THÈSE DE DOCTORAT D'AVIGNON UNIVERSITÉ

École Doctorale N° 536
Agrosciences & Sciences

Spécialité / Discipline de doctorat :
Sciences Agronomiques

Unité de Pathologie végétale (PV)
Unité de Génétique et Amélioration des Fruits et Légumes (GAFL)
Centre INRAE Provence-Alpes-Côte d'Azur, Avignon

Présentée par
Awa SANGARÉ

Bases génétiques de la défense induite et de la résistance aux stress biotiques chez la tomate

Soutenue publiquement le 17/09/2026 devant le jury composé de :

Mme Elsa Ballini, Professeur des universités, Institut Agro Montpellier	**Rapporteure**
Mme Laurence Godiard, Chargée de recherche, INRAE	**Rapporteure**
Mme Julie Ferreira-De-Carvalho, Chargée de recherche INRAE	**Examinatrice**
M. Laurent Legendre, Professeur des universités, Avignon Université **(Président du jury)**	**Examinateur**
M. Marc Bardin, Directeur de recherche, INRAE	**Directeur de thèse**
M. Bernard Caromel, Ingénieur de recherche, INRAE	**Co-directeur de thèse**

Résumé

Les plantes sont continuellement confrontées à des agents pathogènes tels que les virus, bactéries, champignons et oomycètes. Si elles disposent de mécanismes de défense efficaces, ceux-ci varient selon les variétés et sont fortement perturbés par le changement climatique. Les stimulateurs de défense des plantes (SDP) constituent une stratégie prometteuse pour renforcer durablement la résistance des cultures. Cependant, leur efficacité dépend fortement du génotype de la plante, et les bases génétiques de cette variabilité restent encore peu connues. L'objectif de cette thèse était d'identifier les déterminants génétiques de la réponse des accessions de tomate aux SDP. Pour cela, une core collection de 148 accessions a été évaluée avec deux SDP commerciaux : Belvine®, appliqué contre *Phytophthora infestans* (agent du mildiou) et phénotypé en tunnel en 2024 et 2025, et Planticare®, appliqué contre *Oidium neolycopersici* (agent de l'oïdium) et évalué en serre selon le même dispositif expérimental.

Les analyses GWAS ont permis d'identifier douze QTL associés à la réponse aux SDP, répartis sur les chromosomes 2, 3, 4, 5, 7, 8 et 12, selon le SDP, le pathogène et l'environnement. Les analyses d'expression génique ont montré que les deux SDP induisent la surexpression de plusieurs gènes de défense, notamment les protéines PR (*PR-1*, *PR-2*, *PR-4*, *PR-5* et *PR-8*), des gènes impliqués dans le stress oxydatif (*Peroxidase* et *Glutathione S-transferase*) et dans la biosynthèse de l'éthylène (ACCO). Parmi les gènes candidats localisés dans les intervalles de confiance des QTL détectés pour Belvine®, trois appartiennent à ces familles de gènes (*PR-2*, *Peroxidase* et *Glutathione S-transferase*), renforçant leur implication dans la réponse aux SDP.

Ce travail a également porté sur l'étude de l'architecture génétique de la résistance de la tomate à *Oidium neolycopersici*. L'évaluation de 31 accessions représentant six espèces du genre *Solanum* a mis en évidence *Solanum cheesmaniae* LA0421 comme une nouvelle source de résistance à l'oïdium. Une population F_2 issue du croisement avec l'accession sensible Rose de Berne (*S. lycopersicum*) a permis, grâce à la combinaison des approches QTL-seq et cartographie QTL, d'identifier un nouveau QTL sur le chromosome 9. Le SNP situé au pic du QTL est localisé dans le gène *ETO1* (régulateur de la voie de biosynthèse de l'éthylène), candidat prometteur pour expliquer cette résistance. Grâce à la proximité génétique entre *S. cheesmaniae* et la tomate cultivée, ainsi qu'à la disponibilité de marqueurs KASP, ce QTL constitue une ressource directement exploitable en sélection assistée par marqueurs. En outre, l'analyse GWAS réalisée sur le panel de 148 accessions a permis d'identifier sept QTL de résistance supplémentaires, localisés sur les chromosomes 1, 4, 5, 8, 9 et 12.

Ce travail est le premier à identifier les régions génomiques impliquées dans la réponse de la tomate aux SDP. Elle met en évidence de nouveaux QTL, des gènes candidats associés à la défense induite et une nouvelle source de résistance à l'oïdium facilement transférable vers la tomate cultivée. Ces résultats ouvrent la voie au développement de variétés combinant résistance génétique et forte réactivité aux SDP, afin de réduire durablement l'usage des produits phytosanitaires.

Mots clés : tomate, stimulateurs de défense des plantes, GWAS, oïdium, mildiou, résistance, variabilité génotypique

Abstract

Plants are continuously exposed to biotic stresses caused by viruses, bacteria, fungi, and oomycetes. Although they have evolved efficient defense mechanisms, the effectiveness of these responses varies among cultivars and is increasingly challenged by climate change. Plant resistance inducers (PRIs) represent a promising strategy to sustainably enhance crop resistance. However, their efficacy strongly depends on the plant genotype, and the genetic basis underlying this variability remains poorly understood. The objective of this thesis was to identify the genetic determinants controlling tomato accession responses to PRIs. To achieve this, a core collection of 148 tomato accessions was evaluated using two commercial PRIs: Belvine®, applied against *Phytophthora infestans* (late blight agent) and phenotyped under tunnel conditions in 2024 and 2025, and Planticare®, applied against *Oidium neolycopersici* (powdery mildew agent) and evaluated under greenhouse conditions using the same experimental design.

Genome-wide association studies (GWAS) identified twelve quantitative trait loci (QTLs) associated with tomato responses to PRIs, distributed across chromosomes 2, 3, 4, 5, 7, 8, and 12, depending on the PRI, the target pathogen, and the experimental environment. Gene expression analyses further showed that both PRIs induced the upregulation of several defense-related genes, including pathogenesis-related (PR) genes (*PR-1*, *PR-2*, *PR-4*, *PR-5*, and *PR-8*), oxidative stress-related genes (*Peroxidase* and *Glutathione S-transferase*), and a key gene involved in ethylene biosynthesis (*ACCO*). Among the candidate genes identified within the confidence intervals of the Belvine®-associated QTLs, three belonged to these induced defense gene families (*PR2*, *Peroxidase*, and *Glutathione S-transferase*), supporting their potential role in PRI responsiveness.

Beyond PRI responses, this work also investigated the genetic architecture of tomato resistance to *Oidium neolycopersici*. The evaluation of 31 accessions representing six species of the genus *Solanum* revealed *Solanum cheesmaniae* LA0421 as a new source of resistance to powdery mildew. An F_2 population derived from a cross between LA0421, and the susceptible accession Rose de Berne (*S. lycopersicum*) was subsequently developed. Combining QTL-seq and linkage mapping enabled the identification of a novel QTL on chromosome 9. The SNP is located at the QTL peak maps within the *ETO1* gene (Regulator of ethylene biosynthetic process), making it a strong candidate for the observed resistance. Owing to the close genetic relationship between *S. cheesmaniae* and cultivated tomato, together with the availability of KASP markers, this QTL represents a valuable resource for marker-assisted breeding. Furthermore, the GWAS conducted on the panel of 148 accessions identified seven additional resistance QTLs distributed across chromosomes 1, 4, 5, 8, 9, and 12.

This work is the first to identify genomic regions associated with tomato responsiveness to plant resistance inducers. It provides new QTLs controlling PRI responses, identifies candidate genes involved in induced defense mechanisms, and uncovers a novel source of powdery mildew resistance readily transferable to cultivated tomato. Together, these findings provide new opportunities for developing tomato cultivars combining genetic resistance with enhanced responsiveness to PRIs, thereby contributing to more sustainable disease management and reduced reliance on chemical plant protection products.

Key words: tomato, Plant resistance inducers, GWAS, powdery mildew, late blight, resistance, genotypic variation

Remerciements

Je tiens à exprimer ma profonde gratitude à toutes les personnes qui ont contribué, de près ou de loin, à la réalisation de cette thèse.

Mes premiers remerciements vont à mes encadrants, **Marc Bardin** et **Bernard Caromel**, pour leur accompagnement, leurs conseils et leur confiance tout au long de ces années. Je remercie particulièrement Bernard Caromel et son épouse **Pascale** pour leur bienveillance au-delà du cadre professionnel, notamment pour leur chaleureuse invitation lors des fêtes de Noël. Merci à Pascale, qui m'a appris à réaliser mes premiers gâteaux.

Mes remerciements vont également aux membres de mon **CSI** : **Fabienne Vailleau**, **Vincent Segura** et **Clémentine Lepinay**, pour leurs recommandations et leurs retours constructifs, qui ont permis d'améliorer significativement mon travail.

Ma gratitude s'étend également aux **membres du jury**, pour avoir accepté d'évaluer mon travail et pour l'attention portée à cette thèse.

Je souhaite également remercier **Magali Duffaud, Jean-François (JF)**, **Anne**, et **Esther** pour leur aide précieuse lors des expérimentations et des manipulations au laboratoire. Ma gratitude va aussi à **Sonia**, **Frédérique**, **Jacques** et **Laure**, dont le soutien en analyses bio-informatiques a été essentiel.

JF, c'est sûr que tu me manqueras. Je n'aurai plus quelqu'un pour me demander à quelle heure je suis arrivée, ni pour m'appeler « **Aawaaaaaa** », à quoi je répondais à voix basse « **Jfffffff** ». Bref, tous ces moments me manqueront.

Un grand merci à **Bénédicte** et **Véronique** pour l'organisation des réunions doctorants, leurs échanges constructifs et leurs conseils toujours avisés. Je remercie également **Lili**, **Fadilatou** et **Simone**, stagiaires impliquées et motivées, ainsi que **Octave** et **Mélanie** pour leur participation aux expérimentations.

Je tiens à saluer les équipes expérimentales **A2M** et **IE** de la pathologie (Prophyle) : **Mara**, **Stéphane**, **Nathalie**, **Jérémie**, **Michel** et **Cindy**. Merci pour votre aide dans les expérimentations, avec une mention toute particulière à **Michel**, qui a toujours pris le plus grand soin des plantes ;

avec lui, on ne peut qu'aimer travailler sur des expérimentations où les plantes sont toujours magnifiques.

Merci à l'équipe **ReDD** pour les gourmandises de 10 h, qui ont souvent apporté douceur et convivialité dans nos journées de travail.

Je remercie chaleureusement le groupe **GQ du GAFL** : **Mathilde**, **Émilie**, **Morgane**, **Bénédicte** et **Véronique**, pour leurs conseils avisés sur mes analyses de données, ainsi que l'ensemble du groupe des doctorants pour les échanges, le soutien et la solidarité.

Je souhaite remercier **Alan Kergunteuil**, **Romain Larbat**, **Florent Magot** et **Matthieu Gaucher** pour leur disponibilité et leur soutien dans l'analyse des données métabolomiques et transcriptomiques. Merci à **Pascale Favier** et **Christelle Lacroix** pour leur patience face à mes demandes de missions et de congés parfois tardives.

Merci au projet **CapZéroPhyto** pour le financement de ma thèse. À tous les membres de la **Patho** et du **GAFL**, merci pour votre soutien, votre collaboration et les belles années passées à vos côtés.

Je remercie les équipes **Aquavit** pour l'organisation d'activités conviviales qui ont contribué à créer un environnement de travail agréable et soudé.

Je souhaite exprimer un remerciement tout particulier à **ma mère**, pour ses prières, son soutien indéfectible et son amour constant. Sans elle, je n'aurais jamais pu prétendre à une thèse. Je remercie également toute **ma famille** pour leur présence et leurs encouragements.

Enfin, je tiens à remercier **mes amis**, en particulier **Yapi Ignace II** (le « jorant ») et **Dosso Moussa**, qui ont su m'écouter, accueillir mes contraintes et toujours me remonter le moral lorsque j'en avais besoin.

Scientific communications

Peer review papers

Awa Sangaré, Jean-François Bourgeay, Anne Massire, Aurelie Berard, Damien Hinsinger, Bernard Caromel, Marc Bardin. New powdery mildew resistance QTL found on chromosome 9 in *Solanum cheesmaniae*. Submitted to the journal Theorical and Applied Genetics. (July 9, 2026)

Documents Intended for Transfer

RMT BESTIM. Immunité des plantes : quelles stratégies pour l'optimiser dans un contexte agroécologique ? (2025)

Communications in International congress

Awa Sangaré, Jean-François Bourgeay, Magali Duffaud, Bernard Caromel, and Marc Bardin. Genetic resistance and plant defense induction to control tomato powdery mildew caused by *Oidium neolycopersici*. Scientific Seminar, Ghana, (November 2023) [Oral presentation]

Awa Sangaré, Jean-François Bourgeay, Magali Duffaud, Bernard Caromel, and Marc Bardin. Implication of the overexpression of PR genes and oxidative stress genes in the efficacy of natural substances against *Oidium neolycopersici*. PhD student Symposium - International Organization for Biological Control (IOBC), Barcelone, Spain, (April 23, 2024) [Oral presentation]

Awa Sangaré, Jean-François Bourgeay, Anne Massire, Marc Bardin, and Bernard Caromel. Breeding tomatoes to improve their responsiveness to biocontrol products against powdery mildew? The XXI Eucarpia Meeting of the Tomato Working Group, Plovdiv, Bulgarie, (June 10-13, 2025) [Oral presentation]

Awa Sangaré, Jean-François Bourgeay, Magali Duffaud, Marc Bardin, and Bernard Caromel. Does plant genetics shape the protective efficacy of biocontrol against diseases? IOBC/the 16th Meeting of the Working Group on Integrated Control in Protected Crops, Mediterranean Climate, Podgorica, Montenegro, (September 02-05, 2025) [Oral presentation]

Communications in National congress

Awa Sangaré, Jean-François Bourgeay, Bernard Caromel, and Marc Bardin. Genetic effect of tomato response to the application of biocontrol products. Journées Jean Chevaugeon - mycologie,

National Meeting on Plant Pathology and Mycology, Aussois, France, (January 18, 2024) [Oral presentation]

Awa Sangaré, Jean-François Bourgeay, Magali Duffaud, Bernard Caromel, and Marc Bardin. The efficacy of Plant resistance inducers (PRI) in controlling *Phytophthora infestans* and *Oidium neolycopersici* is heritable in tomato. Réunion des sélectionneurs, GAFL, Avignon (March 12, 2024) [Oral presentation]

Awa Sangaré, Jean-François Bourgeay, Anne Massire, Magali Duffaud, Marc Bardin, and Bernard Caromel. Genetic resistance and Plant Resistance induction (PRI) to control three Tomato Pathogens: *Oidium neolycopersici*, *Phytophthora infestans* and *Botrytis cinerea*. Scientific Seminar, GAFL, Avignon, (March 2024) [Oral presentation]

Awa Sangaré, Jean-François Bourgeay, Magali Duffaud, Bernard Caromel, and Marc Bardin. Genetic mapping of tomato responses to plant resistance inducers (Talk: Fous de la Tomate Conference, National tomato research meeting, Angers, France, October 3, 2024) [Oral presentation]

Awa Sangaré, Jean-François Bourgeay, Anne Massire, Magali Duffaud, Marc Bardin, and Bernard Caromel. La réponse de la tomate aux stimulateurs de défense des plantes a une composante génétique. Réunion des sélectionneurs, GAFL, Avignon (January 30, 2025) [Oral presentation]

Awa Sangaré, Jean-François Bourgeay, Anne Massire, Marc Bardin, and Bernard Caromel. Tomato responses to plant resistance inducers a genetic component. Young Researchers Meeting of BAP department, Bordeaux, France, (May 15, 2025) [Oral presentation]

Awa Sangaré, Jean-François Bourgeay, Anne Massire, Magali Duffaud, Marc Bardin, and Bernard Caromel. The response of tomato plants to biocontrol can be improved through breeding - Late blight. Journées Jean Chevaugeon - mycologie, National Meeting on Plant Pathology and Mycology, Aussois, (January 12-16, 2026) [Oral presentation]

Awa Sangaré, Jean-François Bourgeay, Anne Massire, Magali Duffaud, Marc Bardin, and Bernard Caromel. La réponse de la tomate aux stimulateurs de défense des plantes a une composante génétique. Réunion des sélectionneurs, GAFL, Avignon (January 29, 2026) [Oral presentation]

Liste des abréviations

ABA	Abscisic Acid
BABA	β-aminobutyric acid
BLINK	Bayesian-information and Linkage-disequilibrium Iteratively Nested Keyway
BLUP	Best Linear Unbiased Predictor
bp	Base Pair
DAMP	Damage-associated molecular pattern
DIS	Disease index
ET	Ethylene
ETI	Effector-triggered immunity
GAPIT	Genomic Association and Prediction Integrated Tool
GWAS	Genome-Wide Association Studies
GxE	Genotype by Environment
H2O2	Hydrogen Peroxide
HR	hypersensitive response
IPM	Integrated Pest Management
ISR	Induced Systemic Resistance
JA	Jasmonic acid
Kb	Kilo base pair
LC-MS	Liquid Chromatography - Mass Spectrometry
LD	Linkage Disequilibrium
LOD	Logarithm of the Odds
MAF	Minor allele frequency
MAMP	Microbe-associated molecular pattern
Mb	Mega base pair
MM4LMM	Min-Max algorithm for the restricted Maximum Likelihood inference in gaussian variance component Mix Model
NBS-LRR	Nucleotide-Binding Site Leucine-Rich Repeat
NTC	Untreated Control
PCA	Principal Component Analysis
PCR	Polymerase chain reaction
POX	Peroxidase
PP	Percentage of protection
PR	Pathogenesis-Related proteins
PRI	Plant resistance inducer
PRR	Pattern-recognition receptor
PTI	Pattern-triggered immunity
PVE	Phenotypic variance explained
QTL	Quantitative trait loci
RLK	receptor-like kinase
RLP	receptor-like protein
ROS	Reactive Oxygen Species
SA	Salicylic acid
SAR	Systemic Acquired Resistance
SC	Self compatibility
SI	Self incompatibility
SL	Solanum lycopersicum
SLC	Solanum lycopersicum var. cerasiforme
SLL	Solanum lycopersicum var. lycopersicum
SNP	Single Nucleotide Polymorphism
SP	Solanum pimpinellifolium

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General introduction

The transition towards more sustainable agricultural systems has become a major challenge at the interface between food security, environmental preservation, and climate change adaptation. In this context, the ability of plants to mobilize their defense mechanisms against pathogens emerges as a key lever for ensuring sustainable crop protection. Consumers are increasingly attentive to the origin of products, their production methods, and their impacts on the environment and biodiversity. These expectations impose new constraints on agricultural systems, which must reconcile potentially conflicting objectives, including productivity, sustainability, and the conservation of natural resources.

At the same time, climate change is intensifying the constraints faced by agriculture. Increased climatic variability affects cropping practices, yields, fruit quality, and plant health. In particular, the development of plant diseases is favored by the extension of periods conducive to pests and pathogens, the increase in their populations, and their geographic redistribution (Bebber et al. 2013; Chaloner et al. 2021; Ma et al. 2025).

In this context, the exclusive reliance on chemical plant protection products is increasingly limited, both in terms of long-term effectiveness and environmental impacts. There is therefore a need to rethink crop protection strategies by promoting more sustainable approaches. The concept of agroecological immunity aligns with this perspective by aiming to enhance the natural defense capacities of plants rather than directly eliminating pests and pathogens relying on the integration of multiple endogenous and exogenous factors (*Immunité des plantes*, 2025).

Plants possess defense mechanisms that enable them to respond to pathogen attacks. However, the effectiveness of these mechanisms varies among species and genotypes and may be insufficient. This variability is partly explained by domestication and breeding processes, which have led to a reduction in natural resistance capacities in favor of agronomic traits such as yield, fruit qualities, and adaptation to cropping systems (Lin et al. 2014; Chen et al. 2015). In addition, pathogens evolve rapidly and can also overcome plant defense mechanisms rapidly.

In this framework, improving plant immunity represents a major challenge. This can be achieved through different approaches, including the introgression of resistance genes and the

induction of natural defense mechanisms. It is now well established that plant immunity can be effectively stimulated against biotic stresses. Plant resistance inducers (PRI), also referred to as resistance inducers, represent a promising alternative and are receiving increasing attention (Zhu et al. 2024; Jiang et al. 2026).

Numerous studies have demonstrated their ability to activate defense mechanisms and confer protection against a wide range of pathogens. However, within a given species, cultivars exhibit contrasting responses to PRI. Several studies have shown that the effectiveness of these stimulators, as well as their modes of action, strongly depend on the plant genotype (Walters et al. 2011, 2013). These observations suggest that the response to PRI constitutes a complex trait, likely quantitative in nature, whose underlying determinants remain largely unknown.

Studies conducted in wheat and apple indicate that this response is partly controlled by genetic factors (Bénéjam et al. 2021; Soleimani et al. 2024). Furthermore, the effectiveness of PRI varies depending on the plant species and the targeted pathogen. These findings suggest that the genetic mechanisms involved may depend on the specific pathosystem. However, the limited number of available studies does not yet allow for a comprehensive assessment of this variability or for determining the extent to which these results can be generalized across different plant–pathogen systems.

Tomato represents a particularly relevant model for addressing these questions. It has been extensively used to investigate the mechanisms underlying PRI activity and to assess how their effectiveness varies across plant genotypes. (Sharma et al. 2010; Satková et al. 2016; Gupta et al. 2020; Catoni et al. 2022; Tanaka et al. 2024). However, most studies conducted so far have relied on a limited number of genotypes, thereby restricting the scope of their conclusions.

Moreover, no study has yet comprehensively explored the variability of the response to PRIs within a large panel of tomato genotypes, nor thoroughly characterized the underlying genetic architecture of this response. Therefore, understanding the genetic determinants that govern the response to PRIs in tomato, as well as their potential specificity depending on the PRI used and the pathogen targeted, represents a key challenge for the development of more sustainable crop protection strategies. Furthermore, the strong synteny between the tomato genome and those of other Solanaceae, including *Nicotiana*, pepper, eggplant and potato, makes it possible to

effectively extrapolate the genetic knowledge gained from tomato to related species. This high level of genomic conservation substantially enhances the translational value of the findings, fully aligning with the objectives of the CapZéroPhyto project.

This thesis is conducted within the framework of the CapZéroPhyto project, which aims to transpose the concept of ecological immunity to crop protection, in species belonging to the Rosaceae and Solanaceae families. Conceived as an integrative initiative, the project seeks to develop innovative protection strategies based on the combined use of immune-related levers capable of modulating plant defense mechanisms. Five complementary levers are investigated, individually and in combination: genetic resistance, companion plants, biocontrol solutions acting through defense stimulation (PRI), UV-C flashes, and nitrogen supply. Apple and tomato serve as model crops for the study of their major pests and pathogens, before extending the approach to other Rosaceae (peach, strawberry) and Solanaceae (pepper).

Because this work is embedded within the CapZéroPhyto project, the following section provides an overview of current knowledge on tomato, its major pathogens, and biological approaches to plant protection. It concludes with the objectives of this thesis and the research strategy developed to address them.

Chapter 1

Literature Review

1. Tomato and its main diseases

Origin and diversity of tomato

The cultivated tomato (*Solanum lycopersicum*) belongs to the Solanaceae family, which also includes potato, eggplant, pepper, and tobacco. It is the most extensively cultivated species within this family (Faino et al. 2012). The species originates from the Andean region of South America, encompassing present-day Chile, Bolivia, Peru, Ecuador, and Colombia. Wild tomato relatives occupy a remarkably broad ecological range, from coastal areas at sea level to elevations exceeding 3,300 m. They occur in arid coastal lowlands and adjacent *lomas*, where pacific winds provide minimal moisture; in isolated valleys of the high Andes; and in hyper-arid environments such as the Atacama Desert in northern Chile (Knapp & Peralta 2016).

Within the genus *Solanum*, tomato is classified in the *Lycopersicon* section. This section comprises the cultivated tomato *S. lycopersicum* and 12 closely related wild species, including *S. arcanum*, *S. cheesmaniae*, *S. chilense*, *S. chmielewskii*, *S. corneliomulleri*, *S. galapagense*, *S. habrochaites*, *S. huaylasense*, *S. neorickii*, *S. pennellii*, *S. peruvianum*, and *S. pimpinellifolium* (Peralta et al. 2008; Rodriguez et al. 2009). Among these, *S. cheesmaniae* and *S. galapagense* are endemic to the Galápagos Islands. Two domesticated forms are recognized within *S. lycopersicum*: semi-domesticated and cherry-sized fruit-bearing tomato *S. lycopersicum* var. *cerasiforme* and fully domesticated and large fruit-bearing tomato *S. lycopersicum* var. *lycopersicum* (Lin et al. 2014; Gao et al. 2019). Among the large-fruited *S. lycopersicum* var. *lycopersicum*, there are early cultivars (vintage), modern domesticates and modern cultivars (fresh market), indicative of breeding goals that have targeted large-fruited tomatoes (Blanca et al. 2015; Yu et al. 2025).

Phylogenetic analyses indicate that species within the *Lycopersicon* section segregate into two principal groups: pigmented-fruited species (bearing red or yellow-orange fruits), which exhibit strong genetic affinity, and green-fruited species (Rodriguez et al. 2009; Mohan et al. 2016; Yu et al. 2025). Integrating morphological and molecular evidence, Peralta et al. (2008) further organized these 13 species into four major groups: the *Lycopersicon* group comprises the domesticated tomato and its closest red- yellow-fruited relatives (*S. pimpinellifolium*, *S. galapagense*, *S. cheesmaniae*). The other members of tomato species belonging to Neolycopersicon (*S. pennelli*), Eriopersicon (*S. chilense*, *S. corneliomulleri*, *S. habrochaites*, *S.*

huaylasense, *S. peruvianum*) and Arcanum (*S. arcacum*, *S. neorickii*, *S. chmielewskii*) groups have green fruits. Tomato has other wild related species belonging to section *Juglandifolia* (*S. ochranthum*, *S. juglandifolium*) and *Lycopersicoides* (*S. lycopersicoides*, *S. sitiens*) (Li et al. 2023; Peralta et al. 2008), completing the number of species to sixteen.

All 16 tomato wild species or relatives exhibit significant differences in their reproductive habits compared with those of cultivated tomato accessions (Bedinger et al. 2011; Baek et al. 2015, 2016; Du et al. 2025): 1) cultivated tomato varieties and certain wild species (such as *S. cheesemaniae*, *S. galapagense*, and *S. neorickii*) are self-compatible (SC), with non-exserted or slightly exserted stigmas, making them primarily autogamous (self-pollinating); 2) some wild species (*S. chilense*, *S. corneliomulleri*, *S. huaylasense*, and four closely related species) are self-incompatible (SI) and possess floral traits, such as strongly exserted stigmas, that promote cross-pollination (allogamy); 3) the remaining wild tomato species exhibit facultative reproductive strategies and can be divided into two categories: (i) SC species displaying floral characteristics that favor cross-pollination, such as larger flowers and exserted stigmas (e.g., *S. pimpinellifolium* and *S. chmielewskii*), and (ii) predominantly SI species, with some SC populations typically found at the margins of their geographic distributions (namely *S. arcanum*, *S. habrochaites*, *S. pennellii*, and *S. peruvianum*)

There are four types of crossability among tomato species (Baek et al. 2015; Muñoz-Sanz et al. 2020; Du et al. 2025): first, the four self-compatible (SC) species (*S. lycopersicum, S. pimpinellifolium*, *S. cheesmaniae*, *S. galapagense*) show full bidirectional compatibility, producing viable seeds in both direct and reciprocal crosses; second, the five SC species (cultivated tomato, *S. pimpinellifolium*, *S. cheesemaniae*, *S. galapagense*, and *S. neorickii*), can act as maternal parents in crosses with self-incompatible (SI) species, but reciprocal crosses fail, indicating unilateral incompatibility; third, similar unidirectional incompatibility occurs with *S. lycopersicoides*, where successful crosses only occur when it is the pollen donor; and finally, these patterns generally follow the "SI × SC" rule, where SI stigmas reject SC pollen, although notable exceptions exist in species such as *S. chmielewskii* and *S. pennellii*.

Domestication and its impact on genetic diversity

Tomato domestication is generally described as a two-stage process (Blanca et al. 2012): one occurred in South America, where the wild species *S. pimpinellifolium* (SP) was domesticated,

giving rise to a semidomesticated intermediate, S. *lycopersicum* var. *cerasiforme* (SLC) and a second transition occurred in Mesoamerica, where SLC was further domesticated into the fully domesticated *S. lycopersicum* var. *lycopersicum* (SLL). Recently, findings from the sequencing of native SLC populations suggest a more complex history (Razifard et al. 2020). They suggest that the presence of SLC in South America is prior to human presence. So, they may have originated prior to its domestication, as many characteristic traits of South American tomatoes are derived from this species. SLC is therefore thought to have declined or disappeared locally after partially domesticated forms migrated northward.

During the early stages of domestication, selection was primarily directed toward traits desirable for human consumption and industrial processing, including fruit quality and yield. These selective pressures markedly enhance phenotypic diversity. At present, tomato fruits exhibit an extensive spectrum of colors (yellow, green, red, etc.), sizes, shapes, and flavors. In addition to fruit-related traits, substantial variation is also observed in plant architecture (Blancard et al. 2009). Collectively, these selection processes have profoundly shaped the genomic landscape of tomato and have been instrumental in the development of improved cultivated varieties (Wang et al. 2024).

Despite this remarkable phenotypic diversification, cultivated tomato has undergone severe genetic bottlenecks throughout domestication, resulting in an exceptionally narrow genetic base that represents only approximately 4.5% of the total genetic variation present across tomato-related species (Ashrafi et al. 2009; Blanca et al. 2012, 2015; Sahu and Chattopadhyay 2017; Razifard et al. 2020). Recent genomic analyses of accessions within the Lycopersicon clade have further documented a reduction in gene and polymorphism numbers (The 100 Tomato Genome Sequencing Consortium et al. 2014; Gao et al. 2019; Blanca et al. 2022). Throughout domestication, strong directional selection not only reduced overall genetic diversity but also altered numerous traits in cultivated tomato (Huang et al. 2022). Genes associated with stress tolerance and disease resistance were either lost or negatively selected, resulting in a depletion of alleles essential for adaptation, particularly those conferring resistance to pathogens (Gao et al. 2019).

Tomato genetic and genomic

The tomato is a diploid species composed of 12 pairs of chromosomes (2n = 24), whose genome has been fully sequenced and annotated. The tomato genome ranges from 730 to 1,200 Mb in size, of which nearly 77% consists of heterochromatin, mainly located in the pericentromeric regions (Sim et al. 2012). Recent advances in high-throughput sequencing technologies have enabled the generation of high-quality genome assemblies for *S. lycopersicum* as well as for several of its wild relatives.

To date, more than 75 genome assemblies are available in the National Center for Biotechnology Information (NCBI) database (http://www.ncbi.nlm.nih.gov), with at least one reference genome for each species (**Fig. 1**). The list of their reference genomes is presented in **Table 1**. Among these resources, the reference genome (SL4.0) from the cultivar "Heinz 1706" produced by the Tomato Genome Consortium represents a major milestone in tomato genomics. This high-quality assembly, first published in 2012, provides a comprehensive framework for gene annotation and comparative genomic analyses. It has enabled the identification of key genes involved in fruit development, ripening, and plant-pathogen interactions, and serves as a cornerstone for genetic and breeding studies in tomato.

This reference genome is available on the Sol Genomics Network (SGN) platform, which integrates genome assemblies and associated genomic tools for various tomato species and cultivars (https://solgenomics.net/). The Heinz 1706 reference genome has been updated several times using multiple sequencing technologies, resulting in fewer assembly gaps and higher completeness (Hosmani et al. 2019; Su et al. 2021). In 2022, the Heinz 1706 genome was further improved using PacBio high-fidelity long reads and Hi-C data (SL5.0) (Zhou et al. 2022). The SL5.0 genome (801.8 Mb) shows higher continuity (contig N50 of 41.7 Mb in SL5.0 vs 5.5 Mb in SL4.0; few gaps (31 in SL5.0 vs 259 in SL4.0) and improved base accuracy (quality value of 49 vs 39) compared to the SL4.0 genome (782.5 Mb) (Wang et al. 2024). Despite substantial improvement, the SL5.0 genome remains incomplete, with 31 unfilled gaps and unresolved centromeres and telomeres. More recently, Heinz 1706 reference genome was sequenced from telomere to telomere, enabling a complete sequencing of Heinz 1706 (Shi et al. 2026; Chen et al. 2026). The novel genome sequence (SL-T2T) had a total size of 831.45 Mb with a contig N50 size of 68.49 Mb and composed of 12 chromosomes with all 24 telomeres. The genome was completely

assembled except for the 45S rDNA region on chromosome 2. A tomato pangenome and two super-pangenomes (chromosome scale and telomere to telomere) have been also sequenced (Gao et al. 2019; Li et al. 2023a; Shi et al. 2026).

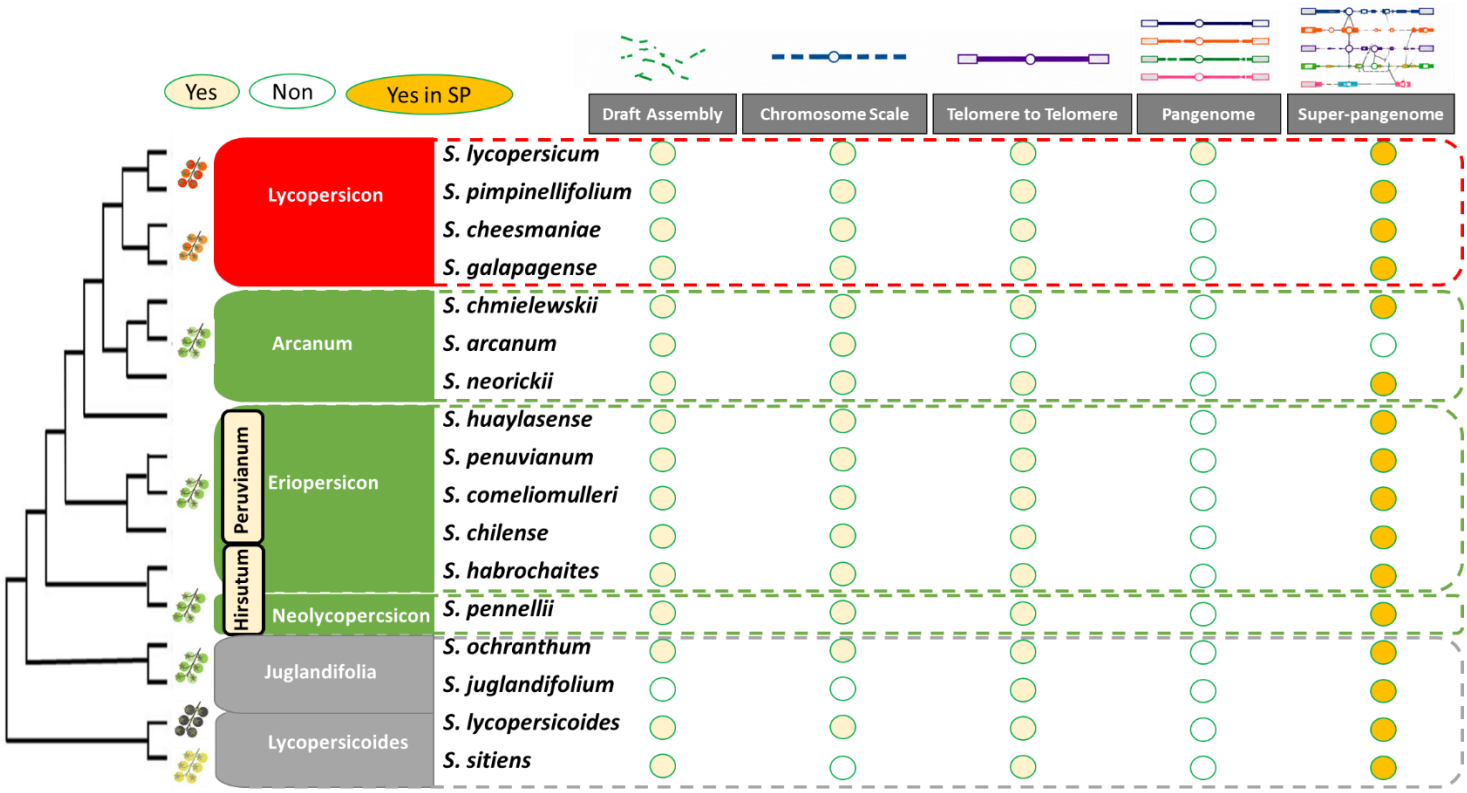


Fig. 1 Current stage of tomato and wild relative genome sequencing (modified from Rose et al. (2025))

High-quality genome assemblies have further enabled advances in comparative genomics, transcriptomics, and genome editing, strengthening our ability to study and improve tomato biology (Bolger et al. 2014). In addition, the overall conservation of genomic regions between tomato and other Solanaceae species provides a useful framework for transferring genetic knowledge across related crops (Anderson et al. 2010; The Tomato Genome Consortium 2012; Peters et al. 2012).

This genomic framework enables the analysis of genetic variation, particularly SNPs, widely used in QTL mapping, genome-wide association studies (GWAS), and genomic selection, facilitating the dissection of complex traits, the identification of key loci and candidate genes, and

accelerating breeding programs through marker-assisted selection (Lin et al. 2014; Gao et al. 2019; Campos et al. 2021; Li et al. 2023a; Prasanna et al. 2023).

Table 1 **List of tomato chromosome-scale reference genomes available to study tomato genomics**

Species	Accession/ species	Genome size (Mbp)	Sequencing technology	Citations
Super-pangenome	All species except *S. arcacum*		PacBio	(Shi et al. 2026)
	Shab, Schi, Sper, Scor, Sneo, Schm, Spim, Sgal, Slcoid, SlycC, Slycl cv. M82		PacBio	(Li et al. 2023a)
Pangenome	Slycl, Spim, Sche, Sgal		PacBio	Gao et al., 2019
S. lycopersicum	Heinz 1706 (SL4.0)	782.52	PacBio	(Hosmani et al. 2019)
	Heinz 1706 (SLT1.0)	799.09	PacBio, HiFi	(Su et al. 2021)
	Heinz 1706 (SL-T2T)	831.45	PacBio	(Chen et al. 2026)
	Micro-Tom	832.8	PacBio Sequel II	(Shirasawa and Ariizumi 2024)
S. pimpinellifolium	LA1589	833	PacBio Sequel	(Han et al. 2024)
S. galapagense	LA0436	802	PacBio	(Li et al. 2023a)
S. cheesmaniae	LA0166	835.6	PacBio Sequel IIe	(Iki et al. 2025)
S. chmielewskii	LA1028	770	PacBio	(Li et al. 2023a)
S. arcanum	LA2157	855.68	Oxford Nanopore PromethION; Illumina HiSeq	(Jiang et al. 2023)
S. neoriiki	LA0747	778	PacBio	(Li et al. 2023a)
S. chilense	LA1972	901	Illumina HiSeq, PacBio	(Molitor et al. 2024)
S. habrochaites	LA1777	960	PacBio	(Li et al. 2023a)
S. pennellii	LA0716	926	Pacbio	(Powell et al. 2022)
	LA0716	1100	PacBio	(Li et al. 2023a)
S. peruvianum	LA0446	867	PacBio	(Li et al. 2023a)
S. lycoprtsicoides	LA2951	1152	PacBio	(Powell et al. 2022)
	LA2951	1200	PacBio	(Li et al. 2023a)
S. stiens	LA1974	1200	Illumina HiSeq; PacBio; BioNano Irys	(Molitor et al. 2021)
S. ochranthum	PI23051901	956.17	PacBio	(Wu et al. 2023)
S. comeliomulleri	LA1331	876.9	PacBio	(Li et al. 2023a)

Shab: *S. habrochaites*; Schi: *S. chilense*; Sper: *S. peruvianum*; Scor: *S. comeliomulleri*; Sneo: *S. neoriiki*; Schm: *S. chmielewskii*; Spim: *S. pimpinellifolium*; Sgal: *S. galapagense*; Slcoid: *S. lycoprtsicoides*; SlycC: *S. lycopersicum var cerisiforme*; Slycl: *S. lycopersicum var lycopersicum*

1.1. **Tomato economic importance**

Tomato is one of the most important crops worldwide, recognized for its high economic value and its ability to supply multiple market segments. It is also among the most widely consumed vegetables globally. In France, households consume more than 13.8 kg of tomatoes per year (Baros et al., 2022), and it is one of the most grown vegetables in home gardens. In 2024, global tomato production was estimated at 188.5 million metric tons, with a harvested area of about 5.12 million

hectares, underscoring its substantial agronomic and economic relevance (FAO 2025). Production is dominated by China, which alone accounts for 61.61 million tons, around 33% of global output. Moreover, the top ten tomato-producing countries collectively contribute nearly 74% of total world production, reflecting a strong geographical concentration of supply (**Fig. 2**).

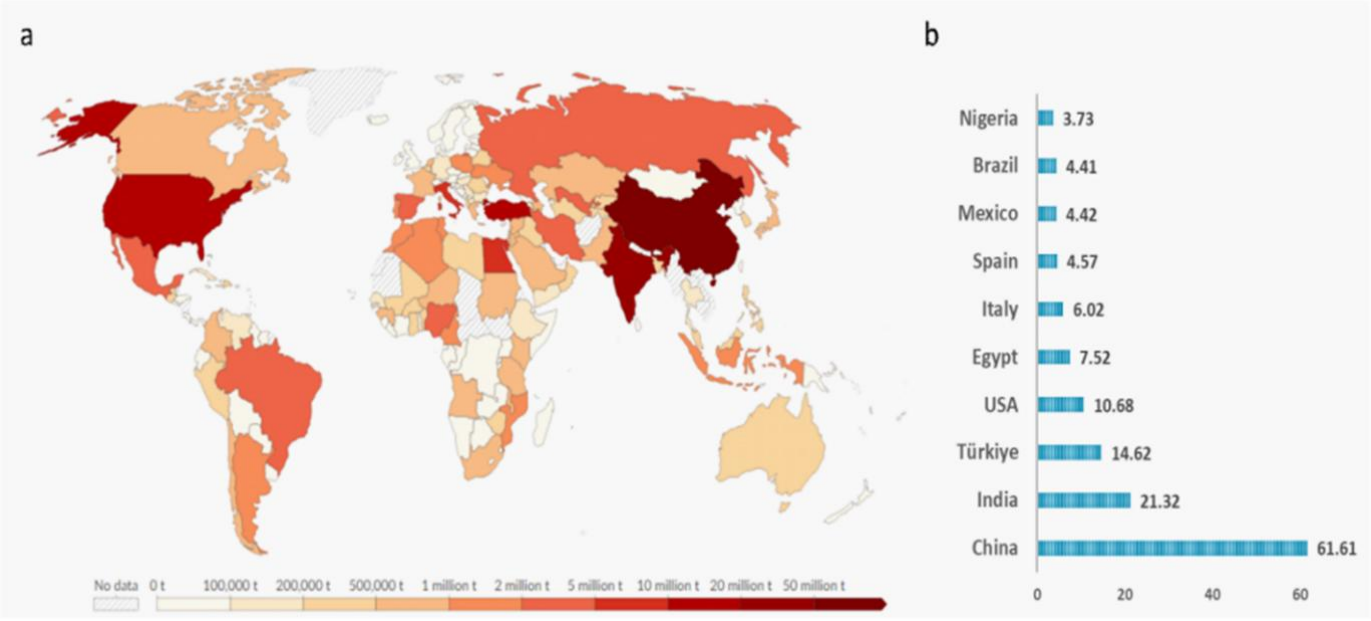


Fig. 2 Global distribution and country-specific quantities. a) World map showing the total quantity produced or emitted per country, expressed in tons (t). The color gradient indicates increasing quantities from light beige (low values) to dark red (high values), with darker shades representing higher totals. b) Bar chart illustrating the quantities (in million tons) for the ten countries with the highest values: China, India, Türkiye, USA, Egypt, Italy, Spain, Mexico, Brazil, and Nigeria. Source: Food and Agriculture Organization of the United Nations (2025) - with major processing by Our World in Data.

Tomato is cultivated worldwide under highly diverse growing conditions, both in terms of climate and production practices. Two main types of tomato varieties are produced: determinate varieties for the processing industry, grown exclusively in open fields, and indeterminate varieties for the fresh market, which can be cultivated under a wide range of conditions, from open-field systems to highly controlled greenhouse environments (Blancard et al. 2009; Causse et al. 2020). Tomato production also represents a key source of income for smallholder farmers in Asia, Africa, and Latin America.

1.2. **Tomato diseases**

The tomato hosts over 200 species of a wide variety of pests and pathogens that can cause significant economic losses (Bai and Lindhout 2007). Plant-parasitic nematodes, particularly

Meloidogyne spp. and *Pratylenchus spp.*, pose a devastating threat to vegetable crops (Orlando et al. 2020; Azlay et al. 2023; Costa et al. 2025). More than 100 viruses affect tomatoes globally, causing mosaic patterns, leaf distortions, and stunted growth. Common viruses include Tomato mosaic virus (ToMV), Tomato yellow leaf curl viruses (TYLCV), Tomato spotted wilt virus (TSWV), Cucumber mosaic virus (CMV), Tobacco mosaic virus (TMV), and Tomato brown rugose fruit virus (ToBRFV). Bacterial pathogens such as *Pseudomonas syringae pv. tomato* (bacterial speck), *Ralstonia solanacearum* (bacterial wilt) and *Clavibacter michiganensis* ssp. *michiganensis* (bacterial canker) are major constraints to tomato production worldwide (Ahmed et al. 2022; Aldayel and Sallam 2026). Tomato crops are also threatened by numerous fungal and oomycetes pathogens of various ecological types, including soil-borne, air-borne and vascular fungi. Greenhouse cropping systems where climate is generally warm, humid and wind free, make it a particularly suitable system for the development of diseases, especially those caused by fungi and fungi-like pathogens. Among them, *Botrytis cinerea*, *Oidium neolycopersici,* and *Phytophthora infestans*, are major concerns on tomato crops in France under greenhouse and field conditions. They were selected as model pathosystems in this study. These diseases cause significant yield losses, and *B. cinerea* and *P. infestans* occur at similar developmental stages under comparable climatic conditions.

1.2.1. Botrytis cinerea

Biology, Diversity, and Host Range

Botrytis cinerea is considered as the second most important plant pathogen in terms of economic significance and academic contribution to the understanding of plant-pathogen interactions (Dean et al. 2012). It is a necrotrophic fungus belonging to the Ascomycota phylum of fungi classified within the Sclerotiniaceae family. The genus *Botrytis* comprises 38 species (Garfinkel 2021) that can infect more than 1400 plant species (Elad et al. 2016). Most of the species have a narrow host range and are even host-specific (Plesken et al. 2015; Steentjes et al. 2021). Among this genus, *B. cinerea* (teleomorph *Botryotinia fuckeliana*), is one of the species holding a wide range of host plants estimated at 586 plant species, including most greenhouse crops (Elad et al. 2016). However, population genomic analyses suggest genetic bases potentially underlying specialization to tomato (Mercier et al. 2021). The pathogen infects many fruits such as strawberries, grapes, blackberries, blueberries, kiwis, raspberries, tomatoes, pears, apples and

many others (Richards et al. 2021; Brito et al. 2021). It can also originate from non-agricultural habitats (Bardin et al. 2018). In addition to its wide host range, typical features of *B. cinerea* are a necrotrophic, macerating mode of infection, abundant production of asexual macroconidia on the surface of colonized host tissue, and formation of melanized sclerotia for long-term survival and sexual reproduction (Plesken et al. 2015). The pathogen also exhibits a high level of phenotypic and genotypic diversity, differentiated by fungicide resistance profiles, host plant specialization, and the season during which isolates are collected (Decognet et al. 2009; Ajouz et al. 2011; Bardin et al. 2014, 2018; Ullah et al. 2024).

Infections and symptoms

Grey mold, caused by *Botrytis cinerea*, develops under moderate temperatures and high humidity, with optimal infection occurring around 15-25 °C and relative humidity above 80% (Ciliberti et al. 2015; Li et al. 2023b; Mahmoud et al. 2023). This airborne necrotrophic pathogen infects leaves, stems, fruits, and flowers, and can damage plant tissues during growth, storage, and transport (Bardin and Gullino 2020; Brito et al. 2021). Disease symptoms include white to grey mycelial growth on necrotic tissues, followed by abundant conidiation and, in some isolates, the formation of melanized sclerotia enabling long-term survival (**Fig. 3**) (Holz et al. 2007; Cheung et al. 2020). The fungus produces an array of phytotoxic metabolites such as botrydial, botcinin, and oxalic acid, which promote severe diseases through cell death and maceration of plant tissue (Bi et al. 2023).

Infection occurs primarily through wounds, although direct penetration through the cuticle is possible (Van Kan 2006). The disease affects both greenhouse and open-field crops, but greenhouse conditions, characterized by sustained high humidity, greatly favor pathogen development (Bardin and Gullino 2020). In greenhouse tomato, severe stem infections often arise

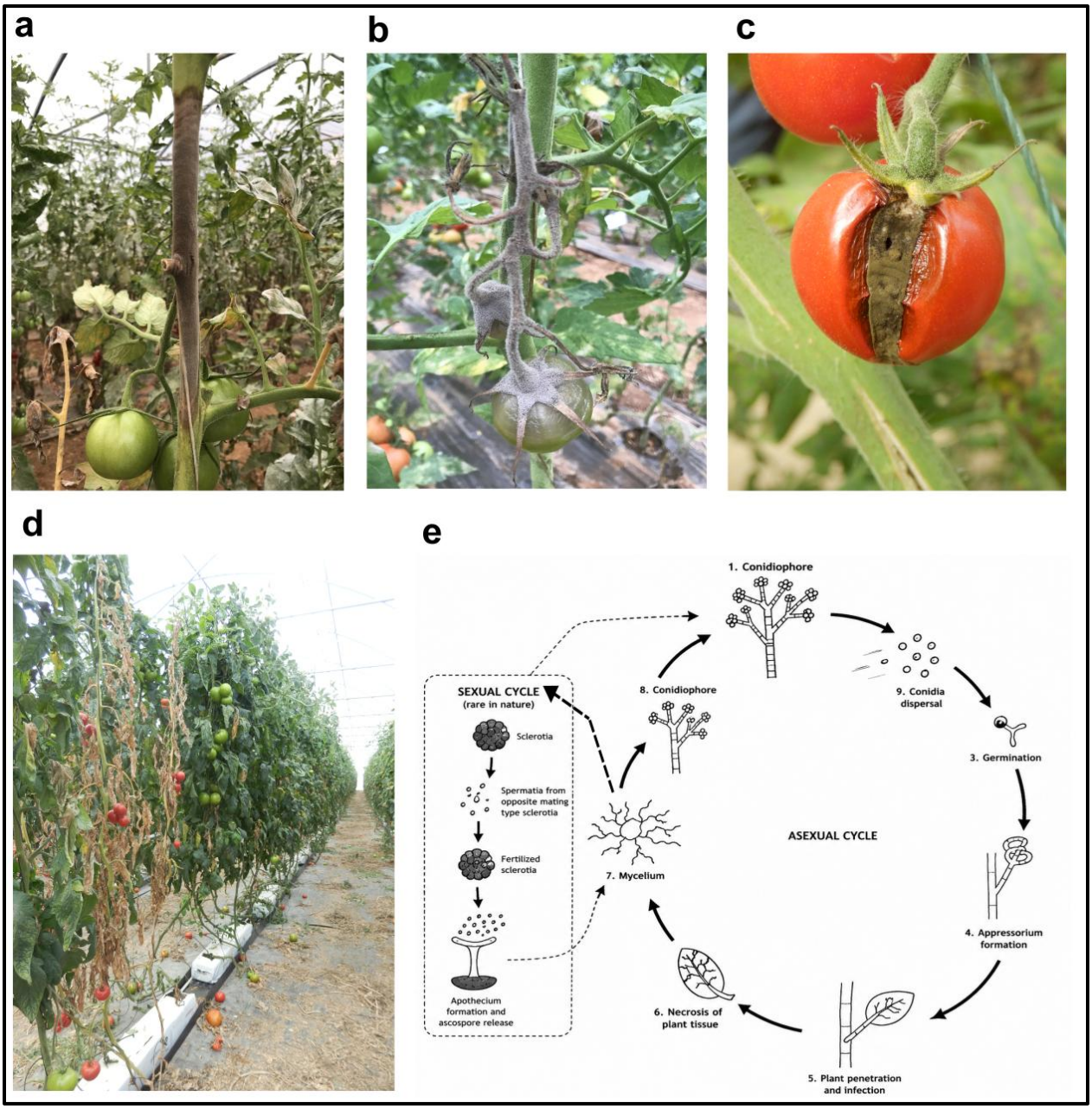


Fig. 3 *Botrytis cinerea* symptoms and life cycle. a-d) grey mold on tomato stem, Fruit cluster rachis, fruit and leaves, respectively. e) *B. cinerea* life cycle, adapted from (Cheung et al. 2020)

at pruning wounds, while post-harvest soft rot is common on mature fruits. The "ghost spot" symptom on unripe fruits reflects an effective host defense yet still compromises marketability (Williamson et al. 2007). Under favorable conditions, *Botrytis cinerea* can cause total crop loss, and global annual losses are estimated at 20-50% (Hao et al. 2017; Brito et al. 2021; Dwivedi et al. 2024). Several management strategies can be implemented to limit the development of *Botrytis cinerea*, including the use of synthetic fungicides. However, the continuous and intensive

application of these fungicides for crop protection has led to major drawbacks, such as high costs, handling difficulties, residual toxicity, environmental concerns, risks to human health, and the emergence of fungicide-resistant strains.

1.2.2. Oidium neolycopersici

Powdery mildew poses a major threat worldwide to various greenhouse crops (Bardin and Gullino 2020). On tomato and other Solanaceae, the disease can be caused by two ascomycete fungi, *Leveillula taurica* (Massire et al. 2025) and *Oidium neolycopersici* (syn. *Pseudoidium neolycopersici*) (Kiss et al. 2001). The latter fungus was first reported in the Netherlands in 1986 (Paternotte, 1988) and in United Kingdom in 1987 (Fletcher et al. 1988) and has since spread widely throughout the world (Lebeda et al. 2017). Herbarium specimens collected since 1947 (Kiss et al. 2001) and the resistance observed in wild tomato species (Lindhout et al. 1994; Ciccarese et al. 1998) suggest that this pathogen has existed for a long time. However, it is only in the last 40 years that it has been recognized as a major cause of outbreaks in tomatoes, likely due to the intensive global production of tomatoes in greenhouses. In recent years, in Europe, it has become the primary causal agent of powdery mildew in tomatoes.

This obligate biotrophic parasite has never been reported to undergo sexual reproduction in natural environments, nor has sexual reproduction been successfully induced or demonstrated under laboratory conditions (Kiss et al. 2001). Apart tomato, *O. neolycopersici* appears to have a broad host range, although reports on its exact host spectrum remain inconsistent (Lebeda et al. 2017). Based on artificial inoculation techniques, this fungus has been shown to infect more than 60 plant species belonging to 13 families, including members of the Solanaceae and Cucurbitaceae (Whipps et al. 1998; Lebeda et Mieslerová 1999; Jones et al. 2001). Field observations of wild and cultivated plants exhibiting symptoms of the disease have shown that this species can occur on a wide range of host plants, including non-solanaceous species such as *Carica papaya*, *Senna bicapsularis*, *Moringa oleifera*, and Euphorbiaceae (Liu et al. 2015b; Mukhtar and Van Peer 2018; Liu et al. 2019b; Mukhtar et al. 2019; Xu et al. 2020).

The disease is characterized by white, powdery spots that expand and coalesce, eventually covering large areas of plant tissue and becoming necrotic. Symptoms are mainly visible on the upper surface of leaves (**Fig. 4**) (Bardin and Gullino 2020). The pathogen penetrates directly through the cuticle and cell walls (Blancard et al. 2009), and subsequently develops within the

epidermal cells of the leaf blade. It infects tomato plants throughout their life cycle, from seedlings to mature plants. Although fruits are not directly infected, the disease can induce etiolation, premature leaf senescence, wilting, and a reduction in both fruit quality and size (Kim et al. 2018).

