Causes and consequences of fungal pathogen infection in grasslands

Inaugural dissertation
of the Faculty of Science,
University of Bern

presented by

Seraina Lisa Cappelli

from Lütisburg SG

Supervisor of the doctoral thesis:
Prof. Eric Allan
Institute of Plant Sciences, University of Bern

Accepted by the Faculty of Science.

Bern, January 21st 2020

The Dean
Prof. Dr. Zoltan Balogh

Public defense at the Institute of Plant Sciences, University of Bern

January 21st 2020

Promotion Committee

Prof. Dr. Eric Allan

Institute of Plant Sciences

University of Bern

Prof. Dr. Jasmin Joshi

Landschaftsarchitektur, ILF

Hochschule für Technik Rapperswil

Chair

Prof. Dr. Matthias Erb

Institute of Plant Sciences

University of Bern

“Have no fear of perfection – you’ll never reach it.”

(Salvador Dalí)

Table of Contents

Chapter 1

General introduction 3

Chapter 2

Sick plants in grassland communities: a growth-defense trade-off is the main driver of fungal pathogen abundance and impact. 17

Chapter 3

Resource use traits predict a growth-defense trade-off between, but not within, species 49

Chapter 4

Consistent biodiversity effects across functions 83

Chapter 5

Summary and general conclusions 133

References 145

Acknowledgements 167

Declaration of consent 171

Curriculum Vitae 173

Chapter 1
General introduction

The kingdom of fungi is highly diverse and is estimated to comprise roughly three million species, occupying a wide range of niches (Hawksworth and Lücking 2017). They range from relatively simple, unicellular microorganisms such as yeasts, to *Armillaria* species stretching out over hundreds of hectares of forest floor. Some mushrooms feed on decaying dead matter, some form symbiosis with plants to access energy rich resources and some use living organisms as food sources, sometimes even actively preying on them or altering the behavior of their host to their advantage (Petersen 2012). To adapt to these highly diverse niches, some fungi have developed complex lifecycles, which can include both sexual and asexual reproduction, multiple sexes and dikaryotic phases (Jennings and Lysek 1996). Fungi are involved in nutrient cycling, help to maintain healthy forests, act as a food source, are part of spiritual ceremonies in many regions of the world and are used in the production of enzymes, medicine and beer, making them vital for human wellbeing and the maintenance of functioning ecosystems (Petersen 2012).

Fungal pathogens

Of particular interest among this staggering diversity of fungi are plant pathogenic fungi. Only approximately 10% of all fungal species are associated with living plants and the majority do not cause diseases (Knogge 1996). Nevertheless, fungal pathogens are responsible for 70% of all known plant diseases (Carris et al. 2012) and fungal diseases cause approximately 12% of yield losses in agricultural crops (Oerke 2006). Foliar pathogenic fungi can cause damage through deprivation of nutrients and inhibition of photosynthesis (Swarbrick et al. 2006; Mitchell 2003). Fungal infection can result in reduced growth (Lively et al. 1995), deformation (Roy 1993; Alexander and Burdon 1984), reduced reproductive output (Alexander et al. 1985; Alexander and Burdon 1984), premature senescence (Goodall et al. 2012) or even mortality (Thrall and Jarosz 1994; Alexander and Burdon 1984; Ridenour and Callaway 2003) in the host plant.

Impact of pathogens on plant communities

The negative impact of fungal pathogens on their host plants is reflected in the biomass reduction of whole plant communities (Mitchell 2003; Allan et al. 2010; Seabloom et al. 2017). In addition to reducing biomass production, fungal pathogens are thought to

influence plant community composition (Burdon 1991). They can alter the outcome of pairwise competition between different species (Paul 1989; Ridenour and Callaway 2003; Paul and Ayres 1990). By changing competitive interactions, fungal pathogens can potentially influence plant community composition and determine whether one or the other species is able to persist.

Fungal pathogens might even help to maintain a high host diversity through both equalizing and stabilizing mechanisms (Chesson 2000). They can have an equalizing effect if they reduce competitive differences between their host species by disproportionately affecting highly competitive species. This can allow subordinate species to persist in the plant communities, which would elsewise have been excluded by competition (Chesson 2000; Mordecai 2011). When pathogens increase the relative importance of intraspecific competition compared to interspecific competition, this can further contribute to coexistence and thus diversity (stabilizing mechanism, Chesson 2000; Mordecai 2011). Specialist pathogens often benefit when their host species is at high abundance, because this facilitates their transmission (Alexander and Mihail 2000; Bever et al. 1997). For the host, this means substantially increased negative consequences of growing in the neighborhood of conspecifics. Specialist pathogens should thus prevent total dominance of their hosts. Rare species in the community benefit from reduced specialist pressure, which may prevent extinction (Chesson 2000; Mordecai 2011; Bever et al. 2015).

The composition of the fungal community might greatly influence the consequences of infection for the host community. Fungal pathogens have a wide variety of strategies to attack and exploit their hosts (Møller, Murphy 2018) which likely influences how much damage they cause. The impact on infection on plant communities might depend on how generalistic (Mordecai 2011) and aggressive (Jarosz and Davelos 1995) the fungal pathogens in the community are and more general on their ecological behavior (Schierenbeck et al. 2016). For example, powdery mildews which mostly grow on leaves likely differ from rust species whose mycelium grows inside leaves (Klenke 2015). Powdery mildews might reduce photosynthesis by blocking the leaves from sunlight, something rusts cannot do due to their internal growth. There is likely also a difference between fungal pathogens, which kill host tissue and eventually the host to access the resources and biotrophic pathogens, which rely on living hosts (Møller, Murphy 2018; Jarosz and Davelos 1995). Thus, the composition of the fungal pathogen community might have a large impact on how destructive the pathogens are in their

host plant community.

Pathogen resistance

Plants have developed strategies to deal with the threat of natural enemies. There are two main strategies that plants can use, defense and tolerance. Their joint effect define pathogen resistance of a plant (Haukioja and Koricheva 2000). Defense mechanisms are of structural or chemical nature and are either constantly present or induced upon enemy attack (Walters 2011). For example, species with though leaves are harder to invade for a pathogen than one with soft, tender leaves (preexistent mechanical defense). An example of an induced defense is controlled cell death to compartmentalize enemy damage (Walters 2011). Pathogen tolerance can be achieved for example with compensatory growth, or allocation of resources away from the pathogen (Paul and Ayres 1986). How strongly a plant community is affected by pathogens, is therefore also dependent on the resistance strategies of the plants in the community.

Fungal pathogens have the potential to substantially alter ecosystems and may contribute to ecosystem stability by maintaining species diversity. In a rapidly changing world, it is crucial to understand the role of fungal pathogens to predict and mitigate the consequences of global change. Anthropogenic influences on global temperatures, atmospheric CO_2 levels or nutrient cycles can alter the impact of fungal diseases, with sometimes detrimental consequences for the hosts (Fisher et al. 2012; Helfer 2014; Mitchell et al. 2003; Liu et al. 2017).

Nitrogen enrichment

A particularly important driver of global change is nitrogen enrichment (Rockstrom et al. 2009; Galloway et al. 2008; Sutton 2011). Humans have roughly doubled the supply of reactive nitrogen into the environment globally since 1990 (Galloway et al. 2008), and in Europe even more than tripled. The intensification of agriculture, of which nitrogen enrichment is a part of, has led to a loss of nutrient poor ecosystems, like extensive meadows. In the Swiss lowlands, nutrient poor and species rich ecosystems, such as extensive grasslands, have shrunk to only 2-5% of their extent in 1950. The change has been partly driven by overall agricultural area decline, but was especially driven by agricultural intensification (Gattlen 2016). Even when no direct fertilization occurs, nitrogen deposition as a consequence of fossil fuel combustion unintentionally fertilizes all ecosystems. While nitrogen deposition has decreased in the last 2-3 decades through various measures, it still remains at high levels (Sutton 2011;

Heldstab et al. 2010). Nitrogen enrichment has far-reaching consequences for ecosystems. It can cause diversity loss in many taxa, such as plants (Vellend et al. 2017), fungal pathogens (Blaser 2014), or insects (Haddad et al. 2000), and disrupt species interactions (Ochoa-Hueso 2016).

Direct effects

Nitrogen enrichment can directly increase pathogen load (Mitchell et al. 2003) and change the influence of pathogens on the competitive ability of the host plant (Paul and Ayres 1990). Good nutritional status of a plant could make it more susceptible to pathogens, because it has more resources to offer for a pathogen. However, it is also possible that a high availability of nutrients could allow the plant to invest more resources into defense against pathogens. Such mechanisms are well studied for agricultural plant species (Dordas 2008). In natural ecosystems evidence for direct effects of nitrogen enrichment on infection are mixed, with often no effects observed (Mitchell et al. 2003; Blaser 2014; Veresoglou et al. 2013).

Indirect effects through changes in community composition

Nitrogen enrichment can also indirectly affect fungal pathogens by changing the host plant community, leading to plant communities dominated by species which are more susceptible to disease (Liu et al. 2017; Liu et al. 2018b; Blumenthal et al. 2009). A reason for this is, that nitrogen or nutrient enrichment in general favors faster growing species (e.g. Vellend et al. 2017; Cleland and Harpole 2010) and fast growing species are often less defended against enemies than slower growing species (growth-defense trade-off, Coley et al. 1985; Endara and Coley 2011; Lind et al. 2013). This has been observed for large browsing herbivores (Lind et al. 2013), insect herbivores (Endara and Coley 2011), as well as for microbial pathogens (Blumenthal et al. 2009; Liu et al. 2017). Plants adapted to nutrient rich environments are more tolerant to natural enemies, as lost tissue can be easily replaced through rapid growth and when resources are available (Gianoli and Salgado-Luarte 2017), while species adapted to nutrient poor habitats typically invest more in defense to avoid tissue loss (Endara and Coley 2011; Lind et al. 2013). These trade-offs are known to exist between species, but less is known about whether they hold between populations of the same species or how they scale up to whole plant communities. It could for example be that fast-growing heavily infected species benefit most from host dilution with increasing diversity, which might mask growth-defense trade-offs at the community level.

Leaf economics spectrum

The growth-strategy of plants is defined by the leaf economics spectrum (Wright et al. 2004; Reich and Cornelissen 2014). Species adapted to different nutrient levels have specific sets of traits adapted to the given environment. These traits covary along environmental gradients of nutrient availability. Nutrient poor habitats favor species with slow growth-rates, tough leaves (low specific leaf area, high leaf dry matter content), low nutrient contents and high defense traits. In nutrient rich habitat, species with traits such as fast growth-rates, high light interception (high specific leaf area), high nutrient content and low investment in defense are favored (Wright et al. 2004; Reich and Cornelissen 2014). The measurement of growth-rates requires multiple successive measures of plant size, but because the growth-rate is well correlated with traits of the leaf economics spectrum, they can be used as proxies for the growth strategy. Especially the specific leaf area is a good predictor of plant growth rates (Pérez-Harguindeguy et al. 2013). Originally, the leaf economics spectrum was used to describe the growth strategies of different plants along large environmental gradients (Wright et al. 2004; Reich and Cornelissen 2014). Nowadays, it is also used to characterize plants growing in very similar habitats and as proxies of within-species adaptation to local variation in nutrient availability. However, it is debated whether this approach is appropriate. Within species the leaf economics spectrum exists, but it does not hold for all species, especially at the extremes of the spectrum (Anderegg et al. 2018). This could be due to different selective pressures at different spacial scales, lower variability of traits within than between species (Anderegg et al. 2018; Shipley 2006) and due to variability in plasticity (Poorter et al. 2009). Therefore, the leaf economics spectrum provides a useful framework to assess plant growh-strategies, but it must be considered that it may not hold in all cases.

Indirect effects through changes in diversity

A further mechanism by which nitrogen enrichment can indirectly alter patterns of fungal infection is through changes in species numbers (Keesing et al. 2006; Vellend et al. 2017). The diversity of the host community can have an impact on encounter rates between hosts and pathogens, pathogen transmission, host susceptibility, and recovery from infection or mortality. Host diversity can therefore significantly alter disease dynamics (Keesing et al. 2006). An important mechanism by which diversity does so is through the density of host species (Janzen 1970; Connell 1971). In less diverse communities, the abundance of each host species is higher, which can promote disease spread, as many pathogens depend on host density. Several studies

have tested the role of host abundance in diverse communities. There are many studies which find that an increase of diversity causes a decrease in infection mainly through reduced host abundances (e.g. Mitchell et al. 2002; Liu et al. 2016; Rottstock et al. 2014). However, there are also others which find less support for this, because the diversity effects were mainly driven by the presence of certain species (e.g. Halliday et al. 2017), which caused pathogen spillover, especially to closely related species (Power and Mitchell 2004; Parker et al. 2015).

Species loss

Species loss caused by nitrogen enrichment and by other anthropogenic drivers is not only a major concern for disease dynamics (Rockstrom et al. 2009). Species diversity has been shown to be important for many ecosystem processes and functions, such as biomass production, nutrient cycling and herbivory (Cardinale et al. 2012; Cardinale et al. 2011). The simultaneous maintenance of multiple ecosystem functions in particular, requires a high species diversity (Hector and Bagchi 2007; Lefcheck et al. 2015). As illustrated by the aforementioned example of pathogen infection, the mechanisms by which diversity impacts ecosystem functions are highly diverse and context dependent. These mechanisms can be broadly classified into selection and complementarity effects (Loreau and Hector 2001). Diversity may increase (/decrease) ecosystem function because the majority of the species benefit (/suffer) from growing in a diverse environment and increase (/decrease) their functioning. These effects are called complementarity effects. Positive complementarity effects are common for biomass production. (Cardinale et al. 2011). This can happen for example due to more efficient overall resource use achieved by species with multiple different resource acquisition strategies (Barry et al. 2019). Diverse communities might also benefit from the presence of high-functioning species. The selection effects quantify the extent by which community functioning is provided by few high-functioning species (Loreau and Hector 2001), however this is less common than positive complementarity effects for biomass production (Cardinale et al. 2011). Single species can drive functioning by either dominating the community at the cost of other species, or by changes in functioning without affecting functioning in the other species. An extended tripartite framework accounts for this, by further partitioning the selection effect into two components (Fox 2005). While the bipartite additive partitioning framework of Loreau and Hector (2001) is widely used (1349 citations in web of knowledge), the tripartite partition of Fox (2005) is less established (151 citations in web of knowledge). It is

common to partition diversity effects into selection and complementarity for biomass production but it can be done for other ecosystem functions as well (Grossiord et al. 2013). This however, has been done only few times (Grossiord et al. 2013; Pires et al. 2018; Roscher et al. 2018b). Quantifying diversity effects for different ecosystem functions allows for quantitative comparisons between functions and helps to gain a mechanistic understanding of the consequences of diversity loss.

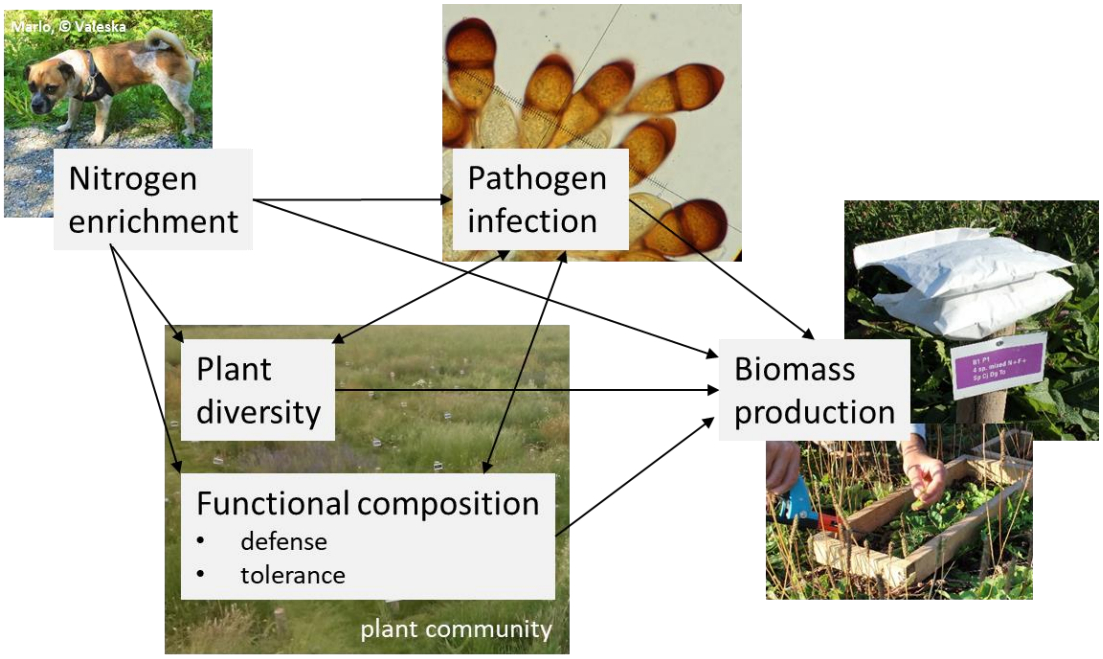


Figure 1 conceptual framework about how nitrogen directly and indirectly impacts pathogen infection and biomass production of diverse plant communities. Photos by Valeska, Beatrice and me.

This thesis

The aim of this thesis was to assess the causes and consequences of foliar fungal pathogens in an experimental grassland. In Chapter 2, the relative importance of mechanisms such as nitrogen disease, host concentration, and growth-defense trade-off in driving infection at the plant community level are presented. Further, we explore the circumstances under which fungal pathogens have negative consequences for biomass production. For a more detailed understanding of the drivers of infection in individual species, differences in population, species and community level mechanisms driving infection are investigated in Chapter 3. Further, in Chapter 4 I compare and relate diversity effects for pathogen infection to diversity effects for other ecosystem functions. Especially interesting in Chapter 4 are the comparisons between the diversity effects for fungal infection with the diversity effects for biomass production, as complementarity and selection effects are commonly calculated for biomass production and with the diversity effects for herbivory, because herbivores occupy the

same ecological niche as fungal pathogens (Raffa et al. 2019). There is evidence for all the introduced mechanisms by which nitrogen can directly or indirectly affect pathogen infection and its consequences. However, this is the first study to simultaneously test all of them to assess their relative importance by systematically manipulating nitrogen, the functional composition and diversity of the plant community, as well as pathogen access to plants. Manipulating these variables independently of each other enables us to mechanistically understand the role of fungal pathogens in natural ecosystems and the consequences of anthropogenic changes in the nitrogen cycle (Figure 1).

Study site

We have established a large field experiment factorially manipulating, species diversity, functional composition, nitrogen enrichment, and fungal pathogen exclusion, called PaNDiv (**Pa**thogens, **N**itrogen, **Div**ersity). The experiment was established on a nutrient rich, rather dry, species rich and extensively managed grassland (Delarze 2015) in the Swiss lowlands. The field has been extensively managed without fertilizer but occasional sheep grazing since 2001. This region of Switzerland has a mean annual temperature of 9.4 ± 0.1 °C and 1021.62 ± 31.89 mm of precipitation (MeteoSchweiz 2019). The soil is characterized as “0.7 to 1m deep brown soil” (Cambisol, soil map of the Canton of Bern Soil map of the Canton of Bern 1970-2005). The experiment is located at the edge of a former peatland. From 1777 on peat was dug to meet the high demand for fuel, since firewood was scarce. The level of the lakes and the groundwater was lowered three times between 1780 and 1920 to make the peat accessible and to gain land for agriculture. During the last melioration (1917-1920), the area where the PaNDiv experiment is located, was drained and the river crossing the parcel of land was put underground (Archivgruppe Moosseedorf 2012; Siegfried 1917-1930, Figure 2). In addition to the cement pipe that was used to channel the river (parallel to where the street runs today), ditches to drain the land were dug. One of them crosses the experimental field through Block 2 and 4 (Katasterplan, Figure 3a). Nowadays we see increased levels of carbon and nitrogen in this area (Figure 3c), which indicates, that they might have used different soil material to fill the ditches. In the late 60s and early 70s, when the land was prepared for building construction, the drainages were renewed and pipes for the wastewater were put under ground. At the same time, the land was levelled off (Lanz 9/4/2019). As filling material household garbage and construction rubble was used. This is still noticeable today, as we found

plastic chips, cement and metal pieces mainly in Block 1. After that, the land was mainly used as grassland, because the rocky underground did not allow soil cultivation.

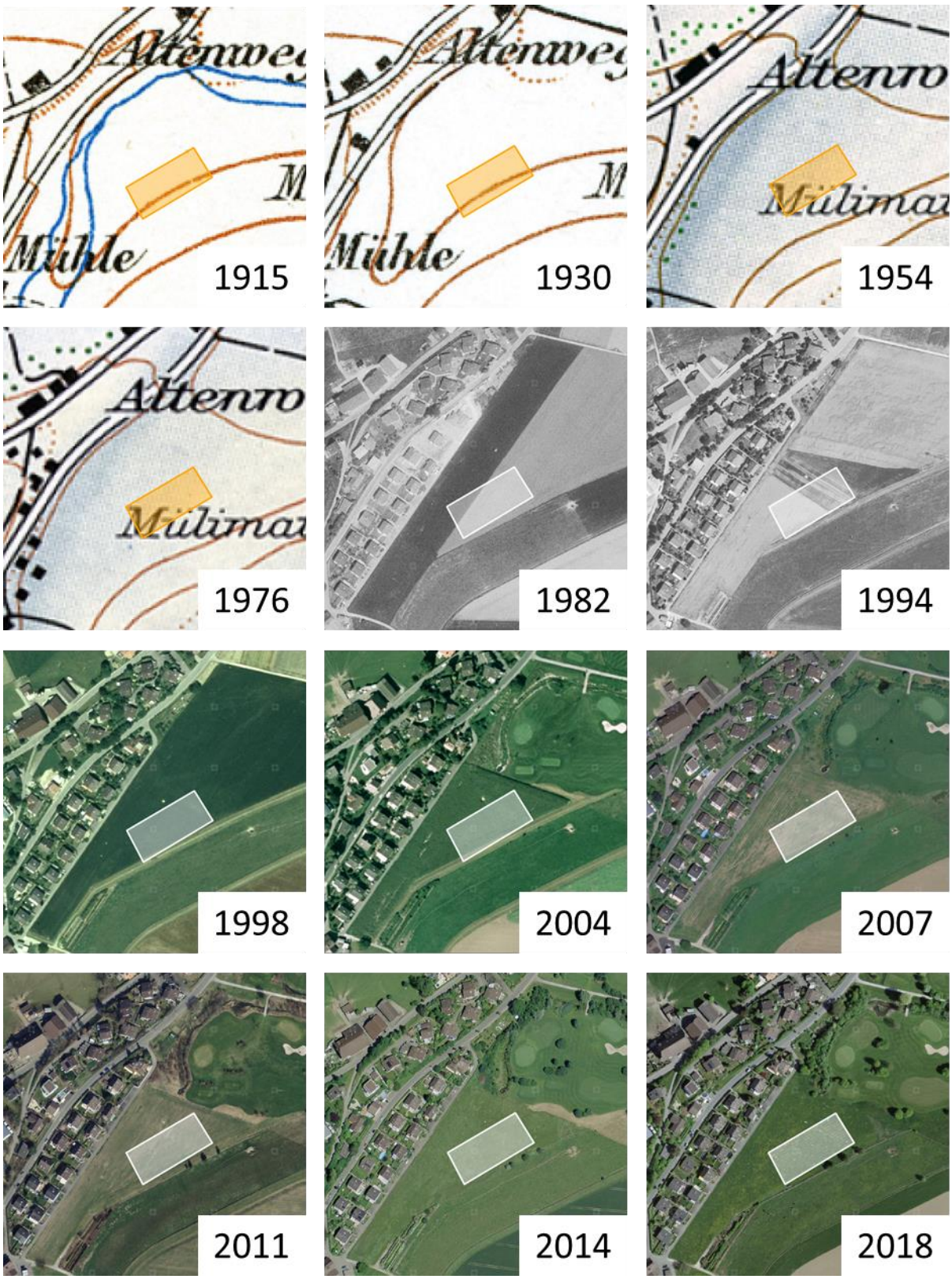


Figure 2 100 years before PaNDiv. The rectangle in the pictures marks the location of the experiment today.

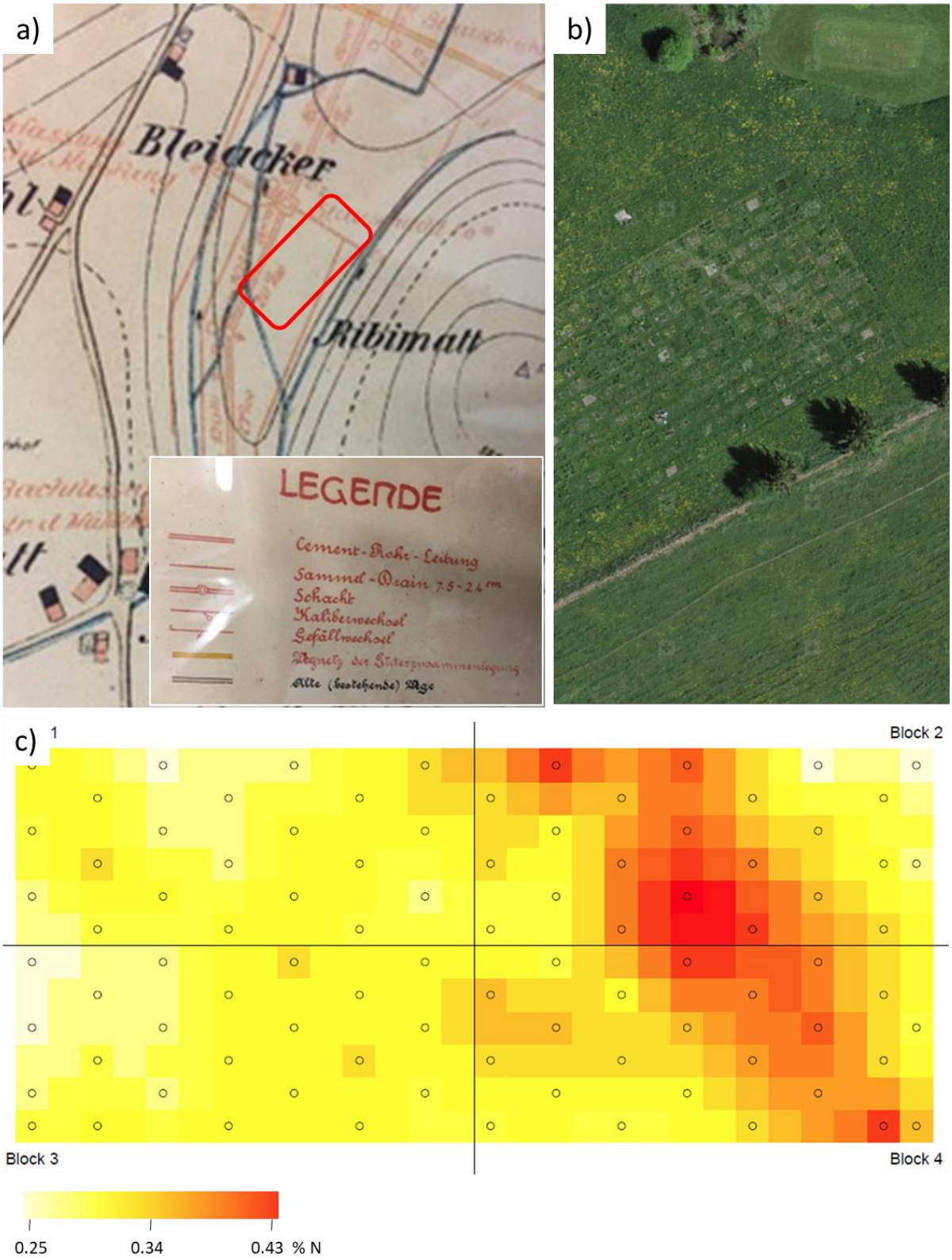


Figure 3 how the drain from the melioration still affects soil and vegetation 100 years later. a) Katasterplan (Katasterplan, estimated 1917-1920) showing where the drain was put (light red line inside of the red square which is roughly where the experiment sl located today, b) aerial view on PaNDiv from Swisstopo 2018, c) more % soil N in 2015 before the experiment was set up along the drain (% soil C looks similar and is also enhanced in this area, min: 2.30%, max 4.17%).

Study design

To set up the experiment, the vegetation was cleared, and the area ploughed prior to sowing the experimental plant communities in fall 2015. Some species were resown in spring 2016 due to poor establishment. The communities were assembled from a species pool of 20 common grassland species (Chapter 2, Table S2). Half of the species were classified as fast-growing, and half as slow-growing based on their specific leaf area and leaf nitrogen content, traits indicative of their growth strategy (Wright et al. 2004; Reich and Cornelissen 2014, Chapter 2, Figure S2). Legumes were not included in the species pool, as they are very functionally different from the other species due to their symbiosis with nitrogen fixing bacteria. Most legumes preferentially grow in nitrogen poor habitats. They could therefore have been only included in the slow-growing species pool, which would have led to large differences between the pools which are not due to the resource traits. We manipulated species diversity (1, 4, 8, and 20 species). Communities with four and eight species could have either only slow, only fast, or a mix of fast and slow species. The communities with four and eight species were chosen randomly from the respective pools, but they contained at least one grass and one herb species. There were 10 communities of each type (fast, slow, mixed, with four and eight species, Table 1). This created a large gradient of average growth strategy and community weighted mean traits. Monocultures of all species which were either fast or slow growing and 20 species communities (4 replicates) which necessarily contained both fast and slow species were planted as well (Table 1). In total, there were 84 unique species compositions (Table 1). The experiment was weeded three times per year to maintain species compositions. As common for extensively managed grasslands in Switzerland, the experiment was mown twice per year (June and August).

Table 1 Community compositions of all 84 communities. Each was grown 4 times with a full cross of nitrogen and fungicide treatment, resulting in total 336 plots.

Nr. of species / Growth strategy	1	4	8	20
Fast	10	10	10	-
Slow	10	10	10	4
Mixed	-	10	10	-

Each of the species compositions was grown with a control treatment (without nitrogen and fungicide), with a fungicide treatment, with a nitrogen treatment and with a joint fungicide and nitrogen treatment, resulting in 336 plots in total. The plots were arranged in four blocks. Each species composition occurred once per block, with one of the four

treatment combinations randomly assigned. Fungicide (Score Profi by Syngenta Agro AG, 24.8% difenoconazole) was sprayed four times during the growing season. Plots without fungicide were sprayed with water. In 2018, we added a second fungicide (Ortiva by Syngenta Agro GmbH, 22.8% azoxystrobin) to the treatment to increase efficacy. The fertilized plots received total 100 kg N $ha^{-1}y^{-1}$ which corresponds to intermediately intensive grassland management (Bluethgen et al. 2012). Fertilizer was applied in spring and after the first mowing in June.

Chapter overviews

In Chapter 2, the effects of community properties on community level infection and consequences of infection are studied. From all tested variables, community weighted mean specific leaf area was the best predictor of infection, indicating that communities dominated by fast growing species had the highest infection. This means that growth-defense trade-off was a major driver of infection at the community level. More diverse communities did not have lower infection, probably due to spillover of generalist pathogens or because susceptible species occurred at high abundances. However, pathogen infection had the strongest negative impact on community biomass at high diversity. This diversity effect was enhanced by fungicide treatment, probably because fungicide altered the composition of the fungal community. These results suggest that nitrogen affects community level infection mainly indirectly by favoring fast-growing species.

The results presented in Chapter 3 show that the strong effect of community weighted mean of specific leaf area at the community level was mainly due to species level growth-defense trade-offs, rather than due to spillover of some fast-growing species to slower-growing species. Within species, variation in leaf traits did not influence infection, indicating that within species trade-off was probably much weaker. However, infection of a species increased with the abundance of the host species and the abundance of closely related species in the surroundings. Diversity had no additional effect on infection apart from diluting the host abundance. Interestingly, fast-growing but heavily infected species were not more tolerant to infection. Tolerance to infection seemed to trade-off with resource use strategy. Nitrogen limitation in species with quick nitrogen acquisition reduced their tolerance to infection, independently of growth strategy. The results of Chapter 3 show that between species growth-defense trade-off is a main driver of infection, but that the consequences of infection are strongly influenced by tolerance and its own trade-offs.

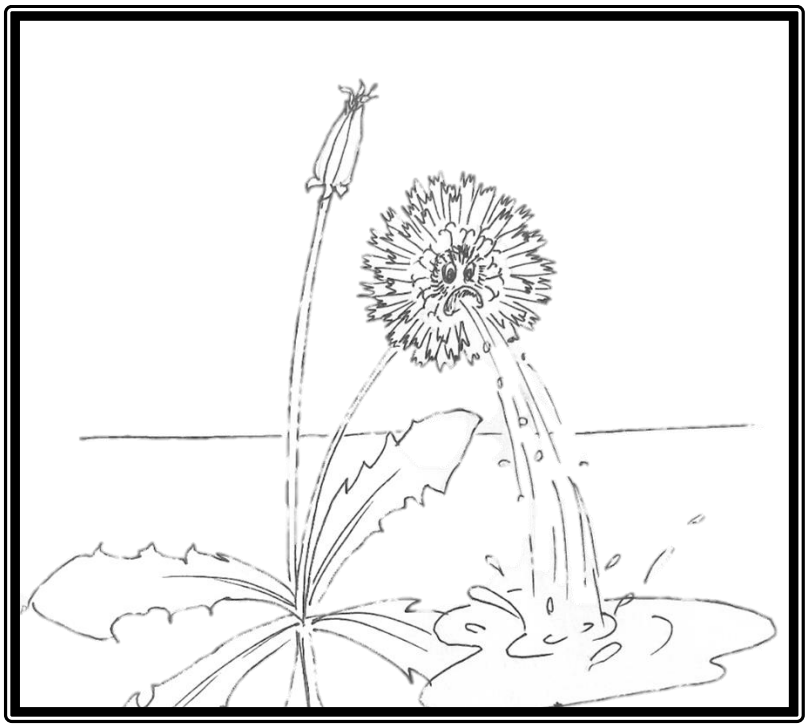
The absence of strong diversity effects in Chapter 2 and Chapter 3 could be explained by contrasting diversity effects, as shown in Chapter 4. Species with rather low infection in monoculture had increased infection when growing in mixtures, likely suffering from spillover, while species with high infection in monoculture had decreased infection, benefitting from host dilution. Even though net effects varied between the functions, diversity had comparable effects on all studied functions: species with low monoculture functioning increased functioning in mixtures, while species with high monoculture functioning decreased functioning in mixtures (negative intraspecific selection effects). On average across the community, most functions increased with diversity (neutral to positive complementarity effects). Diversity effects across functions were mostly not linked to each other and had different context dependencies. This means that different species were contributing most to different functions. The lack of correlations between the diversity effects of pathogen infection and herbivory is probably the most surprising. Pathogens and herbivores share a niche as primary consumers of plants and many theories about plant-pest interactions are applied to both (e.g. Raffa et al. 2019). The results of Chapter 4 hint that the underlying mechanisms driving diversity effects varied between the functions despite similar overall patterns. This might explain why high levels of multiple ecosystem functions can only be provided by diverse communities.

Finally, in Chapter 5, I summarize the most important findings and identify knowledge gaps, which should be addressed in future research.

Chapter 2

Sick plants in grassland communities: a growth-defense trade-off is the main driver of fungal pathogen abundance and impact.

Seraina L. Cappelli, Noémie A. Pichon, Anne Kempel, Eric Allan



ABSTRACT

Aboveground fungal pathogens can substantially reduce biomass production in grasslands. However, we lack a mechanistic understanding of the drivers of fungal infection and impact. Using a global change biodiversity experiment, we show that the trade-off between plant growth and defense is the main determinant of fungal infection in grasslands. Nitrogen addition only indirectly increased infection via shifting plant communities towards more fast-growing species. Plant diversity did not decrease infection, likely because the spillover of generalist pathogens or dominance of susceptible species counteracted dilution effects. There was also evidence that fungal pathogens reduced biomass more strongly in diverse communities. Further, fungicide altered plant-pathogen interactions beyond just removing pathogens, probably by removing certain fungi more efficiently than others. Our results show that fungal pathogens have large effects on plant functional composition and biomass production and highlight the importance of considering changes in pathogen community composition to understand their effects.

INTRODUCTION

Pathogenic fungi are omnipresent in the environment and have large impacts on their hosts (Fisher et al. 2012). Many studies have looked at species-specific (fungus-plant) interactions (e.g. Thrall and Burdon 2003, Roscher et al. 2007a), however, only a few experiments have investigated fungal pathogens in whole plant communities, by manipulating pathogen access to their hosts (Peters and Shaw 1996; Mitchell 2003; Allan et al. 2010; Borer et al. 2015; Heckman et al. 2017). These studies show fungal pathogens can have large top-down effects, even reducing grassland biomass production as much as insect herbivores (Allan et al. 2010; Seabloom et al. 2017). However, effects can be context dependent and factors such as plant species composition (Mitchell et al. 2002; Rottstock et al. 2014) or environmental factors (Mitchell et al. 2003) can determine infection rates and pathogen impact. Increasing our knowledge about causes and consequences of fungal pathogens is important to predict effects of global change, e.g. nitrogen enrichment. Nitrogen input can alter pathogen infection (Burdon et al. 2006) but the mechanisms by which it does so and the consequences for pathogen abundance and impact are poorly understood.

Key determinants of infection success and consequences of infection are related to pathogen transmission, host resistance and host tolerance to infection (as discussed in detail by Keesing et al. (2006)). Transmission and resistance should directly influence the observed levels of infection, while tolerance should alter the negative consequences of infection for fitness or biomass production and, if a tolerant species is a good reservoir host, the infection levels in other species (spillover, Power and Mitchell 2004). All of these factors can be influenced by environmental variables and might trade-off with each other.

Pathogen resistance and tolerance are linked to plant growth strategy. Plants face a trade-off between growth and enemy defense (***growth-defense trade-off***). Plant species adapted to resource-rich environments grow fast but are often less defended against enemies, including herbivores (Endara and Coley 2011; Lind et al. 2013) and fungal pathogens (Blumenthal et al. 2009; Liu et al. 2017). Fast-growing species are likely to better tolerate enemies, as the loss of plant tissue can easily be replaced (Gianoli and Salgado-Luarte 2017). Hence, plant communities dominated by fast-growing species should display higher pathogen infection but lose less biomass to pathogens than communities dominated by slow-growing plants from resource-poor

environments. The leaf economics spectrum distinguishes these strategies and is indicated by several functional traits. Slow-growing species with long-lived, structurally expensive leaves, with low nutrient contents occur at one end of the spectrum and fast-growing species with a high turnover of short-lived, nutrient-rich leaves at the other end (Wright et al. 2004). Some of these traits are also directly related to resistance to natural enemies, e.g. leaf nutrient concentrations (Robinson and Hodges 1981). Although the growth-defense trade-off hypothesis is well supported for individual plant-pathogen interactions, we know little about how it scales up to whole plant communities and its importance relative to other drivers of infection.

Another possible driver of infection is the nutrient supply in plant communities. High nitrogen supply can lead to decreased infection resistance with fungal pathogens (***nitrogen-disease hypothesis***, Dordas 2008). Nitrogen disease effects are mainly known from agriculture, while studies in natural ecosystems show more variable results (Mitchell et al. 2003; Veresoglou et al. 2013). This variation may be partly because nitrogen enrichment can also have complex indirect effects on fungal infection. Nitrogen enrichment often reduces plant species richness and changes plant functional composition by promoting fast-growing over slow-growing species (Bobbink et al. 2010; De Schrijver et al. 2011; Isbell et al. 2013) both of which could indirectly alter fungal infection and its consequences. However, nitrogen could also directly lead to healthier and more tolerant plants. To mechanistically understand nitrogen effects on fungal infection, studies therefore need to assess the direct and indirect effects independently.

Plant diversity can also be a key driver of pathogen infection, through different mechanisms. Pathogen infection has been shown to decrease with greater host diversity in grasslands through changes in plant abundances (e.g. Mitchell et al. 2002; Liu et al. 2016; Rottstock et al. 2014; but see Halliday et al. 2017), which reduces host-pathogen transmission (***host dilution hypothesis,*** Civitello et al. 2015). However, other studies showed that diverse communities are more infected, potentially due to the spillover of generalist pathogens between plant species or due to an increase of host-density independent pathogens, such as vector-transmitted ones (Power and Mitchell 2004; Halliday et al. 2017). The impact of plant diversity on pathogen infection may therefore depend on the relative abundance of specialist and generalist pathogens and on their transmission mode. Plant diversity might also change pathogen community composition by selecting for more generalist species (Thrall et al. 2007)

and this could potentially alter the impact of pathogens if specialists and generalists differ in their virulence (Leggett et al. 2013). However, relatively little is known about the impact of pathogens in low and high diversity plant communities (but see Seabloom et al. 2017; Halliday et al. (2017)).

Changes in plant functional composition, diversity, and nutrients could all affect pathogen communities by changing plant biomass and thereby altering microclimatic conditions. Pathogens generally grow better in warmer and humid conditions, but this varies between pathogen groups (Barrett et al. 2009). The availability of free water if often an important driver of infection (Bregaglio *et al.* 2013; Chen *et al.* 2014; Sun *et al.* 2017; Bradley *et al.* 2003), suggesting that the microclimatic humidity is important. Further, increased temperature may promote overall pathogen infection (Liu et al. 2016), but again, different groups of fungal pathogens may react differently (Helfer 2014). We therefore lack a good understanding of how temperature and humidity influences different pathogen groups and how these effects relate to other drivers of infection.

The impact of pathogen infection on plant communities mainly depends on the resistance and the tolerance of plants. Impact can be assessed in two ways: comparing plots with and without fungicide, and, assessing the amount of fungal infection in a plant community and relating it to the biomass produced. The first approach would be ideal if fungicide reduced infection to zero. However, most fungicides do not completely wipe out all infection, and might be selective for certain fungal groups (Paul et al. 1989; Parker et al. 2015; Karlsson et al. 2014), changing fungal community composition. For example, if a fungicide is selective against the rather specialized rusts, then the fungicide might cause a shift from specialized to more generalist fungal communities. The second approach, relating infection and biomass, allows for more quantitative comparisons. However, here the direction of causality is hard to establish, as higher plant biomass might also lead to higher fungal infection, obscuring the relationship. It is therefore advantageous to use both methods; however, no previous studies have done so.

Here we tested the relative importance of nitrogen, plant diversity and functional composition as drivers of fungal pathogen abundance, in an experiment that manipulated these variables factorially (Figure S1, Table S1). Specifically, we tested the growth-defense trade-off hypothesis, the nitrogen-disease hypothesis, and the

dilution-effect hypothesis (Figure 1). Further, we assessed the fungal impact on plant biomass by comparing biomass from plots with and without fungicide, and by relating plant biomass to infection intensity in the same plots. In addition, we tested if our experimental treatments altered pathogen abundance and impact through changes in microclimatic conditions.

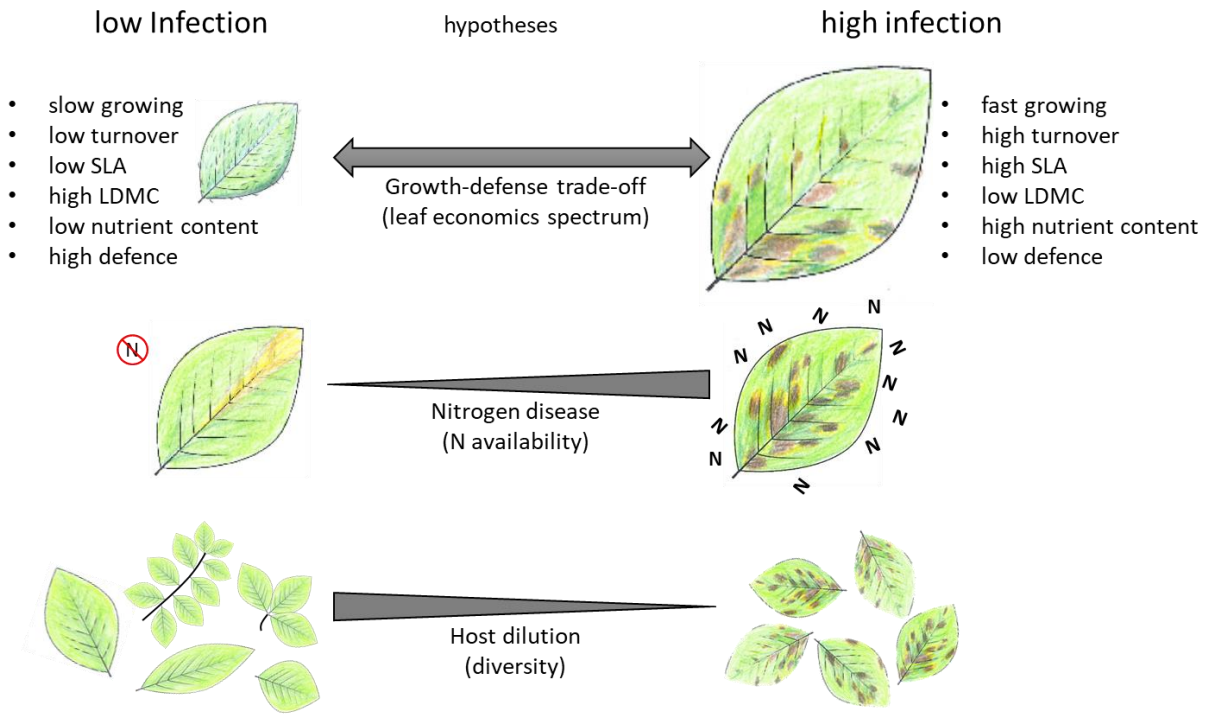


Figure 1 Overview over the main hypotheses, which we tested. Growth-defense trade-off hypothesis: Plant species adapted to resource-rich environments and able to compete well under nutrient rich conditions are often less defended against natural enemies (Blumenthal et al. 2009; Liu et al. 2017). The growth strategy is defined by the leaf economics spectrum (Wright et al. 2004), which has been linked to certain disease resistance mechanisms (Cronin et al. 2014; Cronin et al. 2010; Huot et al. 2014). Nitrogen disease hypothesis: Higher nutrient content of the plant material following nitrogen fertilization should promote disease. This is known for agricultural systems (Dordas 2008), but results from natural ecosystems vary (Mitchell et al. 2003; Veresoglou et al. 2013). Host dilution hypothesis: Many pathogens are dependent on the availability and density of host plants. At high plant diversity the abundance of each host plant is in average lower than in species poor communities (Civitello et al. 2015), which is suggested to be the underlying mechanism of observed negative diversity-disease relationships (Lau et al. 2008; Knops et al. 1999; Mitchell 2003; Mitchell et al. 2003).

MATERIALS AND METHODS

Experiment

We set up a large field experiment (PaNDiv Experiment) in the Swiss lowlands (mean annual temperature and precipitation 9.4±0.1°C, respectively 1021.62±31.89mm, MeteoSchweiz 2019) on a formerly extensively managed grassland in autumn 2015. The experiment consisted of 336 2m x 2m plots. We factorially manipulated plant species richness, plant functional composition (gradient of specific leaf area as a measure of growth strategy), nitrogen addition, and foliar fungal pathogen exclusion.

We used a set of 20 common grassland species spanning a large gradient of specific leaf area (SLA) to establish the experimental plant communities and divided them into fast (high SLA) and slow (Low SLA) growing (Table S2). The experimental communities contained either 1, 4, 8 or 20 species. Plots with four or eight species could contain only slow, only fast or a mix of species, creating a large gradient in community mean SLA values. Monocultures spanned the full range in SLA values while plots with 20 species inevitably had an intermediate mean SLA. The communities had fully developed by late summer 2016. To maintain species compositions, the plots were weeded three times a year. Plots were mown once in the middle of June and once in August (for more details see Supplementary methods and Pichon et al. (2019)).

Each specific community composition received crossed nitrogen and fungicide treatments. Nitrogen (N) enrichment plots received 100 $kgNha^{-1}y^{-1}$, added once in April and once after the first mowing, in the form of urea. This is typical of fertilization experiments (Hautier et al. 2014) and medium - intensive farming (Bluethgen et al. 2012). Foliar fungal pathogen exclusion was done with fungicide (Score Profi by Syngenta Agro AG, 24.8% difenoconazole and Ortiva by Syngenta Agro GmbH, 22.8% azoxystrobin) applied four times during the growing season (0.2ml of Score Profi and 0.4ml of Ortiva mixed with 0.062l of water per treated plot each time). Plots without fungicide were sprayed with water. Difenoconazole interrupts the synthesis of ergosterol (IUPAC 2016), a fungal cell membrane component. If applied on top of the vegetation, it has no effect on soil (Dahmen, Staub 1992). Azoxystrobin blocks the cell respiration by inhibiting the proenzyme coenzyme Q, which prevents the production of ATP. Studies have shown no phytotoxic effects of azoxystrobin (Sundravadana et al. 2007; Khalko et al. 2009) or difenoconazole (Nithyameenakshi et al. 2006). To account for potential soil heterogeneity across the study site, plots were arranged in four blocks. Each community composition was grown once per block and the nitrogen and fungicide treatments were assigned randomly to the communities in the blocks.

Measurements

We measured plant aboveground **biomass** by harvesting two subplots of $0.1m^2$, 5cm above ground level, in mid-June and at the beginning of August 2018. Biomass was dried and weighed. Percentage cover of all sown plant species, plus weeds and bare ground, was visually estimated in the central square meter of the plots shortly before the biomass harvest (June and August). The sum of all estimates per plot could exceed 100% but here we analyze proportional abundances of each species. **Total plant**

cover was calculated as 1- the proportion of bare ground. To describe the functional composition of the communities, we measured SLA (Garnier et al. 2001) on one leaf each from five plants, growing in the central square meter of all the monoculture plots (if possible, otherwise elsewhere in the plot) in June and in August, at the same time as we measured percentage cover. We then calculated several measures of plant functional composition. We calculated **the realized SLA**, i.e. the community weighted mean SLA per plot, using the percentage cover measurements and the mean SLA per monoculture as the baseline SLA for each species under a given treatment (the four combinations of nitrogen x fungicide). Because plant community composition can shift in abundance in response to the nitrogen and fungicide treatments, we also calculated the **shift in SLA** of the whole plant community relative to the **sown SLA** (mean SLA of all species sown in a community), by subtracting the sown SLA from the realized SLA (see also Supplementary methods).

Overall **fungal infection,** and infection with rusts, smuts, powdery mildews, downy mildews and leaf spots (see Rottstock et al. (2014), was assessed for each plant species in each plot, in July and in early October 2018. Ten randomly chosen individuals per plant species, growing in the central square meter of the plot (if possible, otherwise elsewhere in the plot), were screened for signs of infection and the percentage of infected individuals was recorded (see also Supplementary methods). If there were less than 10 individuals in total, the percentage of infected individuals was calculated based on the observed number of individuals. Based on the species level infection, and the percentage cover of each plant species, we calculated an abundance weighted mean fungal infection per plot and season for total infection and infection by separate fungal groups (rusts, powdery and downy mildews and leaf spots). The smut fungi were excluded, because they were very rare (observed only eight times).

Further, we measured the **microclimate** (temperature and relative humidity logger iButton DS1923-F5, Maxim Integrated, USA) in each plot, in the center of one of the biomass subplots for a period of 2-3 days, with hourly measurements between 16.07.2018 and 13.08.2018. Due to a lack of data loggers, we could only measure 28 plots at the same time (Table S3). Therefore, to account for differences in daily temperatures we subtracted the temperature and humidity measured in the plots from temperature and humidity measured at the same time in a nearby meteorological station in Zollikhofen (3.81 km away, MeteoSchweiz 2019).

Analysis

Biomass, infection, and trait data correlated well between the two time points when they were measured. For this reason, we used the total biomass (sum of the two harvests) and mean values of community shift SLA and fungal infection between the two time points.

We conducted two analyses to test for the causes and consequences of pathogen infection. We first analyzed the overall effects of fungicide on fungal infection and biomass production at the plot level, using linear mixed effect models, with fungicide as the independent variable and nitrogen addition, sown species diversity and realized SLA and all possible interactions as covariates. Block and species combination (84 levels) were included as random effects. We stepwise excluded non-significant terms from the model based on likelihood-ratio tests (Zuur 2009) . We also ran separate models for each fungal group.

