



Computer use and cardiovascular risk biomarkers in midlife and older adults

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ABSTRACT

With increased computer usage amongst midlife and older adults, concerns are emerging with regards to the potential adverse health effects of computer use given the sedentary habits it may encourage. The current study aims to investigate the relationship between computer use and cardiovascular risk in midlife and older adults. From the National Survey of Midlife Development in the United States II: Biomarker Project (2004–2009) and the National Survey of Midlife Development in the United States (MIDUS II), 2004–2006, we examined five cardiovascular risk biomarkers—high-density lipoprotein (HDL) cholesterol, low-density lipoprotein (LDL) cholesterol, triglycerides, interleukin-6, and C-reactive protein—in relation to self-reported general computer use frequency and computer use at work frequency. Our results show that general computer use frequency and computer use at work frequency were not significant predictors of any of the five cardiovascular risk biomarkers—HDL cholesterol, LDL cholesterol, triglycerides, interleukin-6, and C-reactive protein. However, our exploratory analysis showed that employment status significantly moderated the relationship between general computer use frequency and LDL cholesterol. Our study highlights the importance of a more nuanced approach to understanding the health implications of computer use and sedentary behaviour in general.

1. Introduction

Digital technology, especially the regular use of computers, has profoundly shaped all facets of modern life, revolutionising the way we work, communicate, and access information (Gell et al., 2015; Graham & Dutton, 2019; Keegan, 2012; Srinivasan, 2018; Tully, 2003). While often associated with younger generations, research indicates that midlife and older adults have also begun to adopt computer use for its utility in work, communication, and leisure activities (Carpenter & Buday, 2007; Nimrod, 2020; Wagner et al., 2010). With increased computer usage, growing concerns are emerging with regards to the potential adverse health effects of widespread computer use in midlife and older adults, especially given the sedentary habits it may encourage (Fotheringham et al., 2000; Gatto & Tak, 2008; Harvey et al., 2013). This focus is particularly relevant for older adults, who often face more susceptibility to health-related issues, including cardiovascular diseases (Lakatta, 2002; Mittelmarm et al., 1993; North & Sinclair, 2012).

Despite the concern regarding the adverse health implications of computer use in midlife and older adults, research findings on the

relationship between computer use and health outcomes remain inconclusive. On one hand, several existing studies have shown that computer use is negatively associated with health outcomes, such as an increased risk of being overweight or obese (Aghasi et al., 2020; Vandelanotte et al., 2009), reduced levels of physical activity (Fotheringham et al., 2000), potential sleep problems when used during leisure time (Andersen & Garde, 2015), and the likelihood of experiencing eye strain (Basnet et al., 2022). On the other hand, recent studies have also reported that computer use enables individuals to engage in health-promoting activities (Stephenson et al., 2017), experience improved cognitive functioning (Almeida et al., 2012; Kamin & Lang, 2020) and foster greater social connections that improve overall well-being (Fingerman et al., 2020; Petersen et al., 2023). Interestingly, one study found that while computer users reported higher subjective health through self-reported measures, there were no significant links between computer use and physical activity or objective health outcomes, such as body mass index and number of chronic diseases (Hartanto et al., 2020).

However, it is noteworthy that most of the existing studies focused

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on subjective health outcomes, which may contribute to the mixed findings (e.g., Fotheringham et al., 2000; Slegers et al., 2008). Thus, it is imperative to shift the focus to objective health biomarkers, which offer a more consistent and precise measure of physiological changes in individuals (Molenaar et al., 2007; Okura et al., 2004). This is particularly pertinent to cardiovascular health, the leading causes of mortality globally (Amini et al., 2021), which often manifest silently, with physiological changes developing unnoticed for years before overt symptoms emerge (Greenland et al., 2004; Soliman, 2019).

Given the mixed findings and global prominence of cardiovascular diseases (Lindstrom et al., 2022), the current study aimed to investigate the association between computer use and its health implications with cardiovascular risk biomarkers. To provide a comprehensive assessment, computer use was operationalised as both general computer use frequency and computer use at work frequency. Five well-established biomarkers linked to cardiovascular risk were included in the current study (Gilstrap & Wang, 2012; Hartanto et al., 2022, 2024; Ridker et al., 2000). The first biomarker, high-density lipoprotein (HDL) cholesterol was included due to its protective role in cardiovascular health by facilitating the removal of excess cholesterol (Kosmas et al., 2018; Trimarco et al., 2022). The second biomarker, low-density lipoprotein (LDL) cholesterol – often referred to as “bad” cholesterol due to its role in transporting cholesterol to the arteries – was included, as it promotes blood clot formation and contributes to increased cardiovascular risk (Jung et al., 2022; Pereira, 2017; Stanculescu et al., 2023). The third biomarker, triglycerides – types of fat found in the blood that store excess energy from the diet – were included because elevated levels of triglycerides are a well-known risk factor for coronary heart disease (Assmann et al., 1998; Sarwar et al., 2007). Lastly, we also included interleukin-6, a cytokine that plays a central role in the body’s immune response (Tanaka et al., 2014), and C-reactive protein, a protein produced by the liver in response to inflammation (Mouliou, 2023). Both have shown to be critical in the development and progression of cardiovascular diseases (Amezcuca-Castillo et al., 2023; Feng et al., 2022; Kuppa et al., 2023; Mossmann et al., 2022). By including these specific biomarkers, this study provides a comprehensive evaluation of cardiovascular risk.

We hypothesised positive relationships between computer use and higher cardiovascular risk in midlife and older adults. This is because prolonged computer use often leads to extended periods of physical inactivity, which may contribute to sedentary behavior (Bertuol et al., 2023; Harvey et al., 2013). Sedentary behaviour has been linked to an increased risk of developing cardiovascular diseases (Bakker et al., 2021; Bezerra et al., 2023) as it promotes obesity, hypertension and insulin resistance, all of which are established risk factors for cardiovascular disease (Kim et al., 2018; Zhang et al., 2022).

2. Materials and methods

2.1. Participants

This cross-sectional study involved 1054 adults from the National Survey of Midlife Development in the United States II (MIDUS II): Biomarker Project (Ryff et al., 2010) and the National Survey of Midlife Development in the United States (MIDUS II), 2004–2006 (Ryff et al., 2021). The MIDUS II: Biomarker Project, conducted between 2004 and 2009, is a subset of a broader, long-term research initiative stemming from the original MIDUS I survey launched in 1995. To be eligible for MIDUS II, participants had to be aged 25 to 74 during the original MIDUS survey and have completed the initial MIDUS I interview (Ryff et al., 2021). For MIDUS II: Biomarker Project, participants were required to have completed the MIDUS II Project 1 Survey (Ryff et al., 2010). Within this study, participants aged 35 to 65 were classified as midlife adults, while those aged above 65 years old were categorised as older adults (Hartanto et al., 2020; Infurna et al., 2020; Kang & Kim, 2022).

