



## Article

# Pathogenicity of *Phytophthora infestans* and *Phytophthora betacei* in Four Solanaceous Species from Southern Colombia

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## Highlights

### What are the main findings?

- Pathogen origin influenced incubation period; whereas, infection growth rate depended on pathogen origin, inoculated host, and their interaction.
- *P. infestans* varied widely and cross-infected hosts. *P. betacei* showed consistently low growth without detectable preference for *S. betaceum*.

### What are the implications of the main findings?

- Host origin alone did not predict aggressiveness or specialization.
- Resistance screening should use diverse isolates and include alternative solanaceous hosts.

## Abstract

Late blight caused by *Phytophthora infestans sensu lato* represents a major threat to solanaceous crops in the Andean region. This study evaluated the aggressiveness of 30 *P. infestans* isolates and 10 *P. betacei* isolates obtained from different solanaceous hosts on four host species using detached-leaf assays. The incubation period and infection growth rate were determined over nine days after inoculation. The incubation period was significantly influenced by the origin of the isolate; whereas, the infection growth rate depended on both the inoculated host and the host–pathogen interaction. *Phytophthora infestans* from *S. tuberosum* showed the highest aggressiveness on its original host, reaching infection rates of up to 20.21% day<sup>−1</sup>. However, several isolates exhibited cross-infection capacity and wide variation in aggressiveness ranges on alternative hosts. In contrast, *P. betacei* showed lower pathogenic fitness and no evident host preference, with a mean infection rate of 1.71% day<sup>−1</sup>. These results reveal a complex pathogenicity structure influenced by the interaction between pathogen origin and host, with implications for breeding programs and late blight management strategies. Several isolates exhibited cross-infection capacity and wide variation in aggressiveness.

**Keywords:** late blight; aggressiveness; plant-pathogen interaction; *Solanum*



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## 1. Introduction

The genus *Phytophthora* comprises some of the most destructive plant pathogens worldwide, causing diseases with substantial economic and ecological impacts in numerous

agricultural crops [1]. *Phytophthora infestans* (Mont.) de Bary remains one of the most devastating plant pathogens globally. It causes late blight, a disease associated with significant economic losses in world agriculture [2].

Its impact is particularly severe in species of the family Solanaceae, including commercially important crops such as potato (*Solanum tuberosum* L.) and tomato (*S. lycopersicum* L.), as well as less extensively cultivated species such as pepino melon (*S. muricatum* Aiton.), which may function as an alternative host in Andean agroecosystems [3,4]. This hemibiotrophic oomycete belongs to the *P. infestans sensu lato* complex, a group of closely related species that also includes *P. betacei* (M.F. Mideros, L.E. Lagos & S. Restrepo), the causal agent of late blight in tree tomato (*S. betaceum* Cav.) [5]. Tree tomato was therefore included as the primary host of *P. betacei* and as a representative crop of this pathosystem. The evolutionary success of this complex lies in its genomic plasticity, which facilitates rapid adaptation to overcome host defenses and fungicide pressure, representing a persistent threat to food security [6,7].

Coevolution between *Phytophthora* and its hosts has resulted in population structuring. Different genetic lineages have been associated with specific hosts, suggesting local adaptation [8,9]. This genetic structure suggests possible host preference, whereby, despite the ability to infect other solanaceous species, virulence, sporulation rate, and aggressiveness are greater on the host of origin; in other words, the pathogen exhibits higher pathogenic fitness on its original host [10,11]. Similarly, *P. betacei* has been defined by its high specificity toward *S. betaceum* [5,12]. This specialization is fundamental for breeding programs, which generally focus on crop resistance.

Nevertheless, the ability of *P. infestans* to cause cross-infections in alternative hosts has also been reported [13,14]. This pathogenic plasticity is relevant in centers of agricultural diversity such as the Andean region, where the coexistence of multiple cultivated and wild solanaceous species may favor the persistence, adaptation, and diversification of pathogen populations [2,4]. It is important to emphasize that the particular conditions of tropical ecosystems markedly increase the susceptibility of solanaceous crops to infection by *P. infestans* and *P. betacei*. Local environmental conditions and inadequate cultural practices have influenced the genetic structure of different pathogen populations [15]. Many of these populations emerge with greater aggressiveness and higher reproductive success, affecting epidemiological dynamics over time [16,17].

This context reveals a knowledge gap regarding variation in pathogenicity and the cross-infection capacity of *P. infestans sensu lato* in southern Colombia, particularly among populations genetically differentiated according to their host of origin. The four solanaceous species were selected to represent two commercially important hosts of *P. infestans* (*S. tuberosum* and *S. lycopersicum*), an alternative host with potential epidemiological relevance (*S. muricatum*) and the primary host of *P. betacei* (*S. betaceum*). Therefore, this study evaluated the establishment and progression of late blight caused by *P. infestans* and *P. betacei* isolates on these four host species using detached-leaf assays.

