

Therapeutic/Humanized Antibodies ELISA Kits

'Humanized antibodies' are **'Animal Antibodies'** that have been modified by recombinant DNA-technology to reduce the overall content of the animal-portion of immunoglobulin so as to increase acceptance by humans or minimize 'rejection'. By analogy, if the hands of a mouse is actually responsible for grabbing things then **'humanized-mouse'** will only contain the **'mouse hands'** and the rest of the body will be human. Since the size of the IgGs is similar in mouse and human, the size of the native mouse IgG and the **'humanized IgG'** does not significantly change. Antigen recognition property of an antibody actually resides in small portion of the IgG molecule called the **'antigen binding site or Fab'**. Therefore, humanized antibodies contain the minimal portion from the Fab or the epitopes necessary for antigen binding. The process of **"humanization of antibodies"** is usually applied to animal (mouse) derived monoclonal antibodies for therapeutic use in humans (for example, antibodies developed as anti-cancer drugs). **Xolair (Anti-IgE) is an example of humanized IgG.** The portion of the mouse IgG that remains in the 'humanized IgG' may be recognized as foreign by humans and may result into the generation of "Human Anti-Drug Antibodies (HADA). The presence of anti-Drug antibody (e.g., Human Anti-Rituximab IgG) that may limit the long-term usage the humanized antibody (Rituximab). Not all monoclonal antibodies designed for human therapeutic use need be humanized since many therapies are short-term. The International Nonproprietary Names of humanized antibodies end in **"-zumab"**, as in **"omalizumab"**.



Humanized antibodies are distinct from **chimeric or fusion antibodies**. The latter also have their protein sequences made more similar to human antibodies, but carry a larger stretch of non-human IgG. An example of **chimeric antibody** is **Rituximab (Anti-CD20)**. The humanization processes is accomplished using recombinant DNA to create

constructs capable of expression in mammalian cell culture and harvested en masse. Not all methods for deriving antibodies intended for human therapy require a humanization step (e.g. phage display) but essentially all are dependent on techniques that similarly allow the "insertion" or "swapping-out" of portions of the antibody molecule. These systems rely on the creation of antibody gene "libraries" which can be wholly derived from human RNA isolated from peripheral blood. The immediate products of these systems are antibody fragments, normally Fab or scFv). This means that, although antibody fragments created using display methods are of fully human sequence, they are not **full antibodies**. Therefore, processes in essence identical to humanization are used to incorporate and express the derived affinities within a full antibody. **Adalimumab (Humira or Anti-TNF-alpha)** is an example of an antibody created through phage display.

ADI has developed specific ELISA kits to measure active therapeutic humanized antibodies in human and animals. Antibodies to therapeutics drugs (HADA/HAMA) after administration into humans can also be detected and measured using ELISA kits.

Humanized Anti-IgE (Xolair/Omalizumab) Assay Kits

Immunoglobulin E (IgE) is an immunoglobulin isotype that is found only in mammals. It is a monomeric antibody with 4 Ig-like domains. Its main function is immunity to parasites. IgE is commonly involved with allergies when present in high amounts in the body. It is especially associated with type I hypersensitivity. IgE is an important target for the treatments for allergy and asthma. IgE elicits an immune response by binding to Fc receptors found on the surface of mast cells and basophils. Fc receptors are of two types: FcεRI, the high-affinity IgE receptor and FcεRII, also known as CD23, is the low-affinity IgE receptor.



Omalizumab (Xolair, Genentech/Novartis) is a recombinant DNA-derived humanized IgG1k monoclonal antibody. Xolair is a glycosylated monoclonal antibody (~149 kda) produced in Chinese hamster ovary cells (CHO) suspension culture. Xolair inhibits the binding of IgE to the high-affinity IgE receptor (FcεRI) on the surface of mast cells and basophils. It is

approved for patients with moderate-to-severe or severe allergic asthma, which is caused by hypersensitivity reactions to certain harmless environmental substances. The drug is administered subcutaneously once every 2 or 4 weeks (150 to 375 mg). Like other protein and antibody drugs, omalizumab causes anaphylaxis (a life-threatening systemic allergic reaction) in 1 to 2 patients per 1,000. Antibodies to Xolair were detected in approximately 1/1723 (< 0.1%) of patients treated with Xolair. **Gomiliximab (IDEC Pharmaceuticals Corporation.)** is a monoclonal IgG1 antibody acting as an immunosuppressive drug for the treatment of allergic asthma. It targets the low affinity IgE receptor (FcεRII). The drug is a chimeric antibody from Macaca irus and Homo sapiens.

