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Ancestry, Telomere Length, and Atherosclerosis Risk

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Cardiovascular profiles in African Americans and European Americans

African Americans (AfAms) and European Americans (EuAms) display differences in cardiovascular risk factors and mortality from cardiovascular disease (CVD). AfAms show a higher prevalence than EuAms in most cardiovascular risk factors[1]. These include high blood pressure[1–3], left ventricular hypertrophy[1], obesity[1] and type 2 diabetes mellitus[1,4]. National US epidemiological data indicate that dyslipidemia is less prevalent in AfAms than EuAms[1]. However, based on the Multi-Ethnic Study of Atherosclerosis (MESA), AfAms and EuAms have a similar prevalence of dyslipidemia, which is less controlled in AfAms[5]. Other studies also found lower rates of cholesterol testing, dyslipidemia awareness, treatment, and therapeutic goal attainments among AfAms than EuAms[6,7]. In some of these studies, ethnic disparities were attenuated by adjustment for social factors and healthcare access[5], while in other studies they were not[7]. Notably, compared with EuAms and Mexican-Americans, AfAms displayed a higher risk of having one or two of the three major cardiovascular risk factors, i.e., diabetes, dyslipidemia and hypertension; compared with EuAms, AfAms were also more likely to have all three conditions[8]. While these ethnic differences might account for the higher CVD mortality in AfAms than EuAms[9,10], the ethnic differences cannot explain consistent observations of less atherosclerosis in the form of plaques, arterial calcifications and occluded coronary arteries in AfAms than in EuAms (Table 1)[1,11–13]. This applies to individuals without or with similar cardiovascular risk factors.[12–18].

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Key Tenet

Here we propose that longer telomeres in AfAms than EuAms[19–21] may attenuate the atherosclerotic risk in AfAms and explain in part the mortality cross-over between elderly AfAms and EuAms. First, however, we provide a brief background on the biology, epidemiology and evolution of telomeres.

Telomere length dynamics

Comprising TTAGGG tandem repeats and their telomere-binding protein complex (shelterin), mammalian telomeres safeguard the ends of the chromosomes[22]. As somatic cells divide, their telomeres undergo progressive shortening, a process that ultimately leads to cessation of replication, i.e., replicative senescence[23]. In humans, embryonic stem cells express the enzyme telomerase, a reverse transcriptase that adds telomere repeats to the ends of the chromosomes, thereby preventing telomere shortening during early intra-uterine life[24]. However, as telomerase is repressed in somatic tissues during extra-uterine life, age-dependent telomere shortening in these tissues largely reflects the replicative histories of stem cells/progenitor cells on top of the somatic cell hierarchy[25]. In this way, TL in skeletal muscle, a minimally proliferative tissue, is longer than TL in leukocytes, which represent the highly proliferative hematopoietic system[26]. Moreover, to accommodate the demands of the growing soma, stem cell/progenitor cell replication is much faster during growth and development. Therefore, the rate of telomere shortening is much faster in childhood than during adulthood[26,27].

Most epidemiological studies have used leukocyte TL (LTL) as a proxy for TL in other somatic tissues, given that TL is ‘proportional’ across somatic tissues within the individual, i.e., a person with short (or long) TL in one somatic tissue has short (or long) TL in other somatic tissues[26], regardless of the replicative history of these tissues. This is because at birth and throughout the life course the variation in TL, which is highly heritable[28], across individuals is much wider than TL variation across somatic tissues within the individual.

Telomere length evolution across mammals

Why is TL in humans as long as it is? Moreover, is this length arbitrary or is it determined by evolutionary principles? TL is short and telomerase activity in somatic tissues is repressed in long-lived, large mammals compared with short-lived, small mammals[29]. One potential explanation for these findings is that short telomeres and repressed telomerase diminish cancer risk by curtailing replicative potential[25,30,31]. The trade-off might be diminished tissue repair through cell replication and therefore increased propensity to degenerative diseases. Thus, across long-lived, large mammals, telomeres might converge to an optimal length that strikes a balance between cancer and degenerative diseases during the life course of animals from a given species.

