Aggressiveness trades-off with host generalism in strains of *Phytophthora infestans* (potato and tomato blight): A synthesis of global cross-inoculation data

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Abstract

Phytophthora infestans is among the most destructive of plant diseases. Pathogen populations have varying degrees of host preference among potato and tomato, from no specificity to extreme specialisation. How host specificity impacts fitness among populations is unclear. Comparing the aggressiveness 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 strains. I assessed the existence of such a trade-off by analysing the global cross-inoculation data on potato and tomato isolates, and tested whether specificity has changed 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. However, tomato isolates became more generalist over the last few decades. High specificity was associated with significantly greater aggressiveness, and strains from potato can generally infect tomato with similar aggressiveness and specificity as in reverse. This synthesis reveals several novel insights on the evolutionary ecology of the blight pathogen, and provides a new way to map and track *P. infestans* populations. Many unresolved questions on host specificity and aggressiveness remain which are discussed.

Keywords: late blight, 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 disease 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 aggressiveness) to obligate specialists (i.e., only infects on one host and zero or near-zero aggressiveness 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 pathogen aggressiveness on hosts (Pariaud *et al.*, 2009). Therefore, a better understanding of how *P. infestans* adapt and specialise to potato and tomato, and the determinants of aggressiveness 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). For practicality, 'aggressiveness' is considered here to be a quantitative measure of pathogenicity that is non-specific to host genotypes, as defined in Pariaud *et al.* (2009).

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). Aggressiveness 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. The earliest cross-inoculation trials done prior to 1910 (from 1847 to 1906) reported no host specificity in isolates from both hosts (Berg, 1926; Oyarzun *et al.*, 1998). However, this changed in the following period from 1915 when remarkable specificity was frequently noted either from 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. For instance, not all US-8 isolates are potato-specific; a relatively generalist US-8 was found to be dominant in British Columbia (Canada) on potato and tomato in the late 1990s (Daayf & Platt, 2003). 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 on their host should become dominant (Gisi *et al.*, 2011). For instance, the greater aggressiveness of US-8 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 partially displaced US-1 from potato, but not tomato (Suassuna *et al.*, 2004). The dominant 13_A2 lineage was more aggressive than other lineages, and was demonstrated to competitively exclude those other strains 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 aggressiveness and fitness is complex. Although the evidence that strains with greater aggressiveness are advantaged is abundant, conversely there have been suggestions that the most aggressive 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 aggressiveness on the host) (Acevedo *et al.*, 2019). However, in the case of *P. infestans* this hypothesis 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 aggressive on potato can have lower aggressiveness on tomato to compensate the lowered transmission rate on potato. Even if high aggressiveness 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) host specific isolates will be more aggressive than generalist isolates, (2) potato isolates will be more host specific than tomato isolates, (3) host specificity will reduce over time, and (4) isolates of the older US-1 lineage will have lower aggressiveness but more specialised to their host of origin 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 aggressiveness 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 aggressiveness (e.g., lesion size, AUDPC, degree of infection). Thus, those reporting only qualitative pathogenicity are excluded. Studies that tested the aggressiveness of 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 aggressiveness 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 because lesion size is a key predictor in epidemic severity in *P. infestans* that was commonly reported (Birhman & Singh, 1995; Pariaud *et al.*, 2009); (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 aggressiveness of isolates on the two hosts were standardised 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 in length per day) was estimated from the lesion size data in the time following inoculation. The standardised 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 test whether specialized isolates are more aggressive on their host, 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). Of these, 31 studies conducted cross-inoculation trials on both potato and tomato isolates of *P. infestans*. 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). Of the 78 data points, 43 reported lesion size. 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 aggressive on the other host) was removed from all analyses (shown in Fig. 3). The trials mainly consisted of leaf inoculations or detached leaf assays. 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.

