



HAL
open science

High-resolution disease phenotyping reveals distinct resistance strategies of wild tomato crop wild relatives against *Sclerotinia sclerotiorum*

Severin Einspanier, Christopher Tominello-Ramirez, Mario Hasler, Adelin Barbacci, Sylvain Raffaele, Remco Stam

► To cite this version:

Severin Einspanier, Christopher Tominello-Ramirez, Mario Hasler, Adelin Barbacci, Sylvain Raffaele, et al.. High-resolution disease phenotyping reveals distinct resistance strategies of wild tomato crop wild relatives against *Sclerotinia sclerotiorum*. 2024. hal-04715349

HAL Id: hal-04715349

<https://hal.inrae.fr/hal-04715349v1>

Preprint submitted on 30 Sep 2024

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.



Distributed under a Creative Commons Attribution - NonCommercial 4.0 International License

1 **High-resolution disease phenotyping reveals distinct resistance**
2 **strategies of wild tomato crop wild relatives against *Sclerotinia***
3 ***sclerotiorum***

4 Severin Einspanier¹, Christopher Tominello-Ramirez¹, Mario Hasler², Adelin Barbacci³, Sylvain
5 Raffaele³, Remco Stam^{1*}

6
7 ¹ Department of Phytopathology and Crop Protection, Institute of Phytopathology, Faculty
8 of Agricultural and Nutritional Sciences, Christian Albrechts University, Kiel, Germany

9 ² Lehrfach Variationsstatistik, Faculty of Agricultural and Nutritional Sciences, Christian-
10 Albrechts-University of Kiel, Hermann-Rodewald-Straße 9, 24118 Kiel, Germany

11 ³ Laboratoire des Interactions Plantes Microorganismes Environnement (LIPME), INRAE,
12 CNRS, Castanet Tolosan Cedex, France.

13 * Address correspondence to: Remco.Stam@phytomed.uni-kiel.de

14

1 **Abstract**

2 Besides the well-understood qualitative disease resistance, plants possess a more complex
3 quantitative form of resistance: quantitative disease resistance (QDR). QDR is commonly defined
4 as a partial but more durable form of resistance and, therefore, might display a valuable target for
5 resistance breeding. The characterization of QDR phenotypes, especially of wild crop relatives,
6 displays a major bottleneck in deciphering QDR's genomic and regulatory background. Moreover,
7 the relationship between QDR parameters, such as infection frequency, lag phase duration, and
8 lesion growth rate, remains elusive. High hurdles for applying modern phenotyping technology,
9 such as the low availability of phenotyping facilities or complex data analysis, further dampen
10 progress in understanding QDR. Here, we applied a low-cost phenotyping system to measure lesion
11 growth dynamics of wild tomato species (e.g., *S. pennellii* or *S. pimpinellifolium*). We provide
12 insight into QDR diversity of wild populations and derive specific QDR strategies and their
13 crosstalk. We show how temporally continuous observations are required to dissect end-point
14 severity into functional resistance strategies. The results of our study show how QDR can be
15 maintained by facilitating different defense strategies during host-parasite interaction and that the
16 capacity of the QDR toolbox highly depends on the host's genetic context. We anticipate that the
17 present findings display a valuable resource for more targeted functional characterization of the
18 processes involved in QDR. Moreover, we show how modest phenotyping technology can be
19 leveraged to help answer highly relevant biological questions.

20

1. Introduction

Quantitative disease resistance in plants

Plant resistance is commonly divided into two concepts with fundamental differences: qualitative and quantitative resistance [1,2]. While qualitative disease resistance provides a highly effective race-specific resistance, quantitative disease resistance (QDR) is a broad-range yet incomplete resistance [2,3]. Qualitative resistance is driven by major race-specific resistance genes (R-genes). They often lead to complete and easily observable resistance and were the dominant research focus for disease resistance breeding programs. However, reports of R-genes losing their efficacy against pathogens have increased recently, and major resistance genes have not been identified for many so-called necrotrophic plant pathogens, like *Botrytis cinerea* or *Sclerotinia sclerotiorum* [3–7]. Commonly, degrees of QDR can't be divided into discrete classes. Quantitative resistance phenotypes are continuously distributed and can only be explained by highly integrated, polygenic regulatory mechanisms [8]. Moreover, QDR can manifest itself in several ways, ranging from differences in infection frequency on the leaf or delayed onset of infection to stalled lesion growth. Numerous studies documented wide distributions of QDR phenotypes against necrotrophic pathogens in both natural and domesticated plant populations, yet the relations of different QDR phenotypes have not yet been studied in detail [1–3,8–12]. Recent reports summarized the diversity in functional QDR, arguing that QDR might be influenced by many independent components such as regulation as a pleiotropic side-effect, weak R-genes, involvement in defense signal transduction, or *cis/trans*-regulatory mechanisms [1,2]. Indeed, many QTLs that influence some degree of QDR have been identified [8,13,14]. Linkage of such QTLs or the underlying loci to exact resistance features, like the lag-phase duration, will be one of the future challenges that would allow understanding and utilizing QDR in pathogen resistance breeding.

24

1 **Phenotyping technology and approaches to quantify QDR**

2 The functional characterization of QDR highly depends on precisely measured phenotypes [2,15].
3 However, the experimental design required to assess QDR phenotypes over entire plant or pathogen
4 populations quickly exceeds the limits of traditional, manual scoring methods and calls for more
5 sophisticated phenotyping technology. The increasing availability of sensor technology (e.g., RGB,
6 multi- or hyperspectral sensors) and analytical methods (e.g., deep-learning or artificial intelligence
7 algorithms) recently have strengthened the attention to plant phenotyping [16]. Many studies have
8 shown how imaging technology can be used to determine plant phenotypes like plant height,
9 nutritional status, or water-use efficiency but also to assist breeder's decisions [14,17,18].
10 Moreover, several reviews recently summarized the potential of modern sensor technology and
11 related software in quantifying phenotypes of host-parasite interactions on multiple levels [19–24].
12 Even advanced applications, like in-field phenotyping or assessing complex features in non-
13 standardized conditions, are possible due to deep-learning models like 'PLPNet' or 'ResNet-9'
14 [25–27]. However, large phenotyping platforms also have limitations. High-end systems often
15 collect a multitude of 3D scanning images or images in multiple spectral wavelengths. Analysis of
16 these data is computationally intensive and often requires very specific knowledge. Thus, such
17 technologies might overwhelm (non-data-science-) researchers with high amounts of complex
18 datasets as significant skills are required to derive easy-to-interpret insights relevant to answering
19 biological research questions [28]. A second challenge lies in adapting an established phenotyping
20 system for various pathosystems, i.e., different crops or pathogens [22,29]. Lastly, most high-end
21 phenotyping systems have very high investment and running costs and thus are less available.
22 Combined with the aforementioned low flexibility, this further limits their use and application in
23 the broad spectrum of plant pathology, where quick and easy screening of QDR in a large panel of
24 plants is one of the main objectives. Recent developments, however, enable researchers to use the
25 generally available consumer-level technology and build low-cost phenotyping platforms like the
26 'Navautron' [30]. In this study, we show the usefulness of such systems in unraveling QDR
27 dynamics in crop wild relatives.

28

29 **Wild tomato populations as a reservoir of potential QDR loci against major pathogens**

30 The domestic tomato (*Solanum lycopersicum*) is a major food crop of global importance [31].
31 However, plant pathogens, including the necrotroph *Sclerotinia sclerotiorum* or species from the

1 genus *Alternaria*, commonly threaten tomato production worldwide [32–35]. Host resistance and
2 fungicides are the standard tools to protect tomatoes against these pathogens. However, strong
3 bottleneck events caused by R-genes or fungicides and higher-than-expected pathogen diversity in
4 the field result in losing fungicide efficacy or plant resistance against such species [36–40].
5 Therefore, highly diverse wild populations are an invaluable source of desirable alleles in breeding,
6 as crosses between wild and domestic lead to increased performance and stress tolerance [41].
7 Integrating phenotyping with screening of genetically highly diverse wild resources will help
8 characterize novel alleles for QDR breeding [42].

9 Wild tomato species originated from several radiation events and can generally be classified into
10 four groups within the so-called section *Lycopersicon*, containing a total of 15 species and two
11 species in the section *Lycopersicoides* [43]. All species have adapted to specific habitats ranging
12 from the edge of the Atacama desert to the Andes, where they withstand diverse (a)biotic stresses.
13 Evolutionary analyses show that different species and populations have evolved drought or salt
14 stress tolerance, as well as adaptation to cold stress [44–48]. Previous studies have also shown
15 substantial variation in susceptibility and resistance of wild *Solanum* spp. against various pathogens
16 but often relied on manual or single time-point disease assessments, thus lacking the temporal
17 resolution and statistical power to describe QDR strategies confidently [38,49,50]. In light of the
18 variation of QDR already shown, wild tomato species are perfectly suited for quantification of
19 QDR mechanisms as proof of principle. Moreover, defining whether specific QDR mechanisms
20 play major roles in resistance will generate much-needed insights into the biology of QDR to help
21 design future durable resistance breeding projects against major pathogens.

22 *Sclerotinia sclerotiorum* is a necrotrophic pathogen that can infect hundreds of host species,
23 including important crops such as rape seed and tomato [30,51,52]. On vegetables, including
24 tomatoes, infection with *S. sclerotiorum* can cause tremendous yield loss due to collapsing stems
25 or damaged fruits [53,54]. Infection in the field can happen through air-dispersed ascospores or via
26 myceliogenic germination of its overwintering structures in the soil, the so-called sclerotia [32,52].
27 In experimental conditions, mycelial inoculation procedures are commonly used, as the preparation
28 of ascospores can display a major challenge [55–59]. No complete form of resistance against the
29 generalist *S. sclerotiorum* has been characterized; therefore, resistance breeding relies on QDR as
30 the source of new alleles [32,52,55,57,60].

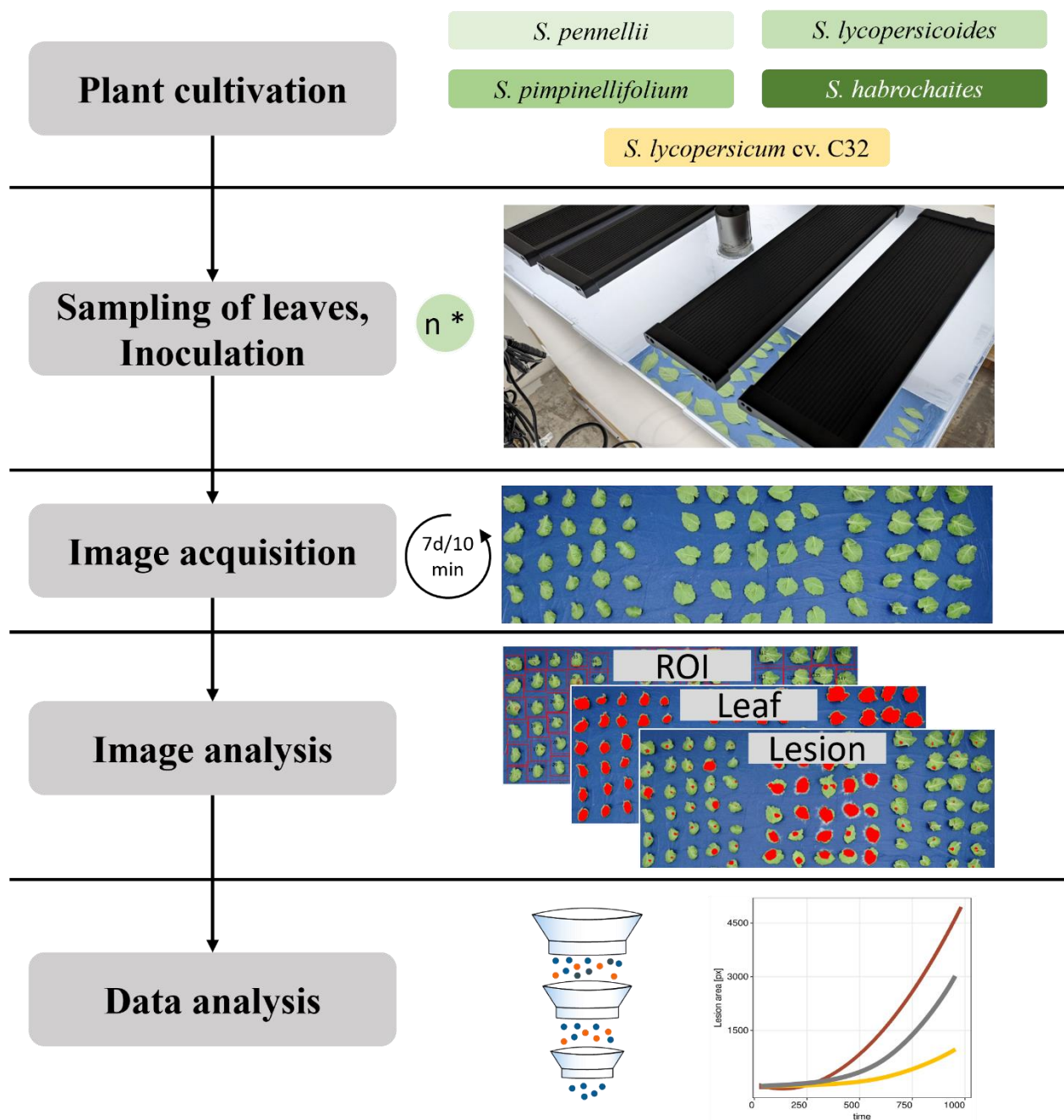
1 In the present work, we build on a low-budget image-based phenotyping system [30] to derive
2 high-resolution time-resolved disease phenotypes and dissect them into three distinct QDR
3 strategies. We show the potential of this system by characterizing the natural diversity of QDR
4 phenotypes of wild *Solanum* species and, therefore, provide insights into the mechanisms
5 underlying QDR against the generalist pathogen *Sclerotinia sclerotiorum*. We use this system as a
6 model to address whether QDR is always represented by a similar mechanism, i.e., infection
7 frequency or lag phase duration, and show that the orchestration of different QDR mechanisms
8 affects the overall QDR on a genotype-specific basis. Accordingly, we argue that the different host
9 species have evolved specific strategies to maintain a defined degree of QDR.

