Exploring the Biogeographic Relationship between Variation in Parasites and Pathogens and Host Plant Dispersal Traits

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Abstract

Plants rely on seed dispersal to track favorable habitats and escape unsuitable conditions in variable environments. One likely driver of phenotypic dispersal trait plasticity is the prevalence and intensity of parasites and pathogens in the plant's environment. If the effects of parasites and pathogens follow broad scale biogeographic patterns, and plant dispersal traits vary with parasite and pathogen prevalence and intensity, then corresponding biogeographic patterns in dispersal trait variation will likely be observed. To test this, I explored the literature to identify potential biogeographic trends in parasites and pathogens and evaluate support for the hypothesis that biotic interactions increase with decreasing latitude. In parallel, I reviewed and synthesized empirical studies that have tested for intraspecific variation in dispersal traits and evaluated the extent of evidence for a latitudinal gradient in dispersal propensity. These literature surveys revealed that there is mixed evidence at best for the hypothesis that parasite and pathogen pressure increase with decreasing latitude, and—consistent with these mixed results—there was little support for a consistent latitudinal gradient in plant dispersal propensity. However, very few studies have quantified biogeographic variation in dispersal, and I found that none of these studies simultaneously tested for covariation between dispersal traits and disease pressure. Nonetheless, it is clear that both disease pressure and plant dispersal traits each show high spatial variation. I propose an experimental design that tests if host plants exhibit phenotypic plasticity in their dispersal traits in response to the presence of parasites or pathogens, as well as the genetic and environmental sources of dispersal trait variation across geographic gradients. Understanding the relationship between dispersal and the presence of parasites and pathogens will be key to predicting the responses of these relationships and the species evolved to a rapidly changing world.

1. Introduction

Dispersal of seeds or spores by maternal plants has evolved as a mechanism to facilitate persistence in spatially and temporally variable environments (Levin et al., 1984), allowing plants to track favorable environments and escape unfavorable environments through generations (Rubio de Casas et al., 2012). In an environment in which local extinctions are possible, selection will always favor some dispersal (Hamilton & May, 1977), though once occupying a favorable patch, dispersal will no longer be favored due to the benefit of remaining there (Hastings, 1983). Dispersal, however, is not as straightforward as individuals simply leaving unfavorable areas in pursuit of better conditions. Due to high costs in energy, time, and opportunity, dispersal can be risky, and a variety of biotic and abiotic conditions (i.e., predators, competitors, resources, dispersal vectors such as wind and water, etc.) may significantly increase or decrease chances of survival during the process of dispersal (Bonte et al., 2012). Furthermore, even if the dispersing organism reaches a suitable habitat, the chances of successful establishment may be low (Ronce, 2007). While plasticity (or "condition-dependence") in dispersal is the optimal strategy compared to a fixed probability of dispersing (McPeek & Holt, 1992), there are few tests of its extent and role in natural ecosystems. Changes in dispersal have been observed in response to unfavorable environments, such as those with high competition, drought conditions, and limited nutrients (Mandák & Pyšek, 1999; Imbert & Ronce, 2001; Brändel, 2007; Martorell & Martínez-López, 2014). Janzen (1970) and Connell (1971) proposed that the favorability of an environment surrounding a dispersing maternal plant decreases when the environment contains enemies such as parasites and pathogens. Indeed, evidence suggests that parasites and pathogens can influence the success of a dispersal event with higher numbers of parasites or pathogens having more deleterious effects on short-distance dispersal events (Augspurger, 1983; Bagchi et al., 2010).

Parasitism has been observed across many taxa and is one of the most widespread relationships in ecology (Poulin & Morand, 2004). Some of the most well-known parasitic relationships are those that include humans as hosts and lead to disease. For example, infection by *Plasmodium falciparum* can lead to malaria, and infection by the cercariae of trematode parasites of the genus *Schistosoma* can lead to schistosomiasis, two diseases that result in hundreds of millions of human cases every year (Vergee, 2019; World malaria report, 2020). Microorganisms that cause disease, like the species of *Lentivirus* that cause *HIV/AIDS* (Haase, 1986), are known as pathogens, though this simple definition is limited in capturing the full complexity of the concept at large (Casadevall & Pirofski, 2002). While certain parasites and pathogens of human hosts may be particularly well-studied, parasites and pathogens of plants also have large human impacts through high economic and ecosystem costs. An outbreak of potato late blight, a disease caused by *Phytophthora infestans*, in the 1840s resulted in the particularly impactful Irish potato famine and diaspora, and, even in 2008, this disease was still causing an estimated annual €1,000,000,000 global loss (Haverkort et al., 2008). Chestnut blight, a disease caused by *Cryphonectria parasitica,* essentially wiped out one of the United States' most economically and ecologically important hardwoods, the chestnut tree (Anagnostakis, 1987). A more recent example in western North America is the widespread destruction of lodgepole pine (*Pinus contorta)* populations by *Ophiostoma clavigerum*, a pathogen carried by mountain pine beetles (*Dendroctonus ponderosae*), which has resulted in significant economic and ecosystem loss in the United States and Canada (DiGuistini et al., 2007; Rosenburger et al., 2013). The impact of parasites and pathogens of plants cannot be understated, and understanding how host plants respond to presence and infection is essential to predicting and mitigating current and future ecosystem and agricultural concerns.

