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Stationary screen time, blood lipids, and atherosclerotic cardiovascular disease incidence

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Abstract

Background and aims The associations between screen time exposure, blood lipids, and Atherosclerotic Cardiovascular Disease (ASCVD) incidence have been less studied. We aimed to examine the associations of exposure to screen time with blood total cholesterol (TC), HDL-C, LDL-C, triglycerides (TG), apolipoprotein A1 (ApoA1), apolipoprotein B (ApoB), and ASCVD risk score, and risk of subsequent ASCVD incidence.

Methods and results A nationwide sample of 7124 China Health and Nutrition Survey 2009 participants were followed up to 2015 for ASCVD incidence. The stationary screen time exposure was assessed through self-reported daily hours of using television, and computers. A total of 292 ASCVD events occurred during 35,310 follow-up person-years. Per 1-h increases in daily screen time exposure were associated with a higher 0.34% (0.12% to 0.56%), 0.47% (0.09% to 0.86%), and 0.51% (0.19% to 0.83%) increases in blood TC, LDL-C, and ApoB levels. A higher risk of incident ASCVD was associated with per log-transformed unit increase in blood LDL-C (adjusted HR = 1.51, 95% CI 1.04 to 2.18), and ApoB (adjusted HR = 1.80, 95% CI 1.12 to 2.92). The elevated blood TC, blood LDL-C, blood ApoA1 and ApoB levels significantly mediated the association between screen time exposure and ASCVD incidence. Urban dwellers, middle-aged adults, and females were particularly associated with a higher ASCVD risk with screen time exposure.

Conclusions The results of this nationwide cohort supported the associations of screen time exposure with elevated blood LDL-C, and ApoB levels, which consistently contributed to an increased risk of subsequent ASCVD incidence.

Keywords ASCVD, Screen time, LDL-C, ApoB

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Introduction

Atherosclerotic Cardiovascular Diseases (ASCVD), which is mainly caused by an unexpected build-up of low-density lipoprotein cholesterol in the arteries, is a leading cause of all cardiovascular disease deaths worldwide [1]. The global disease burden report (2019) showed that the prevalent cases of ASCVD reached 523 million in 2019, causing 18.6 million deaths worldwide [2]. Although the incidence and mortality of ASCVDs have decreased in high-income countries since the mid-twentieth century, many middle-income or low-income countries, such as Eastern European and Asian countries, still report gradually increasing trends of incident ASCVD [3]. In China, hospitalizations for ST-segment-elevation myocardial infarction (MI) increased rapidly from 3.5 per 100,000 population in 2001 to 15.4 per 100,000 population in 2011 [4]. Therefore, it is crucial to examine the risk factors related to ASCVD to reduce the associated disease burden.

People are increasingly exposed to screen time, which mainly includes the daily use of computers, smartphones, and televisions for entertainment and work. A recent report indicated that the average American spends approximately 7 h per day on screens, which is slightly more than the global average of about 6 h and 40 min [5]. Existing evidence has documented various adverse health effects associated with excessive exposure to screen time, including elevated disease mortality, mental and cognitive impairment in children and adults [6–8]. However, the evidence on the associations of screen time exposure with ASCVD is still limited. Several studies have reported the associations of screen time exposure with ASCVD biomarkers, such as fasting triglycerides, HDL cholesterol, and LDL cholesterol, yet with inconsistent findings [9–13]. As far as we know, only two prospective studies have examined the associations of screen time exposure with the incidence of ASCVD, both of which support the associations [6, 14].

