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

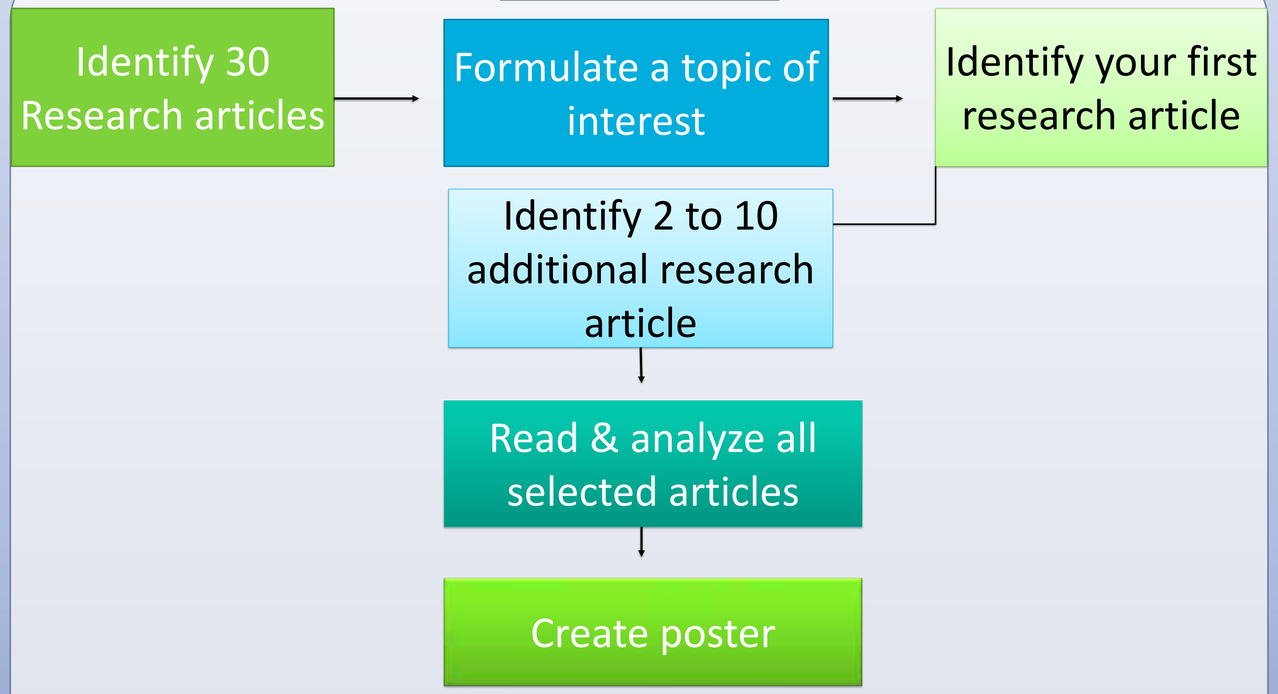
ABSTRACT

The outcome of unaddressed mental illness and delayed intervention can have detrimental long-term effects on an individual's quality of life as well as their overall health, such as relationships with family and friends and self harm. Over the years, there has been a number of studies conducted in order to further investigate this issue as well as factors that may have contributed to this problem. Upon reviewing such as articles the word stigma became more and more apparent and was established as one of the leading causes of such delay, specifically self stigma. Some secondary factors include the incorrect perception concerning mental disorders and the lack of awareness regarding the effectiveness of mental health treatment. Although the current findings have contributed to the advanced knowledge of those issues, it lacks continuous studies that focus on this problem as well as providing directives in how individuals can overcome such obstacles.

INTRODUCTION

- Mental Health has been and continues to remain a critical societal issue with rising numbers of diagnosed individuals on a yearly basis.
- Common diagnosis includes bipolar disorder, anxiety disorders, schizophrenia, obsessive compulsive disorder and post traumatic stress disorder
- General symptoms consist of changes in moods, anxiety, self isolation, extended period of saddest and irritably, and patterns of eating and sleeping
- Untreated mental illness in adults often leads to setbacks and disruptions in one's quality of life.
- Although some articles have partially analyzed good practices to mitigate such issue, there is still a lot of unknown directives in good practices that are left unstudied. This is partially due to the fact that those studies were conducted on small groups. Thus, there exists a need for studies using larger populations.

MATERIALS & METHODS



RESULTS

High stigma is correlated to negative effects on behaviors and well-being

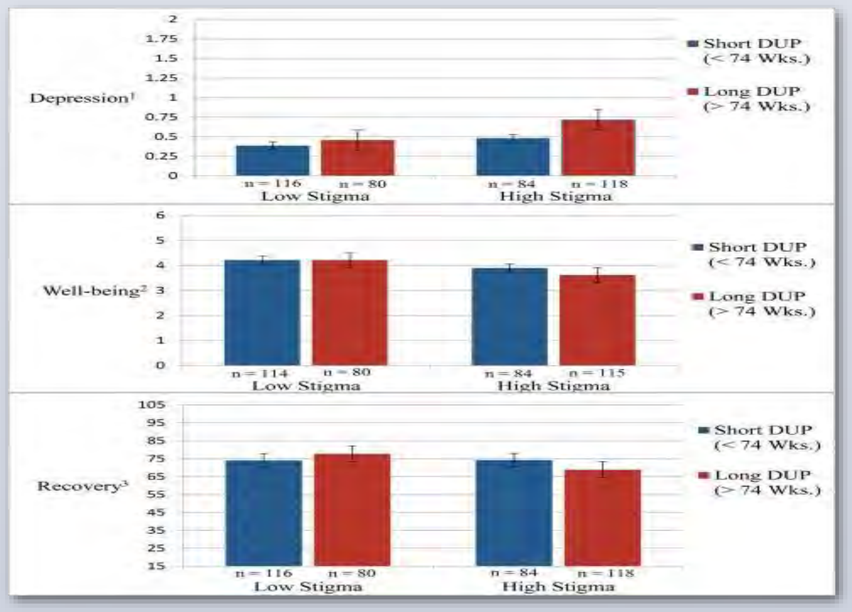


Fig 1. Perceived stigma was notably connected to depression, well-being and recovery. The figure displays these ratings for individuals with low perceived stigma vs. high stigma, and short duration of untreated psychosis (DUP) vs. long DUP.

Outcomes for seeking Mental Health Treatment

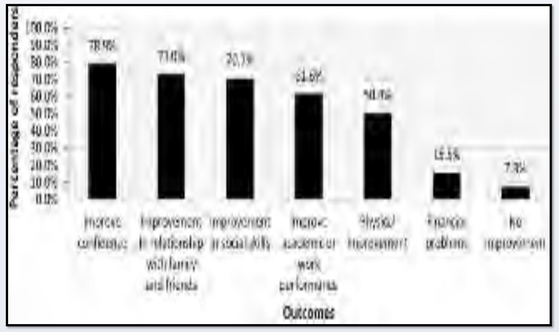


Fig 2. Demonstration of the outcomes of seeking professional treatment for mental health issues

CONCLUSION

- The study of the long-term effects of untreated mental health disorder in adults is important because it focuses on a problem that affects many individuals, not just those affected by mental illnesses.
- It can definitely provide knowledge where it is needed in helping not only the patients themselves but also physicians to better understand the psychological aspect of symptoms, as well as the causes and reasons why some live under fear of stigma.
- This research can lead to an individual's increased capability to make progress and draw accomplishments in their life.

REFERENCES

1. Brouwers, E. P. M. (2020). Social stigma is an underestimated contributing factor to unemployment in people with mental illness or mental health issues: position paper and future directions. BMC Psychology, 8(1), 36-0. https://10.1186/s40359-020-00399-0 [doi]
2. Mueser, K. T., DeTore, N. R., Kredlow, M. A., Bourgeois, M. L., Penn, D. L., & Hintz, K. (2020a). Clinical and demographic correlates of stigma in first-episode psychosis: the impact of duration of untreated psychosis. Acta Psychiatrica Scandinavica, 141(2), 157-166. https://10.1111/acps.13102 [doi]
3. Mohamed Ibrahim, O. H., Ibrahim, R. M., Al-Tameemi, N. K., & Riley, K. (2020). Challenges associated with mental health management: Barriers and consequences. Saudi Pharmaceutical Journal : SPJ : The Official Publication of the Saudi Pharmaceutical Society, 28(8), 971-976. https://10.1016/j.jsps.2020.06.018 [doi]

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The Effects of Physical Exercise On Telomere Lengthening and Cancer

Myazia Daye

Department of Behavioral Science, School of Social & Behavioral Science, Mercy college

