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Can inflammation predict social media use? Linking a biological marker of systemic inflammation with social media use among college students and middle-aged adults

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ABSTRACT

Drawing on recent evidence that inflammation may promote social affiliative motivation, the present research proposes a novel perspective that inflammation may be associated with more social media use. In a cross-sectional analysis of a nationally representative sample, Study 1 (N = 863) found a positive association between C-reactive protein (CRP), a biomarker of systemic inflammation, and the amount of social media use by middle-aged adults. Study 2 (N = 228) showed that among college students CRP was prospectively associated with more social media use 6 weeks later. Providing stronger evidence of the directionality of this effect, Study 3 (N = 171) showed that in college students CRP predicted increased social media use in the subsequent week even after controlling for current week's use. Additionally, in exploratory analyses of CRP and different types of social media use in the same week, CRP was only associated with using social media for social interaction and not for other purposes (e.g., entertainment). The present research sheds light on the social effects of inflammation and highlights potential benefits of using social media as a context for studying the impact of inflammation on social motivation and behavior.

1. Introduction

Inflammation, the body's response to injury and infection, is increasingly considered to influence social behavior (Eisenberger et al., 2017; Muscatell & Inagaki, 2021; Slavich, 2020). Although initial research in animal models of sickness behavior showed that acute elevations in inflammation increase social distancing and withdrawal (Dantzer, 2001; Dantzer and Kelley, 2007; Kelley et al., 2003), an emerging body of work suggests that the effects of inflammation on social behavior may be more nuanced (Eisenberger et al., 2017; Muscatell & Inagaki, 2021; Jolink et al., 2022). Specifically, under some circumstances, inflammation can increase social approach behaviors in animals and neural sensitivity to positive social stimuli in humans (Inagaki et al., 2015; Muscatell et al., 2016). For example, injection of a component of bacteria (Lipopolysaccharide; LPS) that triggers increases in inflammation led rhesus monkeys to spend more time clinging to their cage mate (Willette et al., 2007). Similarly, injecting LPS in humans increases self-reported desire to be around a close other and a potentiation of the neural response to pictures of a close other in a motivation-related brain region. The magnitude of this neural response was correlated with levels of an inflammatory protein (IL-6) in the blood after LPS injection (Inagaki et al., 2015). Moreover, flu vaccination, which also acutely increases levels of IL-6 (Jolink et al., 2022; Kuhlman et al., 2018), has been shown to increase the number of social interactions the person receiving the injection engages in (Reiber et al., 2010). Collectively, these findings indicate that when inflammation is experimentally elevated, it can promote affiliative behavior and motivation. Because cytokines can also be elevated by factors like diet (Li et al., 2022) and psychological stress (Marsland et al., 2017) in addition to pathogens, a critical question for the field is to what extent normal circulating levels of cytokines, or downstream markers of them (i.e., C-Reactive Protein), are associated with affiliative motivations and behavior. One context in which to examine potential inflammatory influences on social affiliation motivations and behavior is via social media use.

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1.1. Social affordances of social media

Social media use has become a key aspect of many people's social lives. People regularly use social media to initiate, maintain, and cultivate relationships; they also engage in meaningful conversations or exchange social support on social media. Although people also engage in non-social activities (e.g., reading the news), a substantial portion of social media use is still considered "social" (Rhee et al., 2021) with many of its features geared toward facilitating self-disclosure and social interactions (Ellison et al., 2007; Wang et al., 2012). Indeed, people frequently receive social support and fulfill their social needs through social media (Ellison et al., 2007; Lee et al., 2015). Further, a growing amount of evidence suggests that people may also perceive social media as social agents that can provide psychological comfort (Hunter et al., 2018; Parent and Shapka, 2020). For instance, in one study participants who had access to their smartphone (vs. did not have access) were buffered against negative psychological effects of social exclusion (Hunter et al., 2018). In another study, participants under stress preferred to use their smartphones (to go on social media) over other objects, expecting relief from stress (Melumad et al., 2020). People with high attachment anxiety tend to use social media excessively to seek emotional comfort and fulfill belonging needs (Costanzo et al., 2021), leading some scholars to argue that people may experience attachment-related processes with social media (D'Arienzo et al., 2019; Roberts & David, 2020; see Musetti et al., 2022 for a review).

1.2. Uses and gratifications approach

According to uses and gratifications (U&G) theory (Katz et al., 1973), people actively choose and use certain types of media to fulfill their needs. Thus, if inflammation can increase social affiliation motivation and behaviors (Eisenberger et al., 2017; Muscatell & Inagaki, 2021; Townsend et al., 2020), inflamed individuals may increasingly turn to social media given its potential to help fulfill their social affiliation needs (Ellison et al., 2007; Rhee et al., 2021; Wang et al., 2012). Moreover, if social affiliative motivation is part of what makes inflamed individuals turn to social media, they may be more inclined to use social media to interact with others (vs. to use social media for entertainment, for example).

1.3. The present Research: Is inflammation related to social media use?

Although emerging evidence suggests that inflammation can at times increase social approach behaviors (Inagaki et al., 2015; Muscatell et al., 2016), extant research on humans to date has largely relied on "proxy measures" of social experience in the laboratory (e.g., performance on computer tasks, neural activity, self-report feelings of social connection), leading to calls for studying social behavior itself (e.g., Muscatell, 2021; Muscatell & Inagaki, 2021). Accordingly, the present research investigated how non-manipulated levels of inflammation are associated with a particular social behavior—namely, social media use, which has become an integral part of many people's social relationships and interactions. To this end, we conducted three studies using diverse samples and study designs, and examined whether heightened inflammation would be associated with more social media use. Study 1 involved a large sample of middle-aged adults in a cross-sectional survey to establish the proposed link between systemic inflammation (CRP) and social media use. Building on this, Study 2 recruited a college student sample—the demographic most actively engaged with social media (Statista, 2020). Using a longitudinal design, we examined whether participants' systemic inflammation levels predict their subsequent self-reported social media use. Finally, Study 3 used a weekly survey design to investigate how inflammation is associated with *increased* social media use (objectively measured via the Screen Time application) over time. Additionally, Study 3 explored how systemic inflammation may propel people to use social media differently. The diverse samples and methods in these studies enabled us to test the generalizability and robustness of the proposed inflammation – social media use link.

