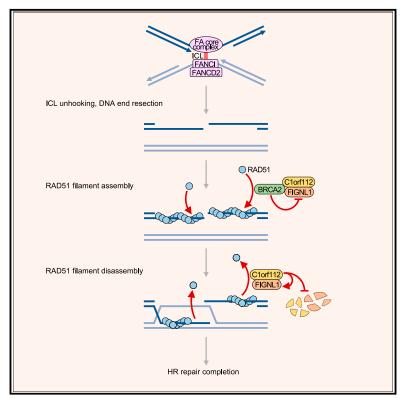
Cell Reports

C1orf112 teams up with FIGNL1 to facilitate RAD51 filament disassembly and DNA interstrand cross-link repair

Graphical abstract



Authors

Zenan Zhou, Han Yang, Xinxin Liang, ..., Yang Yang, Jiadong Wang, Weibin Wang

Correspondence

wangjd@bjmu.edu.cn (J.W.), weibinwang@bjmu.edu.cn (W.W.)

In brief

Zhou et al. perform RAD51 proximity proteomics and identify the factor C1orf112. C1orf112 can enhance FIGNL1 protein stability and stimulate the RAD51 filament disassembly by FIGNL1. Via regulating RAD51 filament, the C1orf112-FIGNL1 complex facilitates the homologous recombination step of ICL repair by the Fanconi anemia pathway.

Highlights

- C1orf112 forms a stable complex with FIGNL1 and physically interacts with RAD51
- The RAD51 filament disassembly activity of FIGNL1 is stimulated by C1orf112
- BRCA2 functions upstream of C1orf112-FIGNL1 to protect RAD51 from premature disassembly
- C1orf112- and FIGNL1-deficient cells are most sensitive to DNA interstrand cross-link agents

Zhou et al., 2023, Cell Reports *42*, 112907 August 29, 2023 © 2023 The Author(s). https://doi.org/10.1016/j.celrep.2023.112907

