Understanding how much TV is too much: A non-linear analysis of the association between television viewing time and adverse health outcomes.

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Abstract

Objective: To inform potential guideline development we investigated non-linear associations between television viewing time (TV time) and adverse health.

Methods: 490,966 UK Biobank participants; aged 37-73 years; recruited in 2006-2010; followed up until 2016-2018. Non-linear associations between self-reported TV time (h/day) and outcomes explored using penalised cubic splines in Cox proportional hazards adjusted for demographics and lifestyle. Population attributable and potential impact fractions calculated to contextualise TV time effects. Non-linear isotemporal substitution analyses used to investigate substituting TV time with alternative activities. Primary outcomes: mortality- all-cause, cardiovascular disease (CVD) and cancer; incidence- CVD and cancer; Secondary outcomes: incident myocardial infarction, stroke, heart-failure; colon, lung, breast, and prostate cancer.

Results: Those with non-communicable disease (109,867[22.4%]), CVD (32,243[6.6%]), and cancer (37,81[7.7%]) at baseline excluded from all-cause mortality, CVD, and cancer analyses, respectively. After 7.0 years (mortality) and 6.2 years (disease incidence) mean follow-up, there were 10,306 (2.7%) deaths, 24,388 (5.3%) CVD events, and 39,121 (8.7%) cancer events. Associations between TV time and all-cause and CVD mortality were curvilinear ($P_{\text{non-linear}} \leq 0.003$) with lowest risk observed <2h/day. Theoretically, 8.64% (95%CI 6.60-10.73) of CVD mortality is attributable to TV time. Limiting TV time to 2h/day, might have prevented, or at least delayed, 7.97% (95%CI 5.54-10.70) of CVD deaths. Substituting TV time with sleeping, walking, moderate or vigorous physical activity was associated with reduced risk for all outcomes when baseline levels of substitute activities were low.

Conclusion: TV time is associated with numerous adverse health outcomes. Future guidelines could suggest limiting TV time to less than 2h/day to reduce most of the associated adverse health.

Abbreviations:

CVD = Cardiovascular disease

NCD = Non-communicable disease

PAF = Population attributable fraction

PIF = Potential impact fraction

TV time = television viewing time

INTRODUCTION

Longer sedentary time is associated with adverse health outcomes.[1] In high income countries, television viewing (TV) predominates discretionary sedentary behaviour.[2,3] In the UK, individuals spend around 5h/day viewing audio-visual content across media devices, including 3.4h/day viewing TV (TV time).[4] TV time is associated with increased risk of type 2 diabetes, cardiovascular disease (CVD), certain cancers, and all-cause mortality.[5,6] TV time has a stronger association with adverse health than other sedentary behaviours (e.g. sitting at work, or whilst driving),[7,8] possibly due to clustering of TV time with other risk factors, such as unhealthy diets,[9] lower physical activity,[10] and poor sleep.[11] A stronger association may also be related to confounding by lower socioeconomic status,[12] as well as improved recall of self-reported TV time compared with other sedentary behaviours,[13] thereby reducing regression dilution bias.[14]

While minimising sedentary time may have health benefits, current national guidelines do not yet provide recommendations for appropriate levels due to the under-developed evidence base.[10,15] For similar reasons, no guidelines provide TV time recommendations. The TV time evidence base is limited partly due to the creation of ordinal or interval TV time variables, arbitrarily carving up what is likely to be a risk continuum. This is driven by a desire to provide clear public health messages but is also related to methodological limitations. Deriving and using categorical, as opposed to continuous variables, is often done to simplify analyses. However, categorising continuous data reduces precision through loss of data, increases the probability of Type I&II errors, and prevents visualisation of the underlying associations. Using continuous variables counters these limitations and provides clearer understanding of underlying associations.[16,17] Previous meta-analyses examining non-linear relationships between sedentary behaviour or TV time and adverse health is based on categorical data.[5,8,18]

We aimed to inform potential future TV time recommendations and provide a maximum TV time estimate by evaluating non-linear associations between TV time and a range of health outcomes and whether the associations differ by demographic and lifestyle factors. Population attributable fractions

were estimated to contextualise the importance of TV time as a modifiable lifestyle factor. Potential impact fractions were calculated to explore potential population benefits of limiting TV time. We used isotemporal substitution analysis to investigate theoretical benefits of replacing TV time with potentially healthier activities (sleeping, walking, moderate and vigorous physical activity).

METHODS

Study design and participants

Data were from UK Biobank, a prospective cohort. 502,536 participants; aged 37–73 years; recruited from the general population by postal invitation between March 13th 2006-October 1st 2010. At 22 locations across England, Scotland, and Wales, participants completed a self-administered, touch-screen questionnaire and a nurse-led interview. Trained staff took measurements (e.g. height, weight, and blood pressure).[19]

Exposure and covariates

TV time was based on free text responses to the touchscreen question at recruitment, "In a typical day, how many hours do you spend watching TV?" Socio-demographic factors such as sex and ethnicity were self-reported. Townsend deprivation index, a measure of socioeconomic status (SES), is derived from residential postcode using data on unemployment, car and home ownership, and household overcrowding.[20]

Height was measured to the nearest centimetre, using a Seca 202 stadiometer, and weight to the nearest 0.1 kg, using a Tania BC-418 body composition analyser. Body mass index (BMI; weight/height^2) was defined using the World Health Organization's categories. Physical activity was self-reported using the validated International Physical Activity Questionnaire.[21] Hand grip strength was measured to the nearest 0.1 kg using a Jamar J00105 hydraulic hand dynamometer and mean values from both hands were used in analyses.

