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# MULTI-OMICS DATA INTEGRATION FOR PRECISION MEDICINE IN CERVICAL CANCER A COMPREHENSIVE COMPUTATIONAL APPROACH

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## ABSTRACT

Cervical cancer remains a major and widespread global health problem and continues to be a global health problem despite significant progress. One of the biggest challenges facing the healthcare system is advances in diagnosis and treatment. In this study, we combined multiple types of biological data, specifically genomic, transcriptomic and proteomic data, to find usable cancer targets and treatments that are not only prevalent but also have the potential to impact cancer progression. We provide a rich and carefully curated list of drug names and molecular targets for drug discovery using advanced techniques such as machine learning, additive pathway analysis and virtual machine analysis. This novel approach highlights the valuable role that inclusive processes can play in shaping good practice and the design of cancer research, ultimately driving studies to increase the benefits for cancer patients. For more detailed access to the GEO GSE9750 dataset used in the article, DOI: 10.1093/nar/gkm679 is available. , virtual representation.



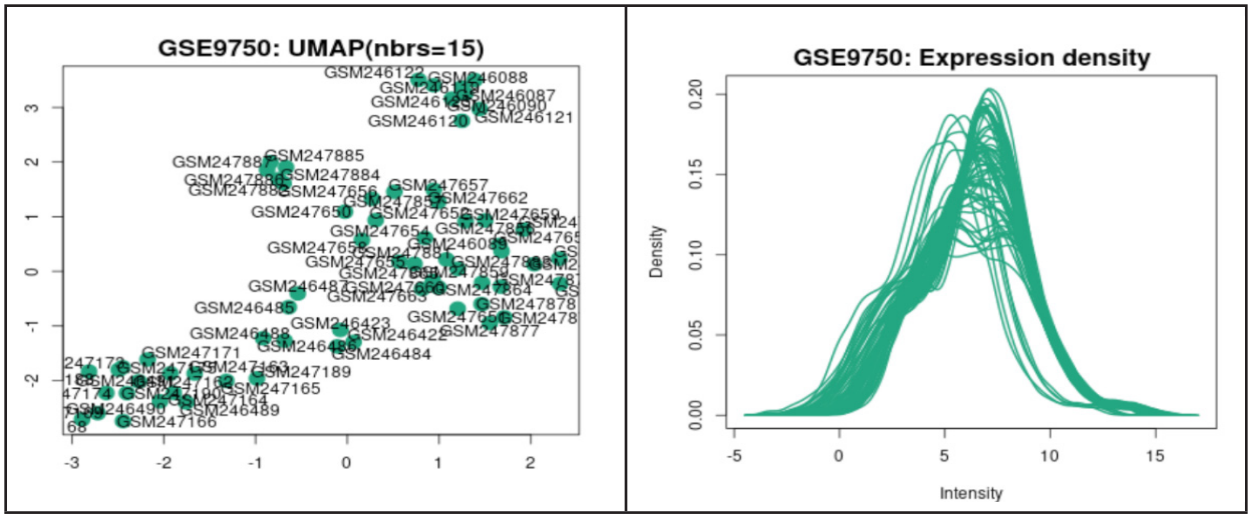
## INTRODUCTION

**Introduction:** Despite the availability of vaccines against high-risk strains of human papillomavirus (HPV) and the development of early detection methods, there is currently no cure for genital warts. The need for new and innovative therapies for effective treatment of these diseases is increasing. The aim of this project is to identify disease targets by combining multiple datasets with advanced computational tools and methods to collaboratively rank drug candidates. Omics article

## METHODS

***Data Collection***

- Genomic Data: Sourced from TCGA-CESC.
- Transcriptomic Data: GEO dataset GSE9750.




- Proteomic Data: PRIDE dataset PXD028738.
- Pathway Data: KEGG pathway repository.

