

Television viewing time independently predicts all-cause and cardiovascular mortality: the EPIC Norfolk Study

Katrien Wijndaele,¹ Søren Brage,¹ Hervé Besson,¹ Kay-Tee Khaw,² Stephen J Sharp,¹ Robert Luben,² Nicholas J Wareham¹ and Ulf Ekelund^{1*}

¹MRC Epidemiology Unit, Cambridge, UK and ²Department of Public Health and Primary Care, Institute of Public Health, University of Cambridge, Cambridge, UK

*Corresponding author. MRC Epidemiology Unit, Institute of Metabolic Science, Box 285, Addenbrooke's Hospital, Hills Road, Cambridge CB2 0QQ, UK. E-mail: ulf.ekelund@mrc-epid.cam.ac.uk

Accepted 20 May 2010

Background Television viewing (TV), a highly prevalent behaviour, is associated with higher cardiovascular risk independently of physical activity. The relationship with mortality, however, is relatively unknown.

Methods We examined the prospective relationship between TV time and all-cause, cardiovascular and cancer mortality in a population-based cohort [The European Prospective Investigation into Cancer and Nutrition (EPIC), Norfolk] of 13 197 men and women {age [SD (standard deviation)]: 61.5 ± 9.0 years}. Participants were free from stroke, myocardial infarction and cancer at baseline in 1998–2000 and were followed up for death ascertainment until 2009 (9.5 ± 1.6 years). TV time, total physical activity energy expenditure (PAEE), education level, smoking status, alcohol consumption, anti-hypertensive and lipid-lowering medication use, participant and family history of disease and total energy intake were self-reported; height and weight were measured by standardized procedures. Hazard ratios (HRs) [95% confidence interval (CI)] for mortality were estimated per 1-h/day increase in TV.

Results Each 1-h/day increase in TV time was associated with increased hazard of all-cause (HR=1.04, 95% CI=1.01–1.09; 1270 deaths) and cardiovascular (HR=1.07, 95% CI=1.01–1.15; 373 deaths), but not cancer mortality (HR=1.04, 95% CI=0.98–1.10; 570 deaths). This was independent of gender, age, education, smoking, alcohol, medication, diabetes history, family history of cardiovascular disease and cancer, body mass index (BMI) and PAEE. They were similar when stratified by gender, age, education, BMI and PAEE. The population-attributable fraction for all-cause mortality comparing the highest TV tertile (>3.6 h/day) with the lowest (<2.5 h/day) was 5.4%.

Conclusions These findings suggest that public health recommendations should consider advising a reduction in TV time, a predominant leisure activity in modern society, in addition to advocating physical activity.

Keywords Cardiovascular diseases, epidemiology, exercise, mortality, television

Introduction

Sedentary behaviour (i.e. excessive sitting, as distinct from insufficient physical activity) has increasingly gained interest as a potential determinant of ill health, particularly given its high prevalence (e.g. 55% of the waking day in the USA).¹ Television viewing (TV) time in particular has been reported as the most widespread leisure-time sedentary behaviour of adults.^{2–4} Cross-sectional and prospective evidence is accumulating for adverse associations between TV time and several intermediate cardiovascular risk factors, including the metabolic syndrome and related cardio-metabolic biomarkers,^{5–13} obesity,^{14,15} abnormal glucose metabolism and type 2 diabetes.^{15–19} Some cancers have also been associated with excessive levels of TV time.^{20,21} These associations were independent of physical activity and an adverse effect of elevated TV time has been observed in physically active individuals.¹³ Some physiological mechanisms supporting detrimental effects of sedentary behaviour are also emerging.¹⁹ Building on the available evidence, so far, two recent studies have examined whether TV time independently predicts specific mortality endpoints (i.e. all-cause, cardiovascular and cancer mortality), of which one only focused on cardiovascular mortality in men.^{22,23}

The purpose of the present study was (i) to examine the prospective association between TV time and mortality from all-causes, cardiovascular disease (CVD) and cancer in a relatively large sample of healthy middle-aged Caucasian men and women and (ii) to examine whether this association is independent of total physical activity energy expenditure (PAEE) and other potentially confounding variables.

Methods

Study participants

EPIC-Norfolk is part of the 10-country collaborative European Prospective Investigation into Cancer and Nutrition Study (EPIC). Between 1993 and 1997, a cohort of 25 633 residents of Norfolk (UK), in the age range of 45–79 years and recruited via participating general practitioners, agreed to participate. The study design and cohort characteristics have previously been described.²⁴ Between January 1998 and October 2000, participants were invited for a follow-up assessment. At this follow-up, the EPIC physical activity questionnaire (EPAQ2) was introduced as a more comprehensive physical activity measure, which also included questions on TV time. This examination constitutes the baseline for the current analyses. A total of 15 784 participants attended, of whom 15 021 provided complete data for variables included in the present study. Participants with a self-reported baseline history of stroke ($n=360$), myocardial infarction ($n=488$) or cancer ($n=1075$)

were excluded. As a result, 13 197 participants (5729 men and 7468 women) were included. The Norwich District Health Authority Ethics Committee approved the study design and all participants provided written informed consent.

Measures

Mortality

All EPIC-Norfolk participants are followed up for mortality and the present study reports on follow-up until 30 April 2009 (9.5 ± 1.6 years). All individuals have been flagged for death certification at the Office of National Statistics (UK) with vital status ascertained on the whole cohort. Coding of death certificates was executed by trained nosologists according to the International Classification of Diseases (ICD). Cardiovascular death was defined as ICD 410–448 (ICD 9) or ICD I10–I79 (ICD 10) as the underlying cause of death. Cancer death was defined as ICD 140–208 (ICD 9) or ICDC00–C97 (ICD 10) as the underlying cause.

