ABSTRACT

INTRODUCTION

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The Long-Term Effects Of Untreated Mental Health Disorder in Adults Keshia Alleyne

Behavioral Science Community Health, School of Social & Behavioral Sciences, Mercy College CSTEP Summer Research Program, Mercy College







Abstract

Telomeres and telomerase are critical for maintaining and stabilizing the chromosomes and for cancer development. Previous research has suggested a role of human behavior on telomere lengthening. As a result, we investigated the influence of physical exercise on telomere length. We hypothesized that telomere length and cancer risk are related and that physical exercise could help prevent abnormal telomere lengthening. We explored current literature, especially original research articles, to understand the relationship between physical exercise and telomere maintenance. The current research results suggest that exercise contributions to the prevention of cancer through regulating telomere length. Thus, regular physical exercise is a valuable activity to help maintain human health against cancer development.

Introduction

• Telomeres are natural chromosomal terminal structures. A telomere is a chromosomal stretch containing repeating nucleotide (organic molecule) sequences .It prevents abnormal chromosomal breakage and fusion.

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• Telomere length has been proposed as a possible cellular

Materials & Methods

Results

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Mean change in relative telomere length over 5 years with lifestyle intervention compared with control

Fig. 2. Increases in telomerase activity were linked with reductions in psychological discomfort, cortisol, dietary fat consumption, and glucose.

Fig. 1. Lifestyle can influence the likelihood of getting cancer by

• Exercise preserves telomere structures.

https://10.1016/S1470-2045

Conclusion



Arsenis, N. C., You, T., Ogawa, E. F., Tinsley, G. M., & Zuo, L. (2017). Physical activity and telomere length: Impact of aging and potential mechanisms of action. Qpeqvctigv. :(27), 45008-45019. https://10.18632/oncotarget.16726

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B. (2018). *Gzgtekug*. "V

Vjg"*Ncpegv*"*Qpeqnqi* {. *36*(11), 1112.

Acknowledgments

College students undergo a lot of sleepless nights for assignments, impending due dates for multiple classes at the same time, and extracurricular activities all the while they are maintaining a job, social life, and personal hurdles that they could have. They are expected to perform at very high levels so that they can get the grades to get into the field or career they're chasing. However, this does not come without sacrifices to their mental health due to all the stress they have undergone.

College students undergo a ridiculous amount of stress because of not only school but their problems outside of

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<u>ABSTRACT</u>

Paxillin is a protein involved in tumor growth, focal adhesion, and motility throughout the plasma membrane and cytoplasm. The roles of paxillin in human malignancies remain unclear. Higher expression levels of paxillin within the nucleus were detected in several studies linking the protein to tumors. Overexpression of paxillin promoted the migration and invasion of cancer cells, while under expression suppressed them. Scientists believe that paxillin may function as an oncogene by regulating tumor cell motility.

INTRODUCTION

Studies determined that paxillin significantly regulates genes involved in the cell cycle. It was, therefore, concluded via knockdown trials that an absence of paxillin resulted in the promotion of prostate cancer cell proliferation by modifying cell cycle progression. It was also found that paxillin regulates apoptotic genes and pathways minimally.

METHODS AND MATERIALS

Specimens from those with normal salivary glands and salivary gland tumors. Western blot analysis was performed to detect the expression levels of paxillin, which used the independent t-test.

Both siRNA and a non-targeted siRNA pool were used in the knockdown experiments.