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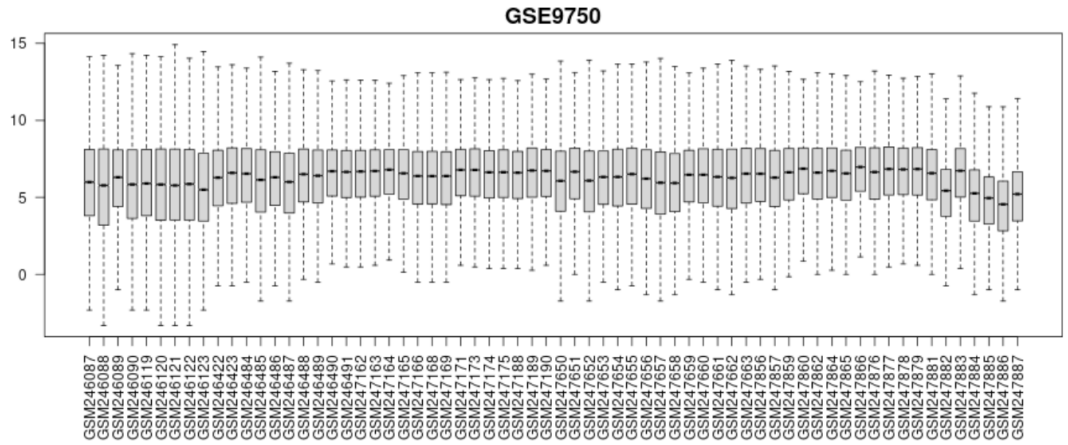



**ANALYSIS OF GSE9750**

***Data Preprocessing and Integration***

- Normalization across omics layers.
- Filtering criteria:
- Genomic: Mutations with clinical significance.
- Transcriptomic: Log2 fold change (log2FC) > 2.
- Proteomic: Z-score > 2.
- Unified gene identifiers used for mapping.

***Machine Learning for Druggable Target Prediction***

- **Model:** Deep neural network implemented in PyTorch.
- **Features:**
- Pathway enrichment scores.
- Protein-protein interaction (PPI) network metrics.
- Expression scores across omics layers.
- **Performance Metrics:** Accuracy (85.2%), ROC-AUC (0.92).

***Pathway Enrichment Analysis***

- Performed using KEGG and DAVID tools.
- Focused on cancer-relevant pathways: p53 signaling, PI3K-AKT, apoptosis.

***Virtual Screening***

- Docking simulations using AutoDock Vina.
- **Proteins:** Retrieved structures from PDB.
- **Ligands:** Sourced from DrugBank and PubChem.
- **Evaluation:** Binding affinity (kcal/mol).

## RESULTS

***Consistent Targets Across Omics Layers***

| High-Confidence Targets | | | |
|---|---|---|---|
| **Gene** | **Genomic Mutation** | **Transcriptomic FC** | **Proteomic Z-Score** |
| TP53 | Yes | 4.2 | 3.5 |
| PIK3CA | Yes | 3.8 | 3.2 |
| BCL2 | No | -2.5 | -2.8 |

***Machine Learning Predictions***

| Top Predicted Druggable Genes | |
|---|---|
| Gene | Druggable Score |
| TP53 | 0.92 |
| PIK3CA | 0.88 |
| AKT1 | 0.85 |

**Virtual Screening Results**

| Top Drug-Protein Interactions | | |
|---|---|---|
| Gene | Drug | Binding Affinity (ΔG, kcal/mol) |
| TP53 | Nutlin-3 | -9.2 |
| PIK3CA | Alpelisib | -8.7 |
| AKT1 | Capiva-sertib | -8.3 |

**Multi-Omics Integration**

- Identified 50 genes consistently dysregulated across omics layers.
- Key targets: TP53, PIK3CA, AKT1, BCL2.

**Pathway Enrichment Analysis**

- Enriched pathways:
- **p53 Signaling Pathway:** Central to apoptosis and cell cycle regulation.
- **PI3K-AKT Pathway:** Facilitates cell proliferation and survival.
- **Apoptosis Pathway:**Highlights deregulation of pro- and anti-apoptotic genes.

