

RUNNING HEAD: TIME SPENT CO-PRESENT WITH PARTNER AND C-REACTIVE
PROTEIN

Everyday co-presence with a romantic partner is associated with lower C-reactive protein

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Abstract

Social relationships are an important driver of health, and inflammation has been proposed as a key neurobiological mechanism to explain this effect. Behavioral researchers have focused on social relationship *quality* to further explain the association, yet recent research indicates that relationship quality may not be as robust a predictor as previously thought. Here, building on animal models of social bonds and recent theory on close relationships, we instead investigated merely being in the physical presence of one's romantic partner. Specifically, we tested the hypothesis that spending more time co-present with a loved partner in everyday life would be associated with lower c-reactive protein (CRP). Three times over the course of one month, 100 people in romantic relationships reported how much time they spent in the same physical space as their partner in the prior 24 hours, in minutes, and provided a sample of blood for CRP assay (n observations = 296). Results from multi-level models showed that when one reported spending more time in the physical presence of their partner they had lower CRP – an effect that was independent from social relationship quality explanations from the prior literature, including romantic relationship quality, hostility, and loneliness. These findings move past global assessments of social isolation to consider a novel everyday behavior that is of great interest in the non-human animal literature – spending time together -- as a potential mechanism linking high-quality relationships and physical health in adult humans. The findings also point to future research on additional behavioral mechanisms that are not dependent on stress pathways: people in high-quality relationships tend to spend enjoyable and affectionate time with one another, which may impact inflammation.

Keywords: inflammation, social relationships, C-reactive protein, social behavior, pair bond, relationship quality, physical proximity

Close social connections confer benefits to physical health that include reduced mortality rates (Holt-Lunstad et al., 2010). Inflammation is one widely-proposed biological pathway through which close social connections contribute to physical health and lower mortality (Kiecolt-Glaser et al., 2010; Leschak & Eisenberger, 2019; Uchino et al., 2018). Understandably, then, quite a bit of research has focused on how the *quality* of those relationships may affect systemic inflammation. For example, researchers have identified hostility (Brooks et al., 2014; Kiecolt-Glaser et al., 2005; Yang et al., 2014), perceptions of support (Jiang et al., 2021; Kiecolt-Glaser et al., 2010; Lee & Way, 2019; Uchino et al., 2018), and even loneliness (Hawkley & Cacioppo, 2003) as being associated with inflammation. Critically, recent work has failed to replicate the link between distress within romantic relationships and systemic inflammation (Bajaj et al. 2016; Jaremka et al., 2020; Nilsson et al., 2020). Thus, exactly *how* close relationships could impact the immune system requires further investigation. Here, rather than quality, we focused on what we believed to be an empirically overlooked factor in the human literature on relationships and systemic inflammation: time spent in the physical presence of a loved partner.

There were many reasons we focused on physical co-presence. The first three relate to the fact that close relationship partners are a key structural element of humans' everyday lives. First, *how* people in non-distressed relationships tend to spend their time together involves a wide variety of relationship processes largely overlooked in the inflammation literature, which has tended to focus on distressing moments (Robles & Kiecolt-Glaser, 2003): relatively satisfying relationships are characterized by interactions that are positively-valenced and affectionate or caring, which may be salubrious in their own right (cf. Algoe, 2019). Second, growing evidence suggests that simply being physically co-present with a loved partner reduces

the need for vigilance to threats (Coan et al., 2006; 2017) and increases physiological regulation of the endocrine (i.e. hypothalamic-pituitary-adrenal axis or HPA) and autonomic pathways that influence peripheral inflammation (Beckes & Coan, 2011; Bourassa et al., 2019; Coan & Maresh, 2015; Cornelius et al., 2020; Gump et al., 2001; Han et al., 2021; Phillips et al., 2006). Third, although time spent physically present with a relationship partner has not been a focal relationship behavior in the human literature, it is regularly used as a marker of a close bond in the non-human animal literature (e.g., Harbart et al., 2020; Lim & Young, 2006; Silk, 2007; Williams et al., 1992), including the theorizing that this time spent co-present with a familiar (close) other helps explain reproductive fitness and longevity (Silk, 2007). In short, in *high-quality* relationships, people tend to simply spend more time with one another in a variety of ways (e.g., Chang, Way, Sheeran, Kurtz, Baucom, & Algoe, 2022), making time spent co-present one logical factor to take seriously as a potential explanatory mechanism linking close relationships and systemic inflammation.

Fourth, in humans, broad-based measures of social *isolation*, which are not specific to what happens within a given relationship but reflect a potential pattern of a lack of contact (that is, *lack* of time spent co-present) with other humans across relationships, have provided indirect evidence that this is a path worth pursuing. Specifically, social isolation has been associated with greater inflammatory markers, such as C-reactive protein (CRP) and interleukin-6 (IL-6) (Heffner et al., 2011; Smith et al., 2020; Yang et al., 2013).

In the everyday ebb and flow of ongoing adult relationships, people have discretion about how much time they spend in the physical presence of the partner. Here, we focused on time spent with romantic relationship partners because these social partners have a large opportunity for influence on proximal measures of health, such as systemic inflammation, due to the fact that

their social contact tends to be frequent and consequential. People are invested in and care about romantic partners (Algoe & Jolink, 2021), who are thus logically more likely to influence physiological patterns relative to many other members of one's social network. Regarding time spent co-present with a romantic partner, people can make dates, eat meals together, and even if living under the same roof, can choose to be home or not, or go to bed at the same time or not, as their partner. In non-clinically distressed or depressed samples of couples such as those represented in prior literature (Bourassa et al., 2019; Coan et al., 2006; 2017; Han et al., 2021), a romantic partner can represent a source of safety (Campa et al., 2009; Collins & Feeney, 2000; Eisenberger et al., 2011). Does more time spent co-present with romantic partners then translate to lower markers of chronic distress in the body?

In the current study, we tested the effect of reported time spent co-present with a romantic partner on systemic inflammation, measured using assay of CRP levels in blood, using the strength of a repeated measures design; we sampled both time spent with the partner and CRP three times across four weeks.¹ As an acute phase protein, CRP is synthesized and released in response to pathogens as well as psychological factors (Pepys & Hirschfield, 2003). CRP levels are not static; in studies of inflammatory clinical conditions, such as rheumatoid arthritis, intraindividual fluctuations in CRP have been evaluated as a marker of disease state and treatment response (England et al., 2019). There has been comparatively little investigation of everyday psychosocial influences on repeated measures of peripheral CRP in non-clinical samples, which is the focus of the present study. Sampling CRP at 3 time points increased

¹ Note that our theorizing is about high-quality, loving relationships providing a source of safety. Whereas there is a large body of clinical literature on distressed couples (cf. Baucom et al., 1998; Christensen et al., 2004), people who sign up for non-therapeutic studies like this tend to be in loving, satisfied romantic relationships. Thus, the sample was expected to meet the threshold of our theoretical assumption.

statistical power to test the association using multi-level statistical modeling and provided the opportunity to assess associations between intraindividual deviations in co-presence with CRP.

Regarding the measure of time spent co-present, participants reported time spent in the physical presence of their partner over the 24 hours prior to the blood sample collection. We believed a 24-hour recall would be more accurate than estimating time spent throughout the prior week. Additionally, this 24-hour recall was proximal to the participant's blood draw and a reasonable approximation of the participant's typical pattern of behavior, especially as one of multiple measurement points. Finally, this 24-hour time point shows good correspondence with the kinetics of CRP. Across laboratory manipulations of systemic inflammation using either lipopolysaccharide injection (Heinzl et al., 2020; Hudgins et al., 2003; Mehta et al., 2010) or Salmonella typhi vaccination (Padfield et al., 2010; Paine et al., 2013), CRP levels consistently reach their peak 24 hours after injection. To assess the relative importance of physical co-presence with the partner, we also ran analyses controlling for weekly relationship satisfaction, hostility toward the partner, and loneliness; in addition to addressing prior findings in the literature on social relationship quality, relationship satisfaction and hostility can be viewed as proxy measures for quality of time spent together. Finally, we tested the reverse-causal explanatory pathway.

Methods

Participants

Individuals ($N = 159$, ages *range* = 18-55) who had been in a romantic relationship for at least six months were recruited from the greater Chapel Hill, North Carolina area for a study examining “Everyday Social Behavior and Health”, conducted October – December 2017. Recruitment and enrollment were conducted online via Informational Email to staff and students of UNC-Chapel Hill, Researchmatch.com, jointheconquest.com, and flyers. Interested

participants completed an online screening questionnaire that determined eligibility and obtained initial consent. Participants had to be at least 18 years of age but younger than 60, and were excluded if they were pregnant or nursing (currently or within the past six months), or currently had any of the following: arthritis, rheumatoid arthritis or joint problems, an immune disorder that might lead to immunodeficiency such as HIV, an auto-immune disorder, a chronic disease of the endocrine system (e.g., Cushing's Disease), a psychiatric disorder other than depression or anxiety, a diagnosed sleep disorder, or were currently diabetic. That is, participants were *not* recruited from a clinical population and did not have a history of pathologies related to immune dysfunction. Participants were excluded if they were currently or regularly taking anti-inflammatory medication, such as Tylenol, Ibuprofen and low-dose aspirin. They were also excluded if they indicated they had six or more alcoholic drinks on one occasion "twice a week" or more, as well as if they indicated using marijuana "several times a week" or more. Finally, tobacco smokers were excluded from the study.

