Product Datasheet

ERBB2 Biosimilar Antibody (orb1238040)

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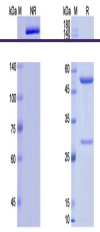
Description	ERBB2 Biosimilar Antibody
Conjugation	Unconjugated
Immunogen	Humanized / ERBB2 (HER2, Tyrosine kinase-type cell surface receptor HER2, MLN19, Metastatic lymph node gene 19 protein, ERBB2, Proto-oncogene Neu, p185erbB2, CD_antigen=CD340, Proto-oncogene c-ErbB-2, MLN 19, NEU, Receptor tyrosine-protein kinase erbB-2, NGL) [Homo sapiens]
Target	ERBB2
Preservatives	PBS buffer pH 7.5
Concentration	batch dependent
Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. Store at +4°C short term (1-2 weeks). Store at -20 °C 12 months. Store at -80°C long term.
Note	For research use only
Application notes	Treatment of HER2-overexpressing breast cancer cell lines with Trastuzumab results in induction of p27KIP1 and the Rb-related protein, p130, which in turn significantly reduces the number of cells undergoing S-phase. A number of other phenotypic changes are observed in vitro as a consequence of Trastuzumab binding to HER2-overexpressing cells. Interaction of Trastuzumab with the human immune system via its human immunoglobulin G1 Fc domain may potentiate its antitumor activities. in vitro studies demonstrate that Trastuzumab is very effective in mediating antibody-dependent cell-mediated cytotoxicity against HER2-overexpressing tumor targets[1]. Trastuzumab consists of two antigen-specific sites that bind to the juxtamembrane portion of the extracellular domain of the HER2 receptor and that prevent the activation of its intracellular tyrosine kinase. Trastuzumab recruits immune effector cells that are responsible for antibody-dependent cytotoxicity[2]. The presence of Trastuzumab IgG significantly increases killing of all breast cancer cell lines. The ADCC activity of PBMCs evoked by Trastuzumab is equally strong against Trastuzumab-sensitive (SKBR-3) or Trastuzumab-resistant (JIMT-1) breast cancer cells, with dose-dependent cell death reaching 50–60% killing at an effector/target ratio of 60:1[3].
Isotype	Human IgG1



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