

**DESCRIPTION**

**Source** Mouse myeloma cell line, NS0-derived  
Ala127-Leu309  
Accession # Q9WU72

**N-terminal Sequence Analysis** Ala127

**Structure / Form** Noncovalently-linked homotrimer

**Predicted Molecular Mass** 21 kDa

**SPECIFICATIONS**

**SDS-PAGE** 19-23 kDa, reducing conditions

**Activity** Measured in a cell proliferation assay using anti-IgM stimulated mouse B cells.  
The ED<sub>50</sub> for this effect is typically 0.1-0.5 ng/mL in the presence of goat anti-mouse IgM  $\mu$  chain.

**Endotoxin Level** <0.10 EU per 1  $\mu$ g of the protein by the LAL method.

**Purity** >85%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

**Formulation** Lyophilized from a 0.2  $\mu$ m filtered solution in MOPS, NaCl and TCEP.  
See Certificate of Analysis for details.

**PREPARATION AND STORAGE**

**Reconstitution** Reconstitute at 100-500  $\mu$ g/mL in sterile water.

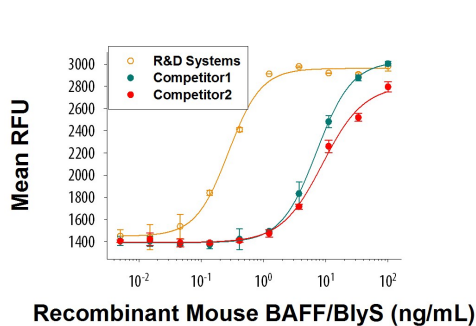
**Shipping** The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.

**Stability & Storage** **Use a manual defrost freezer and avoid repeated freeze-thaw cycles.**

- 12 months from date of receipt, -20 to -70 °C as supplied.
- 1 month, 2 to 8 °C under sterile conditions after reconstitution.
- 3 months, -20 to -70 °C under sterile conditions after reconstitution.

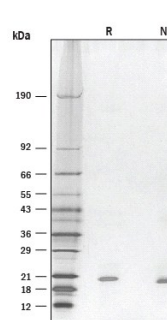
**DATA**

**Bioactivity**



Recombinant Mouse BAFF/BLyS/TNFSF13B (Catalog # 8876-BF/CF) induces mouse B cell proliferation. The ED<sub>50</sub> for this effect is typically 0.1-0.5 ng/mL. The ED<sub>50</sub> for the two competitors is >5 ng/mL, which is more than 25-fold weaker.

**SDS-PAGE**



1  $\mu$ g/lane of Recombinant Mouse BAFF/BLyS/TNFSF13B was resolved with SDS-PAGE under either reducing (R) or non-reducing (NR) conditions and visualized by silver staining, showing a single band at 20 kDa

**BACKGROUND**

B-cell activating factor (BAFF), also known as BLyS, TALL-1, THANK, and TNFSF13B, is a 32 kDa transmembrane glycoprotein in the TNF ligand superfamily. It is involved in multiple aspects of immune system regulation, particularly towards B cells (1, 2). Mature mouse BAFF consists of a 47 amino acid (aa) cytoplasmic domain, a 21 aa transmembrane segment, and a 241 aa extracellular domain (ECD) with a stalk region and one TNF-like domain (3, 4). Within aa 127-309 of the ECD, mouse BAFF shares 72% aa sequence identity with human BAFF. It can be expressed as a homotrimer or as a heteromer in association with the related TNFSF member APRIL (4, 5). A 18 kDa fragment containing the TNF-like domain can be released by proteolysis between Arg126 and Ala127 (4). Soluble BAFF is stored intracellularly in neutrophils and released upon inflammatory stimulation (6). Alternative splicing generates an isoform termed deltaBAFF that lacks 19 aa between the proteolytic cleavage site and the TNF-like domain. deltaBAFF can form heteromers with BAFF and negatively regulates BAFF function (7). BAFF is produced by many hematopoietic cell types including by monocytes, macrophages, neutrophils, dendritic cells, and T cells and also by adipocytes (1, 2, 8). Both BAFF and APRIL are functional ligands for the TNF receptor superfamily members BCMA and TACI, and BAFF additionally binds and signals through BAFF R (9, 10). All three receptors are primarily expressed by B cells (10). BAFF plays a critical role in the development and survival of B lineage cells (2, 11, 12). Mice that over-express BAFF exhibit elevated B cell numbers, increased formation and size of germinal centers, and symptoms of autoimmunity (13). Soluble BAFF is elevated in B cell malignancies, autoimmunity, and other immune disorders (1). In addition, BAFF costimulates T cell activation, promotes a Th1 biased immune response, and promotes the expansion of Treg cells (14-16). BAFF also promotes monocyte survival, proinflammatory cytokine secretion, and differentiation to macrophages (17).

**References:**

1. Lied, G.A. and A. Berstad (2011) *Scand. J. Immunol.* **73**:1.
2. Mackay, F. *et al.* (2010) *Immunol. Rev.* **237**:205.
3. Moore, P.A. *et al.* (1999) *Science* **285**:260.
4. Schneider, P. *et al.* (1999) *J. Exp. Med.* **189**:1747.
5. Roschke, V. *et al.* (2002) *J. Immunol.* **169**:4314.
6. Scapini, P. *et al.* (2003) *J. Exp. Med.* **197**:297.
7. Gavin, A.L. *et al.* (2003) *J. Biol. Chem.* **278**:38220.
8. Alexaki, V.-I. *et al.* (2009) *J. Immunol.* **183**:5948.
9. Yu, G. *et al.* (2000) *Nat. Immunol.* **1**:252.
10. Thompson, J.S. *et al.* (2001) *Science* **293**:2108.
11. Schiemann, B. *et al.* (2001) *Science* **293**:2111.
12. Litinskiy, M.B. *et al.* (2002) *Nat. Immunol.* **3**:822.
13. Batten, M. *et al.* (2000) *J. Exp. Med.* **192**:1453.
14. Huard, B. *et al.* (2001) *J. Immunol.* **167**:6225.
15. Sutherland, A.P.R. *et al.* (2005) *J. Immunol.* **174**:5537.
16. Walters, S. *et al.* (2009) *J. Immunol.* **182**:793.
17. Chang, S.K. *et al.* (2006) *Blood* **108**:2687.