Received: 14 April 2020 | Accepted: 20 November 2020

DOI: 10.1002/2688-8319.12040

Ecological Solutions and Evidence | BRITISH ECOLOGICAL SOCIETY

RESEARCH ARTICLE

Assessing the phylogenetic host breadth of millet pathogens and its implication for disease spillover

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Handling Editor: Alicia Foxx

Abstract

1. Increasing agriculture intensification has led to dramatically improved crop yields; however, this shift in agricultural practice has been accompanied by increasing threats from new and emerging plant pathogens. While the pathogens associated with crop species are often well studied, especially within North America and Europe, less is known about pathogen pressures on crops elsewhere, and our ability to predict the emergence of novel pathogens is limited. Here, we model phylogenetic constraints on the distribution of pathogens of millet – one of the most important crops in Africa.
2. We conducted a literature review to compile a database of common millet pathogens and the non-millet host crops associated with each. We then characterized the phylogenetic host range for each pathogen using measures of mean pairwise distance (MPD) and mean nearest taxon distance (MNTD) separating crop hosts.
3. We detected robust phylogenetic clustering for both metrics of phylogenetic dispersion (MPD and MNTD). Evidence for phylogenetic clustering tended to be stronger (more negative standard effect sizes) and more variable for MPD than for MNTD.
4. Although patterns for individual pathogens were variable, we did not find significant differences in phylogenetic dispersion of hosts among pathogen types (bacteria, viruses and fungi). However, in several cases, we observed evidence of phylogenetic clustering in evolutionarily distant host clades, a possible signal of occasional large phylogenetic host jumps.
5. We show that pathogens cluster on closely related hosts, and it is thus likely that closely related millets also share similar pathogen communities. On average, the probability of a pathogen host shift may, therefore, be predicted by the phylogenetic relatedness between host species. However, host shifts between distantly related hosts are not infrequent. This finding has relevance not only for the design of agronomic systems to reduce disease spillover but also for biological control agents risk analysis, quarantine regulations in international trade and our understanding of the distribution and abundance of plants in natural systems.

KEYWORDS
disease spillover, mean nearest taxon distance, mean pairwise distance, millet pathogens, pathogen host range, phylogenetic distribution

1 | INTRODUCTION

Plant pathogens are a threat to native and agricultural species, including widely planted species such as millet and maize that are the staple crops in some regions (Patil, 2017). Plant pathogens have shown an increase in emergence over time, likely due to the global movement of agricultural species and climate change (Bebber, Holmes, & Gurr, 2014). Some of these pathogens can infect a broad range of plant hosts, but in most cases pathogens tend to be specialized on a subset of the plant species found within any local community assemblage (Gilbert & Webb, 2007; Pearse & Altermatt, 2013). Predicting the likely host range of emerging plant pathogens is important in designing agronomic systems, global regulatory policies in international trade, and risk analysis of biological control agents (Gilbert & Webb, 2007). For example, knowing which crop species are susceptible to which pathogens can help increase yields by reducing crop losses to disease through informing growers about which crops can be grown together and which should not (Brooker et al., 2015). While the majority of research on plant pathogens has focused on commercial and commonly grown crop species (Altman, 2018; Gilligan, 2008), we still lack knowledge about their full host range, and we are thus limited in our ability to predict the likelihood of pathogen host shifts, including spillover to other agricultural species.

The distribution of pathogens across host species is a product of multiple processes, including co-diversification, biogeography of both hosts and pathogens and host shifts. Within an agricultural setting, we are most interested in the latter. The evolutionary arms race between specialist plant pathogens and their plant hosts has been well characterized (Anderson et al., 2010; Barrett, Kniskern, Bodenhausen, Zhang, & Bergelson, 2009): as plant hosts evolve new defences, pathogens evolve ways to neutralize these defences. This arms race leads to increased pathogen specialization (Anderson et al., 2010; Barrett et al., 2009). Because host defence traits tend to be phylogenetically conserved (Gilbert & Parker, 2016), such that closely related host plants share similar defence traits, the host breadth of plant pathogens also tends to be phylogenetically constrained, with more closely related host species more likely to share pathogens (Gilbert & Webb, 2007; Gilbert, Briggs, & Magarey, 2015; Pearse & Altermatt, 2013). Phylogeny can thus help make predictions of likely novel pathogen–host associations (Fountain-Jones et al., 2018), but to make accurate models the breadth of hosts a pathogen currently infests also needs to be considered (see, e.g. Farrell, Elmasri, Stephens, & Davies, 2020). Here, we develop a model to describe the host range of millet pathogens, an important component of the staple diet in the semi-arid tropic regions of Asia and Africa (Hazeltine & Bull, 2003).

The major pathogenic microorganisms infesting millet crops are viruses, bacteria and fungi (Haq & Ijaz, 2020). These pathogen types differ in their intrinsic biological properties, for example, in terms of size, shape, function, genetic content and virulence on the host, which might be expected to influence the breadth of hosts they infect. Most plant viruses rely on biotic vectors for transmission and survival, with the majority transmitted by insect vectors. The mode of transmission/infection can be classified as persistent (e.g. continued feeding by the vector), semi-persistent or non-persistent (e.g. stylet borne) (Dietzgen et al., 2016). Bacteria need to invade the plant tissue to infect, commonly through wounds caused by weather damage, humans, insects and nematodes, or through natural openings such as stomata (Vidaver and Lambrecht, 2004). Bacterial diseases are significantly influenced by temperature and moisture, and a difference of a few degrees can determine whether or not a bacterial disease will develop (Hays and Watson, 2019). Fungi cause the majority of infectious plant diseases (Isleib, 2012) and may be biotrophic (requiring a live host) or necrotrophic (living off nutrients from dead tissue) (Oliver & Ipcho, 2004). Fungal pathogens are spread primarily by spores, which are often produced in abundance, and can be disseminated by wind currents, water, soil, insects and birds or through transport of infected dead plant materials. Fungal spores are highly resistant to temperature and humidity changes (Jennings & Lysek, 1999; Kader, 2002). The large differences in modes of transmission and routes of infection among these broad pathogen types could influence both the opportunity to encounter and the likelihood to successfully infect novel hosts.