Isolates generally had a modest specificity 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), where the aggressiveness on the other host is 55% and 66% of that on the original host for potato and tomato isolates, respectively. This difference between host specificity between potato and tomato isolates is 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 non-aggressive 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 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). Avirulence genes for infecting potato and tomato are likely independent of each other, so a generalist would need to counter the resistance factors of both potato and tomato (Oyarzun *et al.*, 1998; Michalska *et al.*, 2016). Third, specialists should have a lower accumulation of deleterious alleles (Whitlock, 1996). Mutation accumulation can hinder the competitiveness of a generalist in the presence of specialists (Kawecki, 1994). 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). In the context of this study, a strain that is generalist on both tomato and potato can also be considered more 'virulent' than one that is specialised to one of the hosts.

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). An explanation for the relatively low prevalence of these populations is migration and stochasticity where they have not spread simply because they have not been introduced to those areas (e.g. due to quarantine measures). Another possible explanation is the epistatic pleiotropy model where alleles can be conditionally beneficial (Remold, 2012). Under this model, highly aggressive generalists can arise in a population consisting of normal (less

aggressive) generalists or specialists through sexual recombination or selection on cryptic variation. This would require some level of mutation or sexual reproduction. Alternatively, compensatory selection against genetic costs of generalism may recover the lost aggressiveness (Pariaud *et al.*, 2009).

Interestingly, aggressive generalists often co-occur with other strains, including specialists. The specific genetic mechanisms for the arising of highly aggressive generalists remain largely unknown. Nonetheless, fitness is related to a suite of environmental and intrinsic factors including host availability, competitor strains, fungicide use and intra-seasonal environmental (temporal) variation. Results from laboratory trials may not always reflect specificity in the field. Finding that highly aggressive generalist populations are not invariably the most dominant is therefore not unsurprising. For instance, 13_A2 and 23_A1 (genetically identical 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 pathogenicity trials, 13_A2 is marginally less aggressive than 23_A1 with a moderate specificity on potato, while the latter is 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 only dominant during late-season on tomato (Belkhiter *et al.*, 2019; Beninal *et al.*, 2021). Pathogenicity trials showed that the genetically identical US-23 has specificity similar to that of 23_A1. Conversely, US-23 has caused major epidemics on both hosts in North America (Danies *et al.*, 2013). There, US-23 has completely displaced the co-occurring generalist US-22 and a very potato-specific US-24 (Saville & Ristaino, 2019). While less aggressive than either generalists (US-22 and US-23), it has been suggested that the specialist US-24 may persist in some areas (i.e. more northern locations) due to its superior performance under cold conditions (Danies *et al.*, 2013). The genetically similar potato-specific US-8 was able to persist mainly at the West Coast of the USA. It has been suggested that different inoculum (potato seed) sources accounts for its persistence (Saville & Ristaino, 2019). It is hence evident that specialists may persist in the presence of more aggressive generalists, and apparently highly aggressive generalists may not always be most successful.

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. A scarce but greater output of successful sexual progeny from viable oospores in tomato plants than potato (e.g., Cohen *et al.*, 1997; Mayton *et al.*, 2000; Yuen & Anderson, 2013) can produce prospective aggressive generalist genotypes such as US-11 (Gavino *et al.*, 2000), and reduce costs of generalism through compensatory mechanisms. It is still much a matter of debate about whether sexual recombination is less successful in potato than tomato. 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 aggressiveness 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 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 clearly shows transitions from generalism to specialism 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). The modern successive populations are much more diverse and relatively less clonal than in the past (Drenth *et al.*, 1995), and this is reflected in the diversity of strains. As these displacements continue and populations shift in properties, it is important to track these changes. The aggressiveness data on both hosts can be used to plot the strategy space occupied by isolates relative to others (across the aggressiveness and specificity continuums), and to track the shifts of lineages within geographical regions (Fig. S2). Aggressiveness can be readily gauged from cross-inoculation trials, so this method is relatively accessible for managers and other experts to characterise disease outbreaks and populations. Identifying common principles among invasions, through the assessment of ecological strategies used by pathogens, would be a key to help disentangle the complexity of the problem.