Dietary intake of red meat, processed meat, oily fish, and fruit/vegetables was collected using the Oxford WebQ; a web-based, 24-hour recall questionnaire. Total calorie intake was estimated by

McCance and Widdowson's 'Composition of Foods. 7th edition'.[22] Frequency of alcohol intake (operationalised as units/week) and smoking status were self-reported. The presence of non-communicable disease (NCD; depression, bipolar disorder, schizophrenia, alcohol problems, substance abuse, eating disorders, cognitive impartment, dementia, Parkinson's disease, chronic pain syndrome, chronic obstructive pulmonary disease, chronic asthma, chronic liver diseases, hypertension, heart disease, stroke, diabetes, inflammatory diseases, arthritis, and cancer) at baseline was based on self-report of a physician's diagnosis verified at nurse-led interview. Medication for hypercholesterolaemia or hypertension was self-reported and confirmed at interview.

Outcomes

Primary outcome measures were mortality: all-cause, CVD and cancer; and incident (fatal and not-fatal events) CVD and cancer. Secondary outcome measures were incident (fatal and non-fatal) myocardial infarction, stroke, heart failure, colon cancer, lung cancer, breast cancer, and prostate cancer.

Death certificates within the National Health Service Information Centre (England and Wales) and the National Health Service Central Register Scotland (Scotland) provided dates of deaths. Date and cause of hospital admissions were obtained via record linkage to Health Episode Statistics (England and Wales) and Scottish Morbidity Records (Scotland). Further information about the data linkage can be found at http://content.digital.nhs.uk/services. The period at risk per participant began on the date of assessment. End of follow-up was recorded as the date of death or the date of end of follow-up for the assessment centre attended (31st January 2018 in Wales or England and 30th November 2016 in Scotland), or the first date of hospitalisation for CVD or cancer outcomes, whichever came first. We defined incident CVD as a hospital admission or death with ICD-10 (International Classification of Diseases, 10th Revision) codes for ischaemic heart diseases (I20-25), stroke (I60, I61, I63, I64), heart failure (I50), and atrial fibrillation (I48) together. For incident cancer, we used ICD-10 codes for any malignant neoplasm (C00-C97). The following ICD-10 codes were used for secondary outcomes: incident myocardial infarction (I21); stroke (I60, I61, I63, I64), heart failure (I50); cancer: colon (D01.0); lung (D02.2); breast (D05); and prostate (D07.5).

Ethics

The UK Biobank study was approved by the North West Multi-Centre Research Ethics Committee; participants provided written informed consent for data collection, analysis, and record linkage. This study is part of UK Biobank project 7155 (NHS National Research Ethics Service 16/NW/0274).

Statistical analyses

The main associations between TV time and health outcomes were examined using a non-linear Cox regression analysis. Non-linear association was modelled by penalised cubic splines with 5 equally spaced knots. Penalised spline is a variation of basis spline which achieves better estimation accuracy in non-linear data than the commonly used restricted cubic spline and is not heavily influenced by the number and location of knots.[23] Median TV time of the sample was the reference point (HR=1). Likelihood ratio tests were used to examine the overall statistical significance and nonlinearity of TV time. To reduce the potential influence of reverse causality we performed landmark analyses with follow-up commencing two years after recruitment, excluding participants who had prior disease events. Further, we excluded individuals who self-reported relevant conditions at baseline for each outcome i.e. excluding those with NCDs at baseline from all-cause mortality analyses, those with CVD from CVD analyses, and those with cancer from cancer analyses. We also excluded participants with improbable TV time (>8 h/day). All results are reported as hazard ratios together with 95% confidence intervals. Analyses were adjusted for age, sex, ethnicity, deprivation index, month of assessment, height, systolic blood-pressure, medication for hypertension or hypercholesterolaemia, smoking, BMI category, physical activity, grip strength, alcohol intake, and intake of red meat, processed meat, oily fish, and fruit/vegetables. The selection of these covariates was based on the causal assumptions depicted in the directed acyclic graph (Appendix Figure 1).

For sensitivity analyses, we examined for differences in strength of association across subgroups. For simplicity we assumed linear association in these analyses with the primary exposure being hour of TV time. Subgroups examined were: age (<60 vs. ≥60), sex, deprivation (< median vs. ≥ median), physical activity level (<1000 vs. ≥1000 MET[metabolic equivalent of task].minutes/week, grip

strength (< sex-specific median vs. ≥ sex-specific median), body height (< sex-specific median vs. ≥ sex-specific median), and BMI categories (normal vs. overweight/obese).

To examine the risk of health outcomes associated with substituting TV time with alternative activities, we conducted an isotemporal substitution analysis. The alternative activities considered were: sleeping, walking, moderate, and vigorous physical activity.[24] Each substitute activity was modelled separately as the primary exposure using penalised cubic spline to avoid making linearity assumption and were mutually adjusted. Similar to linear isotemporal substitution analysis, TV time was omitted from models while all alternative activities, as well as total time, were included as covariates to ensure any change in substitute activity was due to change in TV time. Median levels of alternative activities were set as the reference points (HR=1). Models were adjusted for the same covariates as the main analyses.

To quantify the potential impact of TV time reduction, we estimated population attributable fractions (PAF) and potential impact factions (PIF) using the standard formula.[25] Assuming causality, as recently supported by a mendelian randomisation study,[26] PAF indicates the proportion of health outcomes reduction if all participants' TV time was reduced to the level associated with lowest risk. Whereas our PIF analysis considered two hypothetical scenarios: limiting participants' TV time to 3 and 2h/day. PIF in this case indicates the proportion of health outcome reduction if participants with >3 and >2h/day of TV time reduced the time to 3 and 2h/day respectively. These provide the most optimistic scenarios for developing TV time guidelines at various thresholds. Statistical significance was set at α <.05 and all analyses were performed using R Statistical Software version 3.5.1 with the packages *survival, mediation*, and *pifpaf*. The proportional hazard assumption was verified by tests based on Schoenfeld residuals.

