



Social role participation and inflammation: A meta-analytic examination of the shape of association and variability with age

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ABSTRACT

Social role participation is theorized to relate to physical health through systemic inflammation. We conducted a meta-analysis quantifying the association between social role participation and systemic inflammatory markers. Specifically, we synthesized studies assessing *social integration* (the degree of participation in a variety of social relationships) and *social isolation* (having few to no social ties), two indicators of social role participation. We further evaluated whether the association between social participation and inflammation followed graded or threshold patterns and whether its magnitude varied across adult development. A systematic search identified 50 articles, contributing 77 effect sizes. Meta-analyses were conducted using random effects models with robust variance estimation and controlled for study quality and effect size conversion confidence. Results indicated that greater social role participation was associated with lower inflammation (age-unadjusted $r = -.054$, $p < .001$; age-adjusted $r = -.037$, $p < .001$). Moreover, we found evidence for a graded pattern, such that compared to the most isolated group, increasingly integrated groups showed progressively stronger inverse effect sizes with inflammation ($b = -.04$, $p = .017$). We found weak evidence that the magnitude of the social role participation-inflammation link was moderated by age. In a subset of the analyses, this link was stronger for older (vs. younger) samples. However, this only emerged in the model that also adjusted for other study characteristics (e.g., inflammatory marker, study design). This review is the first to synthesize evidence across social integration and isolation, showing that greater social role participation is associated with progressively lower levels of inflammation.

1. Overview

Social role participation is consistently associated with physical health outcomes. For example, individuals who engage in more social roles – such as being a spouse, friend, or neighbor – exhibit lower risk for chronic diseases, including cardiovascular disease, Type 2 diabetes, and some forms of cancer (Christiansen et al., 2021; Everson-Rose and Lewis, 2005; Orlewska et al., 2018). Some of the most rigorous observational studies on this phenomenon assessed social role participation at baseline and then followed participants over time to document the onset or progression of diseases (e.g., Eng et al., 2002; Kreibig et al., 2014; Rutledge et al., 2008), providing evidence that social role participation precedes subsequent disease outcomes. Prompted by these findings, conceptual models have proposed various underlying mechanisms connecting social roles to health (Chin and

Cohen, 2020; Cohen, 1988), with several highlighting systemic inflammation as a biological pathway given its sensitivity to social factors (Hänsel et al., 2010; Uchino, 2006) and involvement in a wide set of health problems (Furman et al., 2019). Social roles are linked to health behaviors such as exercise, diet and sleep, and can influence physiological responses to stress, all of which may contribute to levels of systemic inflammation (Kiecolt-Glaser et al., 2010). Over the past three decades, numerous studies have examined whether holding more social roles would be associated with lower levels of systemic inflammation (Kiecolt-Glaser et al., 2010; Smith et al., 2020; Uchino et al., 2018). Yet, despite this growing body of work, the overall magnitude of the association remains unclear, and questions of theoretical and clinical relevance, including its shape and developmental variability, have not been systematically evaluated. Accordingly, this meta-analysis aimed to synthesize evidence on the link between number of

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social roles and systemic inflammation, characterize whether the association is graded or threshold-based, and evaluate whether its strength varied across adult development.

1.1. Social role participation

Humans naturally seek social connections, which can provide a greater sense of purpose in life (Pinquart, 2001) and enhance life satisfaction and emotional well-being (Huxhold et al., 2013; Sandstrom and Dunn, 2014). One way to quantify social connectedness is through social roles – the distinct types of relationships an individual regularly participates in, such as being a spouse, parent, and friend (Cohen, 1988). Conceptually, the number of roles one participates in reflects an objective aspect of relationships, indicating the extent to which one is embedded in a diverse social network. Role participation typically refers to having regular contact with others or consistent involvement in group activities (e.g., volunteer work), but the frequency regarded as “regular” varies by relationship type. For example, weekly contact is often considered regular for close ties, such as family members or friends (Elliot et al., 2018; Häfner et al., 2011; Jiang et al., 2022), whereas monthly contact is more common for peripheral roles, such as community engagement or volunteer activities (Elliot et al., 2018; Ford et al., 2019; Gleit et al., 2012). Nonetheless, some studies use a consistent timeframe across all relationship types (Shankar et al., 2011), or assess role participation based on the number of relationships individuals perceived themselves to hold, regardless of contact frequency (Loucks et al., 2006; Loucks et al., 2005).

Among studies examining the link between social role participation and inflammation, the most common assessment of social roles captures regular participation in at least four different social relationships: marital status, contact with children or close relatives/friends, church involvement, and other community group membership, typically over the past two-weeks to a month (Berkman and Syme, 1979). Another common social network index captures participation in up to 12 unique roles, including additional relationships like neighbors, classmates, or coworkers, over the past two weeks (Cohen, 1988). These measures typically yield sum scores that reflect the total number of social roles across various social relationship types. Furthermore, some measures, such as the Interview Schedule for Social Interaction, assess both objective social role participation and subjective appraisals of social relationships, including perceived social support and relationship quality (Henderson et al., 1980). This distinction reflects a broader categorization in the literature between objective aspects of social relationships (e.g., number of social roles, contact frequency, network size) and subjective (e.g., emotional support, relationship satisfaction, and conflict) aspects of social relationships (Holt-Lunstad, 2018). One conceptual model for understanding how these dimensions influence health highlights the distinction between main effects and stress buffering mechanisms (Cohen and Wills, 1985). Main effects models propose that relationships benefit health regardless of stress exposure, whereas buffering models propose that social relationships are health protective by attenuating the effects of stress. Objective aspects of social relationships, like social role participation, may exert main effects directly through enhancing positive psychological states or by providing information and motivation to promote healthy behaviors (Cohen and Wills, 1985). Alternatively, subjective aspects of social relationships, like social support, may influence health when employed as a coping strategy during stressors (Cohen and Wills, 1985). Guided by a main effects framework, the present review aimed to isolate the inflammatory correlates of objective social role participation by synthesizing only studies that included at least one objective measure of social roles, excluding studies that focus only on subjective aspects (e.g., Friedman et al., 2005; Vollmer-Conna et al., 2015).