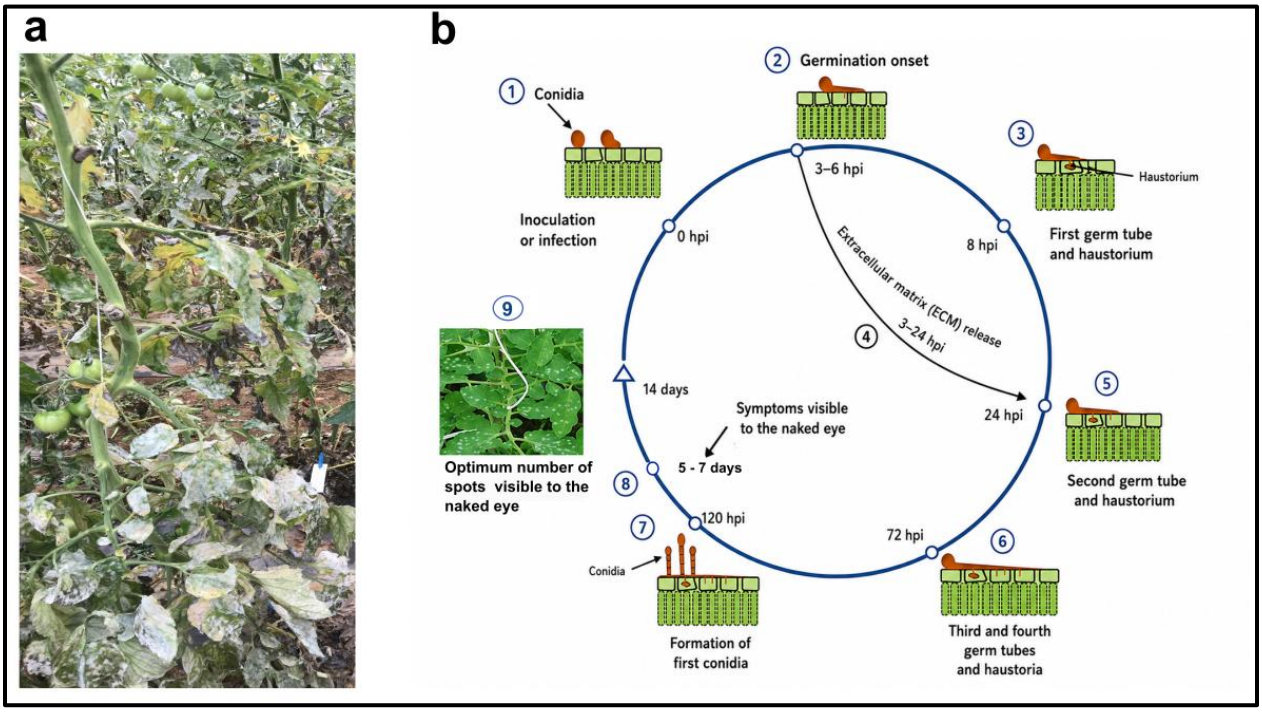


Fig. 4 *Oidium neolycopersici* symptoms and life cycle. a) symptoms of a highly infested plant. b) development and infection cycle of *Oidium neolycopersici* based on information reported by Lebeda et al. (2017) and Liu et al. (2008). 1: leaf infection by *O. neolycopersici* conidia at 0 hpi. 2: germination onset between 3 and 6 hpi. 3: formation of a short primary germ tube ending in a primary appressorium at 8 hpi, from which a primary haustorium develops. 4: extracellular matrix (ECM) deposition occurs between 3 and 24 hpi. 5: formation of a secondary appressorium and secondary haustorium at 24 hpi. 6: formation of the third and fourth germ tubes up to 72 hpi. 7: formation of the first conidiophores between 89 and 120 hpi. 8: first symptoms visible to the naked eye between 5 and 7 days after inoculation. 9: maximum development of *O. neolycopersici* on leaves, with optimum number of lesions visible appearing after 14 days

Oidium neolycopersici exhibits high genetic diversity (Jankovics et al. 2008) and significantly impacts tomato production under both greenhouse and field conditions. Yield losses in field-grown tomatoes can exceed 50% (Qinggui Lian et al. 2022; Park et al. 2022).

Environmental conditions strongly influence powdery mildew disease development. Optimal temperatures for conidial germination, pathogen development, and sporulation range from 20 to 25 °C, with moderate relative humidity (above 60%) (Fletcher et al. 1988; Jacob et al. 2008;

Blancard et al. 2009; Mieslerová and Lebeda 2010; Lebeda et al. 2017). However, excessive moisture is generally detrimental to disease development (Jarvis 1977). Under favorable conditions, symptoms typically appear within 5-7 days after infection (Liu et al. 2008). The disease is mainly controlled by fungicides such as sulfur.

1.2.3. Phytophthora infestans

Phytophthora infestans is an oomycete responsible for late blight in tomato and potato. It primarily infects plants belonging to the *Solanaceae* family, particularly tomato and potato. This hemibiotrophic pathogen can reproduce both sexually and asexually (**Fig. 5e**), depending on its life-cycle stage and the geographical region. The pathogen may affect the leaves, stems, and fruits on tomato and potato (**Fig. 5a-d**), additionaly to tubers on potato.

Sexual cycle of *P. infestans*

Phytophthora infestans is heterothallic with two distinct mating typesand can produce sexual resting spores (oospores). Sexual reproduction requires the presence of both mating types, A1 and A2. However, self-fertility phenomena have been described and may be induced by various factors, such as the presence of mating types from other *Phytophthora* species, culture aging, certain growth media (oat flakes, Lima beans, V8 juice, etc.), physical injuries, or the presence of *Trichoderma viride*, which stimulates the production of gametangia (Fry 2008). Oospores are survival structures that allow the pathogen to survive between seasons in the soil and under adverse climatic conditions (Turkensteen et al. 2000; Babadoost and Pavon 2013). Generally, oospores are produced in the stems because they decay less rapidly to blight (Mosa et al. 1991). However, in tomatoes, Rubin & Cohen (2004), found that oospores were formed in fruits and that *P. infestans* may be transmitted by seeds. In addition, the coexistence of both mating types promotes increased genetic diversity within populations, the development of fungicide resistance, and the breakdown of resistance genes in host plants (Fry 2008). Only in the center origin, Mexico, both mating types could be found at equal frequencies. In other regions the main type is A1. It seems like the type A2 was introduced after A1 with migration from Mexico with the first report in Switzerland, then in other Northern European countries, Japan, and Korea (Panthee et al. 2024).

Asexual cycle of *P. infestans*

The asexual cycle enables dramatically rapid population growth in susceptible host tissue. Infection is initiated when sporangia encounter a moist leaf surface. Depending on the temperature, sporangia can germinate directly at higher temperature (optimum between 20-25 °C) or release biflagellate zoospores at lower temperature (optimum between 10-15 °C) (Fry 2008). Following appressorium formation, infection tubes emerge and penetrate epidermal cells, cuticles, or stomata. Plant infection is also facilitated by wounds. Optimal conditions for its development correspond to a relative humidity above 90% and moderate temperatures, although it can develop across a wide range of temperatures, from cold to warm (Blancard et al. 2009; Chen et al. 2014). The most important factor for its development is an alternation of warm days and cool nights. According to the isolate, the optimum temperature is variable (Fry 2008). After a latent period of 3 to 4 days new sporangia are formed and emerge through the stomata on the lower leaf surface and spread to infect new plants via wind and splash dispersal (Fry 2008).

Tomatoes affected by late blight disease exhibit small, water-soaked brownish lesions along the margin of the leaves, which may have chlorotic borders. These lesions rapidly expand, causing necrosis in entire leaves. Under high humid conditions, the pathogen produces sporangiophores and sporangia on the surface of the infected tissue. This results in the whitish color of sporulation on the lower surface of infected leaves, characteristic of the disease. The leaf lesions coalesce and cause the foliage to appear blighted as the disease progresses quickly. While the disease primarily causes foliar blight, it can also result in black discoloration in the stem and shoulders of ripe fruits at a later stage (**Fig. 5a-d**). Infected fruits show brown lesions on their surface that may enlarge and destroy the entire tomato fruit (Schumann & D'Arcy 2000). In the absence of control measures, yield losses can reach up to 100% in a few days (Fry 2008; Panthee et al. 2024). Fungicides are widely used to control this pathogen, including copper-based compounds. Numerous products with diverse active ingredients are registered worldwide (Blancard et al. 2009).

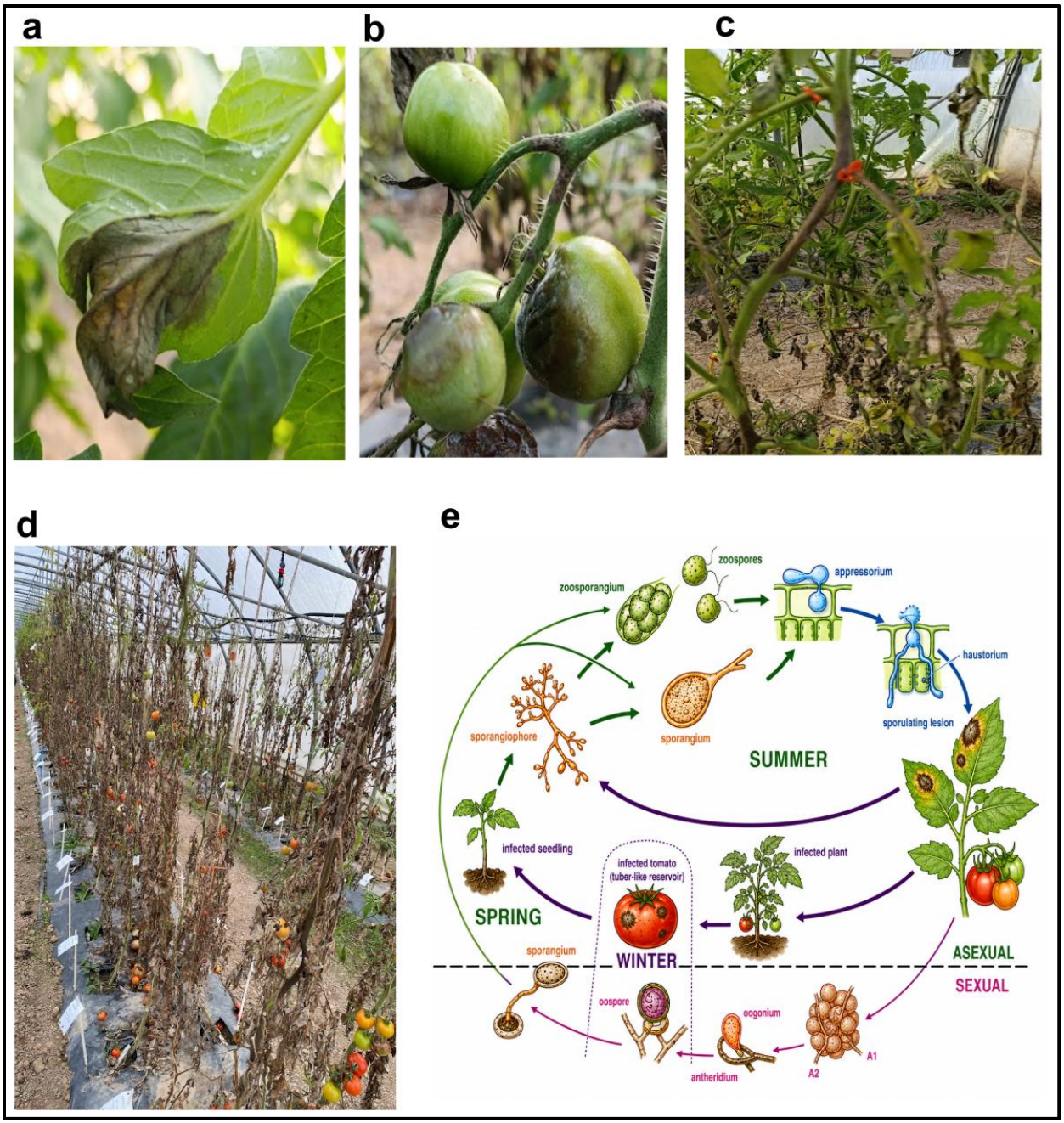


Fig. 5 *Phytophthora infestans* symptoms and life cycle. a-c) late blight on tomato leaf, fruit and stem, respectively. d) photo of a tunnel infested by *Phytophthora infestans* one month after inoculation. e) *P. infestans* life cycle, adapted from (Drenth and Guest 2004) with chatGPT

Host specialization of *Phytophthora infestans* strains on tomato and potato

A host specialization of *P. infestans* has been demonstrated. Some strains show increased virulence on tomato, whereas others are better adapted to potato (Kröner et al. 2017). Genetic differences between isolates collected from tomato and potato have also been reported in Ecuador and Colombia (Oyarzun et al. 1998; Olave-Achury et al. 2022). Additional differences involve the distribution of mating types and fungicide resistance: potato-derived isolates are generally of the A1 mating type and tend to exhibit higher fungicide resistance, while tomato-infecting populations show a more balanced distribution of A1 and A2 mating types (Knapova and Gisi 2002; Srisawad et al. 2023), even if it depends on the country.

Specialization, however, does not exclude pathogenicity toward the alternate host. Legard et al. (1995), initially showed that a tomato-nonaggressive isolate caused much less disease on tomato than on potato, whereas a tomato-aggressive isolate caused severe disease on both hosts but was significantly more damaging on tomato. This interpretation is nuanced by Olave-Achury et al. (2022), who reported that potato isolates were able to infect both host species in host-preference assays, suggesting that potato-derived isolates may still pose a risk to nearby tomato crops even when host specialization is observed. Another specificity is at phenotypic level. It has been reported that the phenotype of a tomato-specialized strain is characterized by an extended biotrophic phase (Vega-Sánchez et al. 2000), sometimes accompanied by abundant sporulation occurring before the onset of necrosis (Smart et al. 2003).

1.3. Disease control

Several management strategies can be implemented to limit pathogen development, including the use of copper-, sulfur- based compounds, synthetic fungicides and various physical methods. However, the continuous and intensive application of synthetic fungicides for crop protection has resulted in major drawbacks, such as high costs, residual toxicity, environmental concerns, risks to human health, and/or the emergence of fungicide-resistant strains (Pokorny et al. 2016; Olave-Achury et al. 2022).

Several studies have reported the development of fungicide resistance in *P. infestans*, with resistance levels varying across geographic regions and fungicide classes (Hermann and Gisi 2012; Saville et al. 2015; Olave-Achury et al. 2022; Abuley et al. 2023). Grünwald et al. (2006),

demonstrated directional selection toward metalaxyl resistance in Mexican populations. Similarly, Srisawad et al. (2023), reported metalaxyl-resistant strains in Thailand affecting both tomato and potato crops. Abuley et al. (2023), documented the first case of resistance to mandipropamid in Danish *P. infestans* populations. Importantly, resistance patterns are not uniform: Qin et al. (2016) reported a low risk of azoxystrobin resistance in Chinese populations, whereas variable sensitivity to mefenoxam has been observed among U.S. lineages (Saville et al. 2015).

Numerous studies have also reported *B. cinerea* resistance to fungicides (Amiri et al. 2014; Rupp et al. 2017; Harper et al. 2022). To date, no direct fungicide resistance has been reported for *O. neolycopersici*. Nevertheless, the continued use of fungicides with identical modes of action poses a substantial risk of resistance development, particularly given the broad host range of this pathogen (Jones et al. 2001) and its high genetic variability (Jankovics et al. 2008).

Therefore, to address these issues, there is a need to explore safe and environmentally friendly alternatives, such as genetic resistance and the induction of plant defense responses.

2. Genetic resistance

2.1. Mechanism of resistance

2.1.1. Plant-pathogen interaction

The plant immune response can be broadly divided into three phases: immune recognition, signal integration, and defense execution (**Fig. 6**) (Wang et al. 2022). Immune recognition represents the stage of plant-pathogen interaction during which plant receptors detect the presence of invading pathogens. It consists of two interconnected layers.

Immune recognition

The plant immune system can be divided into two immune systems based on their location: pattern-triggered immunity (PTI) and effector-triggered immunity (ETI) (Jones and Dangl 2006; Ngou et al. 2021; Dodds et al. 2024). The first layer corresponds to the basal immune system, which relies on the perception of conserved molecular signatures, microbe-associated molecular patterns (MAMPs), damage-associated molecular patterns (DAMPs), or microbial effectors, by pattern-recognition receptors (PRRs) located at the cell surface (Ngou et al. 2021, 2022). PRR-mediated recognition of MAMPs or DAMPs activates PTI, a defense response aimed at preventing

pathogen colonization of host tissues. Most PRRs have been identified as transmembrane receptor-like kinases (RLKs) and receptor-like proteins (RLPs), which frequently contain extracellular leucine-rich repeat (LRR) domains or lysin motifs (LysM) involved in ligand recognition (Liebrand et al. 2013; Tang et al. 2017; Ngou et al. 2022; Dodds et al. 2024). As RLPs lack a cytoplasmic kinase domain, it is hypothesized that they require RLKs to activate downstream signaling (Liebrand et al. 2013). The ectodomains of RLKs and RLPs are highly variable, enabling the recognition of a wide range of ligands, including steroids, peptides, polysaccharides, and lipopolysaccharides (Tang et al. 2017). PTI immune system provides partial resistance to biotrophic, hemibiotrophic, and necrotrophic pathogens.

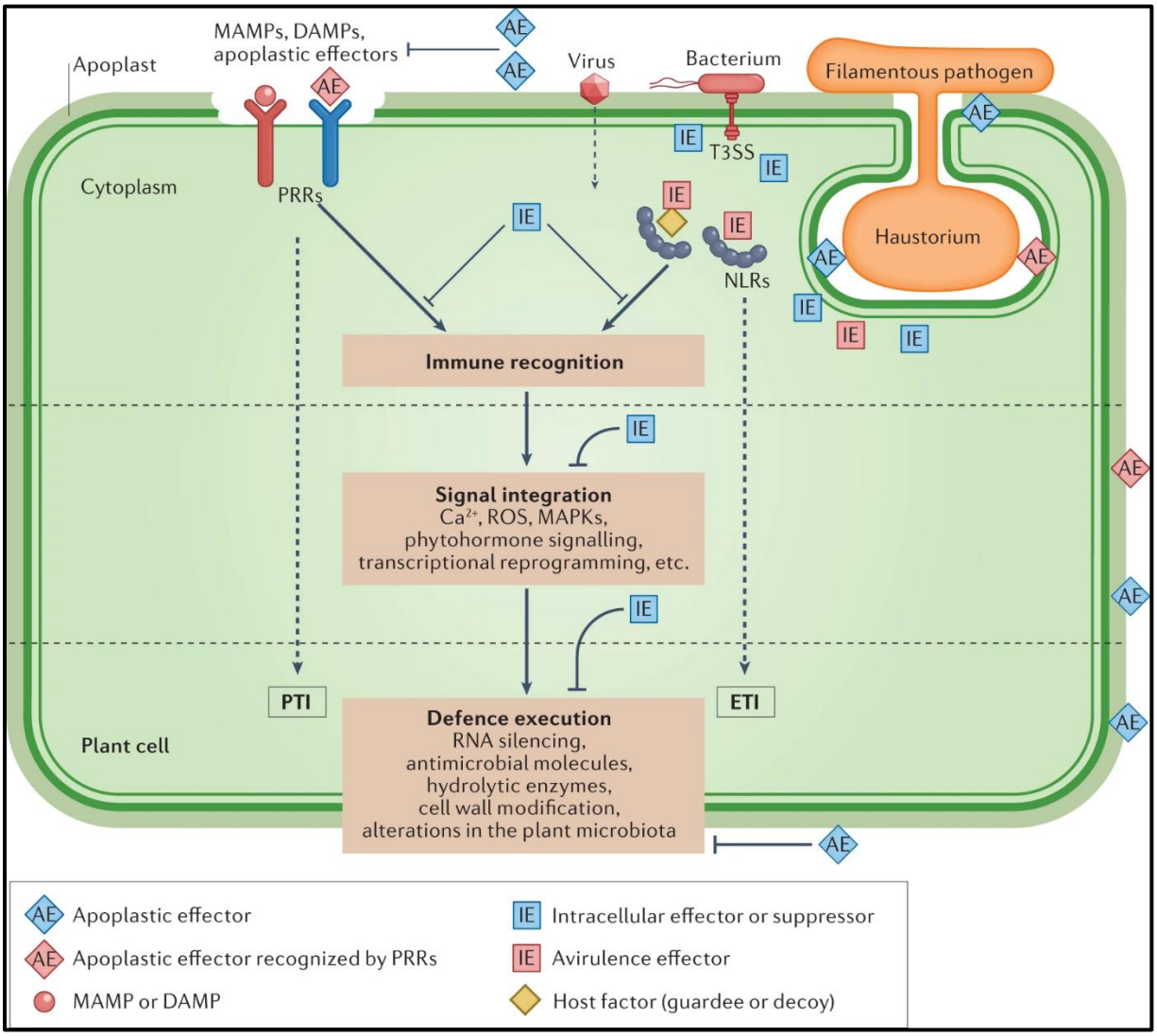


Fig. 6: Stepwise activation of plant innate immunity took from (Wang et al. 2022)

The second layer of immune recognition occurs inside the cell. Pathogen effectors delivered into host cells are detected by intracellular immune receptors, mainly members of the nucleotide-binding site leucine-rich repeat (NBS-LRR) protein family, encoded by most resistance (R) genes. Activation of NBS-LRR proteins, either through direct recognition of effectors or indirectly via effector-mediated perturbations of host targets, triggers effector-triggered immunity (ETI). ETI is generally considered an amplified form of PTI, resulting in strong disease resistance responses that often include localized programmed cell death and systemic defense signaling (Sun et al. 2019; Chai et al. 2023; Hudson et al. 2024). The ETI immune system confers total resistance, associated with qualitative resistance. However, NBS-LRR-mediated resistance is typically effective against obligate biotrophic and hemibiotrophic pathogens, but less effective against necrotrophic pathogens (Glazebrook 2005; Jones and Dangl 2006; Mengiste and Liao 2025).

While the terms PTI and ETI are widely used, their ability to accurately describe specific immune responses is increasingly questioned (Ngou et al. 2022). For example, some transmembrane receptors can perceive apoplastic effectors and trigger ETI-like responses (Thomma et al. 2011; Ngou et al. 2022; Dodds 2023). Thus, ETI is not restricted to NLR-mediated intracellular effector recognition but can also involve RLP-mediated detection of apoplastic effectors. In tomato, race-specific resistance to *Cladosporium fulvum* (*Cf-2*, *Cf-4*, *Cf-5*, *Cf-9*), *Verticillium dahliae* (*Ve1*), and *Fusarium oxysporum f. sp. lycopersici* (*I*, *I-3*, *I-7*) is mediated by LRR-RLPs (Fradin et al. 2009; Catanzariti et al. 2015, 2017; Gonzalez-Cendales et al. 2016; De Wit 2016; Ngou et al. 2022; Hudson et al. 2024). Another example is *LepR3*, which confers race-specific resistance to *Leptosphaeria maculans* in *Brassica napus* (Larkan et al. 2013).

Originally, ETI was defined as the recognition of a specific avirulence protein by a matching R protein in a gene-for-gene relationship (Dodds 2023). However, it is now clear that single NLRs can detect multiple unrelated effectors (Ngou et al. 2022; Remick et al. 2023). For instance, *AtZAR1* indirectly recognizes *HopZ1a*, *HopF2*, *HopBA1*, *HopO1*, *HopX1*, and *AvrAC* from *Pseudomonas syringae* or *Xanthomonas campestris*, as well as *XopJ4* from *Xanthomonas perforans* (Wang et al. 2015; Laflamme et al. 2020). In barley, the Resistance Locus A (RLA) NLRs recognize a broad range of *Blumeria graminis* effectors (Lu et al. 2016) and can even detect races of wheat stripe rust (Bettgenhaeuser et al. 2021).

Due to these ambiguities, additional terminology has been introduced to classify immune responses according to different criteria: the type of receptor involved, such as PRR-mediated immunity and NLR-mediated immunity (Lacaze and Joly 2020) or the localization of the receptor, such as extracellularly triggered immunity and intracellularly triggered immunity (Van Der Burgh and Joosten 2019), or the broader distinction between surface-receptor-mediated immunity and intracellular-receptor-mediated immunity (Van Der Burgh and Joosten 2019; Ding et al. 2021).

Regardless of the mode of recognition, these immune responses rely on distinct activation mechanisms and early signaling components. For PTI and ETI, recent studies have shown that they mutually potentiate each other, leading to enhanced and robust resistance (Ngou et al. 2021).

Signal transduction

Once pathogens are recognized, plants activate a signaling cascade that transmits information from the plasma membrane to the nucleus, where defense-related genes are induced. Following stress perception, plants exhibit a rapid accumulation of intracellular Ca^{2+} and enhanced ROS production, which act as early messengers. These signals activate complex pathways such as the MAPK cascade, which phosphorylates and activates target proteins, including enzymes and transcription factors (WRKY, bHLH, MYB, bZIP, Hsf, ERF), to amplify the signal (Meng and Zhang 2013; Pitzschke 2015). This ultimately leads to the synthesis and/or signaling of defense hormones (salicylic acid (SA), jasmonic acid (JA), ethylene (ET), and abscisic acid (ABA)), large-scale reprogramming of gene expression, and the redirection of metabolic fluxes toward antimicrobial metabolite production (alkaloids, terpenoids, carbohydrates, phenylpropanoids, etc), among other defense responses (Meng and Zhang 2013).

Defense execution

The third phase, defense execution, involves a wide range of cellular responses aimed at restricting pathogen development. These responses include callose and lignin deposition, reinforcement of the cell wall, production of reactive oxygen species (ROS), activation of RNA interference mechanisms, secretion of proteases and protease inhibitors, accumulation of pathogenesis-related (PR) proteins, biosynthesis of secondary metabolites such as antimicrobial compounds, and rapid host cell death known as the hypersensitive response (HR).

These defense responses operate at two interconnected levels: locally at the site of infection and systemically throughout the plant. At the local level, plants strengthen their structural barriers through the deposition of callose and lignin, as well as through the production of phytoalexins and the induction of the hypersensitive response (HR) (Meng and Zhang 2013). Together, these mechanisms reinforce cell walls or trigger localized cell death, thereby limiting pathogen growth and spread.

Beyond these local defenses, uninfected tissues develop systemic resistance, resulting in enhanced protection against subsequent pathogen attacks. This systemic response can be divided into two main types: systemic acquired resistance (SAR), which is induced by pathogen infection, and induced systemic resistance (ISR), which is triggered by beneficial microorganisms.

Together, these local and systemic responses rely on a complex network of signaling molecules and defense components. The roles and functions of key players, including ROS, PR proteins, and defense-related hormones, are discussed in the following section

2.1.2. Reactive Oxygen Species (ROS)

During normal metabolic processes, plant cells produce a variety of reactive oxygen species (ROS), including the superoxide radical ($O_2^{\bullet -}$), hydrogen peroxide (H_2O_2), the hydroxyl radical (•OH), and nitric oxide (NO) (Nanda et al. 2010). These molecules are generated across multiple cellular compartments, including the apoplast, mitochondria, chloroplasts, peroxisomes, and cytosol, through metabolic processes and enzymatic activities, notably those mediated by respiratory burst oxidase homologs (RBOHs) (Baral and Brosché 2026).

Under biotic stress conditions, ROS production increases rapidly and transiently at infection sites, a phenomenon referred to as the oxidative burst. This accumulation represents a key early event in plant defense, contributing to pathogen inhibition while functioning both as antimicrobial agents and signaling molecules. ROS are involved in defense gene activation, the hypersensitive response (HR), cell wall protein cross-linking, phytoalexin production, callose deposition, and systemic acquired resistance (SAR) (O'Brien et al. 2012; Waszczak et al. 2018).

In tomato, resistance to *Oidium neolycopersici* has been associated with ROS accumulation (Bai et al. 2005; Lebeda et al. 2014; Sun et al. 2019). While ROS generally restrict infection by biotrophic and hemibiotrophic pathogens, they may conversely promote susceptibility to

necrotrophic pathogens such as *Botrytis cinerea*, which benefit from host cell death. This pathogen can tolerate or detoxify ROS, for instance through the secretion of antioxidant compounds such as mannitol (Patel et al. 2015). Furthermore, infection by *B. cinerea* induces ROS accumulation in host tissues, contributing to host cell death and thereby facilitating pathogen colonization (Nakajima and Akutsu 2014; Hao et al. 2017).

However, despite their beneficial roles in regulating responses to both biotic and abiotic stresses, excessive ROS accumulation can cause oxidative damage to DNA, RNA, proteins, lipids, and other redox-sensitive molecules, ultimately impairing aerobic metabolism and plant growth (Khan et al. 2020; Li et al. 2024). To maintain cellular redox homeostasis, plants have therefore evolved a complex antioxidant system to tightly regulate ROS levels. Among these, peroxidases (POXs) constitute a major group of enzymes involved in ROS metabolism (Lebeda et al. 2014). They exhibit a dual role in ROS homeostasis, functioning both as scavengers through H_2O_2-dependent oxidation of substrates and as contributors to ROS generation under specific conditions (Lebeda et al. 2014).

2.1.3. Pathogenesis-related proteins (PR proteins)

PR proteins constitute a diverse group of proteins induced upon pathogen attack or activation of plant defense signaling pathways. Transcripts encoding PR proteins accumulate rapidly following pathogen recognition, and they are commonly used as molecular markers of systemic acquired resistance (SAR), largely regulated by key phytohormones such as salicylic acid (Yang et al. 2018; Mishra et al. 2024).

PR proteins are classified into nineteen families (PR-1 to PR-19), including 17 verified and two hypothetical groups, based on their biochemical properties (Islam et al. 2023). The validated PR proteins are organized into 17 distinct families, designated PR1-PR17 (Van Loon et al. 2006; Sels et al. 2008; Ghorbel et al. 2021; Guo et al. 2022). These proteins have been extensively reviewed for their properties and functions (**Table 2**).

Functionally, PR proteins contribute to plant defense through multiple mechanisms, including direct antimicrobial activity (e.g., chitinases and β-1,3-glucanases that degrade fungal cell walls), reinforcement of structural barriers, and modulation of signaling pathways. Some PR proteins also amplify defense responses and contribute to the establishment of long-lasting immunity. Enhanced

Table 2 Recognized families of pathogenesis-related proteins (from Sels et al. 2008 and Han et Schneiter, 2024)

Family	Type member	Activity	Function/Properties	References
PR-1	Tobacco PR-1a	Immune signaling, Lipid-binding	• Antimicrobial • Abundant induced protein in the apoplast	(Chen et al. 2014b; Gamir et al. 2017; Breen et al. 2017; Han et al. 2023)
PR-2	Tobacco PR-2	β-1,3-Glucanase	• Antimicrobial • Cell wall degradation	(Levy et al. 2007; Balasubramanian et al. 2012; Perrot et al. 2022)
PR-3 PR-4 PR-8 PR-11	Tobacco P, Q Tobacco 'R' Cucumber chitinase Tobacco 'class V' chitinase	Chitinase (class I, II, IV, V, VI, VI) Chitinase class I, II Chitinase class III Chitinase class I	• Antimicrobial • Cell wall degradation • Synergistic with PR2	(Oyeleye and Normi 2018; Fukamizo and Shinya 2019; Poria et al. 2021)
PR-5	Tobacco S	Thaumatin-like Osmotin Zeamatin-like	• Antifungal • Glucan binding • Plasma membrane permeability • Sweet tasting • Anti-freeze activity	(Zhang et al. 2018; De Jesús-Pires et al. 2020; Sharma et al. 2022)
PR-6	Tomato Inhibitor I	Proteinase-inhibitor/MEROPS family	• Nematocidal • Insecticidal • Synergistic with PR13	(Ryan 1989; Terras et al. 1993; Sels et al. 2008; Grosse-Holz and Van Der Hoorn 2016; Rawlings et al. 2018)
PR-7	Tomato P_{69}	Subtilisin-like endoproteinase	• Antifungal • Dissociation of microbial cell wall • Phytocytokine signaling	(Figueiredo et al. 2018; Schaller et al. 2018; Yang et al. 2023a)
PR-9	Tobacco 'lignin-forming peroxidase'	Heme-containing peroxidase	• Lignin-forming peroxidase	(Passardi et al. 2004; Almag 2009; Liu et al. 2018b; Cesa
PR-10	Parsley 'PR1'	'Ribonuclease-like'	• Antimicrobial • Cytoplasmic protein • Related to Bet v 1, a major birch pollen allergen	(Radauer et al. 2008; Choi et al. 2012; Fernandes et al. 2013; Aglas et al. 2020)
PR-12	Radish Rs-AFP3	Defensin	• Antimicrobial • Induction of ion efflux • Interaction with fungal sphingolipids	(Terras et al. 1995; Thevissen et al. 2000; Sels et al. 2008; Tam et al. 2015; Parisi et al. 2019)
PR-13	*Arabidopsis* THI2.1	Thionin	• Antimicrobial • Membrane permeating • Synergistic with PR14	(Stec 2006; Sels et al. 2008; Tam et al. 2015; Höng et al. 2021)

Continued

Table 2 Recognized families of pathogenesis-related proteins (from Sels et al. 2008 and Han et Schneiter, 2024)

Family	Type member	Activity	Function/Properties	References
PR-14	Barley LTP4	Lipid-transfer protein	• Antimicrobial	(Sels et al. 2008; Liu et al. 2015a; McLaughlin et al. 2021; Gao et al. 2022; Melnikova et al. 2022)
PR-15	Barley OxOa (germin)	Oxalate oxidase	• Antimicrobial • ROS generation • Germin • Cupin family	(Mittler 2002; Dunwell et al. 2004; Farvardin et al. 2020; Joshi et al. 2021)
PR-16	Barley OxOLP	Oxalate oxidase-like		
PR-17	Tobacco PRp27	metalloprotease	• Poorly characterized	(Okushima et al. 2000; Christensen et al. 2002; Joshi et al. 2021; Morimoto et al. 2022)

resistance to pathogens is often observed upon PR protein overexpression, whereas their downregulation increases susceptibility to infection (Han and Schneiter 2024).

For instance, overexpression of *PR-1* enhances disease resistance against a broad range of pathogens (Sarowar et al. 2005; Floryszak-Wieczorek et al. 2015; Sun et al. 2019). In cacao, constitutive expression of *TcPR-4b* during *Moniliophthora perniciosa* infection has been reported to contribute to resistance (Pereira Menezes et al. 2014).

In addition to their role in biotic stresses, certain PR proteins, such as PR-1, PR-3, and PR-5, have been associated with increased tolerance to abiotic stresses, including heavy metals and salinity (Islam et al. 2023).

2.1.4. Plant hormones

Phytohormones are small molecules produced within plants that regulate diverse physiological processes, including plant defense. Among them, jasmonic acid (JA), salicylic acid (SA), ethylene (ET), abscisic acid (ABA), auxin/indole-3-acetic acid (Aux/IAA), gibberellins (GAs), cytokinins (CKs), and brassinosteroids (BRs) are involved in defense responses (Robert-Seilaniantz et al. 2011; Berens et al. 2017; Mengiste and Liao 2025). However, the most extensively studied in plant immunity are JA, SA, ET, and ABA. At the systemic level, SA is primarily associated with systemic acquired resistance (SAR), whereas JA and ET are linked to induced systemic resistance (ISR), and ABA modulates systemic responses depending on environmental context and stress conditions (Berens et al. 2017; Mengiste and Liao 2025).

In general, JA and ET are associated with resistance to necrotrophic pathogens and chewing insects, whereas SA is required for resistance to biotrophic and hemibiotrophic pathogens (Glazebrook 2005; Pandey and Somssich 2009; Mengiste and Liao 2025). However, this dichotomy is not absolute and depends on both pathogen lifestyle and host genetic background. For example, in tomato, resistance to *B. cinerea* is SA-dependent (Achuo et al. 2004; Van Loon et al. 2006), whereas resistance to the biotrophic pathogen *O. neolycopersici* involves distinct hormonal pathways depending on the gene: *Ol-4* relies on SA, *Ol-1*/*Ol-QTLs* on ET, and *ol-2* is compromised by JA deficiency (Sun et al. 2019). Several studies have demonstrated that ET also contributes to resistance against biotrophic and hemibiotrophic pathogens in *Arabidopsis thaliana*, tobacco, and rice (Shibata et al. 2010; Helliwell et al. 2013; Tintor et al. 2013).

Altogether, this highlights that hormone signaling in plant defense is complex, particularly due to extensive interactions among pathways (Berens et al. 2017). Interactions between SA and JA, ET, and ABA generate a regulatory network that is often dose-, species-, tissue-, and inducer-specific (Mishra et al. 2024).

SA, JA, and ET biosynthesis pathway (Fig. 7)

Salicylic acid: SA is synthesized via two pathways: the phenylalanine ammonium lyase (PAL) pathway (in the cytosol) and the isochorismate (IC) pathway (in chloroplasts), both of which utilize chorismate, the end product of the shikimate pathway, as a precursor (Dempsey et al. 2011; Berens et al. 2017). Thus, in eudicots, the IC pathway seems to be the major route for SA biosynthesis in immunity, but the PAL pathway also contributes (Berens et al. 2017).

Jasmonic acid: JA biosynthesis starts by the conversion of chloroplast membrane lipids to 12-oxophytodienoic acid (OPDA) via sequential enzymatic reactions of lipoxygenases (LOXs), allene oxide synthase (AOS), and allene oxide cyclase. OPDA is further converted to jasmonic acid via 2-oxo-phytodienoic acid reductases (OPRs). The isoleucine-conjugate of jasmonic acid (JA-Ile) is formed by JA amido synthetase 1 (JAR1) and is known as the most potent endogenous form of JA (Berens et al. 2017).

Ethylene: Two enzymatic steps are unique to ethylene biosynthesis: conversion of S-adenosyl-methionine (SAM), a common metabolic precursor, to 1-aminocyclopropane-1-carboxylic acid (ACC) by ACC synthase (ACS) and oxidative cleavage of ACC to form ethylene by ACC oxidase (ACO) (Berens et al. 2017).

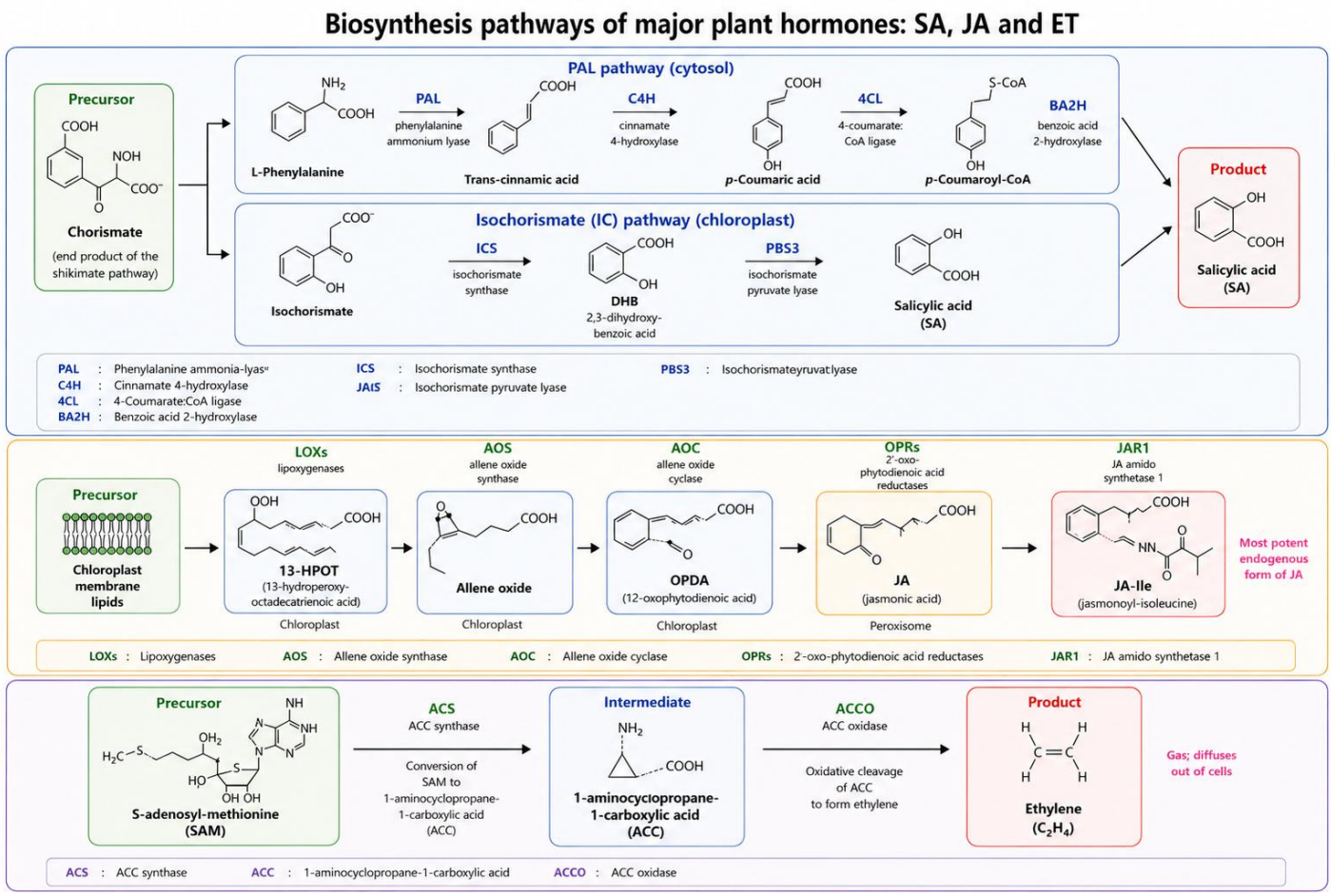


Fig. 7 Illustration of the biosynthesis pathway of the phytohormones salicylic acid (SA), jasmonic acid (JA) and Ethylene (ET). Adapted from (Berens et al. 2017)

2.2. Current knowledge on tomato resistance genes against pathogens

Resistance is defined as the ability of a plant to prevent or limit the infection or development of harmful agents such as pathogens, or pests. Genetic resistance falls into two categories: qualitative or quantitative, based on the number of loci involved (Corwin and Kliebenstein 2017).

Qualitative resistance is typically conferred by single major resistance (R) genes (monogenic resistance) with large effects, providing strong and specific protection against pests or pathogens. This type of resistance is generally more effective against biotrophic pathogens (Balint-Kurti 2019). Referred to as “gene-for-gene” resistance, it occurs when a pathogen effector (encoded by an avirulence gene, Avr) is recognized by a plant defense protein (encoded by a resistance gene, R). It is therefore described as “race-specific” resistance, as it relies on a specific interaction

between a specific R gene and a specific Avr gene, with recognition depending on the pathogen and the strain (Kushalappa et al. 2016).

In contrast, quantitative resistance is a more complex genetic basis conferred by several genes (polygenic resistance or quantitative trait loci (QTL)), which contribute, each with variable effect, to the phenotype of resistance to a pathogen (Niks et al. 2015; Pilet-Nayel et al. 2017). Quantitative resistance often confers a partial level of resistance to the plant and typically results in resistance that, while sometimes partial, is often more durable because it involves a diversity of mechanisms (Pilet-Nayel et al. 2017; Langlands-Perry et al. 2023). It governs the outcome of most host-pathogen interactions. Loci controlling quantitative resistance contain some of the same causal genes that mediate qualitative resistance, but the predominant mechanisms of quantitative resistance extend beyond pathogen recognition (Corwin and Kliebenstein 2017). Indeed, most causal genes for quantitative resistance may be genes involved in PAMP recognition responsible for basal defense (PTI), genes involved in defense signal transduction, genes regulating the phytoalexin synthesis, weak effect alleles of R-genes, genes regulating developmental phenotypes, or other genes not yet identified (Gou et al. 2023; Million et al. 2023).

2.2.1. Genetic sources of resistance

Most cultivated tomato are susceptible to disease. Cultivated tomato has a narrow genetic base due to domestication bottlenecks, limiting available resistance alleles (Gao et al. 2019; Blanca et al. 2022). Wild tomato relatives harbor extensive genetic diversity for disease resistance traits. These species have co-evolved with diverse pathogens in their native habitats and represent invaluable genetic resources for resistance breeding. Wild tomato species have been utilized as the source of disease resistance, except in a few cases. Resistance sources have been identified mainly in wild species particularly *S. pimpinellifolium*, *S. habrochaites*, *S. peruvianum*, and *S. pennellii* (Foolad 2007).

2.2.2. Tomato resistance genes and QTLs

Resistance genes are broadly grouped into two classes: typical resistance genes, which mainly recognize specific pathogenic proteins or effectors, include extracellular receptor-like kinases or proteins (RLKs/RLPs), intracellular receptors (NLRs) and atypical resistance genes, which possess

diverse architectures and functions associated with transcriptional regulation, kinases, translocation of substrates and hormone signaling (Sun et al. 2024; Wang et al. 2025).

Screening of wild tomato germplasm has revealed a rich reservoir of disease resistance, with approximately 40 major resistance traits identified across related species, spanning diverse pathogen and pest classes (Causse et al. 2020). Advances in molecular genetics have markedly accelerated the discovery and mapping of resistance determinants, leading to the characterization of resistance to nearly 30 major tomato diseases (Foolad et al. 2008). To date, more than 80 resistance genes and quantitative trait loci (QTLs) have been mapped across all 12 chromosomes, conferring resistance to bacteria, nematodes, viruses, fungi, and oomycetes (**Table 3**). While most resistance genes are dominant, a few recessive genes have been identified, including *pot-1*, *ty-5*, *py-1*, and *ol-2*, which confer resistance to potyviruses (PVY and TEV), Tomato yellow leaf curl virus (TYLCV), and *O. neolycopersici*, respectively (Bai et al. 2008; Doganlar et al. 1998; Lapidot et al. 2015; Ruffel et al. 2005). Additional recessive resistance has also been reported for Tomato spotted wilt virus (TSWV) (Foolad and Panthee 2012).

The resistance loci are predominantly derived from a limited subset of wild species, notably *S. pimpinellifolium*, *S. habrochaites*, *S. peruvianum*, and *S. pennellii*. In contrast, other wild relatives, including *S. chilense*, *S. cheesmaniae*, *S. neorickii*, and *S. arcanum*, remain comparatively underexplored. Nevertheless, targeted studies highlight their potential: six QTLs of resistance to *Alternaria solani* have been mapped in *S. arcanum* (LA2157) on chromosomes 1, 2, 5, 7, and 9 (Chaerani et al. 2007), while *S. cheesmaniae* and *S. neorickii* have been identified as sources of resistance to key fungal pathogens, including *Alternaria alternata*, *Verticillium dahliae*, *Oidium neolycopersici*, and *Botrytis cinerea* (Diwan et al. 1999; Robert et al. 2001; Bai et al. 2003; Finkers et al. 2008; Usami et al. 2017; Chavarro-Carrero et al. 2021).

Despite its relatively narrow genetic diversity, *S. lycopersicum* also harbors exploitable resistance variation. The major resistance gene *ol-2*, conferring complete resistance to *O. neolycopersici,* originates from *S. lycopersicum* var. *cerasiforme* (Ciccarese et al. 1998), while one resistance QTL to bacterial canker has been mapped to chromosomes 6 in *S. lycopersicum* accession Hawaii 7998 (Abebe et al. 2022). In addition, more than 30 QTLs associated with resistance to *Ralstonia solanacearum* have been identified across multiple chromosomes (Shin et

al. 2020; Méline et al. 2023). However, it is important to note that the resistance source may also originate from wild species.

Table 3: List of disease resistance genes and QTL mapped in tomato. (Q): indicates that the locus is a QTL when is not specified in the name.

Disease	Gene/QTL	Pathogen	Resistance source	Chromosome	Reference
Powdery mildew	*Lv*	*Leveillula taurica*	*S. chilense*	12	(Chunwongse et al. 1994)
Powdery mildew	*Ol-1*, o*l-2*, Ol-3, Ol-4, Ol-5, Ol-6	*Oidium lycopersicum*	*S. esculentum,* *S. habrochaites*	4,6	(Van Der Beek et al. 1994; Ciccarese et al. 1998; Huang et al. 2000a, b; De Giovanni et al. 2004)
	Ol-qtl1 (Q), Ol-qtl2 (Q), Ol-qtl3 (Q)		*S. neorickii*	6,12	(Bai et al. 2003)
	9 (Q)		*Various*	1, 4, 6, 8, 12	(Park et al. 2022)
Gray mold	Fbc1-4 (Q), Fbc9 (Q)	*Botrytis cinerea*	*S. lycopersicoides*	1,2,3,4, 9	(Davis et al. 2009)
	Fbc5 (Q), Fbc-11 (Q)	*Botrytis cinerea susceptibility*	*S. lycopersicoides*	5, 11	(Davis et al. 2009)
	Snbc3, Snbc*4*	*Botrytis cinerea*	*S. neorickii*	3,4	(Finkers et al. 2008)
	Rbcq1, Rbcq2, Rbcq3, Rbcq4a, Rbcq4b, Rbcq6, Rbcq9a, Rbcq9b, Rbcq11, and Rbcq12		*S. habrochaites*	1,2,3,4,6,9,11,12	(Finkers et al. 2007a, b)
Late blight	*Ph-1*, *Ph-2*, *Ph-3*	*Phytophthora infestans*	*S. pimpinellifolium,* *S. habrochaites,* *S. pennellii*	7,9,10	(Gallegly and Marvel 1955; Moreau et al. 1998; Chunwongse et al. 2002; Zhang et al. 2014a)
	1 (Q)		*S. pennellii*	6	(Smart et al. 2007)
	3 (Q)		*Breeding line*	9,10,12	(Panthee et al. 2017)
	Ph-4, *Ph-5-1*, *PH-5-2,* + 4 (Q)		*S. pimpinellifolium*	1,2,10,11	(Merk et al. 2012; Chen et al. 2014a; Sullenberger et al. 2022)
	10 (Q)		*S. pimpinellifolium*	1,2,5,6,10,11,12	(Sullenberger and Foolad 2024)
	LBRQTL- 1.1, LBRQTL- 1.2,		*S. pimpinellifolium*	1,10,12	(Jia et al. 2025)

Table 3: List of disease resistance genes and QTL mapped in tomato. (Q): indicates that the locus is a QTL when is not specified in the name.

Disease	Gene/QTL	Pathogen	Resistance source	Chromosome	Reference
	LBRQTL- 1.3, LBRQTL- 10.1, LBRQTL- 10.2, LBRQTL- 12 (Q)				
	LBRQTL-6, LBRQTL-9, LBRQTL-10 (Q)		*S. pimpinellifolium*	6,9,10	(Gao and Foolad 2024)
	2 (Q), lb1–lb12 (Q)		*S. pimpinellifolium, S. habrochaites,*	1,2,3,4,5,6,7,8, 9,10,11,12	(Frary et al. 1998; Brouwer and St. Clair 2004; Brouwer et al. 2004)
Early blight	24 (Q)	*Alternaria solani*	*S. habrochaites*	1,2,3,5,8,9,10,11,12	(Foolad et al. 2002)
	7 (Q)		*S. habrochaites*	1,2,3,5,8,9,10,11,12	(Zhang et al. 2003)
	10 (Q)		*S. pimpinellifolium*	1,3,4,5,6,9,11	(Foolad et al. 2008)
	6 (Q)		*S. arcanum*	1,2,5,7,9	(Chaerani et al. 2007)
Alternaria stem canker	Asc	*Alternaria alternata f. sp. lycopersici*	*S. pennellii*	3	(Van Der Biezen et al. 1995; Mesbah et al. 1999)
Bacterial canker	Rcm1.0–10.0 (Q), Rcm5.1, Rcm7.1	*Clavibacter michiganensis*	*S. peruvianum*	1,2,5,6,7,8,9,10	(Sandbrink et al. 1995; Van Heusden et al. 1999)
	Rcm5.0, Rcm7.1, Rcm9.0 (Q)		*S. peruvianum*	5,7,9	(Kabelka et al. 2002)
	Rcm2.0, Rcm5.1 (Q)		*S. habrochaites*	2,5	(Coaker and Francis 2004)
	Rcm6 (Q)		*Solanum lycopersicum*	6	(Abebe et al. 2022)
	5 (Q)		*S. pimpinellifolium*	1,2,7,8,12	(Sen et al. 2021)
Bacterial speck	Pto , 6 (Q) (bsRr1-1, bsRr1-2, bsRr1-12a and bsRr1-12b, qRph1; qRph2)	*Pseudomonas syringae pv. Tomato*	*S. pimpinellifolium; Solanum habrochaites*	1,2,5,8,12	(Martin et al. 1991; Bao et al. 2015)
	Prf	Required for resistance to Pst	*S. pimpinellifolium*	5	(Salmeron et al. 1996)
Bacterial spot	Rx-1, Rx-2, Rx-3, Rx-4 (Q), Bs4, Xv4	*Xanthomonas spp.*	*S. esculentum, S. pennellii*	1,3,4,5	(Yu et al. 1995; Astua-Monge et al. 2000; Ballvora et

Table 3: List of disease resistance genes and QTL mapped in tomato. (Q): indicates that the locus is a QTL when is not specified in the name.

Disease	Gene/QTL	Pathogen	Resistance source	Chromosome	Reference
					al. 2001; Yang et al. 2005)
	15 (Q)		*S. lycopersicum,* *S. pimpinellifolium*	1,2,3,4,6,7,8,9, 11,12	(Adhikari et al. 2023)
Bacterial wilt	Rrs3.0–12.0 (Q)	*Ralstonia solanacearum*	*S. pimpinellifolium,* *S. peruvianum*	3,4,6,7,10	(Danesh 1994; Thoquet 1996a, b; Mangin et al. 1999; Deberdt et al. 1999)
	Bwr2.1-12.3 (Q)		*Solanum lycopersicum*	2,3,4,6,8,10,12	(Shin et al. 2020; Méline et al. 2023)
	18 (Q)		*Various*	1,2,3,4,6,7,8,9, 10,12	(Belny et al. 2025)
Corky root rot	Py-1	*Pyrenochaeta lycopersici*	*S. peruvianum*	3	(Doganlar et al. 1998)
Blackmold	Blackmold (Q)	*Alternaria alternata*	*S. cheesmaniae*	2,3,9,12	(Robert et al. 2001)
Cucumber mosaic virus	Cmr	CMV	*S. chilense*	12	(Stamova and Chetelat 2000)
Fusarium crown/root rot	Frl	*F. oxysporum f.sp. radicis-lycopersici*	*S. peruvianum*	9	(Vakalounakis et al. 1997)
Fusarium wilt	I, I1, I2, I2C, I3 I4, I5, I6, I7	*F. oxysporum*	*S. pimpinellifolium,* *S. pennellii*	2,7,8,10,11	(Bournival et al. 1989, 1990; Sarfatti et al. 1989, 1991; Segal et al. 1992; Ori et al. 1997; Simons et al. 1998)
Leaf mould	Cf-1, Cf-2, Cf-4, Cf-5, Cf-9, Cf-ECP2 (Cf-19), Cf-10, Cf-12, Cf-13, Cf-16	*Cladosporium fulvum*	*S. habrochaites,* *S. pimpinellifolium*	1,2,3,4,5,6,7,8, 11	(Van Der Beek et al. 1992; Balint-Kurti et al. 1994; Thomas et al. 1998; Haanstra et al. 1999; Zhao et al. 2016; Xue et al. 2017; Liu et al. 2018a, 2019a; Chai et al. 2020)
Nematode (cyst)	Hero	*Globodera rostochiensis*	*S. pimpinellifolium*	4	(Ganal 1995)

Table 3: List of disease resistance genes and QTL mapped in tomato. (Q): indicates that the locus is a QTL when is not specified in the name.

