scientific reports

Check for updates

OPEN

Inflammation is associated with greater social media use over face-to-face interaction, especially among individuals high in introversion or neuroticism

David S. Lee[1]✉, Tao Jiang[2] & Baldwin M. Way[3,4]

Emerging research suggests that whether inflammation promotes social approach or social avoidance behavior may depend on the context. However, little is known about what such contexts are. Addressing this gap, the present research examined how inflammation is associated with two common daily social behaviors varying in interaction modality. Building on work showing inflammation's role in psychological states such as fatigue and vigilance toward physical and psychological threats and research on social media's role as an energy efficient, mediated communication tool, we hypothesized that inflammation would be associated with more social media use (SMU) over face-to-face interactions (FtF) and with more social media use for social interaction (SMUSI) over FtF. To test our hypotheses, we recruited college students who provided their blood samples to be assayed for C-reactive protein (CRP), a biomarker of systemic inflammation, and completed questionnaires assessing personality, SMU, SMUSI, FtF, and other measures. Extending prior work, CRP was associated with SMU over FtF and SMUSI over FtF. Importantly, these patterns were stronger among individuals with higher introversion and individuals with higher neuroticism. These results provide initial evidence that naturally occurring inflammation may be linked to a stronger preference for a particular social behavior (SMU) over another (FtF) and suggest that such tendency may vary by people with different personality traits. Broadly, the present research contributes to the burgeoning research on inflammation and social behavior and highlights for whom and when social media may be used to fulfill affiliative needs.

Keywords Inflammation, Social media use, Social behavior, Personality, Social compensation

Inflammation is a part of the immune system that evolved to combat infections and heal from injuries. When the body is infected or injured, the immune system triggers inflammatory responses to prevent further spread of the infection (e.g., swelling at the infection site). In addition to these events at the infection site, inflammatory signals also travel to the brain, triggering a cascade of psychological processes in response. Notably, inflammation is now considered to play an important role in how people think, feel, and behave[1,2]; with effects that include altering mood[3], person perception[4], feelings of social connection[5], social judgment[6], and sensitivity to reward[7].

One key research area has focused on how inflammation affects social behavior[1,2,8]. When the immune system is triggered, it releases pro-inflammatory signaling molecules that can result in "sickness behaviors" such as fatigue, anhedonia, and weakness[1,8]. From an evolutionary perspective, these behavioral tendencies not only help the body recuperate, but also protect the individual from further harms and infecting others. Consistent with this idea, initial research on animals showed that inflammation promotes social avoidance[1,8,9]. Similarly, studies that experimentally induced inflammation in humans showed that inflammation led to greater feelings of social disconnection[5], as well as greater neural activity in response to socially threatening images[4] and

[1]University at Buffalo, The State University of New York, 327 Baldy Hall, Buffalo, NY 14228, USA. [2]University of California, Irvine, 4201 Social and Behavioral Sciences Gateway, Irvine, CA 92697, USA. [3]The Ohio State University, 100G Lazenby Hall, Columbus, OH 43201, USA. [4]Institute for Brain, Behavior, and Immunology, Columbus, OH, USA. ✉email: dslee9@buffalo.edu

receiving negative social feedback[10]. These studies contributed to the initial belief that inflammation promotes social avoidance, as part of a hallmark sickness behavior[1,8].

However, recent work has begun to suggest that the effects of inflammation on social behavior may be more nuanced than uniform social avoidance[2,10–13]. Specifically, inflammation at times can increase social *approach* behaviors and neural sensitivity to positive social stimuli[10,11]. For instance, compared with those who received a placebo, people who were injected with a compound that triggers an inflammatory response reported a stronger desire to be around a close other and showed stronger neural response to pictures of close others in a motivation-related brain region[11]. In another study using similar methods, inflammation was associated with a greater willingness to receive social support from a caring individual (e.g., nurse) relative to non-caring individuals[14]. Moreover, flu vaccination, which acutely raises inflammation levels, led people to approach a support figure faster on a laboratory approach/avoidance task[12]. These findings are consistent with the idea that approaching others who can provide social support and care may be adaptive[15,16], especially when in a potentially vulnerable state (i.e., inflammation). They also indicate that inflammation can, in fact, *increase* social approach behaviors under certain contexts—for example, when the context presents an opportunity to interact with close others or receive social support. Thus, one key question surrounding the social effects of inflammation is understanding the various conditions under which inflammation facilitates or undermines affiliative behavior.

Building on prior work on social effects of inflammation[1,2,13] and inspired by recent findings highlighting that different contexts (e.g., whom people are interacting with, what their needs are) can modulate how inflammation affects social behavior[12,16], the goal of the present research was to examine how inflammation is associated with two common—yet varying in contexts—daily social behaviors, namely, interacting with others via face-to-face (FtF) and social media use (SMU). Specifically, we test the novel idea that inflammation would be associated with more SMU over FtF. Extending this, we also test whether the relation between inflammation and social behavior would vary across individuals with different personality traits.

Social affordances of social media

Although social media platforms have diverse features and are constantly evolving, they are largely perceived as "social" platforms[17–19]. For instance, common lay definitions of social media often emphasize how social media "provide new opportunities for finding, observing, and interacting efficiently with others across time and space[20]." Indeed, key features shared across social media platforms, such as removal of geographical/temporal barriers and ease of disseminating self-relevant information, help facilitate self-disclosure and social interactions, which are key to maintaining and strengthening interpersonal relationships[21–23]. Moreover, the mediated (and sometimes asynchronous) nature of communication on social media enables people to strategically present themselves or be more deliberate in communicating their needs[24]. As a result, people can receive social support and fulfill their social needs through social media[25,26]. Unsurprisingly, social media platforms are often perceived and treated by users as social, relational, and communication tools[19,20,26]. This seems to be the case despite the fact that individuals spend much of their time on social media without directly communicating with others[27,28].

Taking this notion further, some scholars contend that fulfilling affiliative needs is a robust, central motive propelling individuals to use social media[17,19,29]. For instance, one study showed that social interaction was one of the primary motives for using Instagram[30]. Similarly, another study discovered that one of the main reasons for using Instagram was related to social purposes (e.g., relationship maintenance), in particular surveillance and gathering knowledge about others[31]. Other researchers have identified seeking new relationships and cultivating existing relationships as two primary motives for Facebook use[32,33]. Collectively, these studies suggest that SMU is an appealing option for fulfilling affiliative needs (e.g., relationship maintenance, exchanging social support).

Inflammation and SMU

While SMU and FtF can both fulfill affiliative needs, we propose that individuals experiencing higher levels of inflammation may be more likely to rely on SMU over FtF. First, inflammation increases fatigue, as demonstrated by experimental studies in which inflammation is raised through either vaccination[34] or administration of a bacteria-mimicking compound[35]. Thus, individuals experiencing higher levels of inflammation may be more motivated to fulfill their affiliative needs in energy-efficient ways. Prior work has shown that compared with other types of social interactions (e.g., FtF, voice calls), social interactions on social media require less energy[36]. Thus, SMU may be an appealing choice for individuals with heightened inflammation to meet their affiliative needs. This notion is consistent with the perspective from the Communicate Bond Belong theory[37], which posits that conserving one's energy is a major factor in people's decisions about *how* to interact with others (i.e., interaction mode).

Second, individuals experiencing higher levels of inflammation (even when symptoms are mild and not explicitly recognizable) are likely to be motivated to protect themselves from potential physical threats (e.g., pathogen) and psychological threats (e.g., social exclusion). Indeed, inflammation has traditionally been thought to trigger sickness behaviors such as fatigue or weakness, in part to protect the body against potential infection risks[1,8]. Similarly, inflammation can activate the behavioral immune system[38], a motivational system that increases vigilance toward potential pathogen cues and promotes avoidance-oriented behaviors[39]. When the behavioral immune system is activated, people become more vigilant against cues of possible contamination and more motivated to engage in behaviors that minimize exposure to others[40]. For example, people higher in disease concern were more likely to classify ambiguous, unfamiliar targets as threatening[6,38,41] and showed higher endorsement of engaging in social distancing behaviors[42]. Thus, individuals experiencing higher inflammation may find the mediated nature of interacting with others on social media more appealing because it allows them to fulfill their affiliative needs while minimizing the likelihood of encountering pathogen cues. Furthermore, recent research has begun to show that inflammation may also motivate people to be protective against psychological threats such as negative feedback or social rejection[10,43,44]. If so, individuals experiencing

higher levels of inflammation should prefer to fulfill their affiliative needs on social media not only because the social media environment is perceived to have fewer explicit rejection cues compared with FtF[45,46] but it allows them to curate messages and have more control over with whom and how to communicate[17,47,48]. Thus, inflammation should lead to a stronger preference for SMU *to* FtF.

In an initial support to this idea, one study showed that C-reactive protein (CRP), a biomarker of systemic inflammation in the blood, predicted more SMU and using social media to interact with others (e.g., sending direct messages)[49]. However, because this prior work did not examine how inflammation is associated with other modes of social interaction (e.g., FtF), it remains unclear whether social media serves as a unique context that enables individuals with higher levels of inflammation to fulfill affiliative needs. Critically, we sought to refine and extend prior work by testing whether inflammation would predict more SMU over FtF. To provide additional supporting evidence, we also examine whether inflammation would be associated with using social media for social interaction (SMUSI) over FtF.