Conclusions

The current synthesis revealed a clear trade-off between generalism and aggressiveness across *Phytophthora infestans* populations on potato and tomato. Host specificity was associated with greater aggressiveness in isolates from both hosts. 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. A new and accessible method to map the strategy space occupied by pathogen populations is presented, that can be used to track how populations are evolving over time and can be used to characterise new invasions. Future studies on ecological strategies employed by pathogen populations and the dependencies of host specificity should prove fruitful in predicting the impacts of epidemics.

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622 **Table 1**

623 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
624 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
625 *Phytophthora infestans* (k = 24 and k = 19 respectively). Bold values indicate statistically significant effects (at $P < 0.05$).
626

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

627 **Table 2**

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

630

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

631 **Table 3**

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

634

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**

635 **Figure Captions**

636 **Fig. 1**

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

639

640 **Fig. 2**

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

644

645 **Fig. 3**

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

652

653 **Fig. 4**

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

659

660 **Fig. 5**

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

663

664

665 **Fig. 6**

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

669 **Supplementary material captions**

670 **Figure S1**

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

673

674 **Figure S2**

675 Relative strategy space occupied by some populations of *Phytophthora infestans* lineages plotted onto Figure
676 4. Aggressiveness is defined as lesion growth rate on hosts (mm per day) plotted against the difference in
677 aggressiveness on the other host. A greater difference in aggressiveness among hosts indicates greater
678 specificity. Potato isolates are in black (circles) and tomato isolates are in grey (triangles). References to
679 isolates are shown below the figure.