1.2. Systemic inflammation

Inflammation plays a critical role in responding to microbial invasion and tissue damage. When immune cells recognize these threats, they release inflammatory cytokines, such as interleukin-6 (IL-6), tumor

necrosis factor alpha (TNF- α), and interleukin-1 beta (IL-1 β). These cytokines have various functions, including helping recruit immune cells to the site, increasing vascular permeability (Turner et al., 2014), and stimulating the release of acute-phase proteins, such as C-reactive protein (CRP) and fibrinogen (Furman et al., 2019; Netea et al., 2019). Acute-phase proteins further help tag pathogens for phagocytosis and support clot formation to aid in tissue repair (Mantovani and Garlanda, 2023). Under typical conditions, this inflammatory response subsides once the threat is resolved (Kotas and Medzhitov, 2015). However, sustained levels of low-grade inflammation can arise if the stimulus has not been resolved or immune regulation is impaired (Furman et al., 2019; Miller et al., 2011; Nathan and Ding, 2010). Additionally, chronic low-grade inflammation can also occur in the absence of acute immune threats and may be influenced by various ongoing biological and psychological processes, including aging, genetic variation, adiposity, and chronic psychological stress (Furman et al., 2019; Ligthart et al., 2018; Papanicolaou, 1998; Reed, 2019). Among studies examining social role participation, systemic inflammation is commonly assessed by quantifying concentrations of inflammatory markers, such as the cytokines and acute phase proteins mentioned above in venous or capillary blood (Justice et al., 2018). Using these measurements, systemic inflammation has been linked to downstream clinical diseases. For example, above and beyond traditional markers of disease (e.g., cholesterol, blood pressure), elevated levels of IL-6 and CRP have been prospectively associated with disease onset or progression, including increased risk of cardiovascular disease, Type 2 diabetes, and various types of cancer (Fernandez et al., 2020; Dos Santos Silva et al., 2010; Thorand et al., 2007), highlighting their predictive utility. In addition, systemic inflammation may contribute to the development of psychiatric disorders (Yuan et al., 2019) and higher levels of CRP predict increases in depressive symptoms over time (Matthews et al., 2010; van den Biggelaar et al., 2007).

1.3. Social role participation and inflammation

Prompted by evidence that linked the number of social roles to clinical disease outcomes (Berkman, 1995) and evidence that linked systemic inflammation to clinical disease (Pradhan, 2001; Smith et al., 1997), conceptual models emerged to explain how social role participation may relate to systemic inflammation (e.g., Berkman et al., 2000; Chin and Cohen, 2020; Fagundes et al., 2011). These models postulate a combination of psychological, biological, and behavioral pathways through which social roles may be associated with inflammation. Psychologically, social role participation contributes to a stronger sense of purpose and enhances overall emotional well-being (Thoits, 1983). Holding a variety of social roles provides sense of purpose by creating more opportunities to help others and feel useful (Lee and Martin, 2023; Pinquart, 2002). These roles also embed individuals in networks that may increase likelihood of perceiving or receiving higher emotional and instrumental support, which can promote positive affect and mitigate the negative impacts of stress (Pinquart and Sörensen, 2000; Rafnsson et al., 2015). In response to stress, sympathetic nervous system (SNS) and Hypothalamic Pituitary Adrenal (HPA) axis activation can influence inflammatory levels. Mechanistically, glucocorticoids released by the HPA axis, commonly cortisol, down-regulate pro-inflammatory signaling, but when released for prolonged periods can result in down-regulation of immune cell glucocorticoid receptor sensitivity. This decreases the sensitivity of immune cells to the anti-inflammatory properties of glucocorticoids, possibly contributing to heightened systemic inflammation (Miller et al., 2002). The SNS can stimulate increases in pro-inflammatory cytokines through the release of catecholamines through sympathetic nerve terminals, which bind directly to receptors on immune cells increasing proinflammatory cytokine production (Elenkov et al., 2000). Additionally, circulating catecholamines can mobilize immune cells from lymphoid tissues, increasing pro-inflammatory immune cells in circulation. Psychological resources may have positive impacts on biological systems by reducing activation of these stress pathways or promoting faster physiological recovery following

a stressor. For example, a greater sense of purpose and more positive affect have been associated with lower basal activation of the SNS and reduced HPA axis activity (Lindfors and Lundberg, 2002; Mezick et al., 2010; Steptoe and Wardle, 2005). Furthermore, emotional and instrumental support derived from social roles can buffer these same systems during acute stress, attenuating stress reactivity and promoting faster physiological recovery (Heinrichs et al., 2003; Kamarck et al., 1990). Over time, both lower basal activation and dampened stress reactivity are hypothesized to correspond with reduced downstream proinflammatory signaling (Miller et al., 2002), thereby contributing to lower levels of systemic inflammation (Lockwood et al., 2016; Marsland et al., 2017; Steptoe et al., 2001). Behaviorally, social role engagement can encourage health-promoting behaviors, such as participating in group exercise, maintaining consistent sleep schedules, or improving medical adherence (Molloy et al., 2008; Tay et al., 2013; Yu et al., 2018) through social modeling or accountability within specific roles. In turn, each one of these improved health-promoting behaviors has been linked to lower levels of systemic inflammation (Dzierzewski et al., 2020; Kronish et al., 2010; Loprini et al., 2013). Over the past three decades, an accumulating body of studies have tested whether the number of social roles is associated with lower levels of systemic inflammation markers. Accordingly, the current meta-analysis synthesizes this literature and leverages the expanded heterogeneity in social role participation and age to examine two theoretically important but unresolved questions.

Unresolved Question One: Graded and Threshold Models of the Social Role–Inflammation Link. First, does the number of social roles relate to systemic inflammation in a *graded* manner, where each additional social role confers incremental reduction in inflammation, or a *threshold* manner, where only social isolation is linked to elevated levels of inflammation, but further social integration does not provide additional benefits? Clarifying the *shape* of this association can refine theories, as different patterns may reflect variations in mechanisms by which social role participation relates to systemic inflammation (Chin and Cohen, 2020). Two conceptual models provide distinct predictions (Chin and Cohen, 2020): the social isolation model (Holt-Lunstad and Steptoe, 2022) and the social integration model (Brissette et al., 2000). Social role participation is a continuum reflecting the objective number of social roles an individual actively participates in. Social isolation represents the lower end of this continuum and is defined as having too few social relationships, often zero or one social ties, or infrequent contact, based on a predefined cutoff indicating limited social participation. By contrast, social integration refers to holding more social roles on a continuous scale. The isolation model posits that being socially isolated elevates negative psychological states, such as increased loneliness and depressive symptoms, which in turn may be associated with higher levels of systemic inflammation over time (Cacioppo et al., 2011;

Holt-Lunstad and Steptoe, 2022). If true, we would expect a threshold effect (Fig. 1A) such that those who are categorized as being isolated have higher levels of inflammation, but gaining additional social roles beyond isolation provides minimal benefit, as the primary mechanism, psychological distress, has been eliminated. By contrast, the social integration model suggests that the continuous number of social roles is associated with reduced inflammation because holding more roles fosters emotional benefits, such as sense of purpose and enhanced positive affect (Chin and Cohen, 2020). If true, we would expect a graded effect (Fig. 1B) where each additional social role contributes to accumulating emotional benefits, and thus lower levels of inflammation (Chin and Cohen, 2020). Notably, these pathways may not be mutually exclusive. As positive and negative affect can co-occur and independently relate with health outcomes (Pressman and Cohen, 2005), it is also possible to observe both threshold and graded patterns (Fig. 1C).

