Social Network Composition and Inflammation at Midlife: A Socioemotional Selectivity Theory Perspective

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Social Network Composition and Inflammation at Midlife: A Socioemotional Selectivity Theory Perspective

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The socioemotional selectivity theory (SST) of aging posits that the narrowing of social networks with age (i.e., pruning peripheral members and maintaining close members) is an adaptive and purposeful process that provides emotional benefits. It has yet to be tested whether the benefits of social network narrowing extend to measures of physical health over time, such as markers of inflammation. The current study aimed to: (1) Characterize age-related changes in social network composition and inflammation; (2) Test whether age-related changes in social network composition associate with levels of inflammation; and (3) Explore whether changes in positive affect mediate the relationship between social network composition and inflammation. Participants were 350 healthy midlife adults enrolled in the longitudinal arm of the Adult Health and Behavior study (45% male, 89% white, wave 1 mean age =45 years); wave 2 data collection occurred approximately 15 years later. At both waves, participants self-reported their social network composition (Social Network Index) and trait positive affect (Positive and Negative Affect Schedule), and blood was drawn to assess markers of systemic inflammation (IL-6, CRP, TNF-α). Social network composition was categorized using ratios of close to peripheral relationships. As expected, social network ratios increased over time and close relationships were more likely to be maintained than peripheral ($t(349)= -2.95$, $p=.003$). Also as expected, most inflammatory markers increased over time (IL-6: $t(349)= -7.70$, $p<.001$; CRP: $t(349)= -3.99$, $p<.001$), but TNF-α decreased ($t(349)= 1.96$, $p=.051$). Unexpectedly, changes in social network

composition were not associated with levels of IL-6, CRP, or TNF-α at wave 2 controlling for wave 1 inflammation (*p*'s >.146), and positive affect did not operate as a mediator. There was a positive association between maintaining peripheral social network members and increases in positive affect (*b*=.12, SE=.047, *t*=2.64, *p*=.008). Overall, these findings support the SST hypothesis of social network narrowing with age, but not the hypothesis that social network narrowing is adaptive for promoting positive affect or lowering inflammation. The association between maintaining peripheral relationships and positive affect may suggest voluntary relationships provide more emotional benefits.

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1.0 Specific Aims

Older adults tend to have fewer social contacts and smaller social networks than younger adults and their own younger selves (Lang et al., 1998; Lang & Carstensen, 1994, 2002). Even as social network size declines, reports of relationship satisfaction, positive affect, and well-being tend to increase with age (Carstensen, 1992). The Socioemotional Selectivity Theory of aging posits that the narrowing of social networks with advancing age is a beneficial and purposeful process that cannot be fully explained by aging-related physical limitations or life events such as retirement or death of network members (Carstensen et al., 1999). Compositionally, network size decreases due to the removal of peripheral social network members, while the closest members in the network are preserved across the lifespan (Carstensen, 1992; Cornwell et al., 2008). In turn, these remaining relationships positively influence reports of daily affect and self-rated health (English & Carstensen, 2014).

Social relationships and associated affective states are robust predictors of physical health, including immune function (Farrell et al., 2018; Glaser & Kiecolt-Glaser, 2005). One immune pathway of considerable importance is systemic inflammation, which predicts mortality and age-related chronic diseases, including atherosclerotic cardiovascular disease, cancer, and neurodegenerative conditions (Harris et al., 1999; Pawelec et al., 2014). To date, no studies have addressed the potential adaptive influence of social network narrowing on inflammation, nor the potential mediating role of affect. Extending knowledge in this area of research can identify which social network compositions are most beneficial or detrimental for healthy aging. The purpose of the current study is to use a longitudinal cohort of midlife adults with two waves of data collected

an average of 15 years apart to test the following aims: (1) To replicate previous research and characterize age-related changes in social network composition and inflammation over time; (2) To test whether age-related changes in social network composition associate with levels of inflammation over time; and (3) To explore whether changes in positive affect mediate the relationship between social network composition and inflammation over time.

2.0 Background and Significance

2.1 Socioemotional Selectivity Theory

The total size and composition of social networks (i.e., the number of members within different categories including friends, family members, and acquaintances) change across the lifespan. Meta-analytic and empirical evidence suggests global network size increases from adolescence to young adulthood (~25-30 years), then declines steadily into old age (Van Tilburg, 1995; Wrzus et al., 2013). The observed change in network size across the lifespan is attributed to the addition or removal of peripheral members, such as friends or acquaintances, at a rate of removing almost one person from the network per decade of age (Wrzus et al., 2013). However, the number of close social contacts, such as family members, remains constant (Antonucci, 2001; Antonucci et al., 2019; Wrzus et al., 2013). These effects largely do not vary by country, cohort differences, or gender, but there is some evidence indicating women may have more family members in their social networks than men (i.e., siblings) (Dykstra, 1995). One longitudinal study to date that assessed the same people at modal ages of 18, 30, 40, and 52 years determined that interactions with friends decreased starting in young adulthood but that interactions and closeness with immediate family members increased and stayed elevated throughout midlife (Carstensen, 1992). Consequently, older adults generally report having social networks primarily composed of close relationships (e.g., family) and fewer peripheral relationships (e.g., acquaintances and friends) (Antonucci & Akiyama, 1987; Bond et al., 2005; Lang & Carstensen, 1994). Thus, there is substantial cross-sectional evidence for age-related social network size decline attributed to the

removal of peripheral social relationships and the preservation of primarily close family relationships.

According to Socioemotional Selectivity Theory (SST), the retention of close relationships and removal of peripheral relationships observed with aging is purposeful (Carstensen, 1992). The SST asserts that the narrowing of networks is occasioned by goal changes from future-oriented goals (e.g., career or parenting) to more affect-rewarding goals, which occur when individuals perceive time as limited (Carstensen et al., 1999; Fung & Carstensen, 2004). In line with this theory, social partner preferences in participants matched on physical health, mental health, and social behavior demonstrated that young adults (age 14-19) were most likely to select novel partners who could provide future benefits, middle-aged adults (age 35-44) were most likely to select social partners to gain information, and older adults (age 69-92) were most likely to select social partners who offer emotional benefits (Carstensen et al., 2003; Fredrickson & Carstensen, 1990; Lang & Carstensen, 2002). Therefore, variation in social partner selection may depend on age, and there is further support that it depends on perception of time and goal shifting. Importantly, although age-related life transitions such as retirement and decline in physical function are associated with alterations in social network sizes (Domènech-Abella et al., 2021; Elovainio et al., 2003), transitional events do not account for all changes seen in social networks; in particular, relationships with family members are especially unaffected by life changes (Wrzus et al., 2013). In sum, theories of aging that focus on physical limitations and life events alone fail to explain the shift in age-related goal setting and the emotional benefits that accompany age-related social network narrowing, whereas the SST offers a plausible explanation of why and how social networks may change with age.

2.2 Social Network Composition Measurement

Previous SST research has measured social relationships by conducting clinical interviews to collect information on participants' total network member roles and their emotional closeness and relationship satisfaction with each member. Through these interviews, Carstensen and colleagues identified lifespan trends of contact, closeness, and satisfaction for different network member groupings, including acquaintances, siblings, parents, close friends, spouses, and children (Carstensen, 1992). Informed by Carstensen's work, the present study aims to analyze how social network compositions change with age by deriving the compositions of close and peripheral members from the Social Network Index (SNI; Cohen et al., 1997). The SNI has not previously been used to assess social network composition in terms of close and peripheral members, but is well suited for this categorization. Broadly the SNI totals the number of social network members, including family members, close friends, close relatives, and other acquaintances, that the respondent has contact with at least once every two weeks. The current study categorizes family members, and in some instances close friends and relatives, as "close" social network members and other acquaintances as "peripheral" members, based on previously demonstrated trajectories of closeness and satisfaction in these categories across adulthood (Carstensen, 1992). Moreover, given that the SST posits that close relationships will remain stable while peripheral relationships will decline over time, the current study uses a ratio of close to peripheral members to summarize changes in network composition. Larger ratios indicate a greater proportion of close members in the network, and smaller ratios indicate a greater proportion of peripheral members in the network.

Previous research has used the SNI to assess different conceptualizations of social network composition and relate it to healthy aging outcomes. For example, Sharifian and colleagues used

only the SNI items pertaining to family and close friends and found that social networks composed of more close friends, versus more family, were associated with better cognitive health two years later in adults (average age at baseline= 68; *N*=10,463) (Sharifian et al., 2019). Others have combined items from the SNI (marital status, number of children) and additional items not in the SNI (contact frequency with friends and children and participation in community events) to create social network typologies and examine their associations with health (Fiori et al., 2006; Litwin & Shiovitz-Ezra, 2006). Individuals in family-type and close friend-type networks had lower mortality rates compared to individuals with isolated networks (i.e., the fewest family relationships, lowest rates of contact with friends, and lowest attendance rates at community events); however, adults in neighborhood type networks (i.e., high contact rates with neighbors but less contact with close friends) had mortality rates comparable to the isolated typology (Litwin & Shiovitz-Ezra, 2006). Taken together, previous research using the SNI has demonstrated associations between social network composition and aging-related health outcomes. However, the theoretical basis for these studies is different from the current analysis, as they focus on the distribution of specific member types within the network (e.g., friends, family, neighbors; Fiori et al., 2006; Litwin & Shiovitz-Ezra, 2006). The only study to assess close relationships failed to address the contribution made by remaining peripheral relationships in the network to health (Sharifian et al., 2019). Considering the existing studies, it remains unknown how the failure to prune acquaintances over time, who are not close or emotionally rewarding, may be disadvantageous for healthy aging.

2.3 Social Relationships and Systemic Inflammation

Social relationships are central to mental and physical health. The effects of social relationships on physical health are of similar magnitude to those of lifestyle factors, such as smoking and physical activity. For instance, individuals with stronger social relationships experience a 50% reduction in mortality rate compared to individuals with weak social relationships (Holt-Lunstad et al., 2010). Having a greater number of social relationships has also been shown to predict reduced susceptibility to acute illness and risk of chronic diseases (Cohen et al., 1997; Valtorta et al., 2018).

The links between social relationships and broader age-related health outcomes (i.e., morbidity and mortality) may be explained in part by systemic inflammation. In the aging process, the immune system upregulates genes involved in inflammation, and systemic inflammation generally increases over time (Franceschi et al., 2017; Furman et al., 2019; López-Otín et al., 2013). Systemic inflammation occurs naturally but can be exacerbated by persistent acute psychological or environmental stressors, causing immune cells to develop a senescence-associated secretory phenotype (SASP) and secrete inflammatory cytokines and molecules in excess (Furman et al., 2019). The stress response includes interleukin-6 (IL-6) release, which among other factors (tumor necrosis factor-alpha (TNF-α) and interleukin-1 beta (IL-1β)), can stimulate production of acute phase C-reactive protein (CRP), a marker for systemic inflammation. On average, for healthy populations, CRP increases 0.2 mg/L per decade (age range= 47-87 years, *N*=2,473) and IL-6 increases from 0.03-0.07pg/mL per year (age range= 21-50 years) (Lassale et al., 2019; Lustig et al., 2017). The potential to slow age-related increases in systemic inflammation is of interest because elevated levels of inflammatory markers (cytokines, chemokines, and acute-

phase proteins) can lead to tissue and organ damage (Furman et al., 2019). Furthermore, systemic inflammation is associated with a greater risk of developing various pathologies, including heart disease, cancer, kidney disease, and auto-immune diseases (Furman et al., 2019). Understanding the potential role of social network composition in predicting inflammatory markers is particularly important during the aging process when individuals are at increased susceptibility for an array of inflammatory diseases.