In MIDUS I, researchers recruited 7108 noninstitutionalized adults through random digit sampling across all 48 contiguous states. In the context of the Biomarker Project, participants underwent an overnight stay at one of three general clinical research centres situated in the United States, which included the University of California, Los Angeles, Georgetown University, and the University of Wisconsin-Madison. A comprehensive physical examination was conducted during participants’ stay, encompassing the collection of fasting blood samples before breakfast on the second day of their hospital visit (Love et al., 2010). Data collection was carried out in strict adherence to approved guidelines and regulations, receiving approval from the Health Sciences Institutional Review Boards at the University of Wisconsin-Madison (H-2008-0060). Prior to their involvement, all participants provided written informed consent. Table 1 provides a detailed breakdown of the sample’s demographics and key variables. Tables 2 and 3 presents the zero-order correlation of the variables examined in this study.

2.2. Measures

2.2.1. General computer use frequency

Participants’ computer use was assessed by: “How often do you use a computer (such as to send e-mail or search the internet)”. Participants rated their frequency of involvement on a scale of 1 (*daily*) to 6 (*never*).

2.2.2. Computer use at work frequency

Participants’ computer use at work frequency was assessed by: “Please indicate how often, during your work-shift, you do each of the following. If you are not currently working, but were employed over the past

Table 1
Participants’ characteristics.

Variable	N	M (SD)	Range
Demographics			
Age	1054	58.04 (11.62)	35.00–86.00
Gender (% male)	1054	0.45 (0.50)	0.00–1.00
Race (% white)	1051	0.94 (0.24)	0.00–1.00
Education Attainment	1051	7.74 (2.45)	1.00–12.00
Income (in thousands)	1032	76.67 (60.41)	0.00–300.00
Employment Status	1052	0.52 (0.50)	0.00–2.00
Health Status and Behaviours			
Alcohol consumption (drinks per month)	1054	2.53 (1.58)	1.00–6.00
Exercise (exercise regularly)	1054	0.79 (0.41)	0.00–1.00
Smoking (current smoker)	1054	0.11 (0.32)	0.00–1.00
Hypertension	1054	0.26 (0.44)	0.00–1.00
Diabetes	1054	0.09 (0.28)	0.00–1.00
Stroke	1054	0.00 (0.07)	0.00–1.00
Antihyperlipidemic Agent Medication	1053	0.31 (0.46)	0.00–1.00
Predictors			
General computer use frequency	1048	4.62 (1.92)	1.00–6.00
Computer use at work frequency	554	3.42 (1.37)	1.00–5.00
Cardiovascular Risk Biomarkers			
HDL cholesterol (mg/dL)	1043	54.58 (17.43)	19.00–107.42
LDL cholesterol (mg/dL)	1043	106.17 (34.62)	16.00–211.77
Triglycerides (mg/mL)	1045	130.92 (78.33)	25.00–554.76
Interleukin-6 (pg/mL)	1044	2.66 (2.17)	0.16–11.15
C-reactive protein (ug/mL)	1040	2.50 (3.00)	0.03–15.55

Note. Values shown are before imputation and winsorization. Education attainment was rated on a scale of 1 (*No school*) to 12 (*PhD, EdD, MD, LLB, LLD, JD, or other professional degree*). HDL = high-density lipoprotein, LDL = low-density lipoprotein. Alcohol consumption was measured based on participants’ frequency of drinking in the past month (1 = *Never drinks*, 6 = *Everyday*). Smoking habits was measured based on whether participants currently smoked regularly (0 = *No*, 1 = *Yes*). Exercise frequency was measured based on whether they engaged in regular exercise, or activity, for at least 20 min, three times per week (0 = *No*, 1 = *Yes*). Only participants who were employed at the time of the study were included in the descriptive analyses related to computer use at work frequency.

Table 2
Correlation matrix of the study variables for the main sample (N = 1054).

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1. General computer use frequency	0.02																		
2. HDL Cholesterol	-0.03	-0.11 ^b																	
3. LDL Cholesterol	-0.07 ^a	-0.50 ^b	0.16 ^b																
4. Triglycerides	-0.13 ^b	-0.14 ^b	-0.07 ^a	0.13 ^b															
5. Interleukin-6	-0.01	-0.11 ^b	0.05	0.10 ^a	0.47 ^b														
6. C-reactive Protein	-0.23 ^b	0.06 ^a	-0.14 ^b	-0.04	0.20 ^b	-0.01													
7. Age	-0.04	-0.41 ^b	-0.03	0.21 ^b	-0.03	-0.15 ^b	0.05												
8. Gender (% Male)	0.05	-0.01	-0.03	0.04	0.01	0.00	0.11 ^b	-0.01											
9. Race (% White)	0.34 ^b	0.03	-0.07 ^a	-0.07 ^a	-0.10 ^a	-0.09 ^a	-0.07 ^a	0.09 ^a	0.04										
10. Education Attainment	0.26 ^b	-0.03	-0.07 ^a	-0.07 ^a	-0.10 ^a	-0.05	-0.22 ^b	0.08 ^a	0.04	0.28 ^b									
11. Income (in thousands)	0.19 ^b	-0.03	0.06 ^a	-0.03	-0.13 ^b	-0.02	-0.42 ^b	0.05	-0.06	0.15 ^b	0.22 ^b								
12. Employment status	0.10 ^a	0.18 ^b	-0.03	-0.01	-0.06	-0.08 ^a	0.07 ^a	0.18 ^b	0.03	0.11 ^b	0.13 ^b	0.02							
13. Alcohol consumption	0.07 ^a	0.11 ^b	0.01	-0.11 ^b	-0.15 ^b	-0.17 ^b	-0.05	-0.01	0.07 ^a	0.09 ^a	0.06	0.04	0.04						
14. Exercise	-0.09 ^a	-0.12 ^b	0.10 ^a	0.05	0.04	0.04	-0.13 ^b	0.01	-0.02	-0.15 ^b	-0.08 ^a	0.00	-0.03	-0.06 ^a					
15. Smoking	-0.11 ^b	-0.10 ^b	-0.10 ^a	0.14 ^b	0.21 ^b	0.13 ^b	0.22 ^b	0.01	0.01	-0.03	-0.06 ^a	-0.12 ^b	0.04	-0.05	-0.03				
16. Hypertension	-0.09 ^a	-0.10 ^b	-0.12 ^b	0.12 ^b	0.11 ^b	0.10 ^a	0.09 ^a	0.05	0.00	-0.03	-0.03	-0.10 ^a	-0.11 ^b	-0.08 ^a	-0.02	0.22 ^b			
17. Diabetes	-0.01	-0.02	-0.05	0.03	0.08 ^a	0.08 ^a	0.05	0.02	0.02	0.02	0.07 ^a	-0.05	-0.01	0.00	0.02	0.09 ^a	0.08 ^a		
18. Stroke	0.00	-0.11 ^b	-0.36 ^b	0.05	0.11 ^b	-0.03	0.30 ^b	0.14 ^b	0.04	-0.01	0.03	-0.12 ^b	0.07 ^a	-0.04	-0.05	0.23 ^b	0.16 ^b	0.04	
19. Antihyperlipidemic Agent Medication																			

Note.

HDL = high-density lipoprotein, LDL = low-density lipoprotein. Education attainment was rated on a scale of 1 (*No school*) to 12 (*PhD, EdD, MD, LLB, LLD, JD, or other professional degree*). Alcohol consumption was measured based on participants' frequency of drinking in the past month (1 = *Never drinks*, 6 = *Everyday*). Smoking habits was measured based on whether participants currently smoked regularly (0 = *No*, 1 = *Yes*). Exercise frequency was measured based on whether they engaged in regular exercise, or activity, for at least 20 min, three times per week (0 = *No*, 1 = *Yes*).