10

1 2. Materials and Methods

2 Experimental Design

3 We screened multiple accessions of four wild tomato species (*S. pennellii*, *S. lycopersicoides*, *S.*
4 *habrochaites*, and *S. lycopersicoides*) with a detached-leaf assay. All accessions of the same species



5 Figure 1: Overview of the high-throughput phenotyping assay.

1 were tested as one batch for up to five independent repetitions. To facilitate comparability between
2 batches, *S. lycopersicum* cv. C32 was used as a control in every experiment. A schematic of the
3 experimental procedures is displayed in fig. 1.

4 ***Sclerotinia sclerotiorum* inoculum preparation**

5 For inoculation experiments, the *Sclerotinia sclerotiorum* isolate 1980 or the OAH1:GFP isolate
6 (for microscopical analysis only, [61]) was used. The fungus was alternatingly cultivated on potato
7 dextrose agar (Sigma Aldrich) and solid malic acid medium [62] at approx. 25°C in the dark. Four
8 1cm pieces of *S. sclerotiorum* inoculum were used to inoculate 100 mL PDB. After four days of
9 incubation on a rotary shaker (24°C, 120rpm), a fungal mycelium suspension was generated: for
10 this, the medium was mixed using a dispenser (IKA T25) for two times 10 sec at 24.000 rpm. The
11 mixture was then vacuum-filtrated through cheesecloth, and the remaining liquid was concentrated
12 to an OD of 1. For the negative control, fungal tissue was removed from the solution by
13 centrifugation, and the supernatant was autoclaved. Tween20 was used as a surfactant. Per leaf,
14 one drop (10 µL) of inoculum was used.

15 **Plant growing conditions**

16 Wild tomato germplasm was obtained from the C. M. Rick Tomato Genetics Resource Center of
17 the University of California, Davis (TGRC UC-Davis, <http://tgrc.ucdavis.edu/>) (see suppl. table
18 5). The species were selected to include genetically diverse species within the section *Lycopersicon*
19 and a species from the section *Lycopersicoides* (fig. 2). All plants were grown at the greenhouse
20 facility of the Department of Phytopathology and Crop Protection, Institute of Phytopathology,
21 Faculty of Agricultural and Nutritional Sciences, Christian Albrechts University, Kiel, Germany.
22 Following seed surface sterilization using 2.75% hypochlorite (15 min. incubation followed by
23 washing twice with dH₂O), seeds were sown in the substrate (STENDER C700, Germany) and
24 cultivated in a growth chamber (21 °C, 65% rH, 450 PAR). From the 3-leaf stage on, plants were
25 cultivated in standard greenhouse conditions with supplement light. Plants were occasionally
26 fertilized via the irrigation system (1% Sagaphos Blue, Germany). Plants were propagated using
27 cuttings (Chryzotop Grün 0.25%) and regularly screened for virus infection.

1 Detached leaf assay

2 Detached leaf assays were conducted to
3 measure quantitative disease resistance of a
4 diverse panel of wild Solanaceae plants. A
5 custom phenotyping system was adapted
6 [30]. A 50cm x 70cm PMMA tray was filled
7 with eight layers of blue tissue paper and
8 flooded with 700mL sterile dH₂O. Plant
9 leaves were placed abaxial side up onto the
10 tissue and inoculated with 10 μL of mock-/*S.*
11 *sclerotiorum*-suspension. Next, the tray is
12 covered with a custom hood. The boxes were
13 placed inside a growth chamber (24°C) and
14 incubated for seven days. The assay was
15 independently repeated five times. We used
16 a representative set of three experiments for
17 all further analysis.

18

19 Phenotyping platform

20 High-resolution images were acquired using RGB cameras (Yealink UVC30) mounted on the box.
21 Cameras were controlled using Raspberry Pi microcomputers or desktop PCs running headless
22 Ubuntu22. A cron daemon launched the image-acquisition script every ten minutes. Plant lights
23 also briefly illuminate during nighttime for image capture to enable images in the dark while
24 maintaining circadian rhythm. This was achieved by using the ‘Shelly Plus Plug S’ wifi plug.

25 Image analysis

26 We adapted the ‘navautron’ software package (<https://github.com/A02101/Navautron>). The image
27 analysis involved manually defining regions of interest (ROI) using ImageJ (ImageJ Version
28 1.530). Further, HSV thresholds were optimized individually per box. For this, ‘assess_noChl.py’
29 was used, and an overlay was generated in Gimp (Version 2.10). Once binary masks represented

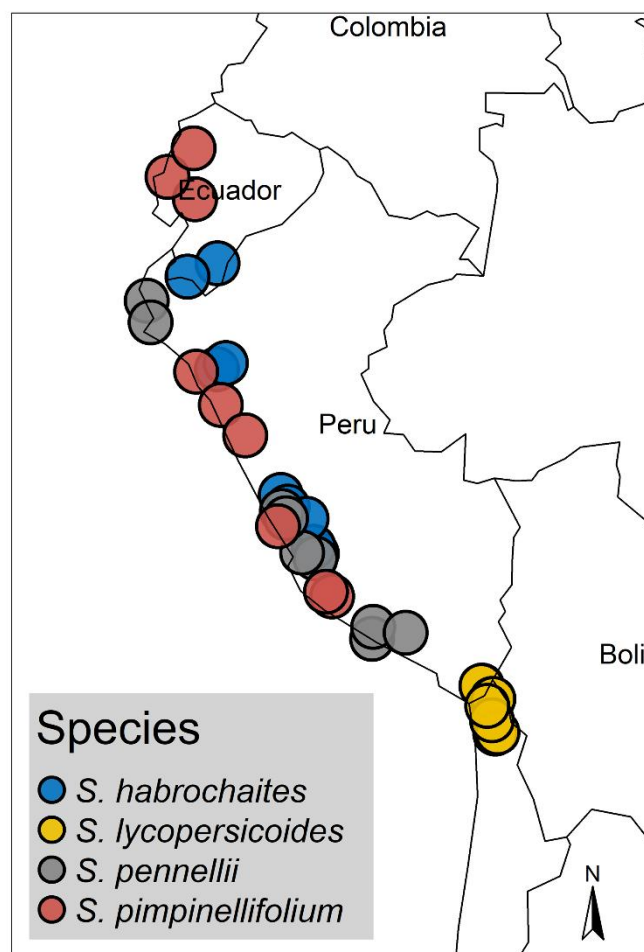


Figure 2: **Sampling localities of wild tomato accessions used in this study.** Seed material of all wild tomato accessions was provided from C. M. Rick Tomato Genetics Resource Center of the University of California, Davis (TGRC UC-Davis, <http://tgrc.ucdavis.edu/>). Individual dots represent the geographical origin of each accession.

1 the respective feature classes (leaf_healthy, leaf_diseased, background), the whole dataset was
2 evaluated using the ‘infest.py’ script. Segmentation was iterated and classified pixel counted. The
3 analysis includes functions from the python3 (Version 3.11.4) libraries ‘numpy’ (Version 1.25.2),
4 ‘opencv’ (Version 4.8.1.78), ‘plantcv’ (Version 4.0.1), and ‘scikit-image’ (Version 0.22.0). The
5 plantcv function ‘dilate’ was used to remove leaf edges containing shadows with ksize=9, i=1 [63].
6 To improve thresholding accuracy (e.g., filling holes) on the lesion, an index filter was applied
7 [ndimage.generic_filter(mask, threshold, size=3, mode=‘constant’)] with a condition to overwrite
8 pixels deviating from the value of the majority of the surrounding pixels. np.sum(mask) was used
9 to quantify the number of pixels in each feature class (lesion and leaf). Code and scripts can be
10 found at https://github.com/seveein/QDR_Wild_Tomatoes.

11 **Microscopy analysis**

12 Plant leaves were harvested and inoculated under standard conditions as described before but with
13 either a GFP-expressing *S. sclerotiorum* strain, the *S. sclerotiorum* wildtype 1980, or the mock
14 suspension. The leaves were evaluated at 12-hour intervals using a Zeiss Discovery V20
15 stereomicroscope under bright light and fluorescent illumination (Zeiss HXP120). Images were
16 taken using an AxioCam MRc camera.

17 **Statistical analysis**

18 An interactive R-script (R-Version 4.3.2, R-Studio 2023.12.1+402) was facilitated to extract lag-
19 phase duration and LDT to quantify resistance characteristics [30]. Each leaf’s lesion size over time
20 was fitted against a 4-degree polynomial regression. The fit to the measured data point was
21 reviewed for each sample. Lesion doubling time (LDT) and lag phase were determined based on a
22 segmented regression analysis, expecting two linear phases. First, a linear phase during the lag
23 period (no symptom development), and second, a linear log growth (symptom) during the
24 exponential growth phase. The start of exp(LDT) is considered the lag phase, while the LDT
25 represents the log(slope) of the linear growing curve in this area.

26 A two-tier filtering pipeline was developed to increase accuracy and remove artifacts from the
27 dataset. First, single time points with an arbitrary high/low leaf area were filtered per leaf. The top
28 and lowest 2.5 % of the data points were trimmed for this. Next, individual leaves with
29 unexpectedly high variability in leaf area were excluded from the data set. Therefore, samples with

1 sd(leaf) > 10% of the mean(leaf) were removed from the dataset using a simple tidyverse (v. 2.0.0)
2 pipeline.

3 As a measure of symptom development over time, the area under the disease progress curve
4 (AUDPC) was calculated using the R-package agricolae (v. 1.3-6). General statistical analysis and
5 visualization were conducted in RStudio (R-Version 4.3.2, R-Studio 2023.12.1+402 [64]), and the
6 packages tidyverse [65], ggplot2 [66], ggpubr [67] and agricolae [68]. AUDPC is defined with
7 i =time and y_i =symptom severity at time= i as: [68]

$$8 \quad AUDPC = \sum_{i=1}^N \frac{(y_i + y_{i+1}) \times (i - 1)}{2}$$

9 For continuous variables (lag phase duration, lesion doubling time, AUDPC, tt100), a statistical
10 model based on a generalized least squares model was defined [69]. In contrast, a generalized linear
11 model was defined for binomial values (infection frequency, 100%/f) [70]. These models included
12 genotype and start date (without interaction effect).

13 The residuals corresponding to the continuous values were assumed to be approximately normally
14 distributed and heteroscedastic concerning the different genotypes. These assumptions are based
15 on a graphical residual analysis (suppl. fig. 7 & 8). Based on these models, a Pseudo R^2 was
16 calculated [71], and an analysis of variances (ANOVA) was conducted, followed by multiple
17 contrast tests [72,73]. User-defined contrast matrices were used i) to compare the species' means
18 with each other and ii) to compare the population means within their specific species with the
19 corresponding species' mean. The individual leaf area was previously found to have no significant
20 influence on lesion area; therefore, it was not included in our statistical model [74]. A linear mixed-
21 effects model was used to determine the relationship between AUDPC and predictors such as
22 genotype, lag phase duration, and LDT. Random intercepts were specified per start date to account
23 for experimental repetitions.

