Biochemical changes in the leaves of mungbean (Vigna radiata) plants infected by phytoplasma

[Fitoplasma ile enfekte olan mungbean (Vigna radiata) bitkilerinin yapraklarındaki biyokimyasal değişiklikler]

Abstract

Objective: Phyllody disease caused by phytoplasma is an emerging problem in mungbean worldwide. However, the alterations in the host physiology and its associated biochemical components induced by the infection with phytoplasma in mungbean plant remain unknown. Hence the present study was performed with the diseased plants in order to determine the patho-physiological changes that take place.

Methods: Under present study, the changes in total phenolic compounds, total soluble proteins, peroxidase (POD), catalase (CAT), polyphenol oxidase (PPO), Chlorophyll a, Chlorophyll b, and total chlorophyll were studied in leaves of phytoplasma-infected and healthy plants of six mungbean genotypes.

Results: Total phenols were decreased significantly in four but increased in one genotype. Protein contents were increased significantly in two genotypes, POD in four, PPO in one and PAL in two genotypes. However, activity of CAT and amount of Chl a, Chl b and total Chl were decreased significantly in all genotypes.

Conclusion: These results suggest that phytoplasma can interfere with host metabolism and photosynthesis to induce disease. In conclusion, this study provides new insights into the mungbean response to phytoplasma infection.

Keywords: Vigna radiata; Phyllody; Antioxidants; PPO; PAL; Pigments; Phenols; Total protein.

Özet

Amaç: Fitil hastalığı, dünyadaki maş fasülyesi fitoplasma’nın yol açtığı bir sorundur. Fakat, maş bitkisindeki fitoplasma enfeksiyonu ile indiklenen konuççu fizyolojisi ve ilişkili olduğu biyokimyasal bileşenlerdeki değişiklikler bilinmemektedir. Bu nedenle, bu çalışma, hasta bitkilerle patofizyolojik-fizyolojik değişiklikleri belirlemek için gerçekleştirilmiştir.

Yöntemler: Bu çalışmada, fitoplasma ile enfekte olmuş yapraklarda total fenolik bileşikler, toplam çözünülen proteinler, peroksidad (POD), katalaz (CAT), polifenol oksidaz (PPO), klorofil a, klorofil b ve toplam klorofil değişiklikleri ile birlikte altı sağlıklı maş fasüljesi genotipi çalışılmıştır.

Bulgular: Toplam fenoller dört genotiphe azalırken total fenolik bileşikler, toplam çözünülen proteinler, peroksidad (POD), katalaz (CAT), polifenol oksidaz (PPO), klorofil a, klorofil b ve toplam klorofil değişiklikleri ile birlikte altı sağlıklı maş fasüljesi genotipi çalışılmıştır.

Sonuç: Bu sonuçlar, fitoplazmanın konuççu metabolizmasını ve fotosentezle hastalığa müdahale edebileceğini
Mainly cultivated in India, Pakistan, China, Thailand, Philippines, Indonesia, Burma, Bangladesh, Vietnam, as well as in hot and dry regions of Southern Europe and the Southern USA. It is the major kharif pulse crop grown in Pakistan [1]. Mungbean is an important source of food for humans and animals in tropical and subtropical countries, and is also used for green manure. Seed of mungbean contains 24.2% protein, 1.3% fat and 60.4% carbohydrates. It is a short duration crop and can be grown twice a year i.e. in spring and autumn seasons [1]. The ecological conditions of Pakistan are very favorable for the mungbean cultivation. Unfortunately, the same conditions are favorable also for some pathogens like viruses, phytoplasma, bacteria and fungi, responsible for significant economic impact, causing a reduction in income for crop producers. During spring 2008, mungbean plants in Faisalabad, Pakistan, showed severe symptoms of phyllody, virescence and a bushy appearance caused by 16SrI-D phytoplasma [2]. Like many other microorganism phytoplasma infection also leads to the appearance of these symptoms characteristic to specific host-pathogen interactions. The appearance of these symptoms occurs due to alteration of physiological, biochemical and metabolic processes within the infected plants. Thus, various antioxidant enzymes such as peroxidase (POD), superoxide dismutase (SOD), catalase (CAT) and polyphenol oxidase (PPO) participate in ROS metabolism during the pathogen attack. High expression of these defence enzymes makes the plant resistant to pathogen invasion [3, 4]. However, these mechanisms fail when the plant is infected by a virulent pathogen.

Studies on physiological relationships between phytoplasmas and some other host plants have been well documented. However, the alterations in the host physiology and its associated biochemical components induced by the infection with phytoplasma in mungbean plant remain unknown. Hence the present study was performed with the diseased plants in order to determine the pathophysiological changes that take place.

