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Screen-Based Entertainment Time, All-Cause Mortality, and Cardiovascular Events

Population-Based Study With Ongoing Mortality and Hospital Events Follow-Up

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Objectives The aim of this study was to examine the independent relationships of television viewing or other screen-based entertainment ("screen time") with all-cause mortality and clinically confirmed cardiovascular disease (CVD) events. A secondary objective was to examine the extent to which metabolic (body mass index, high-density lipoprotein and total cholesterol) and inflammatory (C-reactive protein) markers mediate the relationship between screen time and CVD events.

Background Although some evidence suggests that prolonged sitting is linked to CVD risk factor development regardless of physical activity participation, studies with hard outcomes are scarce.

Methods A population sample of 4,512 (1,945 men) Scottish Health Survey 2003 respondents (≥35 years) were followed up to 2007 for all-cause mortality and CVD events (fatal and nonfatal combined). Main exposures were interviewer-assessed screen time (<2 h/day; 2 to <4 h/day; and ≥4 h/day) and moderate to vigorous intensity physical activity.

Results Two hundred fifteen CVD events and 325 any-cause deaths occurred during 19,364 follow-up person-years. The covariable (age, sex, ethnicity, obesity, smoking, social class, long-standing illness, marital status, diabetes, hypertension)-adjusted hazard ratio (HR) for all-cause mortality was 1.52 (95% confidence interval [CI]: 1.06 to 2.16) and for CVD events was 2.30 (95% CI: 1.33 to 3.96) for participants engaging in ≥4 h/day of screen time relative to <2 h/day. Adjusting for physical activity attenuated these associations only slightly (all-cause mortality: HR: 1.48, 95% CI: 1.04 to 2.13; CVD events: HR: 2.25, 95% CI: 1.30 to 3.89). Exclusion of participants with CVD events in the first 2 years of follow-up and previous cancer registrations did not change these results appreciably. Approximately 25% of the association between screen time and CVD events was explained collectively by C-reactive protein, body mass index, and high-density lipoprotein cholesterol.

Conclusions Recreational sitting, as reflected by television/screen viewing time, is related to raised mortality and CVD risk regardless of physical activity participation. Inflammatory and metabolic risk factors partly explain this relationship. (J Am Coll Cardiol 2011;57:292–9) © 2011 by the American College of Cardiology Foundation

There is indisputable evidence on the links between physical activity and risk for premature death (1). Some emerging published reports consistently suggest that excessive sedentary behavior (as characterized by those activities involving sitting) might be linked to increased risk for obesity (2,3), dyslipidemia (4), plasma glucose levels (5), and the metabolic syndrome (6) independently of moderate-to-vigorous physical activity participation. Television viewing and screen-based entertainment (screen time) in general seems to be the most important indicator of nonoccupational sitting behavior (7). Recent time-use surveys (8–10) indicate that, aside from sleeping, watching TV is the behavior that occupies the most time in the domestic setting.

If sitting or total sedentary time is established to be independently associated with cardiovascular disease (CVD), clinical and public health recommendations should explicitly address sitting in addition to physical activity; currently they do

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not (11). Because only a minority of adults in western populations participate regularly in sport and exercise activities (12,13), and those who do not take part in sports are more likely to develop CVD or die prematurely (14), it might be possible to reduce the risk of nonparticipants by restricting sitting time and increasing nonexercise activity (e.g., standing and ambulating) throughout the day (15). There is no conclusive evidence obtained from comparing the feasibility or long-term effectiveness of interventions designed to increase formal exercise versus decreasing sitting behavior during the day. However, the latter approach might be more promising in terms of long-term adherence, because it will involve more subtle lifestyle changes and fewer of the commonly cited barriers (16) for joining a sporting or lifestyle exercise program.

The primary aim of this study was to examine the relationships of leisure-time sitting behavior (indexed from screen time) with all-cause mortality and CVD events while taking multiple measures to address reverse causality. Because it is also important to understand the mechanisms through which sedentary behavior might influence cardiovascular risk, a secondary aim was to determine the extent to which several biomarkers explain these relationships.

