Cell Communication and Signaling BioMed Central

Meeting abstract

Open Access

Analysis of matrix-dependent cell migration by a barrier migration assay

S Kroening*, C Streiff and M Goppelt-Struebe

Address: Department of Nephrology and Hypertension, Medical Clinic 4, University Hospital of Erlangen-Nürnberg, Erlangen, Germany

* Corresponding author

from 12th Joint Meeting of the Signal Transduction Society (STS). Signal Transduction: Receptors, Mediators and Genes
Weimar, Germany. 29–31 October 2008

Published: 26 February 2009

Cell Communication and Signaling 2009, **7**(Suppl 1):A100 doi:10.1186/1478-811X-7-S1-A100

This abstract is available from: http://www.biosignaling.com/content/7/S1/A100

© 2009 Kroening et al; licensee BioMed Central Ltd.

Background

Cell migration plays a pivotal role in many biological processes. During embryogenesis, the orchestrated movement of cells is required for organ development. Wound healing depends on the migration of fibroblasts and endothelial cells. Contrariwise, misregulation of migratory processes may lead to serious consequences including metastasis and mental retardation.

A variety of cell migration assays are applied to investigate the molecular mechanisms that contribute to migration. An important objective of cell migration assays is to approach the situation in the body in order to get physiologically meaningful information. In vivo, cells are embedded in an extracellular matrix (ECM). Matrix proteins are linked to the cellular cytoskeleton via integrins, which convert information about the substrate to the inside of the cell thereby providing essential signals for cell migration.

Methods and results

The wound healing or scratch assay classically investigates migration of adherent cells. Thereby, scratching a confluent cell layer with a pipet tip creates a cell-free gap. The subsequent migration of cells into the newly created gap is analyzed. However, the scratch leads to cell injury, which may affect cell migration. And, most importantly, protein coating is scratched off implicating that cells migrate into the gap on an unphysiological surface such as glass or plastic.

To overcome these inherent problems of the scratch assay, we developed a barrier cell migration assay. After substrate coating with a matrix protein, a rigid barrier is carefully placed in the dish. Cells are seeded around the barrier to form a monolayer. Removal of the barrier generates a cell-free gap, and the subsequent cell migration into the gap is analyzed. In contrast to the scratch assay, surface coating is preserved in the barrier assay.

To show the effect of ECM proteins on cell migration we analyzed the migration of human tubular epithelial cells (HKC-8) on coverslips, which were either uncoated or coated with collagen IV and fibronectin. Collagen IV constitutes the main ECM protein of the renal basal membrane, whereas fibronectin accumulates during tubulointerstitial fibrosis. Coating with fibronectin increased migration velocity in comparison to uncoated glass coverslips, whereas collagen IV exerted an inhibitory effect. To exemplify the role of cytokines we analyzed migration of HKC-8 cells on different substrates upon treatment with transforming growth factor beta (TGF-beta). TGF-beta enhanced cell migration on uncoated glass and on collagen IV. However, there was no increase in cell motility on fibronectin-coated coverslips.

Conclusion

The novel barrier cell migration assay offers the possibility to analyze cell migration on defined ECM proteins, and thus represents a simple device to investigate the role of cell-matrix interactions in migration of adherent cells under more physiological conditions.