Based on an administrative error, participants who considered themselves to be in a long distance relationship were admitted into the study. Because study hypotheses about inflammation are contingent upon participants interacting with their partner *in person*, at the final in-lab visit we asked two questions assessing whether the participant had had the opportunity to be physically present with their partner: 1) "Are you in a long distance relationship?" (Answer options: *yes* or *no*); 2) "Does your partner typically live in this area? (Greater Research Triangle area including Chapel Hill, Durham, Raleigh, and Carrboro)?" (Answer options: *yes* or *no*). To be conservative, we only included people who indicated that they were both *not* in a long distance relationship *and* that their partner lived locally ($N = 100$). All other responses ($N = 59$) were excluded from analyses. In an *a priori* power analysis conducted using G*Power, a target

sample size of 98 participants was estimated to have 80% power to detect a small effect ($f = 0.1$) across three repeated measures. See Table 1 for descriptive characteristics of the final sample of 100 participants. Demographic characteristics of the full sample and exploratory analyses using the full sample can both be found in the Supplemental Material (SM).

Table 1

Sample Characteristics.

	<i>M (SD)</i>	<i>% (n)</i>
Age	25.45 (8.13)	
Biologically Female		82% (82)
BMI	24.38 (4.07)	
Race/Ethnicity ¹		
White/Caucasian		82% (82)
Black/African American		6% (6)
Hispanic		2% (2)
Latino		2% (2)
East Asian		8% (8)
South Asian		6% (6)
Pacific Islander or Native Hawai'ian		1% (1)
Education Level ²		
High school graduation or equivalent		5% (5)
Some college		46% (46)
College graduation		33% (33)
Professional/post-graduate degree		16% (16)

¹Groups are not mutually exclusive as participants could endorse more than one race/ethnicity.

²We note education level may be confounded with age in this sample ($r = .71, p < .001$)

Procedure

Eligible participants were scheduled for a set of three in-lab appointments, at study entry (Time 1), two weeks later (Time 2), and again four weeks later (Time 3). Any given participant came at the same time of day and day of the week for all three of their appointments (e.g., Tuesdays at 10am). The primary purpose of the lab visits was to obtain a blood sample to measure systemic inflammation, as indexed by C-reactive protein (CRP). Due to a lack of consistent evidence for diurnal variability in CRP within blood (e.g., Meier-Ewert et al., 2001; Miles et al., 2008; Mills et al., 2009), session times were offered between 8am and 8pm.

Participants attended their lab sessions between Oct 30, 2017 and Dec 3, 2017 or between Nov 13, 2017 and Dec 17, 2017.

At each appointment, the participant was greeted by an experimenter who took the participant's height and weight, pricked the participant's finger with a small lancet, and collected drops of blood on a protein saver card. Then, the participant was escorted to another private lab room to complete confidential online questionnaires. These questionnaires assessed factors that may have influenced blood work that day (e.g., medication use), then behavioral and psychological factors of interest for the research questions. This included the key independent measure of how much time the participant spent in the physical presence of their partner in the prior 24 hours. The visit typically took 20-30 minutes. The day prior to attending the first and final lab sessions, participants completed longer online questionnaires; they also completed brief online questionnaires at weeks 1 and 3 from home; these additional measures are not relevant to the present study (Algoe, 2022). Of the 100 eligible participants who attended the first lab visit, only one participant did not attend or provide survey data at the second lab visit and all 100 attended the third lab visit (299 viable in-lab visits/surveys total).

Measures

Total time spent co-present with partner in past 24 hours. At each measurement point, participants estimated the amount of time spent co-present with their partner in the past 24 hours (i.e., "you were in the same room with the person, whether awake or sleeping"). Time spent co-present ranged from 0 – 1440 minutes (M time 1 = 490.1 minutes; M time 2 = 521 minutes; M time 3 = 545.7 minutes). Two reports were missing data on this item ($n = 297$).

Social quality alternative explanations. At each time point, we assessed three constructs of interest in the broader literature on relationships and inflammation: relationship quality,

hostility, and loneliness. To assess *relationship quality*, participants reported how terrible (1) to terrific (9) their relationship with their partner was, “on average, over this past week”.

Confirming our assumption that these were individuals in satisfied relationships, the average rating across all people at all time points was 7.43 ($SD = 1.5$); the modal response was 8 ($N = 96$ out of 298). *Hostility* was measured with three items, measured from not at all (0) to very much (6): “I fought with my partner this week”; “I was upset with my partner this week”; “At times this week, I felt like screaming at my partner”. On average, hostility was rather low at 1.02 ($SD = 1.28$) across all people at all time points. Participants also reported on feelings of *loneliness* in the past week ($M = 1.55$, $SD = 0.56$), with the average of three items measured from hardly ever (1), some of the time (2), and often (3): “How often do you feel that you lack companionship?”; “How often do you feel left out?”; “How often do you feel isolated from others?”. We tested models controlling for these variables. One report was missing responses to all alternative explanation items ($n = 298$).

Covariates. We controlled for sociodemographic and health factors known to be associated with inflammation. All analyses controlled for biological sex, age, baseline BMI and over-the-counter (OTC) medicine use prior to providing their blood sample, specifically probing, “did [the participant] take over-the-counter medications for a cold, flu, or any infection in the last 24 hours?” (O’Connor et al., 2009).² No data were missing from any covariate measure except the use of over-the-counter medicine at the second lab visit, of which a response was missing from one report ($N = 298$).

² Over-the-counter medicines were reported on only 17 of 298 occasions; medications are detailed in SM. Note that participants were screened out for *regular* use of anti-inflammatory medication; this variable was to control for *occasional* use of medication on appointment days.

We explored two additional sets of covariates. In the first, we explored covariates of race/ethnicity, contraceptive use, and use of anti-depressants. In the second, we explored whether participants exercised the day of the blood collection (“have you done any aerobic exercise today (e.g., jogging, tennis, karate?”, *yes*=1 or *no*=0) and sleep quality from the night prior to the blood collection (“how well did you sleep last night?” from *extremely poorly*=1 to *extremely well*=10).

C-reactive protein. C-reactive protein (CRP) was measured via dried blood spots, a method which has shown excellent correspondence with CRP concentrations assayed via traditional venipuncture (McDade et al., 2004). For collection, the experimenter swabbed the participant’s finger with alcohol, then pricked it with an 18-gauge needle (Owen Mumford Unistick 3). Blood drops were collected on a Whatman 903 Protein Saver Card. Samples were dried for 24 hours, then punched using a 3mm Biopsy Punch (Henry Schein) and stored in microcentrifuge tubes at -80°C until assay. Samples were shipped on dry ice to the Analytical and Development Laboratory at the Ohio State University (<https://ccts.osu.edu/content/ccrm-crc-analytical-specimen-labs>) for analysis. Following procedures from McDade, Burhop, & Dohnal (2004), a single 3mm punch was thawed and 200µL of buffer (phosphate-buffered saline with 0.1 percent Tween 20) was added, followed by overnight (~16 hours) incubation at 4°C while shaking at 60 rpm. The following morning, eluate was diluted 1:10 and two separate 25µl aliquots (due to experimenter error, one sample was not processed in duplicate) were assayed according to the manufacturer’s instructions using the Meso Scale Delivery Vplex Plus Kits (K151STG). Manufacturer-provided low and high standards were run in each of the 12 plates. Across all 12 plates, the intraassay coefficient of variation was 1.95%, while the interassay coefficient of variation was 3.24%. Blood was not attained for one participant at the third lab visit. All processed samples were successfully assayed and were well within the linear range

(across all plates, the lowest sample averaged 3.19 the lowest standard). Additionally, two CRP values greater than 10 ug/mL were removed from analyses, as they may indicate an acute infection (Pearson et al., 2003),³ resulting in 296 total observations of CRP. As is common with inflammatory markers (Jaremka et al., 2020; Lee & Way, 2019; Nilsson et al., 2020), CRP values were right skewed, so the variable was log-transformed before analyses. CRP was reliably and positively associated with BMI at every time point: Time 1 $r = .33$, $p < .001$; Time 2 $r = .28$, $p = .006$; Time 3 $r = .30$, $p = .002$.

Data analysis plan

This was a within-subjects design with three repeated measures, so we used multilevel analyses to test the association between time spent in the physical presence of the partner and CRP. Linear mixed models were conducted using lmer from the lme4 package in R (Bates et al., 2014). All models used maximum likelihood estimates where intercepts were allowed to vary randomly, but slopes were fixed. Effect sizes (r) for individual coefficients for each time spent or social relationship quality variable presented in main text were calculated based on the method used by Kashdan and Steger (2006): $r = \sqrt{t^2 / (t^2 + df)}$. Full model results can be found in SM.

In addition to the models using the raw time spent value, to further facilitate interpretation using this powerful inferential design, we probed week-to-week fluctuations of time spent. Specifically, the average time spent across the three time points was calculated for each participant. That value – the participant’s grand mean – was then subtracted from each time point’s time spent values (Paccagnella, 2006) using the center function in the misty package in R (Yanagida, 2020). These three new values reflected the amount of time spent relative to the participant’s average across the three measurements, such that positive values reflected having

³ Sensitivity analyses including individuals with high CRP (>10 ug/mL) can be found in Supplemental Material.

spent *more* time with their partner than the participant's own average and negative values reflected having spent *less* time with their partner than their own average. The statistics from these supplementary models are reported in the SM. Finally, we explored whether a specific portion of the time spent together – time spent co-sleeping – predicted CRP; those additional models are reported in the SM.

Results

See Table 2 for zero-order correlations of main study variables and covariates.

Table 2

Bivariate Correlations with Time Spent Co-Present, CRP, and Sociodemographic Covariates.

