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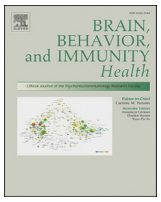

Full Length Article

Social media use and systemic inflammation: The moderating role of self-esteem

David S. Lee [a,*], Baldwin M. Way [b]

[a] University at Buffalo, The State University of New York, Buffalo, NY, USA
[b] The Ohio State University, Columbus, OH, USA

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ABSTRACT

Social media use has become an important part of social life. However, little is known about its relation to physical health. Extending prior work on social media use and psychological well-being, the present research investigated how social media use is associated with a key indicator of health, systemic inflammation. Based on research on self-esteem and work on inflammation, the current study examined whether the link between social media use and inflammatory biomarkers would be moderated by self-esteem. A nationally probablistic sample of middle-aged adults (N = 863) completed self-report questionnaires on social media use, self-esteem, socio-demographic information, and health related behaviors. Approximately two years later, they provided a blood sample that was analyzed for C-reactive protein (CRP) and interleukin-6 (IL-6), biomarkers of systemic inflammation. Consistent with our hypothesis, self-esteem moderated the association between social media use and these markers of inflammation. Specifically, as self-esteem decreased, the positive association of social media use with CRP and IL-6 became stronger. These results held after controlling for socio-demographic information, health status, depressive symptoms, and medication usage. Social media use was not significantly correlated with either CRP or IL-6. The present research demonstrates physical health correlates of social media use and suggests self-esteem as a key variable that can moderate the relation between social media use and health.

1. Introduction

Social media use has become a key part of people's social lives. As of 2019, over 70 % of American adults had used at least one social media site (Perrin and Anderson, 2019). American adults report spending over 2 h per day on social media interacting with others (Statista, 2020); by comparison, they spent less than 2 h per day on eating/drinking, household activities, socializing face-to-face, or exercising (Bureau of Labor Statistics, 2020).

As social media use became more integrated into people's social lives, research examining its impact on psychological well-being has exploded in the past decade (e.g., Coyne et al., 2020; Heffer et al., 2019; Huang, 2017; Hunt et al., 2018; Kross et al., 2013; Liu et al., 2019; Orben et al., 2019; Primack et al., 2017; Shakya and Christakis, 2017; Song et al., 2014; Tromholt, 2016; Verduyn et al., 2015). However, in contrast to the vast amount of work on social media's impact on psychological well-being, little is known about how social media use relates to physical health. This is surprising in light of emerging work showing that the use of media technology can contribute to stress (e.g., Fox and Moreland, 2015; Lee et al., 2016; Morin-Major et al., 2016; Reinecke et al., 2016), depression (e.g., Riehm et al., 2019; Twenge et al., 2018), or lower quality of sleep (e.g., Cain and Gradisar, 2010; Thomee et al., 2011)—processes closely linked to physical health (e.g., Cohen et al., 2007; Murray and Lopez, 1997; Wingard et al., 1982).

1.1. Social media use, self-esteem, and health

Although there is no consensus, a large body of research indicates a small, negative association between social media use and psychological well-being (Liu et al., 2019; Kross et al., 2021; also see Orben et al., 2019). For example, in a longitudinal study, Facebook use predicted declines in affective and cognitive well-being (Kross et al., 2013). Other studies found that limiting or abstaining from social media use reduced loneliness and depression, as well as enhancing life satisfaction (Hunt et al., 2018; Tromholt, 2016). However, recent work has also reported no significant link between social media use and well-being (Odgers and Jensen, 2020; Orben and Przybylski, 2019) and suggested that social media use itself may not be a strong predictor of well-being outcomes

* Corresponding author. 327 Baldy Hall, Buffalo, NY, 14228.
E-mail addresses: dslee9@buffalo.edu (D.S. Lee), way.37@osu.edu (B.M. Way).

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(Coyne et al., 2020; Hall et al., 2019; Heffer et al., 2019; Orben et al., 2019; Stieger and Lewetz, 2018). Instead, emerging perspectives suggest that the link between social media use and well-being may be moderated by an individual's personality characteristics or goals (Lee et al., 2015; Rae and Lonborg, 2015; Sheldon and Bryant, 2016; Sheldon et al., 2021; Tobin et al., 2020; see Orben, 2020). Drawing on this perspective, the present research focuses on people's self-esteem—a key personality construct that critically shapes one's interpersonal life and influences mental and physical health (Leary and Baumeister, 2000; Wood and Forest, 2016).

