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# Anti-Human CD52 (Alemtuzumab Biosimilar)

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| **Catalog Number:** | 500501, 500502, 500503 |
| **Size:** | 1 mg, 5 mg, 20 mg |
| **Regulatory Status:** | RUO |

## PRODUCT DETAILS

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| **Clone:** | Alemtuzumab |
| **Application:** | Flow cytometry, animal model study |
| **Format:** | Liquid |
| **Product Description:** | Anti-Human CD52 (Alemtuzumab Biosimilar) |
| **Isotype:** | Human IgG1 |
| **Clonality:** | Recombinant |
| **Immunogen:** | Human CD52 |
| **Species specificity:** | Human |
| **Purity:** | >95% by reducing SDS-PAGE |
| **Grade:** | In vivo |
| **Storage Conditions:** | 4ºC |
| **Maximal Shelf Life:** | 12 months |
| **Synonyms:** | Campath-1 |

## BACKGROUND INFORMATION

Alemtuzumab is a recombinant humanized monoclonal antibody directed against the cell surface antigen CD52. Structurally, it belongs to the immunoglobulin G1 kappa (IgG1κ) subclass and is composed of two identical heavy chains and two identical light chains, forming a Y-shaped molecule with a molecular weight of approximately 150 kilodaltons (kDa). The antibody was developed by grafting complementarity-determining regions (CDRs) from a murine antibody (Campath-1) into a human IgG1 framework, which preserves antigen-binding specificity while reducing nonhuman immunogenic elements.

Each Fab region of Alemtuzumab is responsible for antigen recognition and binds with high affinity to CD52, a small glycoprotein anchored in the plasma membrane via a glycosylphosphatidylinositol (GPI) linkage. CD52 is widely expressed on the surface of mature lymphocytes, including B and T cells, as well as on monocytes and some granulocytes. The binding epitope for Alemtuzumab lies within the peptide and carbohydrate portions of the CD52 molecule, and the antibody interacts through its variable domains, primarily via hydrogen bonding and shape complementarity.

The Fc region of Alemtuzumab mediates immune effector functions after antigen binding through interactions with complement component C1q and Fc gamma receptors (FcγRs) on immune cells. These interactions can trigger immune mechanisms such as complement-dependent cytotoxicity (CDC) and antibody-dependent cellular cytotoxicity (ADCC), leading to targeted cell depletion in experimental models. Biophysically, Alemtuzumab displays a long half-life in circulation due to neonatal Fc receptor (FcRn)

recycling, contributing to sustained presence at target sites.