680

681 **Table S1**

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

684

Fig. 1

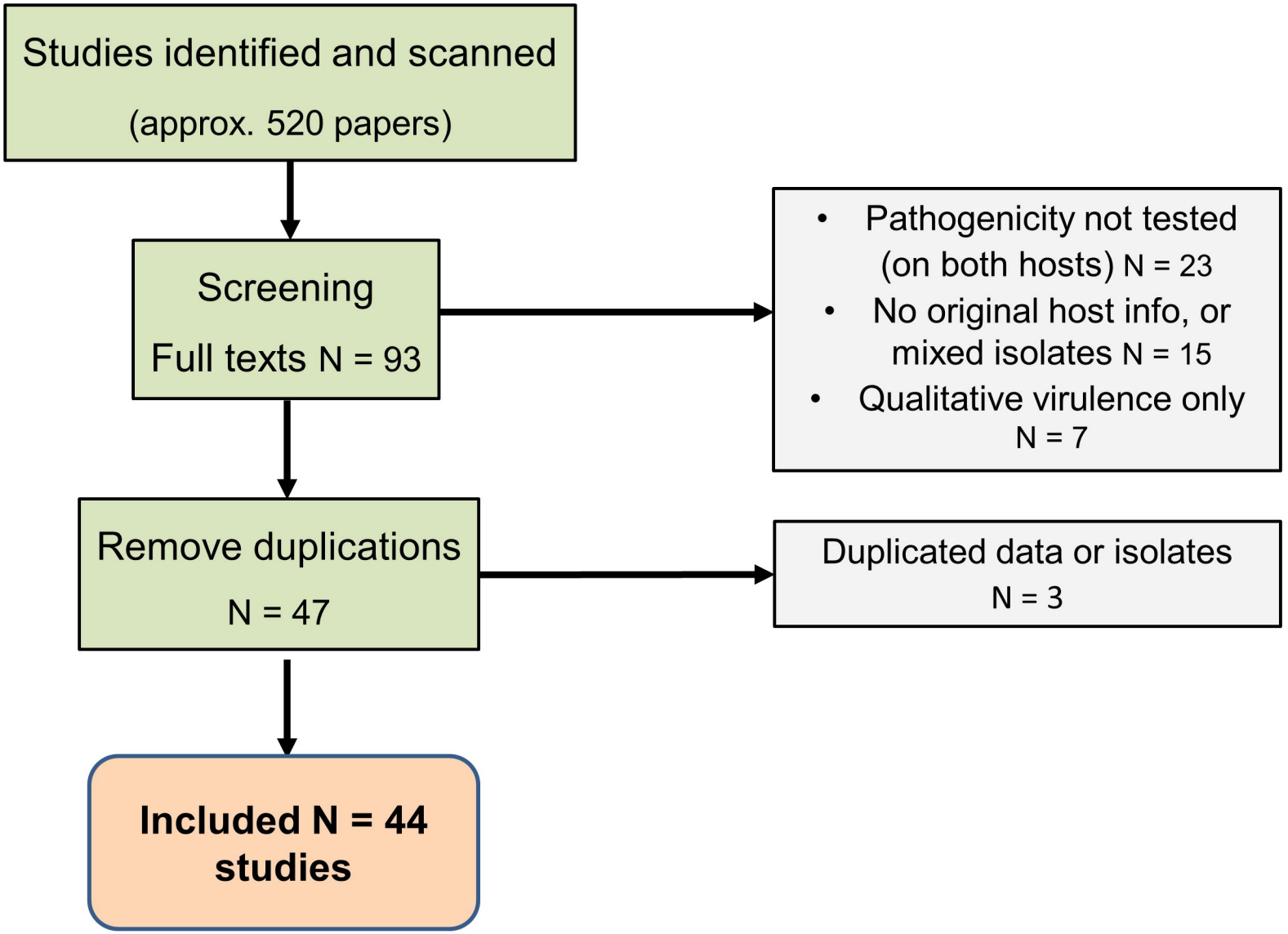

Studies identified and scanned
(approx. 520 papers)
Screening
Full texts N = 93
• Pathogenicity not tested (on both hosts) N = 23
• No original host info, or mixed isolates N = 15
• Qualitative virulence only N = 7
Remove duplications
N = 47
Duplicated data or isolates
N = 3
Included N = 44 studies