RESULTS

Of 502,536 recruited participants, 5,392 (1.1%) with missing and 2,388 (0.5%) with improbable TV time were excluded; 490,966 participants remained for analysis. 109,867 (22.4%) had prevalent NCDs at recruitment and were excluded for all-cause mortality analyses; 32,243 (6.6%) had prior

CVD and were excluded from CVD analyses; and 37,812 (7.7%) had prior cancer and were excluded from cancer analyses. By landmark analysis, mean follow-up up for mortality was 7.0 years (ranging from 5.4-9.9) and 6.2 years (ranging from 4.5-9.0) for disease incidence. Of participants included in respective analyses, 10,306 (2.7%) died, 24,388 (5.3%) developed CVD, and 39,121 (8.7%) developed cancer.

Participants' characteristics are shown in Table 1 using categories of TV time to aid cohort description. Participants in the highest category for TV time (>7 h/day) were more likely to be older, of lower SES, a smoker, overweight/obese, have higher blood pressure, report lower physical activity, and have lower grip strength than those who watched <1 h/day. These participants also tended to report lower intakes of fruit/vegetables, oily fish, and alcohol; and higher intakes of red and processed meat compared with the lowest TV time category. Approximately half of participants reported a TV time of ≤3 h/day. Appendix Figure 2 shows the distribution of TV time among participants.

TV time was generally associated with higher risk of mortality and disease incidence, except for incident stroke, and colon, breast, and prostate cancers (Figure 1). Associations between TV time and incident CVD, incident myocardial infarction, stroke and heart failure, and all cancers were close to linear ($P_{\text{non-linear}} \geq .07$). In contrast, the associations between TV time and all-cause and CVD mortality were curvilinear ($P_{\text{non-linear}} \leq .003$) with minimum risk observed at <2h/day (Figure 1).

[Insert Figure 1 here]

Results of the non-linear isotemporal substitution analysis are shown in Figure 2. These findings indicate that the associated health benefits of substituting TV time depend on the baseline level of substitute activity. Generally, substituting TV time with more of any one of sleeping, walking, moderate or vigorous physical activity was associated with improvement for all outcomes when baseline levels of substitute activities were low. For example, for participants that reported walking 0.5h/day, the associated all-cause mortality hazard ratio was 1 ($HR_{\text{walking}=0.5} = 1$; Figure 2). Substituting 0.5h/day of TV time with 0.5h/day more walking for these participants was associated

with a 10% reduction in all-cause mortality risk ($HR_{walking=1}$ = 0.9). Whereas, for participants that reported walking 1h/day ($HR_{walking=1}$=0.9), substituting 0.5h/day of TV time with 0.5h/day more walking ($HR_{walking=1.5}$=0.9) was not associated with reduction in all-cause mortality risk. Replacing TV time with longer durations of sleep was associated with lower risk of all-cause, CVD, and cancer mortality when participants reported sleeping <6 h/day. Except for sleep, replacing TV time with any of the substitute activities had negligible effect when levels of the activity were high. Replacing TV time with sleep was associated with worse outcomes when sleep was reported >8-9 h/day.

[Insert Figure 2 here]

Moderators of the association between TV time and health outcomes are presented in Table 2. The association between TV time and all-cause mortality was stronger among: younger individuals; males; individuals of lower SES; and less physically active participants. Associations between TV time and incident CVD were stronger in younger participants, and the association between TV time and incident cancer was stronger among those who had lower dietary energy intake. There was no evidence for variation in the strength of associations between TV time and outcomes by grip strength or BMI category (Table 2).

Population attributable (PAF) and potential impact fractions (PIF) of TV time are shown in Table 3. Assuming causal associations, 6.02% (95% CI 4.86 to 7.26) of all deaths, 8.64% (95% CI 6.60 to 10.73) of CVD deaths, 6.62% (95% CI 5.39 to 7.97) of cancer deaths, 6.95% (95% CI 6.23 to 7.70) incident CVD disease, and 5.28% (95% CI 4.71 to 5.78) of incident cancer were attributable to TV time. Further, if all participants limited TV time to 2 h/day, 5.62% (95% CI 4.30 to 7.00) of all deaths and 7.97% (95% CI 5.54 to 10.70) of CVD deaths could have been prevented or delayed, which is equivalent to 93.4% of all deaths and 92.2% of CVD deaths associated with TV time.

DISCUSSION

Our analysis of a large UK-based dataset shows a linear association between TV time and incident CVD, incident cancer and cancer mortality. Whereas associations between TV time and all-cause mortality and CVD mortality were curvilinear. This fits with previous meta-analyses which found non-linear relationships between TV time all-cause mortality,[5,8,18] and between TV time and CVD mortality,[8] but linear relationships between TV time and CVD incidence,[5] and between TV time and cancer mortality.[8]

In our study, the lowest risk for the majority of adverse health outcomes was between 0-2 h/day of TV time. Our estimate is lower than that previously identified however there are a number of limitations in previous meta-analyses examining non-linear associations. Grøntved et al. detected a significantly increased all-cause mortality risk at around 3h/day however this is based on only 4 studies with all-cause mortality data.[5] Sun et al.'s meta-analysis of 10 studies, examining all-cause-mortality only, also detected a higher threshold of increased risk at 4h/day.[18] However, significant heterogeneity was identified among studies (I^2 =66.7%). A meta-analysis of 34 prospective studies (29 studies with TV time data) by Patterson et al., also found higher thresholds of 3.5h/day of TV time for all-cause mortality and 4h/day for CVD mortality.[8] However, there was substantial heterogeneity (I^2 >50% for many outcomes) across included studies, which may have influenced the results. Specifically, there was significant variation in the measurement of TV time, most frequently measured as a self-reported categorical variable. Our results also suggest a potential J-shaped relationship between TV time and at least some adverse health outcomes (e.g. all-cause and CVD mortality; Figure 1) where 1.5 to 2h/day of TV time was associated with the lowest levels of risk. This finding could either represent imprecise estimation (thus wider confidence intervals) or a true protective effect of a small amount of TV time, which warrants further investigation.