Social behaviors of individuals high in introversion or neuroticism under inflammation

A growing body of evidence suggests that the motivation for using social media may differ based on the individual's personality traits and goals[31,50]. Consistent with this idea, the link between inflammation and SMU over FtF may be stronger for individuals higher in introversion or neuroticism.

According to the Five Factor Model[51], introversion is a trait characterized by preference for low stimulation environments and focus on one's internal thoughts and feelings. Compared with extroverts who are sociable and outgoing, introverts tend to be quiet and reserved[52]. Importantly, a key distinction between introverts and extroverts is how they derive energy from social interactions[53]: In general, whereas extroverts are energized by most social interactions, introverts often find them depleting. For example, introverts report having to exert more energy during FtF and needing time alone to recuperate after[54,55]. They prefer interacting with a smaller (vs. larger) group of people and maintain smaller social networks[55–57], presumably because this helps them conserve energy. In contrast, extroverts not only engage in more frequent FtF[58] but find them to be more rewarding[59]. Because they are more socially skilled and comfortable interacting with others[60,61], extroverts find FtF energizing rather than energy depleting[62,63].

The above findings suggest that introverts and extroverts may differ in how they typically fulfill their affiliative needs. However, we further propose that their difference will be more pronounced under higher levels of inflammation. Specifically, introverts who usually find FtF energy depleting may find SMU and SMUSI as an appealing option to fulfill their affiliative needs. Critically, this preference should be greater with higher levels of inflammation, which is associated with more fatigue[64]. In contrast, because extroverts do not find interacting with others—whether on social media or FtF—to be energy depleting, they may not show a strong preference between SMU/SMUSI and FtF even when experiencing higher levels of inflammation.

Neuroticism is a personality trait characterized by emotional instability[51]. People high in trait neuroticism often have high anxiety and difficulties in FtF[65,66]. They report more negative FtF with others[67,68] and experience lower relationship satisfaction[69]. According to the social compensation hypothesis[70–72], individuals who lack high social resources or skills offline seek to compensate by investing their resources into online relationships (e.g., social media). Importantly, this tendency may be more pronounced among people high on internalizing symptoms such as neuroticism or rejection sensitivity[73,74], in part because online environments contain fewer explicit rejection cues[45,46].

Given that inflammation motivates people to avoid situations that could be psychologically threatening[10,43], these findings suggest that individuals with varying levels of neuroticism may also differ in how they fulfill affiliative needs under higher levels of inflammation. Specifically, individuals high in neuroticism may find SMU and SMUSI more appealing than FtF because the social media environment may have fewer rejection cues and allow them to engage in selective self-presentation[75]. Importantly, this preference should be stronger with higher levels of inflammation, which can make people more vigilant for physical or psychological threats[43,44]. In contrast, for individuals low in neuroticism, such affordances may not be as salient of a factor in *how* they fulfill their affiliative needs. Thus, they may not show a strong preference between SMU/SMUSI and FtF even when experiencing higher levels of inflammation.

Present research

Despite recent advances in understanding the social effects of inflammation, important gaps remain. First, although a growing amount of evidence suggests that the impact of inflammation on social behavior is context-dependent (e.g., interacting with a close other vs. a stranger), little is known about additional conditions under which inflammation promotes social approach or avoidance behavior. Building on prior work[49], we propose interaction modality (e.g., face-to-face, social media) as one understudied context that can potentially modulate the link between inflammation and social behavior. Specifically, we argue that social media is an especially appealing environment to fulfill affiliative needs for people experiencing higher levels of inflammation.

Second, most studies to date have examined proxy measures of social experience in the laboratory rather than actual daily social behaviors[13]. For example, prior studies have measured performance on computer-based social behavior tasks[12], self-reported desire to approach a close other[11], feelings of social disconnection[5], or neural response to social stimuli[10]. Related, these studies have used experimental manipulation of inflammation (e.g., flu vaccination, endotoxin administration), which provides strong causal evidence. However, less is known about how naturally occurring, systemic, inflammation relates to daily social behaviors. This gap is notable because inflammation can be affected by common daily experiences such as diet, sleep, exercise, or stress[76,77], and emerging perspectives suggest that elevations in inflammation can impact daily life more broadly than previously recognized[78]. Thus, to address this gap, the present research examined how inflammation relates to two common daily social behaviors: social media use (SMU) and face-to-face interaction (FtF). Examining

SMU and FtF together allows us to control, to some extent, for general social motivation within individuals and provides more information about one's daily social behaviors (see an approach championing assessing multiple modes of interaction in a study[79,80]). Moreover, the focus on SMU and FtF helps isolate daily social behaviors from broader social desire or subjective feelings of social connection, thereby extending prior work on the social effects of inflammation.

The present research had two primary goals. First, we tested whether people experiencing higher levels of inflammation would engage in more social media (SMU) over face-to-face interaction (FtF). Based on prior work indicating how inflammation can enhance social affiliative motivation under some contexts[10,11] and our theoretical framework linking inflammation to psychological states such as fatigue and vigilance against physical and psychological threats, we hypothesized that inflammation would be associated with more objective SMU over FtF (H1a). Following prior work[81,82], we defined SMU as time spent on social media, objectively recorded via participants' smartphone. However, given that people can use social media for purposes other than social interaction (e.g., reading the news), we also examined whether inflammation would be associated with more self-reported social media use for social interaction (SMUSI) over FtF specifically. We defined SMUSI as amount of social media use for social interaction (e.g., directly communicating with others through functions such as direct messaging), based on relevant prior work[27,83,84]. Because measuring SMUSI objectively was not available to our knowledge, we assessed it via self-report, consistent with prior work[83]. By the same logic as in H1a, we predicted that inflammation wouldbe associated with more SMUSI over FtF (H1b). Second, we explored whether these associations would vary across individuals with different personality traits—namely, introversion and neuroticism. Given that people high in introversion find FtF more energy-depleting and their tendency to prefer lower stimulation environments, we hypothesized that introversion would moderate the relation between inflammation and objective SMU over FtF (H2a), as well as self-reported SMUSI over FtF (H2b). Specifically, we expected the relations to be stronger among individuals higher in introversion. Finally, because people high in neuroticism tend to be more sensitive to psychological threat cues and thus may prefer environments with fewer explicit rejection cues (i.e., social media vs. FtF), we predicted that neuroticism would also moderate the links between inflammation and objective SMU over FtF (H3a) and self-reported SMUSI over FtF (H3b). Specifically, we expected the relations to be stronger among individuals higher in neuroticism.

Method

Participants and procedure

One hundred and seventy-one college students (102 females; $M_{age} = 19.24$, $SD_{age} = 2.68$) from the Ohio State University participated in this study for partial course credit in their Introductory Psychology course. Participants first completed background questionnaires assessing factors such as sociodemographic information, personality, social media use, face-to-face interactions, and various social and health behaviors on Qualtrics in the laboratory. Once participants completed the questionnaires, they were taken to another room where a trained research assistant collected their blood samples. To collect blood, each participant's finger was swabbed with alcohol and pricked with an 18-gauge lancet, following prior work[85]. We then collected the blood drops on a protein saver card. Participants were assured that they could opt out of the blood collection without losing compensation or dropping out of the study. Fourteen participants (8.19%) opted out of blood sample collection, and 3 participants (1.75%) provided samples that were judged to have an insufficient amount of blood for assay. Thus, we did not assay the blood from the latter three participants. Because inflammation (from the blood) data from these participants were not available, we excluded these 17 participants from all analyses. Thus, the final sample size for this study was 154 (93 females; $M_{age} = 19.28$, $SD_{age} = 2.81$).

This study and its research protocol were approved by the Institutional Review Board at The Ohio State University (Protocol 2018H0452). All procedures were performed in accordance with relevant guidelines and regulations, including the Declaration of Helsinki. Informed consent was obtained from all participants prior to their participation.

Measures

Objective social media use (SMU)

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 five social media platforms (i.e., TikTok, Snapchat, Instagram, Twitter/X, and Facebook) during the week for which they visited our laboratory to provide their blood samples. Because the Screen Time app records SMU for the entire week from one Sunday to the next Sunday, we contacted participants during the week immediately following their blood collection so that they can provide their SMU for the entire week that overlapped with when the blood was collected. We assessed SMU across these five platforms because they are considered to be the most popular social media platforms among college students[86]. This approach is also consistent with recent recommendation to collect social media use data across multiple platforms[20,87]. We summed the weekly averages across the five social media platforms to create a composite weekly *social media use (SMU)* variable ($M = 870.00$ min, $SD = 653.01$ min). At the time of our data collection, objective SMU information was not reliably accessible for non-iPhone users, making it difficult to create consistent and reliable research materials for non-iPhone users. Thus, we decided to only collect SMU data from iPhone users. Therefore, SMU data were not available from 17 non-iPhone users.

