# abcam

### Product datasheet

## Human FANCD2 peptide ab31576

**Description** 

Product name Human FANCD2 peptide

Purity > 90 % HPLC.

Animal free No

Nature Synthetic

**Species** Human

#### **Specifications**

Our Abpromise guarantee covers the use of ab31576 in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

**Applications** Blocking

Form Liquid

Additional notes - First try to dissolve a small amount of peptide in either water or buffer. The more charged

residues on a peptide, the more soluble it is in aqueous solutions.

- If the peptide doesn't dissolve try an organic solvent e.g. DMSO, then dilute using water or

buffer.

- Consider that any solvent used must be compatible with your assay. If a peptide does not

dissolve and you need to recover it, lyophilise to remove the solvent.

- Gentle warming and sonication can effectively aid peptide solubilisation. If the solution is

cloudy or has gelled the peptide may be in suspension rather than solubilised.

- Peptides containing cysteine are easily oxidised, so should be prepared in solution just prior

to use.

#### **Preparation and Storage**

Stability and Storage Shipped at 4°C. Upon delivery aliquot and store at -20°C or -80°C. Avoid repeated freeze / thaw

cycles.

Information available upon request.

#### **General Info**

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**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. Promotes BRCA2/FANCD1 loading onto

damaged chromatin. May also be involved in B-cell immunoglobulin isotype switching.

**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.

Involvement in disease Defects in FANCD2 are a cause of Fanconi anemia complementation group D type 2 (FANCD2)

[MIM:227646]. It is a disorder affecting all bone marrow elements and resulting in anemia, leukopenia and thrombopenia. It is associated with cardiac, renal and limb malformations, dermal pigmentary changes, and a predisposition to the development of malignancies. At the cellular level it is associated with hypersensitivity to DNA-damaging agents, chromosomal instability

(increased chromosome breakage) and defective DNA repair.

**Developmental stage** Highly expressed in fetal oocytes, and in hematopoietic cells of the fetal liver and bone marrow (at

protein level).

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

Post-translational Monoubiquitinated on Lys-561 during S phase and upon genotoxic stress (isoform 1 and isoform

2). Deubiquitinated by USP1 as cells enter G2/M, or once DNA repair is completed.

Monoubiquitination requires the FANCA-FANCB-FANCC-FANCE-FANCF-FANCG-FANCM complex, RPA1 and ATR, 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 S-phase checkpoint activation, but not for ubiquitination, foci formation, or DNA repair. In contrast, phosphorylation by ATR on other sites may be required for ubiquitination and

foci formation.

**Cellular localization** Nucleus. Concentrates in nuclear foci during S phase and upon genotoxic stress.

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modifications

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