Early evidence reported the important role of lipids metabolism dysfunctions in triggering inflammatory reactions, and causally contributing to the pathological progression of atherosclerosis [15]. Mechanistic evidence further confirmed the casual roles of elevated LDL-C in the pathological progression of CVD by causing endothelial dysfunction, activating the NLRP3 inflammasome in macrophages, inducing the generation of pro-inflammatory cytokines, reactive oxygen species, or triggering arterial thrombosis [15]. The population evidence has documented direct associations of elevated blood levels of total cholesterol (TC) and low-density lipoprotein (LDL)-cholesterol (LDL-C), and low levels of blood high-density lipoprotein (HDL)-cholesterol (HDL-C), with increasing subsequent cardiovascular events [16, 17]. However, evidence reported in recent decades, has revealed that the cytokine production and inflammatory reactions caused by the abnormal accumulation of apolipoprotein B (ApoB) lipoproteins, a main apolipoprotein constituent of LDL-C, within the arterial wall is an initial driver underlying the association between abnormal lipids metabolism and CVD progression [18]. Similarly, a large international observational study indicated that the ApoB/ Apolipoprotein A1(ApoA1) ratio was the strongest predictor, of CVD risk compared with other conventional lipids' biomarkers, such as LDL-C, and HDL-C [19]. A subsequent study also reported a dose–response association between blood ApoB levels and the risk of incident ASCVD, even after adjustment for blood levels of HDL cholesterol levels and reducing lipids medication uses [20]. Therefore, examining the associations of screen time exposure with blood ApoB levels may document an important pathway of screen time exposure associated with ASCVD events. However, to our knowledge, only one study examined the association of screen time exposure with blood APOB levels and reported null associations [21]. Importantly, it is still unknown whether excessive screen time exposure could increase the subsequent risk of incident ASCVD through elevated blood APOB levels.

In this study, based on a nationwide cohort, we firstly aimed to examine the associations of baseline screen time exposure with blood lipids biomarkers such as total cholesterol, HDL-C, LDL-C, triglycerides (TG), ApoA1, ApoB, and ASCVD risk score. Secondly, we aimed to examine the mediating effects of screen time induced-changes in blood lipids biomarkers on the association between screen time exposure and subsequent ASCVD incidence.

Methods

Population and study design

The study participants were from a nationwide ongoing study of the China Health and Nutrition Survey (CHNS). Details of the CHNS have been introduced elsewhere [22–24]. In brief, the CHNS is a large-scale longitudinal study that began in 1989 and aims to examine socioeconomic and behavioral changes, diet and physical activities at the household and individual level with health outcomes. Initially, the CHNS took a multistage random cluster design based on eight provinces (Heilongjiang, Liaoning, Shandong, Henan, Jiangsu, Hubei, Hunan, Guizhou and Guangxi) across China to select a stratified probability sample. In each province, two cities (usually the provincial capital and a lower-income city) and four counties stratified by income (usually one high-income, two middle-income, and one low-income country) were randomly selected. Then, two urban and two suburban

communities from each city, and one community and three rural villages from each county were randomly selected. Finally, 20 households in each community or village were randomly selected and all household members were interviewed [22]. The follow-up studies were conducted every 2 to 4 years [25]. The study protocol was reviewed and approved by the Ethics Committee of the Chinese Center for Disease Control and Prevention. Each participant provided a written informant consent at the time of the investigation.

In this study, we included the participants who participated in the CHNS in 2009, provided information on blood lipids data, and provided at least one-time follow-up records between waves 2011 and 2015. Initially, the 7488 participants satisfied the defined criteria. We excluded the participants who were diagnosed with ASCVD diseases (heart diseases, myocardial infarction, or stroke) at baseline (N = 70), missed the information on screen time measurements (N = 17), missed the information for covariates (N = 239), or were under 16 years of age(N=38). Therefore, 7124 participants were included in the analysis. Details of the procedures for including the study participants are shown **i**n Supplementary Figure S1.

Evaluation of screen time exposures

The information on daily screen exposure during leisure time was collected through face-to-face questionnaire interviews. In this study, we included the following screen activities: watching television, watching videotapes/VCDs/ DVDs, watching movies and videos online, playing television video games, surfing the Internet, chatting via the Internet, and playing computer games. Daily exposures to screen devices (hours) on workdays (Monday to Friday) and weekend days were separately collected. We calculated the average hours of screen exposure between workdays and weekend days to indicate long-term screen exposure.