Secondly, we tested drivers and effects of quantitative levels of fungal pathogen infection in structural equation models (SEM, Figure S1, Table S1). As fungicide did not completely remove infection we fitted a multi-group SEM to test the drivers and effects of pathogen infection on control and fungicide plots separately (Grace 2006). All other treatment variables were also included in the SEM with direct effects on both fungal infection and biomass production. In the SEM we were therefore able to test for the effect of quantitative levels of pathogen infection and whether it varied with fungicide application. We included the deviation between plot and air humidity and temperature and plant cover, to account for indirect effects of the treatment variables through changes in microclimate. This considers potential impacts of the plant community on fungal infection, which elsewise would have likely influenced the path between fungal infection and biomass. We also incorporated an interaction between diversity and pathogen infection, which could affect plot biomass production, by constructing a dummy variable by multiplying the standardized values of fungal infection and species diversity (path 14 in Figure S1, Table S1).

We fitted a multi-group SEM, with the groups being the two levels of fungicide treatment, which allowed fungicide to interact with all the paths of the models. We checked whether each path and intercept differed significantly with fungicide, by comparing the AIC values of a fully unconstrained model, where all paths and intercepts were allowed to differ, with a model where a particular path was constrained

to be equal between fungicide treatments. All paths that did not differ significantly were kept constrained (Table S2). We used the same SEM to analyze the separate fungal groups (rusts, powdery mildews, downy mildews, and leaf spots).

To test for diversity effects through host dilution, we calculated host concentration effects for each plant species, as the relationship between host cover and infection. We fitted separate linear mixed effect models per plant species, nitrogen, and fungicide treatment with block as a random effect. The slopes of these models were analyzed using another mixed effect model with nitrogen and fungicide as explanatory variables and species as a random effect. This allowed us to test whether nitrogen enrichment and fungicide alter any host dilution effects. All analyses were conducted in R (R Core Team 2018), using the package lme4 for linear mixed effects models (Bates et al. 2015) and lavaan for SEMs (Rosseel 2012).

RESULTS

Effects of fungicide application on fungal infection and plant biomass

Fungicide reduced fungal infection by 25.33% on average (Figure 2a). The fungicide was most effective in high SLA communities, especially at high species diversity and in the absence of nitrogen fertilization (Figure S4). Fungicide also increased plant biomass but only in plots with high SLA (Figure 2b). This agrees with the idea that fungicide was most effective in fast growing communities. Comparing the intercepts in the SEM between fungicide and non-fungicide plots showed similar results (Figure 4h).

Drivers of infection

We then used SEM to look in more detail at the drivers of pathogen infection and its impacts on biomass (SEM: Figure 3; selected partial plots: Figure 4; path coefficients, significances, etc.: Table S8). The most important driver was functional composition, i.e. whether plant communities contained slow or fast growing plants. Both the sown SLA (Figure 4a) and the shift in SLA (Figure 4b) increased fungal infection. Communities with low sown SLA and a highly negative SLA shift had lower infection than high SLA communities. Leaf spots, rusts and to some degree powdery mildews increased with increasing SLA, whereas downy mildews were unaffected (Figure S6).

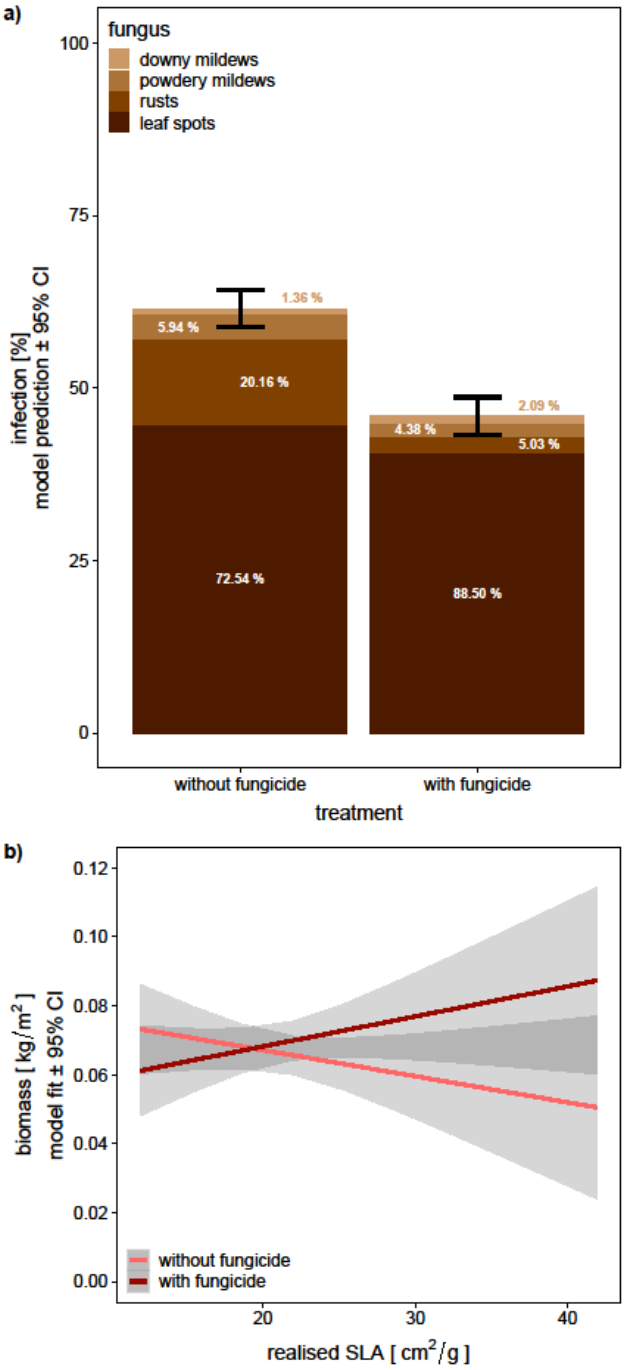


Figure 2 Selected results from the linear mixed effects models: model predictions and 95% confidence interval of a) impact of fungicide treatment on fungal infection and the contribution of single fungal groups to overall infection The numbers in the bars indicate the percentage contribution of each fungal group to the total infection. Main fungicide effects of the linear mixed effects models per fungal group: Fungicide reduced total infection from 61.50 ± 1.38 % to 45.92 ± 1.39 % ($p < 0.001$), leaf spots from 59.1 2 ± 1.58 % to 46.90 ± 1.57 % ($p < 0.001$), rusts from 16.43 ± 0.81 to 2.67 ± 0.81 % ($p < 0.001$) and powdery mildews from 4.85 ± 0.53 % to 2.32 ± 0.83 % ($p < 0.001$), while downy mildews were unaffected by fungicide ($p = 0.623$) and were generally very low (1.11 ± 0.39 %). b) Interactive effect of realized SLA and fungicide on biomass production. Plots dominated by fast-growing species produced less biomass than plots dominated by slow-growing species. Fungicide increased biomass production, but only in plots dominated by fast-growing species. Under fungicide treatment there was even an increase of biomass with increasing realized SLA. Estimates and CI were derived from the effects package (Fox 2003). The whole model results can be found in Table S4 and Table S5.

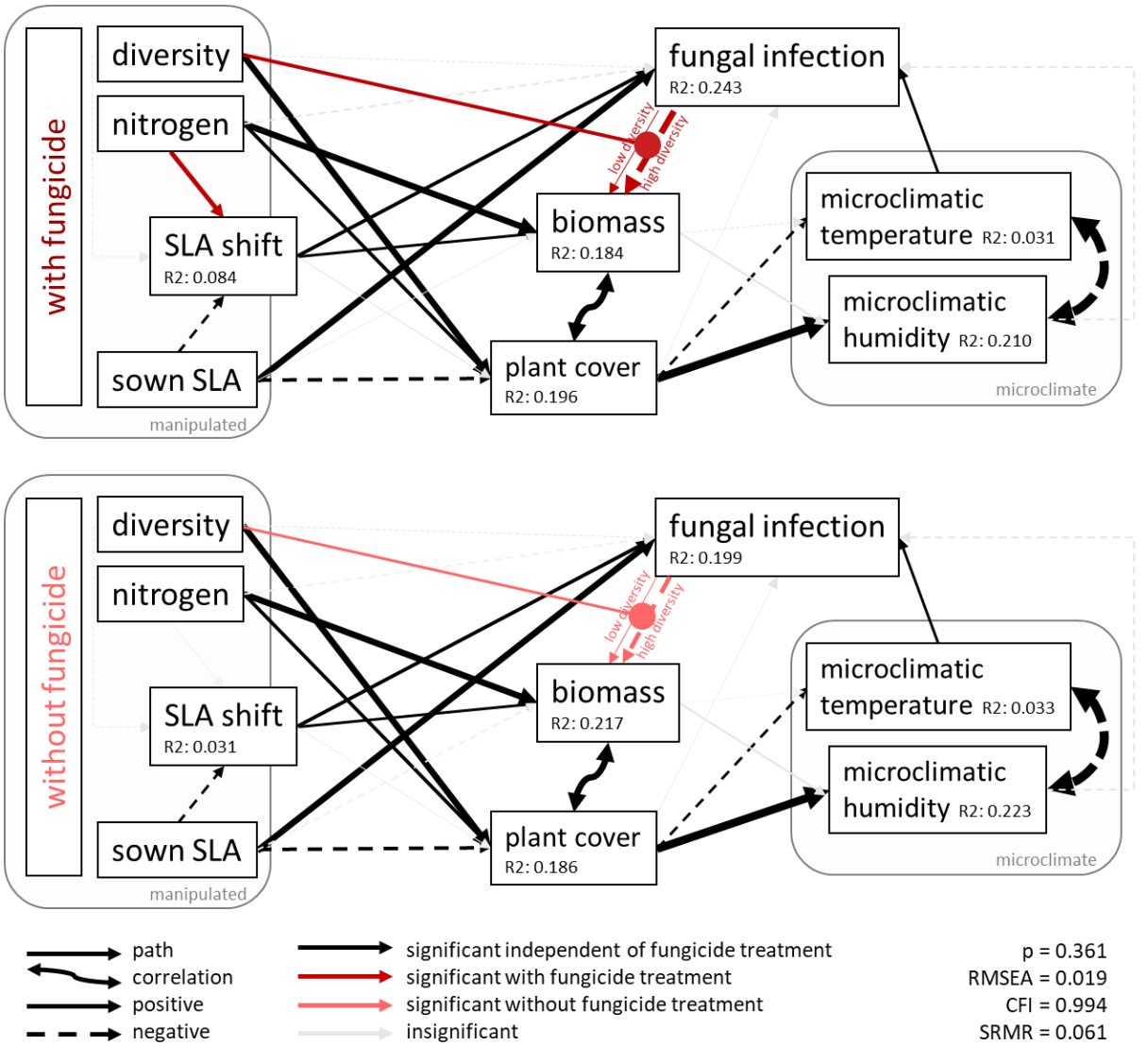


Figure 3 SEM: drivers and consequences of fungal infection. Dashed lines: negative effects. Solid lines: positive effects. Double headed arrows: correlations. Single headed arrows: paths. Black: significant constrained paths, red: significant unconstrained paths between fungicide (dark red) and no fungicide (light red). Light grey: not significant paths. Thickness: strength of the path/correlation.

Microclimate was also important and an increase in temperature increased fungal infection (Figure 4f). Humidity had no significant effect on fungal infection (Figure 4e). However, humidity and temperature were negatively correlated (Figure 3), which makes it hard to fully separate their effects. The impact of microclimate varied between fungal groups: rusts and leaf spots, the most abundant groups, increased with increasing temperature, while powdery and downy mildews were unaffected (Figure S6). Nitrogen and plant species diversity did not affect fungal infection directly (Figure 4c-d).

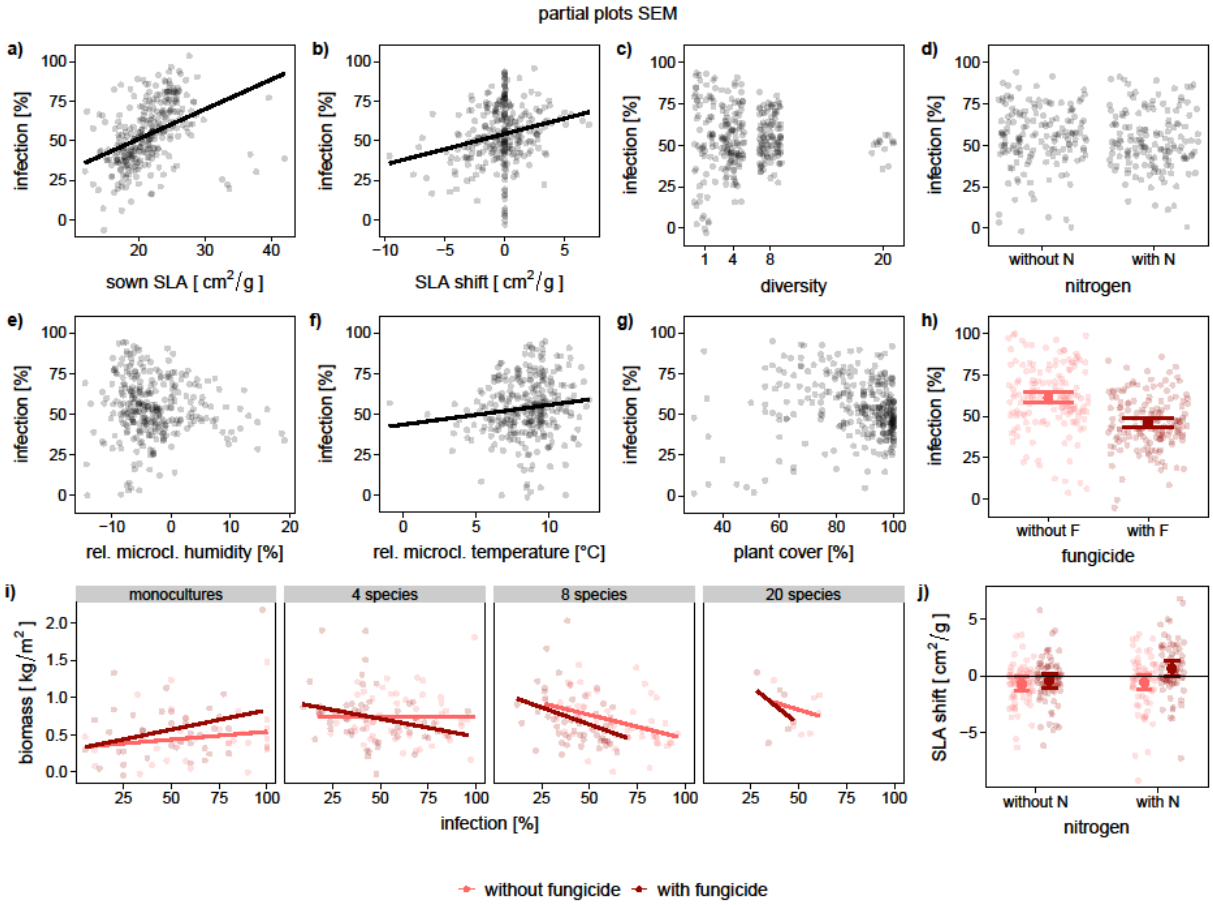


Figure 4 Partial plots of the SEM: impact of selected variables on fungal infection (a-h), biomass production (i) and SLA shift (j) after removing all effects of all the other variables which are not plotted. Effects on fungal infection of a) sown SLA (0.331, p<0.001), b) SLA shift (0.186, p<0.001), c) diversity (-0.030, p=0.551), d) nitrogen (-0.079, p=0.056), e) microclimatic humidity (-0.082, p=0.125), f) microclimatic temperature (0.121, p=0.012), g) plant cover (-0.061, p=0.227) and h) fungicide ±95% CI (-0.689, p<0.001) Interactive effects on biomass of i) fungicide, fungal infection and diversity and j) Interactive effects on SLA shift of nitrogen and fungicide estimate±95 & CI

Several factors indirectly affected infection via changing microclimate. Temperature varied by 13.6°C between plots and was reduced by plant cover, but not by biomass. Plant cover was increased by plant diversity and nitrogen but reduced by sown SLA. Therefore, in addition to its positive direct effect, sown SLA also had a positive indirect effect on fungal infection, but this indirect path was non-significant overall. Species diversity and nitrogen enrichment indirectly decreased infection by increasing plant cover and reducing temperature, but again the indirect effects were not significant overall.

The absence of a direct diversity effect on fungal infection cannot be explained by an absence of host concentration effects, as on average plant species cover was positively related to species-specific infection, suggesting additional mechanisms such as spillover or additionally the presence of density independent pathogens. The

application of fungicide removed host concentration effects (Figure S8).

Impact of fungal infection

In the SEM (SEM: Figure 3; selected partial plots: Figure 4; path coefficients, significances, etc.: Table S8), fungal infection also affected plant biomass production, however this depended on plant diversity (Figure 4i): in species rich communities fungal infection was negatively related to plant biomass, indicating that fungi had strong impacts on biomass, whereas in monocultures, fungal infection was even weakly positively related to biomass (Figure 4i). Adding fungicide increased the effect of diversity on the disease-productivity relationship, which means a stronger negative correlation at high diversity and a stronger positive correlation between infection and biomass in monocultures (Figure 4i). The SEMs per fungal group revealed that the leaf spots and to some degree the rusts drove the negative relationship between infection and biomass (Figure S6). Powdery mildew had no impact on biomass production, while the downy mildews even increased biomass. The downy mildew and rust models did not fit well (both $p<0.001$) but the fit was good for the leaf spots ($p = 0.268$) and adequate for the powdery mildews ($p = 0.088$).

Biomass was also affected by several other factors. Nitrogen enrichment increased biomass production independently of the fungicide treatment, a shift in SLA towards faster growing species increased biomass production, while the effects of sown SLA on biomass depended on the fungicide treatment.

Fungicide also altered the SLA shift in the experimental plant communities to favor faster growing species (Figure 4j), but there was a lot of unexplained variation in SLA shift (R^2=0.084 under fungicide and R^2=0.031 under no fungicide treatment). The effect of fungicide on the SLA shift was amplified by nitrogen enrichment so that plots with nitrogen added and pathogens reduced shifted towards dominance by faster growing species (Figure 4j).

DISCUSSION

Growth-defense trade-off

We found strong support for the growth-defense trade-off hypothesis as the key driver of pathogen abundance and impact. Plant communities dominated by fast growing species had increased infection, and fungicide was most effective at reducing fungal infection in high SLA communities. There is an inherent trade-off between plant growth and the production of certain defense compounds (Huot et al. 2014), and species which are at the fast end of the leaf economics spectrum have been shown to have lower

structural and chemical defenses (Mason et al. 2016; Coley 1988) and higher tissue nutrient levels (Wright et al. 2004). Both could explain the increased pathogen attack on fast growing species, however, the absence of support for the nitrogen disease hypothesis, see below, may indicate that changes in defenses are more important. Our results show that growth-defense trade-offs are not only a major predictor of herbivory (e.g. Lind et al.) and pathogen attack on individual plant species but also scale up to be the key driver of community level pathogen infection.

Fungal pathogen impact was also mostly determined by growth-defense trade-offs. Fungicide allowed fast growing species to increase in abundance, especially under nitrogen. This is in line with findings that plants originating from nutrient rich habitats benefitted most from enemy release (Blumenthal et al. 2009, but see e.g. Heckman et al. 2017). Fast growing species are expected to be good competitors in nutrient rich environments (Wright et al. 2004; Poorter et al. 2009), but our results suggest that pathogens reduce their competitive advantage. Pathogens may therefore equalize competitive abilities and promote diversity in nitrogen rich conditions. In nutrient poor habitats, slow growing plants are expected to be more competitive and in such an environment, pathogens might reduce diversity by excluding faster growing species. Previous studies have shown pathogens can alter the outcome of plant competition (Paul 1989; Ridenour and Callaway 2003) and change plant community composition (Allan et al. 2010). Our results suggest that the growth strategy of plants is the key predictor of plant community responses to pathogens, and that pathogens promote slow growing species. Over time, this would be expected to reduce pathogen abundance and therefore impact. Such feedbacks could cause temporal dynamics between plant community composition and fungal infection, which could only be tested with long-term data on fungal infection and plant functional composition.

Nitrogen disease

We did not find support for the nitrogen disease hypothesis. Nitrogen can increase disease in crops but findings from grasslands are contradictory, with some studies finding support (Mitchell et al. 2003), but others not (Lau et al. 2008). Compared to agricultural systems, grassland plants could evolve increased disease resistance with nitrogen fertilization (Snaydon and Davies 1972), which might offset any benefits the pathogens would derive from higher plant nutrient contents. In addition, plant community composition changes with nitrogen enrichment. Mitchell et al. (2003) did not control for changes in composition but showed that the "disease proneness" of the

plants was an important driver of infection. Liu et al. (2018b) showed that nitrogen addition favors disease prone species (but see Welsh et al. 2016). However, these studies did not explain what drives disease resistance and could not separate compositional change effects from direct effects of nitrogen. Our results indicate that trade-offs linked to the leaf-economics spectrum are likely the underlying mechanism and that an increase in fast growing species is responsible for an increase in infection with nitrogen. Further, nitrogen enrichment can increase humidity and decrease temperature through increased shading in denser vegetation. In the dry summer of 2018, N fertilization may have decreased water and temperature stress and made the plants more resistant to fungal infection, which would explain why we found a negative indirect effect of nitrogen enrichment on infection. This all suggests that the direct effect of nitrogen on community infection in grasslands is weak to non-existent. Nitrogen enrichment rather drives infection through indirect effects of community shift and changes in microclimatic conditions.

Impact of plant diversity

Plant diversity did not affect fungal infection in our study, apart from a small indirect effect through microclimate. This is contrary to most other studies, which found that an increase in diversity leads to a decrease in infection (e.g. Mitchell et al. 2002; Liu et al. 2016; Rottstock et al. 2014; but see Halliday et al. 2017). We expected that host abundance would be diluted at high plant diversity and that this would reduce infection. However, while infection on individual plant species was lower when the plants were rarer (at least when pathogens were not suppressed by fungicide), this did not lead to a negative diversity-infection relationship for the community. Other diversity related mechanisms may have counteracted this relationship. Several other studies reported unexplained effects of diversity on fungal infection, in addition to host dilution, and different plant species and diseases varied in their response to diversity (Rottstock et al. 2014; Mitchell et al. 2002; Knops et al. 1999). One mechanism by which diversity can counteract dilution effects is increased spillover of generalist pathogens at high diversity or an increase of density independent pathogens such as vector-transmitted ones (Power and Mitchell 2004; Halliday et al. 2017). Another possibility is that diverse communities become dominated by susceptible species, limiting host dilution effects. Both mechanisms might explain why plant diversity did not affect fungal infection in our study.

Interestingly, our results suggest that the impact of fungal pathogens on biomass

production was higher in species rich plant communities. Even though plant diversity did not alter overall pathogen infection, it could still have altered fungal community composition or diversity and might have led to more aggressive fungi at high plant diversity or reduced pathogen tolerance of the plants. However, we did not find that diversity altered the abundance of our four fungal guilds. It is therefore also possible that the ability of the plants to deal with infection varies with diversity. A higher pathogen pressure in species poor communities might select for better-defended plant genotypes, leading over time to reduced pathogen impact in monocultures. Results from the Jena Experiment support this idea and show that plants in monocultures have evolved to be more resistant against belowground pathogens (Zuppinger-Dingley et al.) and aboveground fungi (Hahl et al. 2017). To better predict variation in pathogen impact in plant communities we may need to consider pathogen community composition and host genetics.

Climatic stress

Temperature also affected fungal infection - leaf spots and rusts both benefitted from an increase in temperature in the vegetation. Other studies also indicate that higher temperatures increase pathogen infection (Liu et al. 2016) and that different fungal groups vary in their responses. Powdery mildews can increase with temperature, while rusts show more variables responses (Gullino et al. 2018; Helfer 2014). Longer periods of 100% humidity lead to water condensation, which has been shown to increase infection (Burdon 1991; Sun et al. 2017). However, we found no effect of humidity on infection, after correcting for temperature. The summer 2018 was extraordinarily hot and dry, with mean July temperatures 1.6°C above the average of the last 30 years and precipitation 18.81% lower (MeteoSchweiz 2019), which likely resulted in intensive drought and heat stress for the plants. Drought stress can increase fungal diseases in trees (Desprez-Loustau et al. 2006) and increase the negative effects of pathogens on competitive ability (Paul and Ayres 1987). The microclimate itself was driven by plant cover, which was determined by plant diversity, nitrogen, and functional composition. These variables indirectly (but weakly) influenced fungal infection through a change in the microclimate. Changes in vegetation microclimate may therefore play an important role in affecting plant community resistance to disease under extreme weather conditions.

Impact of fungicide and infection intensity on plant biomass

In our study, we used two approaches to assess the impact of fungal pathogens:

exclusion with fungicide and SEMs testing the effect of infection intensity on plant biomass production. Fungicide application increased plant biomass but only in plots dominated by fast growing plants, which suggests that fast growing plants are not entirely tolerant. The magnitude of biomass reduction was lower than in other studies (Allan et al. 2010; Seabloom et al. 2017), perhaps because, unlike in the other studies, we mowed the field regularly, preventing the build-up of large pathogen populations over the season. Our analysis relating infection and biomass suggested that the negative impact of fungal infection on biomass in high diversity plots was amplified by fungicide, even though fungicide generally decreased infection. Fungicide shifted the functional composition of the fungal community by mainly removing the rather specialized rusts and powdery mildews (Klenke 2015) and it removed host concentration effects, which also suggests a shift from specialists towards more generalist pathogens (Bever et al. 2015). Fungicide may therefore have selected for more aggressive, generalist pathogens, which would also explain its small overall effect on biomass production. These results suggest that a shift in pathogen community composition could be a major driver of pathogen impact. Many studies assess the impact of fungal infection on ecosystem functioning by comparing plant biomass in fungicide and non-fungicide plots (Mitchell 2003; Allan et al. 2010; Seabloom et al. 2017; Heckman et al. 2017). Our results show the importance of complementing these experiments with measures of infection severity and pathogen community composition. To increase our mechanistic understanding of the role of pathogens in affecting ecosystem functioning it is crucial to combine both approaches.

One alternative explanation for the altered impact of fungal infection on biomass under fungicide treatment might be non-target effects of the fungicide. However, studies show that the fungicides used here do not have phytotoxic effects when they are used in the recommended concentrations (Sundravadana et al. 2007; Khalko et al. 2009; Nithyameenakshi et al. 2006). Fungicides might also reduce beneficial fungi, like mycorrhiza belowground, or other mutualistic leaf-endophytes (Fokkema and Nooij 1981; Henriksen and Elen 2005). However, root samples of a subset of the experimental plant species showed no difference in mycorrhizal colonization between plants from fungicide and non-fungicide plots in 2017 (data not shown) and a loss of mutualists would be expected to reduce biomass production with fungicide application. This suggests that while non-target effects cannot completely be excluded, they are unlikely to be the key driver of our results.

Conclusions

We found strong support for growth-defense trade-off as a main driver of fungal infection. Fungal infection had an impact on biomass production, but this impact was context dependent, with greatest biomass loss due to pathogens in species rich communities receiving fungicide treatment. Fungicide altered the complex plant-pathogen interactions, beyond just removing pathogens, probably by removing certain fungi more efficiently than others. Fungicide may therefore have a wider range of effects in ecosystems than previously considered. This is both a challenge and an opportunity for studies using fungicide treatments

AUTHORS' CONTRIBUTIONS

SC, NP and EA designed and set up the PaNDiv experiment. NP and SC collected the data. SC analyzed the data and wrote the manuscript with substantial input from EA, NP and AK.

AKNOWLEDGEMENTS

We thank Hugo Vincent and the whole PaNDiv team and helpers, without whom maintaining such a large experiment would not have been possible. Thanks to Nadia Maaroufi and Tosca Mannall for their feedback on the manuscript and to Fletcher Halliday for the friendly review. This study was supported by funding of the Swiss National Science Foundation.

SUPPELMENTARY

Supplementary methods

Experiment

We set up a large field experiment (PaNDiv Experiment) in the Swiss lowlands, close to the city of Bern (mean annual temperature and precipitation 9.4±0.1°C, respectively 1021.62±31.89mm, MeteoSchweiz 2019). The grassland contains a species composition typical for a nutrient rich, rather dry, grazed grassland (Delarze 2015). We cleared an area of 3145m2 (85m x 37m) of all vegetation in autumn 2015 and sowed our experimental plant communities. Some species were resown in spring 2016, because of poor establishment. The experiment consisted of 336 2m x 2m plots, separated by a 1m path sown with a grass seed mixture consisting of *Lolium perenne* and *Poa pratensis* (UFA-Regeneration Highspeed) and mown regularly during the growing season.

We factorially manipulated plant species richness, plant functional composition, nitrogen addition, and foliar fungal pathogen exclusion. We used a set of 20 common grassland species to establish the experimental plant communities (Table S2). Half of the species were classified as fast, half as slow-growing based on specific leaf area (SLA) and leaf nitrogen content (Figure S2), which are traits indicative of the leaf economics spectrum (Reich and Cornelissen 2014; Wright et al. 2004). We did not include legumes in the species pool, because most legumes are adapted to low nitrogen levels and could therefore have been only included in the slow species pool only, making the species pools phylogenetically biased. The experimental communities contained either 1, 4, 8 or 20 species. Plots with 4 or 8 species could have either only slow-growing species, only fast-growing species or a mixture of both, which created a large gradient in community weighted mean traits. We grew monocultures of all species, which were either fast- or slow-growing, and the plots containing all 20 species inevitably had mixed functional compositions. The species for 4 and 8 species communities were chosen randomly from their respective species pools. To maintain species compositions, the plots were weeded three times a year. Plots were mown once in the middle of June and once in August, close to the dates when the farmers usually mow their extensive meadows (for more details see Pichon et al. (2019)).

Conceptual SEM

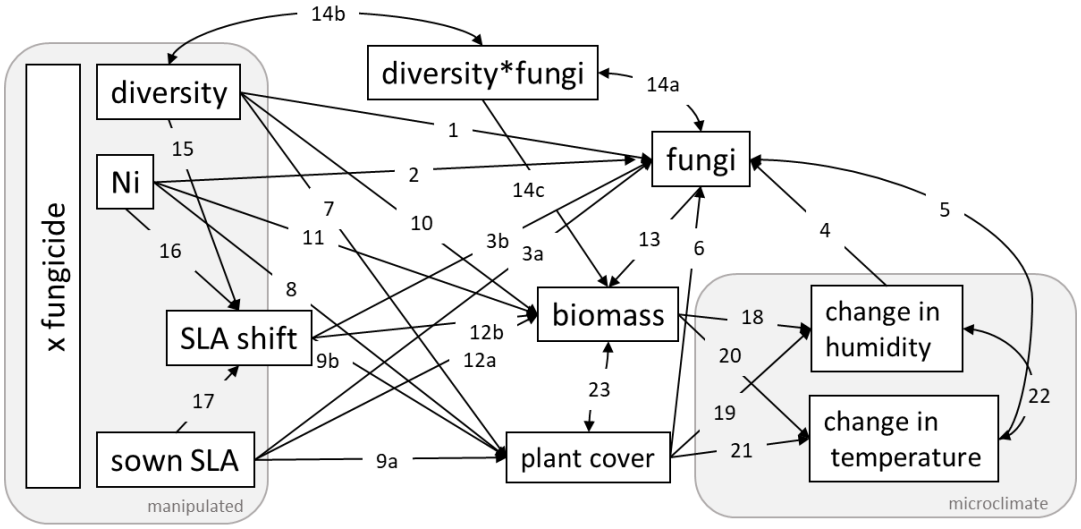


Figure S1 The full SEM that we tested. We tested all paths from the manipulated variables (left box) to the measured variables. The numbered paths are explained in Table S1

Table S1 Hypothesized mechanism driving fungal infection and biomass production in the SEM model (Figure S1)

Path	Hypothesized mechanism	Reference
1	(?/-) mostly negative diversity effect on infection in grasslands, through host dilution	Rottstock et al. (2014), Mitchell et al. (2003), Mitchell et al. (2002)
2	(+) nitrogen disease	Dordas (2008)
3	(+) growth-defense trade-off	Wright et al. (2004)
4	(+) high humidity is often beneficial for fungal pathogen growth and sporulation	Bregaglio et al. (2013), Chen et al. (2014), Sun et al. (2017), Bradley et al. (2003)
5	(+) an increase in temperature can increase fungal infection	Liu et al. (2016), Roy et al. (2004)
6	(?/+) spillover of generalist fungi because of higher density of potential hosts	Power and Mitchell (2004), Parker et al. (2015)
7,10	(+) positive diversity-productivity relationship	Tilman et al. (2001)
8,11	(+) nitrogen increases productivity due to nutrient limitation	Whitehead (1970), DiTommaso and Aarssen (1989), Fay et al. (2015)
9,12	(+) high growth rate at high SLA	Reich et al. (1992), Lavorel and Grigulis (2012)
13	(-) consumption of biomass through fungal pathogens	Allan et al. (2010), Seabloom et al. (2017)
14	(±) different selection pressure (due to host dilution), pathogen community composition shift	Laine (2006), Roy et al. (2000)
15	(±) increase in competition for light, sampling effect	Bachmann et al. (2018)
16	(+) nitrogen enrichment favors the abundance of fast growing plants	Lavorel and Grigulis (2012), Liu et al. (2018b), De Vries et al. (2012)
17	(±) likely the communities with extremely high/low sown SLA have the biggest negative/positive shifts towards intermediate SLA	
18, 19	(+) more plant transpiration in denser vegetation, shelter against wind that could remove humid air	Procházka et al. (2011)
20, 21	(-) more shading	Procházka et al. (2011)

Table S2 List of species used for the study design, their growth strategy, which was classified based on their specific leaf area and leaf nitrogen concentration and the supplier company of the seeds.

Grasses	Growth strategy	Supplier	Supplier resown
Poa trivialis	Fast	UFA	
Lolium perenne		UFA	
Holcus lanatus		UFA	UFA & field
Dactylis glomerata		R Hoffmann	R Hoffmann 2x
Helichotrichon pubescens	Slow	UFA	R Hoffmann
Festuca rubra		UFA	
Bromus erectus		R Hoffmann	R Hoffmann
Anthoxanthum odoratum		R Hoffmann	R Hoffmann 2x
Herbs			
Crepis biennis	Fast	UFA	
Taraxacum officinale		UFA	
Anthriscus sylvestris		UFA	R Hoffmann
Heracleum sphondylium		R Hoffmann	R Hoffmann
Galium album		R Hoffmann	
Rumex acetosa		R Hoffmann	
Achillea millefolium	Slow	UFA	
Centaurea jacea		UFA	
Daucus carota		UFA	UFA
Salvia pratensis		UFA	UFA
Prunella grandiflora		UFA	UFA
Plantago media		UFA	UFA

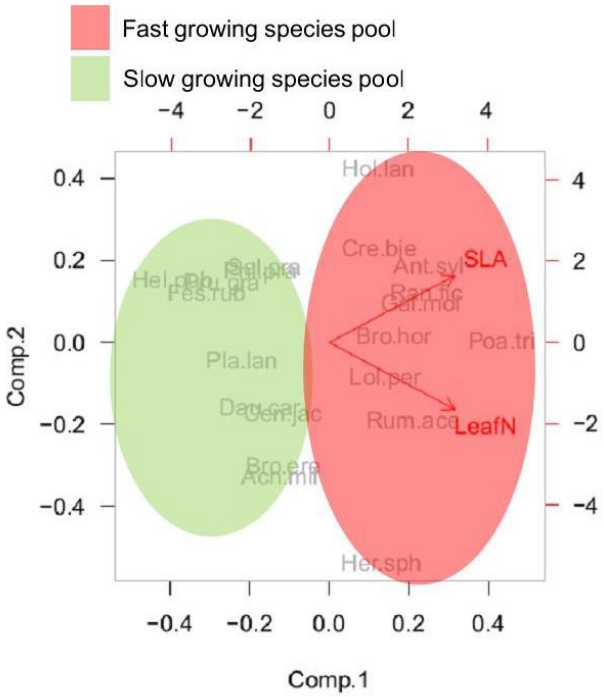


Figure S2 PCA categorizing the experimental species as fast and slow growing based on their values of SLA and leaf nitrogen

Measurements

Measures of SLA

By including the sown SLA and the SLA shift per plot, calculated based on the monoculture measurements with the corresponding nitrogen and fungicide treatment we accounted for abundance shifts and for plastic shifts following nitrogen and fungicide treatments, but not plastic shifts as a response to diversity. The latter, however is not significant compared to the plastic shifts as a response to nitrogen and fungicide (data not shown).

Infection

Measuring the % of infected individuals is different from many studies, which measure infeaction as damaged leaf area (e.g. Mitchell (2003), Halliday et al. (2017)), but likely more suitable to compare different fungal groups, as some (e.g. powdery mildews) mainly grow on the leaf , while others (e.g. rusts) mainly grow in the leaves, which makes a big part of the infection invisible (Klenke 2015). Percent leaf area damaged and percent infected individuals are log-correlated (Figure S3) and are therefore not fundamentally different from each other.

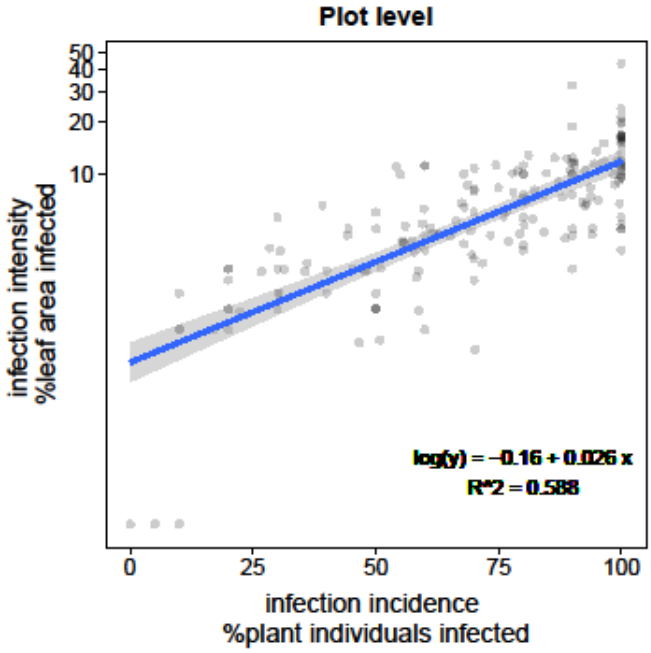


Figure S3 Correlation between community weighted mean of infection intensity based on % leaf area infected and community weighted mean of infection incidence based on % infected individuals. Data from fall 2018, as damaged leaf area was only assessed in fall 2018.

Microclimate

Table S3 Dates when humidity and temperature loggers were placed in which plots

Start	End	Plot numbers
16.7.2018	18.7.2018	239-252, 323-336
18.7.2018	20.7.2018	225-238, 309-322
20.7.2018	23.7.2018	211-224, 295-308
23.7.2018	28.7.2018	197-210, 281-294
28.7.2018	27.7.2018	183-196, 267-280
27.7.2018	30.7.2018	169-182, 253-266
30.7.2018	1.8.2018	71-84, 155-186
1.8.2018	3.8.2018	57-70, 141-154
3.8.2018	6.8.2018	43-56, 127-140
6.8.2018	8.8.2018	29-42, 113-126
8.8.2018	10.8.2018	15-28, 99-112
10.8.2018	13.8.2018	1-14, 85-89

Supplementary Analyses

Table S4 fixed effects of the fungi lmer

Fixed Effects	Estimate	SE	t value	Chi²	p-value
Intercept	0.42213	0.09771	4.32		marginal
Nitrogen	-0.10653	0.08237	-1.293		marginal
Fungicide	-0.78739	0.08393	-9.382		marginal
Species Diversity	-0.02767	0.08368	-0.331		marginal
Realized SLA	0.48125	0.08995	5.35		marginal
Nitrogen x Fungicide	0.08203	0.12109	0.677		marginal
Fungicide x Species Diversity	-0.06974	0.05908	-1.18		marginal
Nitrogen x Realized SLA	-0.13115	0.08343	-1.572		marginal
Fungicide x Realized SLA	-0.40054	0.09638	-4.156		marginal
Species Diversity x Realized SLA	0.23772	0.08665	2.743		marginal
Nitrogen x Fungicide x Realized SLA	0.36719	0.12227	3.003	9.187	0.002
Species Diversity x Fungicide x Realized SLA	-0.223	0.09118	-2.446	6.128	0.013
Random Effects	**Variance**	**SD**			
Composition	0.4232	0.6505			
Block	0.0031	0.0560			

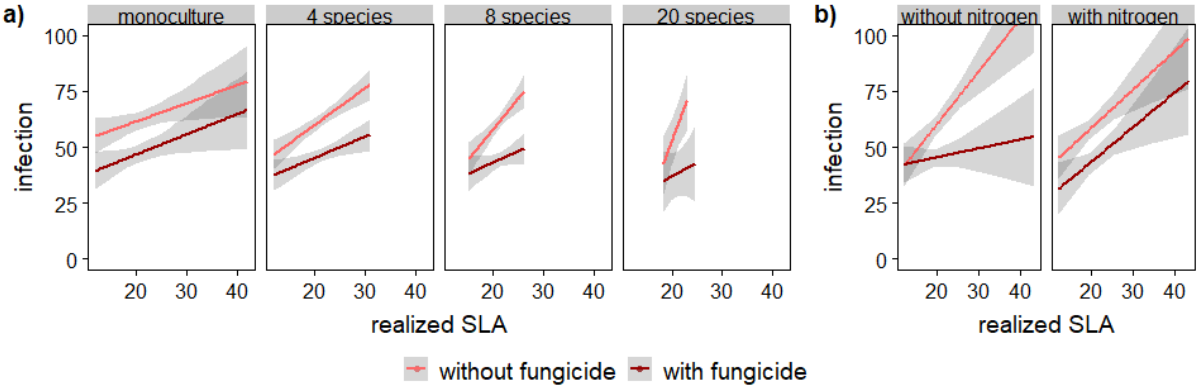


Figure S4 model predictions of lmer for fungal infection of all significant interactions terms (obtained from the effect package in r (Fox 2003)). Fungicide had significant interactions with a) fungicide, realized SLA and plant species diversity in explaining fungal infection, and with b) SLA as well as nitrogen in explaining biomass production. Fungicide reduced fungal infection on average (t-value= -11.942, infection without fungicide: 61.50 ± 1.38 %, infection with fungicide: 45.92 ± 1.39 %). Estimates and CI were derived from the effects package (Fox 2003)

Table S5 fixed effects of the biomass lmer

Fixed Effects	**Estimate**	**SE**	**t value**	**Chi²**	**p-value**
Intercept	-0.3259	0.1393	-2.3400		marginal
Nitrogen	0.5828	0.0881	6.6120	41.49	<0.001
Fungicide	0.0779	0.0864	0.9019		marginal
Realized SLA	-0.0865	0.0768	-1.1266		marginal
Species Diversity	0.1374	0.0688	1.9966	3.999	0.046
Fungicide x Realized SLA	0.1859	0.0879	2.1138	4.518	0.034
Random Effects	**Variance**	**SD**			
Composition	0.2340	0.4837			
Block	0.0330	0.1817			

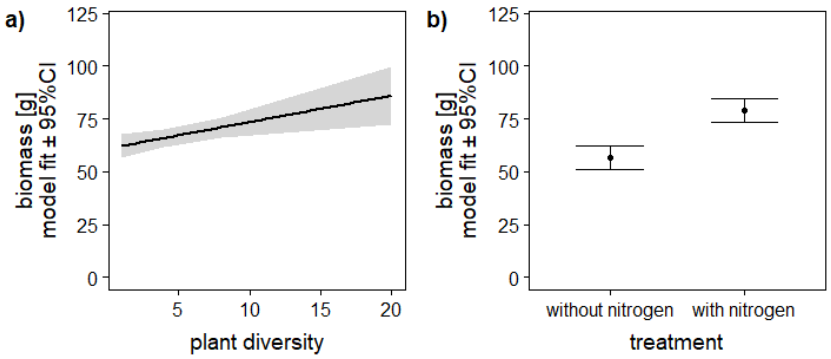


Figure S5 model predictions of the biomass lmer for a) plant diversity and b) nitrogen treatment (obtained from the effect package in r (Fox 2003)).

Table S6 Results from the SEM model constrain critera, where we tested whether paths and intercepts differed significantly with fungicide, by comparing the AIC values of a fully unconstrained model with a model where a particular paths was constrained to be equal between fungicide treatments. Note that we did not constrain the path between biomass and fungal infection, even though it does not significantly differ between treatments. Fungal infection is part of the interaction term, which cannot be constrained, we therefore did not constrain any paths that are part of this interaction.

		p value	
SLA shift ~	Intercept	0.00184	**
	Nitrogen	0.03828	*
	Plant Diversity	0.1991	
	Sown SLA	0.3178	
Fungal Infection ~	Intercept	1.36E-11	***
	Nitrogen	0.9706	
	Plant Diversity	0.3293	
	Sown SLA	0.7754	
	SLA Shift	0.7412	
	Humidity	0.7573	
	Temperature	0.5982	
	Plant Cover	0.6994	
Plant cover~	Intercept	0.143	
	Nitrogen	0.5777	
	Plant Diversity	0.9497	
	Sown SLA	0.07047	.
	SLA Shift	0.441	
Humidity~	Intercept	0.8263	
	Plant Cover	0.8938	
	Biomass	0.838	
Temperature~	Intercept	0.8052	
	Plant Cover	0.9976	
	Biomass	0.5632	
Biomass~	Intercept	0.4208	
	Nitrogen	0.8657	
	Plant Diversity	0.02808	*
	Sown SLA	0.01775	*
	Fungal Infection	0.1295	
	Fungi x Plant Diversity	0.02808	*
	SLA Shift	0.1629	
Humidity ~~	Temperature	0.9887	
Plant cover ~~	Biomass	0.2556	
Fungal Infection x Plant Diversity ~~	Plant Diversity	1.47E-11	***
	Fungi	0.4388	

Table S7 model fit indices of fully unconstrained model ant the final constrained model.

model	DF	AIC	P	RMSEA	CFI	SRMR
unconstrained	34	7945.6	0.204	0.036	0.989	0.045
constrained	64	7912.4	0.361	0.019	0.994	0.061

Table S8 SEM path, correlation and intercept estimates with and without fungicide treatment for the standardized data. Paths/correlations/intercepts labelled with c have been constrained, because they do not significantly differ between fungicide treatments.

Path			with fungicide Esti-mate	S.E.	z-value	p-value	without fungicide Esti-mate	S.E.	z-value	--value
Regressions:										
Response	Predictor									
SLA shift	nitrogen		0.245	0.077	3.196	0.001	0.027	0.075	0.364	0.716
	plant diversity	c	-0.069	0.054	-1.266	0.206	-0.069	0.054	-1.266	0.206
	sown SLA	c	-0.144	0.054	-2.672	0.008	-0.144	0.054	-2.672	0.008
fungal infection	nitrogen	c	-0.079	0.042	-1.908	0.056	-0.079	0.042	-1.908	0.056
	plant diversity	c	-0.03	0.05	-0.597	0.551	-0.03	0.05	-0.597	0.551
	sown SLA	c	0.331	0.042	7.933	0	0.331	0.042	7.933	0
	SLA shift	c	0.186	0.042	4.426	0	0.186	0.042	4.426	0
	micr. humidity	c	-0.082	0.054	-1.535	0.125	-0.082	0.054	-1.535	0.125
	micr. temperature	c	0.121	0.048	2.509	0.012	0.121	0.048	2.509	0.012
	plant cover	c	-0.061	0.051	-1.209	0.227	-0.061	0.051	-1.209	0.227
plant cover	nitrogen	c	0.195	0.051	3.835	0	0.195	0.051	3.835	0
	plant diversity	c	0.355	0.051	6.937	0	0.355	0.051	6.937	0
	sown SLA	c	-0.192	0.051	-3.742	0	-0.192	0.051	-3.742	0
	SLA shift	c	0.006	0.052	0.107	0.915	0.006	0.052	0.107	0.915
micr. humidity	plant cover	c	0.431	0.059	7.273	0	0.431	0.059	7.273	0
	biomass	c	0.071	0.06	1.198	0.231	0.071	0.06	1.198	0.231
micr. temperature	plant cover	c	-0.164	0.066	-2.488	0.013	-0.164	0.066	-2.488	0.013
	biomass	c	-0.027	0.067	-0.409	0.683	-0.027	0.067	-0.409	0.683
biomass	nitrogen	c	0.313	0.051	6.193	0	0.313	0.051	6.193	0
	sown SLA		0.031	0.081	0.384	0.701	-0.119	0.067	-1.779	0.075
	SLA shift	c	0.137	0.053	2.592	0.01	0.137	0.053	2.592	0.01
	fungal infection		-0.168	0.096	-1.747	0.081	-0.056	0.069	-0.814	0.416
	plant diversity		-0.054	0.099	-0.544	0.586	0.191	0.065	2.921	0.003
	infection x diversity		-0.362	0.115	-3.148	0.002	-0.124	0.08	-1.55	0.121
Indirect paths										
Fungal infection	p. div. – humidity	c	-0.013	0.009	-1.458	0.145	-0.013	0.009	-1.458	0.145
	N. – humidity	c	-0.007	0.005	-1.390	0.164	-0.007	0.005	-1.390	0.164
	s. SLA - humidity	c	0.007	0.005	1.386	0.166	0.007	0.005	1.386	0.166
Covariances:										
micr. humidity	micr. temperature	c	-0.463	0.057	-8.168	0	-0.463	0.057	-8.168	0
plant cover	biomass	c	0.328	0.048	6.793	0	0.328	0.048	6.793	0
fungal infection	infection x diversity	c	-0.292	0.038	-7.668	0	-0.292	0.038	-7.668	0
plant diversity	infection x diversity		-0.518	0.077	-6.74	0	0.195	0.067	2.909	0.004
nitrogen	plant diversity		0				0			
plant diversity	sown SLA		0				0			
nitrogen	sown SLA		0				0			
Intercepts:										
	SLA shift		0.177	0.077	2.306	0.021	-0.164	0.075	-2.185	0.029
	fungal infection		-0.352	0.061	-5.786	0	0.337	0.074	4.57	0
	plant cover	c	0.017	0.05	0.339	0.735	0.017	0.05	0.339	0.735
	micr. humidity	c	0.008	0.051	0.153	0.878	0.008	0.051	0.153	0.878
	micr. temperature	c	-0.005	0.056	-0.082	0.934	-0.005	0.056	-0.082	0.934
	biomass	c	-0.044	0.054	-0.824	0.41	-0.044	0.054	-0.824	0.41
	plant diversity	c	-0.002	0.056	-0.041	0.967	-0.002	0.056	-0.041	0.967
	nitrogen	c	-0.02	0.057	-0.344	0.731	-0.02	0.057	-0.344	0.731
	sown SLA	c	-0.005	0.057	-0.081	0.935	-0.005	0.057	-0.081	0.935
	infection x diversity		-0.102	0.062	-1.644	0.1	-0.072	0.068	-1.06	0.289
Variances:										
	SLA shift		0.917	0.104	8.832	0	0.83	0.096	8.602	0
	fungal infection		0.568	0.059	9.7	0	0.798	0.084	9.501	0
	plant cover		0.689	0.072	9.567	0	0.87	0.093	9.362	0
	micr. humidity		0.76	0.076	9.951	0	0.812	0.083	9.766	0
	micr. temperature		0.941	0.095	9.95	0	1.003	0.103	9.765	0
	biomass		0.891	0.093	9.556	0	0.656	0.07	9.354	0
	nitrogen		0.997	0.113	8.832	0	0.996	0.116	8.602	0
	plant diversity		0.961	0.109	8.832	0	0.98	0.114	8.602	0
	sown SLA		0.949	0.107	8.832	0	1.027	0.119	8.602	0
	infection x diversity		0.741	0.078	9.462	0	0.706	0.075	9.447	0

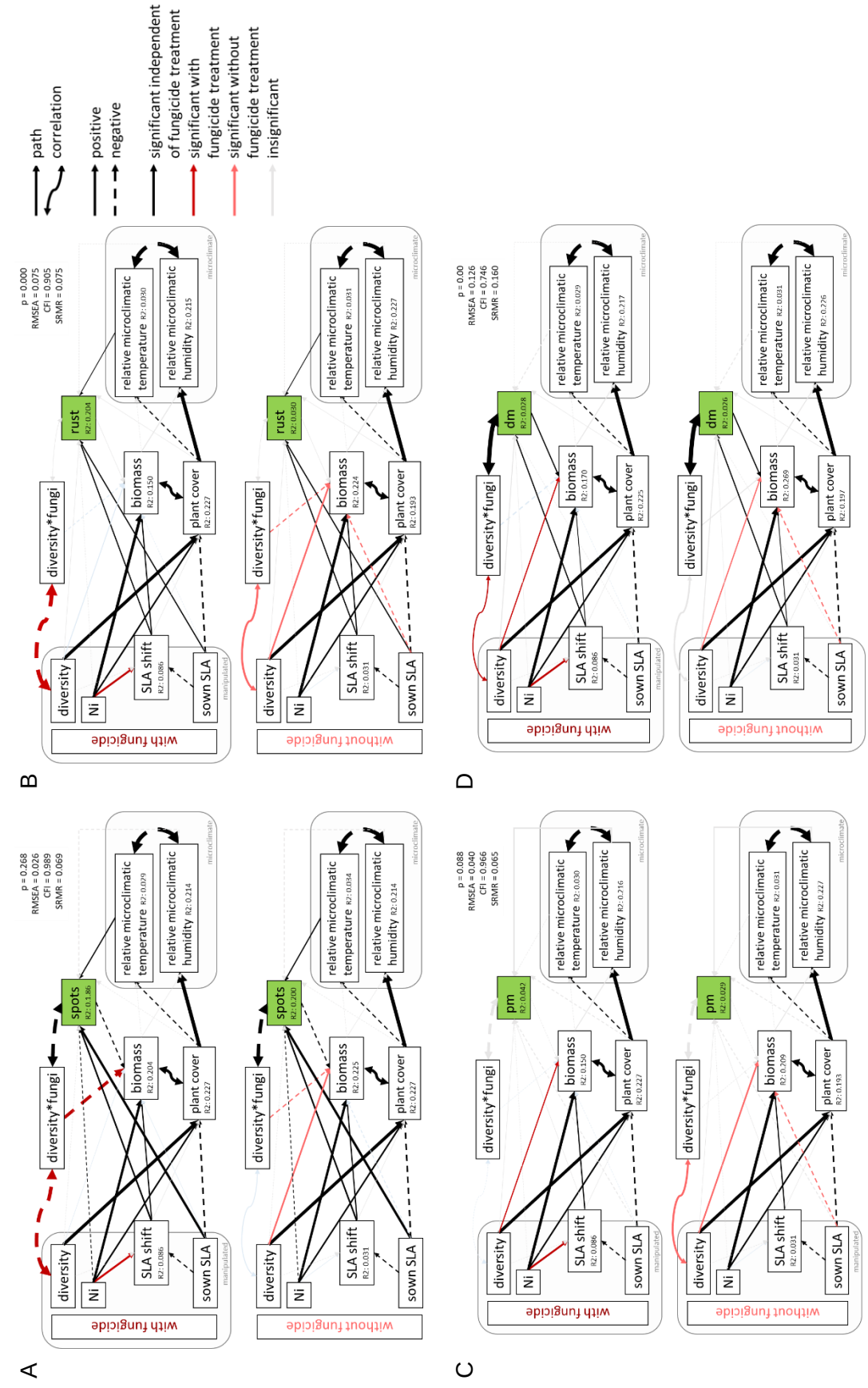


Figure S6 SEM results for the different fungal groups (A: leaf spots, B: rusts, C: powdery mildews, D: downy mildews)Figure S7

Table S9 Fixed effects of the host concentration lmer (Bates et al. 2015), with helmert contrasts. Model: host concentration slope ~ Nitrogen + Fungicide + Nitrogen x Fungicide + (1|Species)

Fixed Effects	**Estimate**	**S.E.**	**t-value**
Intercept	0.2262	0.0678	3.34
Nitrogen	-0.0681	0.0505	-1.35
Fungicide	-0.1003	0.0505	-1.99
Nitrogen x Fungicide	-0.0006	0.0505	-0.01
Random Effects	**Variance**	**SD**	
Species	0.0410	0.2025	

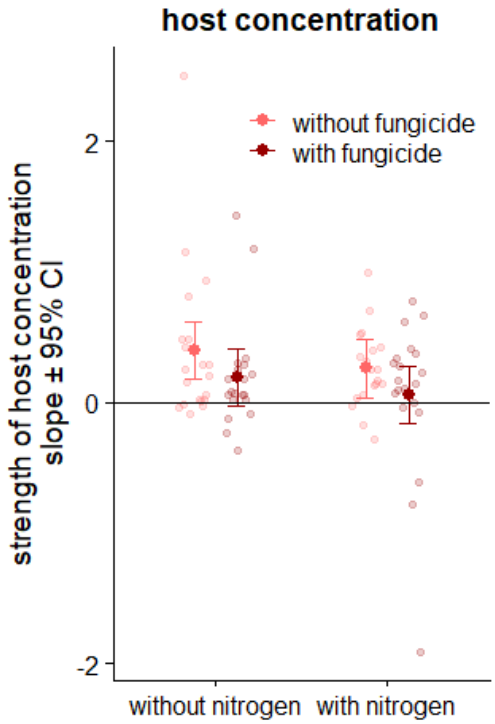


Figure S8 host concentration effect per nitrogen and fungicide treatment, raw data, predicted values and 95% confidence interval (obtained from the effect package in r (Fox 2003)).

