

FIG S5 (A) Functional analyses of CopC N-terminal truncation mutants. The p20/p10 form of caspase-7, -8 or -9 were reacted with CopC (full-length (FL), ΔN50, or ΔN92) at the molar ration of 10:1 in the presence of CaM. (B) Gel-filtration chromatography analyses of CopC ΔN50, CaM and caspase-7 p30 complex formation. The peak eluted from the column, the protein samples used for crystallization as well as the crystal samples were assessed by Coomassie-stained SDS-PAGE gels. (C) A cartoon scheme of the 2:2:2 CaM-CopC-CASP7 complex structure. The dimeric ternary complex is mediated by caspase-7 molecules from two neighboring asymmetric units. The 2-fold crystallographic axis is shown. (D) Cartoon models of Ca²⁺-bound CaM alone (PDB code: 1CDL, left) and Ca²⁺-free CaM in complex with the IQ motif of myosin V (PDB code: 2IX7, right). Ca²⁺ are shown as green sphere. The middle shows CaM-C lobe in the ternary complex (magenta) overlaid with that in the Ca²⁺-bound CaM (upper) or Ca²⁺-free CaM (lower). (E) Dali search results of CopC-NTD structure. (F) Close-up view of the deduced NAD+-binding site in CopC-NTD and its position relative to the R233-containing loop of CASP7. Structures of isolated CASP7-p30 dimer (PDB code: 1K88) and CASP7-p20/p10 dimer (PDB code: 1K86) were superposed onto that of CASP7 in the ternary complex. The deduced NAD+-binding site in CopC-NTD is highlighted by a magenta dash-line box with binding residues in stick models.