Collegiate Science & Technology Entry Program (CSTEP) , Mercy College

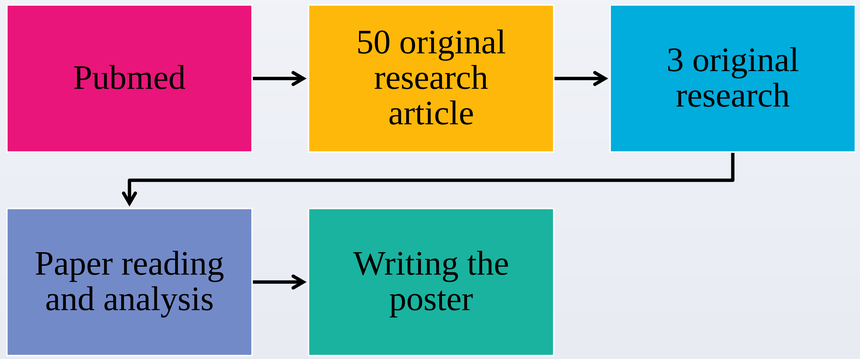
Abstract

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.
- Telomere length has been proposed as a possible cellular marker for biological aging.
- Telomeres shorten with each cell division cycle and oxidative stress.
- Cancer is a condition in which some cells in the body grow out of control and spread to other areas of the body.
- The purpose of this study was to determine if exercise has a role in the relationship between shorter telomeres, higher telomerase activity, and cancer.
- We hypothesized that poor quality of life associated with cancer therapy may promote sedentism and telomere degradation. Those with shorter telomere length probably had a bad lifestyle and were more prone to cancer.
- Exercise has been shown to reduce biochemical factors linked to telomere loss, including-oxidative stress and chronic inflammation
- Lifelong activity and sedentary behavior have been shown to extend telomeres.
- Our investigation may shed light to the understanding on the usefulness of physical exercise on human health.

Materials & Methods



Results

Lifestyle without exercise leads to shortened telomere length and/or cancer

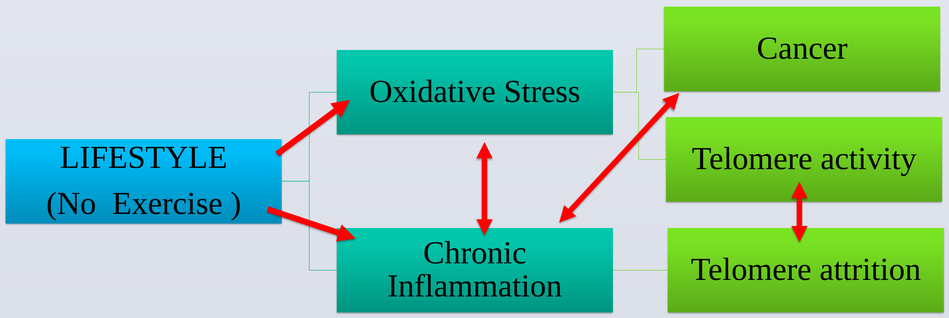


Fig. 1. Lifestyle can influence the likelihood of getting cancer by regulating telomere activity. One of the mechanism is via regulating oxidative stress and chronic inflammation.

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

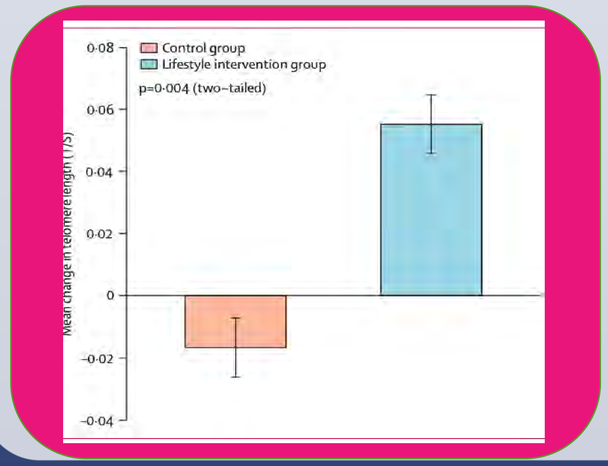


Fig. 2. Increases in telomerase activity were linked with reductions in psychological discomfort, cortisol, dietary fat consumption, and glucose.

Changes in telomere length

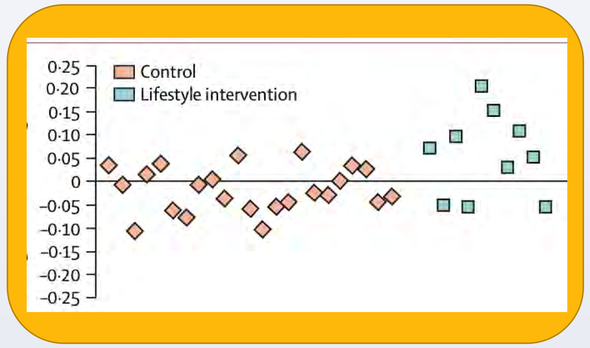


Fig. 3. There is a strong dose-response relationship between the degree of lifestyle modification and the amount of telomere length alteration.

Conclusion

- Patients with cancer may have decreased quality of life, which may lead to increased dental behavior and telomere length.
- The poor quality of life that individuals with shorter telomer length have increases their risk of cancer in later age.
- Poor lifestyle choices increase oxidative stress and decrease telomerase activity.
- Stress, diet, body composition, sleep quality, and ageing all influence telomere length.
- Telomere length may be maintained by lifestyle.
- Exercise preserves telomere structures.

References

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. *Oncotarget, 8*(27), 45008-45019. https://10.18632/oncotarget.16726

Hagstrom, A. D., & Denham, J. (2018). The Effect of Resistance Training on Telomere Length in Women Recovering from Breast Cancer. *Journal of Functional Morphology and Kinesiology, 3*(1), 9. https://10.3390/jfmk3010009

Nomikos, N. N., Nikolaidis, P. T., Sousa, C. V., Papalois, A. E., Rosemann, T., & Knechtle, B. (2018). *Exercise, Telomeres, and Cancer: "The Exercise-Telomere Hypothesis".* Frontiers Media SA. https://10.3389/fphys.2018.01798

Ornish, D., Lin, J., Chan, J. M., Epel, E., Kemp, C., Weidner, G., Marlin, R., Frenda, S. J., Magbanua, M. J. M., Daubenmier, J., Estay, I., Hills, N. K., Chainani-Wu, N., Carroll, P. R., & Blackburn, E. H. (2013). Abiraterone acetate plus prednisone versus prednisone alone in chemotherapy-naïve men with metastatic castration-resistant prostate cancer: patient-reported outcome results of a randomized phase 3 trial. *The Lancet Oncology, 14*(11), 1112. https://10.1016/S1470-2045

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How do students cope with mental health issues?

Nelson Alcantara

Department of Natural Sciences, School of Health and Natural Sciences, Mercy College

CSTEP Summer Research Program, Mercy College

Contact information:
nelson.a.1536@gmail.com

ABSTRACT

Negative mental health for students due to constant stress can hinder the way by which they are able to excel in their academic careers. Mental health issues involving stress are a leading impediment to the academic success of many students around the nation. About 64% of college dropouts are not attending college because of a mental health related issue that includes depression and posttraumatic stress disorder which are the primary diagnoses of young adults that dropout. Almost 73% of students who had mental health issues reported having a mental health crisis on campus however, 34.2% reported that their college didn't know what had happened to them.

INTRODUCTION

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 school, like financial troubles, relationships, family issues and even maintaining a healthy social battery. Adding on top of those troubles' due dates and rigorous assignments college students are under a severe amount of stress and usually do not know how to cope with or seek it help for it. Some students sometimes don't cope with stress in the correct ways harming them even more.

Student Mental Health

Students reporting mental health issues and how it led to substance abuse

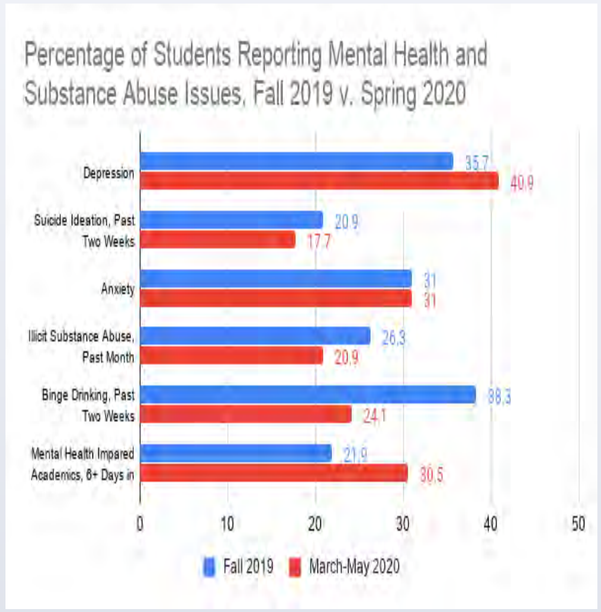

Fig. 1.