Empirically testing whether social role participation relates to inflammation in a graded or threshold manner requires analyzing social roles continuously and categorically within the same sample. Yet, few studies have done so (Berkman and Syme, 1979; Brummett et al., 2001; Yang et al., 2013) with most relying on a single analytic approach. One prior narrative review addressed this gap in the context of cardiovascular disease, finding that the most common pattern of results for men was a graded effect, whereas women demonstrated a combination of graded and threshold effects (Chin and Cohen, 2020). While this review shed light on the shape of the link between social roles and health, it relied on a visual approach to differentiate graded vs. threshold effects, lacking statistical inference necessary for definitive conclusions. Therefore, the current meta-analysis synthesized within-study comparisons across adjacent levels of integration, crucially allowing formal statistical tests of whether this association manifests as graded, threshold-based, or both.

Unresolved Question Two: Magnitude of the Social Role–Inflammation Link Across Age. Second, does the magnitude of the link between social role participation and inflammation vary with age? Social network size changes throughout the adult lifespan. During young adulthood, networks often expand as individuals take on new roles. For example, entering the workforce can introduce new coworkers and friends, while long-term romantic partnerships can bring in in-laws and partners' friends (Wrzus et al., 2013). After young adulthood, however, aging is accompanied by a steady decline in social network size (Van Tilburg, 1995; Wrzus et al., 2013), with some of these changes driven by normative life events, such as parenthood, retirement, or loss of a spouse or friends (Heckhausen, 1999).

How then does the link between social role participation and inflammation vary with age, in light of these normative developmental shifts in social network size? One possibility is that the association is stronger in older adulthood, when social networks are smaller and remaining roles

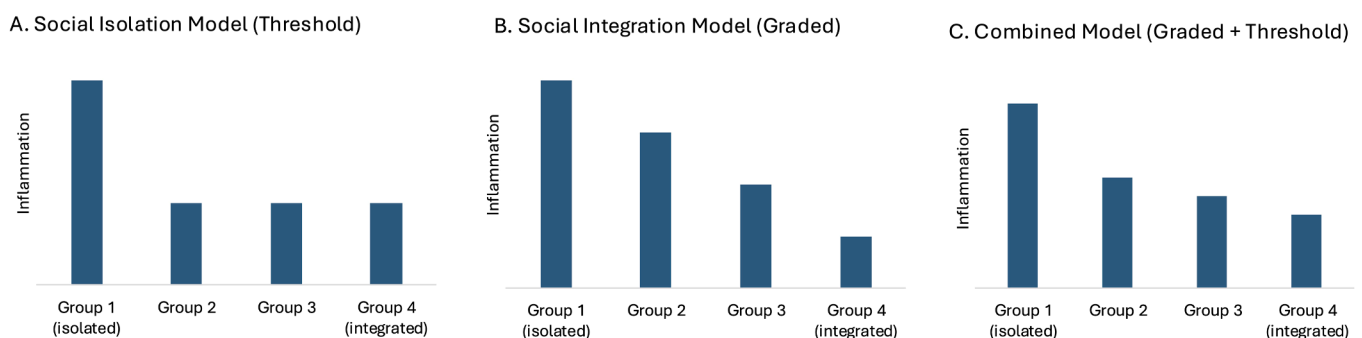


Fig. 1. Hypothesized Shapes of the Link Between Social Role Participation and Inflammation. This figure is adapted from Chin and Cohen, (2020), illustrating the predicted level of inflammation across four levels of social role participation, from most isolated (Group 1) to most integrated (Group 4). Panel A depicts the threshold pattern, such that the isolated group has higher levels of inflammation, but gaining additional social roles beyond isolation provides minimal benefit. Panel B depicts the social integration model, or graded pattern, such that each additional level of social integration is associated with incremental reduction in inflammation. Panel C depicts a combination of both patterns such that the isolated group has a higher level of inflammation compared to the other groups, but there are little benefits from continuing to hold additional social roles.

may become more critical for well-being and stress regulation (English and Carstensen, 2014; Lim-Soh, 2022). Consistent with the socioemotional selectivity theory, older adults may be more emotionally invested in their core relationships and be more sensitive to their presence or absence (Carstensen, 1992). Supporting this hypothesis, prospective empirical studies found that social role participation is more strongly associated with lower systemic inflammation in older adults compared to younger adults (Elliot et al., 2018; Yang et al., 2016).

Alternatively, the association may be stronger earlier in adulthood. Young and midlife adults maintain a diverse set of social relationships in order to seek information and novel experiences (Carstensen et al., 2003). Therefore, although younger adults may not prioritize holding close and emotionally rewarding relationships like older adults, having a diverse set of social roles is still essential for meeting their knowledge-seeking goals. One study supports this idea, finding that social isolation measured in midlife (largely between the ages of 50–65) was a stronger predictor of mortality compared to when it was measured in older adulthood (aged 65+) (Holt-Lunstad et al., 2015). One reason older adults may be less affected by isolation is that it may be culturally normative in later life whereas social isolation in early adulthood is not. Alternatively, these findings could be driven by survivor effects such that the oldest adults included in this meta-analysis may have been a particularly resilient group with better health than some of the younger samples.

Finally, it is also possible that age-related variations depend on whether the social role participation-inflammation link follows the integration model or the isolation model. For example, social integration may be more beneficial for older adults as they may more strongly rely on their social roles as sources of purpose in life and as a buffer of heightened uncontrollable stressors later in adulthood (e.g., death of close friends). By contrast, the link between social isolation and inflammation may be more stable across age because social isolation is a chronic stressor for all age groups (Eisenberger et al., 2017; Smith et al., 2020). Because few studies have examined age as a moderator of the association between number of social roles and inflammation, this question remains unresolved. Leveraging that both integration and isolation have been studied across diverse age groups, the current meta-analysis aimed to test whether the strength of the social role participation-inflammation association varies by age for each.

