SANTA CRUZ BIOTECHNOLOGY, INC.

G2A (N-20): sc-19488

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The Power to Question

BACKGROUND

G2A (for G_2 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_2/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_2/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_1/S when p53 is transactivation competent and G_2/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_2/M. Proc. Natl. Acad. Sci. USA 95: 12334-12339.
5. Shimizu, A., et al. 1998. CyclinG contributes to G_2/M arrest of cells in response to DNA damage. Biochem. Biophys. Res. Commun. 242: 529-533.
6. Aguda, B.D. 1999. A quantitative analysis of the kinetics of the G_2 DNA damage checkpoint system. Proc. Natl. Acad. Sci. USA 96: 11352-11357.

CHROMOSOMAL LOCATION

Genetic locus: GPR132 (human) mapping to 14q32.33.

SOURCE

G2A (N-20) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the N-terminus of G2A of human 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-19488 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.

APPLICATIONS

G2A (N-20) is recommended for detection of G2A of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), 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 (h): sc-43776, G2A shRNA Plasmid (h): sc-43776-SH and G2A shRNA (h) Lentiviral Particles: sc-43776-V.

Molecular Weight of G2A: 42-46 kDa.

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) 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. Jin, Y., et al. 2005. Human resting $CD16^-$, $CD16^+$ and IL-2-, IL-12-, IL-15- or IFN-α-activated natural killer cells differentially respond to sphingosylphos-phorylcholine, lysophosphatidylcholine and platelet-activating factor. Eur. J. Immunol. 35: 2699-2708.
2. Frasch, S.C., et al. 2007. Lysophospholipids of different classes mobilize neutrophil secretory vesicles and induce redundant signaling through G2A. J. Immunol. 178: 6540-6548.
3. Khan, S.Y., et al. 2010. Lysophosphatidylcholines activate G2A inducing $G_{\alpha\ i\text{-}1}$-$G_{\alpha\ q/11}$-Ca^{2+} flux, $G_{\beta\ \gamma}$-Hck activation and clathrin/β-arrestin-1/GRK6 recruitment in PMNs. Biochem. J. 432: 35-45.
4. Ogawa, A., et al. 2010. Identification and analysis of two splice variants of human G2A generated by alternative splicing. J. Pharmacol. Exp. Ther. 332: 469-478.
5. Hasegawa, H., et al. 2011. Lysophosphatidylcholine enhances the suppressive function of human naturally occurring regulatory T cells through TGF-β production. Biochem. Biophys. Res. Commun. 415: 526-531.

RESEARCH USE

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