


# Rapid adaptation of the Irish potato famine pathogen *Phytophthora infestans* to changing temperature

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## Abstract

Temperature plays a multidimensional role in host–pathogen interactions. As an important element of climate change, elevated world temperature resulting from global warming presents new challenges to sustainable disease management. Knowledge of pathogen adaptation to global warming is needed to predict future disease epidemiology and formulate mitigating strategies. In this study, 21 *Phytophthora infestans* isolates originating from seven thermal environments were acclimated for 200 days under stepwise increase or decrease of experimental temperatures and evolutionary responses of the isolates to the thermal changes were evaluated. We found temperature acclimation significantly increased the fitness and genetic adaptation of *P. infestans* isolates at both low and high temperatures. Low-temperature acclimation enforced the countergradient adaptation of the pathogen to its past selection and enhanced the positive association between the pathogen's intrinsic growth rate and aggressiveness. At high temperatures, we found that pathogen growth collapsed near the maximum temperature for growth, suggesting a thermal niche boundary may exist in the evolutionary adaptation of *P. infestans*. These results indicate that pathogens can quickly adapt to temperature shifts in global warming. If this is associated with environmental conditions favoring pathogen spread, it will threaten future food security and human health and require the establishment of mitigating actions.

## KEYWORDS

acclimation, aggressiveness, fitness, *Phytophthora infestans*, thermal adaptation

## 1 | INTRODUCTION

The epidemiological development of infectious diseases results from the interaction of three main factors: a conducive environment, a susceptible host, and a virulent pathogen (Madden, Hughes, & Bosch, 2007). Climate change is expected to exert a strong impact on the epidemics and ecology of plant and animal diseases (Garrett, Dendy, Frank, Rouse, & Travers, 2006; Sparks, Forbes, Hijmans, & Garrett, 2014; Zhan, Ericson, & Burdon, 2018), greatly threatening food security, natural landscapes, and human health (Boland, Melzer, Hopkin, Higgins, & Nassuth, 2004; Kalinda, Chimbari, & Mukaratirwa, 2017; Myers et al., 2017). These epidemic effects on infectious diseases can result from the influences of climate changes on the survival, reproduction, and transmission of both pathogens and hosts and can occur at various spatial and temporal scales (Koelle, Pascual, & Yunus, 2005).

Global warming is an important element of climate change of great concern to society. In recent times, the Earth's climate has warmed at an unprecedented rate—a phenomenon set to continue, rising by 4°C above the pre-industrial era by the end of the 21st century (IPCC, 2014). As a major abiotic environmental factor, temperature can impact on a very broad range of biological and biochemical activities of living organisms from the subcellular molecular level to community-wide interactions (Huey & Kingsolver, ; Padfield, Buckling, Warfield, Lowe, & Yvon-Durocher, 2018; Schaum et al., 2017; Yvon-Durocher et al., 2015). In host-pathogen interactions, temperature can exert a strong influence on the ecology and severity of diseases (Elad & Pertot, 2014), and the evolutionary dynamics of hosts and pathogens (Gillman, Keeling, Gardner, & Wright, 2010). It does this by affecting key stages of the life cycles of either or both hosts and pathogens (Addison, Powell, Six, Moore, & Bentz, 2013; Dysthe, Bracewell, & Six, 2015; Zhan & McDonald, 2011) including survivals (Mariette et al., 2016), reproductive modes (Angilletta, Oufiero, & Leaché, 2006), dispersal abilities (Urban Mark, Tewksbury Josh, & Sheldon Kimberly, 2012), and geographic distribution (Rutherford, D'Hondt, & Prell, 1999) as well as their interactions with other biotic and abiotic elements in the environment (Eastburn, McElrone, & Bilgin, 2011). Given the multidimensional effects of temperature on host-pathogen interactions, a warmer world resulting from climate change may well present new disease management challenges. In this context, it is crucial to analyze the patterns of thermal adaptation of pathogens to future temperature change in order to develop better predictions of future disease epidemiology and formulate relevant management strategies (Mboup et al., 2012).

It is well known that living organisms can adapt to environmental changes by acclimation (Ghalambor, McKay, Carroll, & Reznick, 2007). Through prior exposure to a stressing environment for a short period of time, living organisms often show a marked increase in their fitness compared to individuals naive to the stress (Hoffmann, 1995). This adaptation can be driven genetically or physiologically and has been documented widely in pathogens, plants, and animals in the context of temperature stresses (Hoffmann & Willi, 2008).

Physiological adaptation to thermal changes caused by phenotypic plasticity does not modify the genetic architecture of living organisms. Rather, it is the near-instant response of living organisms to environmental stresses achieved through temporal adjustment of gene expression and/or enzymatic activity (de Nadal, Ammerer, & Posas, 2011; Wu, Wang, Shen, et al., 2019). On the other hand, genetic adaptation involves permanent or quasi-permanent (epigenetic control) changes in the genomic composition of living organisms followed by directional selection favoring beneficial change and usually occurs in addition to physiological adaptation (Hoekstra & Coyne, 2007). The two adaptation processes are interconnected to provide an opportunity for the organism in question to survive, reproduce, and compete in new environments (Willmott et al., 2018). Many empirical studies of thermal adaptation have focused on the scales and patterns of physiological adaptation, that is, phenotypic plasticity (Bacigalupe et al., 2018; Chown, Addo-Bediako, & Gaston, 2002; Garrett et al., 2006; Willmott et al., 2018). In contrast, evolutionary inferences on how fast genetic adaptation can occur and how such adaptations may affect biological interactions among traits are relatively limited, particularly in plant pathogens. For example, rapidly physiological adaptation to temperature stresses during thermal acclimation has been documented in some animals, plants, and microbes (Colinet, Overgaard, Com, & Sorensen, 2013; Crowther & Bradford, 2013; Donelson, Munday, McCormick, & Pitcher, 2012; Fahey, Winter, Slot, & Kitajima, 2016), but no such phenomenon has been reported in a pathogen (Paull, Raffel, LaFonte, & Johnson, 2015). All of these reports on thermal adaptation during acclimation were conducted by continuing passage of the concerned species in a fixed temperature regime.

The potato *Phytophthora infestans* association is a particularly relevant one in which to assess the potential for thermal adaptation in pathogens. Potato has been and is playing an ever-increasing role, as a major food crop (currently ranked as the third largest food crop globally) feeding the world's growing population (Birch et al., 2012). *Phytophthora infestans* is the world's most devastating potato pathogen (Fry, 2008), causing annual economic losses of approximately 8 billion US dollars (Birch et al., 2012; Haverkort et al., 2008; Runno-Paurson et al., 2013). It infects all parts of the potato plant including leaves, stems, and tubers, and under moderate temperature (16–22°C) and high humidity (over 97%), can destroy entire crops within a few days of infection (Harrison, 1992; Latijnhouwers, Ligterink, Vleeshouwers, Van West, & Govers, 2004; Sujkowski, 1987). While resistant genes are available in cultivated and wild potatoes, most of the resistance is difficult to integrate into modern cultivars and can be quickly rendered ineffective due to evolution in the pathogen (Forbes, 2012; Yang et al., 2019). Consequently, the disease is mainly managed by fungicide applications, causing big challenges to environmental sustainability and biodiversity conservation.

*Phytophthora infestans* is considered to be a heterothallic oomycete with two mating types designated A1 and A2, despite the existence of self-fertile isolates (Judelson, 1997). It can reproduce sexually, asexually, and/or parasexually (Billiard et al., 2011; Zhu et al., 2016) and has a potential for long distance wind

dispersal (Granke, Windstam, Hoch, Smart, & Hausbeck, 2009). The pathogen has a large genome (~240 Mbp) rich in transposable elements (>75% of total genome) (Haas et al., 2009; Vetukuri et al., 2012). Many genes critical to the ecological functioning of the pathogen are located around these highly mutable regions, providing a unique opportunity to generate large amount of variation available for quick evolutionary adaptation to changing biotic environments such as host resistance (Lozoya-Saldaña, 2011; Rietman et al., 2012) or abiotic environments such as temperature changes (Mariette et al., 2016). Previous analysis of the association between historical thermal selection and evolution in *P. infestans* found a countergradient adaptation in metabolic rate and fungicide resistance, a trade-off between temperature niche breadth and intrinsic growth rate, and a greater physiological than genetic contribution to the observed thermal adaptation (Qin et al., 2016; Yang et al., 2016).

Here we move beyond this essentially observational approach by using an experimental evolution (acclimation) approach to understand the patterns of thermal adaptation in *P. infestans*. According to our best knowledge, no such information is available in this important pathogen. Unlike other studies that acclimated a pathogen, plant, or animal to a fixed temperature (Crowther & Bradford, 2013; Wos & Willi, 2018; Zhang et al., 2015), we mimicked gradual increases or decreases in air temperatures in nature by small incremental or decremental “stepping-stone” changes in experimental temperatures, by 1°C each time, starting from the lower (12°C) and upper (26°C) thermal boundaries usually required for the epidemic development of *P. infestans* in the field (Yang et al., 2016) and are unaware this incremental (decremental) approach has been used to study the thermal acclimation of species in literatures. The isolates, collected from

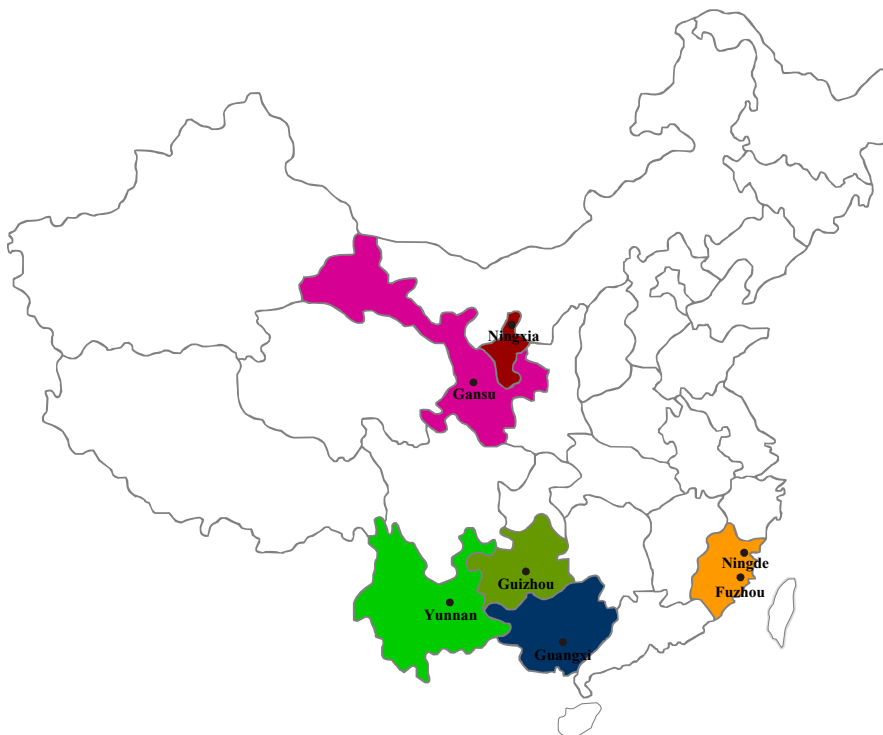
different field sites, representing various evolutionary histories of thermal adaptation of the pathogen were used as parental populations and acclimated continuously for 40 days at each experimental temperature step to increase the chance of mutation and selection.