2. Study 1

2.1. Method

2.1.1. Participants and procedure

Study 1 used data from the Midlife in the United States Refresher study (MIDUS-R; Ryff et al., 2011–2014) and the Midlife in the United States Refresher Biomarker Project (Weinstein et al., 2012–2016). The MIDUS-R recruited a nationally representative sample of 3577 adults who responded to a wide range of questionnaires assessing psychosocial, behavioral, and health factors. We retained 1873 participants who indicated that they had used social media in the past year. Some participants from the MIDUS-R project also participated in the MIDUS Refresher Biomarker Project (N = 863), which focused on assessing key biological factors related to mental and physical health. For this part of the study, participants were admitted to one of three clinical research centers (Georgetown University, University of Wisconsin-Madison, and University of California, Los Angeles (see Weinstein et al., 2012–2016 for study procedure details and IRB approval information) in the afternoon for a 24-hour stay. The blood draw was performed the following morning after fasting overnight (see Weinstein et al., 2012–2016). The final sample for this study was 524 social media users (age range 25–75 years old, $M = 49.69$, $SD = 13.52$; 286 females) who participated in both the Biomarker Project and the MIDUS-R study. Data collection was approved by Institutional Review Boards at Georgetown University, University of Wisconsin-Madison, and University of California, Los Angeles. All participants provided written consent.

2.1.2. Measures

2.1.2.1. CRP. Our predictor variable was CRP ($M = 2.84$, $SD = 5.51$), a biological marker of systemic inflammation (Padfield, et al., 2010; Paine et al., 2013). CRP was measured using two methodologies. First, CRP was measured from plasma using a particle enhanced immunonephelometric assay (BNII nephelometer; Dade Behring Inc., Deerfield, IL). The intra-assay coefficients of variance (CV) ranged from 2.3% to 4.3% and inter-assay CV ranged from 1.1% to 4.4%. The final batch of samples (collected after February 1, 2015; N = 393) as well as those below the limit of detection (0.164 mg/L) using the immunonephelometric assay (N = 27) were assayed from serum using the Meso Scale Diagnostics (MSD) immunoelectrochemiluminescent platform (intra-assay CV: 2.2 to 4.1%; inter-assay CV: 4.7 to 5.2%), which had a lower limit of detection of 0.014 μg/L. The data from the two platforms was integrated using the adjustment formulas described in the study documentation (see Weinstein et al., 2012–2016 for details).

2.1.2.2. Social media use. Participants rated how often they used social media (e.g., Facebook, Twitter, MySpace, etc.) to interact with family members who did not live with them ($M = 3.40$, $SD = 1.70$) and their friends ($M = 3.15$, $SD = 1.72$) in the past year using a 7-point scale (1 = *several times a day*, 7 = *less than once a month*). These two items were reverse-coded and averaged to reflect a composite *social media use* variable, with higher scores indicating more social media usage ($\alpha = 0.63$, $M = 4.73$, $SD = 1.46$).

2.1.2.3. Covariates. Our covariates included several extraneous variables that can influence social media use. Specifically, we included sociodemographic factors (i.e., gender, age, education level, and income) and personality (i.e., the Big Five dimensions; openness, conscientiousness, extraversion, agreeableness, neuroticism) because they can influence social media use (e.g., Correa et al., 2010; Muscanell & Guadagno, 2012). See Supplementary Material for the verbatim wording

and descriptives of the personality measures. Finally, we measured depressive symptoms with the Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977). Extraneous variables known to influence the predictor variable but not the outcome variable (e.g., cigarette smoking, BMI) were not included as covariates in the analyses (Cohen et al., 2017; Permutt, 1990).

2.2. Results

Table 1 presents zero-order correlations among all variables. To test our main hypothesis, we conducted a series of multiple regression analyses with CRP as a predictor of social media use. The models sequentially controlled for an increasing number of covariates to provide greater clarity on how covariates influenced the association between CRP and social media use: no covariates (Model 1), sociodemographic factors (Model 2), personality (Model 3), and depressive symptoms (Model 4). Consistent with our hypothesis, CRP was correlated with more social media use in Model 1 ($\beta = 0.11$, $p = .017$), Model 2 ($\beta = 0.09$, $p = .048$), Model 3 ($\beta = 0.09$, $p = .046$), and Model 4 ($\beta = 0.09$, $p = .041$). These results are summarized in Table 2. No covariates interacted with CRP to predict social media use significantly (all $ps > 0.27$). Additionally, social media use itself did not significantly predict CRP ($ps > 0.39$; see Supplementary Material for details on the analyses).

2.3. Discussion

These results provide initial evidence for a link between systemic inflammation and social media use. Based on recent perspectives, we proposed that people with heightened inflammation may be more motivated to connect with others. Turning to social media in this context makes sense because social media may enable them to connect with others and seek social support. Nevertheless, this study has some limitations. First, the cross-sectional design cannot establish the directionality of the association between CRP and social media use. It is possible that social media use predicts elevated CRP levels (Lee et al., 2022), though this was not the case in this study. Second, although the number of middle-aged social media users is growing (Pew Research Center, 2021), most participants used social media less than once a day (in 2011–2014). Thus, selection bias may have affected the results, so it is unclear whether they would generalize to other populations who use social media more frequently. To address these issues, Study 2 employed a longitudinal design using a college student sample.

3. Study 2

3.1. Method

3.1.1. Participants and procedure

Data collection for Study 2 occurred from August 2018 to October 2018. Two-hundred and twenty-eight undergraduate students participated in this study for partial course credit (136 female; $M_{age} = 18.95$, $SD_{age} = 2.48$). There were three Phases to this longitudinal study: an initial laboratory session (Phase 1; N = 228), an online follow-up survey six weeks later (Phase 2; N = 179), and a follow-up laboratory session approximately one week later (Phase 3; N = 149).