Much research indicates links between social relationships and systemic inflammation. However, the majority of this research has focused on social integration, or the number of unique social roles (e.g., spouse, parent, child, close friend, volunteer) an individual participates in frequently within their social network. For example, meta-analytic evidence links social integration with lower levels of inflammatory markers, including IL-6 and CRP (Uchino et al., 2018). Results from a nationally representative US sample demonstrated a relationship between low social integration and heightened CRP in men; this association is stronger with age, such that men over the age of 60 who held the fewest social roles (0-1 roles) were more likely to have higher CRP levels than men who held the most social roles (4 roles) (odds ratio=1.80; Ford et al., 2006). Two other large cohort studies (*N*=963 and *N*=3267) corroborated these findings and reported associations between low social integration and heightened IL-6 in both men and women, specifically in older age groups (ages 70+ showed the largest effects) (Elliot et al., 2018; Loucks et al., 2006). Holding fewer social roles may be associated with having smaller social networks, so the above findings linking low social integration with higher inflammation may seem contradictory to the predictions of the current study. However, the SST posits that *who is in your social networ*k may be more relevant to health outcomes, above and beyond the total network size. Therefore, the current study distinguishes specific aspects of social network composition and

examines whether pruning peripheral members relative to close members is associated with lower inflammation.

2.4 The Mediating Role of Positive Affect

Social relationships have been linked to positive affect in older adults, further solidifying the critical role of positive social relationships in healthy aging (S. Cohen & Lemay, 2007; Scholz et al., 2012). Due to the age-related decrease in social network size, it might be expected that reports of support and positive affect would decline with age. However, as social network size decreases with age, ratings of relationship satisfaction and positive affect tend to increase or stay stable (Carstensen, 1991, 1992; Due et al., 1999; Inglehart, 2018). These findings could be explained by the fact that the remaining relationships, likely close others such as family members, are more emotionally rewarding, and therefore promote positive affective responses and overall emotional well-being (Antonucci et al., 2014; English & Carstensen, 2014; Yeung & Fung, 2007). Compared to middle-aged and young adults, older adults report a greater quality and closeness of relationships with spouses and family members (Fingerman et al., 2004; Schnittker, 2007). Similarly, reported relationship satisfaction with close family members, such as spouses, children, siblings, and parents, increases starting at midlife (Carstensen, 1992). Collectively, these findings suggest that although social networks are smaller, they may provide more emotional benefits because they are composed of more close members relative to peripheral members.

Age-related narrowing of social networks may lead to positive psychological well-being, including positive affect that, in turn, influences physical health. Meta-analytic evidence from 35

prospective studies in healthy adults indicates that greater reports of positive psychological well-being is protective of mortality rates in healthy populations (HR=0.82; Chida & Steptoe, 2008). There is additional meta-analytic support that well-being is predictive of both short-term and long-term health outcomes, as well as disease symptom severity and recovery (S. Cohen & Pressman, 2006; Howell et al., 2007; Lamers et al., 2012). These findings have been replicated in various studies, demonstrating that well-being is protective of illness, similar to the effect sizes of other health behaviors such as smoking (Veenhoven, 2008). One possible explanation for these associations could be the inverse relationship between positive affect and inflammation (Jones & Graham-Engeland, 2020; Stellar et al., 2015). In longitudinal studies with large representative cohorts (*N*=2,544 and *N*=13,775), lower reports of positive affect at baseline predicted heightened CRP levels up to five years later (Deverts et al., 2010; Niles et al., 2018). Nuances to this association also exist, with one longitudinal study linking positive affect correlates with some inflammatory markers (i.e., IL-6 and TNF-α) but not others (i.e., hsCRP) (Brouwers et al., 2013;), and a cross-sectional study demonstrating no association between positive affect and inflammation (Andreasson et al., 2013). Overall, findings from longitudinal retrospective studies indicates affective responses that may be susceptible to social network variation could have the potential to alter immune function and inflammation. Still, associations may depend on the measurement of inflammatory markers.

2.5 Current Study

Most research on the sequelae of aging-related network narrowing, as posited by the SST, has focused on its promotion of emotional well-being as the outcome. It is unknown how the promotion of emotional well-being may, in turn, predict positive health outcomes as adults age, including levels of inflammation. Therefore, the aim of the current study is to assess changes in social network composition, positive affect, and the progression of systemic inflammation (IL-6, CRP, and TNF-α) in a community cohort of midlife adults from the Adult Health and Behavior (AHAB) studies assessed at two time points (W1 and W2), approximately 15 years apart. The specific aims and hypotheses are as follows:

(1) To characterize age-related changes in 1) social network composition and 2) inflammation over time. I hypothesize 1) there will be an increase in the social network composition ratio of close to peripheral members over time, attributed to the removal of peripheral social network members, and 2) there will be an increase in inflammatory markers over time.

(2) To test whether age-related changes in social network composition associate with changes in inflammation over time. I hypothesize that an increase in the social network composition ratio will associate with lower levels of inflammatory markers at W2 while controlling for inflammatory markers at W1.

(3) To explore whether positive affect mediates the association between changes in social network composition and inflammation over time. I hypothesize the increase in social network composition ratio will correlate with increases in positive affect from W1 to W2, which, in turn, will be associated with lower levels of inflammation at W2.

Much of the current literature has assessed lifespan trajectories of social network composition using cross-sectional age-group comparisons and lacks sufficient evidence of the SST in longitudinal data. In addition, no study to date has assessed the physiological health correlates of social network composition as it relates to predictions of SST, despite physical health being an important outcome for older adults, in addition to emotional well-being, where the majority of research has focused. The sample selected for this study offers notable advantages. In this midlife sample, it's less likely that the pruning of social network members would be due to aging-related physical limitations or life events, such as the death of a network member, and is more likely to align with the SST's position on intentional pruning. Additionally, midlife is a developmental period when adults are at an increased risk for inflammatory diseases, thus offering the potential to observe inflammation trajectories when they are likely to accelerate. Furthermore, this study utilizes data collected at two time points, providing a stronger test of the SST predictions, as well as the characterization of inflammation trajectories from midlife to later adulthood as they relate to changes in social network composition.

3.0 Research Design & Methods

3.1 Participant Characteristics

Study data were derived from the University of Pittsburgh Adult Health and Behavior (AHAB) project. The AHAB project provides a registry of behavioral and biological measurements, plus DNA for genetic analysis of registry phenotypes, on midlife community volunteers recruited via mass-mail solicitation from communities of southwestern Pennsylvania (principally Allegheny County). AHAB data collection was conducted in two phases, termed AHAB I (conducted from 2001 to 2005) and AHAB II (conducted from 2008 to 2011). General study exclusions for both samples included a reported clinical history of atherosclerotic cardiovascular disease, chronic kidney or liver disease, cancer treatment in the preceding year, major neurologic disorders, schizophrenia, or other psychotic illness. Other AHAB study exclusions included pregnancy and use of insulin, glucocorticoid, antiarrhythmic, psychotropic, or prescription weight-loss medications, systolic/diastolic blood pressure >180/110 mmHg (AHAB I) or >160/100 mmHg (AHAB II), alcohol consumption >25 drinks per week (AHAB I) or >5 drinks 3-4 times per week (AHAB II). The AHAB II cohort had additional exclusion criteria, including use of diabetic medications, antihypertensives, lipid lowering drugs, fish oil supplements, and participation in shift work. AHAB II participants were all employed >25 hours per week. Data collection occurred over multiple laboratory sessions, and informed consent was obtained in accordance with approved protocols and guidelines of the University of Pittsburgh Institutional Review Board.

Of the 600 participants who provided some data at both waves, 135 participants were ineligible due to confounds with the immune system (based on meeting any of the following immune confounds: autoimmune disorders, HIV/AIDS, inflammatory bowel disease, chronic hepatitis, chronic lung disease, oral glucocorticoid medication use, acute or chronic infection being treated with antibiotics, regular use of allergy shots, recent vaccination, cold or flu within the past two weeks). An additional 100 participants had invalid inflammatory assays due to loss of samples, phlebotomy failure, or values/CVs outside of the expected range. Of the remaining 365 participants, an additional 15 were removed because they were missing primary predictor variables (SNI: *n*= 10; PANAS: *n*= 4) or missing covariates (BMI: n=1) at either time point. Two participants had one missing SNI relationship item at wave 2; these participants were retained, and these items were treated as zero when taking ratio sums because they had reported not having these relationships at the previous time point. Participants were only retained if they had complete data for all inflammatory markers to enable comparisons across mediation pathways; this resulted in 350 participants for the final sample (see missing data section 3.4.1 below).

3.2 Measures

3.2.1 Social Network Composition

Social network composition was assessed at both time points using the Social Network Index (SNI; Cohen et al., 1997). The SNI assesses the individual's participation in 12 social relationships: spouse, children, parents, in-laws, close relatives, close friends, church members,

classmates, workmates, neighbors, volunteer workmates, other social/recreational/professional groups. Participants report how many total people in these 12 relationship categories they interact with either in person or on the phone at least once in a two-week period.

The SNI was used to create a ratio of the number of close social network members relative to the number of peripheral members. Two versions of the ratio were tested (see Supplemental Table S1)– one with a stricter interpretation of what constitutes "close" social members (i.e., only immediate family members including spouse, children, parents, and in-laws) and a second version with a broader interpretation (i.e., immediate family members as well as members categorized as “close relatives” and “close friends”). Although close friends are indeed considered close, their interactions fluctuate. The broader interpretation accounts for evidence that contact with close friends fluctuates across the lifespan and the preservation of close friends may depend on the need to compensate for the loss of immediate family members (Carstensen, 1992; Lang & Carstensen, 1994). For the strict ratio, the sum of 4 relationships that make up the numerator are referred to as the close sum and for the broader ratio the sum of 6 relationships that make up the numerator are referred to as the moderate sum. For both ratios, the denominator of peripheral social members was calculated as the sum of church/religious group members, school members, work members, neighbors, volunteer members, and other group members. The ratios are interpreted as follows: a larger ratio corresponds with a narrow social network, more close members than peripheral, and a smaller ratio corresponds with a wider social network, fewer close members than peripheral. To avoid having zeroes in the ratio calculation, a 1 was added to all the numerator and denominator before the final ratio was calculated.

3.2.2 Positive Affect

Positive affect was measured at both time points using the Positive and Negative Affect Schedule (PANAS; Watson et al., 1988). Participants reported how much they have generally experienced positive affect, items include feelings such as interested, excited, and enthusiastic, on a 5-point Likert scale from 1 (*never*) to 5 (*always*). Responses were summed, with higher numbers indicating greater positive affect (total sum ranges from 10-50). The scale was reliable between people across both time points (R_{KF}= .94) and within person over time (R_C= .83).

3.2.3 Inflammation

Assays for IL-6, CRP, and TNF-α were conducted in the same way at W1 and W2. IL-6, CRP, and TNF-α were natural log-transformed for analyses. To account for the effects of acute inflammation, people with CRP levels >10mg/L were excluded in analyses (N wave 1= 11; N wave 2= 19), as previously described in section 3.1 (Pearson et al., 2003).

IL-6 levels (pg/mL) were determined by the University of Pittsburgh's Behavioral Immunology Laboratory using a high sensitivity quantitative sandwich enzyme immunoassay kit (R&D Systems, Cat # HS600B) run according to the manufacturer's directions. The standard range of detection for the assay is 0.156-10pg/mL. Plasma **CRP** (mg/L) levels were measured by the Laboratory of Clinical Biochemistry at the University of Vermont and assayed with a BNII nephelometer using a particle enhanced immunophelometric assay (Dade-Behring, Inc., Deerfield, Illinois, USA). The range of detection for the assay is 0.175-1100 mg/L. **TNF-α** levels were measured by the University of Pittsburgh's Behavioral Immunology Laboratory using

ProteinSimple's Human TNF-α second generation Ella cartridge. The range of quantification is .30-1160 pg/mL, and the limit of detection is .03pg/mL. Intra- and inter-assay CVs for all assays at both time points were less than 10% (individual CVs reported in Supplemental Table S2).

3.2.4 Control Variables

Covariates that may influence inflammatory outcomes included age at W1 (in years), AHAB cohort (1 or 2), time between W1 and W2, sex (0=male, 1=female), smoking status at W2 (0=current non-smoker, 1=current smoker), and BMI at W2 (weight/height^2). In addition, all analyses included total network size at W1 (calculated as the total number of members reported in all SNI items) as a covariate.