Research on self-esteem suggests that interpersonal experiences can have vastly different effects on people with different levels of self-esteem.[1] In general, people low in self-esteem (LSEs) feel anxious and stressed in social situations because they fear of being disapproved or rejected by others (Wood and Forest, 2016). Notably, LSEs tend to interpret interpersonal events in ways that validate their negative self-views (Murray et al., 1998). Further, they are more likely to fixate on negative aspects of themselves and maintain negative mood (Wood and Forest, 2016), which can lead to undermined physical health (e.g., Cohen et al., 2007; Glassman and Shapiro, 1998; Rozanski et al., 1999; Wulsin et al., 1999). In contrast, people high in self-esteem (HSEs) tend to cultivate high-quality relationships with others and use them as a resource for self-affirmation (Murray et al., 1998). For example, unlike LSEs who feel insecure after receiving a compliment, HSEs benefit from receiving compliments and social support as it validates and bolsters their positive self-views (Lee and Way, 2019; Marigold et al., 2007).

The overly flattering and self-focused nature of the social media environment may not be a nurturing social space for LSEs (Forest and Wood, 2012). On social media, people tend to portray themselves in overly positive manners and share positive life events more than negative ones (Kross et al., 2013). Constant exposure to positive information about others can trigger upward social comparison and elicit envy, which is linked to lower well-being (Verduyn et al., 2015). Such an environment may be especially stressful for LSEs who tend to perceive others' positive events as a threat to their belonging (Forest and Wood, 2012; see Fox and Moreland, 2015 for a discussion on Facebook stress; also see Lee et al., 2016). In contrast, HSEs may be buffered from these processes as they tend to focus on their positive aspects and are less affected by the potential for rejection (Wood and Forest, 2016).

To test the potential physical health implications of social media use for HSEs and LSEs, the present research focused on systemic inflammation as a key health indicator. The reason to focus on inflammation is that chronic psychological stress, such as experienced by LSEs relative to HSEs when using social media, can lead to elevated levels of systemic inflammation over time (Johnson et al., 2013; Nersesian et al., 2018; Walker et al., 2019). Such inflammation is a potent driver of diseases such as cardiovascular disease, cancers, and diabetes (Emerging Risk Factors Collaboration, 2010; Kiecolt-Glaser et al., 2010; Uchino et al., 2018; Zhou et al., 2012). It is often assessed by measuring blood levels of inflammatory markers such as the proinflammatory cytokine interleukin-6 (IL-6) and the acute phase reactant C-reactive protein (CRP). Emerging research indicates that social integration and related social processes (e.g., social support, quality of social interactions) are reliably associated with lower levels of IL-6 and CRP (see Uchino et al., 2018 for a meta-analysis). For example, older women who had satisfying relationships with others had lower IL-6 (Friedman et al., 2005). However, recent work found that perceived social support was associated with lower CRP only for HSEs, presumably because LSEs' negative self-view often prevents them from benefiting from supportive relationships (Lee and Way, 2019).

[1] For convenience and ease of interpretability, we use terms "HSEs" and "LSEs" to refer to "people high in self-esteem" and "people low in self-esteem," respectively. Self-esteem is typically measured on a continuum, and the labels do not mean that they are two distinct groups.

The collective evidence linking systemic inflammation to the quality of social relationships and interactions is consistent with theory that threats to social safety (e.g., social conflict, isolation, rejection, exclusion) can undermine health (Slavich, 2020; also see Cole et al., 2015 for a related perspective on perceived social isolation and gene expression). Taken together, extant research suggests that self-esteem may play a moderating role in the link between social media use and inflammation. Specifically, we hypothesized that social media use would be associated with higher inflammation for LSEs. This should be the case because LSEs may interpret information on social media in ways that validate their negative self-view (Wood and Forest, 2016). However, HSEs' tendency to focus on their positive aspects (Murray et al., 1998) may buffer them from the negative health impact of social media use (Deters and Mehl, 2013). Thus, we predicted that social media use would not be associated with increased chronic inflammation for HSEs.

2. Method

2.1. Participants and procedure

Data for this study came 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). From 2011 to 2014, the MIDUS-R study recruited a nationally probablistic sample of 3577 adults and collected survey data on a variety of variables including socio-demographic information, psychosocial factors, and health assessments. A subsample of these participants (N = 863) subsequently participated in the MIDUS Refresher Biomarker Project study from 2012 to 2016, during which they provided blood samples and were assessed for physical health and physiological function. Data collection for the Biomarker study (i.e. CRP and IL-6 measurements) occurred about two years (M = 22.16 months, SD = 8.87 months) after the completion of the MIDUS-R survey. Thus, the final analytic sample for this study is the 863 adults (age range 26–78 years old, M = 52.72 years old, SD = 13.44 years old; 450 females; 70.2 % White) in the Biomarker study who had also participated in the MIDUS-R study (i.e., reported on social media use and self-esteem).

2.2. Measures

2.2.1. Social media use

Using an 8-point scale (1 = *several times a day*, 8 = *never or hardly ever*), participants indicated how often they used social media (e.g., Facebook, Twitter, MySpace, etc.) to contact family members (e.g., brothers, sisters, parents, or children) who did not live with them in the past year (M = 3.08, SD = 1.61). Using the same item, they also rated how often they used social media to contact their friends in the past year (M = 4.66, SD = 2.56). We reverse-coded and averaged the two items to create a composite *social media use* variable, with higher scores reflecting more social media use (α = 0.80, M = 4.56, SD = 2.30).

