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1. Anticancer Res. 2017 Feb;37(2):637-644.

Telomere Length and Risk of Hepatocellular Carcinoma: A Nested Case-control Study in Taiwan Cancer Screening Program Cohort.

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Abstract

BACKGROUND:

Telomere length (TL) measured in peripheral blood leucocytes (PBL) might be a useful biomarker to identify elevated cancer risk.

PATIENTS AND METHODS:

A case-control study which included 268 newly-diagnosed HCC cases and 536 matched controls, was conducted. Absolute TL in PBL was analyzed by quantitative real-time PCR.

RESULTS:

The overall median length of TL was not statistically shorter in HCC cases compared to healthy controls. However, we found a significant synergistic effect of longer TL and HCV infection to increase HCC risk with a relative excess risk of 6.86 (95% CI: 2.14-11.58). Among HCC cases, significant shorter TLs were observed for <5 years (OR=3.93, 95% CI: 2.00-7.72); 5-10 years (OR=2.16, 95% CI: 1.10-4.24) compared to >10 years prior to diagnosis.

CONCLUSION:

Shorter PBL TL alone was not significantly associated with increased HCC risk. Among HCC cases, significant shorter TLs were observed for <5 years prior to diagnosis.

PMCID: PMC5377930 [Available on 2018-02-01]
PMID: 28179311 [Indexed for MEDLINE]
Similar articles

2. BMC Cancer. 2017 Jan 5;17(1):24. doi: 10.1186/s12885-016-2997-3.

Association between telomere length and the risk of colorectal cancer: a meta-analysis of observational studies.

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Abstract

BACKGROUND:

Human chromosomes are capped and stabilized by telomeres. Telomere length regulates a 'cellular mitotic clock' that defines the number of cell divisions and hence, cellular life span. This study aimed to synthesize the evidence on the association between peripheral blood leucocytes (PBL) telomere length and the risk of colorectal cancer (CRC).

METHODS:

We searched relevant studies in electronic databases. When two or more observational studies reported the same outcome measures, we performed pooled analysis. All the analyses were performed on PBL using PCR. The odds ratio (OR) and its 95% confidence interval (CI) were used to assess the strength of association.

RESULTS:

Seven studies (with 8 datasets) were included in this meta-analysis; 3 prospective studies, 3 retrospective studies and 1 study with a separate prospective and retrospective designs. The pooled analysis of 4 prospective studies (summary OR 1.01, 95% CI: 0.77-1.34, I^2:30%) and 4 retrospective studies (summary OR 1.65, 95% CI: 0.96-2.83, I^2:96%) showed no relationship between PBL telomere length and the CRC risk. A subgroup analysis of 2 prospective studies exclusively on females also showed no association between PBL telomere length and the CRC risk (summary OR, 1.17, 95% CI:0.72-1.91, I^2:57%).

CONCLUSION:

The current analysis is insufficient to provide evidence on the relationship between PBL telomere length and the risk of CRC. Findings suggest that there may be a complex relationship between PBL telomere length and the CRC risk or discrepancy between genetics, age of patients and clinical studies. Future well powered, large prospective studies on the relationship between telomere length and the risk of CRC, and the investigations of the biologic mechanisms are recommended.

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3. PLoS One. 2016 Nov 23;11(11):e0166828. doi: 10.1371/journal.pone.0166828. eCollection 2016.

Telomere Length and Survival of Patients with Hepatocellular Carcinoma in the United States.

Yang B[1], Shebl FM[2], Sternberg LR[3], Warner AC[3], Kleiner DE[4], Edelman DC[4], Gomez A[4], Dagnall CL[1,5], Hicks BD[1,5], Altekruse SF[6], Hernandez BY[7], Lynch CF[8], Meltzer PS[4], McGlynn KA[1].

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Abstract

BACKGROUND:

Telomere shortening is an important molecular event in hepatocellular carcinoma (HCC) initiation; however, its role in HCC progression and prognosis is less clear. Our study aimed to examine the association of telomere length with survival of patients with HCC.

METHODS:

We measured telomere length in tumor and adjacent non-tumor tissues from 126 persons with HCC in the United States (U.S.) who were followed for mortality outcomes. Relative telomere length (RTL) was measured by a monochrome multiplex quantitative polymerase chain reaction assay. Multivariable Cox proportional hazards modeling was used to calculate hazard ratios (HRs) and 95% CIs for the association between telomere length and all-cause mortality. We also examined associations between telomere length and patient characteristics using multiple linear regression.

RESULTS:

During a mean follow-up of 6.0 years, 79 deaths occurred among 114 individuals for whom survival data were available. The ratio of RTL in tumor relative to non-tumor tissue was greater for individuals with regional or distant stage tumors (0.97) than localized stage tumors (0.77), and for individuals with grade III or IV tumors (0.95) than grade II (0.88) or grade I (0.67) tumors. An RTL ratio ≥1 was not associated with survival (HR 0.92, 95% CI 0.55, 1.55) compared to a ratio <1, after adjusting for age at diagnosis, sex, tumor stage and tumor size. Similarly, RTL in the tumor and non-tumor tissue, respectively, were not associated with survival.

