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SHLD2/FAM35A co-operates with REV7 to coordinate DNA double-strand break repair pathway choice

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Genomic instability is a prerequisite for the emergence of cancer cells. Thus, understanding how to maintain genomic stability is critical. In particular, DNA double strand breaks are one of the most cytotoxic forms of lesion and represent a major threat for genome integrity. This paper identifies a previously unknown factor, SHLD2, that cooperates with REV7 to initiate DNA double-strand break repair.

Dysregulation of the DNA double-strand break response has been extensively linked to the development of several cancers, including breast and ovarian cancers. Interestingly, the authors observe that SHLD2 levels correlate with a poor prognosis in patients affected by a subtype of breast cancer. Furthermore, their data suggest that SHLD2 loss may result in the development of drug resistance to PARP inhibitors. This is the first evidence that SHLD2 would be an accurate clinical biomarker, which could be effective in both diagnosis and prognosis for a subset of breast cancer.

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