1.4. The current meta-analysis

To our knowledge, there are two prior meta-analyses synthesizing the link between social role participation and inflammation. Uchino and colleagues (2018) synthesized 22 studies, focusing on social integration, and Smith and colleagues (2020) synthesized 16 studies, focusing on social isolation. These meta-analyses separately linked more social integration and less isolation to lower levels of systemic inflammation, observing small effect sizes (Smith et al., 2020; Uchino et al., 2018). However, these meta-analyses examined social integration and isolation separately, precluding empirical tests of whether the link between social role participation and inflammation follows a graded or threshold pattern. Furthermore, to maintain independence of observations, these meta-analyses either averaged multiple relevant effect sizes across various inflammatory markers within each study (Uchino et al., 2018) or conducted separate meta-analyses by inflammatory marker and focused only on commonly assessed biomarkers, namely IL-6, CRP, and fibrinogen (Smith et al., 2020). Both approaches have merit in ensuring homogeneity and promoting comparability across studies; however, these approaches can reduce the pool of relevant effect sizes, limiting power for moderator analyses (e.g., by age), and preclude within-study comparisons across inflammatory biomarkers and levels of social role participation. Yet, such within-study contrasts are particularly valuable for testing graded vs. threshold effects, as they hold constant many sources of between-study heterogeneity (Moeyaert et al., 2017). The current meta-analysis utilizes robust variance estimation to incorporate a larger pool of effect sizes across multiple inflammatory markers, increasing power and generalizability of

findings while accounting for within-study dependencies (Pustejovsky and Tipton, 2022). Our goals were to: 1) synthesize the overall association between social role participation and systemic inflammation, bringing together peer-reviewed studies of social integration and isolation; 2) determine whether this association follows a graded pattern (supporting integration model), a threshold effect (supporting the social isolation model), or a combination of both; and 3) examine whether the strength of this association varied with age.

Consistent with prior meta-analyses on this topic (Smith et al., 2020; Uchino et al., 2018), we also examined whether methodological characteristics moderated the overall effect size. Specifically, we evaluated the type of social measure, year at which social role participation was assessed, type of inflammatory marker, study design, and the proportion of male participants. These moderators were selected a priori based on prior empirical evidence demonstrating that factors such as sex and inflammatory marker type can yield different effects (Loucks et al., 2006; Loucks et al., 2005) and theoretical predictions that methodological differences in the type of and timing of social role assessment (Siette et al., 2021) and in study design (Barbosa-Leiker et al., 2014) may contribute to variability in associations between social factors and inflammation. Moreover, socioeconomic status (SES), mental health, and physical health may influence the link between social role participation and inflammation in at least two ways. First, because each is associated with both social role participation (Griffith et al., 2017; Smith and Victor, 2019; Van Groenou and Van Tilburg, 2003) and inflammation (Liu et al., 2023; Mac Giollabhui et al., 2021; Muscatell et al., 2020), they may confound or partially account for observed associations between the two. Second, these factors may shape the magnitude of the association itself. For example, on one hand, social role participation may confer greater benefits for individuals with lower SES because this population has higher inflammation (Muscatell et al., 2020) creating greater variability in inflammation levels that could be associated with social participation. Conversely, emerging conceptual models suggest that individuals facing social disadvantages, such as lower SES, may receive diminished health returns from otherwise protective factors (Assari, 2018). Structural barriers and chronic adversity may constrain the extent to which resources are translated into health benefits (Assari, 2018; Assari and Zare, 2024), thus potentially attenuating the association between social role participation and inflammation among individuals with lower SES. Physical and mental health may similarly shape the link between social role participation and inflammation: individuals with poorer physical or mental health often exhibit greater inflammatory burden, providing greater opportunity for social participation to buffer disease- or distress-related physiological processes (Furman et al., 2019). Alternatively, when inflammation becomes increasingly driven by underlying disease pathology (rather than as an early sign of future disease), it may be less amenable to psychosocial influences, thereby attenuating the link between social role participation and inflammation. Therefore, supplementary analyses examined whether the link between social role participation and inflammation remained after accounting for sample-level SES, mental health, and physical health, and whether these factors also moderated the association.

2. Method

Pre-registration of the study was initiated on May 15, 2024 on the Open Science Framework (https://osf.io/5q4ce/?view_only=714cf6bf1720416c8472c948f4d06267). The following sections follow the guidelines of the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement (Page et al., 2021).

2.1. Study retrieval strategy

Electronic searches were performed in two databases (PubMed and PsycINFO) using search terms to capture studies that combined measures of social role participation and inflammation. The results were filtered to include peer reviewed studies reported in the English language and in

human subjects. Search terms for social role participation were: “social* network*”, “social* isolation*”, “social* integration*”, “social ties”, “social engagement”, “social participation”, “contact frequency”, “social contact”, “social connection”, “social roles”; search terms for inflammation were: “inflammation”, “interleukin”, “IL-6”, “c-reactive protein”, “CRP”, “TNF- α ”, “fibrinogen”, “cytokines”, “e-selectin”, “IL-8”, “IL-10”, “interferon gamma”, “allostatic load”, “SuPAR”.

2.2. Inclusion and exclusion

The search strategy was completed on April 15, 2026. The titles and abstracts of the identified articles were screened based on our inclusion criteria: a) peer reviewed; b) empirical in nature; c) published in the English language; d) examined human subjects; e) sample was not immunocompromised; f) included at least one measure of social role participation and one marker of inflammation; g) sample was above the age 18 and did not include children. Only peer reviewed studies were included to ensure sufficient methodological detail and consistent and reliable reporting of effect sizes. Review articles (i.e., narrative, systematic, meta-analyses, commentaries, and protocol descriptions) were excluded. Studies that exclusively examined immunocompromised samples (i.e., Human Immunodeficiency Virus infection) were excluded (Blanco et al., 2025). Studies that focused on subjective momentary social experiences (Hasselmo et al., 2018; West et al., 2026) or relationship quality (e.g., Friedman et al., 2005; Vollmer-Conna et al., 2015) instead of social role participation were excluded. Studies that reported composites of social relationships that included an objective assessment of social network structure and some other component related to relationship quality (e.g., Gustafsson et al., 2012; McClendon et al., 2021; Mukamal et al., 2006) were included. Due to the focus on the adult lifespan, studies that examined social role participation in childhood (age < 18) were not included (Danese et al., 2009; Lacey et al., 2014; Matthews et al., 2024; Yang et al., 2017).