Measurements for blood lipids biomarkers and ASCVD incidence

Blood lipids including total cholesterol (TC), low-density lipoproteins cholesterol (LDL-C), high-density lipoproteins cholesterol (HDL-C), and triglycerides (TG) were measured using standardized methods. Beyond the traditional blood lipids biomarkers, the 2009 wave of CHNS additionally examined a primary protein of LDL-C (Apolipoprotein B, ApoB) and a primary protein of HDL-C (Apolipoprotein A1, ApoA1), which have increasingly been documented as more efficient biomarkers for predicting subsequent risks of incident cardiovascular diseases. We also calculated the 10-year ASCVD risk score for the participants, who were aged from 20 to 80 years, to evaluate the overall risk of incident cardiovascular diseases, according to the guideline of American College of Cardiovascular guidelines (ASCVD Risk Estimator + (acc.org)).

In the prospective cohort analyses, we defined the ASCVD incident cases by self-reported medical diagnoses or hospitalization records collected from the follow-up periods. According to the data availability from CHNS, we included the diseases of stroke, heart disease, and myocardial infarction (MI) as ASCVD phenotypes. The follow-up periods for each participant were clarity the duration between the date of interviewing date of the 2009 wave and the date of being diagnosed with ASCVD diseases or the end date of the study (2015).

Covariates

We firstly collected the demographic variables and socioeconomic status including age (quartiles, years), gender (male, female), ethnicity (Han, ethnic minority group), marital status (unmarried, married, divorced or widowed or separated), education (illiterate, primary to middle school, high school or above), residence areas (rural, urban), and annual household income (quintiles, yuan). We included smoking status (never smoker, ever smoker, current smoker), and alcohol consumption (never or rarely, monthly, weekly, daily). We calculated the daily outdoor physical activity duration by averaging the duration of all outdoor activities, including both high-intensity and moderate-intensity activities. The total duration was then categorized into the following groups: 0 min, 1–60 min, and greater than 60 min. The dietary consumption levels of fat were estimated through a 24-h diet recall survey on three consecutive days.

Statistical analyses

We summarized the baseline distributions of demographic, socioeconomic, and behavioral differences by different daily screen time exposure levels (0–1.4, 1.5–2, 2–3, > 3 h) using counts and proportions. The results of the significant tests for the baseline characteristics across screen time exposures were obtained through Chi-square tests, Fisher exact tests for categorical variables, and variance analyses for continuous variables. The associations of per 60-min incremental change of daily screen time exposure with blood lipids-related biomarkers and ASCVD risk score were estimated by linear mixed-effect models with a random effect at the provincial level to allow for the unmeasured confounders. To approximate the normal distribution, we log-transformed the blood lipids biomarker and ASCVD risk scores, and the results of regression analyses were transformed into percentage

difference by using the transformation method of " (exp (β)−1) *100". In prospective cohort analyses, we conducted Cox-Proportional hazards models to examine the associations of baseline screen time exposureand baseline blood lipids biomarkers with the risk of subsequent incident ASCVD diseases. The "proportional hazards" were examined using simulation-based Supremum Tests. Finally, we used High Dimensional Mediation Analysis methods to estimate the indirect associations of baseline exposure to screen time with risks of subsequent ASCVD incidence through increased blood lipids [26].

We determined the confounders in multivariable adjusted analyses using the Directed Acyclic Graphs (DAG) methods, and the fully DAG-defined confounders are reported in Supplementary Material Figure S2. We first conducted minimally adjusted analyses to allow for the confounding from age. Then, we conducted multivariable adjusted analyses, which included all DAG defined confounders. In the analyses of screen time with ASCVD incidence, we further included mutually adjusted analyses to allow for the confounding from other lipids biomarkers. To avoid multi-collinearity, we calculated the paired correlation coefficients for all lipids biomarkers and included the blood lipids biomarkers with a paired correlation coefficient less than 0.6 in the mutually adjusted analyses (Supplementary Table S1).

We further examined variations across the subpopulations of residential areas, age, and sex using stratified analyses. To evaluate the overall significance of these variations, we calculated values of "*P* for interaction" through log-likelihood ratio tests comparing the model with screen time × stratified variable (residential areas, age, and sex) interaction to the model without that interaction. We conducted several sensitivity analyses to test the robustness of the study associations by additionally adjusting for the baseline diseases histories of hypertension and diabetes, or additionally adjusting for total dietary fat consumption levels.

All regression methods analyses were performed by SAS 9.4, The HIMA analyses were conducted through R program 4.0, and the significance level was determined at 0.05 with a two-sided test.