TV time and PAEE

Participants completed the EPAQ2, providing information on their physical (in)activity in a disaggregated way (in and around the home, to work, at work and during leisure time), using the past year as a reference frame.²⁵ PAEE [metabolic equivalent (MET) \times h/week] in each domain was calculated by multiplying participation (h/week) by the metabolic cost of each activity (MET).²⁶ Total PAEE was calculated by summing energy expenditure of the different (mutually exclusive) domains, as previously described in detail.²⁷ Time spent viewing television and video (h/week) was calculated by summing responses to four questions about viewing before and after 6 pm on week and weekend days. Repeatability of the EPAQ2 was high both in terms of TV time and total PAEE.²⁵ Comparison against minute-by-minute heart rate monitoring and maximal aerobic capacity (VO_{2max}) suggested that the questionnaire is valid for ranking individuals.²⁵ For ease of interpretation, TV time and PAEE were expressed in h/day and MET \times h/day, respectively. The two measures were mutually exclusive and weakly inversely correlated (Spearman correlation: -0.18 , $P < 0.01$).

Covariates

Education level (low, O, A, degree), social class (manual, non-manual), smoking status (current, former and never), alcohol consumption (U/week), anti-hypertensive medication (yes, no), medication for dyslipidaemia (yes, no), baseline history of myocardial infarction or stroke (yes, no), cancer (yes, no) and diabetes (yes, no), family history of CVD (yes, no) and cancer (yes, no) were self-reported using a detailed health and lifestyle questionnaire.²⁴ Total energy intake (kJ/day) was derived from a validated

130-item semi-quantitative food-frequency questionnaire.²⁸ Trained nurses measured height, weight and waist circumference according to standardized protocols.²⁴ Body mass index (BMI) was calculated as weight/height² (kg/m²).

Statistical analysis

Baseline characteristics were compared by vital status (independent samples *t*-tests and chi-square tests) and by TV tertiles (lowest: <2.5, middle: 2.5–3.6, highest: >3.6 h/day; one-way analysis of variance and chi-squared tests).

Cox proportional hazards regression was used to examine the association between baseline TV time (h/day) and all-cause, cardiovascular and cancer mortality. The proportional hazards assumption was checked using Schoenfeld residuals and Kaplan–Meier plots for all three outcomes. The Schoenfeld residuals did not suggest evidence of deviations from proportionality, and this was consistent with observations in the Kaplan–Meier plots. To examine linearity of the association between TV time and the outcomes, unadjusted mortality rates [95% confidence interval (CI)] per 10 000 person-years of follow-up were plotted by 1-h TV-time increments. Additionally, a log likelihood-ratio test examined whether addition of a quadratic term for TV time to the adjusted model (Model C) resulted in a statistically significant improvement in model fit. Based on evidence for a linear association, hazard ratios (HRs) (95% CI) per h/day increase in TV time were estimated, first adjusting for baseline age and gender (Model A). Further adjustment (Model B) was made for baseline education level, smoking status, alcohol consumption, medication (hypertension and dyslipidaemia; not included for cancer mortality), diabetes history and family history of CVD and cancer. We then introduced baseline PAEE to the model to examine whether TV time was independently associated with mortality (Model C).

To determine whether associations were modified by gender, age (≤60, >60 years), education (low or O, A or degree) or BMI (normal weight: <25, overweight or obese: ≥25 kg/m²), multiplicative interaction terms were included in Model C. Effect modification by physical activity was examined by including a (TV × total PAEE_{median split}) interaction term in Model C (cut-offs, total PAEE: ≤15.7, >15.7 MET h/day).

Continuous BMI and waist circumference were then added (consecutively) to Model C, to examine a potential mediation effect of overall and central adiposity. By including total energy intake (kJ/day), a potential mediation effect of diet was evaluated in a subsample, excluding 21% of participants with missing data for total energy intake.

Finally, the population-attributable fraction (PAF) for all-cause mortality associated with TV >3.6 (highest tertile) compared with <2.5 h/day

(lowest tertile) was calculated. PAF is the estimated proportion by which mortality would be reduced in the entire population if the exposure were eliminated (assuming causality). It was calculated using the formula

$$\text{PAF} = 100 \times \left(\frac{\text{Px} \times (\text{HR} - 1)}{1 + (\text{Px} \times (\text{HR} - 1))} \right)$$

where Px is prevalence of the exposure, and HR is the hazard ratio of the highest compared with the lowest TV tertile (derived from Model C including PAEE).

To minimize the potential effect of reverse causality, analyses were repeated after additionally excluding participants who died within the first 2 years of follow-up. Analyses were conducted using the Statistical Package for Social Sciences, version 15.0 (SPSS 15.0; SPSS, Inc., IL, USA) and STATA10.0 (Stata, Corp., TX, USA). Statistical significance was set at *P* < 0.05.