Our PAF estimates suggest that 6% of all deaths and between 5-9% of disease-specific incidence and mortality can be attributed to TV viewing. PAF estimates from Patterson et al. found 8% of all-cause mortality and 5% of both CVD and cancer mortality was associated with TV time.[8] However, these estimates may be less accurate due to lack of TV time data from all countries included. Theoretically, because of the curvilinear association, our PIF estimates suggest limiting TV time to ≤2h/day could

prevent or delay the vast majority of deaths associated with TV viewing. However, the benefits of reducing TV time are likely to be dependent on the activities that replace TV time.[27] Our isotemporal analysis shows, for those who sleep or exercise little, reducing TV time and replacing it with more sleep and physical activity are associated with the greatest reduction in mortality and morbidity risk.

Biological mechanisms

Causal pathways that explain associations between TV time and adverse health remains unclear. However, results from prospective cohorts are consistent with a pathway where TV time increases inflammatory mediators.[28,29] Results of a genome-wide association study (GWAS) of objectively measured physical activity suggest that higher 'overall activity' might causally lower blood pressure.[30] More recently, a combined GWAS and mendelian randomisation analysis found that TV time in UK Biobank was causally linked to coronary artery disease whereas the other sedentary behaviours (leisure computer use and driving) examined were not.[26] While long term trials are lacking, results from short term trials support a causal link between sedentary time and health outcomes via glucose metabolism.[31,32]

Our moderation analysis showed the adverse health associations were strongest among the most socioeconomically deprived with a significant interaction between TV time and deprivation for all-cause mortality. This fits with previous research that demonstrated an interaction between deprivation and a broad range of lifestyle factors, including TV time.[33]

Strengths and Limitations

UK Biobank is a prospective cohort with linked routine health outcome data providing a unique resource to explore risks associated with TV time. UK Biobank's large size allows analyses with sufficient statistical power despite excluding participants with self-reported NCDs at baseline or with events within 2 years of recruitment. These analyses reduce the chance that results are due to reverse causality (i.e. poor health leading to longer TV time and adverse outcomes). Nevertheless, reverse causality remains a possible explanation. Due to the lack of evidence for the effect of individual disease on TV viewing, we grouped the comorbidities as a binary variable. Our analyses, performed

with TV time as a continuous variable to estimate associated risks, clarifies underlying associations and provides more accurate estimates of PAFs and PIFs.[34] However, PAF and PIF are theoretical estimates that rely on assumptions, such as a lack of bias in study design and that reducing TV time does not affect other risk factors.[35] UK Biobank's low response rate (5%) and over-representation of affluent participants and those from a white ethnic background compared with the UK general population means prevalence estimates are not generalisable to the UK general population.[36] however, both socioeconomic deprivation and non-white ethnicity are associated with longer TV time.[37,38] Therefore, our PAF estimates could represent an underestimate for the general population.

Our isotemporal substitution analysis assumes that individuals are able to make healthy replacement choices, however some lifestyle factors may lie outside an individual's control especially when considering the wider socioeconomic environment.[39] For example, living conditions can influence sleep duration.[40]

All self-reported data have limitations and we are unable to quantify the effect that both intentional and non-intentional factors at participant-level may have had on the accuracy of UK Biobank's self-reported data.[41,42] For example, the dietary variables used here are categorical and we are unable exclude potential outliers or implausible values due to the nature of the data. Therefore, our results may have been influenced by unknown inaccuracies in the self-reported data. However, recall-based assessment methods remain reasonable representations for health behaviours with alternative biases and problems inherent in observed assessment methods.[43,44] Some self-reported exposures and covariates captured by UK Biobank at baseline will have changed during follow up (e.g. average TV time may have reduced for some individuals) and our results do not account for these changes.

Although frequently more accurate than other self-reported sedentary time measures,[13] TV time, like other self-reported factors, is often under-reported,[45,46] and we cannot exclude self-report bias in our results. The UK Biobank single-item question for TV time, although not validated, is based on previous research.[7] TV time in this study does not differentiate between TV time that is physically active versus inactive. Active TV time is time spent watching TV whilst simultaneously being physically active; e.g. treadmill TV time) and active TV time may have attenuated the observed

associations with adverse health seen here. Additionally, TV time is just one of an array of potentially modifiable sedentary behaviours. However, TV time likely bears unique risks to health and therefore warrants investigation.

A constellation of factors likely mediate, moderate, and or confound relationships between TV time and adverse health and it is not possible to adjust for all of these factors. For example, many of the comorbid conditions have low prevalence and adjusting for them mutually would create a multicollinearity issue. However, we propose, a pragmatic approach, accepting that it may be because of these closely linked factors that limiting TV time could confer health benefits. Furthermore, there are potential benefits of TV time, for example, in education,[47] or improving understanding,[48] that should be noted within a TV time recommendation.