CONCLUSIONS:

This U.S. based study found that telomeres may be longer in more aggressive HCCs. There was no evidence, however, that telomere length was associated with survival of patients with HCC. Future investigations are warranted to clarify the role of telomere length in HCC prognosis.

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Conflict of interest statement

The authors have declared that no competing interests exist.

4. Cancer Epidemiol Biomarkers Prev. 2016 Dec;25(12):1537-1549. Epub 2016 Aug 26.

Systematic Review of Genetic Variation in Chromosome 5p15.33 and Telomere Length as Predictive and Prognostic

Biomarkers for Lung Cancer.

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Abstract

Lung cancer remains the leading cause of cancer mortality worldwide. Known histomolecular characteristics and genomic profiles provide limited insight into factors influencing patient outcomes. Telomere length (TL) is important for genomic integrity and has been a growing area of interest as agents targeting telomerase are being evaluated. Chromosome 5p15.33, an established cancer susceptibility locus, contains a telomerase-regulatory gene, TERT, and CLPTM1L, a gene associated with cisplatin-induced apoptosis. This review offers a summary of the clinical utility of 5p15.33 polymorphisms and TL. A total of 621 abstracts were screened, and 14 studies (7 for 5p15.33, 7 for TL) were reviewed. Endpoints included overall survival (OS), progression-free survival (PFS), therapy response, and toxicity. Of the 23 genetic variants identified, significant associations with OS and/or PFS were reported for rs401681 (CLPTM1L), rs4975616 (TERT-CLPTM1L), and rs2736109 (TERT). Both shorter and longer TL, in tumor and blood, was linked to OS and PFS. Overall, consistent evidence across multiple studies of 5p15.33 polymorphisms and TL was lacking. Despite the potential to become useful prognostic biomarkers in lung cancer, the limited number of reports and their methodologic limitations highlight the need for larger, carefully designed studies with clinically defined subpopulations and higher resolution genetic analyses. Cancer Epidemiol Biomarkers Prev; 25(12); 1537-49. ©2016 AACR.

PMID: 27566420
Similar articles

5. Cancer Med. 2016 Sep;5(9):2657-65. doi: 10.1002/cam4.810. Epub 2016 Jul 6.

Leukocyte telomere length in relation to the risk of Barrett's esophagus and esophageal adenocarcinoma.

Wennerström EC[1,2], Risques RA[3], Prunkard D[3], Giffen C[4], Corley DA[5], Murray LJ[6], Whiteman DC[7,8], Wu AH[9], Bernstein L[10], Ye W[11], Chow WH[12], Vaughan TL[13], Liao LM[14].

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Abstract

Chronic inflammation and oxidative damage caused by obesity, cigarette smoking, and chronic gastroesophageal reflux disease (GERD) are major risk factors associated with Barrett's esophagus (BE) and esophageal adenocarcinoma (EAC). EAC has been increasing the past few decades, and early discovery and treatment are crucial for survival. Telomere shortening due to cell division and oxidative damage may reflect the impact of chronic inflammation and could possibly be used as predictor for disease development. We examined the prevalence of shorter leukocyte telomere length (LTL) among individuals with GERD, BE, or EAC using a pooled analysis of studies from the Barrett's and Esophageal Adenocarcinoma Consortium (BEACON). Telomere length was measured in leukocyte DNA samples by Q-PCR. Participants included 1173 patients (386 with GERD, 384 with EAC, 403 with BE) and 736 population-based controls. The association of LTL (in tertiles) along the continuum of disease progression from GERD to BE to EAC was calculated using study-specific odds ratios (ORs) and 95% confidence intervals (CIs) from logistic regression models adjusted for potential confounders. Shorter LTL were less prevalent among GERD patients (OR 0.57; 95% CI: 0.35-0.93), compared to population-based controls. No statistically significant increased prevalence of short/long LTL among individuals with BE or EAC was observed. In contrast to some earlier reports, our findings add to the evidence that leukocyte telomere length is not a biomarker of risk related to the etiology of EAC. The findings do not suggest a relationship between LTL and BE or EAC.

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6. J Dermatol Sci. 2015 Dec;80(3):168-74. doi: 10.1016/j.jdermsci.2015.08.003. Epub 2015 Aug 22.

Telomere length and the risk of cutaneous melanoma and non-melanoma skin cancer: a review of the literature and meta-analysis.