TL is short in humans compared to most mammals[29]. Still, TL variance across the general population is considerable, amounting to 3–4 kilobases (kb)[27,32]. Detrimental mutations in telomere maintenance genes can result in specific diseases that are the outcome of extremely short TL[29], but these diseases are relatively rare. The question then is whether in the general

population, individuals with constitutively short telomeres have a higher risk for degenerative diseases. This might be the case for atherosclerosis, which is perhaps the main degenerative disease that afflicts contemporary humans.

Telomere length and atherosclerosis

Two meta-analyses concluded that short LTL is associated with CVD, based on data from individuals of principally European ancestry[33,34]. In general, short LTL is associated with arterial aging, as expressed in arterial stiffness[35], coronary artery calcifications[36,37], and severity of atherosclerotic plaques in the coronary arteries[38,39] and the carotid arteries[40–43].

What might be the explanation for these associations? For years, the dominant thinking in telomere epidemiology has been that LTL is a passive biomarker of the cumulative burden of inflammation and oxidative stress during adult life. Thus, individuals with a higher burden of inflammation and oxidative stress, including obese individuals, smokers, patients with diabetes and those with atherosclerotic CVD, were presumed to have a faster age-dependent LTL shortening during adult life[44].

However, the variation in LTL across adults of the same age are largely the outcome of the inter-individual variation in LTL during the first two decades of life[27,32] rather than inter-individual variation in the rate of LTL attrition during adulthood[45]. Thus, short LTL might precede by several decades the manifestations of atherosclerotic CVD and related metabolic risks[46,47]. For instance, short LTL is observed long before the onset of carotid lesions and predicts their progression over time[40]. Short LTL also precedes the clinical manifestation of insulin resistance[47,48]. Moreover, not only short LTL, but also single nucleotide polymorphisms (SNPs) associated with short LTL[39,49] are associated with increased propensity for atherosclerotic CVD. These findings principally exclude reverse causality. i.e., atherosclerotic CVD engenders short LTL, supporting the thesis that short LTL antecedes the clinical manifestations of atherosclerotic CVD.

Longer leukocyte telomere length in individuals of recent African ancestry than in individuals of recent European ancestry

In light of the short LTL-CVD association, it is noteworthy that compared with EuAms, AfAms display not only a longer LTL[19–21,50], but also a higher prevalence than EuAMs of SNPs associated with a longer TL[51]. Although the underlying reasons are not well understood, findings that LTL of sub-Saharan Africans is even longer than that of AfAms suggest that the northbound migration from equatorial Africa might have resulted in TL shortening in Europeans[20]. One potential cause of telomere shortening in Europeans might be melanoma. Previous investigators had excluded melanoma as an evolutionary force[52]. However, given that not only longer LTL but also alleles associated with longer LTL are over-represented in patients with melanoma[53,54], without evolutionary-driven telomere shortening, whites of European ancestry would have been more susceptible to melanoma than they are at present[20].

Telomere length and “mortality crossover” between AfAms and EuAms

Overall, AfAms have a shorter life expectancy than EuAms. However, after the age of 80, AfAms display a longer life expectancy than EuAms– a phenomenon referred to as the “mortality crossover”[55,56]. Corti et al.[55] reported that after the age of 80 AfAms had a 25% lower risk of all-cause mortality than EuAms, a difference that was attributed to a 55% lower atherosclerotic coronary heart disease mortality in AfAms. Other studies suggested that socio-economic status and other environmental factors explain the mortality crossover between AfAms and EuAms[56,57]. However, in most of these studies the mortality crossover remained significant after adjustments for environmental factors[55]. Thus, the mortality crossover might be related to different trajectories of coronary heart disease, which could be partially explained by the longer LTL in AfAms vs. EuAms.

Conclusions and perspectives

Since the inception of the discipline of telomere epidemiology, LTL has been regarded as a passive “biomarker” of aging and atherosclerotic CVD. However, an emerging body of research points to an active role of TL in atherosclerosis, which is reason enough to seek further insights into the potential role of TL in the different propensities of AfAms and EuAms to atherosclerotic CVD. The longer LTL in AfAms than EuAms might partially explain the less susceptibility of AfAms than EuAms to atherosclerosis and their longer life expectancy after the age of eighty years. Perhaps, the first step towards gaining further insight into these intriguing ethnic differences is to perform large-scale genome-wide association studies of LTL in multiple ethnic groups. Identifying new LTL- associated SNPs might help construct a comprehensive genetic map leading to mechanistic insights into the role of TL, as expressed in LTL, in a host of human diseases..

