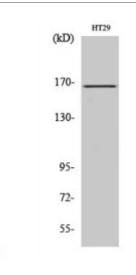


## **Anti-FANCD2** antibody



**Description** Rabbit polyclonal to FANCD2.

Model STJ93040

**Host** Rabbit

**Reactivity** Human, Mouse, Rat

**Applications** ELISA, IHC, WB

**Immunogen** Synthesized peptide derived from human FANCD2 around the non-

phosphorylation site of S222.

**Immunogen Region** 160-240 aa

**Gene ID** 2177

Gene Symbol FANCD2

**Dilution range** WB 1:500-1:2000IHC 1:100-1:300ELISA 1:10000

Specificity FANCD2 Polyclonal Antibody detects endogenous levels of FANCD2

protein.

**Tissue Specificity** Highly expressed in germinal center cells of the spleen, tonsil, and reactive

lymph nodes, and in the proliferating basal layer of squamous epithelium of

tonsil, esophagus, oropharynx, larynx and cervix. Expressed in

cytotrophoblastic cells of the placenta and exocrine cells of the pancreas (at protein level). Highly expressed in testis, where expression is restricted to

maturing spermatocytes.

**Purification** The antibody was affinity-purified from rabbit antiserum by affinity-

chromatography using epitope-specific immunogen.

**Note** For Research Use Only (RUO).

**Protein Name** Fanconi anemia group D2 protein Protein FACD2

Molecular Weight 166 kDa

**Clonality** Polyclonal

**Conjugation** Unconjugated

**Isotype** IgG

**Formulation** Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

**Concentration** 1 mg/ml

**Storage Instruction** Store at -20°C, and avoid repeat freeze-thaw cycles.

Database Links <u>HGNC:3585OMIM:227646</u>

Alternative Names Fanconi anemia group D2 protein Protein FACD2

**Function** Required for maintenance of chromosomal stability. Promotes accurate and

efficient pairing of homologs during meiosis. Involved in the repair of DNA double-strand breaks, both by homologous recombination and single-strand annealing. May participate in S phase and G2 phase checkpoint activation upon DNA damage. Plays a role in preventing breakage and loss of missegregating chromatin at the end of cell division, particularly after replication stress. Required for the targeting, or stabilization, of BLM to non-centromeric abnormal structures induced by replicative stress. Promotes BRCA2/FANCD1 loading onto damaged chromatin. May also be involved in

B-cell immunoglobulin isotype switching.

**Sequence and Domain Family** The C-terminal 24 residues of isoform 2 are required for its function.

Cellular Localization Nucleus. Concentrates in nuclear foci during S phase and upon genotoxic

stress. At the onset of mitosis, excluded from chromosomes and diffuses into the cytoplasm, returning to the nucleus at the end of cell division. Observed in a few spots localized in pairs on the sister chromatids of mitotic chromosome arms and not centromeres, one on each chromatids. These foci coincide with common fragile sites and could be sites of replication fork stalling. The foci are frequently interlinked through BLM-associated ultra-fine DNA bridges.

Following aphidicolin treatment, targets chromatid gaps and breaks.

**Post-translational** Monoubiquitinated on Lys-561 during S phase and upon genotoxic stress by **Modifications** FANCL in complex with E2 ligases UBE2T or UBE2W (isoform 1 and

isoform 2). Deubiquitinated by USP1 as cells enter G2/M, or once DNA repair is completed. Monoubiquitination requires the joint intervention of the FANC core complex, including FANCA, FANCB, FANCC, FANCE, FANCF, FANCG, and FANCM, and proteins involved in cell cycle checkpoints and DNA repair, including RPA1, ATR, CHEK1 and BRCA1, and is mediated by

FANCL/PHF9. Ubiquitination is required for binding to chromatin,

interaction with BRCA1, BRCA2 and MTMR15/FAN1, DNA repair, and normal cell cycle progression, but not for phosphorylation on Ser-222 or interaction with MEN1. Phosphorylated in response to various genotoxic stresses by ATM and/or ATR. Upon ionizing radiation, phosphorylated by ATM on Ser-222 and Ser-1404. Phosphorylation on Ser-222 is required for Sphase checkpoint activation, but not for ubiquitination, foci formation, or

DNA repair. In contrast, phosphorylation by ATR on other sites may be

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