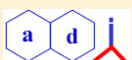
Omalizumab inhibits the binding of IgE to the high-affinity IgE receptor FcεRI by binding to an epitope on IgE that overlaps with the site to which FcεRI binds. This feature is critical to omalizumab's pharmacological effects because a typical anti-IgE antibody can cross-link cell surface FcεRI-bound IgE and induce mediator release from basophils and mast cells.

ADI has developed new ELISA kits that measure **total IgE** (free and Xolair-bound) and more importantly **"Free IgE (Xolair-unbound)"** in patients treated with Xolair. Highly sensitive ELISA kits were also developed to measure the concentration of Xolair in human or animal sera that measure **"Free Xolair or IgE-unbound or Active Xolair"**. ADI has also developed ELISA kits to detect antibodies to Xolair (**Human Anti-Xolair Antibodies**) in patients receiving long-term treatments. These kits will be useful to develop better immunotherapeutics. We are also working to develop assays that may predict patients who are likely to respond to Xolair therapy and who may be more prone to develop antibodies.

Product Details and Ordering Information

http://4adi.com/commerce/catalog/spcategory.jsp?category_id=2767

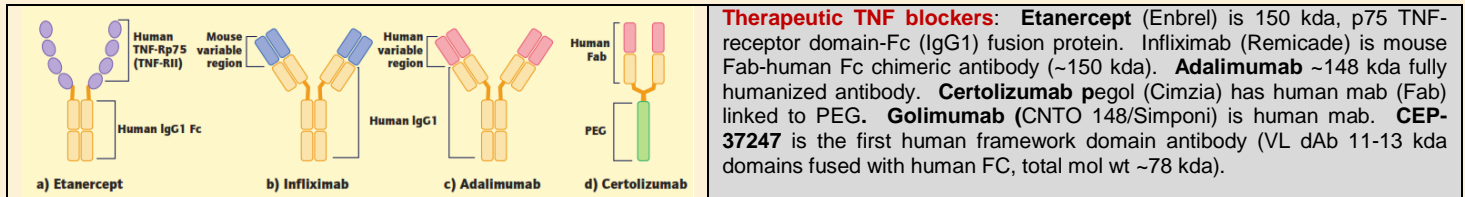
Catalog#	Product Description
200-410-XLG	Xolair/Omalizumab ELISA Kit for human
200-420-XLG	Human Anti-Xolair/Omalizumab Antibody ELISA
200-430-XET	Human IgE (Total; Free and Xolair-bound) ELISA Kit for Xolair-treated samples, 96 tests
200-440-XEF	Human IgE (Free; Xolair unbound) ELISA Kit for Xolair-treated samples, 96 tests
1800	Human serum IgE ELISA kit



Humanized Anti-TNF-alpha (Humira/Adalimumab) Assay Kits

Tumor necrosis factor (TNF, or TNF- α) is a cytokine involved in systemic inflammation. TNF was thought to be produced primarily by macrophages, but it is produced also by a broad variety of cell types including lymphoid cells, mast cells, endothelial cells, cardiac myocytes, adipose tissue, fibroblasts, and neuronal tissue. Large amounts of TNF are released in response to lipopolysaccharide, other bacterial products, and Interleukin-1 (IL-1). In the skin, mast cells appear to be the predominant source of pre-formed TNF, which can be released upon inflammatory stimulus (e.g., LPS). It is a 212-amino acid-long type II transmembrane protein arranged in stable homotrimers. From this membrane-integrated form the soluble homotrimeric cytokine (sTNF) is released via proteolytic cleavage by the metalloprotease **TNF alpha** converting enzyme (TACE, also called ADAM17). TNF can bind two receptors, TNF-R1 (TNF receptor type 1; CD120a; p55/60) and TNF-R2 (TNF receptor type 2; CD120b; p75/80). TNF-R1 is expressed in most tissues, and can be fully activated by both the membrane-bound and soluble trimeric forms of TNF, whereas TNF-R2 is found only in cells of the immune system, and respond to the membrane-bound form of the TNF homotrimer.

