Is there a trade-off between aggressiveness and host generalism across *Phytophthora infestans* (potato and tomato blight) populations? A synthesis of global cross-inoculation data

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Abstract

Phytophthora infestans is among the most destructive of plant diseases. Pathogen lineages have varying degrees of host preference among potato and tomato, from no specificity to extreme specialisation. How host specificity impacts fitness among pathogen populations is unclear. Comparing the pathogenicity of both population types can shed light on the determinants of host specificity in *P. infestans*. A trade-off between generalism and quantitative aggressiveness can explain the variation among pathogen populations. I assessed the existence of such a trade-off by analysing the global cross-inoculation data on potato and tomato isolates. In addition, host specificity was assessed for change over time. The synthesis included 44 and 34 data points for potato and tomato isolates respectively. Potato isolates overall did not prefer their original host significantly more than tomato isolates. Isolates from potato can generally infect tomato with similar aggressiveness (in terms of lesion growth rate) and specificity as in reverse. There was a modest trade-off between generalism and aggressiveness, where high specificity was associated with significantly greater aggressiveness. However, this trade-off did not explain the presence of generalist strains that are equally aggressive as specialist strains. A portion of generalists has high aggressiveness equal to the most host specific strains. For tomato isolates, there was a significant change towards generalism over the last few decades. This synthesis reveals several novel observations on the evolutionary ecology of the blight pathogen. Many unresolved questions on host specificity and aggressiveness remain which are discussed.

Keywords: Local adaptation, specialisation, fitness, *Solanum tuberosum*, *Solanum lycopersicum*

Introduction

Local adaptation and specialisation to hosts can determine the spatial and temporal evolutionary dynamics of plant pathogens (Kröner *et al.*, 2017). It is a process where populations under divergent selection achieve better performance in their habitat of origin than all other possible habitats. In plant pathogens, local adaptation manifests as specificity for the original host over other potential hosts; and in the extreme case, host specialisation where the pathogen is only pathogenic to the original host. Host specificity can be readily quantified in cross-inoculation trials of isolates from different host species or varieties. Adaptation to hosts, environmental conditions, and fungicide application can change the genetic composition of pathogen populations and thus their epidemiological properties (Cohen, 2002; Pangga *et al.*, 2011; Engering *et al.*, 2013). Understanding the effects of these drivers is important in predicting their impacts under climate change (Anderson *et al.*, 2004; Pangga *et al.*, 2011).

Phytophthora infestans de Bary is a globally distributed heterothallic oomycete pathogen on solanaceous plants. It was the causal agent of the Great Irish Potato Famine that was responsible for millions of deaths. It is among the most destructive of plant pathogens (Chowdappa *et al.*, 2015; Fry *et al.*, 2015). The pathogen was formally described in the 1800s (as *Botrytis infestans* by M.J. Berkeley 1846) and primarily causes disease on potato and tomato (termed late / potato and tomato blight respectively). Populations of *P. infestans* have been continuously re-emerging through time and space (Fry *et al.*, 2015). Strains vary dramatically in host specificity among the two host species, from non-specific generalists (i.e., infects both hosts with equal pathogenicity) to extreme obligate specialists (i.e., only infects on one host and zero or near-zero pathogenicity on the other host). The dominance and turnover of genotypes through time have been as intriguing to investigators as it was impeding to managers (Fry *et al.*, 2015). The global blight populations have undergone several remarkable changes throughout their history, from pan-globally dominant lineages (FAM-1 followed by US-1) to the diversity of genotypes seen today (reviewed in Yoshida *et al.*, 2013; Saville *et al.*, 2016). Adaptive processes within the pathogen and environmental conditions underlie the severity of the impacts on each host (Sicard *et al.*, 2007; Fry *et al.*, 2015). The fluxes in the global blight populations are likely explained by variation in the effects of pathogens on their hosts (Pariaud *et al.*, 2009). Therefore, a better understanding of how *P. infestans* adapt and specialise to potato and tomato, and the determinants of pathogenicity is needed to effectively predict the emergence of disease. *Phytophthora infestans* on potato and tomato hosts also presents a great opportunity to test whether generalism tends to carry fitness costs or 'no-cost' generalism prevails (Remold, 2012). In the literature, aggressiveness is broadly defined as the negative effect that a pathogen has on the host, and can be considered to be any quantitative component of pathogenicity that is non-specific to host genotypes (reviewed in Pariaud *et al.* 2009). In this study, aggressiveness is defined explicitly by lesion growth rate on host tissue. In *P. infestans* it is a life-history trait that is a key predictor of

epidemic severity, and is widely evaluated across studies (Birhman & Singh, 1995; Pariaud et al., 2009; Montarry *et al*. 2010).

Potato and tomato are traditionally recognised as primary and secondary hosts for *P. infestans* respectively. Previous reports often found that potato isolates have a higher preference for potato, but tomato isolates are generalist on both hosts (Legard *et al.*, 1995; Michalska *et al.*, 2016; Kröner *et al.*, 2017). Infectivity on tomato likely evolved relatively recently during initial invasion from the centre of origin in the 1800s, because isolates from the native range were non-pathogenic to tomato (Legard *et al.*, 1995). Blight was long believed to spread from potato to adjacent tomato crops, but not vice versa (Berg, 1926; Small, 1938; Legard *et al.*, 1995; Cohen, 2002). However, more recently it has been revealed that transmission regularly occurs from tomato to potato in relatively new strains such as 13_A2, US-22, and US-23 (Hu *et al.*, 2012; Frost *et al.*, 2016). The disease is now generally considered to be as impactful on tomato as it is on potato (Fry *et al.*, 2015; Kröner *et al.*, 2017).