Results

Descriptive characteristics

A total of 1270 participants (725 men and 545 women) died during 124 902 person-years of follow-up (men: 53 330, women: 71 572). There were 373 cardiovascular deaths (men: 211, women: 162) and 570 cancer deaths (men: 321, women: 249). Descriptive baseline characteristics are shown by vital status in Table 1. Participants who died from any cause watched TV for on average 0.4 h/day more than survivors. This difference was greater for cardiovascular mortality (0.6 h/day) and smaller, although significant, for cancer mortality (0.3 h/day). Deceased participants were also less physically active, and showed a less favourable profile for several other characteristics, including waist circumference and BMI. Baseline characteristics by TV tertiles are shown in Table 2, indicating that participants watching more TV scored less favourable for several characteristics.

Cox proportional hazards regression

The increases found for unadjusted mortality rates per 10 000 person-years by 1-h increments in TV time suggested a linear association between TV time and all three mortality outcomes (Figure 1). There was no significant improvement in model fit after adding the quadratic TV term to the adjusted model (Model C), for any of the three outcomes.

Results for the Cox regression (Table 3) showed that TV time (h/day) was positively associated with all-cause, cardiovascular and cancer mortality after adjustment for age and gender (Model A). For all-cause and cardiovascular mortality, associations remained significant after further adjustment for education level, smoking status, alcohol consumption, medication for hypertension and dyslipidaemia,

Table 1 Descriptive characteristics [mean (SD) or *N* (%)] at baseline (second health check) by vital status in 13 197 men and women in EPIC-Norfolk, 1998–2009

Characteristics	All-cause mortality		Cardiovascular mortality		Cancer mortality	
	Survivors <i>n</i> = 11 927	Deceased <i>n</i> = 1270	Survivors <i>n</i> = 12 824	Deceased <i>n</i> = 373	Survivors <i>n</i> = 12 627	Deceased <i>n</i> = 570
Follow-up time (years)	9.8 (0.7)	6.1 (2.7)***	9.6 (1.4)	5.8 (2.8)***	9.6 (1.3)	5.9 (2.7)***
Male gender, <i>N</i> (%)	5004 (42.0)	725 (57.1)***	5518 (43.0)	211 (56.6)***	5408 (42.8)	321 (56.3)***
Age (years)	60.7 (8.7)	69.5 (7.7)***	61.3 (8.9)	70.5 (7.1)***	61.3 (9.0)	67.7 (8.1)***
Education level, <i>N</i> (%)						
Low	3748 (31.4)	532 (41.9)***	4117 (32.1)	163 (43.7)***	4061 (32.2)	219 (38.4)**
O level	1347 (11.3)	119 (9.4)	1428 (11.1)	38 (10.2)	1406 (11.1)	60 (10.5)
A level	5032 (42.2)	496 (39.1)	5388 (42.0)	140 (37.5)	5294 (41.9)	234 (41.1)
Degree	1800 (15.1)	123 (9.6)	1891 (14.8)	32 (8.6)	1866 (14.8)	57 (10.0)
Cigarette smoking, <i>N</i> (%)						
Current	933 (7.8)	154 (12.1)***	1025 (8.0)	62 (16.6)***	1031 (8.2)	56 (9.8)***
Former	4857 (40.7)	654 (51.5)	5343 (41.7)	168 (45.0)	5198 (41.2)	313 (54.9)
Never	6137 (51.5)	462 (36.4)	6456 (50.3)	143 (38.4)	6398 (50.6)	201 (35.3)
Alcohol consumption (U/week)	6.9 (9.0)	7.2 (10.8)	6.9 (9.1)	6.4 (10.3)	6.9 (9.1)	7.6 (10.8)
Antihypertensive drug, <i>N</i> (%)	2286 (19.2)	416 (32.8)***	2545 (19.8)	157 (42.1)***	2547 (20.2)	155 (27.2)***
Lipid-lowering drug, <i>N</i> (%)	400 (3.4)	52 (4.1)	429 (3.3)	23 (6.2)**	439 (3.5)	13 (2.3)
History of diabetes (%)	303 (2.5)	60 (4.7)***	344 (2.7)	19 (5.1)**	342 (2.7)	21 (3.7)
Family history of CVD (%)	6063 (50.8)	663 (52.2)	6515 (50.8)	211 (56.6)*	6433 (50.9)	293 (51.4)
Family history of cancer (%)	4806 (40.3)	495 (39.0)	5173 (40.3)	128 (34.3)*	5062 (40.1)	239 (41.9)
Waist circumference (cm)	87.5 (12.4)	92.3 (12.6)***	87.8 (12.4)	93.2 (13.7)***	87.7 (12.5)	91.9 (11.9)***
BMI (kg/m ²)	26.6 (3.9)	26.9 (4.1)**	26.6 (3.9)	27.2 (4.5)*	26.6 (4.0)	27.1 (3.9)**
Total energy intake (kJ/day)	8217.8 (2381.7)	8436.3 (2411.4)**	8231.0 (2381.4)	8487.4 (2510.1)	8229.5 (2392.1)	8426.3 (2218.2)
Physical activity (MET × h/day)	17.2 (7.9)	13.2 (7.3)***	17.0 (7.9)	12.4 (7.0)***	17.0 (7.9)	14.6 (7.8)***
TV time (h/day)	3.1 (1.4)	3.5 (1.6)***	3.1 (1.4)	3.7 (1.6)***	3.1 (1.4)	3.4 (1.5)***

P* < 0.05; *P* < 0.01; ****P* < 0.001 by vital status.

