

affinity for both U-87 MG and neoplastic tissue homogenate, demonstrating higher internalization of [131I]I-DEDEYFELV in human glioblastoma cells. Nevertheless, it is crucial to emphasize that additional in vivo investigations are necessary.

**Keywords:** EGFr-targeting peptide, Glioblastoma, U-87 MG cells, [131I]I-labeled-peptide.

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#### SYNTHESIS AND EVALUATION OF BIOACTIVE PEPTIDES FROM LAMININ-111 IN TRIPLE-NEGATIVE BREAST CANCER CELLS

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**Introduction/Justification:** Breast cancer stands as the neoplasm group with the highest incidence rate among women worldwide. Laminin-111 a constituent of the tissue basement membrane, is implicated in the development of breast tumors. Biologically active peptides of laminin-111 such as YIGSR and IKVAV, play crucial roles in tumor growth, metastasis, protease secretion, and angiogenesis. These peptides exert significant influence on various aspects of cancer progression, emphasizing their potential as key targets for therapeutic intervention. **Objectives:** The aim of the study was to synthesize the bioactive peptides of laminin-111 (YIKVAV and YIGSR) and assess their interactions in triple-negative breast cancer cells. **Materials and Methods:** The YIKVAV and YIGSR peptides were synthesized through solid-phase peptide synthesis using the Fmoc/tBut strategy. Subsequently, the peptides underwent a characterization and purification process employing high performance liquid chromatography (HPLC) and mass spectrometry. The MDA-MB-231 breast tumor cell line was cultured in supplemented RPMI-1640 medium at 37°C and 5% CO<sub>2</sub> until reaching 90% confluence. The growth curve of the MDA-MB-231 cell line was conducted in sextuplicate over a 7-day period, with cell counts performed on days 1, 3, 5, and 7. To assess the effect of peptides on cell proliferation, cells were seeded at a concentration of  $2 \times 10^4$  in 6-well plates, with the inclusion of both YIKVAV and YIGSR peptides at 50  $\mu$ M ( $n = 6$ ). Cell viability in the presence of YIKVAV and YIGSR was determined using the MTT assay. For this analysis, cells were plated at a concentration of  $2 \times 10^4$ , with peptide concentration of 50  $\mu$ M. Spectrophotometric analyses were realized after 24 h and 7 days of incubation at 595 nm. **Results:** The YIKVAV and YIGSR peptides were efficiently synthesized, yielding approximately 80% for both. Chromatographic analyzes conducted by HPLC and mass spectrometry confirmed the efficiency of the entire synthesis, cleavage, and characterization process of the peptides by the presence of only a single peak corresponding to the synthesized peptides. The growth curve profile determination of MDA-MB-231 cell line revealed exponential growth

between days 5 and 7 of cell culture. The results indicate that the YIGSR peptide significantly inhibited cell growth by approximately 45%, whereas the YIKVAV fragment promoted cell growth by approximately 38% and ( $p < 0.0001$ ) on the seventh day of cell culture. Regarding the MTT analysis after 24 h, no significant differences were observed between the control and treated groups for both fragments, suggesting that the peptides exhibited no toxicity at concentration of 50  $\mu$ M. Additionally, on the 6th day, a reduction in tumor cell viability was observed for the YIGSR fragment, while an increase in viability was noted for YIKVAV ( $p < 0.0001$ ). **Conclusion:** The YIKVAV and YIGSR peptides were effectively synthesized, characterized, and purified. The YIKVAV peptide, at a concentration of 50  $\mu$ M, promoted cell growth, while the YIGSR peptide significantly hindered the growth of the MDA-MB-231 cell line. This observation is consistent with the findings of cell viability assays conducted using MTT. This study holds significance for enhancing our comprehension of peptide actions in their interaction with triple-negative breast cancer cells, with the ultimate aim of proposing more effective targets for treatment.

**Keywords:** Breast cancer, Laminin-111-peptides, MDA-MB-231 cells.

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#### ASSESSMENT OF IN VITRO INTERACTIONS BETWEEN RADIOLABELED EGFR-TARGETING PEPTIDE INHIBITORS AND GLIOBLASTOMA CELLS

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**Introduction/Justification:** Peptides are implicated in various physiological responses and hold significant potential as targeting molecules, especially in cancer diagnosis or treatment. Radiolabeled peptides have been investigated for their potential as theranostic agents, holding considerable promise for precisely targeting tumorigenic cells. Previous studies indicate that biologically active peptides exhibit a high affinity for the Epidermal Growth Factor receptor (EGFr), which is overexpressed in various tumor cells, including glioblastoma, the most prevalent and aggressive malignant brain tumor. **Objectives:** To evaluate the in vitro interactions involving two radiolabeled peptide inhibitors targeting the EGFr overexpressed in glioblastoma cells. **Materials and Methods:** Two EGFr-targeting peptide inhibitors, anti-EGFr-LP and anti-EGFr-LG, were radiolabeled with [131I]NaI (11.1–14.8 MBq) using