During Phase 1, participants completed questionnaires assessing social media use, sociodemographic information, health behaviors, and medical conditions. Then, we collected participants' blood samples to be assayed for CRP. Six weeks later (Phase 2), we emailed participants an online survey that assessed their social media (i.e., Instagram) use. Finally, participants returned to the laboratory one week later to provide another blood sample (Phase 3). The Institutional Review Board at the Ohio State University (Protocol 2012B0343) approved this study. All participants provided informed consent.

3.1.2. Measures

3.1.2.1. Phases 1 and 3 CRP. We assayed CRP from dried blood spots using a protocol similar to the one in McDade and colleagues (2004). Specifically, we swabbed each participant's finger with alcohol and pricked it with an 18-gauge needle (Owen Mumford Unistick 3; https://www.owenmumford.com/us/). We collected the blood drops on a Whatman 903 Protein Saver Card and left them to dry at room temperature for 24 h. Then, we punched the samples (3 mm Biopsy punch) and stored them in microcentrifuge tubes at −80 °C until they were assayed. To assay, we thawed a single 3 mm punch and added 200 μl of buffer (Phosphate Buffered Saline with 0.1% Tween 20) for overnight incubation at 4 °C while shaking at 60 rpm. We then diluted this eluate 1:10 and assayed CRP the following morning using Meso Scale Delivery Vplex Plus kits [K151STG] for which the reported lower limit of detection is 0.0013 μg/L. All samples were within the linear range of the standard curve (the ratio of the minimum sample for each plate to the lowest standard across all plates averaged 3.27) and therefore all samples were included in the analyses. The intraassay CV was 1.81% and inter-assay CV was 11.73% ($M_{Phase1} = 1.09$ mg/L, $SD_{Phase1} = 2.97$ mg/L; $M_{Phase3} = 1.31$ mg/L, $SD_{Phase3} = 2.21$ mg/L). Because CRP does not fluctuate diurnally, we collected the samples at various times throughout the day (Meier-Ewert et al., 2001; Mills et al., 2009).

3.1.2.2. Phase 2 Instagram use. Participants indicated how much time they spent on Instagram daily on average (1 = *10 min or less*, 2 = *11–30 min*, 3 = *31–60 min*, 4 = *1–2 h*, 5 = *2–3 h or more*; $M = 2.63$, $SD = 1.18$). We chose to focus on Instagram use because our pilot study revealed that Instagram was one of the most popular social media platforms among college students at the time of data collection in 2018 (also see Statista, 2020).

3.1.2.3. Covariates. Following Study 1, our sociodemographic covariates included gender (1 = *male*, 2 = *female*) and age. Covariates such as income, education level, personality traits, and depressive symptoms were not available in this study. For analyses adjusting for other potential confounders (e.g., substance use or immune-related conditions),

Table 1
Zero-order correlations for all variables in Study 1.

Variables	1	2	3	4	5	6	7	8	9	10
1. Social media use	–									
2. CRP	0.11*	–								
3. Age	−0.08†	0.01	–							
4. Gender	0.11*	0.14***	−10*	–						
5. Openness	0.06	0.03	0.01	−0.08†	–					
6. Conscientious	0.09*	0.04	0.05	0.12**	0.28***	–				
7. Extraversion	0.24***	0.02	0.03	0.06	0.40***	0.25***	–			
8. Agreeableness	0.17***	0.06	0.03	0.23***	0.33***	0.27***	0.50***	–		
9. Neuroticism	−0.04	−0.02	−0.17***	0.15***	−0.23***	−0.18***	−0.20***	−0.10*	–	
10. Depress	−0.05	0.12**	−0.16***	0.11*	−0.13**	−0.21***	−0.28***	−0.11*	0.43***	–

Notes. $^{\dagger}p \leq 0.10$ *$p \leq 0.05$. $^{**}p \leq 0.01$. $^{***}p \leq 0.001$ (two-tailed). Gender was coded with 1 (male) and 2 (female). Depress = depressive symptoms.

Table 2
Coefficients from linear regression models predicting social media use in Study 1.

	Model 1		Model 2		Model 3		Model 4	
Predictor	b (*p*)	95% CI	b (*p*)	95% CI	b (*p*)	95% CI	b (*p*)	95% CI
CRP	0.03 (0.017)	[0.01, 0.05]	0.02 (0.048)	[0.01, 0.05]	0.02 (0.046)	[0.01, 0.05]	0.02 (0.04)	[0.01, 0.05]
Gender			0.28 (0.04)	[0.02, 0.54]	0.20 (0.15)	[−0.07, 0.47]	0.20 (0.14)	[−0.07, 0.47]
Age			−0.01 (0.19)	[.−0.02, 0.01]	−0.01 (0.10)	[−0.02, 0.01]	−0.01 (0.09)	[−0.02, 0.01]
Income			−0.01 (0.96)	[0.00, 0.00]	−0.01 (0.75)	[0.00, 0.00]	−0.01 (0.73)	[0.00, 0.00]
Education			−0.12 (0.19)	[−0.29, 0.06]	−0.09 (0.34)	[−0.26, 0.09]	−0.09 (0.33)	[−0.27, 09]
Openness					−0.12 (0.39)	[−0.40, 0.16]	−0.12 (0.41)	[−0.40, 0.16]
Conscientious					0.07 (0.62)	[−0.21, 0.35]	0.06 (0.67)	[−0.22, 0.34]
Extraversion					0.54 (<0.001)	[0.29, 0.79]	0.53 (<0.001)	[0.27, 0.78]
Agreeableness					0.12 (0.42)	[−0.17, 0.40]	0.12 (0.41)	[−0.17, 0.41]
Neuroticism					−0.06 (0.56)	[−0.27, 0.15]	−0.04 (0.71)	[−0.26, 0.18]
Depress							−0.01 (0.61)	[−0.03, 0.02]
R^2		0.01		0.03		0.09		0.09

Notes. Gender was coded with 1 (male) and 2 (female). Conscientious = conscientiousness.

please see Supplementary Material.

