targeted therapies that can disrupt these oncogenic pathways. Peptide-based drugs represent a promising avenue for precision oncology, offering high specificity, low toxicity, and potential to inhibit key molecular drivers of cancer. This study investigates the YHWYGYTPQNVT peptide, designed to interact with EGFR, and evaluates its effect on proliferation and migration of HeLa cells, a widely used model of cervical cancer. **Objectives:** This research aims to determine whether the YHWYGYTPQNVT peptide can effectively suppress EGFR-mediated growth signaling in HeLa cells, by analyzing cell proliferation, metabolic activity, and migration, aiming at establishing its potential as a therapeutic alternative to traditional cancer treatments. **Materials and Methods:** The YHWYGYTPQNVT peptide was synthesized using solid-phase peptide synthesis, purified via HPLC, and confirmed by mass spectrometry. HeLa cells were cultured in DMEM + 10% fetal bovine serum and incubated at 37°C with 5% CO_2. To establish a baseline proliferation rate, HeLa cells were plated at 5×104 cells in 6-well plates, and the growth curve was performed in sextuplicate over a 5-days period, with cell counts conducted on days 1, 3 and 5. For the experimental group, cells were treated with YHWYGYTPQNVT (80 μmol/mL). Statistical analysis was conducted using GraphPad Prism, with significance set at $p \leq 0.05$. **Results:** The YHWYGYTPQNVT peptide was synthesized efficiently with yield of approximately 45%. Chromatographic analyzes obtained by HPLC and mass spectrometry confirmed that the entire synthesis, cleavage, and purification process of peptides were performed efficiently. Control group displayed an aggressive proliferation rate with an exponential growth, reaching $\sim 96.8 \times 10^4$ cells, consistent with the known oncogenic potential of HeLa cells. In contrast, the peptide-treated group showed a significant reduction in proliferation, with final cell counts averaging 61.5×10^4 cells - corresponding to a 25.5% decrease compared to untreated cells. **Conclusion:** Our findings highlight YHWYGYTPQNVT as a promising EGFR-targeting agent capable of reducing cervical cancer cell proliferation. By directly interfering with EGFR-driven oncogenic pathways, this approach could lay the groundwork for a new class of peptide-based therapeutics in oncology. Further in vivo validation and molecular pathway analysis are necessary to determine its potential clinical application in patients with EGFR-overexpressing cervical tumors.

Keywords: Anti-EGFr-peptide, Cell proliferation, Cervical cancer, HeLa cells.

https://doi.org/10.1016/j.htct.2025.103785

COMPARISON BETWEEN 18F-PSMA AND 18F-FDG RADIOTRACERS FOR PET/CT IN THE EVALUATION OF PATIENTS WITH METASTATIC MELANOMA

Thiago Soare Rocha Alves, Diego Mendanha, Ellen Lima, Natália Tobar, Juliana Souza, Ligia Macedo, Allan Oliveira Santos, Mariana Cunha Lopes Lima, Elba Etchebehere, Carmen Silva Passos Lima

Universidade Estadual de Campinas (UNICAMP), Campinas, SP, Brazil

A B S T R A C T

Introduction/Justification: PET/CT has emerged in the last two decades as a dominant imaging modality used for staging, monitoring response and surveillance of melanoma using 18F-FDG as radiotracer. Recent publications have demonstrated the possibility of use of 18F-PSMA PET/CT as an additional resource to the evaluation of melanoma, due to the expression of Prostate-Specific Membrane Antigen protein (PSMA) in these cancer cells and because anti-PSMA antibodies react with malignant melanoma neo vasculature. **Objectives:** Would 18F-PSMA PET/CT have the potential role of a novel diagnostic imaging technique in melanoma cases? **Materials and Methods:** Eleven participants with diagnoses of metastatic melanoma underwent 18F-FDG PET/CT and 18F-PSMA PET/CT (24-hours interval), and the lesions uptakes were evaluated with both radiotracers. The results were grouped in three categories: A - greater expression of 18F-PSMA compared to 18F-FDG; B – equivalent uptake between the radiotracers; and C - greater expression of 18F-FDG compared to 18F-PSMA. **Results:** 18,1% of participants were in category A, 54,5% in category B and 27,2% in category C. The lesions with greater 18F-PSMA uptake compared to 18F-FDG were mainly in the brain, lungs, adrenals, and scattered throughout the chest. Furthermore, one subjects presented only 18F-PSMA uptake in brain metastasis, showing the importance of this method to the clinical follow-up of these patients. Our findings align with the Chang et al.'s, who demonstrated in vitro expression of PSMA in the neovasculature of melanoma lesion and with Snow et al.'s who observed PSMA positivity in endothelial cells of capillaries within stage III/IV melanoma metastases. **Conclusion:** Therefore, apart from the use of 18F-PSMA PET/CT in staging prostate cancer patients, this method shows a great potential in the evaluating of metastatic melanoma, still needing further and longer studies to confirm these advantages.