^a $p < .05$.

^b $p < .001$.

10 years, please tell us about your most recent job – How often does your job require you to work on a computer?“. Participants rated their frequency of computer use at work frequency on a scale of 1 (*All of the time*) to 5 (*Never*).

2.2.3. Serum lipid

Enzymatic colorimetric assays were used to determine total cholesterol, HDL cholesterol and triglyceride levels. The inter-assay and intra-assay coefficients of variability for total cholesterol were 2.65% and 0.51–0.81%. while for HDL cholesterol, it is 6.52% and 1.1–1.4%. As for the triglycerides levels, the inter-assay and intra-assay coefficient of variability were 1.01% and 1.6%. Low-density was estimated using the Friedewald formula (Friedewald et al., 1972). Triglycerides levels above 400 mg/dl were replaced with 400 mg/dl to calculate LDL cholesterol levels. Lastly, the inter-assay coefficient of variability for LDL cholesterol was 10.11%. Higher levels of HDL cholesterol indicates lower levels of cardiovascular risk (Kosmas et al., 2018; Trimarco et al., 2022), while higher levels of LDL cholesterol and triglycerides levels indicates higher levels of cardiovascular risk (Aberra et al., 2020; Harchaoui et al., 2009; Jung et al., 2022; Stanciulescu et al., 2023).

2.2.4. C-reactive protein

BNII nephelometer (N Antiserum to Human Fibrinogen; Dade Behring, Inc., Deerfield, IL) was used to measure C-reactive protein with a particle-enhanced immunonephelometric assay range of 0.175–1100 µg/mL (reference range <3 µg/ml). Samples falling below the assay range for CRP by this method were re-assayed by immunoelectrochemiluminescence using a high-sensitivity assay kit (Meso Scale Diagnostics #K151STG). The laboratory intra- and inter-assay coefficients of variance for CRP were in acceptable ranges, with the ranges of the intra-assay variance coefficient between 2.3% and 4.4% and the interassay variance between 2.1% and 5.7%. Higher levels of C-reactive protein indicates higher levels of cardiovascular risk (Amezcuca-Castillo et al., 2023; Kuppa et al., 2023).

2.2.5. Interleukin-6

Interleukin-6 was measured using the Quantikine® High-sensitivity enzyme-linked immunosorbent assay (ELISA) kit #HS600B (R&D Systems, Minneapolis, MN), with an assay range of 0.156–10 pg/mL. All samples were tested in duplicate. The laboratory intra-assay and inter-assay coefficients of variance for interleukin-6 were 3.25% and 12.31%, respectively. The reference range spanned from 0.45 to 9.96 pg/mL. Higher levels of interleukin-6 indicates higher levels of cardiovascular risk (Feng et al., 2022; Mossmann et al., 2022).

2.2.6. Demographics

Age, education, income were mean-centered while gender, race, employment status were dichotomized (gender: 0 = *Female*, 1 = *Male*; race: 0 = *White*, 1 = *Non-white*; employment status: 0 = *Employed*, 1 = *Unemployed*).

2.2.7. Health status and related behaviours

Health status and behaviours of participants was measured through a series of questions. Hypertension, diabetes, stroke history were evaluated and coded by asking participants whether they had experienced or been treated for these conditions in the past twelve months (0 = *No*, 1 = *Yes*). Smoking habits of participants were evaluated and coded by asking participants whether participants currently smoked cigarettes regularly (0 = *No*, 1 = *Yes*). Alcohol consumption was measured and coded based on the participants' frequency of drinking in the past month (1 = *Never drinks*, 6 = *Everyday*). Exercise frequency was measured and coded by asking participants on whether they engaged in regular exercise, or activity, for at least 20 min, three times per week (0 = *No*, 1 = *Yes*). Lastly, participants' use of antihyperlipidemic agent medication was measured and coded based on whether any type of antihyperlipidemic agent medication was used such as HMG-CoA reductase inhibitor, fibric

Table 3
Correlation matrix of the study variables for the employed-only sample (N = 573).

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	
1. Computer use at work frequency																			
2. HDL Cholesterol	-0.02																		
3. LDL Cholesterol	0.00	-0.15 ^b																	
4. Triglycerides	-0.02	-0.51 ^b	0.24 ^b																
5. Interleukin-6	-0.04	-0.13 ^a	-0.08	0.06															
6. C-reactive Protein	0.04	-0.08	0.06	0.06	0.47														
7. Age	-0.12 ^a	0.12 ^a	-0.13 ^a	-0.07	0.13 ^a	-0.04													
8. Gender (% Male)	-0.09 ^b	-0.41 ^a	0.05	0.27	-0.04	-0.22 ^b	-0.03												
9. Race (% White)	-0.05	-0.02	0.01	0.05	-0.02	-0.04	0.09 ^a	-0.02											
10. Education Attainment	0.24 ^b	0.03	-0.05	-0.03	-0.06	-0.09 ^a	-0.06	0.00	0.06										
11. Income (in thousands)	0.23 ^b	-0.06	-0.07	-0.03	-0.04	-0.15 ^b	0.07	0.21 ^b	0.05	0.28 ^b									
12. Alcohol consumption	0.04	0.18 ^b	-0.01	0.01	-0.09 ^a	-0.17 ^b	0.07	0.09 ^a	0.04	0.10 ^a	0.15 ^b								
13. Exercise	0.04	0.10 ^a	0.03	-0.05	-0.18	-0.17 ^b	-0.03	0.01	0.08	0.07	0.07	0.02							
14. Smoking	-0.15 ^b	-0.12 ^a	0.11 ^a	0.07	0.03	0.01	-0.07	0.08	-0.04	-0.11 ^a	-0.08	0.00	-0.08						
15. Hypertension	0.02	-0.06	-0.09 ^a	0.08 ^a	0.14	0.07	0.23 ^b	-0.04	-0.02	-0.03	-0.03	0.02	0.00	-0.07					
16. Diabetes	0.00	-0.07	-0.11 ^a	0.02	0.11 ^a	0.14 ^a	0.06	0.00	-0.01	-0.02	0.01	-0.07	-0.13 ^a	0.02	0.24 ^b				
17. Stroke	0.02	0.06	0.02	0.00	-0.01	0.06	0.05	-0.04	0.01	-0.04	0.01	0.02	0.01	-0.01	0.08	-0.01			
18. Antihyperlipidemic Agent Medication	0.04	0.13 ^a	-0.30 ^b	0.01	0.13 ^a	-0.04	0.34 ^b	0.08 ^a	0.00	-0.01	0.06	0.05	-0.05	-0.05	0.23 ^b	0.18 ^b	-0.02		

Note.
HDL = high-density lipoprotein, LDL = low-density lipoprotein. Education attainment was rated on a scale of 1 (*No school*) to 12 (*PhD, EdD, MD, LLB, LLD, JD, or other professional degree*). Alcohol consumption was measured based on participants' frequency of drinking in the past month (1 = *Never drinks*, 6 = *Everyday*). Smoking habits was measured based on whether participants currently smoked regularly (0 = *No*, 1 = *Yes*). Exercise frequency was measured based on whether they engaged in regular exercise, or activity, for at least 20 min, three times per week (0 = *No*, 1 = *Yes*).
^a $p < .05$.
^b $p < .001$.

