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Research article

Alteration of secondary metabolites' profiles in potato leaves in response to weakly and highly aggressive isolates of *Phytophthora infestans*

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ABSTRACT

Phytophthora infestans is the cause of late blight, a devastating disease in potato and tomato. Many of the mechanisms underlying *P. infestans* pathogenesis and defense responses in potato are still unclear. We investigated the effects of *P. infestans* on the changes in the accumulation of secondary metabolites in potato cultivars using whole plants. Four preformed flavonoids and one terpenoid compound produced in potato tissues were differentially affected by the *P. infestans* inoculation. In Russet Burbank, the accumulation of catechin and rutin was suppressed by both *P. infestans* isolates US-11 and US-8, while the flavanone P3 was associated with susceptibility to this pathogen. On the other hand, catechin, flavonol −glycoside P2, and an unidentified terpenoid (T1), may be involved in the defense of cultivar Defender to both tested *P. infestans* isolates, providing new evidence that different preformed flavonoids and terpenoids in potato may play important roles in its defense or susceptibility to *P. infestans*. These results add to the pool of data showing the involvement of other phenolics and terpenes in potato resistance to microbial pathogens.

1. Introduction

Late blight caused by the oomycete *Phytophthora infestans* (Mont.) de Bary is the most devastating disease of potato (*Solanum tuberosum*) and was responsible for the Irish potato famine in 1845 [1]. This pathogen uses either sporangia or zoospores to infect leaves, stems, and tubers [2–4]. Zoospores and sporangia penetrate the leaf surface either through stomata or directly through the epidermal cell wall. Germination of sporangia occurs mainly at temperatures above 12 °C. The germ tube differentiates into an appressorium and a penetration peg is formed to aid the passage of the pathogen through the host cell wall. Three to four days later, secondary sporulation and infection occur to initiate, once more, the disease cycle of *P. infestans* [5]. In response to such infection, potato sets up a series of reactions including the upregulation of several defense-related genes followed by the accumulation of defense proteins and secondary metabolites. Wang et al. [6] used cDNA microarrays to investigate potato genes associated with quantitative resistance to late blight and showed that approximately 37.2% of all the known *P. infestans*-responsive genes had a general or secondary metabolism function. Many genes that encode enzymes participating in the potato defense-related metabolic pathways, such as that of phenylpropanoids and alkaloids, were activated by *P. infestans* [6].

The majority of antimicrobial secondary metabolites are derived from the phenylpropanoid, isoprenoid, alkaloid or fatty acid pathways [7]. During plant–pathogen interactions, cell wall reinforcement with lignin and the accumulation of phytoalexins, including secondary metabolites from such pathways [8–10] are well-known plant defense responses. Phenylpropanoids exhibit a broad-spectrum antimicrobial activity and are therefore believed to help the plant fight microbial diseases [11]. Cell wall-bound phenolics also accumulate locally to restrict fungal penetration. However, the precise functions of phenolics in such reaction are still poorly understood [12,13].

Phenolic compounds associated with potato resistance to *P. infestans* include chlorogenic acid, caffeic acid, scopoletin, scopolin [14], and *p*-coumaroyloctopamine [15]. Isoprenoids related to potato resistance to *P. infestans* include rishitin, phytuberin and lubimin [15,19]. However, these compounds have a broad activity spectrum and their involvement has often been shown in single interactions where one potato line was inoculated with one isolate of *P. infestans*. In addition, most previous studies were run *in vitro* and often on tubers. The complexity of this interaction requires a better-defined structure of the model used in the study. Wang et al. [16–19] and Henriquez and Daayf [20] used a quadratic system including one susceptible and one partially resistant potato cultivar inoculated

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with one weakly or one highly aggressive isolate of *P. infestans*. Such a system has provided a new perspective in better detecting differential reactions from either the plant or the pathogen at the gene level. The objective of the study presented here was to investigate the accumulation of secondary metabolites in potato after inoculation with *P. infestans*, using whole plants of the susceptible cultivar "Russet Burbank", and the moderately resistant cultivar "Defender", inoculated with a weakly aggressive or a highly aggressive isolates of *P. infestans*. This quadratic system is meant to provide quick access to meaningful differences in plant responses.

2. Results

2.1. Disease development

We used two isolates of *P. infestans*, D-03 (US-11) and D1901 (US-8) to inoculate cultivars Russet Burbank (highly susceptible) and Defender (moderately resistant). No symptoms were visible within the first 48 hpi on either cultivar and only small lesions became noticeable at 72 hpi in Russet Burbank and Defender inoculated with US-8. Late blight symptoms were visible in Russet Burbank inoculated with US-11 only at 120 hpi. Disease progress observations from 72 to 144 hpi are shown in Fig. 1. The US-8 isolate caused spreading disease lesions and extensive tissue damage in Russet Burbank, whereas it ended up causing only limited disease lesions in Defender. The US-11 isolate caused small lesions in Russet Burbank and failed to cause disease in Defender.