	1	2	3	4	5	6	7	8	9
1. CRP Time 1	--								
2. CRP Time 2	.82***	--							
3. CRP Time 3	.82***	.82***	--						
4. Time spent T1	.01	.04	.04	--					
5. Time spent T2	-.02	-.06	-.07	.63***	--				
6. Time spent T3	.02	.02	-.07	.52***	.54***	--			
7. Biological sex	.20*	.17	.21*	.02	-.07	-.05	--		
8. Age	.12	.14	.10	.25*	.17	.19	.06	--	
9. BMI	.33***	.28**	.30**	.04	.04	-.08	-.07	.39***	--

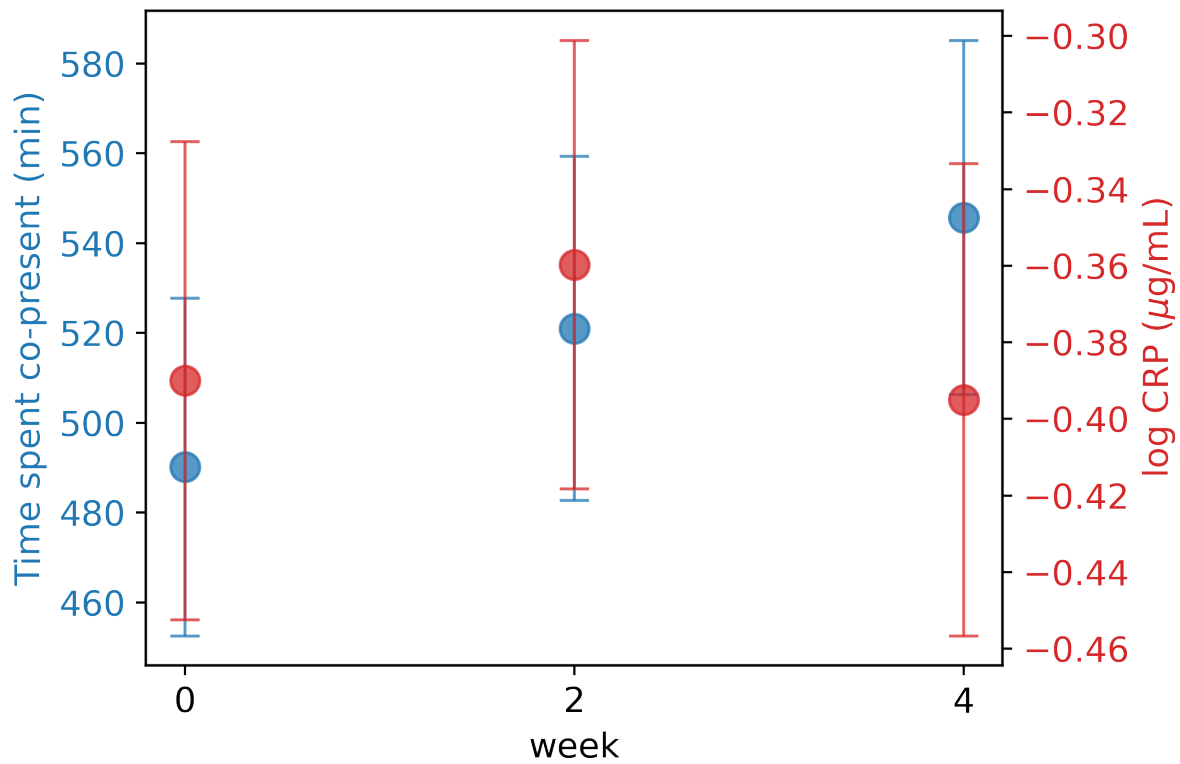
* $p < .05$, ** $p < .01$, *** $p < .001$

Note: Time spent are raw values, in minutes. CRP are log-transformed. T = time point.

Is time spent in the physical presence of the partner associated with CRP?

Consistent with our hypotheses, participants who spent more time co-present with their partner in the prior 24 hours had lower CRP, $b = -.0001$, $r = 0.13$, $p = .043$, CI95% [-.0003, -.00001]. The model controlled for standard sociodemographic and health covariates of biological sex, age, BMI, and recent over-the-counter medicine use. Additionally, results remained consistent when also accounting for race and/or ethnicity, anti-depressant use, and birth control use, as well as when controlling for exercise and sleep quality. See SM for full statistical report. See Figure 1 for a dual axis plot of means at each time point.

Figure 1



Note. Dual-axis plot depicting mean levels of raw time spent co-present (in minutes) and log-transformed CRP values at each time point. Error bars represent the unbiased standard error of the mean.

Weekly fluctuations of time spent. In supplementary analyses, we found similar results using mean-centered deviation scores, where the value of time spent reflected participant's *deviation* from their overall average. Participants who spent more time spent co-present with their partner in the prior 24 hours, beyond their average across three time points, had significantly lower CRP, $b = -.0002$, $r = 0.16$, $p = .029$, CI95% [-.0003, -.00002]. Models once again controlled for biological sex, age, BMI, and recent over-the-counter medicine use, and held when also accounting for race and/or ethnicity, anti-depressant use, and birth control use, and additionally when controlling for exercise and sleep quality. See SM for full model results.

Testing the reverse pathway: inflammation to time spent co-present. We did not find evidence of the reverse direction: CRP did not predict the time spent together in the prior 24 hours, accounting for biological sex, age, BMI and over-the-counter medicine use (see SM).

Addressing social quality alternative explanations: relationship quality, hostility toward partner, and loneliness

The association between total time spent co-present and CRP robustly held when controlling for each social relationship quality alternative explanation. Time spent co-present significantly negatively predicted CRP, $b = -.0001$, $r = .13$, $p = .04$, CI95% [-.0003, -.00001], when controlling for relationship quality that week, $b = .01$, $r = .03$, $p = .67$, CI95% [-.02, .04]. Time spent co-present was significantly negatively associated with CRP, $b = -.0001$, $r = .12$, $p = .05$, CI95% [-.0003, .0000001], when controlling for hostility toward the partner that week, $b = -.04$, $r = .13$, $p = .04$, CI95% = [-.07, -.002]. Lastly, time spent co-present was significantly negatively associated with CRP, $b = -.0001$, $r = .13$, $p = .032$, CI95% [-.0003, -.00001], when controlling for loneliness that week, $b = -.06$, $r = .08$, $p = .20$, CI95% [-.15, .03]. Each model controlled for biological sex, age, BMI and over-the-counter medicine use. Full statistics can be found in the SM, including main effects models of each alternative explanation predicting CRP without time spent in the model.

Weekly fluctuations of time spent. Supplementary analyses showed that, at a given time point, more time spent co-present with one's partner relative to one's own average was negatively significantly associated with CRP, when controlling for weekly relationship quality, hostility toward the partner, or loneliness. Full model results, including standard covariates, can be found in SM.

Discussion

The present study examined the question of how time spent co-present with a romantic partner relates to systemic inflammation, measured with CRP. Specifically, for the first time to our knowledge, we showed that simply spending more time in the physical presence of a loved partner was associated with lower levels of CRP the next day. We showed this using three time points sampled from across the course of a month. Indeed, supplementary analyses showed that at assessments when the participant had spent more time with the partner than their own average, they had lower CRP. Moreover, we put time spent co-present head-to-head with commonly studied explanations for links between social relationships and inflammation in the health literature – relationship quality, hostility toward the partner, and loneliness – showing that total time spent co-present consistently predicted CRP, regardless of these other factors relating to social relationship quality. These findings reveal a largely unexplored potential pathway through which close relationships may affect health.

The findings for time spent co-present are largely consistent with the social isolation literature (Heffner et al., 2011; Smith et al., 2020; Yang et al., 2013) but push it further. First, using the context of one of humans' most important relationships – with a romantic partner – we showed in a fine-grained way that one possible mechanism for effects of isolation is *not* being physically co-present with people. More broadly, we emphasize that, whereas excellent work has demonstrated the potential buffering effects of a partner's presence on physiological outcomes during times of distress (e.g., Bourassa et al., 2019; Coan et al., 2006; Feeney & Kirkpatrick, 1996), here, we do not make the assumption that stress-buffering was the mechanism. For example, in addition to stress buffering that may happen throughout a 24-hour period, social baseline theory suggests that being alone *heightens* vigilance, whereas co-presence may be the

“baseline” optimal state (Gunnar et al., 1996; Heffner et al., 2011; Smith et al., 2020; Yang et al., 2013).

Research on positive interpersonal processes emphasizes that people in high-quality relationships (like the people in our sample) tend to have social interactions with one another that are emotionally positively-valenced and caring (not negative and hostile) (Algoe, 2019), which could be salubrious in their own right, through positive emotions (Cohen & Pressman, 2006; Folkman & Moskowitz, 2000; Pressman et al., 2019), physical affection (Holt-Lunstad et al., 2008; Thomas & Kim, 2021), or other unknown mechanisms. So, in addition to time spent co-present as a new potential avenue of inquiry in this literature, we believe these data push health researchers to carefully examine features of relationships that happen the most *frequently* in everyday life (e.g., shared laughter, calm or happy states). Even if the momentary impact of such features were to be less intense than that of distress or hostility, for example (see Baumeister et al. 2001), frequency should undergird the *cumulative impact* of being in a high-quality relationship on health; biological mechanisms stemming from such moments might include physiological benefits from affectionate touch or physiological attunement (e.g., while co-sleeping), among others.

We acknowledge that these findings were correlational, so although we hypothesize causality, we await stronger tests of the causal hypothesis. Further, theory and evidence could suggest the reverse direction explanation. For example, early theory suggested that the release of pro-inflammatory cytokines was associated with the prototypical “sickness behavior” of social withdrawal, and some human studies provided initial support for that using a broad array of relationship types (Eisenberger et al., 2009; 2010; Inagaki et al., 2012). However, newer theorizing suggests that whether one withdraws may depend on the specific social target, or who

the relationship partner is (Muscatell & Inagaki, 2021), with the potential for people to want to *approach* close partners, such as romantic partners. Indeed, heightened inflammation has been associated with or caused people to more readily approach close relationship partners (Inagaki et al., 2015; Jolink et al., 2021). Those new data would suggest that if inflammation was causing social behavior, one would expect to see a significantly *positive* association between CRP and time spent co-present with the partner, not the significant negative association that we show in the present study. Finally, the test of CRP predicting time spent with the partner was not significant. Altogether, we believe our theoretical explanation to be a better match to the present data than the reverse causal pathway, but we await further testing.