Conclusion

Our findings support a recommendation that the UK adult population limit TV time to <2h/day. Recommendations for TV time could also advise that any reduction in TV time coincides with increasing time spent in healthier alternatives to ensure positive replacement effects.

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PATIENT AND PUBLIC INVOLVEMENT

There was no patient or public involvement in the design or conduct of this study.

TRANSPARENCY DECLARATION

Dr Carlos Celis-Morales (senior and corresponding author) affirms that the manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned (and, if relevant, registered) have been explained.

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HF, FH and CC-M designed the study. FH performed the statistical analysis with assistance from HF and CC-M. HF, FH and CC-M wrote the first draft of the manuscript and all authors contributed to interpretation of the results and redrafting of the manuscript. The authors thank the UK Biobank participants for providing their data.

REFERENCES

1. Biswas A, Oh PI, Faulkner GE, et al. Sedentary Time and Its Association With Risk for Disease Incidence, Mortality, and Hospitalization in Adults A Systematic Review and Meta-analysis. *Ann Intern Med* 2015; **162**(2): 123-+.
2. Eurostat - Statistical Office of the European Communities. The life of women and men in Europe: A statistical portrait., 2008.
3. Nielson Co. The Nielson total audience report: Q1 2018. 2018.
4. Media Nations: UK 2018. Ofcom's annual Media Nations report, 2018.
5. Grontved A, Hu FB. Television viewing and risk of type 2 diabetes, cardiovascular disease, and all-cause mortality: a meta-analysis. *JAMA* 2011; **305**(23): 2448-55.
6. Schmid D, Leitzmann MF. Television viewing and time spent sedentary in relation to cancer risk: a meta-analysis. *J Natl Cancer Inst* 2014; **106**(7).
7. Hu FB, Li TY, Colditz GA, Willett WC, Manson JE. Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women. *Jama-J Am Med Assoc* 2003; **289**(14): 1785-91.
8. Patterson R, McNamara E, Tainio M, et al. Sedentary behaviour and risk of all-cause, cardiovascular and cancer mortality, and incident type 2 diabetes: a systematic review and dose response meta-analysis. *Eur J Epidemiol* 2018; **33**(9): 811-29.
9. Pearson N, Biddle SJ. Sedentary behavior and dietary intake in children, adolescents, and adults. A systematic review. *Am J Prev Med* 2011; **41**(2): 178-88.
10. Hamilton MT, Healy GN, Dunstan DW, Zderic TW, Owen N. Too Little Exercise and Too Much Sitting: Inactivity Physiology and the Need for New Recommendations on Sedentary Behavior. *Curr Cardiovasc Risk Rep* 2008; **2**(4): 292-8.
11. Lewis O, Odeyemi Y, Joseph V, Mehari A, Gillum RF. Screen Hours and Sleep Symptoms: The US National Health and Nutrition Examination Survey. *Fam Community Health* 2017; **40**(3): 231-5.
12. Stamatakis E, Hillsdon M, Mishra G, Hamer M, Marmot M. Television viewing and other screen-based entertainment in relation to multiple socioeconomic status indicators and area deprivation: the Scottish Health Survey 2003. *J Epidemiol Community Health* 2009; **63**(9): 734-40.
13. Healy GN, Clark BK, Winkler EA, Gardiner PA, Brown WJ, Matthews CE. Measurement of adults' sedentary time in population-based studies. *Am J Prev Med* 2011; **41**(2): 216-27.
14. Hutcheon JA, Chiolero A, Hanley JA. Random measurement error and regression dilution bias. *Bmj-Brit Med J* 2010; **340**.
15. Stamatakis E, Ekelund U, Ding D, Hamer M, Bauman AE, Lee IM. Is the time right for quantitative public health guidelines on sitting? A narrative review of sedentary behaviour research paradigms and findings. *Br J Sports Med* 2018.
16. Altman D, G., . Categorising continuous covariates (letter to the editor). *British Journal of Cancer* 1991; **64**: 975.
17. Wainer H. Finding what is not there through the unfortunate binning of results: The Mendel effect.

19, 20. *Chance* 2006; **19**(1): 45-56.
18. Sun JW, Zhao LG, Yang Y, Ma X, Wang YY, Xiang YB. Association Between Television Viewing Time and All-Cause Mortality: A Meta-Analysis of Cohort Studies. *American Journal of Epidemiology* 2015; **182**(11): 908-16.
19. Sudlow C, Gallacher J, Allen N, et al. UK Biobank: An Open Access Resource for Identifying the Causes of a Wide Range of Complex Diseases of Middle and Old Age. *Plos Medicine* 2015; **12**(3).
20. Townsend P, Philimore P, Beattie A. Health and Deprivation: Inequality and the North: Croom Helm; 1987.
21. Craig CL, Marshall AL, Sjostrom M, et al. International physical activity questionnaire: 12-country reliability and validity. *Med Sci Sports Exerc* 2003; **35**(8): 1381-95.