2.2. Analysis of secondary metabolites

The time points selected for secondary metabolites profiling in potato after inoculation with *P. infestans* were determined based on the development of late blight symptoms. Seventy hours post-inoculation was selected as a time point when small lesions became noticeable in both Russet Burbank and Defender. Then, 120 hpi was selected when Russet Burbank inoculated with US-8 showed extensive tissue damage, whereas in Defender spreading lesions were just observed. The 120 hpi time also coincided with small lesions that became noticeable in Russet Burbank inoculated with US-11 (Fig. 1). Finally, an early stage of the interaction was

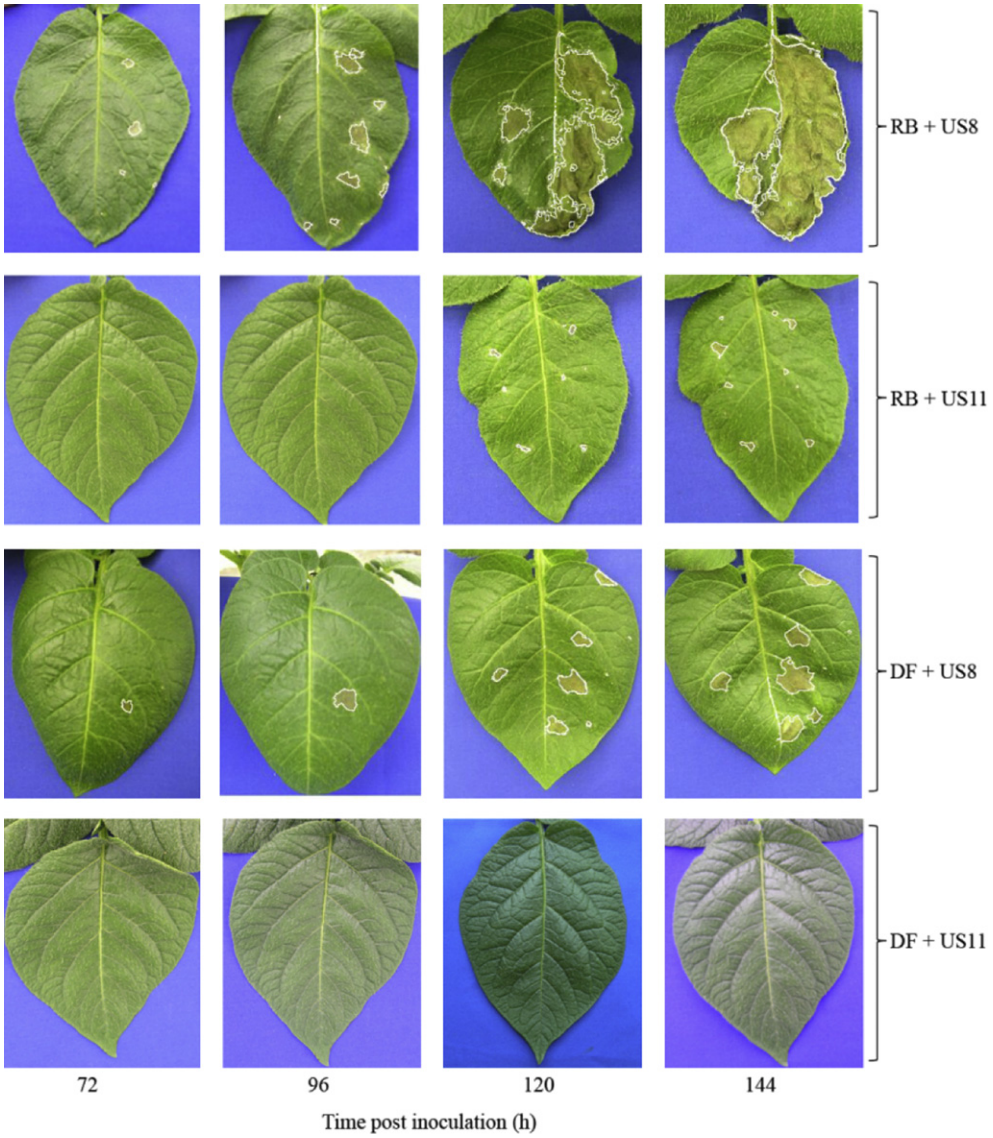

Fig. 1. Infection development by *Phytophthora infestans* in potato. Russet Burbank inoculated with US-8 (RB + US-8), Russet Burbank inoculated with US-11 (RB + US-11), Defender inoculated with US-8 (DF + US-8), Defender inoculated with US-11 (DF + US-11). A white line was added using the image analysis software Assess to denote the lesion boundaries.