The specific objectives of the study are as follows: (a) to investigate the pattern of thermal adaptation in *P. infestans* by analyzing its phenotypic responses to changing temperature; (b) to infer the possible mechanisms contributing to the thermal adaptation of *P. infestans* during acclimation; and (c) to analyze the effect of thermal adaptation on the interaction among biological traits of the pathogen.

## 2 | MATERIALS AND METHODS

### 2.1 | Parental isolates for the temperature acclimation experiment

A total of 21 genetically distinct *P. infestans* isolates previously characterized by SSR assays (Lees et al., 2006; Qin et al., 2016; Wu, Wang, Shen, et al., 2019) and sequence analysis of several functional genes (Cárdenas et al., 2011; Yang et al., 2018) were selected for the temperature acclimation experiment. These isolates were randomly collected from seven potato fields (three isolates from each field) located in Gansu, Guangxi, Guizhou, Ningxia, Yunnan, and Fujian (Fuzhou and Xiapu) along a climatic gradient of China at the early stages of epidemics in 2010 and 2011 (Figure 1, Wu, Wang, Shen, et al., 2019). Among these locations, Gansu and Ningxia, representing a continental climate, and Guizhou and Yunnan, representing a temperate climate, are the four top potato production areas in China, while Guangxi and Fujian, representing



**FIGURE 1** Map showing the geographic locations of the seven *Phytophthora infestans* populations included in this study

a subtropical climate, are the two provinces with the highest potential of developing a potato industry in coming decades (Yang et al., 2016). Detailed information on the collection, isolation, and molecular characterization of the pathogen isolates is provided in our previous publications (Qin et al., 2016; Wu, Wang, Shen, et al., 2019).

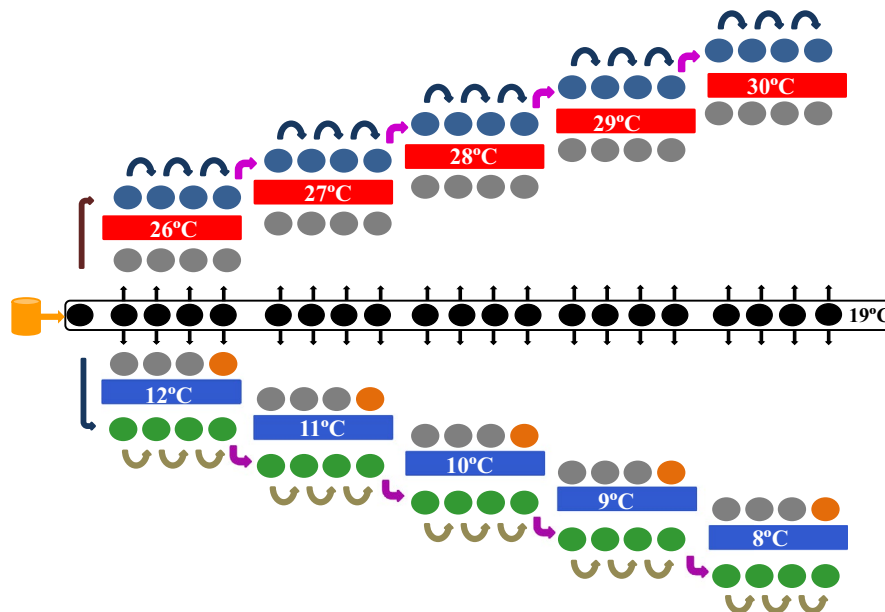
## 2.2 | Temperature acclimation

*Phytophthora infestans* isolates revived from long-term storage were acclimated under a pattern of stepwise increases or decreases of temperature over a total of 20 generations (10 days per generation) for 200 days on rye B agar (50 g/L rye and 12 g/L agar) in 9-cm petri dishes (Figure 2). In the high-temperature acclimation experiment, pathogen isolates were grown under a 1°C incremental sequence of increasing experimental temperatures starting at 26°C and ending at 30°C (five temperature regimes in total). At each temperature step, pathogen isolates were acclimated for four generations (40 days = 10 days/generation). At the end of each generation, the colony size of each isolate was digitalized and a mycelial plug ( $\phi = 5$  mm) taken to initiate a new generation on a fresh rye B agar plate. Similarly, in the low-temperature acclimation, pathogen isolates were subject to a steady 1°C stepwise decrease of experimental temperatures starting at 12°C and ending at 8°C (again a total of five temperature regimes). The low and high thermal limits were chosen to reflect extreme temperature regimes to which, our previous studies have shown one or more of the

pathogen populations was exposed during the potato growing season (i.e., during the pathogenic phase of its life cycle) across the sampled climatic gradient. Globally, most potato-growing regions fall within these extreme cold and hot limits of temperatures (Haverkort, 1990). For both the low- and high-temperature acclimations, controls were set by directly transferring the unacclimated parental isolates maintained in the long term at the pathogen's optimum of 19°C (Yang et al., 2016) to the current acclimation temperature regime anew at the beginning of each generation (Figure 2, gray circles). Control plates inoculated with unacclimated parental isolates were cultured together with the acclimated isolates in each temperature regime. The experimental units (isolates) of both acclimated isolates and unacclimated controls at each temperature regime were repeated three times (i.e., grown on three petri dishes filled with rye B agar), batched together according to temperature regimes and laid out on a completely randomized design in the same growth chamber supplemented with lights. To minimize experimental errors, the entire inoculation procedure associated with the particular temperature regime was completed by the same person in the same day.

## 2.3 | Colony growth and aggressiveness measurement of acclimated and unacclimated parental *Phytophthora infestans* isolates

Colonies of *P. infestans* isolates were photographed using a Nikon-26 camera at the end of each generation (10th day after



**FIGURE 2** The flowchart of *Phytophthora infestans* temperature acclimatization: The yellow cylinder represents the parental isolates preserved in microtubes at 13°C for long-term storage. The black circles represent the parental isolates revived and maintained on rye B plates at 19°C (the optimum temperature of the pathogen); gray circles represent parental isolates without trained (controls); blue circles represent isolates continuously trained under increasing temperatures; and green circles represent isolates continuously trained under reducing temperatures. Arrows represent the transfer of isolates to fresh rye B plates after 10 days (one generation), and orange circles indicate the stages when aggressiveness of acclimated isolates was tested. The acclimation lasted for 20 generations (transfers) with 10 days in each generation

inoculation) for both acclimated and unacclimated parental isolates. Plates with concerned *P. infestans* colonies were placed 60 cm under the camera mounted on a tripod, creating images with a resolution of ~300 dpi. Artificial lights produced by a fluorescent lamp were used for photographing. As a result, a total of 5,040 [21 isolates × 3 repeats × 4 generations × 10 temperatures (5 low temperatures + 5 high temperatures) × 2 treatments (1 acclimation + 1 unacclimated)] colonies were digitalized in the experiment and their sizes were estimated using the image analysis software ASSESS (Lamari, 2002). The software measures colony area (cm<sup>2</sup>) based on the number of pixels inside the delimited polygon. For most of images, the software can recognize the boundaries between agar plates and pathogen colonies automatically and determine the delimited polygon for the colonies using the color planes embedded. Some manual manipulations might be required for the images with low contrast such as those taken from thinner colonies. Aggressiveness of the isolates was evaluated at the end of each four generations at a particular temperature regime by measuring lesion development on detached potato leaves (Zhan et al., 2005). Leaf-based aggressiveness tests were performed on detached leaflets taken from the potato cultivar Favorita, a cultivar universally susceptible to *P. infestans* (Foolad, Sullenberger, & Ashrafi, 2015). To do this, fully expanded leaflets excised from Favorita plants grown in a disease-free field for about 8 weeks were placed in petri dishes ( $\phi = 9$  cm, where  $\phi$  represents diameter) filled with 2% water agar and then inoculated on the abaxial side with mycelial plugs ( $\phi = 5$  mm) taken from the colonies of both the acclimated and unacclimated parental isolates at the end of the last generation (i.e., 10th day of the 4th, 8th, 12th, 16th, and 20th generations) of each temperature regime. The petri dishes containing the inoculated leaflets were kept in a growth chamber with a programmed temperature corresponding to the temperature regimes of the acclimation experiment. For example, if the assay was tested for the aggressiveness of isolates taken from the last generation of acclimation at 10°C, the temperature in the growth chamber was also programmed to 10°C. Disease lesions formed on inoculated leaflets were photographed 10 days after inoculation. Lesion sizes were also measured by the image analysis software ASSESS (Lamari, 2002) and used as an indicator of pathogen aggressiveness. Aggressiveness measured by growth on potato leaflets was tested for all acclimated and unacclimated parental isolates from both high- and low-temperature acclimation experiments. However, only data from the low-temperature acclimation experiment were available for further analysis of thermal adaptation as the high-temperature acclimation experiment failed because most leaflets held under the corresponding temperature regime rotted before disease lesions appeared. As a result, a total of 840 disease data points were generated [21 isolates × 4 replicates × 5 experimental temperatures × 2 treatments (1 acclimation + 1 unacclimated)] and included in the further analysis of thermal adaptation in the pathogen. As for colony growth assessments, the entire measurements of *P. infestans* aggressiveness were completed by the same person to reduce any possible errors

associated with selection of plant materials, inoculation procedure, image analysis, etc.

