

DATA SHEET

Product Name:	Calmodulin-SH, Wheat (<i>Triticum aestivum</i>), Immobilized High Loading; CaMTag™ HD Capture.
Catalog #	C-1011-1
Source:	Isolated from wheat germ
Molecular Mass:	-
Supplied As:	50% slurry in 3 M NaCl, 20mM sodium phosphate buffer, pH, 7.4 and 1 mM EGTA.
Protein Purity:	Calmodulin Content: 1 mL of resin bound with 5 –5.5 mg of wheat germ calmodulin.
Activity:	Covalently bound to the resin products retain their ability to bind calmodulin target proteins in a calcium-dependent manner.

Storage: +4°C. Do not freeze.

Description: Calmodulin (CaM) *Triticum aestivum* (Wheat) sequence is unique in that it contains cysteine at residue position 26 (Cys26). Wheat germ calmodulin coupled primarily through SH group (Cys26) to epoxy activated resin. Coupled at 5 mg per mL and useful for pull down experiments and proteomic applications requiring a high capacity precipitation reagent. Dense resin can be easily separated from extracts by centrifugation using batch or spin column procedures. Resin can be reused but proteolysis from crude samples will reduce binding capacity on repeat cycles. Loading conditions: 100 uM Ca²⁺, with protease inhibitors and appropriate buffers. Elution conditions: Buffers with 1 mM EDTA or EGTA.

Calmodulin (CaM) is a ubiquitous, calcium-binding protein that binds and regulates a multitude of protein targets, many of which are involved in the Alzheimer's and the Parkinson's pathways^{1, 4}. CaM has a molecular weight of about 17kDa, containing 148 amino acids, and pI of 3.9. CaM is characterized by two domains, connected by an alpha-helix chain. Each domain has the capacity to bind two calcium ions. Binding Ca²⁺ ions causes a conformational change in CaM, making it available for interaction with target proteins. Hence, CaM functions as an intracellular calcium ion bridge to mediate cellular reactions and responds appropriately to calcium ion concentration. In Alzheimer's disease (AD), irregular calcium homeostasis seems to trigger CaM and its binding proteins, to enhance plaque formation and neurofibrillary degeneration, which results in cell death¹. The increased cytosolic levels of Ca²⁺ in AD neurons promotes CaM binding and regulation of available Ca²⁺/CaM-dependent CaM-binding proteins, associated with amyloid beta (Ab) formation. In addition, the increased level of Ca²⁺ triggers Calmodulin to activate calcium/CaM-dependent kinase II and precede neurofibrillary tangle formation^{2, 4}. In Parkinson's disease (PD), Calmodulin has been found to interact, in a calcium dependent manner, with Alpha-Synuclein, which is associated with the progression of PD. CaM was identified as one of the synuclein-interacting proteins that regulate synuclein conformation³.

- References:**
1. FM LaFerla. 2002, Nature Reviews Neurosci. **3**: 862-872.
 2. O'Day DH & Myre M., 2004, Biochem Biophys Res Comm. **320**: 1051.
 3. Martinez J., et al., 2003, J. of Biological Chemistry. **278**:17379.
 4. Picconi B, et al., 2004, The J. of Neurosci. **24(23)**: 5283.



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Note: *The animal source of this product was collected at a USDA licensed establishment. The animals received ante and post mortem health inspection at the abattoir by a US FSIS inspector and they were apparently free from infectious and contagious diseases. All donor animals were sourced from the United States.*

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