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Review

Consideration of the Disease Complexes, the Missing Link to Correctly Analyze the Impact of Intercropping on Disease Development

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Abstract: Diversification at the plot level, through the use of intercropping (mixtures of crops), is an alternative to the conventional system of intensive agriculture, based on monospecific, usually single-variety canopies. Intercropping has been shown to provide benefits in terms of disease control. However, competition phenomena and the heterogeneity of the associated crops raise new ecological questions, particularly with regard to the dynamics and evolution of parasite populations. No study has assessed the potential impact of these associations on the dynamics of pathogenic species complexes. Changes in the nutritional status of plants and therefore in their physiological susceptibility to infection within intercropping systems could contribute to an increased diversity of ecological niches and thus affect the composition of the parasitic complex and its spatiotemporal dynamics. In this review, focusing on foliar diseases of fungal origin, and after outlining some elements of the biology and epidemiology of these fungal diseases, we will (i) describe the mechanisms that contribute to the composition of disease clusters and that drive interactions, but we will also review the strategies that these foliar diseases have adopted to deal with these co-infections; (ii) define how intercropping can lead to changes in epidemic dynamics, in particular by presenting the mechanisms that have a direct and indirect effect on disease evolution; and (iii) present the approach that should be adopted to properly study intercropping correctly in a multi-infection situation.

Keywords: disease complex; life history traits allocation; competition; sequential events; trade-off; intercropping; ecological niches

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1. Introduction

In recent decades, the loss of cultivated biodiversity has increased, with intensive agricultural production focusing on only a few species [1]. Indeed, homogenization has been the dominant paradigm of agriculture in industrialized countries, with standardization and optimization of growing conditions based on buffering environmental variability through mechanization and synthetic inputs [2]. These growing conditions implicate tillage systems including various operations that manipulate the soil in order to optimize the production of the crop and act on the dynamics and the structure of the canopy. In all the operations, crop residue management is particularly important to manage necrotroph pathogens that are able to survive on stubbles. However, this agricultural model is reaching its limits, with growing awareness of its unsustainability, its contribution to climate change, and its impact on biodiversity [3], while critical human health issues have been highlighted by the hidden costs of pesticides [4]. The genetic homogenization of crops is well known to facilitate the emergence and spread of pests, diseases, and weeds within fields, landscapes, and countries [5]. Conversely, diversification is an important prophylactic measure that is

essential for pesticide-free agriculture [6]: (i) production with a wider range of crops stabilizes global production at the country level [7] and (ii) extending crop rotations decisively improves weeds and pest control at the farm level [8–10], while the ecological mechanisms are sampling effects and interspecies complementarity. The same findings apply to cover crops, mixed crops, or cultivar mixtures [11].

Modern agricultural practices have dramatically reduced crop diversity at multiple scales (intra and inter-varietal genetic diversity, variety richness, and evenness of varieties in landscapes; [1,12]. The ever-growing list of environmental and health drawbacks of industrialized agriculture has increased the need for a paradigm shift, embodied in the agroecology movement in society and science [13]. A central principle of agroecology is the mobilization of crop biodiversity to enhance natural regulation and replace pesticides and synthetic fertilizers. Indeed, the benefits of diversifying cropping systems have been described in numerous studies and meta-analyses [14]. However, a global decline in crop diversity has been reported in Europe [15]. In France, simplification in crop rotations has also been reported [16] and has been identified as a key explanation for yield stagnation in wheat [17].

The mobilization of diversity within a field, whether varietal or specific, can critically promote crop health [11,18]. However, the combination of varieties and crops remains a challenge due to a lack of knowledge on how to manage heterogeneous canopies [19]. Recently, authors have proposed the transfer of concepts from functional ecology to crops [20–22], in particular, to promote niche complementarity and synergies and minimize competition between crops and/or genotypes. However, biotic regulatory mechanisms, such as disease control by cultivar mixtures [23], have rarely been hybridized with these concepts. Ideas such as the use of competition to promote weed control [24] or synergies between architecture and resistance traits [25] deserve to be better explored, especially in combination with trait-based modeling approaches [19] and experiments. Meanwhile, while most ecological theories predict higher disease resistance and higher productivity in genetically diverse stands [26], some evolutionary theories predict the opposite [27–30]. Thus, there is a paradox regarding the effect of diversity on disease resistance in plant communities. In addition, interspecific neighborhoods can alter plant physiology, sometimes increasing disease resistance [31], and some reports indicate that immunity can be altered by the plants neighboring other plants [32]. Except for some pieces of evidence [30,33,34], the induction of plant immunity by intra-specific plant–plant interactions is largely unknown. The impact of such physiological changes due to plant–plant interactions in the field is also poorly documented. Crop diversification could reduce the incidence of pathogens, but also hinder their adaptation and provide more durable resistance [35,36]. However, higher pathogen diversity could also be induced by host diversity, increasing recombination potential and possibly adaptation. Therefore, describing pathogen evolution in mixtures is an important issue in assessing the persistence of such systems.

To date, none of the studies on intercropping and its impact on disease have considered disease complexes. In particular, none of the various studies have addressed the issue of ecological balances between pathogen communities and the fact that the creation of different niches in intercropped crops compared to pure crops could alter these balances and change the composition and prevalence of the pathogen species that make up these complexes. Indeed, with the development of molecular tools, a high prevalence of multi-infections has been demonstrated [37–39]. In the literature, the term “species complex” can have different meanings, ranging from the complexity of the taxonomic dissection of what a species name encompasses to “complex disease” where many species are associated with the disease (co-occurrence), without understanding which are actually the causal agent(s) and which microbial species are systematically associated with the disease without playing a role in the infection or exploiting the symptoms as secondary invaders. Taken together, three kinds of species complexes can be defined: (i) disease complexes with pathogens coexisting in different ecological niches and without any physical interaction, (ii) disease complexes where associations between different species are the norm but the impact of each pathogen remains unknown, and (iii) actual disease complexes, where there is at least minimal

evidence of the involvement of more than one species in disease severity [39]. Multiple infections, either simultaneous or sequential, affecting a single plant or crop are now recognized as common in plant disease epidemics. These multiple infections thus generate a series of competitive interactions (exploitation competition, apparent competition, or interference competition) that directly affect the life history traits of competitors and, hence, their fitness. Therefore, to define an integrative experimental approach, we need (i) to know how disease complexes function; in this case, we will focus on fungal foliar diseases, because they alone bring together different groups of pathogens due to their trophic mode; (ii) to redefine how intercropping can lead to changes in epidemic dynamics, in particular by identifying the mechanisms that have a direct and indirect effect on disease development; and (iii) to determine which approach to adopt in order to properly study intercropping correctly in multi-infection situations.

2. Trophic Status and Biological Characteristics of Foliar Pathogens

Plants are attacked by numerous pathogens during their life cycle [40]. Among these pathogens, fungi are among the most important causal agents of plant diseases and likely represent the most diverse group of ecologically and economically relevant threats. In fact, they are responsible for more than 60% of plant diseases worldwide. Fungi have developed a plethora of strategies to colonize plants, and these interactions lead to a wide range of outcomes, from beneficial interactions to host death. Pathogenic fungi use a diversity of strategies to colonize plants and cause disease. Plant pathogenic fungi are divided into three main groups: biotrophic pathogens, which form intimate interactions with plants and can persist in and use living tissue (biotrophs); necrotrophic pathogens, which kill tissue to extract nutrients (necrotrophs); and hemibiotrophic pathogens, which start out as biotrophs before becoming necrotrophs. To successfully invade plant organs, pathogen development is tightly regulated, and specialized infection structures, such as appressoria, are formed. Although all pathogens interfere with the plant's primary defenses, necrotrophs secrete toxins that kill plant tissue. In contrast, biotrophs use effector molecules to suppress plant cell death and manipulate plant metabolism in favor of the pathogen [41].

Plant pathogenic fungi are species that extract nutrients from plants and have a negative effect on plant health. Some types of pathogens are completely dependent on their host (obligate parasites); others form a close association with the host but can also complete the life cycle of the plant. These facultative pathogens can thrive in a variety of environments other than the organism (or organisms) in which they cause disease. Pathogens with a biotrophic lifestyle are either obligate or non-obligate. Obligate biotrophs are the pathogens responsible for powdery mildew (Ascomycota) and rust (Basidiomycota). The latter have evolved to adapt to the life cycle of their host plants and are dependent on them to complete their life cycle. Rusts, in particular, have developed different spore types and developmental patterns, including infection of an alternate host after the senescence of the primary host [42]. Obligate biotrophic species are thought to have a limited ability to use common substrates as energy sources and are therefore completely dependent on their host for an energy source, making them obligate parasites [40,41].

Necrotrophic plant pathogens, such as *Trichoderma* spp., *Rhizoctonia solani*, or *Macrophomina phaseolina*, feed on dead tissue. The first stages after the initial encounter with the host are the most critical; to survive, the pathogen must subvert the plant's defenses and generate a zone of dead (necrotic) tissue in which it is protected from the host and from which it can feed. The subsequent stages of necrotrophic diseases are characterized by the spread of necrosis around the initial zone of infection, which precedes the progression of the pathogen. It is convenient to divide all necrotrophic pathogens into species with narrow and broad host ranges [43]. The host specificity of necrotrophic species is conferred by the production of host-specific toxins, which are essential factors for pathogenicity in a compatible host.

Hemibiotrophic pathogens are species that combine biotrophic and necrotrophic lifestyles, with a variable duration of the biotrophic phase before switching to necrotrophy [40]. During the biotrophic phase, fungal pathogens secrete effectors to suppress plant

defenses, in the same way as a strictly biotrophic agent. At the end of the transitional biotrophic phase, the fungus undergoes a massive developmental change that ensures the transition from a biotrophic to a necrotrophic mode. Hemibiotrophic species appear to have developed highly efficient infection strategies and are among the most aggressive plant pathogens. For example, in terms of host range, *Magnaporthe oryzae* can cause disease on a limited number of grasses, but it is best known for rice blast, the most destructive disease of rice [44]. *Colletotrichum* spp. cause anthracnose on over 600 dicotyledonous and monocotyledonous plant species and are serious pathogens of important crops [45].

