**Abstract**

Although many studies have examined the impact of social media use on mental health, little is known about the association of social media use with health-relevant biomarkers. Addressing this gap, we conducted a longitudinal study examining the link between social media use and C-reactive protein (CRP), a biological marker of systemic inflammation predictive of chronic diseases and mortality. Specifically, we measured 171 college students’ amount of social media use objectively via screen time application and collected blood samples at baseline and four weeks later. Social media use was associated with elevated CRP cross-sectionally. Critically, more social media use at baseline predicted increased CRP four weeks later, suggesting that increased social media use led to heightened inflammation during that period. Although more research is needed to understand why social media use led to higher inflammation, the association between objective social media use and a marker of a biological process critical to physical health presents an intriguing opportunity for future research on social media effects.

*Keywords:* Social media use, inflammation, physical health, well-being, screen time

**Introduction**

The past decade has witnessed a plethora of studies examining the impact of social media use on daily lives (e.g., Boyd & Ellison, 2007; Kross et al., 2013; Valkenburg, 2022). For example, more than 80 meta-analyses and reviews have examined the impact of social media use on psychological well-being and mental health (Orben, 2020). By comparison however, much less work has investigated the impact of social media use on health-relevant biology. This is surprising given the importance of physical health and the recent public dialogue on the potentially harmful effects of social media use (e.g., the Guardian, 2021). The goal of the present research is to begin addressing this knowledge gap. Specifically, we examine how social media use is associated with a biological process that influences physical health, namely, systemic inflammation—a potent driver of chronic illnesses such as cardiovascular diseases, Type 2 diabetes, and multiple cancers (see Kiecolt-Glaser et al., 2010).

*Physical Health implications of Inflammation*

Inflammation, a form of activation of the immune system, is a key biological process that affects physical health (Fagundes & Way, 2014). Whereas acute, local inflammation promotes healing by facilitating elimination of viruses and pathogens, chronic, systemic, low-grade inflammation may have detrimental health consequences by affecting many health-relevant systems in the body (Bennett et al., 2018), including the brain (Couzin-Frankel, 2010). While multiple biomarkers in the blood have been used to assess inflammation, the most commonly used biomarker is C-Reactive Protein (CRP). Elevated CRP is associated with increased risk of cardiovascular disease (Emerging Risk Factors Collaboration, 2010), and can predict multiple forms of cancer, including lung, breast, and prostate cancers (Michels et al., 2021), Type 2 diabetes (Pradhan et al., 2001), and earlier mortality (Ni et al., 2020). Thus, the broad health consequences of inflammation suggest that it is an important biological pathway that can influence ~~make it an important indicator or risk marker of~~ physical health. Importantly, recent research indicates that inflammation can also be elevated by factors such as psychological stress, poor diet, inactivity, and sleep deprivation (Furman et al., 2019). In line with this, several studies have investigated inflammation in the context of understanding how various psychosocial and socio-environmental factors (e.g., stress, socioeconomic status, inequality) can contribute to the development of chronic diseases or mortality (see Li et al., 2017; Singh-Manoux et al., 2017). Thus, for our purpose, inflammation is an excellent starting point to study the potential biological effects of social media use.

*Social Media Use and Inflammation*

How might social media use influence inflammation? Several perspectives suggest a potential link. One possibility is that social media use may influence health-related behaviors. For instance, some studies show that excessive social media use or screen time may undermine the quality and quantity of sleep (Cain & Gradisar, 2010; Thomée et al., 2011). Poor quality or insufficient amount of sleep is linked to higher inflammation (Irwin et al., 2016; Mullington et al., 2010). Similarly, prolonged social media use may contribute to sedentary lifestyles and physical inactivity (Allen et al., 2016; Odiaga & Doucette, 2017), which can increase inflammation levels (Henson et al., 2013; Yates et al., 2012). This perspective is consistent with the displacement hypothesis, which argues that social media use may have a negative impact on individuals if the time spent using it displaces health-promoting activities (Kushlev & Leitao, 2020; Verduyn et al., 2021; see Liu et al., 2019).

