IN BRIEF

Sick as a...Grass? Viral Infection Causes Massive Changes in Alternative Splicing in *Brachypodium distachyon*

It’s the season to come down with a virus, and whether you have a cold or the flu, you know that being sick changes everything. However, you may not know how deep into your cellular functions this “everything” extends. Runny nose, yes—but RNA splicing? Oh yes—high-throughput RNA sequencing in multiple systems, including plants, has shown that many different conditions affect alternative splicing. For example, in plants, alternative splicing of genes changes throughout development and regulates growth, flowering, the circadian clock, and the responses to biotic and abiotic stress (reviewed in Reddy et al., 2013; Staiger and Brown, 2013).

To reveal the genome-wide changes in alternative splicing that occur during a plant-virus interaction, Mandadi and Scholthof (2015) infected *Brachypodium distachyon* plants with *Panicum mosaic virus* (PMV) and its satellite virus (SPMV), a combination that causes severe disease and symptoms (see figure). They then used RNA-seq to profile changes in alternative splicing. Of the more than 44,000 transcripts identified, ~42% of those with more than one exon underwent alternative splicing. Among these alternative splicing events, 36% involved intron retention, 41% involved alternate donor or acceptor sites, and 9% involved exon skipping—a striking contrast to animal systems, where exon skipping occurs most frequently. About 14% of alternative splicing events were complex, consisting of duplications or combinations of the aforementioned types. Comparison of multiple species indicated that monocots and dicots have largely similar ratios of the different splicing types. Virus infection increased the overall number of alternative splicing events in *B. distachyon*, but did not change the ratio of different types. The authors identified ~600 genes that showed changes in splicing pattern in response to virus infection; these included ~100 defense-related genes. Cluster analysis of the expression of specific defense-related gene families also showed that the splicing forms were coregulated in response to PMV and PMV+SPMV infection (see figure).

Virus infection also affected alternative splicing of gene transcripts encoding splicing factors, and the authors conducted a detailed examination of the *B. distachyon* ortholog of the *Arabidopsis thaliana* spliceosome component SC35-LIKE SPlicing FACTOr33 (SCL33), identifying seven splice variants of SCL33. Five of these variants contain premature stop codons, which might produce truncated proteins or target the mRNA for degradation by nonsense-mediated decay. The variants changed in abundance during development and viral infection, and the pattern of change was conserved between *B. distachyon* and Arabidopsis. Thus, analysis of this factor shows the complexity and conservation of alternative splicing.

Plants don’t get a runny nose, but the genome-wide effects on alternative splicing identified here show that viral infection causes massive alterations in the splicing landscape. Moreover, the alternative splicing of resistance genes, such as NB-LRR proteins, indicates that these changes may affect disease resistance. Determining whether the observed changes improve adaptation to stress during viral infection, or promote viral spread/infection, and identifying the mechanisms that cause these changes, remain intriguing topics for further research and possibly will inform disease prevention in plants, and maybe even in humans—a development that might make you feel a bit better someday.

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