The primary role of TNF is in the regulation of immune cells. TNF, being an endogenous pyrogen, is able to induce fever, to induce apoptotic cell death, to induce sepsis (through IL1 & IL6 production), to induce cachexia, induce inflammation, and to inhibit tumorigenesis and viral replication. TNF promotes inflammatory response, which in turn causes many of the clinical problems associated with autoimmune disorders such as rheumatoid arthritis, spondylitis, Crohn's disease, psoriasis, hidradenitis suppurativa and refractory asthma. These disorders are treated by using a **TNF inhibitor**. Inhibition can be achieved with a monoclonal antibody such as infliximab (Remicade), adalimumab (Humira) or certolizumab pegol (Cimzia).



Adalimumab was the first fully human monoclonal antibody drug approved by the FDA. It was derived from phage display. Humira is a recombinant human IgG1k monoclonal antibody with human derived heavy and light chain variable regions and human IgG1:k constant regions. It consists of 1330 amino acids (~148 Kda). Humira binds to a single epitope on the N-terminus of the TNF-alpha and blocks its interaction with the p55 and p75 cell surface TNF-receptors. Adalimumab also lyses surface TNF expressing cells in vitro in the presence of complement. Adalimumab does not bind or inactivate lymphotoxin (TNF-beta). Treatment with HUMIRA may result in the formation of autoantibodies and, rarely, in the development of a lupus-like syndrome. In the rheumatoid arthritis controlled trials, 12% of patients treated with

HUMIRA and 7% of placebo-treated patients that had negative baseline ANA titers developed positive titers at week 24. Approximately 5% (58 of 1062) of adult rheumatoid arthritis patients receiving HUMIRA developed low-titer antibodies to adalimumab at least once during treatment, which were neutralizing in vitro.

Adalimumab/Humira was constructed from a fully human monoclonal antibody, while infliximab is a mouse-human chimeric antibody and etanercept is a TNF receptor-IgG fusion protein. Adalimumab has been approved by the FDA for the treatment of rheumatoid arthritis, psoriatic arthritis, ankylosing spondylitis, Crohn's disease, moderate to severe chronic psoriasis and juvenile idiopathic arthritis.

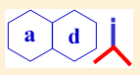
ADI has developed new ELISA kits that measure **TNF-alpha, Free Humira** (TNF-unbound)* in patients treated with Humira. ADI has also developed ELISA kits to detect antibodies to Humira (**Human Anti-Humira Antibodies**) in patients receiving long-term treatments. These kits will allow to research better ways to monitor humira treatment. Additional ELISA kits are available to monitor the increase other autoimmune diseases (ANA, anti-dsDNA IgGs, tuberculosis).

Catalog#	Product Description	Product Type
100-210-TNF	Mouse TNF-alpha ELISA Kit, 96 tests, Quantitative	Kit
100-215-TNH	Human TNF-alpha ELISA Kit, 96 tests, Quantitative	Kit
200-300-ADG	Humira/Adalimumab (Human Anti-TNF-alpha) ELISA Kit for dog, 96 tests	Kit
200-310-AHG	Humira/Adalimumab (Human Anti-TNF-alpha) ELISA Kit for human, 96 tests	Kit
200-320-ADG	Human Anti-Humira/Adalimumab (Human Anti-TNF-alpha) IgG ELISA Kit, 96 tests	Kit
200-320-AHG	Human Anti-Humira/Adalimumab (Human Anti-TNF-alpha) ELISA Kit for human, 96 tests	Kit
200-330-TNF	Human TNF-alpha ELISA Kit, 96 tests	Kit

Related Antibodies and Proteins Ordering Information (http://4adi.com/commerce/catalog/spcategory.jsp?category_id=2768)

MA-20161	Mouse Monoclonal Anti-Human TNF-alpha	Antibodies
SP-101331-5	Tumor necrosis factor alpha (TNF- α) (71-82),	Pure Peptide
SP-55385-1	P55-TNFR	Pure Peptide
SP-55386-1	P75-TNFR	Pure Peptide
SP-86636-1	Tumor necrosis factor alpha TNF- α (31 - 45)	Pure Peptide
SP-89728-1	Tumor necrosis factor alpha TNF- α (46-65)	Pure Peptide
SP-89729-5	[Ile76]-TNF-a (70-80) (human)	Pure Peptide
SP-89730-1	Tumor necrosis factor alpha TNF- α (78-96)	Pure Peptide
TACE11-A	Anti-Rat TACE	Antibodies