## 2. Materials and Methods

### 2.1. Plant Material Preparation

Plants of *S. tuberosum* Phureja Group var. Solandina, *S. lycopersicum* var. Etereí, and *S. betaceum* cv. ‘Común’ were established from commercial seed; whereas, *S. muricatum* was propagated from plants grown in local orchards because of the limited availability of commercial planting material. Plants were grown in a greenhouse in peat-based substrate and irrigated every two days, with average temperatures of 20 °C during the day and 15 °C at night, and 70% relative humidity. Twenty plants of each species were established. After nine weeks of growth, mature leaf material was collected for pathogen inoculation. For all

host species, fully expanded leaves or leaflets, as appropriate, were selected from the upper third of the plant. Only plant material without visible disease symptoms, insect damage, mechanical injury, or signs of senescence was used.

## 2.2. Inoculum Preparation and Infection

The 30 *P. infestans* isolates and 10 *P. betacei* isolates were obtained from the culture collection of the Genetics and Evolution of Tropical Organisms Research Group at the Universidad de Nariño (Supplementary Table S1). Each isolate was re-cultured on tree tomato–pea agar medium (200 mL tree tomato extract, 200 mL pea extract, 20 g sucrose, 15 g agar-agar, 0.5 g CaCO<sub>3</sub>, and 600 mL distilled water) and incubated for 15 days in darkness at room temperature (15–18 °C). The sporangial suspension was prepared by scraping the mycelium in 20 mL sterile distilled water until a concentration of  $4 \times 10^5$  sporangia mL<sup>-1</sup> was obtained. The suspension was refrigerated at 4 °C for three hours to stimulate zoospore release [10,14].

Each isolate was inoculated onto four detached leaves per host species, with each leaf obtained from a different plant individual. Leaves collected from the same plant individual were distributed among different isolates, and their assignment to isolates was randomized. The leaves were arranged in 30 cm-diameter moist chambers maintained at room temperature (15–18 °C) and at a relative humidity above 70%. Four 20 µL droplets of inoculum were applied to each leaf, with two droplets placed on each side of the midrib. Four leaves per host species were treated with sterile distilled water as negative controls and maintained under the same experimental conditions. Photographic records were obtained daily for nine days after inoculation [18].

## 2.3. Experimental Design

The assay was conducted using a completely randomized design with a two-factor factorial arrangement. The experimental factors were pathogen origin and inoculated host species. Pathogen origin comprised four levels: *S. tuberosum*, *S. lycopersicum*, and *S. muricatum* for *P. infestans*, and *S. betaceum* for *P. betacei*. The inoculated-host factor comprised *S. tuberosum*, *S. lycopersicum*, *S. muricatum*, and *S. betaceum*. Each pathogen-origin group was represented by ten independent isolates, which constituted the biological replicates within each origin. Each isolate was evaluated on all four host species. The only exception was the inoculation of *S. muricatum* with *P. betacei*, for which five isolates were evaluated because of the limited availability of plant material.

For each isolate–host combination, four leaves obtained from different plant individuals were considered technical replicates. These replicates were not treated as independent experimental units. Their measurements were averaged to obtain a single value for each isolate–host combination, which constituted the experimental unit used in the statistical analyses. Similarly, the four inoculum droplets applied to each leaf were considered inoculation sites rather than independent replicates.

Each pathogen-origin × inoculated-host treatment therefore comprised ten experimental units, except for the *P. betacei* × *S. muricatum* treatment, which comprised five experimental units. Overall, the experiment included 155 isolate–host experimental units, corresponding to 620 inoculated detached leaves, in addition to the negative-control leaves.

## 2.4. Statistical Analysis

The experiment generated data for two response variables: incubation period and infection percentage. Incubation period was defined as the number of days elapsed between inoculation and the first appearance of visible symptoms [19]. To improve conformity with model assumptions, these data were transformed using the orderNorm function of the bestNormalize v. 1.9.1 package [20,21] in R v. 4.4.1 [22].

The percentage of infected leaf area was quantified daily from digital photographs using ImageJ v. 1.54p [23]. The grid forming the base of each moist chamber was visible in the photographs and served as an internal spatial reference because the dimensions of its cells were known ( $1.3 \times 2$  cm). Before each image was analyzed, the spatial scale was calibrated in ImageJ using the “Set scale” function and the known distance between two grid lines. The perimeter of each leaf or leaflet was manually outlined using the freehand selection tool to determine the total leaf area. Visible lesions originating from the inoculation sites were then individually delineated using the same tool, and their areas were summed to obtain the total infected area. The percentage of infected area for each leaf was calculated as the ratio between infected area and total leaf area multiplied by 100.

For each isolate–host combination and evaluation day, the infection percentages measured in the four leaf-level biological subsamples were averaged to obtain a single daily value. To obtain a biologically interpretable estimate of disease progression, the untransformed mean infection percentage was fitted against time after inoculation using an ordinary least-squares linear regression model:

$$P_t = \beta_0 + \beta_1 t + \varepsilon_t, \quad (1)$$

where  $P_t$  is the percentage of infected leaf area at day  $t$ ,  $\beta_0$  is the intercept,  $\beta_1$  is the estimated increase in infected leaf area over time, and  $\varepsilon_t$  is the residual error. The slope estimated on the original scale was expressed as percentage points of infected leaf area per day ( $\% \text{ day}^{-1}$ ) and was used to report infection growth rates in the text, tables, and figures.

To improve compliance with the assumptions of linearity, normality, and homoscedasticity, daily infection percentages greater than zero were transformed using the base-10 logarithm, and a separate linear regression was fitted to the transformed data for each isolate–host combination. Model fit was assessed using the coefficient of determination ( $R^2$ ). Regression models were fitted using observations collected from days 1 to 9 after inoculation.