24 Based on this model, fixed effect values were extracted and used to predict AUDPC per
25 genotype _{$i=1,2,3$} in relationship to varying lag and LDT values.

$$26 \quad AUDPC_i = Intercept_i + Coefficient_{lag_i} \times lag + Coefficient_{LDT_i} \times LDT \\ 27 \quad + Coefficient_{lag_i \times LDT_i} \times lag \times LDT$$

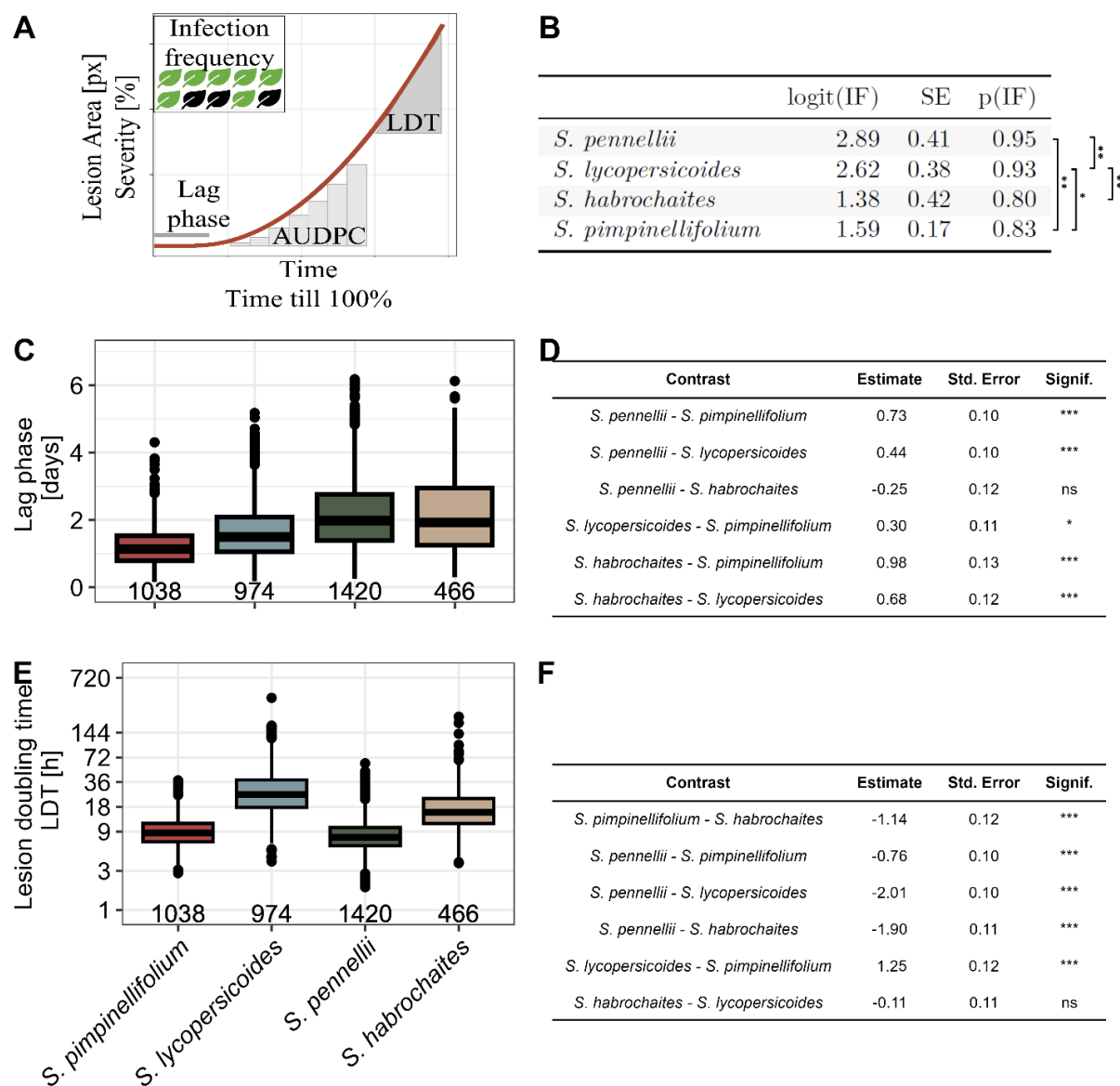
1 The associated R-codes can be found at https://github.com/seveein/QDR_Wild_Tomatoes.

2

1 **3. Results**

2 **Wild tomato species carry different levels of quantitative resistance against *Sclerotinia*** 3 ***sclerotiorum* depending on defense-parameters**

4 We investigated the phenotypic diversity in quantitative disease resistance in four wild tomato
5 species (*S. habrochaites*, *S. lycopersicoides*, *S. pennellii*, and *S. pimpinellifolium*) against the
6 *Sclerotinia sclerotiorum* isolate 1980 [13]. We used the “Navautron” automated phenotyping
7 system for continuous image acquisition and applied a threshold-based segmentation algorithm to
8 extract phenotypic data. Hence, we calculated different QDR parameters such as infection
9 frequency, lag-phase duration, lesion doubling time (LDT), or area under the disease progress curve
10 (AUDPC) to quantify temporal dynamics of infection (fig. 3). High variability between
11 experimental runs with wild tomatoes has been described before [6,49,74]. To account for this, we
12 applied a generalized least squares model (gls, continuous variables) and a generalized linear model
13 (glm, discrete variables) for statistical analysis [69]. Overall, we discovered a great diversity of
14 resistance phenotypes among the tested plant species. We found no 100% resistant accessions
15 (suppl. fig. 3). We observed a significant difference in lag-phase duration among plant species,
16 which we define as the time from infection until the first symptoms appear (see fig. 3 A, C, D). For
17 instance, *S. pimpinellifolium* showed the shortest time from inoculation until lesion development
18 (adjusted mean = 36.2 hrs). In contrast, *S. habrochaites* and *S. pennellii* displayed a significantly
19 prolonged lag phase (both approx. 59 hours) (see suppl. table 1). Using segmented regression
20 analysis, we determined the speed of lesion growth on individual leaves of the panel. The fastest-
21 growing lesions were found on the species *S. pimpinellifolium* and *S. pennellii*. Lesions on *S.*
22 *pennellii* and *S. pimpinellifolium* leaves doubled in size within approx. eleven hours (6.56 log(LDT)
23 and 6.55 log(LDT), respectively), while lesions on *S. habrochaites* and *S. lycopersicoides* spread
24 significantly slower. Those lesions expanded with an average rate of approximately 7.7 log(LDT),
25 corresponding to roughly 36 (*S. habrochaites*) and 41 hours (*S. lycopersicoides*)(see suppl. table
26 2). Moreover, we observed that the success of disease establishment (infection frequency) depends
27 highly on the host species. We identified a significantly lower infection rate on *S. habrochaites*
28



1 **Figure 3: Wild tomato species possess a broad diversity of resistance against *S. sclerotiorum*.** A) Exemplary
2 illustration of different QDR parameters used in this study. The infection frequency is defined as the percentage of
3 leaves showing a lesion after seven days of incubation. The lag phase, or lag-phase, is defined as the time till first
4 visual symptoms appear. We used a segmented regression analysis to determine the lag phase's end mathematically.
5 The absolute lesion size is represented as pixel counts, whereas normalization against leaf area results in symptom
6 severity. The area under the disease progress curve (AUDPC) is defined as the integral area under the severity curve,
7 which depicts the severity over time. As a measure of the lesion spread, the Lesion Doubling Time (LDT) describes
8 the time till a lesion doubles its size. The time till a lesion covers 100% of a leaf is described by tt100%. B) The
9 infection frequency of *S. sclerotiorum* inoculum differs significantly between the host species. The table shows a
10 meta-analysis of pooled accessions collected from three independent experiments. C) Time till lesion formation (in
11 days). The number on the x-axis indicates the count of individual leaves tested. D) Statistical analysis of pairwise
12 differences in lag phase duration between the tested wild tomato species. Values are displayed in days and derived
13 from a generalized least squares model. E) Lesion growth rate during the exponential growth phase hours, plotted on
14 a log scale. The number on the x-axis indicates the count of individual leaves tested. Raw values are plotted. F)
15 Statistical analysis of pairwise differences between the tested wild tomato species regarding lesion doubling time.
16 Values are displayed as log(LDT[h]). Levels of significance are displayed as *** P < 0.001, ** P < 0.01, * P < 0.05,
17 P < 0.1.

1 (corrected infection frequency estimate 80 %), whereas *S. lycopersicoides* and *S. pennellii*
2 displayed significantly higher infection frequency (~93 and 95%, respectively) (fig. 3 B).

3 **Individual QDR measures show different levels of intraspecific variation and conservation** 4 **on *S. pennellii* and *S. lycopersicoides* accessions.**

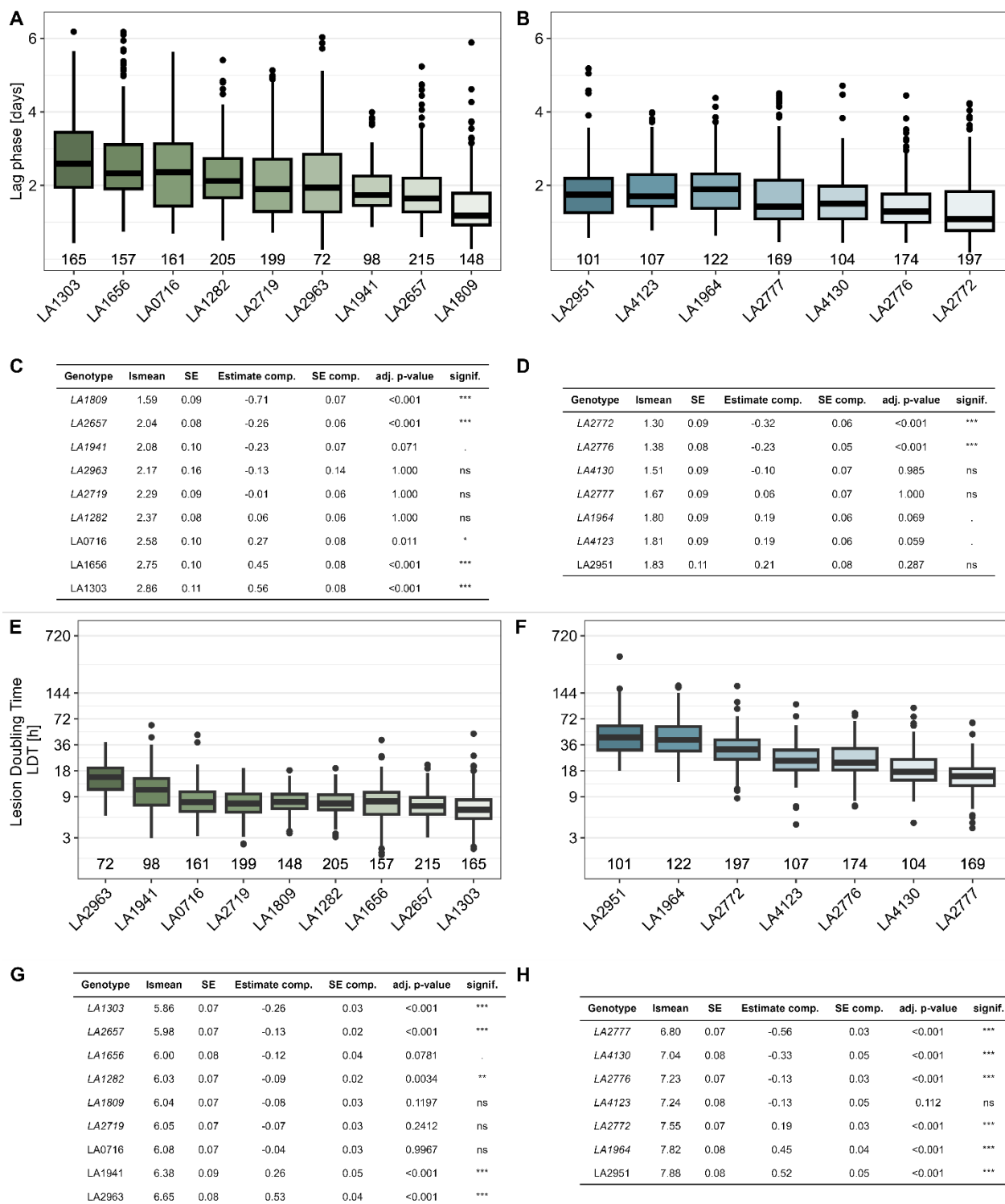
5 To assess the within-species diversity of QDR phenotypes, we tested different accessions of each
6 represented species. We collected phenotypic data from seven *S. lycopersicoides* and nine *S.*
7 *pennellii* populations (fig. 4), as well as eight populations of *S. habrochaites* and ten of the species
8 *S. pimpinellifolium* (see suppl. fig 1 and suppl. fig. 2). Especially the comparison of *S.*
9 *lycopersicoides* and *S. pennellii* highlights that QDR diversity differs between species. We
10 observed that the (adjusted) mean duration of the lag phase on different *S. pennellii* accessions
11 ranged from 1.59 days (38 hours, LA1809) to 2.86 days (68 hours, LA1303) (fig. 4 A, C). Using a
12 generalized least squares model, we identified accessions with a significantly shorter lag phase than
13 the grand mean of the species (LA1809 and LA2657). In contrast, the accessions LA1656 and
14 LA1303 displayed a significantly longer lag phase (2.75 days [66 h] and 2.86 days [68 h],
15 respectively) (fig. 4 A, C). Next, we observed a significantly shorter overall lag phase duration of
16 *S. lycopersicoides* accessions than *S. pennellii*. Accordingly, the first symptoms appeared after 1.3
17 days (31 hours, LA2772) and the latest at 1.83 days after inoculation (43 hours LA1966). The
18 overall time till initial symptom development was more conserved; only two *S. lycopersicoides*
19 accessions deviated significantly from the grand mean, being more susceptible than the overall
20 species level (LA2776 and LA2772) (fig. 4 B, D). Similarly, we found a lack of variation in lag-
21 phase-duration in the populations of *S. pimpinellifolium*. At the same time, *S. habrochaites*-
22 accessions displayed a wider variability of lag-phase phenotypes (suppl. fig. 1, suppl. table 3).