Results

Of the 7124 participants with a total follow-up of 35,310 person-years, 292 individuals developed into ASCVD (Supplementary Figure S3). The baseline demographic, socioeconomic, and behavioral differences among the study participants across different strata of screen time exposure are summarized in Table 1. The high levels of screen time exposure were reported in the participants who were younger, male, unmarried, better educated, from urban areas, never-smokers, and/or had a higher alcohol consumption level.

The results of multivariable regression analyses indicated that exposure to higher levels of daily screen time was associated with higher risks of blood lipids damages (Table 2). Per 1-h increase in daily screen time exposure was associated with 0.34% (95% CI 0.12% to 0.56%) higher increase in blood TC levels, a 0.47% (95% CI 0.09% to 0.86%) higher increase in blood LDL-C levels, and a 0.51% (95% CI 0.19% to 0.83%) higher increase in blood ApoB levels. No significant associations of screen time exposure with other blood lipids biomarkers were observed. The results of screen time exposure and baseline blood lipids biomarkers with subsequent risk of incident ASCVD were reported in Table 3. We did not observe significant direct associations of screen time exposures with subsequent risks of incidence ASCVD (adjusted HR = 1.04, 95% CI: 0.97 to 1.10). The results of minimum-adjusted analyses indicated that the blood TC, TG, HDL-C, ApoB, and APoA1 were significantly associated with risks of subsequent of incident ASCVD. The multivariable-adjusted analyses and mutually adjusted analyses still supported the significant associations of higher levels of blood LDL-C (adjusted HR = 1.51, 95% CI: 1.04 to 2.18), and ApoB (adjusted HR = 1.80, 95% CI: 1.12 to 2.92) with elevated risks of subsequent ASCVD incidence. No evidence indicated the violation of proportional hazards for Cox regression analyses of screen time and lipids with ASCVD incidence (Supplementary Table S2). The further HIMA analyses identified blood LDL-C, and ApoB as the potential mediators of the association between screen time exposure and blood lipids (Table 4). The HIMA analyses revealed a significant pathway of screen time exposure related increases in blood LDL-C levels, which was intermediately contributed to increases in the subsequent risks of incident ASCVD (P for mediation = 0.032). While, more robust evidence was observed in examining the mediation role of blood ApoB on the association between screen time exposure and subsequent ASCVD incidence (P for mediation < 0.001). Both evidences consistently highlighted an important pathway of "screen time exposure-elevated blood LDL-C/ApoB" in predicting subsequent ASCVD incidence.

The results of stratified analyses revealed that the associations of longer screen exposure time with worse ASCVD outcomes were more pronounced in the participants who resided in urban areas, were middle-aged adults, or were females (Fig. 1and Supplementary Table S3-S5). For example, in subgroup analyses of females, per 1-h increase of daily screen time exposure was associated with a 0.50% (95% CI 0.11% to 0.88%) higher increase in blood APOB level. However, these

Table 1 The study participants' baseline demographic and socioeconomic characteristics by screen time exposures

