Clubroot is a devastating plant pathogen that, without the excellent work of breeders, would be difficult and expensive to manage. Defining the earliest recognition events occurring when plants are exposed to clubroot spores and understanding how the pathogen is able to surmount host defences is very important. This project aims to examine the role of chitin in these events, information which will be of great use in the development of disease control strategies whether by biocontrol or through crop improvement.

The cell walls of fungi, insects and crustaceans are formed largely of chitin, making these chains of N-acetylglucosamine residues one of the most abundant biopolymers in the world. The ubiquity of chitin amongst the parasites and herbivores that assail plants make it a potent motif for their detection of non-self. The receptors and signalling components that mediate recognition of chitin oligomers and initiation of defence responses have been characterised in Arabidopsis, rice and wheat. *Lotus japonica* and *Medicago trunculata* employ parallel recognition machinery to detect chitin related lipochitooligosaccharides derived from beneficial mycorrhizae, in this instance triggering symbiosis programmes. Thus, for the appropriate response to friend or foe, recognition of chitin is an important asset for plants. Successful, adapted pathogens must overcome the surveillance mechanisms of plants and subvert defence responses in order to colonise and parasitise their hosts. One strategy adopted by plant pathogens is the secretion of effector proteins that can act extracellularly or inside host cells to manipulate events in their favour. The mode of action of plant pathogen effectors is highly varied but recent discoveries concerning the rice blast fungi *Magnaporthe oryzae,* the wheat pathogen *Mycosphaerella graminicola* and the tomato leaf mould *Cladosporium fulvum* have revealed the significance of chitin binding effectors in both the evasion of detection and defeat of an important component in plants' defensive arsenal. Amongst the first pathogen responsive proteins to be identified in plants, chitinases hydrolyse chitin and play an antimicrobial role by digesting pathogen cell walls, with the additional effect of releasing chitin oligomers for host detection and signal amplification. Fungal effectors with LysM domains can bind to chitin, blocking its recognition by receptors and preventing chitinase hydrolysis, thus diminishing host defence signalling and protecting the fungi from degradation.

Cell walls of *Plasmodiophora brassicae* resting spores contain 25% chitin. This obligate pathogen is the causal agent of clubroot disease, responsible for significant losses to brassica crops each year. Arabidopsis seedlings exposed to *P. brassicae* spores and seedlings treated with chitin will respond similarly by up-regulating expression of defence genes. However, at later stages of infection hundreds of chitin-induced genes, including chitin receptors, are suppressed by *P. brassicae*. We hypothesise that silencing of chitin-mediated immune signalling is an important facet of clubroot virulence. Chitinase genes are among those suppressed during infection and there is evidence of differential timing in chitinase activity between resistant and susceptible lines of Chinese cabbage responding to *P. brassicae*. Assessing the contribution of host chitinases to defence against clubroot infection should complement our investigation of chitin mediated responses. The genome of *P. brassicae* has recently been sequenced; while no LysM domain proteins were identified the predicted secretome was enriched with proteins containing CBM18 chitin binding motifs. Several of these are likely chitin deacetylases, enzymes which remodel chitin to chitosan, a less potent elicitor of immunity and poorer substrate for chitinases. Other chitin binding proteins have no clear functional designation and we propose characterising their potential roles in *P. brassicae* infectivity.

This project aims to develop a sophisticated understanding of the role of chitin in the Arabidopsis – *P. brassicae* interaction from the perspective of both host and pathogen. Regulation of host chitin perception components will be monitored throughout the infection process and their contribution to resistance assessed by analysis of mutant lines. Similarly, chitinase activity will be measured at key time-points and, by combining multiple knock-out lines and artificial miRNAs that silence gene-clusters, we aim to substantially reduce chitinase activity and quantify the effect on *P. brassicae* infection. To complement this approach we will generate and characterise transgenic lines ectopically expressing endogenous chitinases as well as heterologous chitinases and chitosanases. The extensive genetic resources available for both Arabidopsis and *P. brassicae* will be harnessed to examine chitin-associated infection characteristics arising from natural variation. Functions of *P. brassicae* secreted proteins with chitin binding motifs will be investigated both *in vitro* and *in planta* to determine their roles in pathogenicity.

Clubroot is a major blight on brassica production; characterising the early events of pathogen recognition and subsequent responses at the cellular level will form an important milestone for strategies to harness and optimise host resistance against *P. brassicae*. It is important that we make good use of the genome information now available by propelling forward our understanding of the plant-pathogen dynamic and developing models for resistance mechanisms that can be tested in crop species and under field conditions. The responses we identify in Arabidopsis will be tested in *Brassica napus* to confirm their conservation and the applicability of these findings to crop research.