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References

1. Heart Disease and Stroke Statistics - 2015 Update A Report From the American Heart Association. Circulation. 2015; 131:e29–e322. [PubMed: 25520374]
2. Muntner P, Lewis CE, Diaz KM, Carson AP, Kim Y, Calhoun D, et al. Racial differences in abnormal ambulatory blood pressure monitoring measures: Results from the Coronary Artery Risk Development in Young Adults (CARDIA) study. Multicenter Study; Randomized Controlled Trial. Am J Hypertens. 2015; 28:640–648. [PubMed: 25376639]
3. Egan BM, Zhao Y, Axon RN. US trends in prevalence, awareness, treatment, and control of hypertension, 1988–2008. JAMA. 2010; 303:2043–2050. [PubMed: 20501926]
4. Cowie CC, Rust KF, Ford ES, Eberhardt MS, Byrd-Holt DD, Li C, et al. Full accounting of diabetes and pre-diabetes in the U.S. population in 1988–1994 and 2005–2006. Diabetes Care. 2009; 32:287–294. [PubMed: 19017771]
5. Goff DC Jr, Bertoni AG, Kramer H, Bonds D, Blumenthal RS, Tsai MY, Psaty BM. Dyslipidemia prevalence, treatment, and control in the Multi-Ethnic Study of Atherosclerosis (MESA): gender, ethnicity, and coronary artery calcium. Circulation. 2006; 113:647–656. [PubMed: 16461837]

