
MATERIAL DATA SHEET

Recombinant Human SUMO1 Mutant K7R/K16R/K17R

Cat. # ULM-722

Human Small Ubiquitin-like Modifier 1 (SUMO1), also known as Sentrin, UBL1, and SMT3C, is synthesized as a 101 amino acid (aa) propeptide with a predicted molecular weight of 11.5 kDa. Human SUMO1 is the most unique of the four identified SUMO proteins and shares only 44%, 47%, and 41% aa sequence identity with SUMO2, SUMO3, and SUMO4, respectively. In contrast, human SUMO1 shares 100% aa sequence identity with the mouse ortholog. SUMOs are a family of small, related proteins that can be enzymatically attached to a target protein by a post-translational modification process termed SUMOylation (1-3). All SUMO proteins share a conserved Ubiquitin domain and a C-terminal diglycine cleavage/attachment site. Following cleavage of a four aa C-terminal prosegment, the C-terminal glycine residue of SUMO1 is enzymatically attached to a lysine residue on a target protein. In humans, SUMO1 is conjugated to a variety of molecules in the presence of the SAE1/UBA2 SUMO-activating (E1) enzyme and the UBE2I/Ubc9 SUMO-conjugating (E2) enzyme (4,5). In yeast, the SUMO-activating (E1) enzyme is Aos1/Uba2p (6). SUMOylation can occur without the requirement of a specific SUMO ligase (E3), where SUMO1 is transferred directly from UBE2I/Ubc9 to specific substrates. In Alzheimer's disease models SUMO1 has been shown to influence the generation of Amyloid-beta peptide by promoting the accumulation of BACE-1 (7). Covalent modification of Phosphatase and Tensin Homolog Deleted on Chromosome (PTEN) by SUMO1 is thought to regulate tumorigenesis by retaining PTEN at the plasma membrane, an effect that suppresses PI 3-Kinase/Akt-dependent tumor growth (8).

Human SUMO-1 does not contain the exact ψ KXE consensus sequence found in SUMO-2 and SUMO-3. Within this motif ψ represents a large hydrophobic amino acid (I, L, or V), K is the lysine that becomes modified, X is any residue and E is glutamic acid. Many known SUMO-1 conjugation sites occur within this consensus sequence, but SUMOylation also occurs on lysine residues located within non-consensus regions. SUMO-1 has been shown to form chains in vitro and in vivo, but often the linkage is uncharacterized, and the function of SUMO chains has not yet been fully elucidated. SUMO-1 multimerization in vitro has been shown to occur predominantly via lysines K7, K16 and K17. Mutation of lysines 16 and 17 to arginine is useful to investigate mono-SUMOylation requirements or to reduce poly-SUMO chain formation.

Product Information

Quantity:	250 µg
Source:	<i>E. coli</i> -derived Accession # NM_003352
Stock:	Supplied as a solution in HEPES, NaCl and DTT.
Purity:	>95%, by SDS-PAGE under reducing conditions and visualized by Colloidal Coomassie® Blue stain.

Use & Storage

Use:	SUMO1 chains do not readily form <i>in vitro</i> . However, the role of SUMO1 in poly-SUMO chain formation is an area of intense research. Utilizing Recombinant Human SUMO1 Mutant K7R/K16R/K17R will ensure that K7, K16, and K17-linked chains will not be formed. Reaction conditions will need to be optimized for each specific application. We recommend an initial Recombinant Human SUMO1 Mutant K7R/K16R/K17R concentration of 10-50 µM.
Storage:	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. • 12 months from date of receipt, -70 °C as supplied. • 3 months, -70 °C under sterile conditions after opening.

Literature

References:

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5. Tatham, M.H. *et al.* (2001) J. Biol. Chem. **276**:35368.
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