SD, standard deviation

Table 2 Descriptive characteristics [mean (SD) or *N* (%)] at baseline (second health check) by TV tertiles (lowest: <2.5, middle: 2.5–3.6, highest tertile: >3.6 h/day) in 13 197 men and women in EPIC-Norfolk

Characteristics	TV tertiles		
	Lowest	Middle	Highest
Men (<i>n</i>)	2015	1952	1762
Age (years)	60.0 (8.7)	61.9 (9.1)	65.1 (8.6)***
Education level, <i>N</i> (%)			
Low	354 (17.6)	510 (26.1)	652 (37.0)***
O level	161 (8.0)	181 (9.3)	176 (10.0)
A level	940 (46.7)	993 (50.8)	792 (44.9)
Degree	560 (27.7)	268 (13.8)	142 (8.1)
Cigarette smoking, <i>N</i> (%)			
Current	134 (6.7)	150 (7.7)	183 (10.4)***
Former	984 (48.8)	1074 (55.0)	1045 (59.3)
Never	897 (44.5)	728 (37.3)	534 (30.3)
Alcohol consumption (U/week)	10.8 (12.0)	9.6 (11.1)	9.4 (11.7)***
Anti-hypertensive drug, <i>N</i> (%)	309 (15.3)	387 (19.8)	448 (25.4)***
Lipid-lowering drug, <i>N</i> (%)	58 (2.9)	71 (3.6)	77 (4.4)*
Waist circumference (cm)	94.2 (9.6)	95.9 (9.5)	97.9 (9.9)***
History of diabetes, <i>N</i> (%)	53 (2.6)	71 (3.6)	78 (4.4)*
Family history of CVD, <i>N</i> (%)	973 (48.3)	963 (49.3)	892 (50.6)
Family history of cancer, <i>N</i> (%)	779 (38.7)	781 (40.0)	687 (39.0)
BMI (kg/m ²)	26.3 (3.2)	26.9 (3.3)	27.4 (3.4)***
Total energy intake (kJ/day)	8874.6 (2532.1)	8909.0 (2477.0)	8872.8 (2507.4)
Physical activity (MET × h/day)	18.4 (9.0)	17.7 (9.2)	14.3 (8.2)***
Women (<i>n</i>)	2382	2509	2577
Age (years)	58.4 (8.9)	60.8 (8.7)	63.7 (8.6)***
Education level, <i>N</i> (%)			
Low	527 (22.1)	915 (36.5)	1322 (51.3)***
O level	288 (12.1)	341 (13.6)	319 (12.4)
A level	993 (41.7)	989 (39.4)	821 (31.9)
Degree	574 (24.1)	264 (10.5)	115 (4.4)
Cigarette smoking, <i>N</i> (%)			
Current	180 (7.6)	200 (8.0)	240 (9.3)**
Former	718 (30.1)	819 (32.6)	871 (33.8)
Never	1484 (62.3)	1490 (59.4)	1466 (56.9)
Alcohol consumption (U/week)	5.1 (6.1)	4.7 (5.7)	3.9 (5.1)***
Antihypertensive drug, <i>N</i> (%)	388 (16.3)	496 (19.8)	674 (26.2)***
Lipid lowering drug, <i>N</i> (%)	45 (1.9)	84 (3.3)	117 (4.5)***
History of diabetes, <i>N</i> (%)	36 (1.5)	52 (2.1)	73 (2.8)**
Family history of CVD, <i>N</i> (%)	1233 (51.8)	1325 (52.8)	1340 (52.0)
Family history of cancer, <i>N</i> (%)	953 (40.0)	1024 (40.8)	1077 (41.8)
Waist circumference (cm)	79.6 (10.2)	81.6 (10.6)	83.9 (11.0)***
BMI (kg/m ²)	25.7 (4.1)	26.5 (4.3)	27.3 (4.5)***
Total energy intake (kJ/day)	7624.1 (2094.2)	7662.7 (2161.5)	7973.0 (2234.0)***
Physical activity (MET × h/day)	17.9 (7.2)	17.1 (6.8)	15.5 (6.6)***

P* < 0.05; *P* < 0.01; ****P* < 0.001 across TV tertiles.