Social media use for social interaction (SMUSI)

Participants indicated the extent to which they used social media (i.e., TikTok, Snapchat, Instagram, Twitter/X, and Facebook, and other social media combined in general) for social interactions and direct communication during the week. We defined for them social interactions as "exchanging messages, tagging, reacting and commenting on others' posts on social media sites," and direct communication as "sharing pictures/videos

and updates, reacting and commenting on others' page, or exchanging direct/private messages." These items were based on relevant prior work[27,83,84]. Participants used a 5-point scale (1 = *not at all*, 5 = *very much*) to indicate their response to the two items ($\alpha = 0.91$, $M = 3.18$, $SD = 1.17$). Just as with the SMU measure, to obtain SMUSI for the week of blood collection, we contacted participants the week immediately following their blood collection so that they can provide their SMUSI that overlapped with when they provided their blood samples. Because SMUSI measures were obtained via self-report, all participants (including non-iPhone users) were able to provide their data (n = 154).

Face-to-face social interactions (FtF)

Participants indicated the extent to which they engaged in a variety of face-to-face social interactions during the week using a 5-point scale (1 = *not at all*, 5 = *very often/all the time*). To capture a variety of in-person social interaction settings, we slightly modified a measure from prior work[88] to better fit our college student sample contexts. Our modified activities were: "get together with friends to hang out in-person, go to a party or other social events (e.g., concerts, sports events) with friends in-person, watch a movie or TV show with friends in-person, have a meal with friends or family together in-person, have a deep face-to-face conversation with someone in-person, volunteer to help others in-person, and attend religious services in-person" ($\alpha = 0.80$, $M = 2.63$, $SD = 0.81$). This FtF measure was negatively associated with introversion in our study, which is consistent with prior work[58]. Similar to SMU and SMUSI, the FtF measure was obtained during the week immediately following their blood collection.

CRP

We assayed CRP from dried blood spots based on prior work[85]. Specifically, we swabbed each participant's 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 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]. All samples were within the linear range of the standard curve and therefore they were included in the analyses. The intraassay CV was 3.8% and the interassay CV was 11.04% ($M = 0.99$ mg/L, $SD = 1.75$ mg/L).

Introversion and neuroticism

Participants indicated the extent to which various pairs of traits apply to them from the Ten-Item Personality Inventory[89] using a 5-point scale (1 = *strongly disagree*, 5 = *strongly agree*). Introversion was measured with "extraverted, enthusiastic" (reverse-coded) and "reserved, quiet" ($\alpha = 0.85$, $M = 2.99$, $SD = 1.26$). Neuroticism was measured with "anxious, easily upset" and "calm, emotionally stable" (reverse-coded) ($\alpha = 0.71$, $M = 2.67$, $SD = 1.01$).

Covariates

Based on prior work[49], we controlled for extraneous factors that can influence social behaviors. 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 depressive symptoms using the CES-D[90] ($\alpha = 0.87$, $M = 1.98$, $SD = 0.59$) and body mass index (BMI; $M = 23.59$, $SD = 4.47$), which we derived from participants' self-reported height and weight.

Analytical strategy

Because our outcome of interest was the extent to which participants engaged in SMU over FtF and in SMUSI over FtF, we created two variables to test our hypotheses: 1) *SMU over FtF* by first standardizing each participant's objective SMU (in minutes) and FtF (5-point scale) scores and subtracting the FtF variable from the SMU variable so that higher scores indicate more objective SMU over FtF ($M = -0.096$, $SD = 1.44$) and 2) *SMUSI over FtF* by standardizing each participant's SMUSI and FtF scores (as they were measured on different scale labels) and subtracting the FtF variable from the SMUSI variable so that higher scores indicate more self-reported SMUSI over FtF ($M = -0.005$, $SD = 1.18$). Based on theoretical grounds that inflammation should have linear effects on dependent measures, CRP was used as a continouous variable without excluding values over 10 mg/L, as is sometimes done when CRP is a dependent variable[91]. We used these variables in all analyses.

In testing our hypotheses, we followed prior work[49] by sequentially controlling for an increasing number of covariates: (1) no covariates (Model 1), (2) socio-demographic factors (Model 2), (3) BMI (Model 3), and (4) depressive symptoms (Model 4). This was done to provide greater details on the nature of the effect. We controlled for sociodemographic factors because they can influence social media usage[92]. Although BMI was not controlled for in prior work, we included it as a covariate given its association with sedentary behaviors, which may influence SMU, SMUSI, and FtF. Finally, given the possibility that depression can influence social media use[93,94] and inflammation[95], we controlled for depressive symptoms. We controlled for these extraneous variables sequentially (vs. simultaneously) to provide more details about how the inclusion of each covariate influenced the association between CRP and SMU/SMUSI.

We first explored how our outcome variables (i.e., SMU over FtF, SMUSI over FtF) were associated with key variables by conducting Pearson correlation using SPSS version 30. We then tested our main hypotheses by conducting a series of multiple regression (H1a and H1b) and moderation analyses (H2a-H3b) using Model 1 of the PROCESS macro (version 4.2) for SPSS in SPSS. To assess the robustness of our main analyses, we also used Structural Equation Modeling (SEM; lavaan, version 0.6–18) to test whether CRP was more strongly associated

Variables	1	2	3	4	5	6	7	8	9	10
1. CRP	–									
2. SMU	.17†	–								
3. SMUSI	.14†	.26***	–							
4. FtF	−.07	−.05	.27***	–						
5. SMU-FtF	.18*	.73***	.02	−.72***	–					
6. SMUSI-FtF	.17*	.25**	.58***	−.62***	.60***	–				
7. Introversion	−.05	−.02	−.25***	−.48***	.31***	.21**	–			
8. Neuroticism	.12	.29***	−.01	−.17*	.33***	.15†	.17*	–		
9. Depress	.12	.25**	.03	−.37***	.46***	.34**	.26***	.42***	–	
10. Gender	−.02	.28***	−.03	.08	.17†	−.09	.05	.24**	.24**	–

Table 1. Zero-order correlations for key variables. †$p \leq .10$ *$p \leq .05$. **$p \leq .01$. ***$p \leq .001$ (two-tailed). SMU, amount of social media use. SMUSI, social media use for social interaction. FtF, face-to-face social interaction frequency; SMU-FtF, SMU over FtF; SMUSI-FtF, SMUSI over FtF; Depress, depressive symptoms. Gender was coded with 1 (male) and 2 (female).

	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	.16 (.033)	[.01, .31]	.17 (.028)	[.02, .33]	.19 (.019)	[.03, .36]	.14 (.07)	[−.01, .29]
Gender			.37 (.19)	[−.18, .92]	.37 (.19)	[−.18, .91]	.14 (.58)	[−.38, .66]
Age			−.02 (.64)	[−.12, .07]	−.02 (.68)	[−.12, .08]	−.01 (.80)	[−.10, .08]
M_edu			.10 (.51)	[−.19, .38]	.08 (.60)	[−.21, .37]	.10 (.47)	[−.17, .37]
F_edu			.05(.69)	[−.20, .30]	.06 (.64)	[−.19, .31]	.05 (.70)	[−.19, .28]
Income			−.04 (.45)	[−.15, .07]	−.05 (.40)	[−.16, .06]	−.03 (.53)	[−.14, .07]
BMI					−.02 (.40)	[−.08, .03]	−.03 (.33)	[−.08, .03]
Depr							.95 (<.001)	[.52, 1.39]
R^2		.04		.06		.07		.20

Table 2. Coefficients from linear regression models predicting SMU over FtF. Gender was coded with 1 (male) and 2 (female). M_edu, education level by mother; F_edu, education level by father; BMI, body mass index; Depr, depressive symptoms.

with SMU and SMUSI than with FtF. As detailed in the Supplemental Materials, the results were consistent across the different analyses.

Results

Table 1 presents zero-order correlations among all key variables. Notably, SMU over FtF was positively correlated with introversion ($r = 0.31$, $p < 0.001$), neuroticism ($r = 0.33$, $p < 0.001$), and depressive symptoms ($r = 0.46$, $p < 0.001$), indicating that people who scored higher on introversion, neuroticism, and depressive symptoms engaged in more SMU over FtF. Similarly, SMUSI over FtF was positively associated with introversion ($r = 0.21$, $p < 0.001$), neuroticism ($r = 0.15$, $p = 0.07$), and depressive symptoms ($r = 0.34$, $p < 0.001$), indicating that people who scored higher on these variables engaged in more SMUSI over FtF. These findings are largely consistent with the idea that individuals high in introversion and individuals high in neuroticism may use more social media[70] and how depression can lead to more social media use[93].

H1a CRP will be positively associated with objective SMU over FtF.

We conducted a series of multiple regression analyses with CRP as a predictor of SMU over FtF (i.e., SMU minus FtF). CRP was positively associated with SMU over FtF in Model 1 ($\beta = 0.19$, $p = 0.033$), Model 2 ($\beta = 0.20$, $p = 0.028$), and Model 3 ($\beta = 0.23$, $p = 0.019$). Although directionally consistent, CRP was not a statistically significant predictor in Model 4 ($\beta = 0.17$, $p = 0.070$). See Table 2 for details.

H1b CRP will be positively associated with self-reported SMUSI over FtF.

We conducted regression analyses with CRP as a predictor of SMUSI over FtF (i.e., SMUSI minus FtF). Although CRP was positively associated with SMU over FtF in Model 1 ($\beta = 0.18$, $p = 0.036$) and Model 2 ($\beta = 0.19$, $p = 0.035$), it was not a statistically significant predictor in Model 3 ($\beta = 0.17$, $p = 0.069$), and Model 4 ($\beta = 0.11$, $p = 0.20$). See Table 3 for details.