## 2.4 | Data analysis

Fitness of the *P. infestans* isolates was inferred from the amount of disease (aggressiveness) they induced on the detached leaflets of the susceptible potato cultivar Favorita and/or colony sizes they formed on rye B agar after 10 days of inoculation. Thermal adaptation of the pathogen was evaluated by comparing the fitness of the acclimated isolates relative to their unacclimated parental isolates in each generation and temperature regime as well as the average across generations and temperature regimes. Analysis of variance (ANOVA) for colony size and aggressiveness was conducted using a general linear model (SAS Institute, 2013) according to the following:

$$Y_{\text{matpi}} = M + A + T + P + I(P) + E_{\text{matpi}}$$

where  $Y_{\text{matpi}}$  is the mean colony size or aggressiveness for isolate  $i(p)$  from population  $p$  at temperature under treatment  $A$  and  $M$ ,  $A$ ,  $T$ ,  $P$ ,  $I(P)$ , and  $E_{\text{matpi}}$  are the overall mean, treatment (acclimated isolates vs. unacclimated parents), experimental temperature, pathogen collection sites, isolate, and the variance among replicates, respectively. In this analysis, variance was estimated separately for high- and low-temperature acclimations and temperature and collection site were treated as fixed variable while isolates and treatment were treated as random variables. Duncan's multiple range and LSD tests (Ott, 1992) were applied to the results to determine the significance of differences in fitness between the acclimated and unacclimated parental isolates and among isolates originating from locations differing in annual thermal conditions based on the SAS software. Monthly temperature data presented as an average over 15–30 years for each collection site were downloaded from World Climate (<http://www.world-climate.com/>) as described previously (He et al., 2018; Yang et al., 2016; Zhan & McDonald, 2011). Association between colony size and aggressiveness in both the acclimated and unacclimated parental isolates was evaluated by Pearson's correlation (Lawrence & Lin, 1989). In this analysis, colony size data of the isolates from different generations within the same temperature regime were pooled together. Pearson's correlation was also used to evaluate the association between fitness components (colony size and aggressiveness) and thermal condition at the pathogen collection sites as well as the impact of temperature acclimation on this association. Colony size and aggressiveness averaged over different generations of the same temperature regimes as well as different isolates derived from the same location were used for this analysis.

## 3 | RESULTS

Temperature acclimation significantly increased *P. infestans* fitness, measured by aggressiveness on detached leaves and/or colony size

**TABLE 1** Duncan's multiple range tests for differences in colony size and aggressiveness among acclimated and unacclimated *Phytophthora infestans* isolates originated from different temperature zones of China

| Groups  | AMT (°C) | Colony size at low-temperature acclimation (cm <sup>2</sup> ) |                            | Colony size at high-temperature acclimation (cm <sup>2</sup> ) |                            | Lesion size at low-temperature acclimation (cm <sup>2</sup> ) |                          |
|---------|----------|---|----------------------------|--|----------------------------|---|--------------------------|
|         |          | Unacclimated  | Acclimated                 | Unacclimated   | Acclimated                 | Unacclimated  | Acclimated               |
| Guizhou | 14.7     | 21.83 ± 1.47 <sup>B</sup>                                     | 29.01 ± 0.94 <sup>B</sup>  | 9.35 ± 1.72 <sup>C</sup>                                       | 10.72 ± 1.75 <sup>C</sup>  | 6.74 ± 1.44 <sup>A</sup>                                      | 7.17 ± 1.13 <sup>A</sup> |
| Fuzhou  | 20.5     | 27.48 ± 2.48 <sup>A</sup>                                     | 30.74 ± 1.64 <sup>A</sup>  | 16.38 ± 2.45 <sup>A</sup>                                      | 16.06 ± 2.36 <sup>A</sup>  | 3.77 ± 1.11 <sup>CD</sup>                                     | 5.43 ± 1.36 <sup>B</sup> |
| Guangxi | 22.6     | 17.38 ± 1.43 <sup>C</sup>                                     | 19.33 ± 1.15 <sup>F</sup>  | 11.91 ± 2.07 <sup>B</sup>                                      | 13.55 ± 1.84 <sup>B</sup>  | 1.07 ± 0.98 <sup>E</sup>                                      | 2.24 ± 0.98 <sup>C</sup> |
| Gansu   | 11.7     | 23.09 ± 1.59 <sup>B</sup>                                     | 30.29 ± 0.84 <sup>AB</sup> | 8.39 ± 1.69 <sup>CD</sup>                                      | 11.41 ± 1.59 <sup>BC</sup> | 7.43 ± 1.39 <sup>A</sup>                                      | 8.71 ± 1.15 <sup>A</sup> |
| Ningxia | 7.0      | 22.82 ± 1.48 <sup>B</sup>                                     | 25.91 ± 1.12 <sup>C</sup>  | 8.30 ± 1.49 <sup>CD</sup>                                      | 11.12 ± 1.52 <sup>C</sup>  | 4.73 ± 1.36 <sup>BC</sup>                                     | 5.32 ± 1.27 <sup>B</sup> |
| Xiapu   | 20.3     | 15.54 ± 1.19 <sup>C</sup>                                     | 21.38 ± 0.80 <sup>E</sup>  | 4.42 ± 0.79 <sup>E</sup>                                       | 7.16 ± 0.93 <sup>D</sup>   | 2.64 ± 1.23 <sup>D</sup>                                      | 4.52 ± 1.22 <sup>B</sup> |
| Yunnan  | 15.6     | 21.37 ± 1.38 <sup>B</sup>                                     | 23.67 ± 0.87 <sup>D</sup>  | 6.36 ± 0.95 <sup>DE</sup>                                      | 8.12 ± 1.05 <sup>D</sup>   | 6.12 ± 1.29 <sup>AB</sup>                                     | 7.64 ± 1.27 <sup>A</sup> |

Note: Values followed by different letters in the same column are significantly different at  $p = .05$ , and data in parentheses are 95% confidence interval.

Abbreviation: AMT, average mean temperature.

**TABLE 2** Comparison of colony size and aggressiveness between acclimated and unacclimated *Phytophthora infestans* isolates

| Treatments   | Colony size at low-temperature acclimation (cm <sup>2</sup> ) | Colony size at high-temperature acclimation (cm <sup>2</sup> ) | Lesion size at low-temperature acclimation (cm <sup>2</sup> ) |
|--------------|---|--|---|
| Acclimated   | 25.76 ± 0.47 <sup>A</sup>                                     | 11.16 ± 0.63 <sup>A</sup>                                      | 5.86 ± 0.48 <sup>A</sup>                                      |
| Unacclimated | 21.36 ± 0.46 <sup>B</sup>                                     | 9.30 ± 0.66 <sup>B</sup>                                       | 4.58 ± 0.49 <sup>B</sup>                                      |
| Gain (%)     | 20.60   | 20.00  | 27.95   |

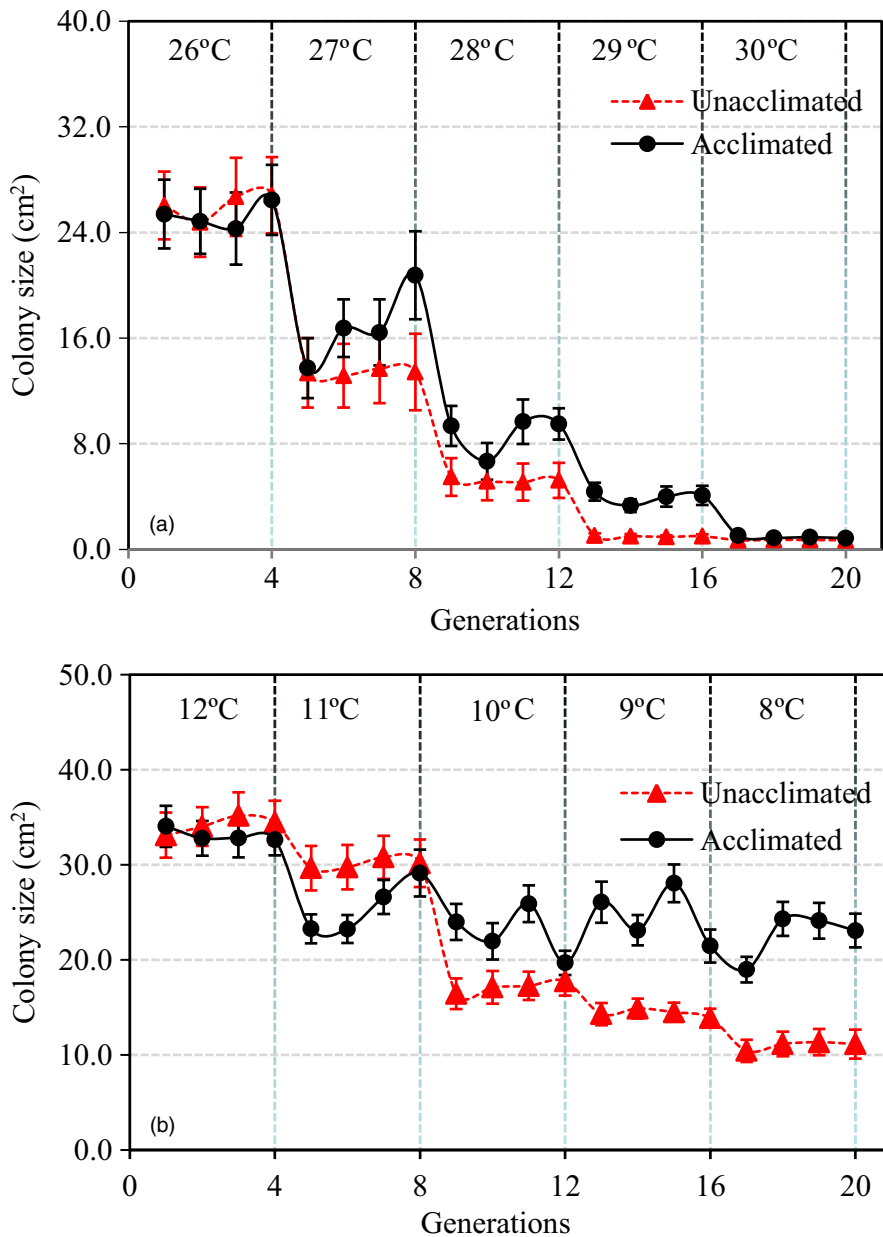
Note: Values followed by different letters in the same column are significantly different at  $p = .05$ , and data in parentheses are 95% confidence interval.

on agar ( $p < .05$ ), although isolates from different geographic locations varied in the extent of the improvement (Tables 1 and 2). Under high-temperature acclimation, the mean colony size across the 20 generations in the seven populations ranged from 7.16 to 16.06 cm<sup>2</sup> with an overall average of 11.16 cm<sup>2</sup> in the acclimated isolates compared to an overall average of 9.30 cm<sup>2</sup> with a range of 4.42 to 16.38 cm<sup>2</sup> in the unacclimated parental isolates. Under low-temperature acclimation, the mean colony size and aggressiveness (lesion size) in the seven populations ranged from 19.33 to 30.74 cm<sup>2</sup> and 2.24 to 8.71 cm<sup>2</sup> with an overall average of 25.76 and 5.86 cm<sup>2</sup> in the acclimated isolates compared to an overall average of 21.36 and 4.58 cm<sup>2</sup> with a range of 15.54–27.48 cm<sup>2</sup> and 1.07–7.43 cm<sup>2</sup> in the unacclimated parental isolates, respectively. In most cases of high- and low-temperature acclimations, trained isolates performed better than untrained parental isolates (Tables 1 and 2).