2.2.2. Inflammatory biomarkers

Two markers of inflammation were used in the analysis: C-reactive protein (CRP) and interleukin-6 (IL-6). CRP was initially measured from plasma using a particle enhanced immunonephelometric assay (BNII nephelometer; Dade Behring Inc., Deerfield, IL). The intra- and inter-assay coefficients of variance (CVs) ranged from 2.3 % to 4.3 % and 1.1–4.4 %, respectively. The final batch of samples (collected after February 1, 2015; N = 393) as well as those below the limit of detection using the immunonephelometric assay (N = 27) were assayed from serum using the Meso Scale Diagnostics (MSD) immunoelectrochemiluminescent platform (intra-assay CV: 2.2–4.1 %; inter-assay CV: 4.7–5.2 %). The data from the two platforms was integrated using the data adjustment formulas described in the study documentation (see Weinstein et al., 2018 for details). IL-6 was measured from serum using an ultrasensitive enzyme-linked immunosorbent assay (R&D Systems,

Table 1
Zero-order correlations for all variables.

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22
1. SNS use																						
2. SE	.03																					
3. log_CRP	.06	-.09*																				
4. log_IL6	-.08*	-.09*	.58**																			
5. Age	-.26**	.21**	.05	.35**																		
6. Sex	.18**	.02	.18**	-.00	-.01																	
7. Income	-.01	.25**	-.02	.02	.12**	-.03*																
8. Edu	.02	.04*	-.08*	-.07	-.01	.00	.03															
9. Married	-.02	-.07**	.09*	.05	-.07**	.12**	-.05**	-.05**														
10. Alcohol	.01	.21**	-.18**	-.10**	.06**	-.07**	.07**	.07**	-.03													
11. Smoking	-.01	.16**	.10**	.19**	.09**	-.02	.02	-.01	.01	.49**												
12. Caffeine	-.06	-.08*	.10**	.17**	.01	.04	-.01	-.01	.06	-.14**	-.05											
13. BMI	.03	-.15**	.51**	.44**	-.03	.04	-.00	-.15**	.09*	-.22**	.05	.14**										
14. IADL	-.12**	.73**	.23**	.35**	.31**	.10**	.18**	-.06**	.04*	.15**	.17**	.08*	.31**									
15. CESD	-.00	-.47**	.13**	.14**	-.17**	.10**	-.04	-.11**	.18**	-.07*	.10**	.10**	.18**	.24**								
16. Antidepr	-.02	-.09*	.07	.06	.01	.06	.06	-.00	.02	-.03	-.01	.02	.06	.10*	.11**							
17. COX-2	-.03	-.03*	-.03	-.04	-.05**	.01	-.00	-.02	.01	-.08**	-.04*	.05	-.01	-.04*	.03	.04						
18. NSAID_O	.01	.04	-.04	.06	.21**	-.08*	-.00	.01	-.03	.07	.03	-.01	.01	.12**	-.02	.01	.04					
19. NSAID_P	.05	-.07	-.07	-.11**	-.12**	-.01	-.06	-.00	-.03	.01	-.02	-.07*	-.04	-.07	.01	.02	.06	.51**				
20. Statin	-.06	.02	-.03	.20**	.36**	-.19**	.06	.04	-.05	.03	.06	.09**	.14**	.18**	-.06	.05	-.10**	.20**	-.10**			
21. ACE	-.08*	.06	.05	.16**	.18**	-.11**	.00	-.04	.04	.02	.12**	.10**	.13**	.17**	.02	-.03	-.02	.07	-.11**	.25**		
22. Beta block	-.09*	.06	.05	.19**	.23**	-.05	.01	-.05	.02	-.05	.04	.11**	.10**	.19**	-.05	.02	-.10**	.11**	-.09*	.29**	.20**	
23. Omega-3	-.04	.05	-.05	.03	.25**	-.09**	.04	.01	-.12**	.11**	.02	-.01	-.01	.05	-.06	-.03	-.04	.02	-.08*	.13**	-.03	.07*

Note. * indicates $p < .05$. ** indicates $p < .01$. Sex was coded with 1 (male) and 2 (female). Edu = highest degree obtained; Married = marital status (1 = married, 2 = not married, widowed, or single); Alcohol = number of drinks consumed in the past month; Smoking = cigarette smoking status (0 = never smoked, 1 = used tobacco regularly or in the past); Caffeine = number of hours since consuming caffeinated beverage; BMI = body mass index; IADL = Instrumental Activities of Daily Living; CESD = depressive symptoms; Antidepr = antidepressant medication usage; COX-2 = COX-2 inhibitor usage; NSAID_O = NSAID oral medication usage; NSAID_P = NSAID parenteral medication usage; Statin = Statin usage; ACE = Angiotensin-converting enzyme inhibitor usage; Beta block = beta-adrenergic blocker usage; Omega-3 = Omega-3 Fatty Acid usage; SNS use = amount of social media use; SE = self-esteem.