Our findings add to the evidence base regarding associations between various measures of social relationships and inflammation (Holt-Lunstad et al. 2010; Smith et al. 2020; Uchino et al 2018), yet stand in contrast to the prior focus on social relationship *quality* factors. Romantic relationship quality and loneliness were not associated with CRP in our sample, despite associations with inflammation in the prior literature (Bajaj et al. 2017; Gouin et al., 2016; Hawkey et al. 2007; Jaremka et al. 2013; Kiecolt-Glaser et al. 2010; Ross et al. 2017; Shankar et al 2011). Additionally, we were somewhat surprised to find that hostility significantly predicted CRP in the opposite direction as the prior literature would suggest, both with and without time spent co-present with the partner in the model: While much of the existing literature has shown hostility and strain in close relationships to be associated with greater inflammation (Brooks et al., 2014; Gouin et al., 2009; Kiecolt-Glaser et al., 2005; 2010; Yang et al., 2014), in this case, hostility was associated with lower inflammation (see Bajaj et al., 2016 for one similar finding). We note that other recent research focusing on negatively-valenced aspects of relationship functioning has also raised questions about the strength of associations with CRP (e. g., Jaremka

et al., 2020) or relevant moderating variables in the link between conflict and inflammation (e.g., synchrony in heartrate variability, Wilson et al. 2018). Moving forward, the results for hostility should be interpreted in the context of the present study, with the primary contextual factor being that these are quite satisfied couples. Hostility ratings were quite low (see Method); however, it is natural for people to get on one another's nerves and plenty of research from affective, clinical, and relationship science suggests that acknowledging negative emotions is healthy (Blackledge & Hayes, 2001; John & Gross, 2004; Overall & McNulty, 2017; Torre & Lieberman, 2018). We look forward to future work that unpacks the meaning of especially low self-reports of hostility (or conversely, modestly higher reports in this happy context), or what else might be happening for couples when hostility is at its nadir, as these insights might guide future predictions regarding inflammation.

We also draw attention to five opportunities for additional research. First, we believe the relationships of participants in this study cross a threshold for feelings of care and safety that underlies our theoretical assumption about the potential value of time spent co-present on inflammation. However, research in distressed couples remains warranted to further refine the theorizing: one possibility, drawn from social baseline theory (Beckes & Coan, 2011), is that even poor relationships still offer slightly more benefit than being alone. Alternatively, relationships with substantially greater stress and negative affect may produce the opposite to what was found here. That question needs to be tested empirically. Second, a related question is whether there are individual differences in the way people view their experiences with their partners, or even whether actual variability in the quality of the time spent together across different days, may moderate the association between time spent and CRP specifically, or another marker of inflammation. Third, the time spent co-present variable was self-reported by

participants. A prior study using this same measure independently reported by both couple members for 35 nights showed corroboration about the validity of participants' time estimates: there was minimal variance between partners in these reports (Chang et al., 2022). That said, there are likely other objective measures of time spent in physical co-presence that would help to augment future study designs. Fourth, inflammatory markers can be influenced by multiple factors. Though the effects held when adjusting for self-reported sleep and exercise, the measurement of these variables would also be enhanced by using objective measures. Similarly, we did not measure potential dietary contributions to these shifts over time (Shivappa, Steck, Hurley, Hussey, & Hébert, 2014). Fifth, these effects may not be unique to CRP, so future work should examine physical co-presence and other markers of inflammation to provide greater insight into the potential cellular pathways contributing to this effect as well as ensure that the effects on CRP are indeed due to peripheral inflammation and not another biological process (Del Giudice & Gangestad, 2018).

In conclusion, people with whom we are in close social relationships, such as a quality romantic partner, are who we want to laugh with, who we want to hug, or who we choose to sit in silence and stillness next to at the end of the day. Enduring, elevated systemic inflammation, as reflected by continued production of higher CRP levels, can produce poor health outcomes (Ershler & Keller, 2000; Kiecolt-Glaser et al., 2010; Ridker, 2009). We sampled CRP on three different days across time, and found evidence suggesting merely being together with a romantic partner was beneficial in the form of lower CRP. By identifying this proximal biological pathway through which being with our closest others may facilitate better health outcomes, these findings reveal yet uncharted avenues for addressing the mechanisms through which close relationships affect long-term health.

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References

- Algoe, S. B. (2019). Positive interpersonal processes. *Current Directions in Psychological Science*, 28(2), 183-188.
- Algoe, S. B. (2022). Everyday social behavior and health, 2017. *UNC Dataverse*, VI.
<https://doi.org/10.15139/S3/1NRDMW>
- Algoe, S. B., & Jolink, T. A. (2020). Social bonds: A new look at an old topic. *Social psychology: Handbook of basic principles*, 140-162.
- Bajaj, A., John-Henderson, N. A., Cundiff, J. M., Marsland, A. L., Manuck, S. B., & Kamarck, T. W. (2016). Daily social interactions, close relationships, and systemic inflammation in two samples: Healthy middle-aged and older adults. *Brain, behavior, and immunity*, 58, 152-164.
- Bates, D., Mächler, M., Bolker, B., & Walker, S. (2014). Fitting linear mixed-effects models using lme4. *arXiv preprint arXiv:1406.5823*.
- Baucom, D. H., Shoham, V., Mueser, K. T., Daiuto, A. D., & Stickle, T. R. (1998). Empirically supported couple and family interventions for marital distress and adult mental health problems. *Journal of consulting and clinical psychology*, 66(1), 53.
- Baumeister, R.F., Bratslavsky, E., Finkenauer, C., & Vohs, K.D. (2001). Bad is stronger than good. *Review of general psychology*, 5(4), 323-370.
- Beckes, L., & Coan, J. A. (2011). Social baseline theory: The role of social proximity in emotion and economy of action. *Social and Personality Psychology Compass*, 5(12), 976-988.
- Blackledge, J. T., & Hayes, S. C. (2001). Emotion regulation in acceptance and commitment therapy. *Journal of clinical psychology*, 57(2), 243-255.

- Bourassa, K. J., Ruiz, J. M., & Sbarra, D. A. (2019). The impact of physical proximity and attachment working models on cardiovascular reactivity: Comparing mental activation and romantic partner presence. *Psychophysiology*, *56*(5), e13324.
- Brooks, K. P., Gruenewald, T., Karlamangla, A., Hu, P., Koretz, B., & Seeman, T. E. (2014). Social relationships and allostatic load in the MIDUS study. *Health Psychology*, *33*, 1373–1381.
- Chang, Y-P., Way, B. M., Sheeran, P., Kurtz, L. E., Baucom, D. H., & Algoe, S. B. (2022). Promoting expressed gratitude in one member of a romantic couple increases time they spend together in everyday life. *Scientific Reports*
- Christensen, A., Atkins, D. C., Berns, S., Wheeler, J., Baucom, D. H., & Simpson, L. E. (2004). Traditional versus integrative behavioral couple therapy for significantly and chronically distressed married couples. *Journal of consulting and clinical psychology*, *72*(2), 176.
- Coan, J. A., Beckes, L., Gonzalez, M. Z., Maresh, E. L., Brown, C. L., & Hasselmo, K. (2017). Relationship status and perceived support in the social regulation of neural responses to threat. *Social Cognitive and Affective Neuroscience*, *12*(10), 1574-1583.
- Coan, J. A., Schaefer, H. S., & Davidson, R. J. (2006). Lending a hand: Social regulation of the neural response to threat. *Psychological science*, *17*(12), 1032-1039.
- Coan, J., & Maresh, E. L. (2014). Social baseline theory and the social regulation of emotion. In J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed.). New York, NY: Guilford Press.
- Cohen, S., & Pressman, S. D. (2006). Positive affect and health. *Current directions in psychological science*, *15*(3), 122-125.

- Cornelius, T., Birk, J. L., Edmondson, D., & Schwartz, J. E. (2020). Ambulatory Blood Pressure Response to Romantic Partner Interactions and Long-Term Cardiovascular Health Outcomes. *Psychosomatic medicine*, 82(4), 393.
- Eisenberger, N.I., Master, S. L., Inagaki, T.K., Taylor, S.E., Shirinyan, D., Lieberman, M.D., & Naliboff, B D. (2011). Attachment figures activate a safety signal-related neural region and reduce pain experience. *Proceedings of the National Academy of Sciences*, 108(28), 11721-11726.
- Eisenberger, N. I., Moieni, M., Inagaki, T. K., Muscatell, K. A., & Irwin, M. R. (2017). In sickness and in health: the co-regulation of inflammation and social behavior. *Neuropsychopharmacology*, 42(1), 242-253.
- Eisenberger, N.I., Inagaki, T.K., Mashal, N.M., Irwin, M.R. (2010). Inflammation and social experience: an inflammatory challenge induces feelings of social disconnection in addition to depressed mood. *Brain, behavior, and Immunity*, 24 (4), 558–563.
[https://doi.org/ 10.1016/j.bbi.2009.12.009](https://doi.org/10.1016/j.bbi.2009.12.009).
- Eisenberger, N.I., Inagaki, T.K., Rameson, L.T., Mashal, N.M., & Irwin, M.R. (2009). An fMRI study of cytokine-induced depressed mood and social pain: the role of sex differences. *Neuroimage*, 47(3), 881–890. <https://doi.org/10.1016/j.neuroimage.2009.04.040>
- England, B. R., Tiong, B. K., Bergman, M. J., Curtis, J. R., Kazi, S., Mikuls, T. R., O’Dell, J. R., Ranganath, V. K., Limanni, A., Suter, L. G., & Michaud, K. (2019). 2019 Update of the American College of Rheumatology Recommended Rheumatoid Arthritis Disease Activity Measures. *Arthritis Care & Research*, 71(12), 1540–1555.
<https://doi.org/10.1002/acr.24042>

