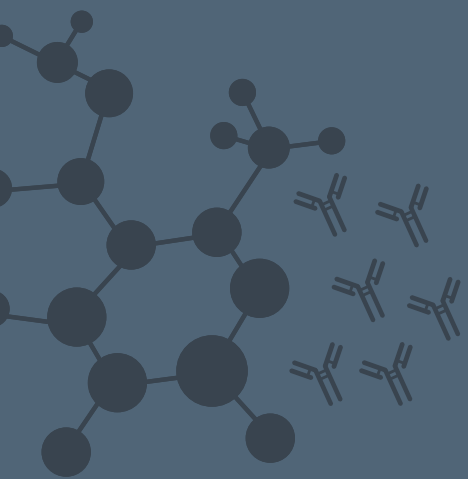
InvivoGen insight

## CONTENTS:






InvivoGen
www.invivogen.com
2025


# TSLP and TL1A cytokines: Promising targets in immune diseases


**TSLP (thymic stromal lymphopoietin) and TL1A (Tumor necrosis factor-like cytokine 1A) have emerged as game-changers in treating immune-mediated inflammation diseases (IMIDs). This review explores the biological activities of TSLP and TL1A, their significance in asthma and Crohn's disease, and recent breakthroughs in drug development. Discover why they are creating a buzz for biopharmas and shaping the future of personalized medicine.**


## The quest for new therapeutic targets

Increased cytokine levels in serum often indicate an ongoing immune response to fight infections or malignancies. However, elevated cytokines can also reflect dysregulated immune responses, leading to inflammatory and autoimmune disorders. The development of recombinant monoclonal antibodies (mAbs) and in vivo mouse disease models has made it easier to identify and manipulate important cytokine(s) as potential therapeutic targets. Today, more than 30 therapeutic mAbs neutralizing cytokines or their receptors have been authorized to treat IMIDs (e.g. Crohn's disease, asthma, atopic dermatitis, rheumatoid arthritis, lupus)[1]. However, long-term cytokine suppression, such as TNF-**α** or IL-4-targeting strategies, can lead to side effects or ineffective treatment for many individuals[2,3].

## Why are TSLP and TL1A promising targets?

The lack of complete efficacy for current therapeutics may be explained by the fact that they target individual, downstream elements of the inflammatory cascade. TSLP and TL1A stand out as they are upstream elements, acting at the early stages of inflammation. Targeting these cytokines offers a strong twofold advantage:

- Modulating cytokines upstream rather than downstream can help achieve **broader and more effective management of inflammation**.
- This approach also significantly improves the likelihood of **preventing dysregulated immune responses**, lowering symptom exacerbation.

The biopharma industry is now setting its sights on TSLP and TL1A, particularly for severe asthma and Crohn's disease. The race is on to produce effective blocking mAbs:

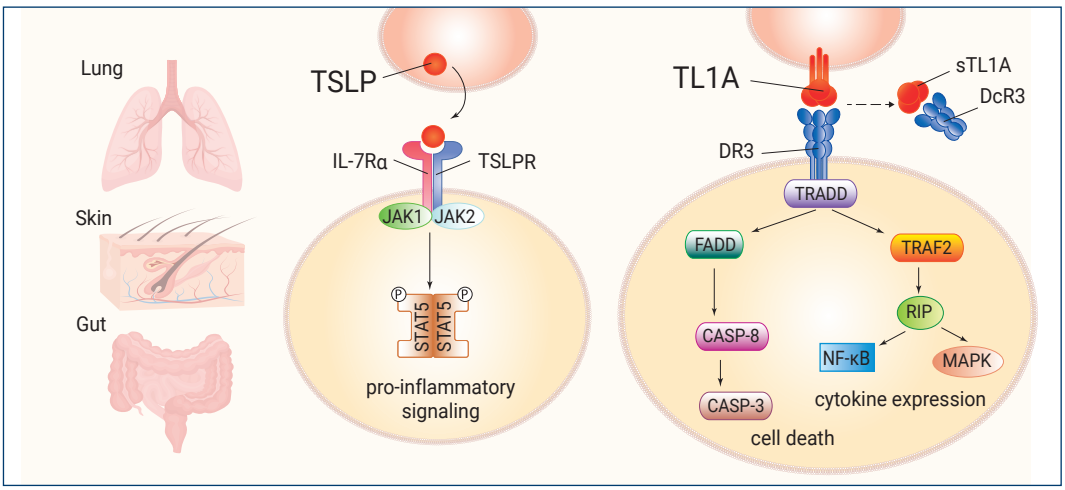
- **TSLP-neutralizing Tezepelumab** was approved by the FDA in 2021 for severe asthma[4]. Other biologics are still in clinical development, such as AZD8630/AMG104, an inhaled anti-TSLP mAb, or Verekitug, an anti-TSLP receptor mAb[5].
- **TL1A-neutralizing Tulisokibart and Duvakitug**, are currently undergoing phase II/III clinical trials for IBD (Crohn's disease and ulcerative colitis)[6-8]. Duvakitug is also being studied for its safety as an asthma therapy[9,10].

## What are the functions of TSLP and TL1A?

TSLP and TL1A exert a common alarmin cytokine function[11]. They are rapidly released from epithelial cells upon infection, allergen exposure, or mechanical injury to induce and propagate inflammation.