At the temporal scale, no differences in colony size were found between the acclimated and unacclimated parental isolates in the earlier acclimation generations (Figure 3). At high and increasing temperature conditions, thermal adaptation, indicated by an increasing fitness in the acclimated isolates compared to unacclimated isolates, was observed starting from 27°C at the 5th generation of acclimation, speeding up gradually until 29°C at the 16th generation of acclimation and ending sharply after the experimental temperature increased to 30°C (Figure 3a, Figure 4a). At low- and decreasing-temperature conditions, adaptation was observed starting from either the 9th generation at 10°C (colony size)—colony size of

unacclimated parental isolates sharply declined at this time—or the 8th generation at 11°C (aggressiveness; Figure 5), and the fitness difference between acclimated and unacclimated parental isolates slowly and waveringly increased as the experimental temperature reduced further (Figures 3b and 4b). In both low- and high-temperature acclimations, more among-generation fluctuation in colony size was found in acclimated isolates than the unacclimated parental isolates (Figure 3a,b). The pattern of temporal changes in fitness was consistent over the isolates originating from different locations (Figure S1).

Acclimation also changed the pattern of association between performance (colony size and aggressiveness) and annual mean temperature in the collection sites of the pathogen. No association ( $r = .22, p = .7222$ ;  $r = -.31, p = .6117$ ) between colony size and annual mean temperature at the pathogen collection sites was found in the high-temperature acclimation (Figure 6a,b). In the low- and decreasing-temperature conditions, the significance of negative association ( $r = .96, p = .0095$ ;  $r = .88, p = .0490$ ) between performance and annual mean temperature at the pathogen collection sites was strongly affected by experimental temperatures in the acclimated isolates as indicated by a correlation analysis (Figure 6d,f), while no such impact was founded in the unacclimated parental isolates ( $r = .51, p = .3800$ , Figure 6c; and  $r = .01, p = .9873$ , Figure 6e). Similarly, acclimation also affected the association between biological and ecological traits of the pathogen. Higher positive correlation was found between aggressiveness and colony size in the acclimated pathogen compared



**FIGURE 3** Temporal changes (fitted smoother) of mean colony size and its 95% confidence interval (CI) in the acclimated and unacclimated *Phytophthora infestans* isolates. The mean colony sizes were estimated over all 21 isolates collected from the seven locations. Acclimation was carried out in two directions under continuing increase or decrease of temperatures over 20 generations (10 days per generation). At the high-temperature acclimation, the pathogen was continually trained under a steady increase of experimental temperatures at 1°C interval starting from 26°C and ending at 30°C (total five temperature regimes). In each temperature regime, the pathogen was acclimated for four generations. Similarly, at the low-temperature acclimation, the pathogen was continually trained under a steady decrease of experimental temperatures at 1°C interval starting from 12°C. The acclimation was also executed under five temperature regimes (ending at 8°C) each with four generations: (a) high-temperature acclimation; and (b) low-temperature acclimation

to the correlation between the two parameters in the unacclimated parental isolates (Figure 7).

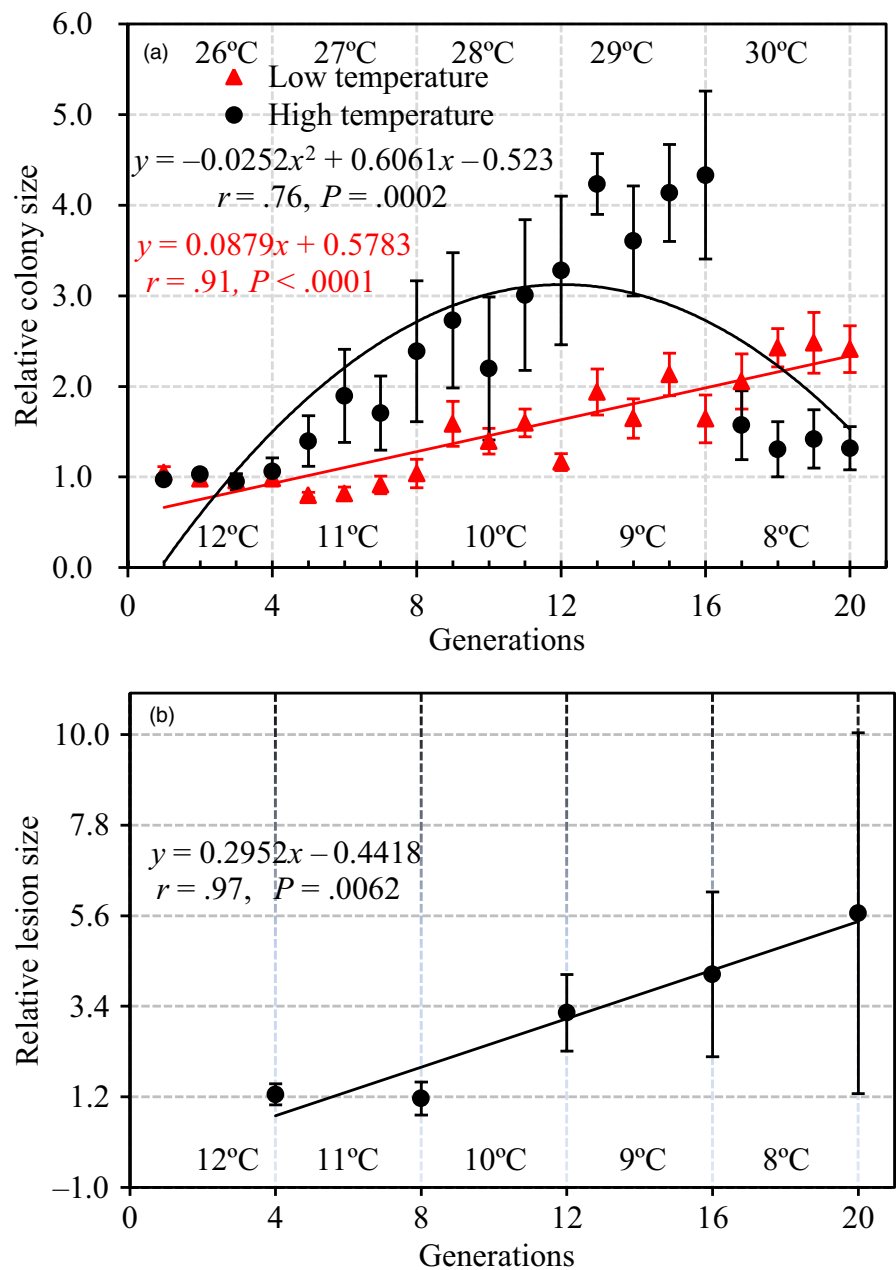
#### 4 | DISCUSSION

Gradual rising global temperature is likely to be one of the major environmental factors significantly affecting future epidemics of infectious diseases such as potato late blight caused by *P. infestans* (Mariette et al., 2016; Sparks et al., 2014; Yang et al., 2016). As a consequence, knowledge of the capacity for thermal adaptation in pathogens is critical to predicting future disease severity and distribution and developing corresponding mitigation strategies (Juroszek & von Tiedemann, 2011). In this study, 21 *P. infestans* genotypes originating from different thermal environments in China were acclimated for 200 days under thermal stresses by a stepwise increasing

or decreasing of experimental temperatures. The evolutionary responses of the isolates to these thermal stresses were evaluated by comparing their fitness component (i.e., intrinsic growth rate measured by colony size on petri dishes and aggressiveness measured by lesion size on detached potato leaves) to the parental isolates. Our result show that acclimated *P. infestans* had higher fitness than unacclimated isolates although the extent of this improvement significantly depended on the thermal environments from which the isolates originated (Table 1). Both the intrinsic growth rate and aggressiveness of the isolates increased ~20% in the 200 days of the acclimation experiment (Table 2), resulting in an average of 4% improvement in each step of temperature change.

Both genetic and physiological adaptation can improve the fitness of living organisms surviving in stressing environments (Draghi & Whitlock, 2012; Narum, Campbell, Meyer, Miller, & Hardy, 2013; Somero, 2010). The two events usually occur

**FIGURE 4** Temporal changes in mean relative colony size, aggressiveness, and their associated 95% confidence intervals of acclimated to unacclimated *Phytophthora infestans* isolates growing in the low- and high-temperature acclimation treatments. The aggressiveness of the isolates was assessed at the end of each four generations, and the mean relative colony sizes and mean aggressiveness were estimated over all 21 isolates collected from the seven locations. Acclimations were carried out in two directions under continuing increase or decrease of temperatures over 20 generations (10 days per generation). At the high-temperature acclimation, pathogen was continually trained under a steady increase of experimental temperatures at 1°C interval starting from 26°C and ending at 30°C (total five temperature regimes). In each temperature regime, the pathogen was acclimated for four generations. Similarly, at the low-temperature acclimation, pathogen was continually trained under a steady decrease of experimental temperatures at 1°C interval starting from 12°C. The acclimation was also executed under five temperature regimes (ending at 8°C) each with four generations: (a) relative colony size under high- and low-temperature acclimation; and (b) relative lesion size under low-temperature acclimation



simultaneously during acclimating processes and are difficult to be fully disentangled. In many experiments involving thermal acclimation, increases in fitness to temperature stresses were thought to be caused primarily by physiological adaptation or phenotypic plasticity (Fierst, 2011; Folguera, Bastías, & Bozinovic, 2009). However, in the current study we have two lines of evidence to support the hypothesis that genetic adaptation to thermal stresses occurred in the pathogen isolates during the 200 days of acclimation.