Predictor	Model 1		Model 2		Model 3		Model 4	
	b (*p*)	95% CI	b (p)	95% CI	b (*p*)	95% CI	b (*p*)	95% CI
CRP	.12 (.036)	[.01, .24]	.13 (.035)	[.01, .25]	.11 (.069)	[−.01, .24]	.08 (.20)	[−.04, .19]
Gender			−.16 (.46)	[−58, .26]	−.16 (.46)	[−.58, .26]	−.33 (.11)	[−.73, .08]
Age			−.01 (.76)	[−.08, .06]	−.01 (.74)	[−.08, .06]	−.01 (.69)	[−.08, .05]
M_edu			−.15 (.18)	[−.37, .07]	−.14 (.23)	[−.36, .09]	−.12 (.28)	[−.33, .10]
F_edu			.02 (.84)	[−.18, .22]	.02 (.87)	[−.18, .21]	−.01 (.90)	[−.20, .17]
Income			−.01 (.88)	[−.09, .08]	−.01 (.92)	[−.09, .08]	.02 (.70)	[−.06, .10]
BMI					.02 (.41)	[−.02, .06]	.02 (.39)	[−.02, .06]
Depr							.71 (<.001)	[.38, 1.04]
R^2		.03		.06		.06		.18

Table 3. Coefficients from linear regression models predicting SMUSI over FtF. Gender was coded with 1 (male) and 2 (female). M_edu, education level by mother; F_edu, education level by father; BMI, Body mass index; Depr, depressive symptoms.

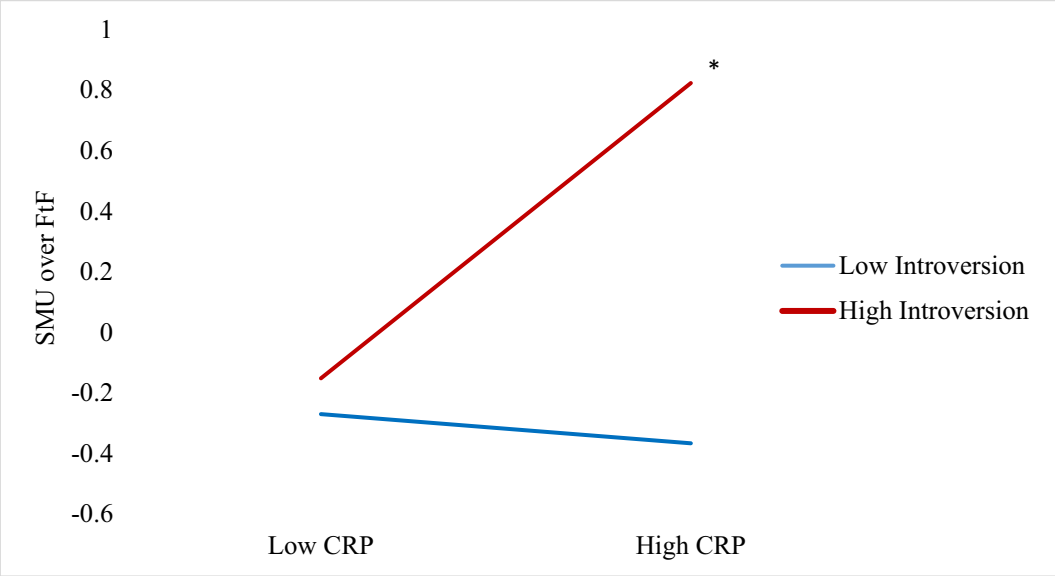

Fig. 1. CRP and SMU over FtF for individuals low in introversion (− 1 SD from mean) and individuals high in introversion (+ 1 SD from mean) with all covariates (Model 4). Note *$p = .002$. 95% CIs for individuals low in introversion: [−.25, .17] and individuals high in introversion: [.14, .61].

H2a Introversion will moderate the link between CRP and objective SMU over FtF.

Moderation analyses yielded a CRP x introversion interaction predicting objective SMU over FtF in three models, Model 1 ($F(1, 124) = 5.33$, $p = 0.053$), Model 2 ($F(1, 114) = 4.51$, $p = 0.036$), Model 3 ($F(1, 113) = 5.00$, $p = 0.027$), Model 4 ($F(1, 112) = 6.05$, $p = 0.016$). To better understand the broader pattern of our results, we further probed the interactions. Simple slopes analyses indicated that for individuals high in introversion (1 SD above the mean), CRP was positively associated with SMU over FtF, Model 1 ($b = 0.36$, $p = 0.003$, 95% CI = [0.12, 0.59]), Model 2 ($b = 0.38$, $p = 0.002$, 95% CI = [0.14, 0.61]), Model 3 ($b = 0.42$, $p = 0.001$, 95% CI = [0.17, 0.66]), Model 4 ($b = 0.37$, $p = 0.002$, 95% CI = [0.14, 0.61]); for individuals low in introversion (1 SD below the mean), CRP did not predict SMU over FtF ($ps = 0.87, 0.98, 0.87, 0.73$). See Fig. 1 depicting Model 4 results.

H2b Introversion will moderate the link between CRP and self-reported SMUSI over FtF.

Moderation analyses yielded a CRP x introversion interaction predicting SMUSI over FtF: Model 1 ($F(1, 138) = 2.90$, $p = 0.091$), Model 2 ($F(1, 126) = 3.75$, $p = 0.055$), Model 3 ($F(1, 124) = 3.80$, $p = 0.054$), Model 4 ($F(1, 123) = 4.80$, $p = 0.030$). Although the interaction was non-significant depending on different analytical approaches, based on theoretical grounds we further explored the interactions: For individuals high in introversion (1 SD above the mean), CRP was positively associated with SMUSI over FtF, Model 1 ($b = 0.26$, $p = 0.008$, 95% CI = [0.07, 0.45]), Model 2 ($b = 0.29$, $p = 0.004$, 95% CI = [0.09, 0.48]), Model 3 ($b = 0.26$, $p = 0.008$, 95% CI = [0.07, 0.45]), Model 4

($b = 0.23$, $p = 0.01$, 95% CI = [0.05, 0.41]); for individuals low in introversion (1 SD below the mean), CRP was not associated with SMUSI over FtF ($ps = 0.81, 0.94, 0.88, 0.49$). See Fig. 2 depicting Model 4 results.

H3a Neuroticism will moderate the link between CRP and objective SMU over FtF.

Moderation analyses yielded a significant CRP x neuroticism interaction predicting objective SMU over FtF in three models: Model 1 ($F(1, 124) = 3.68$, $p = 0.057$), Model 2 ($F(1, 114) = 4.38$, $p = 0.039$), Model 3 ($F(1, 113) = 5.29$, $p = 0.023$), Model 4 ($F(1, 112) = 5.29$, $p = 0.026$). Simple slopes analyses indicated that for individuals high in neuroticism (1 SD above the mean), CRP was positively associated with SMU over FtF, Model 1 ($b = 0.30$, $p = 0.008$, 95% CI = [0.08, 0.52]), Model 2 ($b = 0.34$, $p = 0.004$, 95% CI = [0.11, 0.56]), Model 3 ($b = 0.39$, $p = 0.001$, 95% CI = [0.16, 0.63]), Model 4 ($b = 0.35$, $p = 0.003$, 95% CI = [0.12, 0.57]); for individuals low in neuroticism (1 SD below the mean), CRP was not associated with SMU over FtF ($ps = 0.69, 0.69, 0.78, 0.59$). See Fig. 3 depicting Model 4 results.

H3b Neuroticism will moderate the link between CRP and self-reported SMUSI over FtF.

The CRP x neuroticism interaction significantly predicted SMUSI over FtF in two models: Model 1 ($F(1, 138) = 3.47$, $p = 0.065$), Model 2 ($F(1, 126) = 4.12$, $p = 0.044$), Model 3 ($F(1, 124) = 3.75$, $p = 0.055$), Model 4 ($F(1, 123) = 3.97$, $p = 0.048$). Simple slopes analyses indicated that for individuals high in neuroticism (1 SD above the mean), CRP was positively associated with SMUSI over FtF, Model 1 ($b = 0.24$, $p = 0.009$, 95% CI = [0.06, 0.41]), Model 2 ($b = 0.26$, $p = 0.005$, 95% CI = [0.08, 0.44]), Model 3 ($b = 0.23$, $p = 0.01$, 95% CI = [0.05, 0.42]), Model 4 ($b = 0.20$, $p = 0.02$, 95% CI = [0.03, 0.38]); for individuals low in neuroticism (1 SD below the mean), as predicted, CRP was not associated with SMUSI over FtF ($ps = 0.70, 0.66, 0.60, 0.42$). See Fig. 4 depicting Model 4 results.

Discussion

The present research examined how inflammation relates to daily social behaviors, namely, SMU, SMUSI, and FtF. Based on emerging research on inflammation and social behavior[11,13] and extant work on interaction modes and their affordances[24,37], we predicted and showed that inflammation was associated with greater SMU and SMUSI over FtF. Although these effects became weaker with some covariates (e.g., depressive symptoms) included in the model, the pattern of results remained the same across all models. Importantly, we found that these patterns were stronger among individuals higher in introversion or neuroticism.