Inferential statistical analyses were conducted using the transformed values; however, the original untransformed data, are presented to facilitate biological interpretation. Subsequently, analysis of variance (ANOVA) and Tukey’s multiple-comparison test ( $\alpha = 0.05$ ) were performed for variables showing significant differences, including incubation period and infection growth rate.

To visualize the relationship between *P. infestans* and *P. betacei* isolates and different host species, a co-occurrence network was constructed in which nodes represent isolates and hosts, and edges represent the infection growth rate (linear regression coefficients). The network was generated using igraph v. 2.1.4 [24] and visualized with ggraph v. 2.2.1 and ggplot2 v. 3.5.1 in R v. 4.4.1 [22].

### 3. Results

Pathogen origin significantly affected ( $p < 0.001$ ) the incubation period of the isolates (Table 1). This indicates that pathogen origin may be a determining factor in the speed of disease establishment. In contrast, no significant influence of the inoculated host species or of the interaction between both factors was observed (Table 1).

Tukey’s mean comparison test separated the incubation period into two statistically different groups ( $p < 0.05$ ) according to pathogen origin (Table 2). The first group consisted of isolates from *S. tuberosum* and *S. betaceum*, which showed the longest incubation period, approximately 1.5 days longer than the second group (Table 2). Thus, isolates from *S. lycopersicum* and *S. muricatum* showed faster disease development (Table 2). This difference in incubation times demonstrates variability in the timing of late blight symptom appearance.

**Table 1.** ANOVA of the influence of pathogen origin and inoculated host species (*Solanum tuberosum*, *S. lycopersicum*, *S. muricatum*, and *S. betaceum*) on the incubation period of *Phytophthora infestans* and *P. betacei*.

|                                          | Degrees of Freedom (df) | Sum of Squares | Mean Square | F-Value | p-Value                |
|------------------------------------------|-------------------------|----------------|-------------|---------|------------------------|
| Pathogen origin                          | 3                       | 37.45          | 12.48 ***   | 19.20   | $3.42 \times 10^{-10}$ |
| Inoculated host                          | 3                       | 2.54           | 0.85        | 1.30    | 0.28                   |
| Pathogen origin $\times$ inoculated host | 9                       | 7.30           | 0.81        | 1.25    | 0.27                   |
| Residuals                                | 117                     | 76.07          | 0.65        |         |                        |

\*\*\* = highly significant differences at  $p < 0.001$ .

**Table 2.** Tukey’s multiple comparison test for the incubation period of *Phytophthora infestans* and *P. betacei* inoculated on *Solanum tuberosum*, *S. lycopersicum*, *S. muricatum*, and *S. betaceum*.

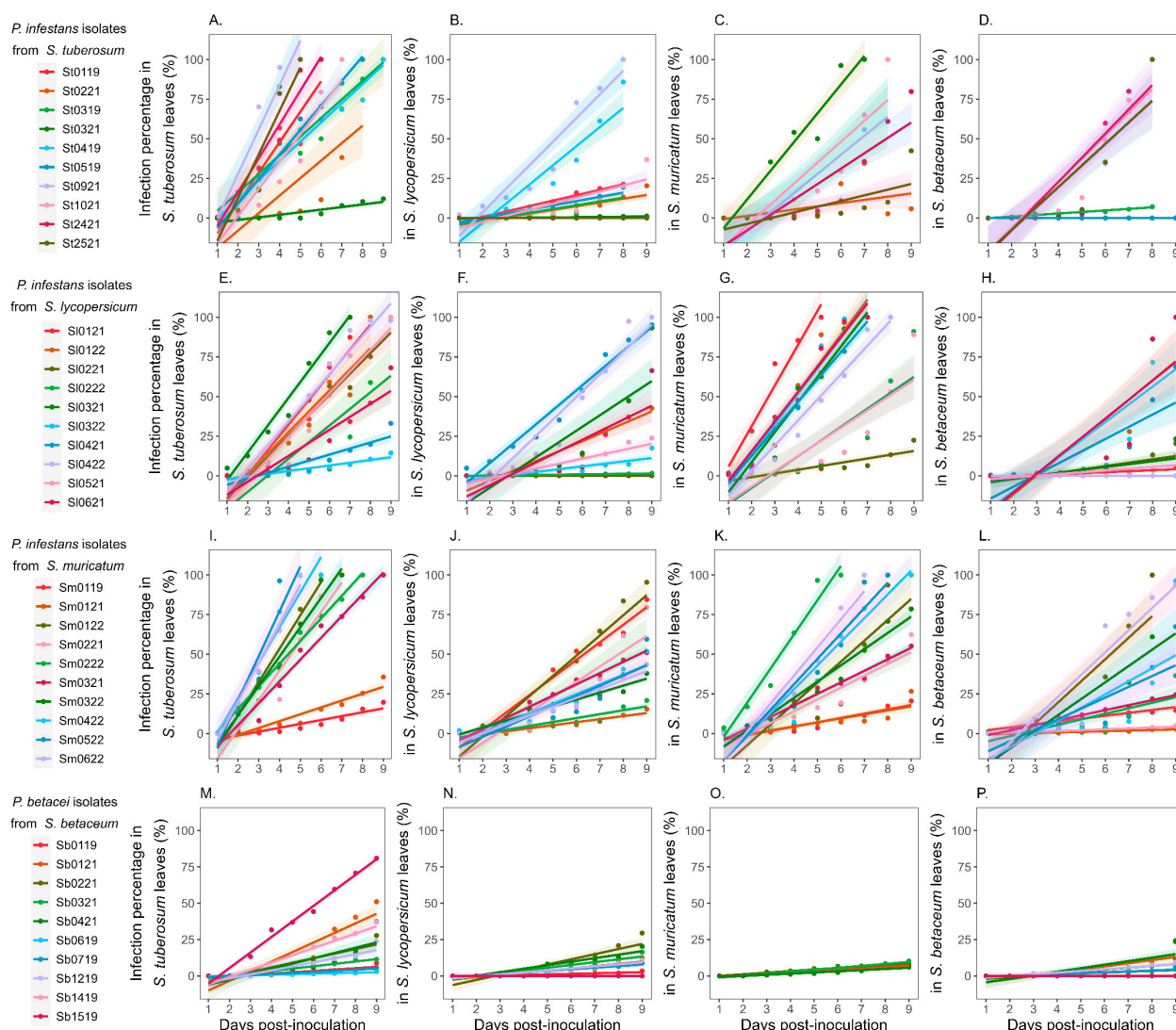
| Pathogen Species    | Pathogen Origin        | Incubation Period (Days) | Groups |
|---------------------|------------------------|--------------------------|--------|
| <i>P. infestans</i> | <i>S. tuberosum</i>    | 3.48                     | a      |
| <i>P. betacei</i>   | <i>S. betaceum</i>     | 3.46                     | a      |
| <i>P. infestans</i> | <i>S. lycopersicum</i> | 2.15                     | b      |
| <i>P. infestans</i> | <i>S. muricatum</i>    | 2.08                     | b      |

