

## Adrenocorticotrophic Hormone (ACTH) (1-39), rat

<b>Cat. No.:</b>	HY-P1477
<b>CAS No.:</b>	77465-10-2
<b>Molecular Formula:</b>	C <sub>210</sub> H <sub>315</sub> N <sub>57</sub> O <sub>57</sub> S
<b>Molecular Weight:</b>	4582.23
<b>Sequence:</b>	Ser-Tyr-Ser-Met-Glu-His-Phe-Arg-Trp-Gly-Lys-Pro-Val-Gly-Lys-Lys-Arg-Arg-Pro-Val-Lys-Val-Tyr-Pro-Asn-Val-Ala-Glu-Asn-Glu-Ser-Ala-Glu-Ala-Phe-Pro-Leu-Glu-Phe
<b>Sequence Shortening:</b>	SYSMEHFRWGGKPVGKKRRPVKVYPNVAENESAEAFPLEF
<b>Target:</b>	Melanocortin Receptor
<b>Pathway:</b>	GPCR/G Protein; Neuronal Signaling
<b>Storage:</b>	Please store the product under the recommended conditions in the Certificate of Analysis.

### BIOLOGICAL ACTIVITY

<b>Description</b>	Adrenocorticotrophic Hormone (ACTH) (1-39), rat is a potent melanocortin 2 (MC2) receptor agonist.
<b>IC<sub>50</sub> &amp; Target</b>	Melanocortin 2 receptor <sup>[1]</sup>
<b>In Vitro</b>	ACTH 1-39 at concentrations of 100-400 nM has no toxic effect on neurons, while ACTH provides protection from excitotoxic neuronal death induced by glutamate (100 μM), NMDA (1 mM), AMPA (50 μM), and kainate (25 μM). ACTH at 400 nM provides substantial protection in each case. ACTH at either 200 or 400 nM protects neurons from quinolinic acid (25 μM). There is also protection by ACTH from cell death induced by 2 μM H <sub>2</sub> O <sub>2</sub> , which gives rise to reactive oxygen species (ROS), with significantly more protection at 400 nM ACTH compared to 200 nM. ACTH gives modest protection against rapid release of nitric oxide (NO) by NOC-12 but not slow release by NOC-18. ACTH (200 or 400 nM) protects neurons from cytotoxic effects of staurosporine (10-20 nM), a classic inducer of cell death via apoptosis. ACTH reduces cell death from 80% to 55% <sup>[1]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.
<b>In Vivo</b>	The icv injection of ACTH significantly reduces cumulative food intake over the observation period compared with the saline/IgG group. The injection of ACTH Ab into the PVN abolishes the anorexigenic effect of ACTH. Infusion icv of ACTH significantly decreases cumulative food intake in rats that receive α-MSH Ab into the PVN and ACTH icv, and food intake is as low as in the group treated with ACTH icv and IgG into the PVN. Injection of either ACTH Ab or α-MSH Ab into the PVN significantly increase cumulative food intake compared with IgG-treated animals; the combined application of both Ab's do not increase food intake further <sup>[2]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.

### PROTOCOL

<b>Animal Administration</b> <sup>[2]</sup>	Rats <sup>[2]</sup> Male Wistar rats (weight range 225-250 g at purchase) are used throughout the study. Animals receive a PVN application of ACTH Ab (2 μg/rat) or IgG (2 μg/rat); administration of either ACTH (1 nM/rat) or saline icv is performed 5 min later <sup>[2]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.
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## REFERENCES

- [1]. Lisak RP, et al. Melanocortin receptor agonist ACTH 1-39 protects rat forebrain neurons from apoptotic, excitotoxic and inflammation-related damage. *Exp Neurol*. 2015 Nov;273:161-7.
- [2]. Schulz C, et al. Endogenous ACTH, not only alpha-melanocyte-stimulating hormone, reduces food intake mediated by hypothalamic mechanisms. *Am J Physiol Endocrinol Metab*. 2010 Feb;298(2):E237-44.
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**Caution: Product has not been fully validated for medical applications. For research use only.**

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