The likely interaction between parasites and pathogens and host plant dispersal leads to the following question to be asked: is there a relationship between geographic variation in parasites and pathogens and variation in host plant dispersal? I hypothesize that geographic variation in parasites and pathogens corresponds with geographic variation in host plant dispersal. The strongest prediction from this hypothesis is that the two mirror each other directly, with patterns of increasing parasite and pathogen prevalence (i.e., the proportion of infected hosts in a population) and intensity (i.e., the number of parasites or pathogens per infected individual) being accompanied by similar patterns in host plant dispersal. The relationship between geographic variation in parasites and pathogens and host plant dispersal has not previously been examined, and the purpose of this thesis is to explore this relationship in depth using the well-known latitudinal gradient as a case study.

In this literature synthesis, I connect host plant dispersal with parasite and pathogen prevalence and intensity, examine the latitudinal patterns of parasites and pathogens, examine the latitudinal patterns of seed dispersal in plants, and synthesize this information to look for patterns in latitudinal variation. I then identify a path forward for future research that could address outstanding research questions on this topic, proposing an experimental design for isolating the effects of parasites and pathogens on host seed dispersal. Finally, I discuss the importance of these studies in the face of shifts in broad scale geographic patterns like climate change, as these shifts are predicted to alter host-parasite and host-pathogen relationships (Hansen et al., 2001; Santini & Ghelardini, 2015), potentially impacting the persistence of both parasite and pathogen species and host plant species involved.

2. Relationship between host plant dispersal and parasites and pathogens

Dispersal phenotypes are the suite of maternal traits that include both seed and adult characteristics that impact dispersal (Donohue, 1998, 1999; Larios & Venable, 2015). Dispersal traits are often grouped by the vector that they take advantage of, and traits within these groups can take on many different forms (Cousens et al., 2008). For example, traits grouped by the use of animals as vectors span a range of values for seed hardness, size, and shape (Boedeltje et al., 2015) as well as the presence or absence of more specialized structures such as the elaiosome to increase dispersal by ants (Mark & Olesen, 1996). Similarly, for traits grouped by the use of water as a vector, traits may include an oil coating around the seed (Ikeda & Itoh, 2001) or a spongy mesocarp to promote flotation (Ridley, 1930). Additionally, traits grouped by the use of humans as a vector may include dispersal through agricultural activities (McCanny & Cavers, 1988), and traits grouped by the use of the self as a vector may include dispersal by rapid, explosive ejection of seeds (Vittoz et al., 2007). The most heavily studied grouping is the suite of dispersal traits in which wind is used as the vector. This grouping includes a wide variety of well categorized traits, such as pappus size (Bartle et al., 2013), wing loading (Tabassum & Leishman, 2018), and seed weight (Hong et al., 2021). While many traits are strongly associated with a particular dispersal vector, this does not mean that each trait is exclusive to that vector. For example, decreased seed mass has been positively associated with wind dispersal in *Gladiolus gueinzii* (Huang et al., 2015) as well as with animal dispersal in *Sparganium emersum* (Pollux et al., 2007). In contrast, seed wing size (measured as “wing index”) has been shown to be positively related to wind dispersal while not being related to water dispersal (Darling et al., 2008). Furthermore, some traits have opposite effects in different species; for example, maternal plant height enhances seed dispersal by wind in *Scrophularia canina* (Rodríguez-Riaño et al., 2019) but restricts dispersal by wind in *Cakile edentulal* var. *lacustris* (LaRue et al., 2018). The selection and expression of these phenotypes is dependent on the quality of environment in which a maternal plant is in (Rubio de Casas et al., 2012).

Janzen (1970) and Connell (1971)**,** through an analysis of tree diversity in the tropics, proposed that pathogens and other enemies of seed-producing plants will decrease the quality of the environment surrounding the maternal plant, which may lead to increased dispersal in susceptible plant hosts. This pattern is observed when the dispersal distance of the plant is further than the mobility distance of the pathogen or enemy (Nathan & Casagrandi, 2004) as parasites and pathogens decrease the quality of life for their plant hosts by stealing their resources and causing diseases which are often accompanied by harmful symptoms (Hall et al., 2009). Disease symptoms are highly variable but often severe. For example, crown gall disease, caused by *Agrobacterium tumefaciens*, can result in the development of tumors in infected plants (Drummond, 1979), and soft rot, caused by *Pectobacterium carotovorum* (formerly known as *Erwinia carotovora)*, produces enzymes that result in a decrease of turgidness in the host plant (Charkowski, 2006). In addition to severe symptoms, some fungal pathogens will produce mycotoxins, which are secondary toxic metabolites that aid in infection (Turner et al., 2009), and ectoparasites, such as *Cuscuta campestris*, will typically directly deplete their host of resources such as nutrients, water, and energy (Irving & Cameron, 2009). To prevent and respond to parasites and pathogens, plants employ a wide variety of often costly defense mechanisms, such as pathogen-inhibiting proteins, the productions of trichomes to prevent initial attachment, and the use of defensive chemical compounds to prevent infection (Freeman & Beattie, 2008). These harms and costs factor into why an environment containing parasites and pathogens is not a favorable environment for susceptible plants to live in. Indeed, evidence from fungal pathogens that cause damping-off, a disease that affects seedlings, suggests that pathogens in the surrounding area of the maternal environment will cause the maternal plant to favor long distance dispersal over short distance dispersal to escape those potential harmful relationships (Augspurger, 1983).