The history of the disease is particularly thought-provoking owing in part to a long timeline of observations on host specificity. Prior to the early 1900s blight on tomato was uncommon (Berg, 1926). Reports from the earliest cross-inoculation trials were mostly published after 1900, when tomato blight was becoming more prevalent globally. Those reports did not mention finding host specificity, or they found that isolates from both hosts were destructive on both hosts under lab or field conditions (Berg, 1926; Oyarzun *et al.*, 1998). However, this changed in the following period after 1915 when remarkable specificity was frequently noted either on both potato and tomato isolates or in potato isolates only (including isolates from Holland, Australia, USA and the UK; Berg, 1926). This was followed by the emergent US-1 lineage which became dominant from the 1930s. During this period, studies on isolates from different regions showed the majority had no remarkable specificity, but potato isolates were still more likely host specific. For example, Wilson and Gallegly (1955) examined isolates from Scotland, the Netherlands, Canada and the USA, and reported that 8 out of 29 potato isolates were host specific versus 2 out of 16 tomato isolates. Isolates from Israel at the time showed similar proportions (9 out of 25 potato isolates and 2 out of 25 for tomato isolates; Kedar *et al.*, 1959). Other cross-inoculation studies conducted during US-1 dominance in Japan (Kishi, 1962), the USA (before 1970, Flier *et al.*, 2003), and Germany (Günther *et al.*, 1970) also reported little or no specificity. This period was followed by the emergence of an array of successive lineages from the mid- to late-1980s up to the present where US-1 becomes either extinct or rare relative to other strains. Studies on surviving (or remaining) US-1 isolates overwhelmingly found host specificity in isolates from either host species within a given site, and mostly in tomato isolates, but not potato isolates (Erselius *et al.*, 1997; Oyazun *et al.*, 1998; Vega-Sanchez *et al.*, 2000; Suassuna *et al.*, 2004; Chen *et al.*, 2009). In North America this pattern is reversed where the remaining US-1 potato isolates tend to be host specific, but not the tomato isolates (Goodwin *et al.*, 1995; Legard *et al.*, 1995; Platt, 1999).

Other more recently emerged strains may or may not be host specific, such as US-8 and BR-1 primarily pathogenic on potato (Legard *et al.*, 1995; Suassuna *et al.*, 2004), US-21 was host specific on tomato (Hu *et al.*, 2012), and EC-1 has low specificity (Oliva *et al.*, 2010). Importantly, host specificity is a population-level trait so there may be variation within a lineage. Different populations of the same lineage can also inhabit the two hosts at the same sites where their host specificity may vary (e.g. Erselius *et al.*, 1997).

On the one hand, *P. infestans* strains that are more aggressive with a greater lesion size and higher sporulation on their host should become dominant (Gisi *et al.*, 2011). For instance, the greater aggressiveness of US-8 (in terms of area under lesion expansion curve) is likely an important factor in the replacement of US-1 in south-western Canada (Miller *et al.*, 1998), and similarly the more aggressive 2_A1 displaced the less aggressive US-1 in east Africa (Njoroge *et al.*, 2018). In Brazil, the more aggressive BR-1 with much faster lesion growth on potato (but less aggressive on tomato) displaced US-1 from potato only, (Suassuna *et al.*, 2004). The 13_A2 lineage is dominant in several continents and is more aggressive than other lineages, and was demonstrated to competitively exclude other lineages in the field, which likely explains its quick expansion across Europe within 3 years (Cooke *et al.*, 2012). On the other hand, strains that can infect a wider range of hosts, including resistant hosts should also be favoured (Seidl Johnson & Gevens, 2014). The 13_A2 lineage was also able to infect previously resistant potato cultivars (Cooke *et al.*, 2012). In Taiwan, the US-11 is aggressive on both potato and tomato and rapidly displaced a potato specific US-1 (Chen *et al.*, 2009). The generalist US-6 and US-7 lineages similarly replaced the potato specific US-1 in North America (Hwang *et al.*, 2014). The question then remains: why aren't pathogen populations all explicitly both highly aggressive and generalist across hosts?

The relationship between pathogen pathogenicity components and fitness is complex. Although there is abundant evidence that greater aggressiveness is advantageous, conversely there have been a few suggestions that the most pathogenic strains are not most transmissible because hosts are weakened or killed too quickly (Pasco *et al.*, 2016; Mariette *et al.*, 2016). This is in line with the 'virulence-transmission trade-off' hypothesis which posits that high host mortality limits transmission rate (note that the "virulence" term was used to mean quantitative pathogenicity on the host) (Acevedo *et al.*, 2019). However, the effects of relative population size (of both pathogens and hosts) are often not considered. In the case of *P. infestans* this hypothesis also does not consider the effects of alternate host habitats, such as tomato plants or wild *Solanum* species (Frost *et al.*, 2016). For instance, a strain that is extremely pathogenic on potato can have lower aggressiveness on tomato to compensate the assumed lowered transmission rate on potato if the other host is locally available. Even if high pathogenicity is indeed linked with lower transmissibility, a trade-off between a generalist strategy (among potato and tomato hosts) versus aggressiveness could explain the variation in host specificity

among *P. infestans* potato and tomato strains (Thrall & Burdon, 2003; Pariaud *et al.*, 2009). Such a trade-off would be in line with generalists being 'the jack of all trades but a master of none' and specialists being 'the master of some' (Remold, 2012). Conversely, it is also evident that strains highly aggressive on both hosts exist (i.e., 'the master of all' strategy), including some invasive isolates of US-11 (Chen *et al.*, 2009) and 13_A2 (Chowdappa *et al.*, 2015). Thus our current understanding of the relationship between aggressiveness and host specificity remains unclear.

A range of cross-inoculation trials on *P. infestans* from potato and tomato have been conducted over the past few decades. Meta-analytic approaches can be used to synthesise the evidence to uncover patterns or processes across studies in plant pathology (Madden & Paul, 2011). A quantitative synthesis of this global data is likely to enable us to draw general conclusions on the relationship between host specificity and aggressiveness. Also, given the historical population changes associated with potential major host specificity shifts, a data synthesis can reveal how host specificity is changing over time. Selection for strains virulent on both hosts can decrease overall host specificity over time. Hence, the following hypotheses were tested: (1) there will be a positive association between host specificity and aggressiveness on the original host (as defined by lesion growth rate), (2) potato isolates will overall have greater host specificity than tomato isolates, (3) host specificity will tend to reduce over time, and (4) isolates of the older US-1 lineage will have lower aggressiveness but higher specificity than those of successive lineages.

Methods

Data collation

To identify studies performing cross-inoculations using potato and tomato isolates of *P. infestans*, I searched ISI Web of Science (Clarivate) and Google Scholar (https://scholar.google.com) in January 2022 using the following terms: 'Phytophthora infestans' AND 'tomato' OR 'potato'. Only trials conducted on potato and tomato hosts were included. Studies that tested pathogenicity on only one out of these two hosts were excluded, as well as studies that did not report the host of origin for isolates (or the host is not determinable based on the isolate information). As such, isolates were classified either as potato isolates or tomato isolates based on the host of origin. Studies must describe some quantitative measure of pathogenicity (e.g., lesion size, AUDPC, degree of infection). Thus, those reporting only qualitative pathogenicity are excluded. Studies that tested multiple isolates were pooled to one data point (for each host of origin) to avoid pseudoreplication.

