

## CEL-BSA

**CATALOG NUMBER:** STA-302

**STORAGE:** -20°C

**QUANTITY AND CONCENTRATION:** 100 µL of 1.0 mg/mL CEL-BSA in 1X PBS.

**SHELF LIFE:** 1 year from date of receipt under proper storage conditions; aliquot to avoid multiple freeze thaw cycles

### **Background**

The non-enzymatic reaction of reducing carbohydrates with lysine side chains and N-terminal amino groups of macromolecules (proteins, phospholipids and nucleic acids) is called the Maillard reaction or glycation. The products of this process, termed advanced glycation end products (AGEs), adversely affect the functional properties of proteins, lipids and DNA. Tissue levels of AGE increase with age and the formation of AGEs is predominantly endogenous, though these products can also be derived from exogenous sources such as food and tobacco smoke. AGE modification of proteins can contribute to the pathophysiology of aging and long-term complications of diabetes, atherosclerosis and renal failure. AGEs also interact with a variety of cell-surface AGE-binding receptors (RAGE), leading either to their endocytosis and degradation or to cellular activation and pro-oxidant or pro-inflammatory events.

Although several AGE structures have been reported, it was demonstrated that N<sup>ε</sup>-(carboxymethyl) lysine (CML) and N<sup>ε</sup>-(carboxyethyl) lysine (CEL) are the major antigenic AGE structures. Next to glucose, reactive di-carbonyl compounds such as methylglyoxal are major precursors in the formation of cellular and extracellular AGEs. Methylglyoxal reacts with lysine residues to form CEL. CEL concentration is increased in patients who have diabetes with complications.

### **Methods**

Dilute the CEL-BSA with SDS-PAGE reducing sample buffer to 1.0-10 µg/mL and boil for 5 minutes. Load 10 µL per lane for western blot analysis of CEL protein adducts.

### **References**

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### **Recent Product Citation**

Liu, Z. et al. (2025). N $\epsilon$ -carboxyethyl-lysine influences atherosclerotic plaque stability through ZKSCAN3 acetylation-regulated macrophage autophagy via the RAGE/LKB1/AMPK1/SIRT1 pathway. *Cardiovasc Diabetol.* **24**(1):36. doi: 10.1186/s12933-025-02586-y.

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