The presence of parasites and pathogens in an environment likely impacts host plant dispersal, but dispersal of host plants also likely impacts the distribution of parasites and pathogens. Parasites and pathogens live in a close relationship with their host, often evolving to specialize on their host, and, as such, are expected to track their hosts through geographic movements (Poulin, 2014). Parasites and pathogens have been shown to be impacted by the presence and density of available hosts (Burdon & Chilvers, 1982; Arneberg et al., 1998), and successful dispersal of host plants may affect parasite and pathogen distribution, though a recent examination of above-ground plant pathogens suggest that host abundance does not impact rates of infection (Schmidt et al., 2020). The relationship between variation in host plant dispersal and parasites and pathogens is likely complex and requires further examination. To identify if there are corresponding geographic patterns between variation in parasites and pathogens and host plant dispersal, I first reviewed the literature for currently known biogeographic patterns in host-parasite and host-pathogen interactions.

3. Biogeographic patterns in host-parasite and host-pathogen interactions

A hypothesis that biotic interactions increase towards the tropics (Schemske et al., 2009) predicts that the intensity of host-parasite and host-pathogen interactions will increase with decreasing latitudes. Consistent with this hypothesis, plant resistance to rust and rust virulence both increase towards lower latitudes (Schemske et al., 2009). Similar patterns have been observed in human pathogens (Guernier et al., 2004), helminth parasites (Preisser, 2019), and ectoparasites of teleost fish (Rhode and Heap, 1998). However, several results from other studies indicate that this pattern is far from universal. For example, the intensity of trematode parasites in snails (Torchin et al., 2015) and the density of helminth parasites of frogs (Johnson & Haas, under review) both appear to decrease towards the tropics, and the presence and intensity of some bacterial, viral, and fungal infective agents in humans have no apparent relationship with latitude (Guernier et al., 2004). This conflicting evidence provides mixed support at most for the hypothesis that host-parasite and host-pathogen interactions are stronger in the tropics than at higher latitudes, though it is clear that the nature and strength of these interactions vary substantially across space.

A canonical biogeographical pattern in diversity that may be relevant to host-parasite interactions is latitudinal diversity gradient (LDG) in ecology. The LDG is the observation that species diversity and richness increase as latitude decreases towards the tropics (Fischer, 1960), and it has been confirmed across many plant and animal taxa on all continents and in every ocean, though there are some exceptions (Willig et al., 2003). Along with the LDG, the 'host-diversity-begets-parasite-diversity' hypothesis, which predicts parasite diversity to increase as host diversity increases, has been confirmed across many parasite and pathogen lifestyles, niches, and host taxa (Hechinger & Lafferty, 2005; Kamiya et al., 2014; Poulin, 2014; Johnson et al., 2016). While this has not been extensively examined in plant parasites and pathogens, diversity in soybean fungal diseases has been shown to increase towards lower latitudes (Yang & Feng, 2001), and crops in the tropics have been observed to be infected by more diseases than crops in the temperate zone (Wellman, 1968). The high diversity in the tropics is in part maintained by increased disease pressure from parasites and pathogens leading to increased dispersal in tropical forests, as is proposed by the Janzen (1970) and Connell (1971) hypothesis.

4. Analyzing published biogeographic data on intraspecific dispersal variation

Several studies have tested for biogeographic patterns in dispersal traits across species. Seed mass and plant height increase towards lower latitudes (Moles et al., 2007; Moles et al., 2009), and, more generally, dispersal distance increases towards lower latitudes as well (Chen et al., 2019). However, by focusing on species-level traits, these studies have limited potential to provide insight into how dispersal traits vary in response to host-pathogen or host-parasite interactions, which are often specialized to individual species pairs (Poulin, 2014). To better evaluate patterns of dispersal trait variation in plant hosts in response to their parasites and pathogens, I reviewed the literature for studies that tested for geographic variation in plant dispersal traits across individual species ranges.