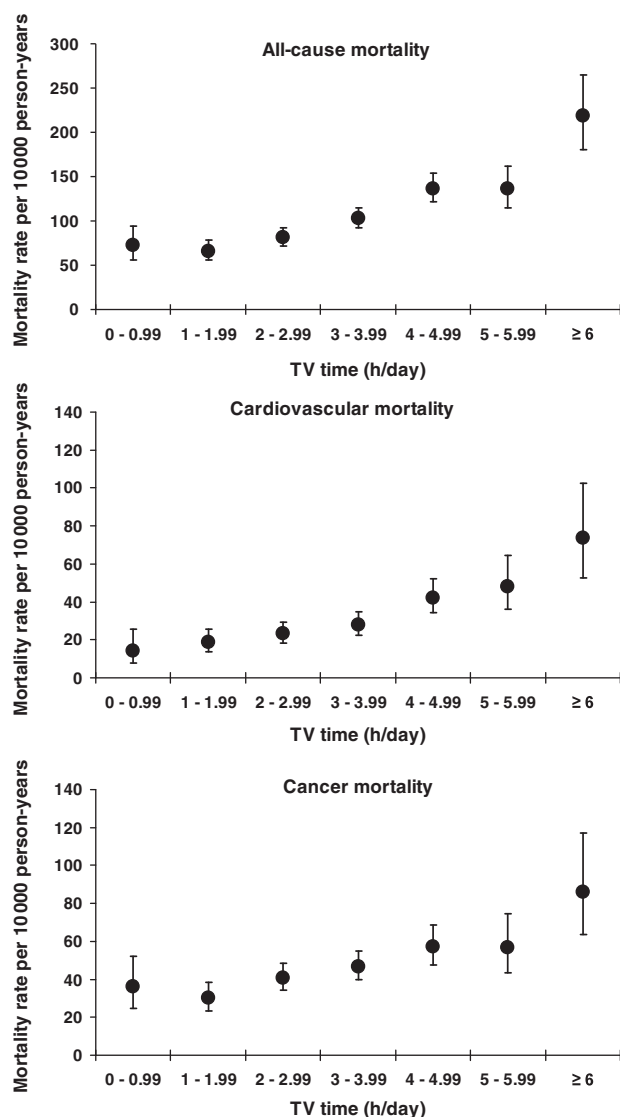


Figure 1 Unadjusted all-cause, cardiovascular and cancer mortality rates (95% CI) per 10 000 person-years by 1-h increments of TV time (h/day) in 13 197 men and women in EPIC-Norfolk, 1998–2009 (*n* television categories: 0–0.99: 818; 1–1.99: 2206; 2–2.99: 3314; 3–3.99: 3192; 4–4.99: 2149 and 5–5.99: 993; ≥6: 525)

diabetes history and family history of CVD and cancer (Model B). These results were virtually unchanged after further adjustment for total PAEE (Model C). A 1-h/day increase in TV time was associated with an increased hazard of death from CVD of 8% and from all causes of 5%. Adjustment for social class (2% missing) instead of education level did not materially change results in any of the analyses (data not shown). Results were also materially unchanged after excluding participants who died within the first 2 years of follow-up (data not shown).

Table 4 shows HRs (95% CI) for all-cause mortality derived from Model C stratified by gender, age, education, BMI and PAEE. HRs were in the same direction and differences between subgroups were small. Consistently, after including multiplicative interaction terms in Model C in the total sample, interactions among TV time and gender, age, education and BMI were non-significant for all three outcomes. Also, no significant interaction was found between TV and total PAEE for all-cause ($P=0.46$), cardiovascular ($P=0.45$) or cancer mortality ($P=0.45$), supporting the similar association between TV and mortality between high and low physically active groups.

To examine whether central and general adiposity mediated the associations between TV and mortality, BMI and waist circumference were added to Model C. The inclusion of BMI had virtually no effect on the results for all-cause (HR=1.04, 95% CI=1.01–1.09, $P=0.03$), or cardiovascular mortality (HR=1.07, 95% CI=1.01–1.15, $P=0.04$; results for cancer mortality: HR=1.04, 95% CI=0.98–1.10, $P=0.20$). Adding waist circumference to Model C attenuated the effect of TV on both all-cause (HR=1.04, 95% CI=0.99–1.08, $P=0.07$) and cardiovascular mortality (HR=1.07, 95% CI=0.99–1.15, $P=0.06$) to a marginally non-significant level (results for cancer mortality: HR=1.03, 95% CI=0.97–1.09, $P=0.31$).

No evidence was found for a potential mediation effect of diet. Comparable significant HRs per h/day increase in TV time were obtained after including total energy intake in Model C ($n=10\,431$): all-cause: HR=1.05, 95% CI=1.01–1.10, $P=0.03$; cardiovascular: HR=1.10, 95% CI=1.02–1.20, $P=0.02$ and

Table 3 HRs (95% CI) for all-cause, cardiovascular and cancer mortality per h/day increase in TV time in 13 197 men and women in EPIC-Norfolk, 1998–2009

Models	All-cause mortality		Cardiovascular mortality		Cancer mortality	
	HR (95% CI)	<i>P</i> -value	HR (95% CI)	<i>P</i> -value	HR (95% CI)	<i>P</i> -value
Model A	1.08 (1.04–1.13)	<0.001	1.14 (1.07–1.22)	<0.001	1.06 (1.01–1.13)	0.03
Model B	1.05 (1.01–1.09)	0.01	1.09 (1.02–1.17)	0.01	1.04 (0.98–1.10)	0.18
Model C	1.05 (1.01–1.09)	0.03	1.08 (1.01–1.16)	0.02	1.04 (0.98–1.10)	0.18

Participants with history of stroke, myocardial infarction or cancer at baseline were excluded. Model A: adjusted for age and gender; Model B: Model A additionally adjusted for education level, smoking status, alcohol consumption, medication for hypertension (not in models examining cancer mortality), medication for dyslipidaemia (not in models examining cancer mortality), baseline history of diabetes, family history of CVD and family history of cancer; Model C: Model B additionally adjusted for total PAEE (MET × h/day). Examination of the Schoenfeld residuals and the Kaplan–Meier plots indicated that the proportional hazards assumption was reasonable for these data.

Table 4 HRs (95% CI) for all-cause mortality per h/day increase in TV time in subgroups according to gender, age, education level, BMI and physical activity energy expenditure level, in 13 197 men and women in EPIC-Norfolk, 1998–2009

Classification	Category (number of deaths/ <i>n</i>)	HR (95% CI)
By gender ^a	Men (725/5729)	1.03 (0.98–1.09)
	Women (545/7468)	1.06 (1.01–1.12)
By age	≤60 years of age (193/6293)	1.09 (0.99–1.20)
	>60 years of age (1077/6904)	1.04 (0.99–1.08)
By education level	Low or O level (651/5746)	1.04 (0.99–1.09)
	A level or degree (619/7451)	1.05 (0.99–1.11)
By BMI	Normal weight (414/4820)	1.07 (1.00–1.15)
	Overweight or obese (856/8377)	1.04 (0.99–1.09)
By PAEE level	Low (890/6486)	1.05 (1.01–1.10)
	High (380/6711)	1.03 (0.96–1.11)

Participants with history of stroke, myocardial infarction or cancer at baseline were excluded. Models are adjusted for age, gender, education level, smoking status, alcohol consumption, medication for hypertension, medication for dyslipidaemia, baseline history of diabetes, family history of CVD, family history of cancer and total physical activity energy expenditure.