Keywords: 18F-FDG PET/CT, 18F-PSMA PET/CT, Melanoma.

https://doi.org/10.1016/j.htct.2025.103786

EVALUATION OF THE AFFINITY OF RADIOLABELED PEPTIDE [^{131}I]I-DEDEYFELV FOR EGFR-OVEREXPRESSING RECEPTORS IN ADULT-TYPE DIFFUSE GLIOMAS

Julia Calviello Giordano [a], Fernanda Paiva Augusto Rodrigues [a], Danielle Vieira Sobral [a], Leonardo Lima Fuscaldi [a], Fabio Luiz Navarro Marques [b], Marycel Figols de Barboza [c], João Luiz vitorino Araújo [a], Luciana Malavolta [a]

[a] *Faculdade de Ciências Médicas da Santa Casa de São Paulo, São Paulo, SP, Brazil*

[b] *Universidade de São Paulo (USP), São Paulo, SP, Brazil*
[c] *Hospital Albert Einstein, São Paulo, SP, Brazil*

A B S T R A C T

Introduction/Justification: Cancer remains one of the leading causes of death worldwide. Among the various tumor sites, the central nervous system is particularly significant, with gliomas accounting for the majority of primary brain tumors. In gliomas, alterations in the tyrosine kinase pathway lead to the overexpression of the Epidermal Growth Factor receptor (EGFr). Over the past decades, radiolabeled peptides with high affinity for EGFr have emerged as promising molecular targets with potential applications in both diagnosis and therapy. **Objectives:** This study aimed to evaluate the affinity of the peptide DEDEYFELV, radiolabeled with iodine-131 (^{131}I), for EGFr-overexpressing receptors in adult-type diffuse gliomas using tumor tissue samples. **Materials and Methods:** The peptide was synthesized using solid-phase peptide synthesis following the Fmoc/tBu strategy. Upon completion of the synthesis, the peptide was characterized and purified via high-performance liquid chromatography (HPLC). DEDEYFELV (20 nmol) was radiolabeled with [^{131}I]NaI (18.5 MBq) using the chloramine-T method. The radiochemical yield of [^{131}I]I-DEDEYFELV was determined via chromatography on Whatman 3MM strips using a 95% MeOH / 5% H_2O eluent. Binding studies of [^{131}I]I-DEDEYFELV with neoplastic tissue homogenates were conducted at 1 and 4 h of incubation and quantified using an automatic gamma counter. Tumor tissue homogenates were obtained from surgical resections performed by a designated neurosurgeon, following informed consent. Gliomas were confirmed through pathological analysis, and tumor samples were preserved at -80°C. All human protocols adhered to local ethical guidelines (Protocol number CEP – FCMSCSP: CAAE 79336124.7.0000.5479). Statistical analysis was conducted using ANOVA or Student's t-test. **Results:** The peptide DEDEYFELV was successfully synthesized with a yield of approximately 92%. Mass spectrometry and HPLC analyses confirmed efficient synthesis, cleavage, and purification, as evidenced by a single peak and a molecular mass corresponding to the expected peptide. Radiolabeling was achieved with a radiochemical yield exceeding 95%. Binding studies of [^{131}I]I-DEDEYFELV with neoplastic tissue homogenates showed values of 3.25 ± 0.31% for high-grade tumors, 2.62 ± 0.34% for low-grade tumors, and 1.61 ± 0.25% for tumors of unknown grade at 1 h of incubation (n = 5). At 4 h, the binding values increased to 6.45 ± 0.66% for high-grade tumors, 10.27 ± 1.58% for low-grade tumors, and 7.74 ± 1.21% for tumors of unknown grade (n = 5). **Conclusion:** These findings demonstrate that the radiolabeled peptide [^{131}I]I-DEDEYFELV exhibits specific binding to EGFr-overexpressing tumor tissues, with an increasing affinity over time. The higher binding observed at 4 h suggests favorable interaction dynamics, particularly in low-grade gliomas. These results highlight the potential of [^{131}I]I-DEDEYFELV as a theranostic agent for EGFr-targeted imaging and therapy, warranting further investigations into its in vivo stability and clinical applicability.

