Summary

Clubroot disease, primarily occurring in crucifers, is caused by a soil-borne protist *Plasmodiophora brassicae* (*Pb*) which obligately leads a biotrophic lifestyle. A conspicuous symptom of disease progression is the formation of complex galls or tumour-like structures on the belowground parts of infected Brassicaceae hosts. This allows the usage of the model plant, *Arabidopsis thaliana*, as an ideotype for studying the biology of this peculiar plant-pathogen interaction in a deep and functional manner. A clubroot gall acts as a strong physiological sink for supporting the metabolic and nutritional demands of the developing pathogen, which is at least partially achieved by strong cellular reprogramming of existing regions of meristematic activities, resulting in increasing cell number and size, as well as vascular tissue repatterning. Xylem development and maturation within galls gets strongly supressed, while phloem preferentially proliferates.

To completely understand how *Pb* triggers cell proliferation to develop galls in a discrete and non-randomised manner to induce and repress formation of different tissue types, deep developmental and anatomical studies are required. This necessitates the optimisation of a standard experimental method that can be effectively applied for tracking and observing internal changes that accompany gall formation by providing a cellular resolution. For this reason, initially, the research work focused on forging and modifying existing fixation and clearing methods to develop a pipeline for permitting good quality fluorescent imaging in dense and opaque clubroot galls to visualise genetically encoded fluorescent proteins and combine fluorescent stains for observing pathogenic structures. This proved instrumental for our next task which focused on understanding the role of *PHLOEM EARLY DOF* (*PEAR*) genes in ectopic phloem production within clubroot galls.

The work on *PEARs* centred mainly around three *PEARs* including *PEAR1*, *DOF6*, and *HIGH CAMBIAL ACTIVITY2* (*HCA2*) which showed transcriptional upregulation in the previously published RNA-Seq dataset, and this was further confirmed by using promoter fusion lines for these genes within proliferating phloem during gall development. We then proceeded by analysing single and higher order mutants of different *PEARs* to see if *Pb* infection could still actuate phloem production in these plants which have inherent phloem-associated defects. However, despite their induction upon infection, the mutants failed to show reduced pathogen driven-phloem proliferation. This indicated that the observed developmental changes within the phloem tissue reflects alterations in upstream regulatory pathways.

Considering that so far the modulation of *PEAR*s has been observed in response to auxin and cytokinins, we compared classical auxin reporter *DR5* and cytokinin reporters *TCS* and *ARR5* within developing galls and found auxin signalling responses to be highly induced across host vascular tissues in comparison to cytokinin signalling responses which appeared to be mostly dampened. Finally, we employed a variety of chemical inhibitors to chemically disturb auxin homeostasis during disease progression to check if it could counteract the induction of *PEARs*, specifically of *DOF6*. Using these chemicals drastically affected host vasculature in terms of development and patterning in different ways; however, in all instances, *DOF6* induction upon *Pb* infection was not deterred.

Overall, from this work we can infer that *PEAR* induction upon *Pb* infection may not be a definitive strategy employed by the pathogen to exclusively overdrive phloem production within galls. Rather, *PEAR* upregulation occurs as a downstream effect of cumulative changes in vascular cell fates resulting from the altered proliferative status of cells destined to enter the phloem cell lineage.