### Materials and methods

Mungbean leaves used in this study were collected from field grown phytoplasma infected and healthy plants of six genotypes (MHP-291, MH-169, MH-933, MH-263, MH-276, and VC-52). To detect phytoplasma total DNA was extracted from leaves collected from symptomatic and non-symptomatic plants using CTAB method [5]. PCR amplification of 16S rRNA gene was performed using phytoplasma specific universal primers P1/P7 [6].

For the estimation of total phenols 1 g leaf samples from phytoplasma infected and healthy plants of each test genotypes were collected after pod formation on healthy plants. Leaves were cut into small pieces and then put into the smearing methanol until the green color was removed. Leaf tissues were homogenized after decanting the methanol in the Polytrone and then again boiled in methanol for further 5 min and filtered through Whatman No. 1 filter paper. Residual material was washed with 80% acidified (0.1% HCL conc.) methanol. The concentration of phenolic compounds in the leaves of phytoplasma infected and healthy plants was determined by using the method described by Bray and Thorpe [7].

To measure total protein contents fully emerged leaves (0.5 g) from phytoplasma infected and healthy plants of each test genotypes were ground in cold extraction potassium phosphate buffer (pH 7.0). Samples were centrifuged and the supernatant was separated. Total soluble protein concentration was measured by dye binding assay as described by Bradford [8].

For the estimation of peroxidase (POD) activity, leaves from phytoplasma infected and healthy plants of each test genotypes were homogenized in a solution composed of 50 mM potassium phosphate buffer (pH 7.0), 0.1 mM EDTA and 1 mM dithiothreitol (DTT). Activity of POD was measured using the method of Chance and Maehly [9] with some modification. One unit POD activity was defined as an absorbance change of 0.01 unit min⁻¹ [10].

To measure catalase (CAT) activity leaves from both phytoplasma infected and healthy plants of each test genotypes were thoroughly mixed in a medium composed of 50 mM potassium phosphate buffer, pH 7.0 and 1 mM dithiothreitol. Enzyme activity was measured using the method described by Hameed et al. [11].

To measure Polyphenol oxidase (PPO) activity leaf tissue samples were cut into small pieces of about 5 mm long from each of the treatment and were ground in liquid nitrogen using a mortar and pestle. PPO activity was measured as described by Jockusch [12]. The absorbance at 546 nm was measured for 4 min at 20 s intervals,
and the values per minute were calculated and the results were presented as U μL⁻¹ min⁻¹.

Leaf tissue samples (2.5 g) from phytoplasma infected and healthy plants of each test genotypes were cut into small pieces of about 5 mm long to measure Phenylalanine ammonia-lyase (PAL) activity as described by Okey et al. [13]. One activity unit was defined as a change in absorbance of 0.01 at 290 nm h⁻¹ g⁻¹ protein. PAL activity was expressed as mg/g fresh weight.

The Chlorophyll a, Chlorophyll b and total chlorophyll contents were estimated from leaves of both phytoplasma infected and healthy plants of each test genotypes using standard procedure of Lichtenthaler [14]. For these purpose leaf samples of 0.25 g were added in 10 mL 80% acetone, grounded in the presence of sand in pestle and mortar and then filtered through muslin cloths. The absorbance of extract was measured at 663, 645, 505, 453 and 470 nm wavelengths, and above mentioned pigments were calculated according to Davis [15].

The data collected from all experiments was analyzed separately for each parameter and subjected to two ways Analysis of Variance (ANOVA) using XL-STAT 2012. The means were compared for significance using Tukey’s HSD test and the values presented are mean of three replicates ± standard error (SE).

**Results and discussion**

All mungbean genotypes tested showed variable levels of infection with susceptible response to phyllody disease in the field. Infection percentage values for mungbean genotypes MHP-291, MH-169, MH-933, MH-263, MH-276, and VC-52 were 45%, 40%, 50%, 48%, 45% and 46%, respectively. The first symptom of disease to be detected was reduction in leaf size on a few plants of all genotypes 30 d after sowing, which later on developed into severe symptoms. Severity of phyllody was found to be associated with the time of infection. Plants infected during early stages of development (before flowering) showed severe symptoms on whole plants while plants infected later (at the time of flowering and pod setting) showed severe symptoms on the upper part only. Early infection caused severe reduction in leaf size, phyllody, inhibition of anther formation; stunting and plant became sterile, resulting in a total loss of yield. However, plants infected at the flowering and pod setting showed severe virecence, as the entire inflorescence was transformed into green leaf-like structures followed by abundant vegetative growth giving bushy appearance as previously reported by Akhtar et al. [2].