- Ershler, W. B., & Keller, E. T. (2000). Age-associated increased interleukin-6 gene expression, late-life diseases, and frailty. *Annual Review of Medicine*, 51, 245–270.
<http://dx.doi.org/10.1146/annurev.med.51.1.245>
- Feeney, B. C., & Kirkpatrick, L. A. (1996). Effects of adult attachment and presence of romantic partners on physiological responses to stress. *Journal of personality and social psychology*, 70(2), 255.
- Folkman, S., & Moskowitz, J. T. (2000). Stress, positive emotion, and coping. *Current directions in psychological science*, 9(4), 115-118.
- Gassen, J., & Hill, S. E. (2019). Why inflammation and the activities of the immune system matter for social and personality psychology (and not only for those who study health). *Social and Personality Psychology Compass*, 13(6), e12471.
- Gouin, J. P., Glaser, R., Loving, T. J., Malarkey, W. B., Stowell, J., Houts, C., & Kiecolt-Glaser, J. K. (2009). Attachment avoidance predicts inflammatory responses to marital conflict. *Brain, behavior, and immunity*, 23(7), 898-904.
- Gouin, J. P., Scarcello, S., da Estrela, C., Paquin, C., & Barker, E. T. (2016). Dyadic coping and inflammation in the context of chronic stress. *Health Psychology*, 35(10), 1081.
- Gump, B. B., Polk, D. E., Kamarck, T. W., & Shiffman, S. M. (2001). Partner interactions are associated with reduced blood pressure in the natural environment: Ambulatory monitoring evidence from a healthy, multiethnic adult sample. *Psychosomatic medicine*, 63(3), 423-433.
- Gunnar, M. R., Brodersen, L., Nachmias, M., Buss, K., & Rigatuso, J. (1996). Stress reactivity and attachment security. *Developmental psychobiology*, 29(3), 191-204.

- Han, S. C., Schacter, H. L., Timmons, A. C., Kim, Y., Sichko, S., Pettit, C., ... & Margolin, G. (2021). Romantic partner presence and physiological responses in daily life: Attachment style as a moderator. *Biological Psychology*, 161, 108082.
- Harbert, K. J., Pellegrini, M., Gordon, K. M., & Donaldson, Z. R. (2020). How prior pair-bonding experience affects future bonding behavior in monogamous prairie voles. *Hormones and Behavior*, 126, 104847.
- Hawkey, L. C., & Cacioppo, J. T. (2003). Loneliness and pathways to disease. *Brain, behavior, and immunity*, 17(1), 98-105.
- Hawkey, L. C., Bosch, J. A., Engeland, C. G., Marucha, P. T., & Cacioppo, J. T. (2007). Loneliness, dysphoria, stress, and immunity: A role for cytokines. In N. P. Plotnikoff, R. E. Faith, A. J. Murgu, R. A. Good (Eds.), *Cytokines: Stress and immunity* (2nd ed., pp. 67–85). Boca Raton, FL: CRC Press.
- Heffner, K. L., Waring, M. E., Roberts, M. B., Eaton, C. B., & Gramling, R. (2011). Social isolation, C-reactive protein, and coronary heart disease mortality among community-dwelling adults. *Social science & medicine*, 72(9), 1482-1488.
- Heinzl, M. W., Resl, M., Klammer, C., Egger, M., Dieplinger, B., & Clodi, M. (2020). Proprotein convertase subtilisin/kexin type 9 (PCSK9) is not induced in artificial human inflammation and is not correlated with inflammatory response. *Infection and Immunity*, 88(3), e00842-19.
- Hennessy, M. B., Deak, T., & Schiml, P. A. (2014). Sociality and sickness: have cytokines evolved to serve social functions beyond times of pathogen exposure?. *Brain, behavior, and immunity*, 37, 15-20.

- Holt-Lunstad, J., Birmingham, W. A., & Light, K. C. (2008). Influence of a “warm touch” support enhancement intervention among married couples on ambulatory blood pressure, oxytocin, alpha amylase, and cortisol. *Psychosomatic medicine*, 70(9), 976-985.
- Holt-Lunstad, J., Smith, T. B., & Layton, J. B. (2010). Social relationships and mortality risk: A meta-analytic review. *PLoS Medicine*, 7, e1000316.
<http://dx.doi.org/10.1371/journal.pmed.1000316>
- Hudgins, L. C., Parker, T. S., Levine, D. M., Gordon, B. R., Saal, S. D., Jiang, X., Seidman, C. E., Tremaroli, J. D., Lai, J., & Rubin, A. L. (2003). A single intravenous dose of endotoxin rapidly alters serum lipoproteins and lipid transfer proteins in normal volunteers. *Journal of Lipid Research*, 44(8), 1489–1498.
- Inagaki, T.K., Muscatell, K.A., Irwin, M.R., Cole, S.W., Eisenberger, N.I., 2012. Inflammation selectively enhances amygdala activity to socially threatening images. *Neuroimage*, 59(4), 3222–3226. <https://doi.org/10.1016/j.neuroimage.2011.10.090>.
- Inagaki, T.K., Muscatell, K.A., Irwin, M.R., Moieni, M., Dutcher, J.M., Jevtic, I., Breen, E.C., & Eisenberger, N.I. (2015). The role of the ventral striatum in inflammatory-induced approach toward support figures. *Brain, behavior, and immunity*, 44, 247–252.
<https://doi.org/10.1016/j.bbi.2014.10.006>.
- Irwin, M. R., Olmstead, R., & Carroll, J. E. (2016). Sleep disturbance, sleep duration, and inflammation: a systematic review and meta-analysis of cohort studies and experimental sleep deprivation. *Biological psychiatry*, 80(1), 40-52.
- Jackowska, M., Kumari, M., & Steptoe, A. (2013). Sleep and biomarkers in the English Longitudinal Study of Ageing: associations with C-reactive protein, fibrinogen,

- dehydroepiandrosterone sulfate and hemoglobin. *Psychoneuroendocrinology*, 38(9), 1484-1493.
- Jaremka, L. M., Fagundes, C. P., Peng, J., Bennett, J. M., Glaser, R., Malarkey, W. B., & Kiecolt-Glaser, J. K. (2013). Loneliness promotes inflammation during acute stress. *Psychological Science*, 24(7), 1089-1097.
- Jaremka, L. M., Kane, H. S., Sunami, N., Lebed, O., & Austin, K. A. (2020). Romantic relationship distress, gender, socioeconomic status, and inflammation: A preregistered report. *Personal Relationships*, 27(3), 708-727.
- Jiang, T., Yakin, S., Crocker, J., & Way, B. M. (2021). Perceived Social Support-Giving Moderates the Association Between Social Relationships and Interleukin-6. *Brain, behavior, and immunity*.
- John, O. P., & Gross, J. J. (2004). Healthy and unhealthy emotion regulation: Personality processes, individual differences, and life span development. *Journal of personality*, 72(6), 1301-1334.
- Jolink, T. A., Fendinger, N. J., Alvarez, G. M., Feldman, M. J., Gaudier-Diaz, M. M., & Muscatell, K. A. (2021). Inflammatory Reactivity to the Influenza Vaccine is Associated with Changes in Automatic Social Behavior. *Brain, behavior, and immunity*.
- Kashdan, T. B., & Steger, M. F. (2006). Expanding the topography of social anxiety: An experience-sampling assessment of positive emotions, positive events, and emotion suppression. *Psychological Science*, 17(2), 120–128. <https://doi.org/10.1111/j.1467-9280.2006.01674.x>

- Kiecolt-Glaser, J. K., Gouin, J. P., & Hantsoo, L. (2010). Close relationships, inflammation, and health. *Neuroscience and Biobehavioral Reviews*, *35*, 33–38.
<http://dx.doi.org/10.1016/j.neubiorev.2009.09.003>
- Kiecolt-Glaser, J. K., Loving, T. J., Stowell, J. R., Malarkey, W. B., Lemeshow, S., Dickinson, S. L., & Glaser, R. (2005). Hostile marital interactions, proinflammatory cytokine production, and wound healing. *Archives of General Psychiatry*, *62*, 1377–1384.
- Kiecolt-Glaser, J. K., Loving, T. J., Stowell, J. R., Malarkey, W. B., Lemeshow, S., Dickinson, S. L., & Glaser, R. (2005). Hostile marital interactions, proinflammatory cytokine production, and wound healing. *Archives of General Psychiatry*, *62*, 1377–1384.
- Lavie, C. J., Church, T. S., Milani, R. V., & Earnest, C. P. (2011). Impact of physical activity, cardiorespiratory fitness, and exercise training on markers of inflammation. *Journal of cardiopulmonary rehabilitation and prevention*, *31*(3), 137-145.
- Lee, D. S., & Way, B. M. (2019). Perceived social support and chronic inflammation: The moderating role of self-esteem. *Health Psychology*, *38*(6), 563.
- Leschak, C. J., & Eisenberger, N. I. (2019). Two distinct immune pathways linking social relationships with health: inflammatory and antiviral processes. *Psychosomatic medicine*, *81*(8), 711.
- Lim, M. M., & Young, L. J. (2006). Neuropeptidergic regulation of affiliative behavior and social bonding in animals. *Hormones and behavior*, *50*(4), 506-517.
- McDade, T. W., Burhop, J., & Dohnal, J. (2004). High-sensitivity enzyme immunoassay for C-reactive protein in dried blood spots. *Clinical Chemistry*, *50*(3), 652–654.
- Mehta, N. N., McGillicuddy, F. C., Anderson, P. D., Hinkle, C. C., Shah, R., Pruscino, L., Tabita-Martinez, J., Sellers, K. F., Rickels, M. R., & Reilly, M. P. (2010). Experimental