- **TSLP** belongs to the common **γ** chain (**γc**) cytokine family and is primarily produced by epithelial and stromal cells in barrier tissues, (lungs, skin, and gastrointestinal tract). It is also produced by innate cells like dendritic cells (DCs), basophils, and mast cells[12]. TSLP binds to a heterodimeric transmembrane receptor comprising the IL-7Rα and TSLPR chains. It signals through tyrosine kinases of the Janus family (JAK1, JAK2) and signal transducer and transcription activators (STATs), notably STAT5[12]. TSLP is a **critical mediator of type 2 (T2) immune responses**, characterized by the release of IL-4, IL-5, IL-13, IL-17A, and IL-22 cytokines. It acts through DC maturation, Th2 and Th17 subsets polarization, as well as activation of group 2 innate lymphoid cells (ILC2s)[11-13].

- **TL1A** is a trimeric molecule belonging to the tumor necrosis factor (TNF) superfamily. It is produced in endothelial cells, activated DCs, and macrophages. It can exist in both membrane-bound and soluble forms[14]. TL1A binds to a homotrimeric



transmembrane death receptor 3 (DR3), or to its soluble decoy counterpart (DcR3). It triggers NF-κB and MAPKs activation to induce **inflammatory gene expression**. Alternatively, it can trigger caspase-8 activation, resulting in **cell death**[14]. TL1A provides a co-stimulatory signal to T cells, influencing their differentiation into Th1, Th2, and Th17 cells[14, 15]. While Th17 and Th2 cells mainly produce T2 cytokines, Th1 essentially secrete IFN-γ and TNF-α[14]. TL1A/DR3 signaling also promotes ILC2s to proliferate and produce large amounts of IL-5, IL-13, and transiently IL-9[11, 14, 15]. TL1A dual signaling capabilities can also induce apoptosis in certain contexts, highlighting its complex role in immune regulation[14].

Overall, although they do not bind to receptors of the same family nor signal through identical pathways, TSLP and TL1A display redundancy in their immune functions. This ensures that the immune system continues to operate even when one cytokine is lacking or compromised. Interestingly, TSLP appears to perform a role upstream of TL1A in ILC2s. In the context of allergic airway inflammation, TSLP induces ILC2s to upregulate DR3 cell-surface expression, thereby exacerbating inflammatory responses[11, 16]. Whether this cooperation holds for other diseases remains to be investigated.

## How do TSLP and TL1A contribute to asthma & Crohn's disease?

TSLP and TL1A mediate early protective immune responses. However, they can also contribute negatively to chronic inflammatory disorders, including asthma and Crohn's disease. They do so by:

- **Genetic predisposition:** TSLP and TL1A polymorphisms have been linked to disease susceptibility and severity[11, 12].
- **Overactivation of downstream actors:** Their target cells foster persistent local inflammation and immune cell infiltration, contributing to tissue damage[14, 18].
- **TSLP and TL1A impact in asthma:** TSLP and TL1A stimulate the production of T2 cytokines by lung-resident immune cells, notably ILC2s, Th2, and Th17 cells[3, 11, 16, 18]. These T2 cytokines drive lung eosinophilia, mucus overproduction, and airway hyperresponsiveness.
- **TL1A impact in Crohn's disease:** TL1A alters the epithelial-mesenchymal transition, resulting in colonic fibrosis and inflammatory reactions[14]. TL1A also increases the production of IFN-γ and TNF-α by Th1 cells, and IL-17 by Th17 cells, exacerbating intestinal inflammation[14, 17].

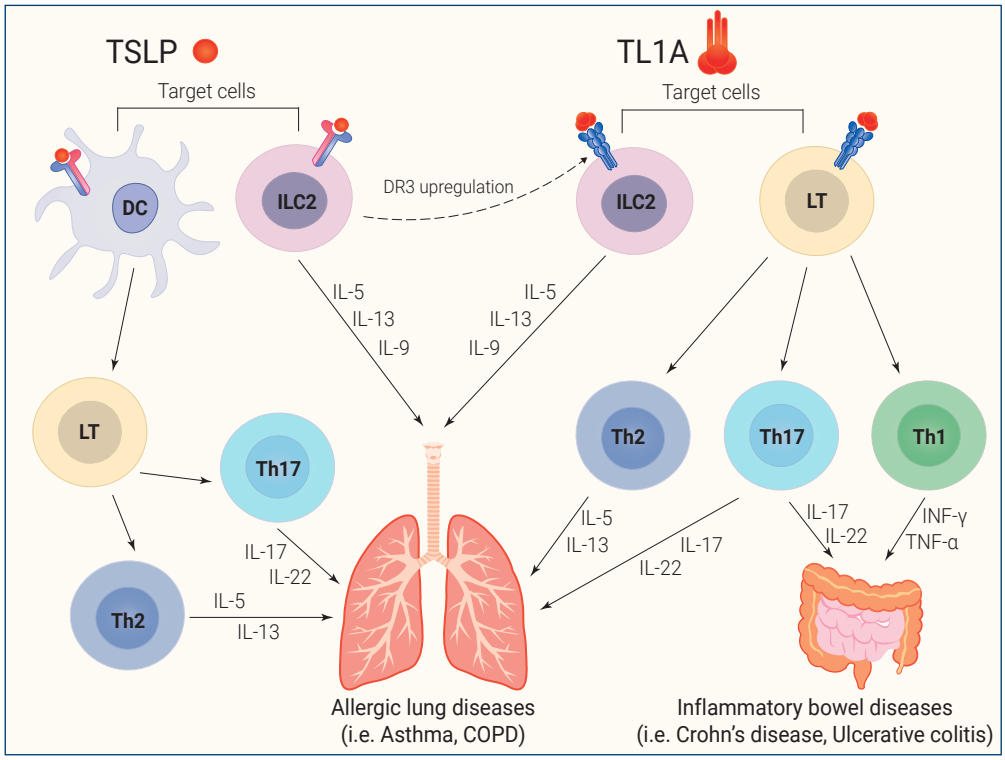
## TSLP and TL1A drug development

Pharmaceutical companies have developed potent mAbs to block TSLP or TL1A signaling and stop the downstream inflammatory cascade in the setting of asthma and IBD.

