

## ANTIOXIDANT RESPONSE OF TOMATO TO LATE BLIGHT (*PHYTOPHTHORA INFESTANS*) INFECTION DEPENDING ON SYMPTOMS INTENSITY

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**ABSTRACT.** Late blight (LB) caused by the *Phytophthora infestans* is a devastating tomato disease, distributed worldwide. Tomato wild species could be a potential source of resistance, however, there are little data about their biochemical response to LB infection. Therefore the aim of the study was to evaluate total phenolic and total flavonoid content and antioxidative activity in the leaves of wild (*Solanum pimpinellifolium*) and cultivated genotype (Bizon) depending on disease severity. *S. pimpinellifolium* compared to Bizon was less susceptible and had five times lower disease severity index (11% and 55% respectively). Additionally, during the disease progression wild genotype showed a much slower decrease of total biochemical parameters compared to the cultivated one. Parameters such as total phenolic content (TP), DPPH (2,2-diphenyl-1-picrylhydrazyl) radical scavenging test and ABTS (2,2'-azinobis-(3-ethylbenzothiazoline-6-sulfonic acid)) radical scavenging activity in the symptomless leaves and the leaves infection 10-25% remained the same in *S. pimpinellifolium*. The susceptible genotype Bizon had about 40% of leaves with the disease severity 25-50%, and a rapid decrease of all biochemical parameters. A strong negative correlation was observed between late blight infection and biochemical parameters in Bizon, while in *S. pimpinellifolium* late blight infection negatively correlated only with TP, TF (total flavonoid content) and total antioxidant activity (TAA).

**Keywords:** biochemical response, late blight, *S. pimpinellifolium*

### INTRODUCTION

Late blight (LB), caused by cosmopolitan pathogen *Phytophthora infestans*, is a destructive disease of tomato (*Solanum lycopersicum*) causing

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severe losses. The yield losses due to tomato late blight can be significant, despite the progress in weather-based software for the disease forecast and the availability of chemical control agents [1]. Changes in *P. infestans* pathogenicity, the introduction of new pathogen isolates, and increased resistance of the pathogen to fungicides, made late blight control highly demanding [2]. All these concerns have made the selection and use of late blight-resistant tomato cultivars a more attractive approach [3]. Since this disease poses problems, especially to organic tomato growers, wide hybridization is considered as a method for introduction of late blight resistance from wild relatives into cultivated tomato [4].

The *Solanum* section *Lycopersicon* is an economically important group of organisms and consists of 14 species including the cultivated tomato *Solanum lycopersicum* and its closest wild relative *Solanum pimpinellifolium*-currant tomato [5,6]. Although *S. pimpinellifolium* is native to Ecuador and Peru [7], it has spread worldwide and due to small red sweet fruits commonly grown as heirloom tomato. Additionally, the tomato wild species bear a wealth of genetic variability for many agriculturally and biologically important characteristics. *S. pimpinellifolium* is regarded as an important source of genes that can confer favourable stress tolerance to cultivated tomato. *S. pimpinellifolium* offers a wealth of breeding potential for desirable traits such as tolerance to abiotic [4] and biotic stresses [8,9], horticultural traits [10] and fruit quality traits [11,12]. Furthermore, possibility of transfer of late-blight resistance from *S. pimpinellifolium* to the cultivated tomato via a traditional backcross breeding approach [13] and its high cross compatibility with the cultivated tomato makes this species very desirable for breeding purposes [14].