From each study, the following were recorded: (1) the host of origin of the isolates (potato or tomato). Data of isolates from the same host species were pooled; (2) a quantitative measurement of pathogen pathogenicity on each of potato and tomato under controlled conditions and variance. Most commonly these are from the inoculation of plant parts or whole plants (e.g., mortality, sporulation rate, lesion

area). Semi-quantitative metrics such as disease severity scores were also accepted as host specificity was analysed using non-parametric tests. Where more than one measure of aggressiveness was reported, lesion size on host tissue (converted to lesion growth rate per day) was preferred over other measures such as AUDPC and plant mortality; (3) original geographical location of the isolate; and (4) time of isolation from hosts (calendar year). The average isolation year was recorded if data from multiple isolates were pooled.

The differences in pathogenicity of isolates on the two hosts were standardized using effect size as $\log_{10}$ response ratio (LRR). Highly negative LRR values (< -1) are associated with host specificity (i.e., non-pathogenic on the other host). Conversely, a highly positive LRR (> 1) indicates the pathogen is only aggressive on the other host and therefore the host of origin is likely a sink habitat (such as the US-8 isolate from tomato in Legard *et al.*, 1995). Lesion growth rate (hereafter 'LGR' in millimetres expansion per day) was estimated from the lesion size data in the time following inoculation. The standardized LGR is used to compare the pathogen aggressiveness on the two hosts across studies.

In addition, I compared the host specificity and aggressiveness of US-1 isolates and other lineages. From all the studies collected from the literature search, studies that tested US-1 isolates were identified. Part of these studies also tested isolates that are not US-1 along with other isolates (which were pooled with US-1 in the main analyses). Those were un-pooled and entered as separate entries comprising of 'US-1' and 'Other' lineage categories. By limiting the analysis on studies that tested both US-1 and successive lineages within the same experiment, it allows for a more direct comparison. Successive lineages are predicted to be more aggressive than US-1 (e.g. Legard *et al.*, 1995) and more generalist across both hosts (e.g. Chen *et al.*, 2009; Njoroge *et al.*, 2016).

Statistical analyses

For the LRR data, a non-parametric bootstrap approach was used to estimate the mean effect sizes and confidence intervals (95% CI). Bootstrapping is based on the method used in Van den Noortgate and Onghena (2005) and involves randomly subsampling with replacement over 1000 iterations. This approach is more suited to cases where many pathogenicity trials did not report the variance (as compared to a classical meta-analysis approach). Non-parametric bootstrapping does not emphasise thresholds for statistical significance, but emphasises effect size and confidence intervals (Rillig *et al.*, 2019). Normality in the lesion growth data was checked using quantile-quantile plots (Q-Q plots), which showed that the distribution of the data was normal (Fig. S1). To assess whether high specificity is associated with greater aggressiveness, the relationship between LGR on the original host and the difference with LGR on other host was analysed using linear regressions. The LGR of US-1 versus other lineages in subset data was analysed using t-tests. The time and LRR data were not

normally distributed, so median-based linear models (mblm) were used to test the relationships between LRR and time (year of isolation), and between LRR and aggressiveness on the original host. All analyses were conducted using R (v.3.6.1, R Core Development Team 2019). Bootstrapping was conducted using the *boot* package (v. 1.3-22) and the *metaphor* package (v. 2.1-0) was used to calculate effect sizes. The *mblm* package (v.0.12.1) was used to run the median-based models.

Results

This search included 78 host specificity comparisons from 44 studies (Fig. 1). Most studies (32 studies) conducted cross-inoculation trials on both potato and tomato isolates of *P. infestans* within the same trial. There were a total of 44 and 34 data points for potato and tomato isolates respectively. The isolates are from all over the globe (Fig. 2), and all inoculation trials were conducted in controlled conditions. Of the 78 data points, 43 reported lesion size (or growth) that was usable for the aggressiveness data. Ten of the studies tested US-1 isolates along with isolates of more successively emergent lineages (including BR-1, EC-1, US-6, US-7, US-8, US-11, and other unnamed isolates of A2 mating type). An outlying data point with LRR value of greater than 1 (i.e., non-pathogenic on the original host but highly pathogenic on the other host) was removed from all analyses (shown in Fig. 3).

For the host specificity data (LRR), the vast majority of pathogenicity traits were directly related to lesions (63 out of 78 cases, e.g., lesion size, growth, percentage, or number), and 5 cases were based on the evaluation of lesions (proportion of plants or leaves with lesions or disease ratings based on the type of lesions – sporulating, expanding or non-expanding). The remainder (10 cases) include sporulation (density or proportion of lesions with sporulation) and overall disease severity. The aggressiveness (lesion growth rate) data were entirely from detached leaf assays. The inoculation approach used was mostly detached leaf assays (64 out of 78 cases, with one combining data from detached stem and leaf inoculations), 4 cases were of leaf inoculation *in planta*, 10 cases used spray inoculation and a single case was of tuber and stem inoculation. Data sources and references are provided in the Supplementary Material (Table S1). The isolation time of pathogen isolates from plants in the field ranged from 1970 to 2017.

Potato and tomato isolates generally had a modest but statistically significant specificities for their host of origin (LRR estimate -0.26 [-0.09, -0.52] and -0.18 [-0.01, -0.41] 95% bootstrap CI for potato and tomato isolates respectively, $P < 0.05$); that is, pathogenicity on the other host is overall reduced to ~55% and ~66% of that on the original host for potato and tomato isolates, respectively. The specificity difference between potato (LRR -0.26) versus tomato isolates (LRR -0.18) was not significant ($P = 0.81$). The host specificity data from each region is summarised in Fig. 3. From the LGR data, there was a significant positive relationship between aggressiveness on the original host

and the difference in aggressiveness among hosts for potato ($P < 0.001$), but not for tomato, although this relationship is also strongly positive ($P = 0.07$, Table 1; Fig. 4). This overall result is also supported in the omnibus analysis between host specificity (LRR) versus aggressiveness (LGR) on the original host ($P < 0.05$, Table 2, Fig. 5). These together indicate that host specificity is associated with greater aggressiveness, and overall the most host specific strains may be up to 3-5 times more aggressive than generalist strains (Fig. 4). There was no significant relationship between LRR and isolation time for potato isolates ($P = 0.54$), but for tomato isolates there was a significant positive relationship ($P < 0.01$, Table 3; Fig. 6).