Methods

Sample and outcomes. The 2003 Scottish Health Survey (SHS03) was a household-based survey that recruited a population sample with multistage, stratified probability sampling with postcode sectors selected at the first stage and household addresses selected at the second stage (17). Ethical approval was granted by the Local Research Ethics Councils. Of eligible adults, 83% consented to take part in the survey. The SHS03 data were linked to the Scottish Information Division Database (ISD) patient-based database of hospital episodes (from 1981 onwards) and deaths up to December 2007. The linked data are of excellent quality—the ISD database has demonstrated 94% accuracy and 99% completeness when samples of computerized CVD records from the Scottish national database were compared with the original patient case notes. Information on deaths was ascertained from the General Registrar Office for Scotland. Classification of the underlying cause of death is based on information collected on the medical certificate of cause of death together with any additional information provided subsequently by the certifying doctor. All the relevant details regarding the ISD can be found at the ISD Scotland website. Diagnoses for CVD cause of death was recorded with the International Classification of Diseases-9 (codes 390-459) and -10 (codes I01-I99). An event was defined as CVD-related hospital episode (including myocardial infarction, coronary artery bypass, angioplasty, stroke, heart failure) or CVD-related death. The potentially eligible sample comprised 6,353 adults (≥35 years), of which 5,814 (91.5% of eligible) consented to their records being linked to records of mortality, hospital episodes, and cancer registration. Among these, 1,302 (22.4% of consenting) were lost to follow-up, leaving 4,512 respondents (1,964 male) who comprised the core sample for the present study (71.0% of eligible). We carried out comparisons between those who consented and those who did not consent to be followed up with likelihood ratios (categorical variables) or Student *t* tests (continuous variables). Compared with those who did not consent, those who consented were older; reported fewer moderate-to-vigorous physical activity and more screen time min/week; and more likely to be from nonmanual social class, white, not to be married, to have a body mass index (BMI) under 30 kg/m^2, to be current or former cigarette smokers, to have long-standing illness, to be inactive at work, to have been diagnosed with hypertension, and not to meet the physical activity recommendations. We also carried out comparisons between those 1,302 participants who were lost to follow-up and those 4,512 who were retained in the analyses. Those who were lost to follow-up were younger and reported more moderate-to-vigorous physical activity and less screen time min/week than those who were retained. They were also were more likely to be from nonmanual social class, to be single, to be current or ex-cigarette smokers, to be free from long-standing illness and doctor-diagnosed hypertension, and to meet the physical activity recommendations.

Abbreviations and Acronyms

BMI = body mass index
CI = confidence interval
CRP = C-reactive protein
CVD = cardiovascular disease
HDL = high-density lipoprotein
HR = hazard ratio

Analyses with cardiovascular events as the outcome excluded 340 respondents who had cardiovascular hospital episodes according to the linked patient-based database between 1981 and before baseline testing. To minimize the chances of reverse causality due to prodromal/undiagnosed disease, we repeated the analyses after excluding another 48 participants with cardiovascular events in the first 24 months of follow-up (CVD analysis). We also repeated the analysis after excluding the 295 participants who had cancer registrations before baseline.

Exposures, confounders, and potentially mediating variables. The main exposure was screen time. Two questions enquired about screen time on weekdays ("Thinking of weekdays, how much time on average day do you spend watching TV or another type of screen such as a computer, or video game? Please do not include any time spent in front of a screen while at school, college or work") and weekend days (with an equivalent question). Although there is no information on the reliability and criterion validity of the screen time questions, the previously reported (2) consistent direct correlations of screen time with waist circumference and BMI and the inverse correlation with physical activity support their convergent validity. Nonoccupational physical activity questions included frequency (days in the last 4 weeks) and duration (min/day) of heavy housework (e.g., scrubbing floors), heavy do-it-yourself/gardening (e.g., digging, building work), walking (14), and any leisure-time exercise (e.g., cycling, swimming, aerobics, calisthenics,

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gym, dancing, football) (12). Occupational physical activity was assessed by asking respondents how physically active they are at work (very/fairly active, not very/not at all active). Their response was combined with information on their occupation with the Standard Occupational Classification 1990 (18) to classify work activity. The criterion validity of the physical activity questionnaire is supported by an accelerometry study on 106 British adults (19). Height, weight, socioeconomic status, health status, and other health behaviors were measured by trained interviewers with standard protocols (2,17). In a separate visit, trained nurses collected nonfasting blood samples with standard protocols and procedures that have been described previously in detail (14,20). Blood sample analytes used in the present analysis were C-reactive protein (CRP), high-density lipoprotein (HDL) cholesterol, and total cholesterol (17,21).