Our findings make several novel contributions. First, by identifying inflammation as a correlate of greater SMU/SMUSI over FtF, we provide insight into *when* and *how* people engage in daily social behaviors. Because interaction modality can have implications for relational and personal well-being[17,96,97], understanding the conditions under which people interact with others on social media versus face-to-face is important. Second, we contribute to the growing body of research on inflammation and social behavior. While most prior studies have focused on demonstrating the effects of experimentally manipulated levels of inflammation on laboratory-based proxies of social behavior[11,12], our findings show that naturally occurring inflammation is related to actual daily social behaviors. Moreover, inflammation's link to greater SMU/SMUSI over FtF corroborates the view that the impact of inflammation on social behavior is context-dependent[10,12]. Finally, our CRP x personality traits

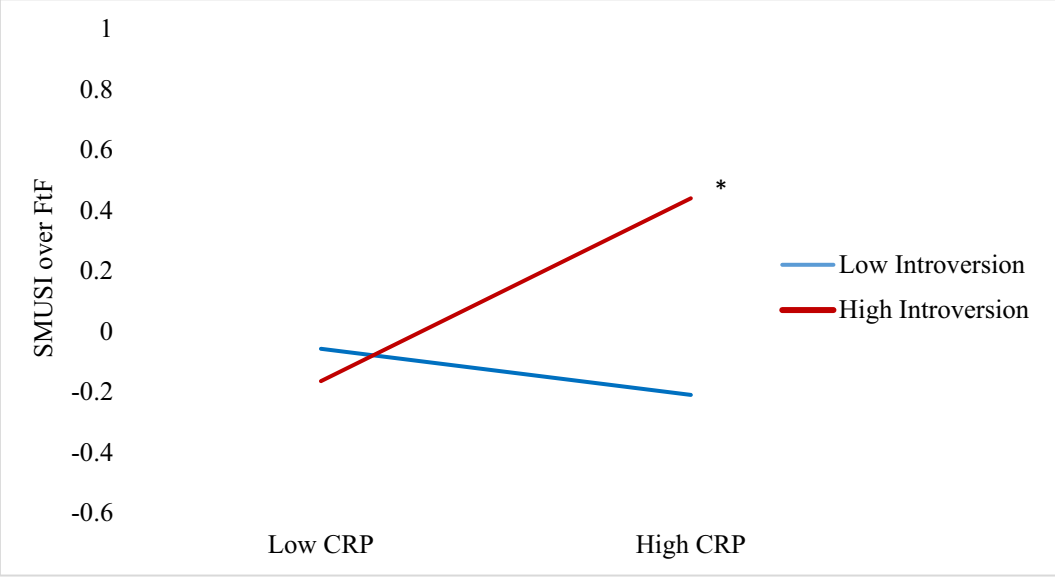

Fig. 2. CRP and SMUSI over FtF for individuals low in introversion (− 1 SD from mean) and individuals high in introversion (+ 1 SD from mean) with all covariates (Model 4). Note $^*p = .01$. 95% CIs for individuals low in introversion: [− .23, .11] and individuals high in introversion: [.05, .41].

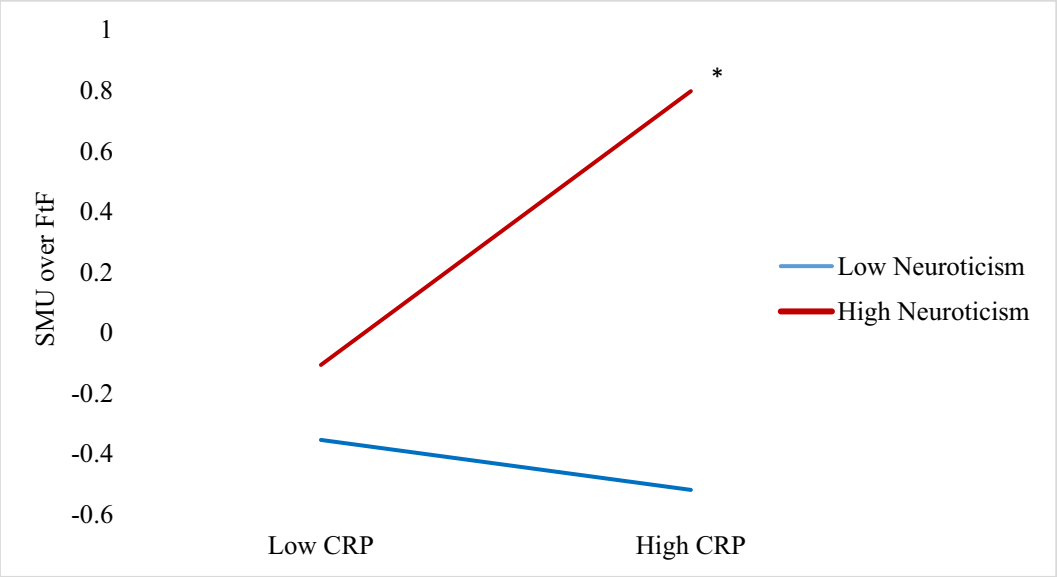

Fig. 3. CRP and SMU over FtF for individuals low in neuroticism (− 1 SD from mean) and individuals high in neuroticism (+ 1 SD from mean) with all covariates (Model 4). Note *$p = .003$. 95% CIs for individuals low in neuroticism: [− .29, .17] and individuals high in neuroticism: [.12, .57].

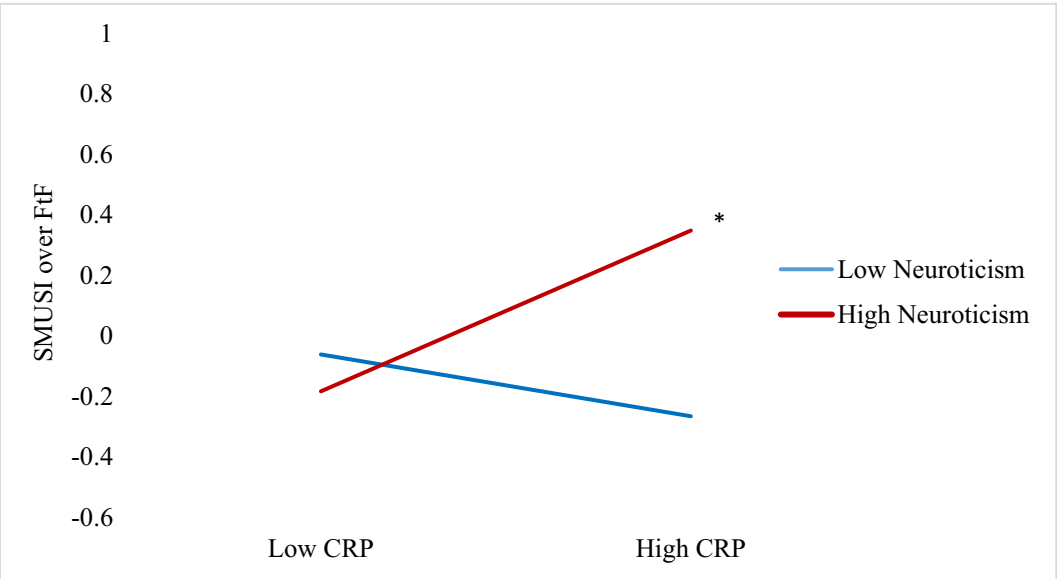

Fig. 4. CRP and SMUSI over FtF for individuals low in neuroticism (− 1 SD from mean) and individuals high in neuroticism (+ 1 SD from mean) with all covariates (Model 4). Note *$p < .01$. 95% CIs for individuals low in neuroticism: [− .11, .11] and individuals high in neuroticism: [.05, .24].

moderation findings provide a biological account for why certain individuals may turn to social media over FtF. Here, we contribute to research on the social compensation hypothesis[71], which to date has predominantly focused on personality traits or sociality (e.g., introversion, rejection sensitivity) to explain why people may prefer online to offline interactions. We add to this literature by showing that a biological marker of inflammation can contribute to social compensation effects.

Broader implications

Social media's role as a social technology

Our findings suggest that social media can play an intriguing role as a social technology, especially for individuals who, for whatever reasons, cannot or prefer not to engage in FtF. Because humans have a fundamental need for social connections[98], such individuals still must find ways to fulfill their affiliative needs. Indeed, research

shows that socially vulnerable individuals (e.g., low self-esteem, high rejection sensitive) desire high-quality social interactions and relationships just as much as others do[99]. Similarly, it is not that individuals with high inflammation do not want social interactions, but rather, they seem to have a particular preference regarding *whom* they interact with (e.g., supportive figures[11]) or, as we suggest, *how* they interact with others (SMU/SMUSI over FtF). We propose that social media may be a particularly appealing space for these individuals as it has lower energy demands, infection risks, or social rejection cues compared with FtF. One interesting future direction is to explore whether our findings would generalize to other social technology, for example, AI companion apps such as ChatGPT, Character.AI, or Replika. Although empirical evidence is needed, these technologies also offer opportunities to fulfill affiliative needs in energy efficient ways with virtually no infection or social rejection risks. Thus, we would suspect that our findings would generalize to many other technologically mediated social contexts.

