## Exploring the Interplay Between Biological Age, Physical Activity and Cancer Incidence Across Educational Strata: a prospective study in the U.K. Biobank

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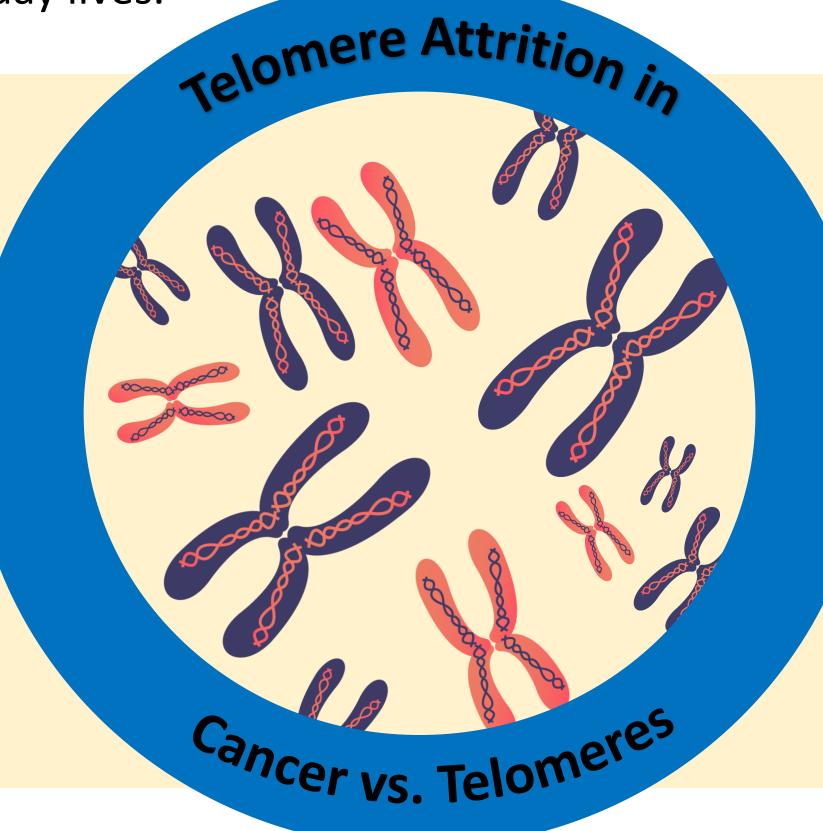
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Cancer incidence is at an ever growing rate with reason due to combination of aging population, emerging unfavorable health behaviours and exposure to environmental carcinogens (e.g. stress) in our everyday lives.

Critical telomere length leads to senescence but some escape and proliferate, becoming *immortal and carcinogenic* 

Telomere shortening is avoided in cancer cells through the reactivation of telomerase via genetic and epigenetic mechanisms



The shortening of telomeres to a critical length forces cells to become senescent, and a reason to loss of tissue function

Oxidative stress is considered to be the primary

Cancer cells present short telomere lengths possibly through genomic instability

factor for the accelerated degradation of telomere length

The "golden standard" proxy for biological age due to association with chronological age, sensitivy to exposures, predictive ability to disease and, inter-individual variation