- Amgen's **Tezepelumab** (AMG157) is the only anti-TSLP antibody currently licensed for treating severe asthma[3-5]. This drug stands out among the panels of asthma treatments as it improves clinical outcomes in all asthma endotypes. Importantly, Tezepelumab can modulate airway inflammation that is not controlled with biologics that block specific downstream elements of the inflammatory cascade (e.g. anti-IL-4/13 receptor antibody Dupilumab, inhaled corticosteroids)[4]. Tezepelumab is being tested in clinical trials for many illnesses, including chronic rhinosinusitis, chronic obstructive pulmonary disease, and chronic spontaneous urticaria[12]. The TSLP-based therapeutic landscape is still expanding, with numerous biologics in the preclinical or clinical development stages. These include mAbs neutralizing TSLP (e.g., AZD8630) or TSLP receptor (Verekitug), as well as a small chemical inhibitor, BP79, targeting the TSLP receptor[5].
- Anti-TL1A inhibitors such as **Tulisokibart** (MK-7240 or PRA023), RVT-3101 (PF-06480605), and **Duvakitug** (TEV-48574) have demonstrated "best-in-class" potential for treating inflammation and scarring in IBD[6-8]. Duvakitug is currently being studied for its safety as an asthma medication[9, 10].

## Stopping the "atopic march"

As the incidence of allergic diseases continues to rise worldwide, strong efforts are being made to understand the mechanisms underlying these conditions for better prevention and control. The concept of "atopic march" is based on studies suggesting that allergic diseases manifest gradually and sequentially: from atopic dermatitis and food allergy in infancy to allergic asthma and allergic rhinitis in childhood[19]. TSLP and TL1A, along with other alarmins found in the skin, gastrointestinal tract, and lungs, may be useful biomarkers for predicting the "atopic march". Future research could lead to the development of tailored treatments based on therapies that block alarmins locally (topical, oral, or inhalation delivery) and temporally (during infancy or youth).




Overall, the upstream positioning of TSLP and TL1A in the inflammation cascade in IMIDs makes them attractive pharmacological targets. According to available data, biopharma is now focusing a lot of work on TSLP and TL1A to enhance current treatments and break through the efficacy ceiling associated with current top medications on the market.


1. **Leonard, W.J. *et al.*, 2023.** Nat Rev Drug Discov, 22(10):827. 2. **Steeland, S. *et al.*, 2018.** Int J Mol Sci, 19(5):1442. 3. **Menzies-Gow, A. *et al.*, 2020.** Respir Res, 21(1):268. 4. **Panettieri, R. *et al.*, 2024.** J Asthma Allergy, 17:219. 5. **Hansi, R.K., *et al.*, 2024.** Biomedicines, 12(10):2312. 6. **Sands, B.E. *et al.*, 2024.** N Engl J Med, 391(12):1119. 7. **Danese, S. *et al.*, 2021.** Clin Gastr Hep. 19(11):2324. 8. **Solitano, V. *et al.*, 2024.** Med. 5(5):386. 9. **Balyan, R. *et al.*, 2024.** Jour Crohn's and Colitis. 18(Supplement_1):i1215. 10. **Raphael, G. *et al.*, 2024.** Jour Crohn's and Colitis. 18(Supplement_1): i1908. 11. **Schmitt, P. *et al.*, 2024.** J Exp Med, 221(6). 12. **Ebina-Shibuya, R. *et al.*, 2023.** Nat Rev Immunol, 23(1):24. 13. **Gauvreau, G.M. *et al.*, 2023.** Allergy, 78(2):402. 14. **Xu, W.D. *et al.*, 2022.** Front Immunol, 13(891328). 15. **Valatas, V. *et al.*, 2019.** 10.3389/fimmu.2019.00583. 16. **Machida, K. *et al.*, 2020.** Am J Resp Crit Care Med, 202(8):1105. 17. **Jin, S. *et al.*, 2013.** Muc Immunol, 6(5):886. 18. **Cecchi, L. *et al.*, 2022.** Eur Ann Allergy Clin Immunol, 54(6):247. 19. **Yang, L. *et al.*, 2020.** doi: 10.3389/fimmu.2020.01907.

# TSLP and TL1A cellular assays

InvivoGen introduces a comprehensive set of tools to study TSLP and TL1A signaling and modulation. These include engineered reporter cell lines, bioactive recombinant human TSLP and TL1A proteins, and biosimilar neutralizing monoclonal antibodies (mAbs). The reliable and consistent performance of these biological tools makes them ideal for release assays of novel activatory and inhibitory molecules.

## TSLP cellular assay NEW

- **HEK-Blue™ TSLP reporter cells**
- **Recombinant human TSLP**
- **Anti-hTSLP-hIgG2 (Tezepelumab)**

### STAT5 reporter cells for TSLP signaling

These cells express the TSLPR and IL-7Rα subunits of the human TSLP receptor, along with human STAT5b and a STAT5-inducible secreted embryonic alkaline phosphatase (SEAP) reporter. STAT5 activation is easily quantified by measuring SEAP activity in the culture supernatant using the QUANTI-Blue™ detection reagent. These cells respond to human TSLP (not murine) *(Fig. 1A)* and are ideal for release assays of activatory or inhibitory molecules, such as Tezepelumab, an anti-TSLP neutralizing mAb *(Fig. 1B)*.

