Revision 1

#8342 Store at -20°C

UV Induced DNA Damage Response Antibody Sampler Kit

1 Kit (7 x 20 microliters)

Cell Signaling TECHNOLOGY®

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Support: 877-678-TECH (8324)

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For Research Use Only. Not for Use in Diagnostic Procedures.

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Phospho-Histone H2A.X (Ser139) (20E3) Rabbit mAb	9718	20 µl	15 kDa	Rabbit IgG
Phospho-cdc25C (Ser216) (63F9) Rabbit mAb	4901	20 µl	60 kDa	Rabbit IgG
Phospho-Chk1 (Ser345) (133D3) Rabbit mAb	2348	20 µl	56 kDa	Rabbit IgG
RPA32/RPA2 (4E4) Rat mAb	2208	20 µl	32 kDa	Rat IgG1
ATRIP Antibody	2737	20 µl	82 kDa	Rabbit
Phospho-ATR (Ser428) Antibody	2853	20 µl	300 kDa	Rabbit
Microcephalin-1/BRIT1 (D38G5) Rabbit mAb	4120	20 µl	100 kDa	Rabbit IgG
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat
Anti-rat IgG, HRP-linked Antibody	7077	100 µl		Goat

Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

Description

The UV Induced DNA Damage Response Antibody Sampler Kit offers an economical means of investigating proteins involved in the cellular response to UV-induced DNA damage. The kit contains enough primary and secondary antibody to perform two western blot experiments per primary.

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.

Background

Exposure to ultraviolet radiation (UV) has a profound impact on human health and disease (1). Low level UV exposure induces the production of vitamin D and is a key regulator of calcium metabolism. Conversely, overexposure to UV is associated with an increased risk of cancer, immunosuppression, and many eye disorders, such as cataracts. Photons of UV light can directly damage DNA causing thymine dimers and other pyrimidine dimers between adjacent bases (2). Free radicals and reactive oxygen species induced by UV exposure also result in DNA lesions and have been linked to malignant melanoma (3). DNA damage from replicative stress and genotoxic agents like UV activate the ATR-mediated checkpoint pathway and stimulate DNA repair, cell cycle arrest, and apoptosis (4). ATR recruitment to sites of DNA damage and activation depends, at least in part, on interaction with the complex of single-stranded DNA, Replication Protein A (RPA), and direct binding to the ATR-associated adapter protein, ATRIP (5). In addition, the Rad17-RFC and Rad9-Rad1-Hus1 (9-1-1) protein complexes are independently recruited with TopBP1 to fully activate the checkpoint response (6,7). BRIT1 (MCPH1) is required for UV-induced formation of ATR, RPA, and p-Rad17 foci at sites of DNA damage (8-10) and may regulate the expression of several DNA damage response proteins (11). Once activated, ATR phosphorylates a number of mediators, including histone H2AX Ser139 and Chk1 kinase at Ser345. H2AX phosphorylation is a marker of DNA damage. Complete loss of H2AX results in reduced Chk1 activation and impaired survival of cells after UV exposure (12). Chk1 and Chk2 kinase activation is essential for checkpoint-mediated control of cell cycle progression (4). Checkpoint kinases stimulate cell cycle arrest by phosphorylation of a group of tyrosine phosphatases known as Cdc25A, Cdc25B, and Cdc25C (13 -15). Both Chk1 and Chk2 kinases phosphorylate Cdc25C at Ser216 in response to DNA damage and stimulate arrest (16-17).

Background References

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