Different letters are significantly different according to Tukey’s multiple comparison test ( $p < 0.05$ ).

The infection percentage caused by *P. infestans* and *P. betacei* in the different hosts was adequately described by a linear regression model for each isolate–host combination (mean  $R^2 = 0.84$ ; Supplementary Figure S1; Supplementary Table S2). This indicates that the temporal progression of infection followed a consistent dynamic during the evaluation period ( $p < 0.05$ ). In cases where disease progress was more aggressive, infection was characterized by wet necrotic lesions that extended from the inoculation point toward the outer leaf area. Conversely, some isolates from *S. tuberosum*, *S. lycopersicum*, and *S. betaceum* caused small, light-colored lesions on *S. lycopersicum* and *S. betaceum* leaves that did not expand over time (Supplementary Table S2). In this latter case, the lesions were identified as a hypersensitive-like response to pathogen infection.

Linear regression analysis of infection by *P. infestans* from *S. tuberosum* on different hosts showed broad variability in disease growth (Figure 1A–D). Among the most aggressive isolates, the lesion caused by St0921 increased by 31.29% of the total leaf area of *S. tuberosum* per day, 14.63% day<sup>−1</sup> on *S. lycopersicum*, 12.04% day<sup>−1</sup> on *S. muricatum*, and 13.60% day<sup>−1</sup> on *S. betaceum* (Figure 1A–D; Supplementary Table S2). In contrast, isolate St0321, which showed low aggressiveness on *S. tuberosum* (1.63% day<sup>−1</sup>) and *S. lycopersicum* (0.16% day<sup>−1</sup>) (Figure 1A,B; Supplementary Table S2) and no growth on *S. betaceum* leaves (Figure 1D; Supplementary Table S2), exhibited high disease expansion on *S. muricatum* (18.29% day<sup>−1</sup>; Figure 1C; Supplementary Table S2).

This variable pattern in growth rate was maintained in isolates from *S. lycopersicum* (Figure 1E–H) and *S. muricatum* (Figure 1I–L) on the four solanaceous hosts. For example, Sl0321 was aggressive on both *S. tuberosum* (Figure 1E) and *S. muricatum* (Figure 1G), with mean infection growth rates of 17.33 and 19.12% day<sup>−1</sup>, respectively. The same isolate showed a moderate infection rate on its host of origin (9.61% day<sup>−1</sup>; Figure 1F) and a low rate on *S. betaceum* (1.78% day<sup>−1</sup>; Figure 1H). Similarly, isolates from *S. muricatum* exhibited a broad aggressiveness spectrum, with infection growth rates on their host of origin ranging from 2.52% day<sup>−1</sup> (Sm0121) to 22.10% day<sup>−1</sup> (Sm0222; Figure 1K; Supplementary Table S2). Sm0121 caused the lowest infection on *S. lycopersicum* and *S. betaceum* leaves, reaching less than 20% of the total leaf area by day nine. In contrast, Sm0122 approached 75% infected leaf area on these same hosts (Figure 1J–L).