Variable handling and statistical analysis. Screen time was grouped as <2 h/day; ≥2 h/day <4 h/day; ≥4 h/day. The choice of 2 h/day as a cutoff for the lowest screen time group is consistent with recommendations for children (20,22) that make specific references to TV. The same cutoff has been used in publications similar to ours (23). The main confounding variable was nonoccupational moderate-to-vigorous physical activity, which was entered in the statistical models as min/day. Other covariables entered into the models were sex, age, BMI (<25, 25 to 30, >30 kg/m^2), social class (I, II, III nonmanual, III manual, IV/V nonmanual), doctor-diagnosed diabetes and hypertension, longstanding illness, marital status (single/never married, married, separated/divorced, and widowed), smoking (never, ex, current smoker), and occupational physical activity (inactive/light/moderate-to-vigorous).

For individuals who survived and remained CVD-free, data were censored to December 2007. The Cox proportional hazards model was used with months as the time scale to estimate the risk of death from any cause or the risk of CVD event by screen time level. The proportional hazards assumption was examined by comparing the cumulative hazard plots grouped on exposure, although no appreciable violations were noted. Test for linear trend was obtained by entering the categorical variables as continuous parameters in the models. We applied Cox models that were adjusted for age and sex (Model 1), plus all covariables minus physical activity (Model 2), plus physical activity (Model 3). To account for the skewed distribution of physical activity, in an alternative analysis we re-ran the Cox models with physical activity as a categorical variable (no physical activity vs. some physical activity, <150 min/week vs. ≥150 min/week), but because results were not appreciably different, we only present the models with the continuous physical activity variables. To further address the issue of reverse causality, we repeated the Cox models after excluding CVD events occurring during the first year of follow-up and cancer registrations before baseline. In another analysis we excluded events in the first 2 years of follow-up and cancer registrations. In these analyses, we dichotomized the screen time variable to <2 and ≥2 h/day to preserve statistical power. For the same reason, we used the same dichotomous screen time variable when we stratified our analyses by sex, physical activity level (<150 min/week vs. ≥150 min/week; no physical activity/any physical activity), BMI level (<25 kg/m^2 vs. ≥25 kg/m^2), and smoking (noncurrent smoker vs. current smoker). To provide a direct comparison for the potential hazard of screen time and the potential benefit of physical activity, we ran analogous Cox models with physical activity as the main exposure with adjustments for: 1) age and sex; 2) plus nonscreen time covariables; 3) plus screen time. To enable direct comparisons, both screen time and physical activity were entered as continuous variables in this analysis.

To test the extent to which certain biological risk factors explained the association between sedentary time and cardiovascular events, we used a method similar to that used by us (21) and others (24). This method involved: 1) separately adding CRP, BMI, total cholesterol, and HDL cholesterol into a basic (sex-, age-, and physical activity-adjusted) Cox model; and 2) using the following formula to calculate proportion of CVD risk explained by each biological risk factor:

$$\frac{\text{HR basic model} - \text{HR adjusted}}{\text{HR basic model} - 1} \times 100$$

The CRP was log transformed to improve normality of distribution. All blood variables and BMI were included as continuous variables. Analyses were also run entering risk markers as categorical variables, although this did not appreciably alter the results. We used analysis of variance with Scheffe post hoc tests and chi-square tests to examine univariable relationships of the confounders or potential mediators with the exposure variables.

Analyses were performed with SPSS (version 13, SPSS, Inc., Chicago, Illinois), and all tests of statistical significance were based on 2-sided probability.

Results

A total of 325 any-cause deaths (153 in men) and 215 incident cardiovascular events (107 in men) occurred during 4.3 (±0.5) years of average follow-up and 19,364 person-years at risk in the core sample. Table 1 presents the descriptive characteristics of the core sample.

