MODULATION OF MEMBRANE RIGIDITY BY THE ESCRT-III COMPLEX

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The ESCRT-III complexes is an evolutionary conserved membrane scission machinery and it is essential in many cellular processes such as cytokinesis, multivesicular bodies formation, HIV release and nuclear membrane repair. Membrane scission by ESCRT-III is accomplished by constricting the negatively curved membrane present inside the neck connecting two membrane-delimited compartments. In Homo Sapiens there are at least 12 ESCRT-III proteins, called CHMPs. However, only a subset of them, namely CHMP4B, CHMP2A/B and CHMP3 appear to be strictly required in all these processes, indicating that they might constitute the minimal scission machinery. Several CHMP2B mutations have been reported to cause neurological disfunctions; however the function of this protein is still unknown. We investigated the mechanical properties of CHMP proteins polymer and found that they can modulate membrane rigidity in a subunit-specific fashion, providing a possible molecular mechanism explaining the pathogenic property of the mutated CHMP2B. Our results indicate that modulation of membrane rigidity is an important aspect of ESCRT-III function and assign a novel function for CHMP2B.