

## Product Information

### DCC/Fc CHIMERA

Mouse, Recombinant  
Expressed in Sf21 insect cells

Product Number **D 0440**  
Storage Temperature  $-20^{\circ}\text{C}$

Synonyms: Deleted in Colorectal Cancer

### Product Description

DCC/Fc chimera is produced from a cDNA sequence encoding the extracellular domain (amino acid residues 1-1097) of mouse DCC fused by means of a polypeptide linker to the Fc portion of human IgG<sub>1</sub> that is histidine-tagged at the carboxyl terminus. The chimeric protein is expressed in Sf21 insect cells infected with baculovirus. Recombinant DCC/Fc chimera is a disulfide-linked homodimer. The amino terminus is Phe<sup>32</sup> based on amino-terminal sequencing. The calculated molecular mass of the reduced DCC/Fc chimera monomer is approximately 146 kDa, but as a result of glycosylation, the recombinant DCC/Fc chimera migrates as a 160-170 kDa protein in SDS-PAGE under reducing conditions.<sup>1</sup>

Deleted in colorectal cancer (dcc, chromosome 18q21) was originally identified as a putative tumor suppressor gene that is lost in more than 70% of colorectal cancers. The gene has also been deleted in several other types of cancer.<sup>2,6,7</sup> The DCC protein is a type I membrane protein belonging to the immunoglobulin (Ig) superfamily.<sup>2,3</sup> The extracellular domain is composed of four Ig-like domains and six fibronectin type III repeats. Native DCC is found in three isoforms. Two forms, a long and a short isoform, are produced from the same gene, but have different initiation sites. The third isoform is produced by alternative splicing and is expressed only in embryonic tissue. In adults, DCC is highly expressed in the brain but can be expressed at lower levels in multiple tissues.<sup>2,3,4</sup> Mouse DCC extracellular domain shares 97% and 99% homology with human and rat DCC, respectively. DCC functions as a receptor or a component of a receptor for netrins and mediates the effects of netrins on commissural axons. Netrins are chemoattractants responsible for the guidance of commissural axons at the midline and of motor axons to their target muscles. DCC may also induce apoptosis and has been shown to be a caspase substrate.<sup>4,5,6</sup>

### Reagents

Recombinant mouse DCC /Fc chimera is lyophilized from a sterile filtered phosphate-buffered saline (PBS) solution containing 50  $\mu\text{g}$  BSA per 1  $\mu\text{g}$  DCC fusion protein.

### Preparation Instructions

Reconstitute the vial contents with sterile PBS. Stock solution concentration should be no less than 200  $\mu\text{g}/\text{ml}$ .

### Storage/Stability

Lyophilized samples of recombinant mouse DCC/Fc chimera are stable for more than six months at  $-20^{\circ}\text{C}$ . Upon reconstitution, store at  $2-4^{\circ}\text{C}$  for up to one month. For extended storage, store in working aliquots at  $-20^{\circ}\text{C}$ . Repeated freeze-thaw cycles should be avoided. Do not store in a frost-free freezer.

### Product Profile

The activity of recombinant mouse DCC/Fc chimera is measured by its ability to bind netrin-2. DCC/Fc chimera is immobilized at 2  $\mu\text{g}/\text{ml}$  on a 96-well plate (100  $\mu\text{l}/\text{well}$ ) in a functional ELISA assay. DCC/Fc chimera can bind rcNetrin with a linear range of 3–100 ng/ml. Optimal dilutions should be determined by each laboratory for each application.

Purity: >90% by SDS-PAGE, visualized by silver stain.

Endotoxin level: < 0.1 ng/ $\mu\text{g}$  of protein as determined by the LAL (Limulus amoebocyte lysate) method.

### References

1. Cooper, H.M., et al., Cloning of the mouse homologue of the deleted in colorectal cancer gene (mDCC) and its expression in developing mouse embryo, *Oncogene*, 11:2243-2254 (1995).
2. Fearon, E.R., et al., Identification of a chromosome 18q gene that is altered in colorectal cancers, *Science*, 247:49-56 (1990).

3. Keino-Masu, K., et al., Deleted in Colorectal Cancer (DCC) Encodes a Netrin Receptor, *Cell*, 87:175-185 (1996).
4. Mehlen, P., The DCC gene product induces apoptosis by a mechanism requiring receptor proteolysis. *Nature*, 395:801-804 (1998).
5. Culotti, J.G. and D.C. Merz, DCC and netrins., *Curr. Opin. Cell Biol.*,10:609-613 (1998).
6. Huerta, S., et al., Human colon cancer cells deficient in DCC product abnormal transcripts in progression of carcinogenesis. *Dig. Dis. Sci.*, 46:1884-1891 (2001).
7. Woodford-Richens, K.L., et al., SMAD4 mutations in colorectal cancer probably occur before chromosomal instability, but after divergence of the microsatellite instability pathway. *Proc. Natl. Acad. Sci. USA*, 98:9719-9723 (2001).

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