In this paper, we investigate the distribution of millet pathogens across their alternative non-millet domesticated hosts using phylogenetic measures of mean pairwise distance (MPD) and mean nearest taxon distance (MNTD). These two metrics quantify the evolutionary relatedness among species, MPD measures the average relatedness among species pairs, whereas MNTD measures the relatedness of closest relatives. Using such measures, we can gain a better understanding of the evolutionary constraints determining the host breadth of millet pathogens, and thus the likelihood of future host shifts. Through the study of millet, we may also gain insights into pathogen risks to other crops, as well as new and emerging threats to millet. The increased pathogen risk from the widespread planting of monocultures (Salaheen & Biswas, 2019) has encouraged the increased application of chemicals such as fungicides and bactericides, many with harmful environmental impacts (Aktar, Sengupta, & Chowdhury, 2009; Dudley and Alexander, 2017). Consequently, there has been growing interest in lower intensity agricultural practices, including inter-cropping and land-sharing approaches that support both agricultural production and wildlife (Singh & Singh, 2017). However, such practices elevate the risk of pathogen spillover (Boudreau, 2013); we attempt to better quantify these risks.

Millets are cereal crops suitable for environments prone to drought and extreme heat with an indefinite storage life, have no grain storage pathogens and have a short growing season, which can be as little as 65 days, reducing the temporal window for pathogen infection (Patil, 2017). They are nutritionally superior to other commonly grown grains, providing a more economical source of protein, vitamins and minerals, making them an important crop species in the world today, particularly in more rain-fed areas lacking well-developed irrigation systems (Patil, 2017). There are seven different millet species, including pearl millet (*Pennisetum glaucum*), which is the most commonly planted millet, finger millet or ragi (*Eleusine coracana)*, Italian or foxtail millet (*Setaria italica*), common or proso millet (*Panicum miliaceum*), kodo millet (*Paspalum scrobiculatum*), little millet (*Panicum miliare/ P. sumatrense*) and barnyard millet (*Echinochloa frumentacea*). Maize (*Zea mays*), common

wheat (*Triticum aestivum*), sorghum (*Sorghum bicolor*) and rice (*Oryza sativa*) that are cultivated in a similar agro-ecological zone as millet and all belonging to the Poaceae family share the highest number of pathogens with millet (Taylor & Duodu, 2018) and are thus likely to pose the greatest immediate risk of pathogen spillover. Pearl millet (*Pennisetum glaucum*), mainly produced in the semi-arid plains of southern Asia and the sub-Saharan regions of Africa (Hazeltine & Bull, 2003), has over 28 different pathogen types recorded as infesting it. However, with over 90% of millet production within developing countries in Africa and Asia, there has been only limited research on the pathogens affecting these indigenous cereal crops (Hazeltine & Bull, 2003). While most pathogens of millet show phylogenetic clustering within Poaceae, there are notable exceptions such as *Athelia rolfsii* – southern blight – which has a very broad host range and infects a large number of distantly related domesticated plant species. A better understanding of the diversity and host range of millet pathogens will contribute significantly towards expanding the planting and agricultural development of this understudied but valuable crop.

2 | MATERIALS AND METHODS

2.1 | Data collection

We first employed a Web of Science (https://www.webofknowledge.com) search – using the following search terms: 'millet' AND 'pathogen' OR 'disease' – followed by a detailed literature review to compile a database of the recorded diseases for millet species, and a list of non-millet host species susceptible to the same suite of pathogens. We included all millet pathogens and their host species within the global pool, irrespective of geographical origin. We then performed a secondary, targeted search on both the scientific and common names of each recorded millet pathogen species to generate a more comprehensive list of non-millet host species. A key source of data on millet pathogens was V. Patil's reference guide *Millets and Sorghum: Biological and Genetics Improvement* (Patil, 2017). These searches returned a list of 48 common millet pathogens (see SI Table 1), which we separated into three broad pathogen types: fungi, viruses and bacteria.

2.2 | Host species and phylogeny

We extracted the phylogenetic relationships of domesticated angiosperms (943 species obtained from Milla et al., 2018) from the Qian and Jin (2016) updated Zanne et al. (2014) megaphylogeny of vascular plants (PhytoPhylo). Unsampled taxa were inserted into the megaphylogeny, using the R library S.PhyloMaker (see Qian & Jin 2016 and Revell, 2012), at the node defining the minimally inclusive clade encompassing the higher taxonomic group within which they are placed. This returned a fully resolved, ultrametric phylogeny of domesticated plants (as short hand, we refer to these plant taxa collectively as crop species), with branch lengths in millions of years. We visually checked the tree topology for accuracy using figtree (http://tree.bio.ed.ac.uk/software/figtree/).

2.3 | Phylogenetic metrics

We used the Picante package in R (Kembel et al., 2010) to calculate the average phylogenetic distance (MPD) between all recorded hosts for each pathogen, returning 48 measures of MPD. We also recorded for each pathogen the mean minimum pairwise distance separating hosts (MNTD). While MPD is thought to reflect phylogenetic structuring across the entire phylogeny, MNTD reflects phylogenetic structures closer to the tips (Che et al., 2018; Kembel et al., 2010; Tucker et al., 2017). Exploring both these metrics allows us to identify taxa that show different phylogenetic structure at different tree depths. A pathogen that occurs only on very closely related hosts will have large negative values of both MPD and MNTD, whereas a pathogen that occurs on close relatives but also infests a distantly related host clade will demonstrate a high degree of clustering using a metric of MNTD, but will show only weak clustering using a metric of MPD.