22. McCance R. McCance and Widdowson's the composition of foods. London: Royal Society of Chemistry; 2002.
23. Govindarajulu US, Malloy EJ, Ganguli B, Spiegelman D, Eisen EA. The comparison of alternative smoothing methods for fitting non-linear exposure-response relationships with Cox models in a simulation study. *The international journal of biostatistics* 2009; **5**(1).
24. Mekary RA, Willett WC, Hu FB, Ding EL. Isotemporal substitution paradigm for physical activity epidemiology and weight change. *Am J Epidemiol* 2009; **170**(4): 519-27.
25. Morgenstern H, Bursic ES. A method for using epidemiologic data to estimate the potential impact of an intervention on the health status of a target population. *Journal of community health* 1982; **7**(4): 292-309.
26. Van De Vegte YJ, Said MA, Rienstra M, Van Der Harst P, Verweij N. Genome-wide association studies and Mendelian randomization analyses for leisure sedentary behaviours. *Nature Communications* 2020; **11**(1).
27. Wijndaele K, Sharp SJ, Wareham NJ, Brage S. Mortality Risk Reductions from Substituting Screen Time by Discretionary Activities. *Med Sci Sports Exerc* 2017; **49**(6): 1111-9.
28. Hamer M, Yates T, Demakakos P. Television viewing and risk of mortality: Exploring the biological plausibility. *Atherosclerosis* 2017; **263**: 151-5.
29. Grace MS, Dillon F, Barr ELM, Keadle SK, Owen N, Dunstan DW. Television Viewing Time and Inflammatory-Related Mortality. *Med Sci Sports Exerc* 2017; **49**(10): 2040-7.
30. Doherty A, Smith-Byrne K, Ferreira T, et al. GWAS identifies 14 loci for device-measured physical activity and sleep duration. *Nat Commun* 2018; **9**(1): 5257.
31. Bailey DP, Locke CD. Breaking up prolonged sitting with light-intensity walking improves postprandial glycemia, but breaking up sitting with standing does not. *J Sci Med Sport* 2015; **18**(3): 294-8.
32. Peddie MC, Bone JL, Rehrer NJ, Skeaff CM, Gray AR, Perry TL. Breaking prolonged sitting reduces postprandial glycemia in healthy, normal-weight adults: a randomized crossover trial. *Am J Clin Nutr* 2013; **98**(2): 358-66.
33. Foster HME, Celis-Morales CA, Nicholl BI, et al. The effect of socioeconomic deprivation on the association between an extended measurement of unhealthy lifestyle factors and health outcomes: a prospective analysis of the UK Biobank cohort. *Lancet Public Health* 2018; **3**(12): e576-e85.
34. Barendregt JJ, Veerman JL. Categorical versus continuous risk factors and the calculation of potential impact fractions. *J Epidemiol Community Health* 2010; **64**(3): 209-12.
35. Mansournia MA, Altman DG. STATISTICS NOTES Population attributable fraction. *Bmj-Brit Med J* 2018; **360**.
36. Fry A, Littlejohns TJ, Sudlow C, et al. Comparison of Sociodemographic and Health-Related Characteristics of UK Biobank Participants With Those of the General Population. *Am J Epidemiol* 2017; **186**(9): 1026-34.
37. Shuval K, Gabriel KP, Leonard T. TV viewing and BMI by race/ethnicity and socio-economic status. *PLoS One* 2013; **8**(5): e63579.
38. Cassidy S, Chau JY, Catt M, Bauman A, Trenell MI. Cross-sectional study of diet, physical activity, television viewing and sleep duration in 233,110 adults from the UK Biobank; the behavioural phenotype of cardiovascular disease and type 2 diabetes. *BMJ Open* 2016; **6**(3): e010038.
39. Katikireddi SV, Higgins M, Smith KE, Williams G. Health inequalities: the need to move beyond bad behaviours: Table 1. *J Epidemiol Commun H* 2013; **67**(9): 715-6.
40. Billings ME, Hale L, Johnson DA. Physical and Social Environment Relationship With Sleep Health and Disorders. *Chest* 2019.
41. Archer E, Pavela G, Lavie CJ. The Inadmissibility of What We Eat in America and NHANES Dietary Data in Nutrition and Obesity Research and the Scientific Formulation of National Dietary Guidelines. *Mayo Clinic Proceedings* 2015; **90**(7): 911-26.

42. Archer E, Marlow ML, Lavie CJ. Controversy and Debate: Memory Based Methods Paper 3: Nutrition's 'Black Swans': Our reply. *Journal of Clinical Epidemiology* 2018; **104**: 130-5.
43. Davy BM, Estabrooks PA. The Validity of Self-reported Dietary Intake Data: Focus on the "What We Eat In America" Component of the National Health and Nutrition Examination Survey Research Initiative. *Mayo Clinic Proceedings* 2015; **90**(7): 845-7.
44. Martín-Calvo N, Martínez-González MÁ. Controversy and debate: Memory-Based Dietary Assessment Methods Paper 2. *Journal of Clinical Epidemiology* 2018; **104**: 125-9.
45. Otten JJ, Littenberg B, Harvey-Berino JR. Relationship Between Self-report and an Objective Measure of Television-viewing Time in Adults. *Obesity* 2010; **18**(6): 1273-5.
46. Newell SA, Girgis A, Sanson-Fisher RW, Savolainen NJ. The accuracy of self-reported health behaviors and risk factors relating to cancer and cardiovascular disease in the general population: a critical review. *Am J Prev Med* 1999; **17**(3): 211-29.
47. Chu GS, W. Learning from Television: What the Research Says.: IAP; 2004.
48. Black J, Barnes JL. Fiction and Social Cognition: The Effect of Viewing Award-Winning Television Dramas on Theory of Mind. *Psychol Aesthet Crea* 2015; **9**(4): 423-9.

FIGURES AND FIGURE LEGENDS

Figure 1. Associations between TV time and all-cause mortality, cardiovascular mortality and incidence, and cancer mortality and incidence.

[Insert Figure 1 here]

Hazard ratio (HR) represented by bold line; 95% confidence interval represented by shaded area. Models adjusted for age, sex, ethnicity, deprivation index, height, systolic blood pressure, medication for hypercholesterolaemia or hypertension, smoking, BMI categories, physical activity, grip strength, alcohol drinking, and dietary intake of red meat, processed meat, oily fish, and fruit/vegetables.