### Biologically active human TSLP cytokine

This high-quality and biologically active cytokine is produced in mammalian cells to ensure protein glycosylation and *bona fide* 3D structure. This guarantees functionality in assays requiring physiologically relevant TSLP interactions.

### Tezepelumab biosimilar neutralizing anti-TSLP mAb

This biosimilar antibody of the FDA approved Tezepelumab can be used for screening and neutralization assays to block TSLP signaling.

## TL1A cellular assay NEW

- **HEK-Blue™ TL1A reporter cells**
- **Recombinant human TL1A**
- **Anti-hTL1A-hIgG1 (Tulisokibart)**

### AP-1/NF-κB reporter cells for TL1A signaling

These cells express the human TL1A receptor, DR3, along with an AP-1/NF-κB-inducible secreted embryonic alkaline phosphatase (SEAP) reporter. Activation of AP-1 and NF-κB is easily quantified by measuring SEAP activity in the culture supernatant using the QUANTI-Blue™ detection reagent. These cells respond to human and mouse TL1A *(Fig. 1C)* and are ideal for release assays of activatory or inhibitory molecules, such as Tulisokibart, an anti-TL1A neutralizing mAb *(Fig. 1D)*.

### Biologically active human TL1A cytokine

This high-quality and biologically active cytokine is produced in mammalian cells to ensure protein glycosylation and *bona fide* 3D trimeric structure. This guarantees functionality in assays requiring physiologically relevant TL1A interactions.

### Tulisokibart biosimilar neutralizing anti-TL1A mAb

This biosimilar antibody of the FDA approved Tulisokibart can be used for screening and neutralization assays to block TSLP signaling.

**CYTOKINE OFFER**
For each cytokine reporter cell line purchased, get a free vial of the matching cytokine.

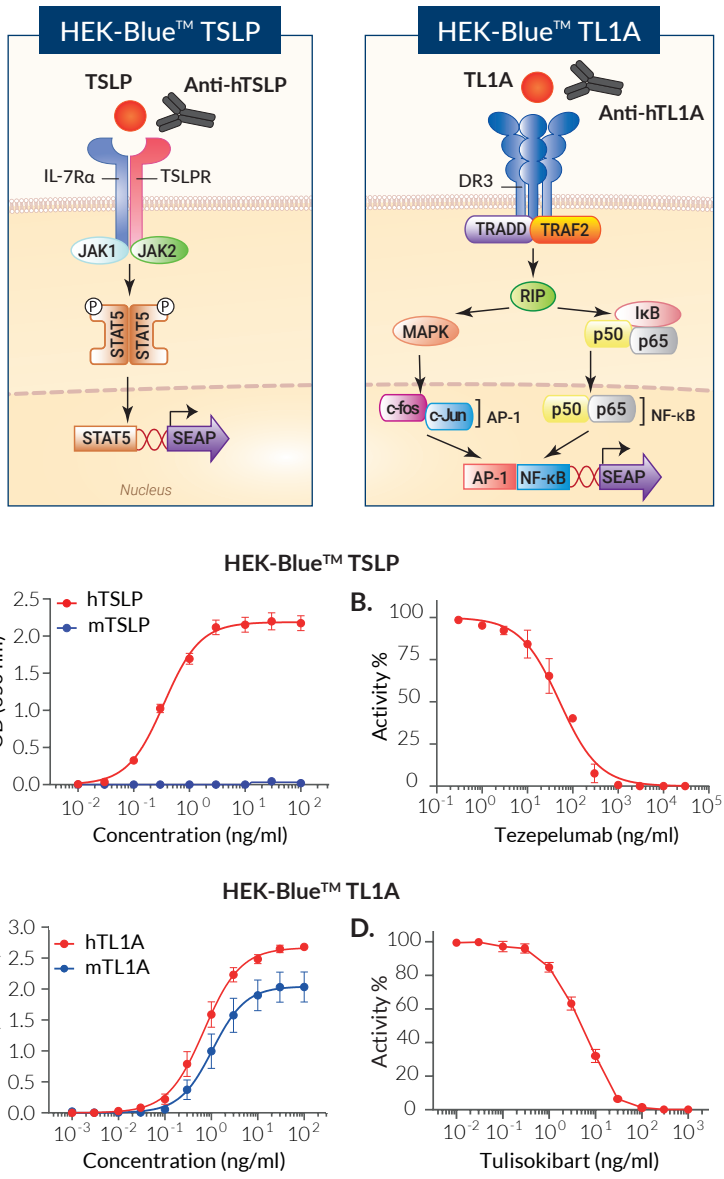
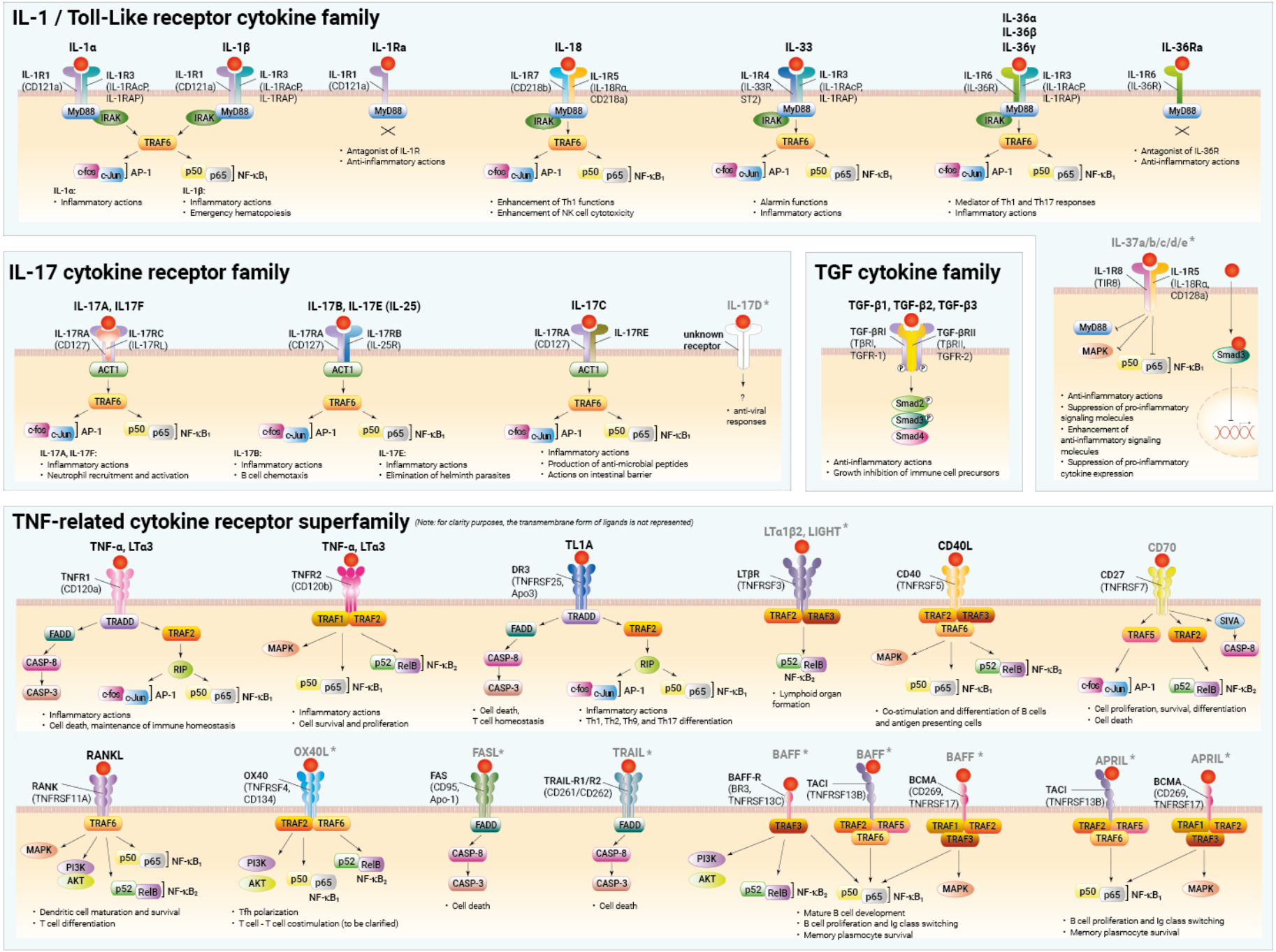