All articles with abstracts that met inclusion criteria or that lacked sufficient detail to determine eligibility were retained for full text review. Reasons for exclusion at the full-text stage are summarized in Supplemental Figure S1. For articles that met inclusion criteria but did not report necessary statistics, we contacted the corresponding authors to request the statistics. A reminder was sent after four weeks, and studies were excluded if no response was received. Data were extracted or obtained from authors for a total of 50 articles. Of these, 35 contributed to the integration analyses, 30 to the isolation analyses, and 19 provided sufficient data to enable the direct comparison of the graded and threshold models.

2.3. Data extraction

Study characteristics. Various sample and methodological characteristics were extracted by the first author (ARH). All extracted variables were then reviewed by a team of four coders (BK, HZ, JD, JT) to ensure accuracy of the data. Specifically, we extracted the measure of social role participation, inflammatory marker, sample mean age and age range at the time of social measure collection, study design (cross-sectional vs. prospective), sample's proportion of male participants, and year of social measure completion. Studies that reported continuous estimates for the association between the social measure and inflammation were categorized as “integration” effects, and studies that reported a categorical comparison between a socially integrated group and a socially isolated group were categorized as “isolation” effects.

Study quality coding. To account for variation in study quality across the articles, a measure adapted from the NHLBI checklist for cross-sectional and observational studies (NHLBI, 2018) that has been used in prior meta-analyses (Chiang et al., 2022) captured three domains of study quality: social relationship measurement, inflammation measurement, and statistical approach (see Supplemental Materials for the full 11-item scale). We calculated a weighted sum so that each scale domain was weighted equally and higher values correspond with better study quality

($M = 12.03$, $SD = 2.32$, range = 6.11–15.89). The primary reviewer (ARH) assessed the quality of each of the studies, and the same secondary coders ensured accuracy.

Effect sizes. We extracted both unadjusted and age-adjusted effect sizes. In health psychology research, statistical models often adjust for the effect of chronological age because of its positive association with systemic inflammation and the likelihood of morbidity (Lassale et al., 2019). However, social network structure changes with age (Sander et al., 2017; Wrzus et al., 2013) meaning it also has shared variance with age. Therefore, the practice of partialling out the variance in the health outcomes explained by age could also dampen the observed effect of social role participation (Wilson, 2024). For age-adjusted effects, we prioritized the least adjusted models that controlled for chronological age. Birth-cohort studies were grouped with age-adjusted effect sizes because the study design removes any variance that could be explained by chronological age. For a small number of studies ($k = 2$; 4% of the total number of studies), partial correlations were grouped with the unadjusted effects because the authors adjusted for other demographics (e.g., sex, race) but not age. Most studies included multiple relevant effect sizes due to the inclusion of multiple social role participation measures and/or multiple inflammatory markers. To minimize variability stemming from differences in the number or type of roles assessed, we prioritized extracting effect sizes from the most commonly used measures—the Berkman and Syme Social Network Index and Cohen Social Network Index (e.g., Berkman and Syme, 1979; Cohen et al., 1997). For inflammation outcomes, we extracted all relevant effect sizes and accounted for within-study dependencies analytically (see data synthesis 3.2).

Extraction. For each unique relationship between social integration/isolation and inflammation, we extracted unadjusted and, when available, age-adjusted statistics. The target effect sizes were Pearson's r for unadjusted links and Partial r for age-adjusted links. We prioritized statistics that would receive the best conversion confidence score (see Supplemental Table S1), meaning the statistics that would require the fewest transformations to achieve the target effect sizes (Chiang et al., 2022). The conversion confidence score was computed such that higher values indicate a greater number of conversion steps, thus yielding lower confidence. The final scores were reversed for analyses so that lower scores reflect less confidence and higher scores reflect more confidence ($M = 3.42$, $SD = 1.21$, range = 1–5).

Conversion. Using the R package *michaela* (Lam and Chiang, 2020), all extracted statistics were converted to their Pearson's or Partial r and then into Fisher's Z . All effect sizes were coded such that more negative values indicate that greater social integration (continuously or vs. social isolation) was associated with lower levels of inflammation. A summary of the types of effect sizes and their corresponding transformations to estimate Fisher's Z s can be found in Supplemental Methods. In the isolation literature, it was common for studies to report comparisons between two or more groups (e.g., quartiles: most isolated, somewhat isolated, integrated, most integrated). For the overall synthesis, we selected the comparison representing the largest difference in social integration between groups (i.e., most isolated vs. most integrated) for analysis. However, for analyses testing graded vs. threshold patterns, we retained all relevant group comparisons. As only a few studies tested both the integration model (e.g., continuous) and isolation model (e.g., categorical comparisons), we contacted all authors to request the alternate form of analysis ($k = 45$; 90%). We also contacted authors ($k = 31$; 62%) if they did not report both age-adjusted and unadjusted estimates. Of the authors contacted, $k = 18$ responded and provided all requested estimates for their studies.

3. Data analysis

3.1. Data preparation

Because several studies drew from the same publicly available cohort data (e.g., Midlife in the United States Study, the Health and Retirement Studies), some effect sizes were based on the same samples of participants.

When studies reported unique associations from the sample (e.g., different inflammation markers or time points), effect sizes were retained and nested within samples in analysis. When studies reported duplicated effect sizes (e.g., two studies reported the same association between social roles and an inflammatory marker at the same time point), we retained the larger sample unless the smaller sample provided more precise statistics (e.g., Pearson's r vs. a t -statistics). Outlier effect sizes greater than 3 SDs from the mean ($es = 3$, 2.1% of the effect sizes reported; Z 's = .50 – 1.07) were removed.

3.2. Data synthesis

Analyses were conducted using R (version 4.4.0) using the *robumeta* package (version 2.1) (Fisher and Tipton, 2023). As 55% of studies included more than one relevant effect size (due to assessing multiple inflammatory markers and/or associations at multiple different time points), we conducted random effects models with robust variance estimation (RVE) to account for correlated effect sizes within studies (Pustejovsky and Tipton, 2022). Furthermore, we also applied small-sample corrections to control for Type 1 error rates, such that unreliable results (i.e., the Satterthwaite degrees of freedom [DF] below 4) were not interpreted (Tipton and Pustejovsky, 2015).

We conducted the data synthesis in two ways: (1) by collapsing across the integration (i.e., continuous) and isolation (i.e., categorical) effect sizes to compute overall associations between social role participation and inflammation, with separate estimates for unadjusted and age-adjusted effect sizes; and (2) by synthesizing integration and isolation effect sizes separately, again distinguishing between unadjusted and age-adjusted estimates. The latter approach resulted in four syntheses: integration unadjusted, integration age-adjusted, isolation unadjusted, isolation age-adjusted. All models controlled for study quality and conversion confidence scores. Synthesized Fisher's Z 's were transformed back into Pearson correlations (r) for ease of interpretation, following conventions (Card, 2015).