A final aspect to consider is the possible link between lifestyle and host specificity. As described above, host range varies considerably, from species specific to a single host to pathogens with a wide host range capable of causing different types of disease in the same host or in many hosts. Obligate biotrophic species appear to have narrower host specificity than species with hemibiotrophic or necrotrophic species. Even non-obligate biotrophic species appear to have the ability to infect a wide range of hosts, as demonstrated by *Claviceps purpurea*, which is capable of infecting over 400 plant species [46]. Furthermore, at the genus level, even obligate biotrophic pathogens can have a wide host range. For example, powdery mildew (*Erysiphe* spp., *Microshaera* spp., *Phyllactina* spp., *Podosphaera* spp., *Sphaerotheca* spp., and *Uncinula* spp.) causes disease in over 10,000 plant species, including monocots and dicots, shrubs, and trees [47]. Consequently, species with a wide host range appear to exist in all types of life forms.

3. Drivers of the Epidemic Dynamics in the Sole Crop Canopy

Disease development in agroecosystems is strongly influenced by the agronomic characteristics of these agrosystems and the local pedoclimatic conditions [48]. Several factors are involved in the dynamics of disease during the growing season: (i) agronomic choices and the surrounding landscape, (ii) inoculum pressure, (iii) pathogen aggressiveness, and (iv) canopy development and changes in host status. The choice of agronomic practices applied to the crop, but also to the surrounding landscape [49], are critical to the constitution of the disease complex infecting a crop: they determine the dynamics of inoculum reservoirs, the connectivity between hosts, and hence, the local disease pressure and kinetics. The development of foliar diseases is strongly influenced by the climatic conditions actually perceived by the pathogens and the plant, and thus the microclimate of a canopy, both at the soil surface and in the soil (temperature, humidity, wind, light, pH, soil composition, and structure) [40]. These factors affect disease development through their influence on host tissue growth and susceptibility, pathogen multiplication and activity, and host–pathogen interactions. Together, these interactions determine disease severity [50].

Disease dynamics are also conditioned by the degree of aggressiveness of the pathogen developing in the canopy. Aggressiveness refers to the quantitative variation in pathogenicity on susceptible hosts [40,51]. Aggressiveness is often measured by a variety of quantitative traits expressed during the host–pathogen interaction: infection efficiency, latent period, number of spores produced, infectious period, and reproduction rate [51]. Measurements of these traits are essential for determining the incidence, prevalence, and severity of a disease. Variability in host and pathogen life history traits will influence disease epidemiology and evolutionary dynamics within pathogen populations [52].

Plants can be viewed as complex structures consisting of a number of components with specific morphological characteristics and organization [52,53]. The architecture of the plant is the spatial distribution of leaves, stems, and flowers on the plant at a given time. Topology includes the physical connection between different components of the plant, as influenced by geometry and the shape, size, orientation, and position of plant organs in space. Architecture depends on geometric and environmental factors that affect growth and therefore changes over the growing season [54]. The architecture of a plant is determined by an interaction between the intrinsic architecture defined by its genome and the characteristics of the population in which it grows (plant density). Plant canopies are complex, dynamic structures. Their architecture depends on growth stage,

sowing density, plant layout, and plant architecture (shape, size, orientation, and spatial positioning of different components). Changes in the canopy structure can lead to changes in the physiological status of hosts (senescence, nutritional and defense status, etc.), thereby influencing the plant's susceptibility to infection.

4. Coinfection and Pathogen Interactions

Diseases in both plants and animals have long been considered to be the result of the interaction between a single host and a single pathogen. However, with the development of molecular tools, a high prevalence of multiple infections has been demonstrated [37–39]. Multiple infections, either simultaneous or sequential, affecting a single plant or crop are now recognized as common in plant disease epidemics. These multiple infections thus generate a range of competitive interactions (exploitation competition, apparent competition, or interference competition) that directly affect the life history traits of competitors, and hence, their fitness. While a theoretical framework based primarily on three ecological theories—niche exclusion, the 'tragedy of the commons', and the virulence-transmission trade-off—provides insights into potential outcomes of multiple infections on pathogen evolution and virulence, as well as disease dynamics, experimental observations, to date, strongly suggest that unexpected outcomes can also occur.

The concept of ecological niche offers an interesting framework from which to analyze pathogen coexistence. This term is often misused to describe only the type of place where each individual lives, which in fact corresponds to the 'habitat' of the individual considered [55,56]. The ecological niche of pathogens is highly dependent on the characteristics of their host plant. While a conducive abiotic environment is also necessary for the infectious success of pathogens, a major determinant of their niche is their host genotype [57]. The niche of a pathogen with a complex life cycle, including host alternation or saprophytic stages, adjusts, changes, and is affected by biotic (including the presence of co-infected pathogens) and abiotic environment, within the limits of its genotype and phenotypic plasticity.

Pathogen coexistence involves commensalism, i.e., the mutual use of at least one common limited resource, necessary for development and reproduction, by at least two individuals (different strains of the same species or several different species). It thus leads to the establishment of interactions between these individuals [58–61]. The exploitation of a common resource by one individual leads to a reduction in the availability of that resource for others. The concepts of coexistence and competition are thus closely linked and can be interpreted through the filter of niche theory, where competitive interactions between individuals depend on the degree of niche overlap [56,58,62,63]. Several studies have shown that resource allocation due to the coexistence of different pathogens can modify host exploitation strategies, alter change epidemic dynamics, and also influence the evolution of the life history traits of individuals [39,50,64–66]. These changes will depend on the type of interactions developed between the pathogens. Three main types of multi-infection interactions have been described in the literature: (i) resource-mediated (exploitation), (ii) host-mediated (apparent), and (iii) interference [37,39,56,61].

Interactions between individuals may depend on the aggressiveness of the strains but also on the sequential arrival of the infection. The first to colonize may adopt a strategy of excluding competitors. Pathogens also interact, either directly or indirectly: directly with some signals that can have a negative or positive effect on the other pathogen and indirectly because they share the same resource, and if one consumes it, it is not available for the other one. Also, if they consume leaves, this could change the microenvironment, which is the ecological niche of the other pathogen. An ecological niche is a concept of environmental opportunity and constraint of the environment for an individual. Any species within that niche is part of the community niche. Any change in the composition of the community means that the niche is evolving slightly. Change can also be seen in the abundance of each species. There are different definitions of ecological niches, ranging from ecological

theories to modeling parameter approaches [67]. The niche in the case of plant pathogens is specific because the habitat and the resources are living organisms [56].

In general, two drivers explain the dynamics of the species complex: (i) extrinsic factors related to the impact of man on the agroecosystem (world trade, use of genetic resistance in the host, and use of fungicides), which are likely to have a long-term effect on restructuring the species complexes and favor the replacement of species by others, and (ii) intrinsic factors (environmental requirements, biological traits of the species, host plant receptivity, and the composition of the parasitic community in the geographic area of cultivation), which structure the local composition of the disease complexes and their dynamics during the cropping season. With regard to extrinsic factors, studies have shown that the distribution of crop species is often accompanied by the establishment of a pathogen species that will eventually coexist with or replace the indigenous, often less harmful, species. This was observed for Sigatoka disease in bananas, where the establishment of Cavendish cultivars favored the dramatic expansion of *Mycosphaerella fijiensis* to the detriment of *Mycosphaerella musae* [68]. Plant resistance and host tissue susceptibility are also known to influence the composition and distribution of disease complexes. Indeed, in most cases, plant genetic resistance is bred to control a pathogen species and if it consists of complete major-gene resistance, it will be highly efficient in eradicating the target species, at least in the short to medium term, if the species population is primarily avirulent. Where species complexes are targeted, resistance will be bred against the dominant, most damaging species. If ecological dogma is followed, it is expected that eradication of a species will open up an ecological niche for other associated fungal species to take over. In the case of disease complexes, this theory has rarely been challenged by field experiments [38,39].

This has been particularly observed for the disease complex of oilseed rape stem canker. Studies showed the use of a specific resistance gene, Rlm11 (known to be efficient against approximately 95% of the *Leptosphaeria maculans* populations and inefficient against *Leptosphaeria biglobosa*; [69]). The systematic colonization of the *Rlm11* isogenic line by *L. biglobosa* further confirmed that the absence of *L. maculans* opened up an ecological or trophic niche that benefits *L. biglobosa.* In the Ascochyta blight model, *Peyronellae pinodes* can be favored and made dominant by the use of resistance genes. For example, in the mid-1960s in Canada, *P. pinodes* became the dominant species on field peas following the introduction of resistance to *Ascochyta pisi* [70]. Host tissue susceptibility varies with host genotype and age [57,71]. This increase in resistance or susceptibility to plant or host tissue age has been demonstrated for several pathosystems [71–75] and may affect members of a species complex differently. For example, Le May et al. (2009) showed that *Phoma medicaginis* var. *pinodella* species developed more readily on senescent or aged organs. Thus, early plant attack by *P. pinodes* weakens plant defenses and accelerates senescence processes, allowing *P. pinodella* to establish itself more rapidly.

Regarding intrinsic factors, three main factors can be considered: (i) environmental requirements, (ii) life history traits, and (iii) genetic exchange. Regarding the first factor, and as shown in part 2 of the manuscript, thermal (and other environmental) biology in fungi requires complex experiments to identify conducive temperature/humidity/light conditions for growth, sporulation, and sexual reproduction [76,77]. Thus, changes in temperature or precipitation during the season can alter and influence the prevalence and fluctuation of pathogen species [78–80]. Life history traits are defined as the biological characteristics of organisms that structure their life history [56]. Assessing the life history traits of the different species involved in disease complexes is essential for understanding the biological and epidemiological differences at the origin of their (possible) niche differentiation and coexistence. Coexistence implies the mutual exploitation of at least one common limited resource necessary for development and reproduction by at least two individuals, often leading to the establishment of competitive interactions [60,61]. The exploitation of the shared resource by one individual leads to a reduction in the availability of this resource for the other individuals, which can lead to a change in the host's exploitation strategy

and affect epidemic dynamics, but also affect the evolution of the life history traits of the individuals [39,65,81].