Students are not seeking help. More than 45% of young adults who stopped attending college because of their mental health never requested accommodations or access the mental health services and supports either . In many cases students believe that they don't have an issue or believe that they shouldn't seek help since it'll make them seem weaker than they are. Concern of stigma is the main reason that students don't seek assistance.

A growing need for help

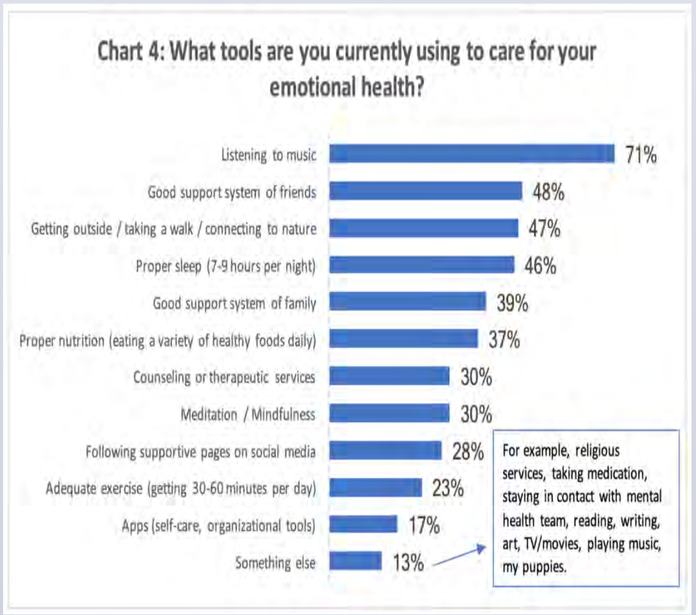

Fig. 2. In this survey done by the JED Foundation on multiple colleges they found various ways in which students would deal with their mental health and as you can see in the chart therapeutic services only amounted for about 30% of the responses to the survey. The survey goes on to elaborate that students are either not aware of the services that they are provided at their school or are simply not willing to go because of what they believe others will say. In most cases students are more concerned over the stigma of their issue than seeking assistance. There is a need for more campus based mental health services as there is an increase in the demand for it since how difficult a lot of students find the pandemic is dealing with alone.

CONCLUSIONS

- **Most students that have mental health related issues do not seek help because of the stigma that surrounds it impacting their academic careers negatively**
- **There is an overall need for more mental health assistance services on campuses and there needs to be a way that allows for students to be able to use these services. If this continues like this for a long time it is more likely that students with mental health issues will receive lower GPA's, drop out of college and be unemployed as compared to people without these issues.**
- **There are many safe ways to cope with mental health that don't need to result in substance abusing that will further hinder your development in your academic career and could even transpire into addiction.**
- **Counseling services need to be accessible to students a lot easier so they can pursue the help they need**

REFERENCES

- Rafat Hussain, Michelle Guppy, Suzanne Robertson and Elizabeth Temple, Physical and mental health perspectives of first year undergraduate rural university students, *BMC Public Health*, 2013 13, 1-11.
- Jenny Hyun, PhD, MPH; Brian Quinn, PhD; Temina Madon, PhD; Steve Lustig, Mental Health Need, Awareness, and Use of Counseling Services Among International Graduate Students, *Journal of American College Health,* 2007 56(2), 109-118
- Michael J. Mason, PhD; Nikola Zaharakis, MS; Eric G. Benotsch, PhD, Social Networks, Substance Use, and Mental Health in College Students, *Journal of American College Health*, 2014 62(7), 440-477
- Jeremy B. Yorgason, PhD; Deanna Linville, PhD; Bryan Zitzman, PhD, "Mental Health Among College Students: Do Those Who Need Services Know About and Use Them?", *Journal of American College Health,* 2008 57(2), 173-182

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Potential Clinical Role of Telomere Length in Human Glioblastoma

Otino Thomas

Department of Natural Sciences, School of Health and Natural Sciences, Mercy College

CSTEP Summer Research Program, Mercy College

ABSTRACT

Glioblastoma Multiforme (GBM) is the most common and lethal of human primary central nervous system (CNS) tumors. Due to the tumor's intrinsic clinical and molecular heterogeneity, the following factors represent some of the greatest challenges in the management of GBM patients: choice of initial treatment, prediction of survival, stratification of patients, and prediction and monitoring of response to therapy. Patients, despite optimal surgery, radiation and chemotherapy, still have a median survival of 14-16 months. A reason for this dismal prognosis is because of the relative inaccuracy of current prognostic markers, so far based on clinical or pathological variables. Molecular markers that effectively predict response to therapy and survival outcomes are limited. Consequently, there is a strong need to develop novel and independent markers of prognosis. Ideal biomarkers for solid tumors would serve one or more important functions. Telomeres, guanine-rich tandem DNA repeats of the chromosomal end, provide chromosomal stability, regulates important cellular processes, and seem to be implicated in human carcinogenesis.

INTRODUCTION

- Gliomas are a heterogeneous group of malignancies with highly variable outcomes
- Pathological diagnosis is largely based on the histological appearance of the tumors. Despite notable recent achievements in oncology, malignant gliomas such as glioblastoma multiforme (GBM) present some of the greatest challenges in the management of cancer patients worldwide.
- Glioblastoma is the most common primary brain tumor in humans and has the most severe prognosis. Even with aggressive surgical resections using state-of-the-art preoperative and intraoperative neuroimaging, along with recent advances in radiotherapy and chemotherapy, the prognosis for GBM patients remains dismal: median survival after diagnosis is about 15 months.
- Although GBM is one of the best-studied brain tumor in terms of genetics and molecular prognostic factors, the true prognostic significance of all potential factors under investigation remain to be clarified.
- It has recently been speculated that changes in telomere domain can result in genetic disorders, genomic variability, and cell immortalization. Telomeres consist of long tandem arrays of TTAGGG repeats bound by proteins collectively termed the shelter in complex, placed at the end of linear chromosomes, which are involved in several essential biological functions.
- Functional telomeres protect chromosome ends from recombination and fusion, and are therefore essential for maintenance of chromosomal stability. Telomere dysfunction occurs as a result of critical shortening of telomeres, followed by sequential bridge–fusion–breakage cycles, leading to numerical chromosomal abnormalities.
- The phenomenon of telomere alteration during tumorigenesis process and progression of solid tumors is well known and established at the molecular level. Cells exhibiting telomere dysfunction, with critical shortening and genomic instability, increase in both the formation of dicentric chromosomes and susceptibility to oncogenic transformation.

Materials and Methods

- ✓ **Patient population:** This study included tumors samples, histologically verified as GBMs, obtained in adult patients who underwent craniotomy for microsurgical tumor resection, at the Department of Neurosurgery of the University of Messina. Only patients who had undergone large, gross total resection of their neoplasms (more than 95 % of the tumor volume) were eligible for the study. All patients underwent Temozolomide chemotherapy (75 mg/m(2)/d x 7 d/wk for 6 weeks) administered orally concomitant with fractionated radiotherapy (60 Gy total dose: 2 Gy x 5 d/wk for 6 weeks) followed by temozolomide monotherapy (200 mg/m(2)/d x 5 days, every 28 days for six cycles).
- ✓ **Tissue Samples:** All tumor tissue samples were obtained from resection specimens, within 15 minutes from surgical tissue removal. Specimens were taken from viable areas of tumor, avoiding areas of gross necrosis and three to seven anatomically separate areas of tumor tissue were sampled from each resection specimen, according to the volume of excised tissue available
- ✓ **Telomere length Analysis:** Genomic DNA was extracted from tumor specimens using standard method phenol/chloroform. Terminal Restriction Fragments (TRF) mean length measurement was performed using Telo TTAGGG Telomere Length Assay kit. Following to analysis, the TRF length was determined as the ratio of the length of tumor tissue TRF and their paired normal tissue TRF.
- ✓ **Statistical Analysis**: The Pearson test was used to obtain correlation values among numerical variables. Data analysis was performed with INSTAT, v. 3.0, and PRISM, v. 4.0. A probability value less than .05 was considered statistically significant. All values are expressed as the means ± SD.