Disease	Gene/QTL	Pathogen	Resistance source	Chromosome	Reference
Nematode (root-knot)	Mi (Mi-1), Mi-2, Mi-3, Mi-5, Mi-9, Mi-HT, Mi-4, Mi-6, Mi-7, Mi-8	*Meloidogyne spp.*	*S. peruvianum*	6,12	(Aarts et al. 1991; Klein-Lankhorst et al. 1991; Williamson et al. 1994; Yaghoobi et al. 1995; Doganlar et al. 1997; Kaloshian et al. 1998; Veremis et al. 1999; El-Sappah et al. 2019)
Potyvirus	pot-1	Potyvirus	*S. habrochaites*	3	(Parrella et al. 2002)
Tobacco mosaic virus	Tm-1, Tm-2, Tm-2^2	TMV/ToMV	*S. habrochaites,* *S. peruvianum*	2,9	(Young et al. 1988; Levesque et al. 1990; Ohmori et al. 1996; Vakalounakis et al. 1997; Tanksley et al. 1998; Luria et al. 2017; Hak et al. 2022)
Tomato spotted wilt virus	Sw-5	TSWV	*S. peruvianum*	9	(Stevens et al. 1995)
Tomato yellow leaf curf virus	Ty-1 (Q), others	TYLCV	Multiple spp.	6,11	(Zamir et al. 1994; Chagué et al. 1997)
	Ty-2		*S. habrochaites*	11	(Dhaliwal et al. 2020)
	Ty-1, Ty-3, Ty-4 and Ty-6		*S. chilense*	3,6,10	(Dhaliwal et al. 2020)
	Ty-5		*S. peruvianum*	4	(Dhaliwal et al. 2020)
Verticillium wilt	Ve (Ve1/Ve2), V2 (chromosome undetermined)	*Verticillium dahliae*	*S. esculentum,* *S. pennellii* *S. neorickii*	9	(Diwan et al. 1999; Fradin et al. 2009; Usami et al. 2017; Chavarro-Carrero et al. 2021)
Tomato brown rugose fruit virus	Tob11	*ToBRFV*	*S. pimpinellifolium*	11	(Jaiswal et al. 2025)

Beyond the Lycopersicon clade, related species such as *S. lycopersicoides* provide complementary resistance sources. In this species, five QTLs (Fbc1, Fbc2, Fbc3, Fbc4, and Fbc9) associated with resistance to *B. cinerea* have been mapped on chromosomes 1, 2, 3, 4, and 9 (Davis et al. 2009), affecting both infection frequency and lesion development.

Even if the 12 chromosomes harbor resistance loci, several hotspots of resistance of genes can be observed in the tomato genome (Foolad 2007; Ashrafi et al. 2009; Causse et al. 2020). Chromosomes 6 and 9 are particularly enriched. Chromosome 6 harbors major resistance genes to root-knot nematodes (*Mi-1.2*, *Mi-9*), *O. neolycopersici* (*Ol-1*, *Ol-3*, *Ol-4*, *Ol-5*, *Ol-6*), *Passalora fulva* (syn. *Cladosporium fulvum*) (*Cf-2*, *Cf-5*), Tomato yellow leaf curl virus (*Ty-1*, *Ty-3*), and Alfalfa mosaic virus (*Am*), along with QTLs for resistance to *R. solanacearum* and Tomato mottle virus (ToMoV) (Agrama and Scott 2006; Ashrafi et al. 2009). Chromosome 9 similarly contains dense resistance clusters, including *Tm-2* and *Tm-2²* (ToMV), *Frl* (Fusarium crown and root rot), *Sw-5* (TSWV), *Ph-3* (*P. infestans*), and *Ve* (*Verticillium dahliae*), as well as QTLs for resistance to *B. cinerea*, *Alternaria solani*, and *C. michiganensis* (Chaerani et al., 2007; Chunwongse et al., 2002; Davis et al., 2009; Foolad et al., 2008; Pillen et al., 1996; Sandbrink et al., 1995; Stevens et al., 1995; Vakalounakis et al., 1997; Van Heusden et al., 1999).

2.2.3. Introgression for resistance genes and QTLs

Introgression refers to the stable incorporation of genetic material (genes, QTLs, or chromosomal segments) from one species into another through hybridization followed by repeated backcrossing with the recurrent parent. Gene introgression from wild tomato relatives into cultivated *Solanum lycopersicum* has become a key strategy for crop improvement. Knowledge of the linkage between molecular markers and resistance loci facilitates efficient, and sometimes rapid, transfer of resistance into diverse genetic backgrounds through marker-assisted selection (MAS) (Foolad et al. 2014). These efforts have led to the development of commercial varieties carrying resistance to major diseases such as late blight, root-knot nematodes, and viruses. For example, breeding lines NC1 CELBR and NC2 CELBR, carry resistance genes to late blight (*Ph-2* and *Ph-3*), to early blight, to verticillium wilt (*Ve* gene) and races 1 and 2 of Fusarium wilt (*I* and *I-2* gene) (Gardner and Panthee 2010; Elsayed et al. 2011). The *Mi-1* gene for nematode resistance is also widely deployed, and varieties such as “Clodano” combine resistance to multiple pathogens (ToMV: 0-2, TMV: 0, Fol: 0-1 (US1-2), Ff: A-E).

However, many identified resistance genes like *Mi-2* to *Mi-9* and *Mi-HT,* have not yet been introgressed into commercial cultivars (El-Sappah et al. 2019). This is largely due to genetic barriers between cultivated tomato and some wild relatives, as well as linkage drag and reduced fertility associated with interspecific crosses (El-Sappah et al. 2019; Foolad et al. 2008; Lin et al. 2014). Consequently, *S. pimpinellifolium* is the most commonly used donor species, owing to its close genetic relationship with cultivated tomato and fewer undesirable agronomic traits compared with more distant species such as *S. habrochaites* or *S. peruvianum* (Foolad et al. 2008).

Relying on a single resistance source can pose a risk to durability, as pathogens can rapidly overcome resistance due to their high evolutionary potential (Lechevalier et al. 2025). Therefore, diversifying resistance sources and pyramiding resistance genes are essential strategies for sustainable disease management. In line with this need to broaden the genetic basis of resistance, closely related species such as *S. cheesmaniae* and *S. galapagense* represent valuable reservoirs of resistance genes and QTLs that can be exploited in future breeding efforts.

Key point on in plant innate immune / resistance gene limits

Resistance breakdown: R-gene-based qualitative resistance often shows limited durability due to gene-for-gene interactions with fungal avirulence (Avr) genes. Pathogens can rapidly evolve through mutations that enable them to evade host recognition mechanisms. For example, the resistance genes *Ph-1*, *Ph-2*, and to a lesser extent *Ph-3,* have been overcome by certain strains of *Phytophthora infestans*.

Introgression constraints: Introgression and pyramiding of resistance genes from wild relatives are often hindered by genetic barriers, linkage drag (i.e., the co-introduction of undesirable agronomic traits), and reduced fertility in progeny.

Defense limitations: In some cases, plant defense is constrained by either the efficiency or the timing of defense responses, which may not be rapid or strong enough to effectively limit pathogen development.

Given the limited durability of R-gene-based resistance, constraints associated with introgression strategies, and the sometimes-insufficient effectiveness of plant defense responses, alternative approaches are needed. In this context, plant defense can be enhanced using plant resistance inducers, offering a complementary strategy to improve both the efficiency and durability of disease control.

3. Induced resistance

Plant resistance inducers (PRI), also called plant defense elicitors, are substances that activate a plant's natural immune system without directly killing pathogens. Their mechanism of action is based on stimulating the plant rather than directly targeting the pathogen, similar to a vaccine that primes the immune system in animals (Marolleau et al. 2013).

Resistance can be induced by a wide range of biotic and abiotic factors, including cell wall fragments, plant extracts, essential oils, alive or dead microorganisms (viruses, bacteria, oomycetes, fungi), algal extracts, polysaccharides, and synthetic compounds. These agents, known as plant resistance inducers (PRIs) or elicitors, enhance plant defense responses and improve resistance to subsequent stresses (Marolleau et al. 2013; Jamiołkowska 2020).

PRIs are broadly classified into chemical and biological elicitors. Chemical elicitors include compounds such as acibenzolar-S-methyl (ASM), probenazole, β-aminobutyric acid (BABA), potassium phosphite, thiamine, and signaling-related molecules like salicylic acid (SA), 2,6-dichloroisonicotinic acid (INA), benzo(1,2,3)thiadiazole-7-carbothioic acid S-methyl ester (BTH), γ-aminobutyric acid (GABA), cytokinins, and hexanoic acid (Walters et al. 2013; Hönig et al. 2023). Biological elicitors include plant growth-promoting rhizobacteria and fungi, arbuscular mycorrhizal fungi, other micro-organisms or micro-organisms extracts, and plant-derived extracts (Walters et al. 2013), which may be in the form of crude or purified extracts.

Depending on their intrinsic nature, these compounds act in different ways. Some function as non-self-determinants by mimicking microbe-associated molecular patterns (MAMPs) or damage-associated molecular patterns (DAMPs) perceived by pattern recognition receptors (PRR) present at the cell surface, while others mimic endogenous plant signaling molecules, such as phytohormone analogs or derivatives (Marolleau et al. 2017). In both cases, they trigger a cascade of cellular reactions either in response to abiotic and biotic stresses or without additional stress.

Induced resistance involves either the immediate activation of defense mechanisms or the establishment of a primed state, in which plants respond more rapidly and/or more strongly upon pathogen attack (Goellner and Conrath 2008; Hilker and Schmülling 2019; Tanaka et al. 2024). In most cases, these two processes act in combination (Ahmad et al. 2010). The early induction of

plant defense responses confers to PRIs the ability to protect plants against a broad spectrum of virulent pathogens.

3.1. PRI as promised alternative to pesticides

The use of plant resistance inducers (PRIs) represents a promising and sustainable strategy for pest and disease management, contributing to environmental protection (Jamiołkowska 2020; Pagliarani et al. 2020). Over the past three decades, PRIs have been extensively studied in phytopathology, with research primarily focusing on their modes of action and efficacy. These studies indicate that PRI-induced resistance is generally more durable, relatively stable over time, and effective against a broad spectrum of pathogens.

Among chemical elicitors, β-aminobutyric acid (BABA) is one of the most studied and effective priming agents, conferring protection in various plant species against a wide range of stresses (Cohen et al. 2016). It has been shown to enhance resistance against *P. infestans* in potato and tomato (Sharma et al. 2010, 2012; Bengtsson et al. 2014; Floryszak-Wieczorek et al. 2015), *B. cinerea* (Luna et al. 2016), *O. neolycopersici*, *Phytophthora parasitica* (Zapletalová et al. 2023), and *Plasmopara viticola* (Tamm et al. 2011).

Among biological elicitors, *Trichoderma* spp. is widely reported to reduce disease severity across multiple plant-pathogen systems. In tomato and cucumber, inoculation of *T. harzianum*, *T. atroviride*, and *T. longibrachiatum* significantly reduced grey mold caused by *Botrytis cinerea* (Tucci et al. 2011; Yuan et al. 2019). Similarly, *T. atroviride* has been shown to be effective against late blight caused by *P. infestans* (Põldmets et al. 2025). Other strains, such as *T. harzianum* T39, induce resistance to downy mildew in grapevine (Perazzolli et al. 2011), while *T. asperellum* SKT-1 enhances resistance in *Arabidopsis* against *P. syringae* pv. *tomato* DC3000 (Yoshioka et al. 2012).

PRI-induced resistance typically corresponds to a form of quantitative resistance, with efficacy ranging from approximately 20% to 100% depending on the inducer and experimental conditions. For example, silicon-based products such as HortiStar® and Actisil® reduced *Alternaria solani* infection in potato by 60-80% under greenhouse conditions (Stridh et al. 2022), showing efficacy comparable to that of beneficial bacteria such as *Bacillus subtilis* 26D and *B. velezensis* M66 (Sorokan et al. 2024). More recently, esculetin has been identified as a novel PRI, providing 45-

69% control of tobacco bacterial wilt caused by *R. solanacearum* (Zhang et al. 2026). Tanaka et al., (2024) showed that the application of L-methionine in tomato reduced disease severity by 30% for *P. syringae* pv. *tomato* and by 50% for *B. cinerea*. Sharma et al., (2010) showed that the application of BABA on young tomato leaves reduced disease severity to *P. infestans* by 43-100%.

Overall, these studies highlight the substantial variability in PRI efficacy, likely reflecting the complexity of the underlying resistance mechanisms and their interaction with plant genotype, pathogen pressure, and environmental conditions.

3.2. **Mechanisms of resistance induced by the PRI**

The interaction between a plant resistance inducer (PRI) and the plant triggers a cascade of reactions that can occur both locally and systemically (Walters and Fountaine 2009). These responses include the accumulation of reactive oxygen species (ROS), such as superoxide and hydrogen peroxide (H_2O_2), the transcriptional activation of genes encoding pathogenesis-related (PR) proteins, and the biosynthesis of defense-related metabolites, including phytohormones, amino acids, phenolics, phospholipids and phytoalexins (Yarullina et al. 2021, 2022; Margaritopoulou et al. 2024).

Systemic resistance is generally classified into two main types: systemic acquired resistance (SAR) and induced systemic resistance (ISR) (Walters et al. 2013). SAR is primarily regulated by salicylic acid (SA) signaling and is typically effective against biotrophic pathogens (Fu and Dong 2013). In contrast, ISR, often triggered by plant growth-promoting rhizobacteria (PGPR), is mainly associated with jasmonic acid (JA) and ethylene (ET) signaling pathways and is particularly effective against necrotrophic pathogens (Pieterse et al. 2012). Activation of systemic resistance involves several defense responses, including hypersensitive response (HR), ROS accumulation, increased activity of defense-related enzymes, enhanced antioxidant capacity, and upregulation of defense-related genes. However, the specific mechanisms underlying induced resistance vary depending on the PRI, the plant species, and the pathogen. To investigate the mode of action of plant resistance inducers (PRIs), researchers typically analyze either the expression of genes associated with hormonal signaling pathways or encoding pathogenesis-related (PR) proteins, as well as the production of reactive oxygen species (ROS), particularly hydrogen peroxide (H_2O_2). A qPCR-based tool was developed (Brisset and Dugé de Bernonville, 2011) to characterize the mode of action of PRIss using a broad panel of defense-related genes. This tool comprises 28

Table 4 List of the defense genes in the tool qPFD (Brisset and Dugé de Bernonville, 2011) adapted to tomato

Gene	**Name**	**Pathway or function**
PR-1	Pathogenesis-related protein 1	PR-protein
PR-2	Pathogenesis-related protein 2 (glucanases)	PR-protein
PR-4	Pathogenesis-related protein 4 (hevein-like)	PR-protein
PR-5	Pathogenesis-related protein 5 (thaumatin-like, osmotin)	PR-protein
PR-8	Pathogenesis-related protein 8 (class III chitinase)	PR-protein
PR-14	Pathogenesis-related protein 14 (lipid transfer protein)	PR-protein
PR-15	Pathogenesis-related protein 15 (oxalate oxidase)	PR-protein
PAL	Phenylalanine ammonia-lyase	Phenylpropanoid pathway
CHS	Chalcone synthase	Phenylpropanoid pathway
DFR	Dihydroflavonol reductase	Phenylpropanoid pathway
ANS	Anthocyanidin synthase	Phenylpropanoid pathway
PPO	Polyphenol oxidase	Phenylpropanoid pathway
HMGR	Hydroxymethyl glutarate-CoA reductase	Isoprenoid pathway
FPPS	Farnesyl pyrophosphate synthase	Isoprenoid pathway
Far	(E,E)-alpha-farnesene synthase	Isoprenoid pathway
CSL	Cysteine sulfoxide lyase	Cysteine sulfoxid pathway
APOX	Ascorbate peroxidase	Oxidative stress
GST	Glutathion S-transférase	Oxidative stress
POX	Peroxidase	Oxidative stress
CalS	Callose synthase	Cell wall modification
Pect	Pectin methyl esterase	Cell wall modification
CAD	Cinnamyl alcool dehydrogenase	Cell wall modification
EDS1	Disease resistance protein EDS1	Salicylic acid pathway
WRKY	WRKY transcription factor 30	Salicylic acid pathway
LOX2	Lipoxygenase AtLOX2	Jasmonic acid pathway
JAR	Jasmonate resistant 1	Jasmonic acid pathway
ACCO	1-aminocyclopropene-1-carboxylate oxidase	Ethylene pathway
EIN3	EIN3-BINDING F-BOX PROTEIN 1	Ethylene pathway
TuA	Tubulin alpha-1 chain	Reference gene
Actin	Actin 7	Reference gene
GAPDH	Glyceraldehyde-3-phosphate dehydrogenase	Reference gene

marker genes, including genes encoding PR proteins, genes involved in oxidative stress responses, related to secondary metabolism, and components of the salicylic acid (SA), jasmonic acid (JA),

and ethylene (ET) signaling pathways (**Table 4**). It has been successfully validated in apple and carrot (Gaucher et al. 2022; Le Clerc et al. 2025). In the interaction between potato and *P. infestans*, priming potato with *Bacillus subtilis* and chito-oligosaccharides upon infection with *P. infestans*, induced systemic resistance with the activation of catalase, peroxidase, the accumulation of hydrogen peroxide, and transcripts of genes encoding amylase inhibitor, PR proteins: PR-1, PR-3, PR-6, and PR-9 (Yarullina et al. 2022). Studies on *Trichoderma* spp. indicate that both SA- and JA-dependent signaling pathways can be activated (Tucci et al. 2011; Yuan et al. 2019). Treatment with β-aminobutyric acid (BABA) induces callose deposition, ROS accumulation, and increased activity of enzymes such as glycolate oxidase, while enhancing the biosynthesis of metabolites from pathways including phenylpropanoids, octadecanoids, flavonoids, polyphenols, and tocopherols (Pastor et al. 2014; Cohen et al. 2016; Alamri et al. 2026). BABA-induced resistance involves multiple hormonal pathways, including SA, JA, ABA and ET (Ton et al. 2005; Hamiduzzaman et al. 2005; Slaughter et al. 2012; Catoni et al. 2022; Zapletalová et al. 2023). The relative contribution of these pathways depends strongly on the plant-pathogen system (Baccelli and Mauch-Mani 2016; Zapletalová et al. 2023). For example, in *Arabidopsis thaliana*, BABA-induced resistance against the hemibiotrophic bacterium *Pseudomonas syringae* pv. *tomato* DC3000 and the necrotrophic fungus *B. cinerea* is associated with faster and stronger induction of SA-dependent defense genes upon pathogen challenge (Slaughter et al. 2012). In contrast, in tomato and potato, BABA-induced resistance appears to rely more on JA- and ET-dependent pathways (Satková et al. 2016; Zapletalová et al. 2023). In grapevine, resistance to downy mildew has been linked to JA signaling following BABA treatment (Hamiduzzaman et al. 2005). Finally, BABA primarily acts through defense priming, enhancing callose deposition and potentiating SA-, JA-, ABA-, and phosphatidylinositol-dependent responses upon pathogen attack (Ton et al. 2005; Hamiduzzaman et al. 2005).

Increasing evidence also indicates that PRI-induced priming can involve stress memory mediated by epigenetic modifications (Catoni et al. 2022), which may be long-lasting and transmitted to subsequent generations (Ramírez-Carrasco et al. 2017; Wilkinson et al. 2018; Martínez-Aguilar et al. 2021). For instance, vegetative progeny of potato plants primed with BABA exhibit enhanced expression of the defense gene *PR1* and increased resistance to *P. infestans* (Floryszak-Wieczorek et al. 2015). Similarly, in *Arabidopsis*, progeny of plants primed either with BABA or with an avirulent strain of *P. syringae* pv. *tomato* show enhanced expression

of defense-related genes and increased resistance to *P. syringae* pv. *tomato* DC3000 and *Hyaloperonospora arabidopsidis* (Slaughter et al. 2012). The same study also showed that additional priming of the progeny leads to an even stronger response, suggesting that plants can inherit a sensitized priming state.

3.3. **Mechanisms of resistance induced in tomatoes**

Tomatoes have served as a successful model system for induction of resistance to many pathogens by various PRI (**Table 5**). The most common pathogens used are *B. cinerea*; *P. infestans*, *O. neolycopersici* and *P. syringae* pv. *tomato*.

Although the general mechanisms underlying plant resistance inducers (PRIs) are well described, their expression in tomato depends strongly on the specific PRI-pathogen interaction. In this species, PRI-induced resistance involves the activation of key hormonal signaling pathways, primarily salicylic acid (SA), jasmonic acid (JA), and ethylene (ET), whose relative contribution varies according to the PRI-pathogen interaction. For example, BABA displays direct activation of the ET-dependent signaling pathway in resistance to *O. neolycopersici* (Satková et al. 2016), while SA- and JA-dependent signaling pathway are primed during *B. cinerea* resistance (Catoni et al. 2022). A comparison of the metabolic profiles of tomato plants treated with hexanoic acid and subsequently infected by *B. cinerea* or *P. syringae* pv. *tomato* (Pst) revealed clear differences (Camañes et al. 2015), reflecting the fact that the plant responses to PRI are highly adapted to specific pathogens.

At the molecular and cellular levels, PRI-induced resistance in tomato is characterized by cell wall reinforcement with callose deposits, the upregulation of pathogenesis-related (PR) genes, the accumulation of reactive oxygen species (ROS), the activation of defense-related enzymes, and the production of secondary metabolites (Koley et al., 2022; Lewis et al., 2023; Mhlongo et al., 2021). It has been demonstrated that treating tomato leaves with polysaccharides extracted from microalgae enhances the activity of phenylalanine ammonia-lyase (PAL), PR proteins (chitinase, 1,3-β-glucanase), and peroxidase (POX) in tomato leaves (Rachidi et al. 2021). In the same study, these polysaccharides also induced modifications in the metabolite profile involved in the construction of the leaf cuticular wax, including fatty acids, alkenes, and phytosterols. Interestingly, the metabolite 1-methyltryptophan was shown to be associated with hexanoic acid-induced resistance (Camañes et al. 2015). Metabolomic analysis of tomato treated by salicylate,

Table 5 non-exhaustive list of plant resistance inducers (PRI) studied on tomato			
Pathogens	**PRI**	**Triggered Response**	**References**
Botrytis cinerea	*T. harzianum* / *T. atroviride*	Activate Jasmonate-responsive genes; upregulation of salicylic acid (SA) (PR1b1 and PR-P2) and TomLoxA	(Tucci et al. 2011)
	β-Aminobutyric acid (BABA)	Priming of both SA- and JA-dependent resistance. BABA treatment results in genome-wide DNA methylation. Earlier and higher expression of PR1 and PR5 genes when it is combined with salt stress	(Luna et al. 2016; Rejeb et al. 2018; Wilkinson et al. 2018; Catoni et al. 2022)
	Hexanoic acid	Antifungal; Induction of resistance to different pathogens. Induction of ABA-dependent callose deposition. Activation of JA-dependent genes; Decreased ROS accumulation in tomato upon pathogen attack. Enhanced expression of similar tomato genes as following Botrytis infection.	(Vicedo et al. 2009; Finiti et al. 2014)
	Cytokinin	modulating cellular trafficking of the pattern recognition receptor (PRR) LeEIX2, which mediates immune responses to Xyn11 family xylanases, and promoting resistance in an SA- and ET-dependent mechanism.	(Gupta et al. 2020)
	L-methionine	Priming effect. Up-regulation of JA-related gene PI2	(Tanaka et al. 2024)
	Levan (β-2,6-linked fructan)	Induce H2O2 accumulation. Enhance POX and phenylalanine ammonia-lyase activities, increased lignin deposition and the upregulation of defense-related genes associated with salicylic acid, jasmonic acid, and ethylene pathways.	(Yang et al. 2026)
	PGPR *Peribacillus frigoritolerans*	Increase the expression of genes related to the SA pathway,	(Mazuecos-Aguilera et al. 2025)

Table 5 non-exhaustive list of plant resistance inducers (PRI) studied on tomato

Pathogens	PRI	Triggered Response	References
	PGPR *Pseudomonas canadensis*	Increase the expression of genes related to the JA/ET hormonal pathway	(Mazuecos-Aguilera et al. 2025)
Oidium neolycopersici	Cytokinin	Modulating cellular trafficking of the pattern recognition receptor (PRR) LeEIX2, which mediates responses to Xyn11 family xylanases. Promoting resistance in an SA- and ET-dependent mechanism.	(Gupta et al. 2020)
	L-methionine	Up-regulation of PR-genes	(Saito et al. 2017)
	BABA	Displaying direct activation of the ET-dependent signaling pathway	(Satková et al. 2016)
	Oligandrin	Displaying direct activation of the ET-dependent signaling pathway	(Satková et al. 2016)
	Planticine® (α(1→4)-linked D-galacturonic acids)	upregulate gene-encoded elicitor and effector recognition receptors (ELRR and ERR), MAPK, WRKY, ERF, MYB, NAC, bZIP, PR proteins, and resistance-related metabolite genes.	(Rakoczy-Lelek et al. 2023)
Leveillula taurica	COS + OGA	Upregulation of PR proteins and SA-related genes	(Van Aubel et al. 2016)
Fusarium oxysporum f.sp. *lycopersici*	*Ulva lactuca*		(El Modafar et al. 2012)
Pseudomonas syringae pv. tomato (Pst)	Hexanoic acid	Induction of ABA-dependent callose deposition, activation of JA-dependent defense responses involving priming of 12-oxo-phytodienoic acid (OPDA) and JA isoleucine accumulation.	(Vicedo et al. 2009)
		Increase in the expression of jasmonic acid carboxyl methyltransferase (JMT) and the SA marker genes PR1 and PR5	(Scalschi et al. 2013)
	L-methionine	Priming effect. Upregulation of SA-related gene PR1b	(Tanaka et al. 2024 ; Sakamoto et al. 2025)
Phytophthora infestans	β-Aminobutyric acid (BABA)		(Sharma et al. 2012)
Ralstonia solanacearum	Acibenzolar-*S*-methyl (ASM)		(Hong et al. 2011)
	Salicylic acid (SA)	Increased the activities of peroxidase and polyphenol oxidase enzymes	(Narasimhamurthy et al. 2019)

Table 5 non-exhaustive list of plant resistance inducers (PRI) studied on tomato

Pathogens	PRI	Triggered Response	References
Rhizoctonia solani	Methyl jasmonate (MeJA)	Induction of biosynthesis of SA, JA and polyphenols. Upregulation of PR1a and PAD4 expression and reduction ROS production.	(Koley et al. 2022)
	Microalgae polysaccharides	Improve the activity of PAL, chitinase, 1,3-betaglucanase, peroxidase. Induce proteins, polyphenols and H2O2 production in leaves treated	(Rachidi et al. 2021)
	Trichoderma pubescens	Upregulated the expression of defense-related genes PAL, CHS, and HQT	(Behiry et al. 2023)
Alternaria solani	Silicon	increased the expression of jasmonic acid (JA) marker genes (PR3, LOXD and JERF3) than salicylic acid (SA) marker genes (PR1, PR2 and WRKYII)	(Gulzar et al. 2021)
	Hexanoic acid	Increase expression of SlNAC1, SlMAPK3 and SlPR1. Induce salicylic acid-dependent signaling pathway	(Rabiei et al. 2022)
	FytoSol (chitosan oligomers + pectin-derived oligogalacturonides)	Upregulate gene expressions of PR3, downregulated the SA-related defense pathway	(Bektas 2022)
Tuta absoluta	Phenylalanine	Increase accumulation of benzenoid/phenylpropanoid volatiles	(Kumar et al. 2024)
Bemisia tabaci	*Beauveria bassiana*	Upregulate defense genes (PIN2, PR2, PAL, and MPK3). Mainly activate tomato phenylpropane metabolic modulated by jasmonate	(Liu et al. 2025)

polyamines, 2,1,3-benzothiadiazole and chitosan, revealed a broad reprogramming of secondary metabolism, including membrane lipid remodeling, phenylpropanoid antioxidants, and phytohormone crosstalk (Iula et al. 2022). characteristics, and the intrinsic properties of the PRI itself (Walters and Fountaine 2009; Sharma et al. 2010; Bruce 2014; López-Vásquez et al. 2024; Pressecq et al. 2025; Põldmets et al. 2025). Generally, induced defense in tomato involves the accumulation of ROS. However, this strategy is not effective against necrotrophic pathogens such as *B. cinerea*, which normally induced ROS production to accelerate cell death. Effective priming against this pathogen generally resulted in decreasing accumulation of ROS and increasing antioxidant activity in tomato upon pathogen attack (Finiti et al. 2014).

3.4. Factors affecting the efficacy of plant resistance inducers in the field

The efficacy of PRIs is considered as highly variable and depends on multiple interacting factors, including host plant traits, biotic and abiotic environmental conditions, and pathogen characteristics (Lee Díaz et al. 2021; Jung et al. 2024; Charpe et al. 2025).

Environmental conditions strongly influence both the expression and durability of induced resistance, and PRI performance under field conditions often differs substantially from that observed in controlled environments (Walters et al. 2005, 2013; Walters and Fountaine 2009; Lee Díaz et al. 2021; Stridh et al. 2022). For instance, Dietrich et al. (2004) showed that nitrogen limitation markedly reduces the induction of defense enzymes following treatment with the PRI BION®, and also delays the induction process, with defense activation occurring later under low-nitrogen conditions. Similarly**,** Stridh et al. (2022) reported that, in potato, HortiSar significantly reduced early blight infection in greenhouse trials, whereas no significant effect was observed under field conditions, highlighting the strong context-dependence of PRI efficacy.

The developmental stage of the plant at the time of application (seedling, flowering, fruiting) is another critical determinant of PRI performance. For instance, Rossi et al., (2025) demonstrated that, in grapevine, the efficacy of four of the most effective PRIs against *Plasmopara viticola* varied markedly across berry developmental stages, with protection increasing as cluster development progressed. This illustrates how physiological changes in the host can affect responsiveness to PRI.

In the present work, we specifically focused on the genotypic variability underlying PRI efficacy. For a more comprehensive overview of the other factors influencing induced resistance, see (Walters et al. 2005, 2013; Walters and Fountaine 2009; Lee Díaz et al. 2021; Prusky and Romanazzi 2023; and Charpe et al. 2025).

3.5. Genotypic variability of PRI efficacy

Plant traits strongly determine whether and how well PRIs activate defenses. Differences among species, cultivars and even wild versus domesticated genotypes cause large variation in basal defense levels, inducibility and downstream signaling. These variations occur at the phenotypic level, the transcriptomic level and the level of metabolic biosynthesis.

The link between the protective efficacy of PRIs and the genotype of plants has been investigated in several plant species, using panels of plants of varying sizes. In tomato, Sharma et al., (2010) demonstrated significant intraspecific variability in BABA-induced resistance against *P. infestans*. Similarly, Tucci et al., (2011) reported that resistance induced by *Trichoderma spp.* against *B. cinerea* varied depending on the tomato genotype. Satková et al., (2016) further showed that both oligandrin and BABA induced significant resistance against *O. neolycopersici* in the susceptible cultivated tomato genotype *S. lycopersicum* 'Amateur', whereas almost no change in resistance was observed in the highly resistant wild accession *S. habrochaites*, likely due to its elevated basal resistance. The authors also identified contrasting transcriptomic responses between the two genotypes. Following BABA treatment, defense gene induction was stronger in the susceptible genotype, whereas oligandrin triggered a greater transcriptional response in the resistant genotype.

Similar genotype-dependent PRI effects have been reported in other plant species. In carrot, Le Clerc et al., (2025) demonstrated that PRI efficacy against *Alternaria dauci* varied according to the plant genotype, both in terms of defense gene induction and resistance enhancement, with little or no protection observed in highly resistant genotypes. In barley, genotype-dependent responses to N-acyl homoserine lactone (AHL) against leaf rust caused by *Puccinia hordei* were reported by Wehner et al., (2019). Comparable results were obtained with acibenzolar-S-methyl (ASM), β-aminobutyric acid (BABA), and cis-jasmone treatments against *Rhynchosporium secalis* and *Blumeria graminis f. sp. hordei,* with genotype × environment interactions also observed (Walters et al. 2011).

Plant genotypic variability in PRI responsiveness has also been described in perennial crops. For example, Marolleau et al., (2017) showed that treatment with acibenzolar-S-methyl (ASM) in apple induced the highest expression of defense-related genes in the moderately susceptible cultivar 'Elstar', whereas the lowest induction levels were observed in the highly susceptible cultivar 'Pink Lady'.

Despite the large number of studies demonstrating plant genotype as an important factor affecting the efficacy of PRIs, only a few studies have investigated the genetic basis of these variations with a large number of genotypes. Ahmad et al., (2011) identified, in *Arabidopsis thaliana* recombinant lines derived from the cross Col-0 × Bur-0, genomic regions associated with

the induction of the salicylic acid-induced defense genes *PR-1* on chromosome IV and two loci regulating flagellin-induced callose on chromosome I and III. Bénéjam et al., (2021), had detected apple scab and fire blight resistance quantitative trait loci (QTLs) under acibenzolar-S-methyl (ASM) treatment conditions. A panel of wheat varieties screened for N-acyl homoserine lactone (AHL)-mediated priming assay identified 15 QTLs on chromosomes 1A, 1B, 2A, 3A, 3B, 3D, 6A, 6B, suggesting the presence of genetic variation in priming efficiency in breeding germplasm (Soleimani et al. 2024).

In tomato, only one study has investigated PRI efficacy across multiple genotypes, using a panel of 32 accessions, but without exploring the genetic basis underlying the observed variability (Sharma 2010). To date, neither the heritability of PRI responsiveness nor the plant genetic loci associated with variation in tomato responses to PRI application have been characterized. However, given the major agronomic and scientific importance of tomato, elucidating the genetic architecture underlying inducibility by PRIs has become essential for improving integrated disease management strategies and for providing transferable knowledge and tools to other Solanaceae species.

Key points about PRI efficacies

- Several factors influence PRI protective efficacy against diseases.

- The underlying molecular causes of these factors remain poorly understood.

- Regarding the plant genotype factor, only a few studies have investigated the genetic basis underlying variability in PRI responsiveness.

- To date, no study has characterized the genetic determinism of PRI responsiveness in tomato, regardless of the pathosystem considered.

4. Concluding remarks

This literature review highlights two major limitations in current tomato disease management strategies involving genetic resistance and plant resistance inducers (PRIs).

First, most resistance genes and QTLs introgressed into cultivated tomato originate from a limited number of wild relatives, particularly *S. pimpinellifolium*, mainly because of its high

crossability with cultivated tomato and the reduced risk of linkage drag. Consequently, despite the economic importance of several diseases, no commercial tomato cultivar currently carries resistance genes or QTLs against *B. cinerea*, because all the genes and QTLs found are originated from green-fruited species. Tomato cultivars resistant to powdery mildew probably all carry the *ol-2* resistance gene. This narrow genetic basis increases the risk of resistance breakdown and highlights the need to identify novel resistance genes and QTLs from other wild tomato relatives close to cultivated tomato to broaden the available usable sources of resistance and facilitate gene pyramiding strategies.

Second, this review emphasizes the limited understanding of the genetic determinants underlying inducibility of resistance by PRIs in tomato. To date, no study has systematically investigated the genetic determinism of tomato responses to PRIs or identified the genomic regions associated with variability in PRI efficacy across genotypes. The few studies conducted in other plant species indicate that host genetic background strongly influences PRI responsiveness and contributes to genotype differences in induced resistance. Therefore, it is essential to determine whether similar genetic effects occur in tomato and whether these responses vary depending on the PRI used and the targeted pathogen.

Objectives and structure of the thesis

The long-term objective of this thesis is to provide breeders with new molecular tools for, on the one hand, the selection of naturally resistant tomato varieties and, on the other hand, the selection of varieties showing an effective response to plant resistance inducers (PRIs). This thesis therefore aims to better understand the genetic basis of plant responses to PRIs through the study of the genetic variability of induced resistance in tomato against two major tomato pathogens.

The aim of this thesis is to map the loci underlying variation in tomato accession responses to PRI application against *Phytophthora infestans* and *Oidium neolycopersici* through a genome-wide association study (GWAS). The results are presented in the **second and third chapters**, respectively.

Additionally, we mapped new resistance QTLs against *O. neolycopersici* and *Botrytis cinerea* in a wild tomato relative that has not yet been explored for these traits. The **fourth chapter** focuses

on the genetic mapping of tomato resistance loci against *O. neolycopersici* using the QTL-seq approach combined with linkage mapping.

The **fifth chapter** reports additional work carried out during the thesis that is not presented in detail in the main manuscript. Finally, the overall results obtained during this thesis are discussed in the **sixth chapter**.

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Chapter 2

GWAS tomato responses to PRI – *Phytophtora infestans*

This chapter is presented as an original article intended for submission to the journal *New Phytologist*.

In this chapter, we focus on identifying QTLs associated with tomato accessions responses to the application of a commercial PRI "Belvine®" against late blight caused by *P. infestans*.

Tomato genomic regions and candidate genes linked to responses to a resistance inducer against *Phytophthora infestans*

Awa Sangaré[1,2], Anne Massire[2], Magali Duffaud[1], Esther Pelpoir[2], Jean-François Bourgeay[1], Matthieu Gaucher[3], Mathilde Causse[2], Marc Bardin[1], Bernard Caromel[2]

Summary

- Plant resistance inducers (PRIs) represent a promising and sustainable strategy for pest and disease management, but their efficacy can vary depending on plant genotype. Our objective was to identify the genetic architecture underlying tomato responses to the application of a PRI.
- We have established that Belvine® is an effective PRI against tomato late blight caused by *Phytophthora infestans*. Analysis of defense-related gene expression revealed that PR proteins, oxidative stress-related, and ethylene-associated genes were upregulated in Belvine®-treated plants.
- A Genome Wide Association Study panel of 148 tomato accessions was grown under plastic tunnel either treated with Belvine® or left untreated, then inoculated with *P. infestans*. We identified five quantitative trait loci (QTLs) associated with genotypic variation in Belvine® efficacy, located on chromosomes 2, 3, 4, and 12. Within the significant regions, we identified several candidate genes, including protein kinases, receptor-like protein kinases, transcription factors, and PR proteins. Notably, several candidate genes encoded proteins with functions analogous to those of genes upregulated under Belvine® treatment, including β-1,3-glucanases (PR-2), glutathione S-transferase, and peroxidase, even though they may correspond to distinct copies of these gene families.
- The identified QTL regions and candidate genes constitute valuable genomic resources for defining tomato breeding strategies aimed at enhancing plant responses to PRIs.

Keywords: Resistance induction, genes, QTL, GWAS, genotypic variation, *late blight*, biocontrol, tomato

1. Introduction

Late blight, caused by the oomycete *Phytophthora infestans*, is a devastating disease affecting both cultivated tomato (*Solanum lycopersicum*) and potato (*Solanum tuberosum*), frequently leading to severe crop losses (Fry 2008; Panthee et al. 2024). Copper-based products and synthetic fungicides are widely used to control the disease. Although these compounds can effectively suppress disease development in the short term, their repeated and intensive use may promote the emergence of resistant pathogen populations (Abuley et al. 2023). Indeed, resistance of *P. infestans* to several fungicide classes has already been reported, highlighting the limitations of chemical control strategies and the need for more sustainable disease management approaches (Saville et al. 2015; Olave-Achury et al. 2022).

The cultivation of resistant varieties is an effective, economical, and environmentally friendly strategy to reduce the negative impacts of *P. infestans*. To date, more than 40 resistance genes and QTLs of resistance to *P. infestans* have been identified in different wild tomato relatives, including *Solanum pimpinellifolium*, *S. pennellii*, and *S. habrochaites* (Brouwer et al. 2004; Smart et al. 2007; Merk et al. 2012; Chen et al. 2014a; Panthee et al. 2017; Sullenberger et al. 2022; Gao and Foolad 2024; Jia et al. 2025). However, only a few of these resistance loci have been introgressed into cultivated tomato. Among the introgressed genes, *Ph-1* and *Ph-3* have already been overcome by certain *P. infestans* isolates (Foolad et al. 2008; Sullenberger and Foolad 2024). Therefore, alternative solutions are needed for improving the durability of resistance.

Biosolutions that induce plant resistance are promising alternatives to conventional fungicides. These plant resistance inducers (PRIs) encompass a wide range of natural and synthetic compounds, including plant extracts, beneficial microorganisms and their derivatives, as well as phytohormones such as salicylic acid (SA) (Jamiołkowska 2020; Hönig et al. 2023). When applied to plants, PRIs activate signaling pathways involved in plant immunity, leading to the regulation of defense-related genes and the production of antimicrobial and protective compounds, including pathogenesis-related (PR) proteins, reactive oxygen species (ROS), secondary metabolites, and cell wall-strengthening components (Cohen et al. 2016; Yarullina et al. 2022; Alamri et al. 2026). These defense responses help restrict pathogen growth and spread and may result in induced systemic resistance (ISR) or systemic acquired resistance (SAR), thereby reducing plant susceptibility to subsequent pathogen attacks (Floryszak-Wieczorek et al. 2015). For instance, the application of β-aminobutyric acid (BABA) induced

the expression of multiple defense-related transcripts and enhanced resistance to powdery mildew in tomato (Satková et al. 2016; Zapletalová et al. 2023). Another important mode of action of PRI is the priming phenomenon. Priming prepares plants for faster and stronger defense responses upon pathogen challenge without constitutively activating defense pathways (Hilker and Schmülling 2019; Hönig et al. 2023). Tanaka et al. (2024) demonstrated that treatment of tomato plants with L-methionine alone did not activate salicylic acid (SA)-, jasmonic acid (JA)-, or ethylene (ET)-mediated defense signaling, but SA- and JA-responsive defense genes were induced more rapidly following *Pseudomonas syringae* pv. *tomato* and *Botrytis cinerea* infection in treated plants.

The induction of resistance in plants is a complex process whose effectiveness depends on the plant genetics, as well as its physiological status, and the environmental conditions (Bruce 2014). In plant, responses to treatments with a PRI have been documented at multiple levels, including phenotypic, transcriptomic, and metabolic levels. Recently, Margaritopoulou et al. (2024) demonstrated that *Reynoutria sachalinensis* extract enhanced phospholipid production and signaling in one zucchini genotype susceptible to *Podosphaera xanthii*, whereas only limited responses were observed in one genotype with intermediate resistance, likely due to genetic resistance mechanisms. In tomato, the use of *Trichoderma* spp. against *B. cinerea* resulted in contrasting outcomes depending on the plant genotype, with no effect or even detrimental effects observed in certain genotypes (Tucci et al. 2011). Similarly, Sharma et al. (2010) reported genotype-dependent variation in BABA-induced resistance against *P. infestans*. Transcriptomic differences were also observed by Satková et al. (2016), who showed that PRI induced defense-related gene expression in the susceptible tomato cultivar *S. lycopersicum* 'Amateur', but not in the resistant accession 'LA 2128' belongings to the wild species *S. habrochaites f. glabratum*. However, the genetic mechanisms underlying this variation remain poorly understood. The identification of genomic regions or candidate genes associated with plant responses to PRIs in tomato could provide new resources to enhance resistance defense.

The objective of this study was to decipher the genetic architecture underlying tomato resistance to *P. infestans* following the application of a biosolution that induces plant defense pathways. After demonstrating that this biosolution can induce defense pathways in the plant and that its protective efficacy depends on the tomato genotype, a Genome Wide Association Study (GWAS) panel of 148 tomato accessions was grown under plastic tunnels and either treated with the PRI or left untreated prior to *P. infestans* inoculation. GWAS identified five

QTLs located on chromosomes 2, 3, 4 and 12 that were associated with genotypic variation in PRI efficacy against *P. infestans*. These findings provide valuable insights for breeding programs aimed at selecting tomato varieties with high responsiveness to PRIs.

2. Material and methods

Plant material, pathogen strains

Plant material

The panel of tomato genotypes used to analyze the genetic response of tomato accessions to the application of PRI was composed of a core collection of 150 homozygous lines, including 116 cherry tomato accessions belonging to *S. lycopersicum* var. *cerasiforme* (SLC), 10 *S. pimpinellifolium* (SP), 18 admixte accessions of SLC and SP, 5 large-fruited tomato *S. lycopersicum* var. *lycopersicum* (SLL), and the commercial cultivar Clodano (Syngenta). Most of them have been used and characterized in previous studies (Albert et al. 2016; Bineau et al. 2021; Héreil et al. 2024). The list of accessions is provided in **Supplementary data 1**. All seeds from the accessions used in this study, with the exception of seeds from the Clodano variety, were produced by the Centre for Vegetable Germplasm (CRB-Leg) of the GAFL research unit (INRAE, Avignon, France). Five of these accessions (Clodano, Criollo, Ecu284, Marpha n2 and Vir884) were used to evaluate the expression of defense genes induced by Belvine®, and eleven accessions (Clodano, Criollo, Vir884, LA2619, Vir2759, Vir744, Ferum, Momor, Rose de Berne, St Pierre, and Vir859) were used to assess whether there is a genotypic variation among tomato accessions regarding the response to the application of the PRI.

Pathogen material

The *P. infestans* strain Pi147 was used in this study. Strain Pi147 was obtained from an infected tomato fruit collected in Bucheley (78, France) in 2021. The strain is routinely maintained by monthly subculturing on V8 agar medium in Petri dishes and incubated in a growth chamber at 18 °C in the dark. Three weeks prior to inoculation, the pathogen was used to infect leaflets of the susceptible tomato accession Momor (INRAE, Avignon, France) and transferred after seven days onto fresh, healthy leaflets of the same accession to restore and maintain its aggressiveness. The pathogen inoculum was prepared by washing heavily infected tomato leaflets in tap water and using them immediately. Sporangia concentration was

estimated with a Malassez counting chamber, and the suspension was adjusted to a final concentration of 5×10^3 sporangia·mL^{-1}.

Biosolution

Preliminary studies have shown that the commercial natural substance Belvine® (Cerience, https://www.cerience.fr/fr/solutions/belvine) is effective in protecting tomato against *P. infestans* (data non-shown). This biosolution is approved in the European Union and has obtained national authorizations in seven-member states, including France, as a plant protection product for the management of grapevine downy mildew. The active substance of the product (ABE-IT 56) consists of components derived from a lysate of the *Saccharomyces cerevisiae* strain DDSF623 (https://ec.europa.eu/food/plant/pesticides/eu-pesticides-database/start/screen/active-substances/details/1307). According to the manufacturer, the product has a preventive effect by stimulating the natural defense responses of grapevine (https://www.cerience.fr/fr/solutions/belvine). In this study, this biosolution was sprayed onto the foliage of tomato plants before inoculation with the pathogen, at a dose of 2% (V/V).

Experimentations and plant growth

This study comprised three independent trials. Trial 1 focused on assessing the induction of tomato defense-related genes following treatment with the biosolution. Trial 2 evaluated genotype-dependent variation in the efficacy of Belvine® against *P. infestans* across a panel of tomato accessions. Trial 3 was designed to map genomic loci associated with the plant response to the biosolution.

Seeds for the trials were directly sown into 9 x 9 cm pots containing seedling substrate (K substrates).

Trial 1: assessment of tomato defense genes induced by the Belvine®

To analyze the expression of tomato defense genes induced by Belvine®, two independent experiments were conducted in 2023 in a glasshouse under semi-controlled conditions, with a two-week interval between each experiment. Five tomato accessions were included in each experiment. Plants were assigned to two treatment groups: a Belvine®-treated group and an untreated control group. For each accession × treatment combination, three plants were used. Four-week-old plants assigned to the treated group were sprayed with approximately 20 mL of Belvine® solution per plant. The treatments were applied twice, at 7 and 2 days before sampling. Following the first treatment (Day -7), a ring was placed near the apical meristem to

mark leaf position. On Day 0, one leaf located below the ring was collected from each of the three plants for each accession × treatment combination. Five leaf discs (10 mm in diameter) were excised from each leaf. The discs collected from the three plants were pooled to constitute a single biological sample for each accession × treatment combination within each experiment. The same sampling procedure was performed in both independent experiments, resulting in two independent biological samples per accession × treatment combination for gene expression analysis, one from each experiment. Samples were immediately frozen in liquid nitrogen to prevent RNA degradation.

Total RNA was extracted using the Macherey-Nagel Nucleospin RNA Plant Kit with slight modifications to the volume and reverse transcription was performed as described by Promega Corp. (M-MLV). Quantitative real-time PCR was performed as described in Gaucher et al. (2022), using a patented set of primers targeting 28 tomato defense genes and three reference genes (Brisset and Dugé de Bernonville, 2011), listed in **Table S1**. Relative changes in defense gene expression ($\log_2$ ratios) were calculated using the $2^{-\Delta\Delta CT}$ method (Schmittgen and Livak 2008) with normalization based on the three reference genes (Vandesompele et al. 2002). An untreated sample of the tomato variety 'Clodano' was used as the calibrator for the calculation of $\log_2$ expression ratios. Genes were considered differentially expressed when the absolute $\log_2$ fold change exceeded 1 and the p-value was ≤ 0.05. For data presentation, gene expression values were averaged across the two independent experiments for each accession × treatment combination.

Trial 2: evaluation of the genotype-dependent variability in Belvine® efficacy

The experiment to evaluate the variability in the protective efficacy of Belvine® against *P. infestans* depending on tomato genotype was carried out in autumn 2023 in an unheated 600 m² plastic tunnel. Eleven tomato accessions with comparable levels of susceptibility to tomato late blight were included. Two treatment conditions were tested: a control group of untreated plants and a group of plants treated with Belvine®. Each treatment × accession combination consisted of four biological replicates. Plants were first grown in a glasshouse for four weeks, then transplanted into the tunnel. The tunnel was organized into 18 blocks, with two blocks randomly assigned to each treatment. Each block consisted of two rows of 11 plants, with one plant per accession randomly distributed within each row. Eight-week-old plants were treated with approximately 40–50 mL per plant of a solution of Belvine®, and two applications performed 8 days and 3 days before pathogen inoculation.

Trial 3: map loci involved in plant response to Belvine®

The experiments were conducted from late summer (August) to late autumn (December), a period conducive to the development of tomato late blight in the Avignon region. Seeds were sown individually in 9 × 9 cm pots filled with seed-starting mix and then grown in a glasshouse under semi-controlled conditions. Six-week-old plants were then transplanted into two 600 m² plastic tunnels to mimic natural production conditions. Each tunnel contained six rows: two single rows used as borders, and two double rows. Each double row was divided into two blocks corresponding to two modalities: modalities: on one side, plants treated with Belvine®, and on the other, untreated control plants (**Fig. S1**).

The two blocks of treated plants were surrounded by one row of untreated tomato plants of the Rose de Berne variety. In each block, 150 tomato accessions were planted randomly within the double row, with a few minor adjustments made to limit the number of times the same accession appeared in the immediate neighborhood of another to a maximum of two. Plants were treated with Belvine® with approximately 20 mL per plant, and two applications performed 7 days and 3 days before pathogen inoculation. Following the last treatment, the height of each plant was recorded by marking its position on the supporting twine. The experiments were repeated over two years, in 2024 and 2025. Although 150 tomato accessions were initially included, only 148 with available genetic data were retained for genetic analysis. Data from one tunnel in 2024 were excluded from the analysis due to insufficient disease development. Consequently, three tunnels were retained for the analysis: B24, B25, and A25, corresponding to tunnel B in 2024, tunnel B in 2025, and tunnel A in 2025, respectively.

Inoculation and disease assessment

Plants were inoculated with 15-20 ml per plant of a 5×10^3 sporangia·mL^{-1} suspension, using a hand-held sprayer.

Resistance to *P. infestans* was evaluated 7-10 days after inoculation. Prior to phenotyping, the first and second leaves located immediately below the plant height reference mark were selected for symptom scoring. In 2024, infestation levels were very low, especially on untreated plants seven days after inoculation. Consequently, plants intended for PRI treatment were retreated twice as previously described and inoculated 3 days after the last treatment. Only leaves that underwent both inoculations and located under the first plant height reference mark were considered for evaluation 7 days after the second inoculation. Phenotyping consisted of recording per leaf (i) the total number of leaflets, (ii) the number of infected leaflets, and (iii)

the severity of infection on infected leaflets. Disease severity on infected leaflets was assessed using a 0-4 ordinal scale, where 0 = no visible symptoms; 1 = 1-25% of leaflet area infected; 2 = 26-50% of leaflet area infected; 3 = 51-75% of leaflet area infected; and 4 = more than 75% of the leaflet area infected (**Fig. S2**). The disease index (DIS) was calculated as follows:

$$DIS = \frac{\mathbf{NIL\ x\ ILAS}}{\mathbf{NL}}$$

where NL is the total number of leaflets per leaf, NIL is the number of infested leaflets per leaf, and ILAS is the disease severity index. For each plant, DIS values from the two assessed leaves were averaged to obtain a single value per plant.

Evaluation of the efficacy of the biosolution

The efficacy of the biosolution was estimated for each plant as the percentage of protection of the plant using the formula according to (Tamm et al. 2011; Zhang et al. 2026) as:

$$\mathrm{PP} = \mathbf{100} * \left(\mathbf{1} - \frac{DISp1(ij)}{DISntc}\right)$$

Where PP is the percentage of protection, DISp1(ij) is the disease index in treated condition of the i^{th} plant of the j^{th} genotype, and DISntc is the average of the disease index in untreated control treatment for the j^{th} genotype. In trial 3, DISntc was calculated for each tunnel independently.

Before assessing efficacy, spatial effects on the disease index were corrected. For trial 2, the spatial correction was performed using the functions of the R package SpATS (Rodríguez-Álvarez et al. 2018). For trial 3, the spatial correction was performed using the functions of the R package statgenHTP (Millet et al., 2026).

Estimation of genotype means and heritabilities

A linear mixed model (**1**) of the Rpackage/lme4 (Bates et al. 2015), was used to estimate the variances of the random effects and to calculate the broad-sense heritability (**2**). Different mixed models were used to estimate variances for heritability and best linear unbiased predictors (BLUPs). The broad-sense heritability was calculated following formula.

$$\mathbf{Y_{ijk} = \mu + G_i + E_j + G_i \times E_j + \mathcal{E}_{ijk}} \quad \textbf{(1)}$$

$$H^2 = \frac{\mathbf{Vg}}{\mathbf{Vg} + \frac{Vge}{T} + \frac{Vr}{RT}} \quad \textbf{(2)}$$

In mixed model, Y is the trait, μ is the general mean of the trait, G_i is the genetic effect of the i^{th} genotype, following a normal distribution $G \sim N(0, Vg)$, E_j is the effect of j^{th} tunnel with E a fixed effect in the model, G x E is the genotype and environment interaction effect, which follows a normal distribution $G \times E \sim N(0, Vge)$, and Ɛ are the residuals assumed to be independent and normally distributed with $\mathcal{E} \sim N(0, Vr)$.

In heritability formula, H^2 is the broad-sense heritability of the trait, Vg is the genetic variance, Vge is the variance of the genotype and environment interaction, and Vr is the residual variance, T is the number of tunnels, and R is the number of replicates per tunnel. The effects of tunnels (E) and GxE were used only for combined dataset.

The genomic heritability was calculated using GBLUP on GAPIT 3. It was calculated with the genetic values of the percentage of protection (BLUP + mean). All statistical analyses were performed using R software. Descriptive statistics and analysis of variance (ANOVA) were used to test the difference between accessions, tunnels and the interaction between accessions and tunnels. When the ANOVA test shows a significant difference, a post-hoc test was performed using Tukey's hsd.test function.

Genotypic data and GWAS

To detect associations between genotypic values and genomic markers, GWAS was performed using a set of 6,251,927 SNPs from genome resequencing (Héreil et al. 2024). SNP positions refer to the Heinz_1706 v.4.0.0 reference genome (SL4.0). SNPs exhibiting more than 3% heterozygosity were excluded from the dataset. All remaining heterozygous genotype calls were subsequently recoded as missing. Missing data were imputed by substituting the major allele, defined as the allele with the highest observed frequency across all samples. Following imputation, SNPs were filtered to retain only those with a minor allele frequency (MAF) greater than 10%. The final set of 82,969 SNP markers was retained and used for the identification of informative markers by GWAS. The LD parameter r^2 between pairs of SNPs was calculated using $-r^2$ command in PLINK software (Purcell et al. 2007) version based on a window size of 1000 kb, following the method described by (Wang et al. 2017).

To identify marker-trait associations to the traits, a GWAS was conducted using the Bayesian-information and Linkage-disequilibrium Iteratively Nested Keyway (BLINK) (Huang et al. 2019) implemented in Genomic Association and Prediction Integrated Tool GAPIT 3 (Wang and Zhang 2021). Both population structure and kinship matrices were used as covariates to reduce false-positive associations due to population structure and familial relatedness. The population structure was calculated as principal component analysis (PCA) with the first three components. The kinship matrix was generated automatically in GAPIT 3 using the VanRaden method (VanRaden 2008). We used a genome-wide threshold ($P < 1.77x10^{-6}$) that was determined based on the effective number of independent markers, Me, estimated using the Gao et al. (2008) method on R software. The genome-wide threshold was calculated with the equation, 0.05/Me.

