**Anti – AGES**

<table>
<thead>
<tr>
<th>Cat No.</th>
<th>Size</th>
<th>Conjugation</th>
<th>Price</th>
<th>Application</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>KH001</td>
<td>10 μg/40 μl</td>
<td>–</td>
<td>£55,000</td>
<td>ELISA, IH, WB</td>
<td></td>
</tr>
<tr>
<td>KH001-01</td>
<td>10 μg/40 μl</td>
<td>Biotin</td>
<td>£70,000</td>
<td>ELISA, IH</td>
<td></td>
</tr>
<tr>
<td>KH001-02</td>
<td>20 μg/200 μl</td>
<td>Peroxidase</td>
<td>£70,000</td>
<td>ELISA, IH, WB</td>
<td>Replacement of KH002</td>
</tr>
<tr>
<td>KH002</td>
<td>20 μg/200 μl</td>
<td>Peroxidase</td>
<td>£70,000</td>
<td>ELISA, IH</td>
<td>Discontinued</td>
</tr>
<tr>
<td>KH001-A</td>
<td>1 mg/ml</td>
<td>–</td>
<td>£70,000</td>
<td>–</td>
<td>Antigen</td>
</tr>
</tbody>
</table>

**Host**: Mouse  
**Specificity**: Every animal species  
**Isotype**: IgG1  
**Clonality**: Monoclonal Antibody (H12)  
**Immunogen**: AGES-BSA  
**Purity**: ProteinG Affinity Purified  
**Cross Reactivity**: Not tested

Reaction of protein amino groups with glucose leads, through the early products such as a Schiff base and Amadori rearrangement products, to the formation of advanced glycation end products (AGEs). Recent immunological studies using anti-AGEs antibody (6D12) demonstrated the presence of AGEs-modified proteins in several human tissues: (i) human lens (nondiabetic and noncortactaneous), (ii) renal proximal tubules in patients with diabetic nephropathy and chronic renal failure, (iii) diabetic retina, (iv) peripheral nerves of diabetic neuropathy, (v) atherosclerotic lesions of arterial walls, (vi) β 2-microglobulin forming amyloid fibrils in patients with hemodialysis-related amyloidosis, (vii) senile plaques of patients with Alzheimer’s disease, (viii) the peritoneum of CAPD patients, (ix) skin elastin in actinic elastosis, and (x) ceroid/lipofuscin deposits. These results suggest a potential role of AGEs-modification in normal aging as well as age-enhanced disease processes. This antibody named as 6D12 has been used to demonstrate AGEs-modified proteins in these human tissues, indicating potential usefulness of this antibody for histochemical identification and biochemical quantification of AGEs-modified proteins.

**Anti – Pyrraline**

<table>
<thead>
<tr>
<th>Cat No.</th>
<th>Size</th>
<th>Conjugation</th>
<th>Price</th>
<th>Application</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>KH010</td>
<td>20 μg/80 μl</td>
<td>–</td>
<td>£55,000</td>
<td>ELISA, IH</td>
<td></td>
</tr>
<tr>
<td>KH010-01</td>
<td>20 μg/80 μl</td>
<td>Biotin</td>
<td>£70,000</td>
<td>ELISA, IH</td>
<td></td>
</tr>
<tr>
<td>KH010-02</td>
<td>20 μg/80 μl</td>
<td>Peroxidase</td>
<td>£70,000</td>
<td>ELISA, IH</td>
<td></td>
</tr>
</tbody>
</table>

**Host**: Mouse  
**Specificity**: Every animal species  
**Isotype**: IgG1  
**Clonality**: Monoclonal Antibody (H12)  
**Immunogen**: Pyrraline-HSA  
**Purity**: ProteinG Affinity Purified  
**Cross Reactivity**: Not tested

Pyrraline is one of the major Maillard compounds resulting from the reaction of glucose and amino compounds at slightly acidic pH. Using anti-pyrraline antibody, pyrraline was detected in sclerosed glomeruli from diabetic and normal old kidneys as well as in renal arteries with arteriosclerosis. Furthermore, it was detected in neurofibrillary tangles and senile plaques in brain tissue from patients with Alzheimer’s disease.