**Figure 1.** Progression of infected leaf area caused by *Phytophthora infestans* and *P. betacei* on four solanaceous host species during nine days after inoculation. Each color represents a different isolate. Plots are arranged in rows according to isolate origin: (A–D) *P. infestans* obtained from *Solanum tuberosum*; (E–H) *P. infestans* obtained from *S. lycopersicum*; (I–L) *P. infestans* obtained from *S. muricatum*; (M–P) *P. betacei* obtained from *S. betaceum*. Plots arranged in columns correspond to the same inoculated host: (A,E,I,M) *S. tuberosum*; (B,F,J,N) *S. lycopersicum*; (C,G,K,O) *S. muricatum*; and (D,H,L,P) *S. betaceum*.

*Phytophthora betacei* caused smaller lesions than *P. infestans* on all hosts, with a reduction in more than 50% in infection growth rate (Figure 1M–P). In addition, 90% of the isolates showed infection rates between 0 and 6.59% day<sup>−1</sup>, indicating their low aggressiveness under the evaluated conditions (Supplementary Table S2). The most aggressive isolates in this group were Sb1519, Sb0121, and Sb1419, which, when inoculated on *S. tuberosum*, produced infections exceeding 25% of the leaf area by day nine (Figure 1M). These results indicate that, although *P. betacei* was able to infect all four hosts, its pathogenic performance was consistently lower than that of *P. infestans*, suggesting limited capacity to colonize plant tissue under the evaluated conditions.

Unlike the incubation period, the infection growth rate differed significantly by pathogen origin ( $p < 0.001$ ), inoculated host ( $p < 0.001$ ), and their interaction ( $p < 0.05$ ; Table 3), highlighting the importance of ecological context in disease dynamics.

**Table 3.** ANOVA of the influence of pathogen origin and inoculated host species (*Solanum tuberosum*, *S. lycopersicum*, *S. muricatum*, and *S. betaceum*) on the infection growth rate of *Phytophthora infestans* and *P. betacei*.

|                                          | Degrees of Freedom (df) | Sum of Squares | Mean Square | F-Value | p-Value                |
|------------------------------------------|-------------------------|----------------|-------------|---------|------------------------|
| Pathogen origin                          | 3                       | 0.48           | 0.16 ***    | 11.96   | $5.51 \times 10^{-7}$  |
| Inoculated host                          | 3                       | 0.78           | 0.26 ***    | 19.31   | $1.86 \times 10^{-10}$ |
| Pathogen origin $\times$ inoculated host | 9                       | 0.30           | 0.03 *      | 2.48    | 0.01                   |
| Residuals                                | 133                     | 1.79           | 0.01        |         |                        |

\* = significant differences at  $p < 0.05$ . \*\*\* = highly significant differences at  $p < 0.001$ .

The analysis of infection growth rates of *P. infestans* and *P. betacei* across different host and pathogen-origin combinations indicated significant variability ( $p < 0.05$ ; Table 4). The highest aggressiveness value was recorded for *P. infestans* from *S. tuberosum* inoculated on the same host, with an infection growth rate of  $20.21\% \text{ day}^{-1}$ . In contrast, *P. infestans* from the same origin but inoculated on *S. lycopersicum* showed the lowest infection values, with a mean value of  $4.25\% \text{ day}^{-1}$ . All *P. betacei* isolates were grouped among the combinations showing lower pathogenic fitness, causing low infection percentages without evident differences among the evaluated hosts.

**Table 4.** Tukey's multiple comparison test for the infection growth rate of *Phytophthora infestans* and *P. betacei* inoculated on *Solanum tuberosum*, *S. lycopersicum*, *S. muricatum*, and *S. betaceum*.

| Species             | Pathogen Origin        | Inoculated Host        | Infection Growth Rate (% day <sup>-1</sup> ) | Groups |
|---------------------|------------------------|------------------------|----------------------------------------------|--------|
| <i>P. infestans</i> | <i>S. tuberosum</i>    | <i>S. tuberosum</i>    | 20.21                                        | a      |
| <i>P. infestans</i> | <i>S. muricatum</i>    | <i>S. tuberosum</i>    | 18.10                                        | ab     |
| <i>P. infestans</i> | <i>S. lycopersicum</i> | <i>S. muricatum</i>    | 17.53                                        | abc    |
| <i>P. infestans</i> | <i>S. lycopersicum</i> | <i>S. tuberosum</i>    | 12.04                                        | abcd   |
| <i>P. infestans</i> | <i>S. tuberosum</i>    | <i>S. muricatum</i>    | 11.53                                        | abcd   |
| <i>P. infestans</i> | <i>S. muricatum</i>    | <i>S. muricatum</i>    | 11.46                                        | abcd   |
| <i>P. infestans</i> | <i>S. muricatum</i>    | <i>S. lycopersicum</i> | 6.78                                         | bcd    |
| <i>P. infestans</i> | <i>S. muricatum</i>    | <i>S. betaceum</i>     | 6.18                                         | bcd    |
| <i>P. infestans</i> | <i>S. tuberosum</i>    | <i>S. betaceum</i>     | 5.80                                         | cd     |
| <i>P. infestans</i> | <i>S. lycopersicum</i> | <i>S. lycopersicum</i> | 5.59                                         | cd     |
| <i>P. infestans</i> | <i>S. lycopersicum</i> | <i>S. betaceum</i>     | 4.67                                         | cd     |
| <i>P. infestans</i> | <i>S. tuberosum</i>    | <i>S. lycopersicum</i> | 4.25                                         | d      |
| <i>P. betacei</i>   | <i>S. betaceum</i>     | <i>S. tuberosum</i>    | 3.55                                         | d      |
| <i>P. betacei</i>   | <i>S. betaceum</i>     | <i>S. lycopersicum</i> | 1.35                                         | d      |
| <i>P. betacei</i>   | <i>S. betaceum</i>     | <i>S. muricatum</i>    | 1.05                                         | d      |
| <i>P. betacei</i>   | <i>S. betaceum</i>     | <i>S. betaceum</i>     | 0.91                                         | d      |

Different letters are significantly different according to Tukey's multiple comparison test ( $p < 0.05$ ).