Cox models. Table 2 shows the hazard ratios (HRs) and 95% confidence intervals (CIs) for all-cause mortality and CVD events. All-cause mortality risk increased with ≥4 h/day of screen time, and CVD event risk increased with ≥2 h/day of screen time. Adjusting for physical activity made very little difference in both types of analyses (Table 2). Excluding deaths or CVD events in the first year of follow-up and participants with previous cancer registration slightly weakened the associations (Table 3). Results were robust to the exclusion of cases with cancer registrations and CVD

Table 1 Descriptive Characteristics of Core Sample by Time Spent on TV Viewing and Other Screen-Based Entertainment

	TV Viewing and Other Screen-Based Entertainment			
	<2 h/day	≥2 and <4 h/day	≥4 h/day	p Value
n	771	2,441	1,300	
Mean age (SD)	55.7 (14.9)	57.2 (13.7)	60.4 (14.1)	<0.001
Sex (% male)	37.9	43.2	47.5	<0.001
Ethnicity (% white)*	96.9	98.4	98.5	0.034
Social class (% manual)*	58.4	65.6	76.0	<0.001
BMI (% >30 kg/m^2)*	18.2	23.6	29.2	<0.001
Marital status (% married/cohabiting)*	59.7	67.8	54.6	<0.001
Smoking status (% never smoked)*	49.7	41.5	32.5	<0.001
Long-standing illness (%)	47.5	51.5	66.5	<0.001
Doctor-diagnosed hypertension	27.3	31.1	39.9	<0.001
Doctor-diagnosed diabetes	3.5	5.2	9.6	<0.001
Mortality				
Died, any cause (%)	5.4	5.6	11.2	<0.001
CVD event, fatal (%)	1.2	2.4	4.9	<0.001
CVD event, nonfatal (%)	6.7	8.6	12.3	<0.001
Person-yrs	3,328	10,548	5,488	
Moderate-to-vigorous physical activity				
Mean (IQR), min/day	41.0 (57.2)	35.4 (49.3)	20.8 (5.7)	<0.001§
Occupational physical activity level, % inactive*	63.4	67.9	82.5	<0.001
Screen-based entertainment				
Median (IQR), min/day	67.3 (35.0)	173.6 (42.9)	381.3 (132.9)	n/a
Explanatory biological risk factors†				
Median CRP (IQR), mg/l ‡	1.3 (2.0)	1.7 (3.1)	2.4 (4.4)	<0.001§
Mean total cholesterol (SD), mmol/l	5.84 (1.09)	5.90 (1.12)	5.99 (1.17)	0.168
Mean HDL cholesterol (SD), mmol/l	1.61 (0.39)	1.54 (0.38)	1.45 (0.37)	<0.001
Mean BMI (SD), kg/m^2	27.0 (4.5)	28.0 (5.0)	28.4 (7.8)	<0.001

Scottish Health Survey 2003, participants 35 years of age and older who consented to their survey data, linked with mortality and hospital stay records. *Only one key category of the variable is shown; †n = 1,928 with valid values in all 4 listed biological variables; ‡C-reactive protein (CRP) was log transformed; §assessed with nonparametric test (Spearman rho) due to its skewed distribution.

BMI = body mass index; CVD = cardiovascular disease; HDL = high-density lipoprotein; IQR = interquartile range.

events in the first 2 years of follow-up (CVD events n = 116): the covariable-adjusted (minus physical activity) HR for those with ≥2 h/day was 1.94 (95% CI: 1.00 to 3.76); further adjustment for physical activity did not appreciably change this result (HR: 1.93, 95% CI: 0.99 to 3.75). When we repeated the main analyses with screen time entered as a continuous variable (min/day), results were similar in terms of direction and strength of the association with CVD events (age- and sex-adjusted HR: 1.0014; 95% CI: 1.0006 to 1.0022, $p < 0.001$; fully adjusted including physical

Table 2 HRs for All-Cause Mortality and CVD Events for Screen-Based Entertainment Groups* Excluding Previous CVD Hospital Stays

		HR (95% CI)		
	Cases/Events	Model 1†	Model 2‡	Model 3§
All-cause mortality				
<2 h/day	791/42	1.00	1.00	1.00
≥2–<4 h/day	2,492/138	1.13 (0.080–1.60)	1.12 (0.79–1.56)	1.14 (0.80–1.62)
≥4 h/day	1,311/146	1.77 (1.25–2.50)	1.52 (1.06–2.16)	1.48 (1.04–2.13)
Trend p value		<0.001	0.013	0.029
CVD events				
<2 h/day	745/18	1.00	1.00	1.00
≥2–<4 h/day	2,333/115	2.20 (1.30–3.71)	2.22 (1.32–3.77)	2.23 (1.31–3.80)
≥4 h/day	1,172/86	2.76 (1.62–7.70)	2.30 (1.33–3.96)	2.25 (1.30–3.89)
Trend p value		0.001	0.009	0.010

*Compared with the referent <2 h/day screen-based entertainment group. †Model 1 covariables: age, sex; ‡Model 2: plus body mass index, smoking, marital status, ethnicity, social class, long-standing illness, occupational physical activity, doctor-diagnosed diabetes and hypertension; §Model 3: plus moderate-to-vigorous physical activity.