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Abstract

There is much evidence supporting the role of telomeres in cancer pathogenesis, however the studies that investigated the association between telomere length and skin cancer risk provided inconsistent results. To help clarify this issue, we performed a systematic review and meta-analysis of published papers on the association between peripheral leukocytes telomere length (PLTL) and the risk of cutaneous melanoma and non-melanoma skin cancer (NMSC). We calculated summary relative risks (SRR) and 95% confidence intervals (95% CI) using random effect models with maximum likelihood estimates, and explored causes of between-studies heterogeneity of risk estimates. We included 1629 cutaneous melanoma and 1439 NMSC from eight independent studies published until March 2015. The SRR of cutaneous melanoma for those in the lowest (vs. highest) category of PLTL distribution was 0.25 (95% CI 0.09-0.67). The results were less clear for NMSC, with two studies reporting no association and one study showing an increase in risk for those in the lowest (vs. highest) category of PLTL distribution. For both cutaneous melanoma and NMSC, the between-studies heterogeneity was large, mainly due to inclusion of hospital-based case-control studies. Our meta-analysis shows evidence of an association between short PLTL and reduced risk for cutaneous melanoma.

PMID: 26341697 [Indexed for MEDLINE]
Similar articles

7. Int J Clin Exp Pathol. 2015 May 1;8(5):5666-73. eCollection 2015.

Correlations of telomere length, P53 mutation, and chromosomal translocation in soft tissue sarcomas.

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Abstract

BACKGROUND:

Soft tissue sarcomas (STSs) are a heterogeneous group of malignant tumors that can be divided into specific reciprocal translocation associated in STSs (SRTSs) and nonspecific reciprocal translocation associated in STSs (NRTSs). Telomeres play a key role in maintaining chromosomal stability; pathological telomere elongation is found in a number of cancers. In this study, we aimed to assess telomere lengths in the two types of sarcomas. Twenty formalin-fixed paraffin-embedded (FFPE) archival tissues, namely, 10 sarcomas with characteristic translocations and 10 without characteristic translocations, were included in this study. Expression levels of special fusion gene transcripts were detected in these tumors by reverse transcription polymerase chain reaction. Telomere lengths were assessed by fluorescence in situ hybridization. Results showed that in 10 of the 10 cases of SRTSs, telomere lengths were similar to or reduced compared with the surrounding normal cells. Telomere lengths were elongated in eight of 10 cases of NRTSs, but reduced in two cases. The difference in telomere length was statistically significant in the two types of sarcomas (P=0.001). Upon combining the P53 mutation status, we found that the telomere length was short in eight cases, and only one case demonstrated p53 mutation. However, the telomere length was long in eight cases, and p53 mutation was observed in five cases. These data suggested that p53 mutation was accompanied with long telomeres, and telomeres possibly play an important role in NRTSs. Therefore, telomere-targeting therapy may lead to novel therapeutic strategies to improve treatment of NRTS patients.

PMCID: PMC4503150 **Free PMC Article**
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Similar articles

8. Trends Biochem Sci. 2015 Sep;40(9):504-15. doi: 10.1016/j.tibs.2015.06.003. Epub 2015 Jul 15.

Replicating through telomeres: a means to an end.

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Abstract

Proper replication of the telomeric DNA at chromosome ends is critical for preserving genome integrity. Yet, telomeres present challenges for the replication machinery, such as their repetitive and heterochromatic nature and their potential to form non-Watson-Crick structures as well as the fact that they are transcribed. Numerous telomere-bound proteins are required to facilitate progression of the replication fork throughout telomeric DNA. In particular, shelterin plays crucial functions in telomere length regulation, protection of telomeres from nuclease degradation, control of DNA damage response at telomeres, and the recruitment of associated factors required for telomere DNA processing and replication. In this review we discuss the recently uncovered functions of mammalian telomere-specific and telomere-associated proteins that facilitate proper telomere replication.

PMID: 26188776 [Indexed for MEDLINE]

Similar articles

9. J Endocrinol Invest. 2015 Nov;38(11):1243-6. doi: 10.1007/s40618-015-0298-3. Epub 2015 May 8.

Telomere length and telomerase expression in pituitary tumors.

Martins CS[1], Santana-Lemos BA[2,3], Saggioro FP[4], Neder L[4], Machado HR[5], Moreira AC[2], Calado RT[2,3], de Castro M[6].

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Abstract

PURPOSE:

Telomere dysfunction and telomerase activation underlie cancer transformation. This study aims to investigate the contribution of telomere biology to pituitary tumor behavior.

SUBJECTS AND METHODS:

Samples from 50 patients with pituitary tumors (11 ACTH-secreting, 18 GH-secreting, and 21 non-secreting tumors) and 7 subjects without pituitary lesions were collected. The expressions of telomerase essential components TERT and TERC and tumor telomere content were measured by quantitative PCR techniques.

RESULTS:

Telomerase (TERT) expression was detected in 36% of tumors. No correlation was observed between TERT and TERC expression level and tumor size in any tumor type. There was no association between gene expression and clinical findings. Telomere content (T/S ratio) was similar between pituitary adenomas (0.39 ± 0.16) and normal pituitaries (0.47 ± 0.12; p = 0.24) and also was between the different adenoma types: ACTH-secreting (0.43 ± 0.08), GH-

secreting (0.31 ± 0.12), and non-secreting (0.42 ± 0.20; p = 0.10) tumors.

CONCLUSIONS:

The telomere content and expression of telomerase components are comparable between normal pituitary glands and tumor tissues, suggesting that telomere biology does not play an important role in pituitary tumor development.

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