3.3 Analytic Plan

Statistical analyses were conducted in R (version 4.0.4), and data management and preparation required the tidyverse and psych packages. Analyses were conducted using both unadjusted and adjusted multiple regression models with IL-6, CRP, and TNF-α as separate outcomes.

3.3.1 Aim 1

To characterize age-related changes in 1) social network composition and 2) inflammation over time.

Hypothesis 1a. There would be an increase in the social network composition ratio of close to peripheral members over time, attributed to the removal of peripheral social network members.

Analytic plan: A dependent t-test compared ratio means of social network composition from W1 and W2. Using the ratios (close members:peripheral members) larger ratios at W2 compared to W1 may indicate "pruning" took place and the number of peripheral members decreased. To confirm this, additional dependent t-tests separately assessed the change in the numerator (close members) and the denominator (peripheral members) from W1 to W2. Analyses were conducted using the base package in R.

Hypothesis 1b. Inflammatory markers IL-6, CRP, and TNF-α were expected to increase from W1 to W2.

Analytic plan: Dependent t-tests assessed change in inflammatory markers (IL-6, CRP, and TNF-α) from W1 and W2. Analyses were conducted using the base package in R.

3.3.2 Aim 2

To test whether age-related changes in social network composition associate with changes in inflammation over time.

Hypothesis 2. The increase in the social network composition ratio (i.e., positive change scores, W2-W1), would be associated with lower levels of inflammatory markers at W2 controlling for inflammatory markers at W1.

Analytic Plan: Change scores (W2-W1) were calculated for social network composition ratios from W1 to W2. Separate multiple regression models predicted IL-6, CRP, and TNF-α levels at W2 from changes in the social network composition ratio, controlling for W1 social network

composition ratio and W1 inflammatory levels. Secondary analyses probed change in close members (the numerator) and change in peripheral members (the denominator) as individual predictors of inflammation outcomes to determine whether the change in ratio size and its correlation with inflammation levels is attributed to decreases in peripheral members or increases in close members. Analyses were conducted using the base package in R.

3.3.3 Aim 3

To explore whether positive affect mediates the observed changes in social network composition and inflammation over time (see *Figure 1*).

Hypothesis 3. The increase in social network composition ratio (i.e., positive change score, W2-W1), would correlate with increases in positive affect (i.e., positive change score, W2-W1), which, in turn, would be associated with lower levels of inflammation at W2.

Analytic plan: Three mediation models (one for IL-6, one for CRP, one for TNF-α) were used to examine whether changes in social network composition associate with changes in positive affect, which in turn, predict inflammation at W2, while controlling for W1 inflammation, W1 positive affect, and W1 social network composition. Secondary analyses probed change in close members (the numerator) and change in peripheral members (the denominator) as individual predictors of inflammation outcomes to determine whether the change in ratio size and its correlation with inflammation levels is attributed to decreases in peripheral members or increases in close members. Standard errors were resampled 5,000 times to create bootstrapped confidence intervals for the direct and indirect paths. Mediation analysis was conducted using the Lavaan package in R.

3.4 Additional Analytic Considerations

3.4.1 Missing Data

To improve the power of the current study, missing data were imputed using Multiple Imputation by Chained Equations (MICE). The majority of missing data was from inflammatory assays at wave 2. 15 resampling imputations were conducted to impute data for 64 individuals (15% of the sample) resulting in a sample of 414 people. The variables selected for data imputation consisted of the covariates included in aim 2 linear models with the addition of race. Analyses were conducted on each imputed data set, and the final estimates were pooled across data sets. We retested aim 2 using only the conservative ratio as a predictor to test whether increasing the sample size altered the original findings.

3.4.2 Sensitivity Analyses

Exploratory sensitivity analyses for aim 1 tested change in ratios, numerators, and denominators after removing participants who reported loss or separation from a spouse, or the loss of a close relationship at some point between wave 1 and 2 to determine if a sample with predominantly intentional pruning would demonstrate even more stability in their close relationships. Additionally, using regression models with change scores as outcomes, sensitivity analyses for aim 1 tested whether age at wave 1, time between waves, sex, IQ at wave 1, or wave 1 inflammation moderated the amount of change in the strict and broader ratios from wave 1 to wave 2. We tested these moderators to determine if any demographic or health variables influenced

the expected trend of pruning (e.g., if a person demonstrated higher inflammation or lower IQ at baseline, they may have had less of an opportunity to prune members intentionally).

In regards to aim 2, when examining the associations between change in social network composition ratio and inflammation, sensitivity analyses included an additional covariate to account for the loss of a close relationship between the waves, extracted from the Stress and Adversity Inventory (STRAIN; Slavich & Shields, 2018). Given the age of the sample and the lower likelihood of loss of close relationships (e.g., spouse, children), it is unlikely that further controlling for the loss of a close relationship would change the results; however, the addition of this covariate ensured the association between change in social network composition ratio and inflammation is attributed to a purposeful pruning of the relationships, as described by the SST. In addition, exploratory sensitivity analyses for aim 2 evaluated changes in each relationship category (i.e., children, parents/in-laws, relatives, close friends, churchmates, neighbors) collected in the SNI in relation to changes in inflammation. Assessment of each relationship category expanded on previous findings that suggest family members and friends may make different contributions to both psychological and physical health outcomes. For example, a longitudinal study attributed weekly contact with friends to lower mortality rates, but contact with family did not emerge as a significant predictor (HR=0.76; Becofsky et al., 2015). Additionally, as previously noted, having a greater percentage of friends compared to family in one's social network has been associated with better cognitive function (Sharifian et al., 2019). Exploratory analyses were corrected for multiple comparisons using the Benjamini-Hochberg correction method (Benjamini & Hochberg, 1995).

4.0 Results

As shown in Tables 1 and 2, the sample was composed of 312 White, 36 Black, and 2 Asian participants with an average age of 45.1 $\pm$ 6.61 at wave 1. The average length of time elapsed between the two waves was 14.9 $\pm$ 2.78 years (range = 9-20 years). The majority of participants were from the AHAB 1 cohort (72.3%) and began participation between 2001-2005 (wave 1). The remaining 27.7% were from AHAB 2, which occurred between 2008-2011 (wave 1). Characteristics of the sample at wave 1 and wave 2 are presented in Table 2 with change scores calculated for applicable variables. Bivariate correlations of the primary variables and change scores used for analyses are presented in Supplemental Table S3. Bivariate correlations for raw variables at wave 1 and wave 2 are presented in Supplemental Tables S4 and S5. Total social network size at wave 1 was correlated with change in the strict ratio from wave 1 to wave 2 (r =.15) and changes in the strict ratio numerator (r =-.22) and denominator (r =-.49); indicating that a larger ratio at wave 1 was accompanied by more removal of both close and peripheral members. Additionally, correlations between social network size at baseline and changes in social network were small to moderate and multicollinearity was not a concern. Neither baseline total social network size (r =.02), ratio change scores (strict: r =.01; broad: r =-.02), change in close sum numerator (r =.06), or change in peripheral sum denominator (r =.05), were strongly correlated with changes in positive affect. Measures of IL-6, CRP, and TNF-α at wave 1 and wave 2 were moderately positively correlated (wave 1: r =.19-.35; wave 2: r =.17-.33) with the smallest correlations between CRP and TNF-α at both waves. Measures of inflammation at time 2 had

weak correlations with change in social network ratios and in positive affect (social ratio: r =-.09-.03; positive affect: r =-.01-.02).

4.1 Aim 1: Characterize age related changes over time

4.1.1 Social network change

I hypothesized there would be an increase in the social network composition ratio of close to peripheral members over time. Table 3 displays the results from paired t-tests. The results indicated that both the strict and broader ratios significantly increased from wave 1 to wave 2 (strict: t(349)= -2.95, p=.003; broad: t(349)= -324, p=.001) (Table 3). Further examination of the change in the numerator of the strict (close-sum) and broader (moderate-sum) ratio and of the change in the denominator, which was the same for both ratio calculations (peripheral-sum), indicated that the number of close members, moderate members, and peripheral members significantly decreased from wave 1 to wave 2 (close sum: t(349)= 10.94, p<.001; moderate sum: t(349)= 6.64, p<.001; peripheral sum: t(349)= 9.49, p<.001). Importantly, the mean number of peripheral members removed was larger than the number of close or moderate members removed, explaining the overall increase in strict and broad ratios from wave 1 to wave 2.

4.1.2 Social network change sensitivity analyses

I conducted a set of sensitivity analyses to further probe the observed change in social network ratios from wave 1 to wave 2. First, I removed 23 individuals who reported a loss of or separation from a *partner* between wave 1 and wave 2 (*N*=327). This did not change the results of the initial t-tests for change in the strict/broad ratios, the numerators, and denominators (Table 4). Second, I removed 44 individuals who reported the loss of *any close relationship* between wave 1 and wave 2 (*N*=306); there were still increases in the strict and broad ratios, but the increase in strict ratio was no longer statistically significant (strict: *p*=.074; broad: *p*=.042) and the change in close, moderate, and peripheral members remained the same (Table 5).

In addition, we tested various moderators to determine if W1 age, time elapsed between waves, sex, W1 IQ, or baseline inflammation affected the observed change in social network ratios from wave 1 to wave 2. Age moderated the observed increase in strict ratio from wave 1 to wave 2, such that individuals who were older at baseline demonstrated less increase in the strict ratio from wave 1 to wave 2 (*b*=-.02, SE=.008, *t*= -2.22, *p*=.027). This association did not withstand correction for multiple comparison (threshold for lowest *p-value*=.025) and was not significant when testing change in the broader ratio. IQ at wave 1 moderated the increase in strict ratio from wave 1 to wave 2, such that having a higher IQ at wave 1 was associated with a greater increase in the strict ratio (*b*=.01, SE=.005, *t*= 2.05, *p*=.041). This association did not withstand correction for multiple comparison (threshold *p*=.025) and was not significant when testing change in the broader ratio. Time elapsed between waves, sex, and wave 1 levels of inflammation had no statistically significant associations with changes in strict or broad ratios (Table 6).

4.1.3 Inflammation change

I hypothesized there would be an increase in inflammatory markers IL-6, CRP, and TNF-α from wave 1 to wave 2. The results are displayed in Table 7. IL-6 significantly increased over time (t(349)= -7.70, p<.001). CRP significantly increased over time (t(349)= -3.99, p<.001). TNF-α decreased, but the result was not statistically significant (t(349)= 1.96, p=.051) (Table 7).

4.1.4 Inflammation change sensitivity analyses

We additionally re-tested aim 1 change in CRP after removing 13 individuals with small (outlier) log transformed CRP values (less than -4) at wave 2. The increase in CRP over time was larger and remained statistically significant (t(336)= -7.32, p<.001) (Table 8).

4.2 Aim 2: Linear associations between change in social network composition and change in inflammation

4.2.1 Linear models

We hypothesized that an increase in the social network composition ratio would be associated with lower levels of inflammatory markers at W2 controlling for inflammatory markers at W1. However, changes in social network composition, assessed by change in the strict ratio, broader ratio, and the corresponding numerator (close/moderate-sum), and denominator

(peripheral sum), were not associated with IL-6, CRP, or TNF-α at W2 controlling for W1 inflammation (results in Tables 9-13, *p*'s >.146).

4.2.2 Linear models sensitivity analyses

In sensitivity analyses, we further added a covariate to account for the loss of a close relationship at any point between the two waves. The results for aim 2 remained unchanged (see Tables 14-18). (There was a weak positive association between reporting the loss of a close relationship between the waves and IL-6 that approached but did not reach significance, *b*=.21, SE=.11, *t*=1.91, *p*=.058).

We also imputed missing data and re-tested linear associations between the conservative ratio and the three inflammatory markers. Increasing the sample size to 414 individuals did not change the direction or significance of the initial associations (Supplemental Table S6).