Chapter 3

Resource use traits predict a growth-defense trade-off between, but not within, species

Seraina L. Cappelli, Noémie A. Pichon, Eric Allan

ABSTRACT

Biotrophic fungal pathogens rely on live hosts as food source. Their establishment and spread depends the availability of hosts, the defense of the hosts against pathogen infection and on the biotic and abiotic environment. A high abundance of host plants facilitates the spread of pathogens within a host population (resource concentration). The defenses of host plants vary, because defense mechanisms are subject to an ecological trade-off with fast growth within and between species (growth-defense trade-off). Further, neighboring plant species can also influence infection in a given host population, for example through spillover of pathogens or through altering microclimatic conditions (associational susceptibility/defense). This study aims to disentangle the relative importance of population and species level growth-defense trade-off, host concentration, and associational susceptibility. We find strong growth-defense trade-off between but not within species and some host concentration as drivers of infection. Additionally, we find associational susceptibility near closely related species, which is a form of host concentration expanded to phylogenetically related host groups. Having high infection does not necessarily cause high biomass loss. Some species are tolerant to infection. Our results hint that tolerance trades-off with quick resource acquisition.

INTRODUCTION

Fungal pathogens are everywhere in the environment and they can have a large impact on their hosts (Fisher et al. 2012). Several theories make predictions about when pathogens should have the largest impact. The **resource concentration hypothesis** predicts that fungal pathogens can readily spread and exploit their host plants when hosts grow at high density. Lower abundances of a given host species should therefore interfere with the transmission of host pathogens and lead to lower infection (Janzen 1970; Connell 1971; Burdon and Chilvers 1982; Knops et al. 1999; Mitchell et al. 2003). In addition to host density, host traits are likely to determine how susceptible a plant species is to infection. The **growth-defense trade-off hypothesis** predicts that defense comes with fitness costs and that plants can therefore invest either in defense against pathogens or in growth, with the optimal strategy depending on resource levels (Coley 1985). Slow growing species adapted to low nutrient environments should be strongly defended against natural enemies, as the production of plant material is costly when resources are scarce. On the other hand with increasing nutrient availability there is increased competition for light and space, which favors species which invest more of their resources into growth and less in defense (Wright et al. 2004; Endara and Coley 2011; Liu et al. 2017; Heckman et al. 2019). The leaf economics spectrum is a set of correlated traits, such as specific leaf area, leaf dry matter content and leaf nutrient concentrations, which distinguishes species adapted to nutrient rich environments, with fast growth, high nutrient acquisition and high leaf turnover, from species adapted to nutrient poor environments, with slow growth, tough leaves and low turnover (Wright et al. 2004). The traits of the leaf economics spectrum are therefore often correlated with infection (Cappelli et al. 2019; Cronin et al. 2010). There is substantial evidence for the resource concentration and growth-defense trade-offs as drivers of pathogen infection but their relative importance and how they manifest at different scales is not well known.

Resource concentration occurs at the population level, as species suffer more infection when they are abundant (Knops et al. 1999; Mitchell et al. 2003). However, resource concentration effects can scale up to the community level and result in reduced infection in diverse plant communities where each species is at lower abundance (Mitchell et al. 2003). In addition, species diversity can have further impacts on infection (Mitchell et al. 2003; Hantsch et al. 2014; Rottstock et al. 2014; Keesing et al. 2006), for example by creating favorable conditions for natural enemies of pathogenic fungi

(Dillen et al. 2017). In general, infection decreases with increased diversity more often than the opposite (Civitello et al. 2015). However, the importance of additional diversity effects beyond reduction in population abundance has rarely been tested.

The growth-defense trade-off is often studied by comparing species with different growth strategies (e.g. Blumenthal et al. 2009; Heckman et al. 2019), but there is evidence that the trade-off also occurs between different genotypes of the same species (Cole et al. 2016; Züst et al. 2015). However, the correlation between traits along the leaf economics trade-off axis is less consistent within than between species (Anderegg et al. 2018). It is therefore not clear if leaf economics traits are suitable to predict within species defense variability at all (Züst and Agrawal 2017). It has further been argued that the link between infection and the leaf economics spectrum might not be due to the growth-defense trade-off, but rather due to microclimatic effects related to leaf size (one component of specific leaf area, Bradley et al. 2003). Big leaves can accumulate droplets of water, which is crucial for spore germination and survival of many fungal pathogens (Bregaglio et al. 2013; Chen et al. 2014; Sun et al. 2017; Bradley et al. 2003). It is therefore important to test multiple traits simultaneously and consider other mechanisms related to these traits. The biotic and abiotic environment can also shape trait expression in plants, which adds additional complexity to the problem. For example nitrogen enrichment can increase leaf nitrogen content and specific leaf area (Firn et al. 2019) or an increase in plant diversity can increase specific leaf area (Lipowsky et al. 2015; Roscher et al. 2018b). It is not clear whether these intraspecific trait changes can happen independently of changes in defense and therefore whether environmental variation could disrupt correlations between leaf economics traits and defense.

The growth strategy of neighboring plants in a community can influence enemy damage in a focal plant, too. For example, certain plant species can function as reservoirs of pathogens. A reservoir host is a species that is infected by a pathogen and contributes to its dispersal but suffers little fitness reduction from pathogens. When reservoir species are present they can increase infection by causing pathogen spillover to other species, leading to associational susceptibility for plants co-occurring with reservoir species (Power and Mitchell 2004; Halliday et al. 2017). Traits related to the leaf economics spectrum can increase reservoir potential (Cronin et al. 2010) if fast growing species have a high tolerance for infection (Power and Mitchell 2004).

Potentially, the opposite could also occur if plants suffer less infection when surrounded by resistant species. The in- or decrease of enemy damage in a focal plant caused by neighboring plants is called associational susceptibility or associational resistance (Barbosa et al. 2009; Iason et al. 2018). In an earlier study we found that high community mean specific leaf area increased community level infection, supporting the idea that communities dominated by fast growing species have higher infection (Cappelli et al. 2019). This could be due to the high abundance of fast growing and thus heavily infected species and/or due to associational susceptibility and increased spillovers to slow growing species when they occur with fast growing ones. However, the relative importance of the two mechanisms remains unclear.

Fast growing species could have high infection but not necessarily suffer more from pathogen infection than slower growing plants. Fast-growing species often cope better with infection or herbivory and are more tolerant (Cronin et al. 2014; Kempel et al. 2019; Gianoli and Salgado-Luarte 2017). Having low defense is likely not so detrimental in a nutrient rich environment since damaged tissue can be replaced easily, especially when the species is good at acquiring the available nutrients (compensatory growth, Goodall et al. 2012; Keary and Hatcher 2004; Kempel et al. 2019). When growing without interspecific competition the species that can profit most from nutrient addition are likely to be those which are able to rapidly acquire nutrients (Tilman 1982). Therefore, a given level of infection should decrease biomass more strongly in slow-growing plants with slower nutrient acquisition rates than in fast-growing plants. To assess fungal pathogen impact we need studies manipulating pathogen abundance on host plants. Under field conditions, this is only possible by using fungicide. The response to fungicide can be expected to be driven by tolerance and defense, as both perfectly defended plants without infection and heavily infected but highly tolerant plants should not react to fungicide. The joint effects of tolerance and defense define the pathogen resistance of a plant (Haukioja and Koricheva 2000). We know that pathogen defense is correlated with plant growth. However, we do not know how strongly tolerance, resistance in general, growth rate and nutrient acquisition are related. Leaf traits indicating fast growth rate might not correlate perfectly with nutrient acquisition rates and pathogen tolerance and overall resistance might therefore trade-off more directly with nutrient acquisition, as tolerance is likely to be more closely linked to nutrient acquisition than growth rate *per se*.

In this study, we aim to test the relative importance of the growth-defense and resource concentration hypotheses in determining pathogen infection. We test how variation in traits at the community, species and population levels influence infection. Further, we test how different plant species react to nitrogen and to enemy exclusion and link these responses to growth traits and infection to test relationships between pathogen defense, pathogen resistance, resource acquisition and growth.

We test the following hypotheses:

The growth-defense trade-off should apply across scales: fast growing plant species and plant populations (with high SLA, low LDMC and high LA) should have higher infection and so should plants growing in fast growing communities (associational susceptibility).

The growth-defense trade-off should also predict pathogen impact and plant responses to nutrients: fast growing plants, with high infection, should increase with nitrogen (quick resource acquisition) and pathogen removal (low pathogen defense and high infection).

The resource concentration hypothesis should also predict infection: plants should have higher infection when their populations are large and when they grow in low diversity communities.

MATERIALS AND METHODS

Experiment

The study was conducted in the PaNDiv Experiment. The experiment was established in 2015 on an extensively managed (no fertilization for at least 10 years), quite dry grassland with naturally high fertility, typical for the Swiss lowlands. This region has a mean annual temperature of 9.4 ± 0.1°C and a mean annual precipitation of 1021.62 ± 31.89 mm (MeteoSchweiz 2019). The experiment factorially crosses nitrogen enrichment (0, 100 kg $ha^{-1}y^{-1}$ N in the form of urea), fungal pathogen exclusion (with fungicide, see below), species diversity (1, 4, 8, 20) and functional composition (gradient from fast to slow growing communities). Twenty common Swiss grassland species were used, 8 grasses and 12 non-leguminous forbs. They were classified as either fast or slow growing based on their SLA and leaf nitrogen traits. Plant communities of 1, 4, 8 and 20 species were established, with species randomly selected from the species pool. In the 4 and 8 species treatments we established plots with combinations of either only fast-growing species, only slow growing or a mixture

of both growth strategies, which produced a large gradient in mean trait values and in trait diversity. All plant combinations were present in 4 plots and received the full cross of nitrogen and fungicide treatments. For the exclusion of foliar fungal pathogens, we used a difenoconazole based systemic fungicide (Score Profi 106 by Syngenta Agro AG, 24.8% difenoconazole). As it was not very effective, we added another fungicide in 2018 (Ortiva by Syngenta Agro GmbH, 22.8% azoxystrobin). The data to test the drivers of pathogen infection at different scales were collected in summer 2017, however, to look at species responses to nitrogen and fungicide we used data also from 2016 and 2018, to look for consistent responses across multiple years. The experiment consists of 336 plots, arranged in four blocks (Pichon et al. 2019). However, for this study a subset of 200 plots was used, which included all monocultures and half of the 4 and 8 species plots but not the 20 species plots (due to the large number of measures that would need to be taken in the 20 species plots).

Measurements

We quantified the abundance of each sown plant species in each plot, using visual estimates of its **percentage cover** in the central square meter of the plot in the middle of August. We also assessed the abundance of weeds and bare ground. The cover was estimated by three well trained persons, who calibrated their measurements in the field. To account for potential remaining differences, each block was measured by only one person (meaning any differences between recorders are accounted for by the block random effect). The sum of all the measures per plot could exceed 100% but species abundances were converted to relative values for the analysis.

We measured three leaf traits on all species in all plots. We measured **leaf area** (LA), **specific leaf area** (SLA) and **leaf dry matter content** (LDMC) on five leaves per species and plot in August 2017, shortly before the biomass harvest, following the protocol of Garnier et al. (2001). The mean of the five LA, SLA and LDMC per species and plot was calculated. *Heracleum sphondylium* had not established very well and there were not enough suitable leaves in many plots, in addition, the measurements are destructive and might have killed the few plants there. For this reason, we did not measure traits for *H. sphondylium*. The *P. trivials* SLA measurements were much higher than all the other SLA measurements and their variation much bigger. The leaves were still very small and very often too young for SLA measurements (Supplementary). We therefore excluded *P. trivialis* from the analysis.

We then calculated measures of functional traits at the community, species, and

individual level. To measure the mean growth strategy of the **community**, we calculated community weighted mean (CWM) values for LA, SLA and LDMC, using the percent cover measurements to weight the traits. To measure the growth strategy for each **species**, we calculated the mean LA, SLA, and LDMC per species across all plots. To measure the growth strategy of the **populations** (defined as one species in one plot), we calculated the difference to the species control (without fungicide, without nitrogen) monoculture. We did this for LA, SLA, and LDMC per species and plot, to calculate ∆LA, ∆SLA and ∆LDMC. These measures reflect the intraspecific variation in the traits, independent of the species mean trait values.

Fungal infection was measured in September 2017 in all species in all the plots, by screening ten individuals (if possible) of the species in the central square meter of the plot. If there were not enough individuals in the central square meter, individuals from the rest of the plot were considered. If fewer than 10 individuals were present in the whole plot, all individuals were screened for infection. Each plant was classified as infected or not infected and the percent of infected individuals was calculated. Fungal infection can be interpreted as a proxy of the outcome of defense against fungal infection, assuming that the sum of all (effective) defense mechanisms define the amount of infection.

In total, there were 800 possible species x plot combinations, of these, 634 were realized (Table S1-2) Eighty had to be removed because of a lack of suitable leaves for SLA measurements (*Poa trivials* and *Heracleum sphondylium*, Supplementary), the remaining missing values represent cases where species did not successfully establish in a given plot.

Finally, we measured plant aboveground biomass production in each plot. We harvested **biomass** in two subplots of 0.1m^2 per plot, 5cm above ground level in August 2017. Biomass was dried and weighed.

In the monocultures, these measurements were taken more often. We have biomass measurements for August 2016, 2017 and 2018 and June 2017 and 2018. The traits and infection were measured in the monocultures as well for the same sampling periods, except in June 2017.

Statistical analysis

Pathogen infection data for all species in all plots was logit transformed and analyzed using linear mixed effects models. The analyses were conducted in R (R Core Team

2018) with the lme4 package (Bates et al. 2015). The full model included the treatments, nitrogen, fungicide and sown plant species diversity, together with all possible interactions between them, as well as the community, species and population level trait values and interactions between the traits and the treatments (only two way interactions were allowed between traits and treatments to avoid fitting very complex models). We also included the percentage cover of the species to test for resource concentration effects. Further, we included a term for plant functional group, i.e. whether the plant species was a grass or an herb. Functional group could interact with all trait measures and with species percentage cover, to account for potentially different growth-defense and resource concentration effects between grasses and herbs. Specific plant species composition, block, plot and species were used as random effects. Fixed effects were stepwise removed if they did not contribute to an improved model fit, based on likelihood ratio tests. To account for potential correlations between the different traits the same model was run for each trait separately, which resulted in the same significant terms.

To further study the (between species) growth-defense trade-off, we compared the responses of species to nitrogen and fungicide in the monocultures. We analyzed whether species, which are able to increase their biomass most strongly following nitrogen enrichment are the same that increase their biomass following fungicide treatment and whether this is related to their monoculture infection level or specific leaf area. This was done by correlating monoculture data across different time points (August 2016 and June and August 2017, depending on the data availability as not all data is available for all time points). We used monoculture data only in order to exclude the influence of interspecific competition on responses to nitrogen and fungicide. For the traits and infection, we used control (no nitrogen, no fungicide) monoculture measurements. The biomass response to fungicide and nitrogen treatment was calculated as the log response ratio. The biomass log response ratio to nitrogen is a good indicator of resource acquisition (Figure S9). In order to test for interactions between response to nitrogen and fungicide, i.e. whether the same species increase with fungicide in the presence and absence of nitrogen, we calculated the log response ratio to nitrogen with and without fungicide separately. The response to fungicide was calculated at without nitrogen addition.

RESULTS

We found that pathogen infection was affected by variables at the community, species

and individual levels (Table 1). Of these, the species level specific leaf area (SLA) was the most important and had the largest effect on pathogen infection. Species with high SLA had higher infection overall. However, this effect was much stronger in herbs than in grasses, as grasses generally had higher infection and even grasses with lower specific leaf area were heavily infected (Figure 1b). The leaf dry matter content and the leaf area of the species were not significantly linked to infection.

At the population level, the relative abundance of a species had an impact on its fungal infection. The more abundant a species was, the higher its infection became. The relative abundance of species is to some degree linked to the plant diversity of the community (Figure S4). The lack of plant diversity effects on infection, suggests that diversity has no additional effect on infection beyond its link to relative abundances.

At the community level, the mean leaf dry matter content increased infection (CWM LDMC) (Figure 1a). As grasses had higher LDMC than herbs, CWM LDMC was strongly linked to grass abundance (sum of the cover of all grass species present in a plant community, Figure S5) this means that a high abundance of grasses in a community increased infection in single species growing in that community. Replacing CWM LDMC with grass cover in the model shows high grass cover increased infection in the grasses, but not the herbs (Table S4, Figure S6). The herbs rather showed a decrease of infection with increasing grass cover, but the confidence interval for this effect was large. In addition, the cover of the individual species became insignificant when community weighted mean of LDMC was replaced with grass cover. This indicates that the grasses drove many of the resource concentration effects observed. As the grasses are phylogenetically related (Figure S7) and share many fungal pathogens (Klenke 2015), they seem to be sensitive to the abundance of closely related species. The full model containing grass cover fitted the data better than the model with CWM LDMC (AIC grass cover model: 2584.4, AIC CWM LDMC model: 2589.2, $p < 0.001$) supporting the idea, that the observed effect of CWM LDMC is mainly due to the effect of grass cover.

Table 1 Results of the mixed effects model explaining fungal infection with plot (orange), species (green) and population (yellow) level trait values as explanatory variables in addition to species abundance (% cover) and treatment variables (blue). Insignificant terms are shown in grey and with lighter colored background. For insignificant higher order interaction terms see Table S3. Significances of lower order interactions (functional group, SLA) were achieved by comparing models without the higher order interaction with and without the specific term. Abbreviations: community weighted mean (CWM), leaf dry matter content (LDMC), specific leaf area (SLA), leaf area (LA)

fixed effects	Estimate	S.E.	X^2	p-value	
Intercept	4.945	0.890			
cover	0.233	0.102	5.141	0.023	*
CWM LDMC	0.216	0.108	3.949	0.047	*
SLA	0.698	1.343	5.699	0.017	*
functional group (Herb)	-0.965	1.173	1.979	0.160	
SLA x functional group (Herb)	4.638	1.951	4.907	0.027	*
Fungicide			3.445	0.063	.
Nitrogen			0.864	0.353	
Plant Diversity			0.114	0.735	
CWM SLA			0.057	0.811	
CWM LA			0.246	0.62	
LA			0.058	0.809	
LDMC			2.126	0.145	
ΔSLA			1.345	0.246	
ΔLA			0.842	0.359	
ΔLDMC			0.132	0.717	

Random effects	Variance	S.D.
Plot	0.451	0.672
Composition	0.000	0.000
Species	6.555	2.560
Block	0.017	0.130

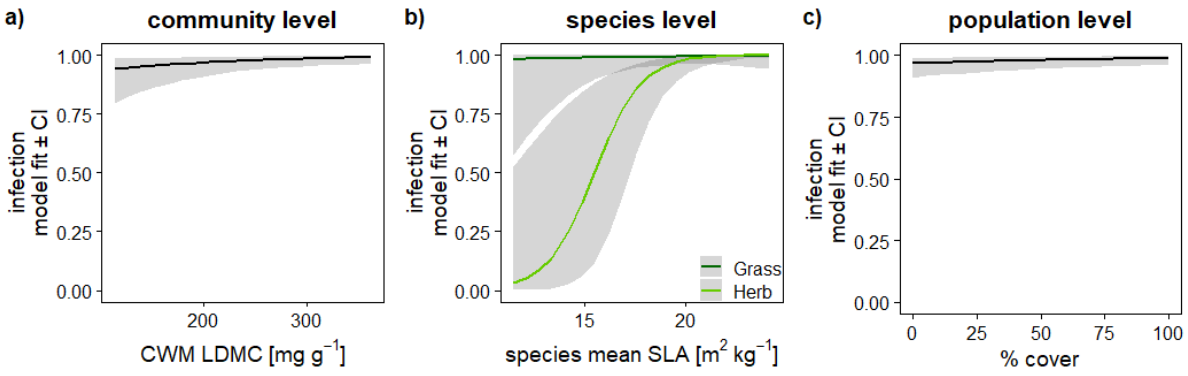


Figure 1 linear mixed effects model predictions for all significant terms ± 95% confidence interval. How a) community weighted mean of leaf dry matter content, b) the mean specific leaf area of a given species depending on the functional group and c) the abundance of a species in a plot measured as percent infection influence the proportion of infected plant individuals in a population. The detailed model results are shown in Table 1.

The species level growth-defense trade-off was the key driver of infection in our experiment and we therefore investigated the trade-off further by correlating species responses to nitrogen and fungicide. Correlated species responses to the treatments in monoculture showed that species which increase their biomass following nitrogen enrichment , were also the ones benefitting most from fungicide treatment (Figure 2a). This correlation between biomass response to nitrogen and fungicide also became stronger over time (Figure 2a). Looking at species in monoculture we also generally saw a positive correlation between SLA and infection, supporting the analysis using the species values across all plots. Although consistent, this correlation was only marginally significant in August 2018, perhaps due to the low power when the analysis is restricted to monocultures. However, neither the response to nitrogen nor the response to fungicide could be linked to SLA. The correlation was never significant and the direction of the correlation was inconsistent across the years (Figure 2e-f). This means that it is not species with high (or low) specific leaf area that increase in biomass following nitrogen enrichment or fungicide treatment. Although insignificant, infection was consistently negatively correlated with biomass response to nitrogen across time, which indicates that species with low infection may benefit from nitrogen enrichment (Figure 2g). There was no correlation between infection and response to fungicide, indicating that high infection levels do not indicate a high impact of pathogens on a given species (Figure 2h).

Nitrogen and fungicide also interacted to affect species responses. The correlation between the biomass log-response ratio to nitrogen with and without fungicide was not significantly correlated, which means that different species were able to increase their biomass in response to nitrogen enrichment in the presence and absence of fungal pathogens (Figure 2b). Species, which increase strongly with nitrogen when fungal pathogens are present (and to fungicide at ambient nitrogen levels) seem not to benefit from nitrogen when fungal pathogens are suppressed. This might indicate that in the absence of fungal pathogens, the species, which typically increase with nitrogen were not able to benefit from additional nutrients. Also, the response of biomass to fungicide with and without nitrogen was not significantly correlated (except for in June 2018), showing that different species benefitted from fungicide at different nitrogen levels (Figure 2c).

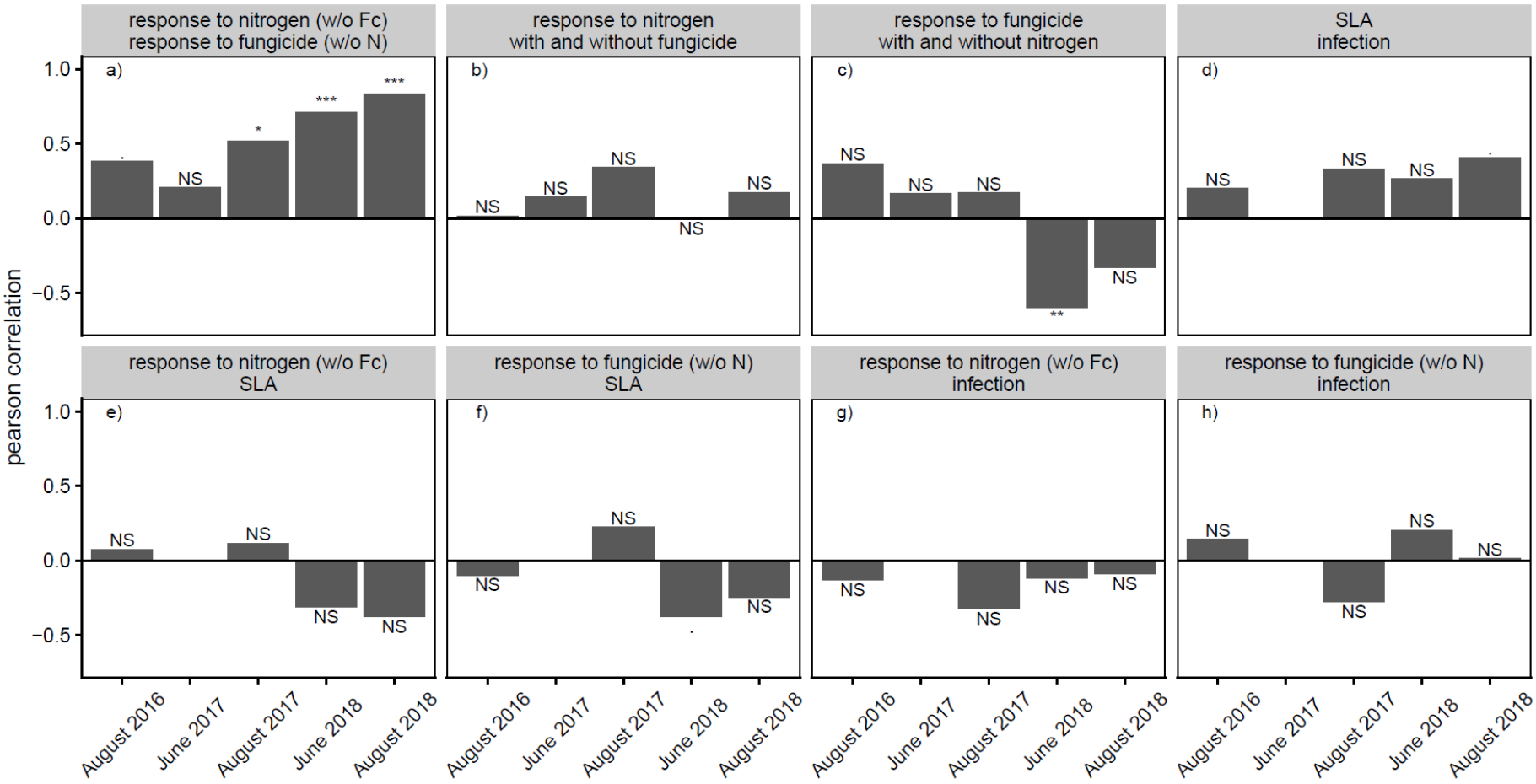


Figure 2 correlations between a) biomass response to fungicide (pathogen resistance) without nitrogen enrichment (N) and response of biomass to nitrogen (resource acquisition, see Figure S9) without fungicide treatment (Fc), b) the biomass response to nitrogen with and without fungicide treatment to show how pathogens change resource acquisition, c) the biomass response to fungicide with and without nitrogen enrichment to show how resources alter pathogen resistance, d) specific leaf area (SLA, as a measure of growth strategy) and infection (as a measure of defense), e) biomass response to nitrogen (resource acquisition) in the presence of fungal pathogens and SLA (growth strategy), f) biomass response to fungicide (pathogen resistance) without nitrogen enrichment. .and SLA (growth strategy), g) biomass response to nitrogen (resource acquisition) in the presence of fungal pathogens) and infection (defense) and h) biomass response to fungicide (pathogen resistance) without nitrogen and infection (defense) across different time points of the experiment. All data was measured in the monocultures of the species. Details about which species contribute how to the observed correlations can be found in Figure S8.

DISCUSSION

We found that the specific leaf area of a species is a key driver of its infection, indicating that the growth-defense trade-off mainly exists at the species level. This is consistent with other studies, which find high infection in species that are adapted to high nutrient environments (Blumenthal et al. 2009; Liu et al. 2017). The increase in infection with increasing SLA was pronounced in herbs, but less so in grasses. We expected fast growing species to be more tolerant, but less resistant, to infection in general. However, we observed high infection in all grasses, even in slow growing ones with low SLA. This could indicate that pathogen tolerance is not linked to the growth-defense trade-off, but that there is an additional trade-off between defense and tolerance (Figure 3a, correlation ?[4]), similar to trade-offs between different components of defense (Kempel et al. 2011; Koricheva et al. 2004, discussed later). Grasses often have a high tolerance to herbivory (Anderson et al. 2013; Anderson and Briske 1995; Coughenour 1985; Barthelemy et al. 2019) and it is possible that they are also more tolerant against fungal pathogens and generally use tolerance instead of defense to cope with natural enemies (Haukioja and Koricheva 2000). As SLA is usually the better predictor of plant growth than LDMC or LA (Pérez-Harguindeguy et al. 2013) we can conclude that a growth-defense trade-off is an important mechanism driving infection. The trade-off holds mainly between species, but breaks down at certain scales, i.e. within species or between grasses.

At the population level, none of the tested traits were significantly correlated with infection. This might suggest that within species growth-defense trade-offs are rather weak compared to between species trade-offs (Heckman et al. 2019). However, it might also be that within species, trade-offs cannot be well captured by resource economic traits. Correlation between the leaf economics traits are less pronounced within than between species. The break down in correlations is partly because there is much less trait variation within species, but it is potentially also due to trait plasticity or different selective forces operating on intraspecific trait variation (Anderegg et al. 2018). Other studies have shown that within species, trait expression is not as tightly linked to growth strategy (Derroire et al. 2018; Roscher et al. 2018a) and species can to some degree change the expression of one resource economics trait without simultaneously changing the expression of other, typically correlated, traits (Chapter 4). SLA can be increased by reducing leaf thickness and increasing leaf area, which can happen independently of changes to dry matter content. Such intraspecific

changes allow adaption to environmental conditions but do not necessarily promote growth (Poorter et al. 2009). For example, species can adjust their SLA to increase light interception in response to shading, without simultaneously changing other leaf economic traits as well (Lipowsky et al. 2015). These findings caution that we might not be able to observe within species growth-defense trade-offs by looking at traits alone.

Leaf area was not linked to infection, at neither population, species nor community level. This indicates that the ability of big leaves to create favorable microclimatic conditions for pathogens was not important in increasing infection in our study. Bradley et al. (2003) showed that large leaves were better at capturing droplets of water, which favored the germination of fungal spores. In the field however, the impact of water retention on infection was context dependent. In a dry study site, species with higher water retention had higher infection, while in wet conditions there was no link between water retention and infection and all species had high infection. (Bradley et al. 2003). Our results suggest that even though leaf area might have an impact on infection under some circumstances, the link between the leaf economics spectrum and infection is mainly due to a growth-defense trade-off.

At the population level, host abundance was the only variable influencing infection. Infection increased with increasing abundance, which supports the resource concentration hypothesis (Burdon and Chilvers 1982; Knops et al. 1999; Mitchell et al. 2003). However, the effect size was rather small compared to the effect size of species SLA. Host abundance was linked to plant species diversity, indicating that diversity can at least weakly decrease infection through host dilution, however, diversity had no additional effects on infection (Keesing et al. 2006). It is likely, that we observed only weak effects of host abundance because of pathogen spillover from neighboring plants (Power and Mitchell 2004; Halliday et al. 2017). Closely related species often share pathogens and pathogen infection may respond to the abundance of closely related species in an area (Parker et al. 2015; Gilbert and Webb 2007). We find some support for this, as the model including grass cover instead of community level LDMC fitted the data better and showed that grasses, but not herbs, suffered more infection when surrounded by a high density of other grasses. This suggests that resource concentration effects are driven not just by the abundance of conspecifics but by the abundance of closely related species. Unfortunately, our study design is not suitable

to fully test for community level resource concentration due to phylogenetic relatedness, as we lack many other confamilials in our species pool and we do not have plots containing only grasses or herbs, which would allow us to separate LDMC from grass cover effects. Another possible explanation for the low effects of host cover and thus diversity relative to other studies (Rottstock et al. 2014; Mitchell et al. 2002; Mitchell et al. 2003; Liu et al. 2016), might be the design of our experiment. The PaNDiv experiment crosses manipulations of diversity and plant growth strategy and the large effects of growth strategy on infection might mask any diversity effects. Host plant abundance was the only population characteristic that had an impact on infection, indicating that resource concentration was more important than growth-defense trade-off at the population level, in fine tuning patterns of infection.

We also found no effect of the community SLA on infection. This indicates that fast growing species have more infection but that they do not cause spillovers to slow growing species. We therefore find no evidence for associational susceptibility for species growing in fast-dominated communities. Spillover happens preferably between closely related species (Gilbert and Webb 2007). Therefore, the community level SLA might be less important than the SLA of closely related species. Plots containing only closely related species covering a large range of SLA would be needed to test for this. Previous results showing that a high community mean SLA increases infection in this experiment (Cappelli et al. 2019) therefore seem to be due to high SLA species supporting high levels of pathogen infection, but not due to increased spread of pathogens between species.

High levels of pathogen infection (indicating low host defense) did not necessarily reduce plant biomass, as the biomass response to fungicide (total host resistance to infection) in monocultures was not correlated with infection across the years (Figure 3a, correlation h). This suggests that tolerance might be a valuable alternative strategy to deal with fungal pathogens (Figure 3a, correlation ?[1]), as proposed by other studies (Roy et al. 2000; Chase et al. 2000; Kempel et al. 2019; Gianoli and Salgado-Luarte 2017). Defense alone (inverse of infection) was consistently negatively linked to growth strategy (Figure 3a, correlation d) but not to overall resistance, in contrast to studies on large herbivore impact (Lind et al. 2013). The lack of correlation between total resistance (response to fungicide) and defense might be explained by a trade-off between tolerance and defense (Chase et al. 2000; Roy et al. 2000, but see Cronin et al. 2014). Both, heavily defended (and thus infection free species), but not tolerant and

highly tolerant, but not defended species should not increase biomass when fungicide is applied. However, if a heavily defended species would be infected nonetheless, it would benefit a lot from fungicide, but this is unlikely to be observed in the field (Figure 3b). Tolerance may therefore play an important role in addition to defense in determining pathogen resistance (Figure 3a, correlation ?[2]). However, to systematically disentangle the effects of resistance and tolerance under different nutrient levels, studies manipulating infection levels through inoculations would be needed, as it is not possible to measure tolerance under field conditions.

Total resistance to infection was positively correlated with biomass response to nitrogen (Figure 3a, correlation a), rather than to SLA and plant growth strategy (Figure 3a, correlation f). As the response to nitrogen was also uncorrelated with growth strategy, nitrogen acquisition strategy may differ from overall growth strategy. A recent experiment with 15N labelled ammonia and nitrate (Walde 2019) showed that different species have high nitrogen uptake under ambient and increased nitrogen availability, suggesting a trade-off between competitive ability for nitrogen and ability to rapidly acquire nitrogen. It is the species most able to take up nitrogen under high nitrogen conditions that increase with nitrogen addition (Figure S9). This agrees with theory stating that coexistence requires that species rapidly draw down the nutrient they find most limiting (Tilman 1982). The species which profit from nitrogen are therefore those able to rapidly acquire nitrogen when it is supplied at high rates and these are the species that suffer from pathogens. Our results therefore suggest a trade-off between nitrogen uptake and ability to cope with pathogens. This is probably linked to the nitrogen limitation of the species with quick nutrient acquisition. Nitrogen limitation might reduce the capacity to compensate for lost tissue (Wise and Abrahamson 2005). The trade-off between nitrogen uptake and tolerance could be linked to the root economics spectrum, which should indicate belowground nutrient acquisition strategy (Mommer and Weemstra 2012; Fort et al. 2016). The root economics spectrum is not strongly linked to the leaf economics spectrum in grassland species (Schroeder-Georgi et al. 2016; Bergmann et al. 2017, but see Reich and Cornelissen 2014) which supports the idea of two (at least partly) independent trade-offs. The growth-defense trade-off has received a lot of attention as a driver of enemy impact but our results suggest that there may be additional trade-offs linked to resource acquisition that determine the impact of fungal pathogens on their hosts.

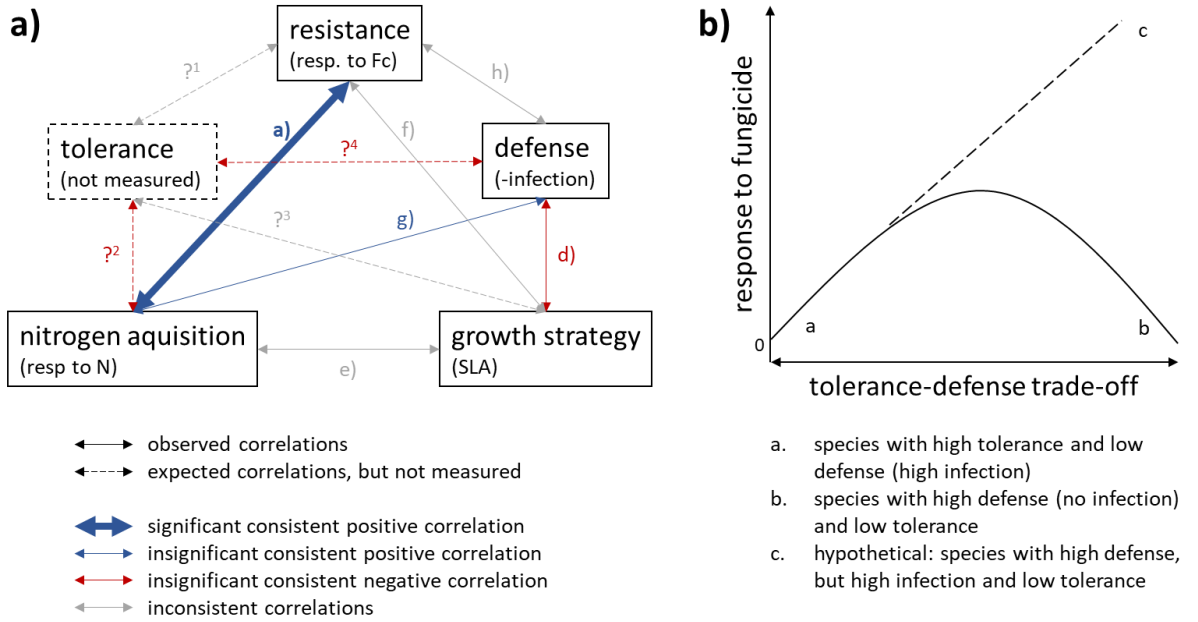


Figure 3 a) overview over the correlation analysis of the monoculture measurements across the year (Figure 2) and what this could mean for infection tolerance. Pathogen resistance represents the joint consequences of tolerance and defense to infection. Of the two, we measured defense (as the inverse of infection), but not tolerance, as this is not possible in our field setting. Total resistance was not consistently linked to defense (h), which means that species with high infection did not benefit most from fungicide. This suggests that tolerance plays an important role (?[1]). The only significant correlation was between total resistance and nitrogen acquisition (a). Given that defense is not significantly linked to resistance, this suggests that tolerance is the main reason for the strong link between resistance and nitrogen acquisition (?[2]). Although not significant, but consistent across the years, defense was negatively correlated with growth strategy (d). We expected fast growing species to be highly tolerant (?[3]), but given that resistance and growth strategy are not related, there is no indication that this is true. b) the lack of a significant correlation between defense and resistance might be explained by the joint effect of tolerance and defense, which are expected to trade-off. Both, highly tolerant (b) and highly resistant (b) species likely do not respond to fungicide. The highly tolerant species would not respond to fungicide, because they are largely unaffected by fungal pathogens, the highly resistant species do not respond, because they should not have infection. If for some reason, even highly defended, but not tolerant species would get infected nonetheless, they should increase biomass strongly following fungicide treatment (c).

Interestingly, species responses to nitrogen and pathogens also interacted. The plants which increased with fungicide under ambient N were not the same as those that benefitted from fungicide under increased nitrogen. A high availability of nitrogen might have allowed certain species to be more tolerant of infection (Kempel et al. 2019; Horgan et al. 2018). It is also possible that when fungi are suppressed insect herbivores invade the shared and now free niche (Raffa et al. 2019; Thaler et al. 2012; Cappelli et al. 2019), which would explain why some species don't benefit so much from fungicide. A shift from strong limitation by nitrogen to increased limitation by other resources in N addition plots could also alter species responses to pathogens. Different species also benefitted most from nitrogen when fungal pathogens were present and when they were suppressed. We know that fungicide doesn't exclude pathogens completely and alters the composition of the fungal community (Cappelli et

al. 2019) and a shift in the pathogen community could therefore alter species responses to nitrogen with and without fungicide. It should be noted, that these results are based on the monoculture data only, which means we have one replication per species and treatment per year. Nonetheless, the results hint at interactive effects of nitrogen and pathogens.

Our results show that the growth-defense trade-off is the major driver of pathogen infection. In general, species level characteristics were the key drivers of pathogen infection, while community context and intraspecific variation were of relatively minor importance. Changes in plant species composition are therefore likely to be the major driver of changes in pathogen abundance. However, although resource economics traits predicted pathogen infection (defense) they did not predict pathogen impact on biomass production. In contrast nitrogen acquisition strategy seems to predict pathogen impact and may trade-off with tolerance against pathogens. It is therefore likely that plant species differentiate along multiple trade-offs axes, between tolerance, defiance and competitive ability for particular nutrients. In order to understand the ecological role of fungal pathogens, it will be important to consider tolerance and to develop frameworks which include multiple trade-offs simultaneously.

ACKNOWLEDGEMENTS

This study is part of the PaNDiv experiment, which is funded by the Swiss National Science Foundation. Such a large project would not be possible without the work of many people. We would like to thank everyone who was involved in field maintenance and data collection, especially Hugo Vincent, Mervi Laitinen, Tosca Mannall, Thu Zar Nwe, Manuel Walde and the many student helpers.

SUPPLEMENTARY

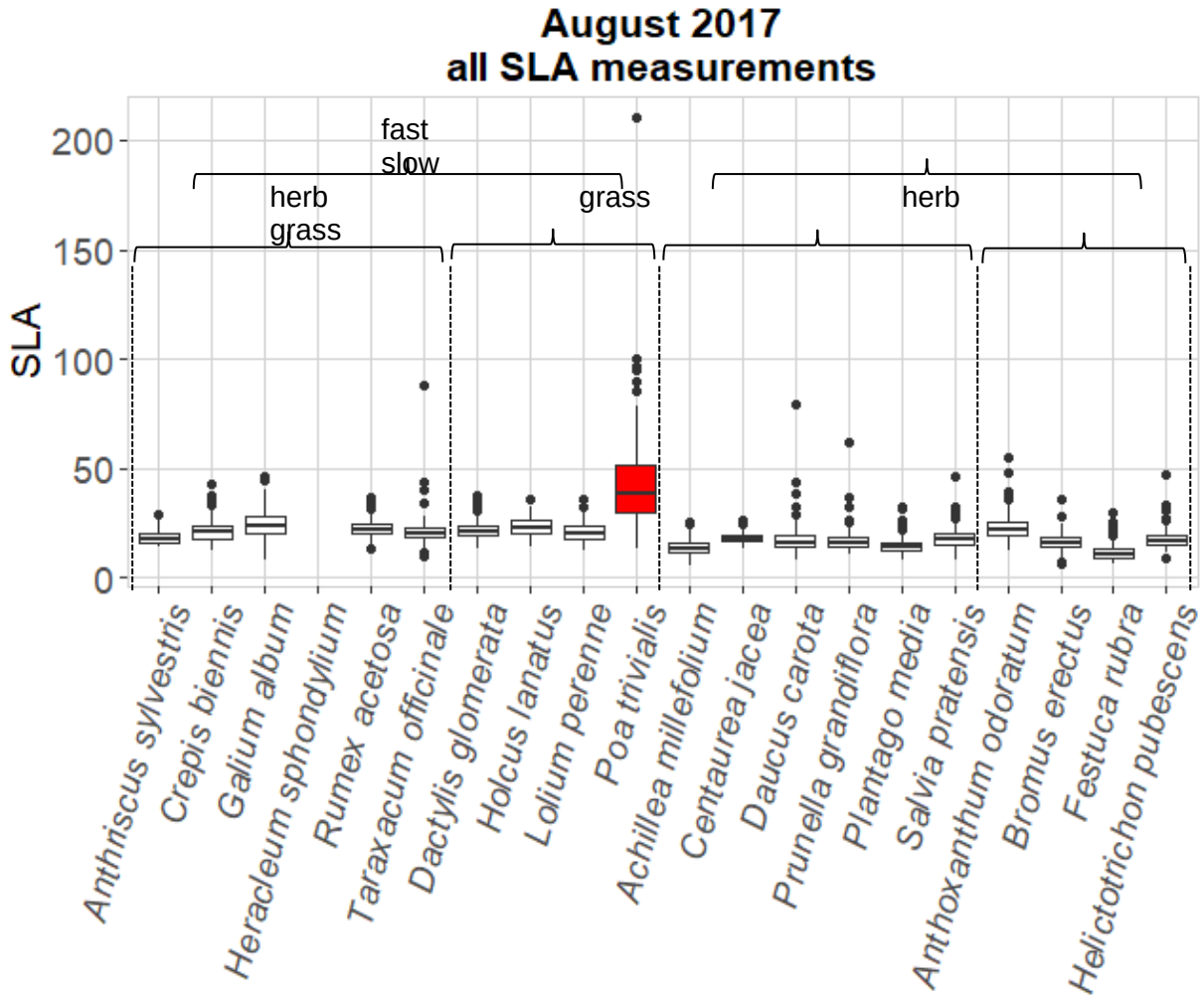


Figure S1 raw SLA measurements per species

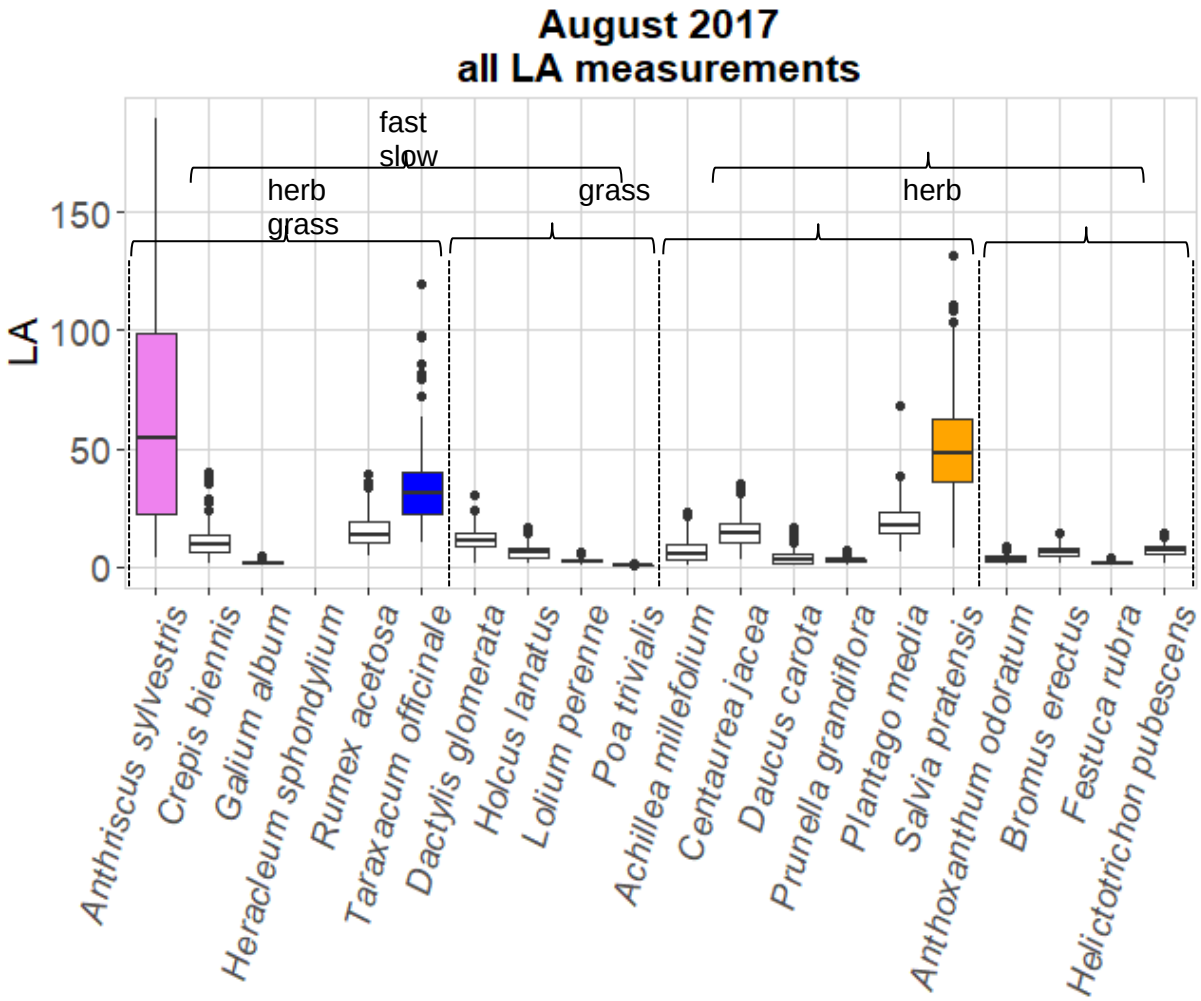


Figure S2 raw leaf area (LA) measurements per species

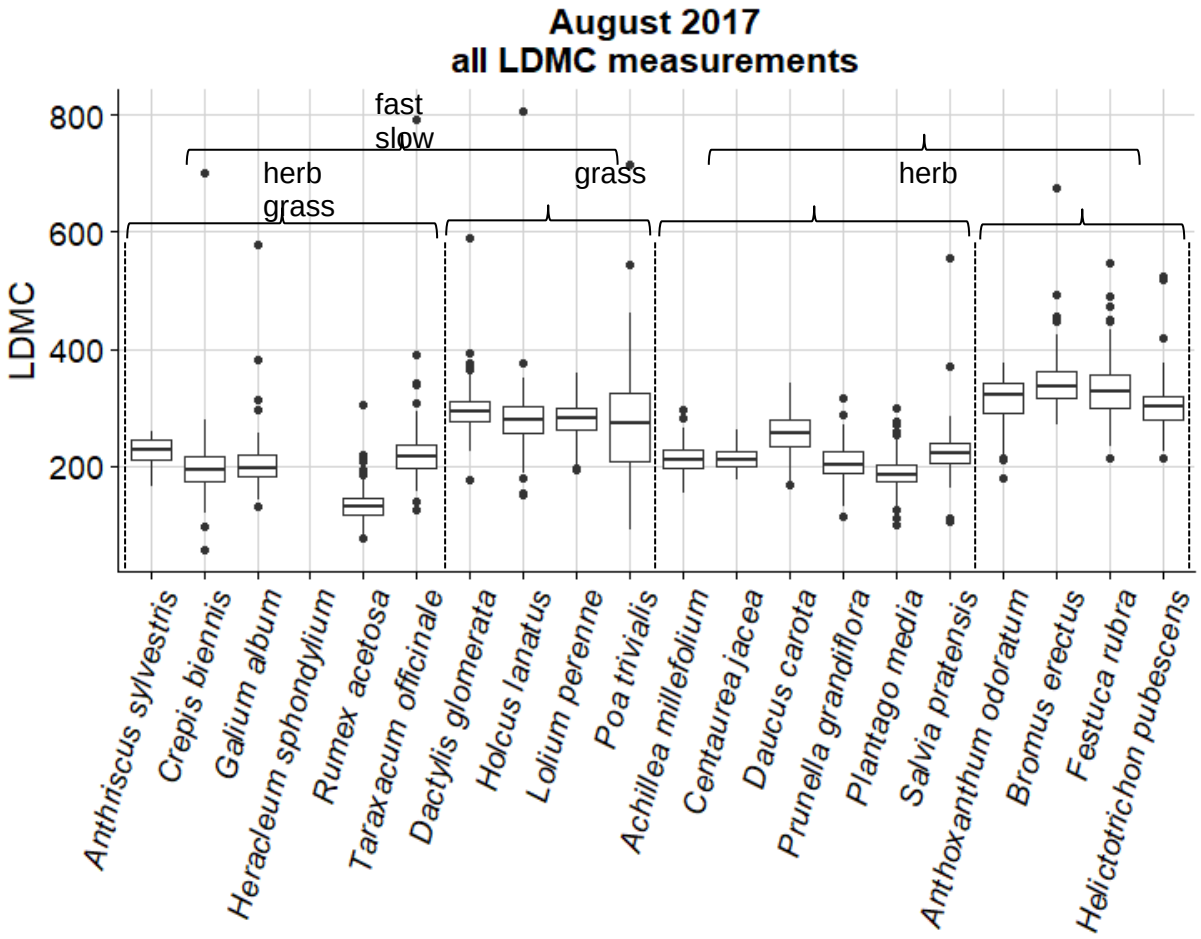


Figure S3 raw leaf dry matter content (LDMC) measurements per species

Table S1 Sample sizes per treatment group. In brackets the maximum possible minus Poa trivialis and Heracleum sphondylium. White: control plots, red: fungicide plots, blue: nitrogen plots, violet: fungicide + nitrogen plots.