^aModels did not include gender.

cancer mortality: HR = 1.02, 95% CI = 0.96–1.09, $P = 0.51$.

The PAF for all-cause mortality of being part of the highest compared with the lowest TV tertile was 5.4%. This suggests that 5.4% of deaths from all causes could be reduced if those watching TV for >3.6 h/day would watch <2.5 h/day instead.

Discussion

The results from this prospective population-based cohort study suggest that high levels of TV time are associated with an increased risk of death from all causes and CVD. These effects are independent of total physical activity and other relevant covariates and are similar between low and high physically active groups. These findings strengthen the available evidence on independent detrimental effects of excess TV time, previously observed in cross-sectional and prospective studies on intermediate risk factors, including cardio-metabolic biomarkers and the metabolic syndrome,^{5–13} obesity,^{14,15} abnormal glucose metabolism and type 2 diabetes.^{15–18} They also extend recent findings from a smaller population-based sample of Australian adults suggesting a linear increase in risk for all-cause (284 cases) and cardiovascular mortality (87 cases), but not cancer mortality (125 cases), with every 1-h increment in TV time, independently of leisure exercise and other confounders.²² Another recent study specifically focusing on cardiovascular mortality in US men did not show an independent association with TV time.²³

Other previous studies have examined the association between daily sitting time and mortality.^{29–31} For example, a population-based study in 17 013

Canadian adults showed a progressively higher risk for mortality across five groups of sitting time ('almost none of the time' to 'almost all of the time') from all causes and CVD (P for trend <0.0001), but not cancer.²⁹ In a population-based sample of 83 034 Japanese middle-aged adults, sitting ≥8 h/day was associated with an 18% higher risk for premature death from all causes compared with sitting <3 h/day, independent of physical activity levels in men, but not women.³¹

Assuming causality, 5.4% of deaths could be reduced if those watching TV >3.6 h/day would watch <2.5 h/day instead. For comparison, we calculated the PAF for all-cause mortality comparing those being part of the lowest PAEE tertile (<12.8 MET × h/day) with the highest (>19.3 MET × h/day), which was 9.8%. Although the public health effect of shifting from the highest to the lowest TV tertile appears to be lower than that of shifting from the lowest to the highest PAEE tertile, an excess rate of 5.4% for total mortality is still substantial. Given the independence of the TV/mortality and PAEE/mortality associations, substituting TV time by physical activity may result in an additive reduction of mortality. TV time, however, increases with increasing age but may also change over time.² Therefore, the PAF for comparing the highest with the lowest TV tertile as shown in this middle-aged cohort may not generalize to younger populations.

Although watching TV is only one component of overall sedentary behaviour, we specifically focused on this behaviour for several reasons. First, it is highly prevalent (e.g. one of the three main activities in the UK besides sleeping and working),² and the most frequently reported sedentary behaviour during leisure time in UK,² the USA³ and Australia.⁴

Consequently, watching TV is likely a strong indicator of overall leisure-time sedentary behaviour.³² Secondly, it is probably one of the sedentary behaviours that is most susceptible to voluntary change compared with other types of prolonged sitting (e.g. occupational sitting). Therefore, reducing TV time is a potential target for behavioural change in future intervention studies. Finally, TV might trigger adverse pathways that are additional to those (physiological) pathways hypothesized for sitting *per se*.¹⁹ For example, a recent study demonstrated that exposure to snack-food advertisement during TV watching triggered automatic eating behaviours that were independent of hunger in children and adults.³³

Potential pathways linking TV time to all-cause and cardiovascular mortalities are likely to be diverse and need further exploration. Some possible mechanisms were examined within the current study. First of all, extensive adjustment for PAEE diminishes the likelihood that our results can be explained by displacement of (even low intensity) physical activity. Secondly, as previously suggested, high exposure to high-caloric food advertisements increases (unconscious) snacking and the overall amount of food consumed.^{33,34} However, the effect of TV time on mortality in our study was independent of total energy intake. Given the amount of measurement error usually associated with self-reported dietary intake, we cannot exclude residual confounding by dietary intake. Our results were, however, independent of overall adiposity (BMI). In contrast, central obesity (i.e. waist circumference) might have a mediating role between TV time and mortality, although the associations were only marginally non-significant after adjustment for this variable. Causal inference cannot be made, however, about the association between TV time and all other variables measured at baseline. Higher levels of adiposity may also lead to increased TV time, as recently shown for objectively measured total sedentary time.³⁵ Other pathways include intermediate cardiovascular and metabolic risk factors previously suggested to be independently associated with TV time.^{5–19} Current and future studies examining pathophysiological processes that are specific for sedentary behaviours and not just the effect of absence of physical activity are an important and emerging area of research, which may support the current epidemiological evidence. One of these biological processes, shown in animal models, is the acute drop in skeletal muscle lipoprotein lipase activity caused by muscle inactivity (absence of muscle contractions) and resulting in disruption of triglyceride and high-density lipoprotein cholesterol metabolism.³⁶ Consistently, observations in free-living humans by objective monitoring of body movement suggested that regular interruptions in sedentary time are beneficially associated with level of triglycerides,³⁷ and urge the need for future epidemiological and experimental studies further exploring these findings.