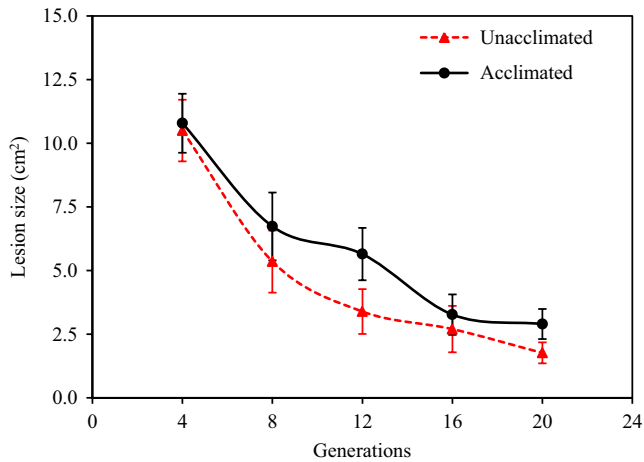
Empirical studies have shown that physiological adaptation during acclimation usually leads to environmental specialists (Bacigalupe et al., 2018; Levins, 1969). Species perform better in the specific environmental niche to which they were acclimated as compared to their ancestors, but their fitness is not changed or even reduced in other environmental niches to which they were not acclimated. On the other

hand, genetic adaptation usually selects for environmental generalists due to heritable change in mean phenotypic value (Chevin, Lande, & Mace, 2010; Walters & Berger, 2019). The adapted species not only demonstrate a higher fitness in the specific environmental niches to which they were acclimated, as relative to their ancestors, but also generally perform better in a wide range of other environmental niches to which they were not acclimated (Rundle & Nosil, 2005). In our experiment, we found that acclimated isolates demonstrated higher fitness at all temperature regimes after five (high temperature) or nine (low temperature) generations of acclimation (Figures 3–5). In this case, the lag of adaptation time in the lower temperature regimes may reflect its longer generation time relative to the higher temperature regimes. However, it has also been documented that genetic adaptation could lead to thermal specialists (Donelson et al., 2012; Seebacher, Ducret, Little, & Adriaenssens, 2015).

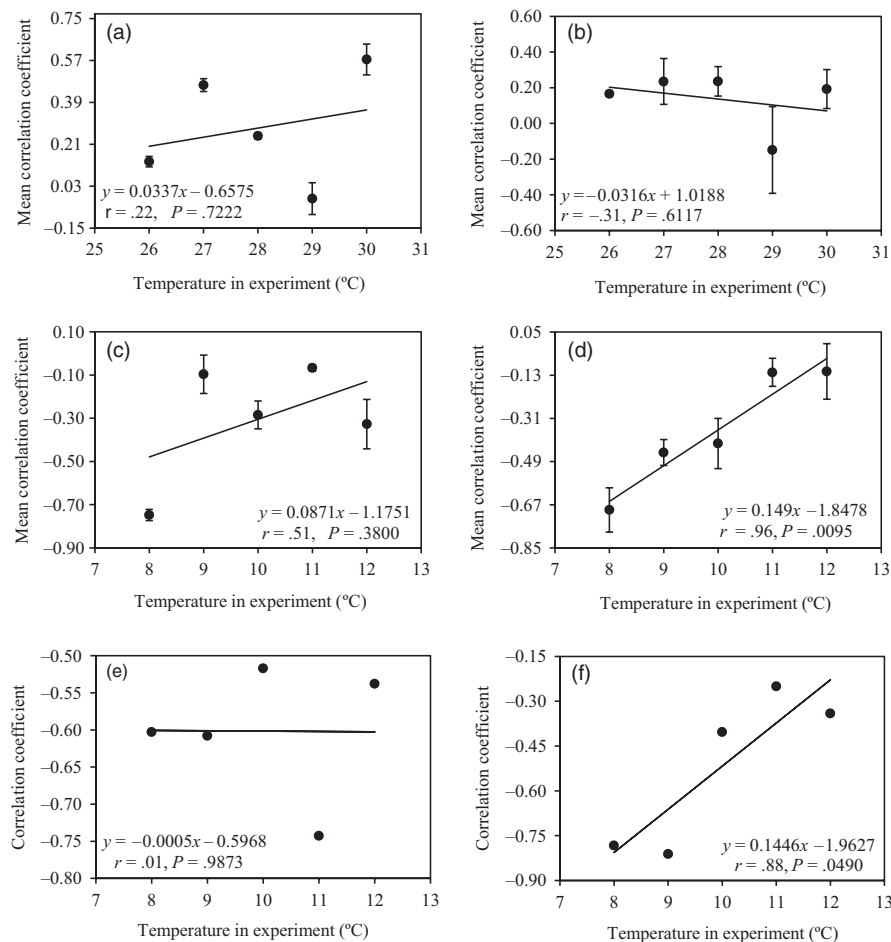


The fitness of the acclimated isolates fluctuated dramatically among generations of the same temperature treatments as compared to the unacclimated ancestry (Figure 3). This pattern of variation is inconsistent with the expectation of a steady increase in

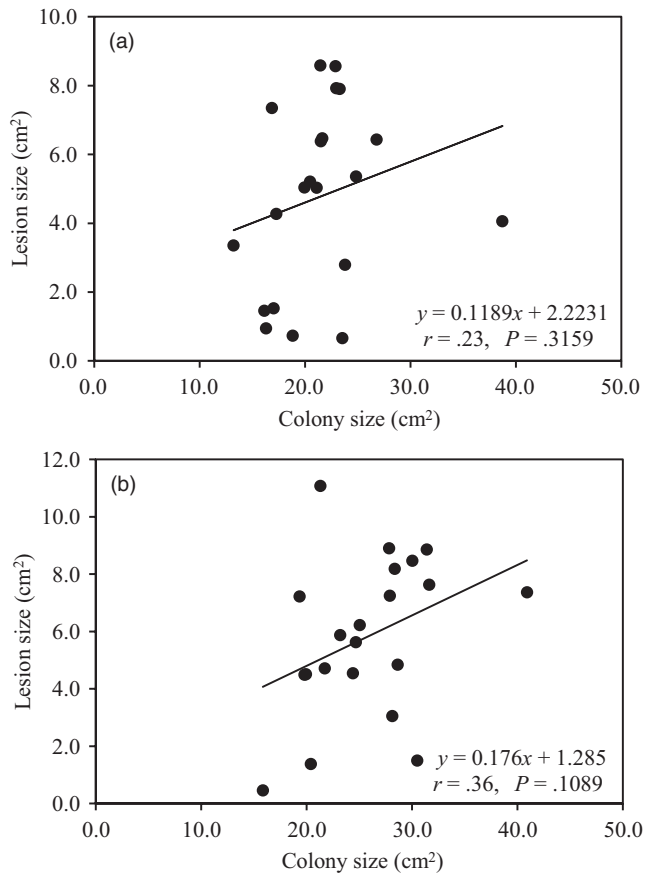
fitness caused by physiological adaptation to an acclimating environment (Leroi, Bennett, & Lenski, 1994), but can be generated by genetic adaptation caused by changes of genomic architecture in the acclimated isolates. It was previously demonstrated that genetic adaptation to thermal conditions can reverse or reinforce the effect of physiological adaptation (Ho & Zhang, 2018), leading to the oscillating fitness in the acclimated *P. infestans* isolates among generations of the same temperature treatments. Large genome size and high mutability of the pathogen may contribute to genetic adaptation occurring during this short period of thermal acclimation (Haas et al., 2009; Vetukuri et al., 2012). The pattern of thermal adaptation in the pathogen differed markedly between the high- and low-temperature acclimations (Figure 4). In the low-temperature acclimation, the fitness of *P. infestans* isolates increased at a later stage and at a slower rate compared to the high-temperature acclimation. Furthermore, in the low-temperature acclimation, the fitness of the pathogen isolates was continually improved over the entire experiment, while in the high-temperature acclimation, experimental pathogen isolates suddenly stopped growing when the experimental temperature reached 30°C (Figure 4). The growth collapse point is close to the maximum growth temperature (31.5°C) of the pathogen we previously estimated for these locations (Yang et al., 2016) and suggests a niche boundary may exist in the evolutionary adaptation of *P. infestans* to high-temperature stresses. Genetic adaptation to extreme stresses requires a range of heritable variation that is broad enough



**FIGURE 5** Temporal changes of mean lesion size (aggressiveness) and its associated 95% confidence interval (CI) in the acclimated and unacclimated *Phytophthora infestans* isolates. Aggressiveness of the isolates was assessed at the end of each four generations at a particular temperature regime, and the mean aggressiveness was estimated over all 21 isolates collected from the seven locations



**FIGURE 6** The response of the association between pathogen colony size or lesion size and the annual mean temperature at collection sites to the changing experimental temperatures in acclimated and unacclimated isolates: (a) without high-temperature acclimation for mycelial growth; (b) with high-temperature acclimation for mycelial growth; (c) without low-temperature acclimation for mycelial growth; (d) with low-temperature acclimation for mycelial growth; (e) without low-temperature acclimation for aggressiveness; and (f) with low-temperature acclimation for aggressiveness



**FIGURE 7** The impact of low-temperature acclimation on the association between colony size and aggressiveness of 21 *Phytophthora infestans* isolates collected from seven potato field geographic sites in China: (a) unacclimated; and (b) acclimated

to provide a sustainable growth of living organisms (Huntley, 2007). Lack of time to accumulate further genetic variation may prevent the survival of the pathogen beyond this upper thermal boundary. Unfortunately, we did not continue the experiment after the collapse point and it is not clear whether the pathogen's fitness will recover if acclimation time periods at this temperature regime were extended. At the other end of the thermal scale, the estimated minimum growth temperature for the pathogen isolates included in the acclimation experiments was 5.8°C (Yang et al., 2016). Inevitably there must be a temperature point below which no growth occurs. However, it is not clear at what temperature this might be or whether such a thermal boundary is approached gradually or abruptly although metabolisms usually reduce less steeply when temperature approaches to lower limit than upper limit, leading to skewed thermal curves (Molnár, Sckrabulis, Altman, & Raffel, 2017). Further experiments with a low-temperature and/or prolonged acclimation to stresses of boundary thermal conditions are required to elucidate these issues.