Finally, we calculated the standard effect sizes (SES) for each measure assuming a simple null model (tip swap) of randomly shuffling tip labels across the tips of phylogeny, as implemented in Picante, with 999 randomizations, as follows:

$$\mathrm{SES}_{\mathrm{metric}} = \frac{\mathrm{Metric}_{\mathrm{observed}} - \mathrm{mean}\,(\mathrm{Metric}_{\mathrm{null}})}{\mathrm{sd}\,(\mathrm{Metric}_{\mathrm{null}})}, \quad (1)$$

where $\mathrm{Metric}_{\mathrm{observed}}$ is the observed MPD or MNTD, and $\mathrm{Metric}_{\mathrm{null}}$ is the MPD or MNTD obtained from the randomly shuffled tip labels. A negative SES value (z-score < 0) indicates that the observed phylogenetic clustering (indexed by MPD or MNTD) is greater than that expected by chance. In contrast, positive SES values (z-score > 0) indicate greater phylogenetic evenness – greater phylogenetic distance among co-occurring species – than expected by chance.

2.4 | Statistical analyses of pathogen host breadth

We calculated the phylogenetic signal in pathogen richness using Blomberg's K and the log-transformed total number of pathogens recorded for each millet species (Revell, 2012). We also contrasted the distribution of MPD vs MNTD across all 48 pathogens. To examine trends in phylogenetic host range, we constructed a linear regression model of the SES for MPD against MNTD and identified the top five pathogen outliers – those pathogens showing the largest departure from the modelled relationship as measured by the magnitude of their residual deviation. These pathogens are characterized as infecting several closely related plant hosts within two or more phylogenetically distant clades. The MNTD and MPD regression allows us to highlight the taxa which show different phylogenetic structure at different tree depths. Finally, we used an analysis of variance (ANOVA) to test for differences in MPD and MNTD between the three pathogen types (viruses, bacteria, fungi).

As sensitivity analyses, we also recalculated strength of phylogenetic signal in pathogen host range for plant hosts just within Poaceae, and additionally estimated SES values for MPD and MNTD excluding

TABLE 1 Observed MPD and MNTD for each pathogen type, showing SES (*z*-values) and significant departure from null expectations of no phylogenetic structure (*p*-values)

Pathogen type	Number of hosts	MPD	*z*-value	*p*-value	MNTD	*z*-value	*p*-value
Alternaria spp.	19	170.83	−6.32	0.001	40.92	−4.66	0.001
Anthracocystis paspali-thunbergii	5	3.52	−7.01	0.001	2.71	−4.85	0.001
Athelia rolfsii	195	201.53	−15.55	0.001	16.48	−9.47	0.001
Balansia oryzae	8	24.78	−8.40	0.001	7.99	−4.83	0.001
Bipolaris setariae	24	98.19	−11.75	0.001	45.09	−4.43	0.001
Claviceps fusiformis	2	0.23	−3.35	0.001	0.23	−3.33	0.001
Curvularia penniseti	28	147.14	−9.05	0.001	9.84	−6.25	0.001
Drechslera nodulosum	8	111.75	−6.08	0.001	53.07	−3.64	0.001
Exserohilum rostratum	8	10.71	−9.55	0.001	4.50	−4.97	0.001
Fusarium moniliforme	13	157.93	−5.64	0.001	9.59	−5.18	0.001
Microdochium sorghi	4	7.52	−5.93	0.001	5.57	−4.62	0.001
Paramyrothecium roridum	21	232.00	−2.90	0.005	27.00	−5.04	0.001
Puccinia substriata var. indica penicillariae	5	227.04	−1.43	0.121	1.17	−4.84	0.001
Pyrenophora dematioidea	21	17.95	−15.17	0.001	2.65	−6.22	0.001
Pyricularia grisea	17	22.08	−13.14	0.001	3.27	−5.68	0.001
Pyricularia setariae	2	0.31	−3.32	0.001	0.31	−3.50	0.001
Rhizoctonia solani	941	281.88	NA	NA	35.71	NA	NA
Sarocladium oryzae	3	25.37	−4.42	0.002	14.79	−4.07	0.001
Sclerophthora macrospora	4	26.47	−5.51	0.001	17.06	−4.27	0.001
Sclerospora graminicola	12	7.39	−12.17	0.001	1.65	−5.33	0.001
Uromyces eragrostidis	2	23.25	−3.11	0.007	23.25	−3.01	0.013
Uromyces setariae-italic	3	1.28	−4.71	0.001	0.79	−4.42	0.001
Ustilago crameri	2	0.31	−3.32	0.001	0.31	−3.50	0.001
Ustilago panici-frumentacei	3	1.82	−4.78	0.001	1.82	−4.30	0.001
Ustilago paradoxa	3	1.82	−4.78	0.001	1.82	−4.30	0.001
Pseudomonas avenae	28	24.70	−18.08	0.001	2.63	−6.53	0.001
Pseudomonas syringae	7	7.83	−8.58	0.001	2.38	−5.00	0.001
Xanthomonas axonopodis pv. Pennamericanum	2	1.82	−3.34	0.002	1.82	−3.19	0.003
Black streaked dwarf virus	11	26.93	−10.93	0.001	8.34	−5.07	0.001
Guinea grass mosaic virus	6	4.74	−7.97	0.001	2.70	−4.86	0.001
Indian peanut clump virus	21	115.77	−10.01	0.001	23.60	−5.36	0.001
Maize dwarf mosaic virus	13	17.16	−12.38	0.001	4.05	−5.37	0.001
Maize streak virus	22	23.75	−15.73	0.001	4.45	−6.03	0.001
Panicum mosaic virus	10	11.19	−10.57	0.001	6.16	−5.27	0.001
Sugarcane mosaic virus	5	12.26	−6.93	0.001	8.33	−4.72	0.001
Wheat streak mosaic virus	17	20.20	−13.65	0.001	1.74	−5.79	0.001

the two most widespread pathogen taxa (*Rhizoctonia solani* and *Athelia rolfsii*). The phylogenetic signal in host range is affected by the phylogenetic universe of hosts against which the patterns are tested. For pathogens that infect all, or nearly all hosts, such as these, it is not possible to generate a meaningful null distribution with which to contrast empirical observations.

