Title
ADHESION, ANTIADHESION, AND MIGRATION OF OLFACTORY NEURONS AND NEURONAL PRECURSORS ARE INDEPENDENTLY REGULATED BY DISTINCT MOLECULAR DOMAINS OF LAMININ

Permalink
https://escholarship.org/uc/item/25t1d860

Authors
CALOF, AL
YURCHENCO, PD
OREAR, JJ
et al.

Publication Date
1992-09-01

License
CC BY 4.0

Peer reviewed
Neuronal precursors and immature neurons of the mouse olfactory epithelium (OE) are motile in vivo, and can be stimulated to migrate and are guided in vitro by the ECM protein laminin (LN) and its homologue merosin (MN). LN and MN are also anti-adhesive, i.e. they cause OE neuronal cells to adhere weakly to substrata that would otherwise be strongly adhesive (Calof and Lander, 1991). Investigations into the domains of laminin responsible for its effects on OE cells have revealed the following: The anti-adhesive activity of LN is highly heat stable and maps to the E1 fragment of the molecule. Although integrin α1β1 is a cell-surface receptor known to interact with this region of LN, function-blocking antibodies directed against this integrin do not inhibit anti-adhesion. The migration-promoting activity of LN is distinct from its anti-adhesive activity, and is not the result of adhesion-altering effects of LN. Migration-promoting activity is heat-labile, maps to the E8 fragment of LN, and can be completely blocked by a monoclonal antibody directed against integrin subunit α6. Migration promoted by MN, however, is only partially blocked by this antibody. Surprisingly, although LN is not detectably adhesive for OE neuronal cells, some of its domains are. E8 is weakly adhesive, and a recombinant G-domain (rG) is strongly adhesive. Adhesion to rG was not dependent on α6-containing integrins, nor could it be blocked by a peptide contained in rG that is thought to represent the binding site of integrin α3β1. Adhesion to rG was blocked, however, by low concentrations of heparin (<20 μg/ml) and by antibodies directed against LN fragment E3 (which is contained in rG). These data suggest that neuronal adhesion, anti-adhesion and migration can be independently regulated by distinct domains of LN and distinct receptors.