The phenotypic variance explained (PVE) by a significant marker was estimated using the equation in R (Kim et al. 2021):

$$\boldsymbol{PVE = \frac{SSsig.marker}{SSallsig.marker + e} x100}$$

where SS is the sum of square and *e* is the residuals from the ANOVA fitted with a linear model incorporating the phenotypic data and all significant markers in decreasing order of their P-value (Knoch et al. 2020). Significant SNP markers in linkage disequilibrium (LD) with each other ($r^2 \geq 0.5$) were considered to represent the same QTL.

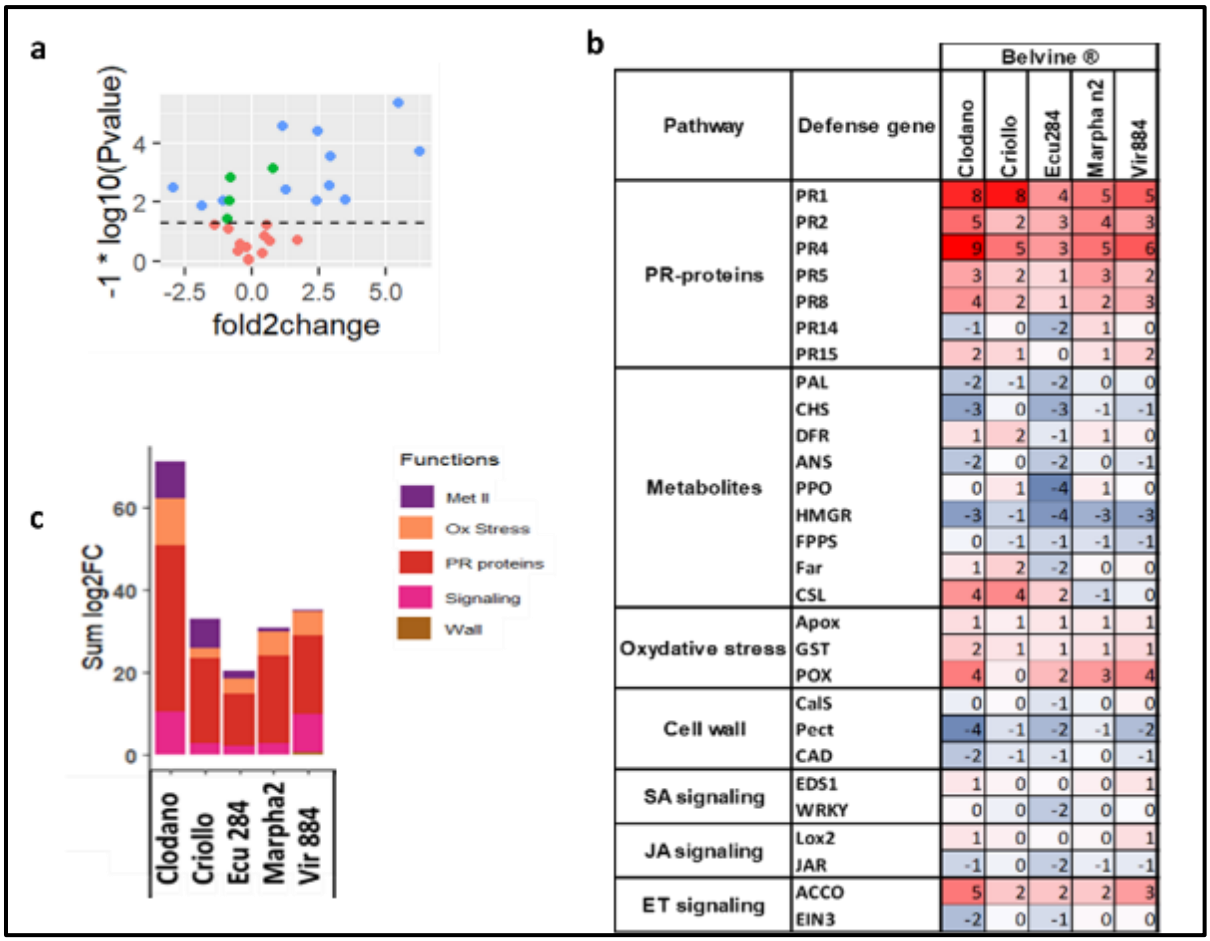
Candidate gene identification

Genome-wide linkage disequilibrium (LD) decay was estimated by plotting the squared allele frequency correlation (r^2) against physical distance between SNPs. LD decay was defined as the chromosomal distance at which r^2 decreased to half of its maximum value (Wang et al. 2017), resulting in intervals ranging from ~600 kb to ~1 Mb. Therefore, these distances were used to define candidate genomic regions around significant SNPs. Candidate genes were investigated using the tomato reference genome assembly SL4.0 and the international tomato annotation group (ITAG) 4.0 at the Sol Genomics Network (https://solgenomics.net). From this subset, candidate genes were identified by manually searching gene descriptions for disease related genes. Candidate genes were also explored through a literature review.

3. Results

Defense genes induced by Belvine®

When the five tomato genotypes were analyzed together, Belvine® treatment resulted in the identification of 12 differentially expressed genes (DEGs) (**Fig. 1a**). Among these, nine genes



		Belvine ®				
Pathway	Defense gene	Clodano	Criollo	Ecu284	Marpha n2	Vir884
PR-proteins	PR1	8	8	4	5	5
	PR2	5	2	3	4	3
	PR4	9	5	3	5	6
	PR5	3	2	1	3	2
	PR8	4	2	1	2	3
	PR14	-1	0	-2	1	0
	PR15	2	1	0	1	2
Metabolites	PAL	-2	-1	-2	0	0
	CHS	-3	0	-3	-1	-1
	DFR	1	2	-1	1	0
	ANS	-2	0	-2	0	-1
	PPO	0	1	-4	1	0
	HMGR	-3	-1	-4	-3	-3
	FPPS	0	-1	-1	-1	-1
	Far	1	2	-2	0	0
	CSL	4	4	2	-1	0
Oxydative stress	Apox	1	1	1	1	1
	GST	2	1	1	1	1
	POX	4	0	2	3	4
Cell wall	CalS	0	0	-1	0	0
	Pect	-4	-1	-2	-1	-2
	CAD	-2	-1	-1	0	-1
SA signaling	EDS1	1	0	0	0	1
	WRKY	0	0	-2	0	0
JA signaling	Lox2	1	0	0	0	1
	JAR	-1	0	-2	-1	-1
ET signaling	ACCO	5	2	2	2	3
	EIN3	-2	0	-1	0	0

Fig. 1 Defense genes induced by Belvine® three days after the second treatment based on the data from the 5 tomato accessions with two independent replicates for each combination accession x treatment. a) volcano plot of the 28 defense genes based on the data from the 5 tomato accessions. The horizontal axis represents the log2 values of the expression ratio, and the vertical axis represents the p-values (-log10). Blue dots indicate differentially expressed genes (|log2foldchange| >= 1 and p-value ≤ 0.05). Orange dots indicate genes not meeting the p-value criterion, and green dots indicate genes reaching the p-value criterion but not the log2 expression ratio criterion. b) relative gene expression in the tomato genotypes Clodano, Criollo, Ecu284, Marpha n2, and Vir884. Values represent the log2folchange values with untreated control plants used as the reference. Each value represents the mean of two independent experiments. Positive values indicate gene upregulation, whereas negative values indicate gene downregulation. c) Genetic variation in the induction of defense-related genes. The y-axis represents the cumulative $\log_2$ fold changes of upregulated genes (log2foldchange >=1). Each color corresponds to the pathways to which belongs the gene: purple = secondary metabolism, orange = oxidative stress, red = pathogenesis-related (PR) proteins, pink = hormonal signaling pathways, and brown = cell wall–related genes

were upregulated (*ACCO*, *POX*, *PR1*, *PR2*, *PR4*, *PR5*, *PR8*, *PR15*, *CSL*) whereas three genes (*HMGR*, *PAL*, *Pect*) were downregulated. Overall, the induction pattern was consistent across the five genotypes. The upregulated genes were mainly associated with PR protein and oxidative stress responses, whereas genes Involved in SA- and JA-signaling showed less induction by the treatment (**Fig. 1b**). Within the ET-signaling pathway, *ACCO* was strongly upregulated, whereas *EIN3* was downregulated. Most genes involved in cell-wall reinforcement and secondary metabolite biosynthesis were downregulated, except *CSL*, which was upregulated in Clodano, Criollo, and Ecu284 (**Fig. 1b**). Differences in gene induction levels were also observed between accessions. Clodano showed the strongest defense gene induction, followed by Criollo, Marpha n2, and Vir884 (**Fig. 1c**), whereas Ecu284 displayed the lowest number of upregulated genes. Altogether, these results highlight substantial genotypic variation in the transcriptional response of tomato to Belvine® treatment. Within the ET-signaling pathway, *ACCO* was strongly upregulated, whereas *EIN3* was downregulated. Most genes involved in cell-wall reinforcement and secondary metabolite biosynthesis were downregulated, except *CSL*, which was upregulated in Clodano, Criollo, and Ecu284 (**Fig. 1b**). Differences in gene induction levels were also observed between accessions. Clodano showed the strongest defense gene induction, followed by Criollo, Marpha n2, and Vir884 (**Fig. 1c**), whereas Ecu284 displayed the lowest number of upregulated genes. Altogether, these results highlight substantial genotypic variation in the transcriptional response of tomato to Belvine® treatment.

Variation in Belvine®-induced resistance across genotypes

All 11 genotypes showed close levels of baseline susceptibility (scores ranging from 2 to 3) (**Fig. S3**). Treatment with Belvine® reduced late blight severity by an average of 89.1%. However, significant genotypic variation in treatment efficacy was observed among the 11 tomato accessions (ANOVA, $p<0.001$; **Fig. 2**). Accessions treated with Belvine® could be assigned into two categories: those that showed no symptoms of disease after inoculation (Clodano, Ferum, and St Pierre) with 100% protection, and all other accessions, which were partially protected, with a disease severity index lower than 1 but a protection level above 70% (**Fig. 2**).

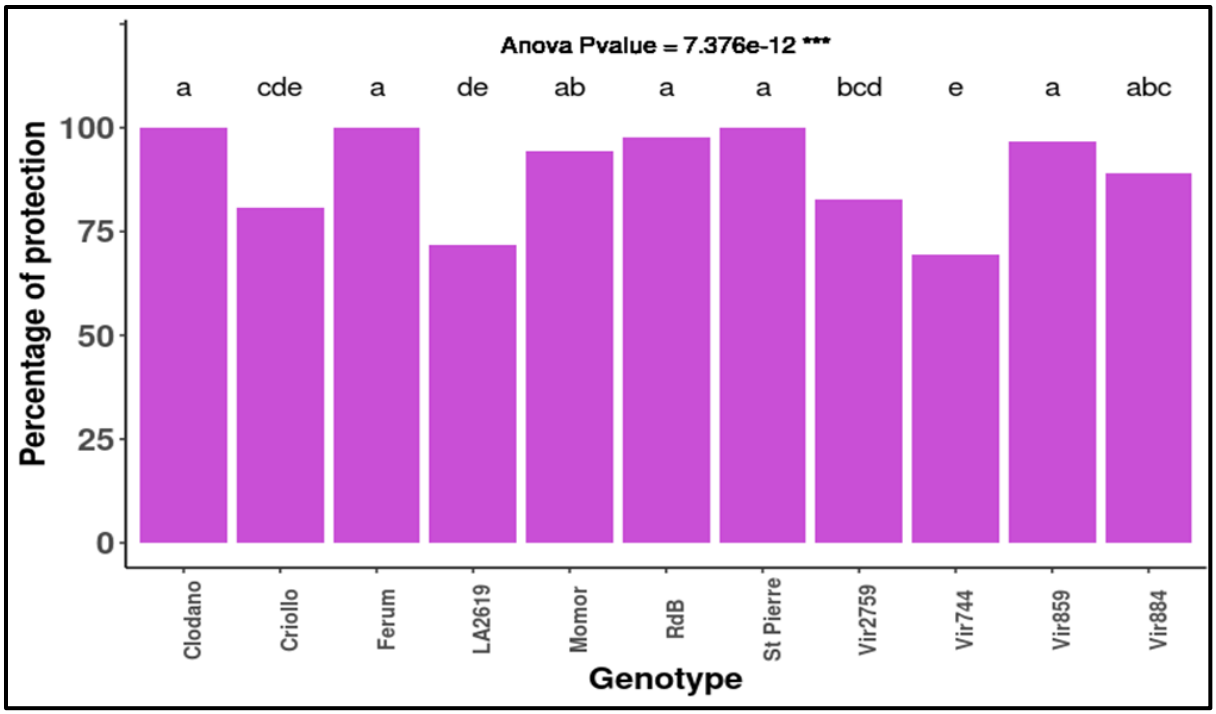


Fig. 2 Genotypic variation of Belvine® efficacy. Efficacy of Belvine® against late blight across the eleven accessions. Different letters indicate statistically distinct groups among genotypes based on Tukey's post hoc test. Asterisks (*) denote significant differences between treated and control modalities according to ANOVA

Phenotypic variation within 148 tomato accessions

The GWAS experiments revealed that Belvine® treatment consistently reduced the average disease index across all tomato accessions in each of the three independent experimental repetitions (tunnels). This trend was also observed for the overall disease index calculated across all replicates (**Fig. 3; Fig. 4**). However, the reduction in disease development following treatment was not uniform among the different accessions. Differences in response were observed even among accessions displaying similar levels of susceptibility under the untreated control conditions (**Fig. 3**). In terms of protection, Belvine® was the most effective in tunnel B25, with a mean protection of 65.73% and a standard deviation of 20.63 (**Fig. 4**). The protective efficacy observed in tunnels B24 and A25 was relatively similar, with mean protection values of 46.14% and 45.21%, respectively. The overall mean protection conferred by Belvine® was estimated at 52.54%. Histograms of the genetic values for percentage of protection (BLUP + mean) showed an approximately normal distribution of this trait in all tunnels as well as in the combined dataset (**Fig. S4**). All 148 accessions exhibited a favorable response to Belvine® application, with protection percentages ranging from 8.71% to 92.76% according to the combined phenotypic data (**Fig. S4**). The correlations between disease index

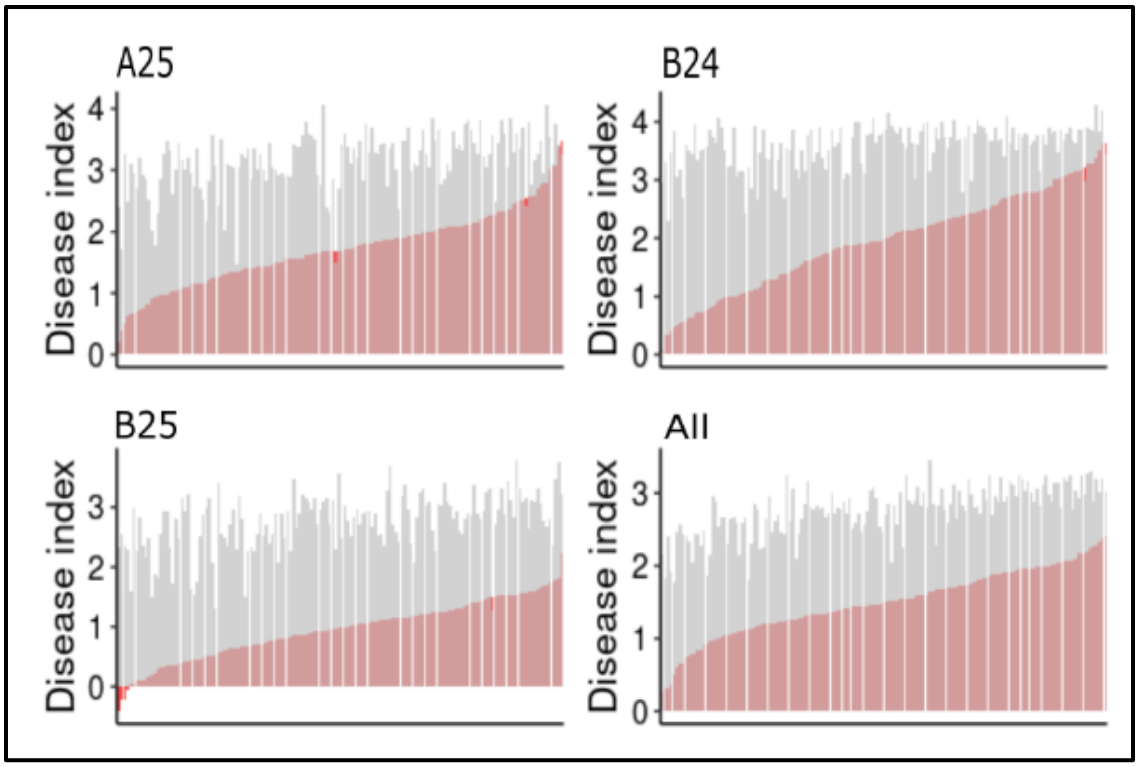


Fig. 3 Variability of 148 tomato accessions resistance to *P. infestans* under control and Belvine® treatment. X axis presents the accessions. Grey represents the disease index under untreated conditions and red color represents disease index under Belvine® condition. A25: Phenotypic data in tunnel A25, B24: Phenotypic data in tunnel B24, B25: Phenotypic data in tunnel B25, and All: the combined phenotypic data of the three tunnels. The order of accessions varies between plots

values under control and Belvine® treatments were positive and statistically significant (p-value < 0.001) in all datasets (**Fig. S5a**). The Correlation coefficients were r = 0.42, r = 0.42, and r = 0.48 in tunnels A25, B24, and B25, respectively, while a higher correlation was observed for the combined dataset (r = 0.59). The correlation between disease index under control conditions and percentage of protection was negative and statistically significant in all cases except for tunnel A25 (**Fig. S5b**). Significant correlations ranged from -0.22 to -0.32, with the strongest negative correlation observed in the combined dataset (r = -0.32).

Genetic effect on tomato accession responses to Belvine® after inoculation

Genetic effects were estimated through heritability, defined as the proportion of total phenotypic variation attributable to genetic variation. Broad-sense heritability was calculated for the disease index under both control and Belvine® treatments, as well as for the percentage of protection, which reflected PRI efficacy and plant response.

Under untreated control conditions, the broad-sense heritability of resistance to *P. infestans*

Environment	Trait	Mean	SE	H^2	Variance decomposition
T4B24	*Disease index in NTC*	3.58	0.03	0.17	0.17 / 0.83
	Disease index in P1	1.95	0.05	0.78	0.78 / 0.22
	Percentage of protection (PP)	46.14	1.40	0.75	0.75 / 0.25
T4B25	*Disease index in NTC*	2.75	0.03	0.53	0.53 / 0.47
	Disease index in P1	0.95	0.03	0.66	0.66 / 0.34
	Percentage of protection (PP)	65.73	1.19	0.62	0.62 / 0.38
T4A25	*Disease index in NTC*	3.13	0.04	0.34	0.34 / 0.66
	Disease index in P1	1.71	0.04	0.62	0.62 / 0.38
	Percentage of protection (PP)	45.21	1.26	0.60	0.6 / 0.4
All	*Disease index in NTC*	3.16	0.02	0.60	0.6 / 0.38
	Disease index in P1	1.53	0.03	0.66	0.66 / 0.18 / 0.16
	Percentage of protection (PP)	52.54	0.80	0.58	0.58 / 0.22 / 0.21

Fig. 4 Statistics of phenotypic data collected from 148 tomato accessions. H^2: Broad-sense heritability, NTC: untreated control condition; P1: Belvine® treated condition; PP: percentage of protection. In column variance decomposition: black represents the percentage of variance explained by genetic variation, orange represents the percentage of variance explained by the interaction between genetic x environments, and green represents the percentage of variance explained by residual variation. Phenotypic means correspond to the intercepts estimated from the linear mixed model

ranged from 17% to 60%, with the lowest value observed in tunnel B24 and the highest obtained using the combined dataset from all tunnels (**Fig. 4**). Under Belvine® treatment, the broad-sense heritability of induced resistance against *P. infestans* ranged from 62% to 78%, with a broad-sense heritability of 66% in the combined dataset. The broad-sense heritability of the percentage of protection was like that observed for resistance induced by Belvine® treatment, ranging from 58% to 75% (**Fig. 4**). The heritability of the combined dataset reached 58%.

The data also highlighted the influence of genotype × environment interactions. These interactions contributed more strongly to traits associated with Belvine® application than to traits measured under untreated control conditions (18-22% vs. 2%) (**Fig. 4**). Genomic heritability estimates were 46.40% in tunnel A25, 45.8% in tunnel B24, 60.1% in tunnel B25, and 63.3% for the mean across the three tunnels (**Fig. S4**).

Population structure

Population genetic diversity analysis was conducted on 148 accessions. The PCA eigenvalue plot (**Fig. S6a**), generated using GAPIT 3, indicating that the first three principal components (PCs) explained most of the variation among the 148 accessions (40.92 %). Phylogenetic trees were constructed using the neighbor-joining (NJ) method in GAPIT 3, resulting in a fan tree (**Fig. S6b**) displaying three distinct sub-populations. A 3D graphical plot of principal component analysis (PCA) (**Fig. S6c**) illustrated the three sub-populations. These plots and

phylogenetic trees revealed the presence of three distinct clusters within the core-collection. The three principal components of the PCA were subsequently used as a Q-matrix in the GWAS analysis. Among the three clusters (sub-populations), G1 consisted of 18 individuals (12.2% of the entire population), G2 included 123 individuals (83.1%), and G3 comprised 7 individuals. The three subpopulations were not classed as *S. lycopersicum*, *S. pimpinellifolium* and admixes (**Supplementary data 2**).

Identification of marker-trait associations for plant responses to Belvine®

A GWAS performed using the BLUPs obtained from the overall dataset identified three significant SNPs associated with tomato response to the application of Belvine® (**Fig. 5**). These SNPs were located on chromosomes 2 (SNP37241), 4 (SNP77453), and 12 (SNP246258). The strongest effect was observed for the SNP located on chromosome 12, followed by those on chromosomes 4 and 2 (**Table 1**). The phenotypic variance explained by these SNPs was 17.06%, 13.46% and 7.56%, respectively. Together, these loci accounted for 38% of the total phenotypic variance.

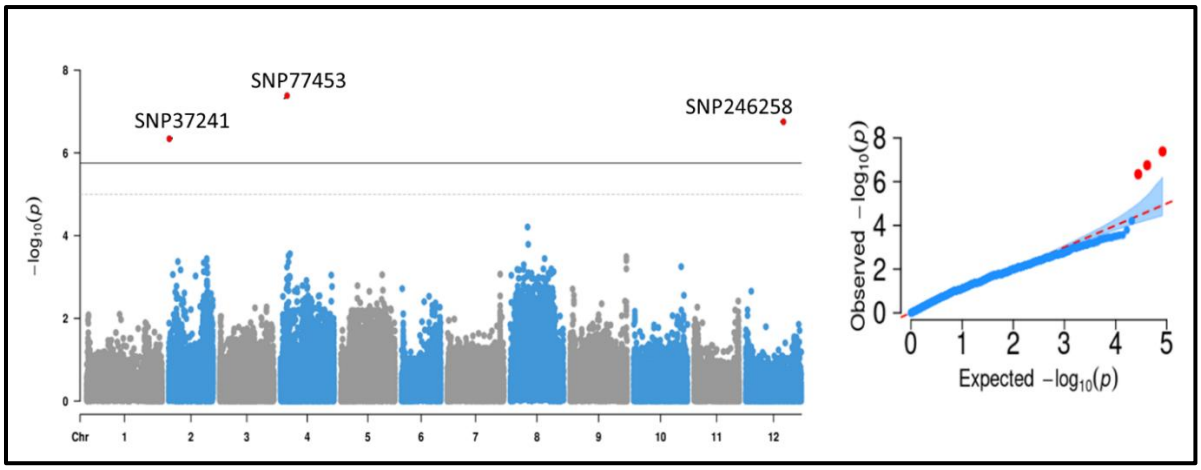


Fig. 5 SNP associated with plant responses to Belvine® application. Left**:** Manhattan plot showing significant SNP associated with plant responses to Belvine®. The X-axis shows SNPs along each chromosome; the Y-axis is the -log10P for the association. The gray lines indicate the Gao significance threshold (-log10(p) = 5.75); Red points indicate the significant SNPs with their name. Right: Corresponding quantile-quantile plots (QQplot) for plant responses. The X-axis shows expected -log10 (P), and the Y-axis shows observed -log10(p). Phenotypic data used for this analysis are the combine dataset (Blups) obtained from the linear mixed model containing GxE interaction

Single-environment GWAS analyses identified five SNPs associated with plant response to Belvine® (**Fig. 6**). Two SNPs located on chromosomes 3 and 4 were detected in tunnel A25, one SNP on chromosome 4 was identified in tunnel B24, and two SNPs located on

Table 1 Statistic of the significant SNP associated with tomato plant responses to Belvine®

Environment	SNP	Chr	Position[a]	P-value	MAF	Effect	PVE[b]
All	SNP37241	2	740 878	4.56e-07	0.17	-3.02	7.56
	SNP77453	4	7 963 272	4.15e-08	0.30	-2.59	13.47
	SNP246258	12	45 309 963	1.77e-07	0.11	-4.09	17.06
A25	SNP55112	3	7 461 290	5.01e-07	0.37	3.48	17.48
	SNP77432	4	7 955 113	1.72e-08	0.31	-3.43	10.31
B24	SNP88909	4	31 269 345	5.97e-11	0.14	-16.48	12.86
B25	SNP41286	2	11 610 659	3.34e-07	0.18	-3.95	21.13
	SNP79715	4	11 286 270	3.59e-07	0.30	-3.46	6.97

[a]The physical map positions of the SNP markers were determined using the tomato genome assembly SL4.0
[b]The phenotypic variance explained (PVE) by each marker

chromosomes 2 and 4 were detected in tunnel B25. These SNPs individually explained 17.48%, 10.31%, 12.86%, 21.13%, and 6.97% of the phenotypic variance in their respective environments. The two SNPs detected in tunnel A25 together explained 27% of the phenotypic variance in this tunnel, while the two SNPs identified in tunnel B25 also jointly explained 27% of the variance. The SNP detected on chromosome 4 in tunnel A25 was located very close to the SNP identified using the combined phenotypic data from all tunnels. Linkage disequilibrium (LD) analysis between SNPs located on the same chromosome showed that the two SNPs identified on chromosome 2 were strongly linked ($r^2 = 0.70$) (**Table S2**). On chromosome 4, two distinct LD groups were observed. SNP77432, SNP77453, and SNP79715 were strongly linked to each other ($r^2 \geq 0.60$), whereas their LD with SNP88909 was weak ($r^2 < 0.1$) (**Table S2**). LD analysis grouped the significant SNPs into five QTL regions: one on chromosome 2, one on chromosome 3, two on chromosome 4, and one on chromosome 12.

For most of the SNPs associated to tomato response to Belvine, the favorable allele corresponded to the reference allele, identical to the one of Heinz 1706 cultivar (SL4.0 genome version) (**Fig. S7, Fig. S8**). However, SNP55112, detected on chromosome 3 in tunnel A25, showed the opposite effect (**Table 1; Fig. S8**). For environments in which several significant SNPs were detected, the effects of different genotypic combinations were further analyzed. Although SNP × SNP interactions were not statistically significant in any dataset, phenotypic distributions showed that, in tunnel B25 and in the combined analysis, accessions carrying the reference genotype at all significant SNPs displayed higher levels of protection, whereas accessions carrying the alternative genotype at all significant SNPs showed the lowest levels of protection (**Fig. S7, Fig. S8**). As expected, in tunnel A25, the highest response levels were

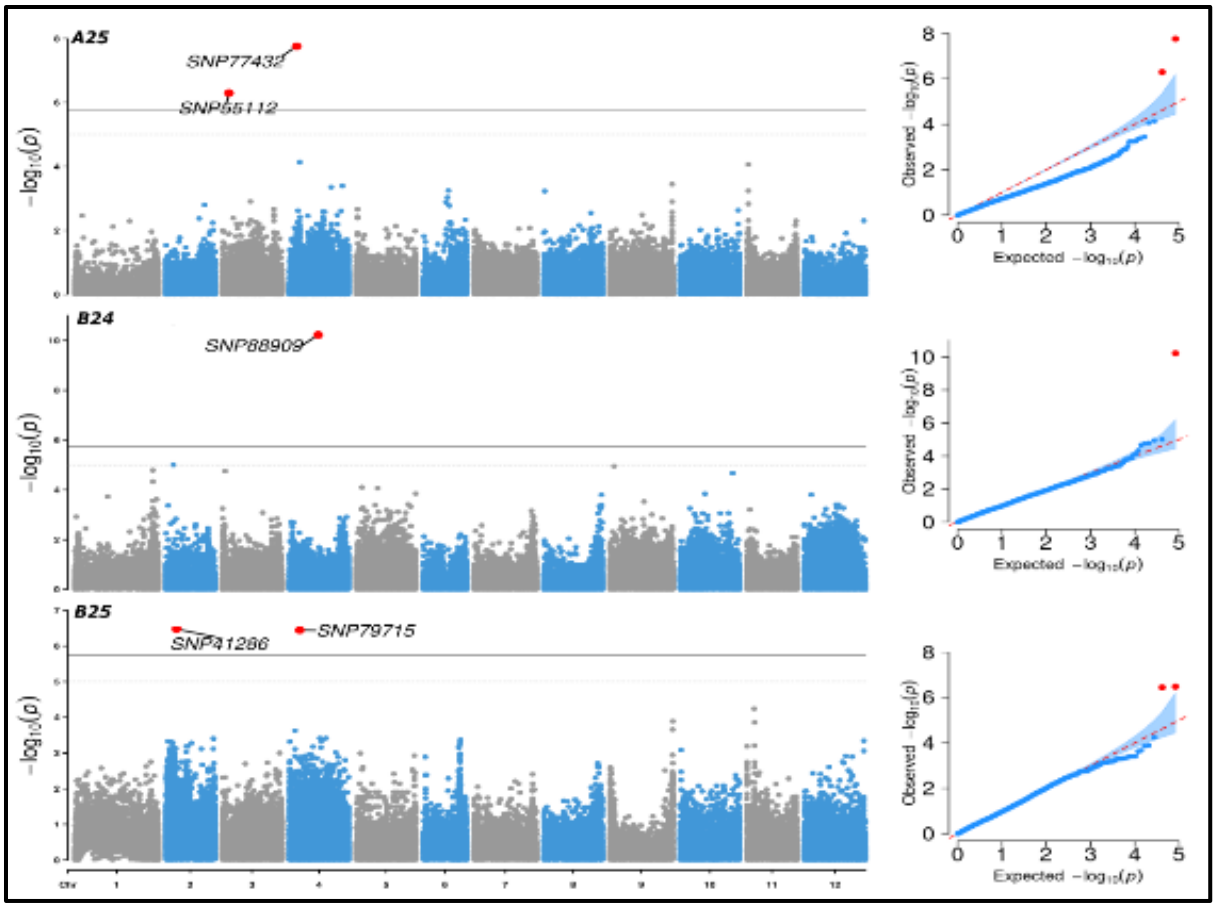


Fig. 6 Left: Manhattan plot showing significant SNP associated with plant responses to Belvine® in each tunnel A25, B24 and B25, respectively. The X-axis shows SNPs along each chromosome; the Y-axis is the -log10(p-value) for the association analysis. The grey lines indicate the Gao significance threshold (-log10(p-value) = 5.75); Red points indicate the significant SNPs with their name. Right: Corresponding quantile-quantile plots (QQplot) for plant responses. The X-axis shows expected -log10 (P), and the Y-axis shows observed -log10(p)

associated with the combination of the reference genotype at the chromosome 4 SNP and the alternative genotype at the chromosome 3 SNP. The other genotypic combinations showed relatively similar responses. Globally, the protection level was not linked to the allele origin. The estimated genotypic effects of significant SNPs across the three environments, together with their 95% confidence intervals, are presented in **Supplementary Fig. S9**. Genotype-by-environment response patterns are also illustrated in **Supplementary Fig. S10**.

Candidate genes surrounding significant SNPs

LD decay varied substantially among chromosomes, and candidate regions were defined based on chromosome-specific LD decay distances. LD decay was defined as the chromosomal distance at which r^2 decreased to half of its maximum value, resulting in intervals ranging from ~600 kb to ~1 Mb (**Table 2**).

Table 2 Candidate genes related to plant defense in the interval of the significant SNP

	SNP	Chr	Position	Regions	Number of genes [b]	Solyc number of candidate genes	Annotation
All	SNP37241	2	740 878	750 kb	21 (2)	Solyc02g044050.1.1	ABC transporter C family member 4
						Solyc02g044000.3.1	ABC transporter C family member 4
	SNP77453	4	7 963 272	650 kb	42 (4)	Solyc04g016470.4.1	β-1,3-glucanases
						Solyc04g016580.1.1	F-box protein
						Solyc04g017610.2.1	F-box protein
						Solyc04g017670.3.1	F-box protein
	SNP246258	12	45 309 963	1000 kb	42 (7)	Solyc12g036320.3.1	Receptor-like protein kinase
						Solyc12g036330.1.1	WAK-associated domain-containing protein
						Solyc12g036430.3.1	Transcription factor bHLH95-like
						Solyc12g036440.1.1	Membrane-associated kinase regulator
						Solyc12g036470.2.1	bHLH transcription factor 071
						Solyc12g036480.2.1	NAC domain
						Solyc12g036560.2.1	Glutathione S-transferase
A25	SNP55112	3	7 461 290	600 kb	45 (4)	Solyc03g044100.4.1	Peroxidase
						Solyc03g044140.3.1	Protein kinase domain
						Solyc03g044160.3.1	Kinase family protein
						Solyc03g044220.1.1	F-box protein
B24	SNP88909	4	31 269 345	650 kb	13 (1)	Solyc04g045290.1.1	Transducin/WD40 repeat-like superfamily protein
B25	SNP41286	2	11 610 659	750 kb	4 (0)	-	-
	SNP79715	4	11 286 270	650 kb	21 (4)	Solyc04g040190.1.1	lycopene beta-cyclase
						Solyc04g040130.1.1	Fatty acid desaturase
						Solyc04g040120.1.1	Fatty acid desaturase
						Solyc04g040090.4.1	Strictosidine synthase-like protein

[a]Regions = interval of the candidate regions (+/-)

[b]Values in parentheses are the number of candidate genes found in the defined regions

A total of 105 annotated genes were identified for the SNPs detected with the combined dataset (**Table 2**; **Supplementary Data 3**). In the region flanking SNP246258 on chromosome 12, 42 genes were annotated. Among these, seven were associated with plant resistance: three protein kinases or kinase regulators (Solyc12g036320.3.1, receptor-like protein kinase; Solyc12g036330.1.1, WAK-associated domain-containing protein; and Solyc12g036440.1.1,

membrane-associated kinase regulator), three transcription factors (Solyc12g036430.3.1, bHLH95-like; Solyc12g036470.2.1, bHLH071; and Solyc12g036480.2.1, NAC domain protein), and one glutathione S-transferase (Solyc12g036560.2.1). For SNP37241 on chromosome 2, 21 genes were annotated, including two genes involved in transport processes, both encoding ABC transporter C family member 4 proteins (Solyc02g044050.1.1 and Solyc02g044000.3.1).

The region flanking SNP77453 on chromosome 4 contained 42 genes, among which four stress-response genes (Solyc04g016470.4.1, Solyc04g016580.1.1, Solyc04g017610.2.1, and Solyc04g017670.3.1) were considered potential candidates. The first gene was annotated as a β-1,3-glucanase, whereas the remaining three encoded F-box proteins. Because SNP77432, detected in tunnel A25, is located very close to SNP77453, the same candidate genes were considered for both SNPs. In the second region detected in tunnel A25, surrounding SNP55112 on chromosome 3, 45 genes were annotated. Four of these were associated with resistance-related functions: one peroxidase (Solyc03g044100.4.1), two protein kinases (Solyc03g044140.3.1 and Solyc03g044160.3.1), and one F-box protein (Solyc03g044220.1.1).

For SNP88909 identified on chromosome 4 in tunnel B24, 13 genes were annotated, including one gene involved in signal transduction (Solyc04g045290.1.1). In tunnel B25, 21 genes were annotated in the region surrounding SNP79715 on chromosome 4, including several genes associated with secondary metabolite biosynthesis (Solyc04g040190.1.1, Solyc04g040130.1.1, Solyc04g040120.1.1, and Solyc04g040090.4.1).

4. Discussion

First report of QTL underlying tomato accession responses to PRI

Our results demonstrate that the efficacy of Belvine® against *P. infestans* is positive but strongly dependent on plant genotype, with a quantitative distribution of protection levels across accessions. This conclusion is further supported by estimates of broad-sense heritability for the percentage of protection, ranging from 60% to 78%, and estimates of narrow-sense heritability ranging from 46% to 63%, depending on the dataset used (A25, B24, B25, or combined data) (**Fig. 4**, **Fig. S4**). These heritability estimates for the percentage of protection are comparable with those reported for genetic resistance to late blight (Merk and Foolad 2012; Ohlson and Foolad 2015, 2016; Sullenberger and Foolad 2018). Overall, these results suggest that an important portion of the variation observed in the response of tomato to Belvine®

treatment is genetically controlled and could therefore be amenable to genetic characterization and selection.

Sharma et al. (2010) also reported that the protection conferred by another PRI, the dl-3-aminobutyric acid (BABA), against *P. infestans* varied according to tomato genotype and leaf age. However, their study was conducted on only 32 accessions belonging exclusively to the species *S. lycopersicum*, whereas our study included 148 accessions originating from several tomato species including *S. lycopersicum* var. *lycopersicum*, *S. lycopersicum* var. *cerasiforme* *S. pimpinellifolium*, thereby encompassing a much broader range of genetic diversity. The consistency of genotype-dependent responses across such a diverse panel strengthens the evidence that responsiveness to PRI is genetically determined and supports the existence of a quantitative genetic basis underlying the efficacy of PRI-induced resistance in tomato.

We detected a total of eight significant SNPs associated with tomato plant responses to the application of Belvine®, three SNPs with the overall dataset on chromosomes 2, 4 and 12, and five SNPs environment specifics, on chromosomes 2, 3, and 4. Based on pairwise linkage disequilibrium, the eight SNPs clustered into five QTL regions. According to Sim et al. (2012) and Jia et al. (2025), recombination is strongly suppressed within approximately the first 30 Mb of chromosome 2. This reduced recombination may explain the high LD observed between the two SNPs identified on chromosome 2, despite their physical separation of approximately 11 Mb. Therefore, these two SNPs were considered to represent a single QTL rather than two independents. To the best of our knowledge, this is the first time that a mapping study of loci involved in the tomato response to PRIs is reported. Our findings are consistent with previous work reporting genomic regions associated with the genotypic variability of PRI efficacy. Indeed, a few studies have identified QTL regions associated with PRI-induced resistance in other species. Soleimani et al. (2024), found 5 QTLs involved in wheat N-acyl homoserine lactone (AHL)-induced resistance against leaf rust caused by *Puccinia triticina*. Similarly, fifteen QTLs have been found to be associated with the variability of *Serendipita indica* efficacy against powdery mildew resistance in wheat (Thielmann et al. 2026). Bénéjam et al. (2021), have also detected QTLs associated with apple tree resistance following treatment with acibenzolar-S-methyl (ASM) against apple scab and fire blight. The difference with that study is that all the QTL found under PRI-treated conditions were also detected in control conditions.

In this study, we mapped loci involved in the response of tomato accessions to Belvine®, using both combined data and single-environment analyses. This approach is supported by previous studies showing that PRI efficacy is strongly influenced by environmental conditions

and genotype-by-environment interactions (Walters et al. 2011; Bruce 2014; Stridh et al. 2022), suggesting that different genes may contribute to plant responses depending on the environment. Consistent with this, we identified distinct genomic regions across our three environments. Integrating detailed environmental descriptors into future genetic analyses will improve the precision of QTL interpretation. Environment-specific loci could then be strategically used in breeding programs, particularly for deployment in regions where specific environmental conditions enhance PRI efficacy.

Previous studies have reported late blight resistance QTLs, LB-qtl2 and LB-qtl12, on chromosomes 2 and 12 in the *S. pimpinellifolium* accessions PI270442, PI 163245 and the *S. lycopersicum* breeding line Fla.8059 (Ohlson et al. 2018; Sullenberger and Foolad 2024). Although these QTLs are located on the same chromosomal arms as the loci identified in the present study, they do not colocalize, suggesting that the genes underlying genetic resistance differ from those involved in the plant response to the PRI treatment.

Candidate genes associated with tomato accessions responses to Belvine®

Within the confidence intervals of the significant SNPs detected in the combined dataset, 105 genes were annotated. Among them, glutathione S-transferase (GST) and β-1,3-glucanase were upregulated under Belvine® treatment. β-1,3-glucanases, members of the PR2 family, contribute to antimicrobial defense through pathogen cell-wall degradation and callose mobilization (Balasubramanian et al. 2012; Perrot et al. 2022). In tomato, the co-expression of β-1,3-glucanase with other defense enzymes (peroxidase, chitinase, polyphenol oxidase, phenylalanine ammonia-lyase) enhances resistance to *Fusarium oxysporum f. sp. lycopersici* and *Alternaria solani* (Mohamed et al. 2007; Salim et al. 2011). Although the specific genes differ from those induced by Belvine®, they belong to the same functional family, suggesting their potential involvement of these gene families in PRI responsiveness. However, further gene expression analyses and functional validation are required to confirm their roles.

Other notable candidates included protein kinases (receptor-like kinases), ABC transporters, and transcription factors such as bHLH95-like, bHLH071, and NAC (NAM, ATAF, and CUC) domain proteins. These families are widely implicated in plant immunity. For example, *SlLRR-RLK94* enhances tomato resistance to *Phytophthora infestans* (Zhu et al., 2025), while in *Nicotiana benthamiana*, ABC transporters *NbABCG1* and *NbABCG2* limit *P. infestans* penetration (Shibata et al. 2016; Rin et al. 2017). It has been reported that NAC and bHLH transcription factors regulate SA-, JA-, ET- and ABA-dependent defense pathways

(Nuruzzaman et al. 2013; Chen et al. 2023; Yan et al. 2024; Xiong et al. 2025). In tomato, the genes SlSRN1 and SlNAC1 positively modulate resistance to *Botrytis cinerea* and *Pseudomonas syringae* pv. *tomato* (Huang et al. 2013; Liu et al. 2014). In rice, bHLH25 contributes to cell-wall reinforcement by repressing miR397b and activating lignin biosynthesis after infection (Liao et al. 2025).

Environment-specific candidate genes were also identified. In tunnel A25, a peroxidase gene co-localized with a significant SNP on chromosome 3. That gene is particularly relevant given that a related gene was upregulated under Belvine®. In tunnel B24, genes involved in signal transduction were detected on chromosome 4, while in tunnel B25, genes associated with alkaloid and terpenoid biosynthesis, as well as fatty-acid desaturases, were identified. These functions are consistent with previous studies showing the accumulation of alkaloids, terpenoids, and lipid-derived defenses in resistant plants or PRI-treated plants during interactions with *P. infestans* (Yogendra et al., 2017; Galeano Garcia et al., 2018; Zhao et al., 2023; Agho et al., 2023; Tsypurskaya et al., 2022).

Together, these candidate genes highlight the diversity of molecular pathways potentially involved in Belvine® responsiveness. Further studies using RNA-seq analysis will enable evaluation of expression variation across a broader set of genes.

5. Conclusion

This study is the first report of chromosomal regions and candidate genes associated with tomato responses to PRI applications. We identified eight significant SNPs associated with tomato accession responses to the application of Belvine®, located on chromosomes 2, 3, 4, and 12, with some SNPs being specific to environmental conditions. The strong linkage disequilibrium observed between some SNPs suggests the presence of five potential QTLs: one on chromosome 2, one on chromosome 3, two on chromosome 4, and one on chromosome 12. The strongest QTLs were identified on chromosome 12 for the combined dataset, and on chromosomes 2 and 3 in tunnels B25 and A25, respectively. Some of these genes share the same function as genes up-regulated under Belvine® treatment in gene expression analysis, notably β-1,3-glucanases (PR-2), glutathione S-transferase, and peroxidase. Although their involvement in PRI responsiveness remains to be validated, the identified QTL regions and candidate genes provide a valuable genetic resource for further investigating the genetic basis of tomato responses to PRIs and may contribute to future breeding efforts.

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SUPPORTING INFORMATION

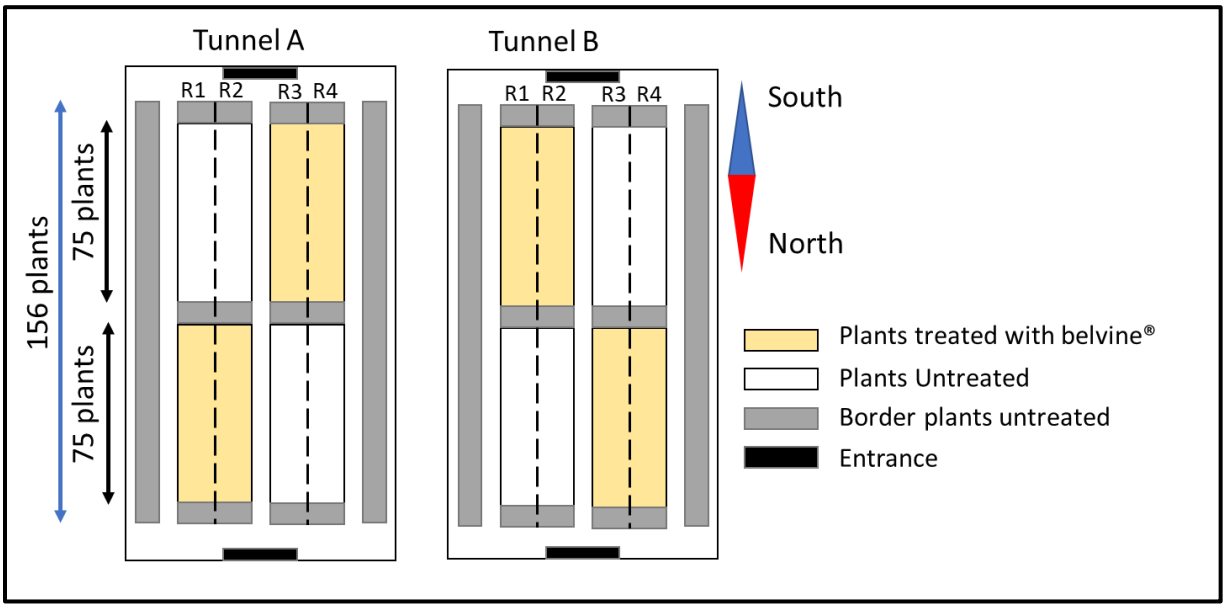


Fig. S1 Experimental design in trial 4. The design presented corresponds to the layout used in 2024. In 2025, the opposite layout was implemented. Each tunnel contained six rows: two single rows used as borders, and two double rows. Each double row was divided into two blocks corresponding to two modalities: one treated with Belvine®, and one untreated control. The two modalities were separated by two untreated Rose de Berne plants per row. Each block contained 150 tomato accessions, randomly assigned within its double row. The experiment was arranged so that each modality faced its opposite

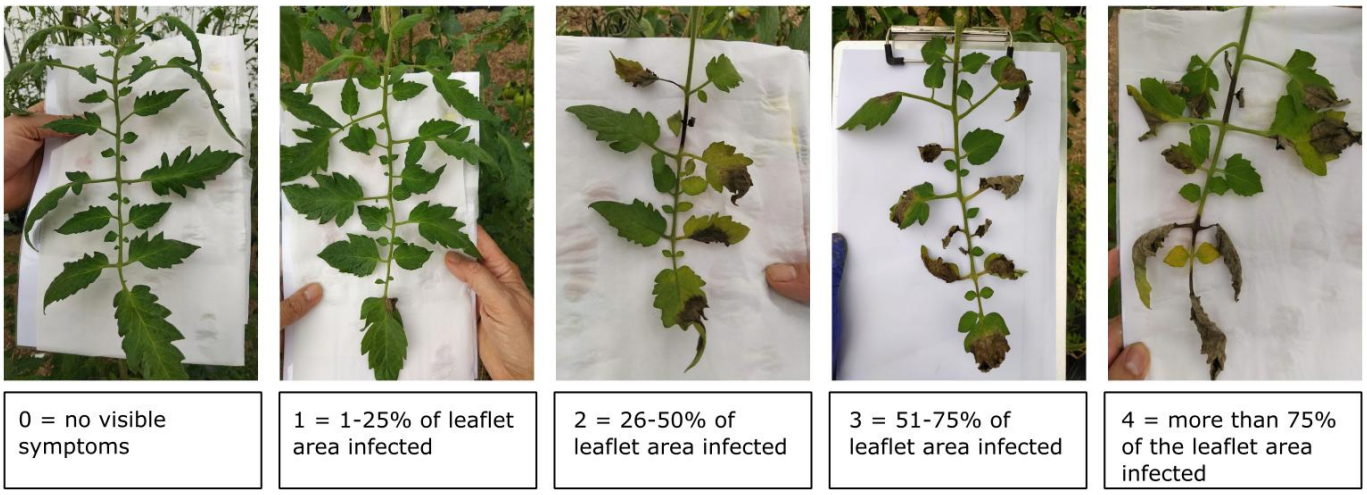


Fig. S2 Scale of disease severity on infected leaflets. Disease severity on infected leaflets was assessed using a 0-4 ordinal scale, where 0 = no visible symptoms; 1 = 1-25% of leaflet area infected; 2 = 26-50% of leaflet area infected; 3 = 51-75% of leaflet area infected; and 4 = more than 75% of the leaflet area infected

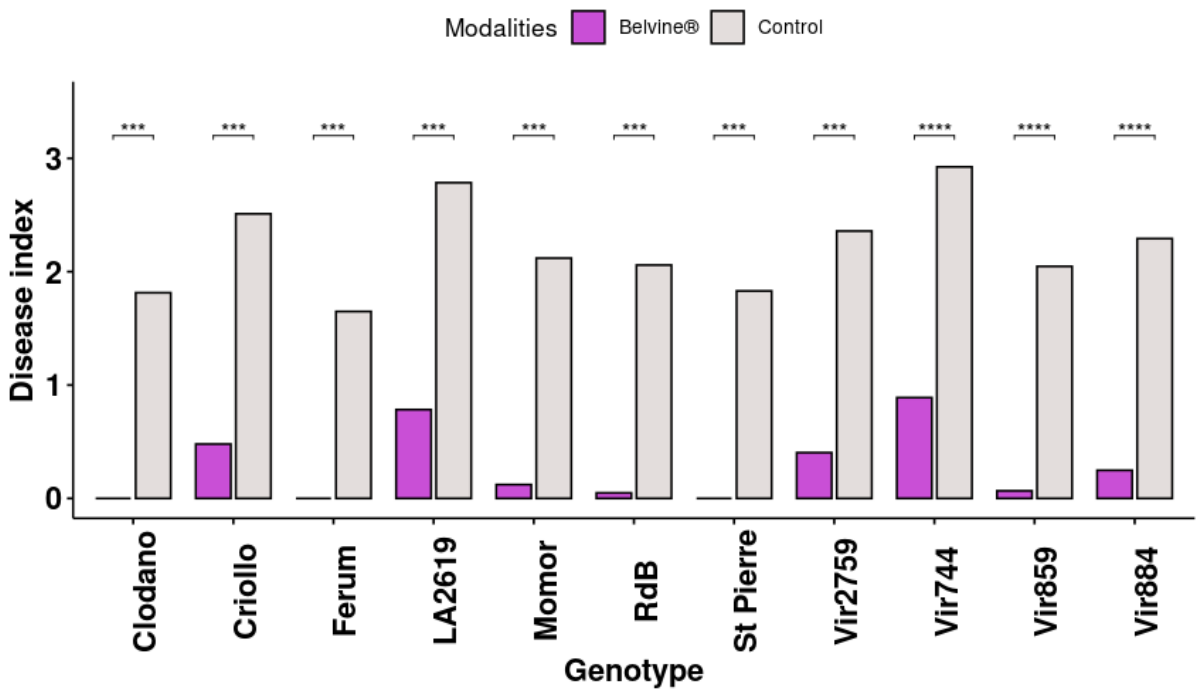


Fig. S3 Genotypic variation of Belvine® efficacy. Disease index of late blight calculated from four biological replicates for eleven tomato genotypes treated with Belvine® (purple), or untreated as controls (grey). Asterisks (*) indicate significant differences between treated and control modalities based on ANOVA.