SD	fast		mixed		slow	
1	8 (10-2)	8 (10-2)			10 (10)	10 (10)
	8 (10-2)	8 (10-2)			10 (10)	10 (10)
4	12 (20-4)	15 (20-4)	16 (20-1)	19 (20-1)	19 (20)	20 (20)
	14 (20-4)	14 (20-4)	18 (20-1)	17 (20-1)	19 (20)	18 (20)
8	22 (40-9)	24 (40-9)	33 (40-4)	28 (40-4)	38 (40)	33 (40)
	24 (40-9)	26 (40-9)	31 (40-4)	32 (40-4)	36 (40)	37 (40)
20					**Total:**	
					637 (800-80)	

Table S2 sample sizes per species. In brackets the maximum possible sample size.

species	**abbreviation**	**sample size**
Achillea millefolium	Am	32 (32)
Anthoxanthum odoratum	Ao	34 (36)
Anthriscus sylvestris	As	6 (44)
Bromus erectus	Be	36 (40)
Crepis biennis	Cb	32 (32)
Centaurea jacea	Cj	36 (36)
Daucus carota	Dc	26 (44)
Dactylis glomerata	Dg	48 (48)
Festuca rubra	Fr	36 (36)
Galium album	Ga	40 (44)
Holcus lanatus	Hl	36 (36)
Helictotrichon pubescens	Hp	40 (40)
Lolium perenne	Lp	32 (32)
Prunella grandiflora	Pg	40 (40)
Plantago media	Pm	48 (48)
Rumex acetosa	Ra	23 (36)
Salvia pratensis	Sp	48 (48)
Taraxacum officinale	To	44 (48)
Poa trivialis	Pt	0 (40)
Heracleum sphondylium	Hs	0 (40)

The four monocultures of *Heracleum sphondylium* had to be excluded due to a lack of enough suitable leaves to measure leaf traits. The four monocultures of *Poa trivialis* were excluded, because the *P. trivialis* SLA measurements were much higher than all the other SLA measurements and their variation much bigger (see Figure S1 – S3). The leaves were still very small and very often too young for SLA measurement. After removing *Poa trivialis* and *Heracleum sphondylium* 720 data points would have been possible of which 634 were realized. Most missing data are due to the species *Anthriscus sylvestris* and *Daucus carota*, which have not established very well and *Rumex acetosa*, which by the time of the measurement had died back, after having flowered.

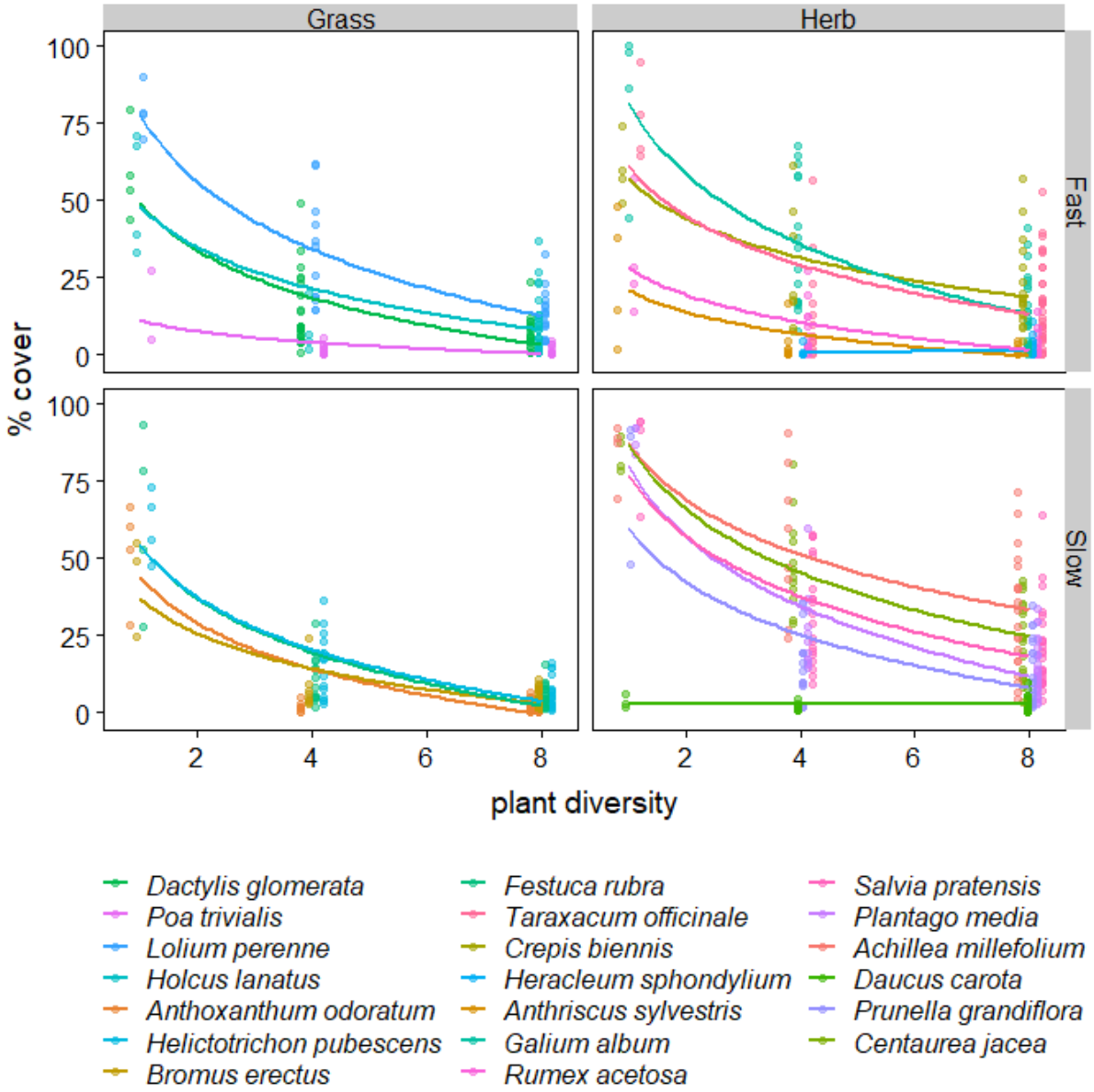


Figure S4 relationship between host plant abundance and plant species diversity for each species.

Table S3 full model results of the linear mixed effects model

fixed effects	Estimate	S.E.	X2	p-value
Intercept	4.945	0.890		
cover	0.233	0.102	5.141	0.023
SLA	0.698	1.343		marginal
CWM LDMC	0.216	0.108	3.949	0.047
functional group Herb	-0.965	1.173		marginal
SLA x functional group Herb	4.638	1.951	4.907	0.027
Fungicide			3.445	0.063
LDMC			2.126	0.145
ΔSLA			1.345	0.246
Nitrogen			0.864	0.353
ΔLA			0.842	0.359
CWM LA			0.246	0.62
ΔLDMC			0.132	0.717
Plant Diversity			0.114	0.735
LA			0.058	0.809
CWM SLA			0.057	0.811
ΔSLA x functional group			3.694	0.055
Nitrogen x SLA			1.252	0.263
Nitrogen x CWM SLA			3.493	0.062
SD x ΔLDMC			3.345	0.067
Nitrogen x ΔLA			3.721	0.054
Nitrogen x LDMC			2.169	0.141
Nitrogen x cover			1.631	0.202
Plant Diversity x Nitrogen			3.502	0.061
Plant Diversity x SLA			2.108	0.146
Nitrogen x Fungicide			1.801	0.18
Plant Diversity x Fungicide			1.771	0.183
CWM LDMC x functional group			1.879	0.17
CWM SLA x functional group			1.743	0.187
Nitrogen x ΔSLA			1.659	0.198
Fungicide x LDMC			1.223	0.269
CWM LA x functional group			0.866	0.352
Plant Diversity x CWM LA			0.79	0.374
Plant Diversity x LA			0.851	0.356
LDMC x functional group			0.5	0.48
Plant Diversity x CWM LDMC			0.424	0.515
Plant Diversity x LDMC			0.487	0.485
Fungicide x ΔSLA			0.4	0.527
Nitrogen x CWM LA			0.355	0.551
Nitrogen x LA			0.375	0.54
LA x functional group			0.38	0.538
ΔLA x functional group			0.299	0.584
Plant Diversity x CWM SLA			0.239	0.625
Nitrogen x ΔLDMC			0.189	0.664
Fungicide x LA			0.15	0.699
Fungicide x CWM LA			0.197	0.657
Plant Diversity x Nitrogen x Fungicide			0.206	0.65
Plant Diversity x cover			0.171	0.679
cover x functional group			0.208	0.648
Fungicide x CWM LDMC			0.146	0.703

Fungicide x ΔLA	0.152	0.696
ΔLDMC x functional group	0.145	0.704
Fungicide x SLA	0.135	0.713
Fungicide x cover	0.083	0.773
SD x ΔSLA	0.051	0.821
SD x ΔLA	0.048	0.827
Fungicide x ΔLDMC	0.037	0.846
Nitrogen x CWM LDMC	0	0.99
Fungicide x CWM SLA	0	0.995

Random effects	**Variance**	**S.D**
Plot	0.451	0.672
Composition	0.000	0.000
Species	6.555	2.560
Block	0.017	0.130

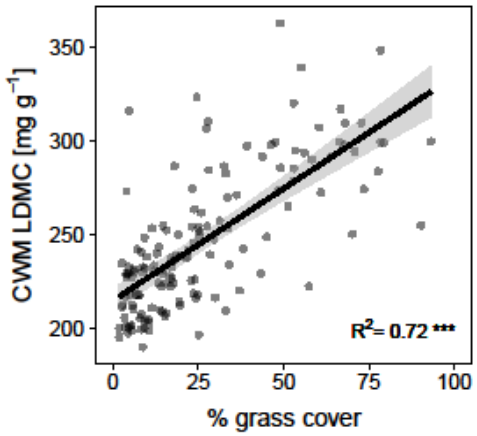


Figure S5 Strong correlation (pearson correlation, $R^2 = 0.72$, $p < 0.001$) between community weighted mean leaf dry matter content (CWM LDMC) and the abundance of grass species.

Supplementary 7: model with grass cover instead of CWM LDMC

Table S4 Results of the mixed effects model explaining fungal infection with plot, species and population level trait values as explanatory variables in addition to species abundance (% cover) and treatment variables) In this model the community weighted mean of LDMC was replaced by the summed abundances of all grass species. Insignificant terms are shown in grey and with lighter colored background. Abbreviations: community weighted mean (CWM), leaf dry matter content (LDMC), specific leaf area (SLA), leaf area (LA)

fixed effects	**Estimate**	**S.E.**	**X2**	**p-value**	
Intercept	5.229	0.954			
grass abundance	0.717	0.175			
SLA	0.561	1.431			
functional group (Herb)	-1.528	1.261			
SLA x functional group (Herb)	4.758	2.082	4.572	0.033	*
grass abundance x functional group (Herb)	-1.142	0.333	11.541	0.001	** *
Fungicide	-0.439	0.219	3.971	0.046	*
Nitrogen			1.511	0.219	
Plant Diversity			1.176	0.278	
CWM SLA			0.185	0.667	
CWM LA			2.155	0.142	
cover			0.351	0.554	
LA			0.629	0.428	
LDMC			2.166	0.141	
ΔSLA			2.351	0.125	
ΔLA			1.279	0.258	
ΔLDMC			0.008	0.930	

Random effects	**Variance**	**S.D.**
Plot	0.424	0.651
Composition	0.000	0.000
Species	7.536	2.745

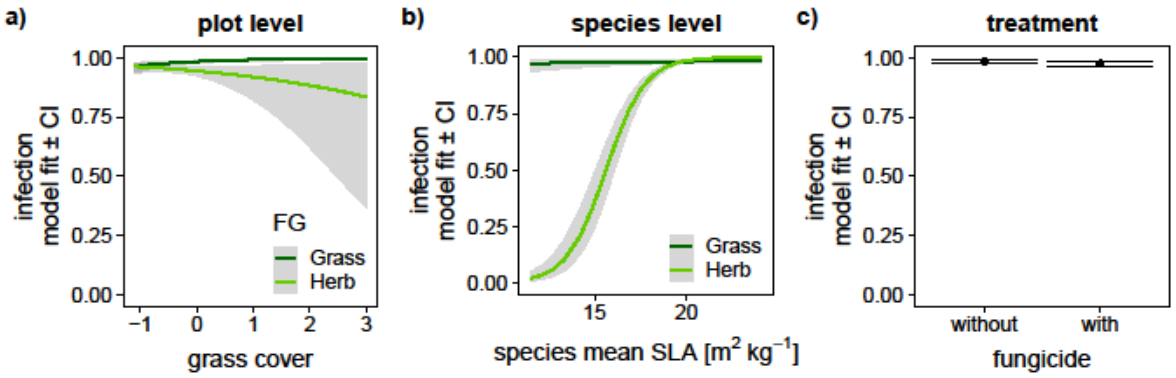


Figure S6 Model prediction plots for the lmer with grass cover instead of community leaf dry matter content

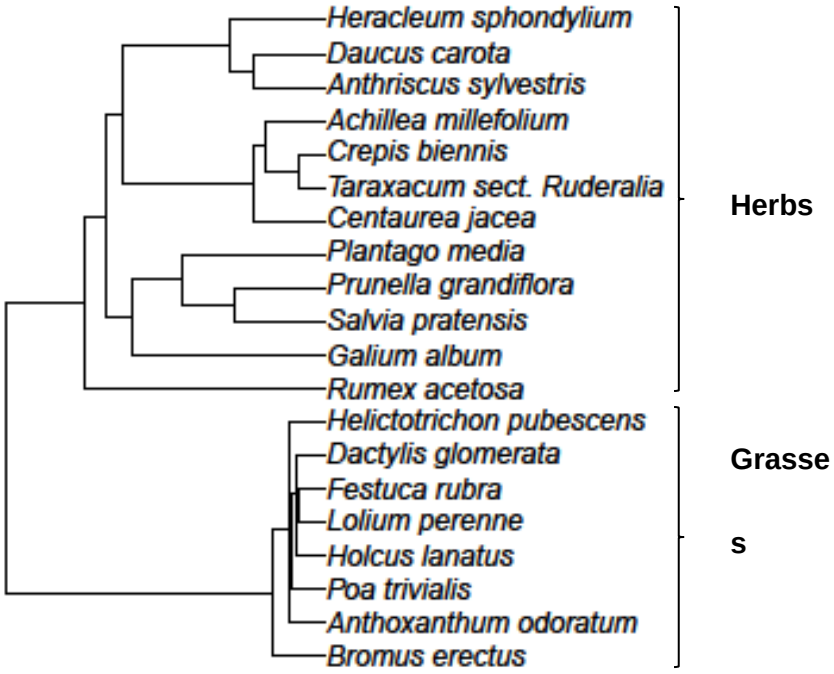


Figure S7 Phylogeny of the PaNDiv experiment species pool. This is a subset of the Daphne phylogeny (Durka and Michalski 2012).[1]

[1] Durka, W. & Michalski, S.G. (2012). Daphne: a dated phylogeny of a large European flora for phylogenetically informed ecological analyses. *Ecology*, 93, 2297.

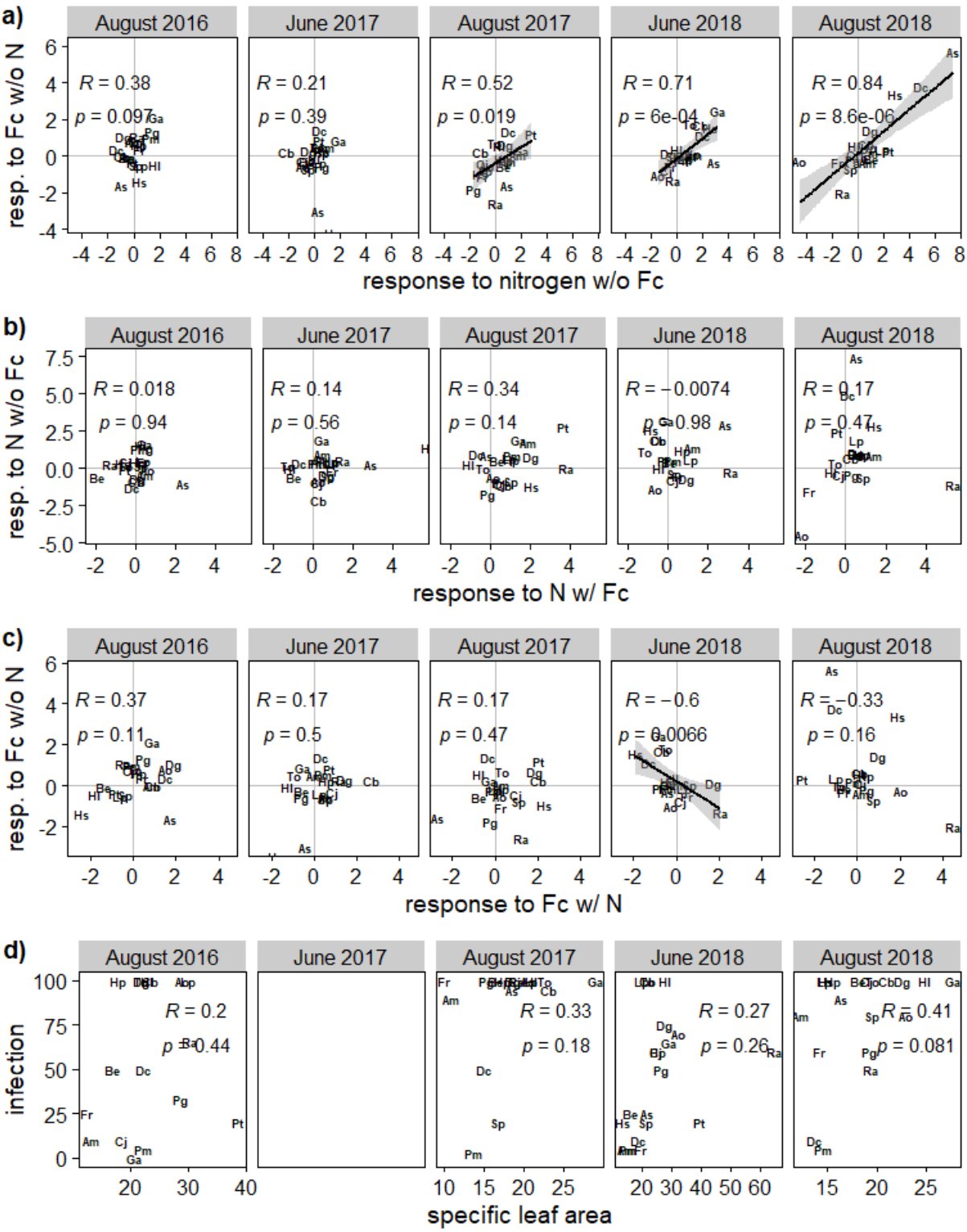
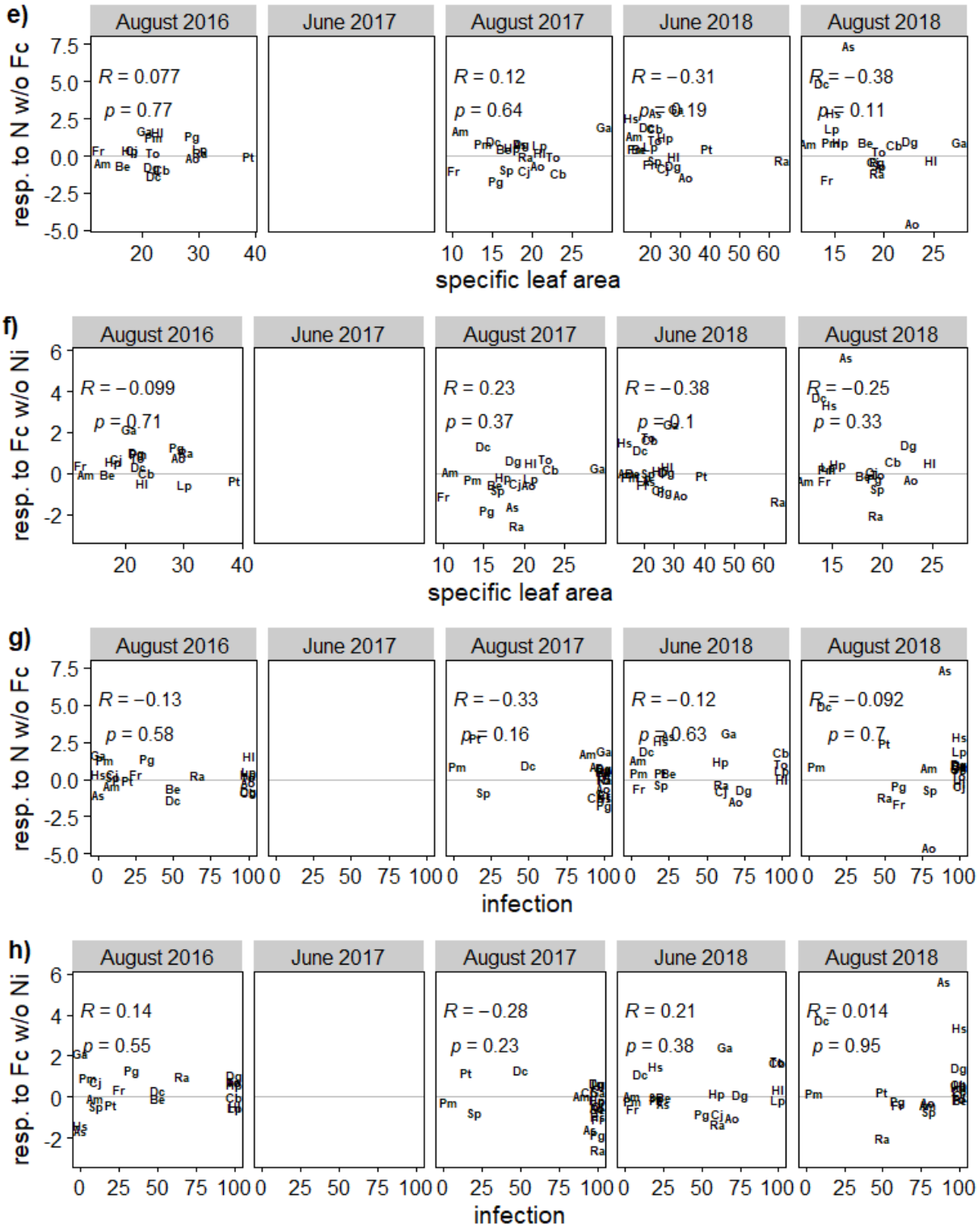


Figure S8 detailed correlations from Figure 2. Correlations between a) biomass response to fungicide (Fc, pathogen resistance) without nitrogen enrichment and response of biomass to nitrogen (N, resource acquisition) in the presence of fungal pathogens, b) the biomass response to nitrogen with and without fungicide treatment to show how pathogens change resource acquisition, c) the biomass response to fungicide with and without nitrogen enrichment to show how resources alter pathogen resistance, and d) specific leaf area (SLA, as a measure of growth strategy) and infection (as a measure of defense) across different time points of the experiment. All data was measured in the monocultures of the species. Note that the range of the x-axis varies between the years when the x-axis represents SLA, as the range in SLA is much larger in June 2018, than in the other sampling periods. The species abbreviations are given in Table S2.


e)
August 2016
June 2017
August 2017
June 2018
August 2018
resp. to N w/o Fc
R = 0.077
p = 0.77
R = 0.12
p = 0.64
R = −0.31
p = 0.19
R = −0.38
p = 0.11
specific leaf area
f)
resp. to Fc w/o Ni
R = −0.099
p = 0.71
R = 0.23
p = 0.37
R = −0.38
p = 0.1
R = −0.25
p = 0.33
specific leaf area
g)
resp. to N w/o Fc
R = −0.13
p = 0.58
R = −0.33
p = 0.16
R = −0.12
p = 0.63
R = −0.092
p = 0.7
infection
h)
resp. to Fc w/o Ni
R = 0.14
p = 0.55
R = −0.28
p = 0.23
R = 0.21
p = 0.38
R = 0.014
p = 0.95
infection

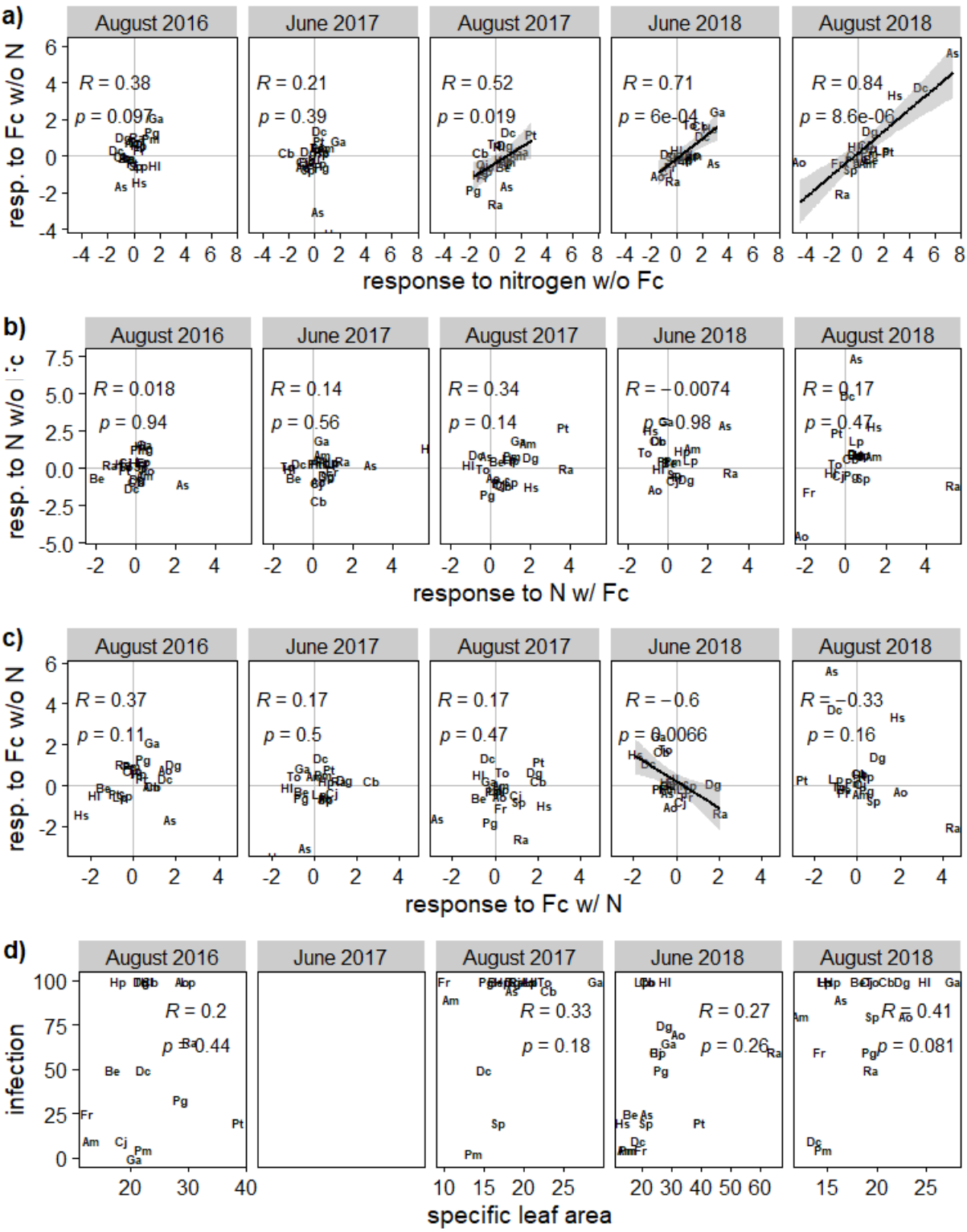


Figure S8 continued. Detailed correlations from Figure 2. Correlations between e) biomass response to nitrogen (N, resource acquisition) in the presence of fungal pathogens and SLA (growth strategy), f) biomass response to fungicide (Fc, pathogen resistance) without nitrogen enrichment and SLA (growth strategy), g) biomass response to nitrogen (resource acquisition) in the presence of fungal pathogens) and infection (defense) and h) biomass response to fungicide (pathogen resistance) without nitrogen and infection (defense) across different time points of the experiment. All data was measured in the monocultures of the species. Note that the range of the x-axis varies between the years when the x-axis represents SLA, as the range in SLA is much larger in June 2018, than in the other sampling periods. The species abbreviations are given in Table S2.

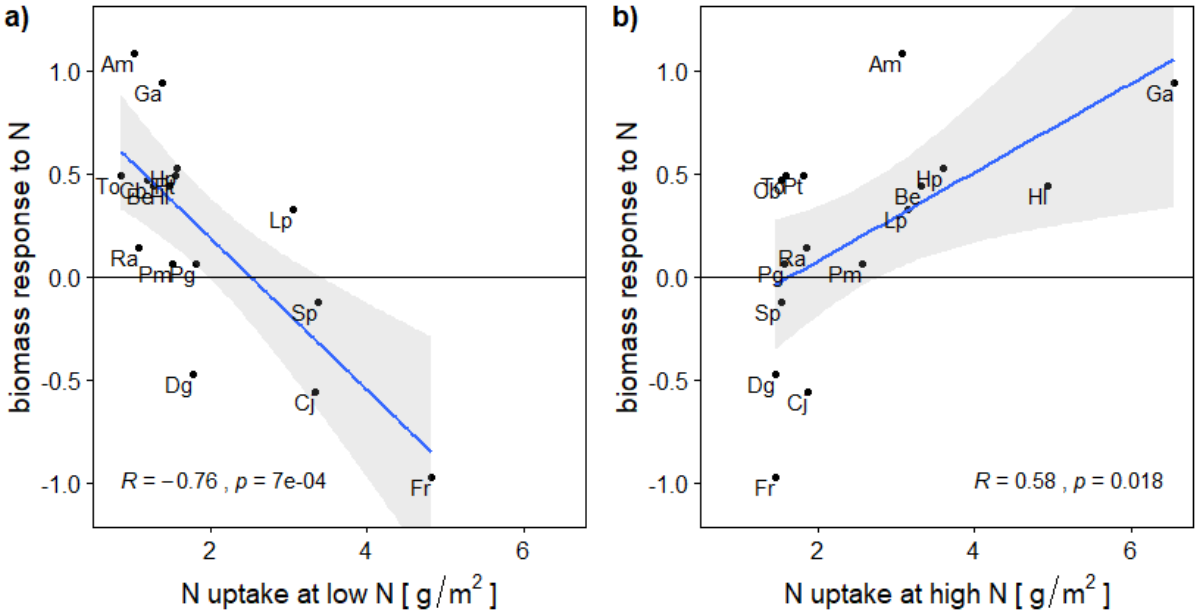


Figure S9 Biomass response to nitrogen enrichment (log response ratio) is large for species which are a) not good at taking up nitrogen at low ambient nitrogen levels (without N fertilization), but b) good at taking up nitrogen at high ambient nitrogen levels (with N fertilization). Data source: Walde (2019)

Chapter 4
Consistent biodiversity effects across functions

Seraina L. Cappelli, Noémie A. Pichon, Tosca Mannall, Eric Allan

ABSTRACT

Effects of biodiversity on ecosystem functioning can occur through a variety of different mechanisms, which can be broadly categorized into selection and complementarity effects. Complementarity effects occur when most species change their functioning in species rich communities, while selection effects quantify the extent to which functioning is driven by a few species. Selection effects can be divided into effects occurring due to interspecific abundance shifts (dominance effects) or due to intraspecific shifts in functioning (species changing functioning but not abundance in diverse communities). Many studies have calculated complementarity and selection effects for biomass production, but we know much less about their importance for other ecosystem functions. We also know little about how the diversity effects of different functions relate to each other, i.e. if the same or different species drive different functions, or whether diversity effects are context dependent. We used data from a large grassland experiment (PaNDiv) in which species diversity, functional composition, nitrogen enrichment and fungal pathogen exclusion were factorially manipulated. We calculated complementarity and selection effects for five different ecosystem functions (plant aboveground biomass, herbivory, pathogen infection and specific leaf area and leaf dry matter content as proxies of nutrient cycling), using bipartite and tripartite partitions. We observed positive complementarity effects across all functions, suggesting that positive diversity effects are typically driven by multiple species. Intraspecific selection effects were negative for all functions, showing that species converge in their functioning, particularly because species with low monoculture functioning increased their functioning in mixture. Despite these overall consistencies, diversity effects on the five functions were not correlated, suggesting different species drive the different functions, and environmental drivers had varying impacts on the functions. This indicates, that different underlying mechanisms can result in similar overall patterns in diversity effects between functions. The variation in underlying mechanisms and species driving different functions suggest that high diversity is needed for the simultaneous provision of multiple ecosystem functions.

INTRODUCTION

The diversity of primary producers affects many different ecosystem functions (Cardinale et al. 2006; Cardinale et al. 2012) through a variety of mechanisms. A more mechanistic understanding of biodiversity-functioning relationships is of fundamental interest and is important to predict and manage functional consequences of biodiversity declines. Biodiversity-ecosystem functioning research groups biodiversity mechanisms into two main categories: complementarity and selection effects, which together sum up to the **net effect** of diversity (additive partitioning sensu Loreau and Hector 2001). The **complementarity effect** summarizes the extent to which species increase or decrease their functioning in mixtures. A large complementarity effect occurs if most species shift their functioning in the same direction. Complementarity effects can be explained by several underlying processes: for example, more efficient resource partitioning or reduced infection from specialist pathogens in diverse communities (for an extensive review of causes of complementarity effects in biomass production see Barry et al. 2019). Alternatively, certain species could contribute disproportionality to the provision of an ecosystem function, leading to **selection effects**. Selection effects are positive if mixture functioning is driven by species with high functioning in monoculture and negative if species with low functioning in monoculture increase their functioning in mixtures (or if high functioning species reduce their functioning). Positive complementarity effects for biomass production is generally observed in biodiversity experiments, however selection effects can vary from slightly positive to negative (Cardinale et al. 2011). This means that positive biodiversity-productivity relationships are driven by increased biomass production in many species. However, we have much less information on the mechanisms by which biodiversity affects other ecosystem functions and therefore how often other functions are driven by few or many species.

It is possible to partition diversity effects into selection and complementarity for other functions. If individual species contributions to function can be calculated, for instance in the case of pathogen infection or herbivory, then diversity effects can be partitioned in the same way. In addition, Grossiord et al. (2013) showed that complementarity and selection effects can be calculated even if the contribution of single species to community functioning cannot be measured, by using proxies for functioning. Functional traits are good proxies of many ecosystem functions (Garnier et al. 2004; Lavorel and Grigulis 2012; Laughlin 2011) and the additive partitioning framework can

be used to analyze community weighted means traits to reveal how diversity affects ecosystem functions related to these traits (Roscher et al. 2018b; Grossiord et al. 2013). For example, Grossiord et al. (2013) showed that water use efficiency in tree communities, using leaf carbon isotope composition as a proxy, had varying net and complementarity effects, but no selection effect. In other words, increased water use efficiency in diverse communities was not driven by single species but by changes in water use efficiency across many species. The diversity interaction modelling approach provides an alternative way to calculate effects analogous to selection and complementarity (Kirwan et al. 2009; Connolly et al. 2013; Dooley et al. 2015; Brophy et al. 2017). However, it requires the estimation of a large number of parameters, especially at high diversity. Calculating selection and complementarity for several different functions measured in the same communities would allow a comparison of diversity mechanisms across functions.

Selection effects can arise from two different processes. Firstly, one or a few species with high (or potentially also with low) functioning can dominate the mixtures and drive community functioning. This is a zero-sum dynamic in which high abundance of one species comes at the cost of decreased abundance of other species. Such interspecific abundance shifts are likely driven by competition: for example, a positive selection effect for herbivory could arise if a species which is highly palatable to herbivores is simultaneously a good competitor (e.g. Kempel et al. 2011) and dominates species mixtures. Secondly, certain species may shift functioning without shifting abundance and provide most of the functioning in mixtures without affecting the functioning of other species, i.e. intraspecific shifts in function. In this case positive selection effects would indicate divergence in functioning in mixtures (high functioning species increase and/or low functioning decrease) and negative selection effects indicate convergence in functioning between species. For example spillover of pathogens in mixtures can lead to more similar levels of infection between species (Power and Mitchell 2004), which could result in a negative selection effect for pathogen infection, if the species with lowest monoculture infection increase in infection the most. As abundance shifts and changes in functioning of single species can potentially have opposing effects on the selection effect, it is important to quantify the two mechanisms separately (Fox 2005, Figure S2), which cannot be achieved by the diversity interaction approach (Kirwan et al. 2009). To our knowledge this tripartite partitioning has mainly been applied to biomass production and rarely to other ecosystem functions (but see Pires et al. 2018).

If the same species are involved in driving different functions then we would expect biodiversity mechanisms for different functions to correlate (Sullivan et al. 2007). However, if different species promote different functions, diversity effects should not be correlated. Very often sets of traits (e.g. Garnier et al. 2001; Wright et al. 2004) and sets of ecosystem functions (e.g. Lavorel and Grigulis 2012; Schädler et al. 2003) correlate. For example, specific leaf area (SLA) is negatively correlated with leaf dry matter content (LDMC, Garnier et al. 2001), herbivory is positively correlated with decomposition (Schädler et al. 2003), or SLA is positively correlated with pathogen infection (Cappelli et al. 2019). These correlations in the traits and in the ecosystem functions would suggest that biodiversity effects should correlate across functions, however, this has not been tested.

It is also likely that diversity effects are context dependent and understanding this context dependency is critical to predict when diversity is an important driver of functioning. Diversity effects can vary depending on which species or species groups occur in the community (Wagg et al. 2017; Marquard et al. 2009). For example, legumes are known to increase complementarity effects (e.g. Marquard et al. 2009). And high functional diversity may lead to stronger positive complementarity and negative selection effects (Wagg et al. 2017; Roscher et al. 2012). However, functional diversity can have complex effects on complementarity and selection effects depending on other community characteristics (Roscher et al. 2012; Isbell et al. 2008; Wagg et al. 2017) and effects of the functional composition of the species pool have rarely been considered.

Further, the abiotic and biotic environment can shape diversity effects. Pires et al. (2018) showed that less frequent but higher intensity rainfall reduced complementarity effects on decomposition and Hector et al. (2012) showed that selection effects for biomass depend on the soil and on the water availability. Studies have also assessed how nutrient availability alters diversity effects on biomass production. Many suggest that nutrient enrichment reduces complementarity effects (Jarchow and Liebman 2012; Roscher et al. 2016; Siebenkaes et al. 2016; Craven et al. 2016, but see Yin et al. 2018; Wacker et al. 2009) likely by removing facilitative interactions between species (Roscher et al. 2016) and increasing dominance effects (Jarchow and Liebman 2012; Siebenkaes et al. 2016; Yin et al. 2018). Further, it is often suggested that reduced impact by natural enemies at high diversity could be an underlying mechanism

explaining positive complementarity in biomass (Eisenhauer 2012; Maron et al. 2011; Schnitzer et al. 2011). However, this has not been tested by calculating complementarity when enemies are excluded. Hardly any studies have calculated context dependency in biodiversity mechanisms for functions other than biomass meaning we do not know whether other functions show similar levels of context dependency or not.

This study analyses data from an experiment manipulating plant species richness, plant functional composition (community mean specific leaf area [SLA]), nitrogen addition and foliar fungal pathogen exclusion. We calculate biodiversity mechanisms across three functions (aboveground biomass, herbivory and pathogen infection) and two community mean traits as proxies for nutrient cycling related functions (SLA and leaf dry matter content). We manipulate the mean SLA of species compositions. Within a given species composition, species can shift in abundance and intraspecific trait values, so we can test how realized community weighted mean SLA changes with diversity and how this depends on nitrogen, pathogens and the functional composition of the species pool. We address the following questions: are the same biodiversity mechanisms important for different functions? Do these mechanisms correlate, suggesting similar processes and species driving effects of diversity on different functions? How strong is context dependency in biodiversity mechanisms and what are the main factors determining the strength of different mechanisms?

MATERIALS AND METHODS

Experiment

The study was conducted in the PaNDiv experiment, located on an extensively managed grassland in the Swiss lowlands. The experiment consists of 336 plots and manipulations of plant diversity (1, 4, 8, 20 species), functional composition and diversity (a gradient of sown SLA was created by grouping species into fast [high SLA] and slow [low SLA] growing species and creating plots with only fast, only slow or a mix of growth strategies), nitrogen enrichment in the form of urea (0, 100 $kg.ha^{-1}.y^{-1}$) and enemy exclusion with foliar fungicide (Score Profi by Syngenta Agro AG, 24.8 % difenoconazole and Ortiva by Syngenta Agro GmbH, 22.8 % azoxystrobin) were factorially manipulated (see Pichon et al. (2019) for a detailed experiment description). Species combinations were randomly selected from the respective species pool (i.e. fast, slow or mixed) and the experiment contained 84 unique species compositions. The plots were arranged in four blocks and all species compositions (diversity x sown

SLA) occurred once per block. Each composition received the four combinations of fungicide x nitrogen treatments, while the particular treatment each composition received was randomly allocated to each block. The plots were separated by 1m wide stripes of grass. To maintain species compositions, the experiment was weeded three times per year. The whole experiment was mown twice a year to mimic the management of an extensively managed grassland in the area.

Ecosystem function measurements

We measured plant species abundances and several different ecosystem functions or function related traits. In total we measured three functions: biomass production, herbivory, pathogen infection and two traits, SLA and LDMC, as proxies of nutrient cycling related functions (Laughlin 2011; Schädler et al. 2003). For simplicity we refer to all functions and traits as “functions”.

We visually estimated % cover of all the sown plant species, the bare ground and the weeds in all plots. The sum of all cover values per plot could exceed 100%. Cover was estimated twice a year (two “**sampling periods**”) between 2016 and 2018, once at the beginning of June and once at the beginning of August. In 2016 we only used the August data, because the field had not fully established before then. The cover values of the target plant species (calculated relative to total target cover, i.e. without the weeds and the bare ground, so that proportional abundance of target species sums to 1) were transformed to relative values and were used as measures of the species’ **abundances**. Shortly after the % cover measurements, we measured **biomass** in all plots in two 50cm x 20cm areas per plot. The samples were dried at 60°C for at least 24h, before weighing and we used the mean biomass of the two measurements. The % cover data were then used to calculate the biomass produced by each species. We multiplied the total biomass per plot by the proportional abundance of each plant species to calculate species specific biomass. Here abundance was proportional to total vegetation cover, i.e. including the weeds, so that the total biomass of all target species does not include the weed abundance. Weed abundances were low, except in the first year (weed cover was 31.9 ± 1.19 % in August 2016 but 7.3 ± 0.34 % across the other sampling periods). To check that these estimates of species specific biomass were accurate we also sorted the biomass from 84 plots (2 samples per plot) in June 2017 and from 216 plots (1 sample per plot) in August 2017. The estimated biomass values per species were close to the sorted biomass values in June, $R^2 = 0.87$ (Figure S1). The correlation was less strong in August ($R^2 = 0.4$) presumably because sorting

only one biomass sample per plot does not account for spatial variation in species abundances. The strong correlation between predicted and observed species biomass means we are confident that our approach is suitable for estimating species biomasses.

We measured the traits **specific leaf area (SLA)** and **leaf dry matter content (LDMC)** in August 2017, simultaneously as the biomass harvest, in a subset of 200 plots (all 80 monocultures and 60 4 and 8 species plots) following the protocol of Garnier et al. (2001). We collected five fully developed, healthy and sun exposed leaves per species per plot. Sometimes we could not find enough suitable leaves and were forced to take fewer leaves (out of 1600 samples possible, in 141 cases only 1-4 leaves were sampled and in 238 cases no leaves could be found). The leaves were hydrated with deionized water overnight, before the leaf area and the fresh weight were measured, for the calculation of SLA and LDMC. Before measuring the dry weight, the leaves were dried at 60°C for at least 48h (Garnier et al. 2001). We estimated **pathogen infection** on 10 plants in the central square meter of the plots in 2016, 2017 and 2018 in September when infection intensity is highest (Rottstock et al. 2014). In 2018 we also estimated infection in June. Infection was measured as the proportion of individuals with signs of infection. If the central square meter had too few individuals, we scored additional plants from the rest of the plot and if less than 10 individuals could be found in the whole plot, the proportion was calculated based on all individuals found. **Herbivory** was assessed at the end of May and August 2018. Five individuals of each target species were haphazardly selected from the central square meter of each plot and five leaves per individual were assessed for damage. Leaves were selected from the middle tier of each individual, excluding juvenile and senescing leaves and we calculated the proportion of damaged leaves per species per plot. We calculated community level fungal infection, insect herbivory, SLA and LDMC as community weighted means. The contribution of each species to the community level function is therefore proportional to its abundance.

Additive partitioning

We calculated net, selection and complementarity effects using the additive partitioning framework of Loreau and Hector (2001) for biomass and the adjusted framework of Grossiord et al. (2013) for fungal infection, insect herbivory, SLA and LDMC. We used equal abundances and monoculture values (from the corresponding nitrogen and fungicide treatment) as the null hypothesis, expected values. We then further

partitioned the selection effects of all functions into intra- and interspecific selection effects using the tripartite partitioning of Fox (2005). For the functions other than biomass we followed the same logic as for the bipartite partitioning of Grossiord et al. (2013), which we extended to a tripartite partition (see Supplementary materials). As the different functions were measured in different units we scaled all functions between 0 and 1, per sampling period, before calculating additive partitioning. For a detailed description of the calculations see Supplementary Methods.

We excluded *Heracleum sphondylium* and *Anthriscus sylvestris*, because of their poor establishment. Further, when a species was missing from a plot, we could not measure traits or enemy damage. The missing values were replaced with the monoculture values of the same species in the same treatment, which leads to conservative estimates of selection and complementarity effects. When monoculture values were missing (2 out of 400 samples), we modelled them based on the other monoculture values, including species identity, sampling period and fungicide and nitrogen treatment as explanatory variables. Zero monoculture values were possible for herbivory and fungal infection and these cause infinitely big complementarity and selection effects. To avoid this, zero values were set to half of the observed minimum function (detailed description in Supplementary Methods).

A caveat of the additive partitioning approach is the importance of the monoculture values, as they are included in all measures of net, complementarity and selection effects. Ideally, we would have replicates of all the monocultures, to have more precise measurements and reduce the impact of random variation. However, because of the high number of species and treatment combinations we could not replicate the monocultures.

Analysis

We first analyzed the overall complementarity, intra and interspecific effects for each variable. All effects on biomass, pathogen infection and herbivory were log transformed, keeping the original sign, to achieve a normal distribution. For SLA and LDMC residuals were normally distributed and variance was homogenous, so these values were not transformed. We then constructed linear mixed effects models including all the treatment variables (nitrogen, fungicide, sown SLA, sown mean pairwise distance in SLA and plant diversity). We excluded interactions between the treatment variables in the analysis even though our experiment design would allow the

full four-way interaction to be tested, to keep our models relatively simple, as we were fitting a large number of different models. We therefore tested how our treatment variables alone change diversity effects. We additionally included random slopes of fungicide and nitrogen against sampling period and random intercepts for the interactive effect of sampling period and plant composition, to account for seasonal effects in these treatments. We simplified the random effect structure using likelihood ratio tests to compare models with and without particular random terms, however, we retained block, plot, species composition and sampling period in all models. Using these mixed effect models, we first calculated the values for each biodiversity effect by fitting intercept only models, using the chosen random structure per effect. We then tested for context dependency in complementarity, intra and interspecific selection effects by examining the fixed effects. Fixed effect structures were simplified by progressively excluding non-significant effects.

