Product Data Sheet

Product Name: ASO-4625
Cat. No.: GC81825

Chemical Properties

Cas. No.			
Formula		M.Wt	
Solubility		Storage	Store at -20°C, away from moisture
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure			

Background

ASO-4625 is a 20-nucleotide gapmer antisense oligonucleotide with a full phosphorothioate backbone and 2'-O-(2-methoxy) ethyl ribose modifications, which targets BCL2 mRNA and BCL2L1 mRNA. ASO-4625 downregulates Bcl-2 expression via an RNase H-dependent antisense mechanism with 100% complementarity, and downregulates Bcl-xL expression via the same mechanism with three mismatches. ASO-4625 induces Apoptosis and acts as a chemosensitizer. ASO-4625 does not significantly modulate the expression of Bcl-W, survivin, Akt, or p53 mRNA. ASO-4625 can be used in studies of breast cancer and non-small cell lung cancer [1].

Caution: Product has not been fully validated for medical applications. For research use only.
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In Vitro, ASO-4625 (2-42 h) downregulates fully matched BCL2 mRNA more potently than mismatched BCL2L1 (Bcl-xL) mRNA in both MDA-MB-231 breast cancer and H125 lung cancer cells, with no significant off-target effects on Bcl-W, survivin, Akt, or p53 expression in MDA-MB-231 cells[1]. ASO-4625 (150-300 nM; 6-hour oligonucleotide incubation, followed by 42-hour further incubation after medium renewal; total 48 hours) reduces both Bcl-2 and Bcl-xL protein levels in MDA-MB-231 and H125 cells, with stronger downregulation of the fully complementary Bcl-2 target relative to the mismatched Bcl-xL target[1]. ASO-4625 (50-100 nM; 72 h) reduces viability of MDA-MB-231 breast cancer and H125 lung cancer cells and enhances the cytotoxic effect of multiple anticancer agents in both cell lines[1].

References:

[1]. Simões-Wüst AP, et al. A functionally improved locked nucleic acid antisense oligonucleotide inhibits Bcl-2 and Bcl-xL expression and facilitates tumor cell apoptosis. Oligonucleotides. 2004;14(3):199-209.

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