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Aducanumab

Cat. No.:	HY-P9967
CAS No.:	1384260-65-4
Target:	Amyloid- β
Pathway:	Neuronal Signaling
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.

BIOLOGICAL ACTIVITY

Description	Aducanumab (BIIB037) is a human monoclonal antibody that selectively targets aggregated amyloid-beta ($A\beta$). Aducanumab shows brain penetration, and can be used for Alzheimer's disease (AD) research ^[1] .																
In Vivo	<p>Aducanumab (30 mg/kg, i.p., single dose) binds all morphological types of brain $A\beta$ plaques in 22-month-old Tg2576 transgenic mice, including diffuse $A\beta$ deposits and compact $A\beta$ plaques^[1].</p> <p>Aducanumab (0.3-30 mg/kg, i.p., weekly, 6 months) reduces soluble and insoluble $A\beta$ in a dose-dependent manner in 9.5- to 15.5-month-old Tg2576 transgenic mice^[1].</p> <p>Aducanumab (10 mg/kg, i.p., weekly, 6 months) restores intracellular calcium to control levels in 18-month-old Tg2576 mice^[2].</p> <p>Aducanumab (0.4-1 mg/mL, ICV, 20 min) leads to rapid decrease in amyloid burden, plaque clearance in Tg2576 mice^[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>9.5- to 15.5-month-old Tg2576 transgenic mice^[1]</td> </tr> <tr> <td>Dosage:</td> <td>0.3-30 mg/kg</td> </tr> <tr> <td>Administration:</td> <td>Intraperitoneal injection (i.p.), weekly, 6 months</td> </tr> <tr> <td>Result:</td> <td>Increased recruitment of Iba-1-positive microglia to $A\beta$ plaques.</td> </tr> </table> <table border="1"> <tr> <td>Animal Model:</td> <td>18-month-old Tg2576 mice^[2]</td> </tr> <tr> <td>Dosage:</td> <td>10 mg/kg</td> </tr> <tr> <td>Administration:</td> <td>Intraperitoneal injection (i.p.), weekly, 6 months</td> </tr> <tr> <td>Result:</td> <td>Restored neurite baseline calcium to control levels. Decreased the number of neurites with elevated levels of calcium after 2 weeks. Decreased the percentage of cell bodies with calcium overload. Restored the levels of VILIP and SERCA to control levels. Increased the cell numbers of NR1 and NR2A.</td> </tr> </table>	Animal Model:	9.5- to 15.5-month-old Tg2576 transgenic mice ^[1]	Dosage:	0.3-30 mg/kg	Administration:	Intraperitoneal injection (i.p.), weekly, 6 months	Result:	Increased recruitment of Iba-1-positive microglia to $A\beta$ plaques.	Animal Model:	18-month-old Tg2576 mice ^[2]	Dosage:	10 mg/kg	Administration:	Intraperitoneal injection (i.p.), weekly, 6 months	Result:	Restored neurite baseline calcium to control levels. Decreased the number of neurites with elevated levels of calcium after 2 weeks. Decreased the percentage of cell bodies with calcium overload. Restored the levels of VILIP and SERCA to control levels. Increased the cell numbers of NR1 and NR2A.
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Animal Model:	22-month-old transgenic Tg2576 mice ^[2]
Dosage:	0.4-1 mg/mL
Administration:	Intracerebroventricular injection (ICV), 20 min
Result:	Decreased the number of amyloid plaque. Decreased the size of the remaining individual plaques. Reduced amyloid plaque burden.

REFERENCES

- [1]. Sevigny J, et al. The antibody aducanumab reduces A β plaques in Alzheimer's disease. *Nature*. 2016 Sep 1;537(7618):50-6.
- [2]. Kastanenka KV, et al. Immunotherapy with Aducanumab Restores Calcium Homeostasis in Tg2576 Mice. *J Neurosci*. 2016 Dec 14;36(50):12549-12558.

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

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