IN BRIEF

Twist of Fate: RID1, a DEAH-box RNA Helicase Involved in Pre-mRNA Splicing, Is Crucial for Development

Pre-mRNA cannot be translated until its introns are removed by the precise process known as pre-mRNA splicing, which is executed by the spliceosome. The spliceosome is a massive, ribosome-sized assembly containing five RNA-protein complexes, called small ribonucleoprotein particles (snRNPs), each of which comprises a specific type of uridine-rich small nuclear RNA (UsnRNA) and its tightly associated proteins and more than 50 non-snRNP proteins (Staley and Guthrie, 1998). One important group of non-snRNP spliceosomal proteins is the DEAD/H-box protein family. These proteins function as motors to unwind short RNA duplexes, thereby influencing intron removal, during the final step of pre-mRNA splicing. DEAD/H-box proteins are present in such diverse organisms as yeast and human and are known to occur in plants, but until recently, little was known about plant spliceosomal DEAD/H-box proteins.

Ohtani et al. (2008) launched into a study of these proteins quite unexpectedly in the course of investigating plant organogenesis using temperature-sensitive Arabidopsis thaliana mutants. One such mutant, shoot redifferentiation defective2-1 (srd2-1), exhibits temperature-sensitive hypocotyl dedifferentiation and de novo meristem formation. The effect of the srd2 mutation varies markedly between tissues and developmental stages. It turns out that SRD2 encodes an activator of snRNA transcription, including all spliceosomal UsnRNAs, and SRD2-mediated upregulation of snRNA transcription is essential for cell proliferation competence as well as several developmental processes, including apical meristem establishment. Another mutant, root initiation defective1-1 (rid1-1), is temperature sensitive for adventitious root formation from hypocotyl explants. When grown in vitro at the restrictive temperature, rid1-1 plants appear quite similar to srd2-1 plants, suggesting that SRD2 and RID1 have similar functions. This prompted Ohtani et al. (pages 2056–2069) to perform a detailed analysis of the rid1-1 mutant with reference to srd2-1. Phenotypic characterization of rid1-1 showed that RID1 and SRD2 play similar roles in various stages of plant development. The requirement for these factors changes dynamically throughout development, and the expression patterns of RID1 and SRD2 overlap considerably. Furthermore, RID1 is preferentially localized in the nucleolus, a common site for snRNP proteins and splicing factors and most likely the site of snRNP biogenesis and spliceosome formation, as determined by examining fluorescent signal patterns in root epidermal cells of transgenic plants expressing RID1-YFP (yellow fluorescent protein; see figure).

Interestingly, chromosome mapping and sequence analysis revealed that RID1 encodes a DEAH-box RNA helicase similar to the splicing factors Prp22 (Prp22) in yeast and DEAH box polypeptide8 (DHX8) in humans. This led to the assumption that RID1, like Prp22 and DHX8, functions in intron removal at the final step of pre-mRNA splicing. However, RID1 failed to complement a Prp22 yeast mutant. Also, phylogenetic tree analysis showed that RID1 is not located in the clade containing Prp22. RT-PCR analysis revealed that the rid1-1 mutation not only influences intron removal, but it also affects the recognition of the splicing site.

The authors speculate that the DEAH-box RNA helicase RID1 might contribute to UsnRNP biogenesis and, consequently, spliceosome assembly. Therefore, genes involved in specific developmental processes may require particularly high spliceosome activity and may in fact be regulated selectively by the capacity of splicing, which is driven by RID1 and the SRD2-mediated production of UsnRNAs. It is amazing what the analysis of temperature-sensitive mutants can lead to.

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REFERENCES


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