

Huntingtin Interaction Antibody Sampler Kit



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1 Kit (6 x 20 microliters)

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

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
PPAR γ (C26H12) Rabbit mAb	2435	20 μ l	53, 57 kDa	Rabbit IgG
CtBP1 (D2D6) Rabbit mAb	8684	20 μ l	47 kDa	Rabbit IgG
p53 (7F5) Rabbit mAb	2527	20 μ l	53 kDa	Rabbit IgG
SUMO-1 (C9H1) Rabbit mAb	4940	20 μ l		Rabbit IgG
NF- κ B1 p105/p50 (D4P4D) Rabbit mAb	13586	20 μ l	50 Active form. 120 Precursor kDa	Rabbit IgG
ACF1 Antibody	6255	20 μ l	203 kDa	Rabbit
Anti-rabbit IgG, HRP-linked Antibody	7074	100 μ l		Goat

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

Description

The Huntingtin Interaction Antibody Sampler kit provides an economical means of detecting transcription-related proteins that interact with Huntingtin (Htt). This kit contains enough antibody to perform two western blot experiments per primary antibody.

Background

Peroxisome proliferator-activated receptor gamma (PPARG) is a member of the ligand-activated nuclear receptor superfamily and functions as a transcriptional activator (1). Besides its role in mediating adipogenesis and lipid metabolism (2), PPAR gamma also modulates insulin sensitivity, cell proliferation and inflammation (3). CtBP1 is able to regulate gene activity through its intrinsic dehydrogenase activity (4,5) and by interacting with Polycomb Group (PcG) proteins during development (6). Along with its homologue, CtBP2, it acts as a transcriptional corepressor of zinc-finger homeodomain factor deltaEF1 to regulate a wide range of cellular processes through transrepression mechanisms (7). The p53 tumor suppressor protein plays a major role in cellular response to DNA damage and other genomic aberrations. Activation of p53 can lead to either cell cycle arrest and DNA repair or apoptosis (8). DNA damage induces phosphorylation of p53 at Ser15 and Ser20 and leads to a reduced interaction between p53 and its negative regulator, the oncoprotein MDM2 (9). MDM2 inhibits p53 accumulation by targeting it for ubiquitination and proteasomal degradation (10,11). Phosphorylation impairs the ability of MDM2 to bind p53, promoting both the accumulation and activation of p53 in response to DNA damage (9,12). Acetylation appears to play a positive role in the accumulation of p53 protein in stress response (13). Deacetylation of p53 occurs through interaction with the SIRT1 protein, a deacetylase that may be involved in cellular aging and the DNA damage response (14). Small ubiquitin-related modifier 1, 2 and 3 (SUMO-1, -2 and -3) are members of the ubiquitin-like protein family (15). The covalent attachment of the SUMO-1, -2 or -3 (SUMOylation) to target proteins is analogous to ubiquitination. Ubiquitin and the individual SUMO family members are all targeted to different proteins with diverse biological functions. Ubiquitin predominantly regulates degradation of its target (1). In contrast, SUMO-1 is conjugated to RanGAP, PML, p53 and I κ B-alpha to regulate nuclear trafficking, formation of subnuclear structures, regulation of transcriptional activity and protein stability (16-20). Transcription factors of the nuclear factor kappaB (NF- κ B)/Rel family play a pivotal role in inflammatory and immune responses (21, 22). In unstimulated cells, NF- κ B is sequestered in the cytoplasm by I κ B inhibitory proteins (23-25). NF- κ B-activating agents can induce the phosphorylation of I κ B proteins, targeting them for rapid degradation through the ubiquitin-proteasome pathway and releasing NF- κ B to enter the nucleus where it regulates gene expression (26-28). ACF1 (BAZ1A) has distinct roles in development (29), regulation of chromatin structure (30), and DNA damage response (31, 32). Different developmental stages dictate the expression of ACF1 in *Drosophila*, and alterations in ACF1 expression during *Drosophila* development leads to deviation from normal chromatin organization (29).

Background References

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