TNFA11-M	Monoclonal Anti-Human TNF-alpha IgG	Antibodies
TNFA15-R-10	Recombinant purified human Tumor Necrosis Factor-Alpha (TNF-alpha), biologically active	Rec. Protein
TNFA25-R-5	Recombinant purified rat Tumor Necrosis Factor-Alpha (TNF-alpha), biologically active	Rec. Protein
TNFA35-R-5	Recombinant purified mouse Tumor Necrosis Factor-Alpha (TNF-alpha), biologically active	Rec. Protein
TNFA35-R-5	Recombinant purified porcine Tumor Necrosis Factor-Alpha (TNF-alpha), biologically active	Rec. Protein
TNFR15-R-10	Recombinant purified human TNF Receptor Inhibitor Fc chimera (TNF-R1/TNF-R1), active	Rec. Protein
TNFR25-R-20	Recombinant purified human TNF Receptor 2 (TNFR2/TNF-RII, Etanercept) protein	Rec. Protein



Humanized Anti-CD20/MS4A1 (Rituximab/Rituxan) Assay Kits

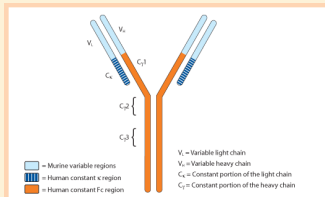


Figure 1.0 Mabthera combines murine anti-CD20 variable regions with a human constant region

CD20 is a 33-36-kDa (297-aa) transmembrane phosphoprotein involved in the activation, proliferation, and differentiation of B-lymphocytes. It is absent in terminally differentiated plasma cells. It is alternatively known as MS4A1, membrane spanning 4-family sub domain A member 1, B-lymphocyte surface antigen

B1, leu-16, Bp35, and CD-antige or CD20. Both N and C-termini located in the cytoplasm and 2 extra cellular regions: a large 44–amino acid loop (aa 142-184), which is the contact site of most anti-CD20 monoclonal antibodies (mAbs), including rituximab, and a small loop (aa 72-80), which is the contact site of human anti-CD20 mAbs. Rituximab also binds to amino acids 170-173 and 182-185 on CD20, which are physically close to each other as a result of a disulfide bond between amino acids 167 and 183. Significant levels of circulating CD20 (cCD20) can be detected in the plasma of CLL patients where they interfere with the binding of rituximab. The cCD20 levels correlated positively with beta-2-microglobulin level and percentage of CD38+ cells and negatively with platelet count and hemoglobin level. Circulating levels of Rituximab after a single dose vary among patients due to difference in tumor burden and clearance rates. Rituximab was detectable in the serum of patients three to six months after completion of treatment. Since Rituximab contains mouse antibody regions, it is more likely to induce antibodies than the humanized antibodies. According to the manufacture of Rituximab, less than 1% (3/355) of patients evaluated for human anti-chimeric antibody (HAMA/HACA) was positive. Availability of more sensitive ELISA for HAMA should be used to confirm the incidences of HAMA in rituximab-treated patients. HAMA/HACA titers may have allergic reactions when treated with this or other murine or chimeric monoclonal antibodies.



Rituximab destroys B cells, and is therefore used to treat diseases which are characterized by having too many B cells, overactive B cells or dysfunctional B cells. This includes many lymphomas, leukemias, transplant rejection and some autoimmune disorders. Rituximab has been shown to be an effective rheumatoid arthritis treatment. It is also used in

autoimmune diseases such as hemolytic anemia, pure red cell aplasia, idiopathic thrombocytopenic purpura (ITP), Evans syndrome, vasculitis (for example Wegener's Granulomatosis), bullous skin disorders (for example pemphigus, pemphigoid—with very encouraging results of approximately 85% rapid recovery, type 1 diabetes mellitus, Sjogren's syndrome, and Devic's disease, and Graves' disease ophthalmopathy.