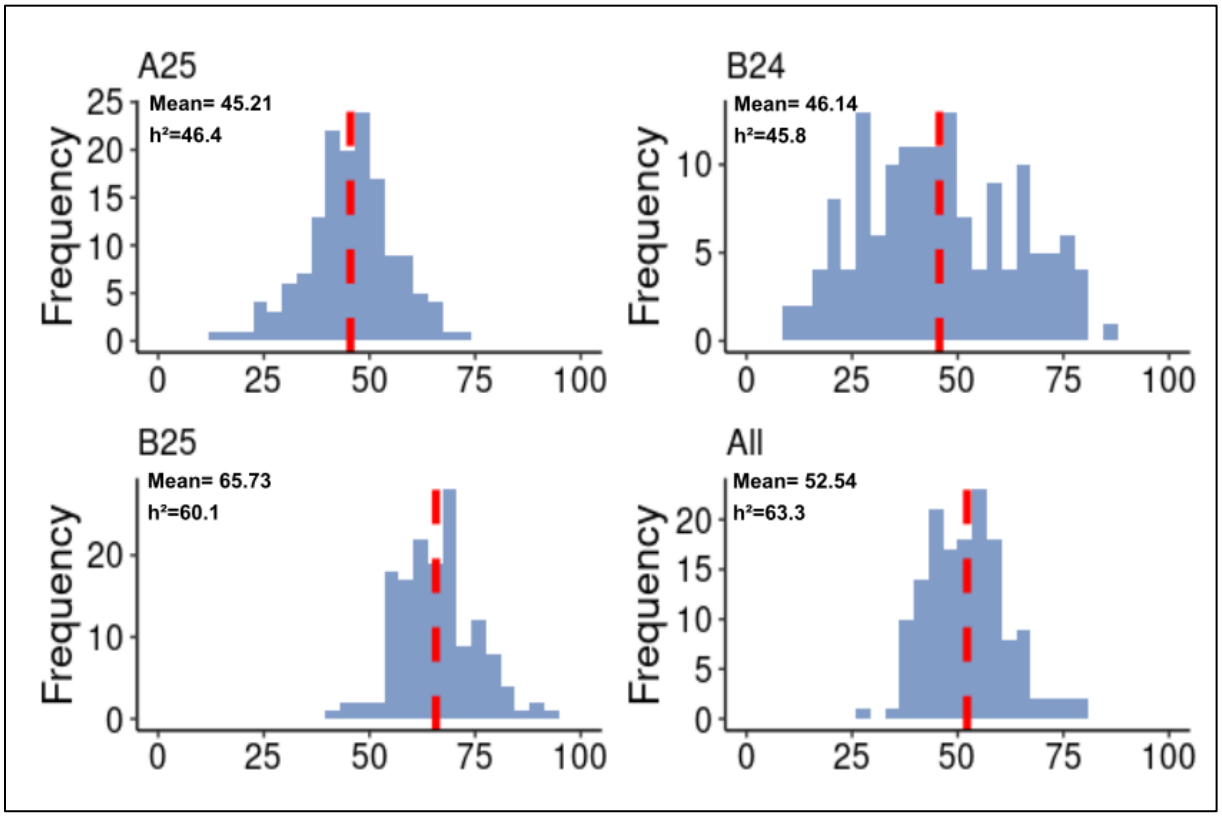


Fig. S4 Phenotypic distribution of the percentage of protection (PP) of 148 tomato accessions for each tunnel and the combined dataset (All). Combined PP represents the average of the three tunnels. PP = BLUP + mean. Blups are from spatial correction with statgenHTP Rpackage. Vertical lines in red indicate the average (mean) of PP. h^2 is the GBLUP calculated genomic heritability

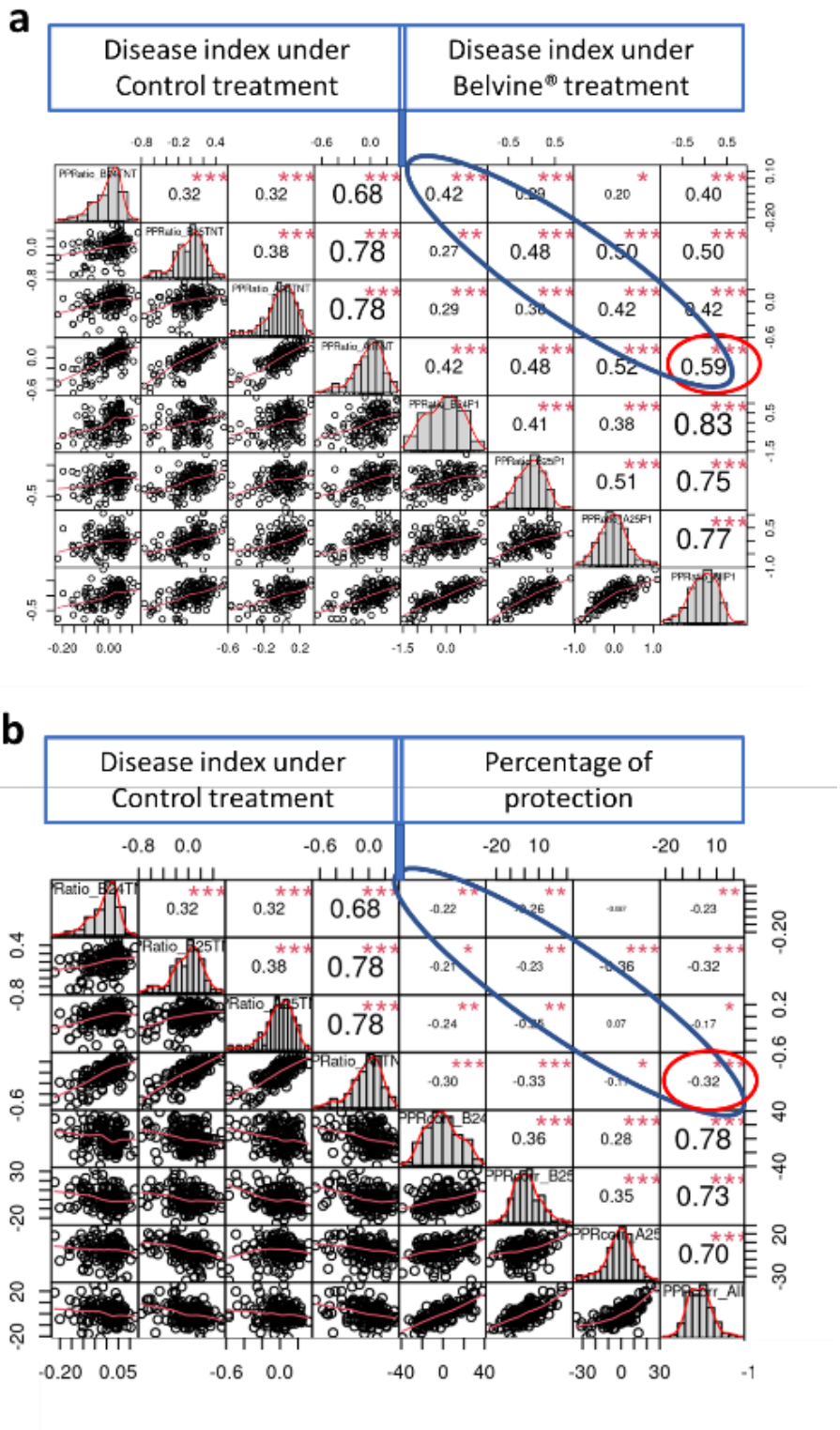


Fig. S5 Pairwise data correlation. a) between the disease index under control and Belvine® treatments. b) between the disease index under control treatment and the percentage of protection. Blue ellipses show the pairwise correlation coefficients within the tunnels, red ellipses emphases correlation coefficient with the combined dataset. (*) denotes significance at a P-value of 0.05, (**) denotes significance at a P-value of 0.01, (***) denotes significance at a P-value of 0.001. no Asterix indicated the correlation coefficient is not statistically significant

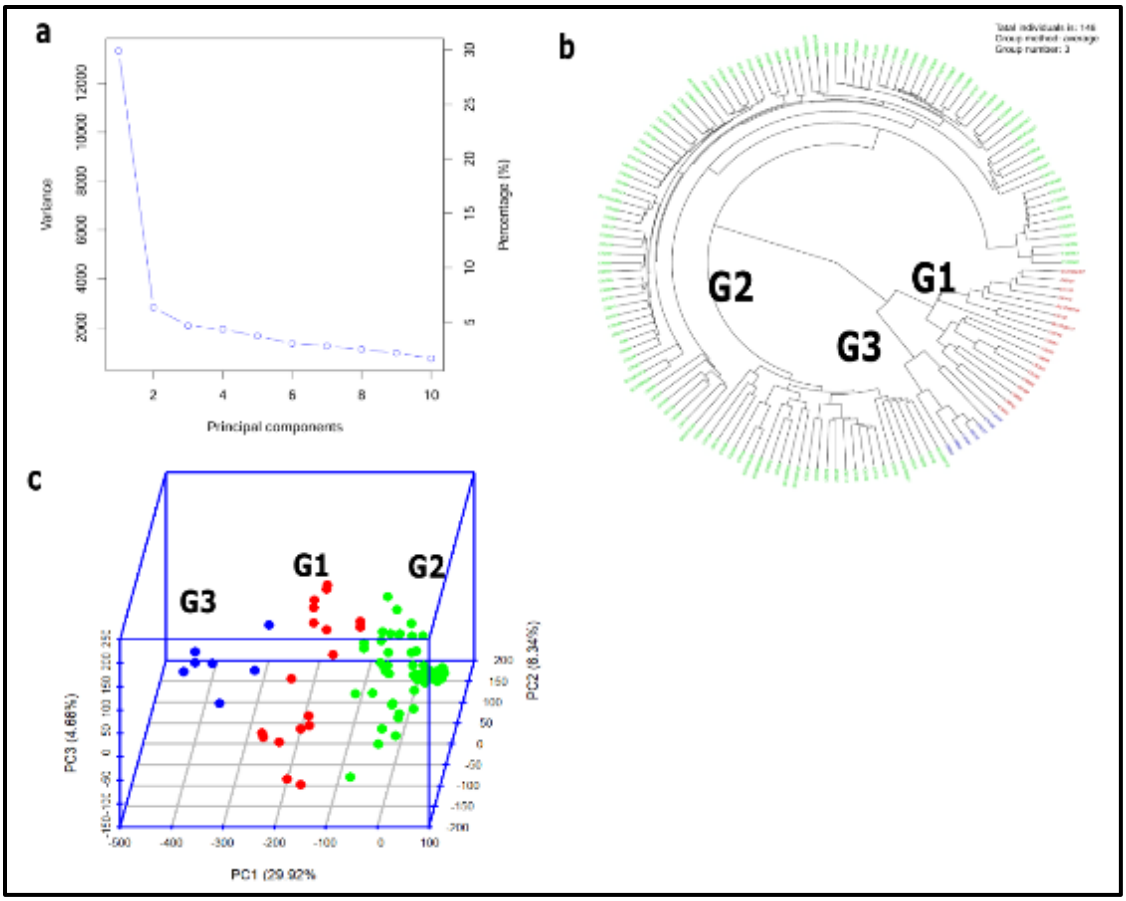


Fig. S6 Population genetic diversity analysis of the 148 tomato accessions. **a)** PCA eigenvalue plot generated using GAPIT 3, showing that the top three principal components (PCA = 3) explain majority of the variation among the 148 individuals; **b)** Phylogenetic tree constructed using the neighbor-joining (NJ) method, showing three subpopulations; and **c)** 3D graphical plot of principal component analysis (PCA), illustrating the three sub-populations

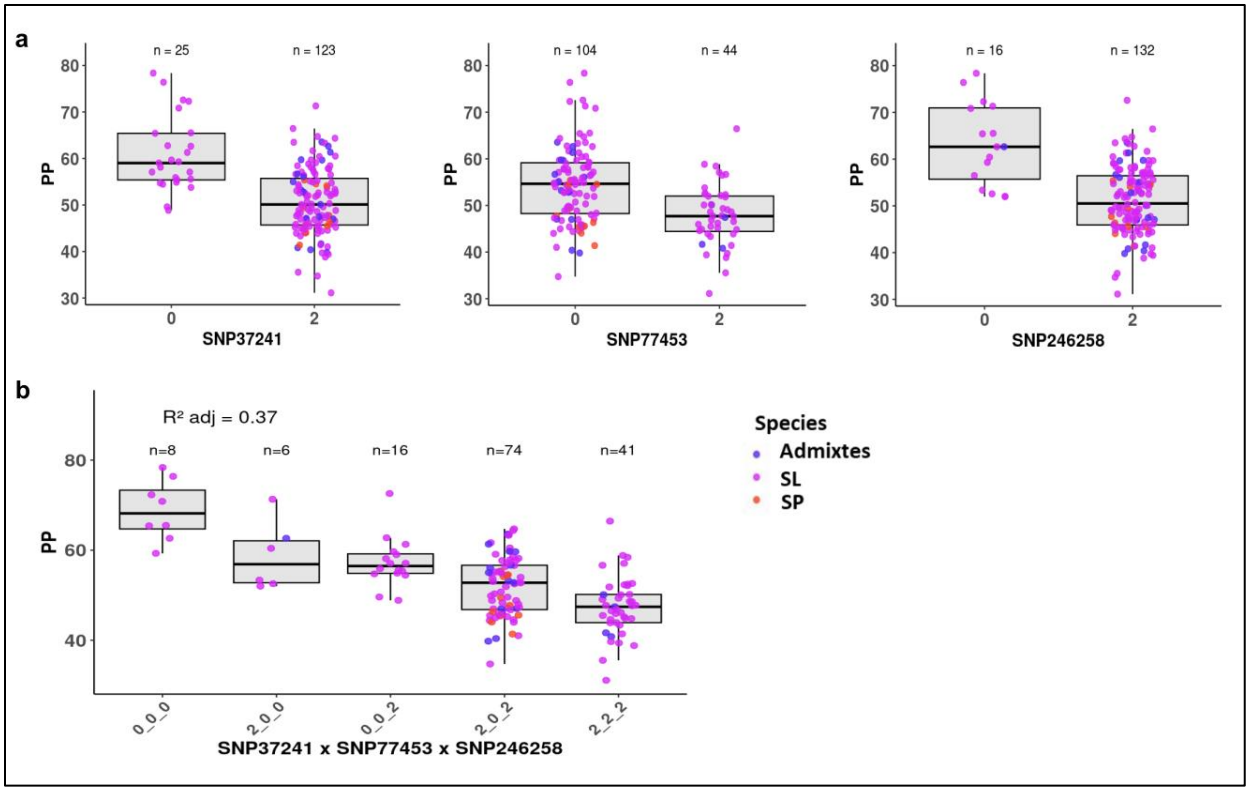


Fig. S7 Phenotypic differences of plant responses among the significant SNPs detected. a) phenotypic differences between the genotypes of the significant SNPs found with combined dataset. b) interaction plot of the three SNPs found with the combination of the data of the three tunnels. Difference point colors represent the species of the plant: SP for *Solanum pimpinellifolium* (red), SL for *Solanum lycopersicum*, including cerasiforme and large-fruited tomato (pink), and admixture for the admixtes of *Solanum lycopersicum* var. *cerasiforme* and *Solanum pimpinellifolium.* The text above the boxplot indicates the number of individuals in the genotypic class

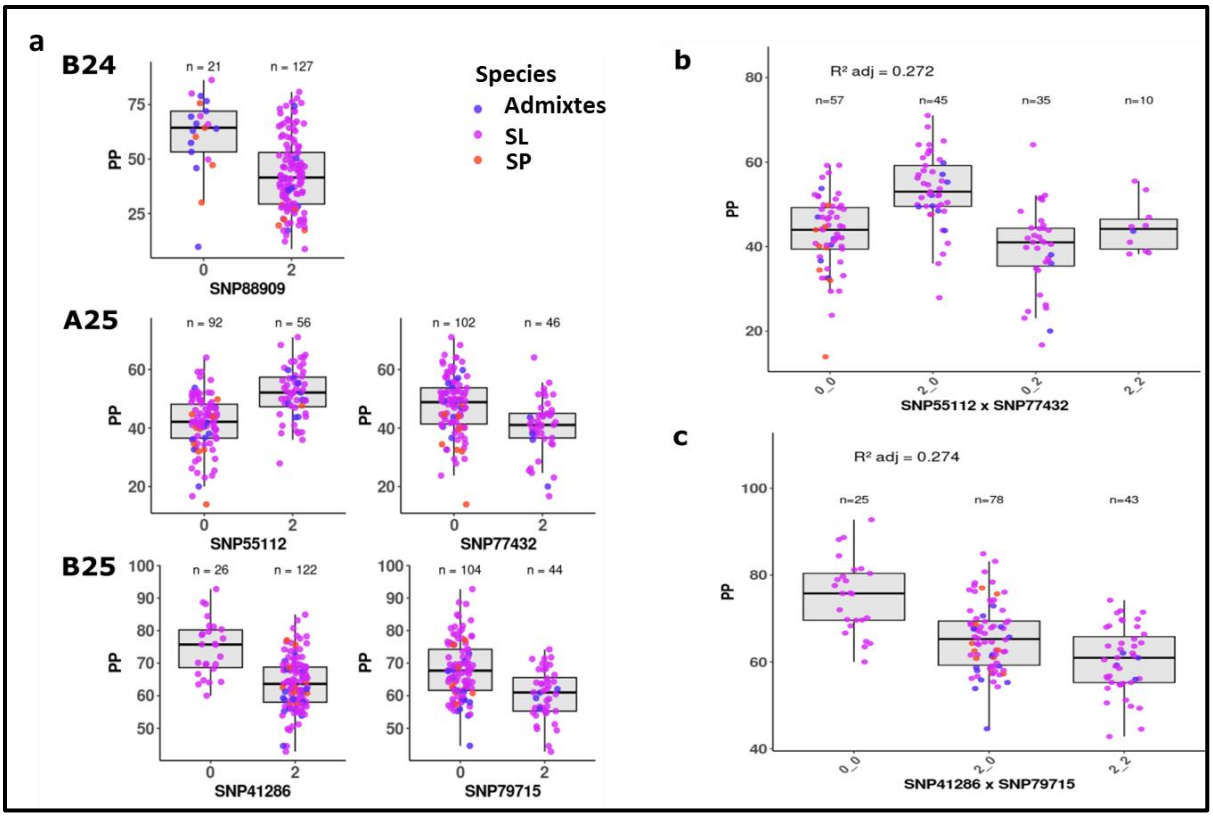


Fig. S8 Phenotypic differences of plant responses among the significant SNP detected. a) phenotypic differences between the genotypes of the significant SNPs found in tunnels B24, A25 and B25. b) interaction plot of the two SNPs found in tunnel A25. c) interaction plot of the three SNPs found in tunnel B25; The text above the boxplot indicates the number of individuals in the genotypic class. Difference point colors represent the species of the plant: SP for *Solanum pimpinellifolium* (red), SL for *Solanum lycopersicum*, including cerasiforme and large-fruited tomato (pink), and mixture for the admixtures of SLC and SP

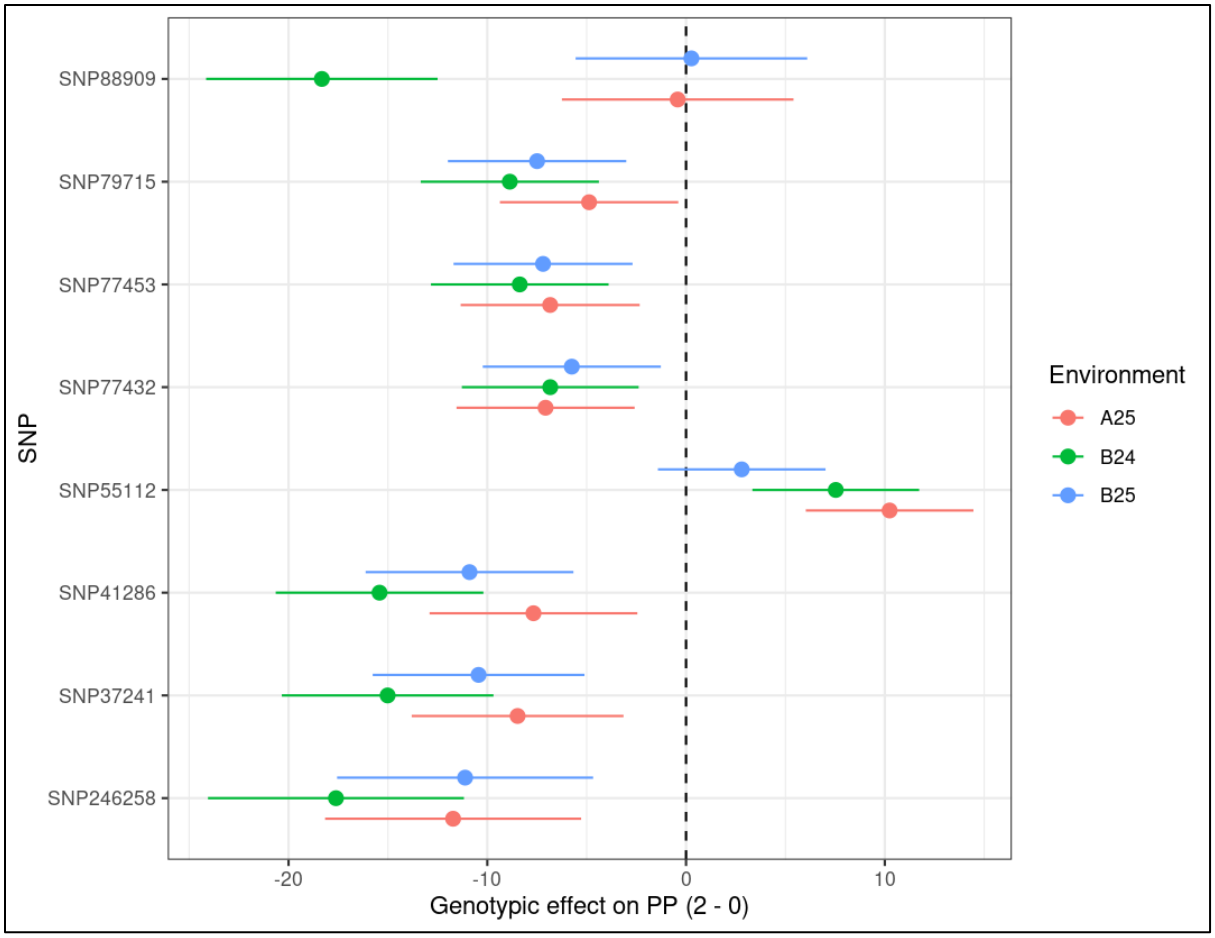


Fig. S9 Genotypic effects of SNP markers on the percentage of protection (PP) conferred by Belvine® against tomato late blight across three environments. Points represent the estimated genotypic effects, calculated as the difference in PP between accessions carrying genotype 2 and those carrying genotype 0 (2 − 0). Horizontal bars indicate 95% confidence intervals estimated using linear mixed-effects models. Colors represent the three experimental environments: A25, B24, and B25. The vertical dashed line indicates a null genotypic effect. Positive and negative values indicate higher PP in accessions carrying genotype 2 and genotype 0, respectively.

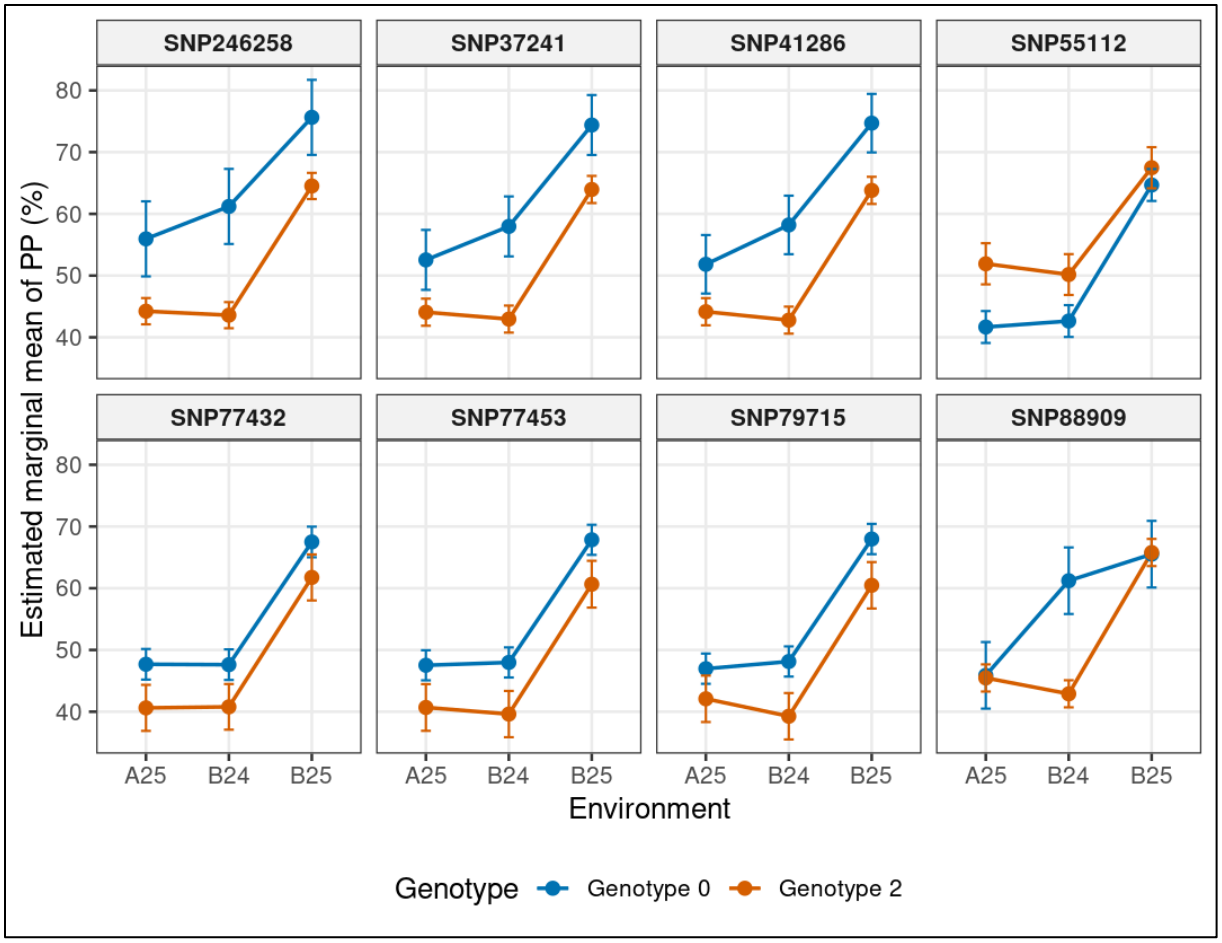


Fig. S10 Genotype-by-environment interactions for SNP markers associated with the percentage of protection (PP) conferred by Belvine® against tomato late blight. Points represent the estimated marginal means of PP for accessions carrying genotype 0 (blue) or genotype 2 (orange), obtained from linear mixed-effects models. Error bars indicate 95% confidence intervals. The three experimental environments are A25, B24, and B25. Lines connect the estimated marginal means of each genotypic class across environments to illustrate variation in genotypic responses

Table S1 List of tomato defense genes targeted for gene expression analysis

Gene	Name	Pathway or functions
PR-1	Pathogenesis-related protein 1	PR-protein
PR-2	Pathogenesis-related protein 2 (glucanases)	PR-protein
PR-4	Pathogenesis-related protein 4 (hevein-like)	PR-protein
PR-5	Pathogenesis-related protein 5 (thaumatin-like, osmotin)	PR-protein
PR-8	Pathogenesis-related protein 8 (class III chitinase)	PR-protein
PR-14	Pathogenesis-related protein 14 (lipid transfer protein)	PR-protein
PR-15	Pathogenesis-related protein 15 (oxalate oxidase)	PR-protein
PAL	Phenylalanine ammonia-lyase	Phenylpropanoid pathway
CHS	Chalcone synthase	Phenylpropanoid pathway
DFR	Dihydroflavonol reductase	Phenylpropanoid pathway
ANS	Anthocyanidin synthase	Phenylpropanoid pathway
PPO	Polyphenol oxidase	Phenylpropanoid pathway
HMGR	Hydroxymethyl glutarate-CoA reductase	Isoprenoid pathway
FPPS	Farnesyl pyrophosphate synthase	Isoprenoid pathway
Far	(E,E)-alpha-farnesene synthase	Isoprenoid pathway
CSL	Cystéine sulfoxide lyase	Cysteine sulfoxid pathway
APOX	Ascorbate peroxidase	Oxidative stress
GST	Glutathion S-transférase	Oxidative stress
POX	Peroxidase	Oxidative stress
CalS	Callose synthase	Cell wall modification
Pect	Pectin methyl esterase	Cell wall modification
CAD	Cinnamyl alcool dehydrogenase	Cell wall modification
EDS1	Disease resistance protein EDS1	Salicylic acid pathway
WRKY	WRKY transcription factor 30	Salicylic acid pathway
LOX2	Lipoxygenase AtLOX2	Jasmonic acid pathway
JAR	Jasmonate resistant 1	Jasmonic acid pathway
ACCO	1-aminocyclopropene-1-carboxylate oxidase	Ethylene pathway
EIN3	EIN3-BINDING F-BOX PROTEIN 1	Ethylene pathway
TuA	Tubulin alpha-1 chain	Reference gene
Actin	Actin 7	Reference gene
GAPDH	Glyceraldehyde-3-phosphate dehydrogenase	Reference gene

Table S2 Linkage disequilibrium between SNPs detected on chromosomes 2 and 4

CHR SNP1	Position SNP1	SNP1	CHR SNP2	Position SNP2	SNP2	R^2
2	740878	SNP37241	2	11610659	SNP41286	0.696259
4	7955113	SNP77432	4	7963272	SNP77453	0.818462
4	7955113	SNP77432	4	11286270	SNP79715	0.603616
4	7963272	SNP77453	4	11286270	SNP79715	0.598459
4	11286270	SNP79715	4	31269345	SNP88909	0.0699576

Chapter 3

GWAS tomato responses to PRI – *Oidium neolycopersici*

This chapter is presented as an original article

In this chapter, we focus on identifying QTLs associated with tomato accessions responses to the application of a commercial PRI "Planticare®" against powdery mildew caused by *O. neolycopersici*.

Deciphering the genetic architecture of tomato accession responses to a plant resistance inducer against *Oidium neolycopersici*

Abstract

Plant resistance inducers (PRIs) enable plants to respond more rapidly and more strongly to biotic stresses; however, their efficacy can vary depending on plant genotype. Only a few studies have investigated the genetic basis underlying this genotypic variation in PRI efficacy, particularly in tomato. Our objective was to identify the genetic architecture governing tomato responses to PRI application. A diverse panel of 148 tomato accessions grown in three greenhouses was either treated with Planticare® or left untreated, then inoculated with *Oidium neolycopersici*. A genome-wide association study was conducted for disease severity under untreated control conditions, under PRI treatment, and for the percentage of protection conferred by the PRI.

We identified five QTLs associated with variation in Planticare® efficacy on chromosomes 4, 7, and 8. These QTL regions contained genes involved in the biosynthesis or modification of secondary metabolites (cytochrome P450 monooxygenases, 4-coumarate-CoA ligase-like proteins, glycosyltransferases), as well as defense-related genes such as antifungal protein ginkbilobin-like, peroxidase, and β-1,3-glucanase. Defense-related gene expression analysis showed that PR proteins (PR-1, PR-2, PR-4, PR-5, PR-8), peroxidase, and the ethylene-associated gene ACCO were upregulated in Planticare®-treated plants. In addition, four QTLs were identified for disease severity under Planticare® treatment on chromosomes 2, 4, and 8, and seven QTLs were detected under untreated control conditions on chromosomes 1, 4, 5, 8, 9, and 12. These QTL regions contained several disease-resistance genes, including receptor-like protein kinases and NBS-LRR proteins.

Overall, the QTL regions and candidate genes identified in this study provide a foundation for the targeted exploitation of PRI-responsive loci in breeding programs aimed at enhancing durable resistance to powdery mildew.

Keywords: Plant resistance inducers, tomato, genotype-dependent, genomic regions, candidate genes, *Oidium neolycopersici*

1. Introduction

Plants are constantly exposed to a wide range of pathogens-including fungi, bacteria, viruses, and oomycetes, that differ in lifestyle, infection strategies, and target tissues (Wang et al. 2022). To cope with these threats, they rely on an innate immune system composed of both constitutive and inducible defenses. Constitutive barriers such as the cuticle, cell wall, and preformed antimicrobial compounds provide the first line of protection by restricting pathogen entry and early colonization. When pathogens overcome these barriers, plants activate inducible immune responses. Recognition of conserved microbial features (PAMPs) by cell-surface pattern-recognition receptors (PRRs) triggers pattern-triggered immunity (PTI) (Mukhtar et al. 2011; Gupta et al. 2015). Alternatively, specific pathogen effectors can be detected by intracellular immune receptors, mainly nucleotide-binding leucine-rich repeat (NLR) proteins encoded by resistance genes, leading to effector-triggered immunity (ETI) (Meng et al. 2019; Mengiste and Liao 2025). These immune responses rely on complex signaling networks involving reactive oxygen species (ROS) production, transcriptional reprogramming of defense-related genes, and hormone-mediated regulation, notably through salicylic acid, jasmonic acid, and ethylene pathways (Waszczak et al. 2018; Dodds et al. 2024).

Beyond local responses, plants can develop systemic immunity, enabling faster and stronger defense activation upon subsequent pathogen encounters and thereby enhancing long-term protection. Plant defense can also be induced following exposure to beneficial microorganisms or defense-inducing compounds, collectively known as plant resistance inducers (PRIs). PRIs allow plants to activate more rapid and robust defense responses upon pathogen challenge (Tang et al. 2017; Hilker and Schmülling 2019; Khan et al. 2020; Hönig et al. 2023; Zapletalová et al. 2023; Tanaka et al. 2024; Zhang et al. 2026). This phenomenon, referred to as induced resistance, has gained considerable attention as a sustainable crop protection strategy because it enhances resistance while minimizing the fitness costs associated with constitutive defense activation (Martinez-Medina et al. 2016; Tanaka et al. 2024).

Over the past three decades, various PRIs have been extensively studied, with research primarily focusing on their modes of action, efficacy, and factors affecting their effects (Yuan et al. 2019; Stridh et al. 2022; Zapletalová et al. 2023; Sorokan et al. 2024; Rossi et al. 2025). It has been shown that the efficacy of these elicitors, as well as their modes of action, strongly depend on plant genotype (Walters et al. 2011, 2013; Satková et al. 2016). For example, Margaritopoulou et al. (2024) demonstrated that application of a *Reynoutria sachalinensis*

extract on courgette enhances phospholipid production and signalling in a genotype susceptible to *Podosphaera xanthii*, whereas the response was limited in an intermediate resistance genotype. A genotype-dependent effect of PRIs has also been demonstrated in carrot for resistance to *Alternaria dauci* by Le Clerc et al. (2025).

Genotypic variation in the efficacy PRIs has been reported for several compounds and across multiple crop species, including tomato, indicating that PRI responsiveness is strongly shaped by plant genetics (Sharma 2010; Sharma et al. 2010, 2012; Tucci et al. 2011; Wehner et al. 2019). However, the genetic bases underlying this variation remain largely unexplored. Only a few genome-wide association studies in wheat, apple, and *Arabidopsis thaliana* have identified QTLs associated with genotype-specific PRI responses (Ahmad et al. 2011; Bénéjam et al. 2021; Soleimani et al. 2024; Thielmann et al. 2026). These pioneering studies demonstrate that PRI responsiveness is heritable, but they also highlight how limited our understanding remains, as the small number of genetic investigations and the QTLs identified so far provide only partial insight into the underlying mechanisms. This gap underscores the need to expand PRI-related genetic mapping to additional species such as tomato, a major crop with rich genetic diversity, where such work can reveal new genomic determinants of PRI efficacy or reinforce previous results and open avenues for breeding PRI-responsive varieties.

The tomato-*Oidium neolycopersici* pathosystem represents a highly relevant model for exploring the genomic determinants underlying tomato accession responses to PRIs. Tomato is the second most widely produced vegetable globally, with an estimated 188.5 million metric tons harvested in 2024 (FAO 2025). *Oidium neolycopersici* is a destructive obligate biotrophic fungus responsible for powdery mildew on more than 60 host species across 13 plant families, particularly and can cause yield losses exceeding 50% (Whipps et al. 1998; Jones et al. 2001; Qinggui Lian et al. 2022; Park et al. 2022). This combination of economic relevance, host diversity, severe disease impact, and high-quality genome assemblies makes the tomato-*O. neolycopersici* interaction an ideal system for uncovering the genetic architecture of PRI responsiveness.

In this work, we aimed to identify the genetic architecture underlying tomato accession responses to a plant resistance inducer (PRI) against *O. neolycopersici*. To this end, we first evaluated the mode of action of six PRIs across five tomato accessions. Based on these results, three PRIs were selected to assess, in a panel of twelve genotypes, the influence of plant genetic background on PRI efficacy. Finally, one PRI was chosen for a genome-wide association study (GWAS) using a panel of 148 tomato accessions that were either treated with the PRI or left

untreated and subsequently inoculated with *O. neolycopersici* under non-heated greenhouse conditions.

2. Material and methods

Plant material, pathogen strains and plant resistance inducers

Plant material

The panel of tomato accessions used to analyze the genetic response of tomato accessions to the application of plant resistance inducers was composed of 150 homozygous lines, including 116 cherry tomato accessions belonging to *Solanum lycopersicum* var. *cerasiforme* (SLC), 10 *S. pimpinellifolium* (SP), 18 admixtes of SLC and SP, 5 large-fruited tomato *Solanum lycopersicum* var. *lycopersicum* (SLL), and one commercial cultivar, Clodano (Syngenta). Most of them have been used and characterized in other studies (Albert et al. 2016; Bineau et al. 2021; Héreil et al. 2024). The list of accessions is provided in **supplementary data 1**. Clodano seeds are from the company Syngenta, while the seeds from others accession were produced by the Centre for Vegetable Germplasm (CRB-Leg) of the GAFL research unit (INRAE, Avignon, France).

Seeds were sown in pots containing seed-starting potting soil (K substrates Easy Growing) and grown under controlled greenhouse conditions. After germination, seedlings were transferred into 9 x 9 cm pots soil substrate TS 3 MB Argile (K substrates).

Pathogen material

A population of *Oidium neolycopersici* originated from tomato (2021, France) was maintained in a growth chamber on the cultivar 'Monalbo' (Avignon, France), under controlled conditions: 24°C during the day, 20°C at night, 75% relative humidity, and a 16-hour photoperiod. The fungus was transferred onto fresh tomato plants every two weeks to produce inoculum. The inoculum was prepared by washing conidial spores from freshly sporulating leaves of heavily infected ‘Monalbo’ plants in tap water and was used immediately.

Experimentations

The study comprised three trials. Trial 1 focused on assessing tomato defense-genes induced by the PRI. Trial 2 evaluated the genotype-dependent variability in PRI efficacies against *Oidium neolycopersici*. Trial 3 were designed to map loci involved in plant response to the PRI.

Table 1 List of plant resistance inducers used in this study and their dose of application	
PRI code	**Application dose**
Planticare®	2%
Messager®	5 mL/L
Tagatose®	8.3 g/L
P11	130 mg/L
P3	0.30%
P6	6.6mL/L
Water	-

Assessing tomato defense genes induced by PRIs

To analyze the expression of tomato defense genes induced by the PRIs, two independent experiments were conducted in 2023, with a two-week interval between each experiment. The experiments were conducted with the five tomato accessions Clodano, Criollo, Marpha n2, Ecu284, and Vir884. Each accession × treatment combination included three biological replicates. Plants were assigned to eight treatment groups: 7 PRIs group (**Table 1**) and untreated control group. Four-week-old plants assigned to be treated were sprayed with approximately 20 mL of PRI solutions per plant. The treatments were applied twice, at 7 days and 3 days before sampling (Day 0). Following the first treatment (Day -7), a ring was placed near the apical meristem to mark leaf position. On day 0, for each accession, one leaf located below the ring was collected from each of the three replicates. Five leaf discs (10 mm in diameter) were excised from each leaf and pooled to form a single biological sample per accession and treatment for gene expression analysis. Samples were immediately frozen in liquid nitrogen to prevent RNA degradation.

Total RNA was extracted using the Macherey-Nagel Nucleospin RNA Plant Kit with slight modifications to the volume and reverse transcription was performed as described by Promega Corp. (M-MLV). Quantitative real-time PCR was performed as described in Gaucher et al., (2022), using a patented set of primers targeting 28 tomato defense genes and 3 reference genes (Brisset and Dugé de Bernonville, 2011), listed in **Table 2**. Relative changes in defense gene expression ($\log_2$ ratios) were calculated using the $2^{-\Delta\Delta CT}$ method (Schmittgen and Livak 2008) with normalization based on the three reference genes (Vandesompele et al. 2002). An untreated sample of the tomato variety ‘Clodano’ was used as the calibrator for the calculation of $\log_2$

expression ratios. Genes were considered differentially expressed when the absolute log_2 fold change exceeded 1 and the p-value was ≤ 0.05.

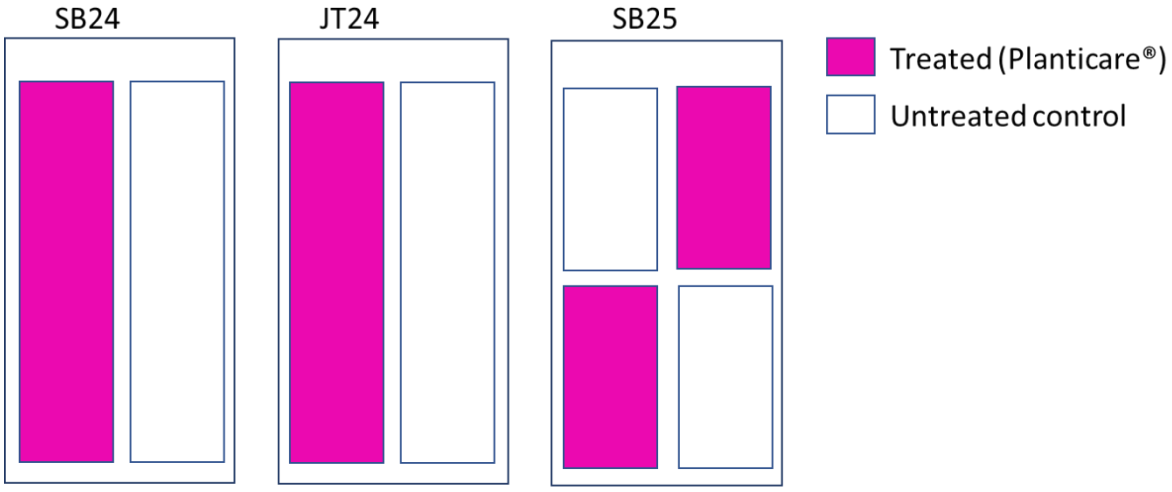
Table 2 List of tomato defense genes targeted for gene expression analysis

Genes	Names	Pathway or functions
PR-1	Pathogenesis-related protein 1	PR-protein
PR-2	Pathogenesis-related protein 2 (glucanases)	PR-protein
PR-4	Pathogenesis-related protein 4 (hevein-like)	PR-protein
PR-5	Pathogenesis-related protein 5 (thaumatin-like, osmotin)	PR-protein
PR-8	Pathogenesis-related protein 8 (class III chitinase)	PR-protein
PR-14	Pathogenesis-related protein 14 (lipid transfer protein)	PR-protein
PR-15	Pathogenesis-related protein 15 (oxalate oxidase)	PR-protein
PAL	Phenylalanine ammonia-lyase	Phenylpropanoid pathway
CHS	Chalcone synthase	Phenylpropanoid pathway
DFR	Dihydroflavonol reductase	Phenylpropanoid pathway
ANS	Anthocyanidin synthase	Phenylpropanoid pathway
PPO	Polyphenol oxidase	Phenylpropanoid pathway
HMGR	Hydroxymethyl glutarate-CoA reductase	Isoprenoid pathway
FPPS	Farnesyl pyrophosphate synthase	Isoprenoid pathway
Far	(E,E)-alpha-farnesene synthase	Isoprenoid pathway
CSL	Cystéine sulfoxide lyase	Cysteine sulfoxid pathway
APOX	Ascorbate peroxidase	Oxidative stress
GST	Glutathion S-transférase	Oxidative stress
POX	Peroxidase	Oxidative stress
CalS	Callose synthase	Cell wall modification
Pect	Pectin methyl esterase	Cell wall modification
CAD	Cinnamyl alcool dehydrogenase	Cell wall modification
EDS1	Disease resistance protein EDS1	Salicylic acid pathway
WRKY	WRKY transcription factor 30	Salicylic acid pathway
LOX2	Lipoxygenase AtLOX2	Jasmonic acid pathway
JAR	Jasmonate resistant 1	Jasmonic acid pathway
ACCO	1-aminocyclopropene-1-carboxylate oxidase	Ethylene pathway
EIN3	EIN3-BINDING F-BOX PROTEIN 1	Ethylene pathway
TuA	Tubulin alpha-1 chain	Reference gene
Actin	Actin 7	Reference gene
GAPDH	Glyceraldehyde-3-phosphate dehydrogenase	Reference gene

Evaluation of the genotype-dependent variability in PRI efficacy

The experiment was conducted in 2022 and 2023 in five non-heated plastic tunnels, with a total cultivated area of 128 m² per tunnel. Each tunnel contained twelve tomato accessions

displaying comparable susceptibility to powdery mildew: Clodano, Criollo, Marpha n2, Ecu284, Vir884, Cisterno, LA1420, LA2619, Red Cherry Small, Red Plum, Vir2759, and Vir744. Plants were assigned to one of the four conditions: an untreated control group (NTC), a Planticare® treated group, a Messager® treated group and a Tagatose® treated group. Each treatment × accession combination included five biological replicates per tunnel. Five-week-old plants were treated following the procedure described in Section 2.2.1, with two applications performed 7 days and 3 days before inoculation. Two days before inoculation, plants were transplanted into tunnels located at the same experimental site in Avignon, France (43°56′N, 4°52′E), following a completely randomized design.



	Year	Date	1st treatment occurred on
JT24	2024	02-19-2024 / 05-15-2024	ten-week-old plants
SB24	2024	02-19-2024 / 04-18-2024	Six-week-old plants
SB25	2025	02-19-2025 / 04-29-2025	Seven-week-old plants

Fig. 1 Schematic representation of the experimental design used in the greenhouse trials. Pink boxes represent treated plants, whereas empty boxes represent untreated control plants. The experiments were conducted in three greenhouses. The table below provides additional information on the year of experimentation, the experiment duration, and the developmental stage of the plants at the time of the first treatment application

Mapping loci involved in plant response to Planticare®

The experiments were conducted from mid-February to April-May. One-month-old plants were individually transplanted into 17-cm-diameter pots filled with TS 3 MB Argile substrate (K Substrates). A total of 150 tomato accessions were included in the trial. Plants were assigned to one of two conditions: an untreated control (NTC) or a Planticare®-treated (P7). Each treatment × accession combination consisted of two biological replicates. Plants were

transferred to the greenhouses one week prior to treatment and were treated as described in Section 2.2.2. The experiments were repeated over two years: in 2024 in two greenhouses (SB and JT), and in 2025 in greenhouse SB only (see **Fig. 1** for details). In 2024, each greenhouse was divided into two blocks (treated and untreated), and each block contained two replicates of each accession. In 2025, the greenhouse was divided into four blocks, with full randomization of one plant per accession. Two blocks received the Planticare® treatment, while the remaining two blocks served as untreated controls. Due to germination issues, some accessions were not included in SB24 and JT24.

Inoculation and disease assessment

Powdery mildew inoculum was prepared by collecting infected leaves, which were placed in a 2 L bottle containing water and shaken to detach conidia. The concentration of conidia was estimated using a Malassez counting chamber, and the inoculum was adjusted to a final concentration of 1×10^4 conidia·mL^{-1}. Plants were inoculated using a hand-held sprayer. Phenotyping was performed between 9 and 14 days after inoculation on the first leaf located below the ring placed after treatment application.

Disease severity was assessed by (i) counting the number of powdery mildew lesions on the leaf and (ii) measuring leaf area using FIJI software from digital images. Disease intensity was expressed as the number of lesions per cm², calculated as the ratio between lesion number and leaf area. In trial 3, disease severity was also assessed on two leaves within the inoculated area using a six-level rating scale based on the appearance and extent of powdery mildew colonies on the leaflet surface (**Fig. 2**). The scores were assigned as follows: 0 = healthy leaflet with no visible symptoms; 1 = very small and sparse colonies; 2 = scattered colonies beginning to expand; 3 = more numerous colonies with early coalescence; 4 = extensive colony development covering large leaflet areas; 5 = dense colony coverage with marked chlorosis; 6 = leaflet almost entirely covered by powdery mildew and showing heavy sporulation.

Evaluation of PRI efficacies

The effectiveness of the PRIs was estimated for each plant as the percentage of protection of the plant using the formula according to (Tamm et al. 2011; Zhang et al. 2026) as:

$$\mathrm{PP} = 100 \text{ x } (1 - \frac{DISp7(ij)}{DISntc(j)})$$

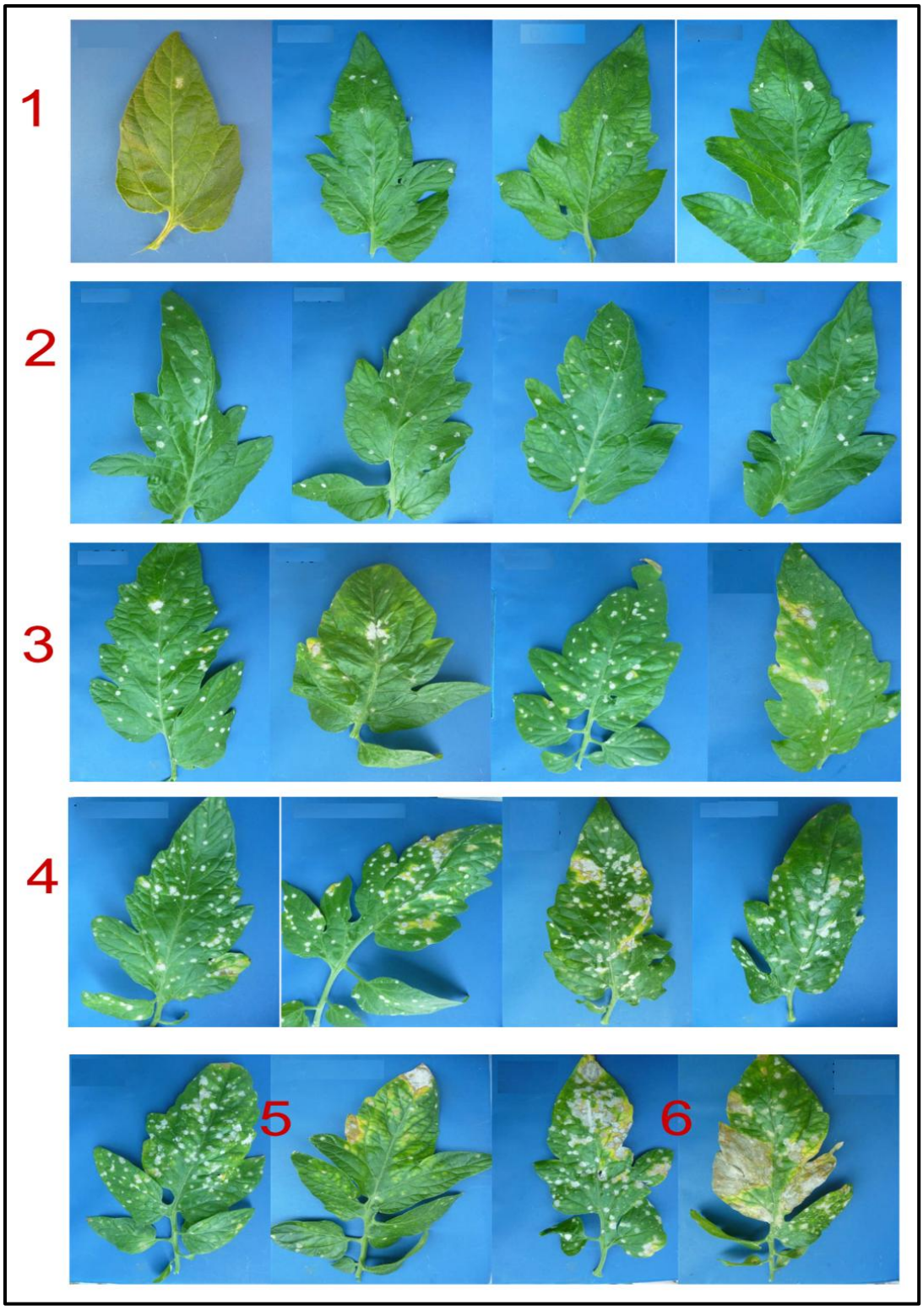


Fig. 2 Whole plant disease severity rating scale used to assess tomato powdery mildew caused by *Oidium neolycopersici*. The score was based on the appearance and extent of powdery mildew colonies on the leaflet surface, 0 = healthy leaflet with no visible symptoms; 1 = very small and sparse colonies; 2 = scattered colonies beginning to expand; 3 = more numerous colonies with early coalescence; 4 = extensive colony development covering large leaflet areas; 5 = dense colony coverage with marked chlorosis; 6 = leaflet almost entirely covered by powdery mildew and showing heavy sporulation

Where PP is the percentage of protection, DISp7(ij) is the disease index in treated condition of the i^{th} plant of the j^{th} genotype, and DISntc is the average of the disease index in NTC conditions for the j^{th} genotype. DISntc was calculated for each tunnel or greenhouse independently.

Before the evaluation of the efficacy in trial 2, the function of the R packages SpATS (Rodríguez-Álvarez et al. 2018) was used to correct the spatial effect on diseases index.

Estimation of genotype means and heritability

A linear mixed model was used to estimate the variances of the random effects and to calculate the broad-sense heritability (H^2). Different mixed models were used to estimate variances for heritability and best linear unbiased predictors (BLUPs). The mixed model and broad-sense heritability formula are shown in **Table 3**. Accessions with basal resistance under NTC condition (number of lesions per cm^2 equal to 0 in trial 2, and disease severity equal to 0 or 1 in trial 3) were removed from analysis concerning the percentage of protection.

Table 3 List of linear mixed model and heritability formula used in this study

Situation	**Linear mixed model**	**Heritability/ repeatability**
Each greenhouse separately Trial 3	$\mathbf{Y_{ik} = \mu + G_i + \varepsilon_{ik}}$	$\boldsymbol{H^2 = \frac{Vg}{Vg + \frac{Vr}{R}}}$
All tunnels Trial 2	$\mathbf{Y_{ijk} = \mu + G_i + T_j + G_i \times T_j + \varepsilon_{ijk}}$	$\boldsymbol{R^2 = \frac{Vg}{Vg + \frac{Vgt}{T} + \frac{Vr}{RT}}}$

In the mixed models, Y is the trait, μ is the general mean of the trait, G_i is the genetic effect of the i^{th} genotype, following a normal distribution $G \sim N(0, Vg)$, T_j is the effect of j^{th} tunnel with T a fixed effect in the model, G x T is the genotype by environment interaction effect, which follows a normal distribution $G \times T \sim N(0, Vgt)$, and Ɛ is the residual assumed to be independent and normally distributed with $\varepsilon \sim N(0, Vr)$.

In heritability and repeatability formula, H^2 is the broad-sense heritability of the trait, R^2 is the repeatability, Vg is the genetic variance, Vgt is the variance of the genotype by environment interaction, and Vr is the residual variance, T is the number of tunnels, and R is the number of replicates.

Genotypic data and Genome-Wide Association Study (GWAS) analysis

To detect associations between genotypic values and genomic markers, GWAS was performed using a set of 6,251,927 SNPs from genome resequencing (Héreil et al. 2024). SNP positions refer to the Heinz_1706 v.4.0.0 reference genome. SNPs exhibiting more than 3% heterozygosity were excluded from the dataset. All remaining heterozygous genotype calls were subsequently recoded as missing. Missing data were imputed by substituting the major allele, defined as the allele with the highest observed frequency across all samples. Following imputation, SNPs were filtered to retain only those with a minor allele frequency (MAF) greater than 10%. The final set of 82,969 SNP markers was retained and used for the identification of informative markers by GWAS.

To identify marker–trait associations, a GWAS was performed using the MM4LMM model (Min-Max algorithm for the Restricted Maximum Likelihood inference in Gaussian Variance Component mixed model) (Laporte et al. 2022), implemented in an in-house bioinformatic pipeline (https://forge.inrae.fr/gafl/users/sophia_marguerit/pipeline_gwas_nf_core, developed by Institut National de Recherche pour l' Agriculture, l'Alimentation et Environnrment, France). Both population structure and kinship matrices were used as covariates to reduce false-positive associations due to population structure and familial relatedness. The population structure was calculated using PCA with the first three components. The kinship matrix was generated using the VanRaden method (VanRaden 2008). We used a genome-wide threshold ($p< 1.77x10^{-6}$) that was determined based on the effective number of independent markers, Me, estimated using the Gao et al., (2008) method on R software. The genome-wide threshold was calculated with the equation, 0.05/Me. No significant marker was identified at $-\log10(p) \geq 5.75$, which was commonly identified in the greenhouses. Therefore, markers with $-\log10(p) \geq 4.75$ were considered significant. All retained markers were assigned to QTL regions based on their chromosomal position and the estimated linkage disequilibrium (LD). The LD parameter r^2 between pairs of SNPs was calculated using $-r^2$ command in PLINK software (Purcell et al. 2007) version 1.9 based on a window size of 1000 kb, following the method described by Wang et al. (2017). SNP pairs with $r^2 > 0.2$ were considered to belong to the same QTL region. High confidence QTLs were defined as QTLs with SNP with $-\log10(p) \geq 5.75$ or significant in at least two GWAS analysis with $-\log10(p) \geq 4.75$. For each high confident QTL, the SNP with the highest -log10(p) was retained as the lead marker. Each QTL name was defined as the associated trait name, followed by the chromosome number and the QTL's order among all QTLs for this trait along the chromosome.

Candidate gene identification

For each QTL, SNP with the lowest p-value was selected as the top SNP to explore candidate genes. The local LD was calculated between the top SNP of the different 'high-confidence' QTLs and all the SNPs belonging in these QTLs by using LDcorSV R package, which takes into account population stratification and the relatedness between genotypes (Mangin et al. 2012). We used $r^2 > 0.2$ to filter the putative candidate genes. For QTLs detected in several greenhouses, the top SNP was selected independently for each greenhouse, and LD analyses were performed as described above.

Candidate genes were investigated using the tomato reference genome assembly SL4.0 and the international tomato annotation group (ITAG) 4.0 at the Sol Genomics Network (https://solgenomics.net). From this subset, candidate genes were identified by manually searching gene descriptions for disease related genes.

3. Results

Expression of defense following PRI treatments

Analysis of the relative defense gene expression in the genotype Clodano (**Fig. 3**) revealed difference regulation level and pathway according to the PRI. Planticare®, P11 and P6 showed the strongest induction level, with 8-14 genes significantly induced, including 3-6 up regulated genes and 5-8 downregulated genes. Downregulated genes were mainly genes involved in the biosynthesis of metabolites and the reinforcement of cell walls, more precisely the gene PECT. The upregulated genes were PR proteins genes PR-1, PR-2 and PR-4 for all the three PRIs, and two additional PR, PR-5 and PR-8 with Planticare. Tagatose® and Messager® showed moderate induction, with 3 and 4 genes induced, respectively, including 3 PR proteins (PR-1, PR-2 and PR-4). An induction was also observed with water treatment. That was only significant on three metabolites genes (DFR, Far, and CSL). P3 was the less effective in terms of genes regulated. Only the gene PPO was downregulated under this condition. These results revealed that PR proteins genes were the most induced by the PRIs. Thus, only the PRIs which showed strong induction of PR proteins genes (P11, Planticare®, Tagatose® and Messager®) were remained to evaluate the genotype-dependent effect of the induction (**Fig. 4**).