The presence or absence of phytoplasma was confirmed by PCR in symptomatic and non-symptomatic plants using the 16S rRNA gene specific universal phytoplasma primers P1/P7. All the samples collected from symptomatic plants gave a 1250 bp PCR product confirming the phytoplasma as disease causative agent, whereas no amplifications were observed from non-symptomatic plants. Symptomatic plants found positive and non-symptomatic plants found negative for phytoplasma were further processed for biochemical studies.

Phenolics are well-known antimicrobial compounds occurring in plants and function as precursors to structural polymers such as lignin, or serve as signal molecules to activate plant defense genes [16, 17]. In present study, there was no significant difference in the total phenols of healthy plants of all tested genotypes except one genotype MH-276 (Figure 1). However, in phytoplasma-infected plants total phenols were significantly higher in leaves of

![Figure 1: Total phenolics of healthy and phytoplasma infected plants of different mungbean genotypes.](image_url)

Different letters on the top of bars indicate significant differences between genotypes for each treatment at p ≤ 0.05.
MH-169 but significantly lower in MH-276 and VC-52 over their healthy plants. But there was no statistical difference in amount of total phenols in healthy and diseased plants of MHP-291, MH-933 and MH-263 (Figure 1). The maximum amount of total phenols was observed in symptomatic plants of genotype MH-169.

Phenolic compounds are thought to be involved in the defense in plants and their increased accumulation after infection could be related to the resistance mechanism of the host [18, 19]. In the present work, all the tested genotypes were susceptible to phytoplasma but total phenols were statistically at par in diseased plants of MHP-291, MH-933 and MH-263 but increased significantly in MH-169. Usually high level of phenolic contents in plants after infection that has been correlated with increased resistance [10] but Junqueira et al. [19] pointed out that phenolic compounds is not only related to the resistance but also that those compounds have other functions in plant tissues. A possible explanation for decrease in phenolic compounds in diseased plants of most genotypes is that many plant pathogens actively suppress the expression of plant defense reactions during successful infection [10, 20].

Many plant pathogenic interactions recognized the involvement of protein components in plant disease resistance [21, 22]. Induction of defense proteins makes the plant resistant to pathogen invasion [23]. In present study, total proteins were statistically at par in healthy plants of MHP-291, MH-169, MH-933 and VC-52 but significantly less in MH-263 and MH-276 (Figure 2). However, significant increase in protein contents was observed in leaves of phytoplasma-infected plants of MH-263 and MH-276 while statistically at par in MHP-291, MH-169, MH-933 and VC-52 over their healthy plants (Figure 2). Generally infected plants show a high protein contents, which could be due to both the activation of the host defense mechanisms and the pathogen attack mechanisms [24].

POD is one of the first antioxidant enzymes responding and providing fast defense against plant pathogens by participating in a variety of defence mechanisms [4, 10, 25]. POD is involved in the production of reactive oxygen species, which are directly toxic to the pathogen or indirectly reduce the spread of the pathogen by increasing the cross-linkage and lignification of the plant cell walls [26, 27]. POD is required for the final polymerization of phenolic derivatives into lignin and involved in suberization or wound healing [28]. Increased POD activity was observed in a number of resistant interactions involving plant pathogenic fungal and bacterial diseases [29, 30, 31]. Under present study, significant differences in the POD activity was observed in healthy plants of all the tested genotypes and maximum POD activity was observed in MH-169 (Figure 3). However, significant increase in POD activity in leaves of infected plants over their healthy plants was observed in MHP-291, MH-169, MH-933 and MH-263 while it remained unchanged in MH-276 and significantly decreased in VC-52 (Figure 3). Observed increase in POD activity in present study can be explained on the bases of earlier observations of Wallis et al. [32] who demonstrated that pathogen infections can increase the level of certain enzymes including peroxidases within plants. Interestingly, in phytoplasma-infected plants total phenols and POD activity were significantly higher in leaves of MH-169 that point out their co-involvement in disease susceptibility. However, this aspect needs to be further tested.

CAT is an oxygen-scavenging enzyme that removes toxic substrates (H$_2$O$_2$) during development, which are otherwise lethal [27, 33]. CAT activity was significantly different in healthy plants of all tested genotypes and maximum...
was observed in healthy plants of VC-52 (Figure 4). However, CAT activity was significantly decreased in leaves of diseased plants of all the tested genotypes (MHP-291, MH-169, MH-933, MH-263, MH-276, and VC-52) over their healthy plants (Figure 4). Results of present studies agree with Hernandez et al. [34], who observed decreased CAT activity in two apricot cultivars infected with plum pox virus. The reduction in CAT activity could be a consequence of enhanced proteolysis caused by peroxisomal endopeptidases, which are induced by oxidative stress [35], as previously is in the case of *Pyricularia oryzae* infection in wheat [15]. However, the reduction of CAT activity usually increases plant resistance to pathogenic attack because plants can maintain high concentrations of H$_2$O$_2$ [36]. Thus, the role of CAT in the plant–pathogen interaction seems to be more complex than for abiotic stress [37], which involves an association between CAT activity and plant tolerance [15, 38].