**Anti – CML**

<table>
<thead>
<tr>
<th>Cat No.</th>
<th>Size</th>
<th>Conjugation</th>
<th>Price</th>
<th>Application</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>KH011</td>
<td>50 μg/200 μl</td>
<td>–</td>
<td>£55,000</td>
<td>ELISA, IH</td>
<td></td>
</tr>
<tr>
<td>KH011-01</td>
<td>50 μg/200 μl</td>
<td>Biotin</td>
<td>£70,000</td>
<td>ELISA, IH</td>
<td></td>
</tr>
<tr>
<td>KH011-02</td>
<td>50 μg/200 μl</td>
<td>Peroxidase</td>
<td>£70,000</td>
<td>ELISA, IH</td>
<td></td>
</tr>
</tbody>
</table>

**Host**: Mouse  
**Specificity**: Every animal species  
**Isotype**: IgG1  
**Clonality**: Monoclonal Antibody (CMS-10)  
**Immunogen**: CML-KLH  
**Purity**: ProteinG Affinity Purified  
**Cross Reactivity**: Not tested

N-(carboxymethyl)lysine (CML) was a major AGEs structure identified by Banes et al. in 1989. Oxidative cleavage of Amadori products is considered as a major route to CML formation in vivo. Banes also revealed that CML was directly formed from the reaction between lipoxidative products and Lysine residue. Thus, CML could become a marker of oxidative stress and long term damage to protein in aging, atherosclerosis, and diabetes.

**Anti – CML**

<table>
<thead>
<tr>
<th>Cat No.</th>
<th>Size</th>
<th>Conjugation</th>
<th>Price</th>
<th>Application</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>KH024</td>
<td>50 μg/200 μl</td>
<td>–</td>
<td>£55,000</td>
<td>ELISA, IH</td>
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<tr>
<td>KH024-01</td>
<td>50 μg/200 μl</td>
<td>Biotin</td>
<td>£70,000</td>
<td>IH</td>
<td></td>
</tr>
<tr>
<td>KH024-02</td>
<td>50 μg/200 μl</td>
<td>Peroxidase</td>
<td>£70,000</td>
<td>IH</td>
<td></td>
</tr>
</tbody>
</table>

**Host**: Mouse  
**Specificity**: Every animal species  
**Isotype**: IgG2a  
**Clonality**: Monoclonal Antibody (NF-1G)  
**Immunogen**: CML-HSA  
**Purity**: ProteinG Affinity Purified  
**Cross Reactivity**: Not tested

N-(carboxymethyl)lysine (CML) is a major antigenic AGEs structure in vivo and is known to be generated from Oxidative cleavage of Amadori product. In addition to amadori product, CML formation also takes place through glyoxal, which is generated from the autoxidation of glucose and unsaturated fatty acids. NF-1G is monoclonal antibody specific for CML and useful for immunohistochemical staining to demonstrate the localization of CML in some pathological tissues.
**Anti – Pentosidine**

<table>
<thead>
<tr>
<th>Cat No.</th>
<th>Size</th>
<th>Conjugation</th>
<th>Price</th>
<th>Application</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>KH012</td>
<td>50 μg/200 μl</td>
<td>—</td>
<td>¥55,000</td>
<td>ELISA, IH</td>
<td></td>
</tr>
<tr>
<td>KH012-01</td>
<td>50 μg/200 μl</td>
<td>Biotin</td>
<td>¥70,000</td>
<td>ELISA, IH</td>
<td></td>
</tr>
<tr>
<td>KH012-02</td>
<td>50 μg/200 μl</td>
<td>Peroxidase</td>
<td>¥70,000</td>
<td>ELISA, IH</td>
<td></td>
</tr>
</tbody>
</table>

**Host**: Mouse  
**Specificity**: Every animal species  
**Isotype**: IgG1  
**Clonality**: Monoclonal Antibody (PEN-12)  
**Immunogen**: Pentosidine-HSA  
**Purity**: ProteinG Affinity Purified  
**Cross Reactivity**: Not tested

Pentosidine is one of the Maillard compounds identified by Monnier et al in 1989. It has been proved to cross-link Arginine to Lysine residue and be detected in β2-microglobulin from patients with hemodialysis-related amyloidosis.  
※ We can not export this products to U.S.A.