5. How Intercropping Will Modify the Epidemic Dynamics of Foliar Diseases

Foliar diseases caused by airborne pathogens are particularly difficult to control as they are usually spread by wind or flying insect vectors. The susceptibility of certain crops to foliar diseases has been attributed to the over-simplification of agro-ecosystems [82]. Systemic changes that can limit pathogen development include (1) the establishment of interspecific cover in the case of annual crops and (2) the development of agroforestry in the case of perennial crops. Intraplot diversification may lead to the emergence of foliar disease regulation processes that do not occur under monospecific cover [83,84]. The resource concentration hypothesis [85] reflects the fact that mixing multiple host and non-host plant species results in the 'dilution' of susceptible individuals within the plant cover, thereby limiting the development of the pathogen population. Increasing the proportion of non-host plants in a mixture reinforces this effect [83]. Variations in host plant availability generate different direct and indirect mechanisms that, individually or in combination, help to reduce parasite pressure on the crop. These processes are fairly well described in the case of association crops (mixtures of two crop species) and varietal associations (mixtures of two varieties of the same crop species). They can be influenced by activating levers that classically allow (i) acting directly on the pathogen to minimize initial parasite pressure by reducing (barrier effect) or diluting inoculum sources and b modifying rainfall and microclimate properties within the canopy (direct effect); (ii) acting indirectly on the host plant to minimize its susceptibility to pathogens by activating allelopathic processes or indirectly inducing plant resistance (premunition) and modifying the physiological properties of plant organs [86]; and (iii) acting indirectly on parasitic antagonist microorganisms that regulate pathogens (Figure 1).

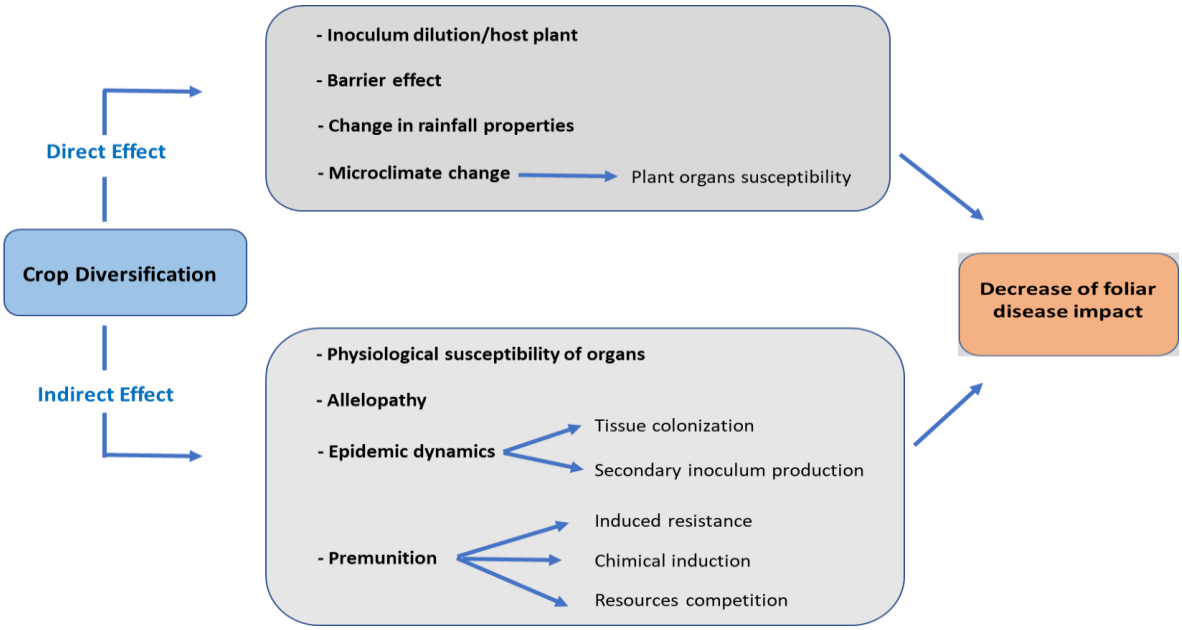

Figure 1. Mechanisms associated with the direct and indirect impact of crop diversification on the epidemic development of foliar diseases.

To activate the first management lever, measures must be taken to limit the dispersal of auto-inoculum (already present on the plot) and allo-inoculum by wind or rain (dispersal by splashing). Auto-inoculum can take different forms: survival structures present in the soil (chlamydospores, sclerotia, and fungal mycelium) on the surface or brought up by tillage, crop residues from the previous season, infected stubble, or even weeds, which can be secondary hosts (especially of phytoviruses). To limit the availability and spread of this

inoculum, in the case of winter crops, the soil can be covered with freeze-dried plants like clover before the cash crop develops. Thus, by covering the soil in early autumn, the clover can prevent the auto-inoculum from spreading to the plants. Allo-inoculum from outside the plot can also come from secondary host plants in the wild parts of the agro-ecosystem. To activate the second management lever, minimizing host plant receptivity, diversification within the field must allow the expression of different epidemiological regulatory mechanisms [84], which involve (1) resource dilution, (2) disruption of the spatial component of epidemic dynamics, (3) disruption of the temporal component of epidemic dynamics, (4) allelopathic effects, (5) immunity or physiological resistance of cash crops, and (6) direct and indirect physical architectural effects (Figure 1). Concerning the direct effects, variation in the availability of host plants generates various mechanisms, which, individually or in combination, help to reduce parasite pressure on the cash crop. The theoretical mechanisms involved include (1) dilution of the inoculum, (2) the barrier effect on inoculum dispersal, (3) change in rainfall properties, and (4) microclimate changes. Concerning the indirect effects, increasing genetic diversity within cropping systems through the use of species mixtures helps to limit disease development and stabilize yields by modifying epidemic dynamics. Plant–plant interactions can induce replacement, facilitation, and niche complementarity effects, but can also lead to competition for resources (water and minerals) and light, thereby modifying the receptivity (physiological susceptibility of organs) of these plants to pathogens or inducing premunition mechanisms and allelopathy.

5.1. Direct Effect Associated with Dilution and Barrier Effects

The amount of inoculum (primary and secondary) that reaches the crop is a critical factor in the epidemic dynamics of pathogens. It determines how early and how fast an epidemic develops. By introducing a degree of heterogeneity into the crop cover (creating microhabitats with different physical and physiological characteristics) and increasing the average distance between two host plants, intercropping reduces the amount of inoculum to which they are exposed. The use of spore traps (vaseline pads) in pure canopies or in associations has shown an effect of the association on the number of spores dispersed in the canopy during the season. For example, the use of an intercrop in a tomato crop helped to limit the dispersal of *Alternaria solani* spores developing on the tomato [87].

Intercropping helps to dilute the inoculum produced by a pathogen in the canopy, thereby reducing the parasite pressure on the crop. In fact, the intensity of an epidemic is determined by the proportions of inoculum available that lead to autoinfection (the spread of spores on the same plant) or allo-infection (the spread of spores on neighboring plants) [88]. However, this dilution effect has a different impact depending on the mode of dispersal, as suggested in the study by [89] on sorghum anthracnose caused by *Colletotrichum sublineolum*, where the inoculum is dispersed by splashing. Although wind dispersal of foliar pathogens also occurs over long distances, it can be disrupted by physical barriers at the intraplot scale [84]. For example, by altering the canopy structure, architecture, and microclimate, intercropping alters access to the most susceptible crops [83]. This mechanism is crucial for regulating epidemics associated with polycyclic diseases such as Ascochyta blight due to *P. pinodes* [90]. Indeed, in this study, the intercropping was responsible for a reduction in disease severity partially due to a change in microclimate conditions, especially the leaf wetness duration during and after flowering. Depending on the organization of the intercropped area (mixed or strip crops), inoculum dispersal by wind and rain is limited. This limitation results either from a physical barrier effect or from an indirect modification of the properties of wind (turbulence level) or rain (drop size and kinetic energy). Research carried out under controlled conditions, simulating different levels of rain intensity, has shown an effect of rain intensity on spore dispersal capacity. In particular, the work of [90] showed that the architecture of associated canopies, through its barrier effect, contributed to a 39 to 78% reduction in the level of spore dispersal.

Another study showed that reducing host density by increasing the land species richness significantly reduced the severity of foliar diseases [87]. In another example, where

the density of *Alternaria solani* spores developing on tomato was reduced by mixing tomato with marigold (*Tagetes erecta*) or pigweed (*Amaranthus hypochondriacus*), the beneficial effect was partly attributed to the physical barrier [91]. In their study, [91] showed that Tomato-Marigold and Tomato-Pigweed intercropping reduced the conidial density near the tomato plants by 64–73% and 27–38%, respectively, showing an important barrier effect. This barrier effect has also been widely described for wheat septoria caused by *Zymoseptoria tritici* in the case of wheat–pea or wheat–clover associations: by acting as a "sieve", the clover undergrowth partially hinders the vertical dispersal of the spores that contribute to the development of the epidemic. Similarly, it has been shown that a cover of Sudan sorghum (*Sorghum bicolor var. sudanensis*) has been shown to reduce the dispersal of *Colletotrichum acutatum* spores responsible for strawberry anthracnose in intercropping systems [92]. According to the stage of the cropping season, the reduction in spore splash ranged from 4 to 9%, and this variation was essentially explained by the recovery of the companion plant and the ability of the pathogen to reproduce and release spores. Similarly, in their study on the Anthracnose of hot pepper due to *Colletotrichum scovillei*, ref. [93] showed that intercropping systems could decrease air spore density by 90%.