Another possibility is that social media use may increase stress, which can elevate inflammation (Kiecolt-Glaser, 2010; Slavich & Irwin, 2014; see Afifi et al., 2018). For example, scholars have argued that hyperconnectivity—the permanent availability of and connectivity to other people and various media contents on social media—can heighten stress (Freytag et al., 2021; Kushlev & Dunn, 2015; Misra & Stokols, 2012; Reinecke et al., 2017; Thomée et al., 2011). Moreover, on social media people may encounter information that can contribute to stress. For example, people may be exposed to offensive or hateful contents and experience online hate (Bilewicz & Soral, 2021; Rieger et al., 2021; see Quandt et al., 2022; also see Walther, 2022). Other times, they may frequently come across other people’s achievements and positive news on social media (Kross et al., 2013); constant exposure to such information about others can trigger upward social comparisons and feelings of envy (Verduyn et al., 2015). Such experience of stress, whether real or imagined, can trigger pro-inflammatory responses (see Eisenberger et al., 2017).

Consistent with the above perspectives, recent cross-sectional studies have provided initial evidence for the link between the amount of social media use and inflammation. For example, Afifi and colleagues (2018) discovered a positive correlation between adolescent’s self-reported Facebook use and blood levels of Interleukin-6 (IL-6; an inflammatory biomarker that triggers CRP synthesis; Sproston & Ashworth, 2018). In another cross-sectional study, Lee and colleagues (2022) showed that college students’ self-reported social media use across four platforms (i.e., Snapchat, Instagram, Facebook, and Twitter) was positively associated with CRP.

Although these initial studies begin to suggest the impact of social media use on inflammation, they also have some limitations. First, these studies measured the amount of social media use by asking participants to retrospectively recall their amount of use over several days, a methodological approach that has been shown to provide imprecise estimates (see Parry et al., 2021). For instance, one study of approximately 50,000 people found that various survey measures of Facebook behavior correlated only moderately (.23 < *r* < .42) with people’s actual Facebook usage (Ernala et al., 2020). Thus, it is unclear how precisely the amount of social media use is associated with inflammation. Second, these studies’ reliance on cross-sectional data limits the ability to draw strong temporal or causal interpretations, which has been noted as a “major weakness” in social media research (Kross et al., 2020; Orben, 2020). Thus, the present research sought to address these issues by measuring social media use objectively via the screen time application and employing a longitudinal design. Specifically, we tested the following hypotheses:

H1: Higher social media use, measured objectively, will be associated with higher levels of inflammation (CRP) concurrently.

H2: Higher social media use, measured objectively, will be associated with increased levels of inflammation (CRP) over time.

**Methods**

*Participants and procedure*

Data collection for this study occurred between September 2021 and May 2022. We recruited one hundred and seventy-one undergraduate students from a large Midwestern university in the United States for partial course credit. This longitudinal study consisted of two parts: a baseline lab session (Phase 1) and a follow-up lab session approximately four weeks after (Phase 2; N = 141). Because the screen time application we used to collect objective social media use data was only offered on the iOS operating system, only 138 iPhone users were retained in the study (87 females; *M*age = 19.13, *SD*age = 2.67; 64.5% White, 23.2% Asian/Pacific Islander, 11.6% African American, 6.5% Other, 5.8% Hispanic/Latin American).

During Phase 1, participants completed several background questionnaires assessing factors such as sociodemographic information, social media use, health behaviors, and other measures not relevant to this investigation. Once participants completed the questionnaires, a trained research assistant collected their blood samples in a separate room. Participants could choose not to provide their blood and continue the study without losing compensation. In Phase 1, thirteen (7.60%) participants opted out of the blood sample collection procedure, and another four participants (2.34%) provided insufficient amount of blood to assay. Approximately four weeks after completing Phase 1, participants returned to the laboratory for Phase 2. We decided on a four-week window between Phase 1 and Phase 2 to allow enough time to observe variability in CRP (Meier-Ewert et al., 2001; Mills et al., 2009). Similar to Phase 1, in Phase 2 participants completed various questionnaires and provided blood samples for CRP. In Phase 2, fourteen (9.93%) participants opted out of the blood collection procedure, and another thirty-six (25.53%) participants did not provide their blood samples because their data collection occurred when we had paused the collection of blood samples due to an IRB related issue. The authors’ university’s Institutional Review Board approved all research reported in this manuscript.