## Health behaviours (HB) and socioeconomic conditions (SEC) have been associated with accelerated biological aging

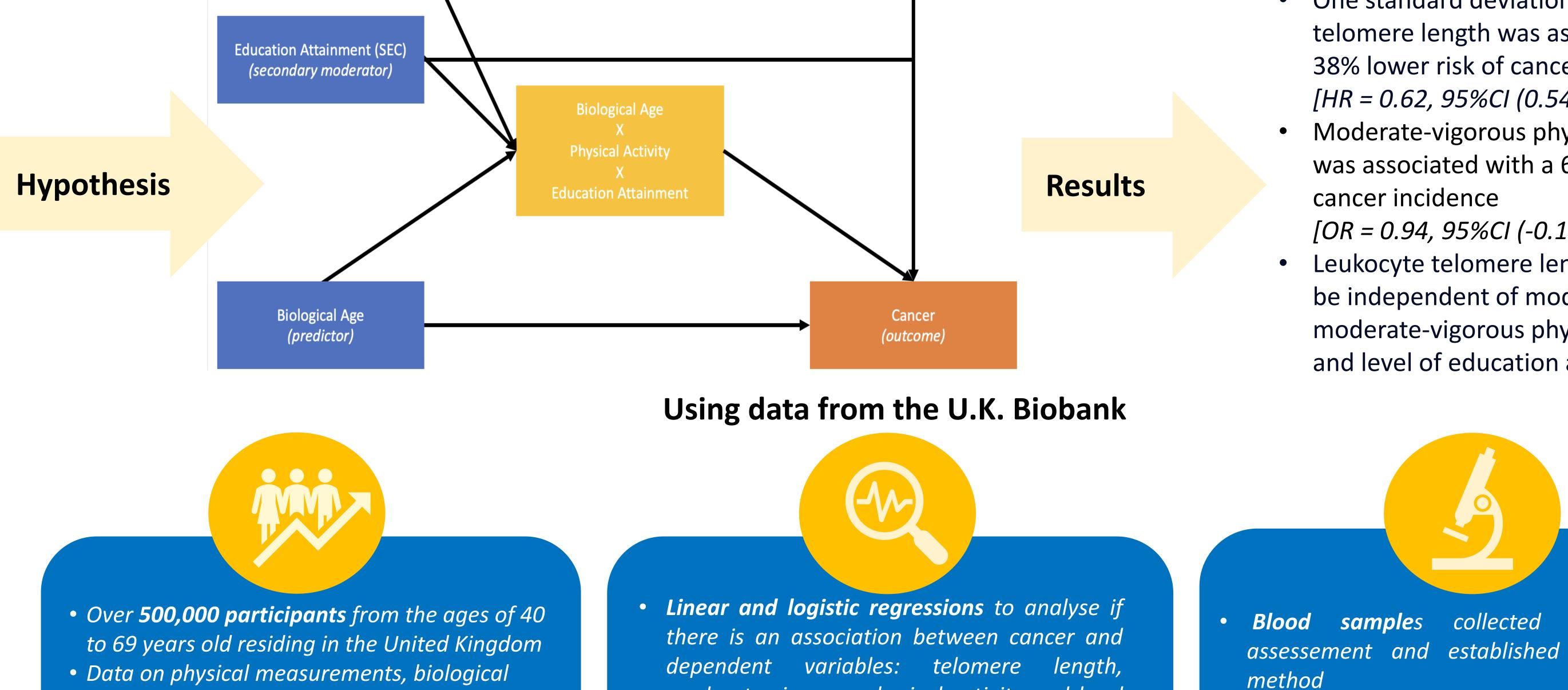
Data presents individuals that engage in high levels of physical activity have 10-20% reduced risk of cancer and exhibit longer telomeres however, results have been inconsistant in terms of telomere length.



Research presents individuals with lower levels of education attainment are associated with higher prevalence of preventable unhealthy behaviours and high cancer incidence.

Education and physical activity may reduce the impact of stress on biological aging and ultimately on the biological age and cancer association.

Physical Activity (HB) (primary moderator)



- **One standard deviation longer** telomere length was associated with 38% lower risk of cancer incidence [HR = 0.62, 95%CI (0.54 - 0.71)]
- Moderate-vigorous physical activity was associated with a 6% lower risk of cancer incidence [OR = 0.94, 95%CI(-0.1 - -0.02)]
- Leukocyte telomere length seems to be independent of moderators of moderate-vigorous physical activity and level of education attainment

samples, imaging scans, genomic sequences, questionnaires on health, lifestyle and medical history repeated annually • Data on cancer diganosis, screenings and deaths from the National Health Services

cancer registry



- Survival analysis using Cox proportional hazard regression with duration set from initial baseline assessment to date of cancer incidence or if no event, end of study

collected

baseline

with Q-PCR

at

- Reported as **T/S ratio**
- Adjusted for sex, age, ethnicity, and white blood cells to consider association of variation in telomere length
- > Leukocyte telomere length can be **a biomarker** for cancer incidence.
- > Factors possible to accelerate or stablize telomere length must be studied deeper with longer observation time.
- > Physical activity should be promoted as a preventative step to cancer incidence but insignificant in moderating the effect of accelerated biological aging on cancer.
- > Level of education attainment cannot be considered to be a moderator of the effect of telomere length on cancer incidence.