3 | RESULTS

We collected data on known pathogens for all seven recognized millet species and recorded their non-millet host range across crop species. We recorded information on 48 pathogens, and 936 susceptible crop species (Supplementary Table 1). While phylogenetic signal in pathogen

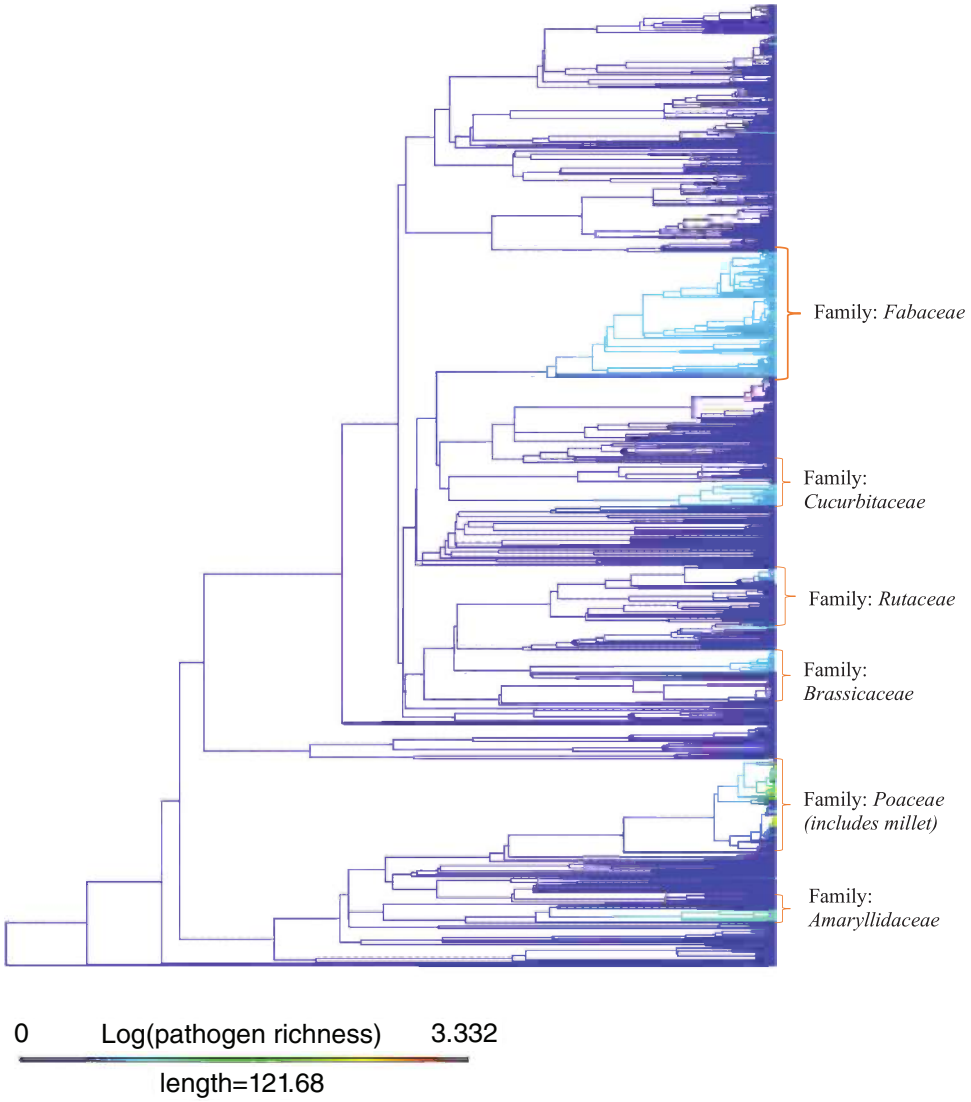

FIGURE 1 Phylogeny of millet-pathogen hosts with branch lengths proportional to t ime (millions of years), and with colours indicating the logarithm of the number of pathogens per host. Phylogenetic signal in pathogen richness is low (Blomberg's $K < 0.01$); however, hosts closely related to millets share more millet pathogens. Internal branches are shaded for illustration only, assuming a form of evolutionary averaging, from the tips of the tree to the root, whereby trait values follow a random walk (Brownian motion) on the tree topology. The two extreme generalists, *Rhizoctonia solani* and *Athelia Rolfsii*, have been excluded from the analysis. Pearl millet (*Pennisetum glaucum*) is shown to have the highest number of recorded pathogens (28 pathogens), with maize (*Zea Mays* – 21 pathogens), common wheat (*Triticum aestivum* – 17 pathogens), sorghum (*Sorghum bicolor* – 17 pathogens) and rice (*Oryza sativa* – 11 pathogens) the non-millet host species sharing the highest number of pathogens with millet

richness was low (Blomberg's $K < 0.01$), hosts closely related to millets tend to share more millet pathogens (see Figure 1). The strength of phylogenetic signal in pathogen richness for crops restricted to Poaceae was also low (Blomberg's $K < 0.01$). The highest recorded pathogen load (28 pathogens) was for pearl millet (*Pennisetum glaucum*), and the lowest (seven pathogens) was for kodo millet (*Paspalum scrobiculatum*), with an average of 15 pathogens per millet species. Maize and sorghum, which are cultivated in a similar agro-ecological zone as millet, shared the greatest number of pathogens with millet species (21 and 17 pathogens, respectively), while most other crop species shared few pathogens with millet with the exception of *Athelia rolfsii*, a broad range facultative fungus, and the soil-borne pathogenic fungus, *Rhizoctonia solani*, also with a very large host range, and which may possibly infect all crop species (Nagaraj, Sunkad, Pramesh, Naik, & Patil, 2017).