**Anti – CEL**

<table>
<thead>
<tr>
<th>Cat No.</th>
<th>Size</th>
<th>Conjugation</th>
<th>Price</th>
<th>Application</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>KH025</td>
<td>50 μg/200 μl</td>
<td>—</td>
<td>¥55,000</td>
<td>ELISA, IH</td>
<td></td>
</tr>
<tr>
<td>KH025-01</td>
<td>50 μg/200 μl</td>
<td>Biotin</td>
<td>¥70,000</td>
<td>ELISA, IH</td>
<td></td>
</tr>
<tr>
<td>KH025-02</td>
<td>50 μg/200 μl</td>
<td>Peroxidase</td>
<td>¥70,000</td>
<td>ELISA, IH</td>
<td></td>
</tr>
</tbody>
</table>

**Host**: Mouse  
**Specificity**: Every animal species  
**Isotype**: IgG1  
**Clonality**: Monoclonal Antibody (KNH-30)  
**Immunogen**: CEL-BSA  
**Purity**: ProteinG Affinity Purified  
**Cross Reactivity**: Not tested

CEL is known to generate from protein modification by methylglyoxal. Mclellan et al. demonstrated that plasma methylglyoxal, which is believed to be generate from Embden-Meyerhof and polyol pathways, concentrations in insulin-dependent diabetic patients were about 7-times higher than those of normal individuals. For examples, CEL was identified in human lens proteins at a concentration similar to that of CML and its accumulation increased with age like CML, indicating that CEL may play an important marker for aging and age-dependent disease such as diabetic complications.

**Anti – RAGE**

<table>
<thead>
<tr>
<th>Cat No.</th>
<th>Size</th>
<th>Conjugation</th>
<th>Price</th>
<th>Application</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>KH039</td>
<td>100 μg/400 μl</td>
<td>—</td>
<td>¥49,000</td>
<td>WB</td>
<td></td>
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</table>

**Host**: Rabbit  
**Specificity**: Human  
**Isotype**: Polyclonal Antibody  
**Clonality**: Partial peptide of human RAGE (C terminal intracellular domain)  
**Immunogen**: Partial peptide of human RAGE  
**Purity**: ProteinG Affinity Purified  
**Cross Reactivity**: Not tested

RAGE is the receptor of AGEs, advanced glycation end products with 35,000 molecular weight and was cloned from bovine lung in 1992 (David Stern et al.). RAGE has been found in several tissues such as monocytes, macrophages, endothelial cells, astrocytes. The ligand of RAGE is demonstrated not only AGEs but also anfoterin, EN-RAGE, N-carboxymethyllysine(CML), β-amylod and so on. The accumulation of AGEs-proteins in vivo has been demonstrated in several disease, it is not clear whether AGEs-proteins accumulated in vivo is a direct cause of the disease or rather reflects its effect. Regarding this issue, AGEs-modified proteins are known to interact with several cells by the AGES-receptors and induce several cellular phenomena. Recently, it has been discovered that RAGE is involved in pathophysiological function of diabetes and Alzheimer’s disease. This antibody is affinity purified rabbit polyclonal antibody raised against partial peptide of human RAGE and should be used for western blotting or immunohistochemistry.

Preparation of antibodies and instruction:  
Prof. S Horuchi, Department of Biochemistry Kumamoto University School of Medicine

**MOK: RAGE1: RAGE: renal tumor antigen**

**Anti – RAGE**

<table>
<thead>
<tr>
<th>Cat No.</th>
<th>Size</th>
<th>Conjugation</th>
<th>Price</th>
<th>Application</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>KG134</td>
<td>50 μg/200 μl</td>
<td>—</td>
<td>¥55,000</td>
<td>ELISA, FCM, IC, WB</td>
<td></td>
</tr>
</tbody>
</table>

**Host**: GANP mouse  
**Specificity**: Human  
**Isotype**: IgG1 κ  
**Clonality**: Monoclonal Antibody (1C5)  
**Immunogen**: Recombinant protein of human RAGE  
**Purity**: ProteinG Affinity Purified  
**Cross Reactivity**: Not tested