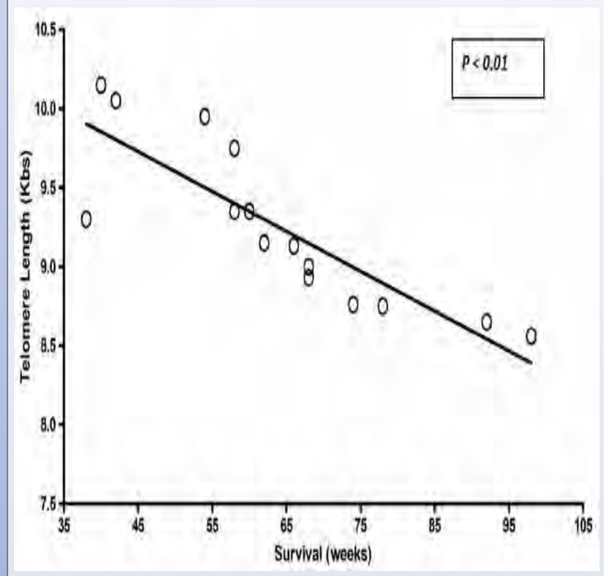
RESULTS

Analysis of telomere length in different patients

Column1	Column2	Column3	Column4	Column5	Column6	Column7	Column8
patients	age (yr) (sex	localization	duration of s	KPS score	survival (we	telomere length (kb)	
1	57/F	R-FP	6	90	42	9.3	
2	**2**	62/M	R-FP	7	100	40	10,15
3	**3**	68/M	L-O	3	100	38	10,05
4	**4**	64/F	R-P	7	90	54	9,95
5	**5**	68/M	L-PT	5	100	58	9,75
6	**6**	67/F	R-FT	11	70	58	9,35
7	**7**	65/M	R-O	10	70	60	9,35
8	**8**	69/M	L-FP	8	80	62	9,15
9	**9**	73/F	R-PO	4	90	66	9,13
10	**10**	71/M	R-TP	1	90	68	9
11	**11**	76/M	R-F	3	90	68	8,93
12	**12**	68/M	R-T	3	90	74	8,76
13	**13**	67/F	L-FT	2	90	78	8,75
14	**14**	62/M	R-F	8	90	92	8,65
15	**15**	63/F	L-F	5	90	98	8.56
N.B.T.							10.93

Table 1. Patients' clinical and molecular parameters. Telomere length was measured by Southern blot analysis in primary, untreated glioblastoma samples and in matched normal tissue TRF (ratio T/N) from 5 patients. Telomeres length was determined as the ratio of the length of tumor tissue TRF and their paired normal tissue TRF (ratio T/N). All cases in which T/N ratio resulted <1 were considered into the group of telomere shortening. When T/N was ≥1, tumors were considered in the group of telomere maintenance. Changes in telomere length, compared with their paired normal brain tissue, were observed in all tumors; were shorter in 13 of 15 tumors (86.66%). While telomeric length in 2 tumors (13.33%), was considered unchanged. **Key:** M: male; F: female; L: left; R: right; F: frontal; P: parietal; T: temporal; O: occipital; KPS: Karnofsky Performance Scale; kb: Kilobase; N.B.T.: normal brain Tissue.

Graph showing inverse correlation between telomere length and survival



Conclusion

- GBM research is being conducted worldwide at a remarkable pace, with some of the more recent promising studies focused on identification of aberrant genetic events and signaling pathways, tumor stem cell identification and characterization, modulation of tumor immunological responses, combination therapies, and understanding of the rare long-term survivors.
- Identification of additional indicators will enable better patients' stratification and individualization of treatment, is needed to more accurately determine the patient's prognosis and to identify novel therapeutic approaches that can optimize the patient's outcome.
- A growing body of knowledge suggest a potential role of telomere length in different tumors. Nevertheless, even if its clinical use its not completely established, a number of studies demonstrated that it can be helpful to patients stratification, to provide useful information about patients prognosis and, in some case, to suggest new therapeutic strategies in cancer diseases.
- The present review shows that the potential clinical use of telomere length information for the prognosis of GBM has been recognized and continues to be validated. Although not entirely consistent in the type of telomere alteration, i.e., attrition vs. elongation, and unclear on the underlying mechanisms, multiple studies in brain tumors have shown that telomere dysfunctions are associated with parameters of clinical outcome in patients with GBMs.

REFERENCES

La Torre D, Aguennouz M, Conti A, Giusa M, Raffa G, Abbritti RV, Germano' A, Angileri FF. Potential clinical role of telomere length in human glioblastoma. Transl Med UniSa. 2011 Oct 17;1:243-70. PMID: 23905037; PMCID: PMC3728840.

Kleihues P, Louis DN, Scheithauer BW, et al. The WHO classification of tumors of the nervous system. *Journal of Neuropathology and Experimental Neurology.* 2002;61(3):215–25.

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The Role of Paxillin in Tumor Cells

RJ Bradley-Ortiz

Department of Natural Sciences, School of Health and Natural Sciences | Mercy College

CSTEP Summer Research Program

ABSTRACT

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

- Three studies were selected based on their commonalities studying paxillin in various tumor and cancer cells, such as salivary glands, gliomas, and prostate cells.
- Found at the intracellular scaffold where the cells adhere to the extracellular matrix, paxillin functions as an oncogene involved in key signal transduction, cell motility, migration, proliferation, survival, angiogenesis, and apoptosis.
- There have been many studies to determine the full effect of paxillin expression to provided insight into tumor development. Paxillin might be an effective therapeutic target in medical science to suppress tumor growth and metastasis as well as to develop superior cancer therapies and treatment.

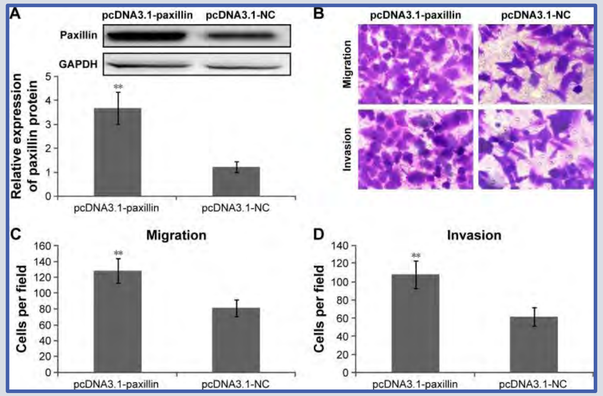
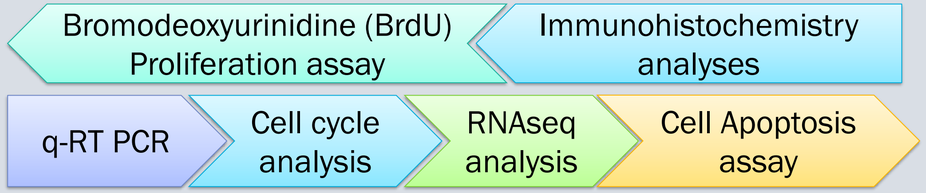


Figure 1:
Paxillin expression promotes migration and invasion of glioma cells in vitro. (A) Western blot detects the expression in U251 cells. (B–D) Migration and invasion abilities of human glioma cells tested.

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.



RESULTS

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.

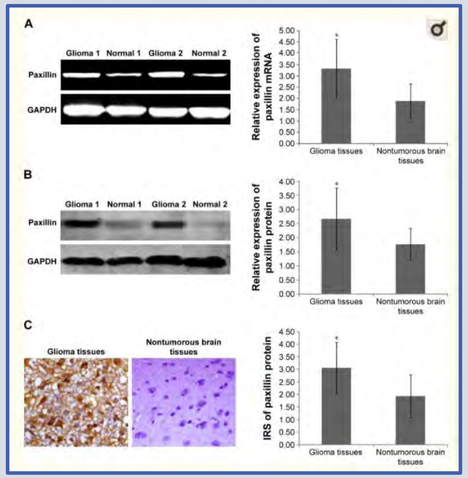


Figure 2:
Paxillin upregulation in human glioma tissues. (A) Expression seen via mRNART-qPCR analysis in glioma and non-tumorous brain tissues. (B) Expression seen via western blot analysis. (C) Immunostaining found paxillin in cytoplasm and cell membrane of tumor cells in human glioma. Little to none found in non-tumorous brain tissues.

CONCLUSIONS

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.

REFERENCES

- Andisheh-Tadbir, A., Afshari, A., & Ashraf, M. J. (2019). Expression of Paxillin in Benign and Malignant Salivary Gland Tumors. Journal of dentistry (Shiraz, Iran), 20(4), 249–254. https://doi.org/10.30476/DENTJODS.2019.44909
- Chen, B., Xia, L., Xu, C. S., Xiao, F., & Wang, Y. F. (2016). Paxillin functions as an oncogene in human gliomas by promoting cell migration and invasion. OncoTargets and therapy, 9, 6935–6943. https://doi.org/10.2147/OTT.S114229
- Ma, X., Biswas, A., & Hammes, S. R. (2019). Paxillin regulated genomic networks in prostate cancer. Steroids, 151, 108463. https://doi.org/10.1016/j.steroids.2019.108463

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Contact: rbradleyortiz@mercy.edu

Vagus Nerve Stimulation Treatment for Drug-Resistant Epilepsy

Anneika Greene
Department of Behavioral Sciences, School of Social & Behavioral Sciences, Mercy College
Collegiate Science & Technology Entry Program (CSTEP), Mercy College

Abstract

VNS treatment is approved for the treatment of partial or focal seizures that fail to respond to pharmacological treatments. It lessens or prevents seizures by regularly transmitting mild pulses containing electric energy to the brain through the vagus nerve. The therapy process involves implanting a device under the skin in the left area of the chest. An electrode is connected to a generator device and put under the skin of the patient engaging in the process, which is followed by the wounding of the wire around the vagus nerve found in the victim's neck. The implanted device is programmed in the patient to deliver stimulation at given regular intervals. The design of VNS aims to change the functionality of the brain cells by providing electrical stimulation to the areas involved in seizures. However, VNS therapy cannot cure epilepsy. Instead, its design helps in lessening the number of seizures a patient may have or reduces their severity. VNS may also help in the faster recovery of the individuals after experiencing seizures.