3.2. Results

3.2.1. Does CRP at Phase 1 predict Instagram use at Phase 2?

To test our hypothesis, we conducted a series of multiple regression analyses with CRP as a predictor of Instagram use. Our analyses consisted of two models: Model 1 included no covariates and Model 2 included sociodemographic covariates (see Table 3 for the detailed results). Consistent with our hypothesis, CRP at Phase 1 was associated with more Instagram use at Phase 2 (6 weeks later) in Model 1 ($\beta = 0.21$, $p = .011$) and Model 2 ($\beta = 0.19$, $p = .018$). Although females reported higher Instagram use than males ($M_{female} = 2.81$, $SD_{female} = 1.15$, $M_{male} = 2.32$, $SD_{male} = 1.17$; Welch's *t*-test, $t(103.57) = 6.25$, $p = .014$), gender did not moderate the association between CRP and Instagram use ($p > .15$).

3.2.2. Does Instagram use at Phase 2 predict CRP at Phase 3?

To test an alternative explanation that social media use may increase CRP levels, we conducted an exploratory analysis with Instagram use at Phase 2 as a predictor of CRP at Phase 3. Although the time between Phase 2 and 3 (one week) was shorter than the time between Phase 1 and 2, this is still sufficient time to measure a change in CRP (Mehta et al., 2010; Paine et al., 2013). To properly test the possibility of social media use increasing inflammation, three additional analytic steps were taken following prior research (Lee et al., 2022; Lee & Way, 2021). First, one participant whose CRP value was over 10 μg/mL was excluded because this value may reflect the presence of an acute infection rather than heightened systemic inflammation (potentially from Instagram use) (Pearson et al., 2003). Second, we log-transformed CRP values to achieve a normal distribution. Finally, we controlled for extraneous variables that can also influence CRP (i.e., BMI, gender, and age). Because CRP was the dependent variable in this analysis (as opposed to being a predictor variable in prior analyses), we included these additional covariates known to influence CRP levels specifically (Permutt, 1990; Cohen et al., 2017). The results indicated that Instagram use at Phase 2 was not a significant predictor of CRP at Phase 3 ($p = .31$; see Supplementary Material for details). These findings suggest that the association between CRP and social media may be partly due to inflammation enhancing the proclivity to use social media rather than higher Instagram usage leading to heightened inflammation.

Table 3
Coefficients from linear regression models of factors at Phase 1 predicting social media use at Phase 2 in Study 2.

Model 1				Model 2			
Predictor	b	*p*	95% CI	Predictor	b	*p*	95% CI
CRP	0.07	0.01	[0.02, 0.13]	CRP	0.07	0.02	[0.01, 0.12]
				Gender	0.46	0.03	[0.05, 0.86]
				Age	−0.02	0.69	[−0.11, 0.08]
R^2		0.04				0.08	

Notes. Gender was coded with 1 (male) and 2 (female).

3.3. Discussion

Study 2 conceptually replicated and extended findings from Study 1 using prospective data. Specifically, while CRP was positively associated with the amount of social media use six weeks later, social media use was not associated with subsequent levels of CRP. These results provide further evidence that inflammation may contribute to increased social media use.

Nevertheless, there are some limitations of this study. First, because social media use and CRP were never measured at the same time, our ability to make a temporal interpretation regarding the relation between CRP and social media use is limited. Second, Study 2 only collected data on Instagram use despite evidence that most college students use several social media platforms (see Perrin and Anderson, 2019). Third, recent research suggests that retrospective estimates of self-reported social media use may be inaccurate (Burnell et al., 2021; Ernala et al., 2020; Parry et al., 2021; Verbeij et al., 2021). In Study 3 we addressed these issues by conducting a longitudinal study and measured social media use objectively over time across multiple platforms through a screen time application.

4. Study 3

4.1. Method

4.1.1. Participants and procedure

Our data came from a larger project investigating college students' lifestyle and well-being. In this project, one hundred and seventy-one college students (102 females; $M_{age} = 19.24$, $SD_{age} = 2.68$) participated for partial course credit between September 2021 and May 2022. For our purpose, we focus on the longitudinal component of this study, which consisted of two parts: a baseline lab session (Phase 1, N = 171) and two follow-up weekly surveys (Phase 2, N = 160; Phase 3, N = 160). During Phase 1, participants completed several background questionnaires assessing factors such as sociodemographic information,

personality, health behaviors, and medical conditions. Once participants completed the questionnaires, they were escorted to another room to provide their blood samples. Participants were assured that they could opt out of the blood sample collection procedure without losing compensation. Excluded from the analyses were thirteen (7.60%) participants who opted out of the blood samples collection procedure and another 4 participants (2.34%) for whom there was deemed to be an insufficient amount of blood. Therefore, no attempt was made to assay these four samples. Approximately one week after completing Phase 1, participants completed a weekly survey assessing their social media use (Phase 2) and another weekly survey one week later (Phase 3). The Institutional Review Board at the Ohio State University (Protocol 2018H0452) approved this study. All participants provided informed consent.

4.1.2. Measures

4.1.2.1 Weekly social media use (Phases 2 and 3). Using the Screen Time application on their iPhone (the iOS operating system), participants retrieved information on how much time they spent on each of the four social media platforms (i.e., Snapchat, Instagram, Twitter, and Facebook) in the past week.[1] For our main dependent variable, we summed the weekly averages across the four social media platforms mentioned above to create a composite *social media use* variable for each week (M_{Week1} = 560.15 min, SD_{Week1} = 462.16 min; M_{Week2} = 595.48 min, SD_{Week2} = 540.85 min).

We decided to assess social media use across four platforms for three reasons. First, Snapchat, Instagram, Twitter, and Facebook were the most popular social media platforms among college students at the time of our study design (Perrin and Anderson, 2019). Second, recent work advocates collecting social media use data across multiple platforms because most people use multiple platforms in varying amounts (Bayer et al., 2020). Third, this method is consistent with our prior work on social media use (Lee et al., 2022).