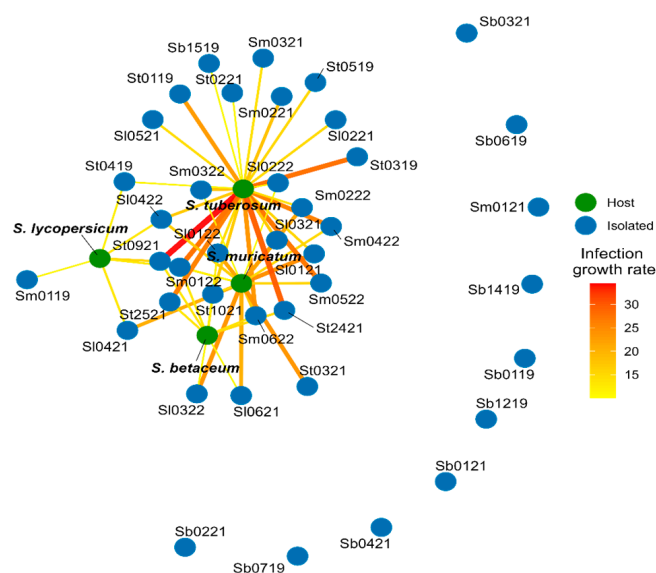
Isolates from *S. tuberosum* showed a high infection rate on their host of origin ( $20.21\% \text{ day}^{-1}$ ), followed by a rate of  $11.53\% \text{ day}^{-1}$  on *S. muricatum* leaves. In contrast, the infection rate on *S. lycopersicum* and *S. betaceum* was 75% lower than on *S. tuberosum* (Table 4). Similarly, isolates obtained from *S. muricatum* showed greater aggressiveness on *S. tuberosum* leaves, with 6.64% more daily infection than on their host of origin. Meanwhile,

infection rates on *S. lycopersicum* and *S. betaceum* were intermediate (6.78 and 6.18% day<sup>-1</sup>, respectively; Table 4).

Conversely, *P. infestans* from *S. lycopersicum* showed a higher infection growth rate on *S. muricatum* leaves (17.53% day<sup>-1</sup>) and *S. tuberosum* leaves (12.04% day<sup>-1</sup>), placing these among the most aggressive interactions (Table 4). However, it showed limited pathogenic fitness on its original host, with a 68.10% reduction compared with *S. muricatum*, reaching values similar to those obtained on *S. betaceum* (4.67% day<sup>-1</sup>; Table 4).

The results show that *S. tuberosum* and *S. muricatum* were the hosts most susceptible to *P. infestans* regardless of pathogen origin, as they presented the highest infection growth rate values, with a mean of 15.14% day<sup>-1</sup>, and no statistical differences between them. In contrast, *P. betacei* caused the lowest infection rates, with values ranging from 3.55% day<sup>-1</sup> on *S. tuberosum* leaves to 0.91% day<sup>-1</sup> on *S. betaceum* (Table 4). In addition, the results reveal that *P. betacei* had no preference among the four evaluated hosts, as pathogen fitness in terms of infection rate was not higher on any particular host. This differs from *P. infestans* from *S. tuberosum* inoculated on its host of origin, which exhibited an infection rate 95.50% higher than that of the group of isolates with the lowest infection rate.

The co-occurrence analysis between isolates and host species revealed a structure that reflects the complexity of plant-pathogen relationships (Figure 2). *Solanum tuberosum* leaves were identified as one of the main nodes, followed by *S. muricatum*, because a large number of *P. infestans* isolates showed plant tissue infection rates above 20% per day (Figure 2; Supplementary Table S2). In turn, the hosts *S. lycopersicum* and *S. betaceum* exhibited weaker connections with isolates regardless of their origin, with infection growth rates between 10 and 15% day<sup>-1</sup> relative to total leaf area (Figure 2; Supplementary Table S2). Ninety percent of *P. betacei* isolates showed no connections with the central nodes (hosts) because of their low infection rate (<10% day<sup>-1</sup>; Figure 2; Supplementary Table S2), such that only Sb1519 progressed moderately on *S. tuberosum* leaves.



**Figure 2.** Co-occurrence network of infection growth rates of *Phytophthora infestans* and *P. betacei* isolates and four host species, where the color and thickness of the connections represent the infection growth rate. Blue nodes represent isolate codes (Pathogen origin; St: *Solanum tuberosum*, Sl: *S. lycopersicum*, Sm: *S. muricatum*, Sb: *S. betaceum*). Green nodes represent the inoculated host species.