Table 2
Coefficients from linear regression models predicting CRP and IL-6.

	Model 0		Model 1		Model 2		Model 3		Model 4	
	CRP	IL-6	CRP	IL-6	CRP	IL-6	CRP	IL-6	CRP	IL-6
Predictor	*β (p)*	*β (p)*	*β (p)*	*β (p)*	*β (p)*	*β (p)*	*β (p)*	*β (p)*	*β (p)*	*β (p)*
Age	–	–	.11 (.029)	.41 (<.001)	.09 (.07)	.36 (<.001)	.09 (.08)	.37 (<.001)	.13 (.017)	.36 (<.001)
Sex	–	–	.09 (.08)	-.09 (.06)	.10 (.038)	-.10 (.023)	-.09 (.048)	-.11 (.012)	.07 (.11)	-.11 (.015)
Income	–	–	.02 (.65)	-.06 (.21)	-.01 (.83)	-.09 (.054)	-.13 (.78)	-.09 (.038)	.01 (.92)	-.09 (.055)
Edu	–	–	-.19 (<.001)	-.11 (.021)	-.07 (.17)	.01 (.82)	-.07 (.16)	-.06 (.88)	-.08 (.11)	.01 (.87)
Married	–	–	.05 (.29)	.03 (.52)	.03 (.45)	-.01 (.97)	.03 (.48)	.01 (.84)	.03 (.49)	-.01 (.81)
Alcohol	–	–	–	–	-.07 (.11)	-.04 (.38)	-.07 (.11)	-.04 (.39)	-.07 (.14)	-.04 (.40)
Smoking	–	–	–	–	.08 (.10)	.04 (.32)	.08 (.10)	.04 (.33)	.09 (.07)	.05 (.30)
Caffeine	–	–	–	–	.04 (.32)	-.06 (.16)	.04 (.31)	-.05 (.20)	.04 (.30)	-.05 (.24)
BMI	–	–	–	–	.43 (<.001)	.33 (<.001)	.42 (<.001)	.31 (<.001)	.44 (<.001)	.30 (<.001)
IADL	–	–	–	–	.01 (.90)	.15 (.002)	.01 (.93)	.14 (.005)	.01 (.95)	.12 (.02)
CESD	–	–	–	–	–	-.01 (.91)	-.01 (.83)	.07 (.17)	.01 (.88)	.08 (.13)
Antidepr	–	–	–	–	–	–	.02 (.63)	.05 (.21)	.04 (.43)	.06 (.18)
COX-2	–	–	–	–	–	–	–	–	-.06 (.16)	-.01 (.83)
NSAID oral	–	–	–	–	–	–	–	–	-.01 (.97)	.01 (.84)
NSAID parent	–	–	–	–	–	–	–	–	-.10 (.08)	-.03 (.57)
Statin	–	–	–	–	–	–	–	–	-.16 (<.001)	-.01 (.83)
ACE	–	–	–	–	–	–	–	–	-.02 (.65)	.05 (.30)
Beta block									-.02 (.64)	.06 (.15)
Omega-3									.01 (.99)	-.05 (.21)
SNS use	.05 (.31)	-.08 (.10)	.07 (.19)	.04 (.40)	.03 (.51)	.03 (.50)	.03 (.49)	.04 (.43)	.03 (.48)	.04 (.41)
SE	-.19 (<.001)	-.09 (.06)	-.19 (<.001)	-.16 (.001)	-.11 (.015)	-.07 (.09)	-.10 (.045)	-.04 (.48)	-.10 (.049)	-.05 (.36)
SNS use x SE	-.12 (.011)	-.08 (.12)	-.12 (.012)	-.10 (.029)	-.09 (.048)	-.08 (.048)	-.09 (.047)	-.08 (.045)	-.09 (.031)	-.09 (.037)
R^2	.05	.02	.10	.21	.29	.35	.30	.36	.33	.37

Notes. Sex was coded with 1 (male) and 2 (female). Edu = highest degree obtained; Married = marital status (1 = married, 2 = not married, widowed, or single); Alcohol = number of drinks consumed in the past month; Smoking = cigarette smoking status (0 = never smoked, 1 = used tobacco regularly or in the past); Caffeine = number of hours since consuming caffeinated beverage; BMI = body mass index; IADL = Instrumental Activities of Daily Living; CESD = depressive symptoms; Antidepr = antidepressant medication usage; COX-2 = COX-2 inhibitor usage; NSAID oral = NSAID oral medication usage; NSAID parent = NSAID parenteral medication usage; Statin = Statin usage; ACE = Angiotensin-converting enzyme inhibitor usage; Beta block = beta-adrenergic blocker usage; Omega-3 = Omega-3 Fatty Acid usage; SNS use = amount of social media use; SE = self-esteem.