We found evidence for strong phylogenetic clustering for both metrics of phylogenetic dispersion (MPD and MNTD), as indicated by negative SES (*z*-values; Table 1). Observed *z*-values for MPD demonstrate both more extreme (minimum value = −18) and more negative median values (median = −8.2) in comparison to MNTD (MNTD minimum = −9, and median = −5), with the latter also demonstrating lower variance (Figure 2). *Athelia rolfsii* is identified as an outlier in its phylogenetic dispersion as measured by MNTD, this pathogen is unusual in infecting a large number of distantly related plant species. Results were broadly unchanged when excluding the two pathogens with the widest host breadth (*Rhizoctonia solani* and *Athelia Rolfsii*), but the mean

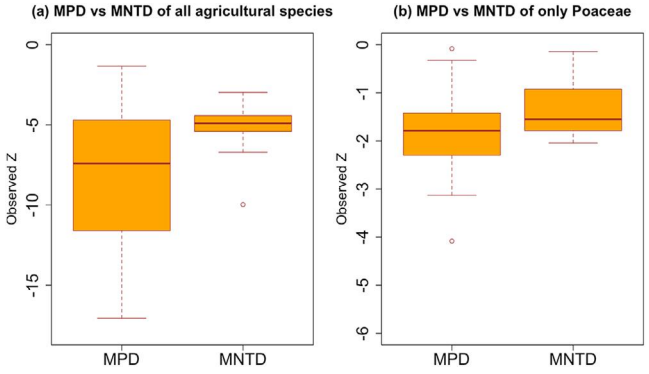

FIGURE 2 SES (*z*-values) for MPD and MNTD for (a) all agricultural host species used in our study and (b) Poaceae host species only. MNTD measures the mean distance between each species within a community and its closest relative. While MPD is thought to reflect phylogenetic structuring across the entire phylogeny, MNTD reflects phylogenetic structures closer to the tips. Strong phylogenetic clustering (more negative SES values) is evident in both metrics, even when we subset the data to Poaceae; however, the magnitude of the phylogenetic clustering within Poaceae hosts is less than that observed when calculated across all hosts, likely because the limited taxon sampling within Poaceae restricts the possible range of values that can be obtained in the null randomizations

SES for MPD and MNTD decreased. Similarly, phylogenetic clustering was also observed even when restricting the analysis to Poaceae hosts only, although the magnitude was decreased somewhat (Figure S1 in the Supporting Information).

Measures of the SES of MPD and MNTD were highly correlated (regression model of MPD against MNTD: intercept = −3.1, slope = 0.23; p-value < 0.001, $r^2 = 0.62$; Figure 3). We identified pathogens showing large departure from the model fit and highlight the top five outlier pathogens with largest residual deviation: *Athelia rolfsii*, *Paramyrothecium roridum*, *Bipolaris setariae*, *Curvularia penniseti*, *Puccinia substriata* var. *indica* (Figures 3 and 4). All these, aside *Puccinia substriata* var. *indica*, are facultative fungal plant pathogens. Notably, all but one fall below the fitted regression line, indicating that their SES for MNTD is lower than predicted from the SES for MPD.

We found no significant statistical difference in phylogenetic dispersion of hosts among pathogen types (bacteria, fungi and viruses) as indexed by either MPD or MNTD (all $p > 0.05$ from the one-way ANOVA of MPD and MNTD among pathogen types; Figure S2 in the Supporting Information). In all cases MPD had a more negative median value and higher variance relative to MNTD.

4 | DISCUSSION

Our observations on millet pathogens suggest a strong phylogenetic signal in the pathogen host range whereby closely related hosts share similar pathogen communities and pathogens cluster on closely related host species. Previous work has illustrated similar patterns in non-domesticated species (e.g. Gilbert & Webb, 2007; Parker et al., 2015; Pearse & Altermatt, 2013); however, only a few studies have explored the phylogenetic host range of pathogens in an agricultural crop (see Gilbert, Magarey, Suiter, & Webb, 2012, for an exception). Similar biogeography of closely related plant species could facilitate pathogen sharing, as close relatives will likely be exposed to the same suite of pathogens (Che et al., 2018). Agricultural crops, however, are often planted beyond their native range and are thus exposed to various novel pathogens for which they would not have (co)evolved defences. That we still detect such a strong phylogenetic signal in their pathogens is, therefore, notable, and indicates a deep phylogenetic conservatism in plant defences and the abilities of the pathogen to overcome those defences.

While we did not find evidence that the number of millet pathogens was phylogenetically structured using a standard measure of phylogenetic signal – Blomberg's *K* (Blomberg, Garland, & Ives, 2003) – hosts closely related to millets share more millet pathogens (Figure 1). However, we also identify likely instances of large phylogenetic host shifts and extraordinary wide host breadth for some pathogens such as *Athelia rolfsii* and *Rhizoctonia solani*. *Athelia rolfsii*, *for example* infects most annual crops in over 100 different families, including multiple species in the diverse crop families Amaryllidaceae and Brassicaceae. By describing the host phylogenetic distribution of millet pathogens, we not only gain a better understanding of the predictors of pathogen host range, but we also improve our ability to make predictions of pathogen range expansion and potential future pathogen spillover (Gilbert & Parker, 2016; Gilbert & Webb, 2007).

We observed large differences in the number of pathogens recorded for different millet species. Pearl millet (*Pennisetum glaucum*) has the greatest number of recorded pathogens, while kodo millet (*Paspalum scrobiculatum*) has the fewest. The difference in pathogen richness among hosts could be due to both differences in their morphological or chemical defence traits (Schuldt et al., 2017) and a reflection of the large variation in the geographical extent of their cultivation (Hazeltine & Bull, 2003; Patil, 2017). For example, pearl millet is extensively grown across Africa, India, China and the southern United States (Hazeltine & Bull, 2003), which might expose this millet to many pathogens native to different geographical regions. In contrast, kodo millet has a much narrower geographical extent of planting, grown mainly in India, and is thus exposed to a more limited suite of pathogens (Patil, 2017). Maize (*Zea mays*), common wheat (*Triticum aestivum*), sorghum (*Sorghum bicolor*) and rice (*Oryza sativa*) share the highest number of pathogen types with millet. These Poaceae species are also grown in the semi-arid tropic regions of Asia and Africa where millet is commonly planted and are currently of even greater economic importance globally (Hazeltine & Bull, 2003). These crops thus not only pose a threat of pathogen spillover to millet, but millet may act as a reservoir for pathogens to which these species are also susceptible. It is worth noting that there is also important variation in the research effort dedicated to different crop species and that this could make it difficult to quantify total pathogen pressure as more pathogens tend to be recorded on better-studied species (Stephens et al., 2016). For example, a simple Web of Science search reveals a large number of papers on pearl millet (1790 papers – 28 recorded pathogens) and