Selection effects can be driven by changes in species with either high and/or low monoculture functioning, e.g. a negative selection effect could arise if species with low monoculture functioning increase their functioning in mixtures and/or because species with high monoculture functioning reduce functioning in mixtures. To better visualize the overall intra and interspecific selection effects we plotted relationships between the mean monoculture function per species, across treatments and seasons (scaled relative to the mean of all monocultures to show species with above or below monoculture functioning), and the mean change in function between monocultures and mixtures (ΔRF_i). We did this for both interspecific changes in function and intraspecific changes. We have two plant groups in the species pool of the experiment, which differ significantly in their LDMC and in their fungal infection: grasses and (non-leguminous) herbs (Figure S9c-d). To visualize, the extent to which observed diversity mechanisms are driven by differences in these two groups, we additionally plotted the relationships for grasses and herbs separately.

We also looked at how the effects correlated with each other across the functions. We calculated the mean of net, complementarity, intraspecific selection and interspecific selection effects across all sampling periods (with the untransformed values), because we did not measure all the functions for all the sampling periods. The mean values where then correlated with each other using Pearson correlation coefficients.

RESULTS

Intercepts

The net effect was negative for SLA, LDMC, herbivory and pathogen infection and positive for biomass, meaning that the mixtures yielded more biomass and had lower infection, herbivory and trait values than expected based on the monocultures (Figure 1). We observed positive or neutral complementarity effects for all functions. Positive complementarity for biomass, pathogen infection and SLA, means that, on average, species had higher than expected functioning in polycultures (Figure 1). Positive complementarity effects were higher when more species increased their functioning in mixtures. For pathogen infection in some cases a minority of the species were driving positive complementarity effects in mixtures (Figure S4).

Selection effects were negative across all functions (Figure 1). We decomposed the selection effect into the contribution of inter- and intraspecific shifts. In the case of the traits negative selection effects were mainly due to interspecific abundance shifts (Figure 1, Figure 2), and to a lesser extent due to intraspecific shifts (Figure 1, Figure 3). For herbivory and pathogen infection it was the opposite and interspecific shifts were more important (Figure 1). This means that species which had below average monoculture enemy damage had more damage in the mixtures and species with above average monoculture enemy damage had less damage in the mixtures (Figure 1, Figure 3). There were contrasting intra and interspecific selection effects on biomass: species with high monoculture biomass increased in abundance and those with low monoculture biomass decreased in the mixtures (positive interspecific selection effect, Figure 1, Figure 2). However, low biomass species increased their biomass per area (negative intraspecific selection effect, Figure 1, Figure 3). Intraspecific shifts outweighed interspecific shifts which led to a negative selection effect in total. Overall, the species which dominated the mixtures were those with high monoculture biomass and to some extent those with low monoculture trait values, monoculture pathogen infection or herbivory did not predict dominance in mixture (Figure 3, Figure 2).

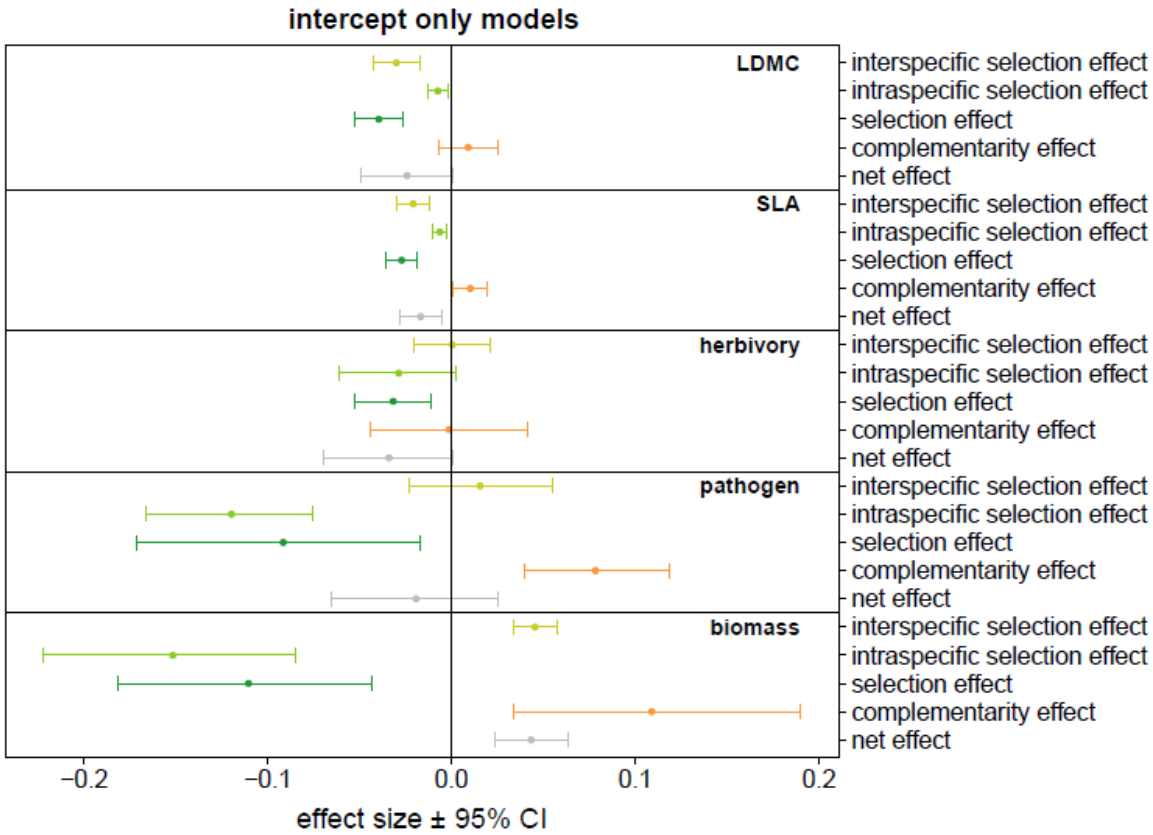


Figure 1 Intercept only models of all additive partitioning measures for all the functions. The data for the herbivory, pathogen and biomass models were log transformed for the analysis. The estimates and the upper and lower boundaries of the confidence intervals were back-transformed to show values on the original standardized scale, which is why the CI bars are asymmetric. Details about how different species contribute to the inter- and intraspecific selection effects of the different functions can be found in Figure 2 and Figure 3.

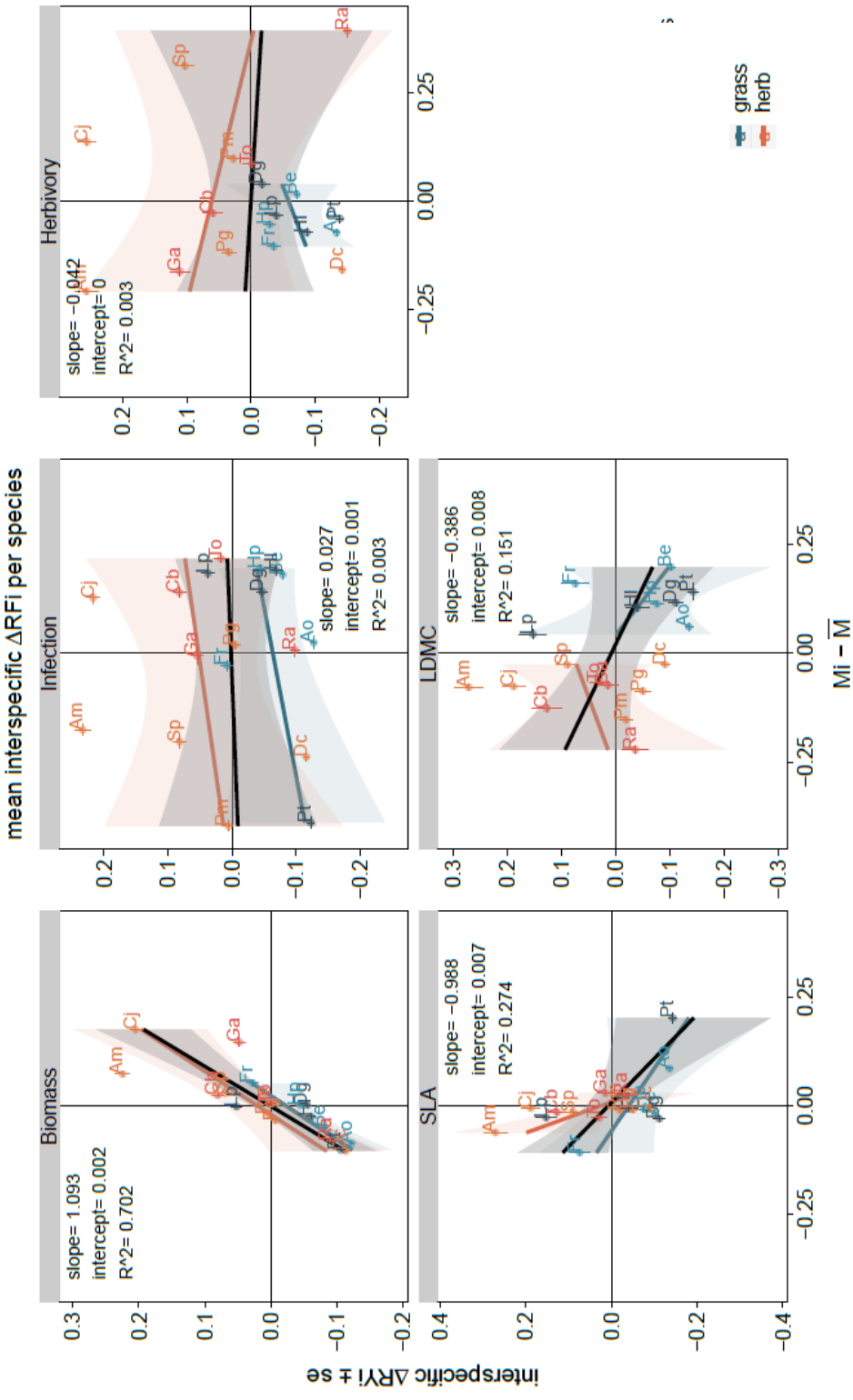


Figure 2 Overall interspecific effects per function. The relationship between mean monoculture function per species across the sampling periods and treatments (scaled to the mean monoculture function) and abundance shift in polyculture (mean interspecific ΔRF_i). Relationship is shown across all species (black) and across herbs (red) and grasses (blue) separately. Species with high monoculture biomass and to some degree with low specific leaf area (SLA) and low leaf dry matter content (LDMC) dominated the mixtures. Monoculture pathogen infection and herbivory did not greatly impact abundance. The negative relationship between ΔRF_i and the monoculture value in the case of LDMC was driven by the dominance of the herbs, which had lower LDMC than the grasses. Within the herbs and to some degree also within the grasses the ones with high LDMC were the species which had higher ΔRF_i and therefore able to increase in abundance more than the ones with lower ΔRF_i. The positive, neutral and negative correlations are reflected in positive, zero and negative interspecific selection effects in the given functions (Figure 1). Slopes, intercepts and R^2 for the relationship across all species are given in the plots.

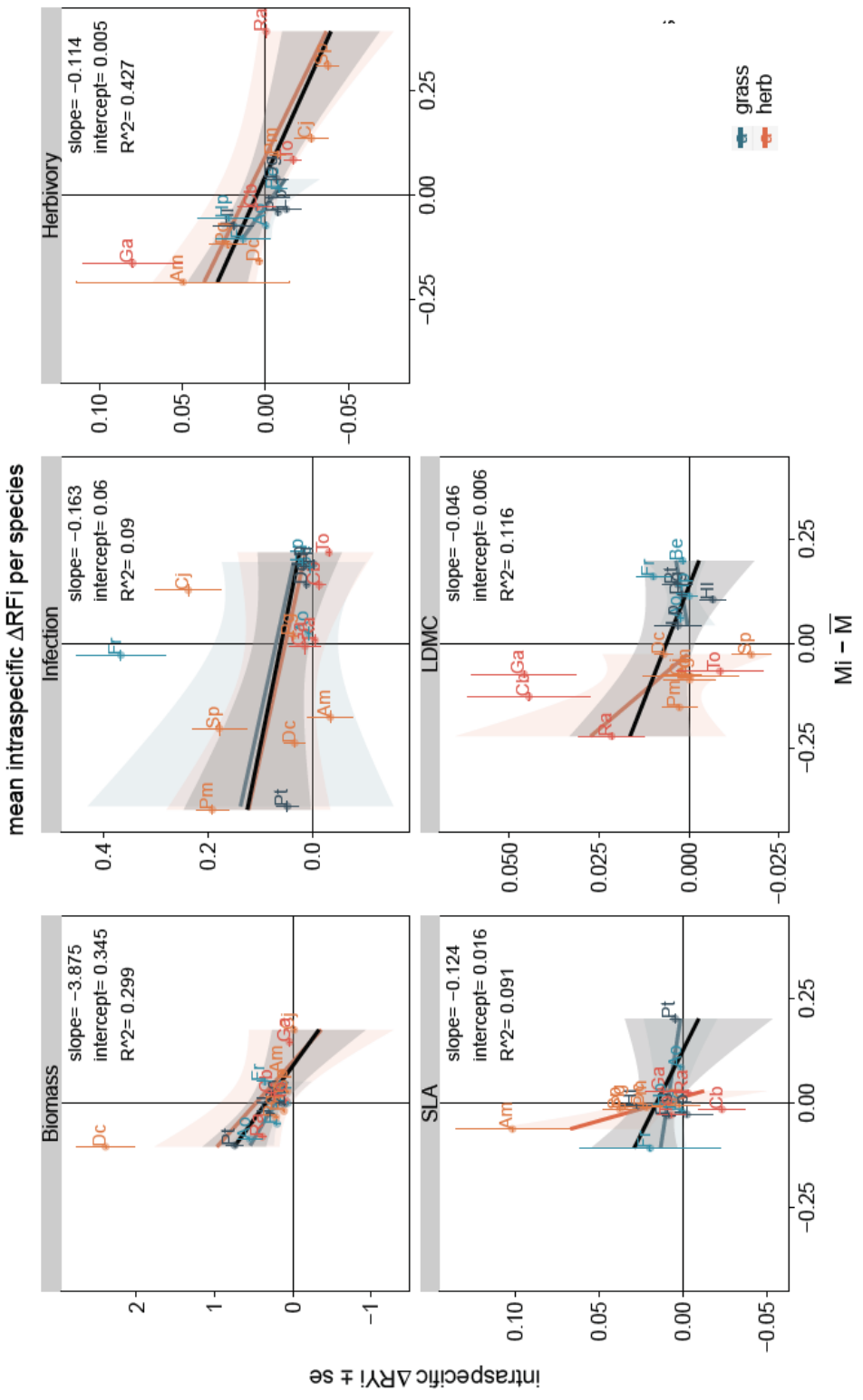


Figure 3 Overall intraspecific effects per function. The relationship between mean monoculture function per species across the sampling periods and treatments (scaled to the mean monoculture function) and intraspecific shift in polyculture (mean intraspecific ΔRF_i). Relationship is shown across all species (black) and across herbs (red) and grasses (blue) separately. The negative correlations are reflected in the negative intraspecific selection effects in all functions (Figure 1). Slopes, intercepts and R^2 for the relationship across all species are given in the plots.

Correlations

To better understand these simultaneous changes in biodiversity effects, we calculated correlations between all effects within and across functions. Within functions, all complementarity and selection effect were highly significantly negatively correlated (blue squares in Table 1), leading to intermediate net effects (Figure 1). The negative correlation between complementarity and selection effects was mainly driven by intraspecific selection effects, while interspecific selection effects had weaker and variable correlations with complementarity effects.

We observed negative correlations between the diversity effects for SLA and LDMC (Table 1n, Figure S8). These correlations mean that when communities shifted towards lower SLA with diversity, they also a shifted towards higher LDMC (i.e. towards a slower growing plant community). We observed negative net effects for both SLA and LDMC, which seems contradictory, but can be explained by the dominance of the slow growing herbs, which have low SLA but also relatively low LDMC (compared to the grasses, Figure S9a).

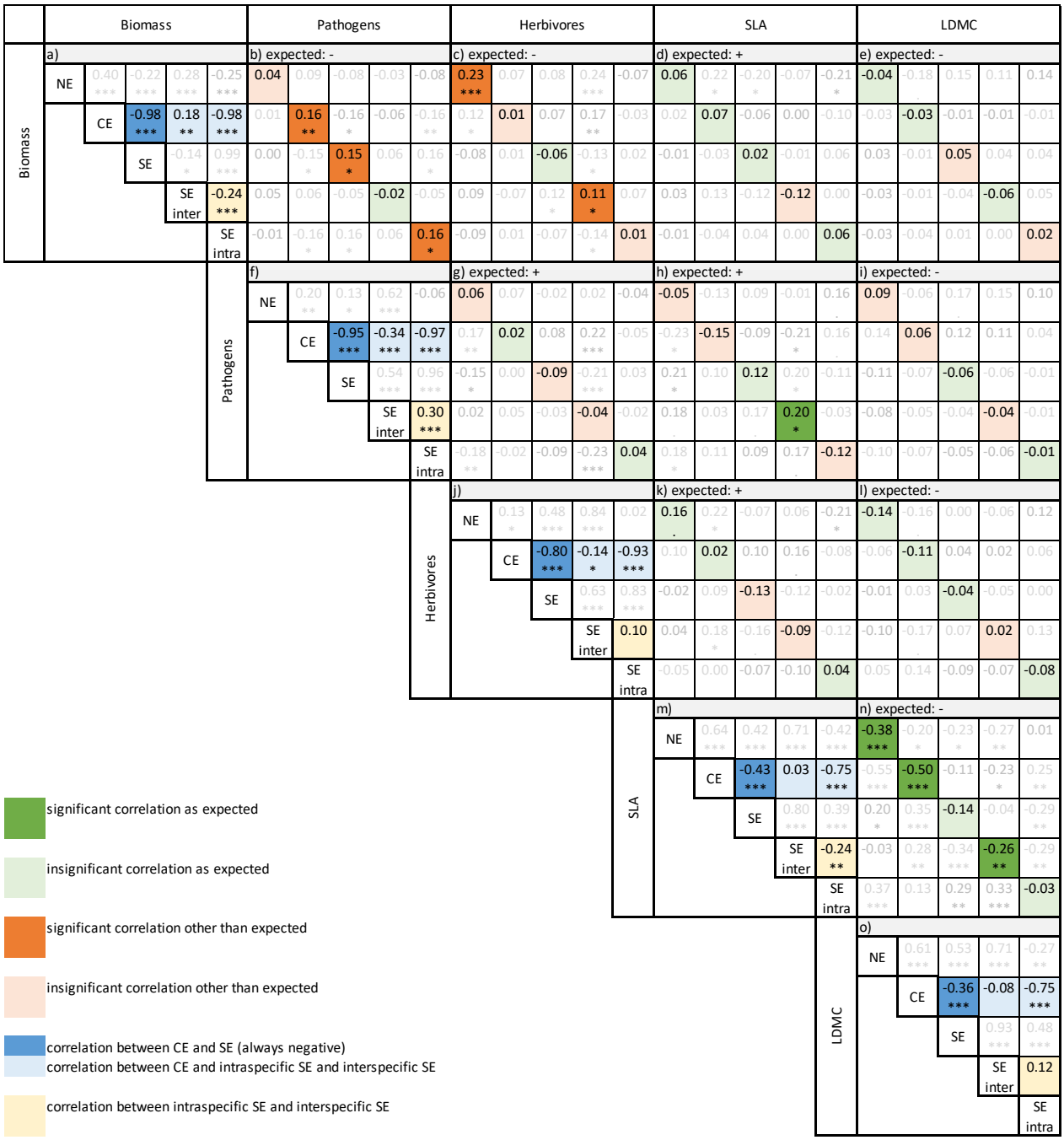
Diversity effects on the consumer (herbivores, pathogens) functions did not correlate strongly with those on the traits, except for a weak positive correlation between the interspecific selection effects of pathogen infection and SLA. (Table 1g-h, k-l). Complementarity and selection effects for biomass were slightly positively correlated with complementarity and selection effects for pathogen infection, due to intraspecific changes (Table 1b). Net effects and interspecific selection effects for biomass were negatively correlated with the net effect of herbivory (Table 1c). These effects were counter to our expectation that where consumers were reduced in diverse communities, this would also increase biomass.

Context dependency

The SLA and LDMC diversity effects were largely unaffected by the experimental treatments (Figure 4). We therefore do not discuss the details of these models.

Diversity effects for biomass were stronger with higher **species richness**. Positive complementarity effects became more positive and negative intraspecific selection effect became more strongly negative with increasing diversity, while interspecific selection effects were not affected (Figure 4).

Table 1 correlations between all measures of net effect (NE), complementarity effect (CE) and total, intra- and interspecific selection effect (SE) of biomass, pathogen infection, herbivory damage, specific leaf area (SLA) and leaf dry matter content (LDMC). More details can be found in Figure S7. The expectations are described in Table S4.



The **growth strategy** of the plant community (sown SLA) had the biggest impact on diversity effects for pathogen infection (Figure 4). The net effect of diversity on pathogen infection was negative in slow growing communities but increased to slightly positive in high SLA communities (Figure 4). Increases in the net effect were driven by increases in intra- and interspecific selection effects (Figure 4): in communities containing only slow-growing species, species with low monoculture infection could increase in abundance (negative interspecific selection effect), while fast growing communities were dominated by species with high monoculture infection. However, the complementarity effects followed the opposite pattern: on average, plants had higher infection than expected based on the monocultures (positive complementarity effect) and this was particularly strong in slow-growing communities. The lower complementarity effects in fast growing communities arise because many fast-growing species have very high monoculture infection (close to 100%) cannot increase infection further in polycultures.

The growth strategy of the plants also determined the effect of diversity on herbivory. The net effect on herbivory shifted from zero in slow growing communities to negative in fast growing and this pattern was driven by the interspecific selection effect (Figure 4). The net effect of diversity on biomass was, on average, slightly lower when the communities contained mainly fast-growing plants than when they contained mainly slow-growing plants (Figure 4). This was driven by complementarity effects, and to some degree by inter- and intraspecific selection effects (Figure 4). Despite these large effects of average growth strategy, functional diversity in terms of differences in SLA never altered diversity effects on any functions.

Nitrogen enrichment increased the interspecific selection effect in biomass and in pathogen infection, but this did not alter the net effect. Weak changes in complementarity and intraspecific selection effects may have balanced changes in the interspecific selection effect. Nitrogen enrichment further decreased positive complementarity and increased negative intraspecific selection effects of herbivory (Figure 4).

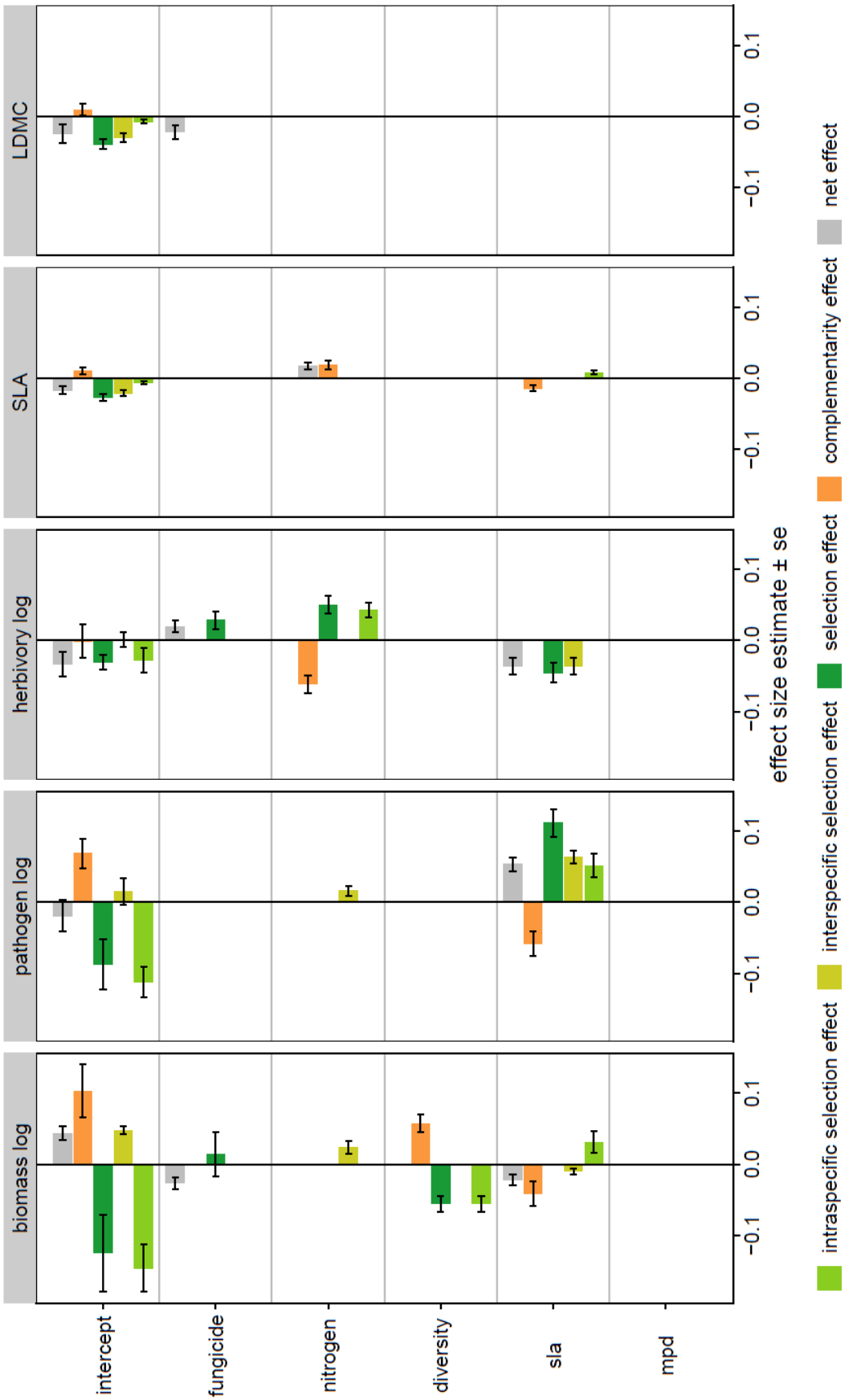


Figure 4 Effect sizes of the linear mixed effects models for net effect (grey), complementarity effect (orange), selection effect (dark green) intraspecific selection effect (yellow-green) and i (light green) of all functions biomass, pathogens, herbivory, SLA and LDMC. All explanatory variables were standardized and centered, which means that the displayed effect sizes reflect the effect size of variable(s) of interest at the mean of all other variables. Selected plots of model predictions can be found in Figure S10 and the detailed model results can be found in -

Fungicide decreased the positive net effect for biomass and removed the negative net effect of herbivory. These changes could not be attributed to complementarity or selection effects, probably because they were too weak to be detected by the models. Fungicide did not change net, complementarity or selection effects of infection (Figure 4a). This doesn't mean that fungicide had no effect on infection (Cappelli et al. 2019), but it means that fungicide doesn't alter diversity effects on infection.

DISCUSSION

We always observed neutral or positive complementarity and negative selection effects across all functions, which shows that broad diversity mechanisms are similar for these functions. Despite similar patterns, the lack of correlations between many diversity effects indicates that different species drove different functions and varying underlying ecological mechanisms are behind the diversity effects on different functions.

We found mostly positive complementarity effects (positive for biomass, pathogen infection and SLA, neutral for herbivory and LDMC). This indicates that multiple species increase their functioning in polycultures, and the functioning of mixed plant communities is typically driven by several species. However, sometimes complementarity effects were driven by a few species with extraordinarily large increases in functioning (especially for pathogen infection, see also Mahaut et al. 2019). This shows the importance of also examining individual species contributions to interpret complementarity effects, in cases where species contributions to function vary dramatically (Roscher et al. 2007b). The lack of significant correlations between complementarity effects for different functions (except between biomass and pathogen infection and between SLA and LDMC), indicate that in most cases different species supplied different functions. This highlights the importance of having a high diversity of species to maintain multiple ecosystem functions simultaneously (Hector and Bagchi 2007; Isbell et al. 2011). Many underlying mechanisms can cause complementarity effects. For biomass production, resource partitioning is often assumed, however there are many other possibilities, such as facilitation between species or decreased pressure from natural enemies (Barry et al. 2019). These mechanisms might directly or indirectly affect other functions: for example decreased aboveground enemy infection should result in a negative complementarity effect for herbivory or pathogen infection and enhanced resource use efficiency might influence the expression of functional traits, e.g. causing increased biomass N pools (Fargione et al. 2007).

However, the generally positive complementarity effects could also have been driven by different mechanisms in different functions. For example, increased density in diverse plant communities can increase SLA through shading (Lipowsky et al. 2015; Roscher et al. 2018b) or diversity can increase the chance for natural enemy spillover (Power and Mitchell 2004; Castagneyrol et al. 2014). Interestingly, even though herbivores and fungal pathogens are both primary consumers and might be expected to respond similarly to plant diversity (e.g. Heckman et al. 2016; Blumenthal 2006), complementarity effects were neutral for herbivory but positive for fungal pathogen infection. Stronger complementarity for pathogen infection might be explained by a greater importance of spillovers for pathogens because pathogens are less mobile than herbivores and more likely to spread only between neighboring hosts (Raffa et al. 2019). Positive complementarity effects indicate that most functions are driven by multiple species; however, it is likely that for different functions, different types of complementarity interactions between species are responsible.

The intraspecific selection effect was always negative, showing that plants converged in their functioning (per area), mostly because species with low monoculture functioning increased their functioning in mixture. Convergence was strong for biomass and pathogen infection and weaker but still present for herbivory, SLA and LDMC. Several other studies have found negative intraspecific selection effects, which supports the idea that this is a common diversity effect across functions (plant traits: Roscher et al. 2018b, decomposition: Pires et al. 2018, biomass: e.g. Liu et al. 2018a; Wagg et al. 2017; Yin et al. 2018 but see Pontes et al. 2012). The species might have converged toward optimum functioning due to synergies between functions, meaning that convergence in one function drives convergence in the others. However, negative intraspecific selection effects were rarely correlated between functions, indicating that different species and mechanisms were responsible for convergence in the different functions. Negative density dependence, caused by strong intraspecific competition or specialist enemies (de Kroon et al. 2012), or even facilitation of low functioning species (Soliveres et al. 2015), could have resulted in increases for low functioning species in mixture and therefore convergence in biomass and possibly the traits. For herbivory and pathogen infection, convergence might have been driven by species with much enemy damage in monoculture benefiting from host dilution (Keesing et al. 2006; Mitchell et al. 2002; Rottstock et al. 2014), while species with low monoculture enemy damage suffered from spillover (Power and Mitchell 2004).

While intraspecific selection effects were consistently negative, interspecific selection effects varied. The contribution of a given species to interspecific selection effects is defined by its abundance and monoculture functioning. Differences between functions arise from different correlations between the abundance shifts and the different monoculture function values. The shifts in abundances were most strongly related to monoculture biomass and to some degree to monoculture trait values (Figure S5). Species with high (monoculture) biomass and low SLA and LDMC increased in abundance at the cost of species with low biomass, high SLA and LDMC. This was expected because these functions often covary (Wright et al. 2004). Monoculture pathogen infection and herbivory were generally not related to abundance shifts, but this varied depending on the experimental treatment, see below. However, the interspecific selection effects of herbivory and biomass were correlated, indicating that at least some of the species which increased in abundance had both high monoculture biomass and high monoculture herbivory. This might indicate that the negative consequences of herbivory could be offset by the benefits of high biomass of the species, leading to dominance of highly productive species, but no visible effect of herbivory on species abundances (Gianoli and Salgado-Luarte 2017).

We found variable context dependency in the diversity effects of different functions. There was weak context dependency for SLA and LDMC but strong effects of the treatments on diversity effects for herbivory, fungal pathogen infection and biomass. Interestingly the diversity effects for fungal pathogens and insect herbivores were affected by different factors. The diversity effects for pathogen infection responded strongly to community functional composition, while diversity effects for herbivory were mainly altered by nitrogen. Diversity effects for biomass mainly changed with increasing plant diversity. This shows that diversity can have different effects on ecosystem functions in different environments.

Diversity effects (complementarity and intraspecific selection effects) only strengthened with increasing plant species richness for biomass. Increasing positive complementarity and decreasing negative selection effects with increasing diversity are common for biomass, showing that mechanisms, such as enhanced nutrient use efficiency or reduced enemy attack, are more effective at higher species richness (Craven et al. 2016). For the other functions this was not the case. Given that diversity effects on plant enemies are often related to the abundance of the host plants (Keesing

et al. 2006) and that the biggest decline in host abundance occur between one and four species (Figure S11), it is not surprising that the diversity effects for herbivory and pathogen infection did not increase as plant species richness changed from 4 to 20 species. Similarly, studies did in most cases not find increasing selection and complementarity effects with higher diversity for different functional traits (Roscher et al. 2018b) and water use efficiency (Grossiord et al. 2013). This indicates that diversity effects on several functions saturate at low diversity levels but that mechanisms promoting biomass complementarity operate more effectively in higher diversity communities.

Community functional composition altered the strength of diversity effects for several functions. Diversity effects on herbivory were enhanced in fast growing communities while diversity effects on biomass and pathogen infection were maximal in slow-growing communities. For biomass production stronger complementarity in slow growing communities might reflect the fact that species from low resource environments are more strongly differentiated in resource competition, allowing more opportunities for coexistence (Tilman 1982). For pathogen infection fast growing species had generally high infection and diversity did not alter infection in fast growing communities (Cappelli et al. 2019). The interspecific selection effect was also affected by functional composition and changed in opposing directions for pathogen infection and herbivory. Species with low pathogen infection and high herbivory increased in abundance in slow growing communities, while species with high infection and low herbivory increased in fast growing communities. This could mean that fast growing plants are more susceptible to herbivores, while the competitive ability of slow growing plants is more reduced by pathogens. This would suggest that different trade-offs between defense, growth and tolerance exist for herbivores and pathogens. Some studies have suggested that high functional diversity should enhance diversity effects (Wagg et al. 2017), which we do not find here. In contrast, community functional composition has rarely been considered as a modifier of diversity-functioning relationships but our results suggest that it alters diversity effects on several functions.

Resource levels and fungal pathogen abundance also altered diversity effects for some functions. Nitrogen enrichment increased the interspecific selection effect for biomass and pathogen infection, showing that nitrogen favors species with high biomass production and high pathogen infection, as expected (Liu et al. 2017; Siebenkaes et

al. 2016; Pontes et al. 2012; Heckman et al. 2016). This is at least partially driven by the same species, as the intraspecific selection effects of biomass and pathogen infection are correlated. Nitrogen enrichment also altered diversity effects for herbivory: complementarity decreased from positive to negative with nitrogen and intraspecific selection became less negative. Fungicide had relatively small effects, but it did weaken the positive net effect of diversity and negative net effect of herbivory. The effects on biomass agree with studies on soil pathogens (Maron et al. de Kroon et al. 2012) and suggest that aboveground pathogens may drive some of the diversity-productivity relationship.

We observed remarkably consistent diversity effects across different functions, showing that diversity affects different functions in broadly similar ways. However, we know relatively little about the underlying mechanisms. It has recently been shown that for biomass production diversity mechanisms link to coexistence mechanisms, so that the most stably coexisting communities produced most biomass (Godoy et al. 2019). This link was not apparent for other functions, agreeing with the idea that different underlying mechanisms are responsible. The low correlations between the diversity effects of the different functions indicate that different species supply different functions in diverse communities and the variable context dependencies indicate that the responses of the different species are likely driven by different mechanisms for different functions. The results of this study illustrate how varying ecological mechanisms affecting different plant species can lead to comparable overall patterns in how diversity impacts different ecosystem functions. Negative selection effects and positive complementarity effects were the rule and led to rather weak net effects. However, the lack of strong correlations between diversity effects on many functions show that different species drive different functions, which highlights the importance of high diversity for the provision of multiple ecosystem functions simultaneously.

ACKNOWLEDGEMENTS

I am very grateful to Eva Spehn and Andy Hector for their help when I was desperately trying to make sense of my results. I am also thankful for the comments on the manuscript of Santi Soliveres and for the much needed emotional support of my colleagues Anne Kempel, Clemens Kleinspehn and Hugo Vincent in the moments when I found another mistake in the code and their service as guinea pigs when I experimented on how to best explain the results. We are thankful to the whole PaNDiv

team especially Hugo Vincent and Mervi Laitinen and the many helpers without whom the PaNDiv Experiment would not be possible

SUPPLEMENTARY

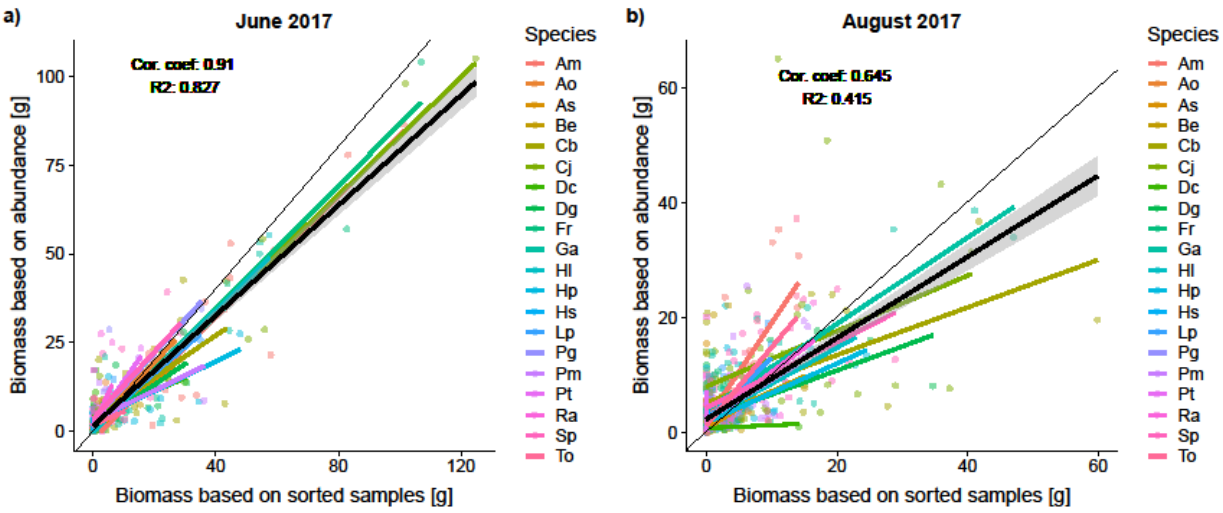


Figure S1 correlation between biomass calculated based on total plot biomass and visual estimates of abundance of each species and biomass per species from sorted samples from a) June and b) August 2017. Note that abundance was measured in the central square meter of the plots, while biomass samples were taken on two subplots (20cm x 50cm) within the central square meter. In August we sorted one sample per plot for 216 plots, while in June we sorted both samples per plot for 84 plots. Sorting only one sample led to a less precise correlation, as we do not account for spatial heterogenity in the plots.

Supplementary Methods

Calculations of additive partitioning

We first used a bipartite partition of diversity effects into complementarity and selection for all functions. We calculated additive partitioning sensu Loreau and Hector (2001) for biomass and used the adjusted framework of Grossiord et al. (2013) for all the other functions, with abundance (relative cover, a_i) as the weighting factor (see Table S1 and Table S2). The **complementarity effect** shows how much higher or lower the biomass of the species in a polyculture is, on average, compared to their monoculture biomass. The **selection effect** is a measure of how much the species with high (or low) monoculture biomass contribute to the polyculture biomass. In all cases, we used the monocultures with the corresponding nitrogen and fungicide treatment as a reference, so that we analyze shifts in response to diversity, under the different treatments.

Table S1 General additive partitioning variables for functions and what they correspond to in the framework of Loreau and Hector (2001) used for biomass and the adjusted framework of Grossiord et al. (2013) used for traits and enemy damage. Note that for calculating $F_{O\,i}$ $T_{mix\,i}$ is weighted with abundance, while $Y_{O\,i}$ is not. The reason for this is that - other than for biomass - for the other functions we do not measure an amount, but a value per species, which is independent of the abundance. For the further calculations it is important that $T_{mix\,i}$ is weighted (Grossiord et al. 2013). More details in Table S2.

variable	explanation	Biomass	Traits & enemy damage
$F_{mono\,i}$	Value of the function of species i measured in the monoculture with the corresponding nitrogen and fungicide treatment	M_i	$T_{mono\,i}$
$F_{mix\,i}$	Value of the function of species i measured in the mixture	$= Y_{O\,i}$, in PaNDiv: $Y_O * a_i$	$T_{mix\,i}$
a_i	Abundance of species i in the mixture	a_i	a_i
$F_{O\,i}$	contribution of species i to the function of the mixture	$Y_{O\,i}$	$T_{O\,i} = T_{mix\,i} * a_i$

The expected value of a certain function of a plant species mixture (F_E) was calculated as the mean of the monoculture values of all the species i in the mixture ($\overline{F_{mono\,i}}$), $F_E = \overline{F_{mono\,i}} = \sum_i (F_{mono\,i} * \frac{1}{N})$, because we sowed the species at equal abundances ($\frac{1}{N}$).

The observed function of a plant species mixture (F_O) in case of biomass, equals the actually measured biomass in the plots and for the other functions it is the community weighted mean of the measured values of the species in the mixture $CWM(T_{O\,i}) = \sum_i T_{O\,i} * a_i$. The net effect quantifies how much the observed function of a plant mixture deviates from the expected function level for that mixture. A positive net effect means

that the mixture has a higher function value than expected based on the monocultures. The net effect can be expressed as the sum of the complementarity effect and the selection effect (Loreau and Hector 2001).

$$NE = CE + SE = N * \overline{\Delta F_{mix\,i}} * \overline{F_{mono\,i}} + N * cov(\Delta F_{mix\,i}, F_{mono\,i}) \quad (1)$$

with the complementarity effect calculated as followed for biomass, based on the number of species in the mixture (N), the biomass yields of all the species in the mixture ($Y_{O\,i}$) and their respective monoculture yields (M_i) (Loreau and Hector 2001).

$$CE = N * \frac{\overline{Y_{O\,i}}}{M_i} * \overline{M_i} \quad (2a)$$

For the other functions, the measures of the function in the mixture ($T_{mix\,i}$) are weighted with a_i to calculate the contribution of each species i to the plotlevel function ($T_{O\,i}$) (Grossiord et al. 2013)

$$CE = N * \frac{\overline{T_{mix\,i} * a_i}}{T_{mono\,i}} * \overline{T_{mono\,i}} \quad (2b)$$

The selection effect for biomass sensu Loreau and Hector (2001) is calculated as

$$SE = \sum_i \left(\frac{Y_{O\,i}}{M_i} - \overline{\frac{Y_{O\,i}}{M_i}}\right) * (M_i - \overline{M_i}) \quad (3a)$$

and for the other functions sensu Grossiord et al. (2013) as

$$SE = \sum_i \left(\frac{T_{mix\,i} * a_i}{T_{mono\,i}} - \overline{\frac{T_{mix\,i} * a_i}{T_{mono\,i}}}\right) * (T_{mono\,i} - \overline{T_{mono\,i}})$$

(3b)

Selection effect: inter- vs. intraspecific shifts

We next partitioned the selection effect into selection effects due to **inter- and intraspecific shifts**. This tripartite partition (complementarity, intra and interspecific selection effects) is the same as the tripartite partition of Fox (2005). To visualize this partition, we can imagine an intermediate community which has the observed species relative abundances (observed cover values), but in which the level of function provided by a species per unit area is the same as in the monoculture (illustrated in Figure S2). We can then in a first step calculate additive partitioning between the intermediate and the expected community. Because an abundance shift of one species always comes at the cost of another species, the complementarity effect is always zero in this first step. The **interspecific selection effect** calculated in this first step is analogous to the dominance effect of Fox (2005). In a second step we can calculate

additive partitioning between the observed and the intermediate community, i.e. assuming that species change their level of function per unit area but their relative cover stays the same. The complementarity effect can be calculated in this second step and is the same as the complementarity effect of the standard bipartite partition and the trait-independent complementarity effect of Fox (2005). The **intraspecific selection effect** of the second step only considers changes in the provision of a given function per species and per unit area (=intraspecific shifts) and is analogous to the trait-dependent complementarity effect of Fox (2005) (mathematical details in Table S2).

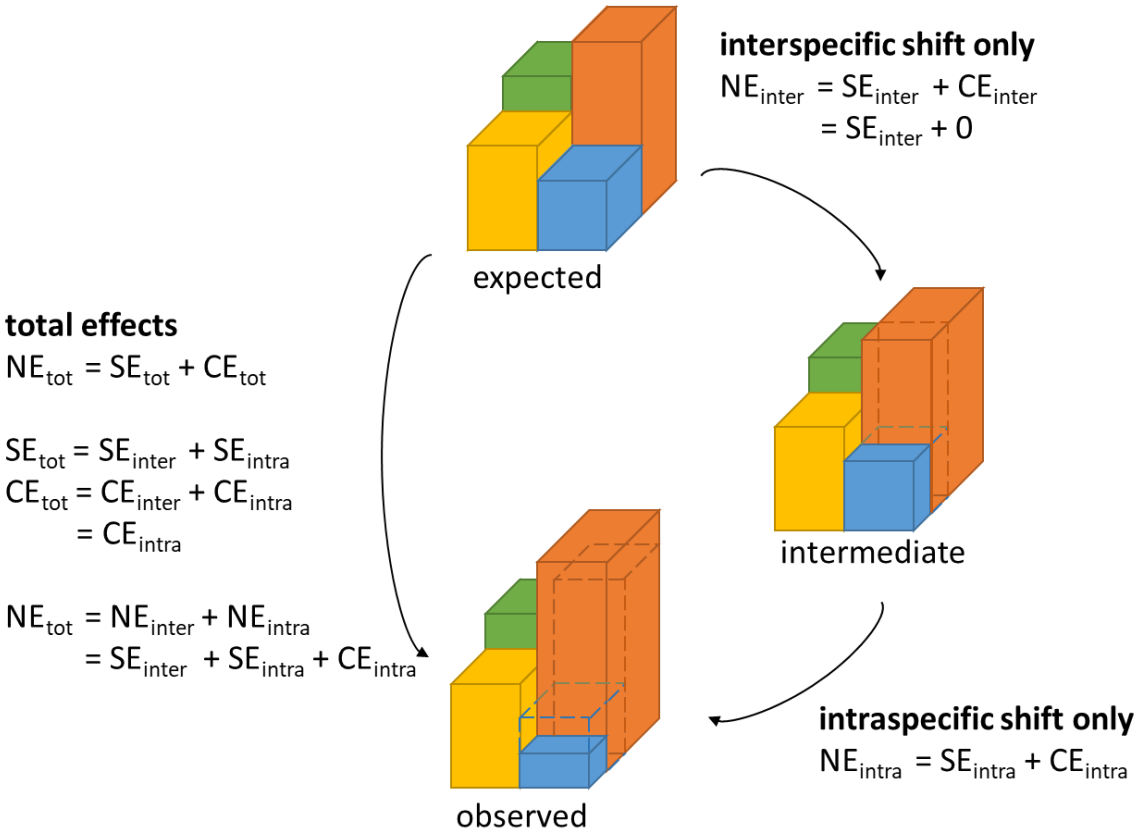


Figure S2 Additive partitioning split up into intra- and interspecific shifts, analogous to Fox (2005). The differently colored cubes illustrate single species. The height of the cubes represents the function provided by the species per unit area, the ground area represents the species abundance (relative cover), which makes the volume of a cube the contribution of the given species to the polyculture function. In this example we assume a plant community of four species. Based on the monocultures we expect the species to have a certain value of the function per area (height), and because of equal sown abundances we expect them to cover exactly one quarter of the total community (top left in the image). However, we observe something different: the blue and orange species have a different function per area than in monoculture (height) and a different abundance (area) than expected (dashed lines represent the expected shape base on the monoculture). Normally, we calculate additive partitioning by comparing the expected and the observed community. Here we want to partition selection effects into inter- or intraspecific shifts. The total selection effect can be split up by comparing an 'intermediate" community, where only the interspecific shift in relative abundances is considered (the orange species increases at the expense of the blue species), with the expected community. This “intermediate” community can then be compared with the actually observed community to evaluate the intraspecific shifts, in which species do not shift their relative abundance but change function per unit area (the red species increases its function per area and the blue species decreases it). Note that the interspecific shift is a zero sum game, which makes complementarity effect inter zero and complementarity effect intra is equal to complementarity effect tot, which makes this illustration analogous to the tripartite partitioning sensu Fox (2005). The mathematical details underlying this concept can be found in Table S2.

Table S2 Additive partitioning variables for biomass and the adaptation for other ecosystem functions such as traits or damage by natural enemies. Yellow: variables per species, white: variables per plot. $F_{mono\,i}$ and $F_{mix\,i}$ are the measured function per species in the monocultures and the mixtures respectively. Abundance (a_i) is the abundance of all the species in all the plots (total abundance is scaled to 1). Diversity (N) is the number of species in a plot, which equals the sown diversity if all species have established. Note that for calculating $F_{O\,i}$ $T_{mix\,i}$ is weighted with abundance, while $Y_{O\,i}$ is not. The reason for this is that - other than for biomass - for the other functions we do not measure an amount, but a value per species, which is independent of the abundance. For the further calculations it is important that Tmix_i is weighted (Grossiord et al. 2013). The expected relative infection equals $\frac{1}{N}$ because we have sown the species at equal abundances.

