

Abstract

Protein Prp22 is a DEAH box RNA helicase, which plays two distinct roles in pre-mRNA splicing: it participates in second transesterification step (ATP independent function) and it releases mature mRNA from the spliceosome (ATP dependent function). Prp45p, yeast ortholog of the human transcription co-regulator SNW/SKIP, is an essential splicing factor, it is included in spliceosome throughout the splicing reaction. Mutant *prp45(1-169)* genetically interacts with some alleles of NTC complex and second step splicing factors, one of them is also gene *PRP22*. Here we present, that mutants *prp22(-158T)* and *prp22(-327A)*, which are synthetically lethal with *prp45(1-169)*, express lower amount of Prp22p due to the mutation in upstream regulation region. Mutants *prp22(-158T)*, *prp22(300PPI)* and *prp22(-327A)* affect splicing of pre-mRNA with mutation in 5' splice site with respect to sequence of the second exon. N-terminal mutants *prp22(Δ301)* and *prp22(Δ350)* are synthetically lethal with *prp45(1-169)*. Synthetic lethality is possibly caused by lower efficiency of Prp22 recruitment to the spliceosomes, which is no more viable for cells.