5.2. Direct Effect Associated with Defense Induction

Many plant-associated microbes are pathogens that impair plant growth and reproduction. Plants respond to infection using a two-branched innate immune system. The first branch recognizes and responds to molecules common to many classes of pathogens, while the second branch responds to pathogen virulence factors, either directly or through their effects on host targets. Resistance induced by non-host species has been demonstrated for several pathogens such as root-knot nematodes, herbivorous insects, *Botrytis cinerea*, and bacteria like *Pseudomonas syringae* pv. *syringae* in the context of intercropping [94]. Two main mechanisms have been identified. The first involves the recognition of a plant-influenced pathogen by a neighboring non-host plant. The second involves the release of molecules (e.g., in root exudates) and/or disturbance of the environment (e.g., shade or competition for nutrients). The volatile or soluble compounds responsible for modifying the expression of the plant immune system in intercrops are still poorly identified. In the case of the pepper–maize association, ref. [94] suggested that root exudates from healthy peppers lead to a reduction in maize attack by *Bipolaris maydis*. More specifically, by looking at the expression of defense genes associated with the plant immune system, the authors showed that these exudates activated the expression of the AOS (Allene Oxide Synthetase) and AOC (Allene Oxide Cyclase) genes, which are involved in the biosynthesis of jasmonic acid, a molecule that is itself involved in defense against necrotrophic pathogens [95]. The induction of genes involved in the biosynthesis of DIMBOA (a benzoxazinoid antibiotic that is part of the chemical defense system of grasses with growth-inhibitory properties against bacteria and fungi) was also observed in maize plants pretreated with pepper exudates [31]. In their study, ref. [96] showed that maize intercropped with legume species showed a 48% decrease in rust disease severity compared to a monoculture of maize. This decrease was closely related to the enhanced acquisition of nutrients (zinc, copper, and iron) that can enhance the resistance to crop diseases, driven by stronger interspecific facilitative effects in the intercropping system.

5.3. Indirect Effect Associated with a Change in Foliar Receptivity

Interactions between two or more species that coexist for a period of time can lead to more efficient use of resources through niche differentiation and complementarity and changes in plant susceptibility to foliar diseases induced by modulating access to soil nutrient resources [97]. Another study showed that growing tomatoes on vetch mulch enhanced plant metabolism and protected against disease spread. In complex, heterogeneous systems, the interception of radiation by each plant species is a competitive factor that can lead to changes in the physiological state of another species and hence its susceptibility to foliar pathogens [98]. The work carried out on protein peas showed that plant architecture and

the distribution of the Leaf area index (LAI) on the nodes of the plant could contribute to modifying radiation penetration, but also accelerating leaf senescence processes [99].

Heterogeneous plant communities have been shown to produce more total biomass than monocultures [18]. This limits negative competitive interactions by reducing niche overlap but also improves resource availability through direct facilitation, such as the secretion of organic acids and phosphatases by some plant species, which increases phosphorus availability in acid soils or the transfer of nitrogen from legume fixers to companion species [97]. Ref. [100] showed that intercropping systems combining several plant species improved the uptake of phosphorus and micro-nutrients such as iron (Fe), zinc (Zn), and manganese (Mn). This coexistence can also lead to competition between species, which modulates access to soil nutrient resources and modifies the susceptibility of the plant to foliar diseases. According to [101], the susceptibility to certain diseases increased by excess nitrogen could be related to high levels of amino acids and reducing sugars in plant tissues, which would facilitate the development of pathogens such as *Pyricularia oryzae* responsible for rice blast. There is a paradox between improved nitrogen uptake in intercrops and reduced disease intensity. Indeed, while nitrogen increases plant susceptibility for most obligate biotrophic pathogens, this can lead to a reduction in powdery mildew (*Peronospora viciae*) attacks in wheat intercropped with faba bean [31].

Competition for light, which modulates the susceptibility of the crop to foliar pathogens depends on the leaf area index (LAI), the relative height of the plants, the growth dynamics of each of the associated species, and also its architecture and height relative to that of the other associated crop [102]. Due to the spatial heterogeneity of the canopy, which varies over the growing season, light energy is primarily available to the dominant species. A previous study [99] demonstrated an increase in the susceptibility of peas to Ascochyta blight caused by *Peyronellaea pinodes* via a modification of canopy architecture and light penetration. This change, which depends on the architectural characteristics of the cultivars, accelerates the senescence of certain organs (stipules and pods) and leads to a reduction in the levels of pisatin, a phytoalexin in the pentose phosphate pathway involved in plant resistance to disease.

5.4. Indirect Effect Associated with Microclimate Change

The development of foliar diseases is strongly influenced by the climatic conditions actually experienced by the pathogens and the plant, and thus the microclimate of a canopy, both at the soil surface and in the soil (temperature, humidity, wind, light, pH, soil composition, and structure) [40]. These factors affect disease development through their influence on host tissue growth and susceptibility, pathogen multiplication and activity, and host–pathogen interactions. Together, these interactions determine disease severity [50]. By modifying the characteristics of a heterogeneous canopy (plant density, spatial arrangement of organs, and distribution of leaf area), intercropping can increase or reduce the duration of leaf wetting and thus favor or limit the development of epidemics [90]. Another work showed that intercropping contributed to changing the architecture of faba beans (internode size and leaf area), which in turn reduced the ability of the fungus *Botrytis fabae* to spread the disease [103]. Similar changes in the temperature of plant organs, particularly the day/night temperature differential, can modulate the development of epidemics, which are often known to be correlated with the sum of mean daily temperatures [99]. The work of [104] reported that shading could reduce brown spot disease caused by *Cercospora coffeicola* by 35% to 65% in dry, low-lying coffee-growing areas of Central America.

Crop diversification in annual systems can alter the microclimate and pathogen development in ways and with intensities that are difficult to predict [102]. For example, intercropping marigold (*T. erecta*) with tomato (*Solanum esculentum*) reduced the duration of periods of high humidity periods (95% relative humidity), creating unfavorable conditions for the development of the tomato pathogen *Alternaria solani* [91]. A reduction in *P. pinodes* attacks on pea pods and stems in pea–cereal combinations could also be partly explained by the combined effect of a change in the microclimate within the canopy, in particular

a reduction in the duration of wetting of sensitive organs. A similar effect was observed in mixed crops of faba beans and cereals (barley, oats, wheat, or triticale) in reducing the severity of botrytis (*Botrytis fabae*) on faba beans [103]. In their study, ref. [93] showed that powdery mildew infection on peas was reduced when peas were intercropped with barley or faba beans (a decrease of 44% and 32% of SAUDPC, respectively). This decrease was explained by changes in microclimate conditions. Indeed, higher biomass and plant height of Barley and faba bean will contribute to the modification of the microclimate within the canopy, particularly by increasing the relative humidity by 1.3 to 4.7% and decreasing the temperature (0.6 to 3.1 °C) and the sunlight intensity (445 to 8194 lux) within the canopy.

6. Pathogen Interactions in an Intercropping Context

While the benefits of diversifying cropping systems are now well established [14,18], effectively combining different species and cultivars remains a challenge due to a lack of knowledge on how to manage heterogeneous canopies. While most current ecological theories predict better disease control and higher productivity in genetically diverse canopies, others have long suggested the opposite [105]. Crop diversification could not only reduce the incidence of foliar diseases but also favor the adaptation of the parasite complex by maintaining greater intra- and interspecific diversity of pathogen populations locally. Understanding how pathogens evolve in intercropping is a critical research issue for assessing the sustainability of these systems. This is all the more important as the scientific literature points to antagonistic effects, particularly in the regulation of leaf diseases associated with biotrophic and necrotrophic fungi. Indeed, plants are in contact with a wide variety of pathogens [39] that share microhabitats but also specific niches within heterogeneous canopies [50,106]. Their coexistence as a parasitic complex and the differentiation of ecological niches within a plant canopy is due to (i) spatial separation, such as two pathogens occupying different plant tissues/organs at the host plant level, (ii) temporal separation, which may correspond to one species developing at the expense of another throughout the course of a season, and (iii) separation in resource use, which may result in different abilities of pathogen species to colonize live/dead plant tissues [60].

Processes that reduce the overall disease incidence act either physically or physiologically on the target pathogens [18]. Some processes (dilution, barrier effect, microclimate, and stand structure) have predominantly physical and spatial determinants, while others (resistance induction and resource provision) have physiological and biochemical determinants. The actual impact of these processes on the ability of plant associations to regulate foliar diseases is therefore highly dependent on the biology of the pathogen, the architecture of the plant, its stage of development and physiology, and, of course, numerous environmental variables. On the temporal scale, two elements are of paramount importance for future work: the state of the host canopy, as influenced by the crop developmental stages, and the dynamics of primary inoculum sources (Figure 2). As highlighted above, the physiological and ecophysiological status of the host (senescence, nutritional and defense status, etc.) is key in shaping the receptivity of the plant to infection, but also in modulating the different types of competitive interactions that take place within disease complexes. Indeed, antagonistic effects are also observed in the competition for resources (light and nutrients) between plants present in a plot, exacerbated by changes in the microclimate. In the case of competition for light, certain diversified cropping systems can lead to an acceleration of senescence processes, favoring the dynamics of leaf diseases caused by necrotrophic pathogens. While canopy aeration is thought to reduce humidity and discourage disease, some studies conducted in agroforestry systems suggest that wind movement may lead to increased plant contact, resulting in injury and increased infection [84]. To date, despite the availability of various sensors, we know of no practical means of reliably assessing the host status in situ. It is likely that rapid advances in multispectral image analysis will provide such means in the near future.

