Revision 1

Store at -20C

Ferroptosis Antibody Sampler Kit

#29650

1 Kit (8 x 20 microliters)

Cell Signaling TECHNOLOGY®

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Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
GPX4 Antibody	52455	20 µl	20, 22 kDa	Rabbit
NCOA4 (E8H8Z) Rabbit mAb	66849	20 µl	80 kDa	Rabbit IgG
KEAP1 (D6B12) Rabbit mAb	8047	20 µl	60-64 kDa	Rabbit IgG
NRF2 (D1Z9C) XP® Rabbit mAb	12721	20 µl	97-100 kDa	Rabbit IgG
4F2hc/SLC3A2 (D3F9D) XP® Rabbit mAb	47213	20 µl	75-120 kDa	Rabbit IgG
FTH1 (D1D4) Rabbit mAb	4393	20 µl	21 kDa	Rabbit IgG
xCT/SLC7A11 (D2M7A) Rabbit mAb	12691	20 µl	35 kDa	Rabbit IgG
DMT1/SLC11A2 (D3V8G) Rabbit mAb	15083	20 µl	55, 70-100 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 Ferroptosis Antibody Sampler Kit provides an economical means of detecting proteins involved in ferroptosis. 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 antibodies.*

Background

Ferroptosis is an iron-dependent form of regulated cell death associated with an increase in lipid peroxides (reviewed in 1,2). Free divalent iron (Fe^{2+}) can lead to spontaneous lipid peroxidation through a Fenton reaction. Ferroptosis is regulated by signaling pathways that control iron storage and oxidative stress. Iron homeostasis is controlled, in part, by ferritin, an iron storage protein consisting of a complex of heavy (FTH1) and light (FTL) chains. Levels of ferritin may be regulated by a selective autophagy process targeting ferritin, termed ferritinophagy. This pathway is mediated by nuclear receptor coactivator 4 (NCOA4), a selective cargo receptor for ferritin (3,4). The divalent metal transporter SLC11A2/DMT1/NRAMP2 regulates iron homeostasis through non-heme absorption in the intestine (5). The glutathione peroxidase pathway has been identified as a key antioxidant defense pathway triggering ferroptosis. The compound RSL3, which directly inhibits GPX4, was identified as an activator of ferroptosis (6). GPX4 converts GSH into oxidized glutathione (GSSH) and reduces cytotoxic lipid peroxides. The glutathione peroxidase pathway is further regulated by System Xc-, an amino acid antiporter consisting of a disulfide-linked heterodimer of SLC7A11/xCT and SLC3A2/4F2hc/CD98, and is inhibited by the ferroptosis inducer erastin (7). Regulation of genes involved in oxidative stress, including *GPX4*, are largely controlled by the transcription factor NRF2 and serves as a defense against ferroptosis (8). Under normal conditions, expression of NRF2 is inhibited through interaction with KEAP1, part of a ubiquitin E3 ligase complex that leads to NRF2 proteasomal degradation. Oxidative stress leads to conformational changes in KEAP1 that disrupts this interaction, resulting in stabilization of NRF2. This process is further regulated through the autophagy pathway in which the autophagy cargo receptor p62/SQSTM1 can competitively inhibit the KEAP1-NRF2 complex, leading to upregulation of NRF2.

Background References

1. Cao, J.Y. and Dixon, S.J. (2016) *Cell Mol Life Sci* 73, 2195-209.
2. Xie, Y. et al. (2016) *Cell Death Differ* 23, 369-79.
3. Mancias, J.D. et al. (2014) *Nature* 509, 105-9.
4. Dowdle, W.E. et al. (2014) *Nat Cell Biol* 16, 1069-79.
5. Gunshin, H. et al. (1997) *Nature* 388, 482-8.
6. Yang, W.S. et al. (2014) *Cell* 156, 317-31.
7. Dixon, S.J. et al. (2014) *Elife* 3, e02523.
8. Fan, Z. et al. (2017) *Oncogenesis* 6, e371.

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