4.1.2.2. Types of social media use. Based on prior work (Liu et al., 2019), we asked participants to report the extent to which they used social media for different purposes in the past week using a 5-point scale (1 = *not at all*, 5 = *very much*). To reduce survey fatigue, these questions referred to how participants used social media generally across platforms (vs. for each platform). The categories were: 1) social interactions (i.e., *"By direct communication, we mean exchanging direct/private messages, tagging, reacting and commenting on others' posts on social media sites."*; M_{Week1} = 3.16, SD_{Week1} = 1.21, M_{Week2} = 3.04, SD_{Week2} = 1.14), 2) self-presentation (i.e., *"People can present photos or update their own status across different social media platforms."*; M_{Week1} = 1.80, SD_{Week1} = 0.91, M_{Week2} = 1.76, SD_{Week2} = 0.94), 3) content consumption (i.e., *"By browsing, people can look at others' profiles, pictures, comments, and updates."*; M_{Week1} = 3.43, SD_{Week1} = 1.25, M_{Week2} = 3.39, SD_{Week2} = 1.14), and 4) entertainment (i.e., *"Entertainment involves leisure use of social media to pass time or entertain oneself. For instance, you can play games alone or with friends or watch entertaining videos on social media."*; M_{Week1} = 3.78, SD_{Week1} = 1.14, M_{Week2} = 3.72, SD_{Week2} = 1.13).

4.1.2.3. CRP. CRP was measured during Phase 1 (i.e., Week 1) via dried blood spots using the same method as in Study 2 (McDade et al., 2004). The intraassay CV was 3.8% and the interassay CV was 11.04% (M = 0.99 mg/L, SD = 1.75 mg/L). All samples were successfully assayed and within the linear range (i.e., the ratio of the lowest sample to the lowest standard across all plates was 5.3).

4.1.2.4. Covariates. Following Study 1, we controlled for extraneous factors that can influence social media use. For our sociodemographic covariates, we measured gender, age, family income, and highest level of education completed by mother and father (1 = *some high school*, 5 = *graduate school*). We also controlled for the Big Five Factor personality using the Ten-Item Personality Inventory (Gosling et al., 2003) and depressive symptoms using the CES-D (Radloff, 1977; α = 0.87, M = 2.07, SD = 0.62). For analyses adjusting for other potential confounders (e.g., substance use or immune-related conditions), please see the Supplementary Material.

4.2. Results

Table 4 presents zero-order correlations among all key variables.

4.2.1. Is CRP associated with more social media use in the same week?

To test this hypothesis, we conducted a series of multiple regression analyses with CRP as a predictor of social media use (Week 1). Following Study 1, four models controlled for an increasing number of covariates (1) no covariates (Model 1), (2) socio-demographic factors (Model 2), (3) personality (Model 3), and (4) depressive symptoms (Model 4). Consistent with our hypothesis, CRP levels were positively associated with social media use in the same week in Model 1 (β = 0.21, p =.021), Model 2 (β = 0.21, p =.021), Model 3 (β = 0.20, p =.026), and Model 4 (β = 0.20, p =.032). The results of these analyses are summarized in Table 5.

4.2.2. Does CRP in week 1 predict increased social media use from week 1 to week 2?

To test whether CRP would predict increased social media use from Week 1 to Week 2, we conducted the same regression analyses with CRP as a predictor of social media use in Week 2, controlling for social media use in Week 1. CRP was associated with increased social media use in Model 1 (β = 0.11, p =.047), Model 2 (β = 0.12, p =.04), Model 3 (β = 0.11, p =.044), and Model 4 (β = 0.11, p =.050). The results of these analyses are summarized in Table 6.

4.2.3. Is CRP associated with different types of social media use in the same week?

To explore how CRP is associated with different types of social media use, we conducted multiple regression analyses with CRP as a predictor of each type of social media use (i.e., social interaction, self-presentation, content consumption, entertainment). CRP was positively associated with using social media for social interaction ($0.18 < \beta s < 0.20$, $0.022 < ps < 0.039$ across 4 models). CRP was not significantly associated with any other types of social media use ($ps > 0.51$).

4.2.4. Does CRP in week 1 predict changes in how people use social media from week 1 to week 2?

Following the analyses above, we conducted several regression analyses with CRP as a predictor of each of the types of social media use in Week 2 controlling for the corresponding use type in Week 1. CRP did

[1] We asked participants to report their weekly social media use in the *past* week (vs. current week). This is to ensure that all participants' weekly average use number is based on their use over a full week (seven days). Because the iOS Screen Time application provides a weekly average use data from Sunday to Sunday, the average use data for the current week can be misleading: For example, a participant coming to the laboratory on a Monday would provide a "weekly" average number based on their use from Sunday to Monday; another participant coming to the laboratory on a Friday would provide a "weekly" average number based on their use from Sunday to Friday. Therefore, to ensure that all participants' weekly average use data are based on their use over 7 days, we asked participants to report their use in the *past* week. Because Phase 2 was completed one week *after* Phase 1 (i.e., CRP measurement), the social media use variable measured in Phase 2 corresponds to social media use during the week of Phase 1, when we measured CRP (i.e., denoted as Week 1 from hereon). Similarly, the social media use variable measured in Phase 3 corresponds to social media use during the week of Phase 2, one week after the CRP measurement (i.e., denoted as Week 2 from hereon).

Table 4
Zero-order correlations for key variables in Study 3.