In addition, publication bias was assessed using the precision-effect test (PET) and the precision-effect estimate with standard error (PEESE). The PET test models a linear relationship between the standard error and the effect size and the PEESE test models a quadratic association. If the PET estimate was non-significant, we interpreted it as final; if significant, we proceeded to PEESE, whose estimate was then considered final (Stanley and Doucouliagos, 2014).

3.3. Graded vs. threshold patterns

We next tested whether the link between social role participation and systemic inflammation followed a graded pattern, threshold pattern, or both. We first evaluated evidence for a graded pattern. Specifically, we computed standardized mean differences between the most socially isolated group (Group 1) and each increasingly integrated group: the next-lowest integration (Group 2), the intermediate level (Group 3), and the highest level of integration (Group 4). In most studies, authors categorized these groups using scale-defined cut-points, but the classifications nonetheless largely mapped onto four ordered categories of social integration. The exception was two studies (Cudjoe et al., 2022; Rizzuto et al., 2017) that stratified participants into three ordered categories of social integration; these studies were retained because they still permitted comparisons across ordered levels of social integration. Differences were expressed in Hedge's g , which characterizes the standardized mean differences while accounting for unequal group sizes (Hedges and Olkin, 2014). We then conducted a meta-regression in which the magnitude of these contrasts was predicted from a linear term representing the comparison between the most isolated group and each increasing integration level (the contrast of Group 1 vs. 2 = 1, Group 1 vs. 3 = 2, and Group 1 vs. 4 = 3). A significant linear term would indicate a graded pattern, as it would suggest incremental decreases in inflammation (i.e., increasingly negative Hedge's g) as we compare each increasingly integrated group to the isolated group.

As both graded and threshold patterns can exist (depicted in Fig. 1), we then evaluated evidence for a threshold pattern. To do so, we recomputed Hedge's g to reflect differences in average inflammation between each pair of adjacent groups: Group 1 vs. 2, Group 2 vs. 3, and Group 3 vs. 4. We then conducted a meta-regression testing whether the magnitude of these contrasts differed significantly from one another. A significant difference among the Hedge's g estimates (e.g., a more negative Hedge's g for Group 1 vs. 2 compared to Group 2 vs. 3) would indicate a threshold pattern, as it would suggest that one step between consecutive integration levels is linked with a steeper reduction in inflammation than the others. Again, all models controlled for study quality, but not conversion confidence, as Hedge's g was uniformly computed from raw mean group differences.

3.4. Moderator analyses

The next set of analyses tested whether effect sizes varied by sample mean age and other extracted methodological characteristics. Specifically, we conducted a series of meta-regressions testing whether sample mean age moderated the overall associations between social role participation and inflammation and then repeated these analyses separately for continuous (integration) and categorical (isolation) effect sizes. In addition to study quality and conversion confidence, these models also adjusted for sample age range to account for between-study variations in how well the mean age represented the overall sample.

We further tested other extracted methodological characteristics as moderators, including social measure type (coded as: Berkman Syme [referent] vs. other measure), year at which social role participation was assessed, inflammatory marker type (CRP, fibrinogen, IL-6, and other [Albumin, MMP9, sE-selectin, sICAM-1, suPAR, and TNF- α]), study design (cross-sectional [referent] vs. prospective), and sex composition (proportion of male participants in the sample). We included the year at which social role participation was assessed to account for macro-level trends, particularly the growing prevalence of virtual social interactions over time. Because most measures of social role participation do not explicitly capture virtual social engagement, and these roles were more salient in recent years, it is possible that this macro-level shift may result in a modest attenuation of effects in more recent studies.

Moderators were first entered in individual models and then tested simultaneously to determine which moderators accounted for variability in effect sizes above and beyond each other. Multi-categorical moderators were deviation effect-coded, binary moderators were dummy-coded, and continuous moderators were mean-centered. Significant moderations were followed up by computing predicted effect sizes at each level of categorical moderators and at ± 1 SD from the mean for continuous moderators. Again, all models included study quality and conversion confidence as covariates.

3.5. Subgroup analyses

Finally, we provide descriptive subgroup analyses showing synthesized correlations, across both social integration and social isolation effect sizes, by inflammatory marker in the results Section 4.7. We also present stratification by other study characteristics for the integration and isolation subsamples. Note that numeric differences in effect size magnitude across study characteristics are descriptive only; statistical comparisons are reported in moderator analyses (Results Section 4.4 and 4.5).

3.6. Supplementary analyses

We additionally examined whether socioeconomic status (SES), physical health, and mental health confounded or moderated the link between social role participation and inflammation. The measurements for each of these variables varied across the studies. For instance, across studies, SES was assessed using education, income, wealth, or composite measures. To maximize comparability across studies, we applied a prespecified order of priority based on how frequently SES indicators were reported across

studies. Specifically, we prioritized the proportion of participants without a college degree (or, when unavailable, without a high school degree, 44% and 18% of studies, respectively). If these data were unavailable, we used the proportion of individuals with annual incomes below \$15,000–20,000 (6%). When categorical indicators were unavailable, we calculated an inverse Proportion of Maximum Possible (POMP) score (0–100%) from continuous education years (12%)¹ or continuous SES composites (8%). SES data were unavailable for 12% of studies. Higher values of the resulting SES variable indicated a greater proportion of the sample with socioeconomic disadvantage ($M = 51\%$, range = 4–95%). Similarly, for **mental health**, we prioritized extracting the proportion of the sample reporting a mental health condition (e.g., depression, PTSD, schizophrenia; 24% of studies), followed by the proportion with two or more adverse experiences (6%),² or POMP scores were derived from continuous sample means of mental health symptoms (28%). Mental health data were unavailable for 42% of studies. Higher values of the resulting mental health variable indicated a greater proportion of the sample with, or at risk for, mental health problems ($M = 19\%$, range = 5–37%). Finally, for **physical health**, we prioritized the proportion of the sample with one or more chronic conditions or limiting illnesses (52%; e.g., Cardiovascular disease, Hypertension, Diabetes etc.), followed by the proportion of the sample taking medications (4%; e.g., aspirin, anti-hypertensive), POMP scores were derived from continuous scales that assessed difficulties in daily life due to physical limitations (6%), or the average number of chronic conditions (6%). Physical health data were unavailable for 32% of studies. Higher values in the resulting physical health variable indicated a greater proportion of the sample with physical health problems or limitations ($M = 37\%$, range = 4–100%).