**Figure 1. Cellular responses to TSLP and TL1A using HEK-Blue™ TSLP (A, B) and HEK-Blue™ TL1A (C, D).** In one set of experiemnts, the cells were incubated with increasing concentrations of human (h) and mouse (m) TSLP or TL1A **(A and C)**. In the second set of experiments, increasing concentrations of Tezepelumab **(B)** or Tulisokibart **(D)** were incubated with recombinant hTSLP (1 ng/ml) or hTL1A (3 ng/ml) for 1 h before the addition the specific reporter cells. After overnight incubation, SEAP activity was measured in the culture supernatant. Data are shown as optical density (OD) at 650 nm **(A, B)** or in percentage of activity **(C, D)** (mean + SEM).

| PRODUCTS | QTY | CAT. CODE |
|---|---|---|
| HEK-Blue™ TSLP Cells | 3 - 7 x $10^6$ | hkb-tslp |
| HEK-Blue™ TL1A Cells | 3 - 7 x $10^6$ | hkb-tl1a |
| Recombinant human TSLP | 10 µg | rcyc-htslp |
| Recombinant human TL1A | 20 µg | rcyc-htl1a |
| Anti-hTSLP-hIgG2 (Tezepelumab) | 100 µg | htslp-mab2 |
| Anti-hTL1A-hIgG1 (Tulisokibart) | 100 µg | htl1a-mab1 |