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. SMU (W1)	–														
2. SMU (W2)	0.64***	–													
3. Soc Inter	0.27***	0.20*	–												
4. Self Pres	0.12	0.10	0.21**	–											
5. Consump	0.21*	0.16†	0.07	0.30***	–										
6. Entertain	0.27***	0.06	0.15†	0.29***	0.50***	–									
7. CRP	0.21*	0.28***	0.20*	0.02	0.07	0.05	–								
8. Age	−0.16†	−0.11	−0.15†	0.01	−0.21**	−0.32***	−0.05	–							
9. Gender	0.17*	0.15†	−0.02	0.16*	0.27***	0.10	−0.02	−0.15†	–						
10. Openness	0.07	0.05	0.18*	0.26***	0.10	0.10	−0.01	0.15†	0.02	–					
11. Conscient	0.06	0.07	−0.02	−0.11	−0.13	−0.10	0.01	−0.13†	−0.01	0.08	–				
12. Extraver	0.10	0.18*	0.26***	0.23**	0.02	−0.05	0.05	−0.07	−0.05	0.38***	0.11	–			
13. Agreeable	−0.01	0.05	0.08	0.12	0.01	0.10	−0.15†	−0.01	0.03	0.24**	0.21**	0.16*	–		
14. Neurotic	0.15†	0.15†	0.01	0.11	0.26***	0.14†	0.12	−0.10	0.24**	−0.14†	−0.27***	−0.17*	−0.35***	–	
15. Depress	0.12	0.06	0.02	0.14†	0.10	0.08	0.12	−0.01	0.24**	−0.17*	−0.26***	−0.26***	−0.39***	0.42***	–

Notes. †$p \leq 0.10$ *$p \leq 0.05$. **$p \leq 0.01$. ***$p \leq 0.001$ (two-tailed). SMU (W1) = Week 1 social media use. SMU (W2) = Week 2 social media use. Soc Inter = social media use for social interaction. Self Pres = social media use for self-presentation. Consump = social media use for content consumption. Entertain = social media use for entertainment. Gender was coded with 1 (male) and 2 (female). Depress = depressive symptoms.

not significantly predict changes in how people used social media from Week 1 to Week 2 (*ps* > 0.39).

4.3. Discussion

If inflammation promotes social affiliative motivation, inflamed individuals may use social media more, given its various social affordances. Consistent with this idea, Study 3 replicated the association between CRP and social media use—even when social media use, across multiple platforms, was assessed objectively through the Screen Time application. Critically, Study 3 extended results from our prior studies by showing that CRP in Week 1 predicted *increased* social media use from Week 1 to Week 2, suggesting that inflamed individuals increased their social media use during that period. Moreover, our exploratory analysis showed that CRP was at least concurrently associated with using social media to socially interact with others (e.g., exchanging direct messages) and not with any other types of social media use (e.g., entertainment). Thus, it appears that part of the increased social media use from higher CRP may be socially motivated, although this potential effect did not extend to the following week. Importantly, our main findings cannot be attributed to self-report biases such as social desirability or hypothesis guessing because we measured inflammation via a biological marker and social media use objectively through the Screen Time application.

5. General discussion

The present research examined how systemic inflammation is associated with social media use. Findings from three studies using different samples and designs provided initial evidence that CRP, a biological marker of systemic inflammation, is positively associated with social media use. The pattern of results remained the same even after adjusting for various factors known to influence social media use such as gender, personality, and depressive symptoms.

Our findings make several novel contributions. To our knowledge, the present research is the first to highlight a potential biopsychosocial antecedent to social media use. Drawing on recent evidence that inflammation may enhance social affiliation motivation and behaviors (Eisenberger et al., 2017; Muscatell & Inagaki, 2021; Townsend et al., 2020), we posited that social media use would be especially appealing to individuals with heightened inflammation, given the possibility that social media use can fulfill their social affiliation motivations. The present results provide initial support to this hypothesis. Critically, these results extend the affiliative effects of inflammation beyond experimentally raised levels of inflammation in prior human work (Inagaki et al., 2015; Jolink et al., 2022; Reiber et al., 2010) to endogenously elevated levels of inflammation in the present work. An important task for future research will be to identify the underlying psychological drivers of this affiliative motivation. For example, experimental increases in inflammation using LPS injection can induce feelings of social disconnection (Eisenberger et al., 2010) and the desire to be alone (Hannestad et al., 2011). Thus, it will be important to build on our findings to determine the degree to which inflammation-induced increases in social media use and social media use for social interaction are driven by the desire for nurturance from a support figure (e.g., Inagaki et al., 2015), amelioration of feelings of loneliness and social disconnection (Tomova et al., 2020), or other motivations arising from social isolation. To this end, future research could examine how inflammation is associated with various goals and types of social media use (e.g., active vs. passive use, seeking social support; e.g., (Verduyn et al., 2015)).

Another important question for the nascent literature studying the effects of inflammation on human social motivation and behavior is the degree to which levels of inflammation influence their expression (Hennessy et al., 2014). Standard laboratory doses of LPS induce high levels of inflammation (i.e., a 1 to 2 order of magnitude increase; Eisenberger et al., 2010), which has resulted in robust increases on a

Table 5
Coefficients from linear regression models predicting Week 1 social media use in Study 3.

	Model 1		Model 2		Model 3		Model 4	
Predictor	b (*p*)	95% CI	b (*p*)	95% CI	b (*p*)	95% CI	b (*p*)	95% CI
CRP	59.87 (0.021)	[9.00, 11.075]	60.21 (0.021)	[9.29, 111.12]	57.94 (0.026)	[7.06, 108.82]	56.76 (0.032)	[4.91, 108.62]
Gender			161.27 (0.07)	[−12.72, 335.25]	140.80 (0.12)	[−36.00, 317.59]	135.98 (0.14)	[−45.21, 317.16]
Age			−31.57 (0.06)	[63.91, 0.78]	−26.83 (0.12)	[−60.95, 7.30]	−26.33 (0.13)	[−60.80, 8.14]
Edu (M)			67.33 (0.16)	[−26.01, 160.67]	60.28 (0.22)	[−35.60, 156.15]	60.55 (0.22)	[−35.75, 156.85]
Edu (F)			4.16 (0.92)	[−76.19, 84.51]	19.99 (0.64)	[−65.30, 105.28]	19.68 (0.65)	[−55.00, 105.37]
Income			−6.35 (0.73)	[−43.09, 30.40]	−9.12 (0.64)	[−48.11, 29.87]	−8.86 (0.66)	[−48.07, 30.35]
Openness					71.50 (0.22)	[−43.64, 186.64]	71.86 (0.22)	[−43.80, 187.52]
Conscientious					33.42 (0.53)	[−72.51, 139.36]	36.07 (0.51)	[−82.15, 144.29]
Extraversion					38.93 (0.30)	[−35.11, 112.97]	40.59 (0.29)	[−34.79, 115.96]
Agreeableness					44.98 (0.51)	[−90.54, 180.51]	50.49 (0.48)	[−91.73, 192.70]
Neuroticism					84.56 (0.07)	[−8.38, 177.51]	82.68 (0.09)	[−11.72, 177.08]
Depress							23.69 (0.79)	[−153.83, 201.22]
R^2		0.04		0.11		0.16		0.16