The present study has several strengths. The large population-based sample enabled us to control for potential reverse causality by excluding participants with relevant health conditions at baseline and by additionally excluding those who died within the first 2 years of follow-up. Furthermore, we were able to adjust for non-categorized overall PAEE and not just (categorized) time spent in leisure, or moderate to vigorous physical activity, minimizing residual confounding. In addition to previously demonstrated criterion validity,²⁵ our PAEE measure has previously also been shown to predict cardiovascular and all-cause mortality in this cohort.²⁷ We also adjusted our analyses for several relevant covariates, including energy intake. However, the following study limitations should also be highlighted. First, TV time was self-reported. Measurement error may have biased the associations observed towards the null, leading to an underestimation of the true associations. Furthermore, as TV time was only measured at baseline, changes in this behaviour might additionally have caused misclassification and, therefore, attenuation of the associations found. The self-report and categorical nature of some covariates may have resulted in residual confounding. Some confounding might still exist for other non-measured psychosocial determinants of health, such as social network interaction/loneliness, which might be correlated with TV time, especially in older individuals.³⁸ Finally, as we excluded participants with baseline chronic disease to minimize the possibility of reverse causality and increase internal validity, the associations found are relative to this cohort of healthy middle-aged Caucasians, and may not generalize to other populations. Large prospective studies, using repeated measurements with longer duration of follow-up and preferably using objective measures of physical activity and sedentary time, are warranted to extend the present results and confirm dose-response effects and indicators of population health risk on different types of mortality in different populations.

In conclusion, in this population-based cohort of middle-aged Caucasians, higher levels of TV time increased the risk for mortality from all causes and CVD, independently of physical activity levels. Given the high prevalence of excessive TV watching, resulting in a substantial PAF, these results indicate the importance of public health recommendations aimed at decreasing TV time and possibly overall sedentary behaviour. These recommendations should be in addition to the established public health recommendations for physical activity.

Funding

Cancer Research Campaign; the Medical Research Council; the Stroke Association; the British Heart Foundation; the Department of Health; the Europe Against Cancer Programme Commission of the

European Union and the Ministry of Agriculture, Fisheries and Food.

Acknowledgements

We are tremendously grateful to Prof. Sheila Rodwell, principal investigator and key contributor to the

EPIC-Norfolk study, who sadly passed away shortly before finalization of this manuscript. The authors also thank the EPIC-Norfolk staff and participants for their invaluable contributions.

Conflict of interest: None declared.

KEY MESSAGES

- The findings from this large population-based cohort of middle-aged Caucasians indicate that each 1-h increment in TV time is independently associated with a 4 and 7% increased risk of all-cause and cardiovascular mortality, respectively.
- Given the high prevalence of excessive TV watching, recommendations aimed at decreasing TV time might have a relevant impact on public health.
- As the associations found are independent of physical activity levels, these recommendations should be in addition to the established public health recommendations for physical activity.

References

- Matthews CE, Chen KY, Freedson PS *et al.* Amount of time spent in sedentary behaviors in the United States, 2003–2004. *Am J Epidemiol* 2008;**167**:875–81.
- Office for National Statistics. *The Time Use Survey, 2005. How We Spend Our Time*. London, UK: Office for National Statistics, 2006.
- Nielsen Media Research. *Nielsen Report on Television*. New York, NY: Nielsen Media Research, 2000.
- Australian Bureau of Statistics. *How Australians Use Their Time, 2006*. Canberra, Australia: Commonwealth of Australia, 2008.
- Dunstan D, Salmon J, Owen N *et al.* Associations of TV viewing and physical activity with the metabolic syndrome in Australian adults. *Diabetologia* 2005;**48**: 2254–61.
- Bertrais S, Beyeme-Ondoua JP, Czernichow S, Galan P, Hercberg S, Oppert JM. Sedentary behaviors, physical activity, and metabolic syndrome in middle-aged French subjects. *Obes Res* 2005;**13**:936–44.
- Ford ES, Kohl HW, Mokdad AH, Ajani UA. Sedentary behavior, physical activity, and the metabolic syndrome among US adults. *Obes Res* 2005;**13**:608–14.
- Aadahl M, Kjaer M, Jorgensen T. Influence of time spent on TV viewing and vigorous intensity physical activity on cardiovascular biomarkers. The Inter 99 study. *Eur J Cardiovasc Prev Rehabil* 2007;**14**:660–65.
- Wijndaele K, Duvigneaud N, Matton L *et al.* Sedentary behaviour, physical activity and a continuous metabolic syndrome risk score in adults. *Eur J Clin Nutr* 2009;**63**: 421–29.
- Burazeri G, Goda A, Kark JD. Television viewing, leisure-time exercise and acute coronary syndrome in transitional Albania. *Prev Med* 2008;**47**:112–15.
- Fung TT, Hu FB, Yu J *et al.* Leisure-time physical activity, television watching, and plasma biomarkers of obesity and cardiovascular disease risk. *Am J Epidemiol* 2000;**152**:1171–78.
- Hancox RJ, Milne BJ, Poulton R. Association between child and adolescent television viewing and adult health: a longitudinal birth cohort study. *Lancet* 2004;**364**:257–62.
- Healy GN, Dunstan DW, Salmon J, Shaw JE, Zimmet PZ, Owen N. Television time and continuous metabolic risk in physically active adults. *Med Sci Sports Exerc* 2008;**40**: 639–45.
- Jakes RW, Day NE, Khaw KT *et al.* Television viewing and low participation in vigorous recreation are independently associated with obesity and markers of cardiovascular disease risk: EPIC-Norfolk population-based study. *Eur J Clin Nutr* 2003;**57**:1089–96.
- 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* 2003;**289**:1785–91.
- Hu FB, Leitzmann MF, Stampfer MJ, Colditz GA, Willett WC, Rimm EB. Physical activity and television watching in relation to risk for type 2 diabetes mellitus in men. *Arch Intern Med* 2001;**161**:1542–48.
- Dunstan DW, Salmon J, Owen N *et al.* Physical activity and television viewing in relation to risk of undiagnosed abnormal glucose metabolism in adults. *Diab Care* 2004;**27**:2603–9.
- Dunstan DW, Salmon J, Healy GN *et al.* Association of television viewing with fasting and 2-h postchallenge plasma glucose levels in adults without diagnosed diabetes. *Diab Care* 2007;**30**:516–22.
- 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 behaviour. *Cur Cardiovasc Risk Reports* 2008;**2**: 292–98.
- Howard RA, Freedman DM, Park Y, Hollenbeck A, Schatzkin A, Leitzman MF. Physical activity, sedentary behavior, and the risk of colon and rectal cancer in the NIH-AARP Diet and Health Study. *Cancer Causes Control* 2008;**19**:939–53.