Additive Partitioning sensu Loreau and Hector (2001)			**Original definition of Loreau and Hector (2001) used for biomass**		**Adapted framework of Grossiord et al. (2013), used for traits, herbivory and**	
var	**explanation**	**calculation**	**var**	**calculation**	**var**	**calculation**
N	Number of species/diversity	given by experimental design	N		N	
a_i	Abundance of species i in the mixture	measured	a_i		a_i	
$F_{mono\,i}$	Value of the function of species i measured in the monoculture with the corresponding nitrogen and fungicide treatment	measured	M_i		$T_{mono\,i}$	$T_{mono\,i}$
$F_{mix\,i}$	Value of the function of species i measured in the mixture	measured	$=$ $Y_{O\,i}$	$Y_O * a_i$ (in PaNDiv, because we don't measure species level biomass)	$T_{mix\,i}$	
$F_{O\,i}$	Contribution of species i to the function of the mixture		$Y_{O\,i}$		$T_{O\,i}$	$T_{mix\,i} * a_i$
F_O	function of the mixture.	$\sum_i F_{O\,i}$	Y_O	$\sum_i Y_{O\,i}$	T_O	$\sum_i T_{O\,i} * a_i = CWM(T_{O\,i})$
$RF_{E\,i}$	Relative expected function of the mixture = sown abundance	$\frac{1}{N}$ (sp. sown at equal abundances!)	$RY_{E\,i}$	$\frac{1}{N}$	$RT_{E\,i}$	$\frac{1}{N}$
$RF_{O\,i}$	Relative observed function of the mixture	$\frac{F_{O\,i}}{F_{mono\,i}}$	$RY_{O\,i}$	$\frac{Y_{O\,i}}{M_i}$	$RT_{O\,i}$	$\frac{T_{O\,i}}{T_{mono\,i}} = \frac{T_{mix\,i} * a_i}{T_{mono\,i}}$
$F_{E\,i}$	Expected function of species I in the mixture	$RF_{E\,i} * F_{mono\,i} = \frac{F_{mono\,i}}{N}$	$Y_{E\,i}$	$RY_{E\,i} * M_i = \frac{M_i}{N}$	$T_{E\,i}$	$RT_{O\,i} * T_{mono\,i} = \frac{T_{mono\,i}}{N}$

F_E	Expected function of the mixture	$\sum_i F_{E\,i} = \sum_i RF_{Ei} * F_{mono\,i}$ $= \sum_i \frac{F_{mono\,i}}{N} = \overline{F_{mono\,i}}$	Y_E $\sum_i Y_{E\,i} = \sum_i RY_{Ei} * M_i$ $= \sum_i \frac{M_i}{N} = \overline{M_i}$	T_E $\sum_i T_{E\,i} = \sum_i RT_{Ei} * T_{mono\,i}$ $= \sum_i \frac{T_{mono\,i}}{N} = \overline{T_{mono\,i}}$
ΔRF_i	Deviation of relative observed and expected function of species i	$RF_{O\,i} - RF_{E\,i}$ $= \frac{F_{O\,i}}{F_{mono\,i}} - \frac{1}{N}$	ΔRY_i $RY_{O\,i} - RY_{E\,i}$ $= \frac{Y_{O\,i}}{M_i} - \frac{1}{N}$	ΔRT $RT_{O\,i} - RT_{E\,i}$ $= \frac{T_{O\,i}}{T_{mono\,i}} - \frac{1}{N}$ $= \frac{T_{mix\,i} * a_i}{T_{mono\,i}} - \frac{1}{N}$
ΔF = NE	Deviation of observed function from expected function in the mixture = **net effect**	$= F_O - F_E$ $= \sum_i F_{O\,i} - \sum_i F_{E\,i}$ $= \sum_i RF_{O\,i} * F_{mix\,i} - \sum_i RF_{Ei} * F_{mix\,i}$ $= \sum_i \Delta RF_i * F_{mix\,i}$ $= N * \overline{\Delta RF_i} * \overline{F_{mono\,i}} +$ $N * cov(\Delta RF_i, F_{mono\,i})$ $=$ **complementarity + selection**	ΔY $= N * \overline{\Delta RY_i} * \overline{M_i} +$ $N * cov(\Delta RY_i, M_i)$ $=$ **complementarity + selection**	ΔT $= N * \overline{\Delta RT_i} * \overline{T_{mono\,i}} +$ $N * cov(\Delta RT_i, T_{mono\,i})$ $=$ **complementarity + selection**
CE	**complementarity effect**	$N * \Delta RF_i * \overline{F_{mono\,i}}$ $= N * \frac{\overline{F_{O\,i}}}{F_{mono\,i}} - \frac{1}{N} * \overline{F_{mono\,i}}$	CE $N * \overline{\Delta RY_i} * \overline{M_i}$ $= N * \frac{\overline{Y_{O\,i}}}{M_i} - \frac{1}{N} * \overline{M_i}$	CE $N * \overline{\Delta RT_i} * \overline{T_{mono\,i}}$ $= N * \frac{\overline{T_{mix\,i} * a_i}}{T_{mono\,i}} - \frac{1}{N} * \overline{T_{mono\,i}}$
SE	**selection effect**	$N * cov(\Delta RF_i, F_{mono\,i})$ $= \sum_i (\Delta RF_i - \overline{\Delta RF_i}) *$ $(F_{mono\,i} - \overline{F_{mono\,i}})$	SE $N * cov(\Delta RY_i, M_i)$ $= \sum_i (\Delta RY_i - \overline{\Delta RY_i}) *$ $(M_i - \overline{M_i})$ $= \sum_i ((\frac{Y_{O\,i}}{M_i} - \frac{1}{N}) - (\frac{\overline{Y_{O\,i}}}{M_i} - \frac{1}{N})) *$ $(M_i - \overline{M_i})$	SE $N * cov(\Delta RT_i, T_{mono\,i})$ $= \sum_i (\Delta RT_i - \overline{\Delta RT_i}) *$ $(T_{mono\,i} - \overline{T_{mono\,i}})$ $= \sum_i ((\frac{T_{mix\,i} * a_i}{T_{mono\,i}} - \frac{1}{N}) -$ $(\frac{\overline{T_{mix\,i} * a_i}}{T_{mono\,i}} - \frac{1}{N})) *$ $(T_{mono\,i} - \overline{T_{mono\,i}})$

Table S3 expresses Figure S2 mathematically. The first column contains all the terms used for additive partitioning when comparing the observed plant community with the expected plant community as it is done by Loreau and Hector (2001). The second column contains all the terms used for additive partitioning when comparing the intermediate to the expected community, only considering intraspecific. Note that we assume $T_{mix\,i}$ to be equal to $T_{mono\,i}$ and because of this $RT_{O\,i}$ is equal to a_i, which then is the reason for zero complementarity effect ($\overline{a_i - \frac{1}{N}}$ illustrates the zero sum game as explained by Fox (2005)). The third column contains all the terms used for additive partitioning when comparing the observed to the intermediate community, only considering interspecific shifts. Note that we assume $RT_{E\,i}$ to be a_i instead of 1/N

	Additive partitioning sensu Loreau and Hector (2001)	**Tripartite partitioning sensu Fox (2005)**	
Va.		**Interspecific shifts only**	**Intraspecific shifts only**
N			
a_i			
$T_{mono\,i}$			
$T_{mix\,i}$		$T_{mono\,i}$	
$T_{O\,i}$	$T_{mix\,i} * a_i$	$T_{mono\,i} * a_i$	$T_{mix\,i} * a_i$
T_O	$\sum_i T_{O\,i} * a_i = CWM(T_{O\,i})$	$\sum_i T_{mono\,i} * a_i = CWM(T_{mono\,i})$	$\sum_i T_{O\,i} * a_i = CWM(T_{O\,i})$
$RT_{E\,i}$	$\frac{1}{N}$	$\frac{1}{N}$	a_i
$RT_{O\,i}$	$\frac{T_{O\,i}}{T_{mono\,i}} = \frac{T_{mix\,i} * a_i}{T_{mono\,i}}$	$\frac{T_{O\,i}}{T_{mono\,i}} = \frac{T_{mono\,i} * a_i}{T_{mono\,i}} = a_i$	$\frac{T_{O\,i}}{T_{mono\,i}} = \frac{T_{mix\,i} * a_i}{T_{mono\,i}}$
$T_{E\,i}$	$RT_{O\,i} * T_{mono\,i} = \frac{T_{mono\,i}}{N}$	$RT_{O\,i} * T_{mono\,i} = \frac{T_{mono\,i}}{N}$	$RT_{O\,i} * T_{mono\,i} = a_i * T_{mono\,i}$
T_E	$\frac{\sum_i T_{E\,i} = \sum_i RT_{Ei} * T_{mono\,i} = \sum_i \frac{T_{mono\,i}}{N} =}{T}$	$\frac{\sum_i T_{E\,i} = \sum_i RT_{Ei} * T_{mono\,i} = \sum_i \frac{T_{mono\,i}}{N} =}{T}$	$\sum_i T_{E\,i} = \sum_i RT_{Ei} * T_{mono\,i} = \sum_i a_i * T_{mono\,i}$

ΔRT_i	$RT_{O\,i} - RT_{E\,i}$ $= \frac{T_{O\,i}}{T_{mono\,i}} - \frac{1}{N} = \frac{T_{mix\,i} * a_i}{T_{mono\,i}} - \frac{1}{N}$	$RT_{O\,i} - RT_{E\,i}$ $= a_i - \frac{1}{N}$	$RT_{O\,i} - RT_{E\,i}$ $= \frac{T_{O\,i}}{T_{mono\,i}} - a_i = \frac{T_{mix\,i} * a_i}{T_{mono\,i}} - a_i$
ΔT	$= N * \overline{\Delta RT_i} * \overline{T_{mono\,i}} + N * cov(\Delta RT_i, T_{mono\,i})$ $=$ **complementarity + selection**	$= N * \overline{\Delta RT_i} * \overline{T_{mono\,i}} + N * cov(\Delta RT_i, T_{mono\,i})$ $=$ **complementarity + selection**	$= N * \overline{\Delta RT_i} * \overline{T_{mono\,i}} + N * cov(\Delta RT_i, T_{mono\,i})$ $=$ **complementarity + selection**
CE	$N * \overline{\Delta RT_i} * \overline{T_{mono\,i}}$ $= N * \overline{\frac{T_{mix\,i} * a_i}{T_{mono\,i}} - \frac{1}{N}} * \overline{T_{mono\,i}}$ = complementarity effect	$N * \overline{\Delta RT_i} * \overline{T_{mono\,i}}$ $= N * \overline{a_i - \frac{1}{N}} * \overline{T_{mono\,i}}$ $= N * 0 * \overline{T_{mono\,i}} = 0$	$N * \overline{\Delta RT_i} * \overline{T_{mono\,i}}$ $= N * \overline{\frac{T_{mix\,i} * a_i}{T_{mono\,i}} - a_I} * \overline{T_{mono\,i}}$ = trait independent complementarity effect
SE	$N * cov(\Delta RT_i, T_{mono\,i})$ $= \sum_i (\Delta RT_i - \overline{\Delta RT_i}) * (T_{mono\,i} - \overline{T_{mono\,i}})$ $= \sum_i ((\frac{T_{mix\,i} * a_i}{T_{mono\,i}} - \frac{1}{N}) - (\overline{\frac{T_{mix\,i} * a_i}{T_{mono\,i}} - \frac{1}{N}})) * (T_{mono\,i} - \overline{T_{mono\,i}})$ = selection effect	$N * cov(\Delta RT_i, T_{mono\,i})$ $= \sum_i (\Delta RT_i - \overline{\Delta RT_i}) * (T_{mono\,i} - \overline{T_{mono\,i}})$ $= \sum_i ((a_i - \frac{1}{N}) - (\overline{a_i - \frac{1}{N}})) * (T_{mono\,i} - \overline{T_{mono\,i}})$ = dominance effect = interspecific selection effect	$N * cov(\Delta RT_i, T_{mono\,i})$ $= \sum_i (\Delta RT_i - \overline{\Delta RT_i}) * (T_{mono\,i} - \overline{T_{mono\,i}})$ $= \sum_i ((\frac{T_{mix\,i} * a_i}{T_{mono\,i}} - a_i) - (\overline{\frac{T_{mix\,i} * a_i}{T_{mono\,i}} - a_i})) * (T_{mono\,i} - \overline{T_{mono\,i}})$ = trait dependent complementarity effect = intraspecific selection effect

Dealing with missing data

Since our plant communities are sown at equal abundances, the expected contribution of the species in a mixture (RF_{e_i}) is 1/plant diversity. Despite resowing, two species, *Heracleum sphondylium* and *Anthriscus silvestris* did not establish in many plots, most likely unrelated to the experimental treatments. We therefore excluded the two species completely and adjusted plant diversity for the calculation of additive partitioning. In many cases the effect sizes of complementarity and selection was unchanged when the two species were excluded, but selection and complementarity effects for biomass were smaller when they were excluded (Figure S3).

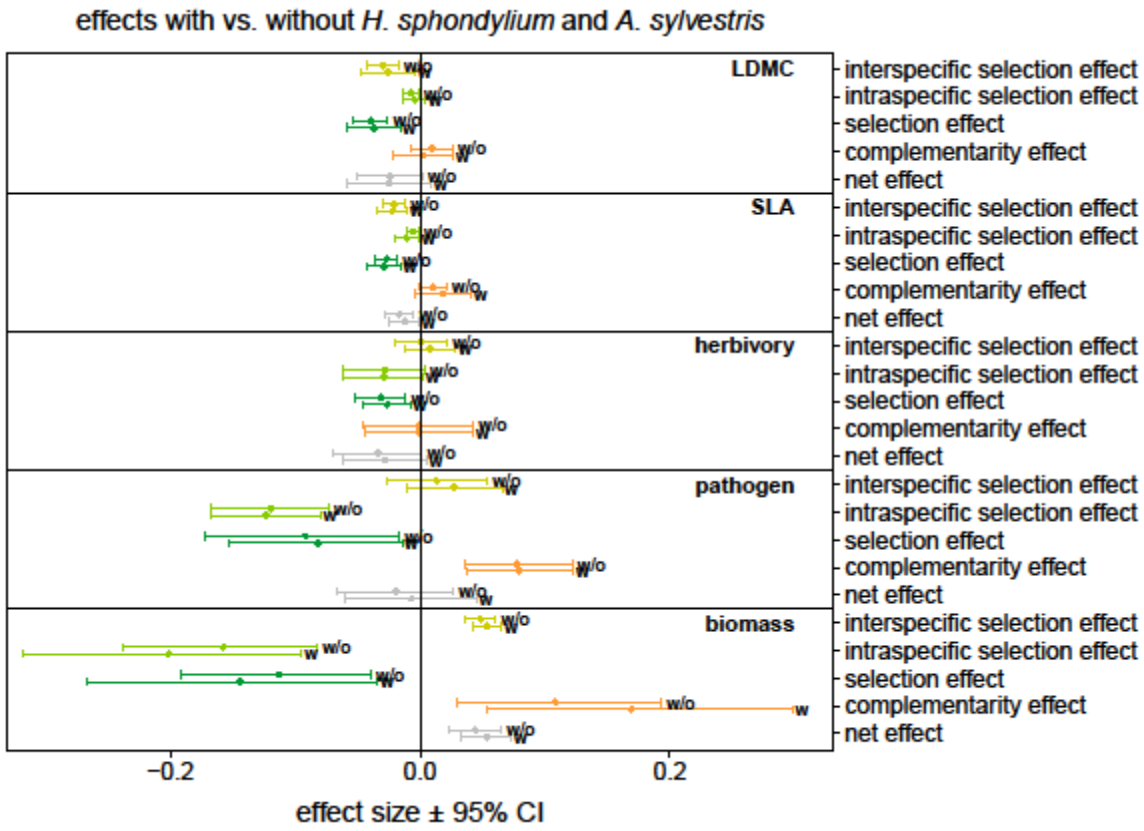


Figure S3 Comparison of intercept only models when *Heracleum sphondylium* and *Anthriscus sylvestris* were included (w) or excluded (w/o) in the analysis. Including them leads to an overestimation of complementarity and selection effect for biomass, but does not change the results of other functions much.

We could not measure traits or enemy damage for species with zero or very low abundance in a plot. We therefore used the monoculture values for these species instead. This is a conservative approach because it assumes that there is no effect of diversity on the functioning of these species. The net effect was not altered by this, because species at low abundance hardly contribute to overall community functioning. The contribution of these species to selection due to intraspecific shifts remains the same and because we used monoculture values, they could not contribute to

interspecific shifts.

We had a few cases with missing monoculture values (traits) and measurements of zero in monocultures (infection, herbivory, pathogens). Both cases cause problems when calculating additive partitioning. The measurements of zero were set to half of the minimum of all the other monocultures, to obtain a reasonably small value, without inflating the calculations of complementarity and selection effects. We had two cases of missing monoculture biomass values due to lost samples (2 out of 400 samples [80 monocultures x 5 sampling period]) and in total four missing values for herbivory, pathogens and traits, because the leaf material was dead after the mowing and had not grown back enough for measurements. In case of missing monoculture values, we predicted the values. We modeled the monoculture values as a function of species, sampling period, nitrogen and fungicide treatment and the interaction between species and sampling period. We then predicted the missing monoculture values from these models.

Supplementary Results

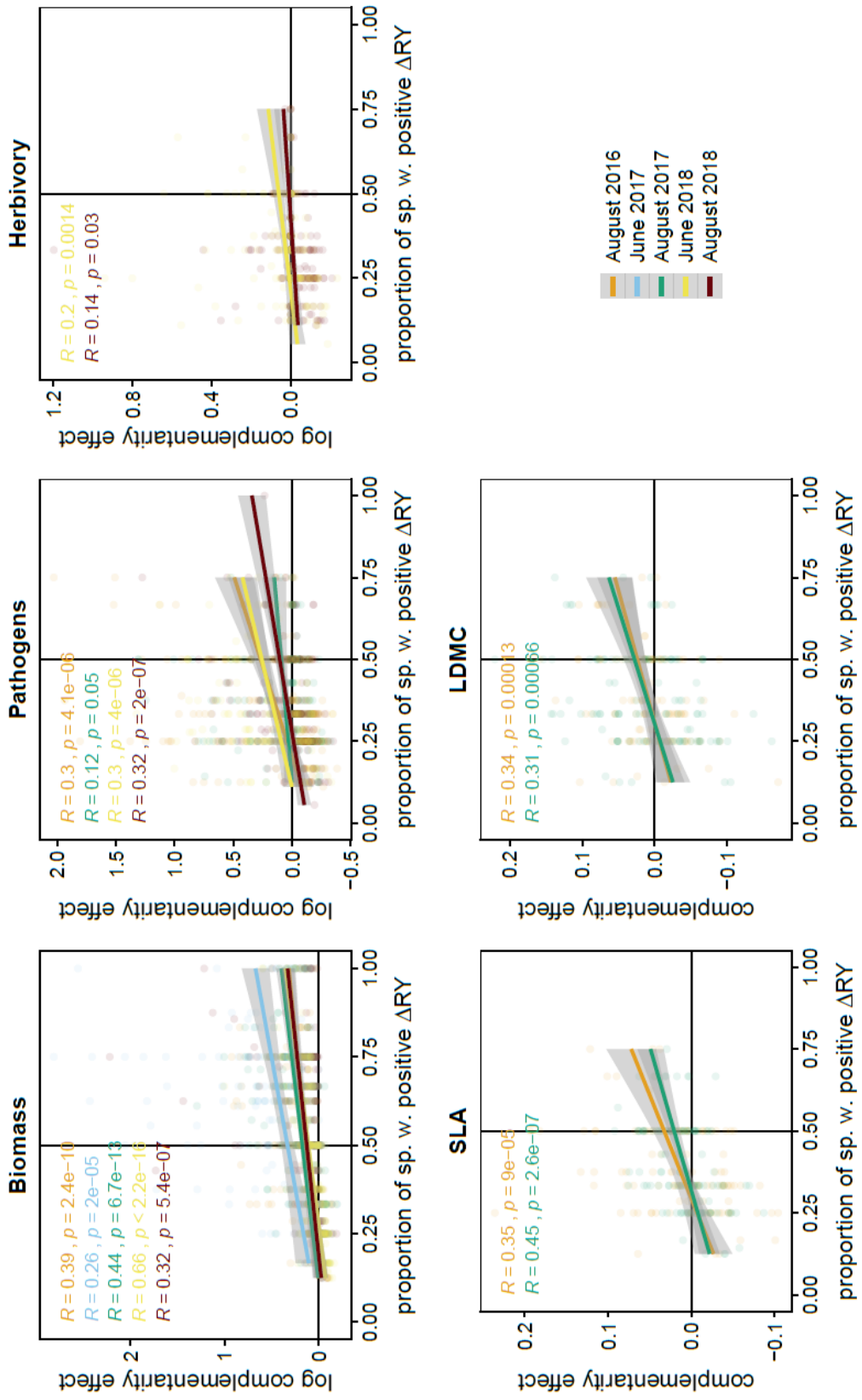


Figure S4 The proportion of species which increase their functioning in polycultures relative to mixtures increased complementarity effects. The proportion of species with positive ΔRY explained between 12 and 45% of the variation in complementarity effects in a Pearson correlation. Note that there are cases where a minority of all species in the mixture were able to drive positive complementarity (top left corner of the graphs).

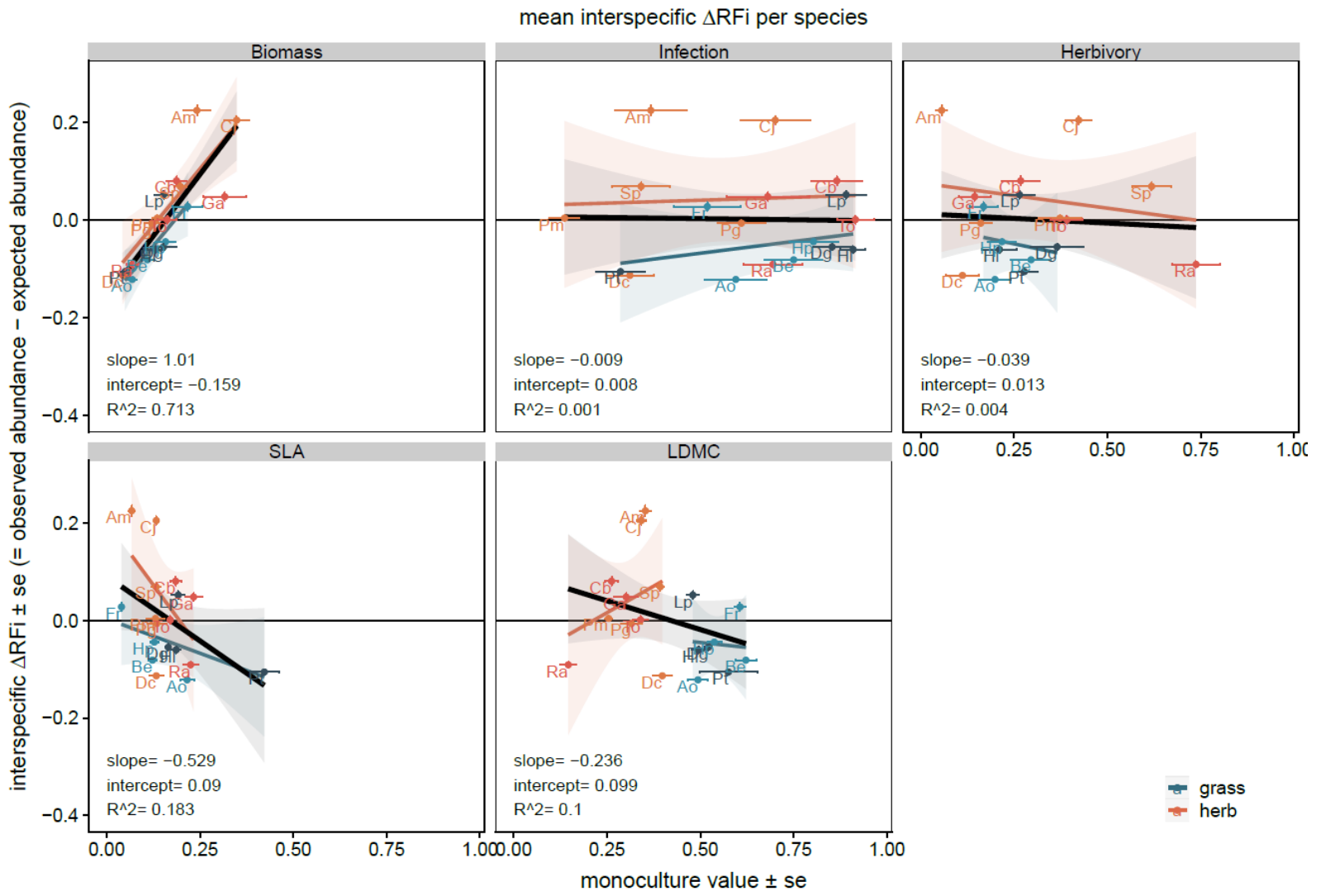


Figure S5 Mean values of monoculture function across the sampling periods and treatments and how well they explain mean interspecific ΔRF_i (=abundance shift) across all species (black) and across herbs (red) and grasses (blue) separately. Species with high monoculture biomass dominated the mixtures. This was to some degree coupled with the traits, but not to infection and herbivory. The negative relationship between ΔRF_i and the monoculture value in the case of LDMC was driven by the dominance of the herbs, which had lower LDMC than the grasses. Within the herbs and to some degree also within the grasses the ones with high LDMC were the species which had higher ΔRF_i and therefore able to increase in abundance more than the ones with lower ΔRF_i

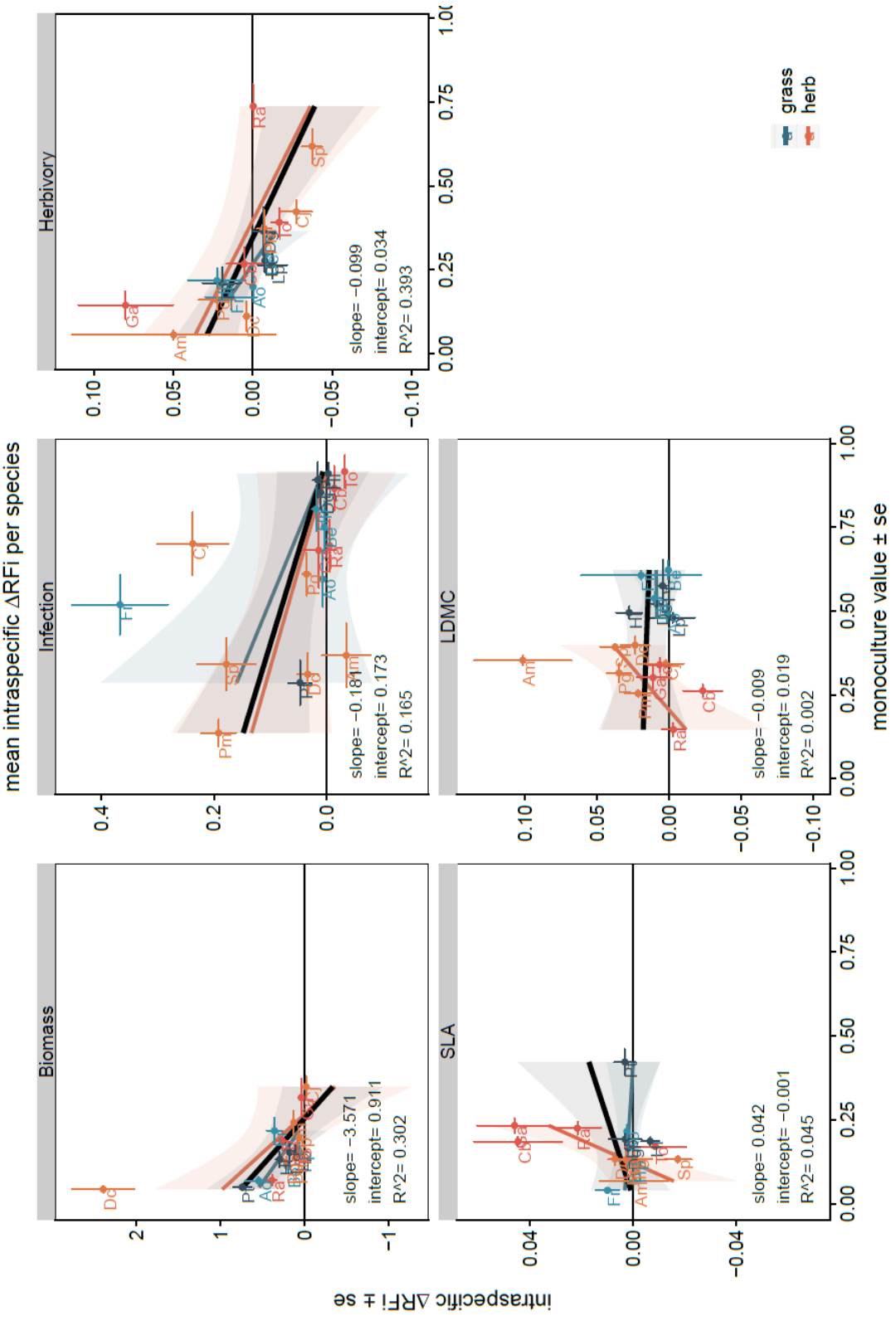


Figure S6 Mean values of monoculture function across the sampling periods and treatments and how well they explain mean intraspecific ΔRF_i (=abundance shift) across all species (black) and across herbs (red) and grasses (blue) separately.

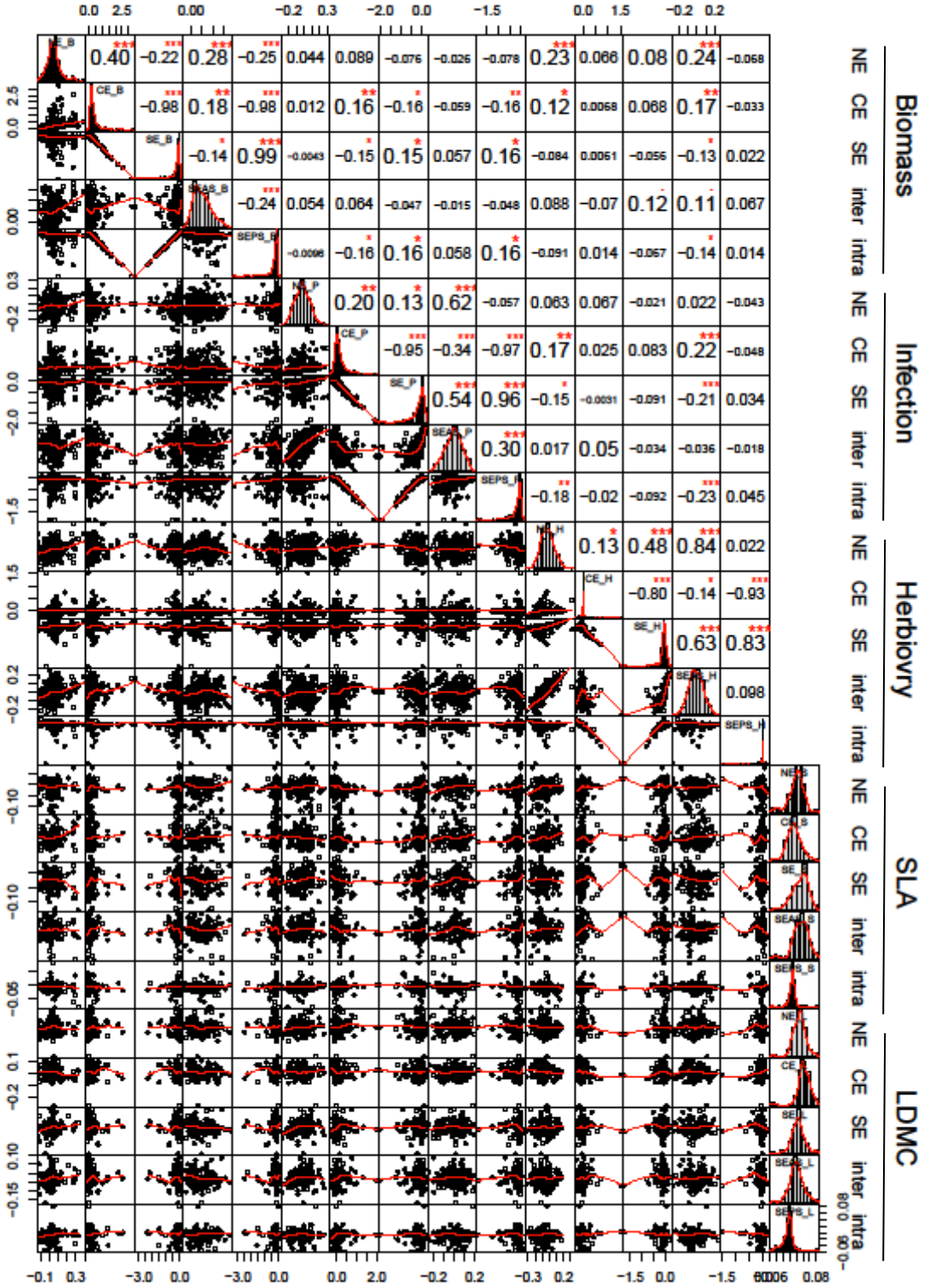


Figure S7 correlations between net effect, selection effect and complementarity effect of all functions (means over the seasons)

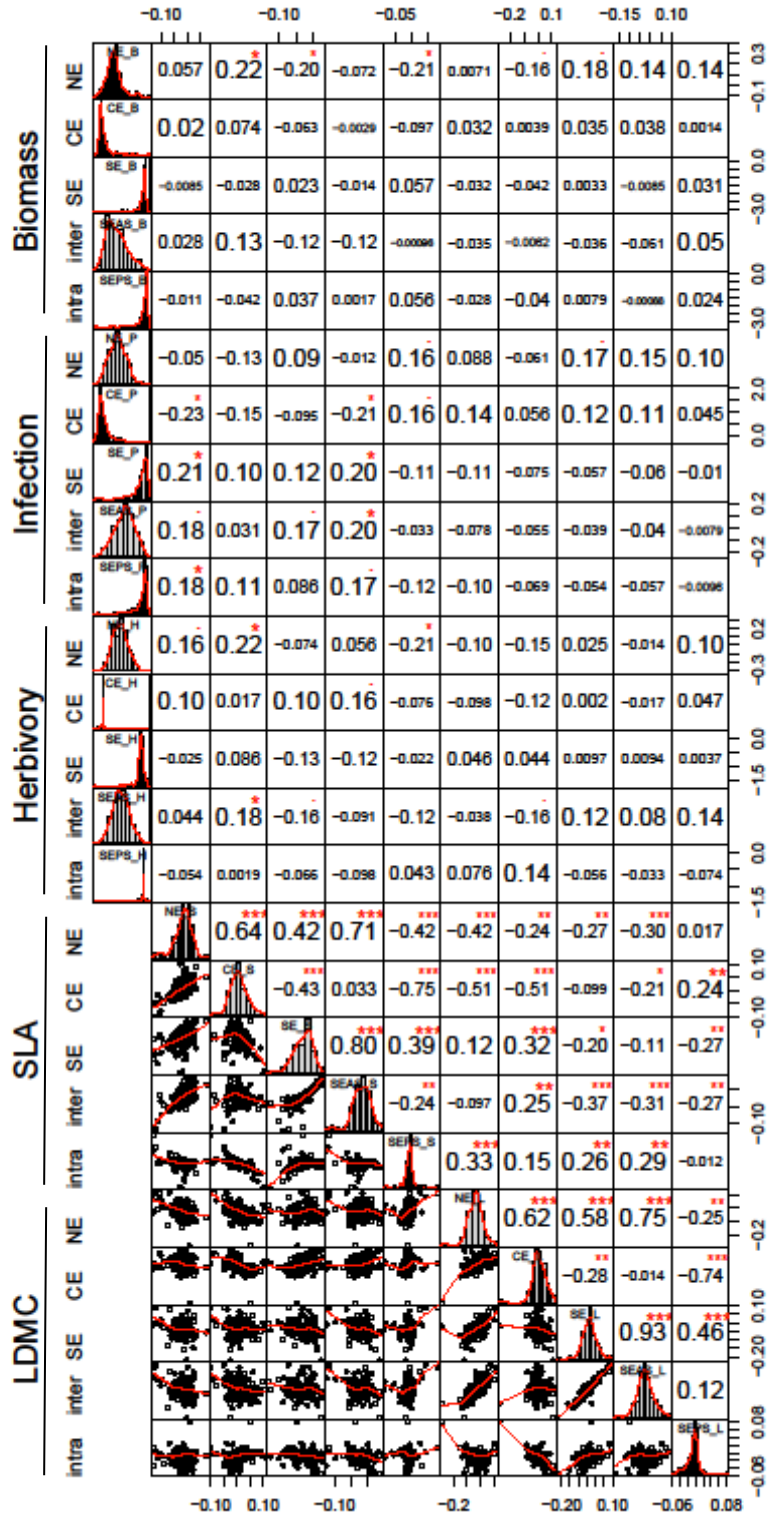


Figure S7 extended

Table S4 expected correlations between the diversity effects of different functions in Table 1

. The first column indicates which panel in Table 1 the expectation refers to.

Panel	Expectation	Reference
b, c	Pathogenic fungi and herbivorous insects cause biomass loss. The more damage by pathogenic fungi and herbivorous insects we observe in the field, the higher the biomass loss should be and diversity effects of biomass production should be negatively related to diversity effects of pathogen infection and herbivory.	Seabloom et al. 2017; Cappelli et al. 2019
d, e	A high SLA and a low LDMC are characteristic for species with fast growth rates and high biomass production. Diversity effects causing a shift towards species with higher SLA and lower LDMC should be linked to diversity effects towards higher biomass production.	Wilson et al. 1999; Smart et al. 2017; Breitschwerdt et al. 2019
g	Pathogenic fungi and herbivorous insects are both primary consumer of plants and many theories about the drivers of infection and herbivory, which are identical for both. There are for example plant characteristics linking to both high infection and high herbivory.	(Schädler et al. 2003; Cappelli et al. 2019; Raffa et al. 2019)
h	Species with fast growth, which is indicated by high SLA are more susceptible to fungal pathogens. If diversity leads to higher SLA, then this should also lead to higher infection and diversity effects of pathogen infection and SLA should be positively correlated.	(Cappelli et al. 2019)
i	Species with fast growth, which is indicated by high SLA are more susceptible to fungal pathogens. As SLA and LDMC are negatively correlated to each other, diversity effects of pathogen infection and LDMC are expected to be negatively correlated to each other	Cappelli et al. 2019; Garnier et al. 2001
k, l	The palatability of plant leaves is positively related to specific leaf area and water content (which is directly negatively related to LDMC). Therefore the diversity effects of herbivory are expected to be positively correlated with SLA and negatively with LDMC.	Schädler et al. 2003
n	SLA and LDMC are usually negatively correlated and thus, the diversity effects on SLA and LDMC are expected to be also negatively correlated	Garnier et al. 2004

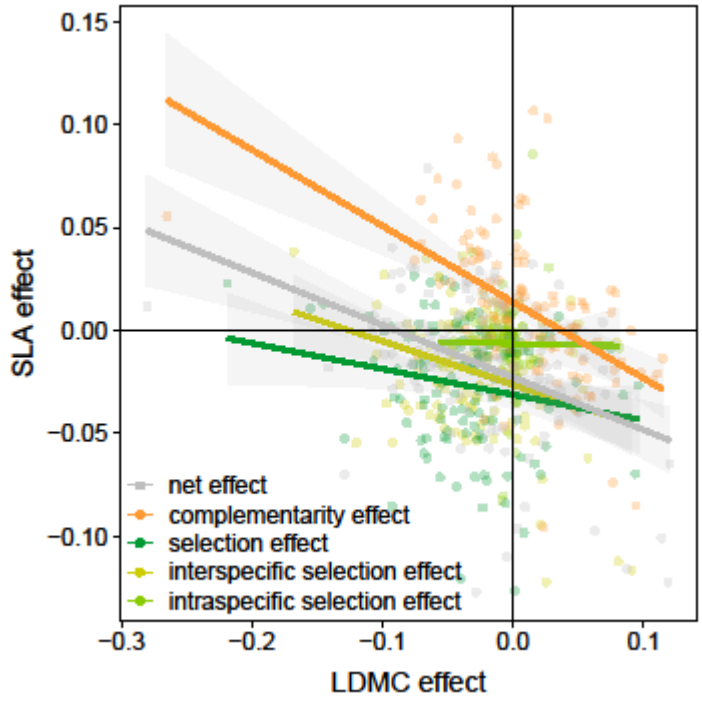


Figure S8 Correlations between net effect, complementarity effect, SE, SEAS and SEPS of SLA and LDMC (means per plot over the seasons)

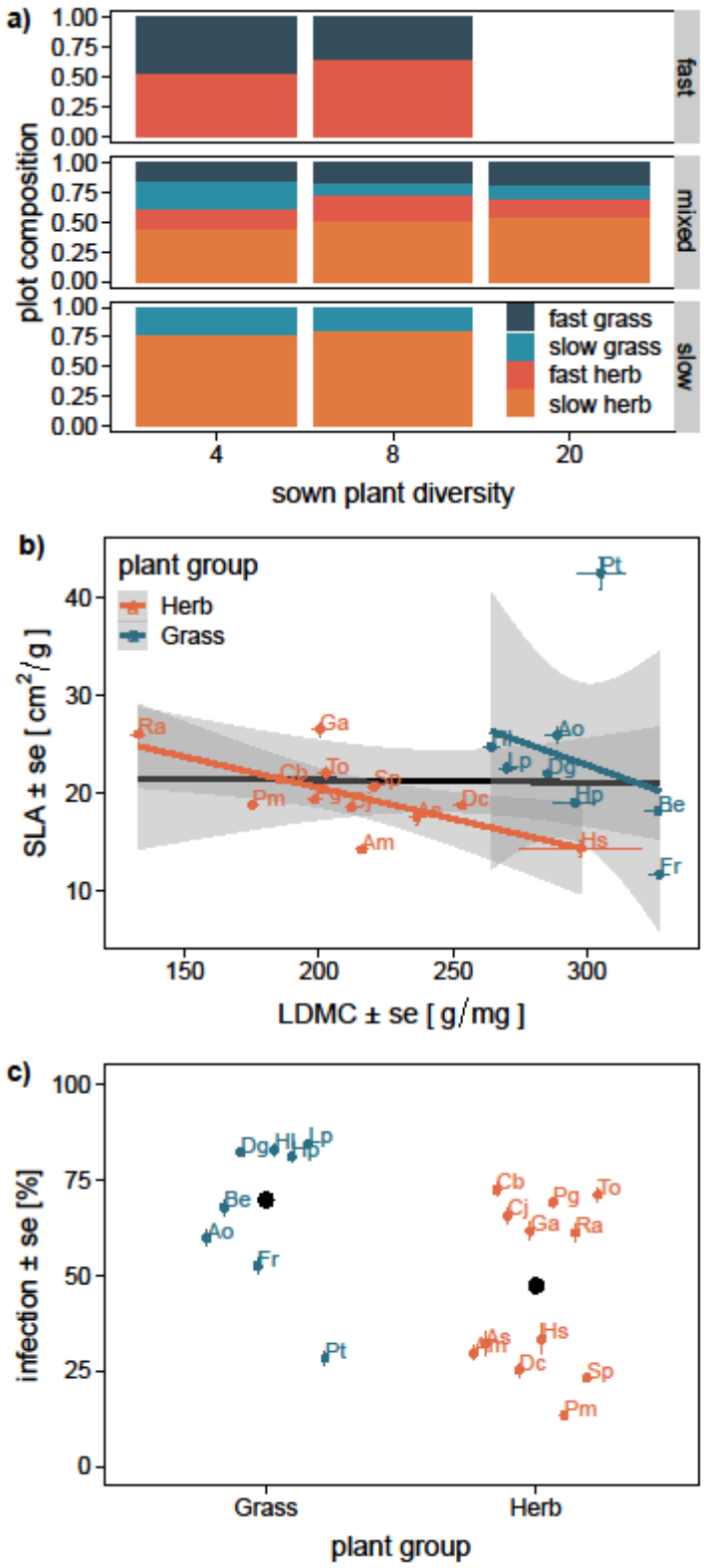


Figure S9 additional illustrations, about how plot composition, mainly the abundance of herbs and grasses affect additive partitioning of SLA, LDMC and infection. Herbs: *Achillea millefolium* (Am), *Anthriscus sylvestris* (As), *Centaurea jacea* (Cj), *Crepis biennis* (Cb), *Daucus carota* (Dc) *Galium album* (Ga), *Heracleum sphondylium* (Hs) *Plantago media* (Pm) *Prunella grandiflora* (Pg) *Rumex acetosa* (Sp), *Taraxacum officinale* (To); Grasses: *Anthoxanthum odoratum* (Ao), *Bromus erectus* (Be), *Dactylis glomerata* (Dg), *Festuca rubra* (Fr), *Helictotrichon pubescens* (Hp), *Holcus lanatus* (Hl), *Lolium perenne* (Lp), *Poa trivialis* (Pt)

a) the average plot composition per diversity and functional composition illustrates thedominance of slow growing herbs over the other plant groups.

b) Correlation between SLA and LDMC for all experimental species (black), herbal species only (blue) and grass species only (red). SLA and LDMC are negatively correlated with each other, but only within plant group and not across al plants, as grasses have generally higher LDMC than herbs.

c) Mean percentage infection ± se of all the species and separated between grasses and herbs. Grasses have in average higher infection than herbs.

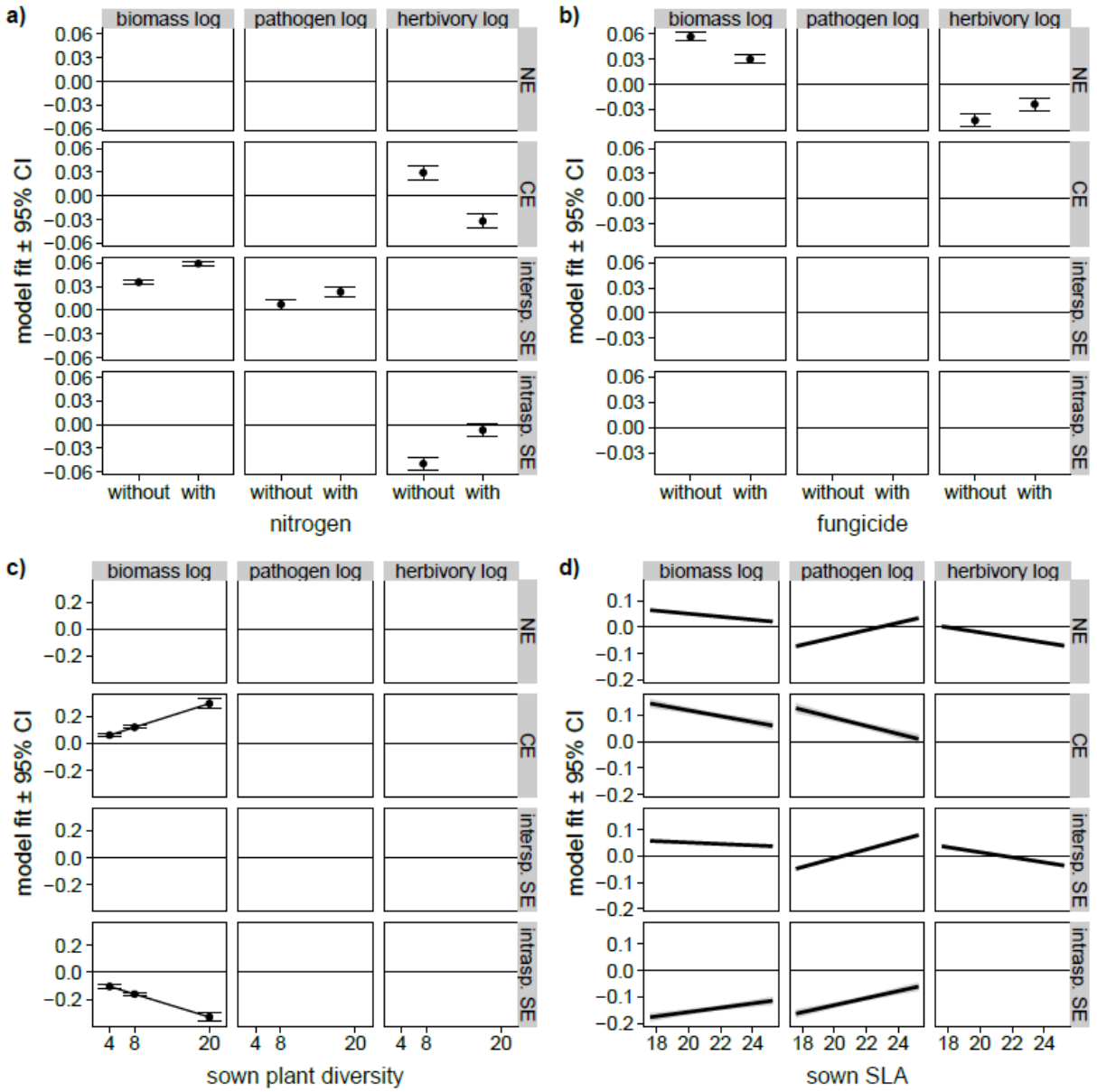


Figure S10 significant effect of a) nitrogen, b) fungicide, c) sown plant species richness and d) sown specific leaf area (SLA) on net effect, complementarity effect, intra- and interspecific selection effect of biomass, pathogen and herbivory. Estimates and CI obtained from the effects package (Fox 2003).

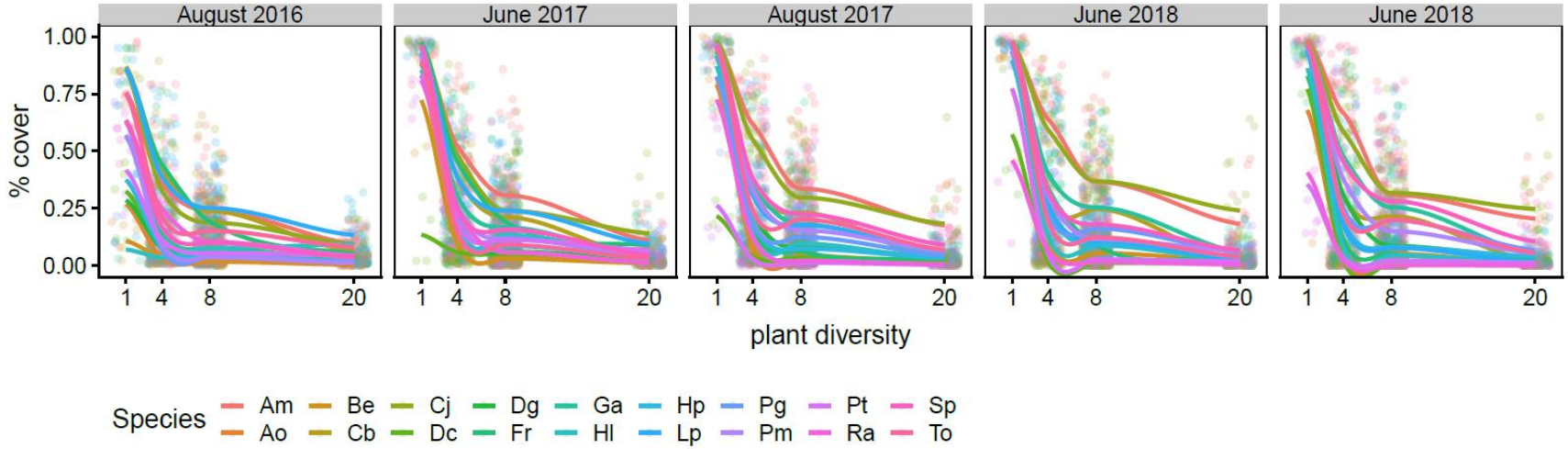


Figure S11 The relationship between plant species abundances and plant species richness in a plot per species. The strongest decrease in abundance occur between the plots with one and four species.

Table S5 Biodiversity effects on biomass production. Linear mixed effects models results.

	Biomass net effect log					complementarity effect log					selection effect log					interspecific selection effect log					intraspecific selection effect log				
Fixed Effects	esti-mate	SE	Df	X2	p-value	esti-mate	SE	Df	X2	p-value	esti-mate	SE	Df	X2	p-value	esti-mate	SE	Df	X2	p-value	esti-mate	SE	Df	X2	p-value
Intercept	0.0433	0.0094				0.1054	0.0355				-0.125	0.0485				0.0474	0.0054				-0.147	0.0318			
Nitrogen			1	-1.609	1.000			1	0.785	0.376			1	-5.993	1.000	0.024	0.008	1	5.906	0.015 *			1	1.425	0.233
Fungicide	-0.027	0.008	1	10.815	0.001 **			1	2.074	0.150	0.014	0.029	1	3.964	0.046 *			1	0.225	0.635			1	0.169	0.681
Plant Diversity			1	1.889	0.169	0.057	0.012	1	20.623	0.000 ***	-0.055	0.011	1	23.479	0.000 ***			1	0.609	0.435	-0.055	0.011	1	25.852	0.000 ***
Sown SLA	-0.022	0.007	1	11.717	0.001 ***	-0.042	0.017	1	5.895	0.015 *			1	2.091	0.148	-0.010	0.004	1	5.615	0.018 *	0.031	0.016	1	3.880	0.049 *
MPD of Sown SLA			1	0.010	0.922			1	0.111	0.739			1	0.115	0.734			1	0.006	0.939			1	0.076	0.783
Random Effects	Var	SD				Var	SD				Var	SD				Var	SD				Var	SD			
1|Block	0.0001	0.0118				0.0004	0.0210				0.0002	0.0151				0.0000	0.0069				0.0004	0.0212			
1|Plot	0.0037	0.0605				0.0155	0.1244				0.0116	0.1078				0.0010	0.0322				0.0115	0.1073			
1|composition	0.0006	0.0252				0.0000	0.0001				0.0000	0.0000				0.0001	0.0116				0.0000	0.0000			
1|compsotion x Harvest						0.0219	0.1480				0.0210	0.1447				0.0006	0.0254				0.0204	0.1430			
1|Harvest	0.0000	0.0062				0.0000	0.0000				0.0000	0.0008				0.0000	0.0007				0.0021	0.0461			
1|Harvest-F						0.0132	0.1150				0.0129	0.1137				0.0001	0.0079				0.0068	0.0825			
Fungicide|Harvest						0.0030	0.0551				0.0037	0.0609				0.0001	0.0119				0.0032	0.0568			
1|Harvest-N	0.0003	0.0168				0.0041	0.0637				0.0042	0.0645				0.0000	0.0029				0.0028	0.0527			
Nitrogen|Harvest	0.0006	0.0248				0.0072	0.0849				0.0069	0.0831				0.0003	0.0164				0.0063	0.0793			

T able S6 Biodiversity effects on pathogen infection. Linear mixed effects models results.