Fig. 2

Fig. 3

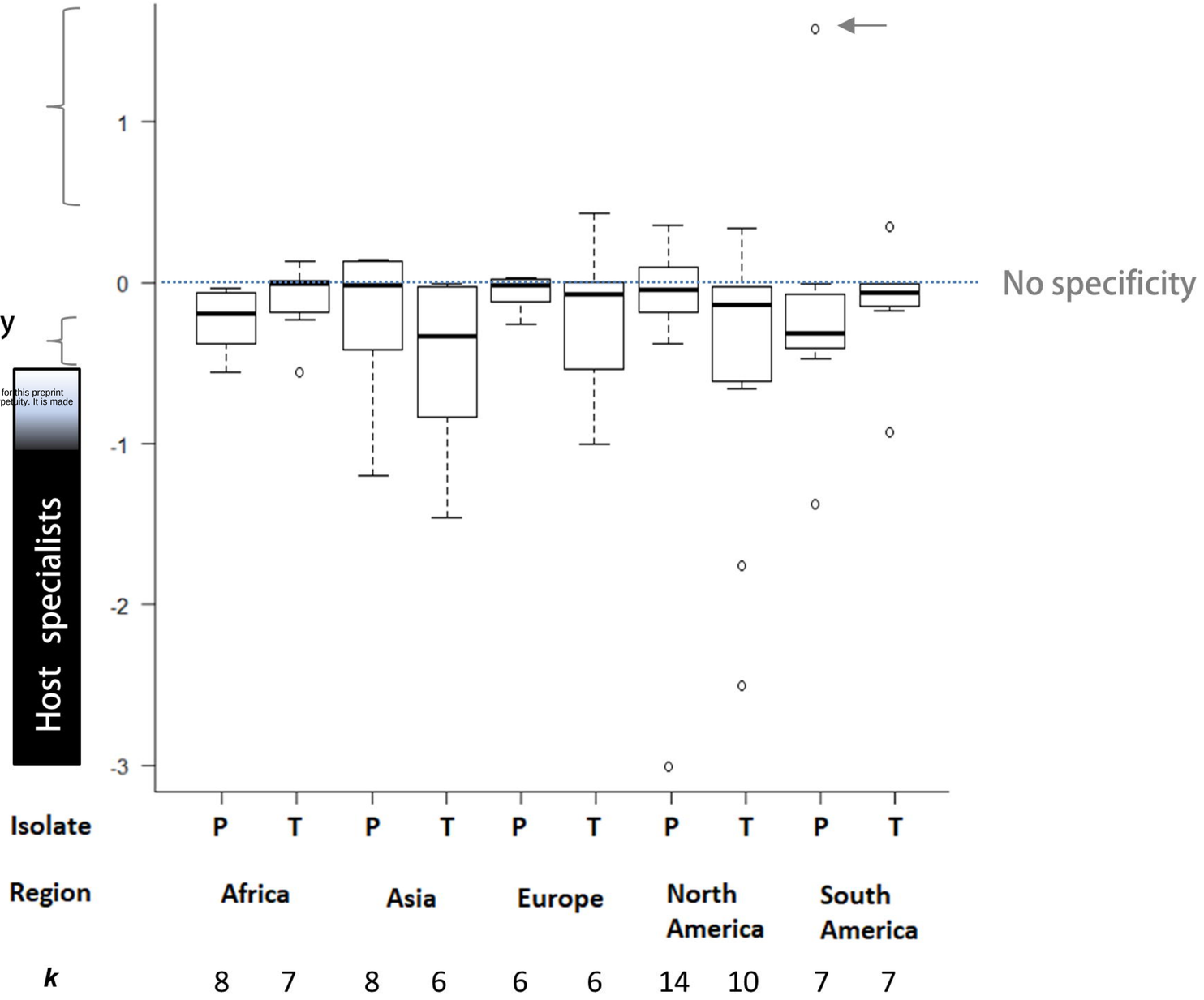


Fig. 4

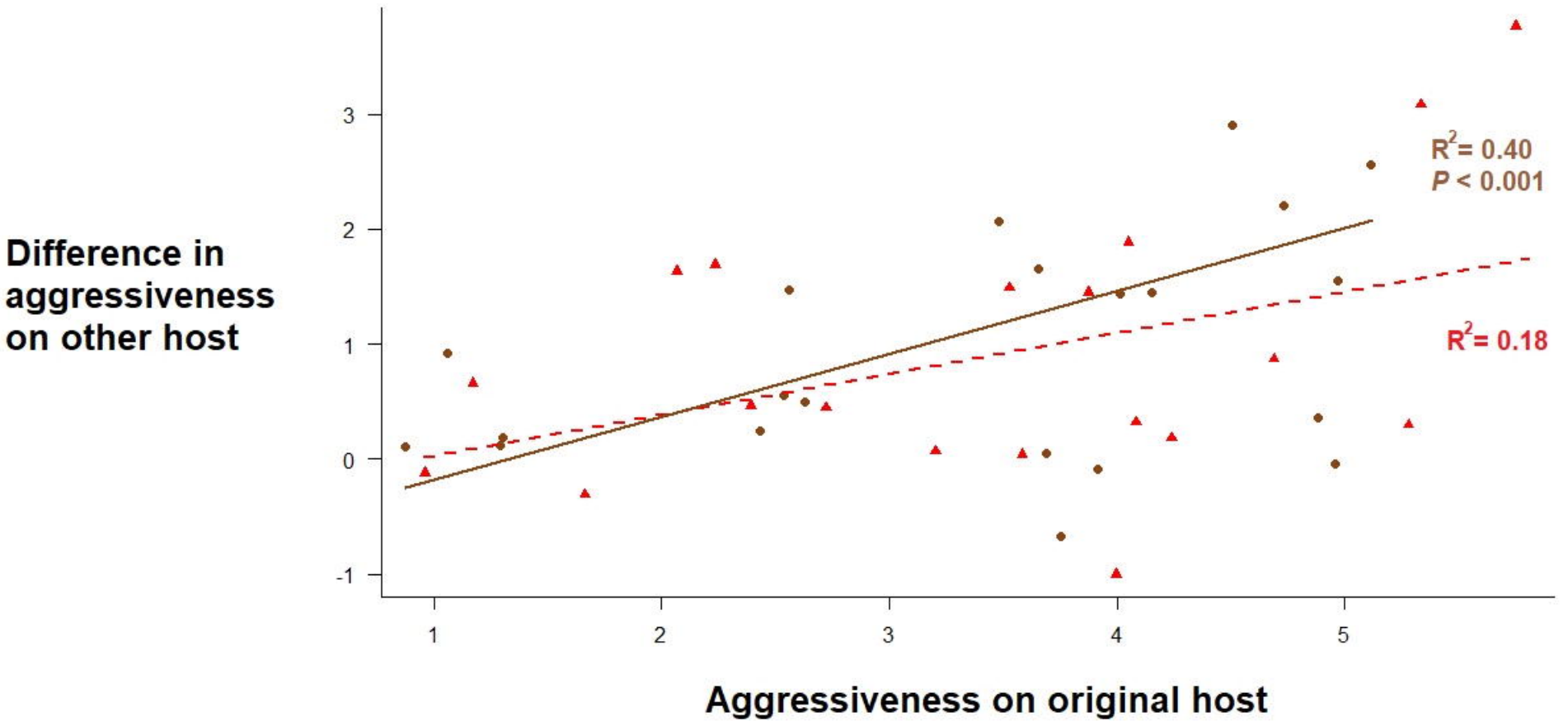

Difference in aggressiveness on other host
3
2
1
0
-1
1
2
3
4
5
R^2= 0.40
P < 0.001
R^2= 0.18
Aggressiveness on original host