Focussing on 'surviving' isolates of the old US-1 lineage versus other successively emerged lineages, the sample size available for analysis was relatively low ($k = 11$ and $k = 10$ for US-1 and other isolates respectively) and fewer reporting lesion growth rates ($k = 7$ and $k = 6$ respectively). Two US-1 tomato isolates had extreme specificity for potato and was non-pathogenic on its original host which were removed from the analyses (LRR 2.31 and 1.58). There were no significant differences in LRR among isolates of US-1 and other lineages (LRR -0.22 vs. -0.30 for US-1 and other lineages respectively; t = -0.30, df = 15.94, $P = 0.77$). Furthermore, there was no significant difference in aggressiveness on the host of origin among US-1 and other successively emergent lineages (LGR 4.12 vs. 3.47 mm per day respectively; t = 0.54, df = 2.84, $P = 0.63$). The low availability of data between US-1 and new isolates means that the results for this comparison should be taken with caution. Nonetheless as the analyses pertains to a subset of studies that assessed US-1 and other isolates in tandem, it is indicated that the remaining US-1 isolates are not necessarily less aggressive than those of other lineages.

Discussion

The current synthesis of the global data suggests a generalist strategy tends to be costly to aggressiveness, which can help explain the persistence of host specific strains in the presence of generalist strains even when both hosts are often abundantly available. *Phytophthora infestans* is a hemibiotrophic pathogen with a clear necrotrophic phase, so aggressiveness towards hosts should generally be under selection in the absence of trade-offs (Jarosz & Davelos, 1995; Montarry *et al.*, 2007). Aggressive strains often succeed less aggressive ones (e.g. Miller *et al.*, 1998; Cooke *et al.*, 2012), therefore a lower aggressiveness is likely a fitness cost for the ability to effectively infect both hosts. This finding is consistent with the first hypothesis that host specific *P. infestans* strains will be more aggressive than generalist strains. Several factors could potentially explain why generalist strains tend to be lower in aggressiveness. First, evolutionary theory predicts that specialisation may be associated with increased fitness in that environment, so naturally specialists would be fitter regardless of any fitness costs associated with generalism (i.e. 'the jack of all trades' strategy; Remold, 2012). Second, genetic change involved in adaptation to the other host environment may involve fitness costs due to antagonistic pleiotropy. This includes costs associated with virulence genes to

overcome host resistance factors (Pariaud *et al.*, 2009; Montarry *et al.*, 2007, 2010). Third, specialists should have a lower accumulation of deleterious alleles (Whitlock, 1996), which in theory can hinder the competitiveness of a generalist in the presence of specialists (Kawecki, 1994). To my knowledge this has not yet been investigated in blight. Yang et al. (2016) found no evidence that mutation accumulation was involved in a trade-off between thermal tolerance and pathogen growth rate in *P. infestans*, but may be explained by antagonistic pleiotropy. Virulence is traditionally defined as the ability of a pathogen to overcome host defences, especially in the gene-for-gene (GFG) model so that high virulence is associated with the ability to infect more hosts (Laine & Barrès, 2013). This finding is in line with the classic trade-off between virulence and aggressiveness in the GFG model within a given host species (Thrall & Burdon, 2003).

This trade-off between generalism and aggressiveness however does not account for the presence of generalist strains that are equally aggressive as specialist strains, or the co-occurrence of specialists and generalist strains (even where strains are of the same lineage, Dey *et al.*, 2018). The synthesis results showed that a portion of generalists (both potato and tomato isolates) has high aggressiveness equal to the most host specific strains (Fig. 4). The underrepresentation of aggressive generalists is unexpected since populations that cause severe disease (such as 13_A2) often quickly becomes prevalent over a very wide area, so more trials should have been conducted on such populations as those become more common. There are a number of possibilities for this. Migration limitations and stochasticity can limit aggressive generalists, where they are restricted to some continents due to the lack of an introduction event (e.g. quarantine measures). Another possible explanation is the epistatic pleiotropy model where alleles can be conditionally beneficial (Remold, 2012). Under this model, generalists with high relative fitness can arise in a population consisting of normal (less fit) generalists or specialists through sexual recombination or selection on cryptic variation. This would require some level of mutation or sexual reproduction. Subsequent compensatory selection against genetic costs of generalism may also recover the lost aggressiveness, as demonstrated in other systems such as insects and fungi (Pariaud *et al.*, 2009). However, the genetic mechanisms for how highly aggressive generalists arise remain largely unknown.

Even though lesion size is regularly measured to gauge the pathogenicity of isolates and the severity of outbreaks (e.g. Miller *et al.* 1998, Pariaud *et al.*, 2009), results from single controlled laboratory trials may be misleading on the actual fitness of pathogen populations across seasons (Andrivon *et al.* 2013). Fitness is related to a suite of factors other than aggressiveness, including environmental and intrinsic factors such as host availability, population size, infection efficiency, competition, fungicide use and seasonal variation. Long-term studies taking account of the complete life cycle of the pathogen conducted over multiple seasons are likely needed to more reliably assess fitness (Andrivon

et al. 2013). Furthermore, studies on the competitive interactions between co-occurring genotypes (e.g. Lebreton et al. 1999) should also shed more light on this problem.

There are indications that aggressive generalists can co-occur with other strains, including specialists under specific conditions. For instance, 13_A2 and 23_A1 (genetically similar to US-23) co-occur in northern Africa (Beninal *et al.*, 2022) and Pakistan (Belkhiter *et al.*, 2019) where both crops are grown in proximity almost year round. In this case, controlled trials showed that 13_A2 was marginally less aggressive than 23_A1 with a moderate specificity on potato, while the latter was overall more aggressive on both hosts (Belkhiter *et al.*, 2019). Yet, the more aggressive 'generalist' 23_A1 is found predominantly on tomato. In Algeria, 23_A1 is similarly only dominant on tomato during late-season (Belkhiter *et al.*, 2019; Beninal *et al.*, 2021). Pathogenicity trials showed that US-23 in North America has specificity similar to that of 23_A1. Conversely, there US-23 has caused major epidemics on both hosts (Danies *et al.*, 2013). It has also completely displaced the co-occurring generalist US-22 and a very potato-specific US-24 (Saville & Ristaino, 2019). These together indicate that the more restricted 23_A1 could potentially be successful, but undetected factors may be limiting its fitness. It has been suggested that the potato specialist US-24 may persist in more northern locations despite being less aggressive than either generalists (US-22 and US-23) due to its superior performance under cold conditions (Danies *et al.*, 2013). The genetically similar potato-specific US-8 is almost entirely displaced, but isolated populations (mainly in the West Coast of the USA) may have been sustained by inoculum sources at certain localities (Saville & Ristaino, 2019).