RAGE (receptor for AGEs, advanced glycation end products) is an around 35 kDa multiligand receptor classified as an immunoglobulin superfamily cell surface molecule. RAGE is found in endothelium, smooth muscle cells, cardiac myocytes, neural tissue, and mononuclear cells and two major truncated forms of RAGE have been also identified (N-terminally truncated, C-terminally truncated). RAGE acts as a coreceptor for not only AGEs, but also high-mobility group box1 (HMGB1), S100/calgranulins, and amyloid-β peptides. Intracellular signaling pathways induced by RAGE include the activation of Cdc42/Rac, MAP kinase, NF-κ B. The C-terminally truncated soluble form of RAGE can bind ligands including AGEs and antagonize RAGE signaling in vitro and in vivo. RAGE plays important role for inflammation, diabetic complications such as nephropathy, vascular injury and Alzheimer’s disease. Several clinical studies have demonstrated that the strong association of RAGE expression with malignant potential of various cancers. It has been showed that engagement of RAGE by HMGB1 plays an important role in regulating the tumor formation, growth, metastasis. It is also suggested that glyceraldehyde- and glycolaldehyde-derived AGEs may be significantly involved in the growth and invasion of melanoma through interactions with RAGE. This antibody is specific to RAGE and will be useful for research for cancer, chronic diseases associated with aging and diabetic complications.
Preparation of antibodies and instruction:

Prof. S Horie, Department of Biochemistry Kumamoto University School of Medicine

**Anti – 3-DG-imidazolone**

<table>
<thead>
<tr>
<th>Cat No.</th>
<th>Size</th>
<th>Conjugation</th>
<th>Price</th>
<th>Application</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>KH043</td>
<td>50 μg/200 μl</td>
<td>—</td>
<td>¥55,000</td>
<td>IH</td>
<td></td>
</tr>
</tbody>
</table>

**Host**: Mouse  
**Specificity**: Every animal species  
**Isotype**: IgG1  
**Clonality**: Monoclonal Antibody (JNH27)  
**Immunogen**: 3-DG-imidazolone-ASA  
**Purity**: ProteinG Affinity Purified  
**Cross Reactivity**: Not tested

It has been shown that Advanced Glycation End products (AGEs) have been involved in chronic disease with aging, such as diabetes or brain disease. So far, several AGES structure has been identified, and these studies shed light on the important role of the growth of the disease. Imidazolone is one of AGEs structure, and has been shown that there are two pathways to generate. One is through 3-deoxyglucosone (3-DG) and another is through methylglyoxal. But it is not clear which pathway is dominant in each chronic disease. This antibody is affinity purified rabbit polyclonal antibody raised against recombinant human galectin-3 and should be used for western blotting.

**Anti – Galectin**

<table>
<thead>
<tr>
<th>Cat No.</th>
<th>Size</th>
<th>Conjugation</th>
<th>Price</th>
<th>Application</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>KG114</td>
<td>100 μg/400 μl</td>
<td>—</td>
<td>¥39,000</td>
<td>WB</td>
<td></td>
</tr>
</tbody>
</table>

**Host**: Rabbit  
**Specificity**: Human  
**Isotype**: −  
**Clonality**: Monoclonal Antibody  
**Immunogen**: Partial peptide of human PPARγ (C terminal)  
**Purity**: Antigen Affinity Purified  
**Cross Reactivity**: Not tested

The galectins are a family of carbohydrate-binding proteins that are distributed widely in metazoan organisms. Many galectin family members are detected primarily intracellularly in most of the systems studied, although certain members can be found both inside and outside of cells. Galectin-3 interacts with beta-galactoside residues of several cell surface and matrix glycoproteins through the carbohydrate recognition domain and is also capable of peptide-peptide associations mediated by its N-terminus domain. Recently, it has been demonstrated that galectin-3 is a new member of AGEs-receptor complex. (Mol.Med.1:634-646,1995)

This antibody is very useful for analyzing the involvement of imidazolone in the chronic disease.

**Anti – PPARγ**

<table>
<thead>
<tr>
<th>Cat No.</th>
<th>Size</th>
<th>Conjugation</th>
<th>Price</th>
<th>Application</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>KG113</td>
<td>100 μg/400 μl</td>
<td>—</td>
<td>¥39,000</td>
<td>WB</td>
<td></td>
</tr>
</tbody>
</table>

**Host**: Rabbit  
**Specificity**: Human  
**Isotype**: −  
**Clonality**: Monoclonal Antibody  
**Immunogen**: Partial peptide of human PPARγ (C terminal)  
**Purity**: Antigen Affinity Purified  
**Cross Reactivity**: Not tested

PPARs (peroxisome proliferator-activated receptors) are a family of transcription factors belonging to the nuclear hormone receptor superfamily. Widely expressed in vertebrates, PPARs play critical roles in metabolism and differentiation of a number of cell types. The PPARγ subtype was originally identified as a factor binding to a fatty acid specific enhancer of the aP2 gene. PPAR-γ actions are mediated by three isoforms resulting from alternative promoter selection and alternative splicing. PPAR-γ 1 is widely expressed while PPAR-γ 2 expression is restricted to adipose tissue and PPAR-γ 3 expression is restricted to adipose tissue, macrophage, and colon. PPAR γ participates in adipose cell differentiation and energy storage. Recently, these roles of PPAR γ have focused attention on PPAR γ as a target of the anti-diabetic thiazolidinedione class of drugs.

