Adiponectin is a multimeric adipocyte-specific secretory protein that regulates energy metabolism, lipid metabolism, and insulin sensitivity with direct anti-diabetic, anti-atherosclerotic effects.

Intracellular signaling pathways induced by RAGE include the activation of Cdc42/Rac, focal adhesion kinase, MAP kinases, and NF-κB. Inhibitors of NF-κB and MEK/ERK are suggested to inhibit the growth and proliferation of tumor cells. RAGE expression is suggested to be associated with the development of various cancers, including breast cancer, prostate cancer, and colorectal cancer. It has been shown that downregulation of RAGE in malignant cells suppresses tumor growth. RAGE expression is suggested to be associated with the development of various cancers, including breast cancer, prostate cancer, and colorectal cancer. It has been shown that downregulation of RAGE in malignant cells suppresses tumor growth.

The products of the nonenzymatic glycation and oxidation of proteins, lipids and nucleic acids, the advanced glycation end (AGE) products, accumulate with age and in various disease conditions, such as diabetes, inflammation, cancer, and aging. AGEs accumulate at sites of microvascular injury or A549 cells, and show strong fluorescence, which is ideal for immunohistochemistry.

The advanced glycation end (AGE) products are typically denatured recombinant Adiponectin. AGEs have been shown to accumulate at sites of microvascular injury or A549 cells, and show strong fluorescence, which is ideal for immunohistochemistry.
AGEs / Metabolic Syndrome

Anti AGEs (Advanced Glycation End Products) monoclonal antibody

AGEs and Metabolic Syndrome

A AGEs, advanced glycation end products, are toxic compounds that accumulate in atherosclerotic plaques and can be dangerous for patients with diabetes or other metabolic diseases.

AGEs can arise not only from glucose, but also from dicarbonyl compounds. A rapid pathway and leads to an insulin-resistant state in L6 muscle cells. Moreover, AGEs can stabilize the PTP in the closed conformation in rat liver mitochondria. Furthermore, AGEs can cause TNF-induced cell death and AGE-4 is formed during TNF-induced cell death in mouse L929 cells. It has been showed that exogenously added MG has a strong synergistic effect on AGE-4-BSA-induced cell death.

Among AGEs, glycolaldehyde-derived AGEs (referred to as AGE-3) have diverse toxic and pro-inflammatory effects, including pericyte dysfunction. AGE-3 also decreases the viability and suppresses the replication rate in human pericytes. AGE-3 is also involved in the pathogenesis of the early stage of diabetic nephropathy.

Anti-AGE monoclonal antibody, clone 6D12, has applications for immunohistochemistry, Western blotting, and IHC staining of pathological tissues. It is a newly developed clone with increased sensitivity and specificity over previous clones. NF-1G allows sensitive detection of CML during immunohistochemical staining of pathological tissues.

In atherosclerotic lesions of stage of human aorta, IHC staining of the early pathological tissues showed that AGE-3 is a marker of inflammation in atherosclerotic lesions.

Non-Fluorescent / Non-Glycated

Anti CML Monoclonal Antibody

CML (carboxymethyl-lysine) is a major AGE formed by reaction of intracellular methylglyoxal with lysine residues and is an important marker for age-dependent disease such as cardiovascular disease in diabetic patients. Imidazaone is formed in tissues through reaction of proteins with deoxyglucosone (3DG) or methylglyoxal.

CML is a marker of oxidative stress and long term damage to protein in aging, atherosclerosis, and diabetes. CML is a major AGE formed by reaction of intracellular methylglyoxal with lysine residues and is an important marker for age-dependent disease such as cardiovascular disease in diabetic patients.

Anti-Cholesterol Monoclonal Antibody

Cholesterol is a major component of atherosclerotic plaques and is involved in the development of atherosclerosis. The myeloperoxidase-H2O2 (MPO) reaction with lipoprotein A (LpA) and glycated collagen. CMA is preferentially generated in glycated collagen.

Anti AGE-4 monoclonal antibody is a tool for studying the development of atherosclerotic plaques. AGE-4, in addition to AGE-3, is a major AGE formed by reaction of intracellular methylglyoxal with lysine residues and is an important marker for age-dependent disease such as cardiovascular disease in diabetic patients.

Anti CMA Monoclonal Antibody

CMA (carboxymethyl-lysine) is formed by reaction of intracellular methylglyoxal with lysine residues and is an important marker for age-dependent disease such as cardiovascular disease in diabetic patients. CMA has been showed that the anti-AGE-4 monoclonal antibody, clone 6D12, is a tool for studying the development of atherosclerotic plaques.

Fluorescent / Crosslinked

Anti AGE-4 Monoclonal Antibody

AGE-4 monoclonal antibody is a tool for studying the development of atherosclerotic plaques. AGE-4, in addition to AGE-3, is a major AGE formed by reaction of intracellular methylglyoxal with lysine residues and is an important marker for age-dependent disease such as cardiovascular disease in diabetic patients.