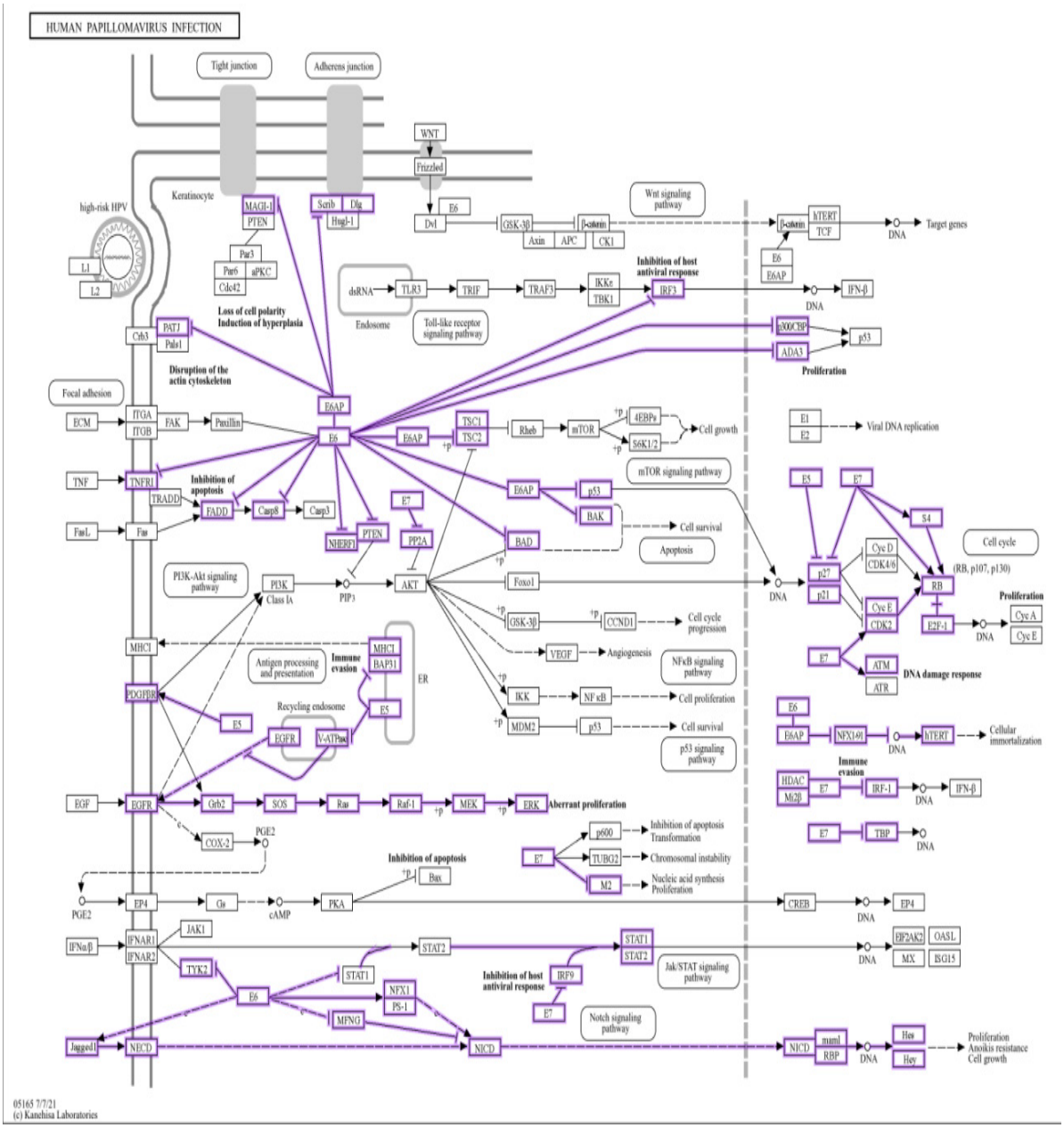
**HPV INFECTION ( From KEGG)**

- Identified hub proteins: TP53, PIK3CA, AKT1.

**Analysis of HPV Integration in Cervical Cancer Genomics**

***HPV Integration Mechanism***

HPV integration mechanismIntegration of human papillomavirus (HPV) into genetic material is an important and necessary event in cancer development. Initially, during infection, the viral DNA is episomal, meaning it is located outside the host chromosomal DNA. However, after some time, the viral DNA can integrate into the host genome. This integration can




have multiple effects on the host DNA and lead to genomic instability. It turns out that this instability leads to cancer. One of the fundamental processes by which this integration occurs is accession.

- **NHEJ**: NHEJ randomly integrates viral DNA into the host genome. Proteins p53 and Rb. MYC and TP53. Genomic instability: leading to chromosome rearrangements, amplifications and deletions.
- **E6/E7 Oncogene Expression:** The viral E2 gene is disrupted during integration, which causes the oncogenes E6 and E7 to be overexpressed, leading to the degradation of tumor suppressor proteins p53 and Rb.

