Formation of BRCA1-A complex at DNA DSBs

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Introduction

Reactome is open-source, open access, manually curated and peer-reviewed pathway database. Pathway annotations are authored by expert biologists, in collaboration with Reactome editorial staff and cross-referenced to many bioinformatics databases. A system of evidence tracking ensures that all assertions are backed up by the primary literature. Reactome is used by clinicians, geneticists, genomics researchers, and molecular biologists to interpret the results of high-throughput experimental studies, by bioinformaticians seeking to develop novel algorithms for mining knowledge from genomic studies, and by systems biologists building predictive models of normal and disease variant pathways.

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Literature references

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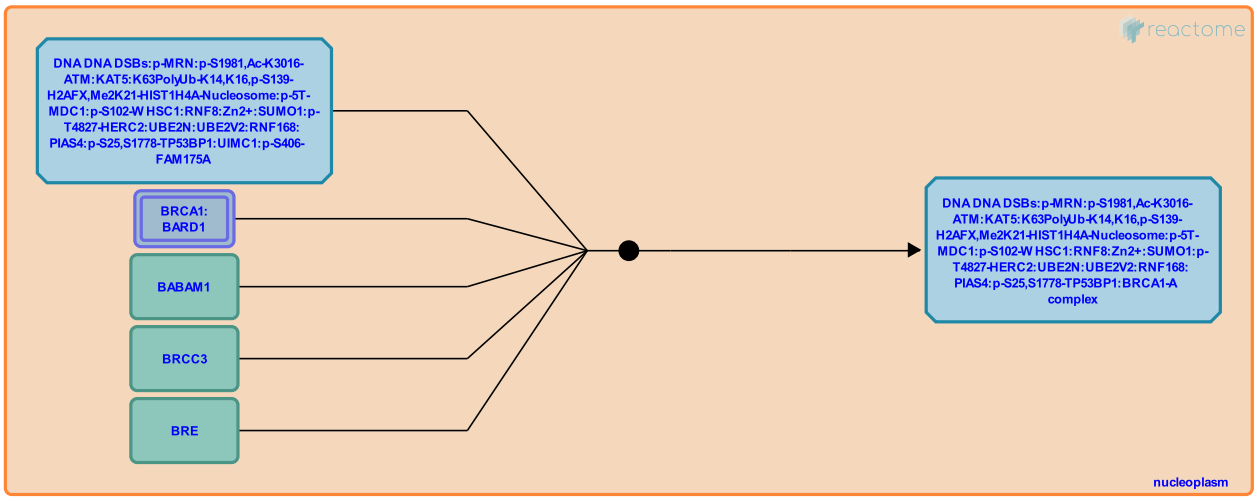
Stable identifier: R-HSA-5683385

Type: binding

Compartments: nucleoplasm



A DNA damage-independent phosphorylation of FAM175A (Abraxas) serine residue S406 creates a pS-X-X-F (phospho-Ser-X-X-Phe) motif that binds BRCT repeats of BRCA1. The BRCA1 cancer predisposing mutation M1775R (Met1775Arg) inhibits BRCA1 binding to FAM175A. FAM175A interaction with UIMC1 (RAP80) enables BRCA1 recruitment to DNA double-strand breaks (DSBs) (Wang et al. 2007). In addition to BRCA1, FAM175A also interacts with BRCC3 (BRCC36) (Wang et al. 2007, Hu et al. 2011) and BABAM1 (MERIT40, NBA1) (Vikrant et al. 2014). BABAM1 simultaneously interacts with BRE (BRCC45) (Hu et al. 2011). Together, BRCA1, BARD1, UIMC1, FAM175A, BRCC36, BRE and BABAM1 form the so-called BRCA1-A complex at DNA DSBs (Wang et al. 2009).

Literature references

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Editions

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