Table 4
Standardized regression coefficients of general computer use frequency on cardiovascular risk biomarkers.

Predictor	HDL Cholesterol				LDL Cholesterol				Triglycerides				Interleukin-6				C-reactive Protein			
	Model 1		Model 2		Model 1		Model 2		Model 1		Model 2		Model 1		Model 2		Model 1		Model 2	
	β	<i>b</i> (SE)	β	<i>b</i> (SE)	β	<i>b</i> (SE)	β	<i>b</i> (SE)	β	<i>b</i> (SE)	β	<i>b</i> (SE)	β	<i>b</i> (SE)	β	<i>b</i> (SE)	β	<i>b</i> (SE)	β	<i>b</i> (SE)
General Computer Use Frequency	-0.00	-0.00 (0.28)	-0.03	-0.32 (0.27)	-0.04	-0.67 (0.61)	-0.01	-0.20 (0.58)	-0.04	-1.46 (1.35)	-0.02	-0.73 (1.34)	-0.06	-0.07 (0.04)	-0.04	-0.04 (0.04)	0.01	0.02 (0.05)	0.04	0.06 (0.05)
Covariates																				
Age	0.10	0.16 (0.05) ^a	0.11	0.16 (0.05) ^a	-0.17	-0.49 (0.01) ^b	-0.04	-0.12 (0.10)	-0.10	-0.69 (0.23) ^a	-0.13	-0.90 (0.24) ^b	0.17	0.03 (0.01) ^b	0.13	0.02 (0.01) ^b	-0.01	-0.00 (0.01)	-0.02	-0.01 (0.01)
Gender (% Male)	-0.42	-14.81 (0.99) ^b	-0.45	-15.82 (0.96) ^b	-0.01	-0.70 (2.14)	0.03	2.29 (2.06)	0.22	35.33 (4.77) ^b	0.22	34.73 (4.81) ^b	-0.03	-0.15 (0.13)	-0.04	-0.16 (0.13)	-0.14	-0.82 (0.19) ^b	-0.13	-0.79 (0.19) ^b
Race (% White)	-0.02	-1.55 (2.05)	-0.03	-2.22 (1.94)	-0.00	-0.68 (4.41)	-0.01	-1.05 (4.17)	0.06	18.30 (9.85)	0.06	21.11 (9.70)*	-0.00	-0.04 (0.28)	0.01	0.05 (0.27)	0.00	0.02 (0.38)	0.01	0.18 (0.37)
Education Attainment	0.07	0.53 (0.22) ^a	0.05	0.33 (0.21)	-0.05	-0.65 (0.47)	-0.05	-0.76 (0.45)	-0.06	-1.92 (1.05)	-0.05	-1.67 (1.04)	-0.05	-0.05 (0.03)	-0.04	-0.03 (0.03)	-0.08	-0.10 (0.04) ^a	-0.07	-0.09 (0.04) ^a
Income	0.00	0.00 (0.01)	-0.02	-0.01 (0.01)	-0.09	-0.05 (0.02) ^a	-0.05	-0.03 (0.02)	-0.07	-0.10 (0.04) ^a	-0.07	-0.09 (0.04) ^a	-0.03	-0.00 (0.00)	-0.02	-0.00 (0.00)	-0.02	-0.00 (0.00)	-0.01	-0.00 (0.00)
Employment Status	0.02	0.76 (1.10)	0.01	0.30 (1.04)	0.03	1.74 (2.37)	0.01	1.00 (2.24)	-0.05	-7.63 (5.29)	-0.04	-5.73 (5.22)	-0.03	-0.12 (0.15)	-0.01	-0.06 (0.14)	-0.01	-0.04 (0.21)	0.01	0.04 (0.20)
Alcohol Consumption			0.25	2.80 (0.30) ^b			-0.00	-0.10 (0.65)			-0.02	-1.04 (1.52)			-0.05	-0.06 (0.04)			-0.04	-0.07 (0.06)
Exercise frequency			0.09	3.91 (1.14) ^b			0.00	0.38 (2.45)			-0.09	-17.13 (5.71) ^a			-0.12	-0.66 (0.16) ^b			-0.15	-1.13 (0.22) ^b
Smoking			-0.09	-4.89 (1.49) ^a			0.06	6.81 (3.20) ^a			0.02	4.84 (7.46)			0.04	0.28 (0.21)			0.02	0.20 (0.30)
Hypertension			-0.11	-4.42 (1.13) ^b			0.01	0.46 (2.42)			0.13	24.09 (5.63) ^b			0.15	0.74 (0.16) ^b			0.12	0.83 (0.22) ^b
Diabetes			-0.02	-1.54 (1.70)			-0.06	-7.40 (3.66) ^a			0.07	18.56 (8.52) ^a			0.04	0.32 (0.24)			0.07	0.74 (0.33) ^a
Stroke			-0.00	-0.64 (6.77)			-0.03	-14.99 (14.54)			0.02	23.12 (33.88)			0.06	1.75 (0.94)			0.07	3.03 (1.31) ^a
Antihyperlipidemic Agent Medication			-0.07	-2.50 (1.09) ^a			-0.34	-25.46 (2.33) ^b			0.01	2.44 (5.44)			0.03	0.16 (0.15)			-0.05	-0.32 (0.21)

Note. β = standardized regression coefficient.

Demographics controlled in Model 1. Demographics and health-related behaviours controlled in Model 2. Alcohol consumption was measured based on participants' frequency of drinking in the past month (1 = *Never drinks*, 6 = *Everyday*). Smoking habits was measured based on whether participants currently smoked regularly (0 = *No*, 1 = *Yes*). Exercise frequency was measured based on whether they engaged in regular exercise, or activity, for at least 20 min, three times per week (0 = *No*, 1 = *Yes*). HDL = high-density lipoprotein, LDL = low-density lipoprotein.

^a $p < .05$.

^b $p < .001$.



Fig. 1. Forest Plot of General Computer Use Frequency on Cardiovascular Risk Biomarkers

Note. Position of each square indicates the effect size contributed by the cardiovascular risk biomarker on general computer use frequency. Size of each square indicates sample size. Whiskers indicate 95% confidence intervals.

acid derivatives, bile acid sequestrants, cholesterol absorption inhibitors (0 = No, 1 = Yes).