23 Next, we analyzed the variability of the lesion growth rate between accessions of each species
24 using the logarithmic lesion doubling time. We observed that all tested *S. pennellii* accessions
25 displayed an average lesion doubling time ranging from 5.84 h (LA1303) to 13.07 hours (LA2963).
26 Five accessions (LA1809, LA1282, LA2719, LA2657, LA1303) have a significantly faster lesion
27 development than the grand mean (LDT < 11 hours). The populations LA2963 and LA1941
28 displayed a significantly longer LDT (13.07 and 9.8 hours, respectively) (fig. 4 E, G). Generally,
29 we found that symptoms of *S. lycopersicoides* grew significantly slower (observed range: 14.9 h to
30 40 hours). However, we still observed a significant within-species variability. For instance,

1 symptoms on leaves of the accession LA2951 doubled within $ls_{mean}=7.88 \log(LDT)$ (approx. 44
2 hours), while lesions of LA2777 expanded much faster at $ls_{mean}=6.8 \log(LDT)$ (15 hours, fig. 4
3 F, H). We observed a high variability among the accessions for *S. pennellii* and *S. lycopersicoides*,
4 mostly deviating from the species-mean in LDT with high significance. Interestingly, the LDT on
5 *S. habrochaites* and *S. pimpinellifolium* appeared much more conserved between the accessions, as
6 only a few samples significantly differed from the grand mean (suppl. fig. 2, suppl. table 4).

7

8



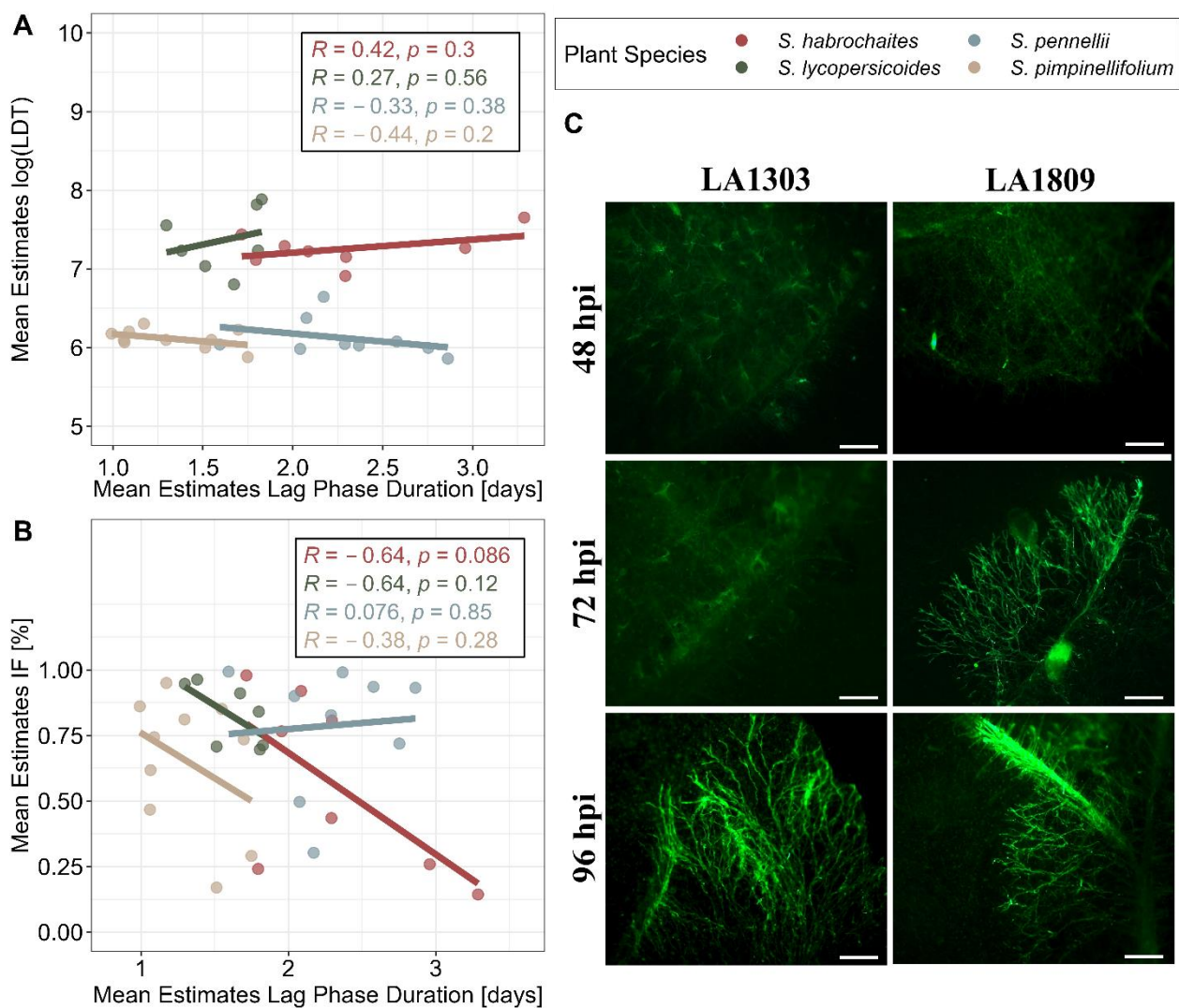
1
2 **Figure 4: QDR parameters show different levels of variation depending on the host species (*S. pennellii* and *S.***
3 ***lycopersicoides*).** A) The lag phase duration (in days after infection) of *S. sclerotiorum* infection on *S. pennellii*
4 *accessions displays a higher level of intraspecific diversity than on accessions of S. lycopersicoides* (B). C) & D)
5 *Variation statistics of the lag phase duration contrasting each accession with the grand mean per species (S. pennellii*
6 *C, S. lycopersicoides D). Estimates are displayed in days post inoculation. E) & F) The lesion doubling time (in*
7 *hours) of S. sclerotiorum infection on S. pennellii accessions is lower than on S. lycopersicoides. G) Variation*
8 *statistics of LDT on S. pennellii and H) S. lycopersicoides. Lsmean-values and SE indicate the adjusted mean and SE*
9 *per population. Estimate comp., SE.comp, and p-values describe pairwise statistics of each accession against the*

1 grand mean. The numbers on the x-axis in panels A, B, E, and F indicate the count of individual leaves tested. Levels
2 of significance are displayed as *** $P < 0.001$, ** $P < 0.01$, * $P < 0.05$, $P < 0.1$.
3

4 **Disease resistance measures are not linked and characterize distinct components of** 5 **quantitative disease resistance.**

6 To test whether fungal infection is directly linked to lesion growth, we conducted microscopy
7 assays using a GFP-tagged *S. sclerotiorum* mutant of the *S. sclerotiorum* isolate 1980 [75]. We
8 selected two accessions from *S. pennellii* with significantly altered lag-phase duration. At 72 hours
9 after inoculation, freshly developed mycelium was observed on leaves of the *S. pennellii* accession
10 with the shortest lag phase duration (LA1809). In contrast, on the less susceptible accession
11 LA1303, the first fungal structures started growing at 96 hpi (suppl. fig. 6). Fluorescent microscopy
12 imaging showed that fungal mycelial structures were always accompanied by clear formation of
13 necrotic lesions but cannot be observed prior visual lesion development (fig. 5 C, suppl. fig. 6).
14 Thus, showing that a longer lag phase does not represent any latent or biotrophic infection and that
15 LDT and lag phase are likely uncoupled phenomena.

16 We performed a correlation analysis to consolidate the relationship between the QDR parameters
17 further. First, we tested the overall relation of lsmean LDT and lsmean lag-phase duration by
18 pooling all accessions of all species. We found that LDT and lag phase were independent ($R=0.14$),
19 with no significant relationship ($p=0.42$) (suppl. fig. 4). We also tested the correlation between
20 QDR strategies at the species level. We found only minor linear relationships between LDT and
21 lag-phase for the four tested species. However, we found a weak, significant negative correlation
22 between infection frequency and the duration of the lag phase (lsmean) in *S. habrochaites* ($R = -$
23 0.64 , $p=0.086$) (fig. 5 A, B). For the remaining species, no significant correlation was found. We
24 did not find a single host accession with high levels of resistance in both, LDT and lag-phase
25 duration.



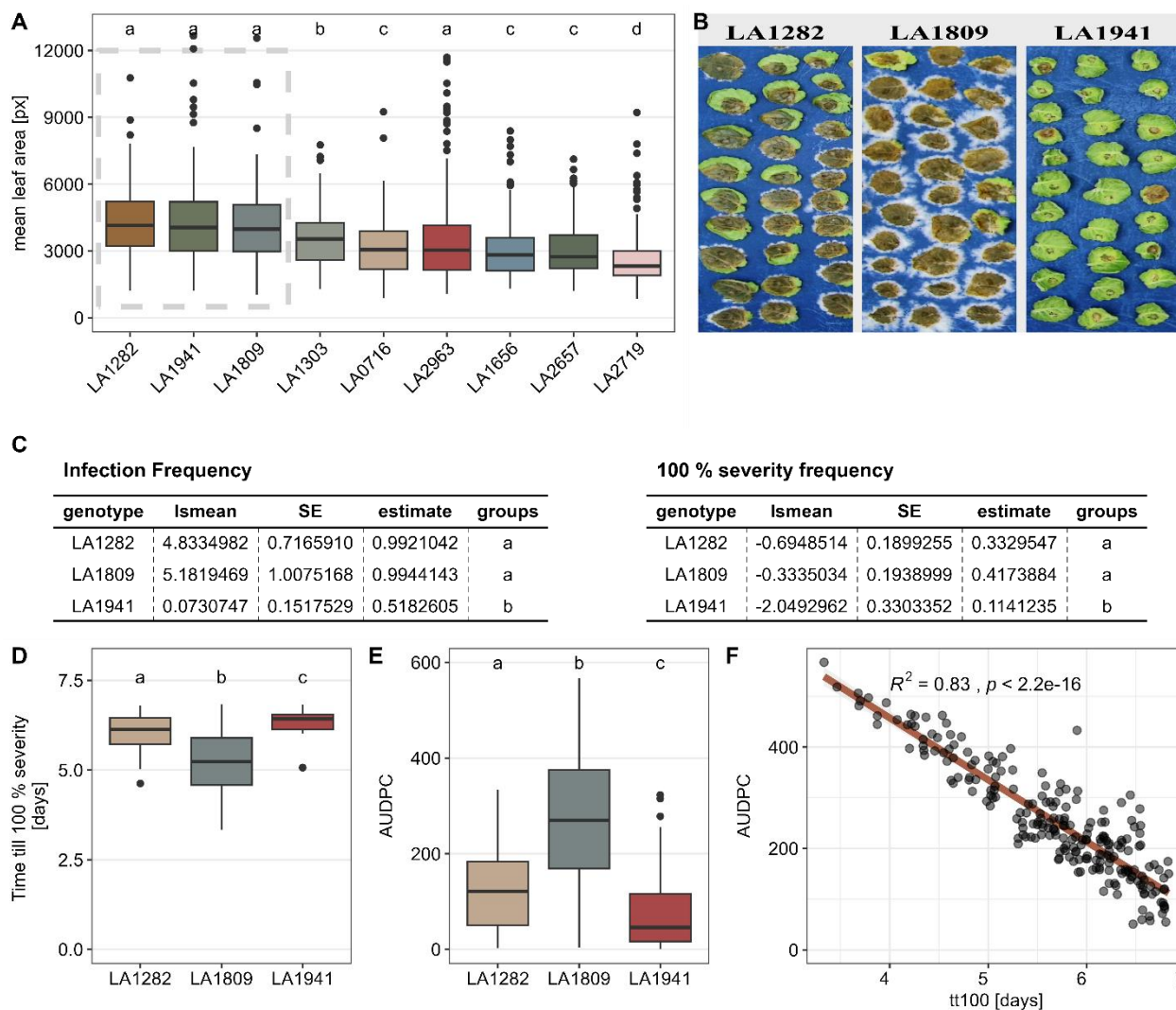
1
2 **Figure 5: Different QDR parameters are independent from each other.** A) Pearson correlation analysis of LDT
3 and lag-phase duration. Dots represent the least squares of each accession. B) Pearson correlation analysis between
4 the infection frequency and lag phase duration. Dots represent infection frequency adjusted per-accession estimates
5 from glm/gls. C) The lag-phase duration of *Ss1980:GFP* infection on *S. pennellii* genotypes is reflected in fungal
6 growth dynamics. Images show a representative selection of 10 biological replicates. The bar indicates 500 μ m.

7 **Severity analysis reveals distinct resistance phenotypes against *Sclerotinia sclerotiorum***
8 **within a single species**

9 For an in-depth analysis of disease severity, we selected three *S. pennellii* accessions with similar
10 leaf sizes: LA1282, LA1809, and LA1941 (fig. 6 A). While symptoms developed on most of the
11 leaves, the impact of infection is highly dependent on the respective accession (see fig. 6 B).
12 Accession LA1941 shows a significantly lower infection frequency (~51%) and a significantly
13 lower rate of fully infected leaves than LA1809 (approx. 11% vs. approx. 41%) or LA1282 (approx.
14 33%, fig. 6 C). It took approx. 6.5 -7 days to cover the whole leaf surface of LA1282 and LA1941.
15 We found a significantly faster lesion spread on LA1809 with approx. 5.5 days till 100 % coverage.

1 This is also reflected by the AUDPC, where LA1809 had the highest mean value (AUDPC approx.
2 250). In contrast, in LA1282 and LA1941, the symptoms spread much slower, leading to
3 significantly lower AUDPC values (100 and 50, respectively, fig. 6 E). Together, the prolonged
4 time till 100% severity and a considerably low AUDPC on LA1282 indicate a late but explosive
5 lesion growth, corresponding to the mean severity-kinetics of those genotypes (fig. 6).

