarmiento et al., 2017) and make pathogen genotypes host-specific (Antonovics et al., 2013). It is the lack of host specificity that allows mutualists to facilitate plants to colonize newly available sites (Bent, Kiekel, Brenton, & Taylor, 2011). When a plant species arrives at a new site (through dispersal/introduction), it can make use of the local mutualistic microbes (e.g. time point A of Figure 2; Callaway, Bedmar, Reinhart, Silvan, & Klironomos, 2011) while escaping host-specific pathogens from their native sites (Mitchell & Power, 2003), so that to experience double benefits of enemy release and local mutualists (Callaway et al., 2011). With the establishment of the host population, it starts to accumulate pathogens (thereby attenuating positive effect of soil biota) and at a certain point (the illustrative time point B of Figure 2) the effect of pathogens on the host plants offsets the benefit of mutualists. Beyond this point, the net PSF effect is reversed from positive to negative, and the expansion of the host population is eventually halted (to prevent any species from becoming predominant, as predicted by the JC hypothesis). It is important to note that a similar thought has been put into the temporal shifts of positive and negative PSF over time to study successional dynamics of community (Kardol et al., 2013).

The time-dependent change in the net PSF as described in Figure 2 may improve the understanding of why detection of the JC effect (i.e. the negative net PSF) could be elusive. Taking both A and C time points in Figure 2, as an example, even though the host population has a similar size (see the black curve of Figure 2), the net PSF effects on the host are contrastingly different (positive at time A while negative at C). The JC effect will be detected at time point C because the negative effect (the red curve) caused by pathogens is stronger than the positive effect of mutualists (the blue curve). Therefore, time-series data on host population dynamics are needed to exclusively evaluate the dynamic change of the net PSF, which has been reported for invasive species with known invasion history (e.g. Dostál et al., 2013). However, such data are limited for forest tree species. Alternatively, local density of adult individuals may be used as a proxy for the establishment time at different sites, as described in the next section.

3.2 | Pathogen accumulation associated with host abundance

In a recent study for understanding how the local densities of a tree species influenced the JC effect, Liu et al. (2015) showed that the seedling recruitment of a subtropical legume tree species, *O. glaberrima*, was greatly suppressed by soil pathogens where the density of the tree species was high (i.e. high-density disadvantage), while the effect disappeared at low tree density due to the lack of pathogen accumulation (i.e. low-density advantage). The result of this study is further confirmed by a subsequent study showing that greater relative abundance of soil pathogens is associated with higher seedling mortality at sites where there are high densities of conspecific adult trees (Liang et al., 2016).

The spatial variation (or temporal dynamics) of the pathogen–host interaction suggests that soil pathogens can only induce plant diseases in the right place and the right time where and when there is sufficient pathogen accumulation. This spatio-temporal variation in pathogen inoculum load further suggests that the JC effect vary in space and time as well. Such variation in the JC effect is not well studied but has important implications to biodiversity maintenance in landscapes (and temporal stability of ecosystems as well) through the mechanism of low-density advantage (lack of pathogen attack in sites where host population is low) and high-density disadvantage (high pathogen accumulation in sites where host population is abundant) (Chesson, 2000).

4 | GENETIC DIVERGENCE OF HOST AND PATHOGENS LEADS TO LOCALIZED PATHOGEN–HOST ASSOCIATIONS

Flor (1956) proposed that host plant populations could be differentiated to resistance (R) genotype and non-resistant genotype and pathogen populations can evolve to specific and non-specific avirulence (Avr) genotypes (also see review of Parker & Gilbert, 2004). As such, a single mutation in the genome of either pathogens (Avr genes) or host plants (R genes) can alter the pathogen–host interaction, that is the "gene for gene" hypothesis in phytopathology (Thompson & Burdon, 1992). For example, *Pseudomonas syringae*, a

genetically polytypic morphospecies causing diseases to plant species of more than 100 families, can be further subdivided into *ca*. 50 pathovars based on their differentiated pathogenicity to host plants. The presence or absence of individual genes in *P. syringae* can determine their host compatibility (Hirano & Upper, 1990).

