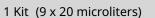
Revision 4

## Double Strand Breaks (DSB) Repair Antibody Sampler Kit



	Cell Signaling
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Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Phospho-ATM (Ser1981) (D25E5) Rabbit mAb	13050	20 µl	350 kDa	Rabbit IgG
ATM (D2E2) Rabbit mAb	2873	20 µl	350 kDa	Rabbit IgG
Phospho-BRCA1 (Ser1524) Antibody	9009	20 µl	220 kDa	Rabbit
DNA-PKcs Antibody	4602	20 µl	450 kDa	Rabbit
Ku80 (C48E7) Rabbit mAb	2180	20 µl	86 kDa	Rabbit IgG
Mre11 (31H4) Rabbit mAb	4847	20 µl	81 kDa	Rabbit IgG
Phospho-p95/NBS1 (Ser343) Antibody	3001	20 µl	95 kDa	Rabbit
Rad50 Antibody	3427	20 µl	153 kDa	Rabbit
XLF Antibody	2854	20 µl	39 kDa	Rabbit
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat

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

The Double Strand Breaks (DSB) Repair Antibody Sampler Kit provides an economical means to investigate repair of double-strand DNA breaks within the cell. The kit contains primary and secondary antibodies to perform two western blots with each antibody.
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.
Double strand DNA breaks (DSB) in mammalian cells can be repaired by the related mechanisms of non-homologous end-joining (NHEJ) and homologous recombination (HR). A DNA-dependent protein kinase composed of DNA-binding subunits Ku70 and Ku86 and the DNA-PKcs catalytic subunit mediates NHEJ repair. The Ku heterodimer binds free DNA ends and recruits DNA-PKcs to the break (1). DNA-PKcs signals areas of DNA damage and recruits additional proteins, such as the Artemis exo- and endonuclease that processes and primes the damaged sequence (2,3). Following replacement DNA synthesis, a ligase complex composed of DNA ligase IV and XRCC4 joins the repaired ends. XRCC4-like factor (XLF) is an essential ligase-associated repair factor that promotes gap-filling during NHEJ (4). Homologous recombination utilizes aligned homologous sequences as a repair template. The MRN complex, composed of Mre11, Rad50, and nibrin (p95/NBS1), plays a critical role in sensing, processing and repairing breaks (5). MRN interacts with BRCA1 and CtIP to facilitate 5' resection of DSB DNA to generate 3' ssDNA ends necessary for repair (6). DNA-binding protein Mre11 exhibits exonuclease and endonuclease activity and is largely responsible for ssDNA end processing (7). Interaction between the MRN complex and ATM kinase promotes association between the kinase and its substrates and likely leads to ATM activation (8). ATM acts a central controller of the cell cycle checkpoint by phosphorylating multiple targets, including c-Abl, BRCA1 and p95/NSB1. Activated c-Abl phosphorylates Rad52, which promotes Rad51 binding to ssDNA and subsequent annealing of ssDNA (7).
1. Gottlieb, T.M. and Jackson, S.P. (1993) <i>Cell</i> 72, 131-42. 2. Franco, S. et al. (2008) <i>J Exp Med</i> 205, 557-64. 3. Collis, S.J. et al. (2005) <i>Oncogene</i> 24, 949-61. 4. Akopiants, K. et al. (2009) <i>Nucleic Acids Res</i> 37, 4055-62. 5. Williams, R.S. et al. (2007) <i>Biochem Cell Biol</i> 85, 509-20. 6. Chen, L. et al. (2008) <i>J Biol Chem</i> 283, 7713-20. 7. Czornak, K. et al. (2008) <i>J Appl Genet</i> 49, 383-96. 8. Lee, J.H. and Paull, T.T. (2007) <i>Oncogene</i> 26, 7741-8.
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