Thermal adaptation to the low-temperature acclimation alters the interaction among life-history and ecological traits in the pathogen as well as the association of these traits with past selection experienced by the pathogen (Figures 4 and 6). For example, low-temperature acclimation enforces a countergradient adaptation (Yang et al.,

2016) of the pathogen to past selection imposed by local temperature regimes (Figure 6c–f) and strengthens the positive association between pathogen's intrinsic growth rate in vitro and aggressiveness (Figure 7). The results suggest that low-temperature-acclimated *P. infestans* isolates, and potentially many other pathogens as well, have an enhanced ability to colonize and reproduce on its potato host although a comparative analysis cannot be performed for the high-temperature acclimation experiment due to the failure to generate aggressiveness data under those temperature regimes.

In conclusion, our study demonstrates that plant pathogen has the ability to rapidly adapt to changing temperature although the underlying genetic or physiological mechanisms cannot be disentangled in the current results. In the future study, comparative analyses between acclimated and unacclimated isolates at genetic or genomic level are required to determine relative contribution of genetic and physiological mechanisms to the observed adaptation. A significant feature of the high-temperature acclimation in the current study was the identification of an apparent thermal mortality threshold for *P. infestans* around 30°C. More importantly though, was the observation that isolates from all collection locations (covering continental, temperate, and subtropical environments) showed significant ability to respond to thermal stresses. While these stresses exceed those currently experienced at the collection sites, they clearly demonstrate *P. infestans* ability to cope with some thermal stresses it is likely to experience in the foreseeable future. Whether these evolutionary trends of pathogen adaptation can be translated into changes in disease severity and distribution under future climatic conditions depends on the relative ability and speed of thermal adaption in the associated hosts (Altizer, Ostfeld, Johnson, Kutz, & Harvell, 2013; Kawecki & Ebert, 2004; Zhan, Thrall, & Burdon, 2014). Therefore, it may not be realistic to robustly predict the future epidemiological landscapes of potato late blight and other main crop diseases without empirical knowledge of thermal adaptation patterns in both plant hosts and pathogens drawn from parallel experiments. However, it is generally believed that pathogens can adapt to temperature shifts more quickly than hosts owing to their faster tissue-specific metabolisms (Gillooly, Brown, West, Savage, & Charnov, 2001), short generation time (Chen & Stillman, 2012), and larger population sizes (Kingsolver & Huey, 2008; Zhan, Thrall, Papaix, Xie, & Burdon, 2015), and in the case of *P. infestans*, in vitro measurements of aggressiveness in a laboratory conditions are generally correlated well with in vivo data from field trials (unpublished data). These factors enhance the risk of more severe epidemics in the future, and adequate action is required to minimize their impact on food security. Due to the massive work required, we only included three isolates from each of the seven locations in the study. However, the finding of consistent patterns in evolutionary responses among isolates from different locations (Figure S1) suggests that small sample size may not affect our conclusions largely.

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## CONFLICT OF INTEREST

None declared.

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## DATA AVAILABILITY STATEMENT

Data are available from the Dryad Digital Repository: <https://doi.org/10.5061/dryad.z08kpr8h> (Wu, Wang, Yahuza, et al., 2019).

## REFERENCES

- Abeles, F., & Ott, L. (1992). *An introduction to statistical methods and data analysis* (5th ed.). Belmont, CA: Duxbury Press. <https://doi.org/10.2307/2288162>
- Addison, A. L., Powell, J. A., Six, D. L., Moore, M., & Bentz, B. J. (2013). The role of temperature variability in stabilizing the mountain pine beetle-fungus mutualism. *Journal of Theoretical Biology*, 335, 40–50. <https://doi.org/10.1016/j.jtbi.2013.06.012>
- Altizer, S., Ostfeld, R. S., Johnson, P. T., Kutz, S., & Harvell, C. D. (2013). Climate change and infectious diseases: From evidence to a predictive framework. *Science*, 341(6145), 514–519. <https://doi.org/10.1126/science.1239401>
- Angilletta, M. J. Jr, Oufiero, C. E., & Leaché, A. D. (2006). Direct and indirect effects of environmental temperature on the evolution of reproductive strategies: An information-theoretic approach. *The American Naturalist*, 168(4), E123–E135. <https://doi.org/10.1086/507880>
- Bacigalupe, L. D., Gaitán-Espitia, J. D., Barria, A. M., Gonzalez-Mendez, A., Ruiz-Aravena, M., Trinder, M., & Sinervo, B. (2018). Natural selection on plasticity of thermal traits in a highly seasonal environment. *Evolutionary Applications*, 11(10), 2004–2013. <https://doi.org/10.1111/eva.12702>
- Billiard, S., López-Villavicencio, M., Devier, B., Hood, M. E., Fairhead, C., & Giraud, T. (2011). Having sex, yes, but with whom? Inferences from fungi on the evolution of anisogamy and mating types. *Biological Reviews*, 86(2), 421–442. <https://doi.org/10.1111/j.1469-185X.2010.00153.x>
- Birch, P. R. J., Bryan, G., Fenton, B., Gilroy, E. M., Hein, I., Jones, J. T., ... Toth, I. K. (2012). Crops that feed the world 8: Potato: Are the trends of increased global production sustainable? *Food Security*, 4(4), 477–508. <https://doi.org/10.1007/s12571-012-0220-1>
- Boland, G. J., Melzer, M. S., Hopkin, A., Higgins, V., & Nassuth, A. (2004). Climate change and plant diseases in Ontario. *Canadian Journal of Plant Pathology*, 26(3), 335–350. <https://doi.org/10.1080/07060660409507151>
- Cárdenas, M., Grajales, A., Sierra, R., Rojas, A., González-Almario, A., Vargas, A., ... Restrepo, S. (2011). Genetic diversity of *Phytophthora infestans* in the Northern Andean region. *BMC Genetics*, 12(1), 23. <https://doi.org/10.1186/1471-2156-12-23>
- Chen, X., & Stillman, J. H. (2012). Multigenerational analysis of temperature and salinity variability affects on metabolic rate, generation time, and acute thermal and salinity tolerance in *Daphnia pulex*. *Journal of Thermal Biology*, 37(3), 185–194. <https://doi.org/10.1016/j.jtherbio.2011.12.010>
- Chevin, L. M., Lande, R., & Mace, G. M. (2010). Adaptation, plasticity, and extinction in a changing environment: Towards a predictive theory. *PLoS Biology*, 8(4), e1000357. <https://doi.org/10.1371/journal.pbio.1000357>
- Chown, S. L., Addo-Bediako, A., & Gaston, K. J. (2002). Physiological variation in insects: Large-scale patterns and their implications. *Comparative Biochemistry and Physiology Part B: Biochemistry and Molecular Biology*, 131(4), 587–602. [https://doi.org/10.1016/S1096-4959\(02\)00017-9](https://doi.org/10.1016/S1096-4959(02)00017-9)
- Colinet, H., Overgaard, J., Com, E., & Sorensen, J. G. (2013). Proteomic profiling of thermal acclimation in *Drosophila melanogaster*. *Insect Biochemistry and Molecular Biology*, 43(4), 352–365. <https://doi.org/10.1016/j.ibmb.2013.01.006>
- Crowther, T. W., & Bradford, M. A. (2013). Thermal acclimation in widespread heterotrophic soil microbes. *Ecology Letters*, 16(4), 469–477. <https://doi.org/10.1111/ele.12069>
- de Nadal, E., Ammerer, G., & Posas, F. (2011). Controlling gene expression in response to stress. *Nature Reviews Genetics*, 12, 833. <https://doi.org/10.1038/nrg3055>
- Donelson, J. M., Munday, P. L., McCormick, M. I., & Pitcher, C. R. (2012). Rapid transgenerational acclimation of a tropical reef fish to climate change. *Nature Climate Change*, 2(1), 30–32. <https://doi.org/10.1038/nclimate1323>
- Draghi, J. A., & Whitlock, M. C. (2012). Phenotypic plasticity facilitates mutational variance, genetic variance, and evolvability along the major axis of environmental variation. *Evolution*, 66, 2891–2902. <https://doi.org/10.1111/j.1558-5646.2012.01649.x>
- Dysthe, J. C., Bracewell, R., & Six, D. L. (2015). Temperature effects on growth of fungal symbionts of the western pine beetle, *Dendroctonus brevicomis*. *Fungal Ecology*, 17, 62–68. <https://doi.org/10.1016/j.funeco.2015.05.010>
- Eastburn, D. M., McElrone, A. J., & Bilgin, D. D. (2011). Influence of atmospheric and climatic change on plant-pathogen interactions. *Plant Pathology*, 60(1), 54–69. <https://doi.org/10.1111/j.1365-3059.2010.02402.x>
- Elad, Y., & Pertot, I. (2014). Climate change impacts on plant pathogens and plant diseases. *Journal of Crop Improvement*, 28(1), 99–139. <https://doi.org/10.1080/15427528.2014.865412>
- Fahey, C., Winter, K., Slot, M., & Kitajima, K. (2016). Influence of arbuscular mycorrhizal colonization on whole-plant respiration and thermal acclimation of tropical tree seedlings. *Ecology and Evolution*, 6(3), 859–870. <https://doi.org/10.1002/ece3.1952>
- Fierst, J. L. (2011). A history of phenotypic plasticity accelerates adaptation to a new environment. *Journal of Evolutionary Biology*, 24(9), 1992–2001. <https://doi.org/10.1111/j.1420-9101.2011.02333.x>
- Folguera, G., Bastias, D. A., & Bozinovic, F. (2009). Impact of experimental thermal amplitude on ectotherm performance: Adaptation to climate change variability? *Comparative Biochemistry and Physiology Part A: Molecular & Integrative Physiology*, 154(3), 389–393. <https://doi.org/10.1016/j.cbpa.2009.07.008>
- Foolad, M. R., Sullenberger, M. T., & Ashrafi, H. (2015). Detached-leaflet evaluation of tomato germplasm for late blight resistance and its correspondence to field and greenhouse screenings. *Plant Disease*, 99(5), 718–722. <https://doi.org/10.1094/PDIS-08-14-0794-RE>
- Forbes, G. A. (2012). Using host resistance to manage potato late blight with particular reference to developing countries. *Potato Research*, 55(3), 205–216. <https://doi.org/10.1007/s11540-012-9222-9>
- Fry, W. (2008). *Phytophthora infestans*: The plant (and R gene) destroyer. *Molecular Plant Pathology*, 9(3), 385–402. <https://doi.org/10.1111/j.1364-3703.2007.00465.x>
- Garrett, K. A., Dendy, S. P., Frank, E. E., Rouse, M. N., & Travers, S. E. (2006). Climate change effects on plant disease: Genomes to ecosystems. *Annual Review of Phytopathology*, 44(1), 489–509. <https://doi.org/10.1146/annurev.phyto.44.070505.143420>
- Ghalambor, C. K., McKay, J. K., Carroll, S. P., & Reznick, D. N. (2007). Adaptive versus non-adaptive phenotypic plasticity and the potential for contemporary adaptation in new environments. *Functional Ecology*, 21, 394–407. <https://doi.org/10.1111/j.1365-2435.2007.01283.x>
- Gillman, L. N., Keeling, D. J., Gardner, R. C., & Wright, S. D. (2010). Faster evolution of highly conserved DNA in tropical plants. *Journal of Evolutionary Biology*, 23(6), 1327–1330. <https://doi.org/10.1111/j.1420-9101.2010.01992.x>

