



# SZABO SCANDIC

Part of Europa Biosite

## Produktinformation



Forschungsprodukte & Biochemikalien



Zellkultur & Verbrauchsmaterial



Diagnostik & molekulare Diagnostik



Laborgeräte & Service

Weitere Information auf den folgenden Seiten!  
See the following pages for more information!



### Lieferung & Zahlungsart

siehe unsere [Liefer- und Versandbedingungen](#)

### Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

### SZABO-SCANDIC HandelsgmbH

Quellenstraße 110, A-1100 Wien

T. +43(0)1 489 3961-0

F. +43(0)1 489 3961-7

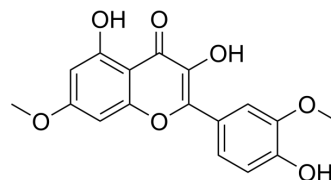
[mail@szabo-scandic.com](mailto:mail@szabo-scandic.com)

[www.szabo-scandic.com](http://www.szabo-scandic.com)

[linkedin.com/company/szaboscandic](https://www.linkedin.com/company/szaboscandic) 

## Rhamnazin

Cat. No.:	HY-N8342
CAS No.:	552-54-5
Molecular Formula:	C <sub>17</sub> H <sub>14</sub> O <sub>7</sub>
Molecular Weight:	330.29
Target:	VEGFR
Pathway:	Protein Tyrosine Kinase/RTK
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



### BIOLOGICAL ACTIVITY

<b>Description</b>	Rhamnazin is an orally active inhibitor of VEGFR2 signaling with an IC <sub>50</sub> of 4.68 μM against VEGFR2 kinase. Rhamnazin shows potent antiangiogenic activity and antitumor efficacy <sup>[1]</sup> . Rhamnazin shows antioxidant and anti-inflammatory properties <sup>[2]</sup> .																		
<b>IC<sub>50</sub> &amp; Target</b>	VEGFR2 4.68 μM (IC <sub>50</sub> )																		
<b>In Vitro</b>	<p>Rhamnazin (5-40 μM) inhibits proliferation, migration and tube formation of HUVECs induced by VEGF<sup>[1]</sup>.            Rhamnazin (0-20 μM) attenuates VEGFR-2 tyrosine kinase activity and VEGFR-2 signaling pathway<sup>[1]</sup>.            Rhamnazin (0-40 μM; 24 h) inhibits the proliferation and VEGFR2 signaling pathway of breast cancer cells<sup>[1]</sup>.            MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <p><b>Cell Migration Assay<sup>[1]</sup></b></p> <table border="1"> <tr> <td>Cell Line:</td> <td>HUVECs</td> </tr> <tr> <td>Concentration:</td> <td>0, 10, 15 and 20 μM</td> </tr> <tr> <td>Incubation Time:</td> <td>6 h</td> </tr> <tr> <td>Result:</td> <td>Strongly inhibited the migration of HUVECs.</td> </tr> </table> <p><b>Western Blot Analysis<sup>[1]</sup></b></p> <table border="1"> <tr> <td>Cell Line:</td> <td>HUVECs</td> </tr> <tr> <td>Concentration:</td> <td>0, 10, 15 and 20 μM</td> </tr> <tr> <td>Incubation Time:</td> <td>24 h</td> </tr> <tr> <td>Result:</td> <td>Decreased VEGF binding to VEGFR2. Reduced VEGF-stimulated phosphorylation of VEGFR2 and its downstream MAPK, AKT, and STAT3 in HUVECs in a concentration dependent manner.</td> </tr> </table> <p><b>Cell Proliferation Assay<sup>[1]</sup></b></p> <table border="1"> <tr> <td>Cell Line:</td> <td>HCC1937, T-47D, SK-BR-3, MCF-7 and MDA-MB-231</td> </tr> </table>	Cell Line:	HUVECs	Concentration:	0, 10, 15 and 20 μM	Incubation Time:	6 h	Result:	Strongly inhibited the migration of HUVECs.	Cell Line:	HUVECs	Concentration:	0, 10, 15 and 20 μM	Incubation Time:	24 h	Result:	Decreased VEGF binding to VEGFR2. Reduced VEGF-stimulated phosphorylation of VEGFR2 and its downstream MAPK, AKT, and STAT3 in HUVECs in a concentration dependent manner.	Cell Line:	HCC1937, T-47D, SK-BR-3, MCF-7 and MDA-MB-231
Cell Line:	HUVECs																		
Concentration:	0, 10, 15 and 20 μM																		
Incubation Time:	6 h																		
Result:	Strongly inhibited the migration of HUVECs.																		
Cell Line:	HUVECs																		
Concentration:	0, 10, 15 and 20 μM																		
Incubation Time:	24 h																		
Result:	Decreased VEGF binding to VEGFR2. Reduced VEGF-stimulated phosphorylation of VEGFR2 and its downstream MAPK, AKT, and STAT3 in HUVECs in a concentration dependent manner.																		
Cell Line:	HCC1937, T-47D, SK-BR-3, MCF-7 and MDA-MB-231																		

	Concentration:	0, 10, 15, 20, 30 and 40 $\mu$ M
	Incubation Time:	24 h
	Result:	Inhibited cell growth with IC <sub>50</sub> s of 19, 27, 32, 41 and 64 $\mu$ M against MDA-MB-231, MCF-7, SK-BR-3, T-47D and HCC1937 in the presence of VEGF, respectively.
<b>In Vivo</b>	Rhamnazin (200 mg/kg; i.g.; daily for 25 days) inhibits breast cancer growth and angiogenesis in mice <sup>[1]</sup> . Rhamnazin (5-20 mg/kg; i.p.; once) shows strong antioxidant and anti-inflammatory properties in the rat acute lung injury model <sup>[2]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.	
	Animal Model:	BALB/c nude mice, breast cancer xenograft model <sup>[1]</sup>
	Dosage:	200 mg/kg
	Administration:	Intragastric administration, daily for 25 days
	Result:	Dramatically suppressed tumor volumes by 47% compared with the vehicle group. Showed a significant reduction of pVEGFR2 <sup>Tyr951</sup> -positive cells in tumors. Resulted in downregulation of VEGFR2 downstream molecules phosphorylation including MAPK, AKT and STAT3.

## REFERENCES

[1]. Yu Y, et al. Rhamnazin, a novel inhibitor of VEGFR2 signaling with potent antiangiogenic activity and antitumor efficacy. *Biochem Biophys Res Commun.* 2015 Mar 20;458(4):913-9.

[2]. Wu G, et al. ANTIOXIDANT AND ANTI-INFLAMMATORY EFFECTS OF RHAMNAZIN ON LIPOPOLYSACCHARIDE-INDUCED ACUTE LUNG INJURY AND INFLAMMATION IN RATS. *Afr J Tradit Complement Altern Med.* 2017 Jun 5;14(4):201-212.

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

Tel: 609-228-6898

Fax: 609-228-5909

E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA