Abstract

SKIP and BIR/Survivin are evolutionarily conserved proteins. SKIP is a known transcription and splicing cofactor while BIR-1/Survivin regulates cell division, gene expression and development. Loss of function of *C. elegans* SKIP (SKP-1) and BIR-1 induces overlapping developmental phenotypes. In order to uncover the possible interactions of SKP-1 and BIR-1 on the protein level, we screened the complete *C. elegans* mRNA library using the yeast two-hybrid system. These experiments identified partially overlapping categories of proteins as SKP-1 and BIR-1 interactors. The interacting proteins included ribosomal proteins, transcription factors, translation factors and cytoskeletal and motor proteins suggesting involvement of the two studied proteins in multiple protein complexes. To visualize the effect of BIR-1 on the proteome of *C. elegans* we induced a short time pulse BIR-1 overexpression in synchronized L1 larvae. This led to a dramatic alteration of the whole proteome pattern indicating that BIR-1 alone has the capacity to alter the chromatographic profile of many target proteins including proteins found to be interactors in yeast two hybrid screens. The results were validated for ribosomal proteins RPS-3, RPL-5, non-muscle myosin and TAC-1, a transcription cofactor and a centrosome associated protein. Together, these results suggest that SKP-1 and BIR-1 are multifunctional proteins that form multiple protein complexes in both shared and distinct pathways and have the potential to connect proteome signals with the regulation of gene expression.

Key words: BIR-1, gene expression, proteome, ribosomal stress, SKIP, Survivin