As stated by some authors [15,16], among numerous defense mechanisms involved in late-blight resistance, alteration of metabolic pathways may be one of the most important disease defense responses [17]. Plants and pathogens are developing a complex relationship during microbial colonization stages generally involving the activation of host immune responses and the intention of the pathogen to manipulate plant cell processes [18]. Additionally, pathogen infection leads to various stress responses, depending on the plant - pathogen combination, the most common is the production of ROS (reactive oxygen species) [19]. Changes in antioxidant status and accumulation of some antioxidant metabolites indicate a plant response against pathogen invasion [20], although the rapid accumulation of plant ROS at the pathogen attack site, a phenomenon called oxidative burst, is toxic to plant directly [21]. Aside from the enzymatic antioxidants such as superoxide dismutase, peroxidase and catalase, low mass compounds such as glutathione, ascorbic acid,  $\alpha$ -tocopherol, carotenoids, and phenolic compounds are involved in the ROS-scavenging [22,23]. Recent findings revealed that some highly toxic ROS components, such as singlet oxygen and hydroxyl radicals, can be scavenged only by nonenzymatic antioxidants ROS [24].

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The expression of phenolic compounds is promoted by biotic and abiotic stresses (e.g., herbivores, pathogens, unfavourable temperature and pH, saline and heavy metal stress, and UV radiation) [25]. Phenolic compounds as a secondary metabolites can participate directly in plant defence as regulatory signals of the response to pathogen attack through their direct toxic effects on the pathogen, as a component in lignin formation and cell wall strengthening, and reductant of cell-wall ROS [26]. Specific phenolic compounds in different plant-pathogen interactions could also be possibly integrated with an automatic plant stress resistance screening programs [27]. Flavonoids, a group of phenolic compounds, have antimicrobial properties based on their abilities to act as antioxidants, inhibit certain enzymes, disrupt cell membranes, prevent viral binding and penetration into cells, and trigger a host cell self-defence mechanisms [28]. Antioxidative activity of plant extract is the capability of the plant to scavenge free radicals to avoid their harmful effect [29]. Therefore the aim of this study was to evaluate the reaction of wild and cultivated tomato genotypes to late blight (caused by invading *P. infestans*) infection through the development of the disease symptoms on the leaves as well as through the phenolic and flavonoid content and antioxidative activity.

## RESULTS AND DISCUSSION

Evaluation of the late blight infection on tomato leaves indicated over 60% of *S. pimpinellifolium* leaves were without symptoms. In addition to 60% symptomless leaves, there were 23.3% of leaves with infection rate up to 5%. On the contrary, Bizon had almost 40% of leaves with infection rate 25-50% and nearly five times higher disease severity index (DSI) (Table 1). Bonferroni test showed a significant difference between the susceptibility of these genotypes.

**Table 1.** Incidence of late blight infection on leaves of two tested genotypes

Genotype		<i>S.pimpinellifolium</i>		Bizon	
leaf disease rate	leaf area infected	leaves per category			
	(%)	(no)	(%)	(no)	(%)
0	0	19	63.33	2	6.67
1	< 5	7	23.33	4	13.3
2	5-10	2	6.67	7	23.33
3	10-25	2	6.67	6	20
4	25-50	0	0	11	36.67
<b>DSI (%)</b>		<b>11.3</b>		<b>53.3**</b>	

\*\* – significant at 1% level

The linkage between the level of infection and antioxidant activity is shown in the Table 2. Genotype (G), late blight infection (LB) and the interaction of these two factors had a strong influence on biochemical parameters in the leaves (Table 2).

**Table 2.** F values for biochemical parameters in tomato leaves

	d.f	TP	TF	DPPH	ABTS	FRAP	NBT test	TRC	TAA
<b>(G)</b>	1	526.13**	460.75**	31.13**	94.13**	1764.4**	126.55**	298.09**	32.30**
<b>(LB)</b>	3	225.74**	862.61**	40.67**	41.39**	790.2**	84.80**	68.45**	96.83**
<b>G × LB</b>	3	85.58**	161.87**	24.07**	23.59**	516.2**	250.08**	95.02**	2.81 ns

d.f. – degree of freedom, \*\* – significant at 1% level; n = 3 QE – quercetin; DW - dry weight;