Differentiation of pathogen virulence does not only occur among different host plant species but can also occur among different genetic variants of a single host plant species. Indeed, even at a very local scale can genetic divergence be detected for long-lived tree species (Audigeos, Brousseau, Traissac, Scotti-Saintagne, & Scotti, 2013). This divergence is also found in mutualistic fungi as reported in a recent study in which genetic divergence of tree species, *Pinus edulis*, has been observed to strongly impact soil mycorrhizae composition which affords a critical function for plants to tolerate drought stress (Gehring, Sthultz, Flores-Renteria, Whipple, & Whitham, 2017). In natural ecosystems, this localized occurrence of genetic variation in host population will decrease the likelihood for a pathogen avirulent genotype to encounter its compatible host genetic variants, thus contributing to lowering outbreak of catastrophic plant disease. This possibly explains why outbreaks of plant diseases are not often observed in nature (Laine, Burdon, Dodds, & Thrall, 2011).

We found that Augspurger and Kelly (1984) is a good example of empirical study that suggests the importance of considering genetic variation in pathogen virulence and in host resistance in testing the JC effect. The study showed that seedlings of *Platypodium elegans* suffered more severe damping-off disease underneath their own parent trees relative to those seedlings planted under non-parent trees of the same species. This result implies a possibility there is a locally co-evolved pathogen–host interaction. We notice that in field/greenhouse experiments for testing the JC effect, plant seeds are often purchased from different seed companies (likely representing different provenances) (e.g. Reinhart & Clay, 2009) and this could lead to underestimating the JC effect due to genetic divergence of different seed provenances. Similarly, the different performance of offspring under an unrelated conspecific relative to its parent can also blur the JC effect in the field. Therefore, accounting for genotype variability is necessary for understanding the JC effect in explaining forest tree species diversity.

To evaluate the effect of genetic divergence (of both pathogens and hosts) on pathogen–host interactions, one may directly isolate genetic strains of the targeted pathogen around individual adults of a tree species and conduct cross-inoculation experiment (considering seed provenances of hosts in the meanwhile). Yet simple, this approach is extremely time-consuming and laborious because of the large number of cross-inoculations involved. For example, Liu et al. (2012) found that the isolate of *F. oxysporum* from *O. glaberrima* (at a high-density site of this host species) not only showed high host specificity (not infecting other tree species co-occurring with *O. glaberrima* at that site), but its virulence could even distinguish seed provenances of *O. glaberrima* (i.e. seedling mortality caused by this particular isolate was significantly lowered for seedlings germinated from seeds of *O. glaberrima ca.* 1.2 km away from that site). Although *F. oxysporum* is a generalist pathogen and is a genetically heterogeneous polytypic morphospecies (Recorbet et al., 2003), it is worth noting that pathogenic variants of *F. oxysporum* are also found to possess a narrow host range in agricultural ecosystems (i.e. forma specialis; Altinok et al., 2017). Meanwhile, localized genetic divergence of long-lived tree species associated with heterogeneous environmental niches (Audigeos et al., 2013) may further intensify the specialized (local) pathogen–host association. The study on the interaction of *O. glaberrima* and *F. oxysporum* is an interesting example showing how the local pathogen–host association can arise from the genetic differentiation of pathogens and host tree species (seed provenances). It showcases the complex interaction between pathogens and host plants in nature and suggests the necessity to take account of genetic variation in pathogens and hosts while testing for the JC effect.

Moreover, on the molecular front, in a recent study aiming to explain why rare species suffer stronger conspecific negative density dependence, Marden et al. (2017) sequenced pathogen resistance (R) genes and found small host plant populations have reduced R gene diversity compared to abundant species populations. This study opens a door for genomic exploration of the JC effect and holds great promise for deciphering the complex pathogen–host interactions. Many well-established genomic methods, for example single nucleotide polymorphisms and spatial genetic structure (see Audigeos et al., 2013), can be useful in this endeavour.