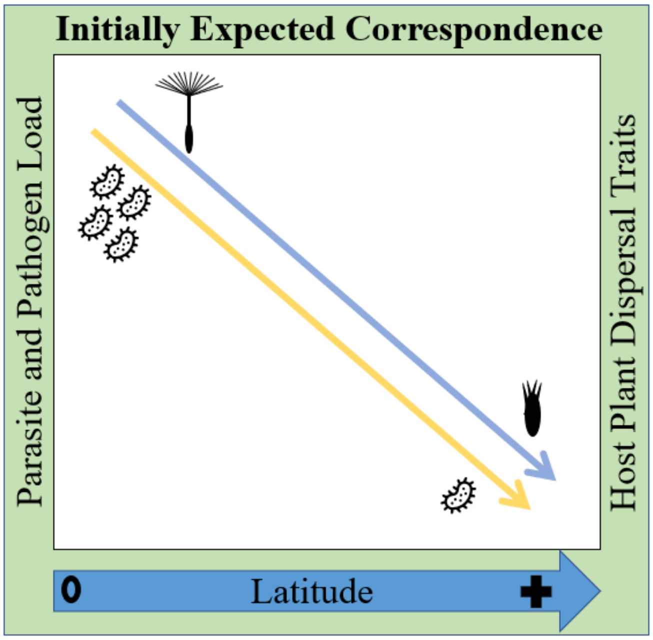


Figure 1: Initial expectations for host plant dispersal trait variation to correspond with increased parasite and pathogen load (i.e., parasite or pathogen intensity and virulence) towards the tropics in a latitudinal gradient.

I initially approached my review of intraspecific geographic variation in dispersal traits with the goal of testing the hypothesis that dispersal traits co-vary with patterns in parasites and pathogens. Based on the latitudinal gradient in biotic interactions hypothesis (Schemske et al. 2009) and the LDG (see above), I aimed to test if I observed a latitudinal gradient in plant dispersal traits that would correspond to increasing disease pressures with decreasing latitudes (*Fig. 1)*. However, my ultimate assessment of the literature on biogeographic variation in parasites and pathogens suggested that latitudinal variation in these interactions varies substantially among taxa (see above). Consequently, I revised my hypothesis to that I would expect to see geographic variation in dispersal traits more generally, which is consistent with (though not definitively related to) the widespread geographic variation in host and parasite interactions that has been reported in the literature.

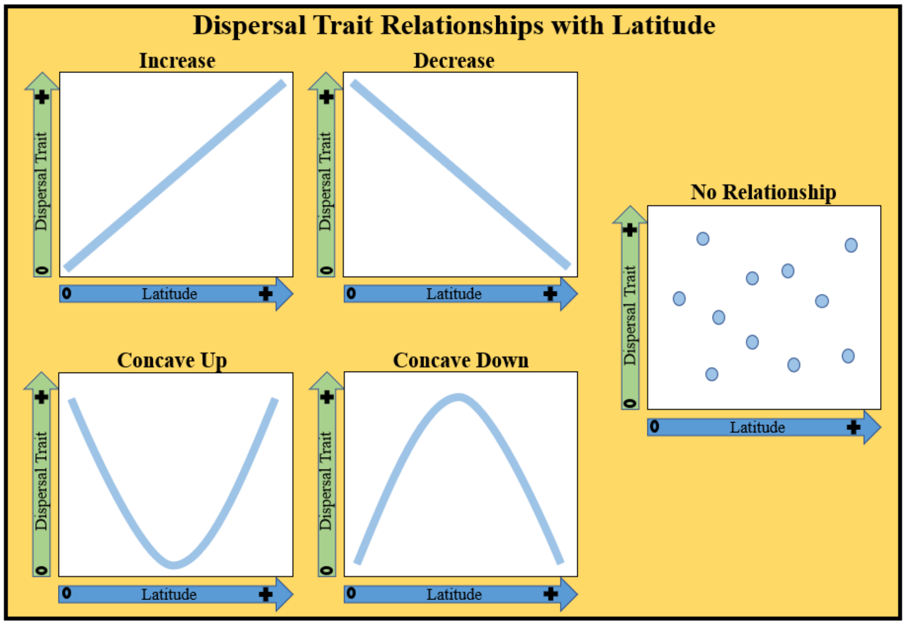
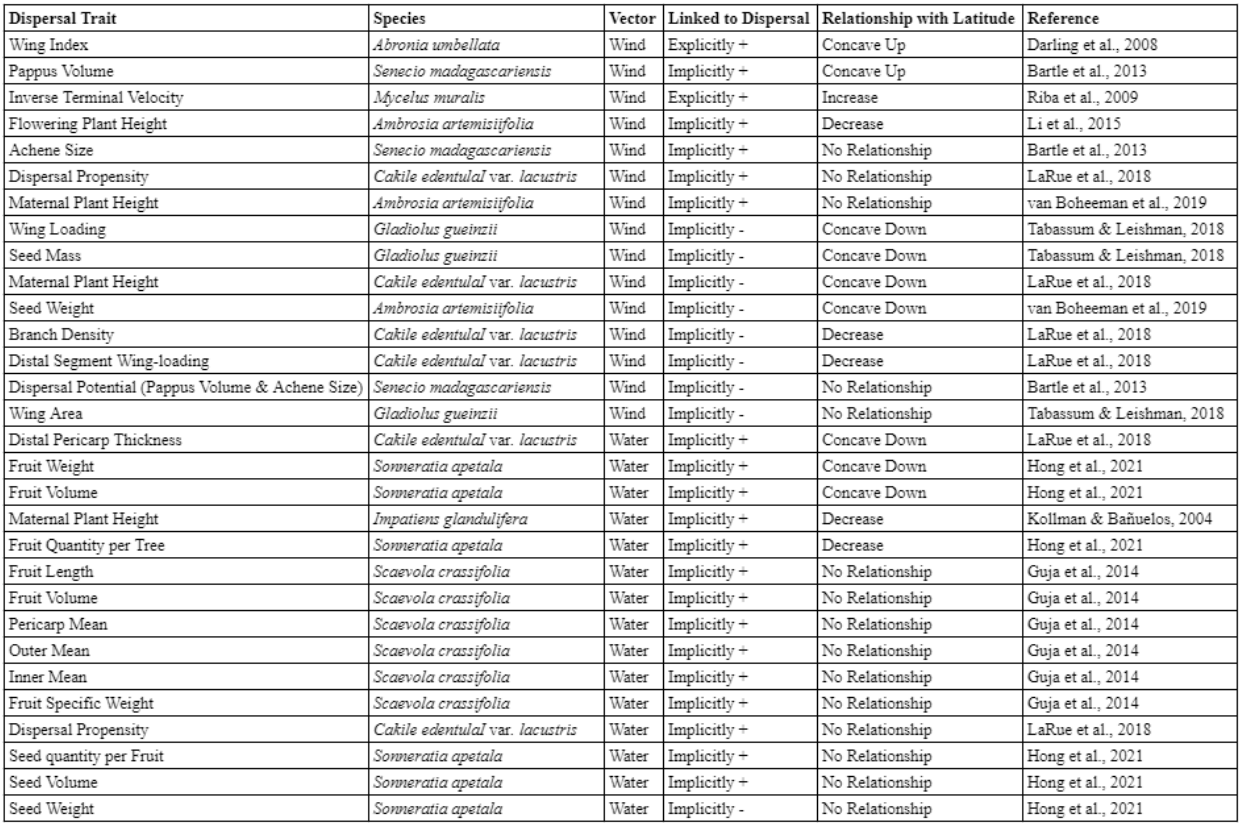