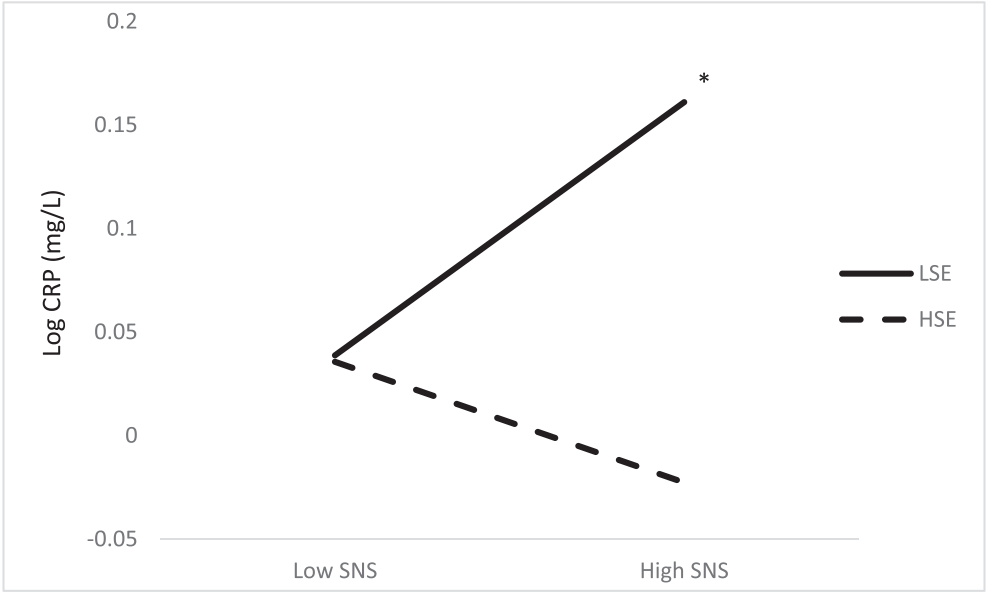

Fig. 1. Social media use and CRP (logged) for LSE (low self-esteem; −1 SD from mean) and HSE (high self-esteem; +1 SD from mean) in model 4. Note. *p < .05. 95 % CIs for LSE: [0.000, 0.05] and HSE: [-0.04, 0.01]. 95 % CI for the conditional effects for Low SNS: [-0.009, 007] and High SNS [-0.02, −0.004].

Minneapolis, MN). The intra-assay CV was 4.7 % and the inter-assay CV ranged from 5 % to 15 %. CRP and IL-6 values were log-transformed to achieve a normal distribution. One participant had missing data for IL-6 and 4 participants had missing data for CRP due to an insufficient amount of blood collected and were thus not included in the analyses.

2.2.3. Self-esteem

Participants responded to seven items from the Rosenberg (1965) self-esteem scale on a 7-point scale ranging from 1 (*strongly agree*) to 7 (*strongly disagree*). Example items include "I certainly feel useless at times" and "I take a positive attitude toward myself (reverse-coded)." We

summed the items to create a *self-esteem* composite, with higher scores reflecting higher levels of self-esteem ($\alpha = 0.78$, $M = 37.08$, $SD = 7.50$).

2.2.4. Covariates

Based on prior work (Horn et al., 2018; Lee and Way, 2019; O'Connor et al., 2009), we controlled for extraneous factors that have been associated with inflammation. Our socio-demographic covariates were age, gender, marital status, income, and highest level of education (1 = *some grade school*, 12 = *Ph.D., MD, etc.*). Health status covariates (measured at the time of the blood draw) included body mass index (BMI), cigarette smoking status (i.e., used tobacco regularly now or in the past), alcohol consumption (i.e., number of drinks in the past month), and caffeine consumption before measuring physiology. Additionally, we controlled participants' ability to carry out daily activities using an adapted version of the Instrumental Activities of Daily Living Scale (Ware and Sherbourne, 1992): Participants used a 4-point scale (1 = *a lot*, 4 = *not at all*) to evaluate their ability to complete daily life activities (e.g., "*How much does your health limit you in lifting or carrying groceries*?"). We also controlled for depressive symptoms using the Center for Epidemiological Studies Depression Scale (Radloff, 1977), medications (0 = *no*, 1 = *yes*) that have been reported to influence IL-6 and/or CRP: antidepressants, antihypertensives (i.e., Angiotensin Convering Enzyme (ACE) inhibitors and Beta-adrenergic receptor blockers), lipid lowering drugs (i.e. statins), nonsteroidal anti-inflammatory drugs (i.e., NSAID (oral and parenteral); COX-2 inhibitors), and omega-3 fatty acids, as they have been shown to have anti-inflammatory properties (see Horn et al., 2018).