PPO is an enzyme of broad distribution in plants and is important in the initial stage of plant defense where membrane damage causes release of phenols such as chlorogenic acid. PPO catalyzes the oxidation of phenolics to free radicals, which can react with biological molecules, thus creating an unfavorable environment for pathogen development [12, 39]. PPO activity was significantly different in healthy plants of all tested genotypes and maximum PPO activity was observed in healthy plants of MH-933 (Figure 5). However, PPO activity was significantly increased in leaves of phytoplasma-infected plants of MH-933 but decreased in MH-169, MH-276 and VC-52 while statistically at par in MHP-291 and MH-263 over their healthy plants (Figure 5). Pathogen infections also increase the level of certain enzymes within plants, including those that could attack cell wall of pathogens and those that create/maintain environments unsuitable for pathogens (i.e. peroxidases and polyphenol oxidases) [32]. These mechanisms against a pathogen fail when the plant is infected by a virulent pathogen, which avoids triggering or suppresses resistance reactions or evades the effects of activated defenses [4].

![Figure 3: Peroxidase activity of healthy and phytoplasma infected plants of different mungbean genotypes. Different letters on the top of bars indicate significant differences between genotypes for each treatment at p ≤ 0.05.](image)

![Figure 4: Catalase activity of healthy and phytoplasma infected plants of different mungbean genotypes. Different letters on the top of bars indicate significant differences between genotypes for each treatment at p ≤ 0.05.](image)
According to Ray et al. [40] antioxidative enzymes (POD and PPO) may participate in the responding defense reaction by inducing plant resistance against pathogenic agents. Present results agree with the findings of Lin and Kao [41] and Borden and Higgins [42] who found a correlation between increased antioxidant enzyme activities and pathogen resistance in plants.

Phenylalanine ammonia-lyase is the primary key enzyme in the synthesis of the secondary compounds, endogenous signaling molecule salicylic acid, which in turn activates the expression of a variety of PR genes [27]. Under present study, PAL activity was statistically at par in healthy plants of MH-169, MH-933 and MH-276 while comparatively high in MH-263 (Figure 6). However, significant increase in PAL activity was observed in leaves of infected plants of genotypes MH-169 and MH-933 while it was significantly decreased in MH-263 and MH-276 over their healthy plants (Figure 6). PAL is the primary enzyme in the phenylpropanoid metabolism and plays a significant role through the formation of cinnamic acid, an important compound in the biosynthesis of phenolics and lignin [43–45]. Inhibition of PAL affects subsequent biosynthetic pathways of phenolic compounds [16]. It was demonstrated that reduction of phenylpropanoid metabolism through inhibition of PAL activity in transgenic tobacco also rendered tissues more susceptible to Cercospora nicotianae [46].

Phytoplasma infected plants of all tested genotypes showed a significant decrease in the concentration of Chl a, Chl b and total Chl compared to non-infected plants (Figure 7A–C). Several studies have revealed that pathogen infections reduce Chl a and b contents [47–49] and here the reduction in chlorophylls shows that the phytoplasma can interfere in photosynthesis as previously reported by Bertamini et al. [50]. Thus, it can be assumed that the observed reduction in chlorophyll levels will probably interfere with the photosynthetic capacity in the mungbean leaves as in papaya dieback and corn infected with maize bushy stunt phytoplasma pathosystems [19]. The phytoplasma infection might affect chlorophyll molecules by degradation or synthesis inhibition.
Conclusion

Leaves infected with phytoplasma depicted damage to physiological and biochemical processes and resulted in diverse and severe symptoms in infected plants. As a general stress response phenomenon, significant decline in H$_2$O$_2$ scavenging antioxidant enzyme CAT and photosynthetic pigments was detected in infected leaves of all genotypes. In phytoplasma-infected plants, total phenols, PAL and POD activities were significantly higher in leaves of MH-169 having least infection percentage (40%) that point out their co-involvement in disease tolerance. However, this aspect needs to be further tested. The information gained can then be used for better understanding the mechanisms of plant response to pathogen infection.

Conflict of interest statement: There is no conflict of interest between authors.

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