*Measures*

*Phases 1 and 2 CRP.* CRP was assayed from dried blood spots following an established protocol from prior work (McDade et al., 2004). First, we swabbed participants’ finger with alcohol and pricked it with an 18-gauge needle (Owen Mumford Unistick 3). We collected the blood drops on a Whatman 903 Protein Saver Card and kept the samples for 24 hours to dry at room temperature. Next, we punched the samples with a 3mm punch and stored them in microcentrifuge tubes at -80°C. To assay, we thawed a single 3mm punch and added 200µl of buffer (Phosphate Buffered Saline with .1% Tween 20) overnight incubation at 4°C while shaking at 60rpm. We then diluted this eluate 1:10 and assayed CRP the following morning using Meso Scale Delivery Vplex Plus kits (K151STG). All samples were successfully assayed (i.e., within the linear range of the standard curve). The intraassay coefficient of variation (CV) was 3.8%, while the interassay CV was 11.04% (*MPhase1* = .99mg/L, *SDPhase1* = 1.75mg/L; *MPhase2* = .98mg/L, *SDPhase2* = 1.48mg/L).

*Phases 1 and 2 Social media use.* Participants reported the amount of time they spent using each of the four social media platforms (i.e., Snapchat, Instagram, Twitter, and Facebook) during the weeks of Phase 1 and Phase 2. Participants were instructed on how to retrieve this information from the screen time application on their iPhone (the iOS operating system). For our predictors, we summed participants’ weekly social media use across the four platforms to create a composite *social media use* variable for each week (*MPhase1* = 579.05 minutes, *SDPhase1* = 436.70 minutes; *MPhase2* = 566.98 minutes, *SDPhase2* = 379.69 minutes).

We measured social media use across four platforms for the following reasons. First, at the time of our study design, Snapchat, Instagram, Twitter, and Facebook were the most commonly used social media platforms among college students (Perrin & Anderson, 2018). Second, recent work suggests collecting social media use across multiple platforms because most people use multiple platforms in varying amounts (Bayer et al., 2020). Third, this approach is consistent with our prior work using self-reported social media use (\*citation masked for blind review).

*Covariates.* Consistent with recent recommendations (Horn et al., 2018) and our prior work (\*citation masked for blind review), we controlled for additional variables that can influence inflammation. Specifically, we included sociodemographic covariates (i.e., gender, age, household income, and highest level of education completed by mother and father (1 = *some high school*, 5 = *graduate school*). We also controlled for health-related behaviors such as body mass index (BMI), cigarette smoking (i.e., number of days participants smoked cigarettes in the past 30 days; 1 = *0 days*, 2 = *1 or 2 days*, 3 = *3 to 5 days*…7 = *20 to 29 days*, 8 = *all 30 days*), frequency of alcohol consumption (1 = *never*, 2 = *several times a year*, 3 = *monthly*, 4 = *2 - 4 times a month*, 5 = *2 – 3 times a week*, 6 = *4 or more times a week*), and amount of time spent sitting in the past month (1 = *none at all*, 2 = *a little*, 3 = *a moderate amount*, 4 = *a lot*, 5 = *a great deal*). Additional covariates included depressive symptoms (Center for Epidemiological Studies Depression Scale; Radloff, 1977) and the use of birth control pill (0 = *no,* 1 = *yes* (*N* = 42), because they can influence inflammation (Horn et al., 2018).

**Results**

*Data cleaning and exclusions*

First, eleven participants who had not had the screen time application enabled at the time of data collection (therefore did not have any screen time information available) were removed from the analysis. Second, consistent with conventional approaches of analyzing CRP, one participant whose CRP value was greater than 10 ug/mL was removed from analyses, because such values are likely to indicate an acute infection rather than heightened inflammation due to psychosocial factors such as social media use (Pearson et al., 2003). Including this participant in the analyses did not change any results substantively. Table 1 includes zero-order correlations for all key variables.

*Testing H1: Is social media use positively associated with CRP cross-sectionally?*

We conducted two sets of multiple regression analyses to test the cross-sectional relations between social media use and CRP (Phases 1 and 2). Consistent with the approach taken in our prior work on social media use and CRP (\*citation masked for blind review),the models sequentially controlled for an increasing number of covariates to provide details on how the association between social media use and CRP is influenced by the covariates: (1) socio-demographic factors, (2) health-related behaviors, (3) depressive symptoms, and (4) the use of birth control. Consistent with our hypothesis, social media use at Phase 1 was positively associated with CRP levels at Phase 1 in Model 1 (*β* = .25, *p* = .018), Model 2 (*β* = .22, *p* = .022), Model 3 (*β* = .23, *p* = .019), and Model 4 (*β* = .24, *p* = .011). Similarly, social media use at Phase 2 was positively associated with CRP levels at Phase 2 in Model 1 (*β* = .35, *p* = .013), Model 2 (*β* = .37, *p* = .011), Model 3 (*β* = .34, *p* = .024), and Model 4 (*β* = .31, *p* = .04). Thus, H1 was supported. The results of these analyses are summarized in Tables 2 and 3.