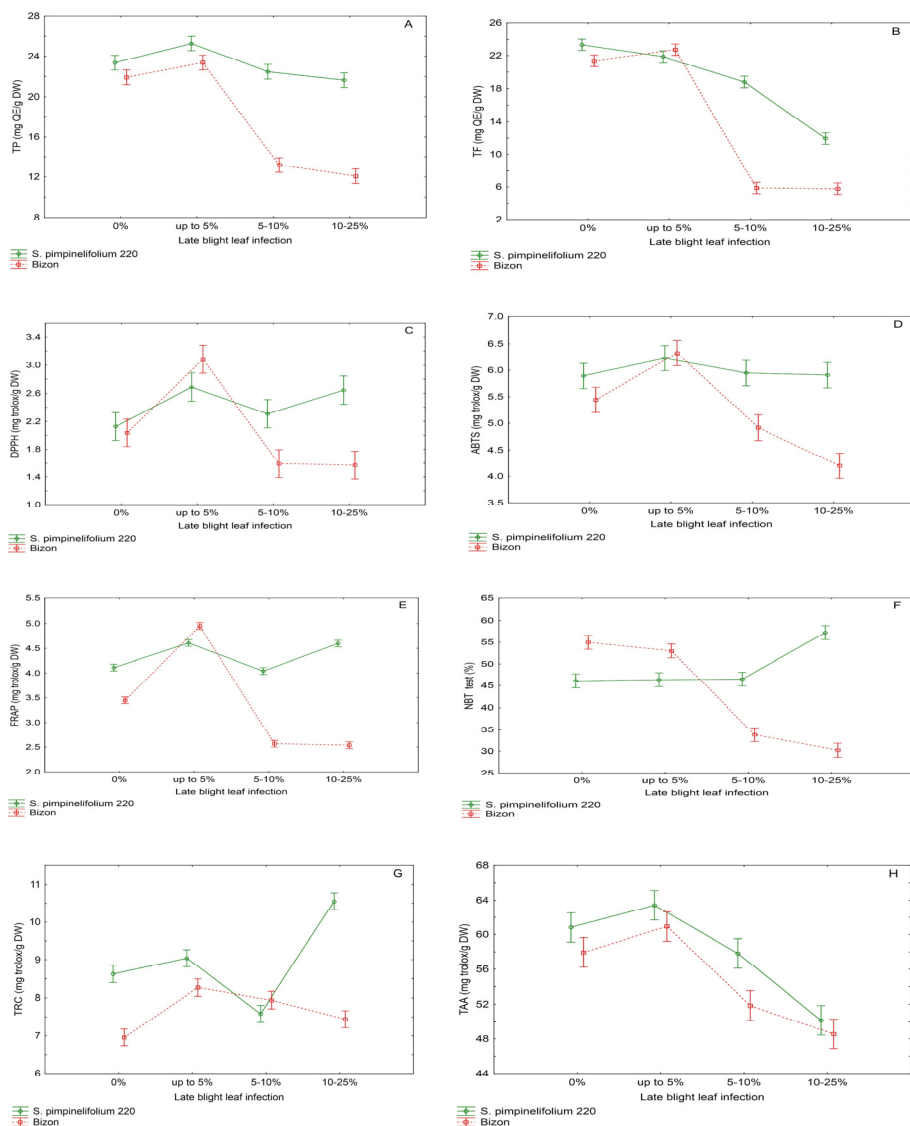
Furthermore, detailed analysis of TP, TF and antioxidant activity tests provide an insight into the physiological response and revealed differences of both tested genotypes reaction to late blight infection. Changes in biochemical parameters depending on the leaf area covered by late blight is shown in Figure 1. Two tomato cultivars with different levels of resistance to late blight express differences in the biochemical response to the disease.

