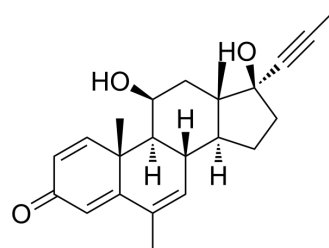


RU28362

Cat. No.:	HY-121859
CAS No.:	74915-64-3
Molecular Formula:	C ₂₃ H ₂₈ O ₃
Molecular Weight:	352.47
Target:	Glucocorticoid Receptor
Pathway:	Immunology/Inflammation; Vitamin D Related/Nuclear Receptor
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	RU28362 is a potent and selective glucocorticoid agonist. RU28362 increases the Bnip3 mRNA levels in neurons. RU28362 inhibits adrenocorticotrophic hormone (ACTH) and corticosterone secretion ^{[1][2]} . RU28362 is a click chemistry reagent, it contains an Alkyne group and can undergo copper-catalyzed azide-alkyne cycloaddition (CuAAC) with molecules containing Azide groups.								
In Vitro	<p>RU28362 (0.1, 1, 5, 10 nM; 72 h) increases the Bnip3 mRNA levels in a dose-dependent manner after 72 h in neurons^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <p>RT-PCR^[1]</p> <table border="1"> <tr> <td>Cell Line:</td> <td>Primary cortical neurons</td> </tr> <tr> <td>Concentration:</td> <td>0.1, 1.0, 5.0, 10.0 nM</td> </tr> <tr> <td>Incubation Time:</td> <td>48, 72 h</td> </tr> <tr> <td>Result:</td> <td>Significantly increased the Bnip3 mRNA levels in a dose-dependent manner after 72 h, Bnip3 mRNA levels were not changed after treatment with RU28362 for 48 h.</td> </tr> </table>	Cell Line:	Primary cortical neurons	Concentration:	0.1, 1.0, 5.0, 10.0 nM	Incubation Time:	48, 72 h	Result:	Significantly increased the Bnip3 mRNA levels in a dose-dependent manner after 72 h, Bnip3 mRNA levels were not changed after treatment with RU28362 for 48 h.
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In Vivo	<p>RU28362 (150 µg/kg; i.p.) inhibits adrenocorticotrophic hormone (ACTH) and corticosterone secretion and selectively suppressed the stress-induced increase in POMC hnRNA in the anterior pituitary gland^[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <table border="1"> <tr> <td>Animal Model:</td> <td>250-350 g, adult male Sprague-Dawley rats^[2]</td> </tr> <tr> <td>Dosage:</td> <td>150 µg/kg</td> </tr> <tr> <td>Administration:</td> <td>I.p.; 60 min before exposure to a 15-min period of restraint stress</td> </tr> <tr> <td>Result:</td> <td>Inhibited adrenocorticotrophic hormone (ACTH) and corticosterone secretion and selectively suppressed the stress-induced increase in POMC hnRNA in the anterior pituitary gland.</td> </tr> </table>	Animal Model:	250-350 g, adult male Sprague-Dawley rats ^[2]	Dosage:	150 µg/kg	Administration:	I.p.; 60 min before exposure to a 15-min period of restraint stress	Result:	Inhibited adrenocorticotrophic hormone (ACTH) and corticosterone secretion and selectively suppressed the stress-induced increase in POMC hnRNA in the anterior pituitary gland.
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REFERENCES

[1]. Sandau US, et al. Glucocorticoids exacerbate hypoxia-induced expression of the pro-apoptotic gene Bnip3 in the developing cortex. *Neuroscience*. 2007 Jan 19;144(2):482-94.

[2]. Ginsberg AB, et al. Specific and time-dependent effects of glucocorticoid receptor agonist RU28362 on stress-induced pro-opiomelanocortin hnRNA, c-fos mRNA and zif268 mRNA in the pituitary. *J Neuroendocrinol*. 2006 Feb;18(2):129-38.

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

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