Nevertheless, whether SMU or SMUSI is an *effective* method for these individuals to address their affiliative needs remains to be an important empirical question[71,72]. On the one hand, studies show that social media can help individuals who struggle to cultivate social relationships face-to-face connect with others online[72,100]. On the other, such individuals also report higher rates of being cyberbullied and harassed online[101]. However, we suspect that higher SMU/SMUSI over FtF may be detrimental to well-being, especially when SMU/SMUSI *displace* FtF[102]. Despite the potential benefits of SMU/SMUSI, studies show that FtF, compared with other interaction modalities, is still the most effective way to achieve feelings of social connectedness and meet various relational needs[17,36]. Similarly, although we cannot rule out bidirectional interpretations, in our study SMU over FtF and SMUSI over FtF were both positively correlated with depressive symptoms. Thus, while more research is needed to understand when and how SMU/SMUSI can effectively fulfill affiliative needs, future work should also consider a broader array of daily social behaviors (e.g., FtF, SMU, texting, parasocial interactions) rather than examining any one behavior in isolation.

Inflammation and social behavior

We note that inflammation levels in the daily lives of young people like those in our study are appreciably lower than inflammation levels present in people with infections (e.g., flu, COVID) and chronic illnesses (e.g., autoimmune or cardiovascular disease). Thus, it is likely that our participants were not necessarily feeling sick or aware of their inflamed state. In this way, our finding that inflammation, even when mild with no drastic symptoms, is associated with SMU/SMUSI over FtF is consistent with evidence that "low-grade" inflammation (i.e. elevated inflammation in the absence of an infection) can have psychological effects similar to those from acute inflammation in response to infection, for example[43,78]. Because inflammation can be elevated by a variety of daily experiences such as stress, poor sleep quality or diet, or low exercise[76,77], its impact on daily social behaviors is likely to occur frequently and for many individuals including those who are healthy. Thus, future research should aim to better understand how inflammation is related to a wider variety of daily social behaviors.

Another future direction is identifying the upstream biological pathways that may be influencing the inflammation seen here. One potential system to investigate in future research is the parasympathetic nervous system (PNS). Stimulation of the PNS inhibits the production of proinflammatory cytokines[103,104] and a non-invasive index of greater PNS activity (High Frequency Heart Rate Variability; HRV) is associated with lower circulating levels of CRP and pro-inflammatory cytokines[105]. A rich body of theoretical work[106] has postulated a social engagement system governed by the PNS. In support of a role for greater PNS activity facilitating social approach, greater HRV is associated with greater social support seeking as a coping response[107] and greater felt affiliation with an ingroup and greater behavior supporting the ingroup[108]. A related measure of greater PNS activity has been associated with greater probability of being with others rather than alone on an event recall diary[109]. Conversely, a dispositional tendency for social avoidance (trait shyness) was strongly associated with lower levels of HRV[110]. Thus, PNS activity does appear to be related to social approach, and whether higher PNS activity is associated with the degree to which one engages in SMU over FtF and mediates the inflammatory effects seen here will be an important area for future research.

Limitations and future directions

The present research has some limitations. First, the correlational nature of this study does not allow us to make strong causal or directional inferences regarding our findings. For example, we cannot conclude that greater SMU over FtF does not lead to higher inflammation[82]. These results do provide a valuable platform justifying more challenging (and expensive) studies that would address limitations such as intensive longitudinal designs that measure inflammation, SMU, SMUSI, and FtF multiple times or experiments that manipulate inflammation levels (e.g., administering a molecule that mimics a bacterial infection). Second, although we have hypothesized that inflammation would be associated with more SMU/SMUSI over FtF through several mechanisms such as increased energy conservation motivation or vigilance against physical or psychological threats, our findings do not provide direct evidence for these mechanisms. Similarly, although we have posited that much of the appeal of SMU is its ability to fulfill affiliative needs, people may use social media for other reasons (e.g., reading the news). Thus, future research should more directly examine the motivations behind using social media (e.g., seeking social support) and different types of SMU (e.g., direct messaging, posting selfies, scrolling). Relatedly, future studies may also examine *whom* (e.g., strong vs. weak ties) people seek to interact with when they are under higher levels of inflammation. Finally, future research should seek to improve measurement of social behaviors. For example, it is possible that some of our participants had imposed Screen Time limit on their phone during the week of the data collection, which can directly influence SMU and SMUSI. Given the growing number of apps and features designed to help users limit their social media or smartphone use, future studies should assess in their study whether participants are using such tools to restrict their usage. Moreover, because self-report measures of SMU frequency estimates can be imprecise[111], future studies should consider developing

more objective measures of SMUSI (e.g., using data donation or passive sensing method to capture time spent on direct messaging) and FtF (e.g., Electorincally Activated Recorder, which unobtrusively records conversations naturalistically[112]). Related, our study was limited in that we collected SMU data from five social media apps, which may not capture the full range of apps participants use. This reflects a broader challenge in social media research: researchers must decide in advance how many apps to assess prior to data collection while participants are likely to be using more apps than can be reasonably included in a survey. Although our approach in collecting usage across five apps is an improvement over prior studies that focus on a single app (e.g., Facebook), future research may benefit from more intensive methodologies such as passive sensing, which can unobtrusively track smartphone use across all apps over time. Such approaches would allow researchers to retrieve comprehensive usage information after data collection and test more targeted hypotheses—for example, examining within-person associations between inflammation and the use of messaging apps such as WhatsApp or Telegram.

Conclusion

The present research examined how inflammation is associated with daily social behaviors. Extending prior work, inflammation was associated with more SMU and SMUSI over FtF. Importantly, this pattern was more pronounced among individuals with higher introversion or neuroticism. These results not only corroborate the idea that the link between inflammation and social behavior may be context-dependent but highlight the unique role of social media as a social technology for certain individuals. Given the increasing awareness of mind–body connections and the importance of daily social lives, more research investigating the relation between biological processes and social behavior is needed.

Data availability

The dataset for this study may be available from the corresponding author on reasonable request.

Received: 17 September 2025; Accepted: 3 February 2026
Published online: 17 February 2026

References

1. Dantzer, R. & Kelley, K. W. Twenty years of research on cytokine-induced sickness behavior. *Brain Behav. Immun.* **21**, 153–160 (2007).
2. Eisenberger, N. I., Moieni, M., Inagaki, T. K., Muscatell, K. A. & Irwin, M. R. In sickness and in health: The co-regulation of inflammation and social behavior. *Neuropsychopharmacol* **42**, 242–253 (2017).
3. D'Acquisto, F. Affective immunology: Where emotions and the immune response converge. *Dialogues Clin. Neurosci.* **19**, 9–19 (2017).
4. Inagaki, T. K., Muscatell, K. A., Irwin, M. R., Cole, S. W. & Eisenberger, N. I. Inflammation selectively enhances amygdala activity to socially threatening images. *Neuroimage* **59**, 3222–3226 (2012).
5. Eisenberger, N. I., Inagaki, T. K., Mashal, N. M. & Irwin, M. R. Inflammation and social experience: An inflammatory challenge induces feelings of social disconnection in addition to depressed mood. *Brain Behav. Immun.* **24**, 558–563 (2010).
6. Makhanova, A. et al. Immune response and intergroup bias: Vaccine-induced increases in cytokine activity are associated with worse evaluations of resume for Latina job applicant. *Brain Behav. Immun.* **122**, 555–564 (2024).
7. Dooley, L. N. et al. The role of inflammation in core features of depression: Insights from paradigms using exogenously-induced inflammation. *Neurosci. Biobehav. Rev.* **94**, 219–237 (2018).
8. Dantzer, R. Cytokine-induced sickness behavior: Mechanisms and implications. *Ann. N. Y. Acad. Sci.* **933**, 222–234 (2001).
9. Kelley, K. W. et al. Cytokine-induced sickness behavior. *Brain Behav. Immun.* **17**, 112–118 (2003).
10. Muscatell, K. A. et al. Exposure to an inflammatory challenge enhances neural sensitivity to negative and positive social feedback. *Brain Behav. Immun.* **57**, 21–29 (2016).
11. Inagaki, T. K. et al. The role of the ventral striatum in inflammatory-induced approach toward support figures. *Brain Behav. Immun.* **44**, 247–252 (2015).
12. Jolink, T. A. et al. Inflammatory reactivity to the influenza vaccine is associated with changes in automatic social behavior. *Brain Behav. Immun.* **99**, 339–349 (2022).
13. Muscatell, K. A. & Inagaki, T. K. Beyond social withdrawal: New perspectives on the effects of inflammation on social behavior. *Brain Behav. Immun. Health* **16**, 100302 (2021).
14. Hansson, L. S. et al. Perception of unfamiliar caregivers during sickness—Using the new caregiver perception task (CgPT) during experimental endotoxemia. *Brain Behav. Immun.* **119**, 741–749 (2024).
15. Cole, S. W. The Complexity of Dynamic Host Networks. In *Complex Systems Science.* In Deisboeck, T. S. & Kresh, J. Y. (eds.) *Biomedicine,* 605–629 (Springer US, 2006). https://doi.org/10.1007/978-0-387-33532-2_26.
16. Hennessy, M. B., Deak, T. & Schiml, P. A. Sociality and sickness: Have cytokines evolved to serve social functions beyond times of pathogen exposure?. *Brain Behav. Immun.* **37**, 15–20 (2014).
17. Hall, J. A. *Relating Through Technology: Everyday Social Interaction*. (Cambridge University Press, 2020). https://doi.org/10.1017/9781108629935.
18. Papacharissi, Z. We Have Always Been Social. *Social Media + Society* **1**, 2056305115581185 (2015).
19. Rhee, L., Bayer, J. B., Lee, D. S. & Kuru, O. Social by definition: How users define social platforms and why it matters. *Telemat. Inform.* **59**, 101538 (2021).
20. Bayer, J. B., Triệu, P. & Ellison, N. B. Social media elements, ecologies, and effects. *Annu. Rev. Psychol.* **71**, 471–497 (2020).
21. Altman, I. & Taylor, D. A. *Social Penetration: The Development of Interpersonal Relationships* (Holt, 1973).
22. Collins, N. L. & Miller, L. C. Self-disclosure and liking: A meta-analytic review. *Psychol. Bull.* **116**, 457–475 (1994).
23. Ellison, N. B., Vitak, J., Gray, R. & Lampe, C. Cultivating social resources on social network sites: Facebook relationship maintenance behaviors and their role in social capital processes. *J Comput-Mediat Comm* **19**, 855–870 (2014).
24. Walther, J. B., Slovacek, C. L. & Tidwell, L. C. Is a picture worth a thousand words?: Photographic images in long-term and short-term computer-mediated communication. *Commun. Res.* **28**, 105–134 (2001).
25. Domahidi, E. The associations between online media use and users' perceived social resources: A meta-analysis. *J. Comput.-Mediat. Commun.* **23**, 181–200 (2018).
26. Ellison, N. B., Steinfield, C. & Lampe, C. The benefits of facebook "Friends:" Social capital and college students' use of online social network sites. *J. Comput.-Mediat. Commun.* **12**, 1143–1168 (2007).
27. Hall, J. A. When is social media use social interaction? Defining mediated social interaction. *New Media Soc.* **20**, 162–179 (2018).
28. Tosun, L. P. Motives for Facebook use and expressing "true self" on the Internet. *Comput. Hum. Behav.* **28**, 1510–1517 (2012).