#### 4. Discussion

Three main findings emerged from this study. First, pathogen origin affected the incubation period; whereas, infection growth rate depended on pathogen origin, the

inoculated host, and their interaction. Second, *P. infestans* isolates showed substantial variation in aggressiveness and frequent cross-infection, indicating that host of origin did not consistently predict the most aggressive interaction. Third, *P. betacei* produced low infection growth rates on all four hosts and showed no detectable preference for *S. betaceum* under the detached-leaf assay conditions.

Significant differences in incubation period according to pathogen origin may be associated with differences in population competitiveness [25]. A shorter period could provide a competitive advantage for isolates from *S. lycopersicum* and *S. muricatum*, as it allows them to complete more infection cycles within a given period and maximize the production of secondary inoculum [19,26]. However, *P. infestans* isolates originating from *S. tuberosum* showed the longest incubation period, it also showed the highest infection growth rate, suggesting a pattern of delayed symptom expression followed by rapid expansion in host tissue. This same behavior was reported for *P. betacei* on the common variety of *S. betaceum* [18]. Nevertheless, in the present study, the relatively long incubation period of *P. betacei* was accompanied by a low infection growth rate, which may indicate lower pathogenic fitness compared with the other evaluated interactions.

Thus, the incubation period alone did not fully represent the infectious behavior of the pathogen in relation to its host, as this behavior depends on multiple physiological and molecular factors. Among the most important are isolate-dependent characteristics such as the duration of the biotrophic phase, the transition to the necrotrophic phase, and the repertoire of effector genes associated with each phase [27]. During the biotrophic phase, the pathogen penetrates plant tissue and obtains nutrients through haustoria, inducing genes related to detoxification, enzyme inhibitors, protection against oxidative stress, and necrosis suppressors [28,29]. Subsequently, the expression of genes that induce cell death in infected tissues leads to the first appearance of wet necrotic lesions and their expansion through the plant tissue [30]. In *P. infestans*, several effectors expressed during the early stages of infection, such as EPI1, AVR3a, SNE1, and AVRblb2, participate in suppressing basal host immune responses, promoting initial colonization by interfering with signaling pathways related to cellular defense and programmed cell death [31–33]. These differences in effector regulation and expression among isolates could contribute to prolonging or shortening the initial asymptomatic period, which may explain the variation observed in isolate evaluation times according to their origin.

This differential pattern in the transition between biotrophic and necrotrophic phases among the different isolates supports the hypothesis that *P. infestans* aggressiveness is determined by a combination of pathogen genetic factors and host response, which may explain the variability in infection rate observed across the different treatments. This is related to the high genetic variability reported in the evaluated populations, which revealed a unique multilocus genotype for each isolate, determined using an analysis of 12 microsatellite markers [9].

Although *P. infestans sensu lato* populations from southern Colombia previously showed high genetic diversity, with a population structuring associated with the host of origin [9], the phenotypic interactions observed in the present study did not consistently correspond to this genetic pattern. *Phytophthora infestans* isolates originating from *S. tuberosum* showed the highest mean infection growth rate on their host of origin; however, this value did not differ significantly from that of isolates originating from *S. muricatum* and inoculated on *S. tuberosum*. Thus, no consistent primary-host advantage in infection growth rate was detected [11,14], indicating that host associated genetic structuring does not necessarily translate into uniform pathogenic specialization. This interpretation is consistent with previous studies in Colombia, in which potato-derived isolates infected both potato and tomato despite genetic differentiation between host-associated populations [11], and

with reports of coexisting specialist and generalist clonal lineages in European *P. infestans* populations [13]. Together, these findings suggest that host adaptation may be isolate- or lineage-specific rather than a fixed population-level characteristic.

Consistent with this interpretation, substantial variation was observed among isolates belonging to the same origin group. Some isolates showed apparent specialist-like responses, as illustrated by St0321, which produced limited or no lesion expansion on three hosts but exhibited a markedly higher infection growth rate on *S. muricatum*. In contrast, isolates such as St0921 maintained relatively high infection growth rates across all four host species, indicating a broader infection profile. These interactions may present lower growth rates as a result of a fitness cost associated with the ability to effectively infect several hosts [13,34]. However, this relationship was not directly tested in the present study. Overall, the contrasting responses among isolates indicate considerable plasticity in the infection process and suggest that disease progression depends on the specific compatibility of each isolate–host combination [28,35].

The observed mismatch between host-associated genetic structure and infection phenotype, together with the dissociation between symptom appearance and lesion expansion, may reflect the combined effects of pathogen genetic background and host response. A comparable dissociation between genotypic and pathogenic characteristics of *P. infestans* was reported by Zheng et al. [36], who found that isolates from ecologically distinct regions shared similar SSR-based genetic backgrounds but differed in their virulence spectra, making it necessary to address these interactions from a molecular perspective.

The genomes of *P. infestans* and *P. betacei* contain more than 500 effector genes encoding proteins associated with pathogenicity [37,38]. These effectors act as molecular tools that facilitate disease and evasion of plant responses [39,40], because they participate in immune suppression by interfering with plant cell signaling pathways and modulating the accumulation of reactive oxygen species. In addition, they can alter hormonal signaling related to salicylic acid, jasmonic acid, and ethylene [35,41], inhibit the expression of genes involved in metabolic pathways and transcription factor regulation, thereby acting as key molecules in plant–pathogen interactions [42]. In this context, differences in effector repertoire, regulation, and expression among species and isolates may contribute to differences in aggressiveness in each host [36].