- ²¹ Zhang M, Xie X, Lee AH, Binns CW. Sedentary behaviours and epithelial ovarian cancer risk. *Cancer Causes Control* 2004;**15**:83–89.
- ²² Dunstan DW, Barr ELM, Healy GN *et al.* Television viewing time and mortality. The Australian Diabetes, Obesity and Lifestyle Study (AusDiab). *Circulation* 2010;**121**:384–91.
- ²³ Warren TY, Barry V, Hooker SP, Sui X, Church TS, Blair SN. Sedentary behaviors increase risk of cardiovascular disease mortality in men. *Med Sci Sports Exerc* 2010;**42**:879–85.
- ²⁴ Day N, Oakes S, Luben R *et al.* EPIC-Norfolk: study design and characteristics of the cohort. *Br J Cancer* 1999;**80**:95–103.
- ²⁵ Wareham NJ, Jakes RW, Rennie KL, Mitchell J, Hennings S, Day NE. Validity and repeatability of the EPIC-Norfolk Physical Activity Questionnaire. *Int J Epidemiol* 2002;**31**:168–74.
- ²⁶ Ainsworth BE, Haskell WL, Whitt MC. Compendium of physical activity: an update of activity codes and MET intensities. *Med Sci Sports Exerc* 2000;**32**:S498–516.
- ²⁷ Besson H, Ekelund U, Brage S *et al.* Relationship between subdomains of total physical activity and mortality. *Med Sci Sports Exerc* 2008;**40**:1909–15.
- ²⁸ Bingham SA, Gill C, Welch A *et al.* Validation of dietary assessment methods in the UK arm of EPIC using weighed records, and 24-hour urinary nitrogen and potassium and serum vitamin C and carotenoids as biomarkers. *Int J Epidemiol* 1997;**26**:S137–51.
- ²⁹ Katzmarzyk PT, Church T, Craig CL, Bouchard C. Sitting time and mortality from all causes, cardiovascular disease and cancer. *Med Sci Sports Exerc* 2009;**41**:998–1005.
- ³⁰ Weller I, Corey P. The impact of excluding non-leisure energy expenditure on the relation between physical activity and mortality in women. *Epidemiology* 1998;**9**:632–35.
- ³¹ Inoue M, Iso H, Yamamoto S *et al.* Daily total physical activity level and premature death in men and women: results from a large-scale population-based cohort study in Japan (JPHC study). *Ann Epidemiol* 2008;**18**:522–30.
- ³² Sugiyama T, Healy GN, Dunstan DW, Salmon J, Owen N. Is television viewing time a marker of a broader pattern of sedentary behavior? *Ann Behav Med* 2008;**35**:245–50.
- ³³ Harris JL, Bargh JA, Brownell KD. Priming effects of television food advertising on eating behavior. *Health Psychol* 2009;**28**:404–13.
- ³⁴ Foster JA, Gore SA, West DS. Altering TV viewing habits: an unexplored strategy for adult obesity intervention? *Am J Health Behav* 2006;**30**:3–14.
- ³⁵ Ekelund U, Brage S, Besson H, Sharp S, Wareham NJ. Time spent being sedentary and weight gain in healthy adults: reverse or bidirectional causality? *Am J Clin Nutr* 2008;**88**:612–17.
- ³⁶ Hamilton MT, Hamilton DG, Zderic TW. Role of low energy expenditure and sitting in obesity, metabolic syndrome, type 2 diabetes, and cardiovascular disease. *Diabetes* 2007;**56**:2655–67.
- ³⁷ Healy GN, Dunstan DW, Salmon J *et al.* Breaks in sedentary time - Beneficial associations with metabolic risk. *Diabetes Care* 2008;**31**:661–66.
- ³⁸ Grajczyk A, Zöllner O. How older people watch television. Telemetric data on the TV use in Germany in 1996. *Gerontology* 1998;**44**:176–81.