4.2.3 Linear models exploratory analyses

In exploratory analyses, we tested whether changes in specific relationship types (children, parents/in-laws, other relatives, close friends, churchmates, and neighbors) were associated with changes in inflammation. Results are presented in Table 19. Descriptive statistics on the changes in the selected relationships can be found in Supplemental Table S7; not all relationships were tested because they lacked sufficient variability. Retaining more relationships from W1 to W2 with neighbors was associated with lower levels of IL-6 at wave 2 (*b*=-.05, SE=.02, *t*=-2.37, *p*=.018), but this result did not withstand correction for multiple comparisons (threshold for lowest *p-value*=

.016). Retaining more relationships with close friends from W1 to W2 was associated with lower levels of CRP at W2, but this was not statistically significant (*b*=-.05, SE=.03, *t*=-1.71, *p*=.088). There were no other statistically significant associations between changes in relationship types and changes in inflammation.

4.3 Aim 3: Positive affect as a mediator of the association between change in social network composition and change in inflammation

I hypothesized that the increase in social network composition ratio would correlate with an increase in positive affect, which, in turn, would be associated with lower levels of inflammation. Changes in social network composition, assessed by change in the strict ratio, broader ratio, and the corresponding numerator (close/moderate sum), were not associated with changes in positive affect (a path) (Tables 20-24, *p'*s >.10). There was a positive association between having less of a decrease in peripheral social network members and increases in positive affect (*b*=.12, SE=.047, *t*=2.64, *p*=.008) (a path). However, change in positive affect was not associated with W2 inflammation (IL-6, CRP, or TNF-α) controlling for W1 inflammation (b path, *p'*s >.169). There were no statistically significant indirect or total effects (Tables 20-24).

5.0 Discussion

The objective of the current analysis was to test the SST, which describes the intentional narrowing of social networks that occurs with age and the subsequent increase in positive affect. These changes in social network composition are thought to be adaptive and therefore may have health benefits (Baltes & Baltes, 1990). The current study sought to replicate previous research by assessing the expected age-related decrease in peripheral social network members and increase in inflammation. We further tested direct associations between social network narrowing and inflammatory markers IL-6, CRP, and TNF-α, and the mediating role of positive affect. We hypothesized that social network narrowing would be associated with less of an increase in systemic inflammation over time, through the pathway of increased positive affect. We found support that social networks narrowed from wave 1 to wave 2, which were an average of 15 years apart. We also identified an increase in two of the three inflammatory markers. However, contrary to our predictions, social network narrowing did not associate with changes in inflammation, and changes in positive affect did not operate as a mediator.

As predicted, social networks narrowed from wave 1 to wave 2, which was indicated by the increase in ratio of close to peripheral network members. We further probed the close and peripheral member sums to confirm that the increase in ratio was attributed to a decrease in peripheral members and not an increase in close members. Indeed, we identified a significant decrease in the peripheral member sums, as well as the close member sums, but the mean decrease in peripheral members was larger. The observed decrease in close members was unexpected and may suggest that using the SNI relationship categorization was not an exact representation of who

the participant is closest to. An additional limitation of the SNI is that it does not capture individuals who the participant does not have at least bi-weekly contact with. A measure that captures less frequent interactions (e.g., monthly, bi-annually) may capture a larger decrease in peripheral members. To create a sample that would demonstrate only intentional pruning, we removed anyone who reported the loss of a partner or other close relationship between the waves. This did not change the observed decrease in close or peripheral members, but it did weaken the significance of the overall increase in social network ratio. This could be explained by the slightly reduced decrease in peripheral members observed in this smaller sample.

We additionally identified baseline age and IQ as potential moderators of network narrowing, although they did not withstand correction for multiple comparisons. Higher baseline age and lower baseline IQ were each associated with less narrowing measured by the strict ratio. This could suggest individuals who are older at baseline have likely already started narrowing their networks, and individuals with a higher IQ may have more capability of narrowing intentionally. It is unlikely that the IQ findings are explained by age considering the correlation between IQ and age at baseline is small (r= -.01) indicating they represent distinct constructs that may be relevant for social network narrowing during the transition from midlife to older age.

As expected, inflammatory markers IL-6 and CRP increased from wave 1 to wave 2, replicating previous research indicating circulating inflammatory markers increase with age. Specifically, IL-6 increased a raw average of .10 (pg/mL) per year during the study, which, given an average sample age of 45 at wave 1 and 60 at wave 2, aligns with previous research demonstrating a raw increase between .03-.14 (pg/mL) per year (Lustig et al., 2017). CRP increased a raw average of .03 (mg/L) per year and in a healthy midlife population we expected a raw increase of .02 (mg/L) per year (Lassale et al., 2019). CRP findings were also robust when

excluding outliers with low values. Unexpectedly, TNF-α decreased over time but the mean of the difference was small and did not meet standard criteria for statistical significance (p=.051). This contradicts previous studies that have demonstrated a .02 (pg/mL) increase in log transformed TNF-α per year in a sample of older adults (Lindbergh et al., 2020). Despite not demonstrating the expected increase over time, TNF-α was positively correlated, as expected, with other variables including age at baseline, BMI, IL-6, and CRP. Additionally, the Midlife in the United States sample, with a mean age of 54, reported an average log transformed TNF-α value of 1.92 (pg/mL) (Ospina et al., 2022); comparatively in our sample at baseline (mean age= 45) there was a log mean of 2.20 (pg/mL). Notably, the samples from wave 1 were stored at -80°C, and then assayed for TNF-α after the wave 2 samples were collected (9-20 years later); TNF-α serum samples stored at -80°C may have lower stability and measuring TNF receptor 1 (TNFR-1) may be more reliable because it is more stable (Justice et al., 2018).

We did not find evidence to support our hypothesis that more social network narrowing would be associated with lower levels of inflammation at wave 2. Sensitivity analyses testing individual relationship types suggest the importance of neighbors (IL-6: p= .018) and close friends (CRP: p= .088), but these findings only applied to one inflammatory marker each and did not withstand correction for multiple comparisons. These overall null findings do not align with previous work linking social network size, diversity, and composition to health benefits (Heffner et al., 2011; Loucks et al., 2006; Uchino et al., 2018). Considering that structural measures of social networks, including the social network ratio and individual relationship types were not associated with inflammation, the health benefits of social relationships in this sample may be better explained by functional measures of social relationships such as quality and support.

Sensitivity analyses also further controlled for the loss of a close relationship as a covariate, but this did not change the original associations. However, endorsing losing a close relationship was positively associated with inflammation (IL-6) at wave 2, but it did not reach statistical significance (p=.058). This association aligns with previous work linking the loss of close relationships and bereavement to higher levels of inflammation (Cohen et al., 2015).

We did not find support for our hypothesis that social network narrowing would be related to increases in positive affect over time. Change in social network composition was largely unrelated to changes in positive affect. Post-hoc, we explored associations between changes in *negative affect* and changes in social network composition, but there were also no significant correlations (Supplemental Table S3). Unexpectedly, retaining more peripheral social network members over time was associated with increases in positive affect. Potentially this is because peripheral members are voluntary relationships (Gallant et al., 2007; McHugh Power et al., 2019) or the activities surrounding these relationships such as volunteering or attending classes have emotional benefits.

We did not find evidence that change in positive affect is related to inflammation. The measure of positive affect (PANAS) was reliable both between and within people over time, ruling out this explanation for the lack of observed associations. However, whereas we expected positive affect to increase from W1 to W2 based on the SST (Boylan & Ryff, 2015), we instead observed a small, statistically significant decrease in positive affect (average decline of .13 PANAS points per year). Previous studies have identified similar slight decreases in positive affect that occur starting at age 60 (Charles et al., 2001), and because this sample ranged from 40-72 it is possible the individuals over 60 are responsible for the observed decrease. However, this possibility is limited by the weak correlation between age and change in positive affect (r= .06) in our sample.

Another possibility is that cross-sectional work demonstrates that positive affect has a positive quadratic association with age (Carstensen et al., 2000; Mroczek & Kolarz, 1998; Stone et al., 2010), but previous longitudinal work and our findings suggest that within-person, there are slight declines from midlife to older age in trait positive affect (decrease of .01 points per year, measured by the PANAS and Affect Balance Scale) (Charles et al., 2001; Hittner et al., 2020). In addition, considering these decreases in trait positive affect are modest, the PANAS measure used in our analyses may not have had sufficient variability to investigate the effects of changes on physical health. An alternative explanation as to why our findings do not support the SST is that the positive affect measure used in our analyses tends to assess high-arousal positive affect (e.g., excited, active) and the SST and previously observed age-related increases in positive affect may only apply to low-arousal positive affect (e.g., calm, placid). Some studies have indicated that older adults are more likely to report greater levels of low-arousal happiness than high-arousal happiness (Bjalkebring et al., 2015). Alternatively, studies focused on negative affect suggest older adults demonstrate a decrease in both low and high-arousal negative affect compared to younger adults (Mak & Schneider, 2022). In post-hoc analyses, negative affect significantly decreased over time in our sample, and we considered testing changes in negative affect as a mediator; however, the correlations between change in negative affect and inflammatory markers were modest and not in the expected direction.

5.1 Strengths and Limitations

Strengths of the current study include having two time points, an important contribution to the literature that has largely relied on cross-sectional evidence. Our sample also captured changes beginning in midlife whereas most research testing the SST has focused on older age groups. Multiple inflammatory markers were included in analyses to capture multiple pro-inflammatory markers relevant for immune responses and in this case identified potentially important methodological considerations for TNF-α. Additionally, this study probed various approaches to quantifying social network composition, including ratios of close to peripheral members, individual aspects of ratios (close vs. peripheral), and individual relationship types. This study has limitations including only having two time points, which limits the ability to test within-person associations between social network composition, positive affect, and inflammation. Our sample was also highly educated and predominantly white, limiting the generalizability of our results. Additionally, despite utilizing various social network composition categories, we had no confirmation that the classifications accurately captured the social network members closest to the participant. Lastly, the use of a trait positive affect measure may have limited variability in changes over time.

5.2 Conclusion

In summary, we found some support that social networks narrow with age primarily through the reduction of peripheral relationships. In addition, two measures of inflammation (CRP

and IL-6) increased as expected. However, in terms of the SST these findings do not strongly support the hypothesis that social network narrowing is adaptive for promoting positive affect or inflammation. Overall, these findings may challenge which samples and study designs the SST applies to. For instance, the previously established positive association between age and positive affect may not extend to older age groups (aged 60+). Additionally, there is cross-sectional evidence for the age related increase in positive affect but these findings have not been replicated in various longitudinal studies including the current analysis (Charles et al., 2001; Hittner et al., 2020; Pinquart, 2001). Another important consideration when examining changes in positive affect is to assess measures of both high- and low-arousal emotions because they may display different trajectories across the lifespan. Our results point toward the importance of using multiple social network measurements and suggest that measures of relationship quality and closeness may also be important to investigate in relation to health. This study provides an example of how the SNI can be used to characterize additional aspects of social network composition including number of close relationships, peripheral relationships, or specific relationship types as opposed to the commonly used measures of social network size and diversity.

