COSMO BIO CO., LTD.
Inspiration for Life Science

Trans Genic Inc.

KG133 Anti AGE-4 Monoclonal Antibody (Clone No. 14B5)

Primary Source	-
Type	Monoclonal
Immunogen	AGE4-BSA
Raised in	GANP mouse
Myeloma	P3U1
Clone number	14B5
Isotype	IgG1 κ
Source	Serum Free Medium
Purification notes	ProteinG
Cross Reactivity	Every animal species
Concentration	0.25 mg/mL
Contents (Volume)	10 µg (40 µL/vial)
Label	Unlabeled
Buffer	PBS [containing 2 % Block Ace as a stabilizer, 0.1 %Proclin as a bacteriostat]
Storage	Store below –20 ℃. Once thawed, store at 4 ℃. Repeated freeze-thaw cycles should be avoided.

Application	
WB	0.1 µg/mL
IHC	Not tested
ICC	Not tested
ELISA	0.05 µg/mL
FCM	Not tested
Neutralization	Not tested
IP	Not tested

GANP

This product is generated from GANP®

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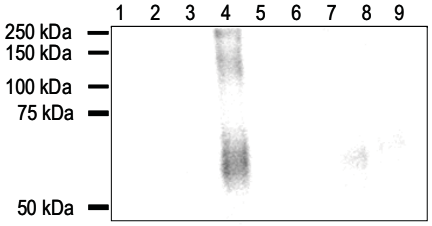

Western blotting

1; AGE1 -BSA (glucose -modified)
2; AGE2 -BSA (glyceraldehyde -modified)
3; AGE3 -BSA (glycolaldehyde -modified)
4; AGE4 -BSA (methylglyoxal -modified)
5; AGE5 -BSA (glyoxal -modified)
6; AGE6 -BSA (3 -DG -modified)
7; CML -BSA (carboxymethyllysine -modified)
8; CEL -BSA (carboxyethyllysine -modified)
9; BSA

Note

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. Methylglyoxal (MG) increases in diabetes and can modify proteins rapidly and form AGE-4. 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 PTP covalently and stabilizes the PTP 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.

AGEs (advanced glycation end-products; 終末糖化産物) はグルコースなどの還元糖とタンパク質、脂質、核酸といった生体分子との間の非酵素的糖化反応で生成され、糖尿病、炎症、腎不全といった疾患や老化に伴い蓄積します。AGEs は、糖尿病網膜症や腎症といった糖尿病血管合併症の発症・進展に強く関与しています。さらに AGEs は、動脈硬化症、アルツハイマー病、末期腎不全、関節リウマチ、肝硬変などの様々な疾患で増加します。
AGEs は、グルコースに由来するだけでなく、ジカルボニル化合物、糖の自動酸化物、糖代謝中間体などからも生成されます。メチルグリオキサール (MG) は糖尿病患者で増加し、タンパク質を修飾し、AGE-4 を生成します。MG は TNF により誘導される細胞死の作用を増強し、細胞死の際、AGE-4 が増加すること、MG 及び AGE-4 がマイコバクテリアに感染したマクロファージにおいてアポトーシスを誘導することが示されています。また細胞死に重要な役割を果たしているミトコンドリア PTP を MG が修飾し、閉口状態に PTP を安定化することが示されています。このほか、MG の増加がインスリンシグナル経路を阻害し、インスリン抵抗性を示すことも明らかとなっております。
本抗体は AGE-4 に特異的な抗体であり、細胞死、加齢に伴う慢性疾患や糖尿病関連疾病などの研究にご使用下さい。

Reference

1	Takeuchi M. et al.:	Immunological evidence that non-carboxymethyllysine advanced glycation end-products are produced from short chain sugars and dicarbonyl compounds in vivo.	Mol Med. 2000 Feb;6(2):114-25.
2	Takeuchi M. et al.:	Immunological detection of a novel advanced glycation end-product.	Mol Med. 2001 Nov;7(11):783-91.
3	Van Herreweghe F. et al.:	Tumor necrosis factor-induced modulation of glyoxalase I activities through phosphorylation by PKA results in cell death and is accompanied by the formation of a specific methylglyoxal-derived AGE.	Proc Natl Acad Sci U S A. 2002 Jan 22;99(2):949-54. Epub 2002 Jan 15.
4	Speer O. et al.:	Rapid suppression of mitochondrial permeability transition by methylglyoxal. Role of reversible arginine modification.	J Biol Chem. 2003 Sep 12;278(37):34757-63. Epub 2003 Jun 18.
5	Riboulet-Chavey A. et al.:	Methylglyoxal impairs the insulin signaling pathways independently of the formation of intracellular reactive oxygen species.	Diabetes. 2006 May;55(5):1289-99.
6	Rachman H. et al.:	Critical role of methylglyoxal and AGE in mycobacteria-induced macrophage apoptosis and activation.	PLoS ONE. 2006 Dec 20;1:e29.

WARNING AND PRECAUTION

1. Not for diagnostic use. The safety and efficacy of product in diagnostic or other clinical uses has not been established.
2. Harmful by inhalation, in contact with skin and if swallowed. Do not breathe dust. Avoid contact with skin and eyes.
3. If contact with skin and eyes, wash all affected areas with large volume of water. If inhaled remove to fresh air. In severe case obtain medical attention.
4. Wash hand thoroughly after handling the product.
5. Do not use this product if container is broken or some contaminants are detected.
6. When preserving the product, Close the container, ensure it does not fall aside or down.
7. Dispose of the container and expired reagents in accordance with federal, state and local government regulations.
8. Do not use the container and accessories of the product for other purpose.

取り扱い上の注意

この添付文書をよく読んでから使用して下さい。

1. 本品は研究用試薬であり、医薬品その他の目的にはご使用になれません。
2. 取り扱い中は皮膚、粘膜、着衣に触れたり、目に入らないように適切な措置を行って下さい。
3. 試薬が誤って目や口に入った場合には、水で十分に洗い流すなどの応急処置を行い、必要があれば医師の手当を受けて下さい。
4. 取り扱い後には手洗いを十分に行って下さい。
5. 容器の破損、異物混入等異常が認められた物は使用しないで下さい。
6. 試薬を保管する場合は、蓋をし、転倒落下防止を確実にし、指定の貯蔵方法で保管して下さい。
7. 使用後の容器は、廃棄物に関する規定に従って処理して下さい。
8. 容器、付属品等の他目的への転用は保証できません。

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