Fig. 5

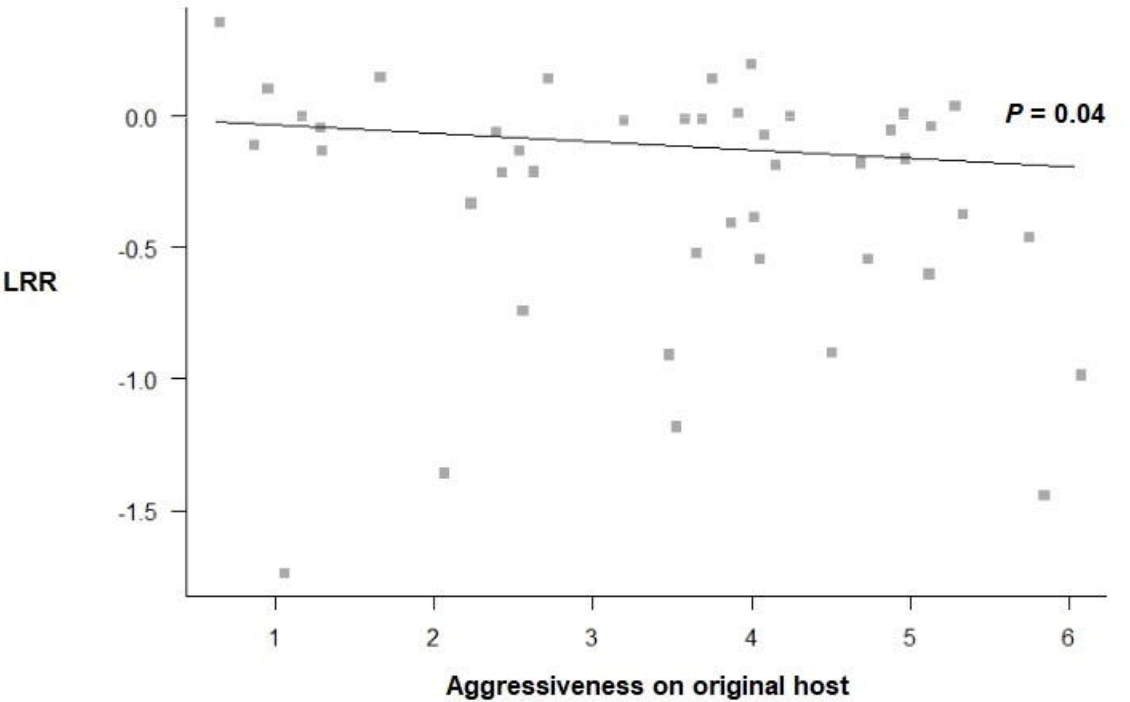
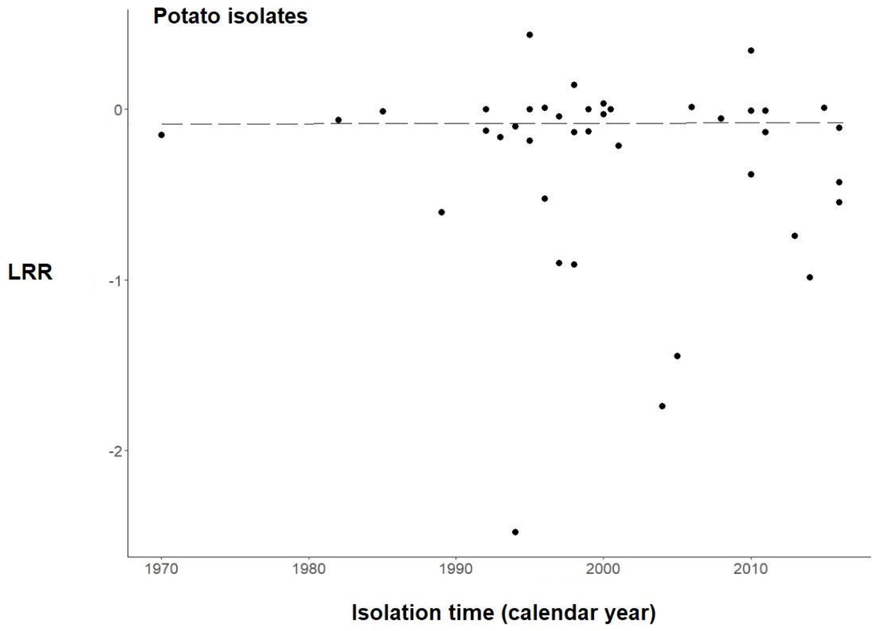
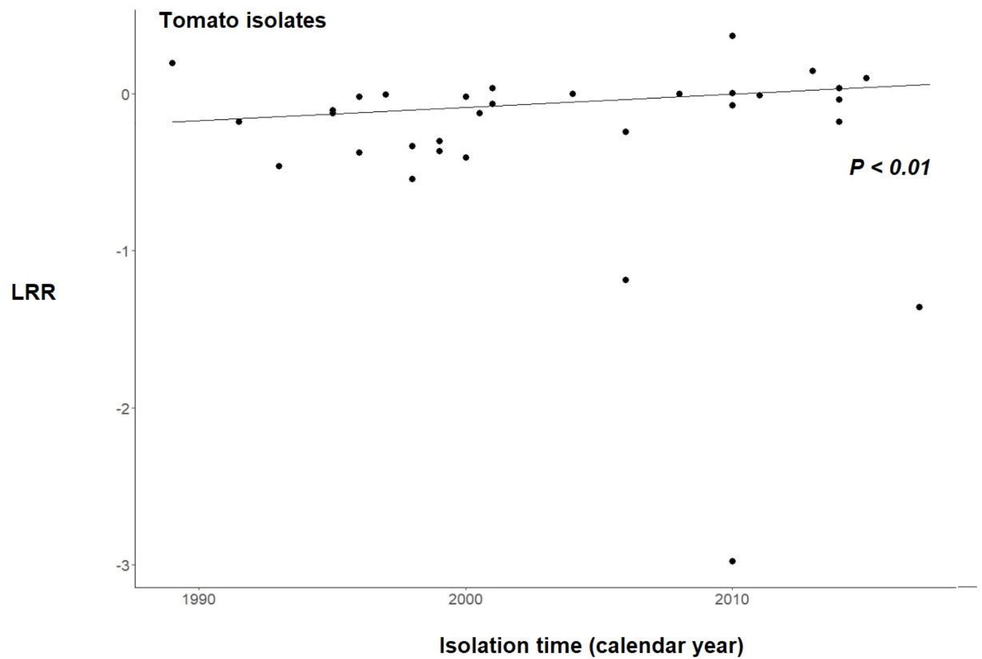


Fig. 5

a)


Potato isolates
LRR
0
-1
-2
1970
1980
1990
2000
2010
Isolation time (calendar year)

b)


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