Covariates	Screen time, hours				*P*-value
	Quartile 1 (0–1.4)	Quartile 2: 1.5–2	Quartile 3: 2–3	Quartile 4: > 3	
Age (years), n(%)					< 0.001
15–40	327 (18.3)	451 (21.3)	336 (22.9)	565 (32.3)	
41–50	399 (22.3)	510 (24.1)	375 (25.6)	408 (23.3)	
51–60	461 (25.8)	612 (28.9)	381 (26.0)	420 (24.0)	
> 60	601 (33.6)	547 (25.8)	373 (25.5)	358 (20.4)	
Gender, n(%)					< 0.001
Male	765 (42.8)	951 (44.9)	689 (47.0)	902 (51.5)	
Female	1023 (57.2)	1169 (55.1)	776 (53.0)	849 (48.5)	
Marital status, n(%)					< 0.001
Unmarried	38 (2.1)	45 (2.1)	41 (2.8)	172 (9.8)	
Married/lived with partner	1513 (84.6)	1917 (90.4)	1312 (89.6)	1445 (82.5)	
Divorce/widowhood/separation	237 (13.3)	158 (7.5)	112 (7.6)	134 (7.7)	
Education, n(%)					< 0.001
Illiterate	624 (34.9)	486 (22.9)	326 (22.3)	261 (14.9)	
Primary school	398 (22.3)	519 (24.5)	296 (20.2)	254 (14.5)	
Low middle school	541 (30.3)	759 (35.8)	505 (34.5)	547 (31.2)	
Upper middle school to university	225 (12.6)	356 (16.8)	338 (23.1)	689 (39.3)	
Residence, n(%)					< 0.001
Urban areas	466 (26.1)	548 (25.8)	452 (30.9)	754 (43.1)	
Rural areas	1322 (73.9)	1572 (74.2)	1013 (69.1)	997 (56.9)	
BMI, n(%)					0.214
Underweight: < 18.5	123 (6.9)	130 (6.1)	68 (4.6)	105 (6.0)	
Normal:18.5–23.9	963 (53.9)	1103 (52.0)	781 (53.3)	906 (51.7)	
Overweight:24–27.9	535 (29.9)	690 (32.5)	463 (31.6)	569 (32.5)	
Obesity: > = 28	167 (9.3)	197 (9.3)	153 (10.4)	171 (9.8)	
Smoking status, n(%)					0.001
Never	1265 (70.7)	1499 (70.7)	1010 (68.9)	1143 (65.3)	
Ever or now	523 (29.3)	621 (29.3)	455 (31.1)	608 (34.7)	
Alcohol consumption, n(%)					< 0.001
Never	1282 (71.7)	1467 (69.2)	995 (67.9)	1078 (61.6)	
1–2 times a Month	150 (8.4)	218 (10.3)	151 (10.3)	258 (14.7)	
1–4 times a Week	169 (9.4)	227 (10.7)	176 (12.0)	263 (15.0)	
Almost every day	187 (10.5)	208 (9.8)	143 (9.8)	152 (8.7)	
Daily outdoor physical activity duration, n(%)					< 0.001
Never	1682 (94.1)	1975 (93.2)	1332 (90.9)	1491 (85.2)	
0–30	71 (4.0)	93 (4.4)	92 (6.3)	178 (10.2)	
> 30	35 (2.0)	52 (2.5)	41 (2.8)	82 (4.7)	

associations were not observed in the female participants. The results of sensitivity analyses indicated that the associations of screen exposure time with blood lipids biomarkers persisted after additional adjustment for dietary fat consumption level, or excluding participants diagnosed with hypertension or diabetes (Fig. 2and Supplementary Table S6).

Discussion

We examined the associations of screen time exposure with blood ASCVD outcomes and subsequent incidence of ASCVD based on a nationwide Chinese prospective cohort. The results indicated consistent patterns of longer exposure to daily screen time with worse blood lipids. Importantly, we highlighted an important lipid pathway of elevated "blood LDL-C/ApoB" levels in the

Table 2 The cross-sectional associations of screen time exposure with blood lipids biomarkers in the study participants

Blood lipids biomarker[a]	Estimated % difference (95% CI)[b]	
	Adjusted for age	Multivariable adjusted
TC	0.38 (0.17, 0.60)*	0.34 (0.12, 0.56)*
TG	1.12 (0.43, 1.82)*	0.60 (−0.07, 1.29)
LDL-C	0.54 (0.17, 0.92)*	0.47 (0.09, 0.86)*
HDL-C	−0.59 (−0.88, −0.29)*	−0.26 (−0.55, 0.03)
ApoA1	−0.37 (−0.63, −0.11)*	−0.23 (−0.49, 0.04)
ApoB	0.66 (0.34, 0.97)*	0.51 (0.19, 0.83)*
ASCVD risk[b]	2.29 (1.43, 3.16)*	0.38 (−0.24, 0.99)

* $P < 0.05$

[a] The blood lipids biomarkers were log-10 transformed to approximate normal distributions

[b] Only calculated for the participants aged from 20 to 80 years old

association between screen time exposure and subsequent ASCVD incidence. The associations persisted after adjustment for various confounders. We finally documented that the participants who resided in urban areas, were middle-aged adults, or females were particularly at higher risks with ASCVD outcomes with exposure to screen time.This study highlights that increased screen time is associated with elevated blood lipids, particularly ApoB, in high-risk individuals. Reducing ApoB levels may help lower cardiovascular risk. These findings suggest that limiting screen time and managing lipid levels could be key strategies in preventing cardiovascular disease, especially in high-risk populations.