Figure 2: Potential patterns in dispersal traits as latitude increases. If the dispersal trait increases as latitude increases, the relationship is labeled 'Increase.' If the dispersal trait decreases as latitude decreases, the relationship is labeled 'Decrease.' If the dispersal trait increases to both the tropics and increasing latitudes, the relationship is labeled 'Concave Up.' If the dispersal trait decreases to both the tropics and increasing latitudes, the relationship is labeled 'Concave Down.' Finally, if the dispersal trait exhibits no relationship with latitude, it is labeled 'No Relationship.'

My literature review focused on empirical studies that specifically quantified intraspecific variation in dispersal traits of plants, with a particular emphasis on variation across latitudinal gradients. To find papers published on this topic, I searched Google Scholar using the following keywords: “Dispersal,” “Trait,” “Plants,” “Species,” “Range,” “Latitude,” “Gradient,” “Center,” “Edge,” “Core,” and “Periphery.” Only papers written in the English language were included in my review. Of the papers that met the search criteria, I summarized the following components: dispersal trait(s) (e.g., seed size), the dispersal vector (ex. wind, water, animal, etc.), the relationship between the trait and dispersal distance (dispersal-enhancing vs. dispersal-restricting). I then identified the latitudinal pattern (*Fig. 2)* as characterized in each paper or, if no pattern was identified, characterized the pattern of variation in each trait from lower latitudes to higher latitudes using linear regressions (R Core Team, 2020). The results of this literature review are displayed in *Table 1*.

Table 1: Each row represents a trait and vector relationship, and each column represents a certain feature of the relationship. The first, second, and third columns are the dispersal trait (ex. seed size), plant species, and the vector (ex. wind, water, animal, etc.) of that dispersal trait. The fourth column describes if the trait is explicitly or implicitly linked to dispersal and whether the relationship was + (dispersal-enhancing) or - (dispersal-restricting) in the literature I pulled from. The fifth column identifies the observational pattern of each trait as latitude increases (see Fig. 2). Finally, the sixth column lists the associated reference (Kollman & Bañuelos, 2004; Darling et al., 2008; Riba et al., 2009; Bartle et al., 2013; Guja et al., 2014; Huang et al., 2015; Li et al., 2015; LaRue et al., 2018; Tabassum & Leishman, 2018; van Boheemen et al., 2019; Hong et al., 2021).



