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ProductInformation

MONOCLONAL ANTI-LCK
CLONE 3A5
Purified Mouse Immunoglobulin

Product Number **L 8414**

Product Description

Monoclonal Anti-Lck (mouse IgG2b isotype) is derived from the hybridoma produced by the fusion of splenocytes from mice immunized with recombinant protein corresponding to the amino acids 1 to 225 of murine p56lck and mouse myeloma p3-NS1-Ag4-1 cells. The antibody is purified by Protein A/Protein G chromatography.

Monoclonal Anti-Lck recognizes human, mouse and rat Lck protein also known as T cell-specific protein-tyrosine kinase, $p56^{Lck}$ or p56Lck. It has been used in ELISA, immunoblotting (56 kDa), and immunoprecipitation.

The T cell antigen receptor (TCR) plays a crucial role in thymocyte differentiation and T cell activation. After antigen binding to the TCR, and with engagement of other co-receptors and their associated ligands (such as CD4, major histocompatibility complex class II, CD28, B7, CD8, and MHC I), signal transduction cascades are activated. The earliest measurable biochemical event is the activation of protein-tyrosine kinases, resulting in the phosphorylation of multiple cellular substrates. At least three protein-tyrosine kinases are known to be involved in TCR signaling at the level of the receptor, including p59Fyn, p56Lck, and ZAP70.[1] STAT5 transcription factor becomes immediately and transiently phosphorylated on tyrosine 694 in response to TCR stimulation. Studies using a Lck-deficient T cell line confirmed the role of Lck in TCR stimulated STAT5 activation.[2]

During T cell development, assembly and expression of a complete gene for the β chain of the TCR blocks further rearrangement at the β locus. This process is referred to as allelic exclusion. This results in the production and expansion of CD4+8+ cells. Studies in transgenic animals suggest that the Lck is the protein tyrosine kinase that regulates allelic exclusion at the T cell receptor β (TCRB) locus.[3]

Mice lacking Lck as well as transgenic mice expressing dominant-negative mutations of Lck demonstrate severe T cell developmental defects, such as severe combined immunodeficiency.[4] T cell survival in transgenic mice was independent of Lck but this kinase played an essential role in TCR-driven homeostatic proliferation of naive T cells in response to the T cell-deficient host environment.[5] Studies on type 1 diabetic patients have shown that peripheral T lymphocyte activation in response to TCR/CD3 stimulation was markedly reduced in correlation with a reduced amount of Lck in resting T lymphocytes. This leads to the conclusion that a primary deficiency in human type 1 diabetes is a defect in TCR/CD3-mediated T cell activation due to the abnormal expression of the Lck tyrosine kinase.[6]

Reagent

Monoclonal Anti-Lck is supplied as a solution in phosphate buffered saline, pH 7.4, with 0.08% sodium azide as a preservative.

Precautions and Disclaimer

Due to the sodium azide content, a material safety data sheet (MSDS) for this product has been sent to the attention of the safety officer of your institution. Consult the MSDS for information regarding hazards and safe handling practices.

Storage/Stability

Store at –20 °C. Freeze the solution in working aliquots for extended storage. Avoid repeated freezing and thawing to prevent denaturing the antibody. The antibody is stable for at least 12 months when stored appropriately. Working dilutions should be discarded if not used within 12 hours.

Product Profile

A recommended working concentration of 1 µg/ml is determined by the immunoblotting using a Jurkat cell lysate.

For ELISA and immunoprecipitation, use a working concentration of 2 µg/mg of protein lysate.

Positive controls include Jurkat cells, T cell hybridoma or T cells in tonsils.

Note: In order to obtain best results using different techniques and preparations we recommend determining optimal working concentration by titration.

References

1. Saint-Ruf, C. et al., Different initiation of pre-TCR and γ-δ-TCR signaling. Nature, **406**, 524-527 (2000).
2. Welte, T., et al., STAT5 interaction with the T cell receptor complex and stimulation of T cell proliferation. Science, **283,** 222-225 (1999).
3. Anderson, S. J., et al., Protein tyrosine kinase p56(lck) controls allelic exclusion of T-cell receptor β-chain genes. Nature, **365**, 552-554 (1993).
4. Goldman, F.D., et al., Defective expression of p56Lck in an infant with severe combined immunodeficiency. J. Clin. Invest., **102,** 421-429 (1998).
5. Seddon, B., Long-term survival but impaired homeostatic proliferation of naive T cells in the absence of p56(Lck). Science, **290**, 127-131, (2000).
6. Nervi, S., et al., Specific deficiency of p56(lck) expression in T lymphocytes from type 1 diabetic patients. J. Immunol, **165,** 5874-5883 (2000).

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