Within the social isolation studies, we also tested whether the threshold used to define social isolation moderated the link between social roles and inflammation. Although many studies classified participants with no social roles as socially isolated, the ranges of the underlying social isolation scales varied across studies, meaning this cutoff represented different proportions of the total scale. To account for this variation, we created a continuous variable representing the percentage of the scale categorized as socially isolated. For instance, on a 5-point scale (0–4), a cutoff of 0 social roles corresponds to the bottom 20% of the scale, whereas on a 10-point scale this same cutoff would correspond to the bottom 10% of the scale.

4. Results

4.1. Study characteristics

As depicted in [Supplemental Figure S1](#), a total of 655 articles were identified (PsycINFO = 209; PubMed = 446). After removing duplicates ($k = 129$), 526 titles and abstracts were screened, and 126 full-text articles were reviewed. Following exclusions detailed in [Supplemental Figure S1](#), the current meta-analysis includes 50 articles. Citations of all articles are provided in [Supplemental References](#). Because the integration, isolation, and graded vs. threshold analyses required different types of statistics, these analytic samples were not mutually exclusive. Individual articles could therefore contribute to more than one analysis when they provided the statistics required for each analysis. In addition, a single article could contribute multiple unique samples (e.g., when separate samples were reported by sex, age, or other demographic characteristics). Across the 50 articles, there were 65 unadjusted and 77 age-adjusted effect sizes based on 38 unadjusted and 38 age-adjusted unique samples, reflecting 535,955 and

510,372 participants, respectively. Of the 50 articles, 35 provided integration statistics, contributing 42 unadjusted and 51 age-adjusted effect sizes based on 24 and 26 unique samples, reflecting 523,530 and 487,504 participants. Of the 50 articles, 30 provided isolation statistics, contributing 51 unadjusted and 37 age-adjusted effect sizes based on 27 and 17 unique samples, reflecting 26,415 and 463,789 participants. Of the 50 articles, 19 provided the statistics needed (see Data Analysis [Section 3.3](#), for details) to conduct the graded vs. threshold pattern analysis, based on 20 unique samples reflecting 72,339 participants. [Table 1](#) presents the overall descriptive statistics of study characteristics, and [Supplemental Table S2](#) details the characteristics of each individual study. The most common inflammatory marker assessed was CRP (54%) followed by IL-6 (22%), and fibrinogen (15%). Most studies used cross-sectional designs (85%). The most common measure of social role participation was the Berkman-Syme Social Network Index (60%; [Berkman and Syme, 1979](#)) followed by Cohen's Social Network Diversity (29%; [Cohen and Wills, 1985](#)).

4.2. Overall effect size

Across both social integration and social isolation effect sizes, greater social role participation was significantly associated with lower inflammation based on both unadjusted ($r = -.054$, $Z = -.054$, $CI_L[-.075, -.034]$, $p < .001$) and age-adjusted effect sizes ($r = -.037$, $Z = -.037$, 95% $CI_L[-.049, -.026]$, $p < .001$). In studies examining social integration, greater social integration was associated with lower levels of inflammation based on both unadjusted ([Fig. 2A](#); $r = -.043$, $Z = -.043$, $CI_L[-.064, -.022]$, $p < .001$) and age-adjusted effect sizes ([Fig. 2B](#); $r = -.042$, $Z = -.042$, 95% $CI_L[-.057, -.027]$, $p < .001$). Similarly, in studies comparing socially isolated vs. integrated individuals, the social integration group exhibited lower levels of inflammation based on both unadjusted ([Fig. 2C](#); $r = -.068$, $Z = -.068$, $CI_L[-.099, -.036]$, $p < .001$) and age-adjusted effect sizes ([Fig. 2D](#); $r = -.036$, $Z = -.036$, 95% $CI_L[-.055, -.016]$, $p = .002$).

We did not observe significant evidence of publication bias; that is, sampling error was not a significant linear predictor of effect sizes in the overall sample (unadjusted: $b = -.80$, 95% $CI [-1.80, .19]$, $p = .107$; age-adjusted: $b = .20$, 95% $CI [-.64, 1.05]$, $p = .621$), within integration studies (unadjusted: $b = -.36$, 95% $CI [-1.41, .69]$, $p = .460$; age-adjusted: $b = .41$, 95% $CI [-.60, 1.42]$, $p = .393$), or within the isolation studies (unadjusted: $b = -.44$, 95% $CI [-1.58, .69]$, $p = .401$; age-adjusted: $b = -.52$, 95% $CI [-1.95, .90]$, $p = .412$). [Supplemental Figure S2](#) provides the funnel plots for visual assessment of publication bias.

4.3. Graded vs. threshold patterns

Next, we tested whether the link between social role participation and systemic inflammation follows a graded pattern, threshold pattern, or both. As depicted in [Fig. 3A](#), there was evidence consistent with a graded pattern. Compared to the most isolated group, increasingly integrated groups showed progressively stronger inverse effect sizes with inflammation ($b = -.04$, 95% $CI [-.070, -.009]$, $p = .017$). Specifically, relative to the most isolated group, the effect size was smallest for the next-lowest integration level ($\hat{g} = -.072$), grew stronger at the intermediate level ($\hat{g} = -.101$), and was largest at the highest level of integration ($\hat{g} = -.124$). This pattern suggests that inflammation decreased incrementally with each additional level of social integration, supporting a graded association.

By contrast, we did not find evidence for the threshold effect of isolation on inflammation. The effect size differences between adjacent groups were not statistically significant (p 's $> .440$). As depicted in [Fig. 3B](#), comparable Hedge's g values were observed between the most isolated and next-lowest integration levels ($\hat{g} = -.072$), the next-lowest and intermediate levels ($\hat{g} = -.030$), and the intermediate and highest integration levels ($\hat{g} = -.030$). That is, each incremental increase in

¹ A sample average of 10 years of education with a range of 6–22 years would have an inverse POMP score of 75% indicating that the sample mean is 75% below the maximum years of education (relative to the observed range).

² Because this indicator more closely represents a risk factor for mental health, we also conducted analyses excluding this measure and results remained the same.

Table 1
Frequency of Effect Sizes by Study Characteristics.