2.3. Data analysis

The primary aim of the current study was to investigate the relationship between general computer use frequency, computer use at work frequency and cardiovascular risk biomarkers, specifically HDL cholesterol, LDL cholesterol, triglycerides, interleukin-6, and C-reactive protein. The predictor variables were taken from the National Survey of Midlife Development in the United States (MIDUS II), 2004–2006 (Ryff et al., 2021) while the cardiovascular risk biomarkers were taken from the National Survey of Midlife Development in the United States II (MIDUS II): Biomarker Project (Ryff et al., 2010). We employed ordinary least squares regression to examine this association, using general computer use frequency and computer use at work frequency as the predictor variables. The full dataset was used to analyse the relationship between general computer use frequency and cardiovascular risk biomarkers ($N = 1054$) while the dataset used to analyse the relationship between computer use at work frequency and cardiovascular risk biomarkers only included participants who were employed at the time of the study ($N = 573$). Two models were estimated for each cardiovascular risk biomarker. In the first model, we controlled for demographic variables including age, gender, education level, household income, race, and employment status which have previously shown associations with cardiovascular risk in existing research (Carson et al., 2009; Hartanto et al., 2021; Hoeymans et al., 1996; Li et al., 2016; Roth et al., 2015; Strand & Tverdal, 2004; Walsemann et al., 2016). However, for the analysis of the relationship between computer use at work and cardiovascular risk biomarkers, employment status was not included as a covariate given that all the participants were employed. The second model included additional controls for health-related variables and behaviours known to impact cardiovascular health, such as smoking status, alcohol consumption, regular exercise frequency, and the use of anti-hyperlipidemic medications (Gastaldelli et al., 2010; Loprinzi et al., 2016; Mozaffarian et al., 2008). Additionally, an exploratory moderation analysis was conducted to examine whether age and employment status could moderate the relationship between general computer use frequency and cardiovascular risk biomarkers. An exploratory mediation analysis was also carried out to determine whether exercise frequency could mediate the relationship between general computer use frequency, computer use at work frequency and cardiovascular risk biomarkers. Specifically, we were interested in the indirect effect of general computer use frequency, computer use at work frequency on cardiovascular risk biomarkers through exercise frequency. To mitigate the influence of extreme outliers, cardiovascular risk biomarker indices underwent winsorization to three standard deviations. Any missing values, accounting for less than 0.54% on any variable, were imputed using the expectation-maximisation (EM) algorithm. All analyses were conducted in R version 4.3.1 (R Core Team, 2023). Descriptive statistics was computed via *psych* version 2.4.6 (Revelle, 2021). The main

analyses and exploratory moderation analysis was conducted with *lme4* version 1.1–35.1 (Bates et al., 2015) while the exploratory moderation analysis was conducted with mediation analysis was conducted with *lavaan* version 0.6–17 (Rosseel, 2012). Visualisations were created with *metafor* version 4.6.0 (Viechtbauer, 2010).

3. Results

3.1. Main analyses

3.1.1. General computer use frequency

After controlling for demographics in Model 1, as seen in Table 4 and Fig. 1, we found that there was no significant relationship between general computer use frequency and HDL cholesterol ($\beta = -0.00$, $b = -0.00$, $SE = 0.28$, 95% CI = $[-0.56, 0.55]$, $p = .987$), LDL cholesterol ($\beta = -0.04$, $b = -0.67$, $SE = 0.61$, 95% CI = $[-1.86, 0.52]$, $p = .267$), triglycerides ($\beta = -0.04$, $b = -1.46$, $SE = 1.35$, 95% CI = $[-4.11, 1.19]$, $p = .279$), interleukin-6 ($\beta = -0.06$, $b = -0.07$, $SE = 0.04$, 95% CI = $[-0.14, 0.01]$, $p = .071$) and C-reactive protein ($\beta = 0.01$, $b = 0.02$, $SE = 0.05$, 95% CI = $[-0.08, 0.12]$, $p = .695$).

After controlling for demographics and health-related behaviours in Model 2, as seen in Table 4 and Fig. 1, we found that there was no significant relationship between general computer use frequency and HDL cholesterol ($\beta = -0.03$, $b = -0.32$, $SE = 0.27$, 95% CI = $[-0.84, 0.21]$, $p = .240$), LDL cholesterol ($\beta = -0.01$, $b = -0.20$, $SE = 0.58$, 95% CI = $[-1.33, 0.93]$, $p = .729$), triglycerides ($\beta = -0.02$, $b = -0.73$, $SE = 1.34$, 95% CI = $[-3.36, 1.91]$, $p = .589$), interleukin-6 ($\beta = -0.04$, $b = -0.04$, $SE = 0.04$, 95% CI = $[-0.12, 0.03]$, $p = .233$) and C-reactive protein ($\beta = 0.04$, $b = 0.06$, $SE = 0.05$, 95% CI = $[-0.04, 0.16]$, $p = .234$).

3.1.2. Computer use at work frequency

After controlling for demographics in Model 1, as seen in Table 5 and Fig. 2, we found that there was no significant relationship between computer use at work frequency and HDL cholesterol ($\beta = -0.06$, $b = -0.74$, $SE = 0.50$, 95% CI = $[-1.72, 0.25]$, $p = .143$), LDL cholesterol ($\beta = 0.02$, $b = 0.43$, $SE = 1.06$, 95% CI = $[-1.66, 2.52]$, $p = .688$), triglycerides ($\beta = 0.02$, $b = 1.00$, $SE = 2.35$, 95% CI = $[-3.61, 5.62]$, $p = .670$), interleukin-6 ($\beta = -0.01$, $b = -0.02$, $SE = 0.06$, 95% CI = $[-0.14, 0.10]$, $p = .750$) and C-reactive protein ($\beta = 0.03$, $b = 0.07$, $SE = 0.09$, 95% CI = $[-0.11, 0.26]$, $p = .427$).

After controlling for demographics and health-related behaviours in Model 2, as seen in Table 5 and Fig. 2, we found that there was no significant relationship between computer use at work frequency and HDL cholesterol ($\beta = -0.07$, $b = -0.89$, $SE = 0.47$, 95% CI = $[-1.82, 0.04]$, $p = .061$), LDL cholesterol ($\beta = 0.05$, $b = 1.32$, $SE = 1.03$, 95% CI = $[-0.72, 3.35]$, $p = .204$), triglycerides ($\beta = 0.02$, $b = 1.18$, $SE = 2.37$, 95% CI = $[-3.48, 5.84]$, $p = .619$), interleukin-6 ($\beta = -0.01$, $b = -0.02$, $SE = 0.06$, 95% CI = $[-0.14, 0.10]$, $p = .737$) and C-reactive protein ($\beta = 0.04$, $b = 0.09$, $SE = 0.09$, 95% CI = $[-0.09, 0.27]$, $p = .344$).

Table 5
Standardized Regression Coefficients of Computer Use at Work Frequency on Cardiovascular risk Biomarkers.