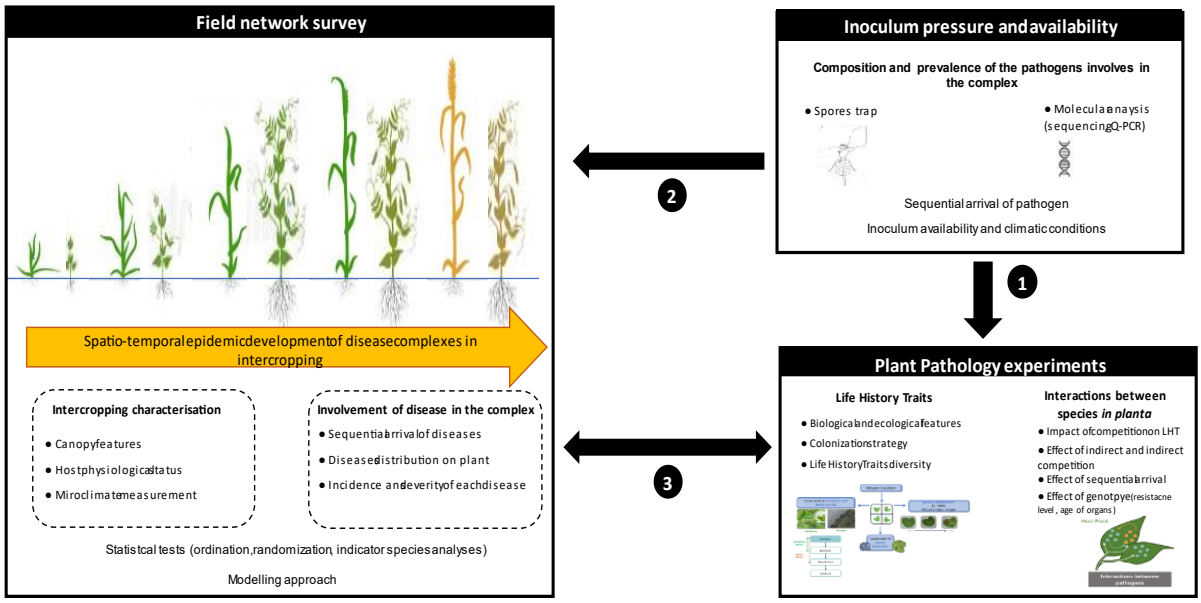

Figure 2. Experimental steps to study disease complexes in intercropping. Methods allowing detection and characterization of plant disease complexes dynamics in intercropping. (1) The identification of causal agents from a pool of potential pathogens through trap systems and molecular diagnosis. (2) The characterization of pathogen prevalence, and sequential arrival on the crop during the growing season. (3) Pathogenicity tests to assess the infection capacity of pathogens in the case of direct or sequential co-infection and help to understand how these pathogens interact in the field.

The dynamics of the primary inoculum also remain poorly understood. Most experimental studies still consider this a 'black box' due to the inherent difficulties in sampling, quantifying, and identifying small, fragmented, and often cryptic populations [40,107]. Progress in this regard could be made by measuring 'proxy' traits for pathogen survival between epidemics. These include the number/spatial density of dormant structures, extinction rates (estimated from genotype frequencies), or the infectious capacity of dormant spores [107]. Dynamics of species complex compositions are often apparent at the time of cropping over one growing season or, for perennial plants, at the scale of the plant's life. The order in which different pathogens infect a plant may condition the type of interactions that will be established. Field surveys can be used to determine the prevalence of each species in the complex and to record the simultaneous or sequential presence of these species. However, this approach is only possible for pathogenic species that are distinguished using diagnostic visual (no confusion between symptoms) and/or molecular (correct assignment of sequences) tools. This highlights the importance of coupling these approaches with molecular diagnostics based on barcode sequences or whole genome sequencing [108], but also establishing tools to monitor the dynamics of disease establishment, particularly in the case of simultaneous infections (e.g., combining spore trapping with real-time qPCR to identify the potential pathogens at play) (Figure 2). Field surveys allow us to identify thermal/environmental preferences, differences in epidemiology, differences in susceptibility to disease, and the effect of co-occurrence on disease severity. While the sequential arrival of pathogens may help to structure the intra-host species complexes within the host and the subsequent epidemiology of disease components, such 'priority effects' [109,110] have rarely been studied for fungal disease complexes in the context of differences in the timing of infection within a growing season [111,112]. In annual crops, differences in epidemiology between different members of a species complex are likely to influence the composition of the species complex at different times of the growing season or on different plant organs. Currently, studies have mainly addressed the epidemiology of different diseases [113,114].

To study the epidemic dynamics of disease complexes, it is essential to have reliable identification and quantification tools. Disease assessment usually begins with the

traditional visual search for symptoms. Symptoms are naturally influenced by environmental conditions, pathogen genotypes, plant genotypes, and their physiological status. Thus, the identification of pathogenic species has always been a challenge for plant pathologists, with observations of a host symptomatic state ranging from typical to atypical and undiagnosable. The development of image-based web and mobile applications for the diagnosis of plant diseases such as those available on the INRAE portal e-phytia (https://ephytia.inra.fr/en/Home/index (accessed on 1 May 2024)) represents a significant breakthrough but remains limited in the case of symptom similarities between different diseases or multiple infections. The development of molecular techniques has often led scientists to use DNA barcode sequences as a new way to bypass the taxonomic expertise required to identify fungal species morphologically [115–117].

This review highlighted the importance of life history traits as predictors and markers of multi-infection performance, and selection both within and between epidemic stages is crucial for the evolution of pathogen populations [118,119]. The analysis of pathogen interactions under controlled conditions has been the subject of numerous publications in recent years, but they have rarely (or only marginally) addressed the specificities of complex fungal pathogens of plants [39,120,121]. The analysis of pathogen interactions on young plants is an approach that makes it possible to mix different inoculation protocols and conditions for the co-occurrence of the pathogens studied. It is also possible to study the effect of the location of competing pathogens on the plant, the sequential arrival of infections, and the age of the host plant (Figure 2).

The final challenge is to link the ecological and evolutionary dimensions of multi-infections. This review has highlighted the importance of life history traits as predictors and markers of multi-infection performance, and selection both within and between epidemic stages is crucial for the evolution of pathogen populations [118,119]. Since co-infections are an integral part of plant disease epidemics and affect crop health in ways that go beyond the simple additive effects of their individual components, risk prediction models and decision support systems should take multi-infection fully into account. Indeed, there cannot be true Integrated Pest Management (IPM) systems without effective management of multi-infections and disease complexes. In parallel, concepts and tools developed in community ecology can be applied to plants infected by species complexes to understand the assembly, dynamics, and management of within-individual host–species ecosystems [122]. Given that many species within disease complexes have identical or very similar infection and host exploitation feeding strategies to exploit hosts, such tools may help to define 'functional groups' into which parasites are grouped in terms of their infection characteristics (infection site and resources consumed) and how the host immune system responds to their infection. This could allow predictions of the likely direct and indirect effects of one pathogen on another and help to determine whether a fungal pathogen will successfully infect a host and how this infection will affect the host state [122]. In the same vein, other authors proposed that evolutionary game theory provides an appropriate theoretical framework to analyze mixed plant–virus infections and predict the long-term evolution of the mixed populations [123]. It would be interesting to evaluate the same approaches to analyze fungal infections due to species complexes. Another possibility is to use the indicator species analysis proposed by [124] to estimate the strength of association of a species with a priori groups of interest and randomization tests to assess the probability of association. This approach was used to identify the causal agent(s) of an unknown squash disease from a pool of 10,000 isolates corresponding to 15 fungal species.

7. Conclusions

When it comes to changing production systems, intercropping is an interesting way of stabilizing yields and reducing the risk of disease. However, like varietal resistance and pesticides, it is a lever that is still being perfected and its long-term effects may be limited if its use is not anticipated and managed in the region. Crop diversification could reduce the incidence of pathogens but also hinder their adaptation and provide more durable

resistance [35,36]. However, higher pathogen diversity could also be induced by host diversity, increasing recombination potential, and possibly adaptation. The generation of heterogeneous canopies could affect the composition and epidemic dynamics of disease complexes. In a context of global change, where the emergence of new diseases has become inevitable, it is necessary to anticipate these changes and to integrate this notion of the evolution of the ecological balance for diseases that develop on crops and therefore within the framework of associated systems.

Therefore, describing pathogen evolution in mixtures is an important issue in assessing the persistence of such systems. Several review papers recently addressed the issues linked to these coinfections with ecological concepts turning the plant and its environment into a whole ecosystem for a cohort of pathogens [39,122,125]. Disease complexes are highly dynamic at all temporal scales, with documented historical replacement of one prior dominant species by another, and, for some annual crops, reports of variation in composition within a growing season. As part of our understanding of how disease complexes work, it is essential to integrate this component, which will affect the ability of pathogen species to establish and develop, for better management of intercropping.

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References

1. Khoury, C.K.; Bjorkman, A.D.; Dempewolf, H.; Ramirez-Villegas, J.; Guarino, L.; Jarvis, A.; Rieseberg, L.H.; Struik, P.C. Increasing Homogeneity in Global Food Supplies and the Implications for Food Security. *Proc. Natl. Acad. Sci. USA* **2014**, *111*, 4001–4006. [CrossRef] [PubMed]
2. Tilman, D. Global Environmental Impacts of Agricultural Expansion: The Need for Sustainable and Efficient Practices. *Proc. Natl. Acad. Sci. USA* **1999**, *96*, 5995. [CrossRef]
3. Gliessman, S.R. *Agroecosystem Sustainability: Developing Practical Strategies*; CRC Press: Boca Raton, FL, USA, 2001. Available online: https://trove.nla.gov.au/version/46552792 (accessed on 1 March 2024).
4. Bourguet, D.; Guillemaud, T. The Hidden and External Costs of Pesticide Use. In *Sustainable Agriculture Reviews*; Lichtfouse, E., Ed.; Springer International Publishing: Cham, Switzerland, 2016; Volume 19, pp. 35–120. [CrossRef]
5. McDonald, B.A.; Stukenbrock, E.H. Rapid Emergence of Pathogens in Agro-Ecosystems: Global Threats to Agricultural Sustainability and Food Security. *Philos. Trans. R. Soc. Lond. B* **2016**, *371*, 20160026. [CrossRef] [PubMed]
6. Frison, E.A.; Cherfas, J.; Hodgkin, T. Agricultural Biodiversity Is Essential for a Sustainable Improvement in Food and Nutrition Security. *Sustainability* **2011**, *3*, 238–253. [CrossRef]
7. Renard, D.; Tilman, D. National Food Production Stabilized by Crop Diversity. *Nature* **2019**, *571*, 257–260. [CrossRef] [PubMed]
8. Cook, R.J.; Veseth, R. *Wheat Health Management*; APS Press: Saint Paul, MN, USA, 1991.
9. Letourneau, D.K.; Armbrecht, I.; Rivera, B.S.; Lerma, J.M.; Carmona, E.J.; Daza, M.C.; Escobar, S.; Galindo, V.; Gutiérrez, C.; López, S.D.; et al. Does Plant Diversity Benefit Agroecosystems? A Synthetic Review. *Ecol. Appl.* **2011**, *21*, 9–21. [CrossRef] [PubMed]
10. Barbieri, P.; Pellerin, S.; Nesme, T. Comparing Crop Rotations between Organic and Conventional Farming. *Sci. Rep.* **2017**, *7*, 13761. [CrossRef] [PubMed]
11. Finckh, M.R. Integration of Breeding and Technology into Diversification Strategies for Disease Control in Modern Agriculture. In *Sustainable Disease Management in a European Context*; Collinge, D.B., Munk, L., Cooke, B.M., Eds.; Springer: Cham, Swizerland, 2008; pp. 399–409. [CrossRef]
12. Bonnin, I.; Bonneuil, C.; Goffaux, R.; Montalent, P.; Goldringer, I. Explaining the Decrease in the Genetic Diversity of Wheat in France over the 20th Century. *Agric. Ecosyst. Environ.* **2014**, *195*, 183–192. [CrossRef]
13. Malézieux, E. Designing Cropping Systems from Nature. *Agron. Sustain. Dev.* **2012**, *2*, 15–29. [CrossRef]
14. Beillouin, D.; Ben-Ari, T.; Makowski, D. Evidence Map of Crop Diversification Strategies at the Global Scale. *Environ. Res. Lett.* **2019**, *14*, 123001. [CrossRef]