- endotoxemia induces adipose inflammation and insulin resistance in humans. *Diabetes*, 59(1), 172–181.
- Meier-Ewert, H. K., Ridker, P. M., Rifai, N., Price, N., Dinges, D. F., & Mullington, J. M. (2001). Absence of diurnal variation of C-reactive protein concentrations in healthy human subjects. *Clinical Chemistry*, 47(3), 426–430.
- Miles, M. P., Andring, J. M., Pearson, S. D., Gordon, L. K., Kasper, C., Depner, C. M., & Kidd, J. R. (2008). Diurnal variation, response to eccentric exercise, and association of inflammatory mediators with muscle damage variables. *Journal of Applied Physiology*, 104(2), 451–458.
- Mills, P. J., Natarajan, L., von Känel, R., Ancoli-Israel, S., & Dimsdale, J. E. (2009). Diurnal variability of C-reactive protein in obstructive sleep apnea. *Sleep and Breathing*, 13(4), 415–420.
- Muscatell, K. A., & Inagaki, T. K. (2021). Beyond social withdrawal: new perspectives on the effects of inflammation on social behavior. *Brain, Behavior, & Immunity-Health*, 16, 100302.
- Nilsson, C. J., Nørgaard, S., Foverskov, E., Bruunsgaard, H., Andersen, P. K., & Lund, R. (2020). Positive and negative aspects of social relations and low-grade inflammation in Copenhagen Aging and Midlife Biobank. *European Journal of Ageing*, 17(4).
- O'Connor, M. F., Bower, J. E., Cho, H. J., Creswell, J. D., Dimitrov, S., Hamby, M. E.,... Irwin, M. R. (2009). To assess, to control, to exclude: Effects of biobehavioral factors on circulating inflammatory markers. *Brain, Behavior, and Immunity*, 23, 887– 897.
<http://dx.doi.org/10.1016/j.bbi.2009.04.005>

- Overall, N. C., & McNulty, J. K. (2017). What type of communication during conflict is beneficial for intimate relationships?. *Current opinion in psychology, 13*, 1-5.
- Paccagnella, O. (2006). Centering or not centering in multilevel models? The role of the group mean and the assessment of group effects. *Evaluation review, 30*(1), 66-85.
- Padfield, G. J., Tura, O., Haeck, M. L., Short, A., Freyer, E., Barclay, G. R., ... & Mills, N. L. (2010). Circulating endothelial progenitor cells are not affected by acute systemic inflammation. *American Journal of Physiology-Heart and Circulatory Physiology, 298*(6), H2054-H2061.
- Paine, N. J., Ring, C., Bosch, J. A., Drayson, M. T., & van Zanten, J. J. V. (2013). The time course of the inflammatory response to the Salmonella typhi vaccination. *Brain, behavior, and immunity, 30*, 73-79.
- Patel, S. R., Zhu, X., Storfer-Isser, A., Mehra, R., Jenny, N. S., Tracy, R., & Redline, S. (2009). Sleep duration and biomarkers of inflammation. *Sleep, 32*(2), 200-204.
- Pearson, T. A., Mensah, G. A., Alexander, R. W., Anderson, J. L., Cannon, R. O., III, Criqui, M., . . . the Centers for Disease Control and Prevention, & the American Heart Association. (2003). Markers of inflammation and cardiovascular disease: Application to clinical and public health practice: A statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. *Circulation, 107*, 499–511. <http://dx.doi.org/10.1161/01.CIR.0000052939.59093.45>
- Pepys, M. B., & Hirschfield, G. M. (2003). C-reactive protein: a critical update. *The Journal of clinical investigation, 111*(12), 1805-1812.

- Phillips, A. C., Carroll, D., Hunt, K., & Der, G. (2006). The effects of the spontaneous presence of a spouse/partner and others on cardiovascular reactions to an acute psychological challenge. *Psychophysiology*, *43*(6), 633-640.
- Prather, A. A., Epel, E. S., Cohen, B. E., Neylan, T. C., & Whooley, M. A. (2013). Gender differences in the prospective associations of self-reported sleep quality with biomarkers of systemic inflammation and coagulation: findings from the Heart and Soul Study. *Journal of psychiatric research*, *47*(9), 1228-1235.
- Pressman, S. D., Jenkins, B. N., & Moskowitz, J. T. (2019). Positive affect and health: what do we know and where next should we go?. *Annual Review of Psychology*, *70*, 627-650.
- Ridker, P. M. (2009). C-reactive protein: Eighty years from discovery to emergence as a major risk marker for cardiovascular disease. *Clinical Chemistry*, *55*, 209 –215.
<http://dx.doi.org/10.1373/clinchem.2008.119214>
- Robles, T. F., & Kiecolt-Glaser, J. K. (2003). The physiology of marriage: Pathways to health. *Physiology & behavior*, *79*(3), 409-416.
- Ross, K. M., Miller, G., Qadir, S., Keenan-Devlin, L., Leigh, A. K., & Borders, A. (2017). Close relationship qualities and maternal peripheral inflammation during pregnancy. *Psychoneuroendocrinology*, *77*, 252-260.
- Shankar, A., McMunn, A., Banks, J., & Steptoe, A. (2011). Loneliness, social isolation, and behavioral and biological health indicators in older adults. *Health Psychology*, *30*(4), 377.
- Shivappa, N., Steck, S. E., Hurley, T. G., Hussey, J. R., & Hébert, J. R. (2014). Designing and developing a literature-derived, population-based dietary inflammatory index. *Public health nutrition*, *17*(8), 1689-1696.

- Silk, J. B. (2007). Social components of fitness in primate groups. *Science*, 317(5843), 1347-1351.
- Smith, K. J., Gavey, S., Riddell, N. E., Kontari, P., & Victor, C. (2020). The association between loneliness, social isolation and inflammation: a systematic review and meta-analysis. *Neuroscience & Biobehavioral Reviews*, 112, 519-541.
- Torre, J. B., & Lieberman, M. D. (2018). Putting feelings into words: Affect labeling as implicit emotion regulation. *Emotion Review*, 10(2), 116-124.
- Uchino, B. N., Trettevik, R., Kent de Grey, R. G., Cronan, S., Hogan, J., & Baucom, B. R. (2018). Social support, social integration, and inflammatory cytokines: A meta-analysis. *Health Psychology*, 37(5), 462.
- Williams, J. R., Carter, C. S., & Insel, T. (1992). Partner Preference Development in Female Prairie Voles Is Facilitated by Mating or the Central Infusion of Oxytocin. *Annals of the New York Academy of Sciences*, 652(1), 487-489.
- Wilson, S. J., Bailey, B. E., Jaremka, L. M., Fagundes, C. P., Andridge, R., Malarkey, W. B., ... & Kiecolt-Glaser, J. K. (2018). When couples' hearts beat together: Synchrony in heart rate variability during conflict predicts heightened inflammation throughout the day. *Psychoneuroendocrinology*, 93, 107-116.
- Yanagida, T., & Yanagida, M. T. (2020). Package 'misty'.
- Yang, Y. C., McClintock, M. K., Kozloski, M., & Li, T. (2013). Social isolation and adult mortality: the role of chronic inflammation and sex differences. *Journal of health and social behavior*, 54(2), 183-203.

Yang, Y. C., Schorpp, K., & Harris, K. M. (2014). Social support, social strain and inflammation: Evidence from a national longitudinal study of US adults. *Social science & medicine*, 107, 124-135.

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Method: Frequencies of Medication Use Across All Three Lab Visits

Of the 298 observations on anti-inflammation medication use, 17 observations indicated using over-the-counter over-the-counter medication within 24-hours of providing their blood sample; 281 observations indicated not using over-the-counter medicine. Responses ($n = 17$) that were coded as “over-the-counter” included Ibuprofen (1), NyQuil (3), Dayquil (2), Sudafed (2), Cetirizine (allergies) (1), Ricola/cough drops (2), Mucinex (2), Advil Congestion and Sinus (1), Aleve (1) anti-histamine (1), acetaminophen (1), Allegra D (1), Dextromethorphan (1), and a yeast infection pill (1). (On three occasions the participant reported taking two of these medications, so the frequencies in parentheses equal 20 rather than 17.)

Of 300 observations, 48 observations included reported anti-depressants use; 252 observations indicated no anti-depressant use. Of 300 observations, 153 observations indicated currently using birth control while the remaining 147 observations indicated *no* birth control use.

Supplementary Table 1: Correlation Table with Total Time Spent Co-Present, CRP, and Alternative Explanation Variables**Supplementary Table 1***Raw Bivariate Correlations for All Study Variables by Time Point.*

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. CRP Time 1	--														
2. CRP Time 2	.79***	--													
3. CRP Time 3	.82***	.81***	--												
4. Time spent co-present T1	.01	.09	.05	--											
5. Time spent co-present T2	-.02	-.02	-.07	.67***	--										
6. Time spent co-present T3	-.01	.02	-.07	.57***	.73***	--									
7. Relationship satisfaction T1	.09	.14	.12	.16*	.09	.11	--								
8. Relationship satisfaction T2	.10	.13	.11	.10	.08	.07	.62***	--							
9. Relationship satisfaction T3	.04	.06	.08	.14	.12	.10	.61***	.57***	--						
10. Loneliness T1	-.02	.06	-.03	-.13	-.08	.04	-.30**	-.27**	-.27**	--					
11. Loneliness T2	.04	.11	.07	-.07	-.11	-.03	-.32***	-.35***	-.29**	.62***	--				
12. Loneliness T3	-.05	.03	-.05	.04	.06	.08	-.32***	-.30**	-.29**	.75***	.64***	--			
13. Hostility T1	-.06	.02	-.004	.005	-.01	-.02	-.59***	-.26*	-.34***	.27**	.28**	.36***	--		
14. Hostility T2	-.13	-.12	-.10	.02	.08	.04	-.28**	-.57***	-.18	.22*	.24*	.23*	.46***	--	
15. Hostility T3	-.07	-.02	-.07	-.07	-.10	-.05	-.42***	-.33**	-.71***	.28**	.28**	.34***	.60***	.34***	--

* $p < .05$, ** $p < .01$, *** $p < .001$

Note: CRP is log-transformed variable. T = time point.