3. Results

First, individuals with CRP values over 10 μg/mL (N = 46; <5.41 %) were excluded from both the CRP and IL-6 analyses as such values may indicate the presence of an acute infection (Pearson et al., 2003). We also excluded 38 participants who had lupus/autoimmune disorder, 46 participants who were under cancer treatment/therapy, 21 participants taking adrenal cortical steroids (i.e., prednisone), 6 participants taking immunosuppressants (i.e. Natalizumab), and one participant taking an immunostimulant. See Supplement for details on sensitivity analyses showing how different exclusion criteria impacted results. Table 1 presents zero-order correlations among key variables. All statistical analyses were conducted with SPSS 26.0.

To test our prediction that the link between social media use and inflammation would be moderated by self-esteem, we conducted a series of multiple regression analyses for each of the inflammatory markers, CRP and IL-6. First, we included standardized variables of social media use and self-esteem, and their interaction term as predictors of CRP. Then, we included the same predictors to predict IL-6 in a separate set of analyses. Based on prior research (Horn et al., 2018; Lee and Way, 2019), the models sequentially controlled for the following covariates: (1) socio-demographic factors, (2) physical health status, (3) depressive symptoms and antidepressants use, and (4) use of drugs with anti-inflammatory properties (i.e., Models 1–4). To show the degree of discrepancy between coefficients produced with differing number of covariates, we additionally included a Model 0, which does not include any covariates (see Lynam et al., 2006). The results of these analyses are detailed in Table 2.

Across Models 0–4, social media use was not significantly associated with CRP. Self-esteem was negatively correlated with CRP in all of the five models. Consistent with our hypothesis, the social media use X self-esteem interaction predicted CRP in Model 0 ($b = -0.003$, $p = .011$, 95 % CI = [-0.006, −0.001]), Model 1 ($b = -0.003$, $p = .012$, 95 % CI = [-0.006, −0.001]), Model 2 ($b = -0.002$, $p = .048$, 95 % CI = [-0.005, 0.000]), Model 3 ($b = -0.002$, $p = .047$, 95 % CI = [-0.005, 0.000]), and Model 4 ($b = -0.003$, $p = .031$, 95 % CI = [-0.005, 0.000]); the small confidence intervals indicated that this effect, while small, was estimated with precision. In all models, as self-esteem decreased, the positive association between social media use and CRP became stronger (see Fig. 1). Simple slopes analyses indicated that for LSEs (1 SD below mean), social media use was associated with higher CRP (Model 0 ($b = 0.04$, $p = .014$, 95 % CI = [0.007, 0.06]), Model 1 ($b = 0.04$, $p = .009$, 95 % CI = [0.01, 0.07]), Model 2 ($b = 0.02$, $p = .074$, 95 % CI = [-0.002, 0.05]), Model 3 ($b = 0.02$, $p = .069$, 95 % CI = [-0.002, 0.05]), and Model 4 ($b = 0.03$, $p = .053$, 95 % CI = [0.000, 0.05]). For example, Model 4 revealed that for LSEs, a unit increase in social media use was associated with 0.13 μg/ml increase in CRP (unlogged). However, this pattern was not observed among HSEs (1 SD above mean) (*ps* = .27, .40, 0.35, 0.36, 0.31).

Across Models 0–4, social media use was not significantly correlated with IL-6. Self-esteem was negatively associated with IL-6 in one of the five models. Consistent with our hypothesis, the social media use X self-esteem interaction significantly predicted IL-6 in four of the five models

