Revision 1

Store at -20C

#16815

Redox Homeostasis and Signaling Antibody Sampler Kit

1 Kit (8 x 20 microliters)

Cell Signaling TECHNOLOGY®

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

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
GPX1 (C8C4) Rabbit mAb	3286	20 µl	22 kDa	Rabbit IgG
GPX4 Antibody	52455	20 µl	20, 22 kDa	Rabbit
Thioredoxin 1 (C63C6) Rabbit mAb	2429	20 µl	12 kDa	Rabbit IgG
Thioredoxin 2 (D1C9L) Rabbit mAb	14907	20 µl	13 kDa	Rabbit IgG
TRXR1 (D1T3D) Rabbit mAb	15140	20 µl	55 kDa	Rabbit IgG
TXNIP (D5F3E) Rabbit mAb	14715	20 µl	55 kDa	Rabbit IgG
Prdx1 (D5G12) Rabbit mAb	8499	20 µl	21 kDa	Rabbit IgG
Phospho-Prdx1 (Tyr194) (D1T9C) Rabbit mAb	14041	20 µl	21 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 Redox Homeostasis and Signaling Antibody Sampler Kit provides an economical means of detecting select components involved in redox homeostasis and signaling. The kit contains enough primary antibodies to perform at least two western blot experiments per 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

Glutathione peroxidase 1 (GPX1) is a cytosolic selenoprotein which reduces hydrogen peroxide to water (1). GPX1 is the most abundant and ubiquitous among the five GPX isoforms identified so far (2). It is an important component in the anti-oxidative defense in cells and is associated with a variety of disease conditions, such as colon cancer (3), coronary artery disease (4), and insulin resistance (1). The selenoprotein glutathione peroxidase 4 (GPX4) is a master regulator of ferroptosis, a form of programmed cell death induced by the iron-dependent lipid peroxidation (5,6). GPX4 converts lipid hydroperoxides to non-toxic lipid alcohols, therefore preventing ferroptosis (6). Research studies show that selenium enhances GPX4 expression and inhibits ferroptotic death to protect neurons (7). In addition, some therapy-resistant cancer cells depend on GPX4 to survive. Loss of GPX4 leads to ferroptosis and thus prevents tumor relapse in mice (8). Furthermore, redox homeostasis mediated by GPX4 is essential for the activation of the cytosolic DNA-sensing cGAS-STING pathway and initiation of the subsequent innate immune response (9). Thioredoxin is a small redox protein found in many eukaryotes and prokaryotes. A pair of cysteines within a highly conserved, active site sequence can be oxidized to form a disulfide bond that is then reduced by thioredoxin reductase (10). Multiple forms of thioredoxin have been identified, including cytosolic thioredoxin 1 (TRX1) and mitochondrial thioredoxin 2 (TRX2). Thioredoxin participates in many cellular processes, including redox signaling, response to oxidative stress, and protein reduction (10). A potential role of thioredoxin in human disorders such as cancer, aging, and heart disease is currently under investigation (11). Thioredoxin can play a key role in cancer progression because it acts as a negative regulator of the proapoptotic kinase ASK1 (12). Changes in thioredoxin expression have been associated with meningococcal septic shock and acute lung injury (13,14). TRXR1 (thioredoxin reductase 1) is a selenocysteine-containing protein that is involved in redox homeostasis (15-20). Its canonical target is thioredoxin, another redox protein (15). Together, they are involved in many functions such as antioxidant regulation (17-20), cell proliferation (16,17,19), DNA replication (16,17), and transcription (17,19). TRXR1 is also capable of reducing a wide array of cellular proteins (15,17). Selenium deficiency, either by diet modification (16,20) or introduction of methylmercury (18), hinders proper expression and function of TRXR1. It is possible that this effect, which results in a higher oxidative state, is a result of the selenocysteine codon (UGA) being read as a STOP codon in the absence of adequate selenium (18). The functions of TRXR1 in cell proliferation and antioxidant defense make it a potential therapeutic target. The ubiquitously expressed thioredoxin-interacting protein (TXNIP) binds and inhibits thioredoxin to regulate cellular redox state (21-23). Research studies demonstrate that hyperglycemia induces TXNIP expression and increases cellular oxidative stress (21). In addition, these studies show that TXNIP reduces glucose

uptake directly by binding the glucose transporter Glut1 to stimulate receptor internalization or indirectly by reducing Glut1 mRNA levels (23). Additional studies indicate that TXNIP plays a role in the regulation of insulin mRNA transcription (24). Microarray analyses indicate that TXNIP acts downstream of PPARγ and is a putative tumor suppressor that may control thyroid cancer cell progression (25). In addition, the TXNIP protein may be a potential therapeutic target for the treatment of type 2 diabetes and some disorders related to ER-stress (26). Prdx1 belongs to a family of non-seleno peroxidases that function as H_2O_2 scavengers. The transient phosphorylation of Prdx1 at Tyr194 leads to inactivation of Prdx1 (27).

Background References

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