



LAMININ IS A KEY COMPONENT OF THE CANCER STEM CELL NICHE, SUSTAINING CELL GROWTH AND GUIDING TUMOR ANGIOGENESIS, INVASION, AND METASTASIS

Laminins are one of the major glycoprotein families present at the basement membrane (BM) of the extracellular matrix (ECM) which underlie most cells in the body and separate epithelial and endothelial cells from connective tissues. Laminins play a vital role in the regulation of normal cellular processes, such as adhesion, migration, proliferation, and differentiation. As a major BM component of the stem cell niche, laminins affect stem cell behavior by maintaining the self-renewal capacity or guide differentiation into a variety of specialized cell types.

Laminins are also heavily involved in the development of early-stage cancers, tumor progression, and invasion. Laminins have been shown to sustain the growth of cancer stem cells and to regulate key cellular events for tumor angiogenesis, cell invasion and metastasis development, including the regulation of epithelial-mesenchymal transition and basement membrane remodeling.

The accumulating evidence in this emerging research area suggests that laminins represent potential therapeutic targets against cancer stem cells, and could potentially be used as predictive and prognostic markers.

FEATURES AND SPECIFICATIONS:

- **Defined and animal origin-free (primary level) substrates**
- **Biologically relevant culture methods**
- **Laminin is a key component of the cancer stem cell (CSC) niche**
- **Laminin sustain the growth of CSC and regulate key cellular events**
- **Potent adhesive and migratory substrate for many cancer cell types**



Direct link to laminin information online



LN511 PROMOTES SELF-RENEWAL OF BREAST CANCER STEM CELLS AND IS A POTENT ADHESIVE AND MIGRATORY SUBSTRATE FOR METASTATIC BREAST TUMOR CELLS IN VITRO

The human recombinant laminin cell culture substrate Biolaminin 511 LN (LN511) is a critical niche component for breast cancer stem cells (CSCs). Breast CSCs produce a LN511 matrix that functions as a ligand for the $\alpha 6 \beta 1$ integrin, promoting self-renewal and tumor initiation but also activation of the Hippo transducer TAZ. TAZ regulates transcription of the laminin $\alpha 5$ subunit and the formation of the LN511 matrix, establishing a positive feedback loop between TAZ and LN511 that contributes to stemness in breast cancer (Chang et al., 2015). LN511 also is a potent adhesive and migratory substrate for metastatic breast tumor cells in vitro and its expression correlates with tumor grade and metastatic potential in vivo. The migration and invasion responses of metastatic breast tumor cells have been shown to be mediated primarily via the $\alpha 3 \beta 1$ integrin cellular receptor (Kusuma et al., 2011).

LN521 PROVIDES A GOOD SUPPORT FOR THE CULTURE OF VARIOUS NEURAL TUMOR CELLS

Biolaminin 521 (LN521) has been shown to provide good support for the culture of various neural cancer cells, such as neuroblastoma, glioblastoma and medulloblastoma cells. In a publication by Ma et al., the authors showed that alpha-4 and alpha-5 laminins have specific effects in promoting the stemness of glioma cells, both at gene and protein expression level. When cultured in a 3D context, U251 glioblastoma cells showed enhanced clonogenicity on Biolaminin 521, 511, 421 and 411, and show a substantial upregulation of integrin 64 (Ma et al., 2016). Gene expression analysis of different neuroblastoma cell lines show high gene expression of alpha-5 and alpha-4, beta-1 and beta-2 laminin chains, indicating that these cells produce laminin 521, 511, 411 and 421. Indeed, LN521 work well for culture of neuroblastoma cell lines (unpublished data). Preliminary data suggest that LN521 even support the culture of medulloblastoma cells which is otherwise difficult to maintain in vitro.

REFERENCES

A laminin 511 matrix is regulated by TAZ and functions as the ligand for the $\alpha 6 \beta 1$ integrin to sustain breast cancer stem cells. Chang C., Lal Goel H., Gao H., Pursell B., Shultz L.D., Greiner D.L., Ingerpuu S., Patarroyo M., Cao S., Lim E., Mao J., Kulju McKee K., Yurchenco P.D., Mercurio A.M. *Research communication*, 2015

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