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Dissecting loci and genes controlling Sclerotinia disease resistance and their genetic and evolutionary relationship with flowering time in *Brassica napus*

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Background:

Sclerotinia disease is a major threat to rapeseed production globally, particularly in China. There exists large difference in resistance to *Sclerotinia* disease between genotypes or varieties and in fact, resistant varieties play in crucial role in rapeseed sustainable production in some regions of China. However, current resistance level is far from demand in rapeseed production. Therefore, to increase resistance level is highly required.

Objective:

The aim of the study was to dissect genetic structure of *Sclerotinia* disease resistance and investigate genetic and evolutionary relationship between resistance and flowering time in *B. napus* rapeseed.

Methods:

GWAS and RIL populations were developed to map loci of disease resistance and flowering time, a pipeline of candidate gene exclusion/selection based on comparative genomics was developed and genome editing, transgenics and other genetic and biochemical approaches were employed to study gene function and molecular mechanisms.

Results:

A dozen of major loci were mapped for disease resistance and flowering time. Among these loci, nine genes for disease resistance and five genes for flowering time were pinpointed by the pipeline of candidate gene exclusion/selection developed in our lab and the functions of these genes were validated by gene editing, Arabidopsis mutants and/or other genetic approaches. Of these genes, three genes are novel pleiotropic genes simultaneously controlling both *Sclerotinia* disease resistance and flowering time. One pleiotropic gene *BnMEB2* was investigated for its detailed genetic, biochemical and evolutionary/ecological mechanisms. The protein MEB2 interacts with a protein PFS of the flowering time pathway and their interaction occurs between specific alternative splicing transcripts – functional differentiation of alternative splicing transcripts of a single gene; PFS.1 interacts with phyB while PFS.2 interacts with both phyB and Flor1. In brief, PFS.1 and PFS.2 orchestrate regulation of phyB turn-over between nucleus and cytoplasm and between day and night, which is connected by MEB2 to disease resistance.

Conclusions:

The *Sclerotinia* disease susceptibility and early flowering were controlled by multiple pleiotropic genes and involve concerted network regulation. The study suggests a strategy to break down negative correlation for breeding varieties with Sclerotinia disease susceptibility and early flowering/maturation.

References:

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