SUMMARY
PART I: INDUCED DISEASE RESISTANCE IN SOLANUM TUBEROsum PLANTS AGAINST NECROTROPHIC, BIOTROPHIC AND HEMIBIOTROPHIC PATHOGEN

In our study, disease resistance in two resistant (BD & BH) and two susceptible (PK & LO) varieties of *S. tuberosum* upon treatment of the FCF of necrotrophic, biotrophic and hemibiotrophic pathogens was studied. Higher FRAP value and lipid peroxidation was seen in *S. tuberosum* plants treated with the FCF of necrotrophic pathogen. Also activity of SOD and CAT was found higher, where as NOD activity was lower in *S. tuberosum* plants treated with the FCF of necrotrophic pathogen. These results showed that high amount of ROS were produced upon necrotrophic pathogen infection. Activities of GR, NOD and GSH concentration were found higher in *S. tuberosum* plants treated with the FCF of the hemibiotrophic and biotrophic pathogen as compared to the FCF of the necrotroph. This showed that *S. tuberosum* plants resisted the hemibiotrophic and biotrophic pathogen by generation of the defense mechanism. Octadecaniod pathway (JA and LOX activity) was induced higher in the *S. tuberosum* plants treated with FCF of necrotrophic pathogen, as compared to the *S. tuberosum* plants treated with FCF of hemibiotrophic and biotrophic pathogens. However Phenyl propanoid (SA and PAL) pathway was induced higher in the *S. tuberosum* plants treated with FCF of hemibiotrophic and biotrophic pathogens, however the induction was poor in the *S. tuberosum* plants treated with FCF of necrotrophic pathogen. Overall from these results it was seen that HR was very weak in the *S. tuberosum* plants' interaction with the necrotrophic pathogen, however strong HR was evidenced in the *S. tuberosum* plants' interaction with the hemibiotrophic and biotrophic pathogens.
Maximum POX activity and higher degradation of the chlorophyll was observed in the *S.tuberosum* plants treated with the FCF of necrotrophic pathogen, whereas comparatively lower POX activity and lower chlorophyll degradation were seen in *S.tuberosum* plants treated with the FCF of biotrophic and hemibiotrophic pathogens. Total phenol induction was higher in the *S.tuberosum* plants treated with the FCF of hemibiotrophic pathogen and biotrophic pathogens and lower in the *S.tuberosum* plants treated with the FCF of necrotrophic pathogen. Susceptible varieties were found to mount quantitatively lower resistance as compared to the resistance varieties.

It was found that Kaempferol diglucoside 7 glucoside and ferulic acid were induced in Necrotrophic, hemibiotrophic and biotrophic interactions. Sinapic acid, Rosmarinic acid were found in the biotrophic interaction, whereas Isochlorogenic acid, caffeic acid and ferulo quinic acid were found induced in the necrotrophic interaction. From the results of the Maule’s test it was seen that S lignin is increased in the biotrophic interaction where as G lignin formation is increased in the necrotrophic interaction, where as hemibiotrophic interaction involves induction of both S and G lignin in *S.tuberosum* plants.
PART II: OXALIC ACID AS A KEY MOLECULE FOR DISEASE RESISTANCE IN 
SOLANUM TUBEROSUM PLANTS AGAINST NECROTROPHIC PATHOGEN

It was observed that FCF of the necrotrophic pathogen showed higher acidic pH and higher amount of OA as compared to the hemibiotrophic and necrotrophic pathogens. Hence effect of role of OA in necrotrophic pathogen interaction with S.tuberosum plant was studied. S.tuberosum plants were treated with 1) A.solani FCF 2) A.solani FCF + 5 mM Oxalic acid 3) 10 mM Oxalic acid 4) Water treated control.

Treatment with exogenous OA produced similar symptoms in the plants as seen after infection with A.solani. DAB staining showed that the combined treatment of A.solani FCF + OA was found to induce higher H$_2$O$_2$.

Hydrogen peroxide detection using DAB staining showed that, hydrogen peroxide was found in the order of A.solani FCF + OA > A.solani FCF > OA on sixth day in S.tuberosum plants.

OA was found to induce rapid cell death and ion leakage in S.tuberosum plants, however once the pathogen established on plants, higher cell death was found in the A.solani FCF and OA+ A.solani FCF treated plants as compared to the OA treated plants. Which showed that OA contributed to the cell death and ion leakage.

Treatment of the OA and A.solani FCF + OA resulted to the increase in the CAT and SOD activity in S.tuberosum plants, which may be to the detoxify H$_2$O$_2$which was generated by the degradation of OA. Results of the NOD activity showed that for the early intervals NOD activity was mainly as a result of the OA treatment, however in the delayed intervals, A.solani FCF treatment mainly contributed to NOD activity.
Lipid peroxidation and FRAP value was also highest in the OA + *A. solani* FCF treated plants. At early stages and late stages higher lipid peroxidation was observed for the OA and OA + *A. solani* FCF treated *S. tuberosum* plants respectively, which showed that OA degraded the OA, which contributed to the H$_2$O$_2$ production. OA and FCF +OA treatment led to rapid and delayed increase respectively in the FRAP value, that resulted from Fenton reaction (H$_2$O$_2$ degradation).

Higher Lignin deposition resulted from the necrotrophic pathogen treatment as compared to OA treatment, which showed that lignifications was resulted as a physical barrier to resist the infection process. Resistance responses were found higher in the resistant varieties as compared to the susceptible ones.
PART III: STUDY OF PATHOGENESIS RELATED PROTEINS (PRs) IN SOLANUM TUBEROSUM PLANTS AGAINST BIOTROPHIC PATHOGEN.

Disease resistance mechanism as seen by induction in the PR proteins in *S. tuberosum* plants upon treatment with the FCF of necrotrophic, biotrophic and hemibiotrophic pathogens was studied.

Treatment with Hemibiotrophic and biotrophic pathogens led to the induction of the higher amount of protein as compared to the necrotrophic pathogen infection. One of the pathogenesis related protein of mass 65.9 kD was induced differentially with the FCF of *P. infestans*.

Based on molecular weight, it was hypothesized to be peroxidase (PR9), however it was found to be absent in activity staining. This pathogenesis related protein was confirmed to be NPR1 by rabbit polyclonal antibodies to *A. thaliana* NPR1. A band size more than 205 kD equivalent to oligomeric form of the NPR1 protein was also detected in native PAGE.

NPR1 protein was found present in both resistant and susceptible varieties of *S. tuberosum*. NPR1 protein was also inducible upon treatment of the FCF of these pathogens. The PR protein β1,3_Glucanase (PR2) and Chitinase (PR3) activities were also induced upon the treatment of the FCF of hemibiotrophic and biotrophic pathogens, where as the induction was low in necrotrophic pathogen.

Induction of SA, NPR1, β1,3_Glucanase (PR2) and Chitinase (PR3) evidenced that SAR was active against hemibiotrophic and biotrophic pathogens, however SAR induction was found weak against necrotrophic pathogen.