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# Effect of *Fusarium oxysporum* f. sp. ciceri on cell membrane stability in chickpea

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**Abstract**
Cell membrane stability in relation to drought tolerance is currently being studied in crop plants. In the present study, this parameter was measured to find out how cell membrane of chickpea is getting affected during disease incidence caused by *Fusarium oxysporum* f. sp. ciceri. The cell membrane injury was found to be directly proportional to severity of infection. It increased with severity of infection thus it was highest in heavily infected seedlings in both samples at both 20th and 30th day of sowing. The changes were very much evident in all samples for naturally infected and artificially inoculated by *Fusarium oxysporum*. In artificially inoculated seedlings cell membrane injury was in between of moderately and heavily infected seedlings. It increased at the time of incubation. Seedlings of 30 days showed higher injury than 20 days seedlings of all the categories.

**Keywords:** Cell membrane injurey, *Fusarium oxysporum* f. sp. ciceri, chickpea etc

## Introduction

Plants face a continuous danger from many pathogens, yet the actual occurrence of illness is rare (O'Connell and Panstruga, 2006) [2]. The success of a disease is primarily determined by the disruptions in the highly organized host immune system (Jones and Dangl, 2006) [3]. Plants and animals with ancient innate immunity exhibit both structural and strategic resemblances (Ronald and Beutler, 2010) [4]. Furthermore, animals possess a specific adaptive immune system that mobilizes mobile defender cells to the locations where pathogens invade. This immune response leads to the elimination or containment of the pathogens (Ausubel, 2005) [5]. Regrettably, plants do not possess this characteristic, and their immobile nature prevents them from evading diseases. However, plants have been endowed by nature with intricate and coordinated signal transduction machinery to make up for the absence of adaptive immunity (Dodds and Rathjen, 2010) [6]. Défense signals initiated by pathogens at a central point activate various subsequent reactions, resulting in the reinforcement of the cell wall, targeted movement of stored defence compounds, production of new protective substances such as phytoalexins, small molecules, and secondary metabolites, creation of reactive oxygen and nitrogen compounds, and buildup of phytohormones and pathogenesis related proteins. The combination of these characteristics results in a hypersensitivity response (HR)-mediated programmed cell death (PCD) at the site of infection (Garcia-Brugger *et al*., 2006). Nevertheless, despite the various signals, the precise sequence of events that result in a highly organized defence network remains unclear.

The seed and/or soil carried pathogen is capable of infiltrating the host by exploiting gaps between the main root and its branches, then establishing itself within the xylem vessels located at the junction of the root and shoot. The pathogen consists of two distinct pathotypes: the yellowing type and the wilt-inducing type. The latter is notorious for causing significant destruction when environmental conditions are suitable (hare and Nene, 1982) [1].

One of the races of *Fusarium oxysporum* f. sp. ciceri, which causes wilting, is widely distributed geographically and leads to significant crop damage. While wilt is mostly controlled by breeding programs, the presence of several pathogenic races and their propensity to change can result in the failure of natural resistance (Gupta *et al*., 2013) [7].

In the present study, we worked to observe *Fusarium oxysporum* induced cell membrane injury in chickpea.

## Sample collection

Chickpea seeds were obtained from 5 different districts of Rajasthan those were- Alwar, Bharatpur, Dholpur, Karauli and Kota. Morphologically unhealthy seeds were incubated on PDA and among all the isolated fungi, *Fusarium oxysporum* was selected for the study.

Chickpea seeds were grown as healthy, weakly infected, moderately infected, heavily infected, and artificially inoculated. After 20 and 30 days, seedlings were studied for membrane stability.

## Determination of Cell membrane injury

The percent cell membrane injury will be measured using the method described by Sullivan (1972) [9], employing the equation as:

% Injury in normal tissue = conductivity of tissue/ Total conductivity *100.

% Injury in stressed tissue = conductivity of tissue/ Total conductivity *100.

% Uninjured tissue = 100-% Injury

% Membrane Injury = 100 – (% uninjured stressed tissue / % uninjured normal tissue) *100

## Results and discussion

Cell membrane stability in relation to drought tolerance is currently being studied in crop plants. In the present study, this parameter will be measured to find out how cell membrane is getting affected during disease incidence. The cell membrane injury was directly proportional to severity of infection. It increased with severity of infection thus it was highest in heavily infected seedlings in both samples at both 20th and 30th day of sowing. The changes were very much evident in all samples for naturally infected and artificially inoculated by *Fusarium oxysporum*. In artificially inoculated seedlings cell membrane injury was in between of moderately and heavily infected seedlings. It increased at the time of incubation. Seedlings of 30 days showed higher injury than 20 days seedlings of all the categories (Table 1 and Figure 1).