**Anti – AdipoR1**

<table>
<thead>
<tr>
<th>Cat No.</th>
<th>Size</th>
<th>Conjugation</th>
<th>Price</th>
<th>Application</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>KG114</td>
<td>100 μg/400 μl</td>
<td>—</td>
<td>¥39,000</td>
<td>WB</td>
<td></td>
</tr>
</tbody>
</table>

**Host**: Rabbit  
**Specificity**: Human  
**Isotype**: −  
**Clonality**: Monoclonal Antibody  
**Immunogen**: Partial peptide of human AdipoR1 (N terminal)  
**Purity**: Antigen Affinity Purified  
**Cross Reactivity**: Not tested

Obesity is a common etiology of diabetes mellitus and other diseases. Certain adipocytokines are considered beneficial due to their ability to enhance insulin sensitivity, while others, considered detrimental, enhance insulin resistance. The beneficial adipocytokine adiponectin displays both anti-diabetic and anti-atherosclerotic effects. Two distinct adiponectin receptors have been identified. Both AdipoR1 and AdipoR2 are seven-pass transmembrane receptors but are structurally, topologically, and functionally distinct from G-protein coupled receptors (GPCR). AdipoR1 is most abundant in muscle whereas AdipoR2 is most abundant in liver. Both receptors promote fatty acid oxidation and glucose uptake by AMP-activated protein kinase and PPARα. PPAR agonists are reported to increase expression of activated adiponectin. PPARα agonists also increase expression of adiponectin receptors. Such findings have focused attention on the role of AdipoR1 in PPAR agonist development.

Preparation of antibodies and instruction:

Prof. S Horie, Department of Biochemistry Kumamoto University School of Medicine
Anti – β 3-AR

<table>
<thead>
<tr>
<th>Cat No.</th>
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<th>Conjugation</th>
<th>Price</th>
<th>Application</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>KG115</td>
<td>100 μg/400 μl</td>
<td>—</td>
<td>¥39,000</td>
<td>WB</td>
<td></td>
</tr>
</tbody>
</table>

Host: Rabbit  
Isotype: —  
Immunogen: Partial peptide of human β 3-AR (C terminal)  
Purity: Antigen Affinity Purified  
Cross Reactivity: Not tested

The neurotransmitter/hormone adrenaline (epinephrine, adrenalin) plays a central role in the mammalian stress response, increasing heart rate, raising blood pressure, and increasing blood glucose levels upon entering the blood stream. Adrenaline is secreted primarily by the adrenal medulla. Adrenaline activates both α-adrenergic receptors and β-adrenergic receptors. Three subtypes of beta adrenergic receptors are known, β1, β2, β3, expressed primarily in heart, respiratory tissue, and adipose tissue, respectively. β3-receptors are particularly abundant in brown adipocytes and play important roles in lipolysis and thermoregulation. Recently this receptor has received attention from researchers interested in type 2 diabetes mellitus and obesity. It is also being considered as a therapeutic target for heart failure.

Anti – AGE-3

<table>
<thead>
<tr>
<th>Cat No.</th>
<th>Size</th>
<th>Conjugation</th>
<th>Price</th>
<th>Application</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>KG122</td>
<td>10 μg/40 μl</td>
<td>—</td>
<td>¥55,000</td>
<td>ELISA, WB</td>
<td></td>
</tr>
</tbody>
</table>

Host: GANP mouse  
Isotype: IgG1  
Immunogen: AGE-3 (glycolaldehyde modified BSA)  
Purity: ProteinG Affinity Purified  
Cross Reactivity: Not tested