Introduction

Vagus nerve stimulation (VNS) is an effective and safe therapy that has been used for more than 20 years for children and adults who have drug-resistant epilepsy (DRE). The implant reduces seizures for the victims from four years and above, having focal seizures that have refractory effects to anti-seizures medications. Anti-seizure medications are effective in treating a patient with epilepsy (Fisher et al., 2021). However, not all individuals with epilepsy receive adequate treatment from the anti-seizures. As a result, seizures that can occur to these individuals are known as drug-resistant or refractory. For the patients who may be victims of refractory epilepsy, different combinations consisting of anti-seizure medications can be considered to provide treatment. The combination of anti-seizure medications may help to reduce the number of seizures in an individual. However, there is a risk of having increased side effects from this kind of medication. In this regard, it may be essential to consider the usage of Non-pharmacologic strategies to treat refractory epilepsy (Schachter, 2017).

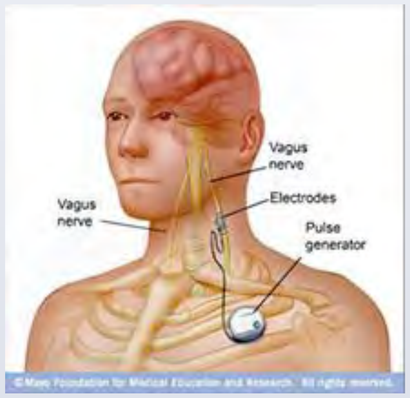
- VNS is an excellent example of such an approach. These may be essential for patients who may not take respective epilepsy surgery. There are various clinics globally that provide manage and provide counsel and treatment for DRE patients having VNS.
- The process has attracted multiple professionals in the healthcare sector, such as physician assistants, nurse practitioners, and clinical nurses, who play essential roles in providing neurological care, including epilepsy (Schachter, 2017). Practice providers have crucial duties in the VNS clinics. In relation, they improve access and promote continuity of care by providing education to patients and families, providing therapy for patients, and ensuring follow-up of the VNS patients (Schachter, 2017). These factors ensure the efficiency of VNS and the well-being of the patients.

Materials & Methods

The study focused on evaluating various data sources and information from secondary studies. The action involved the secondary sources that contain research that is relevant to the topic of the study. The relevancy of the data was essential in ensuring the research could answer the study's objectives (Arcand et al., 2017).

- In this consideration, the study's method considered a retrospective assessment of the efficacy regarding VNS among 30 patient adults having epilepsy treatment and a follow-up of more than six months (Arcand et al., 2017). The following was the method's criteria implantation; no involvement of respective epilepsy surgery candidate, drug-resistant epilepsy, impairing the quality of life, limitation of another form of treatment, and patients with idiopathic generalized epilepsy can fail to be under the control of the necessary AEDs.

- What follows is the assessment of various factors, including the seizure etiology sociodemographic AEDs used in the treatment process using VNS. These assessments were essential in determining the results of the study. Also, there was the studying of various effects and efficacy (Arcand et al., 2017). In relation, the definition of responder rate was defined as> 50% improvement of seizure from the baseline.



Results

The study included thirty participants in total. These were comprised of 18 females and 12 males. The range of their years was between 21 years and 48 years. The follow-up was conducted for six months, 12 months, 32 months, and 36 months. In relation, the response rates were 13/30 (43%) for the six months, 13/27 (48%) for the 12 months, 9/22 (41%) for the 24 months, and 8/16 (50%) for the 36 months. Consequently, no individual was free from the seizures. Individuals comprising 587%, 33%, 59%, and 81% encountered the changes in medication or the type of dosage in the four periods, respectively. For the more significant number of the patients, their medication change involved increasing the AEDs dosage.

Table 3: Characteristics of VNS patients at follow-up

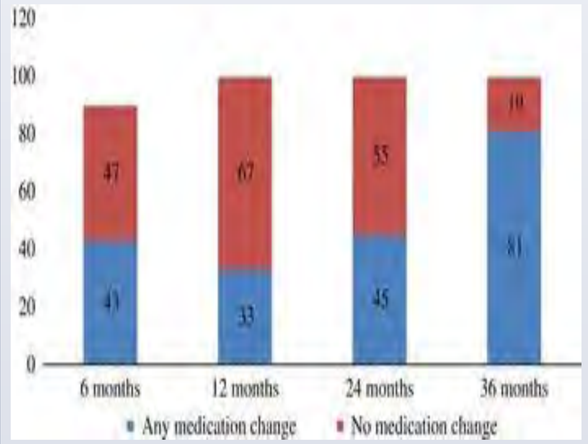
Variable	Pre-VNS	6 months post-VNS (n = 30)	12 months post-VNS (n = 27)	24 months post-VNS (n = 22)	36 months post-VNS (n = 16)	p
Patients (n)	30	30	27	22	16	NA
Percent responders	NA	13 (43%)	13 (48%)	9 (41%)	8 (50%)	0.82
Seizure frequency per month	84.2 ± 143 (1-600)	41.6 ± 73 (0-300)*	31.6 ± 56 (0-210)*	41.5 ± 74 (0-300)*	22.3 ± 26 (0.3-98)*	<0.005
Mean reduction in seizures (%)	NA	59%	67%	56%	55%	0.34
Less admission to ER	NA	10 (33%)	8 (30%)	7 (32%)	4 (25%)	0.63
Less severe seizures	NA	13 (43%)	14 (52%)	13 (59%)	7 (44%)	0.68
Improved quality of life	NA	16 (53%)	15 (56%)	11 (50%)	6 (38%)	0.33

*All median seizure frequency per month values from the different follow-up periods were different than the frequency before implantation of the VNS ($p < 0.005$).
ER = emergency room; NA = not available.

Conclusions

It is essential to conduct a proper experiment and study to determine the effectiveness of vagus nerve stimulation treatment for drug-resistant epilepsy.

- From this study, there is an indication that VNS can be an effective strategy for providing therapy to epilepsy. However, the effectiveness of the therapy is accompanied by high changes in the medication conducted while giving treatment.
- As a result, the action may pose difficulties in determining the real impact from the VNS, therefore, the real effect of VNS could be controversial.



References

Arcand, J., Waterhouse, K., Hernandez-Ronquillo, L., Vitali, A., & Tellez-Zenteno, J. F. (2017). Efficacy of vagal nerve stimulation for drug-resistant epilepsy: Is it the stimulation or medication? *Canadian Journal of Neurological Sciences / Journal Canadien des Sciences Neurologiques*, *44*(5), 532-537. https://doi.org/10.1017/cjn.2017.46

Fisher, B., DesMarteau, J. A., Koontz, E. H., Wilks, S. J., & Melamed, S. E. (2021). Responsive vagus nerve stimulation for drug-resistant epilepsy: A review of new features and practical guidance for advanced practice providers. *Frontiers in Neurology*, *11*. https://doi.org/10.3389/fneur.2020.610379

Hao, M. (2020). Effect of vagus nerve stimulation for the treatment of drug-resistant epilepsy: A protocol of systematic review and meta-analysis. https://doi.org/10.37766/inplasy2020.4.0086

Sajko, T., & Rotim, K. (2018). Vagus nerve stimulation for refractory epilepsy. *Epilepsy Surgery and Intrinsic Brain Tumor Surgery*, 197-204. https://doi.org/10.1007/978-3-319-95918-4_18

Schachter, S. C. (2017). Vagus nerve stimulation. *The Treatment of Epilepsy*, 873-883. https://doi.org/10.1002/9780470752463.ch73

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How does the death penalty affect minorities?

Carmaniola Benjamin

Department of Behavioral Science, School of Social and Behavioral Sciences, CSTEP, Mercy college

ABSTRACT

The death penalty plays a critical role in this country's justice system. Something everyone regardless of your sex, age, and or social class you belong to. As this topic is immense, my research questions focuses specifically on how the death penalty affects minorities. As the years went on, minorities grew in the U.S prison population and white population shrunk.to answer this question, my findings show a pattern of an increase in minorities that are given the death penalty. Meaning more minorities contribute to the U.S. prison population and eventually contribute to the population of capital punishment recipients. These results indicate that the federal death penalty is used disproportionately against people of color. Of the 18 prisoners currently on federal death row, 16 are either African-American, Hispanic or Asian.