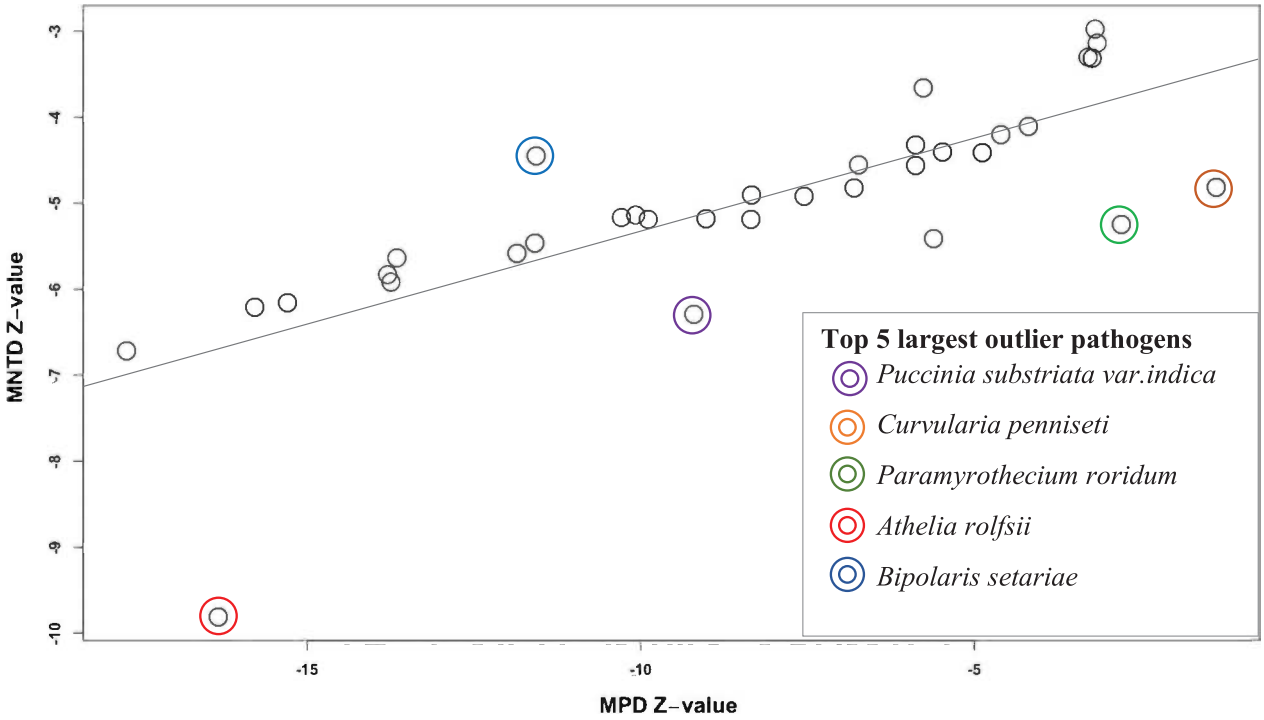

FIGURE 3 Scatter plot of the SES (*z*-values) for MPD against the SES for MNTD, with the fitted line from the linear regression model (see main text), and highlighting the top five largest outlier pathogens (red = *Athelia rolfsii* , blue = *Bipolaris setariae*, green = *Paramyrothecium roridum*, orange = *Curvularia penniseti*, purple = *Puccinia substriata*. var. *indica*). The measures of the SES of MPD and MNTD are highly correlated supporting our claim of strong phylogenetic lustering using both metrics. Outliers highlight pathogens in which the strength of clustering differs with phylogenetic depth relative to the general fit of the relationship between the standard effects sizes of MPD and MNTD observed across all pathogens

finger millet (875 papers – 20 pathogens), but few papers on barnyard millet (76 papers – 9 pathogens) and kodo millet (90 papers – 7 pathogens).

Emerging pathogens are classified as infectious agents with increasing incidence following introduction into a new host population (Engering, Hogerwerf, & Slingenbergh, 2013). Among the many emerging and re-emerging pathogens infesting millet, *Rhizoctonia solani*, *Puccinia* spp., *Fusarium* spp. and *Ustilago* spp. are seen as the main threat to crop yield, causing the greatest ecological or economic impact (Dean et al., 2012). These pathogens of millet are also among those with the widest host range, with *Rhizoctonia solani* potentially capable of infecting all agricultural species (e.g. Gonzalez et al., 2011). The heteroecious rusts, such as *Puccinia substrata*, infest phylogenetically distant clades of obligate hosts, with tight phylogenetic clustering within each host group (Zhao, Wang, Chen and Kang, 2016). These pathogens are mostly common in the semi-arid plains, such as sub-Saharan Africa (Hazeltine & Bull, 2003), where subsistence farming is common, and there is a strong reliance on locally grown crops (e.g. Hazeltine & Bull, 2003). Predicting the risk of emergence of crop pathogens in this region would have large socio-economic benefits, improve food security, and support more sustainable agricultural development (Gilbert & Parker, 2016; Gilbert & Webb, 2007).