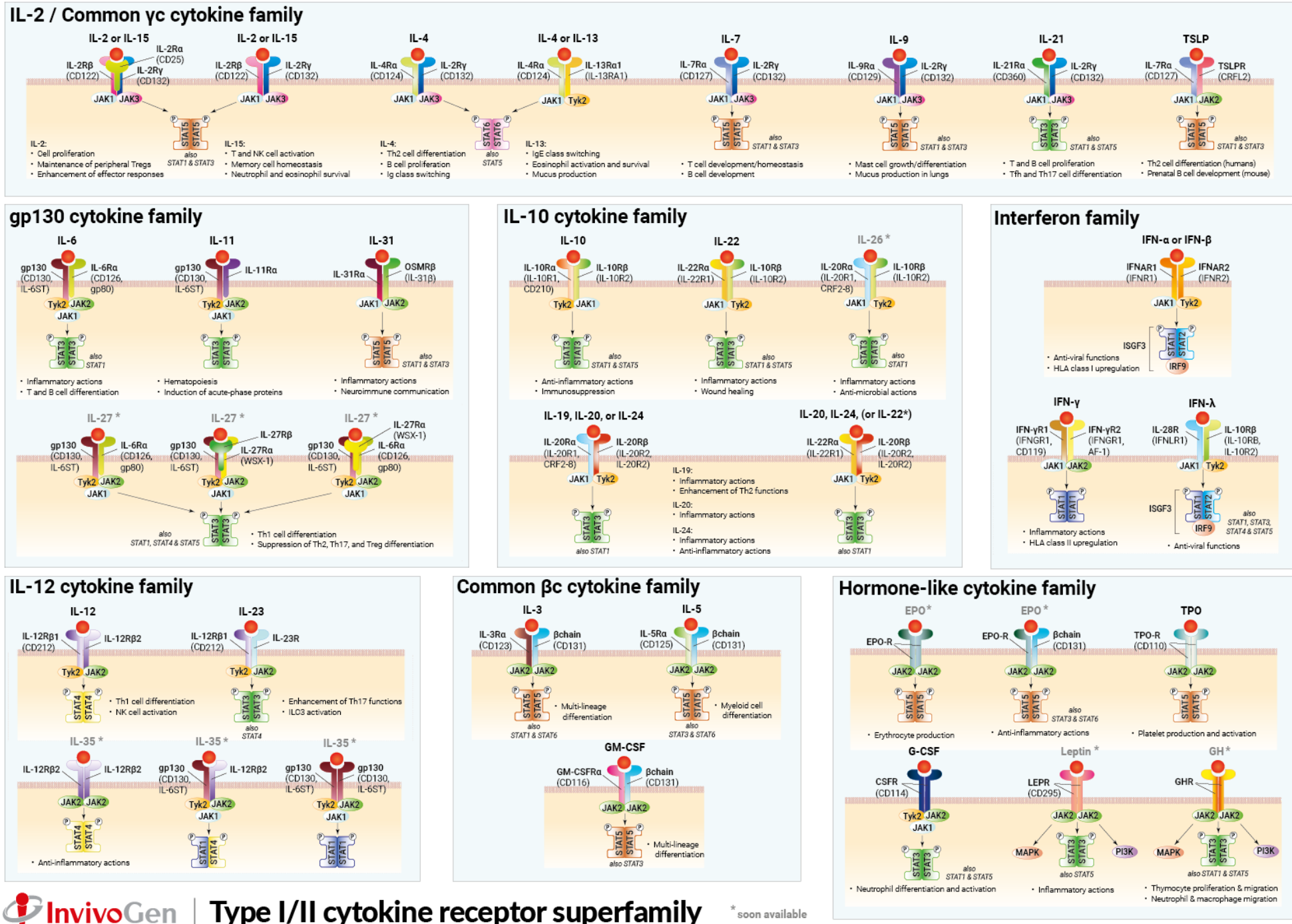



InvivoGen | **IL-1, IL-17, TGF, and TNF-related cytokine receptor families**

* Not yet available

InvivoGen cytokine reporter cell lines

Download our posters here:
www.invivogen.com/resources/


IL-2 / Common γc cytokine family
IL-2 or IL-15
IL-2Rα (CD25)
IL-2Rβ (CD122)
IL-2Rγ (CD132)
JAK1 JAK3
STAT5 STAT5
also STAT1 & STAT3
IL-2:
• Cell proliferation
• Maintenance of peripheral Tregs
• Enhancement of effector responses
IL-2 or IL-15
IL-15:
• T and NK cell activation
• Memory cell homeostasis
• Neutrophil and eosinophil survival
IL-4
IL-4Rα (CD124)
IL-2Rγ (CD132)
JAK1 JAK3
STAT6 STAT6
also STAT5
IL-4:
• Th2 cell differentiation
• B cell proliferation
• Ig class switching
IL-4 or IL-13
IL-4Rα (CD124)
IL-13Rα1 (IL-13RA1)
JAK1 Tyk2
IL-13:
• IgE class switching
• Eosinophil activation and survival
• Mucus production
IL-7
IL-7Rα (CD127)
IL-2Rγ (CD132)
JAK1 JAK3
STAT5 STAT5
also STAT1 & STAT3
• T cell development/homeostasis
• B cell development
IL-9
IL-9Rα (CD129)
IL-2Rγ (CD132)
JAK1 JAK3
STAT5 STAT5
also STAT1 & STAT3
• Mast cell growth/differentiation
• Mucus production in lungs
IL-21
IL-21Rα (CD360)
IL-2Rγ (CD132)
JAK1 JAK3
STAT3 STAT3
also STAT1 & STAT5
• T and B cell proliferation
• Tfh and Th17 cell differentiation
TSLP
IL-7Rα (CD127)
TSLPR (CRFL2)
JAK1 JAK2
STAT5 STAT5
also STAT1 & STAT3
• Th2 cell differentiation (humans)
• Prenatal B cell development (mouse)
gp130 cytokine family
IL-6
gp130 (CD130, IL-6ST)
IL-6Rα (CD126, gp80)
Tyk2 JAK2 JAK1
STAT3 STAT3
also STAT1
• Inflammatory actions
• T and B cell differentiation
IL-11
gp130 (CD130, IL-6ST)
IL-11Rα
Tyk2 JAK2 JAK1
STAT3 STAT3
• Hematopoiesis
• Induction of acute-phase proteins
IL-31
IL-31Rα
OSMRβ (IL-31β)
JAK1 JAK2
STAT5 STAT5
also STAT1 & STAT3
• Inflammatory actions
• Neuroimmune communication
IL-27 *
gp130 (CD130, IL-6ST)
IL-6Rα (CD126, gp80)
IL-27Rα (WSX-1)
Tyk2 JAK2 JAK1
STAT3 STAT3
also STAT1, STAT4 & STAT5
• Th1 cell differentiation
• Suppression of Th2, Th17, and Treg differentiation
IL-10 cytokine family
IL-10
IL-10Rα (IL-10R1, CD210)
IL-10Rβ (IL-10R2)
Tyk2 JAK1
STAT3 STAT3
also STAT1 & STAT5
• Anti-inflammatory actions
• Immunosuppression
IL-22
IL-22Rα (IL-22R1)
IL-10Rβ (IL-10R2)
JAK1 Tyk2
STAT3 STAT3
also STAT1 & STAT5
• Inflammatory actions
• Wound healing
IL-26 *
IL-20Rα (IL-20R1, CRF2-8)
IL-10Rβ (IL-10R2)
JAK1 Tyk2
STAT3 STAT3
also STAT1
• Inflammatory actions
• Anti-microbial actions
IL-19, IL-20, or IL-24
IL-20Rα (IL-20R1, CRF2-8)
IL-20Rβ (IL-20R2, IL-20R2)
JAK1 Tyk2