Related Antibodies and Proteins Ordering Information (http://4adi.com/commerce/catalog/spcategory.jsp?category_id=2768)

Catalog#	Product Description
200-210-RAG	Rituximab/Rituxan (Active) ELISA Kit (Human/mouse/rat), 96 tests
200-245-HAM	Human Anti-Rituximab/Rituxan (HACA/HAMA/HAHA) IgG ELISA kit for human, 96 tests

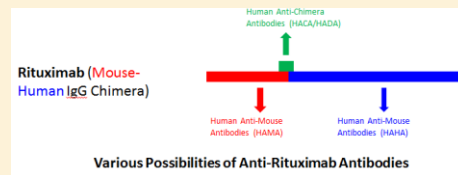
CD20-141-R
CD20-145-R
CD20-146-R
CD20-147-R
CD20-1731-P
CD20-1732-P
CD20-1733-P
CD20-21-M
CD20-22-A
CD20-RP5L-P
CD20-142-P
CD20-1731-P

Recombinant Human CD20/MS4A1-mFc fusion Protein (141-184, 44-aa, ECD, rituximab-binding region)
Recombinant (HEK cells) purified human CD20/MS4A1 (213-297 aa) his tag Protein
Recombinant (HEK cells) purified human CD20/MS4A1 (141-184 aa) his tag Protein
Recombinant (HEK cells) purified Ferret CD20/MS4A1 (213-297 aa) his tag Protein
Human CD20/MS4A1 peptide (Acetyl-cPYaNPSLc, 9-aa, Cyclic Cys1-Cys9); contains ANPS motif and reactivity with Rituximab
Human CD20/MS4A1 cyclic peptide (Acetyl-cWAANPSMac, 11 aa, Cys1-Cys11); contains the ANPS motif and avidity for rituximab
Human CD20/MS4A1 cyclic peptide (Acetyl-cPYsNPSLc; 9aa, Cys1-Cys9; contains NPS motif and react with rituximab
Monoclonal Anti-Human CD20/MS4A1 peptide (EC-domain, rituximab binding domain) IgG, ascites
Anti-Human CD20/MS4A1 peptide (EC-domain, rituximab binding domain) IgG, aff pure
CD20/MS4A1 linear peptide (QDKLTQWPWKLEg, 13-aa) contains WPXWLE motif and reacts with rituximab
Human CD20/MS4A1 linear peptide (142-184, 43-aa, extracellular domain) rituximab-binding peptide, >95% pure
Human CD20/MS4A1 peptide (Acetyl-cPYaNPSLc, 9-aa, Cyclic Cys1-Cys9); contains ANPS motif and reactivity with Rituximab

Rituxan (rituximab) is a genetically engineered chimeric murine/human monoclonal IgG1 kappa antibody (~145 kda). It is produced by mammalian cell (Chinese Hamster Ovary) suspension culture. The mouse/human chimeric CD20 mAb rituximab was the first cancer therapeutic mAb to be given Food and Drug Administration (FDA) approval and since then has become the most important new treatment for B cell malignancies in the last decade.

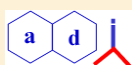
The efficacy and success of Rituximab has led to some other anti-CD20 monoclonal antibodies being developed: **Ocrelizumab** (90%-95% humanized), **Ofatumumab** (HuMax-CD20, fully humanized), **Ibritumomab tiuxetan/Zevalin** (mouse mab IgG1 in conjunction with the chelator tiuxetan) **Tositumomab/Bexxar** (mouse IgG2a is applied with iodine 131). Third-generation anti-CD20s have a glycol-engineered Fc fragment (Fc) with enhanced binding to Fc gamma receptors, which increase ADCC (antibody-dependent cellular cytotoxicity). Rituximab destroys both normal and malignant B cells that have CD20 on their surfaces. Therefore, it may be advisable to carefully monitor patients for circulating CD20, drug concentrations during the treatments, and the development of antibodies (HAMA/HADA). ADI has developed new ELISA kits that measure **circulating CD20**, **“Free Rituximab** (CD20-unbound)” in patients treated with Rituximab.