Supplementary Table 2: Descriptives of Main Study Variables**Supplementary Table 2***Means, SDs, and Ranges for All Study Variables by Time Point.*

	M (SD)	Min-Max
1. CRP Time 1	-0.39 (.57)	-2-.82
2. CRP Time 2	-0.33 (.59)	-1.83-0.96
3. CRP Time 3	-0.4 (.59)	-1.89-0.98
4. Time spent co-present T1	383.73 (415.32)	0-1440
5. Time spent co-present T2	392.19 (405.23)	0-1380
6. Time spent co-present T3	419.08 (433.04)	0-1440
7. Relationship satisfaction T1	7.26 (1.57)	2-9
8. Relationship satisfaction T2	7.46 (1.33)	3-9
9. Relationship satisfaction T3	7.46 (1.66)	1-9
10. Loneliness T1	1.74 (0.55)	1-3
11. Loneliness T2	1.33 (0.5)	1-3
12. Loneliness T3	1.59 (0.55)	1-3
13. Hostility T1	1.16 (1.38)	0-6
14. Hostility T2	0.98 (1.12)	0-4.7
15. Hostility T3	1 (1.31)	0-6

Note: CRP values log-transformed.

Supplementary Table 3: Primary Analyses

Supplementary Table 3 shows full model results for time spent co-present predicting CRP, controlling for standard covariates of biological sex, age, BMI, and over-the-counter (OTC) medicine use in the prior 24 hours.

Supplementary Table 3*Total Time Spent Co-Present Predicting CRP*

Predictors	<i>b</i> [95% CI]	<i>t</i>
Time spent co-present	-.0001 [-.0003, -.000004]	-2.03*
Biological sex	.34 [.07, .61]	2.47*
Age	.001 [-.01, .01]	0.09
BMI	.05 [.02, .07]	3.33**
OTC medicine use	.22 [.06, .37]	2.71**

* $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Tables 4 & 5: Primary Analyses Controlling for All Covariates

Supplementary Table 4 shows full model results for time spent co-present predicting CRP, controlling for all standard covariates (see Supplementary Table 3) as well as an additional set of exploratory covariates: race, birth control use, and anti-depressant use. Conclusions hold.

Next, Supplementary Table 5 show time spent co-present predicting CRP, controlling for standard covariates and a second set of exploratory covariates, two additional factors associated with inflammation in prior literature: sleep quality (Jackowska et al., 2013; Prather et al., 2013), here assessed from the night prior to the CRP measurement (measured 1 = *slept extremely poorly* to 10 = *slept extremely well*) and whether or not the participant had exercised the day of the CRP measurement (Lavie et al., 2011). Conclusions hold.

Supplementary Table 4

Total Time Spent Co-Present Predicting CRP, Controlling for First Set of Exploratory Covariates.

Predictors	<i>b</i> [95% CI]	<i>t</i>
Time spent co-present	-.0001 [-.0003, -.00001]	-2.14*
Biological sex	.10 [-.16, .36]	0.75
Age	.01 [-.01, .02]	0.89
BMI	.05 [.03, .08]	4.43***
OTC medicine use	.20 [.04, .36]	2.50*
White	.29 [-.08, .67]	1.54
Black	-.31 [-.73, .11]	-1.45
Hispanic	.78 [.05, 1.52]	2.11*
Latino	-.16 [-.90, .59]	-0.42
East Asian	-.13 [-.51, .24]	-0.68
South Asian	.43 [-.10, .95]	1.60
Hawaiian/Pacific Islander	-.45 [-1.40, .49]	-.95
Birth control use	.43 [.22, .63]	4.16***
Anti-depressant use	-.19 [-.43, .05]	-1.57

* $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Table 5

Total Time Spent Co-Present Predicting CRP, Controlling for Second Set of Exploratory Covariates.

Predictors	<i>b</i> [95% CI]	<i>t</i>
Time spent co-present	-.0001 [-.0003, .00003]	-2.00*
Biological sex	.33 [.06, .60]	2.39*
Age	.001 [-.01, .01]	0.12
BMI	.05 [.02, .07]	3.27**
OTC medicine use	.22 [.06, .38]	2.75**
Sleep quality prior night	-.001 [-.02, .02]	-0.13
Exercised today	-.09 [-.22, .04]	-1.41

* $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Tables 6-11: Analyses with Alternative Explanations: Relationship Quality, Hostility Toward the Partner, and Loneliness

Primary analyses (see Supplementary Table 3) were also conducted controlling for relationship quality, hostility with the partner, and loneliness. Supplementary Tables 5-7 first establish the effect of each alternative explanation from the prior literature, showing no associations between relationship quality and loneliness with CRP, but a significant negative association between hostility and CRP. Supplementary Tables 8-10 show primary analysis results of time spent co-present predicting CRP (see Supplementary Table 4) when controlling for each alternative explanation, separately.

Supplementary Table 6

Weekly Relationship Quality Predicting CRP.

Predictors	<i>b</i> [95% CI]	<i>t</i>
Relationship quality	.004 [-.03, .04]	0.27
Biological sex	.34 [.07, .61]	2.53*
Age	-.001 [-.01, .01]	-0.11
BMI	.05 [.02, .08]	3.43***
OTC medicine use	.21 [.05, .37]	2.62**

* $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Table 7

Weekly Hostility toward Partner Predicting CRP.

Predictors	<i>b</i> [95% CI]	<i>t</i>
Hostility	-.04 [-.07, -.002]	-2.08*
Biological sex	.33 [.06, .59]	2.49*
Age	.0002 [-.01, .02]	0.03
BMI	.05 [.02, .08]	3.56***
OTC medicine use	.22 [.07, .38]	2.74**

* $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Table 8*Weekly Loneliness Predicting CRP.*

Predictors	<i>b</i> [95% CI]	<i>t</i>
Loneliness	-.04 [-.13, .05]	-0.95
Biological sex	.34 [.07, .61]	2.51*
Age	-.001 [-.01, .01]	-0.11
BMI	.05 [.02, .08]	3.47***
OTC medicine use	.22 [.06, .38]	2.70**

* $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Table 9*Total Time Spent Co-Present Predicting CRP, Controlling for Weekly Relationship Quality*

Predictors	<i>b</i> [95% CI]	<i>t</i>
Time spent co-present	-.0001 [-.0003, -.00001]	-2.06*
Relationship quality	.01 [-.02, .04]	0.43
Biological sex	.33 [.06, .60]	2.44*
Age	.001 [-.01, .01]	0.14
BMI	.05 [.02, .07]	3.35**
OTC medicine use	.21 [.06, .37]	2.68**

* $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Table 10*Total Time Spent Co-Present Predicting CRP, Controlling for Weekly Hostility Toward Partner.*

Predictors	<i>b</i> [95% CI]	<i>t</i>
Time spent co-present	-.0001 [-.0003, -.0000001]	-1.97*
Hostility	-.04 [-.07, -.002]	-2.07*
Biological sex	.33 [.06, .59]	2.42*
Age	.001 [-.01, .02]	0.24
BMI	.05 [.02, .08]	3.46***
OTC medicine use	.22 [.07, .38]	2.81**

* $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Table 11*Total Time Spent Co-Present Predicting CRP, Controlling for Weekly Loneliness.*

Predictors	<i>b</i> [95% CI]	<i>t</i>
Time spent co-present	-.0001 [-.0002, -.00001]	-2.16*
Loneliness	-.06 [-.15, .03]	-1.28
Biological sex	.33 [.06, .60]	2.42*
Age	.001 [-.01, .01]	0.14
BMI	.05 [.02, .08]	3.39**
OTC medicine use	.22 [.07, .38]	2.80**

* $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Table 12: Reverse Pathway of CRP to Time Spent Co-Present**Supplementary Table 12***CRP Predicting Time Spent Co-Present in Prior 24 Hours*

Predictors	<i>b</i> [95% CI]	<i>t</i>
CRP	-62.60 [-154.74, 28.33]	-1.36
Biological sex	-36.32 [-198.23, 126.66]	0.44
Age	11.23 [3.02, 19.45]	2.70**
BMI	-6.32 [-23.22, 10.70]	-0.74
OTC medicine use	2.26 [-154.18, 157.53]	0.03

* $p < .05$, ** $p < .01$, *** $p < .001$

**Supplementary Table 13:
Supplementary Analyses Using Weekly Fluctuations of Time Spent**

Supplementary Table 13 shows results using the supplemental group-mean-centered version of time spent co-present predicting CRP, in which numbers reflect deviations from participant's own average across the three time points. We control for standard covariates of biological sex, age, BMI, and over-the-counter medicine use in the prior 24 hours.

Supplementary Table 13

Weekly Fluctuations of Total Time Spent Co-Present Predicting CRP.

Predictors	<i>b</i> [95% CI]	<i>t</i>
Time spent co-present	-.0002 [-.0003, -.00002]	-2.21*
Biological sex	.34 [.08, .61]	2.54*
Age	-.001 [-.01, .01]	-0.12
BMI	.05 [.02, .08]	3.43***
OTC medicine use	.22 [.06, .38]	2.74**

* $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Table 14 & 15: Supplementary Analyses Controlling for All Covariates

Supplementary Tables 14 and 15 show results using weekly fluctuations of time spent (e.g., group-mean-centered, in which each measurement was centered, or subtracted from, the participant's mean level of time spent averaged across the three time points/measurements) version of time spent co-present, controlling for all standard covariates (see Table 2 in the main text) as well as both sets of additional exploratory covariates, 1) race, birth control use, and anti-depressant use, and 2) exercised today and sleep quality prior night.

Supplementary Table 14

Weekly Fluctuations of Total Time Spent Predicting CRP, Controlling for First Set of Exploratory Covariates.