CI = confidence interval; CVD = cardiovascular disease; HR = hazard ratio.

Table 3 HRs for All-Cause Mortality and CVD Events With Other Exclusions*

Screen Time	Cases/Events	HR (95% CI) Model 1†	Model 2‡	Model 3§
All-cause mortality				
<2 h/day	742/35	1.00	1.00	1.00
≥2–<4 h/day	1,204/97	1.11 (0.77–1.61)	1.11 (0.77–1.61)	1.14 (0.78–1.65)
≥4 h/day	1,204/115	1.81 (1.26–2.60)	1.57 (1.09–2.28)	1.54 (1.06–2.24)
Trend p value		<0.001	0.008	0.017
CVD events				
<2 h/day	696/14	1.00	1.00	1.00
≥2–<4 h/day	2,176/84	1.95 (1.08–3.50)	1.97 (1.09–3.57)	1.98 (1.09–3.59)
≥4 h/day	1,072/65	2.56 (1.41–4.65)	2.14 (1.16–3.94)	2.10 (1.14–3.88)
Trend p value		0.007	0.047	0.052

*Excluding previous events, cancer registration before baseline, and events during the first year of follow-up, for screen-based entertainment groups (compared with the referent <2 h/day screen-based entertainment group). †Model 1 covariables: age, sex; ‡Model 2: plus body mass index, smoking, marital status, ethnicity, social class, long-standing illness, occupational physical activity, doctor-diagnosed diabetes and hypertension; §Model 3: plus moderate to vigorous physical activity.

Abbreviations as in Table 2.

activity: HR: 1.0010, 95% CI: 1.0002 to 1.0018, p = 0.02) and all-cause mortality (age- and sex-adjusted HR: 1.0018; 95% CI: 1.0013 to 1.0024, p < 0.001; fully adjusted including physical activity: HR: 1.0011, 95% CI: 1.0005 to 1.0017, p < 0.001). Repeating the same analysis with physical activity as the main exposure showed that the protective effect of physical activity on all-cause mortality is independent of screen time (nonscreen time covariable-adjusted HR: 0.9912; 95% CI: 0.9862 to 0.9962, p = 0.001; fully adjusted including screen time: HR: 0.9919, 95% CI: 0.9870 to 0.9968, p = 0.001). The inclusion of screen time weakened the association between physical activity and CVD events (nonscreen time covariable-adjusted HR: 0.9956; 95% CI: 0.9913 to 0.9998, p = 0.041; fully adjusted including screen time: HR: 0.9960, 95% CI: 0.9918 to 1.003, p = 0.07).

Online Table 1 presents analyses stratified by physical activity and other key risk factors. Although the statistical power in certain strata was low due to low number of events, effect estimates were markedly consistent in direction. There was little evidence for an association between screen time and CVD events among those who reported long-standing illness (n = 2,234; 158 CVD events): fully adjusted HR for ≥4 h/day: 1.70, 95% CI: 0.94 to 3.09, p = 0.15. Despite the small number of events among those with no long-standing illness (n = 2,016, 55 CVD events) and the corresponding wide CIs, there was some evidence for an association: HR for ≥4 h/day: 6.51, 95% CI: 1.47 to 28.8, p = 0.046).

Explanatory analysis. A total of 1,928 cases had valid data in all 4 potentially mediating variables (BMI, CRP, total cholesterol, HDL cholesterol), corresponding to 70 CVD events, and were entered in the explanatory analysis. Compared with those excluded, those included had lower mean age, higher physical activity (p = 0.002), and lower screen time (p = 0.001) and were more likely to be married (p = 0.02), have a BMI over 30 kg/m^2, have a long-standing illness (p < 0.001), be inactive at work (p < 0.001), not to have been diagnosed with hypertension (p < 0.001), not to have had a CVD event (p < 0.001), and to have met the physical activity recommendations (p < 0.001) (data not shown). Figure 1 presents the extent to which BMI, HDL and total cholesterol, and CRP explain the associations between screen time and CVD events. CRP explained CVD events to the greatest extent (18%), which was equal to the amount explained by the 3 metabolic factors together. These 4 biological factors explained approximately 28% of the screen time–CVD association, with 25% explained by BMI, HDL cholesterol, and CRP. These 3 variables (but not total cholesterol) met the statistical criteria for being a mediator variable (25).