STAT3 STAT3
also STAT1
IL-19:
• Inflammatory actions
• Enhancement of Th2 functions
IL-20:
• Inflammatory actions
IL-24:
• Inflammatory actions
• Anti-inflammatory actions
IL-20, IL-24, (or IL-22*)
IL-22Rα (IL-22R1)
IL-20Rβ (IL-20R2, IL-20R2)
JAK1 Tyk2
STAT3 STAT3
also STAT1
Interferon family
IFN-α or IFN-β
IFNAR1 (IFNR1)
IFNAR2 (IFNR2)
JAK1 Tyk2
ISGF3
STAT1 STAT2
IRF9
• Anti-viral functions
• HLA class I upregulation
IFN-γ
IFN-γR1 (IFNGR1, CD119)
IFN-γR2 (IFNGR1, AF-1)
JAK1 JAK2
STAT1 STAT1
• Inflammatory actions
• HLA class II upregulation
IFN-λ
IL-28R (IFNLR1)
IL-10Rβ (IL-10RB, IL-10R2)
JAK1 Tyk2
ISGF3
STAT1 STAT2
IRF9
also STAT1, STAT3, STAT4 & STAT5
• Anti-viral functions
IL-12 cytokine family
IL-12
IL-12Rβ1 (CD212)
IL-12Rβ2
Tyk2 JAK2
STAT4 STAT4
• Th1 cell differentiation
• NK cell activation
IL-23
IL-12Rβ1 (CD212)
IL-23R
Tyk2 JAK2
STAT3 STAT3
also STAT4
• Enhancement of Th17 functions
• ILC3 activation
IL-35 *
IL-12Rβ2
IL-12Rβ2
JAK2 JAK2
STAT4 STAT4
• Anti-inflammatory actions
IL-35 *
IL-12Rβ2
gp130 (CD130, IL-6ST)
Tyk2 JAK2 JAK1
STAT1 STAT4
IL-35 *
gp130 (CD130, IL-6ST)
gp130 (CD130, IL-6ST)
Tyk2 JAK2 JAK1
STAT1 STAT1
Common βc cytokine family
IL-3
IL-3Rα (CD123)
βchain (CD131)
JAK2 JAK2
STAT5 STAT5
also STAT1 & STAT6
• Multi-lineage differentiation
IL-5
IL-5Rα (CD125)
βchain (CD131)
JAK2 JAK2
STAT5 STAT5
also STAT3 & STAT6
• Myeloid cell differentiation
GM-CSF
GM-CSFRα (CD116)
βchain (CD131)
JAK2 JAK2
STAT5 STAT5
also STAT3
• Multi-lineage differentiation
Hormone-like cytokine family
EPO *
EPO-R
JAK2 JAK2
STAT5 STAT5
• Erythrocyte production
EPO *
EPO-R
βchain (CD131)
JAK2 JAK2
STAT5 STAT5
also STAT3 & STAT6
• Anti-inflammatory actions
TPO
TPO-R (CD110)
JAK2 JAK2
STAT5 STAT5
• Platelet production and activation
G-CSF
CSFR (CD114)
Tyk2 JAK2 JAK1
STAT3 STAT3
also STAT1 & STAT5
• Neutrophil differentiation and activation
Leptin *
LEPR (CD295)
JAK2 JAK2
STAT3 STAT3
MAPK
PI3K
also STAT5
• Inflammatory actions
GH *
GHR
JAK2 JAK2
STAT3 STAT3
MAPK
PI3K
also STAT1 & STAT5
• Thymocyte proliferation & migration
• Neutrophil & macrophage migration
InvivoGen
Type I/II cytokine receptor superfamily
* soon available

# Bispecific T cell engager for efficient T lymphocyte activation and expansion

Bispecific antibodies bring together specificities of two antibodies that are able to bind simultaneously to two separate unique antigens or epitopes. The most widely used application for this tool is in the study and development of cancer immunotherapies that directly target cancer cells.