Our findings of longer screen time exposure with worse blood lipids are consistent with existing evidence. The early evidence indicated the longer night-time TV viewing durations are associated with lower levels of blood HDL-C and higher levels of blood total cholesterol/HDL-C ratio [27]. Subsequent population studies with larger sample sizes have consecutively documented the associations of longer exposure to TV screen viewing or video games with worse levels of blood triglycerides, LDL-C, and total cholesterol in adolescents or young adults [11, 28, 29]. In this study, we consistently found that the associations of longer daily screen time exposures, which include TV viewing, video games, computer use for social contact and playing games with worse levels of blood LDL-C levels. Additionally, we reported the associations of longer exposure to daily screen time with higher blood ApoB levels. Finally, the analyses of prospective cohort provided consistent evidence linking higher levels of blood LDL-C, and ApoB levels in predicting subsequent risks of ASCVD incidence after adjusting for other lipids biomarkers.

We provided initial evidence which highlighted the mediating roles of elevated ApoB-containing lipoproteins levels underlying the association between screen time exposure and ASCVD incidence. We documented these odds from

Table 4 The results of HIMA analyses for blood lipids on the association between exposure to screen time and risks of incident ASCVD in the study participants[a]

Blood lipids biomarkers[b]	$\hat{\alpha}$ (SE)	$\hat{\beta}$ (SE)	P for mediation
LDL-C	0.004 (0.002)*	0.498 (0.175)*	0.032
ApoB	0.006 (0.002)*	0.773 (0.190)*	< 0.001

HIMA High Dimensional Mediation Analyses

[a] The 14 participants were excluded out of the analyses because of missing information for blood LDL-C, or ApoB data

[b] The blood lipids biomarkers were log-10 transformed to approximate normal distribution

α Coefficient estimates of screen time (X) –> lipids (M) (adjusted for covariates)

β Coefficient estimates of lipids (M) –> ASCVD (Y) (adjusted for covariates and exposure)

* $P < 0.05$

Table 3 The associations of baseline blood lipids biomarkers with subsequent risk of incident CVD outcomes[a]

Screen time/Blood lipids biomarkers	Adjusted HRs (95% CI)		
	Adjusted for age	Multivariable-adjusted[b]	Mutually adjusted[c]
Screen time	1.04 (0.98, 1.10)	1.04 (0.97, 1.10)	-
TC	2.16 (1.24, 3.77)*	1.7 (0.95, 3.06)	1.66 (0.87, 3.16)
TG	1.39 (1.15, 1.67)*	1.16 (0.95, 1.42)	1.08 (0.80, 1.45)
LDL-C	1.72 (1.19, 2.48)*	1.48 (1.02, 2.15)*	1.51 (1.04, 2.18)*
HDL-C	0.58 (0.38, 0.89)*	0.86 (0.55, 1.35)	1.42 (0.78, 2.57)
ApoA1	0.58 (0.38, 0.89)*	0.75 (0.49, 1.16)	0.61 (0.37, 0.98)
ApoB	2.39 (1.57, 3.63)*	1.78 (1.15, 2.75)*	1.80 (1.12, 2.92)*

* $P < 0.05$

[a] The blood lipids biomarkers were log-10 transformed to approximate normal distributions

[b] Adjusted for residence,age,gender,marital status,education,BMI,alcohol consumption,smoking status and daily outdoor psychical.activity duration