Predictors	<i>b</i> [95% CI]	<i>t</i>
Time spent co-present	-.0002 [-.0003, -.00002]	-2.21*
Biological sex	.12 [-.14, .38]	0.88
Age	.004 [-.01, .02]	0.65
BMI	.06 [.03, .08]	4.57***
OTC medicine use	.20 [.04, .36]	2.53*
White	.30 [-.09, .67]	1.53
Black	-.29 [-.72, .12]	-1.40
Hispanic	.78 [.05, 1.52]	2.11*
Latino	-.19 [-.93, .56]	-0.50
East Asian	-.13 [-.50, .25]	-0.66
South Asian	.43 [-.09, .96]	1.63
Hawaiian/Pacific Islander	-.47 [-1.42, .47]	-0.99
Birth control use	.42 [.22, .62]	4.08***
Anti-depressant use	-.22 [-.46, .03]	-1.77

* $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Table 15

*Weekly Fluctuations of Total Time Spent Co-Present Predicting CRP,
Controlling for Second Set of Exploratory Covariates.*

Predictors	<i>b</i> [95% CI]	<i>t</i>
Time spent co-present	-.0002 [-.0003, .00002]	-2.17*
Biological sex	.33 [.06, .60]	2.46*
Age	-.001 [-.01, .01]	-0.10
BMI	.05 [.02, .07]	3.37**
OTC medicine use	.22 [.06, .38]	2.78**
Sleep quality prior night	.0001 [-.02, .02]	0.18
Exercised today	-.09 [-.22, .04]	-1.40

* $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Tables 16-18: Supplementary Analyses with Alternative Explanations: Relationship Quality, Hostility Toward the Partner, and Loneliness

See Supplementary Tables 16-18 for results with the group-mean-centered total time spent co-present when controlling for relationship quality, hostility with the partner, and loneliness, separately.

Supplementary Table 16

Weekly Fluctuations of Total Time Spent Co-Present Predicting CRP, Controlling for Weekly Relationship Quality.

Predictors	<i>b</i> [95% CI]	<i>t</i>
Time spent co-present	-.0002 [-.0003, -.00002]	-2.23*
Relationship quality	.006 [-.03, .04]	0.38
Biological sex	.34 [.07, .61]	2.51*
Age	-.001 [-.01, .01]	-0.08
BMI	.05 [.02, .08]	3.45***
OTC medicine use	.22 [.06, .38]	2.71**

* $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Table 17

Weekly Fluctuations of Total Time Spent Co-Present Predicting CRP, Controlling for Weekly Hostility Toward Partner.

Predictors	<i>b</i> [95% CI]	<i>t</i>
Time spent co-present	-.0001 [-.0003, -.00001]	-2.12*
Hostility	-.04 [-.07, -.001]	-2.04*
Biological sex	.33 [.07, .60]	2.48*
Age	.0003 [-.01, .01]	0.04
BMI	.05 [.02, .08]	3.56***
OTC medicine use	.23 [.07, .38]	2.84**

* $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Table 18

Weekly Fluctuations of Total Time Spent Co-Present Predicting CRP, Controlling for Weekly Loneliness.

Predictors	<i>b</i> [95% CI]	<i>t</i>
Time spent co-present	-.0002 [-.0003, -.00003]	-2.34*
Loneliness	-.06 [-.15, .03]	-1.29
Biological sex	.34 [.07, .61]	2.49*
Age	-.001 [-.01, .01]	-0.09
BMI	.05 [.02, .08]	3.50***
OTC medicine use	.23 [.07, .38]	2.83**

* $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Table 19: Characteristics of Long-Distance Sample**Supplementary Table 19***Characteristics of Sample Including Participants in Long-Distance Relationships (N = 159).*

	<i>M (SD)</i>	<i>% (n)</i>
Age	24.70 (7.56)	
Biologically Female		84% (135)
BMI	23.27 (3.9)	
Race/Ethnicity ¹		
White/Caucasian		75.2% (121)
Black/African American		9.3% (15)
Hispanic		3.7% (6)
Latino		3.1% (5)
East Asian		8.7% (14)
South Asian		6.2% (10)
Pacific Islander/Native Hawaiian		0.6% (1)
Middle Eastern		0.6% (1)
Southeast Asian		0.6% (1)
Education Level ²		
High school graduation or equivalent		5.6% (9)
Some college		49.4% (79)
College graduation		30.0% (48)
Professional/post-graduate degree		15.0% (24)

¹Groups are not mutually exclusive as participants could endorse more than one race/ethnicity.²We note education level may be confounded with age in this sample ($r = .71, p < .001$)

Supplementary Table 20: Primary Analyses Including Long-Distance Sample

Although in the main text we present results for participants who reported not being in a long-distance relationship with their partner and/or having a partner who lived locally, being long distance does not mean the participant didn't see their partner in person on any given week of the study. Because it is within the realm of possibility that the couple members saw each other periodically, we tested if time spent co-present were associated with CRP with the full sample, regardless of long-distance relationship status. See Supplementary Table 20 for model results. Conclusions hold.

Supplementary Table 20*Total Time Spent Co-Present Predicting CRP in Sample Including Long-Distance Relationships.*

Predictors	<i>b</i> [95% CI]	<i>t</i>
Time spent co-present	-.0001 [-.0002, -.00003]	-2.65**
Biological sex	.26 [.04, .47]	2.34*
Age	-.001 [-.01, .01]	-0.12
BMI	.04 [.02, .07]	4.02***
OTC medicine use	.34 [.22, .46]	5.71***

* $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Table 21: Sensitivity Analysis Including Individuals with High (>10) CRP

Supplementary Table 21 displays main model results, controlling for standard covariates, using time spent co-present to predict the full range of CRP values (including those greater than 10 $\mu\text{g/mL}$). Of the 298 CRP samples, two were above 10 $\mu\text{g/mL}$, and measured at 23.21 and 24.80 $\mu\text{g/mL}$ raw, assessed from two different people. These values are 6.17 and 6.62 standard deviations, respectively, above the (raw) mean value of CRP across all time points. Once log-transformed, the outlying values are 2.72 and 2.77 standard deviations, respectively above the log-transformed mean.

Supplementary Table 21

Total Time Spent Co-Present Predicting CRP, Including the Sample Values above 10 $\mu\text{g/mL}$.

Predictors	<i>b</i> [95% CI]	<i>t</i>
Time spent co-present	-.0001 [-.0002, .00002]	-1.64
Biological sex	.35 [.07, .62]	2.51*
Age	.001 [-.01, .02]	0.17
BMI	.04 [.02, .07]	3.12**
OTC medicine use	.30 [.13, .47]	3.50***

* $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Tables 22-24: Exploratory Analyses Using Time Spent Co-Sleeping

Our hypotheses focused on total time spent co-present over the course of a day. However, we also collected exploratory estimates of *how* that time was spent. Given prior associations between sleep duration and inflammation (Irwin et al. 2016; Patel et al. 2009), here we took the opportunity to explore whether the primary analysis could be explained solely by the amount of time spent sleeping next to their partner. To measure time spent co-sleeping, participants estimated how much of the *total* time spent with their partner that they reported from the prior 24 hours was spent sleeping next to them (*range*: 0 – 600 minutes; *M* time 1 = 215.4 minutes; *M* time 2 = 239.9 minutes; *M* time 3 = 225.2 minutes). Two reports of time spent co-sleeping were missing from the dataset ($n = 297$).

We present full models controlling for biological sex, age, BMI and over-the-counter medicine use in Supplementary Table 22. We then control for both sets of exploratory covariates in Supplementary Tables 23 and 24 (next page). Time spent co-sleeping was not significantly associated with CRP in either model.

Supplementary Table 22*Time Spent Co-Sleeping Predicting CRP.*

Predictors	<i>b</i> [95% CI]	<i>t</i>
Time spent co-sleeping	-.0002 [-.0004, .00002]	-1.79
Biological sex	.34 [.07, .61]	2.50*
Age	..001 [-.01, .02]	0.12
BMI	.05 [.02, .07]	3.31**
OTC medicine use	.21 [.05, .36]	2.56*

* $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Table 23*Time Spent Co-Sleeping Predicting CRP, Controlling for First Set of Covariates.*

Predictors	<i>b</i> [95% CI]	<i>t</i>
Time spent co-sleeping	-.0002 [-.0004, -.00002]	-1.82
Biological sex	.10 [-.16, .36]	0.77
Age	.01 [-.01, .02]	0.91
BMI	.05 [.03, .08]	4.39***
OTC medicine use	.19 [.03, .35]	2.35*
White	.29 [-.09, .67]	1.52
Black	-.30 [-.72, .12]	-1.41
Hispanic	.78 [.04, 1.52]	2.09
Latino	-.15 [-.90, .60]	-0.39
East Asian	-.13 [-.51, .25]	-0.67
South Asian	.43 [-.10, .96]	1.59
Hawaiian/Pacific Islander	-.45 [-1.40, 0.51]	-0.93
Birth control use	.43 [.23, .64]	4.17***
Anti-depressant use	-.20 [-.44, .05]	-1.61

* $p < .05$, ** $p < .01$, *** $p < .001$

Supplementary Table 24*Time Spent Co-Sleeping Predicting CRP, Controlling for Second Set of Covariates.*

Predictors	<i>b</i> [95% CI]	<i>t</i>
Time spent co-sleeping	-.0002 [-.0004, .00004]	-1.66
Biological sex	.33 [.06, .60]	2.43
Age	.001 [-.01, .01]	0.12
BMI	.05 [.02, .07]	3.26**
OTC medicine use	.21 [.05, .37]	2.61**
Sleep quality prior night	.001 [-.02, .02]	-1.30
Exercised today	-.08 [-.21, .04]	0.05

* $p < .05$, ** $p < .01$, *** $p < .001$