Dispersal Trait	**Species**	**Vector**	**Linked to Dispersal**	**Relationship with Latitude**	**Reference**
Wing Index	*Abronia umbellata*	Wind	Explicitly +	Concave Up	Darling et al., 2008
Pappus Volume	*Senecio madagascariensis*	Wind	Implicitly +	Concave Up	Bartle et al., 2013
Inverse Terminal Velocity	*Mycelus muralis*	Wind	Explicitly +	Increase	Riba et al., 2009
Flowering Plant Height	*Ambrosia artemisiifolia*	Wind	Implicitly +	Decrease	Li et al., 2015
Achene Size	*Senecio madagascariensis*	Wind	Implicitly +	No Relationship	Bartle et al., 2013
Dispersal Propensity	*Cakile edentulal* var. *lacustris*	Wind	Implicitly +	No Relationship	LaRue et al., 2018
Maternal Plant Height	*Ambrosia artemisiifolia*	Wind	Implicitly +	No Relationship	van Boheeman et al., 2019
Wing Loading	*Gladiolus gueinzii*	Wind	Implicitly -	Concave Down	Tabassum & Leishman, 2018
Seed Mass	*Gladiolus gueinzii*	Wind	Implicitly -	Concave Down	Tabassum & Leishman, 2018
Maternal Plant Height	*Cakile edentulal* var. *lacustris*	Wind	Implicitly -	Concave Down	LaRue et al., 2018
Seed Weight	*Ambrosia artemisiifolia*	Wind	Implicitly -	Concave Down	van Boheeman et al., 2019
Branch Density	*Cakile edentulal* var. *lacustris*	Wind	Implicitly -	Decrease	LaRue et al., 2018
Distal Segment Wing-loading	*Cakile edentulal* var. *lacustris*	Wind	Implicitly -	Decrease	LaRue et al., 2018
Dispersal Potential (Pappus Volume & Achene Size)	*Senecio madagascariensis*	Wind	Implicitly -	No Relationship	Bartle et al., 2013
Wing Area	*Gladiolus gueinzii*	Wind	Implicitly -	No Relationship	Tabassum & Leishman, 2018
Distal Pericarp Thickness	*Cakile edentulal* var. *lacustris*	Water	Implicitly +	Concave Down	LaRue et al., 2018
Fruit Weight	*Sonneratia apetala*	Water	Implicitly +	Concave Down	Hong et al., 2021
Fruit Volume	*Sonneratia apetala*	Water	Implicitly +	Concave Down	Hong et al., 2021
Maternal Plant Height	*Impatiens glandulifera*	Water	Implicitly +	Decrease	Kollman & Bañuelos, 2004
Fruit Quantity per Tree	*Sonneratia apetala*	Water	Implicitly +	Decrease	Hong et al., 2021
Fruit Length	*Scaevola crassifolia*	Water	Implicitly +	No Relationship	Guja et al., 2014
Fruit Volume	*Scaevola crassifolia*	Water	Implicitly +	No Relationship	Guja et al., 2014
Pericarp Mean	*Scaevola crassifolia*	Water	Implicitly +	No Relationship	Guja et al., 2014
Outer Mean	*Scaevola crassifolia*	Water	Implicitly +	No Relationship	Guja et al., 2014
Inner Mean	*Scaevola crassifolia*	Water	Implicitly +	No Relationship	Guja et al., 2014
Fruit Specific Weight	*Scaevola crassifolia*	Water	Implicitly +	No Relationship	Guja et al., 2014
Dispersal Propensity	*Cakile edentulal* var. *lacustris*	Water	Implicitly +	No Relationship	LaRue et al., 2018
Seed quantity per Fruit	*Sonneratia apetala*	Water	Implicitly +	No Relationship	Hong et al., 2021
Seed Volume	*Sonneratia apetala*	Water	Implicitly +	No Relationship	Hong et al., 2021
Seed Weight	*Sonneratia apetala*	Water	Implicitly -	No Relationship	Hong et al., 2021

In total, I found only ten studies that fit the search criteria. These ten studies collectively evaluated 30 dispersal traits in nine different species. All studies focused on wind and water vectors of dispersal. Of the 30 dispersal traits, 15 had statistically significant relationships with latitude. Of the significant relationships, dispersal-restricting traits (i.e., higher trait values reduce dispersal distance) increased with latitude or were higher at latitudinal range limits than in the range core. There was no instance of a dispersal-restricting trait increasing away from the tropics. Of the significant relationships, dispersal-enhancing traits (i.e., higher values of the trait increase dispersal distance) exhibited more variable latitudinal patterns than the dispersal-restricting traits. Some significant dispersal-enhancing traits were highest at lower latitudes or highest at latitudinal limits but not in the range core, while the remaining dispersal-enhancing traits were lowest at lower latitudes or lowest at latitudinal limits but not in the range core. When grouped by vector, wind-dispersed traits exhibited highest dispersal values at latitudinal range limits and away from the tropics, and while most water dispersed traits showed no relationship with latitude, those that did showed highest dispersal values at the center of their ranges or towards the tropics.

To further examine this review, a global model could be built using the data points from each paper with the response variable being dispersal trait values standardized to z-scores and the predictor variables being latitude and species. This global model would provide a more general analysis of these results. However, given time restraints and limited access to data points, this was beyond the scope of this review.

Overall, my review of the literature provides clear evidence that dispersal traits do vary across plant species ranges, though, there is no consistent pattern with latitude. The existence of geographic variation in both dispersal traits and parasites and pathogens, and the tendency for each to co-vary with latitude (though in different ways), is consistent with the hypothesis that plant dispersal traits may respond to parasite or pathogen prevalence and intensity through local adaptation or phenotypic plasticity. However, this evidence is far from conclusive as I found no studies that simultaneously quantified dispersal trait variation and parasites and pathogens over geographic scales. Given that relatively few studies have actually quantified intraspecific variation in dispersal traits, it is not surprising that we lack studies that have directly linked patterns in dispersal trait variation to patterns in parasites and pathogens. To determine a causal relationship, experimental tests that isolate and identify the extent to which geographic patterns in parasite and pathogen pressure relates to geographic variation in host plant dispersal traits are needed.