INTRODUCTION

- This article direct results and percentage as to minority population involved in the death penalty.
- If you are a minority or committed a crime against whites, then you are most likely going to be charge with the death penalty.
- This paper shows how much more often people in the U.S are getting the death penalty.
- As this is a huge underrated problem in these times, people tend to see the lack of importance to change the systematic racism that so clearly exists.
- This questions matters because any minority could be affected and put on death row.
- The article also shows a study that demonstrates the chance of a defendant getting sentenced to death if the victim was white.

OBJECTIVES

- These articles focus on the percentage of minorities that are affected by the death penalty.
- The chance of being on death row based on who the victim of the crime is.
- The state where you are charged in can increase or decrease the possibility of getting capital punishment. Along with the type of jury and prosecutor you may have.
- The amount of poverty in a county may drive executions and the overall prevalence of murder. This variable is coded as the percentage of the population of the county that is in poverty

MATERIALS AND METHODS

- Research question is also falls under event dependence in U.S. executions.
- The distributions of executions would follow a stretched distribution that would suggest evidence of event dependence.

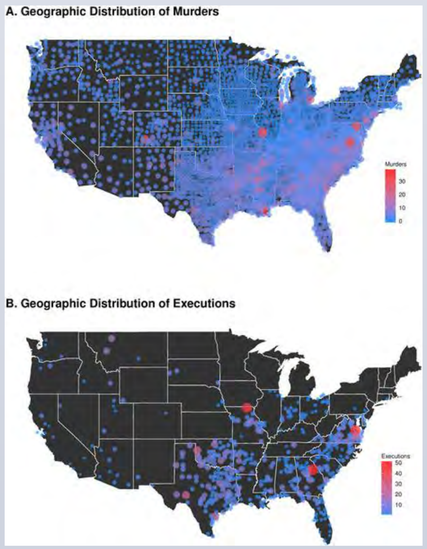
Table 2. Conditional frailty model results for controls

	Model 1
Homicides	−0.00 (0.00)
Percent in Poverty	−0.01 (0.01)
Racial Threat	0.01* (0.00)
ln Population	0.80** (0.04)
AIC	9285.39
BIC	9212.73
R^2	0.30
Max. R^2	0.96
Num. events	832
Num. obs.	3136
Missings	471
PH test	0.15

**$p < 0.001$,
*$p < 0.01$

https://doi.org/10.1371/journal.pone.0190244.t002

RESULTS



- Maps present the average number of murders and executions experienced per year per 100,000 residents by each county between 1977 and 2014.

CONCLUSIONS

- The federal death penalty, like its application in the states, is used disproportionately against people of color. Of the 18 prisoners currently on federal death row, 16 are either African-American, Hispanic or Asian.
- From 1995-2000, 80% of all the federal capital cases recommended by U.S. Attorneys.
- The DOJ study also revealed the influence that the race of the victim has in determining potential capital cases.
- U.S. Attorneys recommended the death penalty in 36 % of the cases with black defendants and non-black victims.
- They only recommended the death penalty in 20 % of the cases with black defendants and black victims.
- People of color have accounted for a disproportionate 43 % of total executions since 1976 and 55 % of those currently awaiting execution.
- While white victims account for approximately one-half of all murder victims, 80% of all Capital cases involve white victims.

REFERENCES

Race and the death penalty. American Civil Liberties Union. (n.d.). https://www.aclu.org/other/race-and-death-penalty.

Baumgartner, F. R., Box-Steffensmeier, J. M., & Campbell, B. W. (n.d.). *Event dependence in u.s. executions*. PLOS ONE. https://dx.doi.org/10.1371%2Fjournal.pone.0190244.

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- Contact information: cbenjamin4@mercy.edu

Telomeres and their effects on cancer

Felipe Marquez
Department of Veterinary Medicine, School of health and science Mercy College
CSTEP Summer Research Program, Mercy college

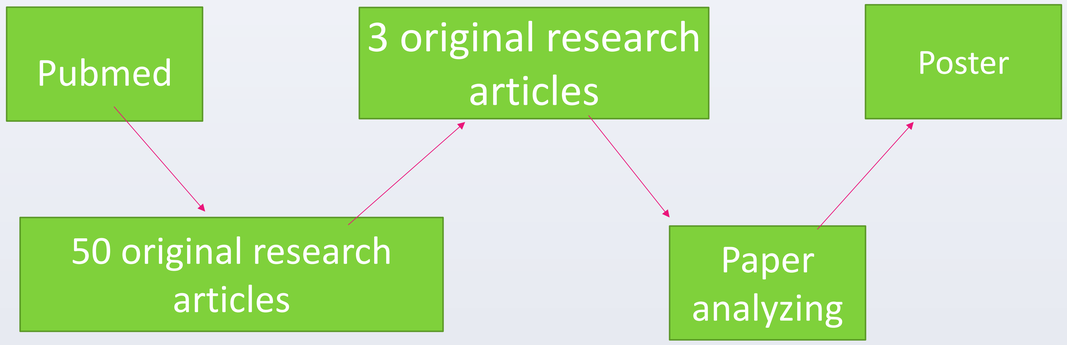
Abstract

Telomeres, the main subject being discussed, are a region of repetitive nucleotide sequences at each end of a chromosome, which protects the end of the chromosome from deterioration or from fusion with neighboring chromosomes. That is why the full study of this topic and any mechanisms that effect and control it is important for the medical community. This led to my finding of the telomere maintenance mechanisms (TMM), which help preserve telomeres from damage. These mechanisms seem to have a connection with cancers when they begin to fail due to age or mutation that stop these TMM from functioning. All this data has led me to believe that these TMM are important for our telomeres and not having them could cause issues like cancers. But, even with all the research that has been done, there has been no direct link between the cause of cancer and either of the two different TMM yet. In fact, some cancers seem to not be linked to a malfunctioning TMM at all. So, I hypothesize that mechanisms like telomerase or alternative lengthening of telomeres (ALT) are in fact just factors that effect the true cancer controlling mechanism in the body.

Introduction

- Telomeres are a region of repetitive nucleotide sequences associated with specialized proteins at the ends of linear chromosomes. As such in a broad sense, telomeres are a widespread genetic feature most found in eukaryotes.
- In most species possessing telomeres, they protect the terminal regions of chromosomal DNA from progressive degradation and ensure the integrity of linear chromosomes by preventing DNA repair systems from mistaking the very ends of the DNA strand for a double strand break.
- Because telomeres have such an important job, there are many mechanisms that prevent telomeres form misfunction or stopping working. One is telomerase (TEL) which, also called terminal transferase, is the enzyme responsible for the maintenance of the length of telomeres by addition of guanine-rich repetitive sequences.
- The other mechanism called alternative lengthening of telomeres (ALT) is still poorly understood but known to rely on telomeric homologous recombination.
- Both mechanisms have activity when it comes to cancerous cell especially the ALT mechanism. The problem though is that not every cancer that humans express show positivity for either ALT or TEL thus forming a direct link of cancer to these mechanisms.
- That is why I predict that there may be another mechanism that can better connect how telomere and cancer prevention are one and the same. This might be able to show why one mechanism may be prevalent in one cancer compared to another.
- Therefore, I choose three specific articles because the key terms found within them centered around telomeres and the two mechanisms we discussed.
- What I have found from those papers shows that again all cancers human attain do not show positive results for either mechanism. The only common feature of all cancers is that they have a more likelihood in being positive for certain ones like with ALT.
- That is why I will conclude that there may be another undiscovered mechanism that can both connect Tel and ALT positivity in a cancer. This possible mechanism might be able to explain how telomeres stop cancers form happening in the first place more directly.

Materials and Methods



Results

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

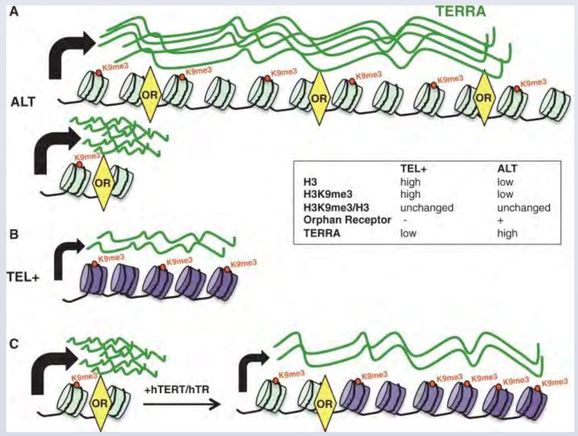


Figure 1: "Possible organization of chromatin at telomeres of ALT and TEL+ cells. (**A**) ALT telomeres are heterogeneous in size, ranging from very long to very short telomeres. They are characterized by a lower nucleosome density than the one detected at telomeres of comparable TEL+ cells. Reduced nucleosome density at ALT telomeres correlates with decreased H3K9me3 occupancy and increased transcription rate, whether at long or at short telomeres. Because of the presence of repeat variants, ALT telomeres are also bound by orphan receptor proteins that, as we speculate, may play important roles in the HR-dependent mechanism of telomere maintenance. (**B**) Telomeric chromatin of TEL+ cells is more condensed; H3 and H3K9me3 densities are increased, and transcription is decreased. (**C**) On overexpression of h*TERT* and h*TR* in ALT cells, short telomeres are elongated. This results in increased H3K9me3 density and telomere transcription downregulation. OR: orphan receptor. H3K9me3 groups are indicated by red dots and TERRA molecules are shown in green.". What this means is that gene factors seems to favor one mechanism over other ones, and these gene have functions that seem to fight cancers.