Appendix A Tables and Figures in Main Document

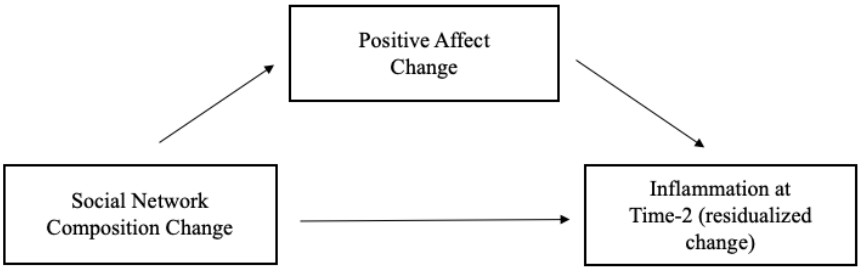

Figure 1. Proposed mediation model for Aim 3

Table 1: Descriptives for constant variables

		Total (N=350)
Sex (reference= male)		45.4%
Years between W1 and W2		14.9 (2.78)
Race		
	White (0)	312 (89.1%)
	Black (1)	36 (10.3%)
	Asian (2)	2 (0.6%)
Current Smoke Status (W2) (reference= no)		92.3%
AHAB Cohort (reference= cohort 1)		72.3%
Years of Education (W2)		16.83 (3.18)

Table 2: Descriptives for wave 1 and wave 2

	W1	W2	Change Score (W2-W1)
Age			
Mean (SD)	45.1 (6.61)	60.0 (7.39)	14.9 (2.78)
Median [Min, Max]	46.0 [30.0, 54.0]	61.0 [40.0, 72.0]	16.0 [9.00, 20.0]
Body Mass Index (BMI)			
Mean (SD)	26.2 (4.93)	27.7 (5.79)	1.53 (2.98)
Median [Min, Max]	25.0 [18.0, 47.0]	26.6 [15.8, 55.6]	1.40 [-10.2, 10.0]
Social Network Total			
Mean (SD)	20.8 (9.33)	16.2 (8.44)	-4.64 (8.04)
Median [Min, Max]	21.0 [0, 52.0]	15.0 [0, 46.0]	-4.00 [-30.0, 17.0]
Strict Ratio			
Mean (SD)	0.564 (0.683)	0.715 (0.843)	0.151 (0.957)
Median [Min, Max]	0.369 [0.0435, 5.00]	0.429 [0.0435, 5.00]	0.00528 [-4.00, 4.67]
Broad Ratio			
Mean (SD)	1.15 (1.31)	1.46 (1.58)	0.319 (1.84)
Median [Min, Max]	0.769 [0.0909, 11.0]	0.931 [0.120, 12.0]	0.0851 [-9.00, 10.7]
Close Total			
Mean (SD)	3.63 (2.04)	2.79 (1.85)	-0.846 (1.45)
Median [Min, Max]	4.00 [0, 9.00]	3.00 [0, 8.00]	-1.00 [-5.00, 5.00]
Moderate Total			
Mean (SD)	8.73 (3.93)	7.43 (3.72)	-1.31 (3.68)
Median [Min, Max]	8.00 [0, 20.0]	7.00 [0, 20.0]	-1.00 [-18.0, 13.0]
Peripheral Total			
Mean (SD)	12.1 (7.28)	8.76 (6.59)	-3.33 (6.57)
Median [Min, Max]	11.0 [0, 35.0]	8.00 [0, 33.0]	-3.00 [-26.0, 14.0]
Positive Affect			
Mean (SD)	35.4 (6.44)	33.4 (6.59)	-2.01 (5.92)
Median [Min, Max]	36.0 [13.0, 50.0]	34.0 [12.0, 50.0]	-2.00 [-24.0, 23.0]
Negative Affect			
Mean (SD)	14.6 (4.80)	13.5 (4.04)	-1.13 (4.45)
Median [Min, Max]	13.0 [10.0, 39.0]	12.0 [10.0, 30.0]	0 [-17.0, 14.0]
IL-6 (log) (pg/mL)			
Mean (SD)	0.143 (0.840)	0.531 (0.740)	0.388 (0.942)
Median [Min, Max]	0.0573 [-3.41, 4.53]	0.475 [-0.970, 6.20]	0.440 [-3.87, 4.57]
CRP (log) (mg/L)			
Mean (SD)	-0.215 (1.03)	0.0213 (1.33)	0.236 (1.11)
Median [Min, Max]	-0.294 [-1.90, 2.30]	0.0723 [-4.61, 2.28]	0.330 [-4.34, 3.02]
TNF-α (log) (pg/mL)			
Mean (SD)	2.19 (0.361)	2.15 (0.298)	-0.0384 (0.366)
Median [Min, Max]	2.15 [1.19, 4.77]	2.12 [1.25, 3.66]	-0.0268 [-2.69, 1.74]

Note. SNI total and other social network sums are raw numbers.

Table 3: Change in SNI paired t-tests

	Strict ratio	Broader Ratio	Close sum (Strict numerator)	Moderate sum (Broader numerator)	Peripheral sum (denominator)
Mean W1	.56	1.15	3.63	8.73	12.09
Mean W2	.71	1.46	2.78	7.42	8.76
t-test statistics					
Mean of difference	-.15	-.32	0.85	1.31	3.33
95% CI	[-.25, -.05]	[-.51, -.13]	[.69, 1.00]	[.92, 1.69]	[2.64, 4.03]
t-value	-2.95	-3.24	10.94	6.64	9.49
Df	349	349	349	349	349
p-value	.003	.001	2.2e-16	1.21e-10	2.2e-16

Table 4: Removing individuals who reported loss or separation from a partner

	Strict ratio	Broader Ratio	Close sum	Moderate sum	Peripheral sum
Mean W1	.56	1.15	3.61	8.73	11.95
Mean W2	.72	1.46	2.83	7.43	8.72
t-test statistics					
Mean of difference	-.16	-.31	.78	1.30	3.23
95% CI	[-.26, -.05]	[-.52, -.11]	[.62, .94]	[.89, 1.70]	[2.53, 3.92]
t-value	-2.94	-3.00	9.88	6.33	9.14
Df	326	326	326	326	326
p-value	.004	.002	2.2e-16	8.2e-10	2.2e-16

Note. 23 people removed based on reporting the loss or separation from a partner.

Table 5: Removing individuals who reported loss of any close member using STRAIN

	Strict ratio	Broader Ratio	Close sum	Moderate sum	Peripheral sum
Mean W1	.57	1.16	3.67	8.83	12.12
Mean W2	.67	1.37	2.75	7.42	9.01
t-test statistics					
Mean of difference	-.095	-.21	.92	1.41	3.12
95% CI	[-.20, .009]	[-.41, -.008]	[.75, 1.08]	[.99, 1.84]	[2.37, 3.85]
t-value	-1.79	-2.04	11.10	6.54	8.25
Df	305	305	305	305	305
p-value	.074	.042	2.2e-16	2.6e-10	4.78e-15

Note. 44 people removed who reported the loss of a close relationship between wave 1 and 2.

Table 6: Testing moderators of change in ratios

	Delta strict ratio			Delta broader ratio	
	b (SE)	p-value		*b* (SE)	p-value
Age visit 1					
Intercept	.15 (.05)	.003	Intercept	.31 (.10)	.001
Age	-.02 (.008)	**.027**	Age	-.01 (.01)	.415
Time between visits					
Intercept	.15 (.05)	.003	Intercept	.32 (.10)	.001
Timelapse	-.02 (.02)	.390	Timelapse	-.02 (.04)	.491
Sex					
Intercept	.20 (.08)	.008	Intercept	.34 (.15)	.022
Sex (0=male, 1= female)	-.09 (.10)	.363	Sex (0=male, 1= female)	-.03 (.20)	.877
IQ at visit 1					
Intercept	.52 (.05)	.003	Intercept	.32 (.10)	.001
IQ	.01 (.005)	**.041**	IQ	.01 (.008)	.298
IL-6 at visit 1					
Intercept	.15 (.05)	.003	Intercept	.32 (.10)	.001
Log IL-6 (pg/mL)	-.08 (.06)	.193	Log IL-6 (pg/mL)	-.09 (.12)	.436
CRP at visit 1					
Intercept	.15 (.05)	.003	Intercept	.32 (.10)	.001
TNF-α at visit 1					
Intercept	.15 (.05)	.003	Intercept	.32 (.10)	.001
Log TNF-α (pg/mL)	.04 (.14)	.801	Log TNF-α (pg/mL)	.01 (.27)	.978

Note. continuous predictors are mean centered.

*Results that survived correction for multiple comparison (threshold *p*=.025 correcting for groups of 2)

Table 7: Change in inflammation paired t-tests

	IL-6 (pg/mL)		CRP (mg/L)		TNF-α (pg/mL)	
	Log mean	Raw mean	Log mean	Raw mean	Log mean	Raw mean
Mean W1	.14	2.14	-.22	1.38	2.19	9.79
Mean W2	.53	3.69	-.02	1.90	2.15	9.07
t-test statistics						
Mean of difference	-.39		-.24		.038	
95% CI	[-.49, -.29]		[-.325, -.11]		[-.0001, .07]	
t-value	-7.70		-3.99		1.96	
Df	349		349		349	
p-value	1.39e-13		8.14e-5		0.051	

Note. Natural log transoformed and corresponding raw means reported.

Table 8: Removing CRP individuals with low levels and re-testing aim 1 change in inflammation

	CRP (mg/L)	
	Log mean	Raw mean
Mean W1	-.16	1.43
Mean W2	.20	1.97
t-test statistics		
Mean of difference	-.36	
95% CI	[-.46, -.27]	
t-value	-7.32	
Df	336	
p-value	1.85e-12	

Note. 13 people removed for having log CRP values below -4

Table 9: Strict ratio predicting inflammation at W2

	Log IL-6				Log CRP				Log TNF-α			
Predictors	*ß*	*B*	*SE*	*p-value*	*ß*	*B*	*SE*	*p-value*	*ß*	*B*	*SE*	*p-value*
(Intercept)	0.01	-0.85	0.90	0.344	-0.12	-2.58	1.39	0.065	0.00	0.93	0.38	**0.014**
Delta strict ratio	-0.04	-0.03	0.04	0.412	0.00	0.00	0.06	0.967	-0.01	-0.00	0.02	0.765
Age	0.16	0.02	0.01	**0.001**	0.03	0.01	0.01	0.455	0.13	0.01	0.00	**0.014**
Sex	0.05	0.04	0.07	0.621	0.09	0.12	0.11	0.290	-0.07	-0.02	0.03	0.509
BMI	0.30	0.04	0.01	**<0.001**	0.29	0.07	0.01	**<0.001**	0.06	0.00	0.00	0.244
Smoke (0=no, 1=yes)	0.62	0.46	0.14	**0.001**	0.11	0.15	0.21	0.494	-0.07	-0.02	0.06	0.711
Cohort	-0.31	-0.23	0.31	0.447	0.24	0.31	0.48	0.512	0.15	0.04	0.13	0.723
Timelapse	-0.15	-0.04	0.05	0.427	0.08	0.04	0.08	0.604	0.15	0.02	0.02	0.413
SNI total	0.05	0.00	0.00	0.269	-0.06	-0.01	0.01	0.148	-0.05	-0.00	0.00	0.342
Log IL-6 (pg/mL)	0.22	0.19	0.04	**<0.001**								
Log CRP (mg/L)					0.47	0.61	0.06	**<0.001**				
Log TNF-α (pg/mL)									0.37	0.30	0.04	**<0.001**
Observations	350				350				350			
R^2 / R^2 adjusted	0.223 / 0.203				0.415 / 0.400				0.188 / 0.167			

Note. covariates at wave 1 include age, SNI total, and inflammation; covariates at wave 2 include BMI, current smoking status, and sex. Timelapse is the time between wave 1 and wave 2.