Notes. Gender was coded with 1 (male) and 2 (female). Edu (M) = highest level of school completed by mother. Edu (F) = highest level of school completed by father. Income = household income. Conscientious = conscientiousness. Depress = depressive symptoms.

Table 6
Coefficients from linear regression models predicting Week 2 social media use in Study 3.

	Model 1		Model 2		Model 3		Model 4	
Predictor	b (*p*)	95% CI	b (*p*)	95% CI	b (*p*)	95% CI	b (*p*)	95% CI
CRP	29.74 (0.047)	[0.38, 59.10]	31.68 (0.04)	[1.51, 61.85]	30.76 (0.044)	[0.84, 60.67]	30.18 (0.05)	[0.04, 60.33]
SMU (Wk1)	0.75 (<0.001)	[0.65, 0.85]	0.76 (<0.001)	[0.66, 0.87]	0.73 (<0.001)	[0.62, 0.84]	0.73 (<0.001)	[0.62, 0.84]
Gender			−4.06 (0.94)	[−106.54, 98.43]	−0.43 (0.99)	[−103.59, 102.73]	−5.77 (0.92)	[−112.15, 100.62]
Age			5.56 (0.56)	[−13.48, 24.60]	12.63 (0.21)	[−7.22, 32.47]	12.85 (0.20)	[−7.10, 32.80]
Edu (M)			−26.26 (0.35)	[−81.30, 28.78]	−34.19 (0.23)	[−90.39, 22.01]	−32.48 (0.26)	[−89.43, 24.48]
Edu (F)			29.84 (0.22)	[−17.84, 77.53]	48.12 (0.06)	[−1.84, 97.98]	46.11 (0.08)	[−4.78, 96.99]
Income			−8.11 (0.46)	[−29.81, 13.60]	−15.95 (0.17)	[−38.71, 6.82]	−15.58 (0.18)	[−38.49, 7.34]
Openness					−8.90 (0.79)	[−75.67, 57.87]	−7.17 (0.83)	[−74.66, 60.32]
Conscientious					46.40 (0.14)	[−14.77, 107.58]	47.73 (0.13)	[−13.98, 109.44]
Extraversion					50.29 (0.02)	[7.14, 93.45]	51.14 (0.02)	[7.65, 94.64]
Agreeableness					11.91 (0.77)	[−67.12, 90.95]	16.02 (0.70)	[−65.50, 97.54]
Neuroticism					52.27 (0.06)	[−2.97, 107.50]	49.28 (0.09)	[−7.82, 106.38]
Depress							21.34 (0.67)	[−76.02, 118.70]
R^2		0.67		0.68		0.70		0.70

Notes. Gender was coded with 1 (male) and 2 (female). SMU (Wk1) = Week 1 social media use. Edu (M) = highest level of school completed by mother. Edu (F) = highest level of school completed by father. Income = household income. Conscientious = conscientiousness. Depress = depressive symptoms.

measure of social disconnection (Eisenberger et al., 2010), particularly among women (Moieni et al., 2015). In contrast, a flu vaccination had weaker effects on the same scale (Kuhlman et al., 2018) or no effects on a scale assessing motivation to foster social connections (Jolink et al., 2022). Likewise, typhoid vaccination did not lead to changes on a scale of social connectedness, assessing the degree to which one felt loved, appreciated, cared for, and hurt (Madison et al., 2023). The elevation in levels of IL-6 after flu vaccination (Kuhlman, et al., 2018; Jolink et al., 2022; Madison et al., 2023) are akin to those seen in chronic, low-grade inflammation and thus a question for future research is whether stress-induced sickness behavior (Hennessy et al., 2014) may be qualitatively different from that induced by an infection (or the mimicking of a host's response to an infection by LPS).

A critical determinant of whether inflammation elicits increased social approach or withdrawal appears to be the context. The self-reported increases in the desire to be alone after LPS (e.g., Hannestad et al., 2011) are akin to findings in the animal literature of pathogen-induced social distancing in order to protect other members of the social group from infection (Stockmaier et al., 2021; Townsend et al., 2020). As social organisms that benefit from social interaction, humans must balance the benefits of social interaction with the costs associated with both the increased risk of pathogen transmission as well as the costs of social isolation. Thus, the social effects of inflammation appear to be context dependent (Hennessy et al., 2014; Lopes, 2014), influenced by the nature of the relationship to the other person, genetic relatedness, energy level, and pathogen virulence (Stockmaier et al., 2021). One contextual factor that has been identified in human studies appears to be the closeness of the relationship (Muscatell & Inagaki, 2021). Experimentally-induced increases in inflammation have led to increases in the desire to approach close others (Inagaki et al., 2015), as opposed to the more commonly seen proclivity to socially distance. An analogue of this effect may occur outside the laboratory as well. Elevations in inflammation due to stress have led to a focusing of one's time on important social relationships over a year (Lindsay et al., 2022). Relatedly, in a nonhuman primate, kinship influences the degree of social interaction between conspecifics, with close kin not changing their social interaction (i.e., grooming) with an infected conspecific while more distant others reduce their social interaction with the infected conspecific (Poirotte & Charpentier, 2020). In this vein, social media use may be a particularly unique form of social interaction because there is lower pathogen exposure risk relative to face-to-face, in-person communication. Therefore, this lower pathogen risk may moderate or alter the affiliative effects of inflammation in comparison with the face-to-face social context.