6. Hyre AD, Muntner P, Menke A, Raggi P, He J. Trends in ATP-III-defined high blood cholesterol prevalence, awareness, treatment and control among US adults. Ann Epidemiol. 2007; 17:548–555. [PubMed: 17395483]
7. Zweifler RM, McClure LA, Howard VJ, Cushman M, Hovater MK, Safford MM, et al. Racial and geographic differences in prevalence, awareness, treatment and control of dyslipidemia: the reasons for geographic and racial differences in stroke (REGARDS) study. Neuroepidemiology. 2011; 37:39–44. [PubMed: 21822024]
8. Fryar CD, Hirsch R, Eberhardt MS, Yoon SS, Wright JD. Hypertension, high serum total cholesterol, and diabetes: racial and ethnic prevalence differences in US adults, 1999–2006. NCHS Data Brief. 2010; 36:1–8.
9. Mosca L, Hammond G, Mochari-Greenberger H, Towfighi A, Albert MA. Fifteen-year trends in awareness of heart disease in women: results of a 2012 American Heart Association national survey. Circulation. 2013; 127:1254–1263. [PubMed: 23429926]
10. Agency for Healthcare Research and Quality. 2008 National Healthcare Disparities Report. Rockville, MD: US Department of Health and Human Services, Agency for Healthcare Research and Quality; Mar. 2009 AHRQ publication No. 09-0002
11. Loria CM, Liu K, Lewis CE, Hulley SB, Sidney S, Schreiner PJ, et al. Early adult risk factor levels and subsequent coronary artery calcification: the CARDIA Study. J Am Coll Cardiol. 2007; 49:2013–2020. [PubMed: 17512357]
12. Bild DE, Detrano R, Peterson D, Guerci A, Liu K, Shahar E, et al. Ethnic differences in coronary calcification: the Multi-Ethnic Study of Atherosclerosis (MESA). Circulation. 2005; 111:1313–1320. [PubMed: 15769774]
13. Stalls CM, Triplette M, Viera A, Pathman D, Cohen M, Rossi J. The association between body mass index and coronary artery disease severity: A comparison of black and white patients. Am Heart J. 2014; 167:514–520. [PubMed: 24655700]
14. Budoff M, Yang T, Shavelle R, Lamont D, Brundage B. Ethnic differences in coronary atherosclerosis. J Am Coll Cardiol. 2002; 39:408–412. [PubMed: 11823077]
15. Wade A, Fedyna S, Mehta NN, St Clair C, Ginwala N, Krishna R, et al. Type 2 diabetes does not attenuate racial differences in coronary calcification. Diabetes Res Clin Pract. 2011; 91:101–107. [PubMed: 21067835]
16. Suzuki T, Voeks J, Zakai NA, Jenny NS, Brown TM, Safford MM, et al. Metabolic syndrome, C-reactive protein, and mortality in U.S. Blacks and Whites: The reasons for geographic and racial differences in stroke (REGARDS) Study. Diabetes Care. 2014; 37:2284–2290. [PubMed: 24879838]
17. Zhao W, Katzmarzyk PT, Horswell R, Wang Y, Johnson J, Hu G. HbA1c and coronary heart disease risk among diabetic patients. Diabetes Care. 2014; 37:428–435. [PubMed: 24130365]
18. Divers J, Wagenknecht LE, Bowden DW, Carr JJ, Hightower RC, Xu J, Langefeld CD, Freedman BI. Ethnic differences in the relationship between albuminuria and calcified atherosclerotic plaque. Diabetes Care. 2010; 33:131–138. [PubMed: 19825824]
19. Hunt SC, Chen W, Gardner JP, Kimura M, Srinivasan SR, Eckfeldt JH, et al. Leukocyte telomeres are longer in African Americans than in whites: the National Heart, Lung, and Blood Institute Family Heart Study and the Bogalusa Heart Study. Aging Cell. 2008; 7:451–458. [PubMed: 18462274]
20. Hansen M, Hunt S, Stone R, Horvath K, Herbig U, Ranciaro A, et al. Shorter telomere length in Europeans than in Africans due to polygenetic adaptation. Hum Mol Genet. 2016; 25:2324–2330. [PubMed: 26936823]
21. Elbers CC, Garcia ME, Kimura M, Cummings SR, Nalls MA, Newman AB, et al. Comparison between southern blots and qPCR analysis of leukocyte telomere length in the health ABC study. Gerontol A Biol Sci Med Sci. 2014; 69:527–531.
22. Blackburn EH. Telomeres and telomerase: their mechanisms of action and the effects of altering their functions. FEBS Lett. 2005; 579:859–862. [PubMed: 15680963]
23. Harley CB. Telomere loss: mitotic clock or genetic time bomb. Mutat Res. 1991; 256:271–282. [PubMed: 1722017]