Table 10: Broad ratio predicting inflammation at W2

	Log IL-6				**Log CRP**				**Log TNF-α**			
Predictors	*ß*	*B*	*SE*	*p-value*	*ß*	*B*	*SE*	*p-value*	*ß*	*B*	*SE*	*p-value*
(Intercept)	0.01	-0.83	0.90	0.357	-0.12	-2.54	1.40	0.070	0.00	0.95	0.38	**0.012**
Delta broad ratio	-0.04	-0.02	0.02	0.387	-0.02	-0.01	0.03	0.683	-0.04	-0.01	0.01	0.378
Age	0.16	0.02	0.01	**0.001**	0.03	0.01	0.01	0.468	0.12	0.01	0.00	**0.013**
Sex	0.05	0.04	0.07	0.608	0.09	0.12	0.11	0.294	-0.07	-0.02	0.03	0.506
BMI	0.29	0.04	0.01	**<0.001**	0.29	0.07	0.01	**<0.001**	0.06	0.00	0.00	0.259
Smoke (0=no, 1=yes)	0.63	0.47	0.14	**0.001**	0.11	0.15	0.21	0.492	-0.06	-0.02	0.06	0.734
Cohort	-0.33	-0.24	0.31	0.428	0.23	0.30	0.48	0.523	0.14	0.04	0.13	0.749
Timelapse	-0.15	-0.04	0.05	0.408	0.08	0.04	0.08	0.619	0.15	0.02	0.02	0.436
SNI total	0.05	0.00	0.00	0.272	-0.06	-0.01	0.01	0.162	-0.05	-0.00	0.00	0.374
Log IL-6 (pg/mL)	0.22	0.20	0.04	**<0.001**								
Log CRP (mg/L)					0.47	0.61	0.06	**<0.001**				
Log TNF-α (pg/mL)									0.37	0.30	0.04	**<0.001**
Observations	350				350				350			
R^2 / R^2 adjusted	0.223 / 0.203				0.416 / 0.400				0.190 / 0.168			

Note. covariates at wave 1 include age, SNI total, and inflammation; covariates at wave 2 include BMI, current smoking status, and sex. Timelapse is the time between wave 1 and wave 2.

Table 11: Close Sum predicting inflammation at W2

	Log IL-6				**Log CRP**				**Log TNF-α**			
Predictors	*ß*	*B*	*SE*	*p-value*	*ß*	*B*	*SE*	*p-value*	*ß*	*B*	*SE*	*p-value*
(Intercept)	0.01	-0.80	0.90	0.374	-0.12	-2.64	1.40	0.059	-0.00	0.91	0.38	**0.016**
Delta close sum	-0.07	-0.04	0.03	0.155	0.03	0.03	0.04	0.466	0.04	0.01	0.01	0.480
Age	0.15	0.02	0.01	**0.002**	0.04	0.01	0.01	0.389	0.13	0.01	0.00	**0.009**
Sex	0.05	0.04	0.07	0.600	0.09	0.12	0.11	0.290	-0.07	-0.02	0.03	0.518
BMI	0.30	0.04	0.01	<**0.001**	0.29	0.07	0.01	<**0.001**	0.06	0.00	0.00	0.267
Smoke (0=no, 1=yes)	0.64	0.47	0.14	**0.001**	0.11	0.14	0.21	0.505	-0.07	-0.02	0.06	0.718
Cohort	-0.32	-0.24	0.31	0.438	0.24	0.32	0.48	0.504	0.16	0.05	0.13	0.714
Timelapse	-0.16	-0.04	0.05	0.396	0.09	0.04	0.08	0.580	0.16	0.02	0.02	0.392
SNI total	0.03	0.00	0.00	0.499	-0.06	-0.01	0.01	0.205	-0.04	-0.00	0.00	0.409
Log IL-6 (pg/mL)	0.22	0.19	0.04	<**0.001**								
Log CRP (mg/L)					0.48	0.61	0.06	<**0.001**				
Log TNF-α (pg/mL)									0.37	0.30	0.04	<**0.001**
Observations	350				350				350			
R^2 / R^2 adjusted	0.226 / 0.206				0.416 / 0.401				0.189 / 0.168			

Note. covariates at wave 1 include age, SNI total, and inflammation; covariates at wave 2 include BMI, current smoking status, and sex. Timelapse is the time between wave 1 and wave 2.

Table 12: Moderate Sum predicting inflammation at W2

	Log IL-6				Log CRP				Log TNF-α			
Predictors	*ß*	*B*	*SE*	*p-value*	*ß*	*B*	*SE*	*p-value*	*ß*	*B*	*SE*	*p-value*
(Intercept)	0.01	-0.84	0.90	0.354	-0.12	-2.58	1.40	0.065	0.00	0.93	0.38	**0.014**
Delta moderate sum	-0.03	-0.01	0.01	0.505	0.00	0.00	0.02	0.936	-0.01	-0.00	0.00	0.842
Age	0.17	0.02	0.01	**0.001**	0.03	0.01	0.01	0.457	0.13	0.01	0.00	**0.012**
Sex	0.05	0.04	0.07	0.593	0.09	0.12	0.11	0.291	-0.07	-0.02	0.03	0.518
BMI	0.30	0.04	0.01	<**0.001**	0.29	0.07	0.01	<**0.001**	0.06	0.00	0.00	0.238
Smoke (0=no, 1=yes)	0.63	0.47	0.14	**0.001**	0.11	0.15	0.21	0.496	-0.06	-0.02	0.06	0.732
Cohort	-0.33	-0.25	0.31	0.421	0.24	0.32	0.48	0.509	0.14	0.04	0.13	0.734
Timelapse	-0.16	-0.04	0.05	0.401	0.08	0.04	0.08	0.601	0.15	0.02	0.02	0.422
SNI total	0.04	0.00	0.00	0.435	-0.06	-0.01	0.01	0.168	-0.05	-0.00	0.00	0.310
Log IL-6 (pg/mL)	0.22	0.20	0.04	<**0.001**								
Log CRP (mg/L)					0.47	0.61	0.06	<**0.001**				
Log TNF-α (pg/mL)									0.37	0.30	0.04	<**0.001**
Observations	350				350				350			
R^2 / R^2 adjusted	0.223 / 0.202				0.415 / 0.400				0.188 / 0.167			

Note. covariates at wave 1 include age, SNI total, and inflammation; covariates at wave 2 include BMI, current smoking status, and sex. Timelapse is the time between wave 1 and wave 2.

Table 13: Peripheral Sum predicting inflammation at W2

	Log IL-6				Log CRP				Log TNF-α			
Predictors	*ß*	*B*	*SE*	*p-value*	*ß*	*B*	*SE*	*p-value*	*ß*	*B*	*SE*	*p-value*
(Intercept)	0.01	-0.88	0.90	0.326	-0.11	-2.53	1.39	0.070	0.00	0.93	0.38	**0.014**
Delta peripheral sum	-0.03	-0.00	0.01	0.648	0.07	0.01	0.01	0.146	0.03	0.00	0.00	0.573
Age	0.17	0.02	0.01	**0.001**	0.03	0.01	0.01	0.456	0.13	0.01	0.00	**0.012**
Sex	0.05	0.04	0.07	0.579	0.08	0.11	0.11	0.335	-0.07	-0.02	0.03	0.495
BMI	0.30	0.04	0.01	<**0.001**	0.29	0.07	0.01	<**0.001**	0.06	0.00	0.00	0.244
Smoke (0=no, 1=yes)	0.63	0.47	0.14	**0.001**	0.11	0.15	0.21	0.480	-0.06	-0.02	0.06	0.739
Cohort	-0.30	-0.22	0.31	0.467	0.21	0.27	0.47	0.565	0.14	0.04	0.13	0.747
Timelapse	-0.14	-0.04	0.05	0.444	0.07	0.03	0.08	0.648	0.15	0.02	0.02	0.425
SNI total	0.04	0.00	0.00	0.518	-0.03	-0.00	0.01	0.580	-0.03	-0.00	0.00	0.554
Log IL-6 (pg/mL)	0.22	0.20	0.04	<**0.001**								
Log CRP (mg/L)					0.48	0.62	0.06	<**0.001**				
Log TNF-α (pg/mL)									0.37	0.30	0.04	<**0.001**
Observations	350				350				350			
R^2 / R^2 adjusted	0.222 / 0.201				0.419 / 0.404				0.189 / 0.167			

Note. covariates at wave 1 include age, SNI total, and inflammation; covariates at wave 2 include BMI, current smoking status, and sex. Timelapse is the time between wave 1 and wave 2.

Table 14: Strict ratio predicting inflammation at W2 with death of close member covariate

	Log IL-6				Log CRP				Log TNF-α			
Predictors	*ß*	*B*	*SE*	*p-value*	*ß*	*B*	*SE*	*p-value*	*ß*	*B*	*SE*	*p-value*
(Intercept)	0.00	-0.83	0.90	0.354	-0.13	-2.56	1.40	0.067	0.00	0.93	0.38	**0.014**
Delta strict ratio	-0.05	-0.04	0.04	0.278	-0.00	-0.00	0.06	0.954	-0.01	-0.00	0.02	0.813
Age	0.18	0.02	0.01	**<0.001**	0.04	0.01	0.01	0.394	0.12	0.01	0.00	**0.019**
Sex	0.04	0.03	0.07	0.716	0.09	0.11	0.11	0.314	-0.06	-0.02	0.03	0.530
BMI	0.30	0.04	0.01	**<0.001**	0.30	0.07	0.01	**<0.001**	0.06	0.00	0.00	0.254
Smoke (0=no, 1=yes)	0.64	0.47	0.14	**0.001**	0.12	0.15	0.21	0.475	-0.07	-0.02	0.06	0.698
Cohort	-0.38	-0.28	0.31	0.353	0.21	0.28	0.48	0.555	0.17	0.05	0.13	0.696
Timelapse	-0.18	-0.05	0.05	0.327	0.07	0.03	0.08	0.656	0.16	0.02	0.02	0.392
SNI total	0.06	0.00	0.00	0.212	-0.06	-0.01	0.01	0.164	-0.05	-0.00	0.00	0.329
Death of close member	0.28	0.21	0.11	0.061	0.09	0.12	0.17	0.495	-0.06	-0.02	0.05	0.683
Log IL-6 (pg/mL)	0.22	0.19	0.04	**<0.001**								
Log CRP (mg/L)					0.48	0.61	0.06	**<0.001**				
Log TNF-α (pg/mL)									0.37	0.30	0.04	**<0.001**
Observations	350				350				350			
R^2 / R^2 adjusted	0.231 / 0.208				0.416 / 0.399				0.189 / 0.165			

Note. covariates at wave 1 include age, SNI total, and inflammation; covariates at wave 2 include BMI, current smoking status, and sex. Timelapse is the time between wave 1 and wave 2. Death of a close member accounts for the loss of any close relationships between wave 1 and wave 2.

Table 15: Broader ratio predicting inflammation at W2 with death of close member covariate

	Log IL-6				Log CRP				Log TNF-α			
Predictors	*ß*	*B*	*SE*	*p-value*	*ß*	*B*	*SE*	*p-value*	*ß*	*B*	*SE*	*p-value*
(Intercept)	0.01	-0.80	0.90	0.373	-0.12	-2.52	1.40	0.072	0.01	0.95	0.38	**0.012**
Delta broad ratio	-0.06	-0.02	0.02	0.241	-0.02	-0.02	0.03	0.597	-0.04	-0.01	0.01	0.413
Age	0.18	0.02	0.01	**<0.001**	0.04	0.01	0.01	0.394	0.12	0.01	0.00	**0.018**
Sex	0.04	0.03	0.07	0.701	0.09	0.11	0.11	0.320	-0.06	-0.02	0.03	0.521
BMI	0.30	0.04	0.01	**<0.001**	0.30	0.07	0.01	**<0.001**	0.06	0.00	0.00	0.267
Smoke (0=no, 1=yes)	0.65	0.48	0.14	**<0.001**	0.12	0.16	0.21	0.466	-0.07	-0.02	0.06	0.721
Cohort	-0.41	-0.30	0.31	0.329	0.20	0.27	0.48	0.575	0.15	0.04	0.13	0.728
Timelapse	-0.19	-0.05	0.05	0.303	0.07	0.03	0.08	0.681	0.15	0.02	0.02	0.420
SNI total	0.06	0.00	0.00	0.213	-0.06	-0.01	0.01	0.182	-0.05	-0.00	0.00	0.363
Death of close member	0.29	0.21	0.11	0.057	0.10	0.13	0.17	0.447	-0.05	-0.01	0.05	0.761
Log IL-6 (pg/mL)	0.22	0.19	0.04	**<0.001**								
Log CRP (mg/L)					0.48	0.61	0.06	**<0.001**				
Log TNF-α (pg/mL)									0.37	0.30	0.04	**<0.001**
Observations	350				350				350			
R^2 / R^2 adjusted	0.232 / 0.209				0.417 / 0.400				0.190 / 0.166			

Note. covariates at wave 1 include age, SNI total, and inflammation; covariates at wave 2 include BMI, current smoking status, and sex. Timelapse is the time between wave 1 and wave 2. Death of a close member accounts for the loss of any close relationships between wave 1 and wave 2.