*Testing H2: Does social media use at Phase 1 predict increased CRP level at Phase 2?*

First, we conducted a series of multiple regression analyses with amount of social media use at Phase 1 as a predictor of CRP at Phase 2 while controlling for CRP at Phase 1. The amount of social media use at Phase 1 predicted CRP at Phase 2 in Model 1 (*β* = .19, *p* = .049), Model 2 (*β* = .23, *p* = .03), Model 3 (*β* = .22, *p* = .034), and Model 4 (*β* = .22, *p* = .037).

Next, we further explored this finding by additionally controlling for Phase 2 social media use in the models. Social media use at Phase 1 predicted CRP at Phase 2 in Model 1 (*β* = .31, *p* = .017), Model 2 (*β* = .32, *p* = .018), Model 3 (*β* = .32, *p* = .022), and Model 4 (*β* = .32, *p* = .026). Interestingly, in all models, Phase 2 social media use was not a significant predictor of CRP at Phase 2 (all *p*s > .67), suggesting that the impact of social media use on CRP may occur over time. Thus, H2 was also supported. The results of these analyses are detailed in Tables 4 and 5.

**Discussion**

The present research investigated how social media use is associated with CRP—a biological marker of inflammation linked with chronic illnesses such as cardiovascular disease and cancers. The results showed that the amount of social media use—assessed objectively via screen time application—was not only associated with higher inflammation cross-sectionally but also an increase in inflammation four weeks later. The findings were consistent across different models adjusting for various covariates.

To our knowledge, this is one of the first studies to demonstrate the link between objective social media use across several platforms with CRP, a biomarker of inflammation. Building on prior work that found a positive correlation between self-reported social media use and inflammation (Afifi et al., 2018; Lee et al., 2022), our longitudinal findings provide initial temporal evidence that social media use can lead to heightened inflammation. Importantly, that social media use predicted increased inflammation even after controlling for depressive symptoms is noteworthy because the finding suggests that social media effects may extend beyond psychological well-being.

Critically, the use of a health-relevant biomarker and the collection of objective social media use data across multiple platforms are key methodological strengths of our study. Compared with most prior studies that relied exclusively on self-report measures, our methodological approach is robust against survey response biases. Given these strengths, we encourage future research to utilize biological markers related to health or well-being and objective social media usage data in their research when applicable.

*Caveats and limitations*

There are some limitations of this study. First, this study tested an aggregate association between amount of social media use across different platforms and inflammation. As an initial attempt to understand the potential link between social media use and inflammation, our goal was *necessarily* broad, focusing on the general metric of amount of social media use—one of the most commonly measured and discussed variables in social media use research. Although this approach allowed us to better connect to extant research and public discourse (e.g., social media use and well-being), fully understanding social media effects requires measuring processes much more nuanced and complex. Given that people use social media for different purposes (e.g., entertainment, following the news, browsing, supporting friends), future research should examine the different ways of using social media to illuminate what aspects of social media use are associated with inflammation. Relatedly, the present research did not collect any data on the types of contents people viewed on social media. Because these contents can drastically influence users' psychological experience (see Valkenburg, 2022), we cannot ascertain the extent to which the contents people interacted with contributed to our findings. Thus, future research should aim to collect data on the contents people view on social media to better capture their experience (e.g., Screenomics approach; see Brinberg et al., 2021; also see Reeves et al., 2019). Moreover, although we had proposed some potential mechanisms for the link between social media use and inflammation, future research should seek to understand specific mediating mechanisms (e.g., sleep quality, stress) for our findings.

Finally, we recruited college students in our study because they represent a demographic with high social media use (Perrin & Anderson, 2018). However, it should also be noted that our sample consisted of relatively healthy young adults who were wealthy enough to own an iPhone. Thus, future research should seek to replicate our findings with a larger sample from more diverse populations to provide more confidence in the generalizability of the results. Similarly, although we did not find any moderators of our results (e.g., gender, depressive symptoms) in this study, future studies may examine additional individual difference variables (e.g., personality, loneliness) that may moderate our findings, given that social media effects are likely to vary at the individual level (Orben ,2020; Valkenburg, 2022).