Genotypic variation was observed at two levels. It concerned both the activation of different defense pathways and the intensity of the response. Regardless the PRI, the metabolite biosynthesis pathway was upregulated mainly in accession Criollo. Moreover, stronger

inductions were observed with accession Clodano, regardless the treatment, except under Tagatose treatment, where Vir884 was the most induced. Focusing on PR protein genes, variation among accessions treated with Planticare® was mainly related to differences in the

		Untreated control	P11	Planticare®	P6	Water	Messager®	P3	Tagatose®
PR-proteins	PR-1	0	5	5	2	1	3	0	2
	PR-2	0	2	6	1	0	2	0	3
	PR-4	0	4	6	2	1	3	1	3
	PR-5	0	1	4	0	0	1	0	1
	PR-8	0	1	3	1	0	1	0	1
	PR-14	0	0	-2	0	-1	1	-1	-1
	PR-15	0	1	-1	0	0	0	0	0
Metabolites	PAL	0	-2	-2	-2	0	-1	-1	-1
	CHS	0	-2	-3	-2	0	0	0	-1
	DFR	0	0	0	0	2	-1	0	0
	ANS	0	-1	-2	-1	0	0	0	-1
	PPO	0	0	-4	1	1	0	-2	0
	HMGR	0	-3	-4	-2	0	-1	0	1
	FPPS	0	0	-1	0	0	0	0	1
	Far	0	0	0	1	2	0	0	0
	CSL	0	0	-1	4	6	1	1	-1
Oxydative stress	Apox	0	1	0	0	0	0	0	0
	GST	0	1	0	0	0	0	0	1
	POX	0	0	1	-1	0	1	0	1
Cell wall	CalS	0	0	0	-1	0	0	0	0
	Pect	0	-3	-3	-2	0	-1	-1	-2
	CAD	0	-1	-2	-1	0	0	-1	0
SA signaling	EDS1	0	0	0	-1	1	0	0	1
	WRKY	0	1	-1	-1	0	-1	0	1
JA signaling	Lox2	0	0	0	1	1	0	0	-1
	JAR	0	-1	0	-1	0	0	0	-1
ET signaling	ACCO	0	2	2	0	1	1	0	0
	EIN3	0	-3	-1	-3	0	-1	-1	-1

Fig. 3 Relative defense gene expression of the genotype Clodano three days after the second treatment with P11, Planticare®, P6, water, Messager®, P3 and Tagatose®. Values represent the log2folchange values with untreated control plants used as the reference. Each value represents the mean of two independent experiments. Positive values indicate gene upregulation, whereas negative values indicate gene downregulation

intensity of gene expression. In contrast, with the other PRIs, some accessions showed upregulation while others showed downregulation. This pattern was particularly pronounced with Tagatose® and Messager®, for which PR protein genes were downregulated in the genotype Ecu284.

		P11					Planticare®					Tagatose®					Messager ®				
		Clodano	Criollo	Ecu 284	Marpha n2	Vir 884	Clodano	Criollo	Ecu 284	Marpha n2	Vir 884	Clodano	Criollo	Ecu 284	Marpha n2	Vir 884	Clodano	Criollo	Ecu 284	Marpha n2	Vir 884
PR-proteins	PR-1	5	3	4	3	3	5	7	3	4	5	2	2	-1	1	-1	3	6	-1	1	3
	PR-2	2	-1	0	0	0	6	2	1	4	4	3	0	0	0	1	2	0	-1	1	4
	PR-4	4	1	0	2	2	6	4	3	4	5	3	0	0	1	0	3	1	-2	1	2
	PR-5	1	-2	-2	-1	0	4	2	1	4	3	1	0	-1	1	0	1	1	0	1	3
	PR-8	1	1	0	0	0	3	2	1	2	2	1	0	0	1	0	1	0	-1	0	1
	PR-14	0	2	-2	1	-1	-2	0	-1	1	0	-1	0	-2	1	0	1	0	-2	1	0
	PR-15	1	1	1	1	0	-1	1	0	0	1	0	0	0	0	0	0	0	-1	0	1
Metabolites	PAL	-2	-2	-1	-2	-4	-2	-1	-2	-2	-2	-1	0	-1	-1	-1	-1	-1	-2	-1	-2
	CHS	-2	0	-1	-1	-2	-3	0	-1	-1	-2	-1	-1	0	0	-1	0	0	-1	-1	-6
	DFR	0	2	1	0	-1	0	2	0	1	0	0	3	1	0	-1	-1	1	-1	0	-1
	ANS	-1	0	-2	-1	-2	-2	0	-1	0	-1	-1	0	0	0	-1	0	1	-1	0	-2
	PPO	0	2	1	0	0	-4	0	-3	0	0	0	1	-1	0	1	0	0	-2	0	-1
	HMGR	-3	-3	-1	-5	-6	-4	-1	-1	-2	-3	1	-1	-1	0	0	-1	-1	-2	-2	-6
	FPPS	0	-2	-1	-2	-3	-1	0	-1	-1	-1	1	0	-1	1	0	0	0	-2	-1	-2
	Far	0	2	-1	-1	0	0	2	-1	0	0	0	3	1	1	-1	0	1	0	0	-1
	CSL	0	4	0	0	4	-1	4	1	-1	3	-1	7	6	0	0	1	4	4	0	1
Oxidative stress	Apox	1	1	0	1	1	0	1	1	1	1	0	0	0	1	0	0	0	0	0	0
	GST	1	1	0	0	0	0	0	0	1	1	1	0	0	0	0	0	0	-1	0	0
	POX	0	0	0	0	1	1	1	1	2	2	1	-1	-2	0	1	1	0	-2	1	2
Cell wall	CalS	0	0	0	-1	0	0	0	-1	0	0	0	0	0	0	0	0	0	-1	0	0
	Pect	-3	0	-2	-2	-4	-3	-2	-2	-1	-2	-2	0	-1	-1	-1	-1	-1	-2	-1	-3
	CAD	-1	-1	-1	-2	-2	-2	-1	-1	-1	-1	0	0	0	0	-1	0	-1	0	-1	-2
SA signaling	EDS1	0	0	0	0	0	0	-1	-1	0	0	1	0	-1	-1	0	0	-1	-1	0	0
	WRKY	1	0	1	0	1	-1	0	0	0	0	1	0	1	1	0	-1	-1	-1	-1	-1
JA signaling	Lox2	0	1	0	0	0	0	0	0	0	0	-1	0	0	0	0	0	0	-1	0	0
	JAR	-1	-1	-1	-1	-1	0	-1	-1	-1	-1	-1	0	0	0	0	0	0	0	0	0
ET signaling	ACCO	2	0	1	1	2	2	0	0	2	2	0	-1	-1	1	-1	1	-1	-2	0	2
	EIN3	-3	-2	0	-3	-3	-1	-2	-3	-2	-2	-1	-1	-1	-1	-1	-1	-1	-2	-2	0

Fig. 4 Relative defense gene expression of five genotypes three days after the second treatment with P11, Planticare®, Tagatose® and Messager®. Values represent the log2folchange values with untreated control plants used as the reference. Each value represents the mean of two independent experiments. Positive values indicate gene upregulation, whereas negative values indicate gene downregulation

Genotypic variation of the efficacy of PRIs against *Oidium neolycopersici*

The experiments revealed significant effects of genotype (**Table S1**, **Figure 5**), tunnel, and genotype × environment interaction (G × E) for all three tested PRIs, except for Tagatose®, where the G × E interaction was not significant. Pairwise comparisons between treated and NTC plants showed significant differences in disease severity for all accessions treated with Planticare®, except for Vir2759 (**Figure 5a**). With Messager®, significant differences were detected in four accessions, whereas eight accessions showed no significant response. Among

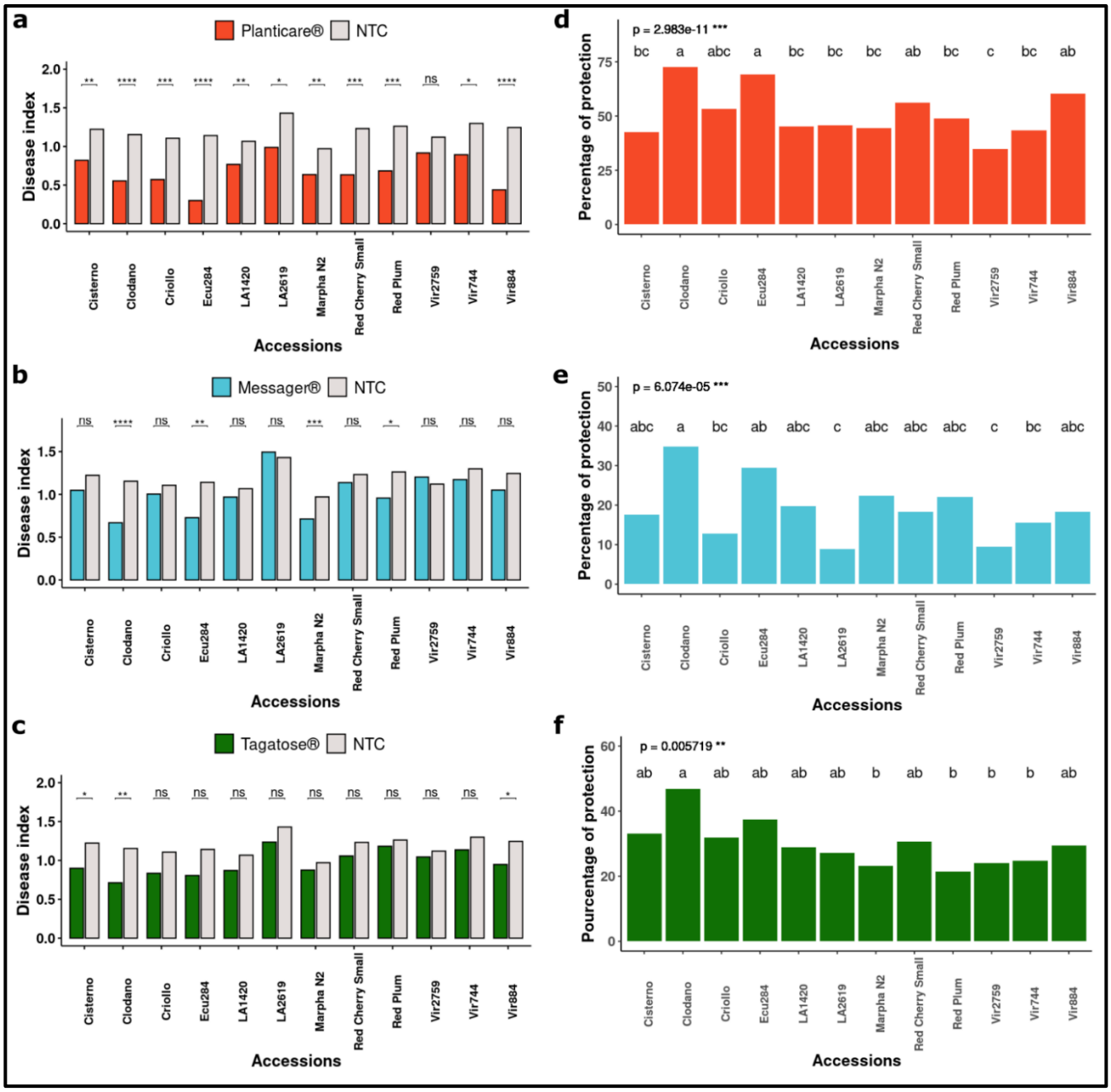


Fig. 5 Genotypic variation of the three PRI efficacy. a-c) Disease index of powdery mildew calculated from five biological replicates per tunnel for twelve tomato accessions treated with the Planticare®, Messager®, and Tagatose®, respectively. Green colors indicate values under treated conditions, and orange colors indicate values under untreated control conditions. Asterisks (*) indicate significant differences between treated and control modalities based on ANOVA. d-f) PRI efficacy against powdery mildew across the twelve accessions for Planticare®, Messager®, and Tagatose®, respectively. Different letters indicate statistically distinct groups among genotypes based on Tukey's post hoc test. Asterisks (*) denote significant differences between treated and control modalities according to ANOVA. (*) = significant at 0.05; (**) = significant at 0.01; and (***) = significant at 0.001.

the non-responsive accessions, LA2619 and Vir2759 nevertheless exhibited increased disease severity following treatment (**Figure 5b**). Under Tagatose® treatment, only three accessions (Criollo, Clodano, and Vir884) showed significant differences between treated and NTC conditions (**Figure 5c**). Among the tested genotypes, Clodano exhibited the strongest response to all PRIs, followed by Ecu284, whereas Vir2759 showed the lowest level of protection, with no significant differences between treated and control plants (**Figure 5 d-f**).

Overall, all PRI treatments reduced the mean severity of *O. neolycopersici* compared with NTC (**Table 4**). Planticare®, Messager®, and Tagatose® reduced powdery mildew severity by 51.37%, 19.15%, and 29.91%, respectively. In addition, relatively high repeatability estimates were obtained for Planticare® ($R^2 = 0.57$) and Tagatose® ($R^2 = 0.52$), whereas Messager® showed lower repeatability ($R^2 = 0.39$).

Planticare® was the most effective treatment, combining the highest protection level with the greatest repeatability. Consequently, it was selected for further mapping of loci involved in tomato accession responses using a larger accession panel.

Table 4 Efficacy of PRI against *O. neolycopersici* and the repeatability of plant response to their application

Treatment	Mean DIS	Mean efficacy (PP)	PP repeatability (R^2)
NTC	1.18 ±0.06		
Tagatose®	0.97 ±0.06	29.91 ±1.76	0.52
Messager®	1,01 ±0.06	19.15 ±1.54	0.39
Planticare®	0.68 ±0.05	51.37 ±1.70	0.57

Phenotypic variation within 148 tomato accessions

Planticare®-treated (P7) plants showed lower average disease index values across all independent greenhouse repetitions (**Table 5** and **Fig. 6a**). Regardless of the treatment, plants in greenhouse SB24 were the most infested, whereas those in JT24 were the least affected. Differences in percentage of protection were also observed among greenhouses, with the highest value recorded in JT24. The average efficacy in SB24, JT24, and SB25 was 35.95%, 57.13%, and 50.20%, respectively (**Table 5**). Although it is difficult in the present study to disentangle the respective effects of environmental conditions and plant age, the efficacy of Planticare® was strongly associated with the developmental stage at which plants were treated. Indeed, plants in SB24 were treated at 6 weeks of age and showed an average protection of

35.95%, whereas plants in SB25 and JT24 were treated at 7 and 10 weeks of age and exhibited substantially higher protection levels of 50.20% and 57.13%, respectively.

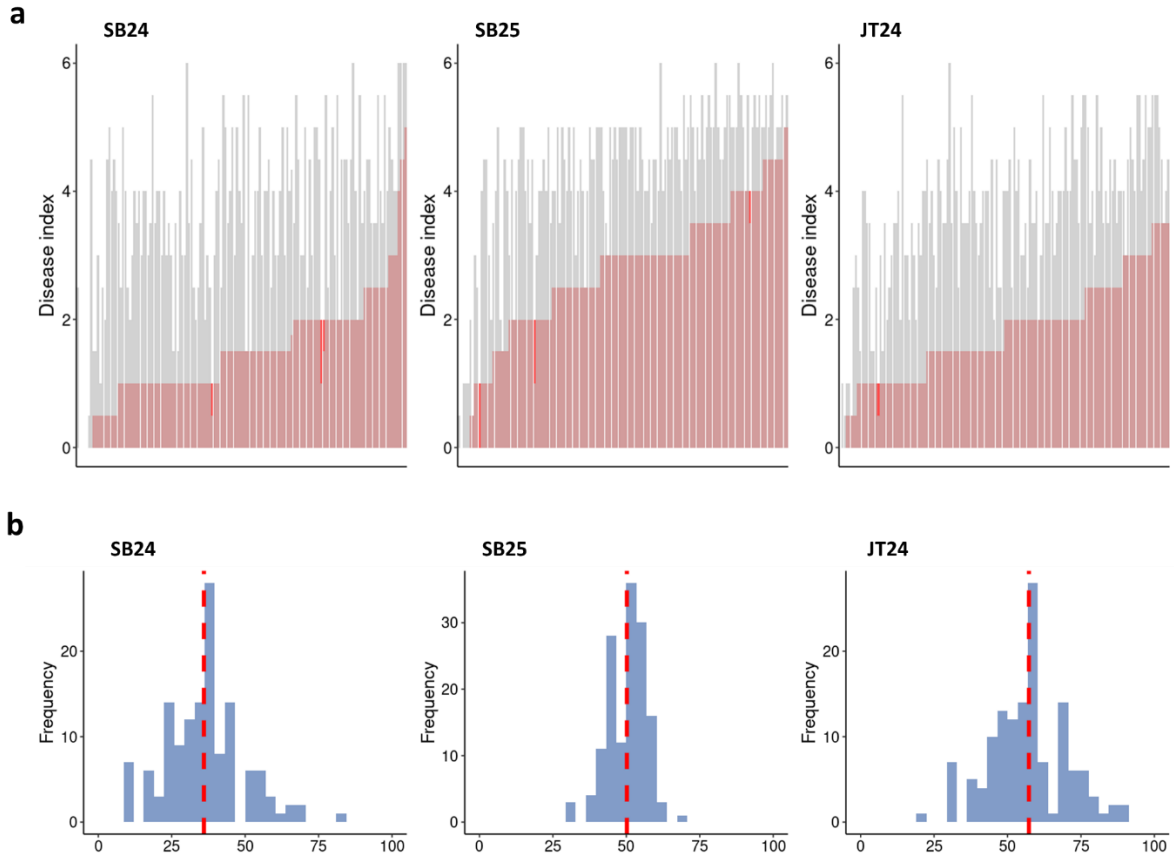


Fig. 6 Phenotypic variation a) Variability of tomato accessions resistance to *O. neolycopersici* under NTC and Planticare® treatment. Gray color represents the control values and red color represents resistance under Planticare® treatment. b) Phenotypic distribution of the percentage of protection (PP) of tomato accessions for each greenhouse. PP = BLUP + mean. Vertical lines in red indicate the average (mean) of PP. SB24: Phenotypic data in greenhouse SB24, SB25: Phenotypic data in greenhouse SB24, JT24: Phenotypic data in greenhouse JT24.

Strong and positive correlations ($p < 0.001$) were detected among greenhouses for disease index under both untreated control and treated conditions. Correlation coefficients between disease indexes recorded in the different greenhouses ranged from 0.63 to 0.73 under untreated conditions, and from 0.47 to 0.52 under treated conditions. Moreover, disease indexes in all greenhouses exhibited a positive correlation between untreated control and treated plants: r = 0.73 in SB24, r = 0.51 in JT24, and r = 0.62 in SB25 (**Fig. S1**).

Results also showed a phenotypic variation within the panel for all the three studied traits (disease in untreated control condition (NTC), disease in P7-treated plants, and percentage of protection (PP), **Fig. 6**). Distributions of the genetic values for percentage of protection (BLUP + mean) showed an approximately normal distribution of this trait in all greenhouses (**Fig. 6b**).

All 148 accessions exhibited a positive reaction to Planticare® application, with protection percentages ranging from 8.97% to 86.62% according to the phenotypic data. In SB24, more than 30 out of 137 accessions exhibited percentages of protection below 25%, whereas in SB25 and JT24, almost all accessions displayed protection levels above 25%.

Table 5 Statistics of the phenotypic data collected from 148 tomato accessions. H^2: Broad-sense heritability. NTC: untreated condition; P7: Planticare® treated condition; PP: percentage of protection

GH[a]	Trait	N accessions[b]	Mean	SE	H^2
SB24	Disease index in NTC (DISntc)	144	4.27	0.08	0.75
	Disease index in P7 (DISp7)	144	2.75	0.07	0.64
	Percentage of protection (PP)	137	35.95	1.25	0.75
SB25	Disease index in NTC (DISntc)	148	3.67	0.08	0.89
	Disease index in P7 (DISp7)	148	1.81	0.05	0.87
	Percentage of protection (PP)	144	50.20	1.33	0.35
JT24	Disease index in NTC (DISntc)	145	3.62	0.09	0.81
	Disease index in P7 (DISp7)	145	1.52	0.06	0.77
	Percentage of protection (PP)	138	57.13	1.44	0.67

[a]Greenhouse
[b]Number of accessions used for the analysis

Tomato responses to Planticare® are modulated by genetics

Broad-sense heritability was calculated for the disease index under both controlled and treated conditions, as well as for the percentage of protection. Except for the percentage of protection in SB25, where the broad-sense heritability was lower (H^2=35%), the broad-sense heritability was above 50% in all the greenhouses for all the traits (**Table 5** and **Fig. S2**). Under NTC, the broad-sense heritability of resistance to *O. neolycopersici* was 75% in SB24, 81% in JT24 and 87% in SB25. Under P7, the broad-sense heritability of resistance induced against *O. neolycopersici* ranged from 64% in SB24 to 87% in SB25. In J24 this value reached 77%. For the percentage of protection, which reflects PRI efficacy and plant response to the PRI, the broad-sense heritability was 75% in SB24, 67% in JT24 and 35% in SB25.

Population structure

Population genetic diversity analysis was conducted on the 148 tomato accessions. The PCA eigenvalue plot (**Fig. 7a**), generated using GAPIT 3, indicating that the first three principal components (PCs) explain the majority of the variation among the 148 accessions (40.92 %). Phylogenetic trees were constructed using the neighbor-joining (NJ) method in GAPIT 3,

resulting in an unrooted tree (**Fig. 7b**) displaying three distinct sub-populations. A 3D graphical plot of principal component analysis (PCA) (**Fig. 7c**) illustrates the three sub-populations. These plots and phylogenetic trees revealed the presence of three distinct clusters within the core-collection. The three principal components of the PCA were subsequently used as a Q-matrix in the GWAS analysis. Among the three clusters (sub-populations), G1 consisted of 18 individuals (12.2% of the entire population), G2 included 123 individuals (83.1%), and G3 comprised 7 individuals.

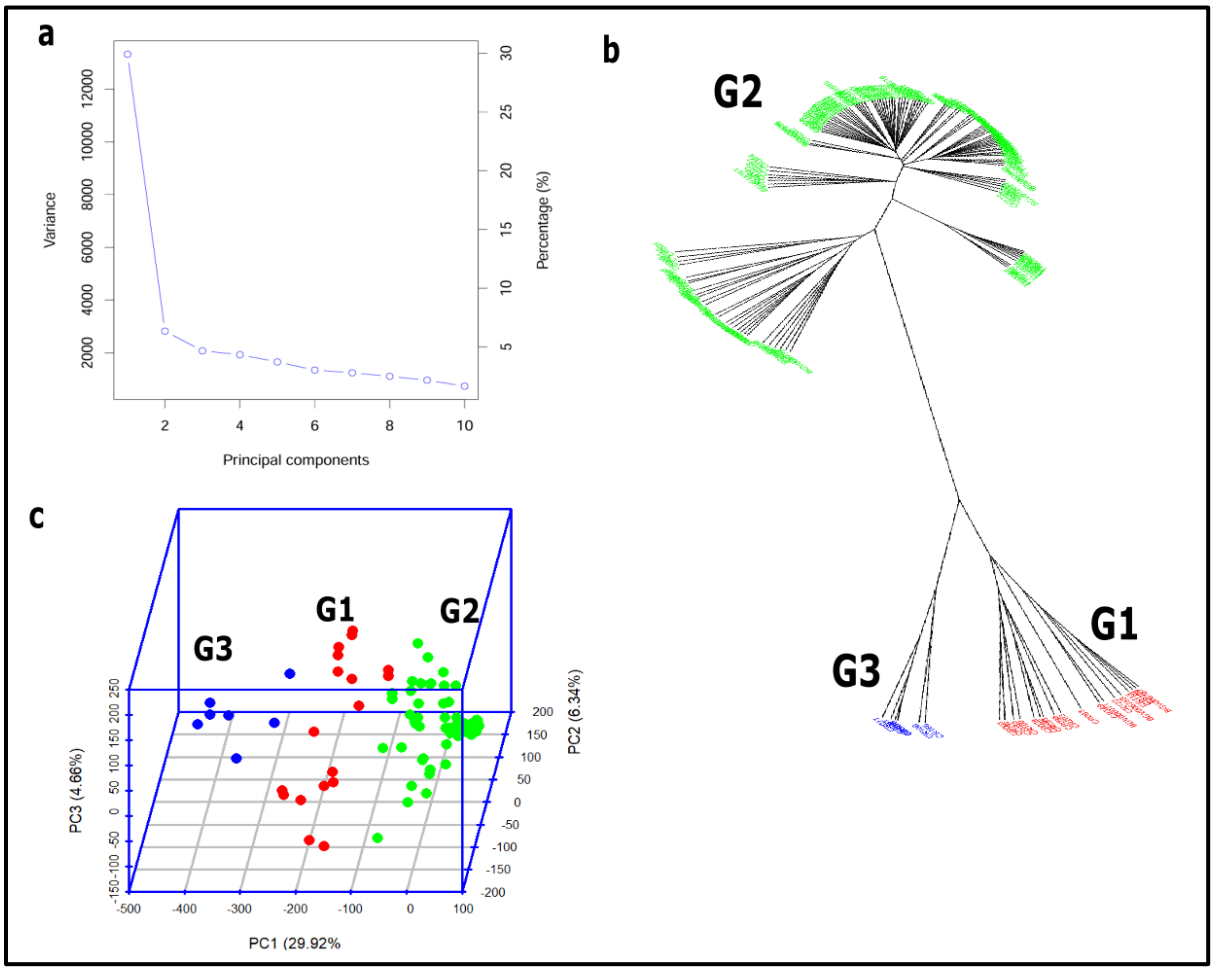


Fig. 7 Population genetic diversity analysis of the 148 tomato accessions. **a)** PCA eigenvalue plot generated using GAPIT 3, showing that the top three principal components (PCA = 3) explain majority of the variation among the 148 individuals; **b)** Phylogenetic tree constructed using the neighbor-joining (NJ) method, showing three subpopulations; and **c)** 3D graphical plot of principal component analysis (PCA), illustrating the three sub-populations

GWAS identified several QTLs annotated to high confidence candidate gene

A total of 27 SNPs was associated with the percentage of protection conferred by Planticare®, including four significant SNPs at log10(p) > 5.75 (**Fig. 8**). These SNPs were

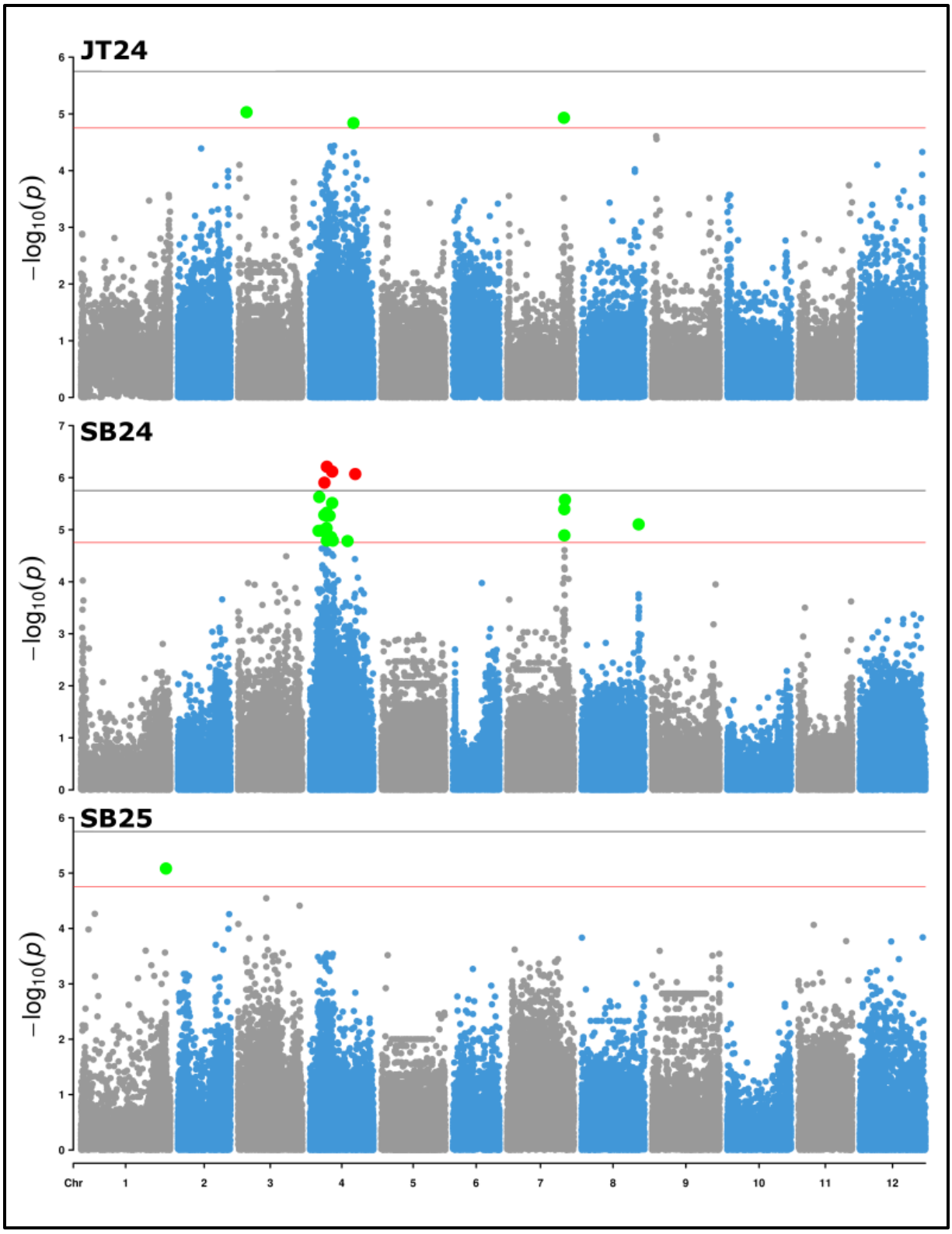


Fig. 8 Manhattan plots showing SNP associated with tomato accession responses to Planticare® (PP) in the greenhouses JT24, SB24 and SB25. The X-axis shows SNPs along each chromosome; the Y-axis is the -log10(p) for the association. The gray lines indicate the Gao significance threshold (-log10(p) = 5.75), and the red lines indicate the Gao significance threshold (-log10(p) = 4 .75); Red points indicate the significant SNPs at log10(p) = 5.75, and green points indicate the significant SNPs at log10(p) = 4.75

grouped into five QTLs. Three of these QTLs were located on chromosome 4 in SB24 and explained between 14% and 15% of the phenotypic variance. One QTL located on chromosome 7 was detected in both JT24 and SB24 and explained 13% and 20% of the phenotypic variance, respectively (**Table 6**). The fifth QTL was located on chromosome 8 in SB24 and explained 16% of the phenotypic variance. For the percentage of protection trait, significant SNPs at the threshold of −log10(p) > 5.75 were identified only in SB24. Interestingly, all SNPs associated with percentage of protection had minor allele frequencies, occurring in only 16-18 accessions, having all a percentage of protection over 20% (**Fig. 9**).

Table 6 Statistic of the significant SNP associated with tomato resistance to *O. neolycopersici* and responses to Planticare® application

Trait	QTL	Greenhouse	SNP	Chr	Position	Pvalue	R^2
% of protection	PP_4_1	SB24	SNP82617	4	17,165,065	6.16e-07	0.15
	PP_4_2	SB24	SNP85877	4	22,535,645	7.64e-07	0.14
	PP_4_3	SB24	SNP93057	4	45,950,336	8.52e-07	0.14
	PP_7_1	JT24	SNP156028	7	57,717,728	1.17e-05	0.13
		SB24	SNP156079		58,127,317	4.04e-06	0.20
	PP_8_1	SB24	SNP184735	8	58,001,418	7.90e-06	0.16

Chr: Chromosome

R^2: Phenotypic variance explained by the SNP, calculated using ANOVA for each SNP independently

In addition to plant responses to PRI, we also mapped loci associated with disease index under both Planticare® treatment and NTC conditions. For disease index under Planticare® treatment (P7), 12 SNPs were identified, including three that exceeded the significance threshold of −log10(p) > 5.75 (**Fig. S3**). These SNPs are grouped into four QTLs: one located on chromosome 2, one on chromosome 4, and two on chromosome 8. All four QTLs were detected in a single greenhouse environment and explained between 3% and 12% of the phenotypic variance in their respective environments (**Table S2**).

Thirty-two SNPs were associated with disease index under NTC conditions, including six that exceeded the significance threshold of −log10(p) > 5.75 (**Fig. S4**). These SNPs are grouped into seven QTLs: one on each of chromosomes 1, 4, 5, 8, and 12, and two on chromosome 9. The QTL on chromosome 5, DISntc_5_1, was detected in both SB24 and SB25, whereas the

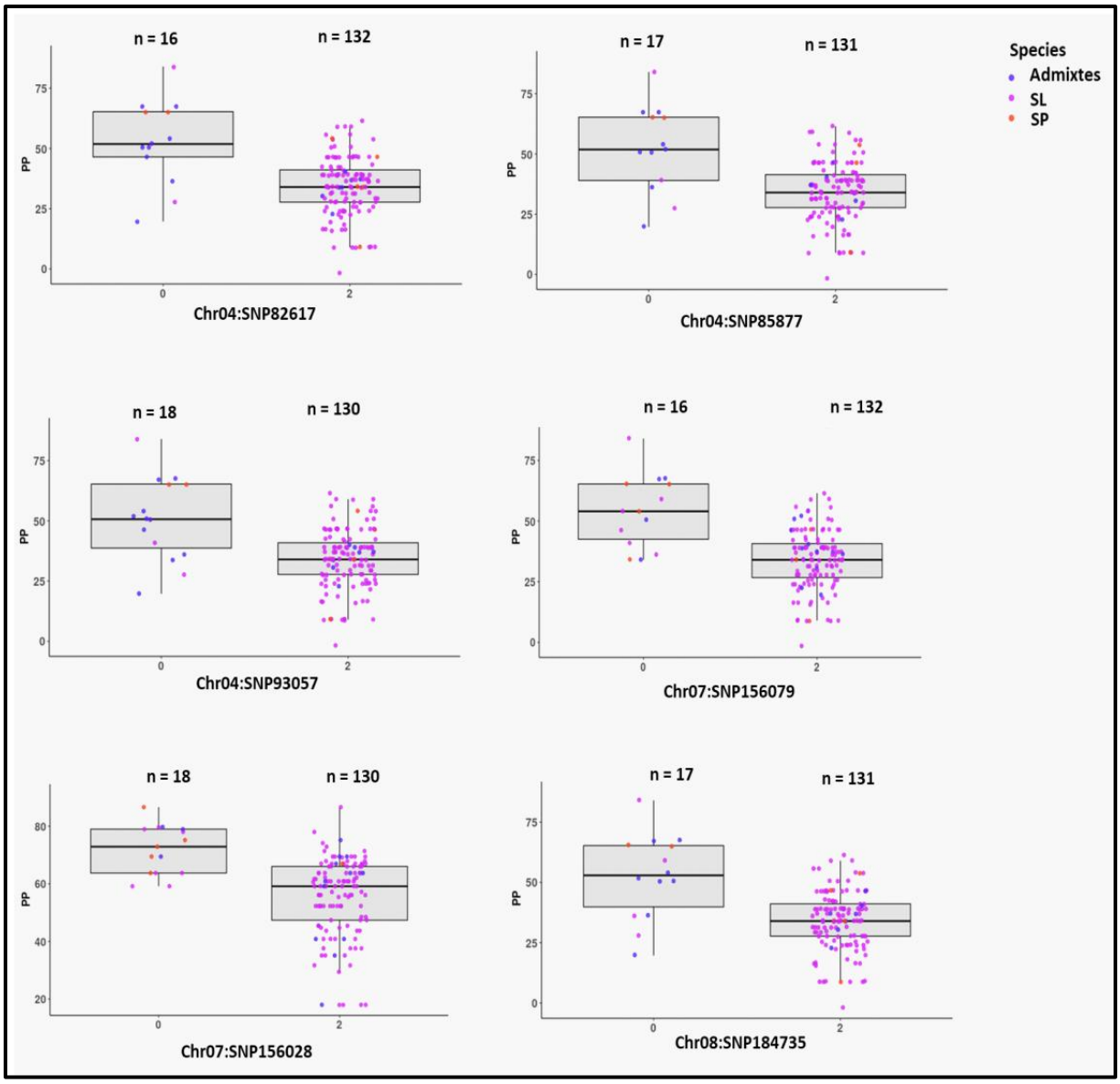


Fig. 9 Phenotypic differences of the percentage of protection among the significant SNP detected in SB24. The text above the boxplot indicates the number of individuals in the genotypic class. Difference point colors represent the species of the plant: SP for *Solanum pimpinellifolium* (red), SL for *Solanum lycopersicum*, including cerasiforme and large-fruited tomato (pink), and mixture for the admixtures of SLC and SP

QTL on chromosome 9, DISntc_9_2, was detected in SB25 and JT24 with the same SNP. Overall, QTLs associated with disease index under NTC conditions explained between 5% and 32% of the phenotypic variance (**Table S2**).

Only the QTL DISp7_8_2 (associated with disease index under Planticare® treatment) was located in close proximity to QTL PP_8_1 (associated with percentage of protection) in the same environment. Aside from this case, no QTLs were shared between percentage of protection and innate resistance, nor between resistance under Planticare® treatment and innate

resistance. However, two QTL hotspots were detected across all traits: one on chromosome 4, which contained five QTLs, and another on chromosome 8, which harbored four QTLs. Some QTLs were located on the same chromosomal arms, notably: on the long arm of chromosome 4 (DISntc_4_1, DISp7_4_1, PP_4_3), and on the long arm of chromosome 8 (DISp7_8_1, DISp7_8_2 = PP_8_1).

Candidate genes surrounding significant SNPs

All the identified QTL regions were further analyzed to look at genes annotated within these regions (**Supplementary data 2**). In total, 307 annotated genes were identified in the regions of the QTLs associated with the percentage of protection according to the tomato reference genome assembly SL4.0 and the international tomato annotation group (ITAG) 4.0.

Several genes with annotated functions related to secondary metabolism and plant defense were identified within the QTL intervals. These include genes encoding cytochrome P450 monooxygenases, glycosyltransferases, HXXXD-type acyltransferases, S-adenosyl-L-methionine-dependent methyltransferases, 2-oxoglutarate-dependent dioxygenases, 4-coumarate-CoA ligase-like 3, 5-epiaristolochene 1,3-dihydroxylase, and phosphomevalonate kinase, all of which are associated with the biosynthesis or modification of specialized metabolites. The intervals also contained genes encoding defense-related proteins, including a peroxidase, a β-1,3-glucanase, an antifungal protein ginkbilobin-like, receptor-like kinases and disease resistance proteins. In addition, several transcription factors belonging to the WRKY, MYB, NAC and bHLH families were identified, together with genes encoding receptor-like kinases, disease resistance proteins and other signaling components. The complete list of annotated genes identified within the QTL intervals is provided in **Supplementary data 2**.

For disease index under Planticare® conditions, 274 genes were annotated within the QTL intervals, including several candidates involved in secondary metabolism and plant defense. These included genes associated with specialized metabolite biosynthesis, such as cytochrome P450 monooxygenases, 1-deoxy-D-xylulose 5-phosphate synthase, phosphomevalonate kinase, 4-coumarate-CoA ligase-like 3, and methyltransferases. Defense-related genes were also identified, including peroxidases, disease resistance proteins (RGA2), MLO-like proteins, receptor-like kinases, lectin receptor kinases, and pleiotropic drug resistance proteins, together with WRKY, MYB, AP2/ERF and bHLH transcription factors.

Under NTC conditions, QTL intervals contained several candidate genes potentially involved in powdery mildew resistance, including LRR- and G-type lectin receptor-like

kinases, defense-related transcription factors (WRKY22, WRKY41, MYB and bHLH), cell wall-associated genes such as *cutin deficient 2* and cellulose synthase-like C4, as well as peroxidase and multiple CC-NBS-LRR and TIR-NBS-LRR resistance genes, reinforcing the presence of classical R-genes within these intervals.

4. Discussion

First report of QTL underlying tomato responses to PRI against *O. neolycopersici*

Our results reveal a clear genotype-dependent effect of Planticare® efficacy across the 148 tomato accessions. This effect was strongly shaped by plant genetics, with broad-sense heritability estimates ranging from 35% to 75%, depending on the repetition. These values are similar to the heritability of disease severity observed under both untreated control and Planticare® treatment conditions. This pattern is consistent with findings in wheat and barley, where the heritability of the efficacy of the PRI N-Acyl Homoserine Lactones closely mirrored the heritability measured under control condition (Wehner et al. 2019; Soleimani et al. 2024).

In our study, we identified five QTLs associated with the percentage of protection on chromosomes 4, 7, and 8, explaining between 13% and 20% of the phenotypic variance. This work provides the first evidence of genomic regions involved in tomato accession responses to a PRI applied against *O. neolycopersici*. These results highlight the value of exploring the genetic diversity present in tomato germplasm, particularly as a source of alleles that could be used in biparental crosses to improve PRI responsiveness.

The protection conferred by Planticare® was strongly associated with the developmental stage at which plants were treated, suggesting that plant developmental stage may influence the efficacy of Planticare® against *O. neolycopersici*. A similar pattern was reported by Rossi et al. (2025), who showed that the efficacy of several resistance-inducing compounds, including Fosetyl-Al, laminarin, COS-OGA, and potassium phosphonate, against grapevine downy mildew (*Plasmopara viticola*) depended on berry developmental stage, with higher levels of protection observed at later stages of development. This observation supports the hypothesis that host developmental status can modulate the efficacy of induced resistance strategies.

In addition to the percentage of protection, we mapped four QTLs associated with disease index under Planticare® treatment, located on chromosomes 2, 4, and 8. We also identified seven QTLs associated with disease index under untreated control conditions, located on chromosomes 1, 4, 5, 8, and 9. In our study, no QTLs were shared between percentage of

protection and innate resistance, nor between resistance under Planticare® treatment and innate resistance. Similarly, in their studies, Soleimani et al. (2024) and Thielmann et al. (2026), also reported no common QTLs between wheat leaf-rust and wheat powdery mildew resistance and PRI efficacies.

Comparing our results with previously reported powdery mildew resistance (Van Der Beek et al. 1994; Ciccarese et al. 1998; Huang et al. 2000b; Bai et al. 2003, 2005; Bai 2004; Faino et al. 2012; Park et al. 2022), some of the QTLs detected in this study were located near two resistance QTLs described by Park et al. (2022). For instance, DISp7_8_2 and PP_8_1 lie very close to QTL-4 on chromosome 8 (approximately 1 Mb from its start position), and DISntc_1_1 is located about 5.6 Mb from QTL-1 on chromosome 1. However, these QTLs cannot be considered identical, as they fall within chromosomal regions known to be highly recombinant (Sim et al. 2012; Torgeman et al. 2024; Jia et al. 2025).

Candidate genes within the QTLs detected

The candidate genes identified within the intervals of QTLs detected for the percentage of protection include several enzymes and regulatory proteins known to participate in the biosynthesis and regulation of plant defense-related secondary metabolites. Among the most promising candidates are cytochrome P450 monooxygenases, which catalyze key oxidation reactions involved in the synthesis of phytohormones, signaling molecules, flavonoids, alkaloids, terpenoids, sterols, fatty acids, lignin (Hansen et al. 2021; Zhang et al. 2023). The presence of a 4-coumarate-CoA ligase-like (4CL) gene further highlights the potential involvement of the phenylpropanoid pathway, as 4-coumarate-CoA ligases contribute to channelize precursors for different phenylpropanoids (Lavhale et al. 2018). Several glycosyltransferases, HXXXD-type acyltransferases and S-adenosyl-L-methionine-dependent methyltransferases were also identified, suggesting active modification of secondary metabolites through glycosylation, acylation and methylation, processes that regulate the stability, transport and biological activity of defense compounds (Gharabli et al. 2023; Majeed et al. 2024; Zhao et al. 2025). Altogether, these results indicate the possible involvement of flavonoid, phenylpropanoids and terpenoid biosynthetic pathways in the defense induce by Planticare®.

In addition, genes encoding a peroxidase and a β-1,3-glucanase were also identified as interesting candidate genes for the PRI response. In our gene expression analysis, members of these gene families were significantly induced following Planticare® treatment. Although the

genes identified within the QTL intervals are not the same as those differentially expressed in response to Planticare®, their belonging to the same functional families suggests that these defence-related pathways may play an important role in the induced resistance triggered by Planticare®.

Almost the same pattern was observed for disease index under Planticare® treatment. Altogether, these genes constitute promising candidate genes potentially involved the resistance induced by Planticare®. Further functional characterization, including transcriptomic analyses, metabolites profiling and reverse genetics approaches, will be required to validate their role.

Under NTC conditions, QTL intervals contained several candidate genes potentially involved in disease resistance, including LRR- and G-type lectin receptor-like kinases (RLKs), cell wall-associated genes such as *cutin deficient 2* and cellulose synthase-like C4, as well as peroxidase and multiple CC-NBS-LRR and TIR-NBS-LRR resistance genes. RLKs, CC-NBS-LRR, and TIR-NBS-LRR proteins represent major classes of resistance genes that perceive pathogens or their effectors and activate downstream defense responses (Wang et al. 2022; Mengiste and Liao 2025). In tomato, resistance gene to several fungal pathogens, including *Cladosporium fulvum* (*Cf-2*, *Cf-4*, *Cf-5*, *Cf-9*), *Verticillium dahlia* (*Ve*), and *Fusarium oxysporum f. sp. lycopersici* (*I*, *I-3*, and *I-7*), is mediated by LRR-containing receptor-like proteins (Fradin et al. 2009; Catanzariti et al. 2015, 2017; Gonzalez-Cendales et al. 2016). Zhu et al. (2025) further demonstrated that SlLRR-RLK94 positively regulates tomato resistance to *Phytophthora infestans*. The *Ph-3* resistance gene, conferring resistance to *Phytophthora infestans*, encodes a CC-NBS-LRR protein (Zhang et al. 2014a). Previously described powdery mildew resistance loci *Ol-4* and *Ol-6*, which confer rapid resistance to *O. neolycopersici*, have been proposed to be homologous to the *Mi-1* locus, a cluster of R genes encoding CC-NBS-LRR proteins (Vos et al. 1998; Bai et al. 2005; Seifi 2011; Seifi et al. 2014).

5. Conclusion

This study provides the first report of QTLs and candidate genes controlling tomato responses to plant resistance inducers (PRIs) in the context of *O. neolycopersici* infection. We identified five QTLs associated with tomato accession responses to the application of Planticare®, which were located on chromosomes 4, 7 and 8. Several candidate genes were identified within the significant SNP regions, including enzymes involved in the biosynthesis and regulation of plant defense-related secondary metabolites and genes encoding defense-

related proteins (peroxidase, a β-1,3-glucanase, an antifungal protein ginkbilobin-like). Additionally, four QTLs were detected for disease index under Planticare® treatment and seven QTLs for disease index under NTC conditions. sixteen QTLs, including seven QTLs associated with powdery mildew resistance under untreated control conditions, four QTLs associated with powdery mildew resistance under Planticare® treatment and several candidate genes were identified within the significant SNP regions, including disease resistance proteins (RLK, NBS-LRR proteins), peroxidase, glutathione S-transferases, cellulose synthase, transcription factors, and pathogenesis-related proteins, among others. These findings provide a first genetic framework for understanding PRI-mediated responses in tomato and open new avenues for dissecting how different classes of defense genes contribute to priming efficacy. Ultimately, this work lays the foundation for the targeted exploitation of PRI-responsive loci in breeding programs aiming to enhance durable resistance to powdery mildew.

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SUPPORTING INFORMATION

Table S1 Analysis of variances for tomato responses to plant responses to PRI applications

PRI	Effect	F-value	P-value
P4	Genotype (G)	2.4852	0.005719 **
	Tunnel (T)	61.0770	< 2.2e-16 ***
	Genotype x Tunnel	1.1962	0.200458
P5	Genotype	3.7388	6.074e-05 ***
	Tunnel	30.0839	< 2.2e-16 ***
	Genotype x Tunnel	3.5742	1.483e-10 ***
P7	Genotype	7.6213	2.983e-11 ***
	Tunnel	42.6348	< 2.2e-16 ***
	Genotype x Tunnel	4.2167	2.725e-13 ***

(*) denotes significance at a P-value of 0.05,

(**) denotes significance at a P-value of 0.01,

(***) denotes significance at a P-value of 0.001.

No Asterix indicated the correlation coefficient is not statistically significant.