Predictor	HDL Cholesterol				LDL Cholesterol				Triglycerides				Interleukin-6				C-reactive Protein			
	Model 1		Model 2		Model 1		Model 2		Model 1		Model 2		Model 1		Model 2		Model 1		Model 2	
	β	<i>b</i> (SE)	β	<i>b</i> (SE)	β	<i>b</i> (SE)	β	<i>b</i> (SE)	β	<i>b</i> (SE)	β	<i>b</i> (SE)	β	<i>b</i> (SE)	β	<i>b</i> (SE)	β	<i>b</i> (SE)	β	<i>b</i> (SE)
Computer Use at Work Frequency	-0.06	-0.74 (0.50)	-0.07	-0.89 (0.47)	0.02	0.43 (1.06)	0.05	1.32 (1.03)	0.02	1.00 (2.35)	0.02	1.18 (2.37)	-0.01	-0.02 (0.06)	-0.01	-0.02 (0.06)	0.03	0.07 (0.09)	0.04	0.09 (0.09)
Covariates																				
Age	0.10	0.19 (0.07) ^a	0.14	0.25 (0.07) ^b	-0.13	-0.48 (0.15) ^a	-0.02	-0.07 (0.16)	-0.07	-0.59 (0.34)	-0.09	-0.75 (0.37) ^a	0.12	0.03 (0.01) ^a	0.08	0.02 (0.01)	-0.04	-0.01 (0.01)	-0.04	-0.01 (0.01)
Gender (% Male)	-0.41	-14.19 (1.32) ^b	-0.45	-15.61 (1.26) ^b	0.06	3.75 (2.79)	0.08	5.30 (2.76)	0.28	42.04 (6.17) ^b	0.29	43.29 (6.33) ^b	-0.04	-0.15 (0.16)	-0.03	-0.11 (0.16)	-0.22	-1.30 (0.25) ^b	-0.19	-1.14 (0.25) ^b
Race (% White)	-0.04	-2.63 (2.49)	-0.07	-4.28 (2.33)	0.03	3.43 (5.28)	-0.02	2.59 (5.09)	0.07	20.28 (11.66)	0.08	23.26 (11.67) ^a	-0.03	-0.19 (0.30)	-0.00	-0.03 (0.30)	-0.03	-0.33 (0.46)	-0.01	-0.15 (0.46)
Education Attainment	0.06	0.40 (0.29) ^a	0.03	0.22 (0.27)	-0.05	-0.62 (0.61)	-0.05	-0.68 (0.58)	-0.02	-0.68 (1.34)	-0.01	-0.32 (1.34)	-0.05	-0.04 (0.03)	-0.02	-0.02 (0.03)	-0.10	-0.13 (0.05) ^a	-0.08	-0.10 (0.05) ^a
Income	-0.01	-0.00 (0.01)	-0.05	-0.01 (0.01)	-0.08	-0.04 (0.02)	-0.05	-0.03 (0.02)	-0.06	-0.08 (0.05)	-0.05	-0.07 (0.05)	-0.01	-0.00 (0.00)	0.01	0.00 (0.00)	0.00	0.00 (0.00)	0.02	0.00 (0.00)
Alcohol Consumption			0.28	3.17 (0.41) ^b			-0.01	-0.12 (0.90)			-0.04	-1.76 (2.06)			-0.09	-0.11 (0.05) ^a			-0.09	-0.18 (0.08) ^a
Exercise			0.10	4.26 (1.56) ^a			0.01	1.18 (3.41)			-0.05	-9.46 (7.82)			-0.16	-0.75 (0.20) ^b			-0.15	-1.12 (0.31) ^b
Smoking			-0.10	-5.37 (1.96) ^a			0.09	9.41 (4.28) ^a			0.05	12.4 (9.80)			0.04	0.21 (0.25)			0.01	0.13 (0.38)
Hypertension			-0.10	-4.09 (1.60) ^a			0.00	0.02 (3.50)			0.12	22.8 (8.04) ^a			0.11	0.49 (0.20) ^a			0.06	0.43 (0.31)
Diabetes			0.01	0.48 (2.62)			-0.06	-8.00 (5.73)			-0.01	-2.61 (13.15)			0.04	0.32 (0.33)			0.11	1.30 (0.51) ^a
Stroke			0.04	14.41 (14.60)			0.01	9.77 (31.91)			0.01	13.49 (73.19)			-0.02	-0.95 (1.85)			0.05	3.29 (2.86)
Antihyperlipidemic Agent Medication			-0.12	-4.72 (1.52) ^a			-0.29	-21.86 (3.33) ^b			-0.01	-0.99 (7.63)			0.07	0.29 (0.19)			-0.05	-0.33 (0.30)

Note. β = standardized regression coefficient.

Demographics controlled in Model 1. Demographics and health-related behaviours controlled in Model 2. Alcohol consumption was measured based on participants' frequency of drinking in the past month (1 = *Never drinks*, 6 = *Everyday*). Smoking habits was measured based on whether participants currently smoked regularly (0 = *No*, 1 = *Yes*). Exercise frequency was measured based on whether they engaged in regular exercise, or activity, for at least 20 min, three times per week (0 = *No*, 1 = *Yes*). HDL = high-density lipoprotein, LDL = low-density lipoprotein.

^a $p < .05$.

^b $p < .001$.

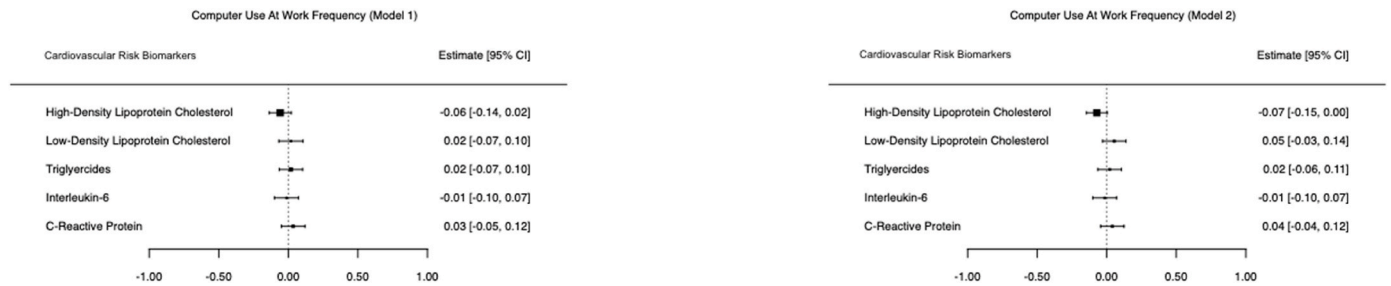


Fig. 2. Forest Plot of Computer Use at Work Frequency on Cardiovascular Risk Biomarkers

Note. Position of each square indicates the effect size contributed by the cardiovascular risk biomarker on computer use at work frequency. Size of each square indicates sample size. Whiskers indicate 95% confidence intervals.

3.2. Exploratory moderation analysis

3.2.1. General computer use frequency

Age. In Model 1, we found that age did not moderate the relationship between general computer use frequency and HDL cholesterol ($\beta = 0.01$, $b = 0.01$, $SE = 0.02$, $95\% \text{ CI} = [-0.04, 0.05]$, $p = .783$), LDL cholesterol ($\beta = -0.03$, $b = -0.05$, $SE = 0.05$, $95\% \text{ CI} = [-0.14, 0.05]$, $p = .315$), triglycerides ($\beta = 0.00$, $b = 0.01$, $SE = 0.11$, $95\% \text{ CI} = [-0.21, 0.22]$, $p = .942$), interleukin-6 ($\beta = -0.03$, $b = -0.00$, $SE = 0.00$, $95\% \text{ CI} = [-0.01, 0.00]$, $p = .322$) and C-reactive protein ($\beta = 0.00$, $b = 0.00$, $SE = 0.00$, $95\% \text{ CI} = [-0.01, 0.01]$, $p = .878$). Similarly, in Model 2, we found that age was not a significant moderator in the relationship between general computer use frequency and HDL cholesterol ($\beta = 0.02$, $b = 0.01$, $SE = 0.02$, $95\% \text{ CI} = [-0.03, 0.06]$, $p = .540$), LDL cholesterol ($\beta = -0.02$, $b = -0.04$, $SE = 0.05$, $95\% \text{ CI} = [-0.13, 0.05]$, $p = .428$), triglycerides ($\beta = 0.01$, $b = 0.03$, $SE = 0.11$, $95\% \text{ CI} = [-0.19, 0.25]$, $p = .780$), interleukin-6 ($\beta = -0.03$, $b = -0.00$, $SE = 0.00$, $95\% \text{ CI} = [-0.01, 0.00]$, $p = .399$) and C-reactive protein ($\beta = -0.01$, $b = -0.00$, $SE = 0.00$, $95\% \text{ CI} = [-0.01, 0.01]$, $p = .772$).