The products of the nonenzymatic glycation and oxidation of proteins, lipids and nucleic acids, the advanced glycation end-products (AGEs), accumulate in various pathological conditions, such as diabetes, inflammation, renal failure, and aging. AGEs accumulate at site of microvascular injury in diabetes, including the kidney, the retina, and within the vasculature. The enhanced formation of AGEs also exists in various disease, such as atherosclerosis, Alzheimer’s disease, end-stage renal disease (ESRD), rheumatoid arthritis and liver cirrhosis. AGEs can arise not only from glucose, but also from dicarbonyl compounds, short chain-reducing sugars and other metabolic pathways of glucose. Among AGEs, glycolaldehyde-derived AGEs (named AGE-3) have diverse toxic biological activities. AGE-3 significantly induces apoptotic cell death, DNA ladder formation and upregulates the secretory forms of VEGF mRNA levels in cultured bovine retinal pericytes. AGE-3 also decreases the viability and suppresses the replication rate in cultured rat Schwann cells, and attenuates cellular insulin sensitivity in 3T3-L1 cells. In human mesenchymal stem cells, AGE-3 increases the apoptotic cell and prevents cognate differentiation into adipose tissue, cartilage, and bone. This antibody is specific to AGE-3 and will be useful to research for chronic diseases associated with aging and diabetic complications.

Anti – AGE-1

<table>
<thead>
<tr>
<th>Cat No.</th>
<th>Size</th>
<th>Conjugation</th>
<th>Price</th>
<th>Application</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>KG132</td>
<td>10 μg/40 μl</td>
<td>—</td>
<td>¥55,000</td>
<td>ELISA, WB</td>
<td></td>
</tr>
</tbody>
</table>

Host: GANP mouse  
Isotype: IgG1, κ  
Immunogen: AGE-1-BSA  
Purity: ProteinG Affinity Purified  
Cross Reactivity: Not tested

It has been shown that glucose-derived AGEs (named AGE-1) causes apoptotic cell death and induces hyperfiltration and microalbuminuria by stimulating secretion of VEGF and MCP-1 proteins in the human mesangial cells. Therefore, AGE-1 may be involved in the pathogenesis of the early stage of diabetic nephropathy. This antibody is specific to AGE-1 and will be useful to research for diabetes, complications of diabetes.

Anti – AGE-4

<table>
<thead>
<tr>
<th>Cat No.</th>
<th>Size</th>
<th>Conjugation</th>
<th>Price</th>
<th>Application</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>KG133</td>
<td>10 μg/40 μl</td>
<td>—</td>
<td>¥55,000</td>
<td>ELISA, WB</td>
<td></td>
</tr>
</tbody>
</table>

Host: GANP mouse  
Isotype: IgG1, κ  
Immunogen: AGE-4-BSA  
Purity: ProteinG Affinity Purified  
Cross Reactivity: Not tested

Methylglyoxal (MG) increases in diabetes and can modify proteins rapidly and form AG. It has been showed that exogenously added MG has a strong synergistic effect on TNF-induced cell death and AGE-4 is formed during TNF-induced cell in death mouse L929 cell, and that increased MG and AGE-4 levels induce apoptosis in mycobacterial-infected macrophages. It also has been demonstrated that MG rapidly modifies the PT after being and stabilizes the PT in the closed conformation in rat liver mitochondria. Moreover, it has been showed that an increase in intracellular MG concentration inhibit the insulin signaling pathway and leads to an insulin-resistant state in L6 muscle cells. This antibody is specific to AGE-4 and will be useful to research for diabetes, chromic diseases associated with aging and diabetic complications, cell death.

http://www.transgenic.co.jp
### Glucose-derived AGEs ELISA Kit

<table>
<thead>
<tr>
<th>Cat No.</th>
<th>Size</th>
<th>Conjugation</th>
<th>Price</th>
<th>Application</th>
<th>Note</th>
</tr>
</thead>
<tbody>
<tr>
<td>KG452</td>
<td>1 kit</td>
<td>--</td>
<td>¥108,000</td>
<td>ELISA</td>
<td></td>
</tr>
</tbody>
</table>

**Components:**
- Antibody-coated microtiter plate: 1 plate
- Glucose-derived AGEs standard: 250 ul x 2
- Reference standard and sample diluent: 15 ml
- HRP- anti AGEs antibody concentrate: 60 ul
- OPD(o-phenylendiamin) tablets: 2 tablets
- Substrate solution: 30 ml
- Stop solution: 15 ml
- Wash buffer concentrate: 30 ml
- Dilution plate: 1 plate
Our International Distributor

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Technical Information

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