All antibodies, humanized or chimeric, have the potential to make antibodies even when injected into a homologous species (humanized antibody injected into human). Chimeric antibodies



(e.g. Rituximab, mouse-human IgG) are expected to make anti-mouse antibodies when injected into humans (**HAMA or human anti-mouse antibodies**). There is also potential to make antibodies to the human portion of the rituximab (**HAHA or human anti-human antibodies**) due to change in confirmation or structure of the chimeric IgG. There may even be antibodies that are directed against the mouse-human fusion regions or chimeric regions (**HACA or human anti-chimeric antibodies or Human Anti-Drug antibodies or HADA**). Therefore, test methods are needed to detect the production of HAMA, HAHA or HACA/HADA.

ADI has also developed ELISA kits to detect antibodies to Rituximab (**Human Anti-Rituximab Antibodies**) in patients receiving long-term treatments. These kits will allow to research better ways to monitor rituximab treatment. Additional ELISA kits are available to monitor the increase other autoimmune diseases (ANA, anti-dsDNA IgGs, tuberculosis). ADI also have variety of antibodies to human, mouse, and rat CD20, and CD-20 peptides involved in rituximab binding.



Humanized Anti-Her2/neu (Herceptin/ trastuzumab) Assay Kits

HER2 (Human Epidermal Growth Factor Receptor 2) also known as Neu, ErbB-2, CD340 (cluster of differentiation 340) or p185 is a protein that in humans is encoded by the ERBB2 gene. Neu is so named because it was derived from a rodent glioblastoma cell line, a type of neural tumor. HER2 is named because it has a similar structure to human epidermal growth factor receptor, or HER1. HER2 is a member of the epidermal growth factor receptor (EGFR/ErbB) family. Amplification or over-expression of this gene has been shown to play an important role in the pathogenesis and progression of certain aggressive types of breast cancer and in recent years it has evolved to become an important biomarker and target of therapy for the disease.

The ErbB family is composed of four plasma membrane-bound receptor tyrosine kinases. All four contain an extracellular ligand binding domain, a transmembrane domain, and an intracellular domain that can interact with a multitude of signaling molecules. Unlike the other family members, HER2 is considered to be an orphan receptor as it has no known ligand. HER2 can heterodimerise with any of the other three receptors. Dimerisation results in the autophosphorylation of tyrosine residues within the cytoplasmic domain of the receptors and initiates a variety of signaling pathways. Signaling through the ErbB family of receptors promotes cell proliferation and opposes apoptosis, and therefore must be tightly regulated to prevent uncontrolled cell growth from occurring.



Amplification or over-expression of the ERBB2 gene occurs in approximately 30% of breast cancers. It is strongly associated with increased disease recurrence and a worse prognosis. Over-expression is also known to occur in ovarian, stomach, and aggressive forms of uterine cancer, such as uterine serous endometrial carcinoma. The extracellular domain of HER2 can be shed from the surface of tumour cells and enter the circulation. HER2 testing is performed in breast cancer

patients to assess prognosis and to determine suitability for Herceptin therapy. It is important that Herceptin is restricted to HER2-positive individuals as it is expensive and has been associated with cardiac toxicity. Measurement of serum HER2 by ELISA offers a far less invasive method of determining HER2 status than a biopsy and consequently has been extensively investigated.

HER2 is the target of the monoclonal antibody trastuzumab (**Herceptin**). Trastuzumab is effective only in cancers where HER2 is over-expressed. Herceptin is fully humanized mab (IgG1 kappa) produced in CHO cell culture. It binds to the domain IV of the extracellular segment of the HER2/neu receptor. Trastuzumab is recommended in 3+Her2 (~2 million her2 per cell) positive breast tumors. Testing is performed on biopsy and immunohistochemistry. Trastuzumab has had a "major impact in the treatment of HER2-positive metastatic breast cancer". The combination of Trastuzumab with chemotherapy has been shown to increase both survival and response rate, in comparison to Trastuzumab alone. Herceptin costs about \$100,000 for a full treatment. Another monoclonal antibody, **Pertuzumab (2c4 or Omnitrag)**, which inhibits dimerization of HER2 and HER3 receptors, is in advanced clinical trials.