Table 16: Close Sum predicting inflammation at W2 with death of close member covariate

	Log IL-6				Log CRP				Log TNF-α			
Predictors	*ß*	*B*	*SE*	*p-value*	*ß*	*B*	*SE*	*p-value*	*ß*	*B*	*SE*	*p-value*
(Intercept)	0.00	-0.78	0.89	0.386	-0.13	-2.63	1.40	0.061	0.00	0.91	0.38	**0.017**
Delta close sum	-0.08	-0.04	0.03	0.106	0.03	0.03	0.04	0.509	0.04	0.01	0.01	0.449
Age	0.17	0.02	0.01	**0.001**	0.04	0.01	0.01	0.342	0.13	0.01	0.00	**0.013**
Sex	0.04	0.03	0.07	0.687	0.09	0.12	0.11	0.309	-0.06	-0.02	0.03	0.542
BMI	0.31	0.04	0.01	**<0.001**	0.29	0.07	0.01	**<0.001**	0.05	0.00	0.00	0.284
Smoke (0=no, 1=yes)	0.66	0.49	0.14	**<0.001**	0.11	0.15	0.21	0.483	-0.07	-0.02	0.06	0.695
Cohort	-0.39	-0.29	0.31	0.343	0.22	0.29	0.48	0.543	0.18	0.05	0.13	0.679
Timelapse	-0.19	-0.05	0.05	0.297	0.08	0.04	0.08	0.627	0.17	0.02	0.02	0.365
SNI total	0.04	0.00	0.00	0.459	-0.05	-0.01	0.01	0.213	-0.04	-0.00	0.00	0.400
Death of close member	0.28	0.21	0.11	0.058	0.08	0.10	0.17	0.542	-0.08	-0.02	0.05	0.600
Log IL-6 (pg/mL)	0.22	0.19	0.04	**<0.001**								
Log CRP (mg/L)					0.48	0.61	0.06	**<0.001**				
Log TNF-α (pg/mL)									0.37	0.31	0.04	**<0.001**
Observations	350				350				350			
R^2 / R^2 adjusted	0.234 / 0.212				0.417 / 0.400				0.190 / 0.166			

Note. covariates at wave 1 include age, SNI total, and inflammation; covariates at wave 2 include BMI, current smoking status, and sex. Timelapse is the time between wave 1 and wave 2. Death of a close member accounts for the loss of any close relationships between wave 1 and wave 2.

Table 17: Moderate Sum predicting inflammation at W2 with death of close member covariate

	Log IL-6				Log CRP				Log TNF-α			
Predictors	*ß*	*B*	*SE*	*p-value*	*ß*	*B*	*SE*	*p-value*	*ß*	*B*	*SE*	*p-value*
(Intercept)	0.01	-0.81	0.90	0.365	-0.13	-2.57	1.40	0.067	0.00	0.93	0.38	**0.014**
Delta moderate sum	-0.04	-0.01	0.01	0.407	0.00	0.00	0.02	0.985	-0.01	-0.00	0.00	0.873
Age	0.18	0.02	0.01	**<0.001**	0.04	0.01	0.01	0.391	0.12	0.01	0.00	**0.017**
Sex	0.04	0.03	0.07	0.673	0.09	0.12	0.11	0.313	-0.06	-0.02	0.03	0.537
BMI	0.30	0.04	0.01	**<0.001**	0.30	0.07	0.01	**<0.001**	0.06	0.00	0.00	0.250
Smoke (0=no, 1=yes)	0.65	0.48	0.14	**<0.001**	0.12	0.15	0.21	0.472	-0.07	-0.02	0.06	0.714
Cohort	-0.41	-0.30	0.31	0.329	0.21	0.28	0.48	0.554	0.16	0.05	0.13	0.704
Timelapse	-0.19	-0.05	0.05	0.304	0.07	0.03	0.08	0.655	0.16	0.02	0.02	0.399
SNI total	0.04	0.00	0.00	0.404	-0.06	-0.01	0.01	0.175	-0.05	-0.00	0.00	0.304
Death of close member	0.27	0.20	0.11	0.072	0.09	0.12	0.17	0.499	-0.07	-0.02	0.05	0.667
Log IL-6 (pg/mL)	0.22	0.20	0.04	**<0.001**								
Log CRP (mg/L)					0.48	0.61	0.06	**<0.001**				
Log TNF-α (pg/mL)									0.37	0.30	0.04	**<0.001**
Observations	350				350				350			
R^2 / R^2 adjusted	0.230 / 0.207				0.416 / 0.399				0.189 / 0.165			

Note. covariates at wave 1 include age, SNI total, and inflammation; covariates at wave 2 include BMI, current smoking status, and sex. Timelapse is the time between wave 1 and wave 2. Death of a close member accounts for the loss of any close relationships between wave 1 and wave 2.

Table 18: Peripheral Sum predicting inflammation at W2 with death of close member covariate

	Log IL-6				Log CRP				Log TNF-α			
Predictors	*ß*	*B*	*SE*	*p-value*	*ß*	*B*	*SE*	*p-value*	*ß*	*B*	*SE*	*p-value*
(Intercept)	-0.00	-0.87	0.90	0.333	-0.11	-2.51	1.39	0.072	0.01	0.93	0.38	**0.014**
Delta peripheral sum	-0.01	-0.00	0.01	0.829	0.08	0.02	0.01	0.118	0.03	0.00	0.00	0.613
Age	0.18	0.02	0.01	**<0.001**	0.04	0.01	0.01	0.367	0.12	0.01	0.00	**0.016**
Sex	0.04	0.03	0.07	0.665	0.08	0.10	0.11	0.371	-0.07	-0.02	0.03	0.515
BMI	0.30	0.04	0.01	**<0.001**	0.29	0.07	0.01	**<0.001**	0.06	0.00	0.00	0.253
Smoke (0=no, 1=yes)	0.65	0.48	0.14	**<0.001**	0.12	0.16	0.21	0.449	-0.07	-0.02	0.06	0.722
Cohort	-0.37	-0.27	0.31	0.372	0.17	0.23	0.48	0.628	0.15	0.05	0.13	0.720
Timelapse	-0.18	-0.05	0.05	0.346	0.06	0.03	0.08	0.720	0.16	0.02	0.02	0.405
SNI total	0.05	0.00	0.00	0.399	-0.02	-0.00	0.01	0.658	-0.04	-0.00	0.00	0.528
Death of close member	0.25	0.19	0.11	0.092	0.12	0.16	0.17	0.368	-0.06	-0.02	0.05	0.710
Log IL-6 (pg/mL)	0.22	0.19	0.04	**<0.001**								
Log CRP (mg/L)					0.48	0.62	0.06	**<0.001**				
Log TNF-α (pg/mL)									0.37	0.31	0.04	**<0.001**
Observations	350				350				350			
R^2 / R^2 adjusted	0.229 / 0.206				0.420 / 0.403				0.189 / 0.165			

Note. covariates at wave 1 include age, SNI total, and inflammation; covariates at wave 2 include BMI, current smoking status, and sex. Timelapse is the time between wave 1 and wave 2. Death of a close member accounts for the loss of any close relationships between wave 1 and wave 2.

Table 19: Relationship Types predicting inflammation at W2

	IL-6		**CRP**		**TNF-α**	
	b (SE)	p-value	*b* (SE)	p-value	*b* (SE)	p-value
Children	-.05(.04)	.267	.05 (.07)	.469	-.004 (.02)	.847
Parents/in-laws	-.02 (.03)	.475	-.01 (.05)	.838	.02 (.01)	.153
Relatives	-.002 (.02)	.914	.04 (.03)	.186	-.001 (.007)	.939
Close friends	-.002 (.02)	.928	-.05 (.03)	.088	-.005 (.007)	.449
Churchmates	-.01 (.02)	.497	.01 (.03)	.70	.002 (.007)	.731
Neighbors	-.05 (.02)	**.018**	-.01 (.03)	.692	-.002 (.008)	.779

*Results that survived correction for multiple comparison (threshold *p*=.016 correcting for groups of 3).

Table 20: Strict ratio change predicting Inflammation through change in Positive Affect

Outcome	Total effect *c*	Indirect Effect (a x b)	Path a	Path b	Direct Effect *c'*
	b (SE)	*b* (SE)	*b* (SE)	*b* (SE)	*b* (SE)
Log IL-6 (pg/mL)	-.037 (.034)	.002 (.004)	-.27 (.34)	-.008 (.006)	-.040 (.034)
Log CRP (mg/L)	.011 (.055)	.003 (.006)	-.27 (.35)	-.011 (.010)	.008 (.055)
Log TNF-α (pg/mL)	-.006 (.012)	-.000 (.001)	-.27 (.36)	.001 (.003)	-.006 (.012)

*p<.05

Note. Covariates for all mediation models at wave 1 include age, SNI total, positive affect, and inflammation; covariates at wave 2 include BMI, current smoking status, and sex. Timelapse is the time between wave 1 and wave 2.

Table 21: Broader ratio change predicting Inflammation through change in Positive Affect

Outcome	Total effect *c*	Indirect Effect (a x b)	Path a	Path b	Direct Effect *c'*
	b (SE)	*b* (SE)	*b* (SE)	*b* (SE)	*b* (SE)
Log IL-6 (pg/mL)	-.019 (.016)	.001 (.002)	-.17 (.18)	-.008 (.006)	-.020 (.017)
Log CRP (mg/L)	-.010 (.033)	.002 (.003)	-.17 (.17)	-.012 (.010)	.012 (.033)
Log TNF-α (pg/mL)	-.007 (.006)	-.000 (.001)	-.17 (.18)	.001 (.003)	-.007 (.007)

*p<.05

Note. Covariates for all mediation models at wave 1 include age, SNI total, positive affect, and inflammation; covariates at wave 2 include BMI, current smoking status, and sex. Timelapse is the time between wave 1 and wave 2.

Table 22: Close sum change predicting Inflammation through change in Positive Affect

Outcome	Total effect *c*	Indirect Effect (a x b)	Path a	Path b	Direct Effect *c'*
	b (SE)	*b* (SE)	*b* (SE)	*b* (SE)	*b* (SE)
Log IL-6 (pg/mL)	-.038 (.027)	-.001 (.002)	.18 (.20)	-.007 (.006)	-.036 (.027)
Log CRP (mg/L)	.030 (.045)	-.002 (.004)	.18 (.20)	-.012 (.010)	.033 (.045)
Log TNF-α (pg/mL)	.007 (.008)	.000 (.001)	.17 (.20)	.001 (.003)	.007 (.008)

*p<.05

Note. Covariates for all mediation models at wave 1 include age, SNI total, positive affect, and inflammation; covariates at wave 2 include BMI, current smoking status, and sex. Timelapse is the time between wave 1 and wave 2.

Table 23: Moderate sum change predicting Inflammation through change in Positive Affect

Outcome	Total effect *c*	Indirect Effect (a x b)	Path a	Path b	Direct Effect *c'*
	b (SE)	*b* (SE)	*b* (SE)	*b* (SE)	*b* (SE)
Log IL-6 (pg/mL)	-.007 (.013)	-.001 (.001)	.15 (.093)	-.007 (.006)	-.006 (.013)
Log CRP (mg/L)	.002 (.015)	-.002 (.002)	.15 (.092)	-.012 (.010)	.004 (.015)
Log TNF-α (pg/mL)	-.001 (.003)	.000 (.001)	.15 (.094)	.001 (.003)	-.001 (.003)

*p<.05

Note. Covariates for all mediation models at wave 1 include age, SNI total, positive affect, and inflammation; covariates at wave 2 include BMI, current smoking status, and sex. Timelapse is the time between wave 1 and wave 2.

