

# PRODUCT DATA SHEET

## N-Dodecanoyl-NBD-L-*threo*-dihydrosphingosine

**Catalog number:** 1623

**Common names:** N-C12:0-NBD-Dihydroceramide; N-C12:0-NBD-L-*threo*-Dihydrosphingosine

**Source:** synthetic

**Solubility:** chloroform/methanol, 2:1; methanol

**CAS number:** 474943-07-2

**Molecular Formula:** C<sub>36</sub>H<sub>63</sub>N<sub>5</sub>O<sub>6</sub>

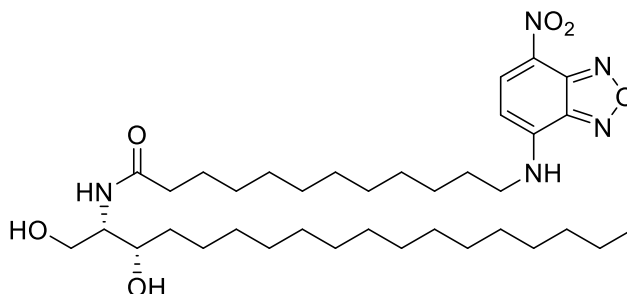
**Molecular Weight:** 662

**Storage:** -20°C

**Purity:** TLC >98%; identity confirmed by MS

**TLC System:** chloroform/methanol (90:10 by vol.)

**Appearance:** orange solid



### Application Notes:

N-Dodecanoyl-NBD-L-*threo*-dihydrosphingosine is a synthetic dihydroceramide analog containing the 7-(4-nitrobenzo-2-oxa-1,3-diazole) (NBD) fluorescent group. NBD has been shown to have only a small influence on lipid adsorption into cells and cellular membranes in many applications. This fluorescent analog of L-*threo*-dihydroceramide is comparable to C12:0-L-*threo*-dihydroceramide in some biological functions. Safingol is a fully saturated, non-natural analog of sphingosine that has anticancer properties and is being investigated for its potential as an antitumor therapy. It has been shown to inhibit both protein kinase C (PKC) and sphingosine kinase. Safingol competitively interacts at the regulatory phorbol-binding domain of PKC, a kinase involved in tumorigenesis. Safingol has been shown to potentiate the effect of doxorubicin (DOX) in tumor-bearing animals.<sup>1</sup> It has been reported that safingol is able to increase the activity of DOX and other chemotherapeutic agents, including mitomycin C, by generating the pro-apoptotic second messenger ceramide, even in tumor cell lines that were resistant to chemotherapy due to mutations.<sup>2</sup> However, a study has recently claimed that safingol induces cell death of an exclusively autophagic character and lacking any of the hallmarks of apoptosis.<sup>3</sup> Safingol inhibited the reactive oxygen intermediates (ROI) released from isolated neutrophils and phorbol ester-induced edema and neutrophil influx. Safingol also demonstrates anti-inflammatory activity. Safingol, like the natural D-*erythro*-sphinganine, is used as a biosynthetic precursor for all complex sphingolipids although the metabolism of the natural and the non-natural compounds are different.<sup>4</sup>

### Selected References:

1. Darges, et al. "Inhibition of leukotriene B4 (LTB4) in human neutrophils by L-*threo*-dihydrosphingosine" *Adv. Exp. Med. Biol.*, Vol. 400A pp. 387-392, 1997
2. G. Schwartz, et al. "A pilot clinical/pharmacological study of the protein kinase C-specific inhibitor safingol alone and in combination with doxorubicin" *Clin. Cancer Res.*, Vol. 3 pp. 537-543, 1997
3. J. Coward et al. "Safingol (L-*threo*-sphinganine) induces autophagy in solid tumor cells through inhibition of PKC and the PI3-kinase pathway" *Autophagy* Vol. 5(2) pp.184-193, 2009
4. M. Dragusin et al. "Metabolism of the unnatural anticancer lipid safingol, L-*threo*-dihydrosphingosine, in cultured cells" Vol. 44 pp. 1772, 2003

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