Future research may benefit from using additional markers of inflammation that elucidate the upstream pathways that lead to increased levels of CRP (Sproston & Ashworth, 2018). For example, a cytokine (IFN-γ) that is particularly involved in defense against viral infection (Cole, 2019) is upregulated in both mammals and insects in social contexts (Filiano et al., 2016) presumably to defend against the increased viral transmission resulting from social interactions. Individual differences in inflammatory profile, such as levels of IFN- γ or other

cytokines, may be related to different social media use patterns because of differential effects on social affiliation motives.

There are some limitations of the present research. First, the present research included three studies that vary in their methodologies and samples, which can make comparing the results across the studies difficult. For instance, Study 1 participants were middle-aged adults who tend to have higher baseline systemic inflammation levels (Franceschi et al., 2018) and different social media usage patterns from college students (Studies 2 and 3). Ideally, a future study may recruit participants across different age groups and examine whether the observed patterns between CRP and social media use would differ by different age groups. Although the conceptual replications across multiple studies (i.e., the consistent pattern between CRP and different measures of social media use) using diverse methods and samples may provide stronger evidence that the constructs of interests are responsible for the findings than findings from a single study (Fabrigar & Wegener, 2016), future research should seek to replicate these results using more consistent methodologies. Second, the correlational findings in the three studies limit our ability to make causal inferences about the relation between inflammation and social media use. To be clear, we do not claim or seek to rule out the possibility that social media use may influence inflammation (Afifi et al., 2018; Lee et al., 2022; Lee & Way, 2021). Rather, we encourage more researchers to consider both possibilities. Here, we used longitudinal design to show that the proposed link between CRP and social media use goes beyond a mere cross-sectional correlation: CRP was prospectively associated not only with more social media use (Study 2) but also with increased social media use from week to week (Study 3). Nevertheless, future research could provide stronger causal evidence by conducting a study that experimentally manipulates inflammation (e.g., typhoid vaccination or LPS administration). Third, the current study mostly focused on a general association between inflammation and amount of social media use. While this broad metric of overall amount of social media use allowed us to link inflammation with a quantifiable, observable measure of (online) social behavior, it is unlikely to reflect the complexity of social media use in daily life. Although this limitation motivated us to measure different types of social media use in Study 3, future research should more comprehensively examine how CRP and other inflammatory markers may be associated with different aspects of social media use. Finally, given that the effects of inflammation on social behavior may vary depending on individual differences (Moieni et al., 2015; Eisenberger et al., 2010) and that social media effects are also heterogenous (e.g., Meier & Johnson, 2022; Valkenburg, 2022), the CRP – social media use link may be different for specific populations (e.g., teenaged girls, individuals with low self-esteem). Thus, future research should seek to examine potential moderators of our findings.

Broadly, our findings highlight the potential benefits of studying social behaviors on social media in understanding the social effects of inflammation. Several scholars have noted that extant research on social effects of inflammation lacks data on actual social behaviors (see Muscatell and Inagaki, 2021). Social media data collected through passive sensing technology can provide *observable* and *quantifiable* information on different types of social behaviors. For instance, in addition to the amount of time spent on social media, researchers can unobtrusively collect data on whom people interacted with on social media or content-analyze the types of posts people shared on social media during heightened inflammatory periods. Although these social behaviors may not be the same as social behaviors in-person, it would be critical to understand how social media use fits into the study of social effects of inflammation, considering the widespread use of social media as a social and communication technology in the current digital age. Moreover, some features that distinguish online social behaviors from offline social behaviors (e.g., differential risk of spreading pathogens) may help provide insight into further understanding the social behavioral effects of inflammation (e.g., withdrawal to recuperate vs. avoid infecting others). To this end, it would be worthwhile for future studies to examine the relations among inflammation, direct social interactions, and social media use to better understand the nuanced social effects of inflammation. For instance, will the social affiliative effect of inflammation promote the frequency of social interactions both on social media and offline? Or, will it lead to more social interactions on social media but not for offline social interactions? If so, is it because social media use simply reflects a substitution of face-to-face interactions with online ones (i.e., displacement hypothesis; e.g., Kraut et al., 1998)? Or, does time spent on social media lead to more in-person social interactions by increasing social contact and maintenance of relationships (i.e., stimulation hypothesis; e.g., Valkenburg & Peter, 2007)? Future research should seek to address these questions.

Finally, our findings also contribute to extant research on the *antecedents of* social media use. To date, a disproportionate amount of research has focused on the consequences of social media use on well-being. However, given the correlational nature of most prior studies, we argue that it is important to also consider the possibility that lower well-being can lead to more social media use (e.g., Aalbers et al., 2019; Griffioen et al., 2021; Kross et al., 2013). Because social stress can heighten inflammation (Eisenberger et al., 2017) and inflammation may also heighten social media use, there could potentially be a positive feedback loop between social media use and inflammation that has compounding effects on well-being. Moreover, understanding and identifying what leads people to use social media more (vs. less) seems critical given the potential negative impact of social media use, especially among teenaged girls (Orben et al., 2022; Twenge and Martin, 2020). This can inform future intervention strategies aimed at fulfilling the needs of those who turn to social media—directly through teaching people how to use social media to address their needs (e.g., information seeking for problem-focused coping, entertainment use for emotion-focused coping; Nabi et al., 2021; see Wolfers & Schneider, 2021 for a review on using media for coping) or indirectly through other means (e.g., strengthen offline social relationships to combat loneliness).

6. Conclusion

The present study found that systemic inflammation is associated with more social media use among middle-aged adults and college students. The study of inflammation and social behaviors on social media presents an intriguing opportunity to understand the social effects of inflammation in daily life.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

Data will be made available on request.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.bbi.2023.05.010.

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