- Gillooly, J. F., Brown, J. H., West, G. B., Savage, V. M., & Charnov, E. L. (2001). Effects of size and temperature on metabolic rate. *Science*, 293(5538), 2248–2251. <https://doi.org/10.1126/science.1061967>
- Granke, L., Windstam, S., Hoch, H., Smart, C., & Hausbeck, M. (2009). Dispersal and movement mechanisms of *Phytophthora capsici* sporangia. *Phytopathology*, 99(11), 1258–1264. <https://doi.org/10.1094/phyto-99-11-1258>
- Haas, B. J., Kamoun, S., Zody, M. C., Jiang, R. H. Y., Handsaker, R. E., Cano, L. M., ... Nusbaum, C. (2009). Genome sequence and analysis of the Irish potato famine pathogen *Phytophthora infestans*. *Nature*, 461(7262), 393–398. <https://doi.org/10.1038/nature08358>
- Harrison, J. G. (1992). Effects of the aerial environment on late blight of potato foliage—a review. *Plant Pathology*, 41(4), 384–416. <https://doi.org/10.1111/j.1365-3059.1992.tb02435.x>
- Haverkort, A. (1990). Ecology of potato cropping systems in relation to latitude and altitude. *Agricultural Systems*, 32(3), 251–272. [https://doi.org/10.1016/0308-521X\(90\)90004-A](https://doi.org/10.1016/0308-521X(90)90004-A)
- Haverkort, A. J., Boonekamp, P. M., Hutten, R., Jacobsen, E., Lotz, L. A. P., Kessel, G. J. T., ... van der Vossen, E. A. G. (2008). Societal costs of late blight in potato and prospects of durable resistance through cisgenic modification. *Potato Research*, 51(1), 47–57. <https://doi.org/10.1007/s11540-008-9089-y>
- He, M.-H., Li, D.-L., Zhu, W., Wu, E.-J., Yang, L.-N., Wang, Y.-P., ... Zhan, J. (2018). Slow and temperature-mediated pathogen adaptation to a nonspecific fungicide in agricultural ecosystem. *Evolutionary Applications*, 11(2), 182–192. <https://doi.org/10.1111/eva.12526>
- Ho, W. C., & Zhang, J. (2018). Evolutionary adaptations to new environments generally reverse plastic phenotypic changes. *Nature Communications*, 9(1), 350. <https://doi.org/10.1038/s41467-017-02724-5>
- Hoekstra, H. E., & Coyne, J. A. (2007). The locus of evolution: Evo devo and the genetics of adaptation. *Evolution: International Journal of Organic Evolution*, 61(5), 995–1016. <https://doi.org/10.1111/j.1558-5646.2007.00105.x>
- Hoffmann, A. A. (1995). Acclimation: Increasing survival at a cost. *Trends in Ecology & Evolution*, 10(1), 1–2. [https://doi.org/10.1016/s0169-5347\(00\)88949-1](https://doi.org/10.1016/s0169-5347(00)88949-1)
- Hoffmann, A. A., & Willi, Y. (2008). Detecting genetic responses to environmental change. *Nature Reviews Genetics*, 9, 421. <https://doi.org/10.1038/nrg2339>
- Huey, R. B., & Kingsolver, J. G. (1989). Evolution of thermal sensitivity of ectotherm performance. *Trends in Ecology & Evolution*, 4, 131–135. [doi:10.1016/0169-5347\(89\)90211-5](https://doi.org/10.1016/0169-5347(89)90211-5)
- Huntley, B. (2007). Limitations on adaptation: Evolutionary response to climatic change? *Heredity*, 98(5), 247–248. <https://doi.org/10.1038/sj.hdy.6800972>
- IPCC (2014). *Impacts, adaptation, and vulnerability. Part A: Global and sectoral aspects. Contribution of working group II to the fifth assessment report of the intergovernmental panel on climate change*. Cambridge, UK: Cambridge University Press.
- Judelson, H. S. (1997). Expression and inheritance of sexual preference and selfing potential in *Phytophthora infestans*. *Fungal Genetics and Biology*, 21(2), 188–197. <https://doi.org/10.1006/fgbi.1997.0973>
- Juroszek, P., & von Tiedemann, A. (2011). Potential strategies and future requirements for plant disease management under a changing climate. *Plant Pathology*, 60(1), 100–112. <https://doi.org/10.1111/j.1365-3059.2010.02410.x>
- Kalinda, C., Chimbari, M., & Mukaratirwa, S. (2017). Implications of changing temperatures on the growth, fecundity and survival of intermediate host snails of schistosomiasis: A systematic review. *International Journal of Environmental Research and Public Health*, 14(1), 80. <https://doi.org/10.3390/ijerph14010080>
- Kawecki, T. J., & Ebert, D. (2004). Conceptual issues in local adaptation. *Ecology Letters*, 7(12), 1225–1241. <https://doi.org/10.1111/j.1461-0248.2004.00684.x>
- Kingsolver, J. G., & Huey, R. B. (2008). Size, temperature, and fitness: Three rules. *Evolutionary Ecology Research*, 10(2), 251–268. <https://doi.org/10.1111/j.1752-4571.2007.00003.x>
- Koelle, K., Pascual, M., & Yunus, M. (2005). Pathogen adaptation to seasonal forcing and climate change. *Proceedings: Biological Sciences*, 272(1566), 971–977. <https://doi.org/10.1098/rspb.2004.3043>
- Lamari, L. (2002). *ASSESS: Image analysis software for plant disease quantification*. St. Paul, MN: The American Phytopathological Society.
- Latijnhouwers, M., Ligterink, W., Vleeshouwers, V. G., Van West, P., & Govers, F. (2004). A Gα subunit controls zoospore motility and virulence in the potato late blight pathogen *Phytophthora infestans*. *Molecular Microbiology*, 51(4), 925–936. <https://doi.org/10.1046/j.1365-2958.2003.03893.x>
- Lawrence, I., & Lin, K. (1989). A concordance correlation coefficient to evaluate reproducibility. *Biometrics*, 45(1), 255–268. <https://doi.org/10.2307/2532051>
- Lees, A., Wattier, R., Shaw, D., Sullivan, L., Williams, N., & Cooke, D. (2006). Novel microsatellite markers for the analysis of *Phytophthora infestans* populations. *Plant Pathology*, 55(3), 311–319. <https://doi.org/10.1111/j.1365-3059.2006.01359.x>
- Leroi, A. M., Bennett, A. F., & Lenski, R. E. (1994). Temperature acclimation and competitive fitness: An experimental test of the beneficial acclimation assumption. *Proceedings of the National Academy of Sciences of the United States of America*, 91(5), 1917–1921. <https://doi.org/10.1073/pnas.91.5.1917>
- Levins, R. (1969). Thermal acclimation and heat resistance in *Drosophila* species. *The American Naturalist*, 103(933), 483–499. <https://doi.org/10.1086/282616>
- Lozoya-Saldaña, H. (2011). Evolution of vertical and horizontal resistance and its application in breeding resistance to potato late blight. *Potato Journal*, 38(1), 1–8.
- Madden, L. V., Hughes, G., & Bosch, F. V. D. (2007). The study of plant disease epidemics. *Study of Plant Disease Epidemics*, 44(7), 2065b–2065. <https://doi.org/10.21273/HORTSCI.44.7.2065b>
- Mariette, N., Androdias, A., Mabon, R., Corbiere, R., Marquer, B., Montarry, J., & Andrivon, D. (2016). Local adaptation to temperature in populations and clonal lineages of the Irish potato famine pathogen *Phytophthora infestans*. *Ecology and Evolution*, 6(17), 6320–6331. <https://doi.org/10.1002/ece3.2282>
- Mboup, M., Bahri, B., Leconte, M., Vallavieille-Pope, C. D., Kaltz, O., & Enjalbert, J. (2012). Genetic structure and local adaptation of European wheat yellow rust populations: The role of temperature-specific adaptation. *Evolutionary Applications*, 5(4), 341–352. <https://doi.org/10.1111/j.1752-4571.2011.00228.x>
- Molnár, I. K., Sckrabulis, J. P., Altman, K. A., & Raffel, T. R. (2017). Thermal performance curves and the metabolic theory of ecology—a practical guide to models and experiments for parasitologists. *Journal of Parasitology*, 103(5), 423–439. <https://doi.org/10.1645/16-148>
- Myers, S. S., Smith, M. R., Guth, S., Golden, C. D., Vaitla, B., Mueller, N. D., ... Huybers, P. (2017). Climate change and global food systems: Potential impacts on food security and undernutrition. *Annual Review of Public Health*, 38(1), 259–277. <https://doi.org/10.1146/annurev-publhealth-031816-044356>
- Narum, S. R., Campbell, N. R., Meyer, K. A., Miller, M. R., & Hardy, R. W. (2013). Thermal adaptation and acclimation of ectotherms from differing aquatic climates. *Molecular Ecology*, 22(11), 3090–3097. <https://doi.org/10.1111/mec.12240>
- Padfield, D., Buckling, A., Warfield, R., Lowe, C., & Yvon-Durocher, G. (2018). Linking phytoplankton community metabolism to the individual size distribution. *Ecology Letters*, 21(8), 1152–1161. <https://doi.org/10.1111/ele.13082>
- Paull, S. H., Raffel, T. R., LaFonte, B. E., & Johnson, P. T. J. (2015). How temperature shifts affect parasite production: Testing the roles of thermal stress and acclimation. *Functional Ecology*, 29(7), 941–950. <https://doi.org/10.1111/1365-2435.12401>

