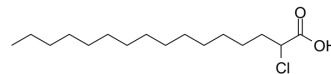


## 2-Chlorohexadecanoic acid

<b>Cat. No.:</b>	HY-131688
<b>CAS No.:</b>	19117-92-1
<b>Molecular Formula:</b>	C <sub>16</sub> H <sub>31</sub> ClO <sub>2</sub>
<b>Molecular Weight:</b>	290.87
<b>Target:</b>	Others
<b>Pathway:</b>	Others
<b>Storage:</b>	Please store the product under the recommended conditions in the Certificate of Analysis.



### BIOLOGICAL ACTIVITY

<b>Description</b>	2-Chlorohexadecanoic acid, an inflammatory lipid mediator, interferes with protein palmitoylation, induces ER-stress markers, reduced the ER ATP content, and activates transcription and secretion of IL-6 as well as IL-8. 2-Chlorohexadecanoic acid disrupts the mitochondrial membrane potential and induces procaspase-3 and PARP cleavage. 2-Chlorohexadecanoic acid can cross blood-brain barrier (BBB) and compromises ER- and mitochondrial functions in the human brain endothelial cell line hCMEC/D3 <sup>[1]</sup> .
<b>In Vitro</b>	2-Chlorohexadecanoic acid (2-ClHA; 10 μM; 4, 6 h) results phosphorylation of eIF2α starting 4 h post treatment, while total eIF2α levels remained unchanged incubation of hCMEC/D3 cells. 2-Chlorohexadecanoic acid increases expression of ATF4, a target gene of eIF2α <sup>[1]</sup> . 2-Chlorohexadecanoic acid (25 μM, 30 min) induces a decrease in the FRET ratio signal of ERAT by 40%, indicating significantly diminished [ATP]ER in cells that were treated with 2-Chlorohexadecanoic acid <sup>[1]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.

### REFERENCES

[1]. Eva Bernhart, et al. 2-Chlorohexadecanoic acid induces ER stress and mitochondrial dysfunction in brain microvascular endothelial cells. *Redox Biol.* 2018 May;15:441-451.

**Caution: Product has not been fully validated for medical applications. For research use only.**

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