Employment Status. In Model 1, we found that employment status did not significantly moderate the relationship between general computer use frequency and HDL cholesterol ($\beta = -0.04$, $b = -0.68$, $SE = 0.57$, $95\% \text{ CI} = [-1.80, 0.44]$, $p = .233$), triglycerides ($\beta = 0.06$, $b = 5.15$, $SE = 2.73$, $95\% \text{ CI} = [-0.20, 10.50]$, $p = .059$), interleukin-6 ($\beta = 0.01$, $b = 0.03$, $SE = 0.08$, $95\% \text{ CI} = [-0.12, 0.18]$, $p = .698$) and C-reactive protein ($\beta = -0.03$, $b = -0.10$, $SE = 0.11$, $95\% \text{ CI} = [-0.31, 0.11]$, $p = .357$). Similar to Model 1, in Model 2, employment status was not a significant moderator in the relationship between general computer use frequency and HDL cholesterol ($\beta = -0.04$, $b = -0.79$, $SE = 0.54$, $95\% \text{ CI} = [-1.85, 0.28]$, $p = .148$), triglycerides ($\beta = 0.06$, $b = 4.90$, $SE = 2.71$, $95\% \text{ CI} = [-0.42, 10.22]$, $p = .071$), interleukin-6 ($\beta = 0.01$, $b = 0.02$, $SE = 0.08$, $95\% \text{ CI} = [-0.13, 0.17]$, $p = .775$) and C-reactive protein ($\beta = -0.03$, $b = -0.11$, $SE = 0.10$, $95\% \text{ CI} = [-0.31, 0.10]$, $p = .309$).

However, in Model 1, employment status significantly moderated between general computer use frequency and LDL cholesterol ($\beta = 0.09$, $b = 3.09$, $SE = 1.22$, $95\% \text{ CI} = [0.69, 5.48]$, $p = .012$). Similarly, in Model 2, employment status was a significant moderator between general computer use frequency and LDL cholesterol ($\beta = 0.08$, $b = 2.71$, $SE = 1.17$, $95\% \text{ CI} = [0.42, 5.00]$, $p = .021$). The simple slope analysis indicated that for unemployed participants, there was no significant relationship between general computer use frequency and LDL cholesterol ($b = -1.15$, $SE = 0.73$, $p = .120$). For employed participants, there was also no significant relationship between general computer use frequency and LDL cholesterol ($b = 1.55$, $SE = 0.92$, $p = .090$). Nevertheless, the correlation between general computer use frequency and LDL cholesterol became positive for participants who are employed.

3.2.2. Computer use at work frequency

Age. In Model 1, we found that age did not significantly moderate the relationship between computer use at work frequency and HDL

cholesterol ($\beta = 0.01$, $b = 0.01$, $SE = 0.05$, $95\% \text{ CI} = [-0.09, 0.11]$, $p = .774$), LDL cholesterol ($\beta = -0.02$, $b = -0.06$, $SE = 0.11$, $95\% \text{ CI} = [-0.27, 0.15]$, $p = .594$), triglycerides ($\beta = 0.03$, $b = 0.18$, $SE = 0.24$, $95\% \text{ CI} = [-0.28, 0.65]$, $p = .440$), interleukin-6 ($\beta = 0.00$, $b = 0.00$, $SE = 0.01$, $95\% \text{ CI} = [-0.01, 0.02]$, $p = .531$) and C-reactive protein ($\beta = -0.00$, $b = -0.00$, $SE = 0.01$, $95\% \text{ CI} = [-0.02, 0.02]$, $p = .957$). Similarly, in Model 2, we found that age did not significantly moderate the relationship between computer use at work frequency HDL cholesterol ($\beta = 0.01$, $b = 0.02$, $SE = 0.05$, $95\% \text{ CI} = [-0.08, 0.11]$, $p = .726$), LDL cholesterol ($\beta = -0.02$, $b = -0.04$, $SE = 0.11$, $95\% \text{ CI} = [-0.25, 0.16]$, $p = .679$), triglycerides ($\beta = 0.03$, $b = 0.17$, $SE = 0.24$, $95\% \text{ CI} = [-0.30, 0.65]$, $p = .481$), interleukin-6 ($\beta = 0.03$, $b = 0.01$, $SE = 0.01$, $95\% \text{ CI} = [-0.01, 0.02]$, $p = .398$) and C-reactive protein ($\beta = -0.00$, $b = -0.00$, $SE = 0.01$, $95\% \text{ CI} = [-0.02, 0.02]$, $p = .908$).

3.3. Exploratory mediation analysis

An exploratory mediation analysis was conducted with cardiovascular risk biomarkers as the outcome variable, general computer use frequency and computer use at work frequency as predictor variables and exercise frequency as the mediator. Demographics such as age and gender were controlled in Model 1, while both demographics and health-related behaviours such as hypertension and diabetes were controlled for in Model 2. The bias-corrected resampling method (1000 sample) showed that exercise frequency was not a significant mediator between general computer use frequency and any of the cardiovascular risk biomarkers. Exercise frequency was also not a significant mediator between computer use at work frequency and any of the cardiovascular risk biomarkers.

3.3.1. General computer use frequency

In Model 1, exercise frequency was not a significant mediator between general computer use frequency and HDL cholesterol ($\beta = 0.00$, $b = 0.03$, $SE = 0.04$, $95\% \text{ CI} = [-0.04, 0.09]$, $p = .461$), LDL cholesterol ($\beta = 0.00$, $b = 0.01$, $SE = 0.02$, $95\% \text{ CI} = [-0.03, 0.04]$, $p = .696$), triglycerides ($\beta = -0.00$, $b = -0.10$, $SE = 0.14$, $95\% \text{ CI} = [-0.38, 0.17]$, $p = .464$), interleukin-6 ($\beta = -0.00$, $b = -0.00$, $SE = 0.01$, $95\% \text{ CI} = [-0.01, 0.01]$, $p = .459$) and C-reactive protein ($\beta = -0.00$, $b = -0.01$, $SE = 0.01$, $95\% \text{ CI} = [-0.02, 0.01]$, $p = .457$). In Model 2, exercise frequency was also not a significant mediator between general computer use frequency and HDL cholesterol ($\beta = 0.00$, $b = 0.01$, $SE = 0.03$, $95\% \text{ CI} = [-0.04, 0.07]$, $p = .668$), LDL cholesterol ($\beta = 0.00$, $b = 0.00$, $SE = 0.01$, $95\% \text{ CI} = [-0.02, 0.02]$, $p = .882$), triglycerides ($\beta = -0.00$, $b = -0.05$, $SE = 0.13$, $95\% \text{ CI} = [-0.30, 0.19]$, $p = .669$), interleukin-6 ($\beta = -0.00$, $b = -0.00$, $SE = 0.01$, $95\% \text{ CI} = [-0.01, 0.01]$, $p = .668$) and C-reactive Protein ($\beta = -0.00$, $b = -0.00$, $SE = 0.01$, $95\% \text{ CI} = [-0.02, 0.01]$, $p = .667$).