6



1
2 **Figure 6: *S. pennellii* accession LA1941 harbours significantly elevated level of quantitative resistance against *S.***
3 ***sclerotiorum*.** A) Mean leaf area of *S. pennellii* accessions quantified during infection experiments. The data of three
4 independent experiments is shown. HSD test was performed to identify cluster with similar leaf size. Selected plants
5 with similar leaf size are indicated by the box. B) Exemplary images of *S. sclerotiorum* infections on the *S. pennellii*
6 populations LA1809 and LA1941 at seven days post-inoculation. C) Statistical analysis of Infection frequency (IF)
7 and frequency of fully infected leaves at the end of experiment. “lsmean” represents the estimate as logits, while
8 ‘estimate’ represents the estimated probability. D) Comparison of time till lesion saturation of *S. sclerotiorum* on *S.*
9 *pennellii* genotypes. E) Area under disease progress curve (AUDPC) of three *S. pennellii* populations with similar leaf
10 size. Wilcoxon-test was performed for levels of significance. Time series data from previous experiments was used.
11 F) Pearson correlation analysis of tt100 vs. AUDPC.
12 All statistics were calculated using a glm/gls with custom contrast matrices. Compact letter display were determined
13 using the package ‘multcompLetters’ with a threshold of $p < 0.05$.

14

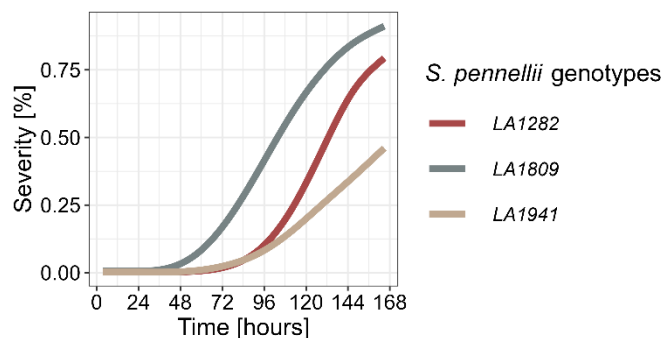
1 The moderation of QDR parameters is genotype-dependent.

2 Next, we used a linear mixed-effect model (lme) to test which of the factors have the strongest
 3 effects on disease severity on those accessions (*S. pennellii* LA1282, LA1809, LA1941). Following
 4 the analysis of variance (ANOVA), we found a significant influence of most tested variables
 5 (genotype, lag phase, LDT) on the AUDPC (tab. 1). Strikingly, we found that the genomic
 6 background of the tested plants is insufficient to explain the observed diversity in AUDPC. In other
 7 words, we observe a significant relationship between lag, LDT, and their interaction with the
 8 genotype. Because of this, we extracted the fixed-effect estimates from the lme and

9
 10 Table 1: **Statistical analysis of the effects of genotype, lag-phase duration, LDT, and their interactions on**
 11 **disease severity (AUDPC) of the *S. pennellii* accessions LA1282, LA1809, and LA1941.** Results of an analysis of
 12 variance (ANOVA) based on a linear mixed-effects model are shown.
 13

	numDF	denDF	F.value	p.value
Intercept	1	437	136.8	<0.001
Genotype	2	437	211.34	<0.001
Lag	1	437	251.68	<0.001
LDT	1	437	90.41	<0.001
Genotype:Lag	2	437	8.54	<0.001
Genotype:LDT	2	437	2.32	0.099
Lag:LDT	1	437	21.91	<0.001
Genotype:Lag:LDT	2	437	3.3	0.038

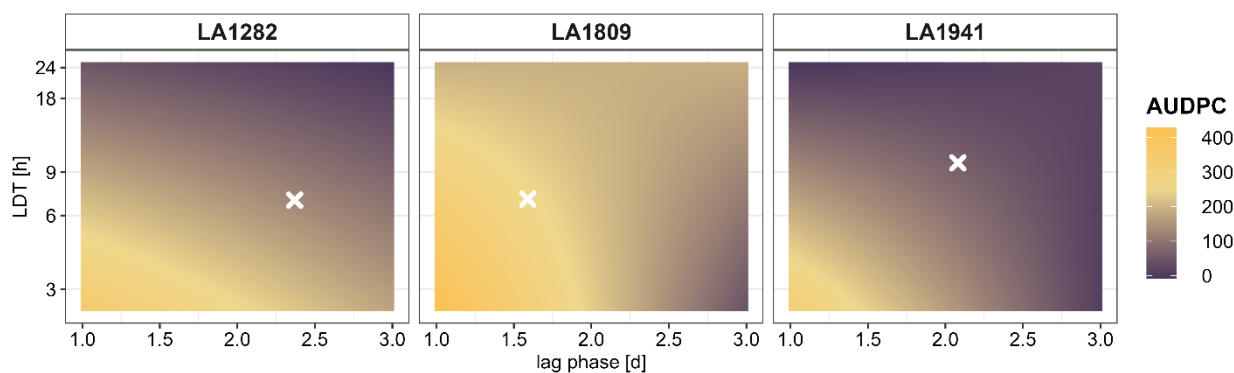
14
 15
 16
 17



18 Figure 7: **Exemplary growth curve of three *S. pennellii* accessions with different resistance levels against *S.***
 19 ***sclerotiorum*.** Shown is the mean symptom-severity of each accession as share of leaf area over the period of seven
 20 days. The experiment was independently repeated three times. $n_{LA1282}=205$, $n_{LA1809}=148$, $n_{LA1941}=98$.

21

1 generated predictor functions for the AUDPC of each genotype. Then, we modeled the AUDPC
2 using high-confidence lag and LDT values from previous observations (see fig. 3 C, D). We
3 observed the highly variable influence of lag-phase duration, LDT, and their interaction on the
4 AUDPC (fig. 8). Strikingly, we found that variation of the LDT has almost no influence on the
5 AUDPC of LA1809 besides the generally elevated severity level (fig. 8). Further, we found that
6 only a prolonged lag phase duration might contribute to an increased potential for lower severity
7 in LA1809 (fig. 8). However, the influence of longer lag-phase is reduced with increasing LDT.
8 For leaves of the accessions LA1282 and LA1941, we found a stronger combined effect of lag-
9 phase and LDT on the severity. More specifically, a prolonged lag phase might lead to a small
10 reduction of the symptom severity on LA1282 while reducing the AUDPC on LA1941 more
11 rapidly. Further, we observe that a prolonged lesion doubling time reduced symptom severity in
12 both LA1282 and LA1941.



13
14 **Figure 8: QDR parameters contribute highly dynamic and host-specific to symptom severity.** We used the *S.*
15 *pennellii* accessions LA1282, LA1809 and LA1941 to test for the genotype-dependent relationship between lag and
16 LDT. Therefore, we extracted the estimates for the factors LDT and lag per each genotype from an ANOVA based
17 on a generalized least square model (tab. 1). The per-genotype AUDPC was modeled using the extracted estimates
18 over a range of values representing the plausible range of lag-/LDT-values. Crosses represent the observed mean
19 AUDPC (fig. 6 E).

20

1 **4. Discussion**

2 **QDR against *Sclerotinia sclerotiorum* is highly diverse in *Solanum* spp..**

3 Wild tomato species have been screened for quantitative resistance phenotypes against many
4 diseases, including Tomato brown rugose fruit virus, *Phytophthora infestans*, *Alternaria solani*,
5 *Fusarium spp.* or *Botrytis cinerea* [8,49,50,74,76–79]. However, high hurdles in characterizing
6 QDR on a phenotypic level limit detailed insights into the functional role of QDR against
7 necrotrophic pathogens. This was mostly due to the lack of affordable high-throughput
8 phenotyping facilities [1,2,5,8,80]. Here, we present a unique dataset of high-resolution QDR
9 phenotypes against *Sclerotinia sclerotiorum* on a diverse set of wild *Solanum* species derived from
10 a low-budget phenotyping set-up. In total, we tested almost 7,000 leaves with approx. 1,000
11 measurements each, resulting in approx. 7 million data points. We used this unique dataset to
12 characterize the lesion development of infected leaves and applied advanced statistical analysis
13 methods to extract more specific descriptors for QDR, such as lag phase, LDT, or AUDPC [30].
14 Because of this system's scale and temporal resolution, we generated novel insights into the
15 phenomena contributing to QDR.

16 **Interspecific QDR phenotypes follow a wide distribution**

17 As expected, we observed a diverse range of disease phenotypes, as demonstrated in previous
18 studies[6,49,78]. None of the tested accessions carried complete resistance against *S. sclerotiorum*,
19 although we found a wide distribution of infection phenotypes. Also, no high ‘universal’ level of
20 partial resistance or tolerance among multiple QDR parameters was found, as none of the species
21 harbors significant advantages in multiple measures (infection frequency, lag phase, or lesion
22 doubling time). Complete resistance against *S. sclerotiorum* is rarely found in cultivated crops
23 [32,52,60,81]. We provide evidence that the time till the emergence of the first lesions (lag-phase)
24 is highly variable within and between host species, with only *S. lycopersicoides* showing a rather
25 conserved lag-phase duration (fig. 4 B). Interestingly, Barbacci et al., (2020) reported that in
26 *Arabidopsis thaliana* the lag-phase duration is mostly influenced by the *S. sclerotiorum* isolate
27 rather than the host accession. The comparably low genetic diversity of the host may have
28 influenced the observed range of QDRs. Standing genetic variation is considered much higher in
29 (predominantly) outcrossing *Solanum* species than in inbreeding *A. thaliana* accessions [82].
30 Accordingly, we assume that the influence of genetic features on the lag-phase duration depends

1 on the specific genomic background of the host plant species. However, fungal influences on
2 pathogenesis cannot be ignored, as the concept of the ‘extended phenotype’, describing the
3 interaction of both genomes i.e. a genotype x genotype (GxG) interaction for host and pathogen,
4 for one phenotype, is well established [37,83]. Furthermore, quantitative host resistance features
5 have been described to interact with the pathogen’s genotype as described for camalexin-associated
6 resistance [50,84].

7 **QDR phenotypes also differ on the intraspecific level but at varying degrees.**

8 High variability of QDR phenotypes among genotypes of the same plant species has been reported
9 on multiple hosts before [6,30,49,74,85,86]. We show that the degree of variability depends on the
10 host species and the respective resistance parameter. Whereas LDT is rather stable among *S.*
11 *pimpinellifolium* accessions, it is highly variable on *S. lycopersicoides* accessions (see fig. 4 &
12 suppl. fig. 2). The specific forms of QDR phenotypes might hint at independent regulatory
13 mechanisms and different evolutionary backgrounds with relatively recent developments, leading
14 to genetic variation, rather than conserved QDR mechanisms. Host adaptation to natural habitats
15 and its influence on disease resistance has been studied before [87,88]. Adaptation might explain
16 disease phenotypes as most *S. lycopersicoides* accessions show significantly prolonged LDT. The
17 habitat of *S. lycopersicoides* faces much more rain than the other species, leading to higher chances
18 of successful infection events than in relatively dry habitats, thus requiring mechanisms to fight
19 established infections. In contrast, drought-resistant *S. pennellii* has high capabilities in delaying
20 infection events, while it lacks defense efficacy once an infection is established (fig. 3), similar to
21 *S. chilense* desert population losing resistance against the fungus *Passalora fulva* [46,87,89].
22 However, to truly test these hypotheses, significantly higher sample sizes and infections under
23 natural conditions would be required, possibly paired with screenings of the morphological
24 properties of the species to assess the pleiotropic influence of habitat adaptation on QDR, e.g., via
25 cuticle thickness or stomata density.

26 **QDR and Genotype x Genotype x Environment interactions**

27 *S. pennellii* accession LA0716 was characterized as relatively resistant against *B. cinerea*, while
28 this genotype is highly susceptible to *S. sclerotiorum* (suppl. fig. 5) [6]. Also in *S. chilense*, QDR
29 phenotypes vary between the pathogen, suggesting the presence of pathogen-specific regulatory
30 mechanisms [78]. However, the pathogen diversity tested in such studies might greatly affect the

1 observed degree of resistance. A study with *Phytophthora infestans* on 85 *S. chilense* accessions
2 showed that the relative differences in resistance phenotypes between individuals were mainly
3 determined by the plant genotype, with modest effects of pathogen isolate used [49]. In contrast,
4 large-scale screenings of infections with different *B. cinerea* isolates showed a clear genotype x
5 genotype (G x G) effect both on panels of wild and domesticated tomatoes, as well as on
6 *Arabidopsis thaliana* [37,50,74,90,91]. In addition, we have shown in *S. chilense* that QDR
7 phenotypes, like the infection frequency, can be correlated with the phytohormone ethylene [92].
8 Knowing that such phytohormonal regulation is also affected by abiotic, environmental (E) factors
9 like temperature, humidity, and light availability, we propose that QDR polymorphism is
10 implemented in a complex signaling network affected by GxGxE interactions [1,5,93,94].