Figure 2. Associations between TV time and all-cause mortality, CVD mortality, incident CVD, and incident cancer for non-linear isotemporal substitution analysis: TV time replaced with sleeping, walking, moderate physical activity, and vigorous activity.

[Insert Figure 2 here]

Hazard ratio (HR) represented by bold line; 95% confidence interval represented by shaded area. For each graph, the horizontal axis indicates the time of substitute activity (h/day). Interpretation is aided by considering a change in position along the horizontal axis. For example, for an increase in substitute activity of 1h/day there is a corresponding 1h/day reduction in TV time.

TABLES

Table 1. Baseline cohort characteristics (n=490,966)

	TV time (Hours of television viewing per day)				
	<1 16,026 (3.26)	1-2 217,820 (44.37)	3-4 195,006 (39.72)	5-6 54,471 (11.09)	≥7 7,643 (1.57)
Mean (SD) age, years	55.09 (8.05)	54.90 (8.12)	57.57 (7.84)	59.40 (7.41)	59.07 (7.61)
Sex					
Female	9069 (56.59)	119073 (54.67)	106491 (54.61)	29504 (54.16)	3983 (52.11)
Male	6957 (43.41)	98747 (45.33)	88515 (45.39)	24967 (45.84)	3660 (47.89)
Ethnicity					
White	14732 (92.72)	204458 (94.20)	185788 (95.54)	51491 (94.81)	7084 (93.06)
South Asian	340 (2.14)	5139 (2.37)	3096 (1.59)	780 (1.44)	143 (1.88)
Black	305 (1.92)	3116 (1.44)	2735 (1.41)	1172 (2.16)	221 (2.90)
Chinese	91 (0.57)	827 (0.38)	466 (0.24)	122 (0.22)	18 (0.24)
Mixed background	147 (0.93)	1287 (0.59)	1054 (0.54)	304 (0.56)	65 (0.85)
Others	273 (1.72)	2215 (1.02)	1332 (0.68)	440 (0.81)	81 (1.06)
Socioeconomic status					
High	3906 (24.40)	77912 (35.82)	66956 (34.38)	15130 (27.81)	1409 (18.47)
Middle	4732 (29.55)	73580 (33.83)	66821 (34.31)	17280 (31.76)	1912 (25.07)
Low	7373 (46.05)	66037 (30.36)	60999 (31.32)	22001 (40.43)	4307 (56.46)
Mean (SD) height, m	1.69 (0.09)	1.69 (0.09)	1.68 (0.09)	1.67 (0.09)	1.67 (0.09)
Mean (SD) systolic blood pressure, mm Hg	133.24 (18.86)	135.60 (18.43)	139.51 (18.53)	141.34 (18.72)	140.83 (19.08)
Smoking status					
Never	9193 (57.56)	128612 (59.21)	102572 (52.81)	25302 (46.67)	3068 (40.39)
Former	4979 (31.18)	70025 (32.24)	70816 (36.46)	21108 (38.94)	2858 (37.63)
Current	1799 (11.26)	18560 (8.55)	20832 (10.73)	7803 (14.39)	1670 (21.99)
Mean (SD) BMI, kg/m^2	25.63 (4.50)	26.57 (4.42)	27.90 (4.75)	29.05 (5.26)	29.93 (5.96)
BMI categories					
Underweight	226 (1.42)	1371 (0.63)	722 (0.37)	192 (0.36)	42 (0.56)
Normal	7856 (49.30)	85426 (39.39)	53900 (27.76)	11290 (20.88)	1358 (18.03)
Overweight	5545 (34.80)	90050 (41.52)	86423 (44.51)	22966 (42.47)	2879 (38.23)
Obese	2309 (14.49)	40027 (18.46)	53113 (27.36)	19625 (36.29)	3251 (43.17)

Mean (SD) MET-min per week	2788.82 (2514.77)	2619.70 (2410.09)	2675.20 (2509.54)	2385.34 (2360.85)	1894.37 (2081.33)
Mean (SD) grip strength, kg	30.69 (10.52)	31.47 (10.90)	30.30 (11.09)	28.83 (10.97)	27.59 (10.78)
Mean (SD) dietary intake per week					
Portions of fruit/vegetables	4.67 (2.93)	4.26 (2.43)	4.03 (2.36)	3.85 (2.53)	3.59 (2.73)
Portions of red meat	1.83 (1.56)	2.02 (1.40)	2.17 (1.44)	2.27 (1.55)	2.35 (1.79)
Frequency of processed meat intake					
Never	3249 (20.34)	23626 (10.86)	15027 (7.72)	3512 (6.47)	514 (6.76)
Less than once a week	5319 (33.30)	69902 (32.14)	58440 (30.02)	14492 (26.68)	1691 (22.24)
Once a week	3686 (23.08)	62920 (28.93)	58596 (30.10)	16030 (29.51)	2074 (27.28)
2-4 times a week	3076 (19.26)	53491 (24.60)	55061 (28.29)	17610 (32.42)	2747 (36.13)
5-6 times a week	480 (3.01)	6050 (2.78)	6008 (3.09)	2087 (3.84)	453 (5.96)
Once or more daily	163 (1.02)	1484 (0.68)	1524 (0.78)	582 (1.07)	125 (1.64)
Frequency of oily fish intake					
Never	1902 (11.94)	20799 (9.58)	21274 (10.97)	7582 (14.05)	1383 (18.37)
Less than once a week	4900 (30.76)	70740 (32.60)	64847 (33.44)	18656 (34.56)	2572 (34.17)
Once a week	5666 (35.56)	84952 (39.15)	73282 (37.79)	19034 (35.26)	2409 (32.00)
2-4 times a week	3184 (19.98)	38328 (17.66)	32884 (16.96)	8264 (15.31)	1070 (14.21)
5-6 times a week	215 (1.35)	1667 (0.77)	1203 (0.62)	314 (0.58)	67 (0.89)
Once or more daily	65 (0.41)	523 (0.24)	405 (0.21)	129 (0.24)	27 (0.36)
Frequency of alcohol drinking					
Daily or almost daily	3807 (23.79)	48151 (22.12)	38079 (19.54)	9148 (16.82)	1126 (14.77)
3-4 times a week	3266 (20.41)	54616 (25.09)	44416 (22.79)	10460 (19.23)	1154 (15.13)
Once or twice a week	3291 (20.56)	54876 (25.21)	52316 (26.85)	14351 (26.38)	1885 (24.72)
1-3 times a month	1616 (10.10)	22895 (10.52)	22648 (11.62)	6673 (12.27)	846 (11.10)
Special occasions only	1938 (12.11)	21351 (9.81)	22937 (11.77)	8358 (15.36)	1466 (19.23)
Never	2086 (13.03)	15795 (7.26)	14469 (7.43)	5411 (9.95)	1148 (15.06)
Self-reported CVD diagnosis	778 (4.85)	9918 (4.55)	14281 (7.32)	6058 (11.12)	1208 (15.81)
Self-reported cancer diagnosis	1161 (7.27)	14836 (6.83)	15971 (8.22)	5085 (9.38)	759 (9.99)