[c] Additionally adjusted for other blood lipids biomarkers

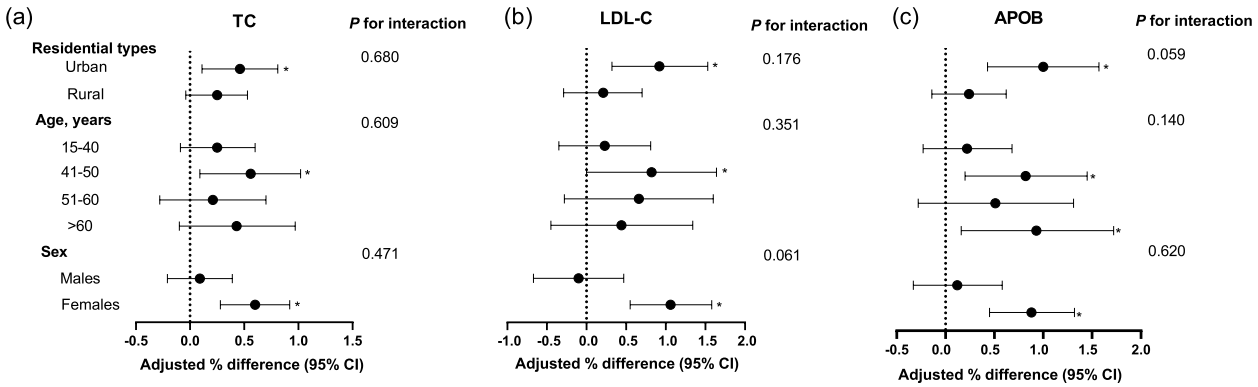

Fig. 1 The results of screen time exposure with blood lipids stratified by: (**a**) residence, (**b**) age, and (**c**) sex

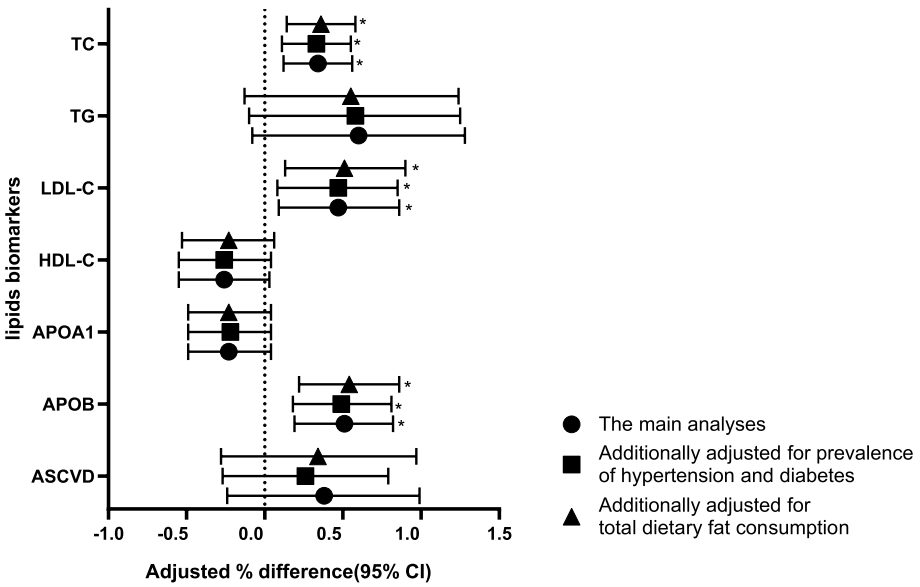

Fig. 2 The results of sensitivity analyses for the associations of screen time exposure with blood lipids

two aspects. Firstly, we documented a significant evidence that elevated blood LDL-C levels mediated the association between screen time exposure and higher subsequent ASCVD incidence. The evidence was consistent with the existing evidence which observed the strong positive associations of blood LDL-C levels with ASCVD incidence [30, 31]. Secondly, we obtained more robust evidence of analyzing the mediation role of blood ApoB levels on the association between screen time exposure and ASCVD incidence. These variations can be explained by several mechanisms. Screen time often leads to prolonged sedentary behavior, which is associated with decreased energy expenditure, reduced lipoprotein lipase activity, and impaired triglyceride metabolism. This can result in elevated levels of triglycerides, LDL-C, and ApoB [32]. Sedentary behavior associated with excessive screen time can induce systemic inflammation, leading to up regulation of pro-inflammatory cytokines such as IL-6 and TNF-α. This inflammation has been linked to deregulated lipid metabolism and increased levels of ApoB-containing lipoproteins [33] Compared with LDL-C, the ApoB has a direct role on assembling the cholesterol and triglycerides into chylomicrons, entering the circulation trough intestinal lymphatic system, contributed to the formation of very-low-density lipoproteins (VLDL) [18]. While the VLDL entering the arterial wall, and causing the oxidation were direct mechanisms related to pathological occurs of atherogenesis [18, 34]. In addition, the measurement of ApoB provides more information about particle types, including LDL-C and VLDL-C, than the measurement of LDL-C alone [35].

