## **Product Data Sheet**

## PE anti-mouse CD134 (OX-40)

Catalog # / Size:	1197045 / 50 μg 1197050 / 200 μg
Clone:	OX-86
Isotype:	Rat IgG1, к
Immunogen:	Recombinant mouse OX-40-CD4 chimeric protein
<b>Reactivity:</b>	Mouse
Preparation:	The antibody was purified by affinity chromatography, and conjugated with PE under optimal conditions. The solution is free of unconjugated PE and unconjugated antibody.
Formulation:	Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide.
<b>Concentration:</b>	0.2

## **Applications:**

Applications:	Flow Cytometry
Recommended Usage:	Each lot of this antibody is quality control tested by immunofluorescent staining with flow cytometric analysis. For flow cytometric staining, the suggested use of this reagent is $\leq 0.25$ microg per $10^6$ cells in 100 microL volume. It is recommended that the reagent be titrated for optimal performance for each application.
Application Notes:	Clone OX-86 has been reported to act as an agonist and stimulate OX-40.
Application References:	1. Higgins LM, <i>et al.</i> 1999. <i>J. Immunol.</i> 162:486. (FC, IHC) 2. Al-Shamkhani A, <i>et al.</i> 1996. <i>Eur. J. Immunol.</i> 26:1695. (Costim) 3. del Rio ML, <i>et al.</i> 2011. <i>Transpl. Int.</i> 24:501. (FC) <u>PubMed</u>

Description: CD134 is a type I integral membrane protein also known as OX-40, ACT35, and tumor necrosis factor receptor superfamily member 4 (TNFRSF4). This receptor is expressed on activated CD4<sup>+</sup> and CD8<sup>+</sup> T cells and B cells. The OX-40 receptor binds to the OX-40 ligand (CD252) to provide a costimulatory signal that is independent of CD28. Blockade of OX40-OX40 ligand interactions has been shown to ameliorate experimental EAE and inflammatory bowel disease, which implies that these interactions are important in the pathogenesis of some autoimmune diseases.

Antigen	1. Al-Shamkhani A, <i>et al.</i> 1996. <i>Eur. J. Immunol.</i> 26:1695.
<b>References:</b>	2. Weinberg AD, <i>et al.</i> 1999. <i>J. Immunol.</i> 162:1818.
	3. Akira H, <i>et al.</i> 1999. <i>J. Immunol.</i> 162:7058.
	4. Pippig SD, <i>et al.</i>

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