Contrary to the second hypothesis that potato isolates will be more host specific, potato and tomato isolates do not differ significantly in specificity, but there were around twice as many highly host-specific populations for potato than for tomato by proportion (12 out of 44 cases or 27% for potato, versus 4 out of 34 cases or 12% with LRR less than -0.5 for tomato; Fig. 5). The reduction in host specificity of tomato strains over the last few decades is interesting, and only partially supports the third hypothesis since the specificity of potato isolates did not change over time. Together, these results generally support the long-held belief that tomato isolates are more generalist than potato isolates. Possible explanations for the reduction in specificity in tomato isolates include a much broader resistance complex for potato than tomato (Lebreton & Andrivon, 1998; Oyarzun *et al.*, 1998; Brouwer *et al.*, 2004) or more successful sexual reproduction in tomato isolates. For tomato isolates, a simpler virulence spectra required to overcome resistance may allow generalism to arise more easily than potato isolates. Rare sexual admixture can produce prospective invasive generalist genotypes such as US-11 (Gavino *et al.*, 2000). Perhaps sexual recombination might help reduce costs of generalism through selective compensatory mechanisms, but sexual populations in *P. infestans* are exceedingly rare and diverse populations are only known to occur in Mexico and northern Europe (Yuen and Andersson 2013). Moreover, in cold regions potato isolates could survive on tubers but

tomato isolates may need other hosts, so specialisation to tomato may not be under selection; although tomato isolates could also be reintroduced from other sources (e.g., seed sources). Differences in regions could not explain the specificity reduction however as tomato isolates from colder regions (i.e., North America and Europe) did not seem different in specificity than warmer regions (i.e., Asia, Africa and South America) (LRR -0.21 and -0.28 respectively). It is clear that the evolutionary forces contributing to host specificity in *P. infestans* are poorly understood, and will require further study before the increasing generalism in tomato isolates (but not potato) could be explained. Nevertheless, this finding provides critical information about how the global pathogen populations are interacting and changing with time. Pathogen transmission from tomato to potato appears likely to increase in the future.

The question of how specialization to hosts impacts on the evolution of pathogens is an important one because of the potential for new and unforeseen outbreaks (Dittmar *et al.*, 2016). For example, the evolution of the sister species *P. mirabilis*, *P. ipomaea*, and *P. phaseoli* closely-related to *P. infestans* is associated with adaptation and specialization on new host species (Raffaele *et al.*, 2010). In *P. infestans*, complete host specialization to potato or tomato is rare, and pertains to only very few isolates (such as certain US-8 populations). Most of the time some level of pathogenicity on the other host is retained (Fig. 3). This may perhaps be due to the relative closeness in physiology among the two hosts and their global metropolitan nature, ensuring genetic exchange among populations.

In the current study, lower aggressiveness or higher specificity in US-1 strains was not found. Past studies often observed the displacement of US-1 by more aggressive strains (Legard *et al.*, 1995; Reis *et al.*, 2003; Suassuna *et al.*, 2004; Chen *et al.*, 2009). The non-significance difference in aggressiveness could be due to the low number of data points available in this case. Alternatively there is a possibility that these studies were testing extant US-1 populations that represent a subset of survivors, which may be equally aggressive as the newer invading populations. This survivorship effect may have also been observed in *P. cinnamomi*, where the A2 mating type is the predominant strain that displaced the less virulent endemic A1 throughout Asia (Arentz, 2017). The remaining extant *P. cinnamomi* A1 has similar aggressiveness (i.e. lesion size) to the A2 strains (Dudzinski *et al.*, 1993; Robin & Desprez-Loustau, 1998). Comparing tomato and potato isolates of US-1 and those of other lineages under controlled conditions (e.g., competitiveness or pathogenicity under different environmental conditions; Young *et al.*, 2009) should shed some light on the selection pressures within each host species.

An overview of the historical literature on host specificity following the early 1900s (when tomato blight was spreading worldwide) clearly shows that transitions from generalism to specialism can occur over time. Specialists are favoured in various models, including selection for their higher

aggressiveness, mutation accumulation, and genetic fixation (i.e., under low gene flow and strong selection, Yeaman & Whitlock, 2011). As these displacements continue and populations shift in properties, it is important to track these changes. Controlled pathogenicity trials (such as detached leaf assays) can be employed to monitor populations. Lesion growth data from pathogenicity trials (i.e. inoculations on potato and tomato) could be plotted onto a Cartesian plane to compare pathogen populations (as in Fig. 4). This approach has limitations; however, as trade-offs involving other life-history traits that are more seldom measured (such as infection efficiency, inter-seasonal transmission and ploidy) could also be important (Andrivon et al. 2013). Identifying common principles among different invasions should aid in disentangling the complexity of the problem. While a trade-off between generalism and aggressiveness may help explain the variation among pathogen populations, host specificity likely interplays with a suite of other dependencies such as competitiveness, infection efficiency, relative host abundance, and other environmental factors.

Conclusions

The current synthesis revealed a slight trade-off between generalism and aggressiveness across *Phytophthora infestans* populations on potato and tomato. In general, host specificity was associated with greater aggressiveness in isolates from both hosts, but a proportion of cases had high aggressiveness on both hosts which this trade-off could not explain. Although the level of specificity among potato and tomato isolates were not significantly different, tomato isolates tended to become more generalist over the last few decades. Due to the variability in how pathogenicity traits relate to fitness, long-term studies (e.g. over multiple seasons) are likely needed to better understand the trade-offs involving pathogenicity. The mechanisms driving the evolution of host specificity are complex, and are likely shaped by demography and other evolutionary factors.

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Table 1

Aggressiveness as defined by lesion growth rate (LGR in millimetres per day) on host of origin versus difference in aggressiveness on the other host (defined as LGR on host of origin minus LGR on other host). The data was collated from cross-inoculation trials conducted on potato and tomato isolates of *Phytophthora infestans* (k = 24 and k = 19 respectively). Bold values indicate statistically significant effects (at $P < 0.05$).

Source	**Estimate**	**SE**	**d.f.**	**t**	***P***
Potato isolates					
Intercept	-0.72	0.55	22	-1.32	0.20
Aggressiveness host of origin	0.55	0.14		3.80	**<0.001**
Tomato isolates					
Intercept	-0.33	0.68	17	-0.49	0.63
Aggressiveness host of origin	0.36	0.18		1.94	0.07

Table 2

Aggressiveness as defined by lesion growth rate (LGR in millimetres per day) on host of origin versus effect size (LRR) for all *Phytophthora infestans* isolates (k = 68). Bold values indicate statistically significant effects (P < 0.05).

Source	**Estimate**	**Median absolute deviation**	**V**	***P***
Intercept	-0.00	0.21	470	0.77
LRR	-0.03	0.10	316	**0.04**

Table 3

Year of isolation (Calendar year) versus effect size (LRR) for *Phytophthora infestans* potato (k = 40) and tomato (k = 32) isolates. Bold values indicate statistically significant effects (at $P < 0.05$).