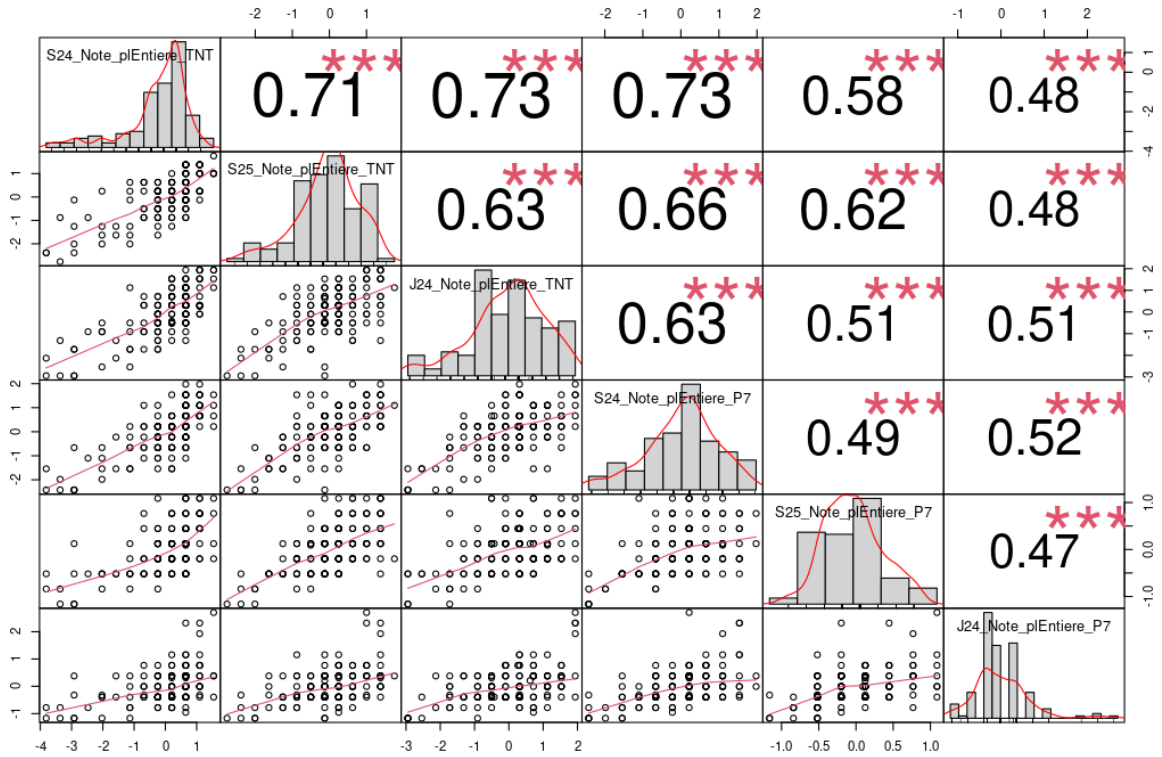


Fig. S1 Correlation plot between disease severity the untreated control condition (NTC), and under the Planticare® treatment condition (P7). S24= SB24, S25=SB25, J24=JT24, Note_plEntiere = Disease severity. (***) denotes significance at a P-value of 0.001

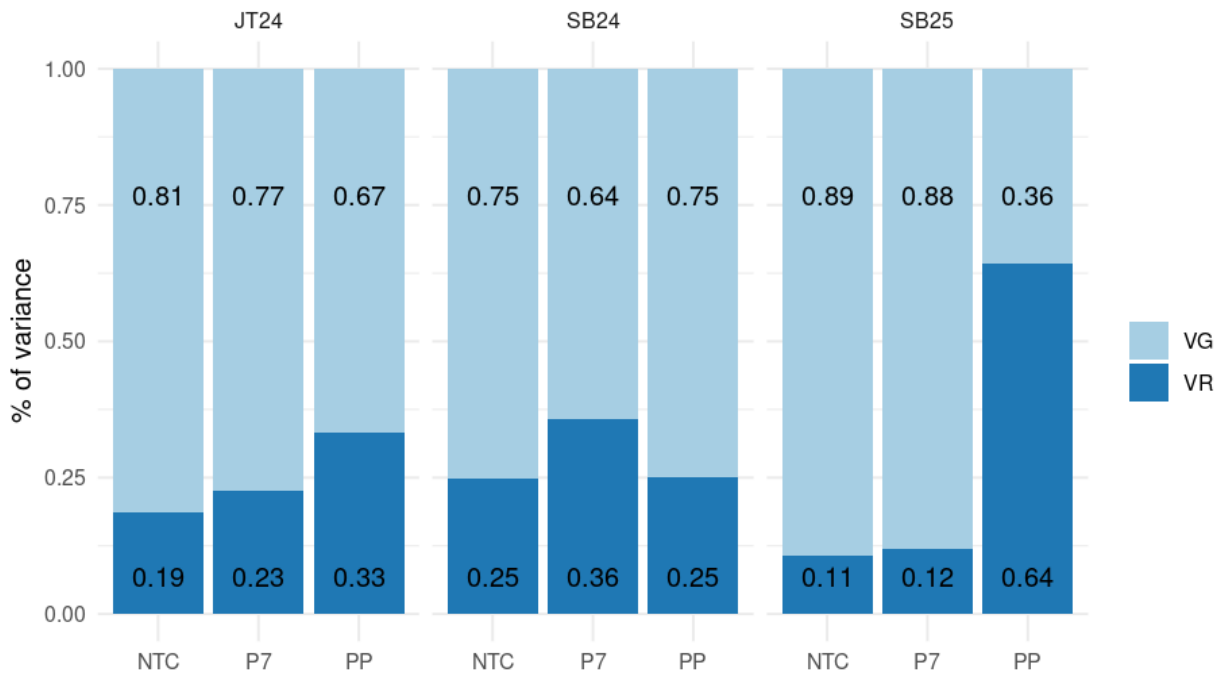


Fig. S2 Variance decomposition of disease severity under the untreated control condition (NTC), under the Planticare® treatment condition (P7), and for the percentage of protection (PP), in the greenhouses JT24, SB24, and SB25. Light blue represents the proportion of genetic variance, and dark blue represents the proportion of residual variance

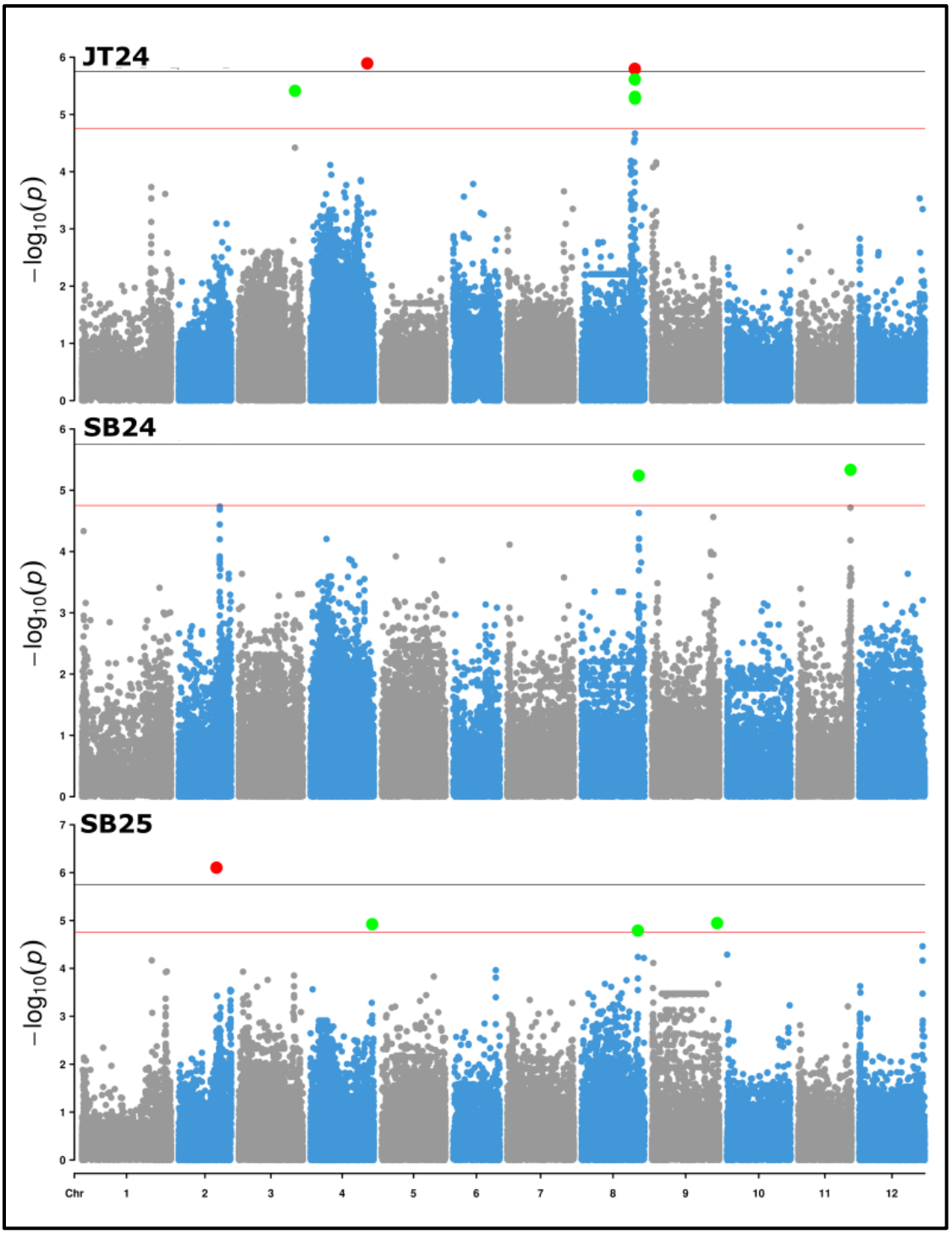


Fig. S3 Manhattan plots showing SNP associated with tomato resistance to *O. neolycopersici* under P7 conditions in the greenhouses JT24, SB24 and SB25. The X-axis shows SNPs along each chromosome; the Y-axis is the -log10(p) for the association. The gray lines indicate the Gao significance threshold (-log10(p) = 5.75), and the red lines indicate the Gao significance threshold (-log10(p) = 4 .75); Red points indicate the significant SNPs at log10(p) = 5.75, and green points indicate the significant SNPs at log10(p) = 4.75

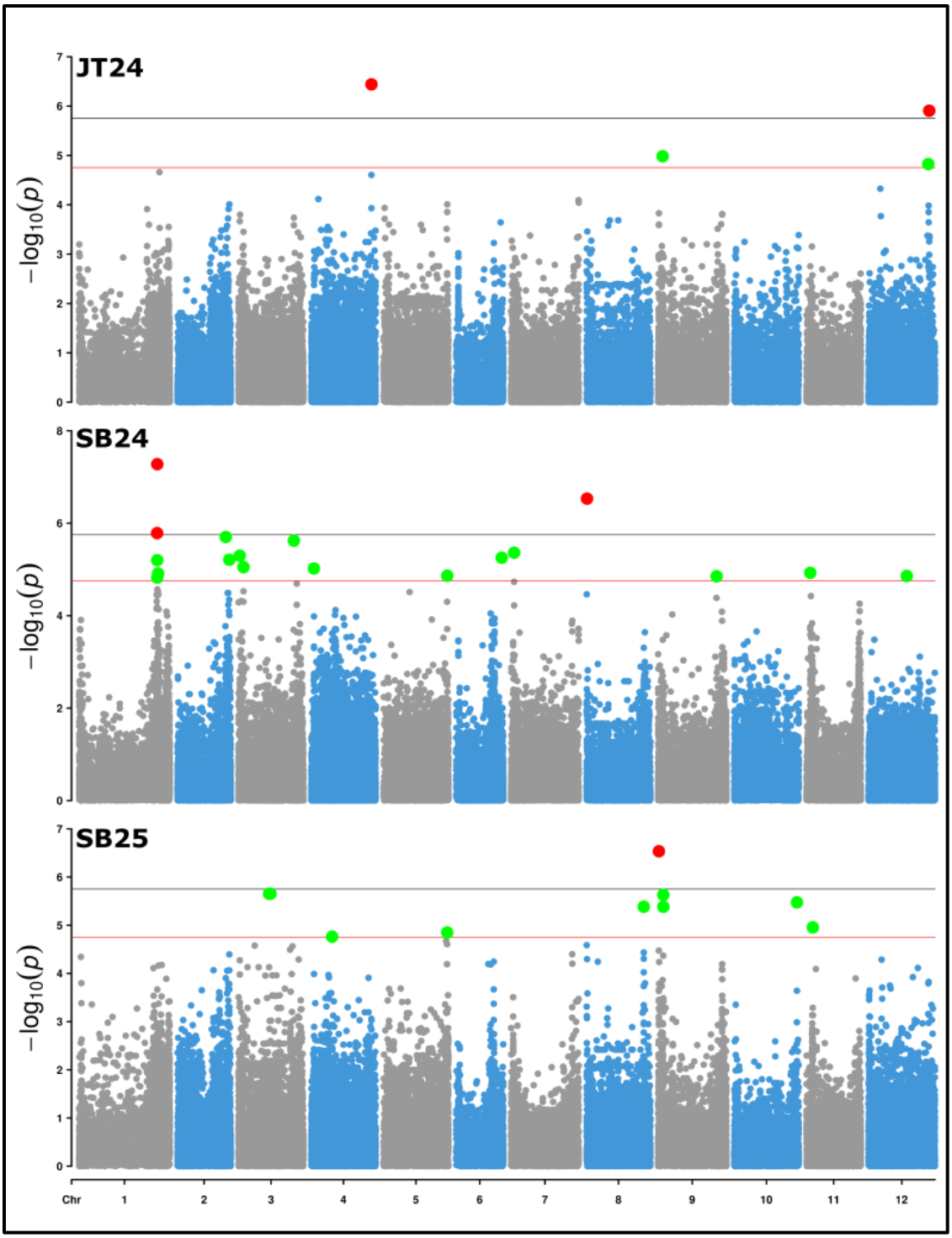


Fig. S4 Manhattan plots showing SNP associated with tomato resistance to *O. neolycopersici* under NTC conditions in the greenhouses JT24, SB24 and SB25. The X-axis shows SNPs along each chromosome; the Y-axis is the -log10(p) for the association. The gray lines indicate the Gao significance threshold (-log10(p) = 5.75), and the red lines indicate the Gao significance threshold (-log10(p) = 4 .75); Red points indicate the significant SNPs at log10(p) = 5.75, and green points indicate the significant SNPs at log10(p) = 4.75

Table S2 Statistic of the significant SNP associated with tomato resistance to *O. neolycopersici* and responses to Planticare® application

Trait	QTL	Greenhouse	SNP	Chr	Position	Pvalue	R^2
Disease index under NTC	DISntc_1_1	SB24	SNP33739	1	78,246,797	5.27e-08	0.22
	DISntc_4_1	JT24	SNP97876	4	60,246,272	3.62e-07	0.11
	DISntc_5_1	SB24	SNP124984	5	64,001,974	1.36e-05	0.22
		SB25	SNP124984		64,001,974	1.41e-05	0.20
	DISntc_8_1	SB24	SNP158246	8	499,728	2.93e-07	0.32
	DISntc_9_1	SB25	SNP186425	9	763,471	2.91e-07	0.05
	DISntc_9_2	JT24	SNP187441	9	4,520,228	1.03e-05	0.11
		SB25	SNP187581		5,168,932	2.34e-06	0.12
	DISntc_12_1	JT24	SNP249788	12	60,830,878	1.23e-06	0.16
Disease index under Planticare®	DISp7_2_1	SB25	SNP48296	2	38,379,206	7.87e-07	0.03
	DISp7_4_1	JT24	SNP97133	4	57,706,419	1.28e-06	0.12
	DISp7_8_1	JT24	SNP183612	8	54,195,197	1.59e-06	0.06
	DISp7_8_2	SB24	SNP184837	8	58,254,233	5.76e-06	0.12

Chr: Chromosome

R^2: Phenotypic variance explained by the SNP, calculated using ANOVA for each SNP independently

Chapter 4

Powdery mildew resistance QTL

This chapter is in the form of an original article submitted to the journal Theorical and Applied Genetics

In this chapter, we focus on identifying QTLs associated with tomato powdery mildew resistance in a wild tomato relative that has not yet been explored for these traits. Before conducting QTL mapping, we screened 31 tomato accessions belonging to six species for resistance to *Oidium neolycopersici*. Among the highly resistant accessions, "LA0421", from the species *Solanum cheesmaniae*, was selected for mapping the resistance loci because it is genetically close to cultivated tomato and has not yet been used to map powdery mildew resistance QTLs. Using a combination of QTL-seq and linkage mapping, we identified a resistance locus on chromosome 9 and highlighted several candidate genes potentially involved in resistance mechanisms. Owing to its close genetic relationship with cultivated tomato, *S. cheesmaniae* represents a novel and more easily introgressable source of resistance, offering strong potential for breeding programs aiming to improve disease resistance in tomato.

Title: New powdery mildew resistance QTL found on chromosome 9 in *Solanum cheesmaniae*

Awa Sangaré [1,2], Jean-François Bourgeay [1], Anne Massire [2], Aurelie Berard [3], Damien Hinsinger [3], Bernard Caromel [2], Marc Bardin [1]

Affiliation

[1]INRAE, Unité de recherche 407 Pathologie Végétale, 84143, Montfavet Cedex, (Provence-Alpes-Côte d'Azur), France

[2]INRAE, Unité de recherche 1052 Génétique et Amélioration des Fruits et Légumes, 84143, Montfavet Cedex, (Provence-Alpes-Côte d'Azur), France

[3]INRAE, Unité de service 1279 Etude du Polymorphisme des Génomes Végétaux, CEA Institut de biologie François Jacob, 91057, Evry Cedex, (Île-de-France), France

Corresponding authors

bernard.caromel@inrae.fr

marc.bardin@inrae.fr / https://orcid.org/0000-0002-2803-7695

Key message

A novel powdery mildew resistance QTL on tomato chromosome 9 was identified in *Solanum cheesmaniae*, providing valuable genetic resources for breeding resistant cultivars.

Abstract

Powdery mildew, caused by *Oidium neolycopersici*, is one of the major fungal diseases affecting tomato production, particularly in greenhouse systems. Although several resistance genes have been identified in wild tomato relatives, their use in breeding programs remains limited due to genetic barriers and linkage drag. This study aimed to identify new resistance loci in the wild species *Solanum cheesmaniae* closely related to the cultivated tomato *S. lycopersicum*. An F_2 mapping population was developed from a cross between the susceptible accession *S. lycopersicum* cv. Rose de Berne and the resistant accession *S. cheesmaniae* LA0421 to identify quantitative trait loci (QTLs) associated with resistance to *O. neolycopersici*. A QTL-seq analysis was performed using deep sequencing of DNA bulks from individuals with extreme phenotypes, resulting in the identification of one resistance QTL located on chromosome 9. In addition, linkage QTL mapping was performed on 143 F_2 individuals to validate the QTL location identified by QTL-seq, designed ***On-q9$^{l}_{che}$***. The QTL was confirmed on the long arm of chromosome 9, explaining 14.60% of the phenotypic variance. Furthermore, we identified several candidate genes potentially involved in

powdery mildew resistance conferred by *S. cheesmaniae*, including genes encoding an ETO1-like protein, a serine/threonine-protein kinase, a NIMA-related kinase 2, a kinase-interacting protein, a 3-ketoacyl-CoA synthase, and the transcription factors UNE12/bHLH59. Overall, this study identified a novel source of resistance to powdery mildew in *S. cheesmaniae*, providing promising perspectives for tomato resistance breeding.

Keywords: QTL-seq, *Oidium neolycopersici*, powdery mildew resistance, *Solanum cheesmaniae*

Acknowledgements

We are thankful to Lili-Rose Miroux, Esther Pelpoir, for genotyping and taking care of the plants for QTL validation, to the centre for vegetable germplasm (CRB-Leg) of GAFL for keeping the seeds available, to the staff of the experiment unit A2M "Arboriculture et Maraichage Méditerranéen", to the staff of the experimental platform PROPHYLE (Unité Pathologie Végétale). We are also grateful to Sonia Elbelt and Jacques Lagnel who helped us in bioinformatics for QTL-seq analysis. We acknowledge the Genoscope Sequencing Laboratory (CEA-IbFJ-Evry) for providing access to sequencing facilities. We thank the ANR 'CapZéroPhyto' project, which is part of the French Priority Research Program "Cultiver et Protéger Autrement" (grant number ANR-20-PCPA-0003) for their financial support to cover the salary of AS and fund the experimental work.

1. Introduction

Tomato (*Solanum lycopersicum L.*) is one of the most widely consumed vegetables in the world. From 2010 to 2024, its production increased by 20%, making it the most produced vegetable (FAO 2025). At the same time, the rapid growth of the global population is expected to drive a substantial increase in food demand, including for tomatoes (Godfray et al. 2010; Vidyarthi and Evans 2019). To meet this current and future demand, high-yielding varieties have been developed. Despite these efforts, yields often fall short of expectations, primarily due to losses caused by diseases. Over 200 species of pathogens and pests are responsible for crop failures, resulting in substantial economic losses (Bai and Lindhout 2007). Among the diseases, powdery mildew is a major concern for tomato production. It can cause substantial damage in both greenhouse and field environments, resulting in yield losses of over 50% (Qinggui Lian et al. 2022; Park et al. 2022), although total crop loss is rare.

On tomato the powdery mildew disease can be caused by the two ascomycete fungi: *Leveillula taurica* and *Oidium neolycopersici*. *Leveillula taurica* appears as white powdery spots on the lower side of the leaf, while the upper side shows light green or yellow spots (Massire et al. 2025). *Oidium neolycopersici* appears also as white powdery spots that can enlarge and coalesce to cover a large area of plant tissues, eventually becoming necrotic, but mainly on the upper surface of leaves (Bardin and Gullino 2020). It can infect tomatoes throughout their life cycle, from seedling to mature plant. Although the disease does not affect tomato fruits, subsequent symptoms can include rapid leaf senescence, wilting, decreased fruit size and reduced fruit quality (Kim et al. 2018). After being reported for the first time in the Netherlands in 1986 (Paternotte 1988) and in England in 1987 (Fletcher et al. 1988), it has since spread rapidly throughout the world and has become a major problem in Europe, particularly for greenhouse-grown tomato.

Tomato is classified in the *Lycopersicon* section of the genus *Solanum*. This section is segregated into two principal groups: pigmented-fruited species (red or yellow/orange fruits), and green-fruits species (Mohan et al. 2016). During domestication and selection, tomato has experienced significant genetic erosion, leading to a loss of genetic diversity (Faino et al. 2012; The 100 Tomato Genome Sequencing Consortium et al. 2014). This loss poses challenges in identifying effective disease resistance traits within cultivated varieties. To overcome these

limitations, research has turned to wild relatives, which, while valuable, are often phylogenetically distant, as the green-fruited species *S. habrochaites, S. peruvianum* or *S. neorickii*.

Among wild species, five dominant resistance genes, acting against *O. neolycopersici*, have been identified on chromosome 6: *Ol-1* from *S. habrochaites* G1.1560 (Van Der Beek et al. 1994), *Ol-3* from *S. habrochaites* G1.1290 (Huang et al. 2000b), *Ol-4* from *S. peruvianum* LA2172 (Bai 2004), *Ol-5* from *S. habrochaites* PI247087 (Bai et al. 2005), and *Ol-6*, whose precise origin remains unknown (Bai et al. 2005). A recessive resistance gene, *ol-2*, has been mapped on chromosome 4 in *S. lycopersicum* var. *cerasiforme* LA1230 (Ciccarese et al. 1998). This gene corresponds to a loss-of-function mutation in the *Slmlo1* gene (Kim et al. 2018). It has been indicated that *Ol-1* and *Ol-3* are in the same chromosomal region (Huang et al. 2000b). Additionally, three resistance QTLs (*Ol-qtl1*, *Ol-qtl2*, and *Ol-qtl3*) have been identified in *S. neorickii* G1.1601: *Ol-qtl1* on chromosome 6, overlapping the region containing *Ol-1*, *Ol-3*, and *Ol-5* (Bai et al. 2003; Faino et al. 2012), and *Ol-qtl2* and *Ol-qtl3* are on the short and long arm of chromosome 12, respectively. Another QTL was found on chromosome 9 in *S. habrochaites* LYC4 (Kissoudis et al. 2015). The *Ol-4* and *Ol-6* genes confer complete resistance by triggering a rapid localized response that halts fungal development at an early stage (Bai et al. 2005; Li et al. 2012; Seifi et al. 2012, 2014). In contrast, *Ol-1*, *Ol-3*, and *Ol-5* confer slow and partial resistance (Seifi et al. 2012). Only the *ol-2* gene originates from a sub-species closely related to the cultivated tomato, namely *S. lycopersicum* var. *cerasiforme*, while all other resistance genes are derived from wild, green-fruited species. The introgression of these genes often causes reduced hybrid fertility and the introgression of genetic drag (Lin et al. 2014; Huang et al. 2022), complicating their use in breeding programs and highlighting the need to explore new resistance sources from closer related species.

QTL identification by linkage mapping approaches, requires genotyping large populations with variable phenotypes for the trait of interest. Michelmore et al. (1991), introduced Bulk Segregant Analysis (BSA) as an alternative approach for major gene detection. This method compares two groups of individuals with contrasting phenotypes. DNA from individuals belonging to each phenotyping group is combined to generate two bulked samples. These bulks were then genotyped using techniques such as Restriction Fragment Length Polymorphism (RFLP) or Random Amplified Polymorphic DNA (RAPD) to identify genomic regions associated with trait variation. More recently, with advances in molecular biology and sequencing technologies, Takagi et al.

(2013), developed the QTL-seq method, which combines BSA with whole-genome resequencing, enabling rapid identification of loci associated with traits in biparental populations. This approach, requires sequencing of only four samples: the two parents and two bulks, each comprising 10-20% of progeny individuals with contrasting phenotypes (Magwene et al. 2011; Takagi et al. 2013). Although powerful, QTL-seq requires precise phenotyping of a large population (300-500 individuals) to ensure robust results (Netpakdee et al. 2022). Despite this requirement, it offers significant advantages in cost efficiency and time savings (Yang et al. 2021). The QTL-seq approach has been successfully applied to various crops, including tomato (Illa-Berenguer et al. 2015; Sullenberger and Foolad 2024), pepper (Park et al. 2019), rice (Yang et al. 2021), cucumber (Lu et al. 2014), and kale (Zhang et al. 2020).

The aim of this study was to apply the QTL-seq approach to identify novel powdery mildew resistance loci in a wild tomato species closely related to the cultivated tomato. To this end, 31 tomato accessions were screened for resistance to *O. neolycopersici*. Among them, the accession LA0421, belonging to the closely related wild species *S. cheesmaniae*, was identified as resistant. This accession was subsequently used for QTL analysis to map new loci associated with powdery mildew resistance.

2. Materials and methods

Plant material and growth conditions

Two sets of plant were used in this study. The first set consisted of 31 tomato accessions representing six species: *S. lycopersicum* var. *lycopersicum*, *S. lycopersicum* var. *cerasiforme*, *S. habrochaites*, *S. pennellii*, *S. galapagense*, and *S. cheesmaniae*. Most of them have been previously used and characterized in another study (Amegan et al. 2024). The list of the accessions is provided in **Table S1**. This panel was used to screen tomato accessions for resistance to *O. neolycopersici*. Seeds were produced by the Centre for Vegetable Germplasm (CRB-Leg) of the GAFL research unit (INRAE, Avignon, France).

The second set consisted of a biparental F_2 population, derived from an interspecific cross between the susceptible *S. lycopersicum* cv. 'Rose de Berne' (hereafter referred to as RdB) and the resistant accession *S. cheesmaniae* 'LA0421'. This cross was performed to generate F_1 and an F_2

progeny, named F2B_LA421. The seeds were produced in 2021 at INRAE-GAFL (Avignon, France).

All seeds were sown in pots containing seed-starting potting soil (K® substrates Easy Growing) and grown under uncontrolled greenhouse conditions. After germination, seedlings were transferred into 9 x 9 cm pots soil substrate TS 3 MB Argile (K® substrates).

Pathogen material and inoculation

A population of *O. neolycopersici*, collected on tomato plants in 2021 at Avignon (France), was maintained in a growth chamber on the cultivar 'Monalbo', under controlled conditions: 24°C during the day, 20°C at night, 75% relative humidity, and a 16-hour photoperiod. The fungus was transferred onto fresh tomato plants showing no symptoms of powdery mildew every two weeks in order to maintain and propagate it. The inoculum was prepared by washing conidial spores from freshly sporulating leaves of heavily infected 'Monalbo' plants in tap water and was used immediately. Conidia concentration was estimated using a Malassez counting chamber, and the suspension was adjusted to a final concentration of 2×10^3 conidia·mL^{-1} of *O. neolycopersici* for the growth-chamber trial and 1×10^4 conidia·mL^{-1} for the tunnel trials. Plants were inoculated with 15-20 mL of the conidial suspension per plant using a hand-held sprayer.

Screening thirty-one tomato accessions for resistance to *O. neolycopersici*

Thirty-one tomato accessions, including *S. lycopersicum* var. *lycopersicum* (2 accessions), *S. lycopersicum* var. *cerasiforme* (6), *S. habrochaites* (7), *S. pennellii* (3), *S. galapagense* (1) and *S. cheesmaniae* (11) (**Table S1**), were evaluated for their resistance to *O. neolycopersici*. Accession G1 1560 belonging to *S. habrochaites* was included as resistant controls, based on previous studies (Lindhout et al. 1993, 1994; Van Der Beek et al. 1994; Huang et al. 2000). Plants were artificially inoculated and placed under controlled conditions conducive to disease expression (21°C, >70% relative humidity, 14-hour photoperiod). Disease severity was evaluated 11 days after inoculation (dpi) as described by Zapletalová et al. (2023). In addition, leaf area was measured using ImageJ, and disease severity was expressed as the number of spots per cm². The experiment was repeated twice, and each trial included five plants per accession.

Disease assessment of F_2 plants

A total of 433 F_2 plants from the F2B_LA421 progeny, was evaluated. One-month-old plants were individually transferred into 17 cm diameter pots containing soil substrate TS 3 MB Argile (K substrates). They were then transplanted into three 128 m² tunnels located at the same

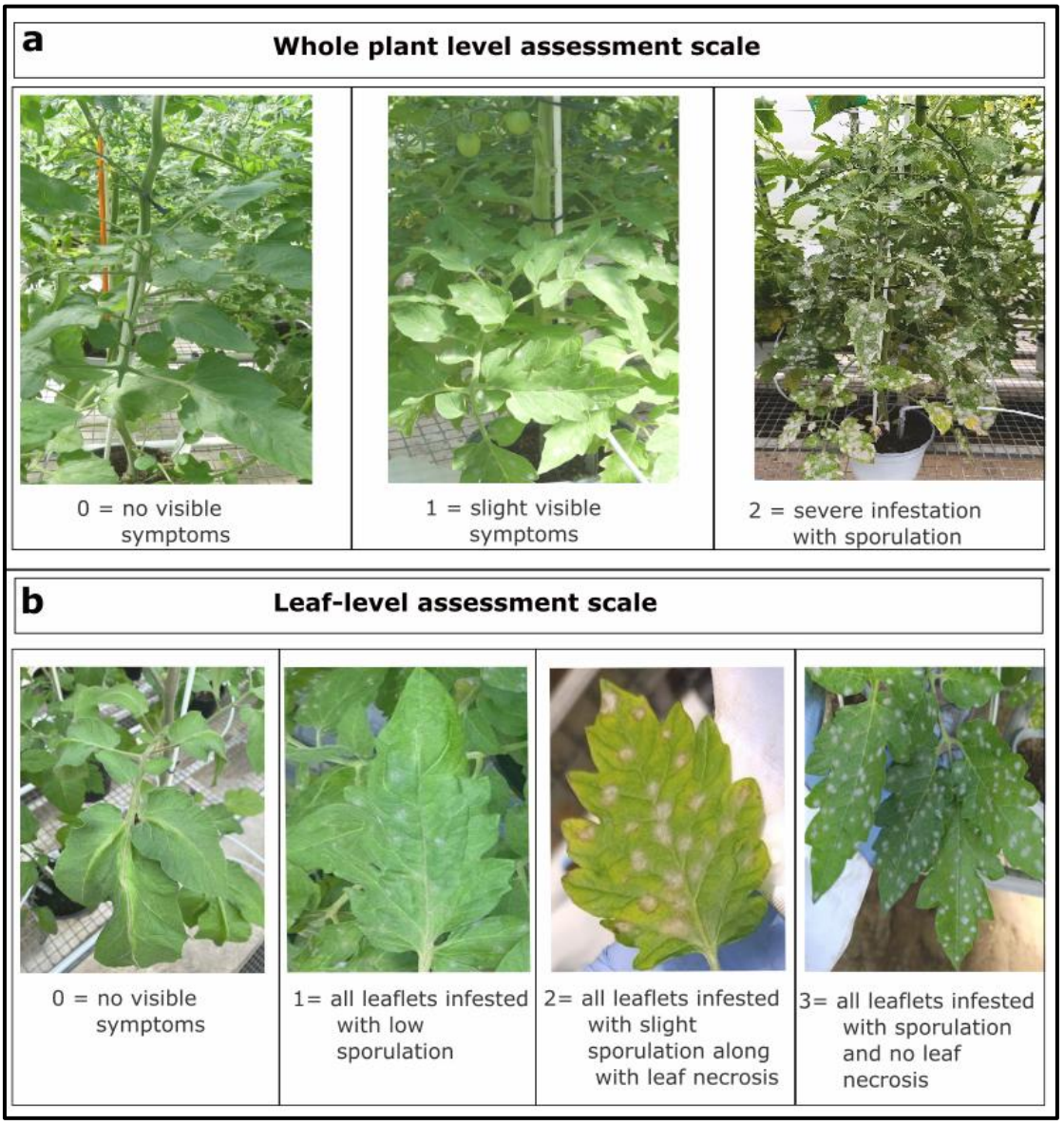


Fig. 1 Phenotypic assessment of *O. neolycopersici* sporulation on tomato, following 14 days of incubation at in spring tunnels conditions. Phenotyping was done at two levels. a) whole-plant assessment scaled from 0 to 2 with 0 = no visible symptoms, 1 = slight symptoms, and 2 = severe infestation with sporulation. b) leaf-level assessment**:** two leaves from the most heavily infested area of the plants were evaluated individually. Each leaf was scored from 0 to 3 with 0 = no visible symptoms, 1 = all leaflets infested with very low sporulation, 2 = all leaflets infested with slight sporulation along with necrosis where the powdery mildew develops, and 3 = all leaflets infested with sporulation and no leaf necrosis. The plant's final phenotype is the sum of its scores at the two different levels, as indicated by the "Disease index" formula: Disease index= whole plant score + leaf1 score + leaf2 score

experimental site in Avignon, France (43°56′N, 4°52′E). Each tunnel contained 6 plants of each parent, 3 plants from the F1 population, and 141-148 F2 plants. All experiments were conducted from early April to late June. Nine-week-old tomato plants were inoculated with a suspension of conidia. Disease symptoms were assessed 14 days after the inoculation (dpi). Phenotyping was performed at two levels. For the whole-plant assessment, symptoms were scored on a scale from 0 to 2 with 0 = no visible symptoms, 1 = slight symptoms, and 2 = severe infestation with sporulation (**Fig. 1a**). For the leaf-level assessment, two leaves from the most heavily infested area of the plants were evaluated individually. Each leaf was scored from 0 to 3 with 0 = no visible symptoms, 1 = all leaflets infested with very low sporulation, 2 = all leaflets infested with slight sporulation along with necrosis where the powdery mildew develops, and 3 = all leaflets infested with sporulation and no leaf necrosis (**Fig. 1b**). The scores from the whole plant and the two leaves were summed to obtain a disease index ranging from 0 to 8, where 0 represents no symptoms and 8 indicates severe infestation with abundant sporulation on leaves.

QTL-seq analysis

To follow the QTL-seq strategy, 176 F_2 plants with extreme phenotypes were selected among the 433 F_2, i.e. 20% of individuals at both ends: 88 resistant plants (disease index = 0) and 88 susceptible plants (disease index = 6 or 8). Three weeks after the first phenotyping, a second phenotyping was carried out on the 176 selected plants only, to confirm their phenotypes. This second phenotyping was used to select 44 individuals for each susceptible and resistant bulks (10% of the progeny) for sequencing.

DNA isolation was performed twice in two ways. DNA from plants included in the bulks was extracted using Qiagen DNeasy 96 Plant Kit. Extracted DNA was quantified with Qubit reagent to determine the concentration of extracted DNA for each individual. DNA quality was assessed using a Nanodrop spectrophotometer through the absorbance ratios at 260/280 nm and 260/230 nm. An agarose gel check was also performed to ensure sample integrity. Equimolar DNA quantity (200 ng) of the selected individuals were pooled to obtain one sample for susceptible bulk (S-bulk) and resistance bulk (R-bulk). For linkage QTL mapping, the DNA was extracted from the 143 F2 plants of tunnel 3 with a home-made extraction protocol adapted from Tai and Tanksley (1990).

DNA from the R and S bulks and from the parents were sequenced at whole genomes level by the Laboratoire Étude des Polymorphismes des Génomes Végétaux (EPGV, Evry, France), on a

MGI DNBSeq-G400 sequencer. QTL-seq analysis was performed on the whole genome sequences of R and S bulks with an in-house bioinformatic pipeline (https://forge.inrae.fr/gafl/pipelines_snakemake/easy_qtlseq/-/blob/master/README.md?ref_type=heads, developed by Institut National de Recherche pour l' Agriculture, l'Alimentation et Environnement, France). The R package QTLseqr developed by (Mansfeld and Grumet 2018) was used. The statistical approache QTL-seq (Takagi et al. 2013) was used for the analysis. Parents' reads were aligned to the reference genome SL4.0 (Hosmani et al. 2019) to identify polymorphisms. The SNP-index was calculated for each SNP in the S-bulk and R-bulk relative to the Heinz 1706 reference genome. The Δ(SNP-index) was then calculated to identify candidate genomic regions. Based on the null hypothesis, a 95% confidence interval was used to determine QTL for powdery mildew resistance.

Genetic map construction and QTL mapping

PACE markers (**Table S2**) were designed on polymorphic SNPs and genotyped using the protocol described in McClean et al. (2024). End-point fluorescence was read on a Varioscan plate reader (Thermo Fisher Scientific). When the three allelic forms (Fam-Fam, Fam-Hex and Hex-Hex were not enough separated, 10 more cycles of PCR were added: denaturation at 94°C for 15 s, annealing at 57°C for 1 min. DNA from both parents and from the F1 were used as controls. Genetic linkage map construction and QTL detection were done using the software QTL IciMapping 4.2 (Meng et al. 2015), with default parameters. QTL mapping was performed using the inclusive composite interval mapping (ICIM) method implemented in QTL IciMapping (Li et al. 2007; Meng et al. 2015).

Candidate genes

Based on the QTL detected using QTL IciMapping, genes located between the flanking markers were identified. Candidate genes within this interval were retrieved from the *S. lycopersicum* genome using the SL4.0 assembly and the International Tomato Annotation Group ITAG4.0 (Hosmani et al. 2019) available at the Sol Genomics Network (https://solgenomics.net/). The sequences of the flanking markers were aligned using BLAST against *S. cheesmaniae* genomes SCH_r1.0 (Iki et al. 2025) available at the Kazusa Genome Atlas (https://genome.kazusa.or.jp/). MUMmer v4 (Marçais et al. 2018) was used to generate a dot plot between SL4.0 and SCH_r1.0. Candidate genes identified in *S. cheesmaniae* were then compared with those annotated in the

SL4.0 reference genome. The genes related to disease resistance were then selected as candidate genes based on the gene function annotation information.

Statistical analysis

Statistical analysis of phenotypic data was carried out using Rstudio v4.5.0. Descriptive statistics and analysis of variance (ANOVA) were used to test: i) the difference between tunnels, ii) the difference in means between RdB, LA0421 and F_1 within and between tunnels. When the ANOVA test shows a significant difference, a post-hoc test was performed using Tukey's HSD test function. A significance threshold of P-value < 0.05 was used to assess pair-wise differences in disease score.

3. Results

Genetic variability in tomato resistances to *O. neolycopersici*

Tomato accessions exhibited a wide diversity in their levels of resistance to powdery mildew caused by *O. neolycopersici*, ranging from highly susceptible to strongly resistant lines (**Fig. 2**). For 2 of the 31 accessions tested, belonging to *S. habrochaites* (PI308182) and *S. galapagense* (LA1401), it was not possible to assess powdery mildew infection because the plants were completely defoliated due to the development of intumescences on the leaves under our controlled testing conditions. All accessions originating from the species *S. habrochaites* and *S. pennelli* exhibited a high level of resistance to *O. neolycopersici*. Among the accessions of the species *S. cheesmaniae*, a wide diversity is observed, including susceptible accessions and one accession exhibiting a high level of resistance (LA0421). This accession is a promising potential source of resistance and offers interesting opportunities for breeding programs, as *S. cheesmaniae* is more closely related to *S. lycopersicum* than *S. habrochaites* and *S. pennelli*. It was crossed with RdB cultivar (*S. lycopersicum*) to map resistance loci.

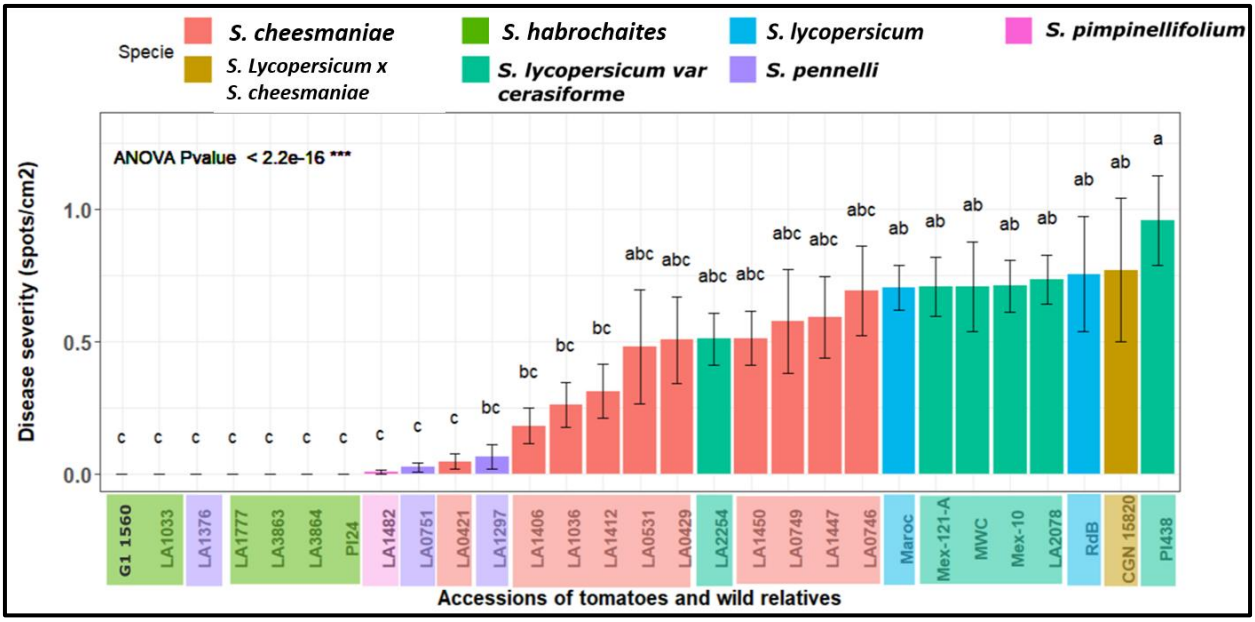


Fig. 2 Bar plot of the severity of the powdery mildew caused by O. neolycopersici (in spots/cm²) across 29 accessions of tomato and related wild species. The histogram shows the mean of two independent replicates (n = 10 plants). The bars represent the mean (n=10) with the associated standard error. Colors indicate the species. The red arrows show LA0421 and RdB, chosen for QTL mapping experiments

Disease evaluations in the interspecific F_2 progeny

The distribution of the disease index among all F_2 individuals (**Fig. 3a**) revealed a quantitative pattern, with a substantial proportion of F_2 plants exhibiting extreme phenotypes corresponding to scores of 0 and 8. The difference-in-means test between parents and F_1 showed a significant overall difference between RdB and LA0421, with respective disease means of 5.44 and 0.78 (**Table 1**). The mean of the F_1 was not significantly different from that of the susceptible parent RdB. The phenotypes of RdB and LA0421 genotypes are perfectly distinct in tunnel 3, unlike in tunnels 1 and 2. Spatial representation of the data, showed a spatial effect in tunnel 1 and 2, where plants planted above the row Y = 22 were more infected by powdery mildew (**Fig. S1a**). In contrast, tunnel 3 was less affected by the spatial effect.

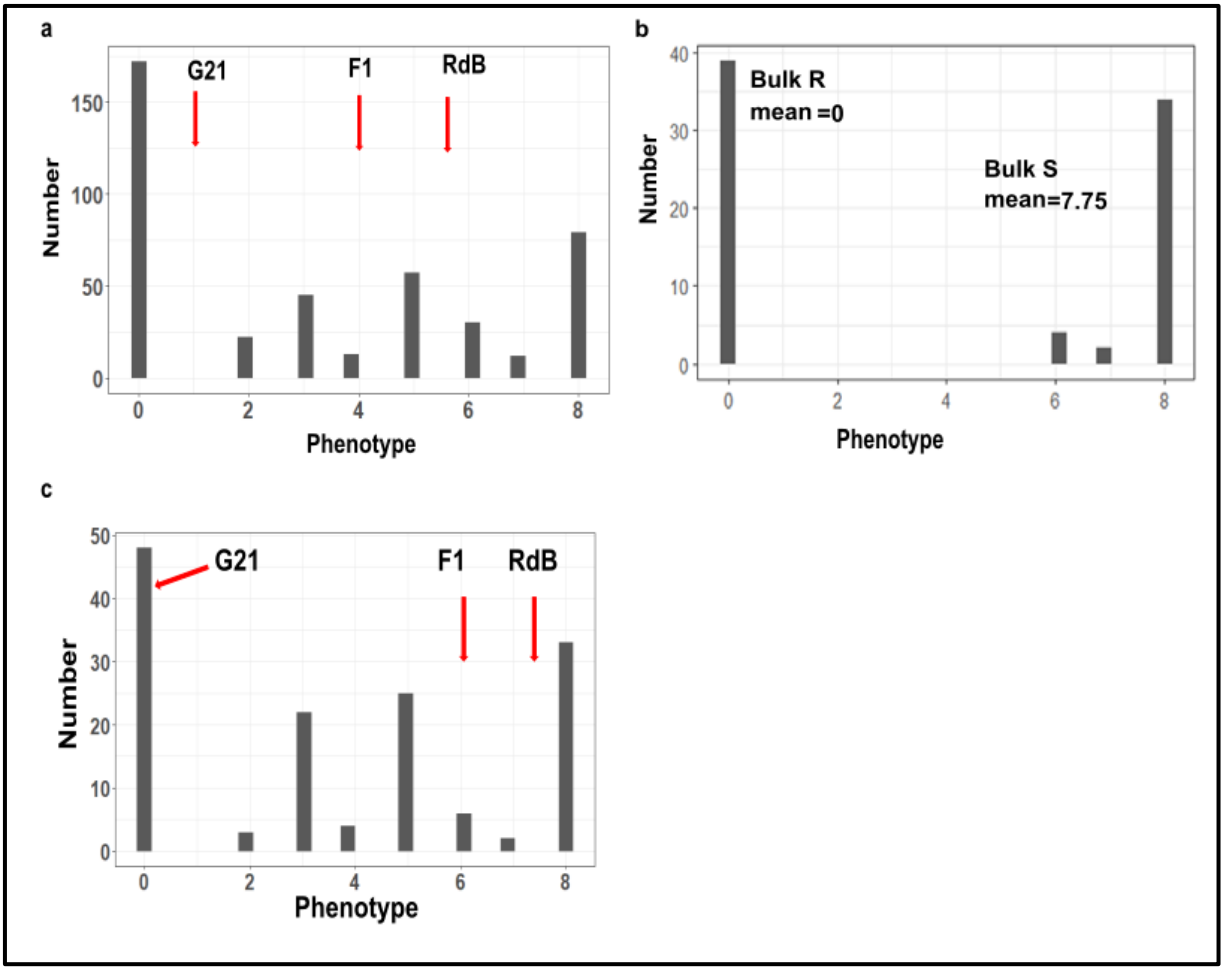


Fig. 3 Phenotypic distribution of F_2 disease index. a) distributions of the 433 F_2, b) distribution of F_2 individuals included in R and S bulks, c) distributions of the 143 F_2 in tunnel3. a,c) The red arrows indicate the level where the average disease index of LA0421, F_1 and RdB genotypes. F_1 plant phenotypes are close to RdB phenotype

Table 1 Average disease index for LA0421, RdB, F_1

	All tunnels		Tunnel 1		Tunnel 2		Tunnel 3	
	Number of plants	Mean ±SE	Number of plants	Mean ±SE	Number of plants	Mean ±SE	Number of plants	Mean ±SE
RdB	18	5.33 ±0.70 [a]	6	4.33 ±1.41 [a]	6	4.33 ±1.28 [a]	6	7.33 ±0.42 [a]
LA0421	18	0.78 ±0.44 [b]	6	1.50 ±1.02 [a]	6	0.83 ±0.83 [a]	6	0.00 ± 0 [b]
F1	9	3.78 ±1.05 [a]	3	2.67 ±2.67 [a]	3	2.67 ±1.45 [a]	3	6.00 ±0.58 [a]

The same letter indicates that the mean values belong to the same group. Different letters indicate significant differences between treatments (Pvalue < 0.05). There is no significant difference between RdB, LA0421 and F1 in tunnel 1 and 2

QTL-seq analysis detected a QTL on chromosome 9

The disease index of the F_2 plants included in bulks was 0 for resistant bulk and 6 to 8 for susceptible bulk (**Fig. 3b**). The susceptible bulk and resistant bulk samples were sequenced at

average depths of 70× and 92×, respectively. The parent LA0421 was sequenced at a depth of 13.9× and the parent RdB at a depth of 20×. The QTL-seq analysis detected a significant genomic region on chromosome 9 between 5 and 60 Mb at 5% tolerance threshold (**Fig. 4**).

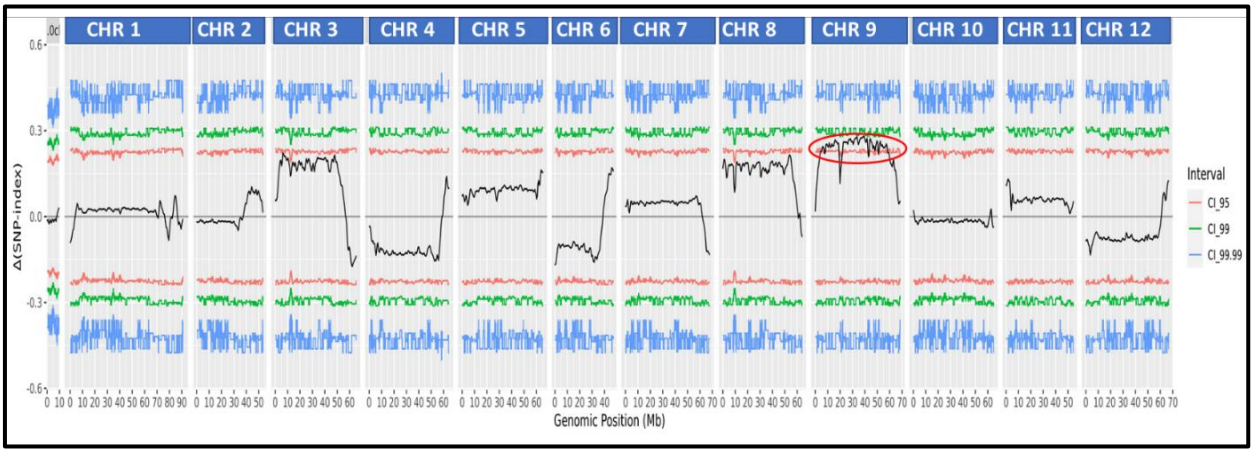


Fig. 4 QTL for powdery mildew resistance detected using the QTL-seq method of Takagi et al. (2013). The red, green and blue lines represent 95%, 99% and 99.99% Confidence Intervals, respectively. The black lines are the average Δ(SNP-index) and were drawn by sliding window analysis. The number on the horizontal coordinate represents the chromosome number. Δ(SNP-index) graph from QTL-seq analysis identified a QTL at the interval of 5-60 Mb on chromosome 9

Genetic map and determination of the confidence interval of the chromosome 9 QTL

To validate the QTL detected on the chromosome 9 by the QTL-seq method, we used the 143 F_2 individuals from tunnel 3 for linkage QTL mapping (**Fig. 3c**). This tunnel was less affected by the spatial effect than the two other ones (**Fig. S1**). The F_2 were genotyped with 20 PACE markers defined on chromosomes 9. All markers were grouped into one linkage group corresponding to the chromosome studied. The total size of the genetic map was 101.16 cM (**Fig. 5a**). The distance between markers ranges from 1.47 to 16.19 cM, with an average genetic distance of 5.06 cM. The order of the markers is the same as their physical order on the chromosome. We didn't define markers in the pericentromeric region, which is known as non-recombinant (Sim et al. 2012). Both markers flanking this pericentromeric region, CH09-11409858_K539 and CH09_57011053_K689, are separated by less than 1 cM. QTL mapping was performed using inclusive composite interval mapping on the 143 F_2 individuals from tunnel 3. The QTL physically located on chromosome 9 by the QTL-seq method, was detected on chromosome 9, with a peak at 48 cM (**Fig. 5a**), explaining 14.60% of the phenotypic variance. The QTL exhibited a positive

additive effect (a = 1.47), indicating that the allele from RdB increased the disease index, whereas the allele from LA0241 decreased it (**Table 2**). The negative dominance effect (d = -0.73) suggested toward the LA0241 allele (**Table 2, Fig. 5b, 5c**).

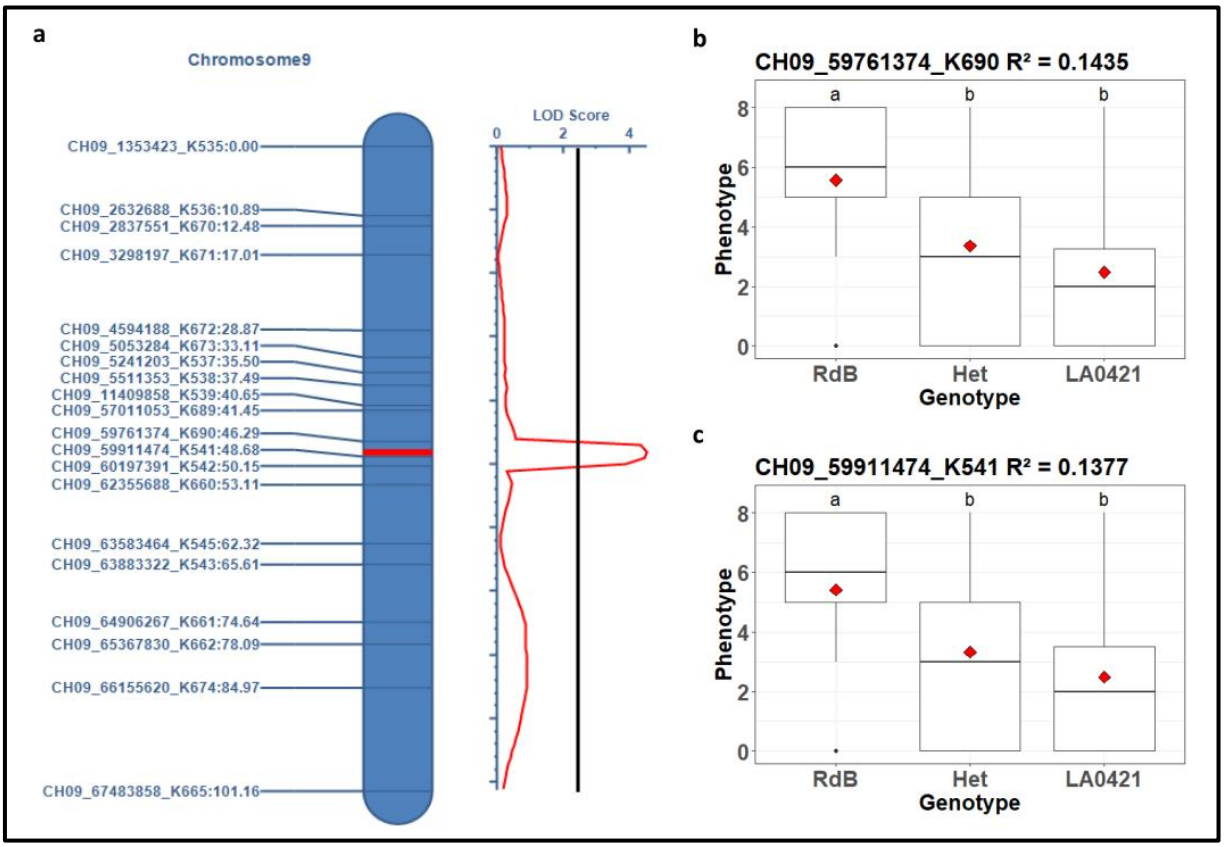


Fig. 5 Linkage analysis with molecular markers confirmed the location of the QTL of powdery mildew resistance on chromosome 9 of *S. cheesmaniae* LA0421. a) Left: Genetic map constructed with the 20 PACE markers defined on chromosome 9. The values to the left of the blue bars correspond to the primer names followed by their genetic positions in centimorgans (cM) (e.g., primer name: position). The primer names include their physical positions after the prefix "CH09_". Right: QTL detected using 143 F_2 individuals in tunnel 3, located at 48 cM, with the closest flanking markers CH09_59761374_K690 and CH09_59911474_K541. b, c) Phenotypic distribution across genotype classes at the flanking markers CH09_59761374_K690 and CH09_59911474_K541 respectively. RdB = RdB-like genotype, G21 = LA0421 -like genotype and Het = heterozygote-like genotype. Red points are phenotypic mean of the F2 plants carrying this genotype. The phenotypic variance explained by the markers are R^2=14.35% and R^2=13.77%, respectively. Letters above boxplot indicate whether the phenotypic mean of the genotypes are different or not.

Table 2 QTL associated with resistance to *Oidium neolycopersici*

QTL	Position	Markers	LOD	PVE	Additive	Dominance	Confidence interval
On-q9$^{l}_{che}$	48	CH09_59761374_K690[a] CH09_59911474_K541[b]	4.5	14.6	1.47	-0.73	46.5 - 50.5

[a] **left marker**
[b] **right marker**

Table 3 Relevant candidate genes within markers CH09_59761374_K690 and CH09_60197391_K542 on SL4.0 and SCH_r1.0 tomato reference genome

Start	End	Gene name	Description	strand
59780341	59789062	*Solyc09g065550.4.1*	Kinase interacting	+
59821112	59822220	*Solyc09g065565.1.1*	NIMA-related kinase 2	+
59824403 69918084	59826491 69922275	*Solyc09g065570.5.1* *SCH1.0ch09.g56380.1*	Serine/threonine-protein kinase Nek2	+
59893373 69988104	59898768 69992683	*Solyc09g065630.3.1* *SCH1.0ch09.g56430.1*	Protein TIFY 4B	+
59908607 70003959	59915336 70009502	*Solyc09g065640.3.1* *SCH1.0ch09.g56440.1*	Ethylene overproducer-like 1	+
59961196	59963018	Solyc09g150148.1.1	Non-specific lipid-transfer protein	+
60124430 70223903	60129781 70224762	*Solyc09g065750.3.1* *SCH1.0ch09.g56560.1*	Beta-carotene isomerase D27	-
60156869 70274140	60158438 70275334	*Solyc09g065780.3.1* *SCH1.0ch09.g56600.1*	3-ketoacyl-CoA synthase 11	-
60169347 70286764	60171149 70287996	*Solyc09g065800.4.1* *SCH1.0ch09.g56610.1*	3-ketoacyl-CoA synthase 11	-
60194165 70313297	60198712 70317324	*Solyc09g065820.4.1* *SCH1.0ch09.g56630.1*	bHLH transcription factor 59/ Transcription factor UNE12	+
Gene names started by “solyc” are genes name on SL4.0 reference genome Gene names started by “SCH1.0” are genes name on SCHR_r1.0 reference genome				

Candidate genes

The QTL is located in the 46.5-50.5 cM confidence interval. As there are no markers at these positions, the markers CH09_59761374_K690 and CH09_60197391_K542, were used to search for candidate genes. This interval corresponds to a 436 kb region on chromosome 9 of the tomato reference genome (SL4.0). The homologous regions ranged from 69,857,354 to 70,316,201 bp (459 kb) in *S. cheesmaniae* SCH_r1.0 genome. The dot plot showed that this region is relatively conserved between SL4.0 and SCH_r1.0 (**Fig. S2**). A total of 32 annotated genes were identified in SL4.0 and 25 in SCH_r1.0 (**Tables S3-S4**). Among these, eleven genes with annotated functions related to biotic stress and defense pathways, were considered particularly relevant. They include three receptors like kinase (RLK): kinase interacting; NIMA-related kinase 2; serine/threonine-protein kinase; two 3-ketoacyl-CoA synthases, an ETO1-like protein, a TIFY protein,

transcriptional factors (TF) such as UNE12 and bHLH59, and a gene encoding for specialized metabolite biosynthesis, beta-carotene isomerase D27(**Table 3**). Seven of these eleven candidate genes showed consistent annotations between SL4.0 and SCH_r1.0, whereas differences were observed only for the transcription factors. Research on Uniprot (https://www.uniprot.org), suggest that bHLH59 is another name of UNE12. It was also observed that the closest marker CH09_59911474_K541 was located in one ETO1-like gene exon.

4. Discussions

New tomato powdery mildew resistance QTL found in *Solanum cheesmaniae*

This study reports the first quantitative trait locus associated with tomato powdery mildew resistance identified in the wild species *S. cheesmaniae*, a species closely related to *S. lycopersicum*. The QTL, named *On-9$^{l}_{che}$*., is located between 46.5 and 50.5 cM from the top of chromosome 9, with a peak at 48 cM. This confidence interval lies close to markers CH09_59761374_K690 and CH09_60197391_K542 positioned at 59,761,374 bp and 60,197,391 bp, respectively, corresponding to a physical distance of 436 kb. This locus shows partial dominance, and explains 14.6% of the phenotypic variance observed in tunnel 3, accounts for about 40% of the difference between the parental lines.

