Rhynchosporium secalis cell wall proteins

UNIVERSITY OF ABERDEEN



A. E. Mackenzie^{a,d}*, M. Münsterkötter^b, W. Knogge^c, A. C. Newton^a, C. A. Munro^d and A. Avrova^a Corresponding author (e-mail): Ashleigh.Mackenzie@hutton.ac.uk

^aThe James Hutton Institute, Dundee, UK; ^bMIPS - Institute of Bioinformatics and Systems Biology,

Munich, Germany: ^cLeibniż-Institute of Plant Biochemistry, Halle, Germany; ^dUniversity of Aberdeen, Aberdeen,

Introduction

Rhynchosporium secalis is one of the most destructive pathogens of barley worldwide. It can lead to yield loss of up to 40% and decrease in grain quality. Populations of R. secalis can change rapidly, defeating new barley resistance (R) genes and fungicides after just a few seasons of their widespread commercial use.

In pathogenic fungi, the cell wall and especially cell wall proteins (CWPs) play a key role in the establishment of pathogenesis. The cell wall forms the outer structure protecting the fungus from the host defence mechanisms. It is involved in initiating the direct contact with the host cells by adhering to their surface. Fungal cell wall also contains important antigens and other compounds modulating host immune responses.

A better understanding of these cell wall proteins and hence, pathogenicity of this fungus may lead to the development of a long term, sustainable mode of management for

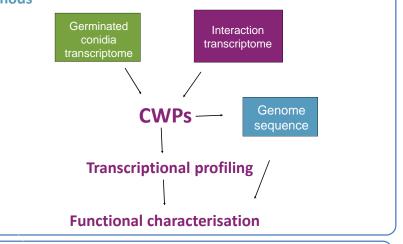
Aims and Objectives

The aim of this project is to investigate the role of R. secalis cell wall proteins in cell wall integrity and pathogenicity.

The main objectives are:

- 1. Identification of R. secalis cell wall proteins using bioinformatics analyses of genome and transcriptome
- 2. Transcription profiling during the development of infection of selected CWPs potentially effecting R. secalis
- 3. Functional characterisation of selected CWPs including their effect on pathogenicity through targeted gene knockout, complementation and biochemical studies

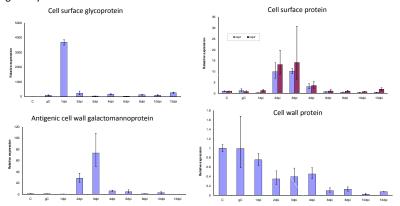
Methods



Results

A list of over 50 CWPs potentially involved in pathogenicity has been generated. The proteins are of very varied function e.g. cell wall biosynthesis and maintenance, adhesion or structural proteins etc. R. secalis genome and interaction transcriptome sequencing provided further information about the extent of CWP families as well as a subset of genes expressed during barley colonization by R. secalis.

Transcription profiling of R. secalis CWPs during the development of infection will help to prioritise them for functional characterisation by measuring how abundant they are through-out the infection time-course. The graphs below show proteins with interesting expression, they are highly abundant early on in infection and therefore could be involved in pathogenicity.



Future Aspects

Genes of particular interest will be selected for targeted gene disruption in order to understand their roles in pathogenicity. I will be using split marker transformation to do this as this technique allows for specific disruption of the gene of interest. Ideally I will be choosing five-six candidate



Acknowledgements
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