	Pathogen Infection net effect log					complementarity effect log					selection effect log					interspecific selection effect log					intraspecific selection effect log				
Fixed Effects	esti-mate	SE	Df	X2	p-value	esti-mate	SE	Df	X2	p-value	esti-mate	SE	Df	X2	p-value	esti-mate	SE	Df	X2	p-value	esti-mate	SE	Df	X2	p-value
Intercept	-0.019	0.0199				0.0747	0.0183				-0.087	0.0316				0.0154	0.0164				-0.112	0.0196			
Nitrogen			1	0.007	0.932			1	1.648	0.199			1	2.501	0.114	0.016	0.007	1	5.469	0.019 *			1	1.127	0.288
Fungicide			1	3.707	0.054			1	1.766	0.184			1	2.206	2.206			1	0.322	0.570			1	0.382	0.537
Plant Diversity			1	1.381	0.240			1	0.174	0.677			1	0.818	0.366			1	0.052	0.820			1	1.258	0.262
Sown SLA	0.053	0.010	1	22.885	0.000 ***	-0.058	0.017	1	9.856	0.002 **	0.111	0.019	1	31.020	0.000 ***	0.064	0.009	1	39.642	0.000 ***	0.051	0.017	1	9.269	0.002 **
MPD of Sown SLA			1	0.050	0.823			1	0.003	0.958			1	0.037	0.848			1	-0.053	1.000			1	0.026	0.871
Random Effects	Var	SD				Var	SD				Var	SD				Var	SD				Var	SD			
1|Block	0.0000	0.0000				0.0002	0.0137				0.0004	0.0190				0.0000	0.0000				0.0004	0.0201			
1|Plot	0.0034	0.0585				0.0232	0.1524				0.0211	0.1452				0.0023	0.0484				0.0162	0.0162			
1|composition	0.0020	0.0449				0.0019	0.0431				0.0000	0.0001				0.0006	0.0245				0.0000	0.0000			
1|compsotion x Harvest											0.0186	0.1362				0.0040	0.0632				0.0146	0.1209			
1|Harvest	0.0018	0.0426				0.0010	0.0317				0.0039	0.0626				0.0012	0.0351				0.0000	0.0000			
1|Harvest-F						0.0000	0.0000									0.0001	0.0086				0.0011	0.0334			
Fungicide|Harvest						0.0087	0.0933									0.0011	0.0325				0.0055	0.0738			
1|Harvest-N																									
Nitrogen|Harvest																									

Table S7 Biodiversity effects on herbivory. Linear mixed effects models results.

	Herbivory net effect log					complementarity effect log					selection effect log					interspecific selection effect log					intraspecific selection effect log				
Fixed Effects	esti-mate	SE	Df	X2	p-value	esti-mate	SE	Df	X2	p-value	esti-mate	SE	Df	X2	p-value	esti-mate	SE	Df	X2	p-value	esti-mate	SE	Df	X2	p-value
Intercept	-0.033	0.0158				-0.001	0.0203				-0.031	0.0097				0.0004	0.0098				-0.028	0.0149			
Nitrogen			1	0.702	0.402	-0.061	0.012	1	24.035	0.000 ***	0.049	0.012	1	15.771	0.000 ***			1	2.910	0.088	0.042	0.010	1	15.703	0.000 ***
Fungicide	0.019	0.008	1	5.452	0.020 *			1	0.799	0.371	0.028	0.012	1	5.218	0.022 *			1	0.941	0.332			1	1.680	0.195
Plant Diversity			1	0.613	0.434			1	0.071	0.789			1	0.863	0.353			1	0.003	0.957			1	2.384	0.123
Sown SLA	-0.037	0.012	1	8.857	0.003 ***			1	1.418	0.234	-0.046	0.013	1	10.741	0.001 **	-0.036	0.012	1	9.264	0.002 **			1	2.271	0.132
MPD of Sown SLA			1	0.025	0.875			1	0.011	0.918			1	0.036	0.851			1	0.005	0.942			1	0.049	0.825
Random Effects	Var	SD				Var	SD				Var	SD				Var	SD				Var	SD			
1|Block	0.0004	0.0196				0.0010	0.0308				0.0001	0.0082				0.0001	0.0111				0.0004	0.0190			
1|Plot	0.0037	0.0610				0.0083	0.0909				0.0085	0.0923				0.0031	0.0554				0.0060	0.0776			
1|composition	0.0033	0.0579				0.0000	0.0000				0.0013	0.0366				0.0025	0.0502				0.0001	0.0090			
1|compsotion x Harvest											0.0039	0.0623				0.0017	0.0415								
1|Harvest	0.0003	0.0167				0.0005	0.0230				0.0000	0.0000				0.0000	0.0000				0.0002	0.0144			
1|Harvest-F																0.0001	0.0098								
Fungicide|Harvest																0.0006	0.0251								
1|Harvest-N																									
Nitrogen|Harvest																									

Table S8 Biodiversity effects on specific leaf area (SLA). Linear mixed effects models results.

	SLA net effect					complementarity effect					selection effect					interspecific selection effect					intraspecific selection effect				
Fixed Effects	esti-mate	SE	Df	X2	p-value	esti-mate	SE	Df	X2	p-value	esti-mate	SE	Df	X2	p-value	esti-mate	SE	Df	X2	p-value	esti-mate	SE	Df	X2	p-value
Intercept	-0.017	0.0055				0.0106	0.0047				-0.027	0.0043				-0.021	0.0046				-0.006	0.002			
Nitrogen	0.017	0.005	1	13.905	0.000 ***	0.019	0.006	1	9.349	0.002 **			1	0.138	0.71			1	0.038	0.844			1	0.794	0.373
Fungicide			1	0.730	0.393			1	1.359	0.244			1	0.588	0.443			1	0.191	0.662			1	0.438	0.508
Plant Diversity			1	0.309	0.578			1	1.566	0.211			1	2.391	0.122			1	0.762	0.383			1	2.202	0.138
Sown SLA			1	2.670	0.102	-0.014	0.004	1	9.681	0.002 **			1	-0.018	1.000			1	0.820	0.365	0.008	0.002	1	9.549	0.002 **
MPD of Sown SLA			1	0.003	0.959			1	0.081	0.777			1	0.133	0.715			1	0.004	0.947			1	0.001	0.970
Random Effects	Var	SD				Var	SD				Var	SD				Var	SD				Var	SD			
1|Block	0.0000	0.0061				0.0000	0.0068				0.0000	0.0000				0.0000	0.0000				0.0000	0.0014			
1|Plot	0.0005	0.0226				0.0008	0.0290				0.0004	0.0206				0.0003	0.0163				0.0003	0.0159			
1|composition	0.0003	0.0177				0.0000	0.0000				4.9250	0.0070				0.0003	0.0175				0.0000	0.0029			
1|compsotion x Harvest	0.0005	0.0214									0.0008	0.0275				0.0005	0.0222								
1|Harvest	0.0000	0.0000				0.0000	0.0041				0.0000	0.0000				0.0000	0.0000				0.0000	0.0019			
1|Harvest-F																0.0000	0.0001								
Fungicide|Harvest																0.0001	0.0095								
1|Harvest-N											0.0014	0.0014				0.0000	0.0000								
Nitrogen|Harvest											0.0003	0.0176				0.0002	0.0135								

Table S9 Biodiversity effects on leaf dry matter content (LDMC). Linear mixed effects models results.

	LDMC net effect					complementarity effect					selection effect					interspecific selection effect					intraspecific selection effect				
Fixed Effects	esti-mate	SE	Df	X2	p-value	esti-mate	SE	Df	X2	p-value	esti-mate	SE	Df	X2	p-value	esti-mate	SE	Df	X2	p-value	esti-mate	SE	Df	X2	p-value
Intercept	-0.024	0.0115				0.0093	0.0084				-0.039	0.007				-0.03	0.0063				-0.007	0.003			
Nitrogen			1	1.924	0.165			1	0.505	0.477			1	0.501	0.479			1	0.280	0.597			1	0.940	0.332
Fungicide	-0.022	0.010	1	5.245	0.022 *			1	0.706	0.401			1	0.686	0.407			1	0.553	0.457			1	2.999	0.083
Plant Diversity			1	0.246	0.620			1	0.112	0.738			1	0.117	0.733			1	0.117	0.732			1	0.121	0.728
Sown SLA			1	0.643	0.423			1	0.212	0.646			1	0.349	0.555			1	1.968	0.161			1	2.226	0.136
MPD of Sown SLA			1	0.012	0.913			1	0.001	0.972			1	0.029	0.865			1	0.002	0.963			1	0.145	0.703
Random Effects	Var	SD				Var	SD				Var	SD				Var	SD				Var	SD			
1|Block	0.0003	0.0167				0.0002	0.0139				0.0000	0.0027				0.0000	0.0067				0.0000	0.0000			
1|Plot	0.0024	0.0492				0.0018	0.0429				0.0016	0.0399				0.0012	0.0344				0.0002	0.0147			
1|composition	0.0000	0.0000				0.0001	0.0081				0.0000	0.0000				0.0003	0.0168				0.0000	0.0054			
1|compsotion x Harvest	0.0019	0.0431									0.0014	0.0377													
1|Harvest	0.0000	0.0045				0.0000	0.0020				0.0000	0.0000				0.0000	0.0000				0.0000	0.0034			
1|Harvest-F											0.0001	0.0119				0.0001	0.0087								
Fungicide|Harvest											0.0008	0.0276				0.0004	0.0192								
1|Harvest-N																									
Nitrogen|Harvest																									

Chapter 5
Summary and general conclusions

Summary

Fungal plant pathogens are ubiquitous and have the potential to substantially influence their host communities (Fisher et al. 2012; Allan et al. 2010; Mordecai 2011). Understanding the role of fungal pathogens in plant communities is important if we are to predict the consequences of global change for these communities.

In this thesis, I quantified the relative importance of different drivers of infection, which are often altered by the global change driver nitrogen enrichment. Nitrogen can directly affect infection by altering the nutritional status of the host plants and indirectly by changing the functional composition and the diversity of the host plant community. As diversity loss is a major concern of global change, I investigated in more detail how different effects of plant diversity influence fungal pathogen infection and how this compares to diversity effects on other ecosystem functions. Further, I investigated how changes in fungal pathogen infection affected the host communities.

We measured infection in the PaNDiv experiment based on incidence rather than severity, (e.g. leaf area infected). We did this because different pathogen groups vary in their visibility and comparisons between groups would have been difficult in case of a severity-based measurement. There is a difference between pathogens whose mycelium grows on the surface of the leave such as powdery mildews and pathogens that grow mostly leaf-internally like rusts. The latter are often only visible where they sporulate, even though their mycelium covers much larger areas (Klenke 2015). Pathogen incidence and severity can vary in their response to different drivers of infection (Blaser 2014), even though they were correlated (Chapter 2). To test for differences between the responses of incidence and severity we measured damaged leaf area on a subset of the plots on 25 random leaves per species in fall 2018. Only fungicide had an influence on the leaf area infected in a community. It reduced the infected leaf area by 40.63% (analysis not shown). This is similar to the results of Blaser (2014), who found relatively few effects of the tested variables on disease severity, compared to disease intensity. The two measures might be useful for studying different aspects of diseases in plant communities. Since disease incidence seems to respond more strongly to drivers of infection, it might be a better measure to

understand disease dynamics in complex environments. The disease severity is perhaps more informative in terms of how much the species are affected by the pathogens (e.g. Mitchell 2003).

Contrary to our expectations, we did not find direct effects of nitrogen enrichment on fungal infection in any of the chapters. Studies which find large effects of nitrogen enrichment on fungal disease looked either at agricultural species (Dordas 2008; Veresoglou et al. 2013) or on whole plant communities (Liu et al. 2016). Studies looking at single species in natural habitats find contrasting results (Blaser 2014; Veresoglou et al. 2013; Mitchell et al. 2003; Lau et al. 2008). This means that the large effects of nitrogen enrichment in other studies is likely due to indirect effects through changes in community composition. The results of Chapter 4 show that under nitrogen fertilization, heavily infected species increased in abundance at the cost of less infected species. This is in line with the results of Liu et al. (2017) and Blumenthal et al. (2009), who found that mainly disease-susceptible species benefitted from nitrogen enrichment. In Chapter 2, we showed that this was linked to the growth strategy of the plants, as mainly fast growing species increased in abundance following nitrogen fertilization.

Growth strategy, as measured by the proxy of specific leaf area was the main driver of infection, supporting the growth-defense trade-off hypothesis. This agrees with studies finding growth-defense trade-offs for mammal (Lind et al. 2013) and insect herbivores (Endara and Coley 2011), as well as for microbial pathogens (Blumenthal et al. 2009). Species with high specific leaf area were the most heavily infected (Chapter 3). This scaled up to whole plant communities. The more fast-growing species dominated a community, the higher community level infection became (Chapter 2). Results from Chapter 3 showed that this was solely due to the high abundance of heavily infected species, but not due to associational susceptibility and spillover of pathogens from fast to slow-growing species. This was further underpinned by the correlation of interspecific diversity effects for specific leaf area and infection (Chapter 4), meaning that when fast growing species increased in abundance, heavily infected species simultaneously increase. The lack of spillover from fast to slow-growing species is surprising, as fast-growth increases the chance that a species becomes a source for spillover (Cronin et al. 2010). These results strongly support the growth-defense trade-off mechanisms between species. However, we did not find an influence of population level variation in specific leaf area on infection, which hints that within species growth-

defense trade-off did not occur (Chapter 3). It may be that traits of the leaf economics spectrum are not suitable to predict within-species growth-defense trade-off (Züst and Agrawal 2017), because the traits are not so tightly correlated with each other between species (Anderegg et al. 2018). However, other studies found mixed results regarding within species growth-defense trade-off and it is likely that between species growth-defense trade-off was more important (Heckman et al. 2019; Cole et al. 2016; Züst et al. 2015).

We did not find evidence for pathogen spillover from fast to slow species, but our results suggest that pathogen spillover occurs between closely related species. With increasing abundance of grasses, all grass species had increased infection (Chapter 3). The grass species we used in the experiment are phylogenetically very closely related (Durka and Michalski 2012) and are known to share many pathogens (Klenke 2015; Spear and Mordecai 2018). This pattern is reflected also within the rust species which I identified: the plant species have specific rusts, but there are two rusts, *Puccinia graminis* and *P. coronata*, which are shared between multiple grass species (Table 2). Studies investigating the role of spillover often use grass communities as a study system, because of shared pathogens between grasses (e.g. Mordecai 2013; Power and Mitchell 2004; Borer et al. 2007; Spear and Mordecai 2018). These studies show how pathogen spillover can significantly alter community assembly. Depending on how strongly each host species is affected by a generalist pathogen and how efficiently each host species passes the pathogen on to con- and heterospecifics, the presence of the pathogen can facilitate invasion, or lead to coexistence or priority effects (Mordecai 2013; Borer et al. 2007). These generalist pathogens depend on the availability of all their hosts together, and not on the abundance single species (Young et al. 2017; Gilbert and Webb 2007; Parker et al. 2015). This might explain why we found rather weak (single) host concentration effects in Chapter 3 compared to other studies which find strong host concentration effects (e.g. Knops et al. 1999; Mitchell et al. 2003). The results of Chapter 4 suggest that mainly species with low infection suffer from spillover when grown in diverse communities. Species with high infection are more likely to benefit from lower abundances in diverse communities. These two mechanisms balance each other out. Spillover from heavily infected species to closely related species in diverse communities is probably the reason why we did not observe overall diversity effects in Chapter 2. Even though we did not measure spillover directly, our results suggest that it plays a major role in diverse communities in driving infection

in species with elsewise low infection.

Table 2 rust species found on plants in October 2017. The two rusts shared between many grasses, Puccinia coronata and P. graminis are highlighted in bold.

Plant species	Rust species
Achillea millefolium	*Puccinia millefolii*
Anthoxanthum odoratum	***Puccinia graminis***
Bromus erectus	***Puccinia coronata*** *Puccinia symphyti-bromorum*
Crepis biennis	*Puccinia praecox*
Centaurea jacea	*Puccinia centaureae* *Puccinia jaceae*
Dactylis glomerata	***Puccinia coronata*** ***Puccinia graminis*** *Puccinia striiformioides* *Uromyces dactylidis*
Festuca rubra	*Uromyces festucae*
Galium album	*Puccinia galii-verni* *Puccinia punctata*
Holcus lanatus	***Puccinia coronata***
Helictotrichon pubescens	***Puccinia graminis***
Lolium perenne	***Puccinia coronata*** ***Puccinia graminis***
Poa trivialis	***Puccinia coronata*** ***Puccinia graminis***
Rumex acetosa	*Puccinia acetosae*
Taraxacum officinale	*Puccinia sylvatica* *Puccinia taraxaci* *Puccinia variabilis*

Infection did not necessarily reduce biomass production. Species with high infection did not benefit most from fungicide treatment (Chapter 3) and the effect of infection on community biomass was context dependent (Chapter 2). How strongly single species are affected by pathogen infection depends on their tolerance (Haukioja and Koricheva 2000). Often it is assumed that fast-growing species are tolerant and that tolerance trades off with defense. (Roy et al. 2000; Chase et al. 2000). However, our results rather suggest a trade-off between tolerance and resource acquisition, in addition to an independent growth-defense trade-off. At the population level, the impact of infection increased with increasing plant diversity. It could be that the species in diverse communities had lower pathogen resistance than in species poor communities, as pathogen pressure and selection for resistance is likely higher in species poor communities. Additionally, the pathogen communities can vary significantly between

species poor and species rich plant communities (e.g. Blaser 2014; Rottstock et al. 2014), which could further influence the consequences of infection. However, from the observed pathogen groups, only the most abundant group, the leaf spots caused biomass loss. How compositional changes in fungal community composition affects plant communities is an interesting field for future studies. In addition to biomass loss, we also observed an impact of fungal infection on the composition of the plant communities. When fungal pathogens were suppressed with fungicide, fast-growing species were able to increase in abundance at the cost of slower growing species (Chapter 2). This effect might have been more pronounced without weeding the communities. It suggests that at least under some circumstances fast-growing species suffer more from infection than slow-growing species. These results show that the impact of fungal pathogen infection is context dependent and complex.

In Chapter 4 we compared diversity effects for pathogen infection with diversity effects for other functions. The additive partitioning framework used to do so was originally developed to understand how diversity affects plant biomass production (Loreau and Hector 2001; Fox 2005), but it has recently been suggested for other functions as well (Grossiord et al. 2013). So far, this has only been attempted a few times (Pires et al. 2018; Roscher et al. 2018b; Fox and Rauch 2009; Grossiord et al. 2013). The reason for this might be that using the additive partitioning framework for other functions requires additional assumptions. A major question that arises is how to deal with species that should have been in a species mixture, but disappeared or failed to establish. The biomass of such species in the polyculture is simply zero. However, we cannot measure the function of these species in the mixture and their hypothetical function at infinitely small abundance is likely not zero. One option would be to ignore the species that are absent and adjust the species diversity. This would mean ignoring processes that have led to the loss of the species. In this thesis, we substituted the missing measurements with the corresponding monoculture value. By doing so, we considered the loss of the species, but ignored their intraspecific shifts and thus potentially underestimated the complementarity effects and the intraspecific selection effects. This could be the reason why we found the strongest diversity effects for biomass. However, we also found rather strong diversity effects for pathogen infection compared to the other functions and the methodological decision cannot explain these differences. Further, it is not clear yet how suitable the additive partitioning framework is for proportional data such as the infection measure used in this thesis. Proportional

data have an upper limit and infection cannot exceed 100%. Biomass production on the contrary can (in theory) always increase. Our data indicates that proportional data could be problematic when many heavily infected species are included in the study. This issue warrants further investigation but exceeds the scope of this thesis.

A potential problem of additive partitioning is the high importance of the monoculture functioning of a species. The monoculture values are included in all the calculations of the diversity effects (Loreau and Hector 2001; Fox 2005). Whether a species has above- or below-average monoculture functioning compared to the other species in a plot, defines in which direction intra- and interspecific changes in this species influence its contribution to the selection effect. Mistakes or random variation in the monoculture measurements can therefore greatly influence selection and complementarity effects. Replicated monocultures would be ideal to ensure accurate monoculture measurements, but for logistical reasons we were not able to do that. Each replication would have required forty additional plots.

Something else which should be considered especially when interpreting the complementarity effect, is that few species with large proportional inter- and intraspecific shifts can drive diversity effects (Mahaut et al. 2019). In our experiment, this was sometimes the case for infection. Few species with large increases in infection caused positive complementarity effects, even though the majority of the species rather had negative changes.

The additive partitioning framework is useful to categorize diversity effects and compare them between functions despite these potential pitfalls. To my knowledge, the diversity interaction modelling approach is the only alternative way to calculate effects analogous to selection and complementarity (Kirwan et al. 2009; Connolly et al. 2013; Dooley et al. 2015; Brophy et al. 2017). However, diversity interaction modelling cannot separate intra- and interspecific shifts and it requires the estimation of a large number of parameters and thus a large amount of data. Therefore, additive partitioning can help to understand the consequences of biodiversity loss and the underlying mechanisms of biodiversity-ecosystem functioning relationships, but results should always be interpreted in light of the above mentioned points.

In PaNDiv, we found consistent negative selection effects and neutral to positive complementarity effects for all functions. On average, the species increased functioning for most functions (positive complementarity effects). The negative

selection effects were mainly due to intraspecific shifts, which means that the species became more similar in functioning in the mixtures relative to their monocultures. Therefore, species with high functioning in monoculture were driving functioning of polycultures less than expected. This shows that diversity has the same broad effects on different functions. However, there were different species involved in driving these patterns for the different functions and the diversity effects for different functions had inconsistent context dependencies. This indicates that the mechanisms underlying the diversity effects varied between the functions. The results highlight the importance of a high species diversity for the maintenance of multiple ecosystem functions (Hector and Bagchi 2007; Isbell et al. 2011). The lacking link between the diversity effects for pathogen infection and herbivory are especially surprising. Foliar fungal pathogens and herbivorous insects are both primary consumers of plants and share a common niche (Raffa et al. 2019; Thaler et al. 2012). Theories, such as the growth-defense trade-off hypothesis or the resource concentration hypothesis are used for both groups (e.g. Halliday et al. 2017; Endara and Coley 2011; Liu et al. 2017) and the same defense mechanisms can be involved in the regulation of both enemy groups (Thaler et al. 2012). The results of Chapter 4 clearly indicate that herbivory and pathogen infection need to be studied separately to fully understand the role of higher trophic levels in ecosystems.

Outlook

We used fungicide to manipulate the access of fungal pathogens to their hosts. The use of pesticides is probably the only way to manipulate infection in a large-scale field experiment. We substantially reduced infection, but we could not completely remove all pathogens, despite adding a second fungicide in 2018. By mostly removing rusts and powdery mildews, we changed the composition of the fungal pathogen community (Chapter 2). Selectively removing pathogen species can influence the consequences of infection, because it likely benefits some plant species more than others, while complete removal of natural enemies would remove pathogen pressure from all host plants (Crawley and Pacala 1991). The results showed that the fungal community surviving the fungicide treatment had greater negative consequences for biomass production. It is possible that we favored more aggressive pathogens with the fungicide treatments, but it might also be that the observed negative consequences were due to the removal of endophytic mutualists or the suppression of hyperparasitic fungi. It would be interesting to study the effects of single fungal guilds alone and in

combination with artificial infection studies in growth chambers to avoid such non-target effects of fungicide. Infection experiments would allow identifying the pathogens that drive the observed patterns. It would help to find out which pathogens have the most detrimental impact on the plant communities, whether all pathogens are regulated with growth-defense trade-offs or which fungal pathogens mostly spillover between species.

To study the impact of pathogen community composition, pathogen species must be identified. I found visual identification difficult, labor intensive, and uncertain. Molecular methods have led and continue to lead, to substantial changes in the nomenclature and phylogeny of fungal pathogens. Many species have been split into several different species, and apparently different species lumped into a single one, which hints that visual identification alone is not appropriate to study pathogen communities (Klenke 2015). I am happy to know that the project “Impact of global change on phyllosphere microbiome in grasslands” of my colleagues Nadia Maaroufi and Anne Kempel was funded to genetically characterize the fungal communities in PaNDiv. I hope that their results will advance our understanding of the ecological role of fungal community composition.

A high diversity in the pathogen community (e.g. Blaser 2014; Rottstock et al. 2014), likely requires a high diversity of defense mechanisms. Microbial pathogens in general first need to enter their host. They can do so by penetrating the cuticle and cell walls by digesting these mechanical barriers with secreted enzymes or toxins, as for example powdery mildews do (Magendans and Dekker 1966). Some, like the haploid-dikaryotic stage (uredo spores) of rust fungi use natural openings such as stomata to enter their hosts (Klebahn 1904). Others rely on the help of other organisms, which create wounds in the plants through their own feeding or even directly transport the pathogen to and into the plant as vectors (Møller, Murphy 2018). Once inside the hosts, the pathogens have different strategies to access the resources of their hosts. There are three main strategies: necrotrophic pathogens attack their hosts with cell wall degrading molecules. This kills the attacked plant cells and makes their content available to the pathogen. Biotrophic pathogens, such as rusts or mildews, feed on substrate provided by their host, but cause only minimal damage to the host cells. There are also pathogens with an initial biotrophic stage, but later become necrotrophic (hemibiotrophic pathogens). To access the resources of the host plants, microbial

pathogens have a large array of effector molecules, which can change the plant's structure, metabolism, or hormonal regulation to the benefit of the pathogen (Møller, Murphy 2018).

To deal with this large variety of pathogen strategies plants have evolved many different defense mechanisms. These mechanisms can be broadly categorized into constitutive and induced structural and chemical defenses (Walters 2011). For example, a thick cuticle or cell wall should reduce the invasion success of pathogens which try to break down this first line of defense or trichomes might hinder the access of vectors to the host plant (Møller, Murphy 2018). An example for a chemical constitutive defense is resin, which apart from mechanically blocking potential entrance ways for pathogens also contains antifungal and antibiotic substances (Kolosova and Bohlmann 2012). For induced defenses, the plant needs to recognize the attack as an attack in the first place. The plants may recognize the pathogen directly or react to inflicted damage (Møller, Murphy 2018). Once detected, the plants can start to react. Many of the induced defense mechanisms are linked to one of two hormonal pathways: the jasmonate and salicylate pathways. Salicylic acid is thought to be mainly involved in fending off biotrophic pathogens like rusts or powdery mildews, while the jasmonic acid pathway is mostly involved in triggering defenses against necrotrophic fungi like many leaf spots (Thaler et al. 2012). However, the attack strategies of individual pathogens are so highly diverse that there is likely no defense strategy against large groups of pathogens and for each pathogen, a very specific set of defense mechanisms is necessary. Many of these very specific defense mechanisms are known from agricultural species or model organisms. Their role in natural communities remains largely unexplored. For example, a high diversity of defense mechanism in a plant community might contribute to positive diversity-ecosystem functioning relationships.

The growth-defense trade-off hypothesis assumes increased defense in slow growing species and indeed our results support that hypothesis. However, given the many possibilities of defense, it is possible that not all of them are necessarily tightly linked to the growth strategy (Züst and Agrawal 2017). In the PaNDiv experiment, specific leaf area linked to infection and thus overall defense (Chapter 2, Chapter 3). Leaf dry weight and leaf area, two physical properties define the specific leaf area. It is possible that specific leaf area captures structural defenses, but chemical defenses less consistently (Abdala-Roberts et al. 2018). This is critical, as different defense

mechanisms might have different trade-offs. For example, induced and constitutive defenses trade off with each other (Koricheva et al. 2004), and only the constitutive defenses trade off with competitive ability (Kempel et al. 2011). Similarly, our results suggest that total defense trades off with growth strategy, while tolerance is rather linked to resource acquisition (Chapter 3). The ideal mixture of defense (and tolerance) mechanisms might be very context dependent. The role of multiple defense trade-offs in whole plant communities is an interesting field for future studies.

As mentioned, tolerance is a strategy to cope with natural enemies in addition to defense. In the field, we were not able to measure tolerance as such. However, the results hint that tolerance might play an important role in plant-pathogen interactions and should be considered in future research, especially since it can have trade-offs that are independent of defense and growth-strategy. While there have been some attempts to study the role of herbivory tolerance (e.g. Kempel et al. 2019; Gianoli and Salgado-Luarte 2017), less is known about pathogen tolerance. The challenge lies in inflicting a given amount of pathogen damage to a plant. Herbivory can be simulated by clipping parts of a plant, but since pathogens do not directly remove plant tissue, mimicking pathogen damage is more complicated. A possibility could be to inoculate fixed proportions of leaves of a plant with fungal spores to manipulate disease intensity. Fitness or biomass in response to the proportion of (successfully) inoculated leaves could serve as a measure of tolerance.

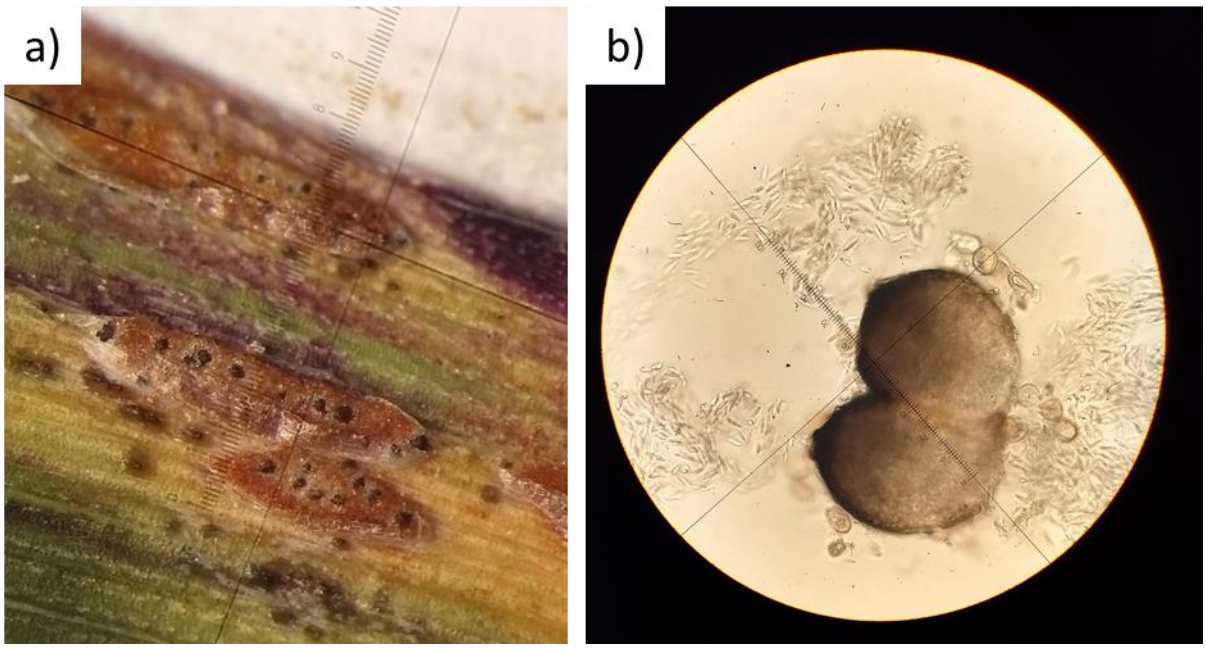


Figure 5 Eudarluca caricis parasitizing Puccinia graminis on a) Helictotrichon pubescens and b) Anthoxanthum odoratum in October 2017.

In this thesis, I focused on interactions between fungal pathogens and their hosts in plant communities. However, there are many other organisms in the ecosystem with which the fungal pathogens can interact, and which might influence the establishment and spread of fungal diseases. There is a large variety of hyperparasitic fungi that exploit fungal pathogens and there are known examples of insects that consume fungal pathogens (Klenke 2015). We observed *Eudarluca caricis* infection on many rusts on PaNDiv (Figure 5), despite not actively searching for them. Thus, natural enemies of fungal pathogens are likely common and abundant, yet their importance for example for top down pathogen control has hardly been studied so far (Klenke 2015, but see Tollenaere et al. 2014).

Final Conclusions

In this thesis, I addressed different drivers of pathogen infection. By factorially manipulating different drivers I could assess their relative importance and understand direct and indirect mechanisms. I showed that growth-defense trade-off of species is a main driver of infection. This trade-off at the species level is reflected at the population level; high abundances of fast-growing species meant high community level infection. Further, we found effects of host concentration. Both species abundances and the abundance of closely related species increased infection. Diversity reduced the abundances of species but seemed to have no additional effects. This was probably because of contrasting effects linked to diversity. Species with high infection benefitted from reduced abundances, while relatively resistant species with low infection when grown alone rather suffered from spillover. Similar effects have been observed for other ecosystem functions. Species, which were able to provide high level of functioning when grown alone, decreased their functioning when grown in mixtures and species, which were not as good at providing the same function when grown only with conspecifics increased functioning in the mixtures. The diversity effects were relatively strong for fungal infection compared to the other functions. Despite the comparable patterns across functions, the underlying mechanisms likely differed. Different species contributed to different functions and the diversity effects for different functions occurred mostly independently from each other. These results contribute to our understanding of the role of fungal pathogens in natural ecosystems.

References

Abdala-Roberts, Luis; Galman, Andrea; Petry, William K.; Covelo, Felisa; La Fuente, Maria de; Glauser, Gaetan; Moreira, Xoaquin (2018): Interspecific variation in leaf functional and defensive traits in oak species and its underlying climatic drivers. In *PLoS One* 13 (8). DOI: 10.1371/journal.pone.0202548.

Alexander, Helen M.; Antonovics, Janis; Rauscher, Mark D. (1985): Relationship of phenotypic and genetic variation in *Plantago lanceolata* to disease caused by *Fusarium moniliforme* var. *subglutinans*. In *Oecologia* 65 (1), pp. 89–93. DOI: 10.1007/BF00384468.

Alexander, Helen M.; Burdon, J. J. (1984): The effect of disease induced by Albugo candida (white rust) and Peronospora parasitica (downy mildew) on the survival and reproduction of Capsella burna-pastoris (shepherd's purse). In *Oecologia* 64 (3), pp. 314–318. DOI: 10.1007/BF00379127.

Alexander, Helen M.; Mihail, J. D. (2000): Seedling disease in an annual legume: consequences for seedling mortality, plant size, and population seed production. In *Oecologia* 122 (3), pp. 346–353. DOI: 10.1007/s004420050040.

Allan, Eric; van Ruijven, Jasper; Crawley, Michael J. (2010): Foliar fungal pathogens and grassland biodiversity. In *Ecology* 91 (9), pp. 2572–2582. DOI: 10.1890/09-0859.1.

Anderegg, Leander D. L.; Berner, Logan T.; Badgley, Grayson; Sethi, Meera L.; Law, Beverly E.; HilleRisLambers, Janneke (2018): Within-species patterns challenge our understanding of the leaf economics spectrum. In *Ecol. Lett.* 21 (5), pp. 734–744. DOI: 10.1111/ele.12945.

Anderson, T. Michael; Kumordzi, Bright B.; Fokkema, Wimke; Fox, Hugo Valls; Olff, Han (2013): Distinct physiological responses underlie defoliation tolerance in African lawn and bunch grasses. In *International journal of plant sciences* 174 (5), pp. 769–778. DOI: 10.1086/670237.

Anderson, V. J.; Briske, D. D. (1995): Herbivore-induced species replacement in grasslands - is it driven by herbivory tolerance or avoidance. In *Ecol. Appl.* 5 (4), pp. 1014–1024. DOI: 10.2307/2269351.

Archivgruppe Moosseedorf (2012): Die drei Absenkungen des Moossees 1780, 1855-1859, 1917-1920. With assistance of Franz Bill, Markus Senn, Daniel Suter, Esther Zesiger. Gemeinde Moosseedorf. Available online at http://www.moosseedorf.ch/de/inhalte/portrait/Absenkungen-Moossee-1780-1920.php, checked on 11/27/2019.

Bachmann, Dorte; Roscher, Christiane; Buchmann, Nina (2018): How do leaf trait values

change spatially and temporally with light availability in a grassland diversity experiment? In *OIKOS* 127 (7), pp. 935–948. DOI: 10.1111/oik.04533.

Barbosa, Pedro; Hines, Jessica; Kaplan, Ian; Martinson, Holly; Szczepaniec, Adrianna; Szendrei, Zsofia (2009): Associational Resistance and Associational Susceptibility: Having Right or Wrong Neighbors. In *Annu. Rev. Ecol. Evol. Syst.* 40, pp. 1–20. DOI: 10.1146/annurev.ecolsys.110308.120242.

Barrett, Luke G.; Kniskern, Joel M.; Bodenhausen, Natacha; Zhang, Wen; Bergelson, Joy (2009): Continua of specificity and virulence in plant host-pathogen interactions: causes and consequences. In *New Phytol.* 183 (3), pp. 513–529. DOI: 10.1111/j.1469-8137.2009.02927.x.

Barry, Kathryn E.; Mommer, Liesje; van Ruijven, Jasper; Wirth, Christian; Wright, Alexandra J.; Bai, Yongfei et al. (2019): The Future of Complementarity: Disentangling Causes from Consequences. In *Trends Ecol Evol* 34 (2), pp. 167–180. DOI: 10.1016/j.tree.2018.10.013.

Barthelemy, Hélène; Dorrepaal, Ellen; Olofsson, Johan (2019): Defoliation of a grass is mediated by the positive effect of dung deposition, moss removal and enhanced soil nutrient contents: results from a reindeer grazing simulation experiment. In *OIKOS* 128 (10), pp. 1515–1524. DOI: 10.1111/oik.06310.

Bates, Douglas; Mächler, Martin; Bolker, Ben; Walker, Steve (2015): Fitting Linear Mixed-Effects Models Using lme4. In *J. Stat. Softw.* 67 (1), pp. 1–48. DOI: 10.18637/jss.v067.i01.

Bergmann, Joana; Ryo, Masahiro; Prati, Daniel; Hempel, Stefan; Rillig, Matthias C. (2017): Root traits are more than analogues of leaf traits: the case for diaspore mass. In *The New phytologist* 216 (4), pp. 1130–1139. DOI: 10.1111/nph.14748.

Bever, James D.; Mangan, Scott A.; Alexander, Helen M. (2015): Maintenance of Plant Species Diversity by Pathogens. In *Annu. Rev. Ecol. Evol. Syst.* 46, pp. 305–325. DOI: 10.1146/annurev-ecolsys-112414-054306.

Bever, James D.; Westover, Kristi M.; Antonovics, Janis (1997): Incorporating the Soil Community into Plant Population Dynamics. The Utility of the Feedback Approach. In *J. Ecol.* 85 (5), pp. 561–573. Available online at http://www.jstor.org/stable/2960528.

Blaser, Stefan (2014): Effects of land use on the diversity of fungi in central European forests and grasslands. PhD. University of Bern, Bern. Institute of Plant Sciences.

Bluethgen, Nico; Dormann, Carsten F.; Prati, Daniel; Klaus, Valentin H.; Kleinebecker, Till; Hoelzel, Norbert et al. (2012): A quantitative index of land-use intensity in grasslands: Integrating mowing, grazing and fertilization. In *Basic Appl. Ecol.* 13 (3), pp. 207–220. DOI: 10.1016/j.baae.2012.04.001.

Blumenthal, Dana; Mitchell, Charles E.; Pysek, Petr; Jarosik, Vojtech (2009): Synergy between

pathogen release and resource availability in plant invasion. In *Proc. Natl. Acad. Sci. U. S. A.* 106 (19), pp. 7899–7904. DOI: 10.1073/pnas.0812607106.

Blumenthal, Dana M. (2006): Interactions between resource availability and enemy release in plant invasion. In *Ecol Lett* 9 (7), pp. 887–895. DOI: 10.1111/j.1461-0248.2006.00934.x.

Bobbink, Roland; Hicks, K.; Galloway, J.; Spranger, T.; Alkemade, R.; Ashmore, M. et al. (2010): Global assessment of nitrogen deposition effects on terrestrial plant diversity: a synthesis. In *Ecol. Appl.* 20 (1), pp. 30–59. DOI: 10.1890/08-1140.1.

Borer, Elizabeth T.; Hosseini, Parviez R.; Seabloom, Eric W.; Dobson, Andrew P. (2007): Pathogen-induced reversal of native dominance in a grassland community. In *Proc. Natl. Acad. Sci. U. S. A.* 104 (13), pp. 5473–5478. DOI: 10.1073/pnas.0608573104.

Borer, Elizabeth T.; Lind, Eric M.; Ogdahl, Eric J.; Seabloom, Eric W.; Tilman, David; Montgomery, Rebecca A.; Kinkel, Linda L. (2015): Food-web composition and plant diversity control foliar nutrient content and stoichiometry. In *J. Ecol.* 103 (6), pp. 1432–1441. DOI: 10.1111/1365-2745.12461.

Bradley, D. J.; Gilbert, G. S.; Im Parker (2003): Susceptibility of clover species to fungal infection: The interaction of leaf surface traits and environment. In *Am. J. Bot.* 90 (6), pp. 857–864. DOI: 10.3732/ajb.90.6.857.

Bregaglio, Simone; Donatelli, Marcello; Confalonieri, Roberto (2013): Fungal infections of rice, wheat, and grape in Europe in 2030-2050. In *Agron. Sustain. Dev.* 33 (4), pp. 767–776. DOI: 10.1007/s13593-013-0149-6.

Breitschwerdt, Eva; Jandt, Ute; Bruelheide, Helge (2019): Trait–performance relationships of grassland plant species differ between common garden and field conditions. In *Ecol Evol* 9 (4), pp. 1691–1701. DOI: 10.1002/ece3.4818.

Brophy, Caroline; Dooley, Áine; Kirwan, Laura; Finn, John A.; McDonnell, Jack; Bell, Thomas et al. (2017): Biodiversity and ecosystem function: making sense of numerous species interactions in multi-species communities. In *Ecology* 98 (7), pp. 1771–1778. DOI: 10.1002/ecy.1872.

Burdon, J. J. (1991): Fungal Pathogens as Selective Forces in Plant Populations and Communities. In *Austral Ecol.* 16 (4), pp. 423–432. DOI: 10.1111/j.1442-9993.1991.tb01072.x.

Burdon, J. J.; Chilvers, G. A. (1982): Host density as a factor in plant-disease ecology. In *Annu. Rev. Phytopathol.* 20, pp. 143–166. DOI: 10.1146/annurev.py.20.090182.001043.

Burdon, Jeremy J.; Thrall, Peter H.; Ericson, Lars (2006): The current and future dynamics of disease in plant communities. In *Annu. Rev. Phytopathol.* 44, pp. 19–39. DOI: 10.1146/annurev.phyto.43.040204.140238.

Cappelli, Seraina L.; Pichon, Noémie A.; Kempel. Anne S. (2019): Sick plants in grassland communities: a growth-defense trade-off is the main driver of fungal pathogen abundance and impact. growth-defense trade-off in a grassland. In *Preprint at bioRxiv*.

Cardinale, Bradley J.; Duffy, J. Emmett; Gonzalez, Andrew; Hooper, David U.; Perrings, Charles; Venail, Patrick et al. (2012): Biodiversity loss and its impact on humanity. In *Nature* 486 (7401), pp. 59–67. DOI: 10.1038/nature11148.

Cardinale, Bradley J.; Matulich, Kristin L.; Hooper, David U.; Byrnes, Jarrett E.; Duffy, Emmett; Gamfeldt, Lars et al. (2011): The functional role of producer diversity in ecosystems. In *Am J Bot* 98 (3), pp. 572–592. DOI: 10.3732/ajb.1000364.

Cardinale, Bradley J.; Srivastava, Diane S.; Duffy, J. Emmett; Wright, Justin P.; Downing, Amy L.; Sankaran, Mahesh; Jouseau, Claire (2006): Effects of biodiversity on the functioning of trophic groups and ecosystems. In *Nature* 443 (7114), pp. 989–992. DOI: 10.1038/nature05202.

Carris, Lori M.; Little, Christopher R.; Stiles, Carol M. (2012): Introduction to fungi. The plant health instructor. The American Phytopathological Society (APS). St. Paul, MN 55121 USA. Available online at https://www.apsnet.org/edcenter/disandpath/fungalasco/intro/Pages/IntroFungi.aspx, checked on 11/12/2019.

Castagneyrol, Bastien; Jactel, Hervé; Vacher, Corinne; Brockerhoff, Eckehard G.; Koricheva, Julia (2014): Effects of plant phylogenetic diversity on herbivory depend on herbivore specialization. In *JOURNAL OF APPLIED ECOLOGY* 51 (1), pp. 134–141. DOI: 10.1111/1365-2664.12175.

Chase, Jonathan M.; Leibold, Mathew A.; Simms, Ellen (2000): Plant tolerance and resistance in food webs: community-level predictions and evolutionary implications. In *Evol. Ecol.* 14 (4), pp. 289–314. DOI: 10.1023/A:1010983611618.

Chen, Wanquan; Wellings, Colin; Chen, Xianming; Kang, Zhengsheng; Liu, Taiguo (2014): Wheat stripe (yellow) rust caused by Puccinia striiformis f. sp.tritici. In *Mol. Plant Pathol.* 15 (5), pp. 433–446. DOI: 10.1111/mpp.12116.

Chesson, P. (2000): Mechanisms of maintenance of species diversity. In *Annu. Rev. Ecol. Syst.* 31, pp. 343–366. DOI: 10.1146/annurev.ecolsys.31.1.343.

Civitello, David J.; Cohen, Jeremy; Fatima, Hiba; Halstead, Neal T.; Liriano, Josue; McMahon, Taegan A. et al. (2015): Biodiversity inhibits parasites: Broad evidence for the dilution effect. In *Proc. Natl. Acad. Sci. U. S. A.* 112 (28), pp. 8667–8671. DOI: 10.1073/pnas.1506279112.

Cleland, Elsa E.; Harpole, W. Stanley (2010): Nitrogen enrichment and plant communities. In

R. S. Ostfeld, W. H. Schlesinger (Eds.): YEAR IN ECOLOGY AND CONSERVATION BIOLOGY 2010, vol. 1195. COMMERCE PLACE, 350 MAIN STREET, MALDEN 02148, MA USA: WILEY-BLACKWELL (Annals of the New York Academy of Sciences), pp. 46–61.

Cole, Christopher T.; Stevens, Michael T.; Anderson, Jon E.; Lindroth, Richard L. (2016): Heterozygosity, gender, and the growth-defense trade-off in quaking aspen. In *Oecologia* 181 (2), pp. 381–390. DOI: 10.1007/s00442-016-3577-6.

Coley, P. D. (1988): Effects of plant growth rate and leaf lifetime on the amount and type of anti-herbivore defense. In *Oecologia* 74 (4), pp. 531–536. DOI: 10.1007/BF00380050.

Coley, P. D.; Bryant, J. P.; Chapin, F. S. (1985): Resource Availability and Plant Antiherbivore Defense. In *Science* 230 (4728), pp. 895–899. DOI: 10.1126/science.230.4728.895.

Connell, J. H. (1971): On the role of natural enemies in preventing competitive exclusion in some marine animals and in rain forest trees. In P. J. den ed Boer, G. R. ed Gradwell (Eds.): Dynamics of populations. Proceedings of the Advanced Study Institute on Dynamics of numbers in populations, Oosterbeek, the Netherlands, 7-18 September 1970 / Ed. by P. J. Den Boer and G. R. Gradwell. Wageningen: Pudoc, pp. 298–312.

Connolly, John; Bell, Thomas; Bolger, Thomas; Brophy, Caroline; Carnus, Timothee; Finn, John A. et al. (2013): An improved model to predict the effects of changing biodiversity levels on ecosystem function. In *Journal of Ecology* 101 (2), pp. 344–355. DOI: 10.1111/1365-2745.12052.

Coughenour, Michael B. (1985): Graminoid Responses to Grazing by Large Herbivores: Adaptations, Exaptations, and Interacting Processes. In *Annals of the Missouri Botanical Garden* 72 (4), p. 852. DOI: 10.2307/2399227.

Craven, Dylan; Isbell, Forest; Manning, Pete; Connolly, John; Bruelheide, Helge; Ebeling, Anne et al. (2016): Plant diversity effects on grassland productivity are robust to both nutrient enrichment and drought. In *Philosophical transactions of the Royal Society of London. Series B, Biological sciences* 371 (1694). DOI: 10.1098/rstb.2015.0277.

Crawley, Michael J.; Pacala, S. W. (1991): Herbivory and plant diversity. In Catherine A. Toft, André Aeschlimann, Liana Bolis (Eds.): Parasite-host associations. Coexistence or conflict? : 9th International congress of comparative physiology : Selected papers / edited by Catherine A Toft, André Aeschlimann and Liana Bolis, pp. 157–173.

Cronin, James Patrick; Rúa, Megan A.; Mitchell, Charles E. (2014): Why Is Living Fast Dangerous? Disentangling the Roles of Resistance and Tolerance of Disease. In *Am. Nat.* 184 (2), pp. 172–187. DOI: 10.1086/676854.

Cronin, James Patrick; Welsh, Miranda E.; Dekkers, Martin G.; Abercrombie, Samuel T.;

Mitchell, Charles E. (2010): Host physiological phenotype explains pathogen reservoir potential. In *Ecol. Lett.* 13 (10), pp. 1221–1232. DOI: 10.1111/j.1461-0248.2010.01513.x.

de Kroon, Hans; Hendriks, Marloes; van Ruijven, Jasper; Ravenek, Janneke; Padilla, Francisco M.; Jongejans, Eelke et al. (2012): Root responses to nutrients and soil biota: drivers of species coexistence and ecosystem productivity. In *Journal of Ecology* 100 (1), pp. 6–15. DOI: 10.1111/j.1365-2745.2011.01906.x.

De Schrijver, An; Frenne, Pieter de; Ampoorter, Evy; van Nevel, Lotte; Demey, Andreas; Wuyts, Karen; Verheyen, Kris (2011): Cumulative nitrogen input drives species loss in terrestrial ecosystems. In *Glob. Ecol. Biogeogr.* 20 (6), pp. 803–816. DOI: 10.1111/j.1466-8238.2011.00652.x.

De Vries, Franciska T.; Manning, Pete; Tallowin, Jerry R. B.; Mortimer, Simon R.; Pilgrim, Emma S.; Harrison, Kathryn A. et al. (2012): Abiotic drivers and plant traits explain landscape-scale patterns in soil microbial communities. In *Ecol. Lett.* 15 (11), pp. 1230–1239. DOI: 10.1111/j.1461-0248.2012.01844.x.

Delarze, Raymond (2015): Lebensräume der Schweiz. Ökologie - Gefährdung - Kennarten. 3., vollst. überarb. Aufl.: Bern : Ott.

Derroire, Géraldine; Powers, Jennifer S.; Hulshof, Catherine M.; Cárdenas Varela, Luis E.; Healey, John R. (2018): Contrasting patterns of leaf trait variation among and within species during tropical dry forest succession in Costa Rica. In *Sci. Rep.* 8 (1), p. 285. DOI: 10.1038/s41598-017-18525-1.

Desprez-Loustau, Marie-Laure; Marçais, Benoit; Nageleisen, Louis-Michel; Piou, Dominique; Vannini, Andrea (2006): Interactive effects of drought and pathogens in forest trees. In *Ann. For. Sci.* 63 (6), pp. 597–612. DOI: 10.1051/forest:2006040.

Dillen, Mathias; Smit, Christian; Buyse, Martijn; Hofte, Monica; Clercq, Patrick de; Verheyen, Kris (2017): Stronger diversity effects with increased environmental stress: A study of multitrophic interactions between oak, powdery mildew and ladybirds. In *PLoS One* 12 (4). DOI: 10.1371/journal.pone.0176104.

DiTommaso, A.; Aarssen, L. W. (1989): Resource manipulations in natural vegetation: a. In *VEGETATIO* 84 (1), pp. 9–29. DOI: 10.1007/BF00054662.

Dooley, Áine; Isbell, Forest; Kirwan, Laura; Connolly, John; Finn, John A.; Brophy, Caroline (2015): Testing the effects of diversity on ecosystem multifunctionality using a multivariate model. In *Ecol Lett* 18 (11), pp. 1242–1251. DOI: 10.1111/ele.12504.

Dordas, Christos (2008): Role of nutrients in controlling plant diseases in sustainable agriculture. A review. In *Agron. Sustain. Dev.* 28 (1), pp. 33–46. DOI: 10.1051/agro:2007051.

Durka, Walter; Michalski, Stefan G. (2012): Daphne: a dated phylogeny of a large European flora for phylogenetically informed ecological analyses. In *Ecology* 93 (10), p. 2297. DOI: 10.1890/12-0743.1.

Eisenhauer, Nico (2012): Aboveground–belowground interactions as a source of complementarity effects in biodiversity experiments. In *Plant Soil* 351 (1-2), pp. 1–22. DOI: 10.1007/s11104-011-1027-0.

Endara, María-José; Coley, Phyllis D. (2011): The resource availability hypothesis revisited: a meta-analysis. In *Funct. Ecol.* 25 (2), pp. 389–398. DOI: 10.1111/j.1365-2435.2010.01803.x.