All but one pathogen examined (exception *Rhizoctonia solani)* showed phylogenetic clustering. Such phylogenetic patterning is consistent with an evolutionary arms race between pathogens and their plant hosts, driving pathogen phylogenetic specialization (Antonovics et al., 2013; Barrett et al., 2009), and strong phylogenetic conservatism in host plant defence traits (Gilbert & Parker, 2016). Under assumptions of phylogenetic conservatism (Wiens et al., 2010), a plant pathogen with the necessary traits to successfully evade the defences of one particular plant host should also overcome the defence mechanisms of closely related plant species (Gilbert et al., 2015). The strength of evolutionary conservatism in plant defence traits may determine the phylogenetic breadth of hosts for a given pathogen. Greater extremes (more negative values) and greater variance for the mean of the pairwise distance separating hosts species in comparison to the distance between most closely related hosts (nearest taxon index) suggests that phylogenetic conservatism in host defence traits extends beyond immediate relatives and has a relatively deep evolutionary signature (Kembel et al., 2010; Mazel et al., 2016).

While close relatives are most likely to share pathogens, we also identify some pathogens that are relatively over-dispersed or demonstrate a pattern of clustering within phylogenetically distant clades. For example, the phylogenetic distribution of *Paramyrothecium roridum* and *Athelia rolfsii*, both fungi, indicates that they can both infect very distantly related plant hosts, perhaps evidence of large phylogenetic host jumps. These pathogens might be uniquely adept at overcoming plant evolved defences, or it is possible that distantly related hosts have converged on similar defence mechanisms through convergent evolutionary processes. The top outliers from the regression model of

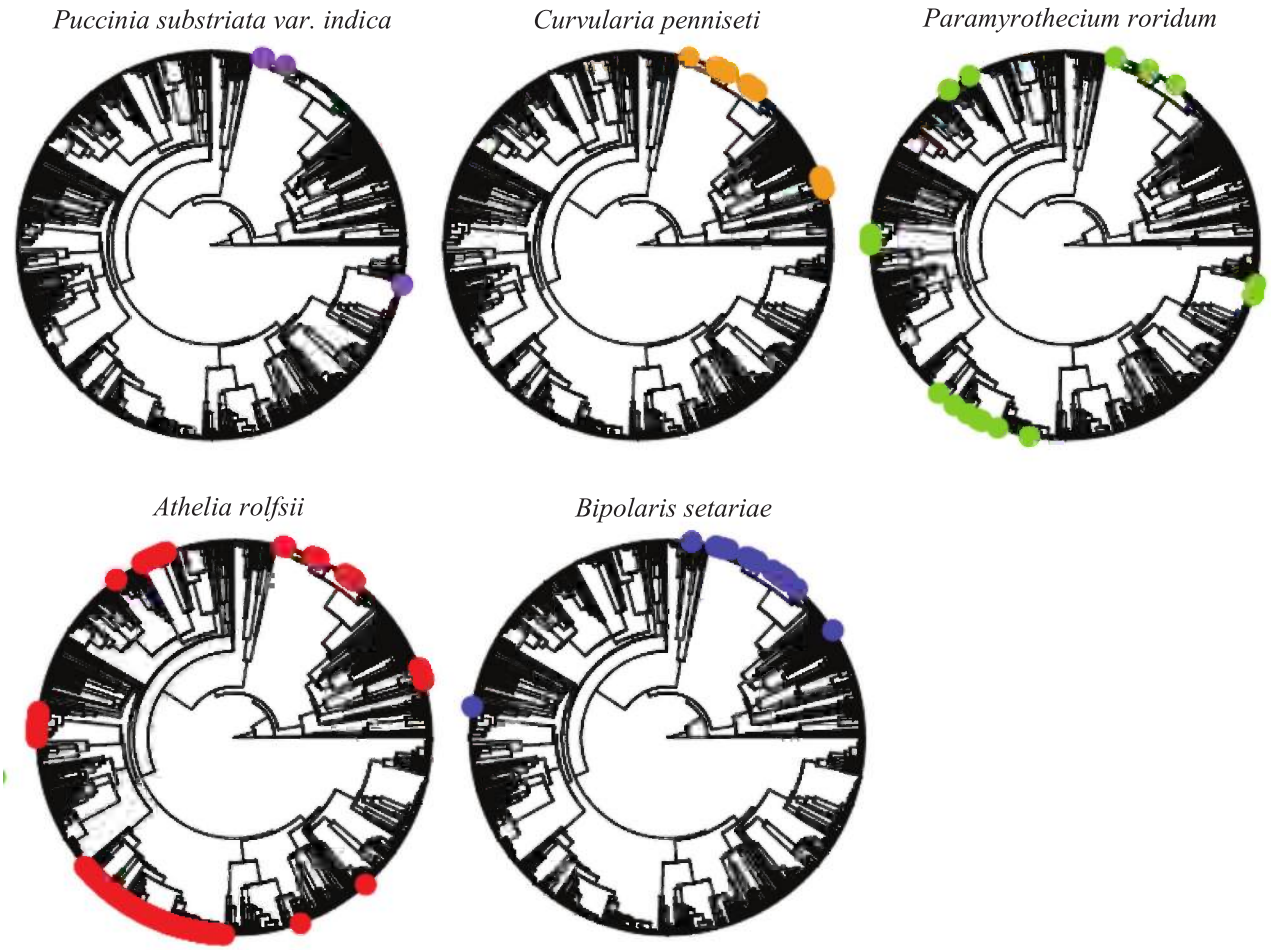

FIGURE 4 Identical phylogenetic trees highlighting the hosts for each of the five pathogens identified as large outliers from the regression of MPD against MNTD, see Figure 3. Colours correspond to the named pathogen highlighted in Figure 3. Pathogens outliers characteristically infect several closely related plant hosts within two or more phylogenetically distant clades

pairwise distances are all fungal pathogens, and most synthesize many enzymes to attack plant cells and are characterized by the formation of oxalic acid, which may allow them to infect and attack a wide host breadth (Brigand, 2019) – high concentration of oxalic acid induces programmed cell death in plants and facilitates necrotrophic fungus development (Lehner et al., 2008). *Rhizoctonia solani* is unusual in that it may be capable of infecting all agricultural crops; it is a cosmopolitan fungal pathogen, composed of over eight different anastomotic and intraspecific groups, each known to cause a variety of diseases in different hosts (e.g. sheath blight, stem rot and dumping off) (Ogoshi, 1987). This intraspecific diversity and broad geographic extent likely capture important ecological and epidemiological diversity, and perhaps helps explain the extreme generalism of this crop pathogen (Nagaraj et al., 2017; Ogoshi, 1987). The intraspecific variation in *Rhizoctonia solani* also highlights a more general challenge to modelling host breadth in widespread pathogens. It can be difficult to reliably differentiate strain diversity, and even more problematic to assess whether they should be better considered as evolutionarily independent taxa, or included as a single, but highly variable, species. Nonetheless, we believe that if data on strain diversity were available it would most likely emphasize the strong phylogenetic clustering we detect, and thus our analyses are likely conservative. To assess robustness of our results, we evaluated strength of clustering with and without including the two most widespread pathogen taxa (*Rhizoctonia solani* and *Athelia Rolfsii*), and show results are broadly similar.

