Infections with parasitic worms are widespread, serious and very prevalent. They are based on an ability to manipulate the host's immune system. The precise mechanisms of the manipulation are not clearly understood. The parasites express a wide range of the molecules with different immunomodulatory properties. The promising option of gastrointestinal nematode control is exploitation of genetic resistance by vaccination. However, these methods use the host immune system and there has been considerable effort put into understanding host immunity.

The interaction between sheep and the gastrointestinal nematode *Teladorsagia circumcincta* is one of the best understood of host–parasite interactions. Immunity to nematodes takes two grazing seasons to become fully established. However, even adult hosts develop incomplete protective immunity against nematodes. Major mechanisms of immunity to nematodes are associated with IgE and IgA activity. Both IgA and IgE recognize multiple parasite molecules and may work by inhibiting parasite proteins or in conjunction with eosinophils and mast cells respectively. However, there are some areas where our understanding of IgE-mediated protection is incomplete. Sheep IgE antibody strongly recognizes bands for galectin.

Based on our previous study we propose that nematodes may have evolved their galectin to specifically manipulate the host immune response. One possibility is parasite galectin may act as a molecular sponge and bind host IgE and inhibit mast cell degranulation.
Therefore the objectives of this project are to combine parasitological, molecular, proteomic, and immunological procedures to clone galectin proteins and confirm galectin as an antibody binding protein and a suppressor of IgE-antigen complex formation and mediator release.

The results of the project will advance our understanding of parasite–host interactions and further, the identification of this mechanism opens up multiple avenues for defining novel targets for immune intervention during nematode infection. The research may also lead to better methods of controlling autoimmune reactions in humans.