Fargione, Joseph; Tilman, David; Dybzinski, Ray; Hille Ris Lambers, Janneke; Clark, Chris; Harpole, W. Stanley et al. (2007): From selection to complementarity: shifts in the causes of biodiversity-productivity relationships in a long-term biodiversity experiment. In *Proc Natl Acad Sci U S A* 274 (1611), pp. 871–876. DOI: 10.1098/rspb.2006.0351.

Fay, Philip A.; Prober, Suzanne M.; Harpole, W. Stanley; Knops, Johannes M. H.; Bakker, Jonathan D.; Borer, Elizabeth T. et al. (2015): Grassland productivity limited by multiple nutrients. In *Nat. Plants* 1 (7). DOI: 10.1038/NPLANTS.2015.80.

Firn, Jennifer; McGree, James M.; Harvey, Eric; Flores-Moreno, Habacuc; Schütz, Martin; Buckley, Yvonne M. et al. (2019): Leaf nutrients, not specific leaf area, are consistent indicators of elevated nutrient inputs. In *Nat. Ecol. Evol.* 3 (3), pp. 400–406. DOI: 10.1038/s41559-018-0790-1.

Fisher, Matthew C.; Henk, Daniel. A.; Briggs, Cheryl J.; Brownstein, John S.; Madoff, Lawrence C.; McCraw, Sarah L.; Gurr, Sarah J. (2012): Emerging fungal threats to animal, plant and ecosystem health. In *Nature* 484 (7393), pp. 186–194. DOI: 10.1038/nature10947.

Fokkema, N. J.; Nooij, M. P. de (1981): The Effect of Fungicides on the Microbial Balance in the Phyllosphere1. In *EPPO Bulletin* 11 (3), pp. 303–310. DOI: 10.1111/j.1365-2338.1981.tb01937.x.

Fort, Florian; Cruz, Pablo; Lecloux, Eric; Oliveira, Leandro Bittencourt de; Stroia, Ciprian; Theau, Jean-Pierre; Jouany, Claire (2016): Grassland root functional parameters vary according to a community-level resource acquisition-conservation trade-off. In *JOURNAL OF VEGETATION SCIENCE* 27 (4), pp. 749–758. DOI: 10.1111/jvs.12405.

Fox, J. W. (2005): Interpreting the ‘selection effect’ of biodiversity on ecosystem function. In *Ecol Lett* 8 (8), pp. 846–856. DOI: 10.1111/j.1461-0248.2005.00795.x.

Fox, Jeremy W.; Rauch, Gisep (2009): Partitioning the mechanisms by which genetic diversity of parasite infections affects total parasite load. In *OIKOS* 118 (10), pp. 1507–1514. DOI: 10.1111/j.1600-0706.2009.17547.x.

Fox, John (2003): Effect Displays in R for Generalised Linear Models. In *J. Stat. Softw.* 8 (15). DOI: 10.18637/jss.v008.i15.

Galloway, James N.; Townsend, Alan R.; Erisman, Jan Willem; Bekunda, Mateete; Cai, Zucong; Freney, John R. et al. (2008): Transformation of the nitrogen cycle: Recent trends, questions, and potential solutions. In *Science* 320 (5878), pp. 889–892. DOI: 10.1126/science.1136674.

Garnier, E.; Cortez, J.; Billes, G.; Navas, M. L.; Roumet, C.; Debussche, M. et al. (2004): Plant functional markers capture ecosystem properties during secondary succession. In *Ecology* 85 (9), pp. 2630–2637. DOI: 10.1890/03-0799.

Garnier, E.; Shipley, B.; Roumet, C.; Laurent, G. (2001): A standardized protocol for the determination of specific leaf area and leaf dry matter content. In *Funct. Ecol.* 15 (5), pp. 688–695. DOI: 10.1046/j.0269-8463.2001.00563.x.

Gattlen, Nicolas (2016): Biodiversitätsförderung in der Landwirtschaft. Monotonie im Kulturland. Edited by Bundesamt für Umwelt (BAFU). Available online at https://www.bafu.admin.ch/bafu/de/home/themen/ernaehrung-wohnen-mobilitaet/dossiers/ernaerung-dossiers/magazin-umwelt-landwirtschaft-ernaehrung/monotonie-im-kulturland--biodiversitaetsfoerderung-in-der-landwi.html, checked on 1/15/2019.

Gianoli, Ernesto; Salgado-Luarte, Cristian (2017): Tolerance to herbivory and the resource availability hypothesis. In *Biology letters* 13 (5). DOI: 10.1098/rsbl.2017.0120.

Gilbert, Gregory S.; Webb, Campbell O. (2007): Phylogenetic signal in plant pathogen-host range. In *Proc. Natl. Acad. Sci. U. S. A.* 104 (12), pp. 4979–4983. DOI: 10.1073/pnas.0607968104.

Godoy, Oscar; Gomez-Aparicio, Lorena; Matias, Luis; Perez-Ramos, Ignacio Manuel; Allan, Eric (2019): A mechanistic path to maximize biomass productivity while maintaining diversity.

Goodall, Jeremy; Witkowski, E. T. F.; McConnachie, Andrew J.; Keen, Chris (2012): Altered growth, population structure and realised niche of the weed Campuloclinium macrocephalum (Asteraceae) after exposure to the naturalised rust Puccinia eupatorii (Pucciniaceae). In *Biol. Invasions* 14 (9), pp. 1947–1962. DOI: 10.1007/s10530-012-0205-1.

Grace, James B. (2006): Structural equation modeling and natural systems. Cambridge: Cambridge University Press.

Grossiord, Charlotte; Granier, André; Gessler, Arthur; Scherer-Lorenzen, Michael; Pollastrini, Martina; Bonal, Damien (2013): Application of Loreau & Hector's (2001) partitioning method to complex functional traits. In *Methods Ecol Evol* 80, n/a-n/a. DOI: 10.1111/2041-210X.12090.

Gullino, Maria Lodovica; Pugliese, Massimo; Gilardi, Giovanna; Garibaldi, Angelo (2018): Effect of increased CO2 and temperature on plant diseases: a critical appraisal of results obtained in studies carried out under controlled environment facilities. In *J. Plant Pathol.* 100 (3), pp. 371–389. DOI: 10.1007/s42161-018-0125-8.

Haddad, Nick M.; Haarstad, John; Tilman, David (2000): The effects of long-term nitrogen loading on grassland insect communities. In *Oecologia* 124 (1), pp. 73–84. DOI: 10.1007/s004420050026.

Hahl, Terhi; van Moorsel, Sofia Julia; Schmid, Marc W.; Zuppinger-Dingley, Debra; Schmid, Bernhard; Wagg, Cameron (2017): Diversity loss selects for altered plant phenotypic responses to local arbuscular mycorrhizal communities. In *Preprint at bioRxiv*. DOI: 10.1101/216986.

Halliday, Fletcher W.; Heckman, Robert W.; Wilfahrt, Peter A.; Mitchell, Charles E. (2017): A multivariate test of disease risk reveals conditions leading to disease amplification. In *Proc. R. Soc. Lond., B, Biol. Sci* 284 (1865). DOI: 10.1098/rspb.2017.1340.

Hantsch, Lydia; Bien, Steffen; Radatz, Stine; Braun, Uwe; Auge, Harald; Bruelheide, Helge (2014): Tree diversity and the role of non-host neighbour tree species in reducing fungal pathogen infestation. In *J. Ecol.* 102 (6), pp. 1673–1687. DOI: 10.1111/1365-2745.12317.

Haukioja, E.; Koricheva, J. (2000): Tolerance to herbivory in woody vs. herbaceous plants. In *Evol. Ecol.* 14 (4-6), pp. 551–562. DOI: 10.1023/A:1011091606022.

Hautier, Yann; Seabloom, Eric W.; Borer, Elizabeth T.; Adler, Peter B.; Harpole, W. Stanley; Hillebrand, Helmut et al. (2014): Eutrophication weakens stabilizing effects of diversity in natural grasslands. In *Nature* 508 (7497), p. 521. DOI: 10.1038/nature13014.

Hawksworth, David L.; Lücking, Robert (2017): Chapter 4 : Fungal Diversity Revisited: 2.2 to 3.8 Million Species. In Heitman, Howlett, Crous, Stukenbrock, James, Gow (Eds.): The Fungal Kingdom: American Society of Microbiology.

Heckman, Robert W.; Halliday, Fletcher W.; Mitchell, Charles E. (2019): A growth-defense trade-off is general across native and exotic grasses. In *Oecologia*. DOI: 10.1007/s00442-019-04507-9.

Heckman, Robert W.; Halliday, Fletcher W.; Wilfahrt, Peter A.; Mitchell, Charles E. (2017): Effects of native diversity, soil nutrients, and natural enemies on exotic invasion in experimental plant communities. In *Ecology* 98 (5), pp. 1409–1418. DOI: 10.1002/ecy.1796.

Heckman, Robert W.; Wright, Justin P.; Mitchell, Charles E. (2016): Joint effects of nutrient addition and enemy exclusion on exotic plant success. In *Ecology* 97 (12), pp. 3337–3345. DOI: 10.1002/ecy.1585.

Hector, Andy; Bagchi, Robert (2007): Biodiversity and ecosystem multifunctionality. In *Nature* 448 (7150), pp. 188–190. DOI: 10.1038/nature05947.

Hector, Andy; Felten, Stefanie von; Hautier, Yann; Weilenmann, Maja; Bruelheide, Helge (2012): Effects of Dominance and Diversity on Productivity along Ellenberg's Experimental Water Table Gradients. In *PloS one* 7 (9). DOI: 10.1371/journal.pone.0043358.

Heldstab, J.; Reutiman, J.; Biedermann, R.; Leu, D. (2010): Stickstoffflüsse in der Schweiz. Stoffflussanalyse für das Jahr 2005. Bundesamt für Umwelt (BAFU). Bern.

Helfer, Stephan (2014): Rust fungi and global change. In *New Phytol.* 201 (3), pp. 770–780. DOI: 10.1111/nph.12570.

Henriksen, B.; Elen, O. (2005): Natural Fusarium grain infection level in wheat, barley and oat after early application of fungicides and herbicides. In *JOURNAL OF PHYTOPATHOLOGY* 153 (4), pp. 214–220. DOI: 10.1111/j.1439-0434.2005.00955.x.

Horgan, Finbarr G.; Cruz, Ainara Penalver; Bernal, Carmencita C.; Ramal, Angelee Fame; Almazan, Maria Liberty P.; Wilby, Andrew (2018): Resistance and tolerance to the brown planthopper, Nilaparvata lugens (Stal), in rice infested at different growth stages across a gradient of nitrogen applications. In *FIELD CROPS RESEARCH* 217, pp. 53–65. DOI: 10.1016/j.fcr.2017.12.008.

Huot, Bethany; Yao, Jian; Montgomery, Beronda L.; He, Sheng Yang (2014): Growth-Defense Tradeoffs in Plants: A Balancing Act to Optimize Fitness. In *Mol. Plant* 7 (8), pp. 1267–1287. DOI: 10.1093/mp/ssu049.

Iason, Glenn R.; Taylor, Joanne; Helfer, Stephan (2018): Community-based biotic effects as determinants of tree resistance to pests and pathogens. In *FOREST ECOLOGY AND MANAGEMENT* 417, pp. 301–312. DOI: 10.1016/j.foreco.2018.01.037.

Isbell, Forest; Calcagno, Vincent; Hector, Andy; Connolly, John; Harpole, W. Stanley; Reich, Peter B. et al. (2011): High plant diversity is needed to maintain ecosystem services. In *Nature* 477 (7363), pp. 199–202. DOI: 10.1038/nature10282.

Isbell, Forest; Reich, Peter B.; Tilman, David; Hobbie, Sarah E.; Polasky, Stephen; Binder, Seth (2013): Nutrient enrichment, biodiversity loss, and consequent declines in ecosystem productivity. In *Proc. Natl. Acad. Sci. U. S. A.* 110 (29), pp. 11911–11916. DOI: 10.1073/pnas.1310880110.

Isbell, Forest I.; Losure, David A.; Yurkonis, Kathryn A.; Wilsey, Brian J. (2008): Diversity-productivity relationships in two ecologically realistic rarity-extinction scenarios. In *OIKOS* 117 (7), pp. 996–1005. DOI: 10.1111/j.0030-1299.2008.16692.x.

IUPAC (2016): Pesticide Properties Database. difenoconazole. International Union of Pure and

Applied Chemistry. Available online at http://sitem.herts.ac.uk/aeru/iupac/Reports/230.htm#none, updated on 10/17/2016, checked on 1/6/2017.

Janzen, Daniel H. (1970): Herbivores and the Number of Tree Species in Tropical Forests. In *Am. Nat.* 104 (940), pp. 501–528. DOI: 10.1086/282687.

Jarchow, Meghann E.; Liebman, Matt (2012): Nutrient enrichment reduces complementarity and increases priority effects in prairies managed for bioenergy. In *BIOMASS & BIOENERGY* 36, pp. 381–389. DOI: 10.1016/j.biombioe.2011.11.010.

Jarosz, Andrew M.; Davelos, A. L. (1995): Effects of disease in wild plant populations and the evolution of pathogen aggressiveness. In *New Phytol.* 129 (3), pp. 371–387. DOI: 10.1111/j.1469-8137.1995.tb04308.x.

Jennings, D. H.; Lysek, G. (1996): Fungal biology. Understanding the fungal lifestyle / D. H. Jennings, G. Lysek. Oxford: Bios.

Karlsson, Ida; Friberg, Hanna; Steinberg, Christian; Persson, Paula (2014): Fungicide Effects on Fungal Community Composition in the Wheat Phyllosphere. In *PLoS One* 9 (11). DOI: 10.1371/journal.pone.0111786.

Katasterplan. Plan of the Melioration in Schupfen, Moos, Affoltern, Münchenbuchsee, Deisswil, Wiggiswil, Moosseedorf und Urtenen Scale 1:5000.

Keary, I. P.; Hatcher, P. E. (2004): Combining competition from *Lolium perenne* and an insect-fungus combination to control *Rumex obtusifolius* seedlings. In *Weed Res.* 44 (1), pp. 33–41. DOI: 10.1046/j.1365-3180.2003.00370.x.

Keesing, F.; Holt, R. D.; Ostfeld, R. S. (2006): Effects of species diversity on disease risk. In *Ecol. Lett.* 9 (4), pp. 485–498. DOI: 10.1111/j.1461-0248.2006.00885.x.

Kempel, Anne; Schädler, Martin; Chrobock, Thomas; Fischer, Markus; van Kleunen, Mark (2011): Tradeoffs associated with constitutive and induced plant resistance against herbivory. In *Proc. Natl. Acad. Sci. U. S. A.* 108 (14), pp. 5685–5689. DOI: 10.1073/pnas.1016508108.

Kempel, Anne; Vincent, Hugo; Prati, Daniel; Fischer, Markus (2019): Context dependency of biotic interactions and its relation to plant rarity. In *Preprint at bioRxiv* 22. DOI: 10.1101/791269.

Khalko, S.; Pan, S. K.; others (2009): Phytotoxicity of some fungicides and their compatibility study with a potential biocontrol agent *Trichoderma harzianum*. In *Journal of Crop and Weed* 5 (2), pp. 151–153.

Kirwan, L.; Connolly, J.; Finn, J. A.; Brophy, C.; Lüscher, A.; Nyfeler, D.; Sebastia, M. T. (2009): Diversity-interaction modeling: estimating contributions of species identities and interactions to ecosystem function. In *Ecology* 90 (8), pp. 2032–2038. DOI: 10.1890/08-1684.1.

Klebahn, H. (1904): Die wirtswechselnden Rostpilze. Berlin: Borntraeger.

Klenke, Friedemann (2015): Pflanzenparasitische Kleinpilze. Bestimmungsbuch für Brand-, Rost-, Mehltau-, Flagellatenpilze und Wucherlingsverwandte in Deutschland, Österreich, der Schweiz und Südtirol. With assistance of Markus Scholler. Berlin, Heidelberg: Springer Berlin Heidelberg.

Knogge, W. (1996): Fungal Infection of Plants. In *The Plant cell* 8 (10), pp. 1711–1722. DOI: 10.1105/tpc.8.10.1711.

Knops, J. M.H.; Tilman, D.; Haddad, N. M.; Naeem, S.; Mitchell, C. E.; Haarstad, J. et al. (1999): Effects of plant species richness on invasion dynamics, disease outbreaks, insect abundances and diversity. In *Ecol. Lett.* 2 (5), pp. 286–293. DOI: 10.1046/j.1461-0248.1999.00083.x.

Kolosova, N.; Bohlmann, J. (2012): Conifer Defense Against Insects and Fungal Pathogens. In Rainer Matyssek, Hans Schnyder, Wolfgang Oßwald, Dieter Ernst, Jean Charles Munch, Hans Pretzsch (Eds.): Growth and Defence in Plants: Resource Allocation at Multiple Scales. Berlin, Heidelberg: Springer Berlin Heidelberg, pp. 85–109.

Koricheva, Julia; Nykänen, Heli; Gianoli, Ernesto (2004): Meta-analysis of Trade-offs among Plant Antiherbivore Defenses: Are Plants Jacks-of-All-Trades, Masters of All? In *Am. Nat.* 163 (4), E64-E75. DOI: 10.1086/382601.

Laine, Anna-Liisa (2006): Evolution of host resistance: looking for coevolutionary hotspots at small spatial scales. In *Proc. R. Soc. Lond., B, Biol. Sci* 273 (1584), pp. 267–273. DOI: 10.1098/rspb.2005.3303.

Lanz, Walter (2019): Münchenbuchse, Gebiet Mühle. phone call to Seraina L. Cappelli. Bern, 9/4/2019.

Lau, Jennifer A.; Strengbom, Joachim; Stone, Laurie R.; Reich, Peter B.; Tiffin, Peter (2008): Direct and indirect effects of CO2, nitrogen, and community diversity on plant-enemy interactions. In *Ecology* 89 (1), pp. 226–236. DOI: 10.1890/07-0423.1.

Laughlin, Daniel C. (2011): Nitrification is linked to dominant leaf traits rather than functional diversity. In *Journal of Ecology* 99 (5), pp. 1091–1099. DOI: 10.1111/j.1365-2745.2011.01856.x.

Lavorel, Sandra; Grigulis, Karl (2012): How fundamental plant functional trait relationships scale-up to trade-offs and synergies in ecosystem services. In *Journal of Ecology* 100 (1), pp. 128–140. DOI: 10.1111/j.1365-2745.2011.01914.x.

Lefcheck, Jonathan S.; Byrnes, Jarrett E. K.; Isbell, Forest; Gamfeldt, Lars; Griffin, John N.; Eisenhauer, Nico et al. (2015): Biodiversity enhances ecosystem multifunctionality across

trophic levels and habitats. In *Nature communications* 6, p. 6936. DOI: 10.1038/ncomms7936.

Leggett, Helen C.; Buckling, Angus; Long, Gráinne H.; Boots, Mike (2013): Generalism and the evolution of parasite virulence. In *Trends Ecol. Evol.* 28 (10), pp. 592–596. DOI: 10.1016/j.tree.2013.07.002.

Lind, Eric M.; Borer, Elizabeth; Seabloom, Eric; Adler, Peter; Bakker, Jonathan D.; Blumenthal, Dana M. et al. (2013): Life-history constraints in grassland plant species: a growth-defence trade-off is the norm. In *Ecol. Lett.* 16 (4), pp. 513–521. DOI: 10.1111/ele.12078.

Lipowsky, Annett; Roscher, Christiane; Schumacher, Jens; Michalski, Stefan G.; Gubsch, Marlén; Buchmann, Nina et al. (2015): Plasticity of functional traits of forb species in response to biodiversity. In *Perspect Plant Ecol Evol Syst* 17 (1), pp. 66–77. DOI: 10.1016/j.ppees.2014.11.003.

Liu, Bing; Zhao, WenZhi; Meng, YangYang; Liu, Chan (2018a): Biodiversity, productivity, and temporal stability in a natural grassland ecosystem of China. In *SCIENCES IN COLD AND ARID REGIONS* 10 (4), pp. 293–304. DOI: 10.3724/SP.J.1226.2018.00293.

Liu, Xiang; Chen, Fei; Lyu, Shengman; Sun, Dexin; Zhou, Shurong (2018b): Random species loss underestimates dilution effects of host diversity on foliar fungal diseases under fertilization. In *Ecol. Evol. (Ecology and Evolution)*, n/a-n/a. DOI: 10.1002/ece3.3749.

Liu, Xiang; Lyu, Shengman; Sun, Dexin; Bradshaw, Corey J. A.; Zhou, Shurong (2017): Species decline under nitrogen fertilization increases community-level competence of fungal diseases. In *Proc. R. Soc. Lond., B, Biol. Sci* 284 (1847). DOI: 10.1098/rspb.2016.2621.

Liu, Xiang; Lyu, Shengman; Zhou, Shurong; Bradshaw, Corey J. A. (2016): Warming and fertilization alter the dilution effect of host diversity on disease severity. In *Ecology* 97 (7), pp. 1680–1689. DOI: 10.1890/15-1784.1.

Lively, C. M.; Johnson, S. G.; Delph, L. F.; Clay, K. (1995): Thinning reduces the effect of rust infection on jewelweed (*Impatiens capensis*). In *Ecology* 76 (6), pp. 1859–1862. DOI: 10.2307/1940718.

Loreau, M.; Hector, A. (2001): Partitioning selection and complementarity in biodiversity experiments. In *Nature* 412 (6842), pp. 72–76. DOI: 10.1038/35083573.

Magendans, J. F. C.; Dekker, J. (1966): A microscopic study of powdery mildew on barley after application of the systemic compound wepsyn. In *Netherlands Journal of Plant Pathology* 72 (3), pp. 274–278. DOI: 10.1007/BF02650218.

Mahaut, Lucie; Fort, Florian; Violle, Cyrille; Freschet, Grégoire T. (2019): Multiple facets of diversity effects on plant productivity: species richness, functional diversity, species identity and intraspecific competition. In *Funct Ecol* 0, pp. 1–12. DOI: 10.1111/1365-2435.13473.

Maron, John L.; Marler, Marilyn; Klironomos, John N.; Cleveland, Cory C. (2011): Soil fungal pathogens and the relationship between plant diversity and productivity. In *Ecol Lett* 14 (1), pp. 36–41. DOI: 10.1111/j.1461-0248.2010.01547.x.

Marquard, Elisabeth; Weigelt, Alexandra; Temperton, Vicky M.; Roscher, Christiane; Schumacher, Jens; Buchmann, Nina et al. (2009): Plant species richness and functional composition drive overyielding in a six-year grassland experiment. In *Ecology* 90 (12), pp. 3290–3302. DOI: 10.1890/09-0069.1.

Mason, Chase M.; Bowsher, Alan W.; Crowell, Breanna L.; Celoy, Rhodesia M.; Tsai, Chung-Jui; Donovan, Lisa A. (2016): Macroevolution of leaf defenses and secondary metabolites across the genus Helianthus. In *New Phytol.* 209 (4), pp. 1720–1733. DOI: 10.1111/nph.13749.

MeteoSchweiz (2019): MeteoSwiss IDAWEB. Available online at https://gate.meteoswiss.ch/idaweb/login.do, checked on 1/17/2019.

Mitchell, C. E. (2003): Trophic control of grassland production and biomass by pathogens. In *Ecol. Lett.* 6 (2), pp. 147–155. DOI: 10.1046/j.1461-0248.2003.00408.x.

Mitchell, C. E.; Reich, P. B.; Tilman, D.; Groth, J. V. (2003): Effects of elevated CO2, nitrogen deposition, and decreased species diversity on foliar fungal plant disease. In *Glob. Change Biol.* 9 (3), pp. 438–451. DOI: 10.1046/j.1365-2486.2003.00602.x.

Mitchell, C. E.; Tilman, D.; Groth, J. V. (2002): Effects of grassland plant species diversity, abundance, and composition on foliar fungal disease. In *Ecology* 83 (6), pp. 1713–1726.

Møller, I. M.; Murphy, Angus S. (2018): Plant physiology and development. [International] sixth edition. Edited by Lincoln Taiz, Eduardo Zeiger. Sunderland, Massachusetts, New York, NY: Sinauer Associates; Oxford Univeristy Press.

Mommer, Liesje; Weemstra, Monique (2012): The role of roots in the resource economics spectrum. In *The New phytologist* 195 (4), pp. 725–727. DOI: 10.1111/j.1469-8137.2012.04247.x.

Mordecai, Erin A. (2011): Pathogen impacts on plant communities: unifying theory, concepts, and empirical work. In *Ecol. Monograph* 81 (3), pp. 429–441. DOI: 10.1890/10-2241.1.

Mordecai, Erin A. (2013): Consequences of Pathogen Spillover for Cheatgrass-Invaded Grasslands: Coexistence, Competitive Exclusion, or Priority Effects. In *Am. Nat.* 181 (6), pp. 737–747. DOI: 10.1086/670190.

Nithyameenakshi, S.; Jeyaramraja, P. R.; Manian, S. (2006): Investigations on Phytotoxicity of Two New Fungicides, Azoxystrobin and Difenoconazole. In *American J. of Plant Physiology* 1 (1), pp. 89–98. DOI: 10.3923/ajpp.2006.89.98.

Ochoa-Hueso, Raúl (2016): Non-linear disruption of ecological interactions in response to nitrogen deposition. In *Ecology*, n/a-n/a. DOI: 10.1002/ecy.1521.

Oerke, E. C. (2006): Crop losses to pests. In *J. Agric. Sci.* 144, pp. 31–43. DOI: 10.1017/S0021859605005708.

Parker, Ingrid M.; Saunders, Megan; Bontrager, Megan; Weitz, Andrew P.; Hendricks, Rebecca; Magarey, Roger et al. (2015): Phylogenetic structure and host abundance drive disease pressure in communities. In *Nature* 520 (7548), p. 542. DOI: 10.1038/nature14372.

Paul, N. D. (1989): The Effects of Puccinia Lagenophorae on Senecio Vulgaris in Competition With Euphorbia Peplus. In *J. Ecol.* 77 (2), pp. 552–564. Available online at http://www.jstor.org/stable/2260769.

Paul, N. D.; Ayres, P. G. (1986): Interference between healthy and rusted groundsel (*Senecio vulgaris* L.) within mixed populations of different densities and proportions. In *New Phytol.* 104 (2), pp. 257–269. DOI: 10.1111/j.1469-8137.1986.tb00650.x.

Paul, N. D.; Ayres, P. G. (1987): Water Stress Modifies Intraspecific Interference between Rust (Puccinia lagenophorae Cooke)-Infected and Healthy Groundsel (Senecio vulgaris L.). In *New Phytol.* 106 (3), pp. 555–566. Available online at http://www.jstor.org/stable/2434820.

Paul, N. D.; Ayres, P. G. (1990): Effects of interactions between nutrient supply and rust infection of Senecio vulgaris L. on competition with Capsella bursa-pastoris (L.) Medic. In *New Phytol.* 114 (4), pp. 667–674. DOI: 10.1111/j.1469-8137.1990.tb00438.x.

Paul, N. D.; Ayres, P. G.; Wyness, L. E. (1989): On the use of fungicides for experimentation in natural vegetation. In *Funct. Ecol.* 3 (6), pp. 759–769. DOI: 10.2307/2389509.

Pérez-Harguindeguy, N.; Díaz, S.; Garnier, E.; Lavorel, S.; Poorter, H.; Jaureguiberry, P. et al. (2013): New handbook for standardised measurement of plant functional traits worldwide. In *Aust. J. Bot.* 61 (3), p. 167. DOI: 10.1071/BT12225.

Peters, J. C.; Shaw, M. W. (1996): Effect of artificial exclusion and augmentation of fungal plant pathogens on a regenerating grassland. In *New Phytol.* 134 (2), pp. 295–307. DOI: 10.1111/j.1469-8137.1996.tb04634.x.

Petersen, Jens H. (2012): The kingdom of fungi. Princeton: Princeton University Press.

Pichon, Noémie A.; Cappelli, Seraina; Soliveres, Santiago; Hölzel, Norbert; Klaus, Valentin H.; Kleinebecker, Till; Allan, Eric (2019): Decomposition disentangled: a test of the multiple mechanisms by which nitrogen enrichment alters litter decomposition. In *in review*. DOI: 10.1101/671545.

Pires, Aliny P. F.; Srivastava, Diane S.; Marino, Nicholas A. C.; MacDonald, A. Andrew M.; Figueiredo-Barros, Marcos Paulo; Farjalla, Vinicius F. (2018): Interactive effects of climate

change and biodiversity loss on ecosystem functioning. In *Ecology* 99 (5), pp. 1203–1213. DOI: 10.1002/ecy.2202.

Pontes, Laise da Silveira; Maire, Vincent; Louault, Frederique; Soussana, Jean-Francois; Carrere, Pascal (2012): Impacts of species interactions on grass community productivity under contrasting management regimes. In *Oecologia* 168 (3), pp. 761–771. DOI: 10.1007/s00442-011-2129-3.

Poorter, Hendrik; Niinemets, Ülo; Poorter, Lourens; Wright, Ian J.; Villar, Rafael (2009): Causes and consequences of variation in leaf mass per area (LMA): a meta-analysis. In *New Phytol.* 182 (3), pp. 565–588. DOI: 10.1111/j.1469-8137.2009.02830.x.

Power, A. G.; Mitchell, C. E. (2004): Pathogen spillover in disease epidemics. In *Am. Nat.* 164 (5), S79-S89. DOI: 10.1086/424610.

Procházka, Jan; Brom, Jakub; Šťastný, Jakub; Pecharová, Emilie (2011): The impact of vegetation cover on temperature and humidity properties in the reclaimed area of a brown coal dump. In *Int. J. Min. Reclam. Environ.* 25 (4), pp. 350–366. DOI: 10.1080/17480930.2011.623830.

R Core Team (2018): R: A Language and Environment for Statistical Computing. Vienna, Austria. Available online at https://www.R-project.org/.

Raffa, Kenneth F.; Bonello, Pierluigi; Orrock, John L. (2019): Why do entomologists and plant pathologists approach trophic relationships so differently? Identifying biological distinctions to foster synthesis. In *The New phytologist*. DOI: 10.1111/nph.16181.

Reich, P. B.; Walters, M. B.; Ellsworth, D. S. (1992): Leaf Life-Span in Relation to Leaf, Plant, and Stand Characteristics among Diverse Ecosystems. In *Ecol. Monograph* 62 (3), pp. 365–392. DOI: 10.2307/2937116.

Reich, Peter B.; Cornelissen, Hans (2014): The world-wide 'fast-slow' plant economics spectrum. A traits manifesto. In *J. Ecol.* 102 (2), pp. 275–301. DOI: 10.1111/1365-2745.12211.

Ridenour, W. L.; Callaway, R. M. (2003): Root herbivores, pathogenic fungi, and competition between Centaurea maculosa and Festuca idahoensis. In *Plant Ecol.* 169 (1), pp. 161–170. DOI: 10.1023/A:1026277401640.

Robinson, Ph. W.; Hodges, C. F. (1981): Nitrogen-Induced Changes in the Sugars and Amino Acids of Sequentially Senescing Leaves of Poa pratensis and Pathogenesis by Drechslera sorokinian1). In *JOURNAL OF PHYTOPATHOLOGY* 101 (4), pp. 348–361. DOI: 10.1111/j.1439-0434.1981.tb03355.x.

Rockstrom, Johan; Steffen, Will; Noone, Kevin; Persson, Asa; Chapin, III, F. Stuart; Lambin, Eric et al. (2009): Planetary Boundaries: Exploring the Safe Operating Space for Humanity. In

Ecol. Soc. 14 (2).

Roscher, Christiane; Schmid, Bernhard; Kolle, Olaf; Schulze, Ernst-Detlef (2016): Complementarity among four highly productive grassland species depends on resource availability. In *Oecologia* 181 (2), pp. 571–582. DOI: 10.1007/s00442-016-3587-4.

Roscher, Christiane; Schumacher, Jens; Foitzik, Oliver; Schulze, Ernst-Detlef (2007a): Resistance to rust fungi in *Lolium perenne* depends on within-species variation and performance of the host species in grasslands of different plant diversity. In *Oecologia* 153 (1), pp. 173–183. DOI: 10.1007/s00442-007-0713-3.

Roscher, Christiane; Schumacher, Jens; Gubsch, Marlen; Lipowsky, Annett; Weigelt, Alexandra; Buchmann, Nina et al. (2018a): Origin context of trait data matters for predictions of community performance in a grassland biodiversity experiment. In *Ecology* 99 (5), pp. 1214–1226. DOI: 10.1002/ecy.2216.

Roscher, Christiane; Schumacher, Jens; Gubsch, Marlén; Lipowsky, Annett; Weigelt, Alexandra; Buchmann, Nina et al. (2012): Using plant functional traits to explain diversity-productivity relationships. In *PloS one* 7 (5), e36760. DOI: 10.1371/journal.pone.0036760.

Roscher, Christiane; Schumacher, Jens; Gubsch, Marlén; Lipowsky, Annett; Weigelt, Alexandra; Buchmann, Nina et al. (2018b): Interspecific trait differences rather than intraspecific trait variation increase the extent and filling of community trait space with increasing plant diversity in experimental grasslands. In *Perspect Plant Ecol Evol Syst* 33, pp. 42–50. DOI: 10.1016/j.ppees.2018.05.001.

Roscher, Christiane; Schumacher, Jens; Weisser, Wolfgang W.; Schmid, Bernhard; Schulze, Ernst-Detlef (2007b): Detecting the role of individual species for overyielding in experimental grassland communities composed of potentially dominant species. In *Oecologia* 154 (3), pp. 535–549. DOI: 10.1007/s00442-007-0846-4.

Rosseel, Yves (2012): lavaan: An R Package for Structural Equation Modeling. In *J. Stat. Softw.* 48 (2), pp. 1–36.

Rottstock, Tanja; Joshi, Jasmin; Kummer, Volker; Fischer, Markus (2014): Higher plant diversity promotes higher diversity of fungal pathogens, while it decreases pathogen infection per plant. In *Ecology* 95 (7), pp. 1907–1917.

Roy, B. A. (1993): Floral mimicry by a plant pathogen. In *Nature* 362 (6415), pp. 56–58. DOI: 10.1038/362056a0.

Roy, B. A.; Kirchner, J. W.; Christian, C. E.; Rose, L. E. (2000): High disease incidence and apparent disease tolerance in a North American Great Basin plant community. In *Evol. Ecol.* 14 (4), p. 421. DOI: 10.1023/A:1010997429365.

Roy, Bitty A.; Güsewell, Sabine; Harte, John (2004): Response of plant pathogens and herbivores to a warming experiment. In *Ecology* 85 (9), pp. 2570–2581. DOI: 10.1890/03-0182.

Schädler, M.; Jung, G.; Auge, H.; Brandl, R. (2003): Palatability, decomposition and insect herbivory: patterns in a successional old-field plant community. In *OIKOS* 103 (1), pp. 121–132. DOI: 10.1034/j.1600-0706.2003.12659.x.

Schierenbeck, Matias; Constanza Fleitas, Maria; Julio Miralles, Daniel; Rosa Simon, Maria (2016): Does radiation interception or radiation use efficiency limit the growth of wheat inoculated with tan spot or leaf rust? In *FIELD CROPS RESEARCH* 199, pp. 65–76. DOI: 10.1016/j.fcr.2016.09.017.

Schnitzer, Stefan A.; Klironomos, John N.; HilleRisLambers, Janneke; Kinkel, Linda L.; Reich, Peter B.; Xiao, Kun et al. (2011): Soil microbes drive the classic plant diversity–productivity pattern. In *Ecology* 92 (2), pp. 296–303. DOI: 10.1890/10-0773.1.

Schroeder-Georgi, Thomas; Wirth, Christian; Nadrowski, Karin; Meyer, Sebastian T.; Mommer, Liesje; Weigelt, Alexandra (2016): From pots to plots: hierarchical trait-based prediction of plant performance in a mesic grassland. In *J. Ecol.* 104 (1), pp. 206–218. DOI: 10.1111/1365-2745.12489.

Seabloom, Eric W.; Kinkel, Linda; Borer, Elizabeth T.; Hautier, Yann; Montgomery, Rebecca A.; Tilman, David (2017): Food webs obscure the strength of plant diversity effects on primary productivity. In *Ecol Lett* 20 (4), pp. 505–512. DOI: 10.1111/ele.12754.

Shipley, B. (2006): Net assimilation rate, specific leaf area and leaf mass ratio: which is most closely correlated with relative growth rate? A meta-analysis. In *Funct. Ecol.* 20 (4), pp. 565–574. DOI: 10.1111/j.1365-2435.2006.01135.x.

Siebenkaes, Alrun; Schumacher, Jens; Roscher, Christiane (2016): Resource Availability Alters Biodiversity Effects in Experimental Grass-Forb Mixtures. In *PloS one* 11 (6). DOI: 10.1371/journal.pone.0158110.

Siegfried, Hermann (1917-1930): Topographischer Atlas der Schweiz (TA). Siegfriedkarte: Swisstopo.

Smart, Simon Mark; Glanville, Helen Catherine; Blanes, Maria del Carmen; Mercado, Lina Maria; Emmett, Bridget Anne; Jones, David Leonard et al. (2017): Leaf dry matter content is better at predicting above-ground net primary production than specific leaf area. In *Funct. Ecol.* 31 (6), pp. 1336–1344. DOI: 10.1111/1365-2435.12832.

Snaydon, R. W.; Davies, M. S. (1972): Rapid Population Differentiation in a Mosaic Environment. II. Morphological Variation in Anthoxanthum odoratum. In *Evolution* 26 (3), p. 390. DOI: 10.2307/2407014.

Soil map of the Canton of Bern (1970-2005): Amt für Landwirtschaft und Natur des Kantons Bern. Available online at www.geo.apps.be.ch, checked on 11/29/2019.

Soliveres, Santiago; Maestre, Fernando T.; Berdugo, Miguel; Allan, Eric (2015): A missing link between facilitation and plant species coexistence: nurses benefit generally rare species more than common ones. In *Journal of Ecology* 103 (5), pp. 1183–1189. DOI: 10.1111/1365-2745.12447.

Spear, Erin R.; Mordecai, Erin A. (2018): Foliar pathogens are unlikely to stabilize coexistence of competing species in a California grassland. In *Ecology* 99 (10), pp. 2250–2259. DOI: 10.1002/ecy.2427.

Sullivan, Gary; Callaway, John C.; Zedler, Joy B. (2007): Plant assemblage composition explains and predicts how biodiversity affects salt marsh functioning. In *Ecol Monogr* 77 (4), pp. 569–590. DOI: 10.1890/06-1947.1.

Sun, Shiling; Lian, Sen; Feng, Shulian; Dong, Xiangli; Wang, Caixian; Li, Baohua; Liang, Wenxing (2017): Effects of Temperature and Moisture on Sporulation and Infection by Pseudoperonospora cubensis. In *Plant Dis.* 101 (4), pp. 562–567. DOI: 10.1094/PDIS-09-16-1232-RE.

Sundravadana, S.; Devadason, Alice; Kuttalam, Subapriya; Ramasamy, Samiyappan (2007): Efficacy of azoxystrobin on Colletotrichum gloeosporiodes penz growth and on controlling mango anthracnose. In *J. Agric. Biol. Sci.* 2.

Sutton, Mark A. (2011): The European nitrogen assessment. Sources, effects, and policy perspectives / edited by Mark A. Sutton … [et al.]. Cambridge: Cambridge University Press.

Swarbrick, P. J.; Schulze-Lefert, P.; Scholes, J. D. (2006): Metabolic consequences of susceptibility and resistance (race-specific and broad-spectrum) in barley leaves challenged with powdery mildew. In *Plant Cell Environ.* 29 (6), pp. 1061–1076. DOI: 10.1111/j.1365-3040.2005.01472.x.

Thaler, Jennifer S.; Humphrey, Parris T.; Whiteman, Noah K. (2012): Evolution of jasmonate and salicylate signal crosstalk. In *Trends Plant Sci.* 17 (5), pp. 260–270. DOI: 10.1016/j.tplants.2012.02.010.

Thrall, P. H.; Burdon, J. J. (2003): Evolution of virulence in a plant host-pathogen metapopulation. In *Science* 299 (5613), pp. 1735–1737. DOI: 10.1126/science.1080070.

Thrall, Peter H.; Hochberg, Michael E.; Burdon, Jeremy J.; Bever, James D. (2007): Coevolution of symbiotic mutualists and parasites in a community context. In *Trends Ecol. Evol.* 22 (3), pp. 120–126. DOI: 10.1016/j.tree.2006.11.007.

Thrall, Peter H.; Jarosz, Andrew M. (1994): Host-pathogen dynamics in experimental

populations of *Silene alba* and *Ustialgo violacea*. I. Eoclogical and genetic determinants of disease spread. In *J. Ecol.* 82 (3), pp. 549–559. DOI: 10.2307/2261263.

Tilman, D.; Reich, P. B.; Knops, J.; Wedin, D.; Mielke, T.; Lehman, C. (2001): Diversity and productivity in a long-term grassland experiment. In *Science* 294 (5543), pp. 843–845. DOI: 10.1126/science.1060391.

Tilman, David (1982): Resource competition and community structure. Princeton, Guildford: Princeton University Press.

Tollenaere, C.; Pernechele, B.; Makinen, H. S.; Parratt, S. R.; Nemeth, M. Z.; Kovacs, G. M. et al. (2014): A hyperparasite affects the population dynamics of a wild plant pathogen. In *MOLECULAR ECOLOGY* 23 (23), pp. 5877–5887. DOI: 10.1111/mec.12908.

Vellend, Mark; Baeten, Lander; Becker-Scarpitta, Antoine; Boucher-Lalonde, Véronique; McCune, Jenny L.; Messier, Julie et al. (2017): Plant Biodiversity Change Across Scales During the Anthropocene. In *Annu. Rev. Plant Biol.* 68 (1), pp. 563–586. DOI: 10.1146/annurev-arplant-042916-040949.

Veresoglou, S. D.; Barto, E. K.; Menexes, G.; Rillig, M. C. (2013): Fertilization affects severity of disease caused by fungal plant pathogens. In *Plant Pathol.* 62 (5), pp. 961–969. DOI: 10.1111/ppa.12014.

Wacker, Luca; Baudois, Oksana; Eichenberger-Glinz, Susann; Schmid, Bernhard (2009): Diversity effects in early- and mid-successional species pools along a nitrogen gradient. In *Ecology* 90 (3), pp. 637–648. DOI: 10.1890/07-1946.1.

Wagg, Cameron; Ebeling, Anne; Roscher, Christiane; Ravenek, Janneke; Bachmann, Doerte; Eisenhauer, Nico et al. (2017): Functional trait dissimilarity drives both species complementarity and competitive disparity. In *Funct Ecol* 31 (12), pp. 2320–2329. DOI: 10.1111/1365-2435.12945.

Walde, Manuel G. (2019): Biodiversity and nutrient cycling in experimental grassland ecosystems. Master. ETH, Zürich. Department of Environmental System Science.

Walters, Dale (2011): Plant defense. Warding off attack by pathogens, herbivores, and parasitic plants. Chichester, Ames, Iowa: WILEY-BLACKWELL.

Welsh, Miranda E.; Cronin, James Patrick; Mitchell, Charles E. (2016): The role of habitat filtering in the leaf economics spectrum and plant susceptibility to pathogen infection. In *J. Ecol.* 104 (6), pp. 1768–1777. DOI: 10.1111/1365-2745.12632.

Whitehead, D. C. (1970): The role of nitrogen in grassland productivity. A review of information from temperate regions. Farnham Royal: Commonwealth Agricultural Bureaux (Bulletin / Commonwealth Bureau of Pastures and Field Crops, 48).

Wilson, Peter J.; Thompson, K. E. N.; Hodgson, John G. (1999): Specific leaf area and leaf dry matter content as alternative predictors of plant strategies. In *New Phytol.* 143 (1), pp. 155–162. DOI: 10.1046/j.1469-8137.1999.00427.x.

Wise, Michael J.; Abrahamson, Warren G. (2005): Beyond the compensatory continuum: environmental resource levels and plant tolerance of herbivory. In *OIKOS* 109 (3), pp. 417–428. DOI: 10.1111/j.0030-1299.2005.13878.x.

Wright, I. J.; Reich, P. B.; Westoby, M.; Ackerly, D. D.; Baruch, Z.; Bongers, F. et al. (2004): The worldwide leaf economics spectrum. In *Nature* 428 (6985), pp. 821–827. DOI: 10.1038/nature02403.

Yin, Xin; Qi, Wei; Knops, Johannes M. H.; Qin, Xuexue; Du, GuoZhen (2018): Aboveground facilitation and not complementary resource use cause overyielding among grasses in Tibetan alpine ecosystems. In *Folia Geobot* 53 (4), pp. 365–376. DOI: 10.1007/s12224-018-9325-4.

Young, Hillary S.; Parker, Ingrid M.; Gilbert, Gregory S.; Guerra, Ana Sofia; Nunn, Charles L. (2017): Introduced Species, Disease Ecology, and Biodiversity–Disease Relationships. In *Trends Ecol Evol* 32 (1), pp. 41–54. DOI: 10.1016/j.tree.2016.09.008.

Zuppinger-Dingley, Debra; Flynn, Dan F. B.; Deyn, Gerlinde B. de; Petermann, Jana S.; Schmid, Bernhard: Plant selection and soil legacy enhance long-term biodiversity effects. In *Ecology* 0 (ja). DOI: 10.1890/15-0599.

Züst, Tobias; Agrawal, Anurag A. (2017): Trade-Offs Between Plant Growth and Defense Against Insect Herbivory: An Emerging Mechanistic Synthesis. In *Annu. Rev. Plant Biol.* 68 (1), pp. 513–534. DOI: 10.1146/annurev-arplant-042916-040856.

Züst, Tobias; Rasmann, Sergio; Agrawal, Anurag A. (2015): Growth-defense tradeoffs for two major anti-herbivore traits of the common milkweed Asclepias syriaca. In *OIKOS* 124 (10), pp. 1404–1415. DOI: 10.1111/oik.02075.

Zuur, Alain F. (2009): Mixed effects models and extensions in ecology with R. Softcover reprint of the hardcover 1 st ed 2009. New York: Springer Science+Bussines Media (Statistics for Biology and Health).

Acknowledgements

I would like to thank the following people for their contribution and support. Only the sum of all their little and big efforts made this thesis possible.

In the first place, I would like to thank my dear colleague and friend Noémie. We started the PaNDiv experiment together and now we finished our theses together as well. I cannot imagine my PhD without you. Thank you for just everything! Of course, our theses would not have been possible without an amazing supervisor. Thanks, Eric, for being there for all big and small questions, for all the discussions about statistics, scientific concepts and manuscripts. I could not have wished for a better supervisor.

I would also like to thank all the people in our group. Thank you, Anne, for contributing so much to the first manuscript and for being a great female scientist role model ;-). Thank you, Tosca, for sharing your data, insect expertise and English skills for the last manuscript and for being such a great friend. Thank you, Santi for your feedback on my manuscripts, for the scientific discussions over a beer or two and for your institute shaking laughs. Thank you, Thu Zar, Rodrigo, Nadia and Hugo S. for the good times.

A large project like the PaNDiv experiment always requires many smart and hardworking people. The maintenance, data collection and organization of all the minions is a huge amount of work. Thank you Hugo V., Mervi and Marlise for keeping the experiment running and for all the great support. I would also like to thank all the field work helpers: Samuel and Nico for contributing a lot during the early phase of the experiment, Sylvain, Barry, Yvonne and Livia for being such amazing and reliable HiWis and all the others who were there for the labor intensive weeding and all the other times when we needed additional hands: Alexa, Aline, Alissa, Alvaro, Ana, Andrea, Andreas, Angel, Anita, Anja, Toni, Armin, Aurore, Barbara, Barbora, Beatrice, Carlos, Cedric, Celia, Chiara, David D., David H., Denise, Dimitry, Elise, Eric U.,

Evelyne, Farshad, Floris, Gemma, Géraldine, Ghulam, Giacomo, Gino, Gjeska, Hannah, Ines, Jaime, Jeronimo, Jil, Juliette, Kaspar, Laura, Lia, Linda, Lucien, Lune, Marta, Mathieu, Maurizia, Melanie, Milena, Mira, Mireia, Miriam, Mohamed, Pascal K., Pascal W., Pascal S., Lynn, Rares, Remy, Rodrigo R., Romain, Roman, Rosalinda, Salim, Sandro, Sara, Sarah, Sonja E., Sonja F., Sophie, Soraya, Stephanie, Tala, Thibault, Tobias, Tom, Valentin, Velina, Viona and Zephyr.

There are many people, who made my life at the institute and at the field easier and to whom I am very grateful too: the owner of the field: Fritz; Baschi and Stifu with their expertise about fungi and stats; the administration team: Helga, Yvonne and Sandra; the librarian Christine, the IT team Peter and Roman; the technical service team Tinu, Willi and Roger; the lab technicians Marianne and Therese, Res and Michaela from HAFL, from who we could borrow the harrow; Charlotte Bühlmann from the Münchenbuchsee Archive who provided a lot of historical background knowledge about our field site.

I am blessed to have worked in an institute full of amazing people, who have shared their knowledge, experience and friendship with me. I was so lucky to have Judith Hinderling, who helped me to get to know all the important people and places at the IPS and fed my brain with so much taxonomic knowledge and “Eselsbrücken” to remember everything. I am very thankful for the many happy hours with Marion Renner, Lisanna Schmidt, Armin Komposch, Graham Prescott, Deborah Schäfer, Clemens Kleinspehn, Sarah Bürli, Ralph Bolliger, Katja Rembold, Lena Neuenkamp, Caterina Penone, Maria Felipe Lucia and everybody else working at the IPS.

I also want to thank my scientific friends Xiang Liu, Fletcher Halliday and Nilsa Bosque-Perez for their inspiration and support.

Ich danke meiner Familie für das warme Zuhause wo ich jederzeit willkommen bin, um

Energie zu tanken. Ohne die Unterstützung meiner Eltern wäre ich nicht hier, wo ich heute bin.

Zum Schluss, möchte ich mich bei meinem Partner Jérôme Oesch bedanken. Danke, dass Du mich während der ganzen Doktorarbeit in den stressigen Phasen wider fit gekuschelst und meine schlechte Laune ertragen hast. Vielen Dank für Deine Liebe und Unterstützung.

Declaration of consent

on the basis of Article30 of the RSL Phil.-nat. 18

Name/First Name:	Cappelli, Seraina
Matriculation Number:	09-916-453
Study program:	Ecology & Evolution
Title of the thesis:	Causes and consequences of fungal pathogen infection in grasslands
	Bachelor ☐ Master ☐ Dissertation ☒
Supervisor:	Prof. Dr. Eric Allan

I declare herewith that this thesis is my own work and that I have not used any sources other than those stated. I have indicated the adoption of quotations as well as thoughts taken from other authors as such in the thesis. I am aware that the Senate pursuant to Article 28. RSL Phil.-nat. 05 is authorized to revoke the title awarded on the basis of this thesis.
For the purposes of evaluation and verification of compliance with the declaration of originality and the regulations governing plagiarism, I hereby grant the University of Bern the right to process my personal data and to perform the acts of use this requires, in particular, to reproduce the written thesis and to store it permanently in a database, and to use said database, or to make said database available, to enable comparison with future theses submitted by others.

Bern, 22nd November 2019 Seraina Cappelli

Curriculum Vitae

Name: Cappelli
First name: Seraina Lisa
Date of birth: June 24th 1990
Nationality: Swiss & Italian

Education:

2015-2020	University of Bern, Switzerland
PhD thesis:	Causes and consequences of fungal pathogen infection Supervisor: Eric Allan
2012-2015	ETH Zürich, Switzerland Master in Ecology and Evolution Master thesis: Tree Choice of Red Wood Ants Supervisor: Anita C. Risch, WSL Birmensdorf
2009-2012	ETH Zürich Bachelor in Environmental Sciences

Publications:

Cappelli, Seraina L.; Pichon, Noémie A.; Kempel. Anne S.; Allan, Eric (2019): Sick plants in grassland communities: a growth-defense trade-off is the main driver of fungal pathogen abundance and impact. Growth-defense trade-off in a grassland. Preprint at *bioRxiv*. DOI: 10.1101/806299.

Pichon, Noémie A.; Cappelli, Seraina L.; Soliveres, Santiago; Hölzel, Norbert; Klaus, Valentin H.; Kleinebecker, Till; Allan, Eric (2019): Decomposition disentangled: a test of the multiple mechanisms by which nitrogen enrichment alters litter decomposition. Preprint at *bioRxiv*. DOI: 10.1101/671545.