Other approaches such as DNA-based vaccines against Her2 using either the full length expression of her2 (pE2A) or extracellular domain or intracellular domains (pE2TM, pSec2, p185 or MVA-BN-Her2) are being used to produce antibodies to Her2. Similarly, Her 2 peptide-derived vaccine (E75, GP2, AE37 peptide or multiple peptides) are being tested to induce antibodies to Her2. Like many humanized antibodies, Herceptin can antibodies or human anti-human antibodies (**HABA**) (1 patient in 903). However, this is highly dependent upon the sensitivity of the assay.

ADI has developed new ELISA kits that measures "**Free Herceptin**" in patients treated with Herceptin. ADI has also developed ELISA kits to detect antibodies to Herceptin (**Human Anti- Herceptin Antibodies**) in patients receiving long-term treatments. These kits will allow to research better ways to monitor herceptin treatment. Additional ELISA kits are available to monitor the increase other autoimmune diseases (ANA, anti-dsDNA IgGs, tuberculosis). ADI also have variety of antibodies to human, mouse, and rat herceptin and Her2 peptides for vaccine studies.

Herceptin Related reagents and ELISAs Ordering Information (http://4adi.com/commerce/catalog/spcategory.jsp?category_id=2770)

Catalog#	Product Description	Product Type
200-510-HLG	Herceptin/Trasuzumab ELISA Kit for human, 96 tests	Kit
200-520-HAG	Human Anti-Herceptin/Trasuzumab Antibody (HABA) ELISA Kit 96 tests	Kit
200-530-HER	Her2/neu/ErbB2/CD340 protein ELISA kit, 96 tests	Kit
HER21-R-10	Recombinant (HEK) human Her2/ErbB2/Neu (1-652)- hlgG-Fc fusion protein	Rec. Protein
HER22-R-5	Recombinant (sf9) human Her2/ErbB2/Neu (676-1255)- GST fusion protein	Rec. Protein
HER23-R-10	Recombinant (HEK) human Her2/ErbB2/Neu (1-652)- his tag fusion protein	Rec. Protein
HER24-R-10	Recombinant (HEK) mouse Her2/ErbB2/Neu (1-653)-his tag fusion protein	Rec. Protein
HER25-R-10	Recombinant (HEK) mouse Her2/ErbB2/Neu (1-653)-hlgG1-Fc fusion protein	Rec. Protein
HER26-R-10	Recombinant (HEK) rat Her2/ErbB2/Neu (4-656)-his tag fusion protein	Rec. Protein
HER27-R-10	Recombinant (HEK) rat Her2/ErbB2/Neu (4-656)-his tag fusion protein	Rec. Protein
HER28-R-10	Recombinant (HEK) rat Her2/ErbB2/Neu (4-656)-hlgG1-Fc fusion protein	Rec. Protein
HER29-R-10	Recombinant (HEK) monkey/rhesus Her2/ErbB2/Neu (1-652)-his tag fusion protein	Rec. Protein
HER30-R-10	Recombinant (HEK) monkey/rhesus Her2/ErbB2/Neu (1-652)-hlgG1-Fc fusion protein	Rec. Protein
HER31-M	Rabbit mono anti-human Her2/ErbB2/Neu (1-652) protein IgG	Antibodies
HER32-A	Anti-human Her2/ErbB2/Neu (1-652) protein IgG	Antibodies
HER33-M	Mouse mono anti-monkey/rhesus Her2/ErbB2/Neu (1-652) protein IgG	Antibodies
HER34-A	Anti-monkey/rhesus Her2/ErbB2/Neu (1-652) protein IgG	Antibodies
HER2-369-P	HER2 peptide, (369 – 377), E 75 vaccine candidate	Peptide
HER2-597-P	HER2 peptide, cyclic, (597-626, disulphide bond) vaccine candidate	Peptide
HER2-654-P	HER2 peptide, (654 – 662), GP2 vaccine candidate	Peptide
HER2-776-P	HER2 peptide, (776 – 790 fused with LRMK), GP2 vaccine candidate	Peptide
HER2-563-P	HER2 peptide, cyclic, (563-598,); vaccine candidate	Peptide
HER2-585-P	HER2 peptide, cyclic, (585-598,); vaccine candidate	Peptide
HER2-613-P	HER2 peptide, cyclic, (613-626,); vaccine candidate	Peptide

ADI is also offering custom testing of animal or human samples for herceptin or antibodies to herceptin, and her2 measurements.

