Revision 1

Store at -20C

#53898

ER Homeostasis Antibody Sampler Kit

1 Kit (9 x 20 microliters)

Cell Signaling TECHNOLOGY®

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Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
FAM134B (E8Y9R) Rabbit mAb	83414	20 µl	70 kDa	Rabbit IgG
CCPG1 (E3C5G) Rabbit mAb	80158	20 µl	105-120 kDa	Rabbit IgG
XBP-1s (E9V3E) Rabbit mAb	40435	20 µl	60 (human), 55 (mouse/rat) kDa	Rabbit IgG
ATF-6 (D4Z8V) Rabbit mAb	65880	20 µl	90-100 kDa	Rabbit IgG
Phospho-eIF2α (Ser51) (D9G8) XP® Rabbit mAb	3398	20 µl	38 kDa	Rabbit IgG
PERK (C33E10) Rabbit mAb	3192	20 µl	140 kDa	Rabbit IgG
ATF-4 (D4B8) Rabbit mAb	11815	20 µl	49 kDa	Rabbit IgG
IRE1α (14C10) Rabbit mAb	3294	20 µl	130 kDa	Rabbit IgG
BiP (C50B12) Rabbit mAb	3177	20 µl	78 kDa	Rabbit IgG
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.

Description

The ER Homeostasis Antibody Sampler Kit provides an economical means of detecting proteins involved in ER homeostasis by regulating ER stress and ER-phagy. The kit includes enough antibodies to perform two western blot experiments with each primary antibody.

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

The endoplasmic reticulum (ER) is a large organelle extending from the nuclear envelope to the plasma membrane with diversity in structure and function (1,2). It functions in calcium storage, lipid and steroid synthesis, and protein folding, processing, and transport. ER structure and function is regulated in a dynamic fashion adapting to situations of ER stress and organelle damage (1,2). When demands on protein processing exceeds capabilities, cells trigger an adaptive mechanism called the unfolded protein response (UPR) which is largely controlled by the activities of three pathways: PERK, IRE1α, and ATF-6 (1). The ER chaperone protein BiP is recruited to unfolded proteins in the ER lumen and its dissociation from PERK, IRE1α, and ATF-6 leads to their activation. PERK is a kinase on the ER membrane that couples ER stress to changes in translation. PERK activation during ER stress leads to phosphorylation of eIF2α, repressing most translation but selectively inducing some targets such as ATF-4, a transcription factor that regulates targets in the recovery of the stress response. IRE1α is an ER protein with endoribonuclease activity that is activated during ER stress and converts XBP-1 from an unspliced XBP-1µ isoform to a spliced XBP-1s isoform functioning as a transcription factor regulating stress response genes. Lastly, during ER stress, ATF-6 is cleaved liberating a mature transcription factor controlling stress response genes.

Subsequent to ER expansion triggered by the UPR, cells may trigger a process of ER-phagy, the degradation of ER fragments through autophagy (3). Autophagy is a process for the bulk degradation of cytoplasmic components by a double membrane autophagosome fusing to the lysosome. Selective autophagy, like ER-phagy, permits the degradation of specific targets. This process generally involves specific cargo receptors containing LIR or GIM domains targeting bound cargo to the autophagosome through interactions with LC3 or GABARAP, respectively. FAM134B was the first ER-phagy receptor discovered. Loss of FAM134B can sensitize cells to apoptosis when challenged by nutrient deprivation or ER stress stimuli. CCPG1 is another ER-phagy cargo receptor that associates with FIP200, a component of the ULK1 complex facilitating ER trafficking to autophagosomes. Importantly, CCPG1 is transcriptionally regulated by ER stress. Taken together, signaling from the UPR and ER-phagy help regulate ER homeostasis. Defects in this process may contribute to pathological conditions, including metabolic and neurological disorders, cancer, and defense against infectious diseases (3).

Background References

1. Sano, R. and Reed, J.C. (2013) *Biochim Biophys Acta* 1833, 3460-3470.
2. Ferro-Novick, S. et al. (2021) *Trends Biochem Sci* 46, 630-639.
3. Hübner, C.A. and Dikic, I. (2020) *Cell Death Differ* 27, 833-842.

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