# *DB029*: STAT3 (C20)

## Background:

Signal Transducers and Activators of Transcription (STATs) are a family of cytoplasmic proteins that participate in cellular responses to cytokines and growth factors (1). Many cytokines involved in immune responses utilize the Jak-STAT signaling pathway. Jaks are receptor-associated protein tyrosine kinases, and STATs are activated by tyrosine phosphorylation (2). Abnormal signaling of the JAK-STAT pathway has been implicated in hematopoietic disorders including severe combined immunodeficiency and leukemia (4). STATs have been implicated in programming gene expression in biological events such as embryonic development, programmed cell death, organogenesis, innate immunity, adaptive immunity and cell growth regulation in many organisms (3). STAT1 knockout mice are defective in interferon-mediated functions. STAT4 and STAT6 knockout mice show defective responses to IL-12 and IL-4, respectively. Analyses of STAT5a and STAT5b knockout mice reveal important roles in prolactin-mediated mammary gland development and growth hormone-mediated induction of sexual dimorphism, respectively. Conditional knockout study of STAT3 demonstrates its critical roles in cytokine-mediated functions in several tissues, including T cells, macrophages, skin, and mammary gland (5). Abnormal activity of certain STAT family members, particularly STAT3 and STAT5, is associated with a wide variety of human malignancies, including hematologic, breast, head and neck, and prostate cancers. (1). STAT5A and STAT6, are selectively activated when the heart is subjected to ischemic injury, whereas activation of STAT3 and STAT5A is involved in myocardial hypertrophy (6). STAT5 and STAT3, as these two STAT molecules are required for normal breast development and involution, respectively, and may play an important role in breast carcinogenesis (7). STAT3 beta, a shorter form of STAT3 (also referred to as STAT3 alpha) has been shown to be transcriptionally active under conditions where STAT3 alpha is not, and STAT3 beta and c-Jun are capable of cooperative activation of certain promoters (8).

## Origin:

STAT3 (C20) is provided as an affinity purified rabbit polyclonal antibody, raised against a peptide mapping to the carboxy terminus of mouse STAT3 p92

## Product Details:

Each vial contains 200 µg/ml of affinity purified rabbit IgG STAT3 (C20) DB029, in 1 ml PBS containing 0.1 % sodium azide and 0.2% gelatin.

## Competition Studies:

A blocking peptide is also available, *DB029P*, for use in competition studies. Each vial contains 100 µg of peptide in 0.5 ml PBS with 0.1% sodium azide and 100 µg BSA.

## Specificity:

STAT3 (C20) DB029 reacts with STAT3 p92 of mouse, rat, and human origin by western blotting, immunoprecipitation, and immunohistochemistry, but does not react to STAT 3b.

## Storage:

Store this product at 4º C, do not freeze. The product is stable for one year from the date of shipment.

## References:


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4. Nosaka T, Kitamura T. Janus kinases (JAKs) and signal transducers and activators of transcription (STATs) in hematopoietic cells. Int J Hematol. 2000 Jun; 71(4): 309-19.
5. Takeda K, Akira S. STAT family of transcription factors in cytokine-mediated biological responses. Cytokine Growth Factor Rev. 2000 Sep; 11(3): 199-207.
6. Mascareno E, Siddiqui MA. The role of Jak/STAT signaling in heart tissue renin-angiotensin system. Mol Cell Biochem. 2000 Sep; 212(1-2): 171-5.
7. Bromberg J. Signal transducers and activators of transcription as regulators of growth, apoptosis and breast development. Breast Cancer Res. 2000; 2(2): 86-90.
8. Schaefer TS, Sanders LK, Nathans D. Cooperative transcriptional activity of Jun and Stat3 beta, a short form of Stat3. Proc Natl Acad Sci U S A. 1995 Sep 26; 92(20): 9097-101.



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