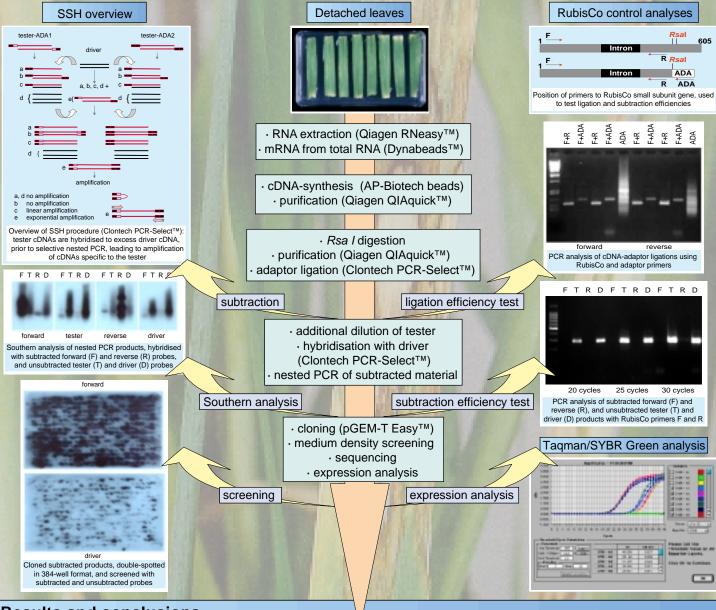
Characterisation of Resistance Pathways to Rhynchosporium secalis in Barley using Suppression Subtractive Hybridisation

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Introduction

Rhynchosporium secalis is the cause of leaf scald in barley. Resistance is conditioned by either race-specific genes, of which at least eleven resistance genes have been identified, or quantitative factors which are probably race-non-specific. The molecular bases of either form of resistance are poorly understood.

We present here the generation of cDNA libraries enriched for sequences induced in barley following activation of *R. secalis* resistance, using an improved suppression subtractive hybridisation (SSH) method.



Results and conclusions

Two SSHs were performed: 1. To enrich for barley cDNAs specifically expressed when *R. secalis* resistance was activated (avirulent); 2. To enrich for barley cDNAs specifically expressed in the susceptible interaction with *R. secalis* (virulent). SSH-derived PCR products were directly cloned into pGEM-T Easy. Preliminary sequencing has revealed a clone with homology to a cysteine protease gene in the avirulent interaction. Cysteine proteases are currently being associated with the plant hypersensitive response (Avrova *et al.*, 1999*). Cloned sequences from the virulent interaction included lipoxygenase, abscisic acid (ABA)-induced gene, osmotin-like gene and potato wound-induced gene, implicating involvement (respectively) of the following stress-related pathways: jasmonate, abscisic acid, pathogenesis-related and wound response. Further sequencing and analyses of gene expression and function will be performed to better characterise these interactions.

Borlov	
Barlow	
	1
Potato	1
Rice	11
Arabidopsis	8
	1
	32
Potato	1
Barley	5
Wild potato	4
Potato	2
Tomato	1
Tobacco	13
Wheat	1
Barley	1
Tomato	1
Tomato	1
	40
	Rice Arabidopsis Henry's Lily Potato Barley Wild potato Potato Tomato Tobacco Wheat Barley Tomato

Origin

% of clones

*Avrova, A.O., Stewart, H.E., De Jong, W., Heilbronn, J., Lyon, G.D. and Birch, P.R.J. 1999. MPMI 12: 1114-1119

Gene