

UC Irvine

UC Irvine Previously Published Works

Title

Angiotensin II(Ang II) type I (AT(1)) receptor-mediated neuronal migration in mouse olfactory epithelium explant cultures.

Permalink

<https://escholarship.org/uc/item/1028h6cd>

Journal

FASEB JOURNAL, 13(4)

ISSN

0892-6638

Authors

Huang, XC
Calof, AL
Summers, C

Publication Date

1999-03-12

License

[CC BY 4.0](#)

Peer reviewed

148.10

ANGIOTENSIN II (Ang II) TYPE 1 (AT₁) RECEPTOR-MEDIATED NEURONAL MIGRATION IN MOUSE OLFACTORY EPITHELIUM EXPLANT CULTURES.

X-C. Huang, A.L. Calof and C. Summers. Dept. of Physiology and Brain Institute, Univ. of Florida and Dept. of Anatomy and Neurobiology, Univ. of California, Irvine.

It is known that both AT₁ and Ang II type 2 (AT₂) receptors participate in the processes of growth, proliferation and differentiation of several non-neuronal cell types. However, it is not clear whether Ang II receptor subtypes have similar roles in the nervous system. We have studied the effects of Ang II on neuronal migration, proliferation and differentiation in a model culture system, the olfactory epithelium (OE) of the mouse. The presence of both AT₁ and AT₂ receptors in embryonic mouse OE and OE explant cultures was demonstrated by RT-PCR and receptor binding assays. Addition of Ang II (10-100 nM; 15-48 hr.) to the culture medium immediately following plating caused time- and concentration-dependent increases in the total number of neurons migrating from OE explants. This effect of Ang II was abolished by the AT₁ receptor antagonist losartan (1 μ M), but not by the AT₂ receptor blocker PD123,319 (1 μ M). The effect of Ang II did not appear to be due to changes in progenitor cell proliferation or neuronal differentiation, since neither the percentage of cells incorporating [³H]-thymidine, nor the number of progenitor cells that differentiated into post mitotic neurons (assessed by acquisition of immunoreactivity for the neural cell adhesion molecule NCAM, a marker of neuronal differentiation) was increased in Ang II-treated cultures. Together, the data suggest that the AT₁ receptor-mediated effect of Ang II on the total number of migratory neurons is due to an enhancement of neuronal migration, rather than an effect on proliferation or differentiation. This effect of Ang II does not involve AT₂ receptors. (Supported by NS-19441).