Source	**Estimate**	**Median absolute deviation**	**V**	***P***
Potato isolates				
Intercept	-0.41	10.29	448	0.62
Year	0.00	0.01	328	0.54
Tomato isolates				
Intercept	-17.00	26.98	126	**0.009**
Year	0.01	0.01	402	**0.009**

Figure Captions

Fig. 1

Flow diagram of the literature search and the screening process, detailing the number of studies excluded during screening up to the final number of studies included.

Fig. 2

World map detailing the locations of *Phytophthora infestans* potato and tomato populations tested in cross-inoculation trials. Symbols with lighter shades represent imprecise locations specified to the general region only. Geographically distant populations tested within the same study are joined by dotted lines.

Fig. 3

Summary median and quantiles for host specificity (effect size LRR) among potato and tomato isolates of *Phytophthora infestans* across continental regions (where *k* is the sample size). More negative LRR values indicate higher specificity. Very negative values (such as < -1) are associated with specialisation to the original host. Populations with very positive values are specialised on the other host and are considered outliers (indicated by the grey arrow). A value of zero indicates equal aggressiveness on both hosts where there is no specificity.

Fig. 4

Relationship between aggressiveness versus the difference in aggressiveness among hosts in *Phytophthora infestans* potato and tomato isolates. Aggressiveness is defined as lesion growth rate on host tissue (in millimetres per day). A greater difference in aggressiveness among hosts indicates greater specificity. Potato isolates are indicated in brown (solid line and circles) and tomato isolates are indicated in red (dashed line and triangles).

Fig. 5

Relationship between host specificity (LRR) and aggressiveness on the original host (LGR) for potato and tomato isolates.

Fig. 6

Relationship between host preference (LRR) versus year of isolation in *Phytophthora infestans* a) potato and b) tomato isolates. More negative LRR values are associated with greater specificity for the host of origin than the other host.

Supplementary material captions

Figure S1

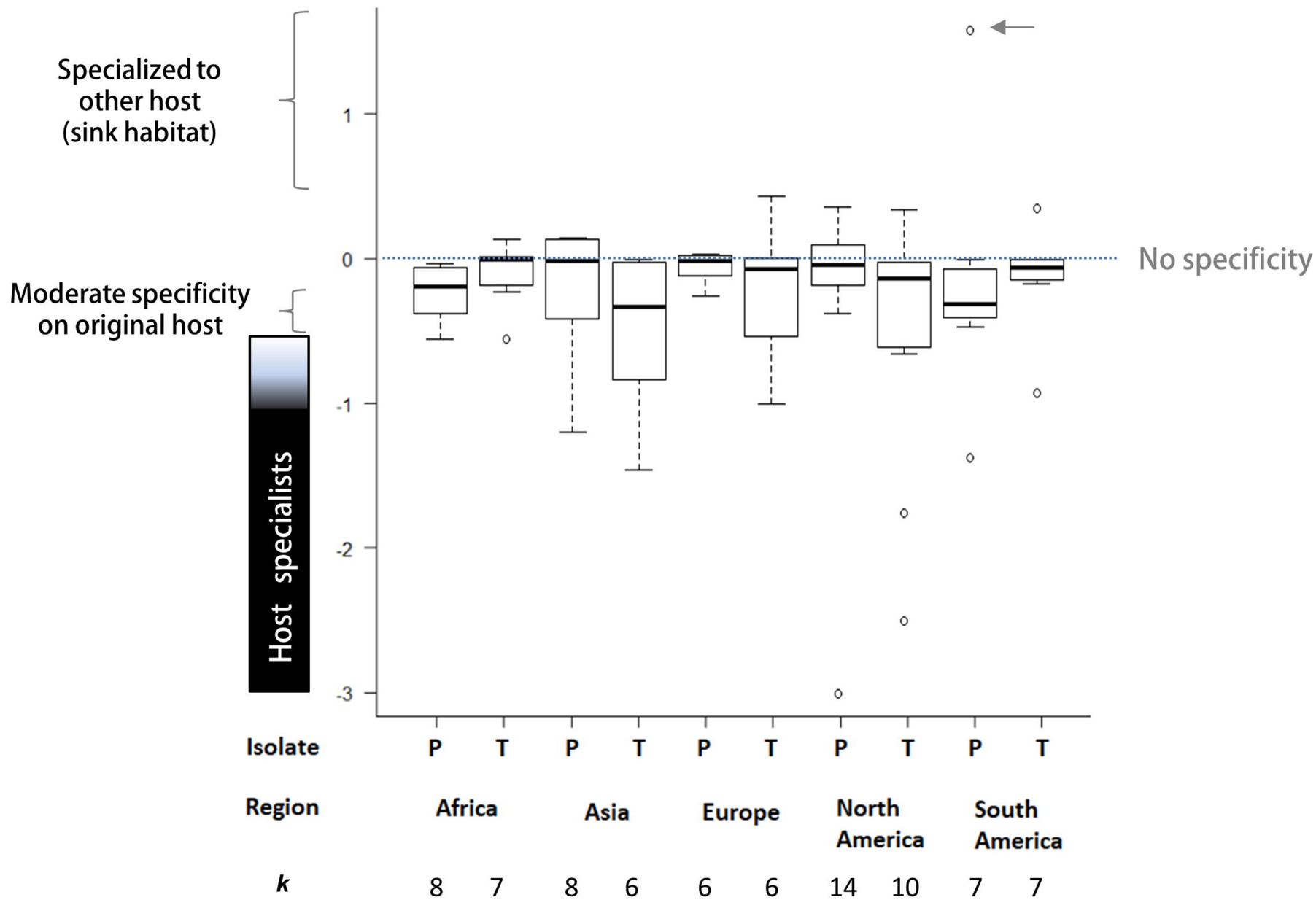
Quantile-quantile plots for the lesion growth rate data of *Phytophthora infestans* from a) potato and b) tomato (LGR on the original host).

Table S1

List of studies and data used in the synthesis of *Phytophthora infestans* host specificity and aggressiveness of potato and tomato isolates.