*Broader Implications*

Recently, social media companies have come under intense public scrutiny over the possibility that social media use can harm the well-being of its users, especially, teenaged girls (e.g., the Guardian, 2021). However, absent from these discussions is the possibility that certain levels of social media use may have physical health implications for certain individuals. As mentioned in the introduction, CRP, and inflammation more generally, have been associated with a variety of undesirable health outcomes ranging from cardiovascular disease (Emerging Risk Factors Collaboration, 2010) and diabetes (Pradhan et al., 2001) to multiple forms of cancer (Michels et al., 2021). Although some of these health outcomes linked with inflammation do not manifest until adulthood, the underlying disease process begins much earlier, including during the adolescent and young adult period when social media use is highest. For example, atherosclerosis, the principal cause of cardiovascular disease, is present in adolescents (Berenson et al., 1998) and an adolescent’s health behaviors (e.g., smoking) or other risk factors (e.g., obesity) predict adulthood cardiovascular disease risk (Raitakari et al., 2003). Therefore, if the initial link between increases in social media use and elevated inflammation shown here is verified and shown to be causal, it would suggest the possibility that excessive social media use could increase risk for health outcomes such as cardiovascular disease. Although the evidence is preliminary at this point, we call for additional research investigating the potential physical health implications of social media use.

**Conclusion**

 The current research discovered that objective social media use is positively associated with inflammation cross-sectionally and increased inflammation over time. The relation between social media use and inflammation presents an intriguing opportunity for future research that integrates social media effects and biological processes. Given the prevalence of social media use in the daily lives of adolescents and young adults, and the societal importance of good physical health, more research investigating the physical health effects of social media use utilizing diverse methodologies is needed.

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Table 1. *Zero order correlations among key variables*

|  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- |
| Variables | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
| 1. SMU (P1) | -- |  |  |  |  |  |  |  |
| 2. SMU (P2) | .55\*\*\* | -- |  |  |  |  |  |  |
| 3. CRP (P1) | .23\* | .13 | -- |  |  |  |  |  |
| 4. CRP (P2) | .36\*\* | .24† | .77\*\*\* | -- |  |  |  |  |
| 5. Gender | .17† | .15† | .10 | .27\* | -- |  |  |  |
| 6. Age | -.18\*  | -.13 | .02 | .02 | -.11 | -- |  |  |
| 7. BMI | -.03 | .003 | .37\*\*\* | .35\*\* | -.04 | .04 | -- |  |
| 8. Depres | .07 | .002 | .11 | -.007 | .17† | -.05 | .04 | -- |

*Notes*. †*p* ≤ .10 \**p* ≤ .05. \*\**p* ≤ .01. \*\*\**p* ≤ .001 (two-tailed). SMU (P1) = social media use at Phase 1. SMU (P2) = social media use at Phase 2. CRP (P1) = logged CRP at Phase 1. CRP (P2) = logged CRP at Phase 2. Gender was coded with 1 (male) and 2 (female). BMI = Body mass index. Depres = depressive symptoms.