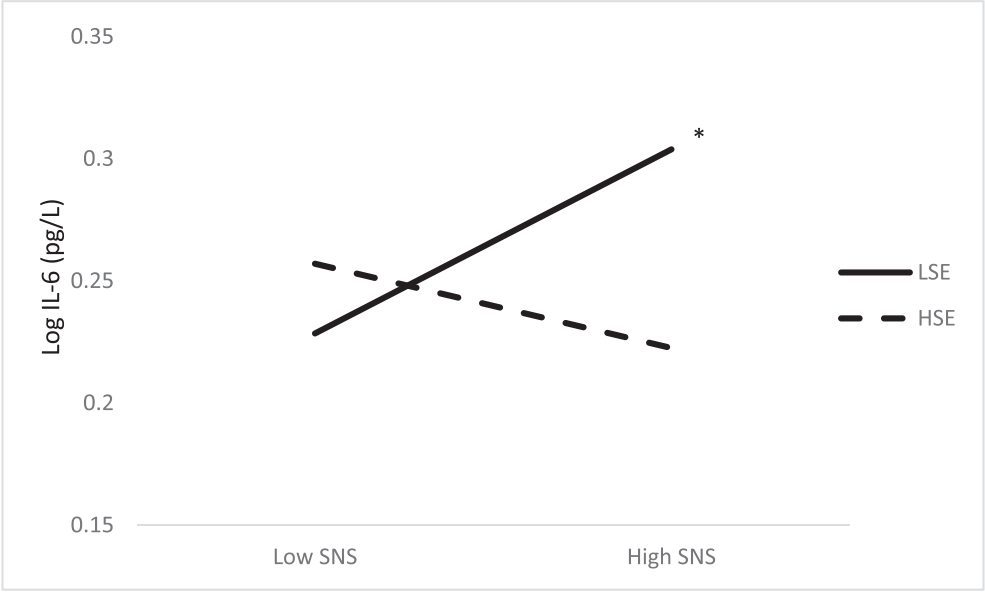

Fig. 2. Social media use and IL-6 (logged) for LSE (low self-esteem; −1 SD from mean) and HSE (high self-esteem; +1 SD from mean) in model 4. Note. *p < .05.95 % CIs for LSE [0.000, 0.04] and HSE: [-0.02, 0.01]. 95 % CI for the conditional effects for Low SNS: [-0.004, 007] and High SNS [-0.01, −0.0002].

(Model 0 ($b = -0.002$, $p = .12$, 95 % CI = [-0.003, 0.000]), Model 1 ($b = -0.002$, $p = .029$, 95 % CI = [-0.004, 0.000]), Model 2 ($b = -0.002$, $p = .048$, 95 % CI = [-0.003, 0.000]), Model 3 ($b = -0.002$, $p = .045$, 95 % CI = [-0.003, 0.000]), and Model 4 ($b = -0.002$, $p = .037$, 95 % CI = [-0.003, 0.000]). As self-esteem decreased, the positive association between social media use and IL-6 became stronger (see Fig. 2). Simple slopes analyses indicated that for LSEs (1 SD below mean), social media use was positively associated with IL-6 (Model 0 ($b = 0.000$, $p = .97$, 95 % CI = [-0.02, 0.02]), Model 1 ($b = 0.02$, $p = .038$, 95 % CI = [0.001, 0.04]), Model 2 ($b = 0.02$, $p = .071$, 95 % CI = [-0.001, 0.03]), Model 3 ($b = 0.02$, $p = .057$, 95 % CI = [-0.001, 0.04]), and Model 4 ($b = 0.02$, $p = .049$, 95 % CI = [0.000, 0.04]). For example, Model 4 revealed that a unit increase in social media use was associated with 0.09 pg/ml increase in IL-6 (unlogged) for LSEs. Although social media use was negatively associated with IL-6 for HSEs (1 SD above mean) in Model 0 ($b = -0.02$, $p = .022$, 95 % CI = [-0.04, −0.003]), this pattern was not observed in other models ($ps = .34, .35, 0.39, 0.38$).

4. Discussion

The present research examined how self-esteem would influence the link between social media use and systemic inflammation, a predictor of multiple disease states such as cancer and cardiovascular disease. The results showed that as self-esteem decreased, the positive association between social media use and inflammation became stronger. Specifically, for LSEs, social media use was related to higher levels of CRP and IL-6. However, the same relation was not observed among HSEs. Although the results differed slightly depending on the covariates included and different exclusion criteria, the pattern of results remained largely consistent and similar for both markers of inflammation. These results provide preliminary evidence that self-esteem may play a role in moderating the relation between social media use and some inflammatory markers.

Our findings make several novel contributions. First, we extend research on social media use and well-being (e.g., Kross et al., 2021) by showing the physical health correlates of social media use utilizing two biological measures. To our knowledge, very little work has examined the relation between social media use and biological measures that are health related (see Afifi et al., 2018 for an exception). Thus, our findings contribute to this nascent yet important research area. Second, by showing the moderating role of self-esteem, the current results indicate that the impact of social media use may depend on "who" uses it. This perspective is consistent with recent recommendations to identify a specific subpopulation (e.g., age, personality) when making claims about emergent technology effects (Orben, 2020). Broadly, the present study contributes to the current debate on the well-being effects of social media by highlighting the importance of people's self-esteem. Finally, our findings may contribute to the social integration and health literature. Although frequent face-to-face social interactions generally promote health (e.g., Cohen, 2004), frequent interactions with others on social media did not result in an improved inflammatory profile. These findings raise the question of how social media use, a novel form of social interaction, should be incorporated into the current conceptualization of social integration. To this end, future research should closely examine the role of social media interactions to better understand social integration in the digital age, and its effects on health.

Why was social media use significantly associated with inflammation among LSEs, but not among HSEs? As described in the introduction, LSE's may find social media use more stressful, which could induce greater inflammation. In contrast, perhaps HSEs are able to cultivate high-quality relationships and interactions on social media (Wood and Forest, 2016). Given that social media can provide access to social resources, HSEs might have built more social capital and received more social support on social media. Relatedly, the approach-oriented nature of HSEs in social situations may lead them to use social media more "actively", which has been shown to buffer the negative well-being impact of social media use (Deters and Mehl, 2013; Verduyn et al., 2015). An interesting future direction would be to examine how self-esteem influences how people use social media (e.g., active vs. passive use, social comparison).