29. Wang, Z., Tchernev, J. M. & Solloway, T. A dynamic longitudinal examination of social media use, needs, and gratifications among college students. *Comput. Hum. Behav.* **28**, 1829–1839 (2012).
30. Lee, E., Lee, J.-A., Moon, J. H. & Sung, Y. Pictures speak louder than words: Motivations for using instagram. *Cyberpsychol. Behav. Soc. Netw.* **18**, 552–556 (2015).
31. Sheldon, P. & Bryant, K. Instagram: Motives for its use and relationship to narcissism and contextual age. *Comput. Hum. Behav.* **58**, 89–97 (2016).
32. Raacke, J. & Bonds-Raacke, J. MySpace and Facebook: Applying the uses and gratifications theory to exploring friend-networking sites. *Cyberpsychol. Behav.* **11**, 169–174 (2008).
33. Sheldon, P. Student favorite: Facebook and motives for its use. *Southwest. Mass Commun. J.* **23**, 39–53 (2008).
34. Harrison, N. A. et al. Inflammation Causes Mood Changes Through Alterations in Subgenual Cingulate Activity and Mesolimbic Connectivity. *Biol. Psychiat.* **66**, 407–414 (2009).
35. Lasselin, J. et al. Fatigue and sleepiness responses to experimental inflammation and exploratory analysis of the effect of baseline inflammation in healthy humans. *Brain Behav. Immun.* **83**, 309–314 (2020).
36. Hall, J. A., Dominguez, J., Merolla, A. J. & Otmar, C. D. Social bandwidth: When and why are social interactions energy intensive?. *J. Soc. Pers. Relat.* **40**, 2614–2636 (2023).
37. Hall, J. A. & Davis, D. C. Proposing the Communicate bond belong theory: Evolutionary intersections with episodic interpersonal communication—Proposing the communicate bond belong theory. *Commun Theor* **27**, 21–47 (2017).
38. Miller, S. L. & Maner, J. K. Sick body, vigilant mind: The biological immune system activates the behavioral immune system. *Psychol Sci* **22**, 1467–1471 (2011).
39. Schaller, M. & Park, J. H. The behavioral immune system (and Why It Matters). *Curr. Dir. Psychol. Sci.* **20**, 99–103 (2011).
40. Ackerman, J. M., Hill, S. E. & Murray, D. R. The behavioral immune system: Current concerns and future directions. *Soc. Personal. Psych.* **12**, e12371 (2018).
41. Makhanova, A., Miller, S. L. & Maner, J. K. Germs and the out-group: Chronic and situational disease concerns affect intergroup categorization. *Evol. Behav. Sci.* **9**, 8–19 (2015).
42. Makhanova, A. & Shepherd, M. A. Behavioral immune system linked to responses to the threat of COVID-19. *Personal. Individ. Differ.* **167**, 110221 (2020).
43. Gassen, J. & Hill, S. E. Why inflammation and the activities of the immune system matter for social and personality psychology (and not only for those who study health). *Soc. Personal. Psych.* **13**, e12471 (2019).
44. Prokosch, M. L., Gassen, J., Ackerman, J. M. & Hill, S. E. Caution in the time of cholera: Pathogen threats decrease risk tolerance. *Evol. Behav. Sci.* **13**, 311–334 (2019).
45. McKenna, K. Y. A. & Bargh, J. A. Plan 9 From Cyberspace: The implications of the internet for personality and social psychology. *Pers. Soc. Psychol. Rev.* **4**, 57–75 (2000).
46. Hance, M. A., Blackhart, G. & Dew, M. Free to be me: The relationship between the true self, rejection sensitivity, and use of online dating sites. *J. Soc. Psychol.* **158**, 421–429 (2018).
47. Luo, M. & Hancock, J. T. Self-disclosure and social media: Motivations, mechanisms and psychological well-being. *Curr. Opin. Psychol.* **31**, 110–115 (2020).
48. Walther, J. B. Computer-mediated communication: Impersonal, interpersonal, and hyperpersonal interaction. *Commun. Res.* **23**, 3–43 (1996).
49. Lee, D. S., Jiang, T., Crocker, J. & Way, B. M. Can inflammation predict social media use? Linking a biological marker of systemic inflammation with social media use among college students and middle-aged adults. *Brain Behav. Immun.* **112**, 1–10 (2023).
50. Seidman, G. Self-presentation and belonging on Facebook: How personality influences social media use and motivations. *Personal. Individ. Differ.* **54**, 402–407 (2013).
51. McCrae, R. R. & Costa, P. T. Personality trait structure as a human universal. *Am. Psychol.* **52**, 509–516 (1997).
52. John, O. P., Robins, R. W. & Pervin, L. A. *Handbook of Personality: Theory and Research* (Guilford press, 2008).
53. Handbook of Personality. *Theory and Research* (Guilford Press, 1999).
54. Schmeck, R. & Lockhart, D. Introverts and extraverts require different learning environments. *Educ. Leadersh.* **40**, 54–55 (1983).
55. Cain, S. Susan Cain: The power of introverts: (549532012–001). https://doi.org/10.1037/e549532012-001 (2012).
56. Asendorpf, J. B. & Wilpers, S. Personality effects on social relationships. *J. Pers. Soc. Psychol.* **74**, 1531–1544 (1998).
57. Burger, J. M. Individual differences in preference for solitude. *J. Res. Pers.* **29**, 85–108 (1995).
58. Barrett, L. F. & Pietromonaco, P. R. Accuracy of the five-factor model in predicting perceptions of daily social interactions. *Pers. Soc. Psychol. Bull.* **23**, 1173–1187 (1997).
59. Smillie, L. D. Extraversion and reward processing. *Curr. Dir. Psychol. Sci.* **22**, 167–172 (2013).
60. Riggio, R. E. Assessment of basic social skills. *J. Pers. Soc. Psychol.* **51**, 649–660 (1986).
61. Eaton, L. G. & Funder, D. C. The creation and consequences of the social world: An interactional analysis of extraversion. *Eur. J. Pers.* **17**, 375–395 (2003).
62. Ackerman, J. M. & Bargh, J. A. Two to Tango : Automatic Social Coordination and the Role of Felt Effort. In Bruya, B. (ed.) *Effortless Attention*. 335–372 (The MIT Press, 2010). https://doi.org/10.7551/mitpress/9780262013840.003.0015.
63. Roskes, M., Elliot, A. J., Nijstad, B. A. & De Dreu, C. K. W. Avoidance motivation and conservation of energy. *Emot. Rev.* **5**, 264–268 (2013).
64. Bower, J. E. Cancer-related fatigue—Mechanisms, risk factors, and treatments. *Nat. Rev. Clin. Oncol.* **11**, 597–609 (2014).
65. Amichai-Hamburger, Y., Wainapel, G. & Fox, S. 'On the Internet No One Knows I'm an Introvert': Extroversion, neuroticism, and internet interaction. *Cyberpsychol. Behav.* **5**, 125–128 (2002).
66. Newby, J. et al. Neuroticism and perfectionism as predictors of social anxiety. *Personal. Individ. Differ.* **106**, 263–267 (2017).
67. McNulty, J. K. Neuroticism and Interpersonal Negativity: The independent contributions of perceptions and behaviors. *Pers. Soc. Psychol. Bull.* **34**, 1439–1450 (2008).
68. Russell, D. W., Booth, B., Reed, D. & Laughlin, P. R. Personality, social networks, and perceived social support among alcoholics: A structural equation analysis. *J. Pers.* **65**, 649–692 (1997).
69. Dehle, C. & Landers, J. E. You can't always get what you want, but can you get what you need? Personality traits and social support in marriage. *J. Soc. Clin. Psychol.* **24**, 1051–1076 (2005).
70. Valkenburg, P. M., Schouten, A. P. & Peter, J. Adolescents' identity experiments on the internet. *New Media Soc.* **7**, 383–402 (2005).
71. Valkenburg, P. M. & Peter, J. Online communication and adolescent well-being: Testing the stimulation versus the displacement hypothesis. *J. Comput.-Mediat. Commun.* **12**, 1169–1182 (2007).
72. Zywica, J. & Danowski, J. The faces of Facebookers: Investigating social enhancement and social compensation hypotheses; predicting Facebook™ and offline popularity from sociability and self-esteem, and mapping the meanings of popularity with semantic networks. *J. Comput.-Mediat. Commun.* **14**, 1–34 (2008).
73. Blackhart, G. C., Hernandez, D. K., Wilson, E. & Hance, M. A. The impact of rejection sensitivity on self-disclosure within the context of online dating. *Cyberpsychol. Behav. Soc. Netw.* **24**, 690–694 (2021).
74. Toma, C. L. Online dating and psychological wellbeing: A social compensation perspective. *Curr. Opin. Psychol.* **46**, 101331 (2022).
75. Michikyan, M., Subrahmanyam, K. & Dennis, J. Can you tell who I am? Neuroticism, extraversion, and online self-presentation among young adults. *Comput. Hum. Behav.* **33**, 179–183 (2014).