Table 2. *Coefficients from linear regression models predicting CRP at Phase 1*

|  |  |  |  |
| --- | --- | --- | --- |
| **Model 1** | **Model 2** | **Model 3** | **Model 4** |
| **Predictor** | ***b*** | ***p*** | **95% CI** | **Predictor** | ***b*** | ***p*** | **95% CI** | **Predictor** | ***b*** | ***p*** | **95% CI** | **Predictor** | ***b*** | ***p*** | **95% CI** |
| Gender | .12 | .27 | [-.10, .34] | Gender | .18 | .10 | [-.04, .39] | Gender | .17 | .11 | [-.04, .39] | Gender | -.02 | .84 | [-.27, .22] |
| Age | .02 | .26 | [-.02, .06] | Age | .02 | .22 | [-.01, .06] | Age | .02 | .17 | [-.01, .06] | Age | .02 | .34 | [-.02, .05] |
| Edu (M) | -.06 | .33 | [-.17, .06] | Edu (M) | -.01 | .97 | [-.11, .11] | Edu (M) | .01 | .86 | [-.10, .12] | Edu (M) | -.02 | .75 | [-.13, .09] |
| Edu (F) | .04 | .42 | [-.06, .14] | Edu (F) | .02 | .69 | [-.07, .11] | Edu (F) | .02 | .70 | [-.07, .11] | Edu (F) | .04 | .38 | [-.05, .13] |
| Income | .03 | .21 | [-.02, .07] | Income | .03 | .18 | [-.01, .07] | Income | .03 | .18 | [-.01, .07] | Income | .02 | .39 | [-.02, .06] |
| SMU (P1) | .01 | .018 | [.0001, .001] | BMI | .05 | <.001 | [.03, .07] | BMI | .05 | <.001 | [.03, .07] | BMI | .05 | <.001 | [.03, .06] |
|  |  |  |  | Smoking | -.03 | .45 | [-.12, .05] | Smoking | -.03 | .47 | [-.12, .06] | Smoking | -.02 | .57 | [-.11, .06] |
|  |  |  |  | Alcohol | .06 | .07 | [-.01, .12] | Alcohol | .06 | .08 | [-.01, .12] | Alcohol | .05 | .15 | [-.02, .11] |
|  |  |  |  | Sit | -.05 | .40 | [-.17, .07] | Sit | -.08 | .21 | [-.20, .05] | Sit | -.05 | .46 | [-.17, .08] |
|  |  |  |  | SMU (P1) | .01 | .022 | [.0001, .001] | Depres | .12 | .18 | [-.06, .31] | Depres | .09 | .29 | [-.08, .27] |
|  |  |  |  |  |  |  |  | SMU (P1) | .01 | .025 | [.0001, .001] | BirthCon  | .33 | .004 | [.11, .55] |
|  |  |  |  |  |  |  |  |  |  |  |  | SMU (P1) | .01 | .011 | [.0001, .001] |
| *R2* |  | .09 |  |  | .29 |  |  | .30 |  |  | .36 |

*Notes*. Gender was coded with 1 (male) and 2 (female). Edu (M) = highest degree obtained by mother; Edu (F) = highest degree obtained by father; Income = family annual income; Smoking = # of cigarettes smoked per day in the last 30 days; Alcohol = frequency of alcohol consumption; Sit = amount of time spent sitting in the past month; BirthCon = consumption of birth control medication. BirthCon was coded with 0 (not currently taking birth control medication) and 1 (currently taking birth control medication). *R2* values reflect those with social media use in the models. †*p* ≤ .10 \**p* ≤ .05. \*\**p* ≤ .01. \*\*\**p* ≤ .001 (two-tailed).

Table 3. *Coefficients from linear regression models predicting CRP at Phase 2 with social media use at Phase 2*

|  |  |  |  |
| --- | --- | --- | --- |
| **Model 1** | **Model 2** | **Model 3** | **Model 4** |
| **Predictor** | ***b*** | ***p*** | **95% CI** | **Predictor** | ***b*** | ***p*** | **95% CI** | **Predictor** | ***b*** | ***p*** | **95% CI** | **Predictor** | ***b*** | ***p*** | **95% CI** |
| Gender | .25 | .09 | [-.04, .54] | Gender | .20 | .16 | [-.08, .47] | Gender | .20 | .16 | [-.08, .47] | Gender | .05 | .80 | [-.31, .40] |
| Age | .04 | .06 | [-.01, .07] | Age | .03 | .06 | [-.02, .07] | Age | .04 | .049 | [.001, .07] | Age | .03 | .08 | [-.01, .07] |
| Edu (M) | -.12 | .09 | [-.27, .02] | Edu (M) | -.09 | .25 | [-.24, .06] | Edu (M) | -.08 | .29 | [-.23, .07] | Edu (M) | -.10 | .20 | [-.25, .05] |
| Edu (F) | .06 | .40 | [-.07, .19] | Edu (F) | .02 | .75 | [-.11, .15] | Edu (F) | .03 | .67 | [-.10, .16] | Edu (F) | .03 | .63 | [-.10, .16] |
| Income | .05 | .09 | [-.01, .11] | Income | .05 | .07 | [-.01, .11] | Income | .05 | .08 | [-.01, .11] | Income | .05 | .08 | [-.01, .11] |
| SMU (P2) | .01 | .013 | [.0001, .001] | BMI | .03 | .02 | [.01, .05] | BMI | .03 | .021 | [.01, .05] | BMI | .03 | .013 | [.01, .05] |
|  |  |  |  | Smoking | -.02 | .80 | [-.17, .13] | Smoking | -.02 | .81 | [-.17, .13] | Smoking | -.01 | .87 | [-.16, .14] |
|  |  |  |  | Alcohol | .07 | .11 | [-.02, .16] | Alcohol | .07 | .12 | [-.02, .16] | Alcohol | .06 | .19 | [-.03, .15] |
|  |  |  |  | Sit | .05 | .57 | [-.12, .21] | Sit | .03 | .77 | [-.15, .20] | Sit | .03 | .74 | [-.14, .20] |
|  |  |  |  | SMU (P2) | .01 | .011 | [.0001, .001] | Depres | .11 | .30 | [-.10, .32] | Depres | .11 | .28 | [-.09, .32] |
|  |  |  |  |  |  |  |  | SMU (P2) | .01 | .024 | [.0001, .001] | BirthCon  | .24 | .17 | [-.11, .58] |
|  |  |  |  |  |  |  |  |  |  |  |  | SMU (P2) | .01 | .04 | [.0001, .001] |
| *R2* |  | .27 |  |  | .40 |  |  | .42 |  |  | .45 |