We identified several susceptible subgroups with exposure to screen time. The urban residents exhibited worse blood lipids with screen time exposure. These urban–rural differences can be explained by variations of individual-level outdoor physical activity and sedentary activity duration, as well as dietary habits [36]. Compared with rural residents, the participants from the urban areas reported less daily duration in outdoor activity, and more time in sedentary occupational activity [36, 37]. Additionally, compared with rural residents, the urban dwellers were more likely to adopt a sodium, sugar, and saturated fat-rich diet [36]. Both are well-documented factors to be strongly associated with longer screen time exposure and worse blood lipids outcomes [38, 39]. The associations of exposure to screen time with worse blood lipids were more pronounced in middle-aged adults. Several potential reasons can be used to explain these characteristics. First, the middle-aged adults were at higher risk of exposure to daily screen time. A nationwide study, which involved 40,337 US participants aged from 2 to 17 years, reported the duration of daily screen time was gradually increased from a mean daily hour of 2.28 (SD 1.72) in children to a mean hour of 4.59 (SD 2.50) in adolescents [40]. We reported a similar trend for age-specific screen time exposure, which indicated relatively higher exposure in the participants aged 35 to 47 years. While the lowest levels of exposure were observed in participants aged above 60 years. Second, the hypercholesteremia prevalence dramatically increased from young adulthood to middle-aged adulthood, peaked from 45 to 54 years of age, and persistently declined after the age of 65 or more years [41]. These differences resulted in the middle-aged adults to be the most susceptible population underlying the screen time exposure. Females with exposure to higher levels of daily screen time had worse blood lipids. Compared with males, females had higher risks of incident lipids abnormalities because they were more likely to experience a sharp escalation with biological aging and menopause-related endocrine changes [42]. The population evidence also documented a pronounced association of screen time exposure with depression incidence in females [43].

Strengths and limitations

Our study has strengths. The analyses were based on a nationwide Chinese cohort covering 7000 participants, which provided enough power to detect the difference in the study associations. The screen time exposures were separately measured by watching TV, playing video games, searching interment, and playing computer games which included wide phenotypes. The screen time duration was calculated by average exposures between workdays and weekends, which represents more accurate individual-level exposure. We simultaneously included traditional blood lipids and lipoproteins biomarkers, and extensively examined the associations of screen time exposure with blood lipids and lipoproteins biomarkers, and subsequent risks of ASCVD incidence.

Several limitations in this study should be mentioned. Firstly, the information on screen time exposure was collected in 2009, which might have some difference with the recent exposures. At that time, television was the predominant screen type, whereas today, mobile phones and other digital devices have become more common forms of screen exposure. This shift in screen type may lead to differences in screen time behaviors and their associated health impacts, limiting the generalizability of the findings to current patterns of screen use. Further studies should replicate our study with more up-to-date data. Secondly, this study included a limited sample size of children, which limited the generalization of the study conclusions.

Conclusions

In this cohort study, exposure to longer daily screen duration was associated with higher blood LDL-C and ApoB levels, and intermediately associated with elevated ASCVD incidence in the subsequent 6-years. These results initially highlight ApoB-containing lipoproteins is a potential pathway linking screen time exposure to ASCVD incidence.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12872-025-04568-0.

Additional file 1.

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Authors' contributions

Prof. Jiaqiang Liao had full access to all of the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. Jiaqiang Liao designed the study and supervised the work. Yunjie Liu, Lanjin Xu, and Panting Liu conducted equally to the data analyses and drafted the manuscript. Xueyan Liao participated interpretation of data and statistical analyses. All authors participated the critical revision of the manuscript for important intellectual content.

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Data availability

All data generated or analysed during this study are included in this published article [and its supplementary information files].

Declarations

Ethics approval and consent to participate
The study protocol was reviewed and approved by the Ethic Committee of the Chinese Center for Disease Control and Prevention. Each participant provided a written informant consent at the time of investigation.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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