5. Identifying the Effects of Parasites & Pathogens on Geographic Variation in Dispersal Traits

The literature review described above (*Table 1)* reveals that substantial variation in dispersal traits exists within plant species, though the patterns of dispersal trait variation do not obviously correspond to the latitudinal gradients that have been documented across species (Moles et al., 2007, Moles et al., 2009). Without direct experimental tests, it is difficult to pinpoint the extent to which the dispersal traits expressed by different plant populations and individuals within the populations are responding to parasites and pathogens and, furthermore, the extent to which parasites and pathogens are responding to dispersal trait variation. Meta-analyses, though useful in identifying broad-scale patterns, can have significant drawbacks in determining causal mechanisms due to mismatching geographical scales, time scales, methods, and analyses of the component parts. One limitation of this meta-analysis in particular is that five of the nine species examined for geographic patterns in dispersal trait variation were invasive species. Invasive species are not as affected by parasites and pathogens as native species (Torchin et al., 2003), leading to further difficulty in matching dispersal variation patterns to parasite and pathogen patterns. Even among invasive species, however, there was still variation in relationships with latitude. Observational data alone cannot determine if the dispersal trait variation observed is due to phenotypic plasticity (i.e., condition-dependent dispersal) or genetic differences among host populations.

Carefully designed experimental studies would be required to separate the genetic and environmental drivers of dispersal trait variation in response to parasites and pathogens and test if that variation is adaptive. Specifically, controlled experiments can test between alternative hypotheses for the underlying mechanisms causing dispersal trait variation in response to parasites and pathogens: (1) the Adaptive Plasticity hypothesis, (2) the Physiological Constraint hypothesis, and (3) the Genetic Differentiation hypothesis. The Adaptive Plasticity hypothesis predicts that host plants affected by parasitism and pathogens will increase their allocation to traits that enhance dispersal to reduce offspring exposure to the parasite or pathogen. In contrast, the Physiological Constraint hypothesis predicts that host plants affected by parasites or pathogens will exhibit a reduction in dispersal because dispersal (i.e., dispersal traits) is costly, and the stress of infection reduces the number of resources a plant can allocate to dispersal. Finally, the Genetic Differentiation hypothesis, predicts that individual plants will not change their dispersal strategy in response to pathogen or parasite pressure, but that populations will exhibit fixed (non-plastic) strategies that reflect local adaptation to the historical patterns of parasite and pathogen pressure in their home locations.

I propose an experimental design with host plants and parasites or pathogens (*Fig. 3*) that tests whether geographic variation in dispersal traits is caused by genetic variability or phenotypic plasticity and, if phenotypic plasticity is the main driver, whether the relationship falls under the Physiological Constraint hypothesis or the Adaptive Plasticity hypothesis. For this experimental design, one should first identify a plant-parasite or plant-pathogen whose host plant exhibits geographic variation in dispersal traits. Seeds from multiple host plant populations are raised in a common garden environment. To specifically test if host-parasite or host-pathogen interactions drive latitudinal variation in host plant dispersal, it would be ideal to include replicate host populations for multiple latitudes (e.g., 5 latitudes with 3 host plant populations/latitude). Individuals within each population are randomly assigned to one of two groups: infected and uninfected. The uninfected treatment serves as the experimental control, and plants in this group are raised with no parasites or pathogens added. For the infected treatment, a set amount of a single parasite or pathogen is added. A single parasite or pathogen is chosen over parasites or pathogens from each plant population due to the ability of parasites and pathogens to specialize on their host. To examine the effect of the intensity of infection, several more treatment groups can be formed with differing amounts of parasites or pathogens added as a modification to this experimental design. The timing of introduction of the parasites or pathogens occurs before the developmental trajectory for dispersal traits has been determined by the host plant. As the plants age, traits that have been mechanistically linked to dispersal distance are measured at regular intervals. The statistical analysis for this design would involve an ANOVA with the dispersal trait values as the response variable and host plant location (e.g., latitude) and treatment as the predictors, with population included as a random intercept term. This experimental design can be extended to testing the effect of parasites and pathogens on host plant dispersal across other geographic gradients as well by replacing latitude with elevation, range position (center vs. edge), humidity, or other environmental factors that vary among populations.