*Notes*. †*p* ≤ .10 \**p* ≤ .05. \*\**p* ≤ .01. \*\*\**p* ≤ .001 (two-tailed).

Table 4. *Coefficients from linear regression models predicting CRP at Phase 2 (controlling for CRP at Phase 1)*

|  |  |  |  |
| --- | --- | --- | --- |
| **Model 1** | **Model 2** | **Model 3** | **Model 4** |
| **Predictor** | ***b*** | ***p*** | **95% CI** | **Predictor** | ***b*** | ***p*** | **95% CI** | **Predictor** | ***b*** | ***p*** | **95% CI** | **Predictor** | ***b*** | ***p*** | **95% CI** |
| Gender | .03 | .79 | [-.18, .24] | Gender | .04 | .73 | [-.19, .26] | Gender | .04 | .74 | [-.19, .26] | Gender | .03 | .86 | [-.25, .30] |
| Age | .02 | .23 | [-.01, .04] | Age | .02 | .29 | [-.01, .05] | Age | .02 | .29 | [-.02, .05] | Age | .02 | .31 | [-.02, .05] |
| Edu (M) | -.08 | .12 | [-.18, .02] | Edu (M) | -.07 | .22 | [-.19, .04] | Edu (M) | -.07 | .22 | [-.19, .05] | Edu (M) | -.07 | .23 | [-.19, .05] |
| Edu (F) | .06 | .22 | [-.04, .15] | Edu (F) | .05 | .31 | [-.05, .15] | Edu (F) | .05 | .32 | [-.05, .16] | Edu (F) | .05 | .32 | [-.05, .16] |
| Income | .01 | .79 | [-.04, .05] | Income | .01 | .69 | [-.04, .06] | Income | .01 | .69 | [-.04, .06] | Income | .01 | .69 | [-.04, .06] |
| CRP (P1) | .72 | <.001 | [.52, .92] | BMI | .01 | .39 | [-.01, .03] | BMI | .01 | .40 | [-.01, .03] | BMI | .01 | .40 | [-.01, .03] |
| SMU (P1) | .01 | .007 | [.0001, .001] | Smoking | .01 | .98 | [-.11, .12] | Smoking | .01 | .98 | [-.11, .12] | Smoking | .01 | .98 | [-.12, .12] |
|  |  |  |  | Alcohol | .02 | .69 | [-.06, .09] | Alcohol | .02 | .69 | [-.06, .09] | Alcohol | .01 | .71 | [-.06, .09] |
|  |  |  |  | Sit | .04 | .54 | [-.08, .15] | Sit | .04 | .57 | [-.09, .16] | Sit | .04 | .57 | [-.09, .16] |
|  |  |  |  | CRP (P1) | .66 | <.001 | [.41, .91] | Depres | .01 | .96 | [-.17, .18] | Depres | .01 | .95 | [-.17, .19] |
|  |  |  |  | SMU (P1) | .01 | .005 | [.0001, .001] | CRP (P1) | .66 | <.001 | [.38, .94] | BirthCon  | .02 | .87 | [-.26, .31] |
|  |  |  |  |  |  |  |  | SMU (P1) | .01 | .006 | [.0001, .001] | CRP (P1) | .65 | <.001 | [.36, .94] |
|  |  |  |  |  |  |  |  |  |  |  |  | SMU (P1) | .01 | .008 | [.0001, .001] |
| *R2* |  | .67 |  |  | .68 |  |  | .68 |  |  | .68 |

*Notes*. †*p* ≤ .10 \**p* ≤ .05. \*\**p* ≤ .01. \*\*\**p* ≤ .001 (two-tailed).