Solanum cheesmaniae represents a valuable genetic resource for improving both abiotic and biotic stress tolerance as well as fruit yield traits in tomato. In particular, *S. cheesmaniae* has been reported as a source of quantitative loci contributing to salinity tolerance, where salt tolerance genes such as LeNHX3, HKT1;1, and HKT1;2 were identified (Villalta et al. 2008; Asins et al. 2013). It is also a valuable source of alleles increasing fruit number under unstressed conditions (Villalta et al. 2007) and improving yield under salinity through rootstock-mediated tolerance controlled by several moderate-effect QTLs (Estañ et al. 2009). In addition to abiotic stress tolerance, *S. cheesmaniae* has also been reported to confer resistance to the plant pests, including *Spodoptera exigua, Bemisia tabaci*, and *Tuta absoluta* (Rakha et al. 2017a, b; Vosman et al. 2018). Other studies have reported the presence of resistance QTL to *Alternaria alternata* and *Verticillium dahlia* in *S. cheesmaniae* (Diwan et al. 1999b; Robert et al. 2001). However, this species displays substantial variability in its responses to biotic stress, as illustrated in this study, in which accession LA0421 shows high resistance to powdery mildew whereas the other nine tested accessions were

less resistant. In addition, this accession has been reported to repel *Tuta absoluta* females (Amegan et al. 2024). Together, these results highlight the potential of *S. cheesmaniae* as a valuable source of genetic variation for improving tomato resistance to multiple stresses, although this potential must be considered with caution since biotic-stress responses vary markedly among accessions and require broader evaluation.

The QTL *On-9$^{l}_{che}$* is the second QTL conferring resistance to powdery mildew mapped on chromosome 9. The first QTL was identified at the beginning of the short arm of chromosome 9 (Kissoudis et al. 2015) in the species *S. habrochaites*, a green-fruited species distantly related. For introgression of resistance to powdery into cultivated tomato *On-9$^{l}_{che}$* may be preferred to avoid linkage drag. Actually, apart from the *ol-2* gene, all the other genes (*Ol-1*, *Ol-3*, *Ol-4*, *Ol-5*, and *Ol-6*) and QTLs already mapped, originate from green-fruited wild species distantly related to cultivated tomato, such as *S. habrochaites*, *S. neorickii*, *S. pennellii* and *S. peruvianum*, likely to carry a significant genetic burden (Huang et al. 2000b; Bai et al. 2003, 2005; Bai 2004; Park et al. 2022). The identification of *On-9$^{l}_{che}$* introduces a new source of resistance that broadens the diversity of resistance genes and QTLs available from a closely related species. Having access to multiple resistance genes or QTLs from tomato-related species would allow the development of cultivars with potentially more durable resistance than relying on a single gene. In addition, we designed PACE markers to map the QTL, therefore, these markers can be directly used in marker-assisted selection (MAS) in breeding programs.

The chromosome 9 of tomato is already known to harbor several other resistance loci against various pathogens and pests. QTL conferring resistance to *A. alternata* and *V. dahlia* was located on chromosome 9 of *S. cheesmaniae* (Diwan et al. 1999b; Robert et al. 2001). A QTL named *TIVd9*, identified in *S. pimpinellifolium*, controls the density of type IV glandular trichomes, which are involved in insect resistance (Mata-Nicolás et al. 2021). Partial resistance QTLs to anthracnose (*Colletotrichum coccodes*) have also been located on this chromosome (Lopez-Ortiz et al. 2023) in the accession PI 272636 (*S. lycopersicum* var. *cerasiforme)*. The major gene *Ph-3*, conferring resistance to late blight (*Phytophthora infestans*), has been cloned on chromosome 9 in the species *S. pimpinellifolium* (Zhang et al. 2014b). This gene is located at 67.5 Mb on the same chromosomal arm as the *On-9$^{l}_{che}$* QTL. These two loci are in a highly recombinant region (Sim et al. 2012) so it will be possible to select individual with resistance alleles at both loci. Pyramiding several resistance genes and QTLs is necessary as tomato face numerous pests and pathogens. Precise

genetic mapping and availability of linked molecular markers will help to introgress several of these resistance loci, located on the same chromosome, in a single tomato cultivar.

Candidate genes for powdery mildew resistance in *S. cheesmaniae*

In this study, we identified seven candidate genes. Among them, the gene Solyc09g065640.3.1/ SCH1.0ch09.g56440.1, encoding an ethylene overproducer-like 1 protein (ETO1), is annotated as involved in ethylene biosynthesis regulation. In *Arabidopsis thaliana*, recessive mutations in ETO1 result in a nearly tenfold overproduction of ethylene, highlighting its role as a negative regulator of ethylene levels (Wang et al. 2004). ETO1 protein family are components in the negative regulation of type 2 ACC synthases, which catalyze the rate-limiting step of ethylene production (Yoshida et al. 2005). Several studies have demonstrated the involvement of ethylene signaling in tomato resistance to *O. neolycopersici*. For instance, Satková et al. (2016) showed that BABA-induced resistance to powdery mildew in tomato depends on the ethylene signaling pathway. Moreover, the SNP closest to the QTL, CH09_59911474_K541, is located within this gene, further supporting its candidacy. Altogether, these results suggest that this gene represents a strong candidate for functional validation. The serine/threonine-protein kinase NEK2, NIMA-related kinase 2 and kinase interacting, may also represent candidate genes, as protein kinases are key components of plant immune signaling pathways, although their specific roles in disease resistance remains to be elucidated. Transcription factor UNE12 is a regulator of defense response (Mukhtar et al. 2011). Studies on *Arabidopsis thaliana*, demonstrated that the gene *AtUNE12* is involved in the regulation of salicylic acid mediated signaling pathway and the response to abscisic acid stimulus (Bruessow et al. 2021; He et al. 2022). The *Arabidopsis* Beta-carotene D27-LIKE1, a part of terpenoid chemical class, contributes to strigolactone biosynthesis and negatively impacts ABA level (Yang et al. 2023b). Although less well known than terpenoids, strigolactones hormones also play a specific role in plant defenses against pathogens (Stes et al. 2015; Marzec 2016). It has been demonstrated that a strigolactone-deficient tomato mutant was susceptible to the foliar fungal pathogens *Botrytis cinerea* and *Alternaria alternata* (Torres-Vera et al. 2014).

New nomenclature for genes and QTLs conferring resistance to *O neolycopersici*

We decided to name this QTL as *On-9$^{l}_{che}$*. The current nomenclature of tomato powdery mildew resistance genes and QTL provides limited information about their genomic location and species of origin. To improve clarity and informativeness, we propose a revised nomenclature based on the

system described by Caromel et al. (2005), with several modifications (**Table 4**). In this system, the name includes the pathogen genus and species, the chromosome number of the plant, the chromosome arm indicated in superscript ('s' for short and 'l' for long), and the species of origin of the resistant allele indicated in subscript. To avoid ambiguity among wild tomato species with similar abbreviations, the species of origin is indicated by the first three letters of the species name (e.g., lycc for *S. lycopersicum* var. *cerasiforme*, pim for *S. pimpinellifolium*, che for *S. cheesmaniae*, hab for *S. habrochaites*). Previously described tomato powdery mildew resistance genes and QTL were named using the prefix "Ol". Because resistance is associated with *O. neolycopersici*, we propose replacing the prefix "Ol" with "On". Recessive genes remain written in lowercase. Following this nomenclature, the QTL identified in this study was named *On-9$^{l}_{che}$*. A number must be added before the locus type when, within the same species, several loci are identified on the same chromosomal arm. For example, *Ol-1* and *Ol-3* located on the long arm of chromosome 6 in *S. habrochaites* will be written as: *On-6$^{l}_{hab}$1* and *On-6$^{l}_{hab}$2* respectively.

Table 4 Names of genes and QTL mapped for resistance to *O. neolycopersici* in tomato

New name	Originate name	Type	Dominance	Species	Chromosome	Arm	References
On-6$^{l}_{hab}$1	*Ol-1*	major gene	dominant	*S. habrochaites*	6	long	Van der Beek et al. 1994
on-4$^{l}_{lycc}$	*ol-2*	major gene	recessive	*S. lycopersicum* var. *cerasiforme*	4	long	Ciccarese et al. 1998
On-6$^{l}_{hab}$2	*Ol-3*	major gene	dominant	*S. habrochaites*	6	long	Huang et al. 2000
On-6$^{s}_{per}$	*Ol-4*	major gene	dominant	*S. peruvianum*	6	short	Bai, 2004
On-6$^{l}_{hab}$3	*Ol-5*	major gene	dominant	*S. habrochaites*	6	long	Bai et al. 2005
On-6^{s}-	*Ol-6*	major gene	dominant	Unknown	6	short	Bai et al. 2005
On-6$^{l}_{neo}$	*Ol-qtl1*	qtl	dominant	*S. neorickii*	6	long	Bai et al. 2003
On-12$^{s}_{neo}$	*Ol-qtl2*	qtl	dominant	*S. neorickii*	12	short	Bai et al. 2003
On-12$^{i}_{neo}$	*Ol-qtl3*	qtl	dominant	*S. neorickii*	12	long	Bai et al. 2003
On-9$^{s}_{hab}$	-	qtl	-	*S. habrochaites*	9	short	Kissoudis et al. 2015
On-9$^{l}_{che}$	-	qtl	dominant	*S. cheesmaniae*	9	long	This study

5. Conclusion

This study identified a new resistance QTL in *S. cheesmaniae*, a species phylogenetically closely related to the cultivated tomato, representing a new source of resistance to powdery mildew caused by *O. neolycopersici*. Seven key candidate genes were identified within the confidence interval of this QTL. Future researches could explore the specific regulatory mechanisms of these genes, as well as their potential used in breeding programs aimed at improving resistance to this disease. The close relationship between *S. cheesmaniae* and the cultivated tomato, along with the availability of KASP markers should make easy to incorporate this QTL into breeding programs, to introgress it, either alone or in combination with other resistance genes, into elite cultivars. This will increase the diversity of resistance genes deployed in tomato cultivars, thereby providing more durable protection against powdery mildew.

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Statements & Declarations

Funding

This work was supported by the 'CapZéroPhyto' project of the French Priority Research Programme "Cultiver et Protéger Autrement" project, the ANR (grant number ANR-20-PCPA-0003).

Competing Interests

There is no conflict of interest in this research.

Author Contributions

A.S. was the principal investigator (PI) for the project. A.S., B.C. and M.B. conceptualized and designed the study. A.S., J.F.B. and M.B. were involved in the phenotyping, A.S. wrote the draft of the manuscript, performed genomic and statistical analysis. A.S. and B.C. performed DNA isolation and quantification for whole genome sequencing. A.B. and D.H. performed whole genome sequencing. A.M. and B.C. perform crossing, M.B. and B.C. supervised the work and revised the manuscript critically for important intellectual content. All authors read and approved the final manuscript.

Data availability

The datasets presented in this study are available in Tables, Figures, Supplementary Tables, and Supplementary Figures. The genotypic and phenotypic data are available https://entrepot.recherche.data.gouv.fr/dataset.xhtml?persistentId=doi:10.57745/ZP3WH2.

SUPPORTING INFORMATION

Table S1 list of the accessions used to screen tomato powdery mildew resistance

Whole name	Name on Fig. 2	Species
MarocFavet	Maroc	*S. lycopersicum var. lycopersicum*
Rose de Berne	RdB	*S. lycopersicum var. lycopersicum*
LA1033	LA1033	*S. habrochaites*
LA3863	LA3863	*S. habrochaites*
LA3864	LA3864	*S. habrochaites*
LA1777	LA1777	*S. habrochaites*
PI247087	PI24	*S. habrochaites*
PI308182	-	*S. habrochaites*
G1 1560	G1560	*S. habrochaites*
Mex-10	Mex-10	*S. lycopersicum var. cerasiforme*
Mex-121-A	Mex-121-A	*S. lycopersicum var. cerasiforme*
Matt"s Wild Cherry	MWC	*S. lycopersicum var. cerasiforme*
LA2078	LA2078	*S. lycopersicum var. cerasiforme*
PI438556	PI438	*S. lycopersicum var. cerasiforme*
LA2254	LA2254	*S. lycopersicum var. cerasiforme*
LA1376	LA1376	*S. pennellii*
LA1297	LA1297	*S. pennellii*
LA0751	LA0751	*S. pennellii*
CGN 15820	CGN 15820	*S. cheesmaniae x S. lycopersicum*
LA0421	LA0421	*S. cheesmaniae*
LA0429	LA0429	*S. cheesmaniae*
LA0531	LA0531	*S. cheesmaniae*
LA0746	LA0746	*S. cheesmaniae*
LA0749	LA0749	*S. cheesmaniae*
LA1036	LA1036	*S. cheesmaniae*
LA1406	LA1406	*S. cheesmaniae*
LA1412	LA1412	*S. cheesmaniae*
LA1447	LA1447	*S. cheesmaniae*
LA1450	LA1450	*S. cheesmaniae*
LA1401	-	*S. galapagense*
LA1482	LA1482	*S. lycopersicum var. cerasiforme*

Table S2 Pace Marker lists with their physical positions based on tomato Heinz genome assembly SL4.0 and their sequences

Marker name	Position	Sens	Sequence marker A1 (RdB)	Sequence marker A2 (G21)	Sequence marker common
CH09_1353423_K535	1 353 423	REV	GACGAACCTGATCTGCTCCAAAAT	GACGAACCTGATCTGCTCCAAAAC	GGCATGTTTGAGACTAATTCCACAA
CH09_2632688_K536	2 632 688	REV	GTTTTGGCCTAAGCATTTCTCCGC	TTTTGGCCTAAGCATTTCTCCGT	AACATTGGTGCTCTGACTAATCTGG
CH09_2837551_K670	2 837 551	FOR	TAAAGTGAGCATCATTTCTCAG	GTCCTAAAGTGAGCATCATTTCTCAA	CACCGAACATCTAGCAATTCTTTTA
CH09_3298197_K671	3 298 197	REV	TGGTTTCGAATAATTGTTTTCACCAA	TGGTTTCGAATAATTGTTTTCACCAT	GTTAGGCAAGAAACCTTCATTTCTCA
CH09_4594188_K672	4 594 188	REV	CCAAAACCTCCTACAAGGATGAGC	CCAAAACCTCCTACAAGGATGAGT	CAGGCAGCCTATTGGTTTTCATC
CH09_5053284_K673	5 053 284	REV	AATCAGAAAATCCCGCCCTCATT	AATCAGAAAATCCCGCCCTCATC	AGAACATCTTTTGATGAGCAACCAG
CH09_5241203_K537	5 241 203	FOR	GACTTGCATAAGGTGGTTCAATACA	GACTTGCATAAGGTGGTTCAATACG	ATTAATGCCTCATGCCTGTCTAGG
CH09_5511353_K538	5 511 353	FOR	TACACCAACTCTTAAGGACACTAGA	TACACCAACTCTTAAGGACACTAGG	GAAATGACTCGGGAGTGCAACATTT
CH09_11409858_K539	11 409 858	FOR	CAAGGAAATACTTCCCTCGGT	CAACAAGGAAATACTTCCCTCGGC	TCTATCAGCCTCATCCAAAACTACA
CH09_57011053_K689	57 011 053	FOR	GATAAGCAACAAGTAGATTTGTTCATTA	AAGCAACAAGTAGATTTGTTCATTG	GCCCTTCCTCTTCTACTTGGT
CH09_59761374_K690	59 761 374	REV	TGCTGCTCTCTGCTCCTATAGA	TGCTGCTCTCTGCTCCTATAGT	ACCTCAAAGTGCTTCATTTTGGT
CH09_59911474_K541	59 911 474	FOR	GAGCTGTGAGATGGTTTTCTGATG	CGAGCTGTGAGATGGTTTTCTGATA	AGCAGAGTACATATGGATCCAAGAAA
CH09_60197391_K542	60 197 391	REV	ACTTGCAGCCGTAGGAATTTGAT	CTTGCAGCCGTAGGAATTTGAC	GTTGCAGACTGATAGAGCTGTAATG
CH09_62355688_K660	62 355 688	REV	AACCGTTCGATGTATTCTTGTTTTT	AACCGTTCGATGTATTCTTGTTTTC	TTGTGTTAATTGGGAATTGCAGAGG
CH09_63583464_K545	63 583 464	REV	GAAATGATTCCGAAGCGATCTCAG	GAAATGATTCCGAAGCGATCTCAA	TACTCATACGTCTAACCGACGATGT
CH09_63883322_K543	63 883 322	REV	GTACGGATGGACTAACACACCCCAA	GTACGGATGGACTAACACACCCCAG	ACTCTCATTTACCTGAAGGATCCAG
CH09_64906267_K661	64 906 267	REV	TCGATAAGAGTTTTATGGTGCTGTT	TCGATAAGAGTTTTATGGTGCTGTA	TCGATTGGACTGCTACATACTTCAA
CH09_65367830_K662	65 367 830	REV	CTATACCTGTCATTGGTGGGTGGC	CTATACCTGTCATTGGTGGGTGGT	TAGTCTAGCATGCAAACCAAGTGAA
CH09_66155620_K674	66 155 620	FOR	TGTGTTAGTGGTGAAGATTATGGTAA	TGTGTTAGTGGTGAAGATTATGGTAT	TTCATTAATGGTTGCACTTTCCTCA
CH09_67483858_K665	67 483 858	FOR	GGTCCGGTTGACCCTATAAAATGC	GGTCCGGTTGACCCTATAAAATGT	CTGTTCCACCGGCGATTCCAG

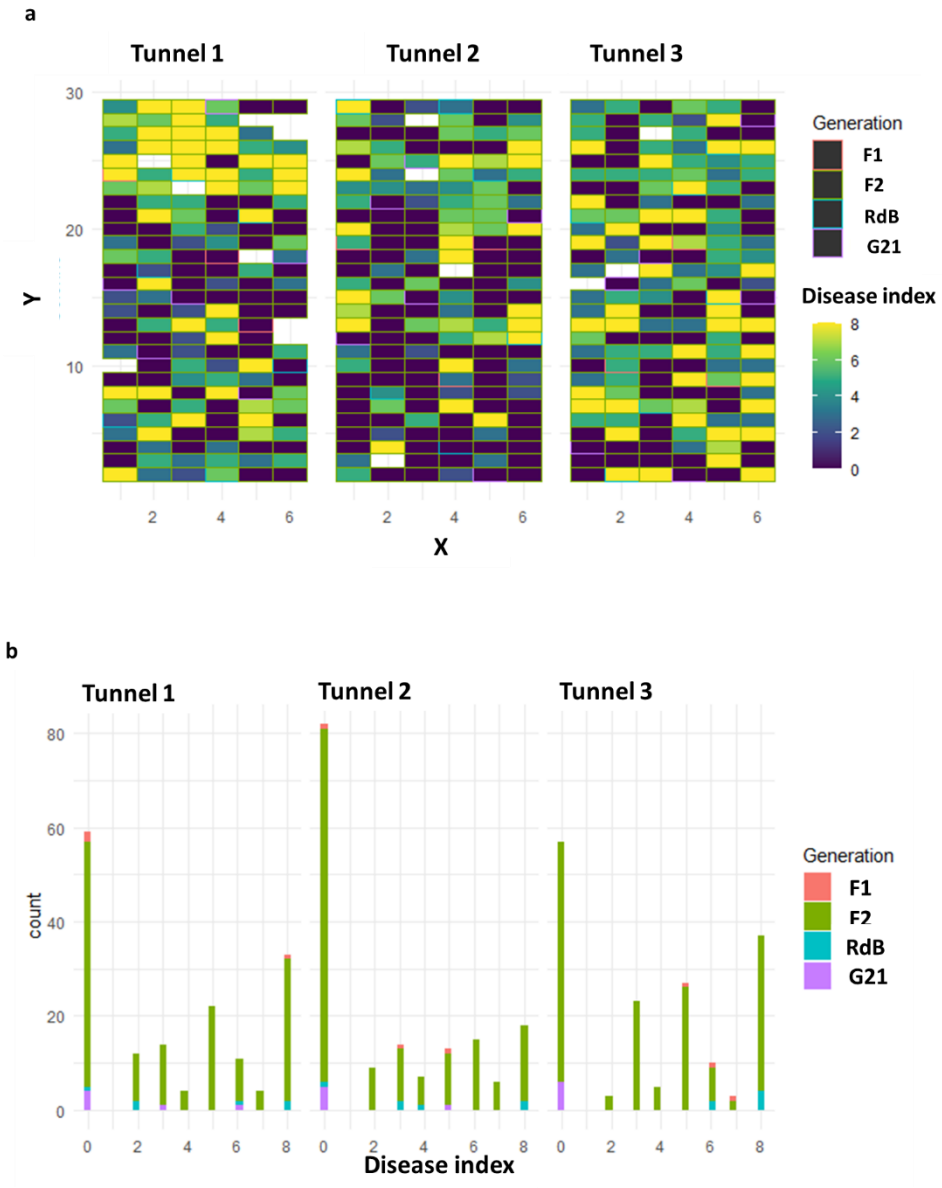


Fig. S1 Representation of total plant score. a) Disease index space in the three tunnels according to their X and Y positions. The color of the boxes corresponds to the values of the total score, ranging from black (total score =0) to yellow (total score =8). The color of the outline of the rectangles designates the nature of the plant: F_1 = orange, F_2 = green, RdB = blue and LA0421 = purple. b) Histogram of the total score of plants in the three tunnels. Scores on the abscissa and number of plants on the ordinate. The different colors represent the nature of the plant: F_1 = orange, F_2 = green, RdB = blue and LA0421 = purple

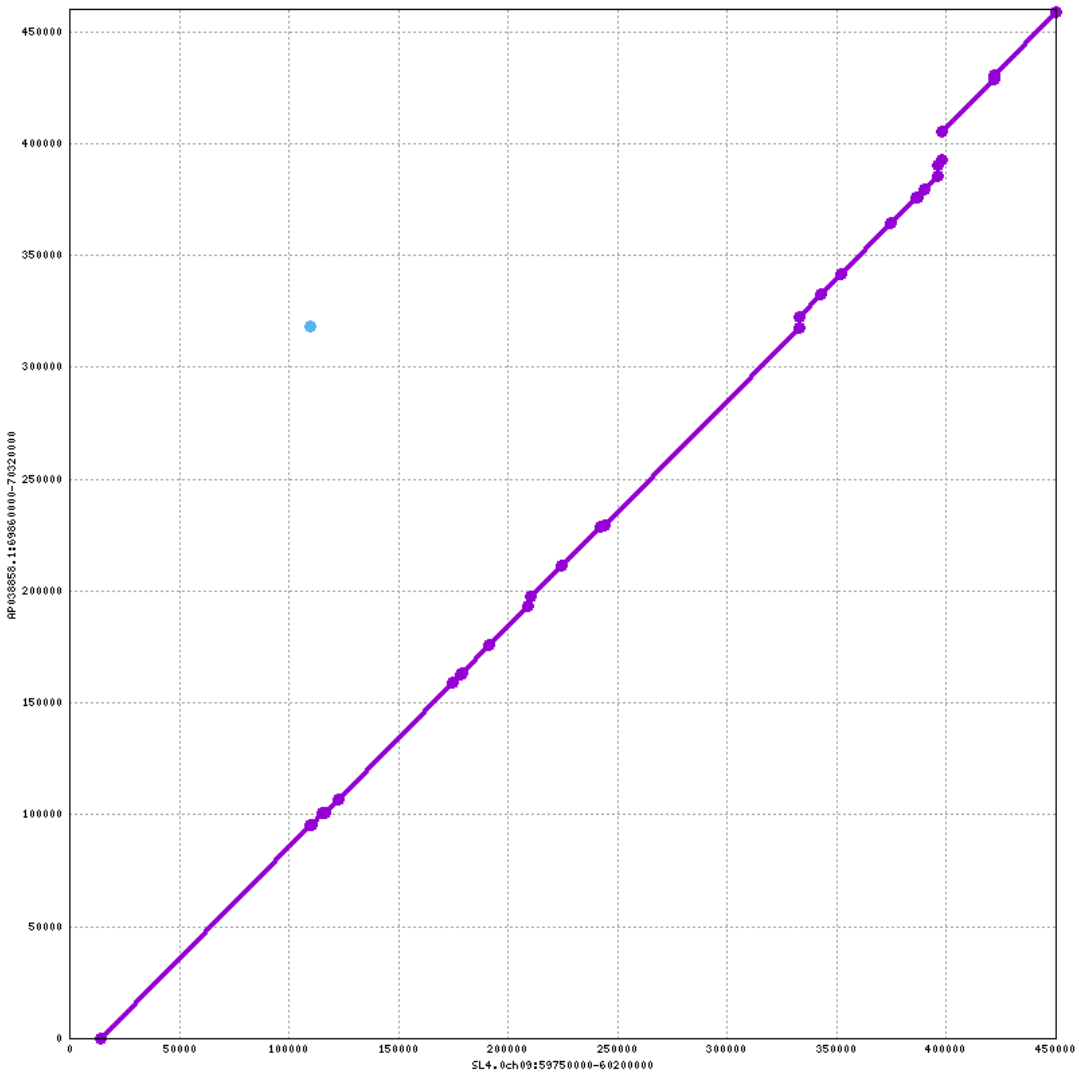


Fig. S2 Dot plot generated using MUMmer v4 to compare sequences of SL4.0 and SCH_r1.0 within the markers CH09_59761374_K690 and CH09_60197391_K542

Table S3 Genes annotated within markers CH09_59761374_K690 and CH09_60197391_K542 on SL4.0 tomato reference genome

Name	Start	End	Strand	Functions
Solyc09g065540.3.1	59756389	59770639	+	Biotin-binding protein
Solyc09g065550.4.1	59780341	59789062	+	Kinase interacting
Solyc09g065560.3.1	59789136	59797686	-	Sulfate transporter
Solyc09g065565.1.1	59821112	59822220	+	NIMA-related kinase 2
Solyc09g065570.5.1	59824403	59826491	+	Serine/threonine-protein kinase Nek2
Solyc09g065580.4.1	59826831	59829907	+	2-oxoglutarate (2OG) and Fe (II)-dependent
Solyc09g065590.3.1	59844028	59848450	+	Auxin canalization protein
Solyc09g065600.4.1	59848293	59856267	-	GDP-mannose transporter GONST4
Solyc09g065610.2.1	59858294	59858470	-	Bax inhibitor-1 family protein
Solyc12g049235.2.1	59868633	59869577	+	Zinc finger BED domain-containing protein
Solyc09g065620.3.1	59880955	59882889	+	Chlorophyllase
Solyc09g065630.3.1	59893373	59898768	+	Tify domain-containing protein/CCT-2 domain-containing protein
Solyc09g065640.3.1	59908607	59915336	+	Ethylene overproducer-like 1
Solyc09g065650.3.1	59921685	59946294	-	Protein phosphatase 2C family protein
Solyc09g150148.1.1	59961196	59963018	+	Non-specific lipid-transfer protein
Solyc09g065660.4.1	59975274	59977539	+	Heat shock transcription
Solyc09g065670.3.1	59999711	60004939	+	Unknown protein
Solyc09g065680.1.1	60014180	60014472	-	Unknown protein
Solyc09g065690.4.1	60014836	60027427	-	2-oxoglutarate (2OG) and Fe (II)-dependent oxygenase superfamily protein
Solyc09g065700.3.1	60040900	60061994	+	BEACH domain
Solyc09g065710.1.1	60063791	60066607	+	Pentatricopeptide repeat-containing protein
Solyc09g065720.4.1	60106675	60114638	+	Peptidyl-prolyl cis-trans isomerase

(Continued)

Name	Start	End	Strand	Functions
Solyc09g065730.3.1	60114946	60117780	-	2-methyl-6-phytyl-1 _Note2=4-hydroquinone methyltransferase_Note3=chloroplastic
Solyc09g065740.2.1	60120672	60123783	+	Ammonium transporter
Solyc09g065750.3.1	60124430	60129781	-	Beta-carotene isomerase D27 _Note2=chloroplastic-like
Solyc09g065755.1.1	60130818	60135349	-	Histone H2A
Solyc09g065760.4.1	60140807	60143746	+	DUF506 domain-containing protein
Solyc09g065770.1.1	60155430	60156482	+	Protein ELC-like
Solyc09g065780.3.1	60156869	60158438	-	3-ketoacyl-CoA synthase
Solyc09g065800.4.1	60169347	60171149	-	3-ketoacyl-CoA synthase
Solyc09g065810.3.1	60179149	60191006	+	Guanylate-binding protein
Solyc09g065820.4.1	60194165	60198712	+	bHLH transcription factor 59

Table S4 Genes annotated within markers CH09_59761374_K690 and CH09_60197391_K542 on SCH_r1.0 tomato reference genome

Name	Start	End	Strand	Functions
SCH1.0ch09.g56340.1	69852387	69864951	+	Methylcrotonoyl-CoA carboxylase subunit alpha _Note2=mitochondrial
SCH1.0ch09.g56360.1	69877103	69884697	+	Protein NETWORKED 1D
SCH1.0ch09.g56370.1	69885297	69893579	-	Sulfate transporter 3.1
SCH1.0ch09.g56380.1	69918084	69922275	+	Serine/threonine-protein kinase Nek2
SCH1.0ch09.g56390.1	69940163	69944265	+	VAN3-binding protein
SCH1.0ch09.g56400.1	69945009	69952242	-	GDP-fucose transporter 1
SCH1.0ch09.g56410.1	69954389	69957341	-	Protein LIFEGUARD 2
SCH1.0ch09.g56420.1	69976186	69977213	+	Chlorophyllase-1 _Note2=chloroplastic
SCH1.0ch09.g56430.1	69988104	69992683	+	Protein TIFY 4B
SCH1.0ch09.g56440.1	70003959	70009502	+	ETO1-like protein 1
SCH1.0ch09.g56460.1	70016855	70045562	-	Probable inactive protein kinase At3g63330
SCH1.0ch09.g56470.1	70072268	70074015	+	Heat stress transcription factor A-6b
SCH1.0ch09.g56480.1	70095245	70100090	+	Zinc finger protein MAGPIE
SCH1.0ch09.g56500.1	70115291	70122628	-	Lysine-specific demethylase JMJ31
SCH1.0ch09.g56510.1	70136175	70156738	+	Protein SPIRRIG
SCH1.0ch09.g56520.1	70159062	70161878	+	Pentatricopeptide repeat-containing protein At3g63370 _Note2=chloroplastic
SCH1.0ch09.g56530.1	70204099	70213852	+	Peptidyl-prolyl cis-trans isomerase CYP63
SCH1.0ch09.g56540.1	70214729	70217160	-	2-methyl-6-phytyl-1 _Note2=4-hydroquinone methyltransferase _Note3=chloroplastic
SCH1.0ch09.g56550.1	70220148	70223257	+	Ammonium transporter 2 member 5
SCH1.0ch09.g56560.1	70223903	70224762	-	Beta-carotene isomerase D27 _Note2=chloroplastic
SCH1.0ch09.g56570.1	70230639	70240157	-	Probable histone H2A variant 1
SCH1.0ch09.g56590.1	70272587	70273639	+	Protein ELC-like

(***Continued***)

Name	Start	End	Strand	Functions
SCH1.0ch09.g56610.1	70286764	70287996	-	3-ketoacyl-CoA synthase 11
SCH1.0ch09.g56620.1	70298198	70309504	+	Guanylate-binding protein 4
SCH1.0ch09.g56630.1	70313297	70317324	+	Transcription factor UNE12

Chapter 5

Supplementary study non-shown

In this chapter, we report additional work carried out during the thesis that is not presented in detail in the main manuscript and explain why they are not shown.

1. Comparative analysis of metabolites accumulated under plant resistance

During this thesis, we also collected samples to analyze the metabolites for which the production was induced after plant treatments with different PRIs, including Planticare®, Belvine®, Messager® and Tagatose®, both in the presence and absence of the pathogens *O. neolycopersici* and *P. infestans*, with the aim of identifying metabolites induced and correlated with resistance. Metabolites were extracted and analyzed by Liquid Chromatography-Mass Spectrometry (LC-MS), and data for Planticare® are already available. However, because the analysis could not be completed to the point of identifying the exact names of the induced and correlated metabolites, this part was not included in the manuscript. We plan to publish a data paper based on these metabolomic datasets.

2. Development of an amplicon sequencing panel

We developed an amplicon-sequencing panel consisting of 384 primer pairs that are polymorphic in at least three accessions from different tomato species, distributed across the genome in recombinant regions and spaced every 300–500 kb. Among these 384 primer pairs, 192 were validated on the accessions Rose de Berne and LA0421, as well as on an F_2 population derived from the Rose de Berne × LA0421 cross. We subsequently optimized a library-construction protocol adapted to these 192 primer pairs. The first validation of this protocol was performed using 384 samples from the Rose de Berne × LA0421 F_2 population, which enabled the construction of a genetic map for this population.

However, we observed a high proportion of missing data per primer pair and per individual. As a result, the genetic map was built using only the markers with sufficient data (approximately half of the initial set). This map was therefore not very dense and contained large gaps. To reduce these gaps, we decided to optimize the library-construction protocol by separating the primers into two pools:

- primer pairs with low amplification efficiency, amplified using 28 cycles during multiplex PCR;
- primer pairs with good amplification efficiency, amplified using 24 cycles.

We also hypothesized that the high rate of missing data could be due to the number of samples. Indeed, sequencing was performed on the Illumina iSeq100, which has a capacity of 1.2 Gb. Using 384 samples and 192 primer pairs drastically reduced sequencing depth. To address this issue, we reduced by half the number of plant samples used for validating the new protocol, from 384 to 192. Unfortunately, the sequencing run produced an occupancy below 25%, resulting in a large proportion of missing data that prevented genetic map construction and therefore did not allow us to determine whether 192 markers were sufficient to saturate the genetic map. The investigation to identify the causes of this result is still ongoing. For this reason, the chapter initially planned for this work was removed from the manuscript.

3. QTL analysis of *Botrytis cinerea* resistance in *Solanum cheesmaniae*

We also phenotyped an F_2 population derived from the Rose de Berne × LA0421 cross to map resistance loci to *Botrytis cinerea* and to analyze secondary metabolites differentiating resistant and susceptible plants. We intended to use the amplicon-sequencing panel to genotype this population. However, it was the sequencing of this population that failed, with occupancy rates consistently below 25%. So, we are now waiting for the issue to be resolved before proceeding further with the genetic analysis.

Chapter 6 General discussion and perspectives

General discussion and perspectives

The work carried out during this thesis made it possible, for the first time, to explore the genomic regions involved in the variation of tomato accession responses to the application of plant resistance inducers (PRIs) and to identify a novel powdery mildew resistance QTL in the wild species *Solanum cheesmaniae*, which is genetically closely related to cultivated tomato. The results obtained have provided insights into the mode of action of PRIs, the genetic determinants underlying tomato responses to their application, and the candidate genes potentially involved in the mechanisms of defense induction and resistance to powdery mildew and late blight. However, each PRI was investigated in interaction with only a single pathogen, and many questions remain regarding the nature and complexity of the interactions among the plant, the PRI, and the pathogen. This final chapter presents a critical assessment of the experimental and methodological choices, a comparative analysis of the results obtained with both PRIs, and a discussion of the significance of the main findings and the research perspectives arising from this work.

1. Critical assessment of the experimental and methodological choices

The synthesis of three years of research has highlighted several limitations in the experiments that were conducted. This paragraph provides a critical discussion of key methodological and strategic choices made both prior to and during the thesis. Recommendations for improving future experimental designs are also proposed.

Choice of phenotyping methods

Accurate phenotyping is essential for genetic research, as measurement errors significantly reduce statistical power (Tuberosa 2012; Cobb et al. 2013; Vukadinovic et al. 2023). Throughout this thesis, different phenotyping methods were used to quantify plant susceptibility to powdery mildew, including the counting of lesions per cm^2 and semi-quantitative scoring, as described in Chapters 3 and 4. Semi-quantitative scores provide a rapid assessment of overall susceptibility and were used as an alternative to lesion counts per cm^2, which constitute a more precise quantitative measure essential for QTL mapping and GWAS analyses. However, lesion counting was replaced because it is extremely time-consuming when applied to large plant populations.

For practical reasons, to count lesions, only one leaf per plant was evaluated in the GWAS experiment, which does not fully capture the overall infection status of the plant. Moreover, to ensure consistency across samples, leaves were collected at a fixed position (the 3rd or 4th leaf from the base). While this approach improves comparability, it also introduces potential biases, as infection patterns are not uniform across the canopy. Depending on the plant, the selected leaf may correspond either to the most susceptible or the least susceptible part of the plant, thereby generating variability and potential bias even among biological replicates.

Another source of bias associated with counting lesions per cm^2 is that this method focuses exclusively on lesion number, even though clear qualitative differences were observed among colonies. In our experiments, several distinct symptom types were recorded: large colonies, small colonies, white sporulation, grey sporulation (indicating low-density sporulation), white sporulation surrounded by necrosis, and fully necrotic spots without visible sporulation. Each of these phenotypic patterns may reflect different underlying defense mechanisms. Necrotic spots are typically associated with a hypersensitive response (HR), whereas small colonies or grey colonies may indicate a restriction of pathogen growth. These responses are consistent with the defense mechanisms described for major resistance genes to powdery mildew.

For example, *Ol-4* and *Ol-6* trigger an early hypersensitive response that rapidly arrests pathogen development (Bai et al. 2005). In contrast, *ol-2* confers a non-hypersensitive resistance characterized by callose deposition, which limits pathogen progression without inducing cell death (Bai et al. 2005). The qualitative variation observed in our phenotyping therefore suggests that plants may express different defense pathways, which are not captured when only lesion number per cm² is measured.

Future phenotyping efforts should incorporate qualitative traits such as lesion size, lesion color, and the extent of sporulating tissue. Quantifying sporulation area is particularly relevant but remains challenging with conventional image-analysis tools because spore and leaf colors vary widely, even more when analyzing a core collection that is as representative as possible of the species' diversity. Developing an artificial intelligence-based program trained on diverse sporulation patterns and leaf backgrounds would greatly improve lesion segmentation, sporulation quantification, and lesion counting, with greater precision than manual scoring. Manual scoring relies heavily on evaluator subjectivity, which introduces bias and reduces the accuracy of heritability estimates, genetic value predictions, and QTL detection. Automated, standardized, and reproducible phenotyping approaches would help overcome these limitations and strengthen quantitative genetic analyses. The same approach could be applied to late blight phenotyping to quantify the pathogen-colonized area.

Plant responses to PRIs vary widely across studies and can be assessed through susceptibility under treated conditions, the difference or ratio between treated and untreated conditions, or the percentage of protection (Tamm et al. 2011; Wehner et al. 2019; Yuan et al. 2019; Põldmets et al. 2025; Zhang et al. 2026). Mapping studies rely either on susceptibility under treated conditions (Bénéjam et al. 2021; Thielmann et al. 2026) or on both treated susceptibility and the PRI response calculated as a difference (Soleimani et al. 2024). However, these traits do not capture the same biological meaning. A simple difference between control and treated severity does not reflect true product efficacy, as accessions with identical differences may show very different levels of protection. For example, three accessions may all show a difference of 2, yet one decreases from 2 to 0 (100% protection), another from 4 to 2 (50% reduction), and a third from 6 to 4 (33% reduction).

In this thesis, PRI efficacy was defined as the percentage of protection, which better represents the plant's response to treatment. For GWAS, both percentage of protection and disease severity

under treated conditions were used to identify specific loci. This dual approach helps distinguish loci involved in disease reduction from those affecting plant responses under treatment.

Overall, these considerations highlight the need for biologically meaningful and statistically robust phenotyping criteria to improve genetic analyses and our understanding of plant responses to PRIs.

Choice of experimental design and methodology

Throughout this thesis, the number of biological replicates per accession had to be limited due to constraints in space, material preparation, and scoring time, especially given the large number of accessions and factor combinations (accession × treatment). In the GWAS experiments, only two biological replicates per accession × treatment was used within a single environment, which inevitably reduces the robustness of mean effect estimates.

A similar limitation was observed during the powdery mildew phenotyping of the F2 population, where 459 F2 plants were distributed across three 128 m² tunnels. Although these tunnels were located side by side, substantial differences were detected among them, including strong spatial effects in some tunnels that ultimately prevented the use of their phenotypic data.

Increasing the number of biological replicates per genotype and per condition remains the most effective strategy to improve the accuracy of mean estimates. It also facilitates the correction of spatial phenotyping biases using dedicated statistical tools such as the R packages SpATS or StatgenHTP (Rodríguez-Álvarez et al. 2018; Millet et al. 2021).

Beyond replication, the choice of treatment number and timing is equally critical for accurately estimating PRI efficacy and comparing results across environments. Most studies report effective protection when PRIs are applied 1–3 days before inoculation, or repeatedly between 10 and 2 days before inoculation (Warneys et al. 2018; Le Clerc et al. 2025; Detcheverry et al. 2026). In our study, two treatments were applied 7–8 days and 3 days before inoculation. Due to climatic constraints, seed germination dynamics, and scoring schedules, plants could not be treated at the same developmental stage, which may have contributed to variability in PRI efficacy across environment and may affect the genomic regions detected for plant response to PRI.

GWAS is a powerful approach for investigating the genetic basis of complex traits. However, the results strongly depend on the statistical model used, the complexity of the trait, and the size and structure of the population. Linkage disequilibrium is a major limitation, as it

complicates the identification of true candidate genes. This issue is particularly pronounced in tomato, where more than half of the genome does not recombine (Sim et al. 2012; Jia et al. 2025). One way to mitigate this limitation is to complement GWAS with other omics approaches, such as metabolomics and transcriptomics. These approaches are especially relevant in our context, as PRIs are known to induce the expression of defense-related genes and the production of secondary metabolites. Moreover, transcriptomic analysis and metabolomic profiling can be used directly as phenotypes in GWAS, enabling the identification of genomic regions involved in the induction of plant defense responses.

Usually, GWAS relies on SNP markers. However, SNPs capture only a fraction of the genetic variation present within a panel. With recent advances in sequencing technologies, genome assemblies, and the availability of reference genomes for all tomato species, GWAS can now incorporate additional types of molecular variation, such as structural variants and k-mers. The K-mers markers have already proven useful for identifying resistance loci, for example for aphid resistance in melon and powdery mildew resistance in wheat (Jaegle et al. 2025; Belinchon-Moreno et al. 2026). Integrating these richer forms of variation would likely improve the resolution and biological relevance of GWAS in tomato.

Studying the mode of action of PRIs requires identifying the genes and metabolites induced after their application. Defense gene induction can be assessed by measuring the expression of specific marker genes such as PR-1 or NPR1 (nonexpressor of pathogenesis-related 1), which are classical indicators of salicylic acid pathway activation and systemic acquired resistance (Walters and Fountaine 2009; Baccelli and Mauch-Mani 2016; Berens et al. 2017; Mazuecos-Aguilera et al. 2025). Alternatively, a broader transcriptomic approach such as RNA sequencing (RNA-seq) can be used (Yuan et al. 2019; Zapletalová et al. 2023). In this study, we chose to use the qPFD tool (Patent WO/2011/161388), developed specifically to evaluate the defense-stimulating activity of biosolutions. This method was selected because it is inexpensive, rapid, and allows the assessment of a limited but representative set of 28 genes involved in major plant defense pathways. It enabled us to identify products that strongly induce defense gene expression in tomato.

However, for treatments that did not result in a strong induction of the defense-genes spotted on the qPFD chip, we cannot definitively conclude that they do not act by resistance induction.

The qPFD chip was originally developed using apple defense genes, and in our case, we relied on homologous tomato genes. We therefore cannot be certain that these genes play major

roles in tomato defense. Moreover, PRI-induced resistance can be highly specific, and it is likely that PRIs activate additional defense genes not represented on the chip. For example, ABA is a phytohormone well known to contribute to resistance (Baccelli and Mauch-Mani 2016; Berens et al. 2017), yet none of the genes on the qPFD list correspond to the ABA biosynthesis pathway. In addition, a single functional category may include multiple gene variants with distinct induction patterns. Satková et al. (2016) showed that among five PR-1 transcripts (P1, P2, P3, P4, and P6), P2 were not induced following application of BABA or oligandrin, whereas the others were induced depending on the treatment and the tomato accession. Interestingly, P2 was induced in the resistant accession after *O. neolycopersici* inoculation, even without PRI treatment. In addition, in our study, sampling was performed three days after the final treatment, which does not capture the temporal dynamics of gene expression. Moreover, the priming effect of the PRIs was not evaluated, as sampling occurred before inoculation. Future studies should include RNA-seq analysis, priming assays and multiple sampling time points before and after inoculation to better characterize the kinetics of defense responses.

Finally, an important experimental choice in this work is that all genetic trials were conducted under field-like conditions. This greatly strengthens the relevance and transferability of our results, as the identified QTLs and PRI effects were observed in an environment that closely mimics real production settings. Conducting experiments outside controlled growth chambers reduces the risk that QTL effects are artefacts of artificial conditions and increases confidence that the detected loci may remain effective when deployed in the field.

2. Comparison between Belvine® and Planticare®

Mode of action and efficacy

Both Belvine® and Planticare® induced the expression of the same defense-related genes, including *PR-1*, *PR-2*, *PR-4*, *PR-5*, *PR-8*, *POX*, *GST*, and *ACCO*. Previous experiments in 2021 and 2022 had also shown that both products were effective against powdery mildew and late blight. However, in the experiment of 2023, Planticare® did not reveal any protective effect against late blight. Consistent with these phenotypic observations, metabolomic analyses from the same experiment did not identify any differentially accumulated metabolites between Planticare®-treated plants and untreated controls, suggesting that Planticare® was not effective that year. These observations were not consistent with the expected biological activity of

Planticare® and suggest a possible experimental inconsistency in the 2023 trial, such as a handling or labeling error during product application. This hypothesis is further supported by the GWAS experiment results (see Chapter 3), which confirms that the batch of Planticare® used in subsequent assays remained biologically active.

In this context, the 2023 dataset was considered with caution and was not interpreted as evidence against the previously reported activity of Planticare®. However, the overall evidence remains consistent with earlier experiments demonstrating the efficacy of Planticare® against both powdery mildew and late blight.

Genomic regions involved in tomato accessions responses to PRI

This thesis identified several SNPs associated with the response of tomato accessions to the application of Belvine® and Planticare®. Among the genomic regions detected, chromosome 4 emerged as a notable focal point, as it contains SNPs significantly associated with the response to both PRIs. The genetic signals are clustered within relatively close intervals: 7.9 Mb, 11.3 Mb, and 31.3 Mb for Belvine® and 17.1 Mb, 22.5 Mb, and 45.9 Mb for Planticare®.

The recurrence of associations on the same chromosome and their distribution across several regions suggests that chromosome 4 may contain gene-rich areas potentially involved in the tomato response to PRIs. These results suggest that certain genomic regions could play a role in the response of tomato to PRIs, regardless of the product applied or the pathogen agent, although further experiments are required to confirm this hypothesis. Our observations are consistent with findings in wheat following the application of N-acyl homoserine lactone (AHL) against *Puccinia triticina* and the inoculation of *Serendipita indica* against *Blumeria graminis* f. sp. *tritici*, where the QTL QTL_PE_1 detected for AHL-priming efficacy and QTL2.Bgt_*S.indica* detected for *S.indica*-priming efficacy are located on the same chromosomal arm of chromosome 1B, less than 10 Mb apart (Soleimani et al. 2024; Thielmann et al. 2026). It is also possible that the similarity observed reflects the fact that both PRIs operate through similar mechanisms of action. Therefore, PRIs that induce different mechanisms could potentially reveal additional or distinct responses.

Another similarity between the results obtained with Belvine® and Planticare® is that, for both PRIs, the defense gene functions induced after treatment, such as peroxidases and β-1,3-glucanases, were also represented among the candidate genes located within the QTL intervals. This strengthens the relevance of these gene functions as potential markers of tomato responses to these PRIs.

This study did not investigate the genomic regions involved in tomato accession responses to the application of the same PRI against different pathogens, so it is not possible to say that the remaining QTLs detected are specific to the PRI or the pathogen inoculated. However, previous studies have provided evidence that specific genomic regions can be associated with plant responses depending on the pathogen and, more broadly, on the traits measured. Thielmann et al. (2026) reported in wheat the detection of different QTLs associated with shoot fresh weight, root fresh weight, and powdery mildew resistance under *Serendipita indica* priming conditions, suggesting that genomic regions associated with *Serendipita indica* effect depend on the traits measured. In apple, distinct genomic regions were associated with resistance to apple scab and to fire blight under ASM treatment (Bénéjam et al. 2021). In that study, PRI-associated loci largely overlapped with known resistance QTLs, suggesting that ASM responsiveness in apple is mainly driven by pathogen-specific resistance determinants rather than by loci dedicated to PRI response. This contrasts with our results, where the QTLs detected under Planticare® treatment do not coincide with genetic resistance loci, and most candidate genes are not classical R genes. This indicates that, in tomato, PRI-induced responses may rely on regulatory or metabolic pathways distinct from innate resistance and therefore may be less constrained by pathogen specificity.

Altogether, our results show that plant response to PRIs is a complex trait whose genetic architecture depends both on the PRI applied and, on the trait, used to quantify plant response. While some chromosomal regions appear to be commonly induced across treatments, others are PRI-specific, highlighting the multifaceted nature of induced resistance.

3. Implication of this study and perspectives

Plant response to PRI as a new target for breeding tomato accessions

This study is the first to report QTLs associated with tomato accession responses to PRI application. Altogether, our results provide new insights into the genetic architecture underlying PRI-induced responses and highlight several candidate genes potentially involved in induced resistance in tomato. Future studies could validate these candidate genes by combining gene expression analyses, targeted knockouts, and functional assays to clarify their contribution to PRI-induced defense pathways. Then, they could serve as molecular targets for marker-assisted selection or for validating PRI-induced resistance mechanisms in breeding lines. However, anonymous markers merely linked to the detected loci can already be used in breeding.

In addition, the detection of both stable and environment-specific QTLs provides breeders with complementary tools: stable loci can support PRI efficacy across a broad range of conditions, whereas environment-specific loci are useful in particular environmental contexts where specific environmental factors favor PRI performance. This is especially relevant in the context of climate change, where developing cultivars adapted to local conditions is becoming increasingly important. Integrating detailed environmental descriptors with genetic analyses will ultimately improve the precision of QTL interpretation and support the development of tomato varieties with more reliable and context-adapted responses to PRIs. Together, these findings demonstrate that PRI-related traits can be effectively integrated into breeding pipelines, paving the way for the development of tomato varieties with more reliable, durable, and context-adapted induced resistance.

In the short term, a multi-environment trial could be conducted with selected accessions, chosen according to their genotype at the significant SNPs, to validate the identified QTLs. Within the same trial, RNA-seq analyses and metabolite profiling could be performed to examine gene expression patterns and metabolic changes associated with PRI responsiveness. Such combined approaches would help highlight the genes and metabolites involved in tomato responses to PRIs and provide a deeper understanding of the molecular mechanisms underlying induced resistance.

In our GWAS analysis, all SNPs with a minor allele frequency (MAF) ≤ 10% were removed. While this filtering step reduces the risk of spurious associations, it also implies that some significant genomic regions carried by rare alleles may have gone undetected. This is supported by the fact that, for most of the significant SNPs detected, the favorable allele had a frequency very close to the MAF threshold. Future research could therefore focus on accessions showing strong PRI responsiveness and use them in biparental crosses to detect additional QTLs that may not have been captured in GWAS. Such crosses would increase allele frequency and provide higher statistical power to identify rare but potentially important loci.

The QTLs identified in this study have strong potential for transferability into breeding programs in long term. Because they were detected in tomato species closely related to cultivated tomato (*S. pimpinellifolium*), and subspecies of *S. lycopersicum* (*S. lycopersicum var. cerasiforme*), their introgression into elite cultivars should be relatively straightforward, with limited risk of linkage drag. Breeders can also verify the genotype of their elite lines at the SNP positions associated with the identified QTLs to determine whether these lines already carry the

favorable allele. If they do, no additional introgression is required; if not, one of the responsive accessions identified in this study can be used as a donor to introduce the allele of interest. This approach strengthens the practical transferability of our results and supports the efficient deployment of PRI-responsive loci in tomato improvement programs.

New resistance QTL to powdery mildew

Our study combined QTL mapping and GWAS to dissect the genetic architecture of tomato resistance to *O. neolycopersici*. QTL mapping revealed a dominant resistance locus (On-9^{I}_{che}) on chromosome 9 originating from *S. cheesmaniae*, explaining approximately 15% of the phenotypic variance. Within this interval, candidate genes include members of the ethylene-associated ETOH pathway, consistent with previous reports implicating ethylene in tomato resistance to *O. neolycopersici* (Sun et al. 2019).

In the short term, this candidate gene could be validated by quantifying ethylene production in segregating populations such as F_2 or backcross lines. This would allow verification of whether variation at the candidate locus translates into measurable differences in ethylene biosynthesis. We did not test the interaction between the QTL on chromosome 9 and other genomic regions that were close to the detection threshold. Therefore, the development of near-isogenic lines (NILs) carrying these regions will be essential to dissect with a high precision their individual effects as well as their potential interactions.

GWAS identified seven additional resistance QTLs within *S. lycopersicum* and *S. pimpinellifolium*, explaining 5-32% of the phenotypic variation of disease index. These intervals contain several strong candidate genes, notably NBS-LRR resistance genes and receptor-like protein kinases (RPK), both central components of pathogen recognition and immune signaling. Because GWAS identifies associations rather than causal genes, these candidate genes will require functional validation, for example through expression profiling under infection, allele mining, or functional complementation in susceptible backgrounds.

All QTLs detected in this study are newly identified, expanding the current repertoire of genomic regions associated with powdery mildew resistance in tomato. Importantly, because these loci originate from species closely related to cultivated tomato, they can be readily introgressed into elite cultivars with minimal risk of genetic incompatibility or linkage drag. This makes them highly valuable targets for breeding programs aiming to enhance durable resistance.

Transferability of the results to field conditions

Several results of this work are directly transferable to growers. The efficacy of PRIs was clearly demonstrated, and some of the products tested are already available commercially. For instance, Belvine® is currently registered and marketed in France as a PRI for grapevine. The company could request an extension of its use to tomato. Planticare® is approved in Canada, and its producer could apply for registration in Europe. Once these steps are completed, both PRIs could be used by growers and integrated rapidly into crop protection programs.

PRIs are applied preventively to prime plant defenses before pathogen infection. However, little is known about the duration of protection they provide. To ensure effective crop protection, PRIs may need to be applied multiple times, ideally guided by the probability of infection risk. Very few studies have investigated the persistence of PRI efficacy over time. Recent work indicates that plants may retain a "memory" of induction and even transmit enhanced responsiveness to their progeny, which could react more efficiently to subsequent treatments (Ramírez-Carrasco et al. 2017; Martínez-Aguilar et al. 2021; Catoni et al. 2022). This suggests that retreating previously induced plants could further enhance their resistance to pathogens. In some cases, the product combines PRI activity with a direct effect on the pathogen, providing dual protection. Therefore, they can be applied both before and after infection. However, understanding their mode of action is essential to ensure proper positioning.

Combining multiple levers for integrated pest management

Genetic resistance and induced resistance should not be considered stand-alone solutions but rather complementary components of integrated pest management (IPM). Effective crop protection relies on combining several levers, including genetic resistance, PRI responsiveness, cultural practices (crop rotation, residue removal, fertilization management), and the use of other biosolutions. Although each lever can be effective individually, their performance is often limited or strongly influenced by environmental conditions. Integrating PRIs into broader IPM strategies can therefore enhance overall protection and reduce reliance on chemical inputs.

The combination of genetic resistance and PRI application is particularly promising. Bénéjam (2021) demonstrated the complementarity between genetic resistance and the PRI ASM, showing additive effects on disease reduction. However, this is not always observed: some studies have reported that certain PRIs are more effective on susceptible plants (Liu et al. 2023; Margaritopoulou et al. 2024). This highlights the importance of studying, beforehand,

the interaction between resistance QTLs and PRI-response QTLs to ensure that combining both traits does not lead to antagonistic effects. Such knowledge is crucial for designing breeding strategies that combine genetic resistance with strong responsiveness to PRIs. Beyond the combination of genetic resistance and PRI application, integrating multiple complementary strategies will be essential. However, before deploying such approaches under real field conditions, it is necessary to verify that they are compatible and potentially synergistic. This question is being addressed within the CapZeroPhyto project, one of whose main objectives is to study the combination of different crop protection levers. The outcomes of these investigations will provide insights that can support future analysis of how PRIs interact with other agronomic factors.

This work directly supports the objectives of the CapZeroPhyto project, which funded this research, and represents a meaningful step toward more agroecological pest management strategies. By integrating genetic resistance, induced resistance, and omics-based analyses, this study outlines a promising pathway toward a more resilient and sustainable tomato production system.

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Équipe d'encadrement

Marc Bardin, Directeur de recherche, INRAE- Pathologie Végétale

Bernard Caromel, Ingénieur de recherche, INRAE- Génétique et Amélioration des Fruits et Légumes

AVIGNON UNIVERSITÉ