ALT Relativity to TEL Prognosis

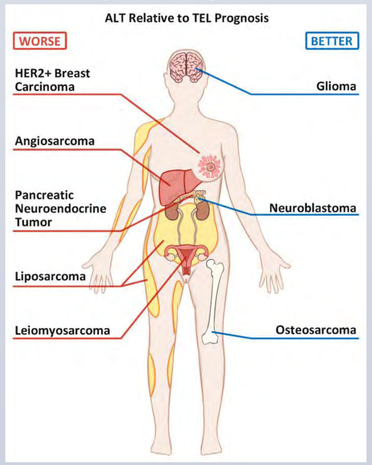


Figure 2
"Commonly seen Alternative Lengthening of Telomeres positive (ALT+) cancers and their prognoses relative to their Telomerase positive (TEL+) counterparts." There is a small group of cancers that rely neither on telomerase nor the ALT pathway for their survival though. Thus this chart show that nether TMM seems to be the number one answer we are looking for and further research is required for this field.

Conclusion

- In summary there is no true connection of both TMM being the main cause for cancers, yet
- This proves my thought and hypothesis that their may be something more to telomeres, possibly an undiscovered TMM
- Though both known TMM are still considered some sort of factor in preventing cancer and underlying mechanisms may yet prove a direct connection
- With a complete understanding of telomeres, humans could possibly discover many positive uses like lifespan increasement and a cure/prevention to cancer

References

1. De Vitis, M., Berardinelli, F., & Sgura, A. (2018). Telomere Length Maintenance in Cancer: At the Crossroad between Telomerase and Alternative Lengthening of Telomeres (ALT). International Journal of Molecular Sciences, 19(2), 606. doi: 10.3390/ijms19020606. https://10.3390/ijms19020606 [doi]
2. Episkopou, H., Draskovic, I., Van Beneden, A., Tilman, G., Mattiussi, M., Gobin, M., Arnoult, N., Londoño-Vallejo, A., & Decottignies, A. (2014). Alternative Lengthening of Telomeres is characterized by reduced compaction of telomeric chromatin. Nucleic Acids Research, 42(7), 4391-4405. https://10.1093/nar/gku114 [doi]
3. MacKenzie, D.,Jr, Watters, A. K., To, J. T., Young, M. W., Muratori, J., Wilkoff, M. H., Abraham, R. G., Plummer, M. M., & Zhang, D. (2021). ALT Positivity in Human Cancers: Prevalence and Clinical Insights. Cancers, 13(10), 2384. doi: 10.3390/cancers13102384. https://10.3390/cancers13102384 [doi]

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Alternative lengthening of telomere in pancreatic tumors

Genesis Lopez

CSTEP Summer Research Program, Mercy College

Department of Natural Sciences, School of Health and Natural Sciences, Mercy College

ABSTRACT

Telomeres are the structures located at the end of chromosomes. A subset of human malignancies is telomerase-negative and rely upon recombination-based components known as ALT (Alternative Lengthening of Telomeres). ALT relies upon proteins that are fundamental for homologous recombination, including BLM and the MRN complex, to expand telomeres. Pancreatic neuroendocrine tumors are the second most normal threat of the pancreas. The ATRX/DAXX complex stores histone variation H3.3 in dreary heterochromatic chromosomal areas, specifically at telomeres and pericentromeric region. The objective of my study is to understand how the telomeres are maintained in pancreatic tumors. I identified a set of original research articles from Pubmed and focused on analyzing three most relevant papers. I hypothesized that ALT contributes to the growth of malignant pancreatic neuroendocrine tumors in patients with the help of the loss of ATRX and DAXX. The overall main findings in these papers were that telomeres could be elongated by telomerase through adding TTAGGG repeats to the chromosome ends. Moreover, ALT was detected in 15 cases; while all ATRX-negative and DAXX-negative tumors were ALT-positive. Finally, ARX expression correlated with larger tumor size. The significance of this topic is to show readers how the tumors in the pancreas arise in the body and the methods by which ATRX-negative and DAXX contribute to the growth of PANET.

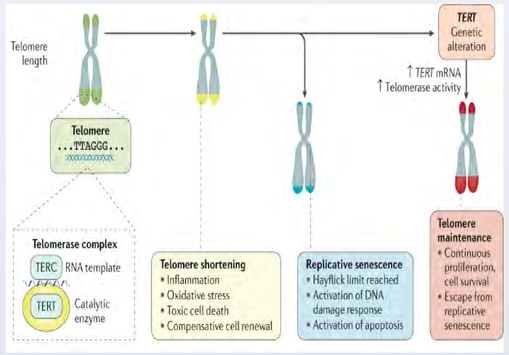
INTRODUCTION

- Telomeres are the complexes of DNA toward the ends of chromosomes that also contain TTAGGG DNA sequences that are repeated. Telomere shortening is firmly related with an expanded danger of malignancy during maturing and different kinds of persistent sicknesses. This leads to chromosomal instability in the body.
- Regularly telomerase is enacted. On the other hand, a subset of human malignancies (Tumors) is telomerase-negative and relies upon recombination-based components known as alternative lengthening of telomeres (ALT). The pancreas secretes enzymes that aid digestion and hormones that help regulate the metabolism of sugars.
- ALT relies upon proteins that are fundamental for homologous recombination, including BLM and the MRN complex, to expand telomeres. This leaves me with the following unsolved questions: How do we prevent these tumors from arising within the human body? What measures can we take in order to prevent them from arising and what would be the genetic makeup of the preventative measures? This leads to my research hypothesis that certain molecular mechanisms regulate ALT to promote pancreatic cancer development. How I selected my three papers to answer my research question is by thoroughly reading the 30 articles I have collected and picking the ones that correlate together and concisely.
- TTAGGG ATRX-negative and DAXX contribute to the ALT lengthening within the body, which causes malignant pancreatic neuroendocrine tumors to arise.
- Tumor cells elongate their telomeres using a recombination-based alternative lengthening mechanism.

MATERIALS & METHODS

- Use of Pubmed to search original research articles using the following keyword: Alternative lengthening telomere, pancreas and tumors.
- Read the title and abstract of the papers to identify the most relevant articles for study
- Summarize the information into a poster

ALT mechanisms



RESULTS

Fig. 1.
Figure one explains and shows the ALT mechanism. It shows the breakdown of how it looks like in the human body and the structural component/ the structural sequence of it. In the figure we are shown the telomerase complex and inside the telomere complex is the RNA template and the catalytic enzyme. We are also shown the telomere shortening mechanisms and the components in that. As well as the replicative senescence and the Telomere maintenance.

Pancreatic tumor

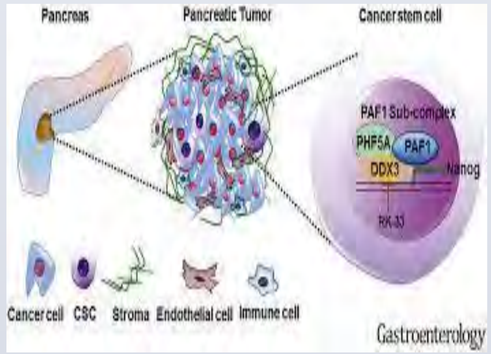


Fig 2.
In figure two we are exposed to to the pancreas and what it looks like when the pancreas has a pancreatic tumor. In the tumor we we see different kinds of components in the tumor. Such as the cancer cells, CSC Stroma, endothelial cell and the immune cell, all in the tumor. In the figure we are shown what the components of a cancer stem cell are. Some of these components include PAF1 sub complex, PHF5A and DDX3.