- **bsAb CD3-CD28** **NEW**

## Key Features

- Simple, versatile, and cost-effective
- Increased solubility and serum half-life
- No need of expensive material or extra steps

## Applications

- Expansion of enriched or PBMC-derived T cells
- Cancer immunotherapy studies
- CAR T cell development

InvivoGen's bsAb CD3-CD28 is a T cell activating and expanding CD3-CD28 bispecific antibody (bsAb), targeting the human CD3 and CD28 receptors. It is a fusion protein dimer comprising tandem single-chain variable fragments (scFv)2 and an IgG1 Fc fragment.
This bispecific T cell engager mimics the natural cross-linking of CD3 and CD28 by APCs and delivers the necessary signal 1 and signal 2 for T cell for activation *(Fig. 1 & Fig. 2)* and expansion *(Fig. 3)*.

For your convenience and to facilitate the selective expansion of T cell subsets, InvivoGen also offers high quality recombinant human cytokines, including IL-2, IL-7 and IL-15 (www.invivogen.com/recombinant-cytokines).

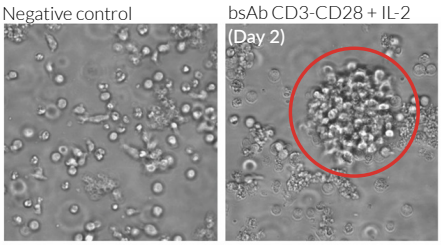



**Figure 1. Morphology of activated human T cells.** Morphology of unstimulated (left) and activated human T cells (right) after 48 h of stimulation with bsAb CD3-CD28 and recombinant human IL-2.

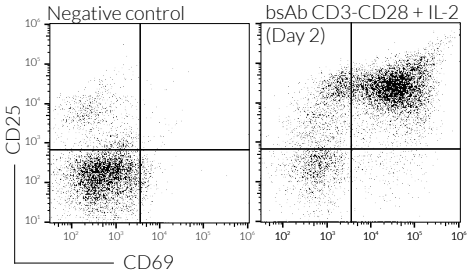
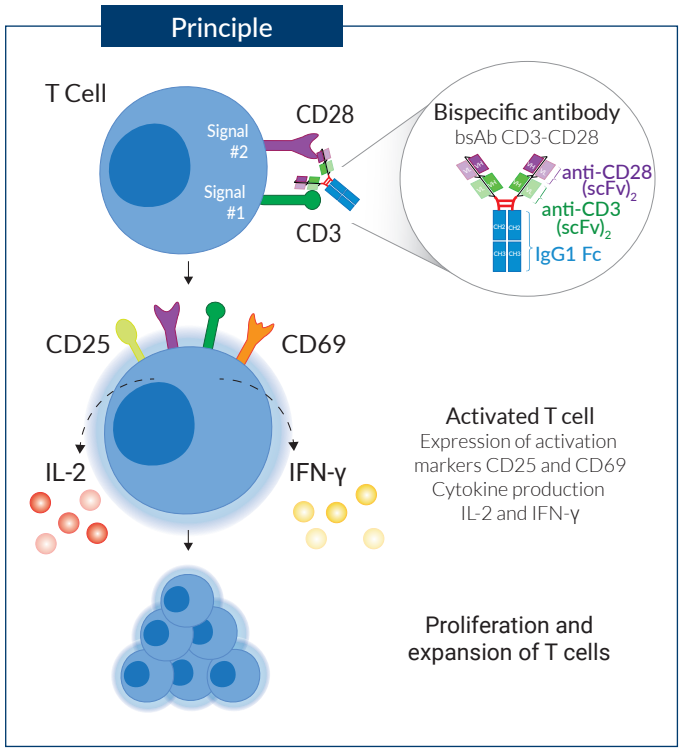



**Figure 2. CD25 & CD69 expression after T cell activation.** Isolated human T cells were stimulated for 48 h using bsAb CD3-CD28 and recombinant human IL-2. Unactivated T cells were used as negative control. Upregulation of CD69 and CD25 surface expression was assessed by flow cytometry.



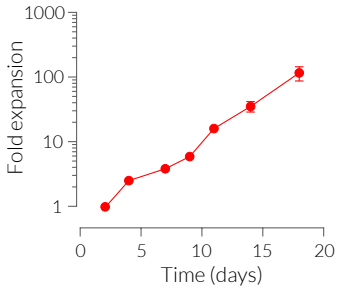




**Figure 3. Human T cell expansion.** Isolated human T cells were expanded over 25 days with bsAb CD3-CD28 in RPMI medium supplemented with recombinant human IL-2. Viable cells were counted on days 2, 4, 7, 9, 11, 14, and 18 (mean ± SEM in 8 experiments with 2 donors).

| PRODUCTS | QTY | CAT. CODE |
|---|---|---|
| bsAb CD3-CD28 | 100 µg | bsab-tex-1 |
| Recombinant human IL-2 | 10 µg | rcyc-hil2-01 |
| Recombinant human IL-7 | 10 µg | rcyc-hil7 |
| Recombinant human IL-15 | 20 µg | rcyc-hil15 |

Learn more
www.invivogen.com/anti-cd3-cd28-t-cell-expansion

InvivoGen

Europe Tel: +33 562 71 69 39 Fax: +33 562 71 69 30 info.eu@invivogen.com
USA Tel: +1 888 457 5873 Fax: +1 858 457 5843 info@invivogen.com
Asia Tel: +852 3622 3480 Fax: +852 3622 3483 info.hk@invivogen.com