11 **QDR is determined by the interplay of QDR strategies**

12 QDR is commonly defined as a highly interconnected regulatory network with an integrated,
13 pleiotropic role in general plant metabolism [1]. Therefore, the linkage of different defense
14 strategies, like IF and lag-phase duration, could be a good perspective for resistance breeding.
15 However, we did not observe strong correlations between QDR parameters and did not find a
16 species or accession with a universal high resistance level for all tested parameters. Disconnected
17 QDR parameters have been reported before: *Xanthomonas axanopodis* mutants showed increased
18 infection frequency but a reduced lesion growth rate on cassava and *B. cinerea* showed
19 unconnected IF and lesion expansion rates on wild tomatoes [6,95]. We used the presented
20 phenotyping platform to show that the moderation or cross-talk between defense strategies is
21 genotype-specific and differs even between accessions of the same species (fig. 8). Based on these
22 findings, we propose a model for QDR against necrotrophic pathogens involving three genetically
23 distinct strategies: 1. Prohibition of initial infection, 2. Retardation of disease outbreaks, and 3.
24 Deceleration of ongoing infections.

25 **Disease severity is specifically determined by genotype-dependent moderation of QDR** 26 **strategies.**

27 We used three differently severely infected *S. pennellii* genotypes to describe the influence of two
28 of the QDR strategies (retardation and deceleration of symptom development) on overall symptom
29 severity. Interestingly, the different accessions possess diverse capabilities in moderating the QDR
30 strategies, as our model-based approach indicates contrasting roles of LDT and lag-phase duration.

1 In *A. thaliana*, it was shown that lesion traits, like lesion size or shape, are also controlled by
2 genetically distinct mechanisms [90]. Previous work showed that defense-associated hormone
3 responses greatly differ between different wild tomato accessions and even within the same
4 population. In *S. chilense*, ethylene responses could only be linked to IF in one population but not
5 in others [92]. Therefore, we argue that the genetic finetuning of QDR measures highly depends
6 on the specific genetic background, and future studies should determine the complex interplay
7 between various QDR-regulating strategies [94].

8 In this study, we used a new phenotyping platform to derive different QDR-related phenotypes.
9 The low cost and high flexibility of the system allowed us to screen a big set of diverse plants
10 relatively fast, and therefore, we identified new genotypes with distinct QDR properties.
11 Accordingly, we characterized accessions and species with beneficial properties as significantly
12 longer lag-phase duration (*S. pennellii*, LA1303 & LA1656) or prolonged LDT (*S. lycopersicoides*,
13 LA2951 & LA1964). Accordingly, we suggest that *S. pennellii* accessions are specialized in
14 delaying lesion development, whereas *S. lycopersicoides* accessions are more capable of slowing
15 down the spread of established lesions. Follow-up research is needed to identify the genes
16 underlying these differences. The resolution of the present dataset will enhance the ability to predict
17 distinct defense phases, facilitating more targeted sampling strategies for transcriptomic or
18 metabolomic analysis. This can help breed durable resistance in tomato crops with delayed and less
19 severe symptoms without inducing strong evolutionary pressure. The sustainability of major R
20 gene-mediated resistance (including pyramiding of such) has regularly been questioned [4,96].
21 Facilitating the concept of QDR is proposed to thwart the arms race between plant hosts and
22 pathogens. QDR phenotypes specifically tolerate disease to a certain extent without applying a
23 strong bottleneck onto the pathogen population [14]. Our findings provide major insights into the
24 architecture of QDR strategies and will help in the targeted functional characterization of QDR. By
25 disentangling end-point QDR phenotypes into discrete resistance mechanisms, the functional
26 characterization of genetic features controlling QDR will become much more targeted. Based on
27 this study, the factors influencing the level of QDR can be explained in much more detail.

1 **Acknowledgments**

2 We gratefully acknowledge Hendrik Seide, Lilly Gieseler, and Ellen Krohn for their technical
3 support and Dr. Farooq Ahmad for his insightful feedback and discussions whilst writing the
4 manuscript. We also thank the TGRC at UC Davis (USA) for seed material.

5 **Availability of data and materials**

6 Additional data can be found in the supplementary information files. All scripts used for this study
7 are available at https://github.com/seveein/QDR_Wild_Tomatoes.

8 **Competing Interest declaration**

9 The authors declare that they have no competing interests.

10 **Funding**

11 This work was partly funded by the DFG (STA1547/6 and SFB924) and the ANR (ANR-21-CE20-
12 30) for the collaborative project ResiDEvo. Exchange visits were supported by the DAAD and the
13 Partenariat Hubert Curien programme of Campus France.

14

15 **Authors' contribution**

16 RS and SR conceived this study, SE planned and performed the experiments. AB and CTR
17 developed the image analysis pipeline. SE conducted all analytical steps and produced the figures.
18 MH developed the statistical tests and models. SE and RS wrote the manuscript. All authors read
19 and approved the final manuscript.

20

1 References

- 2 [1] J.A. Poland, P.J. Balint-Kurti, R.J. Wisser, R.C. Pratt, R.J. Nelson, Shades of gray: the world of quantitative
3 disease resistance, *Trends Plant Sci.* 14 (2009) 21–29. <https://doi.org/10.1016/j.tplants.2008.10.006>.
- 4 [2] F. Roux, D. Voisin, T. Badet, C. Balagué, X. Barlet, C. Huard-Chauveau, D. Roby, S. Raffaele, Resistance to
5 phytopathogens *e tutti quanti* : placing plant quantitative disease resistance on the map: Quantitative disease
6 resistance in plants, *Mol. Plant Pathol.* 15 (2014) 427–432. <https://doi.org/10.1111/mpp.12138>.
- 7 [3] M. Mbengue, O. Navaud, R. Peyraud, M. Barascud, T. Badet, R. Vincent, A. Barbacci, S. Raffaele, Emerging
8 Trends in Molecular Interactions between Plants and the Broad Host Range Fungal Pathogens *Botrytis cinerea*
9 and *Sclerotinia sclerotiorum*, *Front. Plant Sci.* 7 (2016). <https://doi.org/10.3389/fpls.2016.00422>.
- 10 [4] J.K.M. Brown, Durable Resistance of Crops to Disease: A Darwinian Perspective, *Annu. Rev. Phytopathol.* 53
11 (2015) 513–539. <https://doi.org/10.1146/annurev-phyto-102313-045914>.
- 12 [5] M. Gou, P. Balint-Kurti, M. Xu, Q. Yang, Quantitative disease resistance: Multifaceted players in plant
13 defense, *J. Integr. Plant Biol.* 65 (2023) 594–610. <https://doi.org/10.1111/jipb.13419>.
- 14 [6] A. ten Have, R. van Berloo, P. Lindhout, J.A.L. van Kan, Partial stem and leaf resistance against the fungal
15 pathogen *Botrytis cinerea* in wild relatives of tomato, *Eur. J. Plant Pathol.* 117 (2007) 153–166.
16 <https://doi.org/10.1007/s10658-006-9081-9>.
- 17 [7] L. Tian, J. Li, Y. Xu, Y. Qiu, Y. Zhang, X. Li, A MAP kinase cascade broadly regulates the lifestyle of
18 *Sclerotinia sclerotiorum* and can be targeted by HIGS for disease control, *Plant J.* (2023) tpj.16606.
19 <https://doi.org/10.1111/tpj.16606>.
- 20 [8] J.A. Corwin, D.J. Kliebenstein, Quantitative Resistance: More Than Just Perception of a Pathogen, *Plant Cell*
21 29 (2017) 655–665. <https://doi.org/10.1105/tpc.16.00915>.
- 22 [9] C. Boudhrioua, M. Bastien, D. Torkamaneh, F. Belzile, Genome-wide association mapping of *Sclerotinia*
23 *sclerotiorum* resistance in soybean using whole-genome resequencing data, *BMC Plant Biol.* 20 (2020) 195.
24 <https://doi.org/10.1186/s12870-020-02401-8>.
- 25 [10] L.A. Frey, T. Vleugels, T. Ruttink, F.X. Schubiger, M. Pégard, L. Skøt, C. Grieder, B. Studer, I. Roldán-Ruiz,
26 R. Kölliker, Phenotypic variation and quantitative trait loci for resistance to southern anthracnose and clover
27 rot in red clover, *Theor. Appl. Genet.* (2022). <https://doi.org/10.1007/s00122-022-04223-8>.
- 28 [11] C.M. Fusari, J.A. Di Rienzo, C. Troglia, V. Nishinakamasu, M.V. Moreno, C. Maringolo, F. Quiroz, D.
29 Álvarez, A. Escande, E. Hopp, R. Heinz, V.V. Lia, N.B. Paniego, Association mapping in sunflower for
30 *sclerotinia* head rot resistance, *BMC Plant Biol.* 12 (2012) 93. <https://doi.org/10.1186/1471-2229-12-93>.
- 31 [12] J. Wu, G. Cai, J. Tu, L. Li, S. Liu, X. Luo, L. Zhou, C. Fan, Y. Zhou, Identification of QTLs for Resistance to
32 *Sclerotinia* Stem Rot and *BnaC.IGMT5.a* as a Candidate Gene of the Major Resistant QTL SRC6 in *Brassica*
33 *napus*, *PLoS ONE* 8 (2013) e67740. <https://doi.org/10.1371/journal.pone.0067740>.
- 34 [13] M. Derbyshire, M. Denton-Giles, D. Hegedus, S. Seifbarghy, J. Rollins, J. Van Kan, M.F. Seidl, L. Faino, M.
35 Mbengue, O. Navaud, S. Raffaele, K. Hammond-Kosack, S. Heard, R. Oliver, The Complete Genome
36 Sequence of the Phytopathogenic Fungus *Sclerotinia sclerotiorum* Reveals Insights into the Genome
37 Architecture of Broad Host Range Pathogens, *Genome Biol. Evol.* 9 (2017) 593–618.
38 <https://doi.org/10.1093/gbe/evx030>.
- 39 [14] L. Willocquet, S. Savary, J. Yuen, Multiscale Phenotyping and Decision Strategies in Breeding for Resistance,
40 *Trends Plant Sci.* 22 (2017) 420–432. <https://doi.org/10.1016/j.tplants.2017.01.009>.
- 41 [15] P.M. Dracatos, S. Lück, D.K. Douchkov, Diversifying Resistance Mechanisms in Cereal Crops Using
42 Microphenomics, *Plant Phenomics* 5 (2023) 0023. <https://doi.org/10.34133/plantphenomics.0023>.
- 43 [16] J.J. Walsh, E. Mangina, S. Negrão, Advancements in Imaging Sensors and AI for Plant Stress Detection: A
44 Systematic Literature Review, *Plant Phenomics* 6 (2024) 0153. <https://doi.org/10.34133/plantphenomics.0153>.
- 45 [17] N. Fahlgren, M. Feldman, M.A. Gehan, M.S. Wilson, C. Shyu, D.W. Bryant, S.T. Hill, C.J. McEntee, S.N.
46 Warnasooriya, I. Kumar, T. Ficor, S. Turnipseed, K.B. Gilbert, T.P. Brutnell, J.C. Carrington, T.C. Mockler, I.
47 Baxter, A Versatile Phenotyping System and Analytics Platform Reveals Diverse Temporal Responses to
48 Water Availability in *Setaria*, *Mol. Plant* 8 (2015) 1520–1535. <https://doi.org/10.1016/j.molp.2015.06.005>.
- 49 [18] M. Watt, F. Fiorani, B. Usadel, U. Rascher, O. Muller, U. Schurr, Phenotyping: New Windows into the Plant
50 for Breeders, *Annu. Rev. Plant Biol.* 71 (2020) 689–712. <https://doi.org/10.1146/annurev-arplant-042916-041124>.
- 51 [19] C.H. Bock, J.G.A. Barbedo, E.M. Del Ponte, D. Bohnenkamp, A.-K. Mahlein, From visual estimates to fully
52 automated sensor-based measurements of plant disease severity: status and challenges for improving accuracy,
53 *Phytopathol. Res.* 2 (2020) 9. <https://doi.org/10.1186/s42483-020-00049-8>.
- 54

