Progress in mapping resistance to Sclerotinia white mold in lentil

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Introduction

Under conditions conducive to the disease, white mold caused by Sclerotinia sclerotiorum can be an important disease of lentils (Fig 1). Management of white mold in lentil is mainly through the planting of resistant cultivars. Some of the current commercial cultivars of lentils show tolerance to white mold, but the level of tolerance is low. There is need to improve the level of resistance/tolerance in lentil to white mold. There are some germplasm lines that show better resistance than currently available in commercial cultivars and could be employed in breeding lentil for resistance to white mold. However, there is no information available on genetics of lentil resistance to white mold. In order to devise efficient strategies to move resistance genes to commercial cultivars, we need to understand the genetics of resistance to white mold in lentils. One approach to study genetics of lentil resistance to white mold is to map the resistance genes using populations of recombinant inbred lines and molecular markers.

Materials and Methods

Lentil populations of RILs: Nine crosses between lentils with different levels of resistance to white mold were made and the progenies are being advanced to generate recombinant inbred lines.

Confirmation of the hybrid nature of the populations: Polymorphic molecular markers were used to verify the hybrid nature of the populations. The parents and six progenies randomly selected from each population were genotyped using markers that are polymorphic between the parents. Occurrence of alleles of both parents in the progeny indicates the hybrid nature of the population.

Generation of additional molecular markers: One approach to generate additional molecular markers is to take advantage of the genomic information available in the model legume plant Medicago truncatula. We tested the applicability of 618 microsatellite markers of M. truncatula in lentils.

Results

Nine populations of RILs have been advanced to the F4 by single seed decent. The number of progenies from the nine crosses varies from 125 to 240 and the total number of lines being advanced is 1752. Molecular markers showed that the populations are indeed hybrids from their respective parents (Fig. 2).

A total of 618 microsatellite markers of M. truncatula were tested on lentils and other grain legumes, and about 580 of the 618 markers were applicable to lentils and other grain legumes. Twenty-five of the markers were found polymorphic in lentil. Twenty-one of the polymorphic markers are co-dominant and the other four markers were dominant markers (Fig. 3). We also have access to a set of microsatellite markers developed at ICARDA specifically for lentil.

Discussion

Progress has been made in studying genetics of lentil resistance to white mold. Defined populations for genetic mapping are being advanced to the F4 and will be advanced further to the F5 and be available in about 9 months for phenotyping their reactions to infection by S. sclerotiorum, and for genotyping polymorphic molecular markers. Taking the advantages of the model legume M. truncatula, twenty-five additional polymorphic markers are identified for genetic mapping in lentil. Microsatellite markers from ICARDA and markers from pea will also be used. The phenotypic and molecular marker data will be used to generate a genetic linkage map of lentil for use in a quantitative trait loci analysis of white mold resistance.

Selected Literature


Eujayl, I., Baum, M., Powell, W., Erskine, W., and Pehu, E. 1998. A genetic linkage map of lentil (Lens sp.) based on RAPD and AFLP markers using recombinant inbred lines. Theoretical and Applied Genetics 95: 83-89


Fig. 1. Field symptoms of white mold of lentil shown in these 2003 photos. Early development of stem rot (above) and massive stem rot (below). Fluffy white mycelium is clearly visible.

Fig. 2. Molecular confirmation of the hybrid populations of lentils. Screening molecular markers on parental lines (left) and testing a polymorphic marker on two parents and randomly selected six progenies (right).

Fig. 3. Screening microsatellite markers of Medicago truncatula on lentils and other grain legumes. Three (SSR07, SSR11 and SSR84) of the polymorphic markers are shown. Lane 1: M. truncatula; Lanes 2-4: Lentils; Lanes 5-6: chickpeas; Lanes 7-8: Peas.