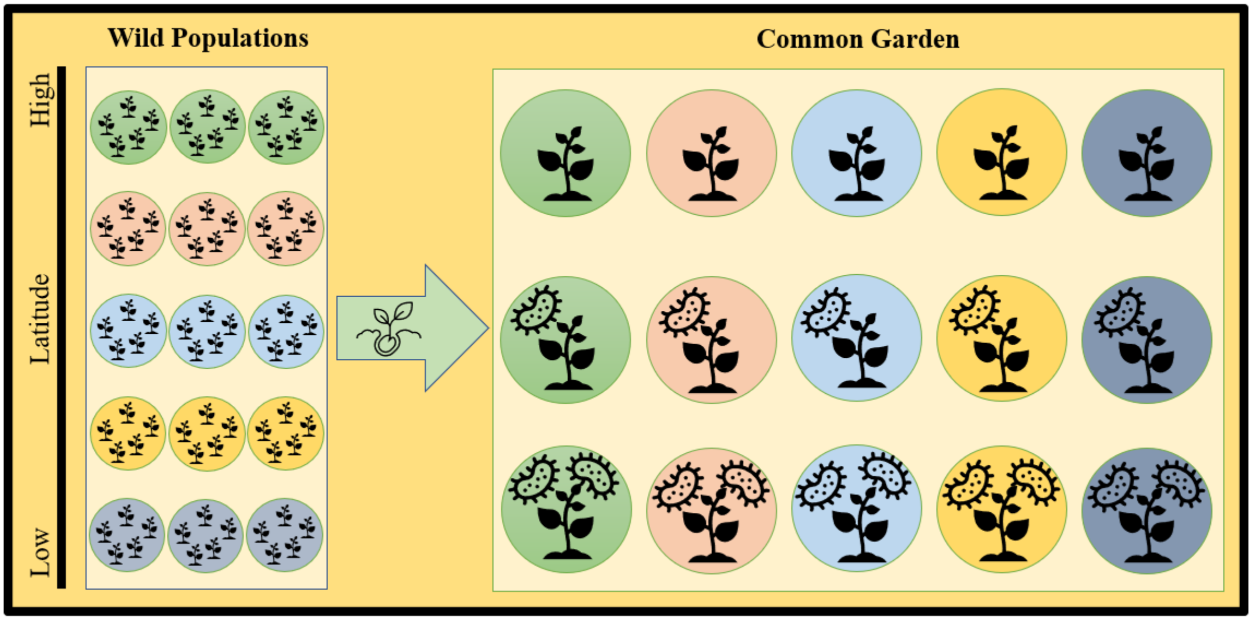


Figure 3: Proposed experimental design to test the effect of parasites and pathogens on dispersal strategies of their hosts across a geographic range (in this case, latitude). Plants of a single species from five source latitudes (three populations per latitude) (left) are selected. Their seeds are planted in a common garden experiment (right) where a single parasite or pathogen is introduced at varying levels to each population before their dispersal strategy is determined. The dispersal traits of these plants are measured, and a model is fit to determine whether plants are responding under the Adaptive Plasticity hypothesis, Physiological Constraint hypothesis, or Genetic Differentiation hypothesis.

Due to this being a common garden experiment, the effect of population location (e.g., latitude) can be evaluated to test if the dispersal traits are affected by genetic differences among the source locations. A significant treatment effect would indicate that dispersal traits are plastic. The direction of this response will show either adaptive plasticity or physiological constraint, as adaptive plasticity would show an increase in dispersal potential with increasing parasite or pathogen pressure while physiological constraint would yield the opposite pattern. If the Physiological constraint hypothesis is at play, I expect plants with a higher parasite or pathogen load to show decreased dispersal. Finally, if the Genetic Differentiation hypothesis is at play, I expect plants with a higher parasite or pathogen load to show fixed dispersal traits, regardless of the parasite or pathogen treatment.

Ideally, this experiment would be repeated across multiple host taxa and multiple parasite and pathogen taxa to test if any resulting patterns are general or contingent on the ecological and evolutionary context of individual species. Furthermore, this experiment can be repeated with multiple parasite or pathogen taxa added to the host plants at the same time to determine the effects of parasite and pathogen diversity and disease combination. This experimental design will isolate the importance of parasite and pathogen variation on host plant dispersal trait variation, allowing for patterns of parasites and pathogens and host plant dispersal traits to be more easily observed and predicted, which is of utmost importance in a rapidly changing world.

6. Significance

Shifts in broad geographic patterns like climate change have the potential to alter biogeographic patterns (Saupe et al., 2019). Current evidence suggests that increasing temperatures and induced stress as a result of climate change leads to higher susceptibility and vulnerability to disease for some plant species (Hansen et al., 2001; Seherm & Coakley, 2003; Desprez-Loustau et al., 2007). Higher temperatures have increased the proportion of soil-borne reservoir pathogens (Delgado-Baquerizo et al., 2020), demonstrating the immediacy of this problem. Similarly, shifts towards increased greenhouse gas outputs are expected to expand plant disease occurrence, and shifts towards increased humidity are expected to enhance harmful mycotoxin production by fungal pathogens (Velásquez et al., 2018). Plant pathogens are highly adaptable to shifts in broad geographic patterns, and, due to this potential, the emergence of new diseases in vulnerable ecosystems is likely (Santini & Ghelardini, 2015). Understanding the relationship between host plant dispersal and the prevalence and intensity of parasites and pathogens is key to successfully predicting and mitigating the future of plant disease and how involved species of plant hosts, parasites, and pathogens may persist in a rapidly changing world.

7. Acknowledgments

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