Generally, initial symptoms of late blight are followed by the increase of antioxidant activity measured by DPPH, ABTS, FRAP, TRC and TAA test in susceptible genotype (Bizon). However, when the leaf area was covered by late blight symptoms more than 5%, all the observed parameters, excluding TRC, decreased rapidly in this genotype, for example flavonoids declined several times. Along with the disease progress, when the symptoms have affected more than 10% of leaf surface, biochemical parameters remained on the same level, except the ABTS which decrease more. Conversely, in *S. pimpinellifolium*, in the case of leaf infection up to 5%, only an increase of TP and DPPH values was obtained, while there was no significant difference in other parameters compared to symptomless leaves. Furthermore, with disease progression, 5-10% of leaf area covered, six out of eight biochemical parameters (TP, DPPH, ABTS, FRAP, NBT and TAA) in *S. pimpinellifolium* were on the level of values measured in leaves without visible symptoms. When the late blight symptoms reached 10-25% of leaves area FRAP, NBT and TRC had the highest values (even higher than in symptomless leaves) in *S. pimpinellifolium*. On the contrary, in genotype Bizon in the same disease rank, FRAP and NBT obtained the lowest values, while TRC remained at the same level as a symptomless leaves.

Various methods have been published about the determination of antioxidant activity in different biological systems. Antioxidant activity of selected natural assays as a complex process, must be measured by more than one mode of action [30].

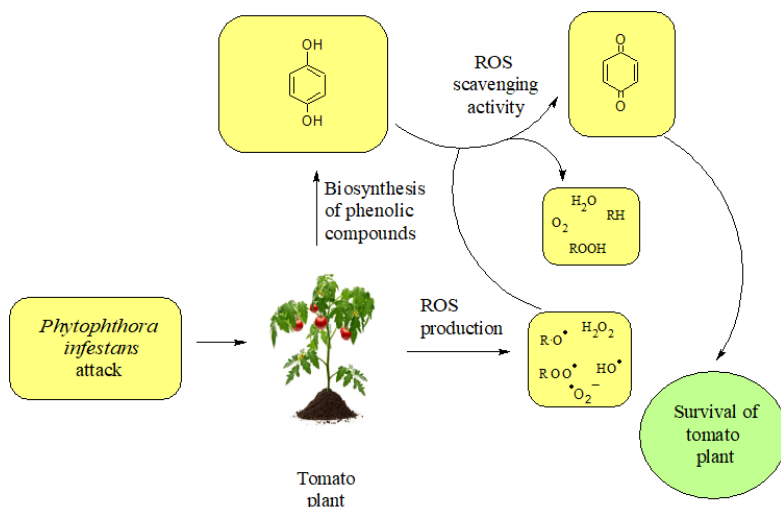
Phenolic compounds belong to the primary defence line in the host-pathogen reaction of tomato and *P. infestans* by inhibiting pathogen penetration [31]. This indicates that at the beginning of the infection plants defend themselves by increasing the antioxidative activity measured by selected assays. Moreover, in our research during the disease development the content of TP and TF in the leaves of tolerant genotype *S. pimpinellifolium* remained at higher concentration values than in Bizon (Figure 1A and B). The similar findings were obtained by Komy et al. [32] who found a significant increase in phenolic compounds in potatoes after infection by *Phytophthora infestans*, with phenolic concentration significantly higher in the resistant plants. These results are in agreement with the earlier reports of Henriquez et al. [33], who suggested the relationship between the accumulation of flavonoids and terpenoids and the late blight level. Also the accumulation of total phenolics correlated with increased potato defence responses to *A. solani* [34]. On the other hand, the high content of TP and TF in tomato leaves is genotype dependent and does not mean the resistance to late blight automatically [35,36].

The delicate balance between ROS generation and ROS scavenging is disturbed by the different types of stress factors among them pathogens infection [37]. The flexible antioxidant system is able to control the optimum ROS levels [38]. In our study, the majority of the antioxidative activity parameters in *S. pimpinellifolium* maintained higher stability than in Bizon, which could influence the disease development. On the contrary, the disease progress (Scheme 1) was much slower in wild genotype. This is in accordance with the results obtained by Liljeroth et al. [39], once the late blight infection had been established, the lesions expanded more rapidly in susceptible than in partially resistant cultivar.