Fig. 2

Fig. 3


Specialized to other host (sink habitat)
Moderate specificity on original host
Host specialists
No specificity
1
0
-1
-2
-3
Isolate
P T P T P T P T P T
Region
Africa
Asia
Europe
North America
South America
k
8 7 8 6 6 6 14 10 7 7

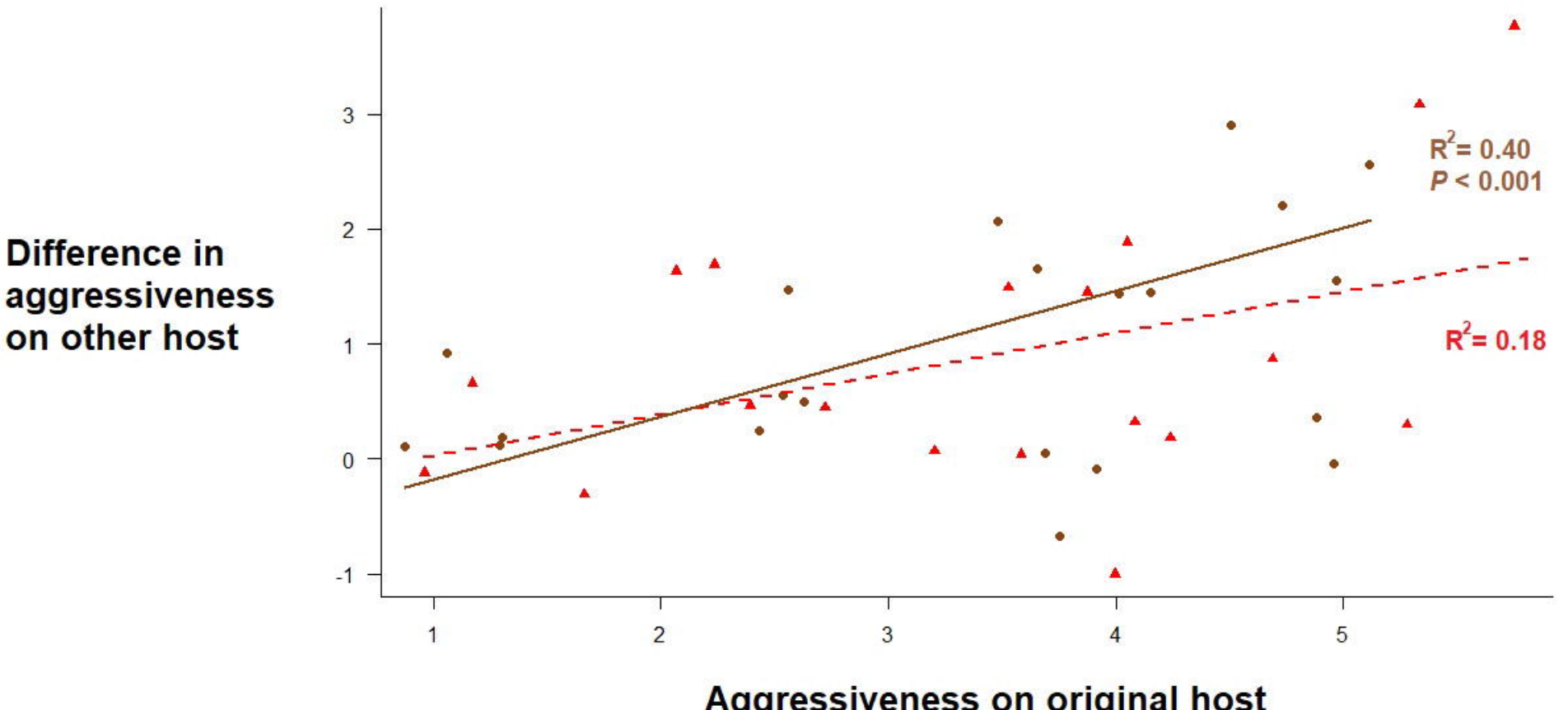

Difference in aggressiveness on other host
3
2
1
0
-1
1
2
3
4
5
Aggressiveness on original host
R2= 0.40
P < 0.001
R2= 0.18

