

## G2A (M-20): sc-9692

### BACKGROUND

G2A (for G<sub>2</sub> accumulation) is a seven transmembrane G protein-coupled receptor that is upregulated in response to DNA damage and stress. G2A is predominantly expressed in hematopoietic tissues and in hematopoietic stem cells, and it is more highly detected in pro-B cells, while lower expression is observed in immature B cells and pre-B cells. G2A is expressed throughout T cell maturation, and it is further increased in response to T-cell activation. Ectopic expression of a G2A fusion protein in NIH/3T3 fibroblasts induces a cell cycle arrest that is consistent with a block at the G<sub>2</sub>/M transition. G2A is also able to attenuate the proliferative effects of Bcr-Abl, a chimeric tyrosine kinase oncogene, suggesting that G2A possesses anti-oncogenic properties. The amino acid sequence of G2A contains a destruction box motif that is consistently observed in cyclins, where it is required for ubiquitination and proteolytic degradation.

### REFERENCES

1. Bedi, A., et al. 1995. Bcr-Abl-mediated inhibition of apoptosis with delay of G<sub>2</sub>/M transition after DNA damage: a mechanism of resistance to multiple anticancer agents. *Blood* 86: 1148-1158.
2. Allday, M.J., et al. 1995. DNA damage in human B cells can induce apoptosis, proceeding from G<sub>1</sub>/S when p53 is transactivation competent and G<sub>2</sub>/M when it is transactivation defective. *EMBO J.* 14: 4994-5005.
3. Hochstrasser, M. 1996. Ubiquitin-dependent protein degradation. *Annu. Rev. Genet.* 30: 405-439.
4. Weng, Z., et al. 1998. A DNA damage and stress inducible G protein-coupled receptor blocks cells in G<sub>2</sub>/M. *Proc. Natl. Acad. Sci. USA* 95: 12334-12339.
5. Shimizu, A., et al. 1998. CyclinG contributes to G<sub>2</sub>/M arrest of cells in response to DNA damage. *Biochem. Biophys. Res. Commun.* 242: 529-533.

### CHROMOSOMAL LOCATION

Genetic locus: Gpr132 (mouse) mapping to 12 F1.

### SOURCE

G2A (M-20) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the N-terminus of G2A of mouse origin.

### PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-9692 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

### STORAGE

Store at 4° C, **\*\*DO NOT FREEZE\*\***. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

### RESEARCH USE

For research use only, not for use in diagnostic procedures.

### APPLICATIONS

G2A (M-20) is recommended for detection of G2A of mouse and rat origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for G2A siRNA (m): sc-44371, G2A shRNA Plasmid (m): sc-44371-SH and G2A shRNA (m) Lentiviral Particles: sc-44371-V.

Molecular Weight of G2A: 42-46 kDa.

Positive Controls: rat skeletal muscle tissue extract: sc-364810.

### RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western Blotting: use donkey anti-goat IgG-HRP: sc-2020 (dilution range: 1:2000-1:100,000) or Cruz Marker™ compatible donkey anti-goat IgG-HRP: sc-2033 (dilution range: 1:2000-1:5000), Cruz Marker™ Molecular Weight Standards: sc-2035, TBS Blotto A Blocking Reagent: sc-2333 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml). 3) Immunofluorescence: use donkey anti-goat IgG-FITC: sc-2024 (dilution range: 1:100-1:400) or donkey anti-goat IgG-TR: sc-2783 (dilution range: 1:100-1:400) with UltraCruz™ Mounting Medium: sc-24941.

### SELECT PRODUCT CITATIONS

1. Yan, J.J., et al. 2004. Therapeutic effects of lysophosphatidylcholine in experimental sepsis. *Nat. Med.* 10: 161-167.
2. Frasch, S.C., et al. 2008. NADPH oxidase-dependent generation of lysophosphatidylserine enhances clearance of activated and dying neutrophils via G2A. *J. Biol. Chem.* 283: 33736-33749.
3. Frasch, S.C., et al. 2011. Signaling via macrophage G2A enhances efferocytosis of dying neutrophils by augmentation of Rac activity. *J. Biol. Chem.* 286: 12108-12122.
4. Garbin, U., et al. 2013. Expansion of necrotic core and shedding of MerTK receptor in human carotid plaques: a role for oxidized polyunsaturated fatty acids? *Cardiovasc. Res.* 97: 125-133.
5. Frasch, S.C., et al. 2013. Neutrophils regulate tissue Neutrophilia in inflammation via the oxidant-modified lipid lysophosphatidylserine. *J. Biol. Chem.* 288: 4583-4593.

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