3.3.2. Computer use at work frequency

In Model 1, exercise frequency was not a significant mediator between computer use at work frequency and HDL cholesterol ($\beta = 0.00$, b

= 0.02, $SE = 0.06$, 95% CI = [-0.10, 0.14], $p = .770$), LDL cholesterol ($\beta = 0.00$, $b = 0.01$, $SE = 0.04$, 95% CI = [-0.06, 0.08], $p = .785$), triglycerides ($\beta = -0.00$, $b = -0.04$, $SE = 0.12$, 95% CI = [-0.28, 0.21], $p = .775$), interleukin-6 ($\beta = -0.00$, $b = -0.00$, $SE = 0.01$, 95% CI = [-0.02, 0.02], $p = .770$) and C-reactive protein ($\beta = -0.00$, $b = -0.00$, $SE = 0.02$, 95% CI = [-0.03, 0.03], $p = .770$). In Model 2, exercise frequency was also not a significant mediator between computer use at work frequency and HDL cholesterol ($\beta = 0.00$, $b = 0.01$, $SE = 0.05$, 95% CI = [-0.10, 0.12], $p = .865$), LDL cholesterol ($\beta = 0.00$, $b = 0.00$, $SE = 0.02$, 95% CI = [-0.03, 0.04], $p = .878$), triglycerides ($\beta = -0.00$, $b = -0.02$, $SE = 0.12$, 95% CI = [-0.26, 0.22], $p = .866$), interleukin-6 ($\beta = -0.00$, $b = -0.00$, $SE = 0.01$, 95% CI = [-0.02, 0.02], $p = .865$) and C-reactive protein ($\beta = -0.00$, $b = -0.00$, $SE = 0.01$, 95% CI = [-0.03, 0.03], $p = .865$).

4. Discussion

In the present study, we examined the relationship between computer use and cardiovascular biomarkers in midlife and older adults to assess potential cardiovascular risk implications. We consistently found that neither general computer use frequency and computer use at work frequency was significantly associated with any of the 5 cardiovascular risk biomarkers included in the current study. Overall, our main findings do not support the hypothesis that computer use is positively associated with heightened cardiovascular risk in this demographic, challenging the concerns that sedentary behaviour related to computer use may negatively affect cardiovascular health.

The absence of significant relationships between computer use and any of the cardiovascular biomarkers suggests that frequent computer use may not be a reliable indicator of sedentary lifestyle. While this might contradict existing research on the relationship between sedentary lifestyle and cardiovascular health (Fotheringham et al., 2000; Gatto & Tak, 2008; Harvey et al., 2013), it highlights the possibility that computer use alone does not fully capture the complexity of sedentary behaviour. This is further supported by our exploratory mediation analysis, which showed that exercise frequency did not significantly mediate the relationship between computer use and cardiovascular risk. This suggests that computer use and cardiovascular risk is not directly affected by exercise frequency alone. In fact, we found a significant positive zero-order correlation between general computer use frequency and exercise frequency (see Table 2), indicating that computer users may engage in regular exercise. This could be explained by how older adults who use computers regularly may be more health-conscious and proactive about mitigating potential health risks (Hunsaker et al., 2021; Wan et al., 2022). Furthermore, the evolving technology landscape has introduced a wide range of tools and apps, such as physical trackers like FitBit and online coaching platforms, that may have been integrated into the daily routines of computer users to help them maintain an active lifestyle (Hurling et al., 2007; Longhini et al., 2024; Newbold et al., 2021; Petersen et al., 2020).

Our exploratory moderation further reinforces the complexity behind the relationship between computer use and cardiovascular risk. This analysis revealed that employment status significantly moderated the relationship between general computer use frequency and LDL cholesterol. Although the simple slope analysis did not find a significant relationship for participants regardless of employment status, we found that the correlation between general computer use frequency and LDL cholesterol became positive among employed participants. Work-related factors may play a role in influencing cardiovascular risks associated with computer use at work as individuals are more likely to spend extended amounts of time in front of a computer with fewer opportunities for movement or breaks (Parry & Straker, 2013; Smith et al., 2015). However, future studies should replicate these findings using larger samples to confirm the robustness of this relationship. These findings have important implications for public health recommendations and interventions, especially in midlife and older adults. Firstly,

public health strategies should emphasize the importance of taking regular breaks and reducing prolonged periods of sitting across multiple contexts. Furthermore, given the complexity of computer use, public health strategies should adopt a more holistic approach, considering other activities associated with sedentary behaviour such as television viewing (Karkauskienė et al., 2023; Katzmarzyk & Lee, 2012; Thorp et al., 2011).

Our study has several limitations that should be acknowledged. Firstly, the cross-sectional nature of this study limits our ability to draw causal conclusions, making the results susceptible to reverse causation (Kramer, 1988; Wang & Cheng, 2020). Future studies should adopt a longitudinal design to better understand the relationship between computer use and cardiovascular risk in midlife and older adults. Secondly, our study utilizes self-reported measures of computer use frequency, which may lack validity (Kastelic & Sarabon, 2019; Parry et al., 2021). This limitation could explain the observed lack of association between both forms of computer use frequency and cardiovascular biomarkers as evidence suggests that self-reported computer use frequency often fails to reflect actual usage patterns (Kramer, 1988; Wang & Cheng, 2020). Moreover, self-reported measures may oversimplify the complexity of computer use behaviour, failing to capture important factors such as the context, purpose and intensity of use, possibly leading to inconsistencies when compared with more objective data on computer use frequency (Araujo et al., 2017). To overcome these limitations, future research could adopt more objective measures such as wearable sensors that can track screen time and movement or continuous biomarker monitoring, offering a more detailed understanding of how computer use may impact cardiovascular health (Group, 2021; Li et al., 2022).

In conclusion, our study's findings highlight the complexity of the relationship between computer use and cardiovascular health among midlife and older adults. While our results do not support a straightforward link between computer use and heightened cardiovascular risk, they encourage further investigation into the diverse factors at play, including the nature of computer use, the context in which computer use occurs, and the individual characteristics and behaviours of computer users. This nuanced approach will contribute to a more comprehensive understanding of how technology and sedentary behaviour intersect with cardiovascular health in the modern age.

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CRediT authorship contribution statement

Meilan Hu: Writing – review & editing, Writing – original draft, Methodology, Investigation, Formal analysis, Conceptualization. **Hu Fen Diong:** Writing – review & editing, Writing – original draft, Project administration, Methodology, Formal analysis, Conceptualization. **K.T. A. Sandeeshwara Kasturiratna:** Writing – review & editing, Formal analysis. **Andree Hartanto:** Writing – review & editing, Supervision, Investigation, Formal analysis, Conceptualization.

Declaration of competing Interest

The authors declare no conflict of interest.

Data availability

All MIDUS datasets and documentation are archived and publicly available at the ICPSR repository (<http://www.icpsr.umich.edu/>) at the University of Michigan.

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