Discussion

Our results suggest that there is an independent, deleterious relationship of screen-based recreational sitting time with CVD events and all-cause mortality. Compared with those spending <2 h/day on screen-based entertainment, there was a 48% increased risk of all-cause mortality in those spending ≥4 h/day and an approximately 125% increase in risk of CVD events in those spending ≥2 h/day (Table 3). These associations were independent of traditional risk factors such as smoking, hypertension, BMI, social class, as well as physical activity. Our all-cause mortality results are in agreement with a large study of Canadian adults who were followed up for 12 years, where the all-cause mortality HR for the highest category of nonrecreational (work, school, housework) daily sitting time ("almost all the time") was 1.54 (26). Another study among >8,000 Australian adults reported a very similar all-cause mortality HR (1.46) for ≥4 h of TV watching compared with the <2 h/day reference group (23). Both studies were also robust for adjustments or stratifications by sex, physical activity level, smoking, and BMI. The Canadian study, however, had minimal control of physical health at baseline, which makes reverse causation a strong possibility (26). In contrast, we

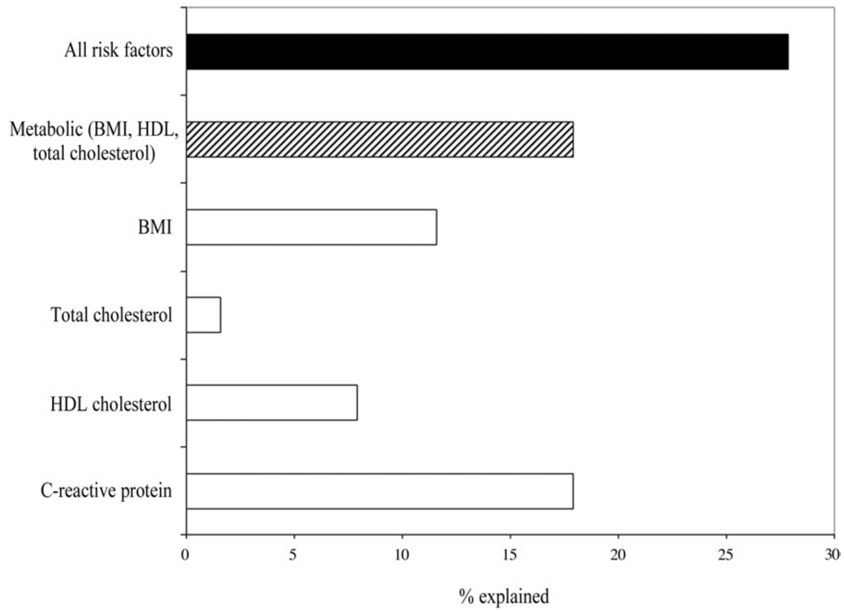

Figure 1 **Extent to Which Biological Risk Factors Explain Association Between Screen-Based Entertainment Time and Cardiovascular Events**

n = 1,928 with valid data on all 4 risk factors. BMI = body mass index; HDL = high-density lipoprotein.

were able to exclude respondents with objectively verified CVD/cancer at baseline and adjust for multiple indicators of health. Our study specifically examined recreational sedentary time. Because the largest proportion of sitting time for many people is spent at work and in many circumstances is difficult to modify, our data imply that reduced recreational sitting time might be linked to reduced risk. Although we found no evidence of a dose-response relationship, our analysis suggests that a threshold of ≥2 h/day of screen time might be linked to an increased risk for a CVD event. The Australian study (23) found that daily TV viewing times in excess of 4 h/day (but not 2 to 4 h/day) were associated with CVD death risk. We speculate that this disagreement occurs because our exposure variable was more inclusive than TV alone that was used in the Australian study and because our CVD outcome included nonfatal as well as fatal events. We were not able to demonstrate, in contrast to the Canadian (26) study, a clear relationship between screen time and CVD events among those who meet the physical activity recommendations and among those with a BMI <25 kg/m^2 (Online Table 1). In this analysis, there were only 50 events, and as such, we speculate that the lack of a robust and statistically important association was due to limited statistical power. Nevertheless, the direction of the association was markedly consistent across all strata, lending support to our main conclusion that screen time is an independent predictor of CVD events. Another large study among U.S. women, Manson et al. (27), found that extreme amounts of sitting (>16 h/day) were linked to an increased risk for incident CVD compared with <4 h/day after 6 years of follow-up. Such levels of sitting imply that an individual spends their entire waking time sitting, but there was no evidence for adverse effects of smaller amounts of daily sitting (27).