All experiments were performed in triplicates. One way analysis of variance (ANOVA) was used to show significance of difference with respect to control. In all experiments ρ value was found to be lower than 0.05 which indicate that difference were statistically significant.

Reactive oxygen species have significant functions in perceiving and recognizing pathogens, as well as in signaling for subsequent defensive mechanisms. However, the mechanism by which these redox alarms coordinate throughout a plant's defensive network remains elusive. This study demonstrates the involvement of redox-responsive genes generated by *Fusarium oxysporum f.sp ciceri* Race1 (Foc1) in modulating downstream defensive signaling in chickpea. Confocal microscopy revealed the infiltration and colonization of pathogens in the xylem vessels of infected chickpea plant roots, resulting in tissue destruction and the accumulation of degraded callose products. These depositions caused blockages in the xylem vessels of suitable hosts, but the resistant plants did not have these obstructions. Fungal activity was found to cause membrane damage, as evidenced by lipid peroxidation experiments. The occurrence of cell shrinkage and the steady movement of the nucleus towards the centre are notable characteristics that indicate the entry of fungi. The application of quantitative real-time polymerase chain reaction revealed distinct expression patterns of redox regulators, cellular transporters, and transcription factors during the course of Foc1. Network research revealed that redox regulators, cellular transporters, and transcription factors collaborate to form a highly organized defense network. Additionally, sugars were identified as internal signal modulators within this network. The respiratory burst oxidase homologue, cationic peroxidase, vacuolar sorting receptor, polyol transporter, sucrose synthase, and zinc finger domain containing transcription factor have been identified as crucial molecular possibilities that regulate major nodes in the defense network. Characterizing the functions of these central controllers could be a valuable approach to gain insights into the relationship between chickpea and Foc1, and to establish a case study that can be used as a model to investigate the intricate nature of wilt diseases in other significant legume crops (Gupta *et al*., 2013) [7].

Cell membrane injury may also be due to damage of structural lipids located in host cell membrane (Rella *et al*., 2015) [8]

**Table 1:** % Cell membrane injury in seedling of healthy (control), naturally infected (weakly, moderately and heavily) and artificially inoculated on 20th and 30th day of sowing

| S. No. | Category of seedlings | % Cell Membrane injury in | |
|---|---|---|---|
| | | Seedling of 20 days | Seedling of 30 days |
| 1. | Healthy | 1.31 ±0.38 | 1.68 ± 0.04 |
| 2. | Weakly infected | 30.40 ± 0.81 | 44.88 ± 1.23 |
| 3. | Moderately infected | 42.71 ± 1.31 | 58.45 ± 1.00 |
| 4. | Heavily infected | 54.95 ± 1.45 | 62.57 ± 0.88 |
| 5. | Artificially inoculated | 50.11 ± 1.47 | 61.32 ± 0.26 |

The values indicated in the table are the mean of three replications with standard error of mean (±SEM)

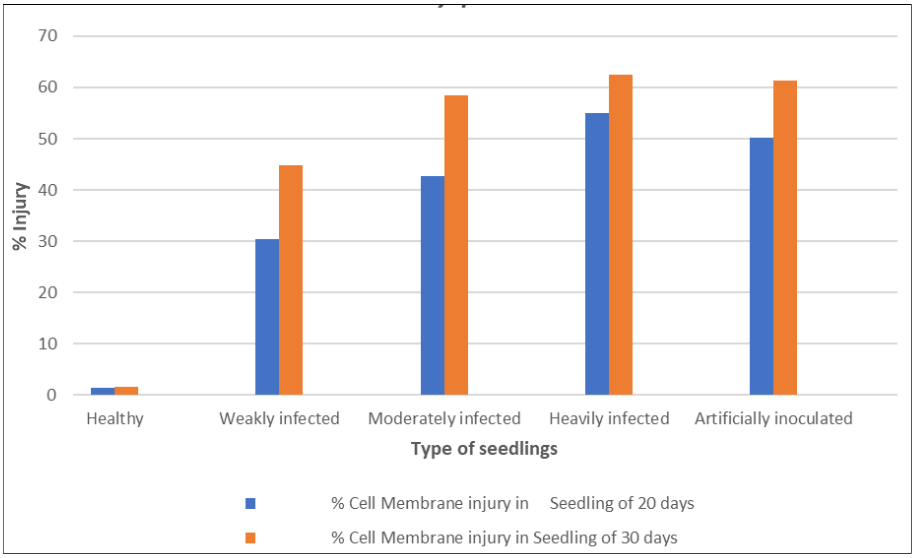


**Fig 1:** Percent cell membrane injury in seedlings infected by *F. oxysporum*


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