- Qin, C.-F., He, M.-H., Chen, F.-P., Zhu, W., Yang, L.-N., Wu, E.-J., ... Zhan, J. (2016). Comparative analyses of fungicide sensitivity and SSR marker variations indicate a low risk of developing azoxystrobin resistance in *Phytophthora infestans*. *Scientific Reports*, 6, 20483. <https://doi.org/10.1038/srep20483>
- Rietman, H., Bijsterbosch, G., Cano, L. M., Lee, H.-R., Vossen, J. H., Jacobsen, E., ... Vleeshouwers, V. G. A. A. (2012). Qualitative and quantitative late blight resistance in the potato cultivar Sarpo Mira is determined by the perception of five distinct RXLR effectors. *Molecular Plant-Microbe Interactions*, 25(7), 910–919. <https://doi.org/10.1094/mpmi-01-12-0010-r>
- Rundle, H. D., & Nosil, P. (2005). Ecological speciation. *Ecology Letters*, 8(3), 336–352. <https://doi.org/10.1111/j.1461-0248.2004.00715.x>
- Runno-Paurson, E., Hannukkala, A. O., Kotkas, K., Koppel, M., Williams, I. H., & Mänd, M. (2013). Impact of phytosanitary quality of seed potato and temporal epidemic progress on the phenotypic diversity of *Phytophthora infestans* populations. *American Journal of Potato Research*, 90(3), 245–254. <https://doi.org/10.1007/s12230-013-9299-y>
- Rutherford, S., D'Hondt, S., & Prell, W. (1999). Environmental controls on the geographic distribution of zooplankton diversity. *Nature*, 400(6746), 749. <https://doi.org/10.1038/23449>
- SAS Institute (2013). *Base SAS® 9.4 procedures guide: Statistical procedures* (2nd ed.). Cary, NC: SAS Institute.
- Schaum, C.-E., Barton, S., Bestion, E., Buckling, A., Garcia-Carreras, B., Lopez, P., ... Yvon-Durocher, G. (2017). Adaptation of phytoplankton to a decade of experimental warming linked to increased photosynthesis. *Nature Ecology & Evolution*, 1(4), 0094. <https://doi.org/10.1038/s41559-017-0094>
- Seebacher, F., Ducret, V., Little, A. G., & Adriaenssens, B. (2015). Generalist-specialist trade-off during thermal acclimation. *Royal Society Open Science*, 2(1), 140251. <https://doi.org/10.1098/rsos.140251>
- Somero, G. (2010). The physiology of climate change: How potentials for acclimatization and genetic adaptation will determine 'winners' and 'losers'. *Journal of Experimental Biology*, 213(6), 912–920. <https://doi.org/10.1242/jeb.037473>
- Sparks, A. H., Forbes, G. A., Hijmans, R. J., & Garrett, K. A. (2014). Climate change may have limited effect on global risk of potato late blight. *Global Change Biology*, 20(12), 3621–3631. <https://doi.org/10.1111/gcb.12587>
- Sujkowski, L. S. (1987). The influence of temperature on *Phytophthora infestans* (Mont.) de Bary. *Journal of Phytopathology*, 120(3), 271–275. <https://doi.org/10.1111/j.1439-0434.1987.tb04441>
- Urban Mark, C., Tewksbury Josh, J., & Sheldon Kimberly, S. (2012). On a collision course: Competition and dispersal differences create no-analogue communities and cause extinctions during climate change. *Proceedings of the Royal Society B: Biological Sciences*, 279(1735), 2072–2080. <https://doi.org/10.1098/rspb.2011.2367>
- Vetukuri, R. R., Åsman, A. K. M., Tellgren-Roth, C., Jahan, S. N., Reimegård, J., Fogelqvist, J., ... Dixelius, C. (2012). Evidence for small RNAs homologous to effector-encoding genes and transposable elements in the oomycete *Phytophthora infestans*. *PLoS ONE*, 7(12), e51399. <https://doi.org/10.1371/journal.pone.0051399>
- Walters, R. J., & Berger, D. (2019). Implications of existing local (mal) adaptations for ecological forecasting under environmental change. *Evolutionary Applications*, 12(7), 1487–1502. <https://doi.org/10.5061/dryad.h7nj6qn>
- Willmott, A. G., Hayes, M., James, C. A., Dekerle, J., Gibson, O. R., & Maxwell, N. S. (2018). Once- and twice-daily heat acclimation confer similar heat adaptations, inflammatory responses and exercise tolerance improvements. *Physiological Reports*, 6(24), e13936. <https://doi.org/10.14814/phy2.13936>
- Wos, G., & Willi, Y. (2018). Thermal acclimation in *Arabidopsis lyrata*: Genotypic costs and transcriptional changes. *Journal of Evolutionary Biology*, 31(1), 123–135. <https://doi.org/10.1111/jeb.13208>
- Wu, E. J., Wang, Y. P., Shen, L. L., Yahuza, L., Tian, J. C., Yang, L. N., ... Zhan, J. (2019). Strategies of *Phytophthora infestans* adaptation to local UV radiation conditions. *Evolutionary Applications*, 12(3), 415–424. <https://doi.org/10.1111/eva.12722>
- Wu, E.-J., Wang, Y.-P., Yahuza, L., He, M.-H., Sun, D.-L., Huang, Y.-M., ... Zhan, J. (2019). Data from: Rapid adaptation of the Irish potato famine pathogen *Phytophthora infestans* to changing temperature, v2. *Dryad Dataset*, <https://doi.org/10.5061/dryad.z08kpr8h>
- Yang, L., Ouyang, H. B., Fang, Z. G., Zhu, W., Wu, E. J., Luo, G. H., ... Zhan, J. (2018). Evidence for intragenic recombination and selective sweep in an effector gene of *Phytophthora infestans*. *Evolutionary Applications*, 11(8), 1342–1353. <https://doi.org/10.1111/eva.12629>
- Yang, L.-N., Pan, Z.-C., Zhu, W., Wu, E.-J., He, D.-C., Yuan, X., ... Zhan, J. (2019). Enhanced agricultural sustainability through within-species diversification. *Nature Sustainability*, 2(1), 46–52. <https://doi.org/10.1038/s41893-018-0201-2>
- Yang, L. N., Zhu, W., Wu, E. J., Yang, C., Thrall, P. H., Burdon, J. J., ... Zhan, J. (2016). Trade-offs and evolution of thermal adaptation in the Irish potato famine pathogen *Phytophthora infestans*. *Molecular Ecology*, 25(16), 4047–4058. <https://doi.org/10.1111/mec.13727>
- Yvon-Durocher, G., Allen, A. P., Cellamare, M., Dossena, M., Gaston, K. J., Leitao, M., ... Trimmer, M. (2015). Five years of experimental warming increases the biodiversity and productivity of phytoplankton. *PLoS Biology*, 13(12), e1002324. <https://doi.org/10.1371/journal.pbio.1002324>
- Zhan, J., Ericson, L., & Burdon, J. J. (2018). Climate change accelerates local disease extinction rates in a long-term wild host-pathogen association. *Global Change Biology*, 24(8), 3526–3536. <https://doi.org/10.1111/gcb.14111>
- Zhan, J., Linde, C. C., Jürgens, T., Merz, U., Steinebrunner, F., & McDonald, B. A. (2005). Variation for neutral markers is correlated with variation for quantitative traits in the plant pathogenic fungus *Mycosphaerella graminicola*. *Molecular Ecology*, 14(9), 2683–2693. <https://doi.org/10.1111/j.1365-294X.2005.02638.x>
- Zhan, J., & McDonald, B. A. (2011). Thermal adaptation in the fungal pathogen *Mycosphaerella graminicola*. *Molecular Ecology*, 20(8), 1689–1701. <https://doi.org/10.1111/j.1365-294X.2011.05023.x>
- Zhan, J., Thrall, P. H., & Burdon, J. J. (2014). Achieving sustainable plant disease management through evolutionary principles. *Trends in Plant Science*, 19(9), 570–575. <https://doi.org/10.1016/j.tplants.2014.04.010>
- Zhan, J., Thrall, P. H., Papaix, J., Xie, L., & Burdon, J. J. (2015). Playing on a pathogen's weakness: Using evolution to guide sustainable plant disease control strategies. *Annual Review of Phytopathology*, 53, 19–43. <https://doi.org/10.1146/annurev-phyto-080614-120040>
- Zhang, L. J., Jing, Y. P., Li, X. H., Li, C. W., Bourguet, D., & Wu, G. (2015). Temperature-sensitive fitness cost of insecticide resistance in Chinese populations of the diamondback moth *Plutella xylostella*. *Molecular Ecology*, 24(7), 1611–1627. <https://doi.org/10.1111/mec.13133>
- Zhu, W., Shen, L. L., Fang, Z. G., Yang, L. N., Zhang, J. F., Sun, D. L., & Zhan, J. (2016). Increased frequency of self-fertile isolates in *Phytophthora infestans* may attribute to their higher fitness relative to the A1 isolates. *Scientific Reports*, 6, 29428. <https://doi.org/10.1038/srep29428>

## SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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