Keywords: EFFr-targeting peptide, Glioma, Tumor tissue, [^{131}I] I-labeled peptide.

https://doi.org/10.1016/j.htct.2025.103787

EXPRESSÃO DE GENES ASSOCIADOS COM RESISTÊNCIA À CISPLATINA EM LINHAGEM DE CÉLULAS DE CÂNCER DE CAVIDADE ORAL: PASSOS INICIAIS PARA O DESENVOLVIMENTO DE UM PAINEL MOLECULAR

Bianca Piovesan Melchiori Peruzza, Carmen Silvia Passos Lima, Juliana Carron, Gustavo Jacob Lourenço

Universidade Estadual de Campinas (UNICAMP), Campinas, SP, Brasil

R E S U M O

Introdução/Justificativa: A cisplatina (CDDP) é um dos principais agentes quimioterápicos utilizados no tratamento do carcinoma de células escamosas de cavidade oral (CCECO). No entanto, a resistência ao tratamento representa um desafio clínico, reduzindo a eficácia terapêutica e impactando negativamente o prognóstico dos pacientes. A quimiorresistência à CDDP envolve mecanismos moleculares complexos, incluindo alterações na expressão de genes relacionados ao reparo do DNA e ao metabolismo do fármaco. Nesse contexto, genes como AKR1C1, CCND1, CCND3, ERCC1 e SLC31A1 têm sido implicados em processos biológicos associados à resistência, como detoxificação de drogas, regulação do ciclo celular e transporte de íons. **Objetivos:** O objetivo deste estudo foi avaliar a expressão desses genes em células do CCECO sensíveis e resistentes à CDDP, buscando identificar potenciais biomarcadores de resistência e novas abordagens para otimizar a terapia em pacientes com o CCECO. **Materiais e Métodos:** A linhagem celular SCC-25 (câncer de língua, CRL-1628, ATCC) sensível à CDDP foi cultivada seguindo protocolo padrão. A resistência celular foi induzida com 10,64 μM de CDDP, conforme protocolo previamente estabelecido. Os modelos experimentais utilizados foram: SCC-25 e SCC-25 resistente à CDDP (SCC-25-R). O cDNA de cada amostra foi amplificado por qPCR para avaliar a expressão dos genes AKR1C1, CCND1, CCND3, ERCC1 e SLC31A1 utilizando iniciadores específicos e reagentes do kit com o corante SYBR green no equipamento QuantStudio 3, seguindo as recomendações do fabricante. O gene GAPDH foi utilizado como controle endógeno. A comparação entre os grupos foi realizada por meio do teste t e os resultados foram expressos como fold change (FC). Valores de $p < 0,05$ foram considerados significativos. **Resultados:** A expressão dos genes AKR1C1 (FC: 74,19, $p = 0,001$), CCND1 (FC: 1,91, $p = 0,003$), CCND3 (FC: 1016,29, $p = 0,009$) e ERCC1 (FC: 14,90, $p = 0,004$) foi maior nas células SCC-25-R em comparação com as células sensíveis à CDDP. Em contraste, o gene SLC31A1 apresentou uma expressão reduzida na linhagem SCC-25-R (FC: 0,56, $p = 0,005$) em relação às células sensíveis ao tratamento. **Conclusão:** Nossos