5 | FUTURE RESEARCH ON THE JC EFFECT

In the context of this review, we identify three future directions for better understanding the role of the JC effect in maintaining forest biodiversity, especially under current global change. These proposed directions reflect the fact that the virulence of pathogens is contingent on many factors, including environmental conditions, establishment time of hosts at a site, and genotypes of hosts and pathogens.

1. Environment-dependent host specificity of pathogens: Host specificity of pathogens is a critical requirement of the JC hypothesis for pathogens to check host populations from becoming dominant through frequency- or distance-dependent effects, but there is only limited direct evidence on host specificity in fact (e.g. Packer & Clay, 2000; Liu et al., 2012). On the contrary, pathogens often infect a variety of hosts and thus are generalist (e.g. Hersh et al., 2012; Sarmiento et al., 2017). As to such contradiction, one previous review suggests effective specialization as a result of the particular host–pathogen–environment interaction (Benítez et al., 2013). At present, empirical evidence supporting this environment-dependent host specificity of pathogens is yet to be collected.

Although identifying the targeted pathogens for crop diseases has been widely and successfully done, testing for the specificity of soil pathogens for host tree species via inoculation experiment (i.e. the application of Koch's application; Agrios, 2005) is near infeasible, given the difficulty of conducting careful cross-inoculation experiments, and

the large number of inoculation combinations required to test any real community. One possible approach may provide partial answer to this problem in the future JC studies. It is to conduct cross-inoculation experiment for selected abundant tree species with considering *in situ* environmental conditions. The rationale behind this approach is that the regulation of abundant tree species in a community is critical in structuring the community and competition among abundant species is likely stronger than among others species. Evidence of (environment-dependent) pathogen specificity with abundant host species is thus informative of understanding the role of pathogens in regulating communities.

2. Host trait-associated JC effect: As plant traits have been widely recognized to reflect their adaptations to abiotic and biotic regimes (Funk et al., 2017), plant traits are expected to affect pathogen–host interaction. The inclusion of host traits adds a new explanation, beyond the variation of host and pathogen genotypes, for interspecific variation in the JC effect across tree species—the JC effect may be strong for some tree species but weak for the other at the same site. Especially, if (environment-dependent) effective specialization of pathogens exists widely in the field, the role that plant traits play in affecting the JC effect across species may be important and worth to explore.

It has been widely known that plant life-history traits such as wood density, shade tolerance and growth rates are closely related to the resistance to pathogen infection (Augspurger & Kelly, 1984; Kobe & Vriesendorp, 2011). For example, Augspurger and Kelly (1984) found that there was an inverse relationship between adult wood density of tree species and their susceptibility to pathogens. Fast-growing and shade-intolerant tree species, having low tissue/wood density, are observed to experience strong CNDD relative to slow-growing and shade-tolerant species as measured by seedling mortality (Kobe & Vriesendorp, 2011), and they are susceptible to pathogen attack and may experience severe JC effect. This shade tolerance-regulated JC effect could have significant implications to species diversity along a light gradient from full light-gaps to closed-canopy understorey where species of different shade tolerance have different colonization success and are subject to differentiated JC effect. We see the importance of conducting analysis to identify life-history traits that may strengthen or weaken the JC effect. It will be especially interesting if root traits (e.g. specific root length) can be included as they may have more direct effects on the susceptibility of tree species to soil pathogens. Furthermore, it is necessary to incorporate phylogenetic data in this analysis so that to understand the evolutionary basis of the trait–pathogen association. The stem-mapped plots of the CTFS-ForestGEO network (Anderson-Teixeira et al., 2015) where detailed trait data are collected for almost every species can provide an ideal system for this proposed research.