We found no significant difference in the strength of clustering between different pathogen types (bacteria, fungi and viruses). There are three possible explanations for this observation. First, a lack of statistical power to detect the differences that might exist between these pathogen types. For example, we only have data on five bacterial pathogens. Second, the distribution of pathogens may be primarily determined by environment and management practices, such as where crops are planted. Third, there is some fundamental evolutionary constraint that limits the phylogenetic breadth of the host that applies across all pathogen types. Currently, we lack the data to distinguish among these explanations, but phylogenetic patterns for a much greater number of tree pathogens appear to reveal similar trends (unpublished data). Nonetheless, it is worth noting that even within each of these three broad pathogen types, there are significant biological differences that might influence the host range of pathogens.

For example, viruses are mostly transmitted by insects, which can be classified as either persistent or non-persistent. Persistent transmission necessitates continued feeding by vectors while non-persistent transmission may be stylet borne, and does not involve sustained feeding (Dietzgen et al., 2016). Such differences in transmission mode could potentially explain why some viruses, such as the non-persistent Indian peanut clump virus, have a wide host plant range, while others, such as the persistent Panicum mosaic virus, are restricted to within a single plant family, in this example, Poaceae (Dietzgen et al., 2016). More data are needed to evaluate whether these differences reflect a general trend across plant viruses.

Our results support growing evidence for strong phylogenetic conservatism in the host species a pathogen can infect (Davies & Pedersen 2008; Gilbert & Webb, 2007; Streicker et al., 2010) but also suggest that simple taxonomic or phylogenetic classifications of pathogen host range, for example, the number of hosts or higher taxonomic designations (e.g. Pedersen, Altizer, Poss, Cunningham, & Nunn, 2005), or phylogenetic breadth of hosts (e.g. Poulin & Mouillot, 2003; Poulin, Krasnov, & Mouillot, 2011), might fail to accurately capture the true distribution of pathogens across host phylogeny (see also Park et al., 2018). We observe occasional, but not uncommon, pathogen distributions consistent with over-dispersion or host jumps that might be overlooked by current phylogenetic approaches (e.g. Gilbert et al., 2012).

4.1 | Management implications

Improving our knowledge of the host range of pathogens can benefit both our understanding of species coexistence in natural populations, for example, via Janzen–Connell mechanisms (Comita et al., 2014; Parker et al., 2015), and agricultural practices by reducing opportunities for disease spillover (Power & Mitchell, 2004). It is now widely appreciated that industrial-scale monocropping, despite increasing yields, can lead to worsening pathogen problems as there are few barriers to pathogen spread (Salaheen & Biswas, 2019). Intercropping is a common alternative practice, especially in the tropics, but could have implications for cross-species pathogen transmission (Boudreau, 2013). Our results support suggestions that an intercropped agronomic system that involves mixing phylogenetically distant host species might be preferable, as this should decrease the possibility of pathogen spillover and reduce the risk of cross-infection (Gilbert & Webb, 2007). However, large phylogenetic host shifts remain difficult to predict and may be more common than previously assumed. Pathogens making large phylogenetic host jumps may have particularly severe impacts on naïve hosts (see Farrell & Davies 2019); identifying such high-risk pathogens is an urgent research priority.

A better knowledge of the pathogen host range may help identify pathogens that pose a high risk of spillover before they emerge. Such proactive surveillance can reduce chemical inputs to control plant disease and promote less environmentally harmful and more sustainable agricultural practices. Through improved understanding of disease ecology, such as the interactions among plants, their pathogens and the environment, it may be possible to implement management practices to reduce cross-species infection when intercropping, while maintaining increasing agricultural yield to support the fast-growing human population (Sokolow et al., 2019; Power & Mitchell, 2004). We have provided a first pass for the common pathogens of millet, examining their phylogenetic distribution across plant hosts. Our approach is easily transferable to different crop species and non-agricultural species planted beyond their native range. The strength of phylogenetic conservatism in the host range may differ for pathogens of different hosts. Ultimately, we should strive towards modelling the full host-pathogen association matrix to generate predictions of unique pathogen–host infestations (see, e.g. Farrell et al. 2020).

ACKNOWLEDGEMENTS

We are grateful to The Davies Lab at the University of British Columbia for their support. We also thank the MasterCard Foundation for financial support for ES.

AUTHORS' CONTRIBUTIONS

ES led the project and designed the methodology under the supervision and approval of JD. ES carried out the literature search for the compiled data base and conducted the phylogenetic metrics and statistical analyses as recommended by JD. Both authors were involved in the writing of the manuscript and gave final approval for publication.

DATA AVAILABILITY STATEMENT

The database on the recorded diseases for millet species and the list of non-millet domesticated host species susceptible to the same suite of pathogens can be accessed at figshare (Ssebuliba, 2020).

PEER REVIEW

The peer review history for this article is available at https://publons.com/publon/10.1002/2688-8319.12040.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

How to cite this article: Ssebuliba E, Davies TJ. Assessing the phylogenetic host breadth of millet pathogens and its implication for disease spillover. *Ecol Solut Evidence*. 2021;2:e12040. https://doi.org/10.1002/2688-8319.12040