15. Aizen, M.A.; Aguiar, S.; Biesmeijer, J.C.; Garibaldi, L.A.; Inouye, D.W.; Jung, C.; Martins, D.J.; Medel, R.; Morales, C.L.; Ngo, H.; et al. Global Agricultural Productivity Is Threatened by Increasing Pollinator Dependence without a Parallel Increase in Crop Diversification. *Glob. Change Biol.* **2019**, *25*, 3516–35127. [CrossRef] [PubMed]
16. Schott, C.; Mignolet, C.; Meynard, J.M. Les Oléoprotéagineux Dans Les Systèmes de Culture: Évolution Des Assolements et Des Successions Culturales Depuis Les Années 1970 Dans Le Bassin de La Seine. *OCL* **2010**, *17*, 276–291. [CrossRef]
17. Brisson, N.; Gate, P.; Gouache, D.; Charmet, G.; Oury, F.-X.; Huard, F. Why Are Wheat Yields Stagnating in Europe? A Comprehensive Data Analysis for France. *Field Crops Res.* **2010**, *119*, 201–212. [CrossRef]
18. Newton, A.C. Exploitation of Diversity within Crops—The Key to Disease Tolerance? *Front. Plant Sci.* **2016**, *7*, 665. [CrossRef] [PubMed]
19. Gaudio, N.; Escobar-Gutiérrez, A.J.; Casadebaig, P.; Evers, J.B.; Gérard, F.; Louarn, G.; Colbach, N.; Munz, S.; Launay, M.; Marrou, H.; et al. Knowledge and Future Research Opportunities for Modeling Annual Crop Mixtures. A Review. *Agron. Sustain. Dev.* **2019**, *39*, 20. [CrossRef]
20. Litrico, I.; Violle, C. Diversity in Plant Breeding: A New Conceptual Framework. *Trends Plant Sci.* **2015**, *20*, 604–613. [CrossRef] [PubMed]
21. Anten, N.P.R.; Vermeulen, P.J. Tragedies and Crops: Understanding Natural Selection To Improve Cropping Systems. *Trends Ecol. Evol.* **2016**, *31*, 429–439. [CrossRef] [PubMed]
22. Barot, S.; Allard, V.; Cantarel, A.; Enjalbert, J.; Gauffreteau, A.; Goldringer, I.; Lata, J.-C.; Le Roux, X.; Niboyet, A.; Porcher, E. Designing Mixtures of Varieties for Multifunctional Agriculture with the Help of Ecology. A Review. *Agron. Sustain. Dev.* **2017**, *37*, 13. [CrossRef]
23. Borg, J.; Kiær, L.P.; Lecarpentier, C.; Goldringer, I.; Gauffreteau, A.; Saint-Jean, S.; Barot, S.; Enjalbert, J. Unfolding the Potential of Wheat Cultivar Mixtures: A Meta-Analysis Perspective and Identification of Knowledge Gaps. *Field Crops Res.* **2018**, *221*, 298–313. [CrossRef]
24. Petit, S.; Cordeau, S.; Chauvel, B.; Bohan, D.; Guillemin, J.-P.; Steinberg, C. Biodiversity-Based Options for Arable Weed Management. A Review. *Agron. Sustain. Dev.* **2018**, *38*, 48. [CrossRef]
25. Vidal, T.; Gigot, C.; de Vallavieille-Pope, C.; Huber, I.; Saint-Jean, S. Contrasting Plant Height Can Improve the Control of Rain-Borne Diseases in Wheat Cultivar Mixture: Modelling Splash Dispersal in 3-D Canopies. *Ann. Bot.* **2018**, *121*, 1299–1308. [CrossRef] [PubMed]
26. Hajjar, R.; Jarvis, D.I.; Gemmill-Herren, B. The Utility of Crop Genetic Diversity in Maintaining Ecosystem Services. *Agric. Ecosyst. Environ.* **2008**, *123*, 261–270. [CrossRef]
27. Hamilton, W.D. The Genetical Evolution of Social Behaviour. I. *J. Theor. Biol.* **1964**, *7*, 1–16. [CrossRef] [PubMed]
28. Hamilton, W.D. The Genetical Evolution of Social Behaviour. II. *J. Theor. Biol.* **1964**, *7*, 17–52. [CrossRef] [PubMed]
29. Karban, R.; Shiojiri, K.; Ishizaki, S.; Wetzel, W.C.; Evans, R.Y. Kin Recognition Affects Plant Communication and Defence. *Proc. R. Soc. Lond. B* **2013**, *280*, 20123062. [CrossRef] [PubMed]
30. Chen, B.J.W.; Hajiboland, R.; Bahrami-Rad, S.; Moradtalab, N.; Anten, N.P.R. Presence of Belowground Neighbors Activates Defense Pathways at the Expense of Growth in Tobacco Plants. *Front. Plant Sci.* **2019**, *10*, 751. [CrossRef] [PubMed]
31. Zhu, S.; Morel, J.-B. Molecular Mechanisms Underlying Microbial Disease Control in Intercropping. *Mol. Plant-Microbe Interact.* **2019**, *32*, 20–24. [CrossRef] [PubMed]
32. Subrahmaniam, H.J.; Libourel, C.; Journet, E.-P.; Morel, J.-B.; Muños, S.; Niebel, A.; Raffaele, S.; Roux, F. The Genetics Underlying Natural Variation of Plant-Plant Interactions, a Beloved but Forgotten Member of the Family of Biotic Interactions. *Plant J.* **2018**, *93*, 747–770. [CrossRef] [PubMed]
33. Biedrzycki, M.L.; Venkatachalam, L.; Harsh, P.B. Transcriptome Analysis of *Arabidopsis thaliana* Plants in Response to Kin and Stranger Recognition. *Plant Signal. Behav.* **2011**, *6*, 1515–1524. [CrossRef]
34. Hu, L.; Robert, C.A.M.; Cadot, S.; Zhang, X.; Ye, M.; Li, B.; Manzo, D.; Chervet, N.; Steinger, T.; van der Heijden, M.G.A.; et al. Root Exudate Metabolites Drive Plant-Soil Feedbacks on Growth and Defense by Shaping the Rhizosphere Microbiota. *Nat. Commun.* **2018**, *9*, 2738. [CrossRef]
35. Mikaberidze, A.; McDonald, B.A.; Bonhoeffer, S. Developing Smarter Host Mixtures to Control Plant Disease. *Plant Pathol.* **2015**, *64*, 996–1004. [CrossRef]
36. Gandon, S.; Michalakis, Y. Local Adaptation, Evolutionary Potential and Host-Parasite Coevolution: Interactions between Migration, Mutation, Population Size and Generation Time: Local Adaptation and Coevolution. *J. Evol. Biol.* **2002**, *15*, 451–462. [CrossRef]
37. Read, A.F.; Taylor, L.H. The ecology of genetically diverse infections. *Science* **2001**, *292*, 1099–1102. [CrossRef] [PubMed]
38. Balmer, O.; Tanner, M. Prevalence and implications of multiple-strain infections. *Lancet Infect. Dis.* **2011**, *11*, 868–878. [CrossRef] [PubMed]
39. Tollenaere, C.; Susi, H.; Laine, A.L. Evolutionary and Epidemiological Implications of Multiple Infection in Plants. *Trends Plant Sci.* **2016**, *21*, 80–90. [CrossRef] [PubMed]
40. Agrios, G.N. *Plant Pathology*, 5th ed.; Academic Press: San Diego, CA, USA, 2005.
41. Doehlemann, G.; Ökmen, B.; Zhu, W.; Sharon, A. Plant pathogenic fungi. *Microbiol. Spectrum* **2017**, *5*, FUNK-0023-2016. [CrossRef]
42. Petersen, R.H. The rust fungus life cycle. *Bot. Rev.* **1974**, *40*, 453–513. [CrossRef]
43. Mengiste, T. Plant immunity to necrotrophs. *Annu. Rev. Phytopathol.* **2012**, *50*, 267–294. [CrossRef] [PubMed]