Fig. 5

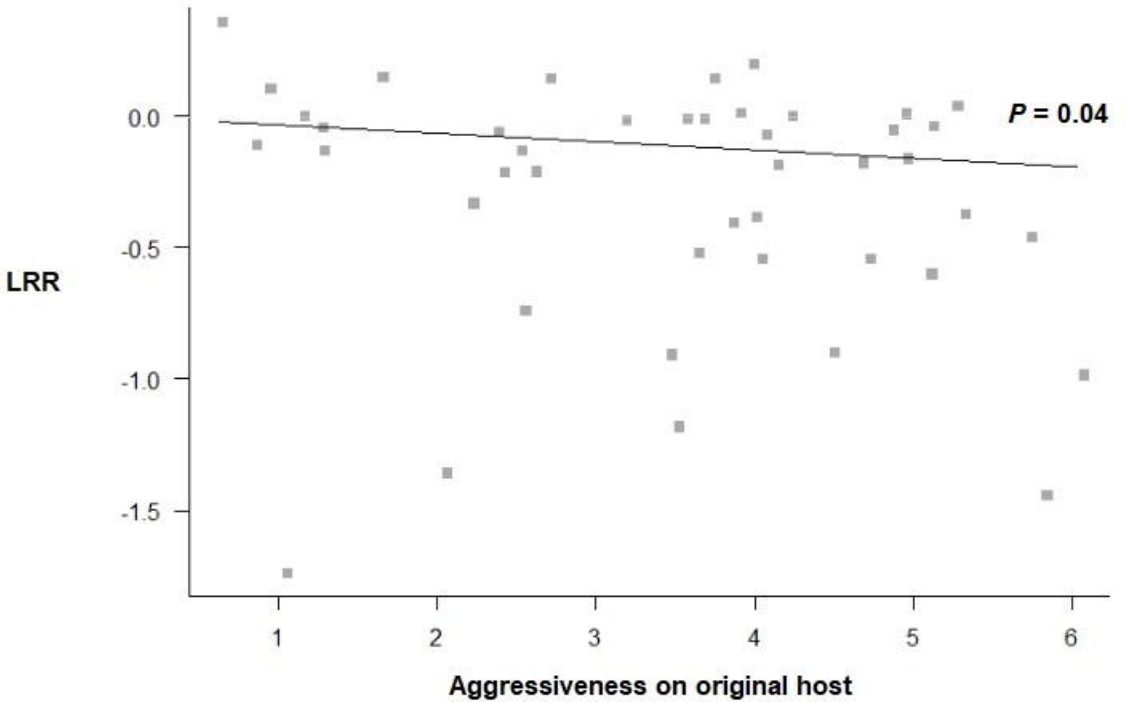


Fig. 5

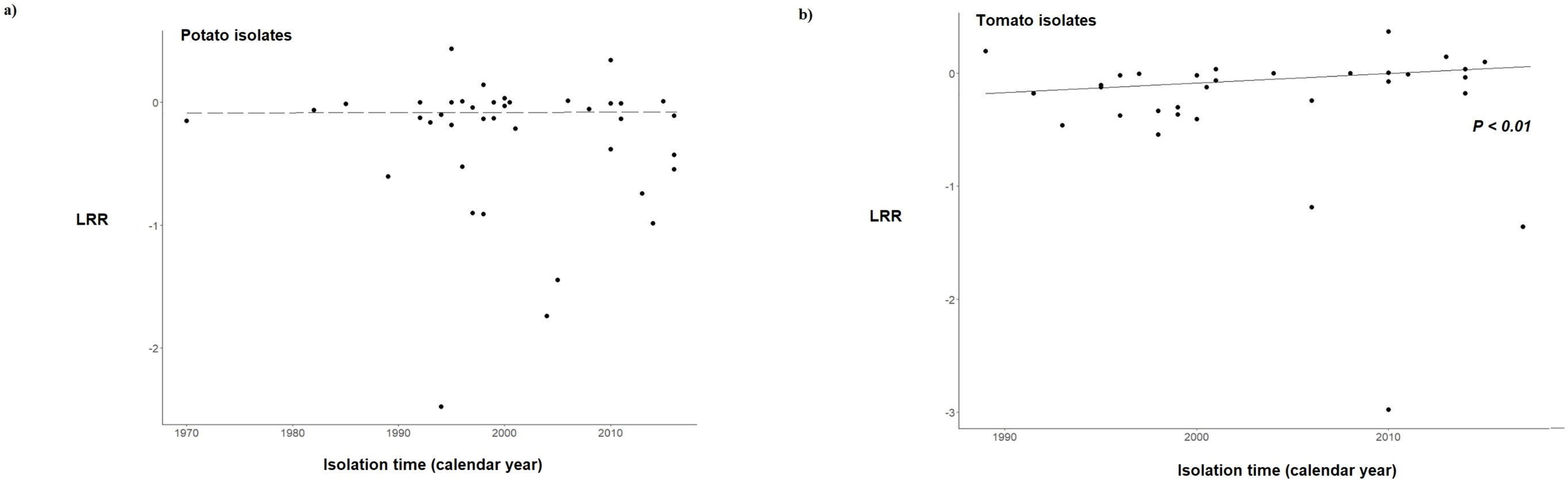
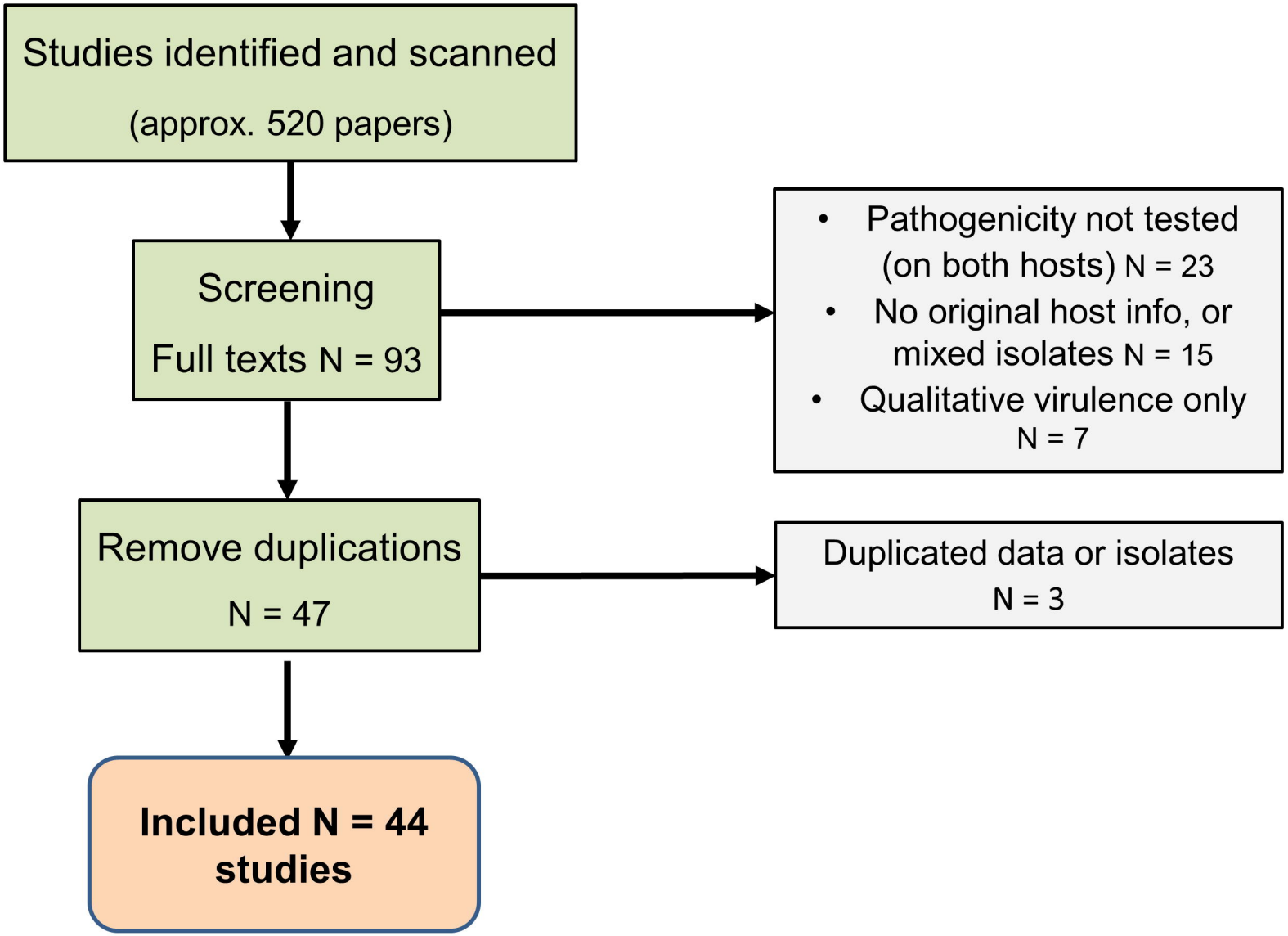

a)
Potato isolates
LRR
0
-1
-2
1970
1980
1990
2000
2010
Isolation time (calendar year)
b)
Tomato isolates
LRR
0
-1
-2
-3
P < 0.01
1990
2000
2010
Isolation time (calendar year)

Fig. 1


Studies identified and scanned
(approx. 520 papers)
Screening
Full texts N = 93
• Pathogenicity not tested (on both hosts) N = 23
• No original host info, or mixed isolates N = 15
• Qualitative virulence only N = 7
Remove duplications
N = 47
Duplicated data or isolates
N = 3
Included N = 44 studies