Data are n (%) or mean (standard deviation; SD) for categorical and continuous variables, as appropriate. MET=metabolic equivalent of task.

Table 2. Association of TV time on all-cause mortality, CVD incidence, and cancer incidence stratified by sociodemographic and behavioural factors

	All-cause mortality		**Incident CVD**		**Incident cancer**	
	HR (95% CI)	$P_{Interaction}$	**HR (95% CI)**	$P_{Interaction}$	**HR (95% CI)**	$P_{Interaction}$
Age		.04		<.001		.73
< 60	1.08 (1.05, 1.11)		1.07 (1.05, 1.09)		1.03 (1.01, 1.05)	
≥ 60	1.04 (1.02, 1.06)		1.02 (1.01, 1.04)		1.02 (1.01, 1.04)	
Sex		.008		.40		.93
Female	1.03 (1.01, 1.06)		1.03 (1.01, 1.05)		1.02 (1.01, 1.04)	
Male	1.06 (1.04, 1.08)		1.04 (1.03, 1.06)		1.03 (1.02, 1.04)	
Deprivation index		<.001		.44		.13
≤ median	1.02 (0.99, 1.04)		1.05 (1.03, 1.07)		1.01 (1.00, 1.03)	
> median	1.08 (1.06, 1.10)		1.03 (1.01, 1.04)		1.03 (1.02, 1.05)	
Daily energy intake		.41		.83		<.001
< 2000 (F) / 2500 (M)	1.06 (1.02, 1.10)		1.02 (1.00, 1.05)		1.05 (1.03, 1.07)	
≥ 2000 (F) / 2500 (M)	1.04 (0.99, 1.10)		1.03 (1.00, 1.06)		0.99 (0.97, 1.01)	
MET minutes / week		.03		.10		.36
< 1000	1.07 (1.04, 1.10)		1.05 (1.03, 1.07)		1.02 (1.00, 1.03)	
≥ 1000	1.04 (1.02, 1.06)		1.03 (1.02, 1.05)		1.03 (1.02, 1.04)	
Grip strength		.63		.27		.64
≤ sex-specific median	1.05 (1.03, 1.07)		1.04 (1.03, 1.06)		1.02 (1.01, 1.03)	
> sex-specific median	1.05 (1.02, 1.08)		1.03 (1.01, 1.05)		1.03 (1.01, 1.04)	
BMI categories		.43		.97		.68
Normal	1.07 (1.03, 1.10)		1.04 (1.01, 1.07)		1.04 (1.02, 1.05)	
Overweight/obese	1.06 (1.04, 1.08)		1.05 (1.03, 1.06)		1.02 (1.01, 1.03)	

Adjusted for age, sex, ethnicity, deprivation index, height, systolic blood pressure, medication for hypercholesterolaemia or hypertension, smoking, BMI categories, physical activity, grip strength, alcohol drinking, and dietary intake of red meat, processed meat, oily fish, and fruit/vegetables. Daily energy intake measured in calories: cut offs at 2000 for female (F) and 2500 for male (M). CVD=cardiovascular disease; HR=hazard ratio for each hour of TV time; CI=confidence interval

Table 3. Population attributable fractions and potential impact fractions of TV time

	Population Attributable Fraction, % (95% CI)	**Potential Impact Fraction, % (95% CI)**	
		≤ 3 hours	**≤ 2 hours**
All-cause mortality	6.02 (4.86–7.26)	3.26 (2.30–4.21)	5.62 (4.30–7.00)
Cardiovascular disease mortality	8.64 (6.60-10.73)	5.35 (3.60–7.03)	7.97 (5.54-10.70)
Cancer mortality	6.62 (5.39–7.97)	2.37 (1.34–3.46)	4.55 (3.04–6.10)
Incident cardiovascular disease	6.95 (6.23–7.70)	1.69 (1.03–2.30)	3.49 (2.61–4.40)
Incident cancer	5.28 (4.71–5.84)	1.32 (0.85–1.80)	2.61 (1.90–3.37)

Based on the HRs shown in Figure 1 and the distribution of TV time in UK Biobank. CI=confidence interval