44. Wilson, R.A.; Talbot, N.J. Under pressure: Investigating the biology of plant infection by *Magnaporthe oryzae*. *Nat. Rev. Microbiol.* **2009**, *7*, 185–195. [CrossRef]
45. Talhinhas, P.; Baroncelli, R. *Colletotrichum* species and complexes: Geographic distribution, host range and conservation status. *Fungal Divers.* **2021**, *110*, 109–198. [CrossRef]
46. Tudzynski, P.; Scheffer, J. *Claviceps purpurea*: Molecular aspects of a unique pathogenic lifestyle. *Mol. Plant Pathol.* **2004**, *5*, 377–388. [CrossRef] [PubMed]
47. Bélanger, R.R.; Bushnell, W.R.; Dik, A.J.; Carver, T.L.W. (Eds.) *The Powdery Mildews: A Comprehensive Treatise*; APS Press: Saint Paul, MN, USA, 2002.
48. Willocquet, L.; Savary, S.; Fernandez, L.; Elazegui, F.A.; Castilla, N.; Zhu, D. Structure and validation of RICEPEST, a production situation-driven, crop growth model simulating rice yield response to multiple pest injuries for tropical Asia. *Ecol. Model.* **2002**, *153*, 247–268. [CrossRef]
49. Plantegenest, M.; Le May, C.; Fabre, F. Landscape epidemiology of plant diseases. *J. Roy. Soc. Interface* **2007**, *4*, 963–972. [CrossRef] [PubMed]
50. Fitt, B.D.L.; Huang, Y.J.; van den Bosch, F.; West, J.S. Coexistence of related pathogen species on arable crops in space and time. *Annu. Rev. Phytopathol.* **2006**, *44*, 163–182. [CrossRef] [PubMed]
51. Pariaud, B.; Ravigné, V.; Halkett, F.; Goyeau, H.; Carlier, J.; Lannou, C. Aggressiveness and its role in the adaptation of plant pathogens. *Plant Pathol.* **2009**, *58*, 409–424. [CrossRef]
52. Barrett, L.G.; Thrall, P.H.; Burdon, J.J.; Linde, C.C. Life history determines genetic structure and evolutionary potential of host–parasite interactions. *Trends Ecol. Evol.* **2008**, *23*, 678–685. [CrossRef] [PubMed]
53. White, J. The plant as a metapopulation. *Ann. Rev. Ecol. Syst.* **1979**, *10*, 109–145. [CrossRef]
54. Barthelemy, D.; Cargaglio, Y. Modélisation et simulation de l'architecture des arbres. *For. Enterp.* **1991**, *73*, 28–39.
55. De Reffye, P.; Houllier, F.; Blaise, F.; Barthelemy, D.; Dauzat, J.; Auclair, D. A model simulating above- and below-ground tree architecture with agroforestry applications. *Agrofor. Syst.* **1995**, *30*, 175–197. [CrossRef]
56. Begon, M.; Harper, J.L.; Towsend, C.R. *Ecology, Individuals, Populations and Communities*; Blackwell Science: London, UK, 2006.
57. Al-Naimi, F.A.; Garrett, K.A.; Bockus, W.W. Competition, facilitation, and niche differentiation in two foliar pathogens. *Oecologia* **2005**, *143*, 449–457. [CrossRef]
58. Pianka, E.R. *Competition and Niche Theory in Theoretical Ecology*; May, R.M., Ed.; Sinauer: Sunderland, MA, USA, 1981; pp. 167–196.
59. Alley, T.R. Competition theory, evolution, and the concept of an ecological niche. *Acta Biotheor.* **1982**, *31*, 165–179. [CrossRef] [PubMed]
60. Amarasekare, P.; Hoopes, M.F.; Mouquet, N.; Holyoa, M. Mechanisms of coexistence in competitive metacommunities. *Am. Nat.* **2004**, *164*, 310–326. [CrossRef] [PubMed]
61. Mideo, N. Parasite adaptations to within-host competition. *Trends Parasitol.* **2009**, *25*, 261–268. [CrossRef] [PubMed]
62. MacArthur, R.H. *Geographical Ecology*; Harper & Row: New York, NY, USA, 1972.
63. Putman, R.J. Competition and coexistence in a multi-species grazing system. *Acta Theriol.* **1986**, *31*, 271–291. [CrossRef]
64. Hellard, E.; Fouchet, D.; Vavre, F.; Pontier, D. Parasite–parasite interactions in the wild: How to detect them? *Trends Parasitol.* **2015**, *31*, 640–652. [CrossRef] [PubMed]
65. Susi, H.; Vale, P.F.; Laine, A.L. Host genotype and coinfection modify the relationship of within and between host transmission. *Am. Nat.* **2015**, *186*, 252–263. [CrossRef] [PubMed]
66. Dutt, A.; Rault, A.; Andrivon, D.; Jumel, S.; Le Roy, G.; Baranger, A.; Le May, C. Competition and facilitation among fungal plant parasites affect their life-history traits. *OIKOS* **2021**, *130*, 652–667. [CrossRef]
67. Fodor, E. Ecological niche of plant pathogen. *Ann. For. Res.* **2011**, *54*, 3–21.
68. Ploetz, R.C. Black Sigatoka of Banana. The Plant Health Instructor. 2001. Available online: https://www.apsnet.org/edcenter/apsnetfeatures/Pages/BlackSigatoka.aspx (accessed on 15 February 2024).
69. Balesdent, M.H.; Fudal, I.; Ollivier, B.; Bally, P.; Grandaubert, J.; Eber, F.; Chèvre, A.M.; Leflon, M.; Rouxel, T. The dispensable chromosome of *Leptosphaeria maculans* shelters an effector gene conferring avirulence toward *Brassica rapa*. *New Phytol.* **2013**, *198*, 887–898. [CrossRef]
70. Tivoli, B.; Banniza, S. Comparison of the epidemiology of ascochyta blights on grain legumes. *Eur. J. Plant Pathol.* **2007**, *119*, 59–76. [CrossRef]
71. Kus, J.V.; Zaton, K.; Sarkar, R.; Cameron, R.K. Age-related resistance in *Arabidopsis* is a developmentally regulated defense response to *Pseudomonas syringae*. *Plant Cell* **2002**, *14*, 479–490. [CrossRef] [PubMed]
72. Miller, M.E. Relationships between onion leaf age and susceptibility to *Alternaria porri*. *Plant Dis.* **1983**, *67*, 284–286. [CrossRef]
73. Reuveni, M.; Tuzun, S.; Cole, J.S.; Siegel, M.R.; Kuć, J. The effects of plant age and leaf position on the susceptibility of tobacco to blue mould caused by *Peronospora tabacina*. *Phytopathology* **1986**, *76*, 455–458. [CrossRef]
74. Pretorius, Z.A.; Rijkenberg, F.H.J.; Wilcoxson, R.D. Effects of growth stage, leaf position and temperature on adult-plant resistance of wheat infected by *Puccinia recondita* f.sp. *tritici*. *Plant Pathol.* **1988**, *37*, 36–44. [CrossRef]
75. Roumen, E.C.; Bonman, J.M.; Parlevliet, J.E. Leaf age related partial resistance to *Pyricularia oryzae* in tropical lowland rice cultivars as measured by the number of sporulating lesions. *Phytopathology* **1992**, *82*, 1414–1417. [CrossRef]
76. Le May, C.; Potage, G.; Andrivon, D.; Tivoli, B.; Outreman, Y. Plant disease complex: Antagonism and synergism between pathogens of the Ascochyta blight complex on pea. *J. Phytopathol.* **2009**, *157*, 715–721. [CrossRef]