**Figure 1.** A) Total polyphenol content (TP) in tomato leaves B) Total flavonoid content (TF) in tomato leaves; Antioxidant activity in tomato leaves measured by C) Radical Cation Scavenging Activity (DPPH test); D) Radical Cation Scavenging Activity (ABTS), E) Ferric-reducing antioxidant power (FRAP); F) Nitroblue tetrazolium test (NBT test); G) Total Reduction Capacity (TRC); H) Total Antioxidant Activity (TAA) Means of three independent experiments with three replications; Bars represent standard errors.

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**Scheme 1.** Defence mechanism of tomato plant against *P. infestans* attack

Correlation coefficients revealed that almost all parameters (excluded TRC) in Bizon were significantly influenced by leaf infection (Table 3). This negative correlation is in accordance with the strong decrease of these parameters during the disease development, especially comparing the leaves with up to 5% and 5-10%. Despite the fact that TP content in tolerant genotype *S. pimpinellifolium*, increased with the initial symptoms and then remained on the level in symptomless leaves, negative correlation between TP and the leaf disease incidence was achieved. Additionally, strong positive correlation of TP and TAA in both genotypes was obtained. These results are in the accordance with Cai et al. [40] who stated that the higher total phenolic content of the plant resulted in higher total antioxidant capacity.

Surely, that TP and TF have some role in the scavenging of the free radicals and increasing TAA in the initial stage of infection, but such results, indicated that some other non-enzymatic factors are also involved in the tomato defence against late blight. This statement could be confirmed by the fact that there was no significant correlation between TP and certain antioxidative tests (DPPH, FRAP and TRC) in tolerant genotype.

**Table 3.** Correlation coefficients in tomato leaves

Variable	G	TP	TF	DPPH	ABTS	FRAP	NBT	TRC	TAA
leaf infection	<i>S.p.</i>	-0.60*	-0.93**	0.43 ns	-0.19 ns	0.26 ns	0,67*	0,39 ns	-0.77**
	<i>Bizon</i>	-0,88**	-0,83**	-0,84**	-0,88**	-0,86**	-0,99**	-0,39 ns	-0,89**
TP	<i>S.p.</i>		0,54 ns	0,28 ns	0,77**	0,28 ns	-0,58*	-0,15 ns	0,79**
	<i>Bizon</i>		0,94**	0,94**	0,98**	0,97**	0,88**	0,67**	0,95**
TF	<i>S.p.</i>				-0,36 ns	0,15	-0,30 ns	-0,68*	-0,30 ns
	<i>Bizon</i>				0,95**	0,92**	0,96**	0,80**	0,62*

\*\* – significant at 1% level; \* – significant at 5% level; ns non-significant

## CONCLUSIONS

*S. pimpinellifolium* showed less susceptibility to the late blight infection compared to *Bizon*. Genotype (G), late blight infection (LB) and their interaction had a strong influence on biochemical parameters in the leaves. During the progression of late blight symptoms TP, TF and antioxidant activity measured by several assays (except TAA) in susceptible genotype *Bizon*, decreased rapidly. On the other side in wild genotype that decline was much lower. The results obtained in this experiment indicated that, beside TP and TF, some other secondary metabolites with antioxidant capacity are also involved in the tomato defence system against late blight infection.

## EXPERIMENTAL SECTION

The field trial was conducted in 2014 at the experimental field of the Institute of Field and Vegetable Crops (IFVCNS) at Rimski Šančevi, Serbia. Two genotypes from the IFVCNS collection were included in the trial. *Bizon* a susceptible one, the accession originated from Bulgaria, is an early heirloom variety with determinate growth type and *S. pimpinellifolium* accession as a tolerant one. According to our previous research [41] these two genotypes showed significant difference in susceptibility to late blight.

Sowing for seedlings production in a glass house was done on 3rd of April and the plants were transplanted on 27th of May into the open field. The trial was set up in completely randomized block design with three replications



and ten plants in each. The between-row spacing was 140 cm, and within-row spacing was 50 cm. There was no fungicide application and *Phytophthora infestans* natural infection was evaluated.