Curves comparing relapse-free survival (RFS) after surgical resection for patients with NF-PanNET

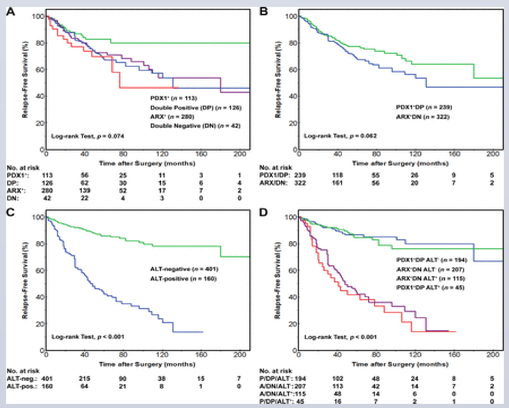


Fig. 3.
After comparing relapse-free survival (RFS) after surgical resection for patients with NF-PanNETs. (A) No statistically significant differences in RFS were identified between patients with NF-PanNETs no RFS difference was seen between patients with ARX^{+}+DN NF-PanNETs and patients with PDX1^{+}+DP NF-PanNETs. However, the RFS for patients with ALT-positive NF-PanNETs was significantly shorter than patients with ALT-negative NF-PanNETs.

Representative ALT-negative and ALT-positive pancreatic neuroendocrine tumors detected on fine needle aspirates

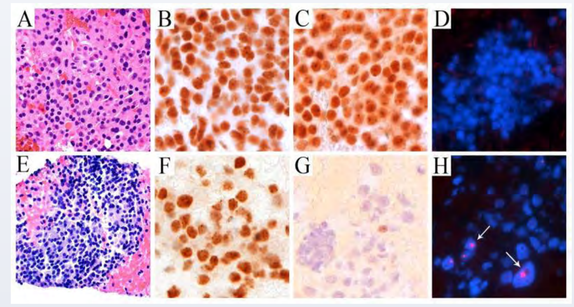


Fig. 4.
The results of immunolabeling for ATRX and DAXX, and of the telomere-specific FISH for ALT. Four specimens were contained. While all ATRX-negative and DAXX-negative tumors were ALT-positive, researchers identify 3 of 14 (21%) ALT-positive cases in which both ATRX and DAXX remained intact.

CONCLUSIONS

- Both ATRX/DAXX IHC and telomere-specific FISH can be accurately performed on FNA specimens to reliably assess ALT status, the cell block material used for analysis is adequate.
- ALT contributes to the growth of malignant pancreatic neuroendocrine tumors in patients with the help of ATRX-negative and DAXX contributing as well.
- Further examinations are important to evaluate the interesting natural attributes and conceivable guess of ALT-positive tumors and will be critical in planning novel enemy of malignant growth therapeutics focusing on the ALT pathway.
- Need to assess the unique biological characteristics and possible prognosis of ALT-positive malignant pancreatic neuroendocrine tumors for designing novel anti-pancreatic therapeutics.

REFERENCES

- Zhao, S., Wang, F., & Liu, L. (2019). Alternative Lengthening of Telomeres (ALT) in Tumors and Pluripotent Stem Cells. *Genes, 10*(12), 10.3390/genes10121030. https://E1030
- VandenBussche, C. J., Allison, D. B., Graham, M. K., Charu, V., Lennon, A. M., Wolfgang, C. L., Hruban, R. H., & Heaphy, C. M. (2017). Alternative lengthening of telomeres and ATRX/DAXX loss can be reliably detected in FNAs of pancreatic neuroendocrine tumors. *Cancer Cytopathology, 125*(7), 544-551. https://10.1002/cncy.21857
- Hackeng, W. M., Brosens, L. A. A., Kim, J. Y., O'Sullivan, R., Sung, Y. N., Liu, T. C., Cao, D., Heayn, M., Brosnan-Cashman, J., An, S., Morsink, F. H. M., Heidsma, C. M., Valk, G. D., Vriens, M. R., Nieveen van Dijkum, E., Offerhaus, G. J. A., Dreijerink, K. M. A., Zeh, H., Zureikat, A. H., . . . Singhi, A. D. (2021). Non-functional pancreatic neuroendocrine tumours: ATRX/DAXX and alternative lengthening of telomeres (ALT) are prognostically independent from ARX/PDX1 expression and tumour size. *Gut,* https://gutjnl-2020-322595

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The Molecular Mechanism of the Hypoplastic Left Heart Syndrome

Giselle Cuahutle
Department of Natural Sciences, School of Health and Natural Sciences, Mercy College
CSTEP Summer Research Program, Mercy College

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.

INTRODUCTIONS

- Hypoplastic left heart syndrome is a birth defect that affects the proper function of the heart flow through the heart. During pregnancy, the left side of a fetus heart does not develop at its full potential. Essentially, this type of condition causes numerous issues such as the left ventricle, the mitral valve, and the aortic valve are either underdeveloped or not developed at all.
- In the current medical research field, there is no correct answer that truly explains the molecular mechanism. However, there are various published articles that try to explain parts of the molecular mechanism. The molecular mechanism is a serious of steps: from transcription, translation, gene expression, and chromosome.
- In the search of the finding good-published articles, I had to learn the foundation on how to identify an article, how to dissect it, how to differentiate between a published and posted article online, and to know whether an article should be read based on the abstract.

MATERIALS AND METHODS

We used the following steps to construct this poster: the use of specific words in relation to the subject, use an online medical database called PubMed, gather 50 research articles, only use 3-5 articles from the gather articles, and lastly, construct the poster

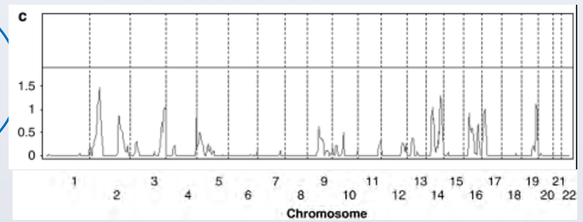
RESULTS

Chromosome

Genes

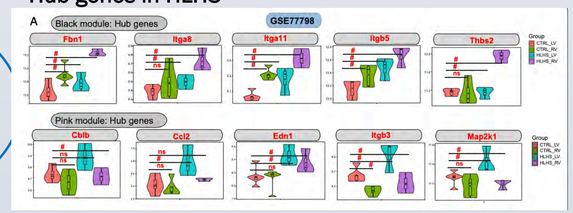
Gene expression and mRNA

Chromosomes related to HLHS



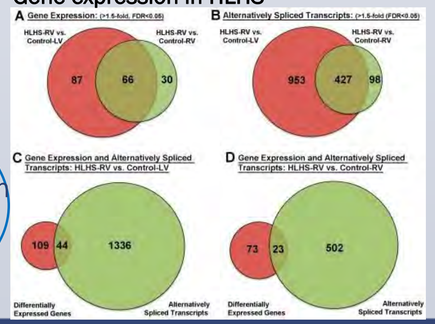
In figure 1, in the letter c portion, the HLOD (the genetic-wide heterogeneity LOD) score results for 43 families with left ventricular outflow tract malformation. In section c, its showing chromosome that is related to HLHS.

Hub genes in HLHS



In figure 2, the examined expression shows hub genes in black and pink model. The controls is the GSE77798 database using WGCNA. (weighted correction network analysis)
The results were gathered from HLHS mRNA expression profiling datasets (specifically HLHS mice)

Gene expression in HLHS



In figure 3, there is comparison between the control gene expression and spliced transcripts and the HLHS gene expression and HLHS spliced transcripts.
The tissue used in this experiment was collected from neonates with HLHS.

CONCLUSION

- In all three articles the important findings are used to articulate a single molecular mechanism that tries to explain why hypoplastic left heart syndrome occurs.
- 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 transcripts.
- The need for further studies is essentially for finalizing a molecular mechanism and the prevention of the hypoplastic left heart syndrome.

REFERENCE

Ricci, M., Xu, Y., Hammond, H. L., Willoughby, D. A., Nathanson, L., Rodriguez, M. M., Vatta, M., Lipshultz, S. E., & Lincoln, J. (2012). Myocardial alternative RNA splicing and gene expression profiling in early stage hypoplastic left heart syndrome. *PloS one*, *7*(1), e29784. https://doi.org/10.1371/journal.pone.0029784

McBride, K. L., Zender, G. A., Fitzgerald-Butt, S. M., Koehler, D., Menesses-Diaz, A., Fernbach, S., Lee, K., Towbin, J. A., Leal, S., & Belmont, J. W. (2009). Linkage analysis of left ventricular outflow tract malformations (aortic valve stenosis, coarctation of the aorta, and hypoplastic left heart syndrome). *European journal of human genetics : EJHG*, *17*(6), 811–819. https://doi.org/10.1038/ejhg.2008.255

Liu, X., Shang, H., Li, B. *et al.* Exploration and validation of hub genes and pathways in the progression of hypoplastic left heart syndrome via weighted gene co-expression network analysis. *BMC Cardiovasc Disord* **21**, 300 (2021). https://doi.org/10.1186/s12872-021-02108-0

Contact

gcuahutle@mercy.edu

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