Table 24: Peripheral sum change predicting Inflammation through change in Positive Affect

Outcome	Total effect *c*	Indirect Effect (a x b)	Path a	Path b	Direct Effect *c'*
	b (SE)	*b* (SE)	*b* (SE)	*b* (SE)	*b* (SE)
Log IL-6 (pg/mL)	-.002 (.006)	-.001 (.001)	.12 (.047)*	-.008 (.006)	-.001 (.006)
Log CRP (mg/L)	.013 (.009)	-.002 (.002)	.13 (.048)*	-.014 (.010)	.0014(.009)
Log TNF-α (pg/mL)	.002 (.003)	.000 (.000)	.12 (.049)*	.001 (.003)	.002 (.003)

*p<.05

Note. Covariates for all mediation models at wave 1 include age, SNI total, positive affect, and inflammation; covariates at wave 2 include BMI, current smoking status, and sex. Timelapse is the time between wave 1 and wave 2.

Appendix B Supplemental Tables

Table S1: Ratio categorizations using items from the SNI

Items	Ratio #1	Ratio #2
Are you married & living together, or living with someone in a marital like relationship?	Close	Close
How many children do you see or talk to at least once every two weeks?	Close	Close
Do you see or talk to either or both of your parents at least once every two weeks?	Close	Close
Do you see or talk to either or both of your partner's parents at least once every two weeks?	Close	Close
How many close relatives do you see or talk to at least once every two weeks?	n/a	Close
How many close friends do you see or talk to at least once every two weeks?	n/a	Close
How many members of religious groups do you see or talk to at least once every two weeks?	Peripheral	Peripheral
How many fellow students or teachers do you see or talk to at least once every two weeks?	Peripheral	Peripheral
How many people from work (other than those you supervise) do you see or talk to at least once every two weeks?	Peripheral	Peripheral
How many of your neighbors do you see or talk to at least once every two weeks?	Peripheral	Peripheral
How many people from volunteer work do you see or talk to at least once every two weeks?	Peripheral	Peripheral
How many people from other groups (e.g., social clubs, recreational groups, groups concerned with children) do you see or talk to at least once every two weeks?	Peripheral	Peripheral

Table S2: Inflammatory Marker CVs

	IL-6		TNF-α		CRP	
	Inter (range)	Intra (average)	Inter (range)	Intra (average)	Inter (range)	Intra (average)
Wave 1	5%	5.78	1.23-1.30		2.1-5.7%	2.3-4.4%
Wave 2	10%	3.68	1.14-1.33	2.13	2.5-4.4%	0.75-3.13%

Note. TNF-α intra-averages were collapsed across waves

Table S3: Bivariate correlations for variables in analyses

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
1. age w1																			
2. bmi w2	-.03																		
3. sni total w1	.02	.02																	
4. delta strict ratio	-.12*	.00	.15**																
5. delta broad ratio	-.04	-.04	.11*	.87**															
6. delta close sum	-.19**	.08	-.22**	.29**	.19**														
7. delta moderate sum	.03	.04	-.29**	.04	.23**	.45**													
8. delta peripheral sum	-.03	-.01	-.49**	-.50**	-.48**	.06	.16**												
9. delta positive affect	.06	.04	.02	.01	-.02	.06	.10	.05											
10. delta negative affect	-.07	-.10	.08	.05	.03	.01	-.07	-.06	-.31**										
11. logil6 w2	.20**	.30**	.03	-.09	-.07	-.10	-.01	-.05	.00	-.05									
12. logil6 w1	.14*	.11*	.02	-.07	-.04	-.12*	.00	-.06	-.03	.03	.29**								
13. logcrp w2	.09	.45**	-.05	-.03	-.05	.04	.05	.04	-.01	-.12*	.33**	.15**							
14. logcrp w1	.14**	.35**	.01	-.04	-.02	-.04	.03	-.08	.07	-.02	.28**	.29**	.58**						
15. logtnfa w2	.18**	.08	-.04	-.03	-.06	-.01	.01	.02	.02	-.16**	.25**	.12*	.17**	.13*					
16. logtnfa w1	.15**	.06	.02	.01	.00	-.05	.02	-.04	-.00	.08	.08	.35**	.09	.19**	.40**				
17. sex (ref=male)	.08	-.18**	.05	-.05	-.01	-.05	-.01	.02	-.04	.08	.01	.01	-.01	.00	-.04	-.03			
18. smoking status (ref=no)	.05	-.06	-.17**	-.11*	-.01	.03	.07	.06	.02	-.05	.18**	.11*	.09	.12*	-.01	-.02	.09		
19. cohort (ref=1)	-.11*	.00	.04	.05	.03	.13*	-.00	.04	.18**	-.11*	-.09	-.28**	-.05	-.13*	-.13*	-.10	-.08	-.08	
20. timelapse	.09	-.01	-.00	-.05	-.04	-.14**	-.03	-.04	-.17**	.09	.08	.29**	.04	.12*	.13*	.08	.07	.09	-.96**

Note. *indicates $p < .05$. ** indicates $p < .01$.

Table S4: Bivariate correlations for wave 1

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. age w1																
2. bmi w1	.13*															
3. sni total w1	.02	.03														
4. strict ratio w1	.11*	.01	-.46**													
5. broad ratio w1	.08	.05	-.39**	.87**												
6. close sum w1	-.04	-.10	.40**	.14*	-.01											
7. moderate sum w1	-.02	-.04	.68**	-.08	.09	.55**										
8. peripheral sum w1	.04	.05	.92**	-.55**	-.55**	.22**	.33**									
9. positive affect w1	.04	-.08	.29**	-.11*	-.07	.10	.28**	.21**								
10. negative affect w1	-.07	.05	-.18**	.15**	.11*	-.06	-.15**	-.15**	-.30**							
11. logil6 w1	.14*	.16**	.02	.02	.02	.00	.00	.02	.01	-.03						
12. logcrp w1	.14**	.42**	.01	.05	.06	-.08	-.03	.02	-.04	-.00	.29**					
13. logtnfa w1	.15**	.12*	.02	-.03	-.04	.01	-.05	.05	-.00	-.03	.35**	.19**				
14. sex (ref= male)	.08	-.23**	.05	.04	.12*	-.01	.19**	-.04	.03	-.09	.01	.00	-.03			
15. smoke (ref= no)	-.00	-.00	-.20**	.21**	.20**	-.13*	-.11*	-.20**	-.13*	.07	.07	.06	-.06	-.00		
16. cohort (ref=1)	-.11*	.03	.04	-.14*	-.14*	-.01	.01	.04	-.04	.11*	-.28**	-.13*	-.10	-.08	-.07	
17. timelapse	.09	-.03	-.00	.11*	.11*	.03	.01	-.01	.05	-.10	.29**	.12*	.08	.07	.06	-.96**

Note. *indicates $p < .05$. ** indicates $p < .01$.

Table S5: Bivariate correlations for wave 2

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1. age w2																
2. bmi w2	-.03															
3. sni total w2	-.01	.03														
4. strict ratio w2	-.02	-.03	-.46**													
5. broad ratio w2	.03	-.05	-.37**	.87**												
6. close sum w2	-.20**	-.07	.28**	.31**	.15**											
7. moderate sum w2	-.00	.02	.66**	-.01	.15**	.52**										
8. peripherea l sum w2	-.01	.03	.91**	-.57**	-.56**	.07	.29**									
9. positive affect w2	.04	-.03	.33**	-.18**	-.17**	.04	.21**	.30**								
10. negative affect w2	-.14**	-.04	-.13*	.02	-.00	-.03	-.10	-.10	-.17**							
11. logil6 w2	.21**	.30**	-.01	-.10	-.09	-.16**	-.03	.00	-.07	.03						
12. logcrp w2	.10	.45**	-.00	-.00	-.01	-.07	.00	-.00	-.02	-.07	.33**					
13. logtnfa w2	.21**	.08	-.02	-.04	-.06	-.09	-.04	.00	-.02	-.07	.25**	.17**				
14. sex (ref= male)	.10	-.18**	.07	-.03	.09	-.05	.19**	-.02	-.01	-.02	.01	-.01	-.04			
15. smoke (ref= no)	.08	-.06	-.11*	.05	.12*	-.07	-.02	-.12*	-.06	.01	.18**	.09	-.01	.09		
16. cohort (ref=1)	-.46**	.00	.07	-.05	-.08	.09	.01	.08	.12*	.01	-.09	-.05	-.13*	-.08	-.08	
17. timelapse	.45**	-.01	-.05	.04	.05	-.07	-.02	-.06	-.10	-.02	.08	.04	.13*	.07	.09	-.96**

Note. *indicates $p < .05$. ** indicates $p < .01$.

Table S6: Conservative ratio and inflammation linear model (MICE)

	IL-6			CRP			TNF-α		
Predictors	*B*	*SE*	*p-value*	*B*	*SE*	*p-value*	*B*	*SE*	*p-value*
(Intercept)	-1.05	0. 73	0.152	2.29	1.14	0.045	1.30	0.32	**<0.001**
Delta strict ratio	-0.01	0.03	0.687	0.02	0.05	0.739	-0.00	0.01	0.862
Age	0.02	0.00	**0.001**	0.00	0.01	0.597	0.01	0.00	**0.004**
Sex	0.03	0.06	0.682	0.09	0.10	0.355	-0.03	0.03	0.236
BMI	0.04	0.01	**<0.001**	0.07	0.01	**<0.001**	0.00	0.00	0.171
Smoke (0=no, 1=yes)	0.45	0.12	**0.001**	0.28	0.19	0.144	-0.05	0.05	0.30
Cohort	-0.22	0.24	0.367	0.20	0.38	0.603	-0.07	0.11	0.490
Timelapse	-0.03	0.04	0.438	0.02	0.06	0.778	0.01	0.02	0.740
SNI total	0.00	0.00	0.279	-0.01	0.01	0.343	-0.00	0.00	0.752
Log IL-6 (pg/mL)	0.16	0.04	**<0.001**						
Log CRP (mg/L)				0.59	0.05	**<0.001**			
Log TNF-α (pg/mL)							0.20	0.03	**<0.001**
Observations	419			419			419		
R^2	0.223			0.425			0.147		

Note. Model estimates from imputed missing data for 69 individuals missing inflammatory data, BMI, and PANAS after 15 multiple imputation iterations.

Table S7: Change in relationship types

	Relationship type W1 (N=350)	Relationship type W2 (N=350)	Change (W2-W1) (N=350)
Children			
Mean (SD)	1.39 (1.25)	1.43 (1.26)	0.0371 (0.816)
Median [Min, Max]	2.00 [0, 6.00]	2.00 [0, 6.00]	0 [-2.00, 4.00]
Parents/in-laws			
Mean (SD)	1.57 (1.13)	0.683 (0.895)	-0.883 (1.09)
Median [Min, Max]	1.00 [0, 4.00]	0 [0, 4.00]	-1.00 [-4.00, 3.00]
Relatives			
Mean (SD)	2.14 (2.10)	2.07 (2.05)	-0.0714 (2.07)
Median [Min, Max]	2.00 [0, 7.00]	1.50 [0, 7.00]	0 [-7.00, 7.00]
Close friends			
Mean (SD)	2.96 (1.98)	2.57 (1.85)	-0.389 (2.07)
Median [Min, Max]	3.00 [0, 7.00]	2.00 [0, 7.00]	0 [-7.00, 7.00]
Churchmates			
Mean (SD)	2.03 (2.77)	1.61 (2.44)	-0.420 (2.27)
Median [Min, Max]	0 [0, 7.00]	0 [0, 7.00]	0 [-7.00, 7.00]
Neighbors			
Mean (SD)	2.23 (2.01)	2.09 (1.65)	-0.140 (1.79)
Median [Min, Max]	2.00 [0, 7.00]	2.00 [0, 6.00]	0 [-6.00, 6.00]

Note. The other SNI groups (students, coworkers, volunteers, other) were not included because there was not enough variation in change.

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