3. Effect of climate change on pathogen–host association: Many studies have shown, either empirically or by modelling, that global warming rapidly changes the distribution, physiology, phenology and interactions of many species which seriously threaten the present and future biodiversity (Bellard, Bertelsmeier, Leadley, Thuiller, & Courchamp, 2012; Parmesan & Yohe, 2003; Thuiller, Lavorel, Araújo, Sykes, & Prentice, 2005). An important yet unexplored question is how global warming may alter pathogen–host plant interactions and jeopardize the capacity of the JC effect in maintaining forest diversity. This is an urgent issue as earth surface temperature continues to rise and by the end of the 21st century the temperature is projected to likely rise at least 1.5°C (IPCC, 2013). Although disturbances, rising temperature included, could influence forest diseases as predicted by the framework of disease triangle (Cobb & Metz, 2017), the repercussion of climate change on soil pathogen–host plant interaction is poorly known. Limited empirical evidence has so far shown that increased temperature can influence pathogen–host interaction (Chakraborty, 2013), and thus, the impact of global warming on the JC effect is expected to vary across different regions.

In temperate regions, there are cold spells in winter by which a high proportion of fungal pathogens will fail to live through possibly due to their resting spore structures damaged by subzero temperatures (Penczykowski et al., 2015). For instance, *ca.* 40% of powdery mildew *Podosphaera plantaginis* population dies during winter (Tack & Laine, 2014). Therefore, in temperate forests increased temperatures not only facilitate the development of pathogens during growing seasons (Weed et al., 2013), but also increase overwintering survival (Penczykowski et al., 2015), whereby the intensity of the JC effect would likely increase. As predicted from the trend that species diversity increases with the strength of CNDD along the latitude (LaManna et al., 2017), tree diversity in temperate forests may benefit from the intensified JC effect (i.e. increased plant disease severity) with warming climate if else being equal, while tree diversity in the tropics may decrease due to the weakened JC effect caused by warming (plant disease severity is expected to decrease with temperature beyond the optimum value; Figure 1c).

We propose to conduct field and greenhouse warming experiments along a latitudinal gradient to investigate the impact of rising temperature on pathogen–host interaction. The network of the CTFS-ForestGEO sites again offers an ideal system for this study. The plots span latitudes 25°S-61°N—where temperatures have risen by an average of 0.61°C over the past 30 years (Anderson-Teixeira et al., 2015). Warming experiments (e.g. open-top chamber, combined infrared canopy or below-ground warming treatment; see Rich et al., 2015) can be used to emulate the global warming to the similar degree as predicted by the IPCC (2013). Regardless whether global warming has positive or negative effect on pathogen–host plant interactions, any change in virulence of soil pathogens driven by global change will likely have profound implications for global biodiversity. Evidence gathered from warming and other change experiments will add novel mechanistic understanding of potential biodiversity change in response to global change (Figure 1c).

Climate change is more than the change in temperature. It is also important to study the effects of other global change processes such

as drought and nitrogen deposition on pathogen–host plant interactions so that to form a more complete understanding of the role of the JC effect in maintaining biodiversity in the face of global change. Although water deficit caused by drought slows tree growth and increases mortality (Desprez-Loustau et al., 2006), it will also suppress activities of soil pathogens (Jactel et al., 2012), thus lowering the role of the JC effect.

6 | CONCLUDING REMARKS

The JC hypothesis is a primary forest biodiversity maintenance hypothesis. The operation of this hypothesis, as induced by soil-borne pathogens, is subjected to the conditions where and when the specific pathogens and host plants occur and co-evolve, as predicted by the disease triangle (Figure 1). The spatial and temporal variations in virulence of pathogens and the resistance of host plants explain the mixed results in the literature about the importance of the JC effect in maintaining forest biodiversity. As such, future tests for the JC hypothesis must consider the conditions under which the JC effect may be environment-dependent, time-dependent (pathogen accumulation) and genotype-dependent (pathogen virulent genotypes versus host resistant genotypes). Following the framework of the disease triangle, we suggest future research on the JC effect to focus on testing environment-dependent effective specialization of soil pathogens, identifying host plant traits that affect the intensity of the JC effect, and exploring the possible responses of negative soil feedback (the JC effect) to climate change.

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AUTHORS' CONTRIBUTIONS

Both authors conceived the review and contributed equally to the writing of the paper.

DATA ACCESSIBILITY

Seedling mortality data for Table 3 are available in the Dryad Digital Repository: https://doi.org/10.5061/dryad.kk6cn22 (Liu & He, 2019).

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SUPPORTING INFORMATION

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