- 1 [20] A.-K. Mahlein, Plant Disease Detection by Imaging Sensors – Parallels and Specific Demands for Precision
2 Agriculture and Plant Phenotyping, *Plant Dis.* 100 (2016) 241–251. [https://doi.org/10.1094/PDIS-03-15-0340-
4 FE](https://doi.org/10.1094/PDIS-03-15-0340-
3 FE).
- 4 [21] A.-K. Mahlein, M.T. Kuska, S. Thomas, M. Wahabzada, J. Behmann, U. Rascher, K. Kersting, Quantitative
5 and qualitative phenotyping of disease resistance of crops by hyperspectral sensors: seamless interlocking of
6 phytopathology, sensors, and machine learning is needed!, *Curr. Opin. Plant Biol.* 50 (2019) 156–162.
7 <https://doi.org/10.1016/j.pbi.2019.06.007>.
- 8 [22] A.M. Mutka, R.S. Bart, Image-based phenotyping of plant disease symptoms, *Front. Plant Sci.* 5 (2015).
9 <https://doi.org/10.3389/fpls.2014.00734>.
- 10 [23] I. Simko, J.A. Jimenez-Berni, X.R.R. Sirault, Phenomic Approaches and Tools for Phytopathologists,
11 *Phytopathology*® 107 (2017) 6–17. <https://doi.org/10.1094/PHYTO-02-16-0082-RVW>.
- 12 [24] F. Tanner, S. Tonn, J. de Wit, G. Van den Ackerveken, B. Berger, D. Plett, Sensor-based phenotyping of
13 above-ground plant-pathogen interactions, *Plant Methods* 18 (2022) 35. [https://doi.org/10.1186/s13007-022-
15 00853-7](https://doi.org/10.1186/s13007-022-
14 00853-7).
- 15 [25] A.O. Anim-Ayeko, C. Schillaci, A. Lipani, Automatic blight disease detection in potato (*Solanum tuberosum*
16 L.) and tomato (*Solanum lycopersicum*, L. 1753) plants using deep learning, *Smart Agric. Technol.* 4 (2023)
17 100178. <https://doi.org/10.1016/j.atech.2023.100178>.
- 18 [26] M.T. Kuska, R.H.J. Heim, I. Geedicke, K.M. Gold, A. Brugger, S. Paulus, Digital plant pathology: a
19 foundation and guide to modern agriculture, *J. Plant Dis. Prot.* 129 (2022) 457–468.
20 <https://doi.org/10.1007/s41348-022-00600-z>.
- 21 [27] Z. Tang, X. He, G. Zhou, A. Chen, Y. Wang, L. Li, Y. Hu, A Precise Image-Based Tomato Leaf Disease
22 Detection Approach Using PLPNet, *Plant Phenomics* 5 (2023) 0042.
23 <https://doi.org/10.34133/plantphenomics.0042>.
- 24 [28] K. Kersting, C. Bauckhage, M. Wahabzada, A.-K. Mahlein, U. Steiner, E.-C. Oerke, C. Römer, L. Plümer,
25 Feeding the World with Big Data: Uncovering Spectral Characteristics and Dynamics of Stressed Plants, in: J.
26 Lässig, K. Kersting, K. Morik (Eds.), *Comput. Sustain.*, Springer International Publishing, Cham, 2016: pp.
27 99–120. https://doi.org/10.1007/978-3-319-31858-5_6.
- 28 [29] H. Poorter, G.M. Hummel, K.A. Nagel, F. Fiorani, P. Von Gillhaussen, O. Virnich, U. Schurr, J.A. Postma, R.
29 Van De Zedde, A. Wiese-Klinkenberg, Pitfalls and potential of high-throughput plant phenotyping platforms,
30 *Front. Plant Sci.* 14 (2023) 1233794. <https://doi.org/10.3389/fpls.2023.1233794>.
- 31 [30] A. Barbacci, O. Navaud, M. Mbengue, M. Barascud, L. Godiard, M. Khafif, A. Lacaze, S. Raffaele, Rapid
32 identification of an Arabidopsis NLR gene as a candidate conferring susceptibility to *Sclerotinia sclerotiorum*
33 using time-resolved automated phenotyping, *Plant J.* 103 (2020) 903–917. <https://doi.org/10.1111/tjp.14747>.
- 34 [31] FAO, Agricultural production statistics 2000–2022, FAO, 2023. <https://doi.org/10.4060/cc9205en>.
- 35 [32] M.D. Bolton, B.P.H.J. Thomma, B.D. Nelson, *Sclerotinia sclerotiorum* (Lib.) de Bary: biology and molecular
36 traits of a cosmopolitan pathogen, *Mol. Plant Pathol.* 7 (2006) 1–16. [https://doi.org/10.1111/j.1364-
38 3703.2005.00316.x](https://doi.org/10.1111/j.1364-
37 3703.2005.00316.x).
- 38 [33] M.R. Foolad, H.L. Merk, H. Ashrafi, Genetics, Genomics and Breeding of Late Blight and Early Blight
39 Resistance in Tomato, *Crit. Rev. Plant Sci.* 27 (2008) 75–107. <https://doi.org/10.1080/07352680802147353>.
- 40 [34] T. Schmey, C.S. Tominello-Ramirez, C. Brune, R. Stam, *Alternaria* diseases on potato and tomato, *Mol. Plant*
41 *Pathol.* 25 (2024) e13435. <https://doi.org/10.1111/mpp.13435>.
- 42 [35] F.G. Zalom, PESTS, ENDANGERED PESTICIDES AND PROCESSING TOMATOES, *Acta Hortic.* (2003)
43 223–233. <https://doi.org/10.17660/ActaHortic.2003.613.35>.
- 44 [36] S. Einspanier, T. Susanto, N. Metz, P.J. Wolters, V.G.A.A. Vleeshouwers, Å. Lankinen, E. Liljeroth, S.
45 Landschoot, Ž. Ivanović, R. Hüchelhoven, H. Hausladen, R. Stam, Whole-genome sequencing elucidates the
46 species-wide diversity and evolution of fungicide resistance in the early blight pathogen *Alternaria solani*,
47 *Evol. Appl.* 15 (2022) 1605–1620. <https://doi.org/10.1111/eva.13350>.
- 48 [37] H.C. Rowe, D.J. Kliebenstein, All Mold Is Not Alike: The Importance of Intraspecific Diversity in
49 Necrotrophic Plant Pathogens, *PLoS Pathog.* 6 (2010) e1000759.
50 <https://doi.org/10.1371/journal.ppat.1000759>.
- 51 [38] T. Schmey, C. Small, S. Einspanier, L.M. Hoyoz, T. Ali, S. Gamboa, B. Mamani, G.C. Sepulveda, M. Thines,
52 R. Stam, Small-spored *Alternaria* spp. (section *Alternaria*) are common pathogens on wild tomato species,
53 *Environ. Microbiol.* (2023) 1462-2920.16394. <https://doi.org/10.1111/1462-2920.16394>.
- 54 [39] R.A. Silva, M.S. Lehner, T.J. Paula Júnior, E.S.G. Mizubuti, Fungicide sensitivity of isolates of *Sclerotinia*
55 *sclerotiorum* from different hosts and regions in Brazil and phenotypic instability of thiophanate-methyl
56 resistant isolates, *Trop. Plant Pathol.* 49 (2024) 93–103. <https://doi.org/10.1007/s40858-023-00629-x>.

- 1 [40] Q. Wang, Y. Mao, S. Li, T. Li, J. Wang, M. Zhou, Y. Duan, Molecular Mechanism of *Sclerotinia sclerotiorum*
2 Resistance to Succinate Dehydrogenase Inhibitor Fungicides, *J. Agric. Food Chem.* 70 (2022) 7039–7048.
3 <https://doi.org/10.1021/acs.jafc.2c02056>.
- 4 [41] A. Bolger, F. Scossa, M.E. Bolger, C. Lanz, F. Maumus, T. Tohge, H. Quesneville, S. Alseekh, I. Sørensen, G.
5 Lichtenstein, E.A. Fich, M. Conte, H. Keller, K. Schneeberger, R. Schwacke, I. Ofner, J. Vrebalov, Y. Xu, S.
6 Osorio, S.A. Aflitos, E. Schijlen, J.M. Jiménez-Goméz, M. Rynagajllo, S. Kimura, R. Kumar, D. Koenig, L.R.
7 Headland, J.N. Maloof, N. Sinha, R.C.H.J. van Ham, R.K. Lankhorst, L. Mao, A. Vogel, B. Arsova, R.
8 Panstruga, Z. Fei, J.K.C. Rose, D. Zamir, F. Carrari, J.J. Giovannoni, D. Weigel, B. Usadel, A.R. Fernie, The
9 genome of the stress-tolerant wild tomato species *Solanum pennellii*, *Nat. Genet.* 46 (2014) 1034–1038.
10 <https://doi.org/10.1038/ng.3046>.
- 11 [42] G.J. Rebetzke, J. Jimenez-Berni, R.A. Fischer, D.M. Deery, D.J. Smith, Review: High-throughput phenotyping
12 to enhance the use of crop genetic resources, *Plant Sci.* 282 (2019) 40–48.
13 <https://doi.org/10.1016/j.plantsci.2018.06.017>.
- 14 [43] J.B. Pease, D.C. Haak, M.W. Hahn, L.C. Moyle, Phylogenomics Reveals Three Sources of Adaptive Variation
15 during a Rapid Radiation, *PLOS Biol.* 14 (2016) e1002379. <https://doi.org/10.1371/journal.pbio.1002379>.
- 16 [44] K.B. Böndel, H. Lainer, T. Nosenko, M. Mboup, A. Tellier, W. Stephan, North–South Colonization
17 Associated with Local Adaptation of the Wild Tomato Species *Solanum chilense*, *Mol. Biol. Evol.* 32 (2015)
18 2932–2943. <https://doi.org/10.1093/molbev/msv166>.
- 19 [45] I. Fischer, L. Camus-Kulandaivelu, F. Allal, W. Stephan, Adaptation to drought in two wild tomato species:
20 the evolution of the *Asr* gene family, *New Phytol.* 190 (2011) 1032–1044. <https://doi.org/10.1111/j.1469-8137.2011.03648.x>.
- 22 [46] T.L. Kahn, S.E. Fender, E.A. Bray, M.A. O’Connell, Characterization of Expression of Drought- and Abscisic
23 Acid-Regulated Tomato Genes in the Drought-Resistant Species *Lycopersicon pennellii*, *Plant Physiol.* 103
24 (1993) 597–605. <https://doi.org/10.1104/pp.103.2.597>.
- 25 [47] T. Nosenko, K.B. Böndel, G. Kumpfmüller, W. Stephan, Adaptation to low temperatures in the wild tomato
26 species *Solanum chilense*, *Mol. Ecol.* 25 (2016) 2853–2869. <https://doi.org/10.1111/mec.13637>.
- 27 [48] R. Stam, T. Nosenko, A.C. Hörger, W. Stephan, M. Seidel, J.M.M. Kuhn, G. Haberer, A. Tellier, The *de Novo*
28 Reference Genome and Transcriptome Assemblies of the Wild Tomato Species *Solanum chilense* Highlights
29 Birth and Death of NLR Genes Between Tomato Species, *G3 GenesGenomesGenetics* 9 (2019) 3933–3941.
30 <https://doi.org/10.1534/g3.119.400529>.
- 31 [49] P.S. Kahlon, M. Verin, R. Hüchelhoven, R. Stam, Quantitative resistance differences between and within
32 natural populations of *Solanum chilense* against the oomycete pathogen *Phytophthora infestans*, *Ecol. Evol.* 11
33 (2021) 7768–7778. <https://doi.org/10.1002/ece3.7610>.
- 34 [50] N.E. Soltis, S. Atwell, G. Shi, R. Fordyce, R. Gwinner, D. Gao, A. Shafi, D.J. Kliebenstein, Interactions of
35 Tomato and *Botrytis cinerea* Genetic Diversity: Parsing the Contributions of Host Differentiation,
36 Domestication, and Pathogen Variation, *Plant Cell* 31 (2019) 502–519. <https://doi.org/10.1105/tpc.18.00857>.
- 37 [51] G.J. Boland, R. Hall, Index of plant hosts of *Sclerotinia sclerotiorum*, *Can. J. Plant Pathol.* 16 (1994) 93–108.
38 <https://doi.org/10.1080/07060669409500766>.
- 39 [52] M.C. Derbyshire, M. Denton-Giles, The control of sclerotinia stem rot on oilseed rape (*Brassica napus*):
40 current practices and future opportunities, *Plant Pathol.* 65 (2016) 859–877. <https://doi.org/10.1111/ppa.12517>.
- 41 [53] P. Mazumdar, Sclerotinia stem rot in tomato: a review on biology, pathogenicity, disease management and
42 future research priorities, *J. Plant Dis. Prot.* 128 (2021) 1403–1431. <https://doi.org/10.1007/s41348-021-00509-z>.
- 44 [54] C.A. O’Sullivan, K. Belt, L.F. Thatcher, Tackling Control of a Cosmopolitan Phytopathogen: Sclerotinia,
45 *Front. Plant Sci.* 12 (2021) 707509. <https://doi.org/10.3389/fpls.2021.707509>.
- 46 [55] J. Chen, C. Ullah, D. Giddings Vassão, M. Reichelt, J. Gershenson, A. Hammerbacher, *Sclerotinia*
47 *sclerotiorum* Infection Triggers Changes in Primary and Secondary Metabolism in *Arabidopsis thaliana*,
48 *Phytopathology* 111 (2021) 559–569. <https://doi.org/10.1094/PHYTO-04-20-0146-R>.
- 49 [56] J. Sucher, M. Mbengue, A. Dresen, M. Barascud, M. Didelon, A. Barbacci, S. Raffaele, Phylotranscriptomics
50 of the Pentapetalae Reveals Frequent Regulatory Variation in Plant Local Responses to the Fungal Pathogen
51 *Sclerotinia sclerotiorum*, *Plant Cell* 32 (2020) 1820–1844. <https://doi.org/10.1105/tpc.19.00806>.
- 52 [57] M.B. Uloth, M.P. You, P.M. Finnegan, S.S. Banga, S.K. Banga, P.S. Sandhu, H. Yi, P.A. Salisbury, M.J.
53 Barbetti, New sources of resistance to *Sclerotinia sclerotiorum* for crucifer crops, *Field Crops Res.* 154 (2013)
54 40–52. <https://doi.org/10.1016/j.fcr.2013.07.013>.
- 55 [58] D. Wei, J. Mei, Y. Fu, J.O. Disi, J. Li, W. Qian, Quantitative trait loci analyses for resistance to *Sclerotinia*
56 *sclerotiorum* and flowering time in *Brassica napus*, *Mol. Breed.* 34 (2014) 1797–1804.
57 <https://doi.org/10.1007/s11032-014-0139-7>.