77. Boixel, A.L.; Chelle, M.; Suffert, F. Patterns of thermal adaptation in a globally distributed plant pathogen: Local diversity and plasticity reveal two-tier dynamics. *Ecol. Evol.* **2022**, *12*, e8515. [CrossRef]
78. Khokhar, M.K.; Hooda, K.S.; Sharma, S.S.; Singh, V. Post flowering stalk rot complex of maize—Present status and future prospects. *Maydica* **2014**, *59*, 226–242.
79. Pfordt, A.; Ramos Romero, L.; Schiwek, S.; Karlovsky, P.; von Tiedemann, A. Impact of environmental conditions and agronomic practices on the prevalence of *Fusarium* species associated with Ear- and Stalk Rot in Maize. *Pathogens* **2020**, *9*, 236. [CrossRef]
80. Abdullah, A.S.; Moffat, C.S.; Lopez-Ruiz, F.J.; Gibberd, M.R.; Hamblin, J.; Zerihun, A. Host–multi-pathogen warfare: Pathogen interactions in co-infected plants. *Front. Plant Sci.* **2017**, *8*, 1806. [CrossRef]
81. Somera, T.S.; Mazzola, M. Toward a holistic view of orchard ecosystem dynamics: A comprehensive review of the multiple factors governing development or suppression of apple replant disease. *Front. Microbiol.* **2022**, *13*, 949404. [CrossRef] [PubMed]
82. Avelino, J.; Romero-Gurdia, A.; Cruz-Cuellar, H.F.; Declerck, F.A.J. Landscape context and scale differentially impact coffee leaf rust, coffee berry borer, and coffee root-knot nematodes. *Ecol. Appl.* **2012**, *22*, 584–596. [CrossRef] [PubMed]
83. Malézieux, E.; Crozat, Y.; Dupraz, C.; Laurans, M.; Makowski, D.; Ozier-Lafontaine, H.; Rapidel, B.; Tourdonnet, S.; Valantin-Morison, M. Mixing plant species in cropping systems: Concepts, tools and models. A review. *Agron. Sustain. Dev.* **2009**, *29*, 43–62. [CrossRef]
84. Ratnadass, F.; Avelino, J.; Fernandes, P.; Letourmy, P.; Babin, R.; Debert, P.; Deguine, J.P.; Grechi, I.; Naudin, K.; Rhino, B.; et al. Synergies and tradeoffs in natural regulation of crop pests and diseases under plant species diversification. *Crop Prot.* **2021**, *146*, 105658. [CrossRef]
85. Root, R.B. Organization of a plant-arthropod association in simple and diverse habitats—fauna of collards (*Brassica oleracea*). *Ecol. Monogr.* **1973**, *43*, 95–120. [CrossRef]
86. Van Bruggen, A.H.C.; Finckh, M.R. Plant diseases and management approaches in organic farming systems. *Ann. Rev. Phytopathol.* **2016**, *54*, 25–54. [CrossRef] [PubMed]
87. Mitchell, C.E.; Tilman, D.; Groth, J.V. Effects of grassland plant species diversity, abundance, and composition on foliar fungal disease. *Ecology* **2002**, *83*, 1713–1726. [CrossRef]
88. Mundt, C.C.; Léonard, K.J. Effect of host genotype unit area on development of focal epidemics of bean rust and common maize rust in mixtures of resistant and susceptible plants. *Phytopathology* **1986**, *76*, 895–900. [CrossRef]
89. Ngugi, H.K.; King, S.B.; Holt, J.; Julian, A.M. Simultaneous temporal progress of sorghum anthracnose and leaf blight in crop mixtures with disparate patterns. *Phytopathology* **2001**, *91*, 720–729. [CrossRef]
90. Schoeny, A.; Jumel, S.; Rouault, F.; Lemarchand, E.; Tivoli, B. Effect and underlying mechanisms of pea-cereal intercropping on the epidemic development of ascochyta blight. *Eur. J. Plant Pathol.* **2010**, *126*, 317–331. [CrossRef]
91. Gomez-Rodriguez, O.; Zavaleta-Mejia, E.; Gonzalez-Hernandez, V.A.; Livera-Munoz, M.; Cardenas-Soriano, E. Allelopathy and microclimatic modification of intercropping with marigold on tomato early blight disease development. *Field Crops Res.* **2003**, *83*, 27–34. [CrossRef]
92. Mills, D.J.; Coffman, C.B.; Teasdale, J.R.; Everts, K.L.; Anderson, J.D. Factors associated with foliar disease of staked fresh market tomatoes grown under differing bed strategies. *Plant Dis.* **2002**, *86*, 356–361. [CrossRef] [PubMed]
93. Gao, Y.; Ren, C.; Liu, Y.; Zhu, J.; Li, B.; Mu, W.; Liu, F. Pepper-Maize intercropping affects the occurrence of anthracnose in hot pepper. *Crop Protec.* **2021**, *148*, 105750. [CrossRef]
94. Ding, X.; Yang, M.; Huang, H.; Chan, Y.; He, X.; Li, C.; Zhu, Y.; Zhu, S. Priming maize resistance by its neighbours: Activating 1.4-benzoxazine-3-ones synthesis and defense gene expression to alleviate leaf disease. *Front. Plant Sci.* **2015**, *6*, 830. [CrossRef]
95. Campos, M.L.; Kang, J.H.; Howe, G.A. Jasmonate-triggered plant immunity. *J. Chem. Ecol.* **2014**, *40*, 657–675. [CrossRef]
96. Wu, J.; Bao, X.; Zhang, J.; Lu, B.; Sun, N.; Wang, Y.; Yang, N.; Xing, Y.; Gallaway, R.M.; Li, L. Facilitation between intercropped species increases micronutrient acquisition and control rust disease in maize. *Field Crops Res.* **2024**, *307*, 109241. [CrossRef]
97. Brooker, R.W.; Karley, A.J.; Newton, A.C.; Pakeman, R.J.; Schöb, C. Facilitation and sustainable agriculture: A mechanistic approach to reconciling crop production and conservation. *Funct. Ecol.* **2016**, *30*, 98–107. [CrossRef]
98. Kumar, V.; Mills, D.J.; Anderson, J.D.; Mattoo, A.K. An alternative agriculture system is defined by a distinct expression profile of select gene transcripts and proteins. *Proc. Natl. Acad. Sci. USA* **2004**, *101*, 10535–10540. [CrossRef]
99. Richard, B.; Jumel, S.; Rouault, F.; Tivoli, B. Influence of plant stage and organ age on the receptivity of *Pisum sativum* to *Mycosphaerella pinodes*. *Eur. J. Plant Pathol.* **2012**, *132*, 367–379. [CrossRef]
100. Li, L.; Tilman, D.; Lambers, H.; Zhang, F.S. Plant diversity and overyielding: Insights from belowground facilitation of intercropping in agriculture. *New Phytol.* **2014**, *203*, 63–69. [CrossRef]
101. Chaboussou, F. *Healthy Crops: A New Agricultural Revolution*; Jon Carpenter Publishing: Charlbury, UK, 2004.
102. Corre-Hellou, G.; Baranger, A.; Bedoussac, L.; Cassagne, N.; Cannavacciuolo, M.; Joelle, J.; Pelzer, E.; Piva, G. Interactions entre facteurs biotiques et fonctionnement des associations végétales. *Innov. Agron.* **2014**, *40*, 25–42.
103. Fernandez-Aparicio, M.; Amri, M.; Kharrat, M.; Rubiales, D. Effect of crop mixtures on chocolate spot development on faba bean grown in Mediterranean climates. *Crop Prot.* **2011**, *30*, 1015–1023. [CrossRef]
104. Staver, C.; Guharay, F.; Monterroso, D.; Muschler, R.G. Designing pest-suppressive multistrata perennial crop systems: Shade-grown coffee in Central America. *Agrofor. Syst.* **2001**, *53*, 151–170. [CrossRef]
105. Hamilton, W.D. The evolution of altruistic behavior. *Am. Nat.* **1963**, *97*, 354–356. [CrossRef]

106. López-Villavicencio, M.; Jonot, O.; Coantic, A.; Hood, M.E.; Enjalbert, J.; Giraud, T. Multiple infections by the Anther Smut Pathogen are frequent and involve related strains. *PLoS Pathog.* **2007**, *3*, e176. [CrossRef] [PubMed]
107. Tack, A.J.M.; Laine, A.-L. Ecological and evolutionary implications of spatial heterogeneity during the off-season for a wild plant pathogen. *New Phytol.* **2014**, *202*, 297–308. [CrossRef]
108. Schneider, D.J.; Collmer, A. Studying plant-pathogen interactions in the genomics era: Beyond molecular Koch's postulates to systems biology. *Annu. Rev. Phytopathol.* **2010**, *48*, 457–479. [CrossRef]
109. Fukami, T. Historical contingency in community assembly: Integrating niches, species pools, and priority effects. *Annu. Rev. Ecol. Evol. Syst.* **2015**, *46*, 1–23. [CrossRef]
110. Fukami, T.; Mordecai, E.A.; Ostling, A. A framework for priority effects. *J. Veg. Sci.* **2016**, *27*, 655–657. [CrossRef]
111. Karvonen, A.; Seppala, O.; Valtonen, E.T. Host immunization shapes interspecific associations in trematode parasites. *J. Anim. Ecol.* **2009**, *78*, 945–952. [CrossRef]
112. Hoverman, J.T.; Hoye, B.J.; Johnson, P.T.J. Does time matter? How priority effects influence the outcome of parasite interactions within hosts. *Oecologia* **2013**, *173*, 1471–1480. [CrossRef] [PubMed]
113. Halliday, F.W.; Umbanhowar, J.; Mitchell, C.E. Interactions among symbionts operate across scales to influence parasite epidemics. *Ecol. Lett.* **2017**, *20*, 1285–1294. [CrossRef] [PubMed]
114. Barbetti, M.J.; Khan, T.N.; Pritchard, I.; Lamichhane, J.R.; Aubertot, J.N.; Corrales, D.C.; You, M.P. Challenges with managing disease complexes during application of different measures against foliar diseases of field pea. *Plant Dis.* **2021**, *105*, 616–627. [CrossRef] [PubMed]
115. Schenk, P.M.; Carvalhais, L.C.; Kazan, K. Unraveling plant–microbe interactions: Can multi-species transcriptomics help? *Trends Biotechnol.* **2012**, *30*, 177–184. [CrossRef] [PubMed]
116. Crandall, S.G.; Gold, K.M.; Jiménez-Gasco, M.D.M.; Filgueiras, C.C.; Willett, D.S. A multi-omics approach to solving problems in plant disease ecology. *PLoS ONE* **2020**, *15*, e0237975. [CrossRef] [PubMed]
117. Massart, S.; Adams, I.; Al Rwahnih, M.; Baeyen, S.; Bilodeau, G.J.; Blouin, A.G.; Lebas, B.S. Guidelines for the reliable use of high throughput sequencing technologies to detect plant pathogens and pests. *Peer Community J.* **2022**, *2*, e62. [CrossRef]
118. Montarry, J.; Cartolaro, P.; Delmotte, F.; Jolivet, J.; Willocquet, L. Genetic structure and aggressiveness of *Erysiphe necator* populations during grapevine powdery mildew epidemics. *Appl. Environ. Microbiol.* **2008**, *74*, 6327–6332. [CrossRef] [PubMed]
119. Castel, M.K.; Mailleret, L.; Andrivon, D.; Ravigné, V.; Hamelin, F.M. Allee effects and the evolution of polymorphism in cyclical parthenogens. *Am. Nat.* **2013**, *183*, 75–88. [CrossRef] [PubMed]
120. Alizon, S.; de Roode, J.C.; Michalakis, Y. Multiple infections and the evolution of virulence. *Ecol. Lett.* **2013**, *16*, 556–567. [CrossRef]
121. Dutt, A.; Andrivon, D.; Jumel, S.; Le Roy, G.; Baranger, A.; Leclerc, M.; Le May, C. Life history traits and trade-offs between two species of the ascochyta blight disease complex of pea. *Plant Pathol.* **2020**, *69*, 1108–1124. [CrossRef]
122. Rynkiewicz, E.C.; Pedersen, A.B.; Fenton, A. An ecosystem approach to understanding and managing within-host parasite community dynamics. *Trends Parasitol.* **2015**, *3*, 212–221. [CrossRef] [PubMed]
123. Elena, S.F.; Bernet, G.P.; Carrasco, J.L. The games plant viruses play. *Curr. Opin. Virol.* **2014**, *8*, 62–67. [CrossRef] [PubMed]
124. Severns, P.M.; Sykes, E.M. Indicator Species Analysis: A useful tool for plant disease studies. *Phytopathology* **2020**, *110*, 1860–1862. [CrossRef] [PubMed]
125. Lamichhane, J.R.; Venturi, V. Synergisms between microbial pathogens in plant disease complexes: A growing trend. *Front. Plant Sci.* **2015**, *6*, 385. [CrossRef] [PubMed]