24. Nugent CI, Lundblad V. The telomerase reverse transcriptase: components and regulation. Genes Dev. 1998; 12:1073–1085. [PubMed: 9553037]
25. Blackburn EH, Epel ES, Lin J. Human telomere biology: A contributory and interactive factor in aging, disease risks, and protection. Science. 2015; 350:1193–1198. [PubMed: 26785477]
26. Daniali L, Benetos A, Susser E, Kark JD, Labat C, Kimura M, et al. Telomeres shorten at equivalent rates in somatic tissues of adults. Nat Commun. 2013; 4:1597. [PubMed: 23511462]
27. Aubert G, Baerlocher GM, Vulto I, Poon SS, Lansdorp PM. Collapse of telomere homeostasis in hematopoietic cells caused by heterozygous mutations in telomerase genes. PLoS Genet. 2012; 8:e1002696. [PubMed: 22661914]
28. Hjelmborg JB, Dalgård C, Möller S, Steenstrup T, Kimura M, Christensen K, et al. The heritability of leucocyte telomere length dynamics. J Med Genet. 2015; 52:297–302. [PubMed: 25770094]
29. Gomes NM, Ryder OA, Houck ML, Charter SJ, Walker W, Forsyth NR, et al. Comparative biology of mammalian telomeres: hypotheses on ancestral states and the roles of telomeres in longevity determination. Aging Cell. 2011; 10:761–768. [PubMed: 21518243]
30. Aviv A, Kark JD, Susser E. Telomeres, atherosclerosis, and human longevity: a causal hypothesis. Epidemiology. 2015; 26:295–299. [PubMed: 25774608]
31. Stanley SE, Armanios M. The short and long telomere syndromes: paired paradigms for molecular medicine. Curr Opin Genet Dev. 2015; 33:1–9. [PubMed: 26232116]
32. Factor-Litvak P, Susser E, Kezios K, McKeague I, Kark JD, Hoffman M, et al. Leukocyte telomere length in newborns: Implications for the role of telomeres in human disease. Pediatrics. 2016; 137:e20153927. [PubMed: 26969272]
33. D'Mello MJ, Ross SA, Briel M, Anand SS, Gerstein H, Paré G. The association between shortened leukocyte telomere length and cardio-metabolic outcomes: a systematic review and meta-analysis. Circ Cardiovasc Genet. 2015; 8:82–90. [PubMed: 25406241]
34. Haycock PC, Heydon EE, Kaptoge S, Butterworth AS, Thompson A, Willeit P. Leucocyte telomere length and risk of cardiovascular disease: systematic review and meta-analysis. BMJ. 2014; 349:g4227. [PubMed: 25006006]
35. Benetos A, Okuda K, Lajemi M, Kimura M, Thomas F, Skurnick J, et al. Telomere length as an indicator of biological aging: the gender effect and relation with pulse pressure and pulse wave velocity. Hypertension. 2001; 37:381–385. [PubMed: 11230304]
36. Hunt SC, Kimura M, Hopkins PN, Carr JJ, Heiss G, Province MA, Aviv A. Leukocyte telomere length and coronary artery calcium. Am J Cardiol. 2015; 116:214–218. [PubMed: 25960381]
37. Ormseth MJ, Solus JF, Oeser AM, Bian A, Gebretsadik T, Shintani A, et al. Telomere length and coronary atherosclerosis in rheumatoid arthritis. Rheumatol. 2016; 43:1469–1474.
38. Calvert PA, Liew TV, Gorenne I, Clarke M, Costopoulos C, Obaid DR, et al. Leukocyte telomere length is associated with high-risk plaques on virtual histology intravascular ultrasound and increased proinflammatory activity. Arterioscler Thromb Vasc Biol. 2011; 31:2157–2164. [PubMed: 21680897]
39. Scheller Madrid A, Rode L, Nordestgaard BG, Bojesen SE. Short telomere length and ischemic heart disease: Observational and genetic studies in 290 022 individuals. Clin Chem. 2016; 62:1140–1149. [PubMed: 27259814]
40. Chen S, Lin J, Matsuguchi T, Blackburn E, Yeh F, Best LG, Devereux RB, Lee ET, Howard BV, Roman MJ, Zhao J. Short leukocyte telomere length predicts incidence and progression of carotid atherosclerosis in American Indians: the Strong Heart Family Study. Aging. 2014; 6:414–27. [PubMed: 24902894]
41. Benetos A, Gardner JP, Zureik M, Labat C, Xiaobin L, Adamopoulos C, et al. Short telomeres are associated with increased carotid atherosclerosis in hypertensive subjects. Hypertension. 2004; 43:182–185. [PubMed: 14732735]
42. Fernández-Alvira JM, Fuster V, Dorado B, Soberón N, Flores I, Gallardo M, et al. Short Telomere Load, Telomere Length, and Subclinical Atherosclerosis: The PESA Study. J Am Coll Cardiol. 2016; 67:2467–2476. [PubMed: 27230041]
43. Huzen J, Peeters W, de Boer RA, Moll FL, Wong LS, Codd V, et al. Circulating leukocyte and carotid atherosclerotic plaque telomere length: interrelation, association with plaque

characteristics, and restenosis after endarterectomy. Arterioscler Thromb Vasc Biol. 2011; 31:1219–1225. [PubMed: 21372300]

