



# Diphtheria Toxoid (17.6B9): sc-80475

## BACKGROUND

Diphtheria Toxoid is a 567 amino acid proenzyme that is produced by Coryne-*phage-β* infection of *Corynebacterium diphtheriae*. Functioning as an enzymatic catalyst, Diphtheria Toxoid participates in the covalent attachment of elongation factor 2 (EF-2) to the ADP ribose moiety of NAD, thereby killing the infected cell. Diphtheria Toxoid works in three sequential steps, the first of which is penetration of the cell via receptor-mediated endocytosis. The toxin then translocates across the membrane into the cytoplasm where it ultimately attaches an ADP-ribosyl group to a modified histidine on EF-2, thus blocking protein synthesis and causing cell death. These steps are performed by three distinct structural domains: the receptor-binding domain (R), the pore-forming membrane-translocation domain (T) and the catalytic domain (C). Although only a single molecule of Diphtheria Toxoid is sufficient to kill a cell, toxicity can be repressed by DtxR, an iron-dependent transcriptional repressor that downregulates the expression of various virulence factors, including Diphtheria Toxoid.

## REFERENCES

- Louie, G.V., Yang, W., Bowman, M.E. and Choe, S. 1997. Crystal structure of the complex of diphtheria toxin with an extracellular fragment of its receptor. *Mol. Cell.* 1: 67-78.
- Parikh, S.L. and Schramm, V.L. 2004. Transition state structure for ADP-ribosylation of eukaryotic elongation factor 2 catalyzed by diphtheria toxin. *Biochemistry* 43: 1204-1212.
- Ortiz, P.A., Ulloque, R., Kihara, G.K., Zheng, H. and Kinzy, T.G. 2006. Translation elongation factor 2 anticodon mimicry domain mutants affect fidelity and diphtheria toxin resistance. *J. Biol. Chem.* 281: 32639-32648.
- Furukawa, N., Saito, M., Hakoshima, T. and Kohno, K. 2006. A diphtheria toxin receptor deficient in epidermal growth factor-like biological activity. *J. Biochem.* 140: 831-841.
- Kageyama, T., Ohishi, M., Miyamoto, S., Mizushima, H., Iwamoto, R. and Mekada, E. 2007. Diphtheria toxin mutant CRM197 possesses weak EF-2-ADP-ribosyl activity that potentiates its anti-tumorigenic activity. *J. Biochem.* 142: 95-104.
- D'Aquino, J.A., Lattimer, J.R., Denninger, A., D'Aquino, K.E. and Ringe, D. 2007. Role of the N-terminal helix in the metal ion-induced activation of the diphtheria toxin repressor DtxR. *Biochemistry* 46: 11761-11770.
- Qiao, J., Ghani, K. and Caruso, M. 2007. Diphtheria toxin mutant CRM197 is an inhibitor of protein synthesis that induces cellular toxicity. *Toxicon* 51: 473-477.
- Perier, A., Gourier, C., Pichard, S., Husson, J., Lajeunesse, E., Babon, A., Menez, A. and Gillet, D. 2007. Creation of intercellular bonds by anchoring protein ligands to membranes using the diphtheria toxin T domain. *FEBS Lett.* 581: 5480-5484.
- Kent, M., Yim, H., Murton, J., Satija, S., Majewski, J. and Kuzmenko, I. 2007. Oligomerization of membrane-bound diphtheria toxin (CRM197) facilitates a transition to the open form and deep insertion. *Biophys. J.* 94: 2115-2127.

## SOURCE

Diphtheria Toxoid (17.6B9) is a mouse monoclonal antibody raised against Diphtheria Toxoid.

## PRODUCT

Each vial contains 100 µg IgG<sub>1</sub> in 1.0 ml PBS with < 0.1% sodium azide and 0.1% gelatin.

## APPLICATIONS

Diphtheria Toxoid (17.6B9) is recommended for detection of Diphtheria Toxoid of *Corynebacterium diphtheriae* origin by solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Molecular Weight of Diphtheria Toxoid: 62 kDa.

## STORAGE

Store at 4° C, **\*\*DO NOT FREEZE\*\***. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

## RESEARCH USE

For research use only, not for use in diagnostic procedures.

## PROTOCOLS

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