Identification of markers closely linked to effective leaf rust resistance genes in wheat

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Leaf rust, caused by *Puccinia triticina*, is the most common and widespread disease of wheat (*Triticum aestivum*) worldwide. Leaf rust causes a reduction of grain quality and yield losses of up to 40%. Epidemics are due to a breakdown of leaf rust resistances (Lr resistances) by virulent rust races. An example for a breakdown is Lr37, which became ineffective within two years in the 2006. One option to avoid epidemics is pyramiding of Lr-genes so that cultivars carry several effective Lr-genes. A prerequisite for pyramiding are closely linked molecular markers. At the moment, more than 80 Lr-genes have been identified, but only a part of these have been deployed in wheat varieties in part due to linkage drag. Resistances showing a high level of resistance in the field are Lr2a, located on chromosome 2DS, and Lr24 on chromosome 3B. In order to get detailed information on their localization and to reduce linkage drag, NILs (near isogenic line) containing one of the Lr-genes were crossed to the susceptible cultivar Monopol. Parental lines and F2 plants were inoculated with leaf rust single spore isolates avirulent to Lr2a and Lr24. The development of fungal structures was analyzed on leaf material of parental lines and of 150 (Lr2a) and 144 (Lr24) F2 plants, each at 72 hours after the inoculation (hai) and uredospore pustule development was scored at 168 hai. First results of the analysis proved the recessive inheritance of Lr2a as a good fit to a 3s:1r segregation ($\chi2=1,5$) and dominant inheritance of Lr24 with 3r:1s segregation ($\chi2=0,39$) were observed. Based on these results kompetitive allele specific PCR markers (KASP) which have been generated based on SNPs detected between near isogenic lines (NILs) carrying the resistances and the susceptible parental line (Thatcher) were genetically mapped and aligned to the reference genome so that candidate genes for Lr2a were identified, already. Next steps will be the analysis of additional F2 populations segregating for effective resistances (e.g. Lr9, Lr19) and pyramiding of these genes.