The evaluation of the early blight symptoms on leaves was performed on 4<sup>th</sup> of August. Ten fully developed leaves per replication were taken from the top of each plant and assessed to the intensity of the late blight infection. Evaluation of the disease intensity on leaves was done according to the EPPO modified scale: 0 - without infection, 1 - less than 5% of leaf affected, 2 - spots covering 5-10%, 3 - spots covering 10-25%, 4 - spots covering 25-50%, 5 - spots covering more than 50% of the leaf.

The severity of the late blight in each leaf was determined using the disease severity index (DSI), calculated according to [42].

$$DSI (\%) = \frac{\sum (n \times v) \times 100}{N \times V}$$

(n = number of leaves per each category; v= value of each category; N= total number of observations; V= maximum value of the category)

Immediately after disease evaluation, leaves were frozen at -80°C for further biochemical analysis.

**Weather conditions** were mostly changeable with temperature fluctuations and higher amounts of precipitation in all months except June. The plants were transplanted in the end of May due to heavy rains. In July, August and September the raining was almost daily with occasional heavy rains which was favourable for the late blight development.

**Analysis of biochemical parameters** were performed after the disease assessment in the Laboratory for Biochemistry, Faculty of Agriculture, Novi Sad, Serbia during 2015. Analysis were done per each genotype and per each category of disease intensity in three replications. Plant material (200 mg) was extracted with 70% aqueous acetone solution (50 mL) by sonication for 20 minutes in an ultrasonic bath at ambient temperature. The extracts were rapidly vacuum-filtered through a sintered glass funnel and kept refrigerated until assayed.

The total phenolic (TP) content was determined in the acetone extracts using a Folin-Ciocalteu colorimetric method [43]. Plant extracts (200 µL) were mixed with 100 µL of saturated sodium carbonate solution and 3 mL of Folin-Ciocalteu reagent diluted with distilled water. The absorbance of the reaction mixture was measured after incubation at ambient temperature for 30 min at

720 nm. Quercetin was used as a standard (covering the concentration range between 0.1 and 1.0 mg/mL). The results were expressed in milligrams of quercetin equivalents per 1 g of leaf dry weight (mg QE/g DW).

The total flavonoid (TP) content was determined spectrophotometrically [44]. Briefly, 0.5 mL of leaf extracts was mixed with 3 mL of 2% AlCl<sub>3</sub> solution. Absorptions were measured spectrophotometrically at 415 nm after 1 h. The amount of flavonoids was calculated as a quercetin equivalent (QE) from the calibration curve of quercetin standard solutions.

Measurement of antioxidative activity. Scavenging of free radicals was tested in a DPPH (2,2-diphenyl-1-picrylhydrazyl) acetone solution [45]. The scavenging efficiency of the substance added is indicated by the degree of discoloration of the solution. Ferric-reducing antioxidant power (FRAP) assay was carried out according to the procedure described by [46]. The results were expressed as mg Trolox equivalents per gram of leaf dry weight (mg TE/g). The ABTS (2,2'-azinobis-(3-ethylbenzothiazoline-6-sulfonic acid)) assay was based on a method developed by [47]. A methanolic solution of known Trolox concentrations was used for calibration and the results were expressed as mg Trolox equivalents per g of dry leaf weight (mg TE/g). The total antioxidant activity (TAA) of leaf extracts was evaluated by phosphomolybdenum method as reported by [48]. The standard curve for total antioxidant activity was plotted using Trolox solution. A reducing power assay (total reduction capacity-TRC) was performed by the method of [44]. Trolox was used as a standard. The superoxide free radical scavenging activity was carried out by NBT (nitroblue tetrazolium) test [48]. The per cent inhibition of superoxide anion generated was calculated using the formula: scavenging activity (%) = (1 - absorbance of sample/absorbance of control) x 100.

**Statistical analysis.** Obtained data were analysed using software *STATISTICA*, ver. 13.2 (Dell, Inc., USA). Values for DSI and biochemical parameters were tested by analysis of variance followed by a comparison of means by Bonferroni test ( $p < 0.01$ ). Correlation coefficients were calculated according to Spearman.

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