- 1 [59] B. Williams, M. Kabbage, H.-J. Kim, R. Britt, M.B. Dickman, Tipping the Balance: Sclerotinia sclerotiorum
2 Secreted Oxalic Acid Suppresses Host Defenses by Manipulating the Host Redox Environment, *PLoS Pathog.*
3 7 (2011) e1002107. <https://doi.org/10.1371/journal.ppat.1002107>.
- 4 [60] Z. Wang, L.-Y. Ma, J. Cao, Y.-L. Li, L.-N. Ding, K.-M. Zhu, Y.-H. Yang, X.-L. Tan, Recent Advances in
5 Mechanisms of Plant Defense to Sclerotinia sclerotiorum, *Front. Plant Sci.* 10 (2019) 1314.
6 <https://doi.org/10.3389/fpls.2019.01314>.
- 7 [61] T. Badet, D. Voisin, M. Mbengue, M. Barascud, J. Sucher, P. Sadon, C. Balagué, D. Roby, S. Raffaele,
8 Parallel evolution of the POQR prolyl oligo peptidase gene conferring plant quantitative disease resistance,
9 *PLOS Genet.* 13 (2017) e1007143. <https://doi.org/10.1371/journal.pgen.1007143>.
- 10 [62] R. Li, R. Rimmer, L. Buchwaldt, A.G. Sharpe, G. Séguin-Swartz, C. Coutu, D.D. Hegedus, Interaction of
11 Sclerotinia sclerotiorum with a resistant Brassica napus cultivar: expressed sequence tag analysis identifies
12 genes associated with fungal pathogenesis, *Fungal Genet. Biol.* 41 (2004) 735–753.
13 <https://doi.org/10.1016/j.fgb.2004.03.001>.
- 14 [63] M.A. Gehan, N. Fahlgren, A. Abbasi, J.C. Berry, S.T. Callen, L. Chavez, A.N. Doust, M.J. Feldman, K.B.
15 Gilbert, J.G. Hodge, J.S. Hoyer, A. Lin, S. Liu, C. Lizárraga, A. Lorence, M. Miller, E. Platon, M. Tessman, T.
16 Sax, PlantCV v2: Image analysis software for high-throughput plant phenotyping, *PeerJ* 5 (2017) e4088.
17 <https://doi.org/10.7717/peerj.4088>.
- 18 [64] R Core Team, R: A Language and Environment for Statistical Computing, R Foundation for Statistical
19 Computing, Vienna, Austria, 2022. <https://www.R-project.org/>.
- 20 [65] H. Wickham, M. Averick, J. Bryan, W. Chang, L.D. McGowan, R. François, G. Grolemund, A. Hayes, L.
21 Henry, J. Hester, M. Kuhn, T.L. Pedersen, E. Miller, S.M. Bache, K. Müller, J. Ooms, D. Robinson, D.P.
22 Seidel, V. Spinu, K. Takahashi, D. Vaughan, C. Wilke, K. Woo, H. Yutani, Welcome to the tidyverse, *J. Open*
23 *Source Softw.* 4 (2019) 1686. <https://doi.org/10.21105/joss.01686>.
- 24 [66] H. Wickham, ggplot2, Springer New York, New York, NY, 2009. <https://doi.org/10.1007/978-0-387-98141-3>.
- 25 [67] A. Kassambara, ggpubr: “ggplot2” Based Publication Ready Plots, 2023. [https://CRAN.R-](https://CRAN.R-project.org/package=ggpubr)
26 [project.org/package=ggpubr](https://CRAN.R-project.org/package=ggpubr).
- 27 [68] Felipe de Mendiburu, Muhammad Yaseen, agricolae: Statistical Procedures for Agricultural Research, 2020.
- 28 [69] R.J. Carroll, D. Ruppert, Transformation and weighting in regression, Chapman and Hall, New York, 1988.
- 29 [70] P. McCullagh, J.A. Nelder, Generalized linear models, 2. ed., [Nachdr.], Chapman & Hall, London, 1999.
- 30 [71] S. Nakagawa, H. Schielzeth, A general and simple method for obtaining R^2 from generalized linear mixed-
31 effects models, *Methods Ecol. Evol.* 4 (2013) 133–142. <https://doi.org/10.1111/j.2041-210x.2012.00261.x>.
- 32 [72] F. Bretz, T. Hothorn, P. Westfall, Multiple Comparisons Using R, 0 ed., Chapman and Hall/CRC, 2016.
33 <https://doi.org/10.1201/9781420010909>.
- 34 [73] T. Hothorn, F. Bretz, P. Westfall, Simultaneous Inference in General Parametric Models, *Biom. J.* 50 (2008)
35 346–363. <https://doi.org/10.1002/bimj.200810425>.
- 36 [74] C. Caseys, G. Shi, N. Soltis, R. Gwinner, J. Corwin, S. Atwell, D.J. Kliebenstein, Quantitative interactions: the
37 disease outcome of *Botrytis cinerea* across the plant kingdom, *G3 GenesGenomesGenetics* 11 (2021) jkab175.
38 <https://doi.org/10.1093/g3journal/jkab175>.
- 39 [75] T. Badet, O. Léger, M. Barascud, D. Voisin, P. Sadon, R. Vincent, A. Le Ru, C. Balagué, D. Roby, S.
40 Raffaele, Expression polymorphism at the *ARPC4* locus links the actin cytoskeleton with quantitative
41 disease resistance to *Sclerotinia sclerotiorum* in *Arabidopsis thaliana*, *New Phytol.* 222 (2019) 480–496.
42 <https://doi.org/10.1111/nph.15580>.
- 43 [76] M. Foolad, L. Zhang, A.A. Khan, D. Niño-Liu, G. Lin, Identification of QTLs for early blight (*Alternaria*
44 *solani*) resistance in tomato using backcross populations of a *Lycopersicon esculentum* × *L. hirsutum* cross,
45 *Theor. Appl. Genet.* 104 (2002) 945–958. <https://doi.org/10.1007/s00122-002-0870-z>.
- 46 [77] A. Kabas, H. Fidan, H. Kucukaydin, H.N. Atan, Screening of wild tomato species and interspecific hybrids for
47 resistance/tolerance to Tomato brown rugose fruit virus (ToBRFV), *Chil. J. Agric. Res.* 82 (2022) 189–196.
48 <https://doi.org/10.4067/S0718-58392022000100189>.
- 49 [78] R. Stam, D. Scheikl, A. Tellier, The wild tomato species *Solanum chilense* shows variation in pathogen
50 resistance between geographically distinct populations, *PeerJ* 5 (2017) e2910.
51 <https://doi.org/10.7717/peerj.2910>.
- 52 [79] L.P. Zhang, G.Y. Lin, D. Niño-Liu, M.R. Foolad, Mapping QTLs conferring early blight (*Alternaria solani*)
53 resistance in a *Lycopersicon esculentum* × *L. hirsutum* cross by selective genotyping, *Mol. Breed.* 12 (2003).
- 54 [80] V. Méline, D.L. Caldwell, B. Kim, R.S. Khangura, S. Baireddy, C. Yang, E.E. Sparks, B. Dilkes, E.J. Delp,
55 A.S. Iyer-Pascuzzi, Image-based assessment of plant disease progression identifies new genetic loci for
56 resistance to *Ralstonia solanacearum* in tomato, *Plant J.* 113 (2023) 887–903.
57 <https://doi.org/10.1111/tpj.16101>.

- 1 [81] L.-N. Ding, T. Li, X.-J. Guo, M. Li, X.-Y. Liu, J. Cao, X.-L. Tan, Sclerotinia Stem Rot Resistance in
2 Rapeseed: Recent Progress and Future Prospects, *J. Agric. Food Chem.* 69 (2021) 2965–2978.
3 <https://doi.org/10.1021/acs.jafc.0c07351>.
- 4 [82] I.E. Peralta, D.M. Spooner, S. Knapp, Taxonomy of wild tomatoes and their relatives (*Solanum* sect.
5 *Lycopersicoides*, sect. *Juglandifolia*, sect. *Lycopersicon*; Solanaceae), American Soc. of Plant Taxonomists,
6 Ann Arbor, Mich, 2008.
- 7 [83] L. Lambrechts, S. Fellous, J.C. Koella, Coevolutionary interactions between host and parasite genotypes,
8 *Trends Parasitol.* 22 (2006) 12–16. <https://doi.org/10.1016/j.pt.2005.11.008>.
- 9 [84] M.S.C. Pedras, S. Hossain, R.B. Snitynsky, Detoxification of cruciferous phytoalexins in *Botrytis cinerea*:
10 Spontaneous dimerization of a camalexin metabolite, *Phytochemistry* 72 (2011) 199–206.
11 <https://doi.org/10.1016/j.phytochem.2010.11.018>.
- 12 [85] S. Chauhan, S. Katoch, S.K. Sharma, P.N. Sharma, J.C. Rana, K. Singh, M. Singh, Screening and
13 identification of resistant sources against *Sclerotinia sclerotiorum* causing white mold disease in common
14 bean, *Crop Sci.* 60 (2020) 1986–1996. <https://doi.org/10.1002/csc2.20160>.
- 15 [86] Y. Yanar, S.A. Miller, Resistance of Pepper Cultivars and Accessions of *Capsicum* spp. to *Sclerotinia*
16 *sclerotiorum*, *Plant Dis.* 87 (2003) 303–307. <https://doi.org/10.1094/PDIS.2003.87.3.303>.
- 17 [87] P.S. Kahlon, S.M. Seta, G. Zander, D. Scheickl, R. Hückelhoven, R. Stam, Population studies of the wild
18 tomato species *Solanum chilense* reveal geographically structured major gene-mediated pathogen resistance,
19 (2020) 9.
- 20 [88] R. Stam, G.A. Silva-Arias, A. Tellier, Subsets of NLR genes show differential signatures of adaptation during
21 colonization of new habitats, *New Phytol.* 224 (2019) 367–379. <https://doi.org/10.1111/nph.16017>.
- 22 [89] C.M. Rick, Potential Genetic Resources in Tomato Species: Clues from Observations in Native Habitats, in:
23 A.M. Srb (Ed.), *Genes Enzym. Popul.*, Springer US, Boston, MA, 1973: pp. 255–269.
24 https://doi.org/10.1007/978-1-4684-2880-3_17.
- 25 [90] R.F. Fordyce, N.E. Soltis, C. Caseys, R. Gwinner, J.A. Corwin, S. Atwell, D. Copeland, J. Feusier, A. Subedy,
26 R. Eshbaugh, D.J. Kliebenstein, Digital Imaging Combined with Genome-Wide Association Mapping Links
27 Loci to Plant-Pathogen Interaction Traits, *Plant Physiol.* 178 (2018) 1406–1422.
28 <https://doi.org/10.1104/pp.18.00851>.
- 29 [91] N.E. Soltis, C. Caseys, W. Zhang, J.A. Corwin, S. Atwell, D.J. Kliebenstein, Pathogen Genetic Control of
30 Transcriptome Variation in the *Arabidopsis thaliana* – *Botrytis cinerea* Pathosystem, (n.d.).
- 31 [92] P.S. Kahlon, A. Förner, M. Muser, M. Oubounyt, M. Gigl, R. Hammerl, J. Baumbach, R. Hückelhoven, C.
32 Dawid, R. Stam, Laminarin-triggered defence responses are geographically dependent in natural populations
33 of *Solanum chilense*, *J. Exp. Bot.* (2023) erad087. <https://doi.org/10.1093/jxb/erad087>.
- 34 [93] M. Altmann, S. Altmann, P.A. Rodriguez, B. Weller, L. Elorduy Vergara, J. Palme, N. Marín-de La Rosa, M.
35 Sauer, M. Wenig, J.A. Villaécija-Aguilar, J. Sales, C.-W. Lin, R. Pandiarajan, V. Young, A. Strobel, L. Gross,
36 S. Carbonnel, K.G. Kugler, A. Garcia-Molina, G.W. Bassel, C. Falter, K.F.X. Mayer, C. Gutjahr, A.C. Vlot, E.
37 Grill, P. Falter-Braun, Extensive signal integration by the phytohormone protein network, *Nature* 583 (2020)
38 271–276. <https://doi.org/10.1038/s41586-020-2460-0>.
- 39 [94] P.S. Kahlon, R. Stam, Polymorphisms in plants to restrict losses to pathogens: From gene family expansions to
40 complex network evolution, *Curr. Opin. Plant Biol.* 62 (2021) 102040.
41 <https://doi.org/10.1016/j.pbi.2021.102040>.
- 42 [95] A.M. Mutka, S.J. Fentress, J.W. Sher, J.C. Berry, C. Pretz, D.A. Nusinow, R. Bart, Quantitative, image-based
43 phenotyping methods provide insight into spatial and temporal dimensions of plant disease, *Plant Physiol.*
44 (2016) pp.00984.2016. <https://doi.org/10.1104/pp.16.00984>.
- 45 [96] R. Stam, B.A. McDonald, When resistance gene pyramids are not durable—the role of pathogen diversity: R-
46 gene pyramid durability and pathogen diversity, *Mol. Plant Pathol.* 19 (2018) 521–524.
47 <https://doi.org/10.1111/mpp.12636>.
- 48