These effector genes are located in genome regions rich in repetitive sequences and poor in genes, favoring high mutation rates [6]. Selection on these polymorphisms enables them to overcome plant defense barriers and colonize a broad host range with differential aggressiveness characteristics, which also result from interaction with the specific host genotype [35,43].

Previous field and genomic studies have suggested a strong association between *P. betacei* and *S. betaceum*, and comparative analyses have identified candidate apoplastic and RXLR effectors that may contribute to host specificity [5,12]. However, this pattern was not reflected in lesion expansion under the detached-leaf conditions used in the present study. *Phytophthora betacei* showed consistently low infection growth rates across the four solanaceous hosts, with no detectable advantage on *S. betaceum*. This discrepancy may reflect differences between host association under natural conditions and the specific component of aggressiveness measured in detached leaves. Variation among local isolates and differences in effector content, regulation, or expression could also contribute to the observed infection patterns [12,37], although genomic and transcriptional analyses would be required to evaluate this hypothesis. Recent pangenomic evidence further indicates a complex evolutionary history involving divergence and gene flow among clade 1c lineages in the Andean region [3]. This could favor the conservation of cross-infection capacities on

shared hosts. However, the contribution of these evolutionary processes to these results observed here remains unresolved.

The co-occurrence analysis identified greater susceptibility to infection by *P. infestans* and *P. betacei* in *S. tuberosum* leaves. In this host, isolates exhibited higher pathogenic fitness, with high infection rates and severe disease characteristics. This indicates that this host provides more suitable conditions for infection and pathogen development, traits related to the evolutionary history of the *P. infestans*-*S. tuberosum* pathosystem [44,45]. *Solanum muricatum* was the second most susceptible species and is considered an alternative host that grows either wild or in home gardens in the absence of strong selection pressure from fungicide application. These conditions allow it to harbor species of *P. infestans sensu lato*, where the inoculum survives and spreads [4,46].

One limitation of this study is that only the incubation period and infection growth rate were evaluated. Although these parameters are commonly used to assess pathogen aggressiveness, they may not represent it accurately in cases where other variables, such as sporulation capacity and latency period, may be determining factors [34]. A clear example of this dynamic was described in *P. infestans* populations in Algeria, where small lesions were observed on *S. lycopersicum* leaves but with greater sporulation than on *S. tuberosum*. This resulted in greater pathogen spread and persistence in the environment (competitive fitness), allowing it to continue infection cycles [47]. Therefore, future research should consider a larger number of variables that more comprehensively represent pathogen fitness in relation to their hosts.

Understanding the complex interaction of *P. infestans* and *P. betacei* with economically important solanaceous species provides important implications for potential long-term disease control strategies in Colombia. The observed variability in infection growth rates depending on pathogen origin and inoculated host highlights the need to consider multiple factors in future late blight management strategies. These include not only host genetics in plant breeding programs, but also the specific genetic and pathogenic characteristics of *P. infestans* and *P. betacei* populations, as well as their interactions across agroecological and molecular scales. From an applied perspective, isolates with greater aggressiveness and infection capacity on multiple hosts represent valuable tools for breeding programs, as they allow selection pressure to be increased during screening processes and facilitate the identification of genotypes with partial or broad-spectrum resistance [48,49]. Therefore, future research should focus on validating these results at a larger scale using field trials and incorporating a comprehensive molecular analysis of plant-pathogen interactions to deepen understanding of the mechanisms underlying pathogenicity.

## 5. Conclusions

The pathogenic behavior of *P. infestans* and *P. betacei* on four solanaceous hosts from southern Colombia was determined by different components of the infection process. Pathogen origin influenced the incubation period; whereas, the infection growth rate was jointly affected by pathogen origin, the inoculated host, and their interaction. *Phytophthora infestans* isolates exhibited substantial variation in aggressiveness and a broad capacity for cross-infection. In contrast, *P. betacei* infected all evaluated hosts but consistently produced lower infection growth rates and showed no clear preference for *S. betaceum* under the detached-leaf conditions used in this study. These findings indicate that pathogen origin alone does not reliably predict host specialization or aggressiveness. Therefore, the marked variability among isolates should be considered in resistance-screening programs and late blight management strategies in production systems.

**Supplementary Materials:** The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/horticulturae12080973/s1>, Figure S1: Log-transformed slope  $R^2$  values for each isolate. The red line indicates the mean; Table S1: Information on the origin of the *P. infestans* and *P. betacei* isolates available in the culture collection of the Genetics and Evolution of Tropical Organisms Research Group at the University of Nariño; Table S2: Regression parameters and goodness-of-fit metrics for infection growth of *P. infestans* and *P. betacei* in four Solanaceae hosts: *S. tuberosum*, *S. muricatum*, *S. lycopersicum*, and *S. betaceum*.

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**Data Availability Statement:** The datasets generated and analyzed during this study are available in the Figshare repository at <https://doi.org/10.6084/m9.figshare.30629153>. The original photographic records of symptom and lesion development on detached leaves are openly available in Figshare at <https://doi.org/10.6084/m9.figshare.32981915>.

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