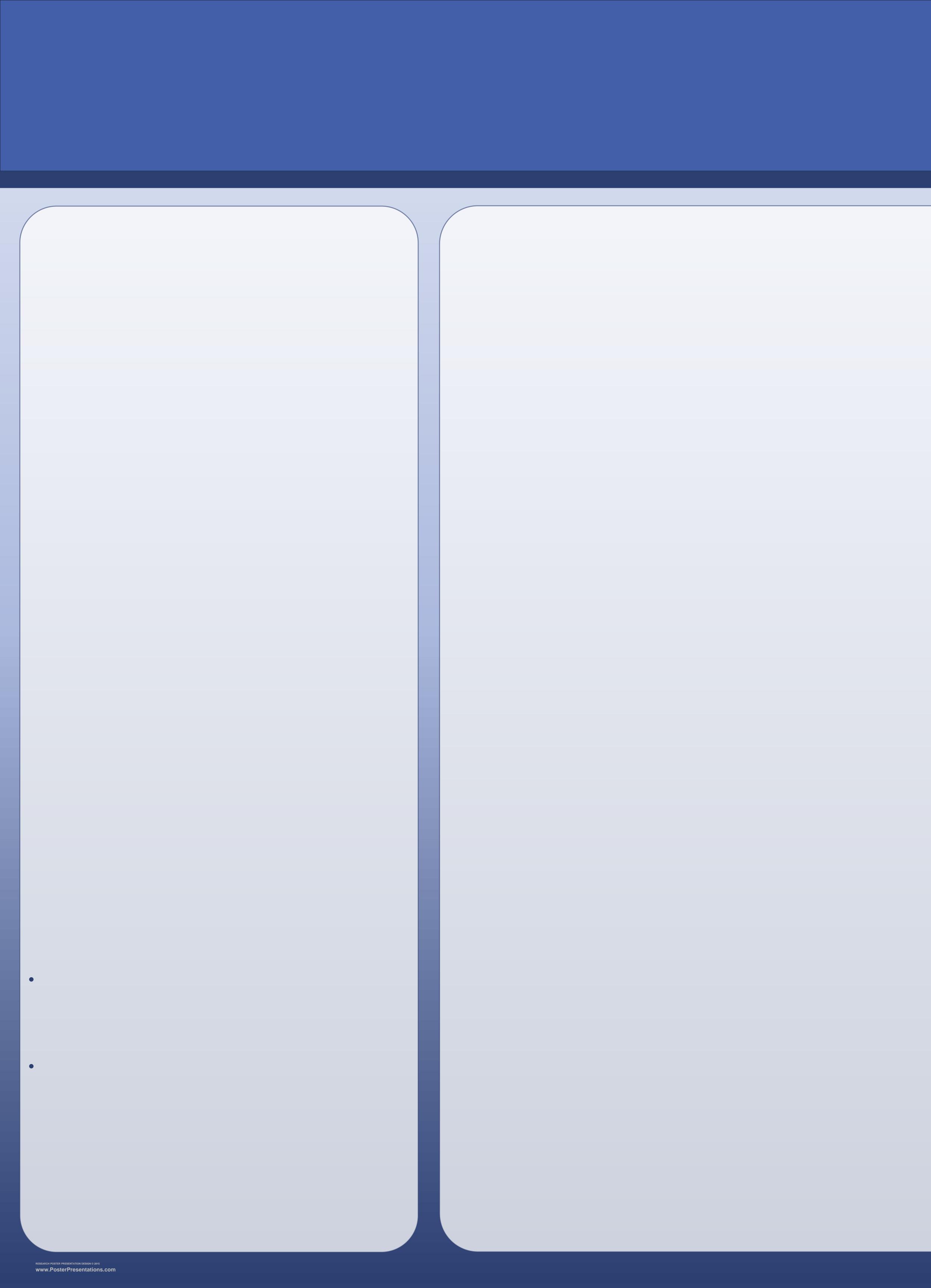
<u>RESULTS</u>

It was observed that paxillin was highly expressed in tumoral tissues in SGTs. It may also function as an oncogene. Overexpression may be closely related to tumor progression of human gliomas. Paxillin is linked to modulating tumor cell motility, leading scientists to believe that there is the potential for use as a therapeutic target for glioma intervention. There may be a genomic network of paxillin found to be upregulated in prostate cancer. Further studies are required to determine how to target paxillin in patients.

Andisheh-Tadbir, A., Afshari, A., & Ashraf, M. J. (2019). Expression of Paxillin in Benign and Malignant Salivary Gland Tumors. Journal of dentistry

<u>CONCLUSIONS</u>

<u>REFERENCES</u>



Epilepsy

Frontiers in Neurology 11

Epilepsy Surgery and Intrinsic Brain Tumor Surgery

The Treatment of



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Abstract

Introduction

Materials and Methods

articles OLNH FDQFHUV %XW HYHQ ZLWK DOO WKH UHVHDUFK WKQW KDV EHHQ GRQH WKHUH 50 original research articles

Results

Telomerase and ALT generate different structures at the end of chromosomes, via histone modification of DNA

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Conclusion

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ABSTRACT

The understanding and learning of the molecular mechanism related to the hypoplastic left heart syndrome(HLHS) can facilitate future findings and the prevention of the syndrome. For this purpose, I identified relevant original research articles from PubMed. The results of the articles suggest that the chromosome 2p23.2 is the most linkage signal in response to HLHS. Moreover, hub genes were identified for HLHS via WGCNA, and analysis of differential gene expression revealed unique mRNA splicing patterns in HLHS. Based on the article findings, I can suggest that this is one way on creating a molecular pathway in HLHS. My analysis of the findings creates a concept map explaining how they are correlated together, shedding light to our understanding on the HLSH pathogenic mechanisms.



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MATERIALS AND METHODS

- syndrome occurs.
- transcripts.

Ricci, M., Xu, Y., Hammond, H. L., Willoughby, D. A., Nathanson, L., Rodriguez, M. M., Vatta

CONCLUSION

• In all three articles the important findings are used to articulate a single molecular mechanism that tries to explain why hypoplastic left heart

• The molecular mechanism of the hypoplastic left heart syndrome is at chromosome 2p23.2, following with multiple examined hub genes, expressed gene expression and spliced

• The need for further studies is essentially for finalizing a molecular mechanism and the prevention of the hypoplastic left heart syndrome.

REFERENCE