PIACENZA, Italy
Institute of Agronomy, Genetics and Field Crops, Università Cattolica del Sacro Cuore (UCSC)

**Mapping candidate genes for *Fusarium* ear rot resistance**

Maschietto, V; Lanubile, A; Marocco, A.

*Fusarium* ear rot is one of the most important disease of maize, that is of concerns because *Fusarium verticillioides* produces the mycotoxins fumonisins. Resistance to *Fusarium* ear rot is polygenic with nearly complete dominance or overdominance of resistance alleles. The availability of molecular markers associated to resistance genes could be a successful strategy to select lines resistant to *F. verticillioides*. Mapping of quantitative trait loci (QTLs) provides a powerful method to understand the genetic relationships between correlated traits.

We attempt to use a genetical-genomics strategy to localize candidate genes for resistance to *F. verticillioides* on the high density molecular map.

Resistant (CO441; R) and susceptible (CO354; S) genotypes were selected and crossed to obtain $F_2$ and $F_3$ mapping populations. In previous works, we found a high level of variability for the response to *F. verticillioides* infection between the two susceptible and resistant maize lines selected (Lanubile et al., 2010, J. Plant Physiol. 167:1398-1406; Lanubile et al., 2011, World Mycotox. J. 4:43-51). Similar functional categories of genes were involved in the response to infection in the two genotypes. In the resistant line, the defense-related genes assayed were transcribed at high level before infection and provided basic defense to the fungus. In the susceptible line, the same genes were induced from a basal level and responded specifically to pathogen infection. The expression level of eleven genes involved in plant defense were validated by qPCR (Table 1). The candidate genes are physically mapped and the polymorphisms relative to 27 maize genotypes are availables in www.panzea.org. About 70 primers were designed on the candidate sequences and SNPs will be identified between CO441 and CO354 parents.

In addition to SNP markers, 217 SSRs were selected, according to their chromosomic positions on the reference map (http://www.maizegdb.org/), for genotyping $F_2$ individuals. 110 SSRs (about 50%) even distributed on the ten chromosomes, resulted polymorphic (Table 1). Furthermore, 200 $F_3$ progenies will be phenotyped for the response to artificial infection and fumonisin contamination.

**Table 1. Number of polymorphic SSR markers and selected candidate genes.**

| Chromosome | SSRs | Candidate genes |
|---|---|---|
| 1 | 12 | Pathogenesis related protein 5<br>WRKY DNA-binding protein 1 |
| 2 | 12 | - |
| 3 | 6 | Chitinase<br>Pathogenesis related protein 6<br>Respiratory burst oxidase |
| 4 | 11 | - |
| 5 | 13 | MYB-like DNA binding protein |
| 6 | 13 | - |
| 7 | 11 | Pathogenesis related protein 1<br>Thaumatin-like protein |
| 8 | 12 | Wound-induced protease inhibitor |
| 9 | 13 | - |
| 10 | 7 | β-glucosidase<br>Peroxydase 1 |