44. Voghel G, Thorin-Trescases N, Farhat N, Nguyen A, Villeneuve L, Mamarbachi AM, et al. Cellular senescence in endothelial cells from atherosclerotic patients is accelerated by oxidative stress associated with cardiovascular risk factors. Mech Ageing Dev. 2007; 128:662–671. [PubMed: 18022214]
45. Benetos A, Kark JD, Susser E, Kimura M, Sinnreich R, Chen W, et al. Tracking and fixed ranking of leukocyte telomere length across the adult life course. Aging Cell. 2013; 12:615–621. [PubMed: 23601089]
46. Zhao J, Zhu Y, Lin J, Matsuguchi T, Blackburn E, Zhang Y, et al. Short leukocyte telomere length predicts risk of diabetes in American Indians: the strong heart family study. Diabetes. 2014; 63:354–362. [PubMed: 23949319]
47. Verhulst S, Dalgård C, Labat C, Kark Jd, Kimura M, Christensen K, et al. A Short Leukocyte Telomere Length is associated with development of Insulin resistance. Diabetologia. 2016; 59:1258–1265. [PubMed: 27020448]
48. Chen S, Yeh F, Lin J, Matsuguchi T, Blackburn E, Lee ET, et al. Short leukocyte telomere length is associated with obesity in American Indians: The strong heart family study. Aging. 2014; 6:380–389. [PubMed: 24861044]
49. Codd V, Nelson CP, Albrecht E, Mangino M, Deelen J, Buxton JL, et al. Identification of seven loci affecting mean telomere length and their association with disease. Nat Genet. 2013; 45:422–427. [PubMed: 23535734]
50. Lynch SM, Peek MK, Mitra N, Ravichandran K, Branas C, Spangler E, et al. Race, ethnicity, psychosocial factors, and telomere length in a multicenter setting. PLoS One. 2016; 11:e0146723. [PubMed: 26752285]
51. Hamad R, Tuljapurkar S, Rehkopf DH. Racial and socioeconomic variation in genetic markers of telomere length: A Cross-Sectional Study of U.S. Older Adults. EBioMedicine. 2016; 11:296–301. [PubMed: 27566956]
52. Osborne DL, Hames R. A life history perspective on skin cancer and the evolution of skin pigmentation. Am J Phys Anthropol. 2014; 153:1–8.
53. Caini S, Raimondi S, Johansson H, De Giorgi V, Zanna I, Palli D, Gandini S. Telomere length and the risk of cutaneous melanoma and non-melanoma skin cancer: a review of the literature and meta-analysis. J Dermatol Sci. 2015; 80:168–174. [PubMed: 26341697]
54. Iles MM, Bishop DT, Taylor JC, Hayward NK, Brossard M, Cust AE, et al. The effect on melanoma risk of genes previously associated with telomere length. J Natl Cancer Inst. 2014; 106
55. Corti MC, Guralnik JM, Ferrucci L, Izmirlian G, Leveille SG, Pahor M, et al. Evidence for a black-white crossover in all-cause and coronary heart disease mortality in an older population: the North Carolina EPESE. Am J Public Health. 1999; 89:308–314. [PubMed: 10076478]
56. Eberstein IW, Nam CB, Heyman KM. Causes of death and mortality crossovers by race. Biodemography Soc Biol. 2008; 54:214–228. [PubMed: 19350756]
57. Masters RK. Uncrossing the U.S. Black-White mortality crossover. The role of cohort forces in life course mortality risk. Demography. 2012; 49:773–796. [PubMed: 22729715]

Table 1

Prevalence of cardiovascular risk factors and of coronary atherosclerosis in African Americans and European Americans.

	EuAm Women	AfrAm Women	EuAm Men	AfrAm Men	Ref #
CV Risk Factors (%)					
Tobacco	19.8	15.4	22.6	22.8	1
HBP (140/90 or medic)	27.7	42.9	30.1	40.1	1
Diabetes (Medic or Gl>1.26g/l)	7.8	16.9	11.6	18.6	1
Dyslipidemia (>200mg/dl)	45.9	40.7	39.9	37.4	1
Obesity (BMI>30 kg/m2)	33.0	58.0	34.0	38.0	1
Coronary Atherosclerosis (%)					
Coronary Artery Calcifications					
33–45 years	11.3	4.9	17.6	5.2	11
45–84 years	44.6	36.5	70.4	52.1	12
*Coronary Stenosis					
% with Gensini score >10	26.7	14.9			13
% with severe stenosis	34.0	20.0			13

EuAm = European American; AfAm = African American;

*men and women together