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Fibrillarin promotes homologous recombination repair by facilitating the recruitment of recombinase RAD51 to DNA damage sites

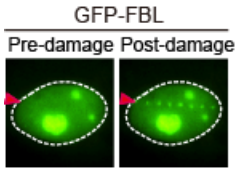
Key words: Genome instability, DNA double-strand breaks, homologous recombination, RAD51; fibrillarin (FBL)

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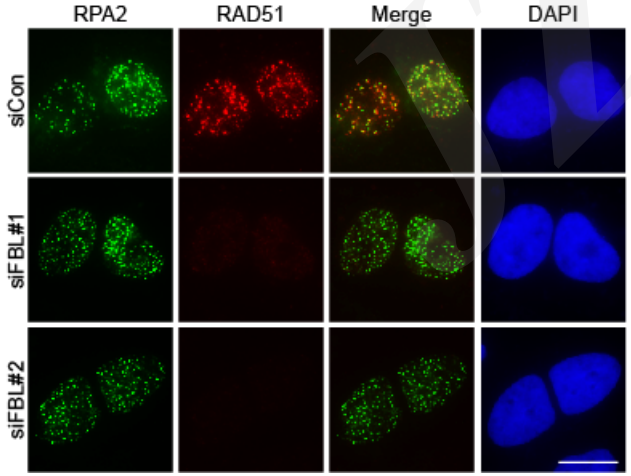
Research Summary

Our findings provide novel insights into the mechanism of RAD51 recruitment to DNA damage sites mediated by FBL, and highlight the significant implications of FBL in cancer therapy.

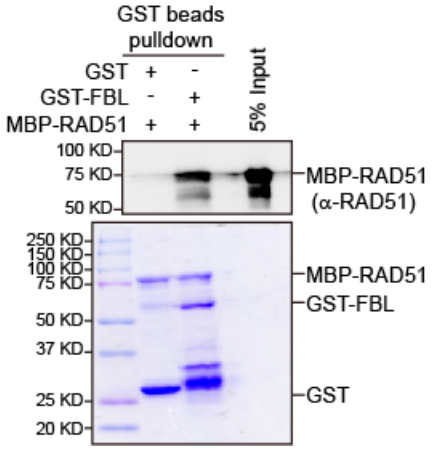
- FBL can be recruited to DNA damage sites.

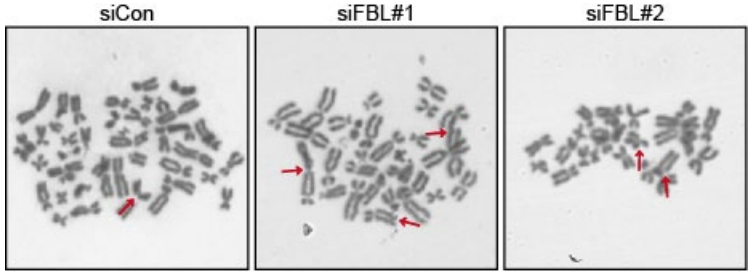


- FBL promotes the recruitment of RAD51 to DNA damage sites.

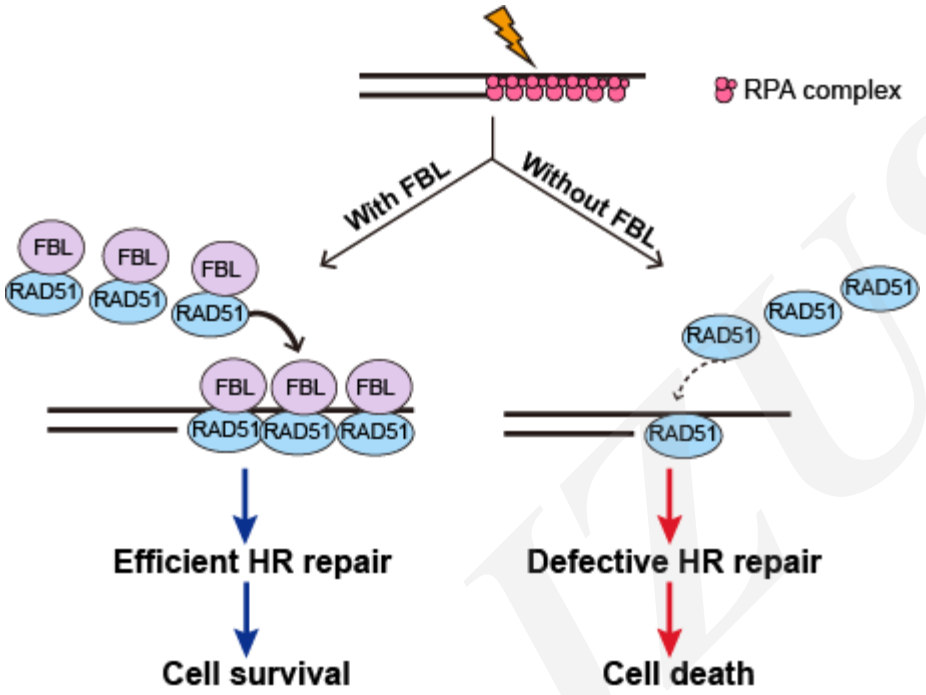


- FBL directly interacts with RAD51 and promotes genome integrity.





Innovation Points



The model depicts the proposed mechanism by which FBL facilitates HR repair. Our study identifies FBL as a regulator of HR. Upon DNA damage, FBL is recruited to DSBs and directly interacts with the recombinase RAD51. Depletion of FBL leads to impaired formation of RAD51 foci and reduced HR efficiency. Additionally, FBL depletion results in an increase in chromosome aberrations and enhances cellular sensitivity to DNA-damaging agents.