Notably, social media use was not associated with higher systemic inflammation in this study: It was not a significant predictor of CRP or IL-6 in any of our regression models; zero-order correlations revealed that social media use was not significantly associated with CRP and negatively associated with IL-6 (unexpectedly). On the surface, these results seem to be inconsistent with findings from Afifi et al. (2018), which discovered a positive correlation between Facebook use and IL-6 among adolescents. However, it is important to note that the present study involved middle-aged adults (mean age: 52.72 years old); studies show that older adults have different goals and motivations for using social media compared with younger adults (Leist, 2013). In fact, Afifi et al. (2018) found a positive association between Facebook use and IL-6 only among adolescents; the same pattern was not observed among the mothers and fathers in their study. Thus, one possibility is that our older participants used social media in ways that buffered them from the negative impact of social media use. However, our findings also indicate that social media use was positively associated with systemic inflammation for middle-aged adults lower in self-esteem. Given recent perspectives suggesting that social media use itself may not be a strong predictor of well-being outcomes (Coyne et al., 2020; Heffer et al., 2019; Orben et al., 2019) and that the link between social media use and well-being may be moderated by an individual's personality characteristics (Sheldon et al., 2021; Tobin et al., 2020), we believe a more plausible story is that the link between social media use and inflammation is moderated by a third variable—for example, self-esteem.

In contrast to the lack of a main effect of social media use on CRP or IL-6, self-esteem was negatively associated with CRP in all models and with IL-6 in models without the drug covariates. This is consistent with some recent work that found a negative association between a construct that includes self-esteem, psychological resources (e.g., optimism, mastery, self-esteem) and various inflammatory markers (e.g., Marteinsdottir et al., 2016; Stokes, 2020). However, other studies also report no reliable link between self-esteem and systemic inflammation (Chiang et al., 2019; Kim and Thomas, 2019) or only a significant correlation under certain contexts (Elliot and Chapman, 2016). Thus, the relationship between self-esteem and CRP seen here is not universally replicated and future research may clarify how and when various individual psychological characteristics are associated with systemic inflammation (Chen and Miller, 2013).

This study has some limitations. First, the present research relied on a correlational design, limiting our ability to make causal inferences about the relation between social media use and systemic inflammation. Thus, future work should employ longitudinal and experimental designs to provide causal evidence. Second, the effect sizes observed in the current study are small, though comparable to those typically found in studies on social media use and psychological well-being (from $r = -0.05$ to $r = -0.15$). This raises questions about whether the findings have clinical or practical significance (see Kross et al., 2021; Odgers and Jensen, 2020; Orben and Przybylski, 2019). Related, in this study the potential increase in family-wise error rate across the analyses was not controlled. Thus, at this point, we caution readers to not over-interpret these preliminary findings and encourage replication; readers may also consider factors that can influence effect size and significance level when interpreting the results (e.g., inclusion of covariates, sample characteristics, exclusion criteria; see Supplement for sensitivity analyses). Third, it is important to note that this study measured one type of social media usage (i.e., contacting family members and friends). Although our social media use measure is in line with how some scholars define "active" usage (e.g., Verduyn et al., 2015), it does not fully capture the wide array of activities people can engage in on social media. Given that people use social media for different purposes, and the different ways in which people use social media can influence their well-being (e.g., Verduyn et al., 2015; see Clark et al., 2018), future research should measure different activities people engage in on social media (e.g., reading the news, sharing photos/videos,

playing video games with others) and how they may relate to health. Fourth, although the MIDUS biomarker sample is large and diverse in terms of its sociodemographic distribution, it is not nationally representative. Thus, we encourage readers to be cautious in generalizing our findings to other populations. Relatedly, future research should examine whether our findings in middle-aged adults generalize to other populations (e.g., adolescents). Finally, there was a time lag ($M = 22.16$ months; $SD = 8.87$ months) between the blood draw and completion of the survey containing the social media use and self-esteem items. Thus, there may have been changes in either the biomarkers or the survey items that could have increased or, most likely, decreased the strength of these effects during this time period. Statistically adjusting for this time lag had no effect on the results (see Supplement for further discussion).

In conclusion, the relation between social media use and chronic inflammation was moderated by self-esteem. Given the prevalence of social media in our social lives and the importance of social relationships to physical health, more research is needed to understand how social media use relates to physical health. To this end, the present research demonstrates that the link between social media use and health may be nuanced and highlights the importance of identifying moderators at the individual difference level.

Disclosures

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Declaration of competing interest

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. The authors declare no competing interests (e.g., personal, financial, organizational) involved with this work.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.bbih.2021.100300.

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