## Abstract

Ubiquitin and ubiquitin-like modifiers (UBLs- SUMO, Nedd8, Hub1, etc.) are a family of related proteins characterized by similarities in sequence and tertiary structure to Ubiquitin. They have been shown to play important roles in eukaryotic cells by binding and modifying cellular function of a wide variety of other proteins. Hub1 is an unconventional member of this family which differs from other canonical UBLs by lacking the characteristic GG motif, C-terminal extension or processing enzymes which recognize it. Other UBLs are known to be conjugated to substrates covalently through the di-glycine motif and have an elaborate conjugation machinery which includes E1, E2 and E3 enzymes facilitate this whereas Hub1 only binds its substrate non-covalently and has no conjugation machinery associated with it. Hub1 has been previously reported to be a part of the spliceosome, mostly through the non-covalent binding to a conserved amino acid sequence motif called HIND (I & II) which has been shown to be present in spliceosomal proteins Snu66 and Prp38 in different organisms. The role Hub1 in pre-mRNA splicing has been previously examined in Humans, S. pombe and S. cerevisiae. Cells deficient in Hub1 in S. pombe and humans have severe defects in general splicing. In the study discussed here, it is shown that Hub1 in S. pombe plays a unique role in the splicing of core snRNP splicing factor U6 snRNA, and some of the sickness observed in temperature sensitive mutant hub1-1 can be rescued by transforming the cells with the cDNA of U6 snRNA. One possible reasons of U6 splicing being hub1-dependent was also investigated. It is shown here that the unique features of U6 snRNA that results from its transcription by RNA polymerase III (as opposed to RNA polymerase II for all other spliceosomal intron containing RNAs and other snRNA molecules) seems not to be the reason for its Hub1 dependency.