Trastuzumab

**Cat. No.:** HY-P9907  
**Molecular Weight:** 145145.09  
**Target:** EGFR  
**Pathway:** JAK/STAT Signaling; Protein Tyrosine Kinase/RTK  
**Storage:** Please store the product under the recommended conditions in the COA.

### Biological Activity

**Description**  
Trastuzumab is a humanized monoclonal antibody for patients with invasive breast cancers that overexpress HER2. Trastuzumab has been clinically used to treat HER2 Positive Metastatic Breast Cancer and HER2 Positive Gastric Cancer.

<table>
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<th>IC₅₀ &amp; Target</th>
<th>HER2&lt;sup&gt;[1]&lt;/sup&gt;</th>
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**In Vitro**  
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<sup>[1]</sup>. 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<sup>[2]</sup>. 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<sup>[3]</sup>.

**In Vivo**  
Trastuzumab treatment of mouse xenograft models results in marked suppression of tumor growth. When given in combination with standard cytotoxic chemotherapeutic agents, Trastuzumab treatment generally results in statistically superior antitumor efficacy compared with either agent given alone<sup>[1]</sup>. Trastuzumab causes a significant growth inhibition of the outgrowth of macroscopic JIMT-1 xenograft tumors in both nude and SCID mice<sup>[3]</sup>.

### Protocol

**Cell Assay**<sup>[3]</sup>  
The effects of Trastuzumab and Trastuzumab-F(ab′)<sub>2</sub> on the growth of JIMT-1, SKBR-3, and BT-474 cells are evaluated using the AlamarBlue method. Exponentially growing cells are harvested and plated in single wells of a 96-well flat-bottomed tissue culture plate at defined densities, ranging from 4,500-8,000 cells per well depending on the cell line. After overnight culture, the regular medium is exchanged to medium containing 0, 1, 10, or 100 μg/mL Trastuzumab.
or Trastuzumab-F(ab′)². Cell viability is tested after 72 h of treatment. Fluorescence is detected at an excitation of 544 nm, and emission is detected at 590 nm\(^3\).

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

| Animal Administration \(^3\) | Trastuzumab and Trastuzumab-F(ab′)² are given at a dose of 5 and 25 \(\mu\)g/g, respectively, by weekly i.p. injection. The five times greater amount of administered F(ab′)² is chosen based on the different half-lives of IgG and F(ab′) F(ab′)². Control mice are treated with weekly i.p. injection of 100 \(\mu\)L physiologic saline (saline). Animals are euthanized by CO \(_2\) inhalation\(^3\). MCE has not independently confirmed the accuracy of these methods. They are for reference only. |

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- Patent. US20190151462A1

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