76. Uchino, B. N. et al. Social support, social integration, and inflammatory cytokines: A meta-analysis. *Health Psychol.* **37**, 462–471 (2018).
77. Uchino, B. N. et al. Self-rated health and inflammation: A Test of depression and sleep quality as mediators. *Psychosom Med* **81**, 328–332 (2019).
78. Lindsay, E. K. Small, "doses" of inflammation initiate social sickness behavior. *Brain Behav. Immun.* **102**, 40–41 (2022).
79. Hall, J. A. & Liu, D. Social media use, social displacement, and well-being. *Curr. Opin. Psychol.* **46**, 101339 (2022).
80. Hall, J., Pennington, N. & Holmstrom, A. Connecting Through Technology During COVID-19. *hct* **3**, (2021).
81. Kross, E. et al. Facebook use predicts declines in subjective well-being in young adults. *PLoS ONE* **8**, e69841 (2013).
82. Lee, D., Jiang, T., Crocker, J. & Way, B. Social media use and its concurrent and subsequent relation to a biological marker of inflammation: short-term longitudinal study. *J. Med. Internet Res.* **25**, e46309 (2023).
83. Verduyn, P. et al. Passive Facebook usage undermines affective well-being: Experimental and longitudinal evidence. *J. Exp. Psychol. Gen.* **144**, 480–488 (2015).
84. Clark, J. L., Algoe, S. B. & Green, M. C. Social network sites and well-being: The role of social connection. *Curr. Dir. Psychol. Sci.* **27**, 32–37 (2018).
85. McDade, T. W., Burhop, J. & Dohnal, J. High-sensitivity enzyme immunoassay for C-reactive protein in dried blood spots. *Clin. Chem.* **50**, 652–654 (2004).
86. Pew Research Center. *Teens and Social Media: Key Findings from Pew Research Center Surveys*. https://www.pewresearch.org/short-reads/2023/04/24/teens-and-social-media-key-findings-from-pew-research-center-surveys/ (2023).
87. Grady, S. M., Tamborini, R., Eden, A. & Van Der Heide, B. The social factors and functions of media use. *J. Commun.* **72**, 523–539 (2022).
88. Twenge, J. M., Spitzberg, B. H. & Campbell, W. K. Less in-person social interaction with peers among U.S. adolescents in the 21st century and links to loneliness. *J. Soc. Person. Relath.* **36**, 1892–1913 (2019).
89. Gosling, S. D., Rentfrow, P. J. & Swann, W. B. A very brief measure of the Big-Five personality domains. *J. Res. Pers.* **37**, 504–528 (2003).
90. Radloff, L. S. The CES-D Scale: A self-report depression scale for research in the general population. *Appl. Psychol. Meas.* **1**, 385–401 (1977).
91. Mac Giollabhui, N. et al. To exclude or not to exclude: Considerations and recommendations for C-reactive protein values higher than 10 mg/L. *Brain Behav. Immun.* **87**, 898–900 (2020).
92. Gottfried, J. & Park, E. Americans' Social Media Use 2025.
93. Aalbers, G., McNally, R. J., Heeren, A., De Wit, S. & Fried, E. I. Social media and depression symptoms: A network perspective. *J. Exp. Psychol. Gen.* **148**, 1454–1462 (2019).
94. Griffioen, N., Lichtwarck-Aschoff, A., Van Rooij, M. & Granic, I. From wellbeing to social media and back: A multi-method approach to assessing the bi-directional relationship between wellbeing and social media use. *Front. Psychol.* **12**, 789302 (2021).
95. Osimo, E. F. et al. Inflammatory markers in depression: A meta-analysis of mean differences and variability in 5,166 patients and 5,083 controls. *Brain Behav. Immun.* **87**, 901–909 (2020).
96. Goodman-Deane, J., Mieczakowski, A., Johnson, D., Goldhaber, T. & Clarkson, P. J. The impact of communication technologies on life and relationship satisfaction. *Comput. Hum. Behav.* **57**, 219–229 (2016).
97. Macdonald, B., Luo, M. & Hülür, G. Daily social interactions and well-being in older adults: The role of interaction modality. *J. Soc. Pers. Relat.* **38**, 3566–3589 (2021).
98. Baumeister, R. F. & Leary, M. R. The need to belong: Desire for interpersonal attachments as a fundamental human motivation. *Psychol. Bull.* **117**, 497–529 (1995).
99. Wood, J. V. & Forest, A. L. Self-Protective yet Self-Defeating. In *Advances in Experimental Social Psychology*. 53 131–188 (Elsevier, 2016).
100. Kim, Y. & Fingerman, K. Daily social media use, social ties, and emotional well-being in later life. *Innov. Aging* **5**, 300–300 (2021).
101. Nesi, J., Mann, S. & Robb, M. B. Common Sense is Grateful for the Generous Support and Underwriting that Funded this Research Report (2023).
102. Kushlev, K. & Leitao, M. R. The effects of smartphones on well-being: theoretical integration and research agenda. *Curr. Opin. Psychol.* **36**, 77–82 (2020).
103. Tracey, K. J. The inflammatory reflex. *Nature* **420**, 853–859 (2002).
104. Way, B. M. & Uchino, B. Physiological pathways mediating between psychological stress and health. In Schneiderman, N. et al. (eds.) *APA handbook of health psychology, Volume 1: Foundations and context of health psychology.* (1), 313–334 (American Psychological Association, 2025). https://doi.org/10.1037/0000394-014.
105. Williams, D. P. et al. Heart rate variability and inflammation: A meta-analysis of human studies. *Brain Behav. Immun.* **80**, 219–226 (2019).
106. Porges, S. W. Polyvagal theory: A biobehavioral journey to sociality. *Compr. Psychoneuroendocrinol.* **7**, 100069 (2021).
107. Geisler, F. C. M., Kubiak, T., Siewert, K. & Weber, H. Cardiac vagal tone is associated with social engagement and self-regulation. *Biol. Psychol.* **93**, 279–286 (2013).
108. Sahdra, B. K., Ciarrochi, J. & Parker, P. D. High-frequency heart rate variability linked to affiliation with a new group. *PLoS ONE* **10**, e0129583 (2015).
109. Isgett, S. F. et al. Influences of oxytocin and respiratory sinus arrhythmia on emotions and social behavior in daily life. *Emotion* **17**, 1156–1165 (2017).
110. Schwerdtfeger, A. R., Rominger, C. & Obser, P. D. A shy heart may benefit from everyday life social interactions with close others: An ecological momentary assessment trial using Bayesian multilevel modeling. *Biol. Psychol.* **152**, 107864 (2020).
111. Parry, D. A. et al. A systematic review and meta-analysis of discrepancies between logged and self-reported digital media use. *Nat. Hum. Behav.* **5**, 1535–1547 (2021).
112. Mehl, M. R. The electronically activated recorder (EAR): A method for the naturalistic observation of daily social behavior. *Curr. Dir. Psychol. Sci.* **26**, 184–190 (2017).

Author contributions

DL was involved in study conceptualization, methodology, data curation and analyses, writing original draft, and reviewing and editing. TJ was involved in methodology, data curation and analyses, reviewing and editing manuscript. BW was involved in study conceptualization, methodology, and reviewing and editing manuscript.

Funding

The project described was supported in part by Award Number [anonymized] from the National Institute of Mental Health (NIMH). The content is solely the responsibility of the authors and does not necessarily represent the official views of the NIMH.

Declarations

Competing interests
The authors declare no competing interests.

Additional information
Supplementary Information The online version contains supplementary material available at https://doi.org/10.1038/s41598-026-39242-8.

Correspondence and requests for materials should be addressed to D.S.L.

Reprints and permissions information is available at www.nature.com/reprints.

Publisher's note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.