Table 5. *Coefficients from linear regression models predicting CRP at Phase 2 (controlling for CRP at Phase 1 and SMU at Phase 2)*

|  |  |  |  |
| --- | --- | --- | --- |
| **Model 1** | **Model 2** | **Model 3** | **Model 4** |
| **Predictor** | ***b*** | ***p*** | **95% CI** | **Predictor** | ***b*** | ***p*** | **95% CI** | **Predictor** | ***b*** | ***p*** | **95% CI** | **Predictor** | ***b*** | ***p*** | **95% CI** |
| Gender | .03 | .76 | [-.18, .25] | Gender | .04 | .73 | [-.19, .26] | Gender | .04 | .73 | [-.19, .27] | Gender | .03 | .85 | [-.25, .31] |
| Age | .02 | .29 | [-.01, .04] | Age | .02 | .29 | [-.01, .05] | Age | .02 | .31 | [-.02, .05] | Age | .02 | .33 | [-.02, .05] |
| Edu (M) | -.08 | .13 | [-.18, .02] | Edu (M) | -.07 | .23 | [-.19, .05] | Edu (M) | -.07 | .24 | [-.19, .05] | Edu (M) | -.07 | .24 | [-.19, .05] |
| Edu (F) | .06 | .22 | [-.04, .16] | Edu (F) | .05 | .32 | [-.05, .16] | Edu (F) | .05 | .32 | [-.05, .16] | Edu (F) | .05 | .32 | [-.05, .16] |
| Income | .01 | .83 | [-.04, .05] | Income | .01 | .71 | [-.04, .06] | Income | .01 | .71 | [-.04, .06] | Income | .01 | .71 | [-.04, .06] |
| CRP (P1) | .73 | <.001 | [.52, .94] | BMI | .01 | .39 | [-.01, .03] | BMI | .01 | .40 | [-.01, .03] | BMI | .01 | .40 | [-.01, .03] |
| SMU (P1) | .01 | .02 | [.0001, .001] | Smoking | .01 | .97 | [-.11, .12] | Smoking | .01 | .98 | [-.12, .12] | Smoking | .01 | .98 | [-.12, .12] |
| SMU (P2) | .001 | .67 | [-.01, .01] | Alcohol | .01 | .70 | [-.06, .09] | Alcohol | .01 | .70 | [-.06, .09] | Alcohol | .01 | .72 | [-.06, .09] |
|  |  |  |  | Sit | .03 | .61 | [-.10, .16] | Sit | .03 | .65 | [-.10, .17] | Sit | .03 | .65 | [-.11, .17] |
|  |  |  |  | CRP (P1) | .66 | <.001 | [.41, .92] | Depres | .01 | .92 | [-.18, .19] | Depres | .01 | .92 | [-.18, .20] |
|  |  |  |  | SMU (P1) | .01 | .018 | [.0001, .001] | CRP (P1) | .66 | <.001 | [.37, .94] | BirthCon  | .02 | .87 | [-.26, .31] |
|  |  |  |  | SMU (P2) | .001 | .86 | [-.01, .01] | SMU (P1) | .01 | .022 | [.0001, .001] | CRP (P1) | .65 | <.001 | [.36, .95] |
|  |  |  |  |  |  |  |  | SMU (P2) | .001 | .85 | [-.01, .01] | SMU (P1) | .01 | .026 | [.0001, .001] |
|  |  |  |  |  |  |  |  |  |  |  |  | SMU (P2) | .001 | .85 | [-.01, .01] |
| *R2* |  | .67 |  |  | .68 |  |  | .68 |  |  | .68 |

*Notes*. †*p* ≤ .10 \**p* ≤ .05. \*\**p* ≤ .01. \*\*\**p* ≤ .001 (two-tailed).