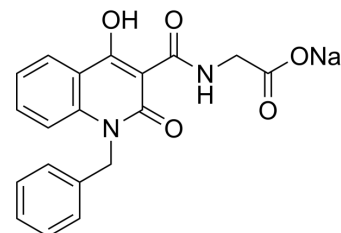


## IOX2 sodium

Cat. No.:	HY-15468A
CAS No.:	2377239-85-3
Molecular Formula:	C <sub>19</sub> H <sub>15</sub> N <sub>2</sub> NaO <sub>5</sub>
Molecular Weight:	374.32
Target:	HIF/HIF Prolyl-Hydroxylase
Pathway:	Metabolic Enzyme/Protease
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



### BIOLOGICAL ACTIVITY

<b>Description</b>	IOX2 sodium is a specific prolyl hydroxylase-2 (PHD2) inhibitor with IC <sub>50</sub> of 22 nM. IOX2 sodium regulates platelet function and arterial thrombosis by upregulating HIF-1α expression and inhibiting ROS production. IOX2 sodium can be used in the study of thrombotic diseases <sup>[1][2]</sup> .									
<b>IC<sub>50</sub> &amp; Target</b>	IC <sub>50</sub> : 22 nM (PHD2) <sup>[2]</sup>									
<b>In Vitro</b>	<p>IOX2 sodium (0, 10, 25, and 50 μM) dose-dependently inhibits collagen-related peptide (CRP; 0.25 μg/mL) or thrombin (0.03 U/mL)-induced platelet aggregation and ATP release. But IOX2 doesn't affect P-selectin expression and the surface levels of glycoprotein (GP)Ibα, GPVI, or αIIbβ<sub>3</sub><sup>[1]</sup>.</p> <p>IOX2 sodium also inhibits the spreading of platelets on fibrinogen or collagen and clot retraction<sup>[1]</sup>.</p> <p>IOX2 sodium (50 μM; 24 h) increases the transcription level of VEGF-A and BNIP3 in Normal human epidermal keratinocytes (NHEK) and Normal human dermal fibroblasts (NHDF), when grown under normoxia and hypoxia condition<sup>[2]</sup>.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>									
<b>In Vivo</b>	<p>IOX2 sodium (10 mg/kg; i.p.; single dose) impaired the in vivo hemostatic function of platelets and arterial thrombus formation in mice<sup>[1]</sup>.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <table border="1" data-bbox="341 1428 1510 1806"> <tr> <td>Animal Model:</td> <td>Mouse<sup>[1]</sup></td> </tr> <tr> <td>Dosage:</td> <td>10 mg/kg</td> </tr> <tr> <td>Administration:</td> <td>Intraperitoneal injection</td> </tr> <tr> <td>Result:</td> <td>Upregulated HIF-1α in platelets, decreased ROS generation, and downregulated NOX1 expression. Increased the phosphorylation level of VASP (Ser157/239), and inhibited the phosphorylation of p38 (Thr180/Tyr182), ERK1/2 (Thr202/Tyr204), AKT (Thr308/Ser473), and PKCδ (Thr505) in CRP- or thrombin-stimulated platelets.</td> </tr> </table>		Animal Model:	Mouse <sup>[1]</sup>	Dosage:	10 mg/kg	Administration:	Intraperitoneal injection	Result:	Upregulated HIF-1α in platelets, decreased ROS generation, and downregulated NOX1 expression. Increased the phosphorylation level of VASP (Ser157/239), and inhibited the phosphorylation of p38 (Thr180/Tyr182), ERK1/2 (Thr202/Tyr204), AKT (Thr308/Ser473), and PKCδ (Thr505) in CRP- or thrombin-stimulated platelets.
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### CUSTOMER VALIDATION

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- Nat Commun. 2022 May 4;13(1):2447.
  - Oxid Med Cell Longev. 2020 Jan 4;2020:4909103.
  - Aging. 2021 May 20;13(10):14355-14371.
  - Thromb Haemostasis. 2022 Apr 27.
  - Sci Rep. 2022 Jan 27;12(1):1443.

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## REFERENCES

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[1]. Gu W, et al. Inhibition of Hypoxia-Inducible Factor Prolyl-Hydroxylase Modulates Platelet Function. Thromb Haemost. 2022 Oct;122(10):1693-1705.

[2]. Deppe J, et al. Impairment of hypoxia-induced HIF-1 $\alpha$  signaling in keratinocytes and fibroblasts by sulfur mustard is counteracted by a selective PHD-2 inhibitor. Arch Toxicol. 2016 May;90(5):1141-50.

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

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