Biological mediators. The precise pathways linking sitting and cardio-metabolic disease are unclear. It has been suggested that metabolic mechanisms might partly explain these links (15), and data from animals have demonstrated that prolonged sitting might disturb lipid metabolism. There is evidence for a dramatic reduction of lipoprotein lipase activity (by 80% to 90%) during sitting compared with standing up or ambulating (28). Lipoprotein lipase is a key enzyme for the catabolism of triglyceride-rich lipoproteins in the endothelium, and its reduced activity might raise the possibility of other metabolic actions being impaired (15). Our study provides novel findings to suggest a role of metabolic and inflammatory pathways in partly explaining the association between sitting and CVD risk. A well-established marker of low-grade inflammation, CRP, was approximately 3-fold higher in participants spending more than 4 h/day in screen time and explained a substantial amount of the screen time–CVD association. Because screen time was assessed at the same time point as the risk markers, we cannot establish the nature of the temporal relationship between these factors. Nevertheless, our results are in concordance with another study of ours with clear temporal element, which found that TV viewing at age 23 years was independently associated with composite factors of metabolic (including HDL and BMI) and hemostatic/inflammatory (including CRP) but not with cholesterol (total or LDL) biomarkers at 42 years (29) (Stamatakis et al., unpublished observations, January to February, 2010). Both sets of our results are partly corroborated by experimental data in humans. The induction of 5 days bed rest, which represents an extreme form of sedentary behavior, had profound effects on various metabolic risk (including insulin resistance and vascular dysfunction) but not on inflammation (30). Thus, low-grade inflammation might only result from chronic exposure to sedentary lifestyle. A further important mechanism might be related to a decreased expression of endothelial nitric oxide synthase that is caused from reduced local shear stress as a result of lower blood flow from excessive sitting. Further experimental studies will be required to determine the exact mechanisms accounting for increased CVD risk during prolonged inactivity in humans.

Strengths. The main strengths of this study are the detailed measures we were able to take to minimize reverse causality, the many potential confounders we included in our models, and the objectively confirmed CVD events. Other strengths include the nationally representative sample that is expected to have adequate variability in terms of primary and secondary exposures, and therefore it is appropriate to examine the relationships of interest.

Study limitations. Screen time was self-reported. The TV and computer use questions have been shown to underestimate sedentary time when compared with accelerometry (31). Although we have no information on the reliability and criterion validity of the SHS03 screen time questions, we observed the expected associations of screen time with various sociodemographic variables, which provides convergent validity evidence of the screen time data. It is also encouraging that a recent review (7) concluded that TV and computer use time questions have the strongest reliability and validity among sitting-related questions. Although screen time is a partial indicator of overall sitting, TV and computer use account for the overwhelming proportion of leisure time sitting among British adults (32). Also, screen entertainment time tends to be associated with excess calorie consumption, but we were unable to account for dietary intake, although our results were independent of BMI. Finally, our explanatory analyses were limited by the small sample size available, due to limited compliance with blood measurements.

Conclusions

We found a deleterious relationship between recreational sitting and all-cause mortality and cardiovascular events. Our analyses suggest the relationship is independent of physical activity, although further studies that employ objective measures of activity and sedentary time are required to confirm this. We also provide evidence to suggest a role of metabolic and inflammatory pathways in partly explaining the association between sitting and CVD risk. Further experimental studies will be required to determine the exact mechanisms. Our results support the inclusion of a sedentary behavior guideline in public health recommendations for CVD prevention.

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Key Words: mortality ■ physical activity ■ sedentary behavior.

APPENDIX

For a supplementary table, please see the online version of this article.