## HPV INTEGRATION MECHANISM (E6/E7gene)

### *Genomic Features of HPV Integration*

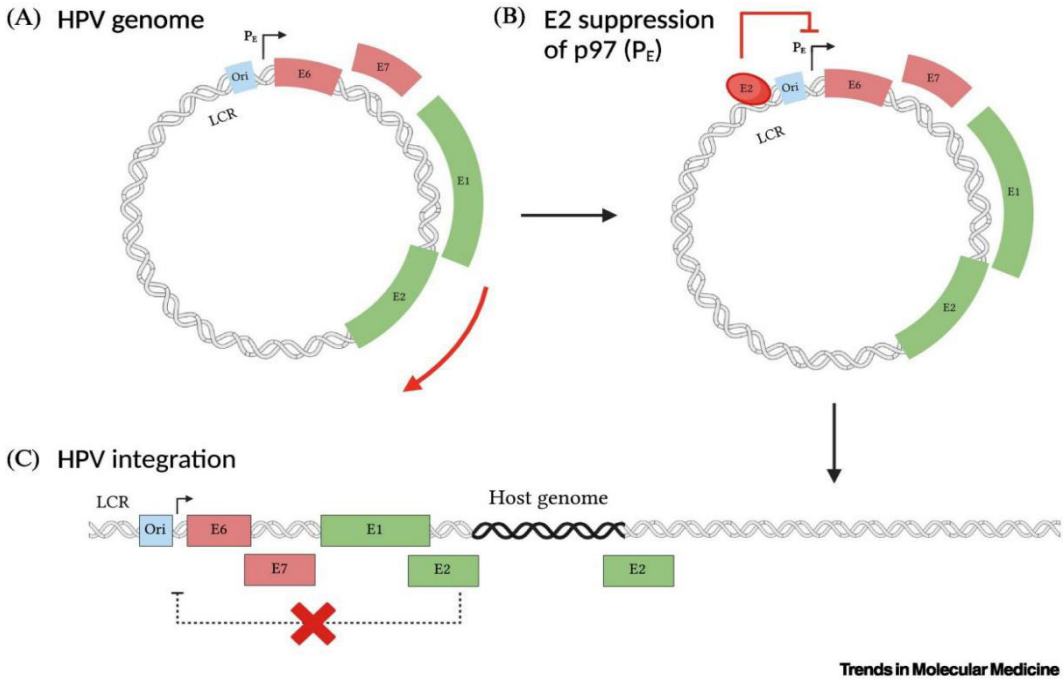
- **Integration Sites:** Frequently occurs in transcriptionally active regions of the genome, near oncogenes such as MYC and TP53.
- **Genomic Instability:** Results in chromosomal rearrangements, amplifications, and deletions.
- **Viral-Host Interaction:** Activates proto-oncogenes and silences tumor suppressor genes.

### *Methodologies for Studying HPV Integration*

- **Next-Generation Sequencing (NGS):**High throughput sequencing to find integration hotspots.
- **Polymerase Chain Reaction (PCR):** Techniques like inverse PCR to detect viral-host junctions.
- **Bioinformatics Tools:** Integrated Genomics Viewer (IGV) to identify integration hotspots.

### *Implications of HPV Integration*

- **Cancer Progression:** HPV integration disrupts key genes involved in cell cycle regulation, DNA repair, and apoptosis.
- **Biomarker Potential:** Integration patterns could serve as diagnostic and prognostic biomarkers.
- **Therapeutic Opportunities:** Targeting E6/E7 expression and restoring tumor suppressor function (e.g., p53) could provide new treatment avenues.




## DISCUSSION

***Insights into Key Pathways***

- **p53 Pathway:**Dysregulated in >50% of cancers. Nutlin-3 reactivates the negative regulator of p53 by inhibiting it.
- **PI3K-AKT Pathway:** Alpelisib and Capivasertib are key target nodes. Synergistic PotentialPromising target aligned to understanding cervical cancer, providing translational potential.

***Clinical Implications***

- Prioritized drugs align with FDA-approved cancer therapies, potentially expediting clinical translation.
- Combination therapies targeting p53 and PI3K-AKT pathways show synergistic potential
- High-confidence targets align with known cervical cancer pathways, offering potential for translational applications.
- FDA-approved drugs (e.g., Alpelisib) provide an expedited route to clinical trials.

## CONCLUSION

This comprehensive study clearly demonstrates the great potential and power of integrating multi-omics data with advanced analytics in the context of cancer research. This study provides a solid foundation for developing oncology strategies for patients by identifying new biological targets and potential drug candidates and providing detailed information on the integration of HPV in clinical carcinogenesis. Future studies and research are needed to validate these important findings in clinical and preclinical settings and ensure their real-world validity.

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