	Integration		Isolation	
	Unadjusted (es=42) Frequency (%) or Mean (SD)	Age-adjusted (es=51) Frequency (%) or Mean (SD)	Unadjusted (es=51) Frequency (%) or Mean (SD)	Age-adjusted (es=37) Frequency (%) or Mean (SD)
Inflammatory marker type				
CRP	27 (64.3%)	27 (52.9%)	25 (49%)	18 (48.7%)
Fibrinogen	4 (9.5%)	7 (13.7%)	11 (21.6%)	6 (16.2%)
IL-6	9 (21.4%)	12 (23.5%)	10 (19.6%)	9 (24.3%)
Other	2 (4.8%)	5 (9.8%)	5 (9.8%)	4 (10.8%)
Study design				
Cross-sectional	38 (90.5%)	34 (66.7%)	47 (92.2%)	33 (89.2%)
Prospective	4 (9.5%)	17 (33.3%)	4 (7.8%)	4 (10.8%)
Social measure type				
Berkman-Syme	19 (45.2%)	29 (56.9%)	30 (58.8%)	30 (81.1%)
Other	23 (54.8%)	22 (43.1%)	21 (41.2%)	7 (18.9%)
Proportion male	.50 (.18)	.48 (.16)	.52 (.38)	.50 (.43)
Study quality	12.51 (2.24)	12.73 (2.35)	11.61 (2.49)	11.10 (1.76)
Conversion confidence	4.95 (.31)	2.73 (.90)	3.12 (1.05)	2.97 (.90)
Sample Mean Age at Social Measurement	56.97 (12.58)	58.70 (10.55)	61.60 (10.68)	66.23 (8.68)
Mental Health*	.23 (.08)	.18 (.09)	.22 (.10)	.14 (.08)
es with data	26	32	21	18
Physical health*	.51 (.21)	.36 (.22)	.32 (.28)	.32 (.21)
es with data	29	33	40	29
Low Socioeconomic Status*	.47 (.20)	.50 (.24)	.48 (.19)	.49 (.15)
es with data	38	48	50	33

*Mental Health, Physical Health, and SES are presented as the proportion of the sample with a mental or physical health condition or in the socioeconomic disadvantage category. See methods Section 3.6 for additional details on how these items are coded.

integration had a similar magnitude of effect on reducing inflammation, suggesting a lack of evidence for a threshold effect.

4.4. Moderator analyses: moderation by sample mean age

We then tested whether the link between social role participation and inflammation varied by sample's mean age. Across both integration and isolation studies, sample mean age did not moderate the link between social role participation and inflammation (unadjusted: $b = -.001$, 95% CI $[-.002, .001]$, $p = .512$; age-adjusted: $b = -.001$, 95% CI $[-.002, .001]$, $p = .134$). This non-significant pattern held when examining only integration studies (unadjusted: $b = -.001$, 95% CI $[-.003, .001]$, $p = .261$; age-adjusted $b = -.001$, 95% CI $[-.003, .001]$, $p = .111$) or only isolation studies (unadjusted $b = -.0001$, 95% CI $[-.004, .003]$, $p = .937$; or age-adjusted: $b = -.001$, 95% CI $[-.004, .001]$, $p = .264$).

4.5. Moderator analyses: moderation by other methodological characteristics

We tested whether the association between social role participation and inflammation varied by other methodological characteristics, including the social role participation measure used, the year at which the social role participation measure was administered, the inflammatory biomarker assessed, the type of study design, and proportion of male participants. When examining only integration studies, study design was a significant moderator of age-adjusted effect sizes ($b = .038$, 95% CI $[-.002, .073]$, $p = .043$), such that the isolation-inflammation link was stronger in cross-sectional studies (cross-sectional predicted $r = -.052$, $Z = -.052$, $CI_z[-.071, -.033]$) compared to prospective (prospective predicted $r = -.014$, $Z = -.014$, $CI_z[-.042, .014]$).

No significant moderations were observed among isolation studies (p 's $> .098$), among integration studies (p 's $> .339$), or when pooling integration and isolation studies (p 's $> .071$).

4.6. Simultaneous moderators analyses

We next examined whether the moderating effects of the methodological characteristics were independent by including all moderators in the same model. As summarized in Table 2, among integration studies,

sample mean age emerged as a significant moderator of age-adjusted effect sizes (integration: $b = -.002$, 95% CI $[-.003, -.0001]$, $p = .035$), such that the link between social integration and inflammation strengthened as sample mean age increased (mean age = 59). Specifically, as depicted in Fig. 4, among samples with younger participants, the effect size was smaller (predicted r at 1 SD below mean age = 48: $r = -.050$, $Z = -.050$, $CI_z[-.084, -.017]$), compared to samples with older participants (predicted r at 1 SD above mean age = 69: $r = -.085$, $Z = -.085$, $CI_z[-.12, -.053]$). Among integration studies, study design was a significant moderator of age-adjusted effect sizes ($b = .043$, 95% CI $[-.006, .080]$, $p = .030$), such that the integration-inflammation link was stronger in cross-sectional studies (cross-sectional predicted $r = -.067$, $Z = -.067$, $CI_z[-.097, -.038]$) compared to prospective (prospective predicted $r = -.046$, $Z = -.046$, $CI_z[-.086, -.006]$).

No other significant moderations were observed among integration studies (p 's $> .067$), among isolation studies (p 's $> .221$), or when pooling integration and isolation studies (p 's $> .074$).

4.7. Inflammatory marker subgroup analyses

Finally, for descriptive purposes, we report synthesized effect sizes stratified by biomarker across both social integration and social isolation effect sizes. Among unadjusted effect sizes, the estimated correlations were $r = -.079$ ($Z = -.079$, $CI_z[-.13, -.028]$, $p = .012$) for fibrinogen and $r = -.048$ ($Z = -.048$, $CI_z[-.071, -.025]$, $p < .001$) for CRP. IL-6 and the "other inflammatory marker" category had Satterthwaite $df < 4$, indicating insufficient information for reliable subgroup estimation. Among adjusted effect sizes, the synthesized correlations were $r = -.053$ ($Z = -.053$, $CI_z[-.088, -.018]$, $p = .012$) for fibrinogen, $r = -.040$ ($Z = -.040$, $CI_z[-.059, -.022]$, $p < .001$) for IL-6, and $r = -.034$ ($Z = -.034$, $CI_z[-.048, -.020]$, $p < .001$) for CRP. The "other inflammatory marker" category again could not be reliably estimated. We present stratification by other study characteristics for the integration and isolation subsamples in Table 3.

4.8. Supplementary analyses

We examined the proportion of sample with socioeconomic disadvantage, or with indicators of poorer mental health or physical health

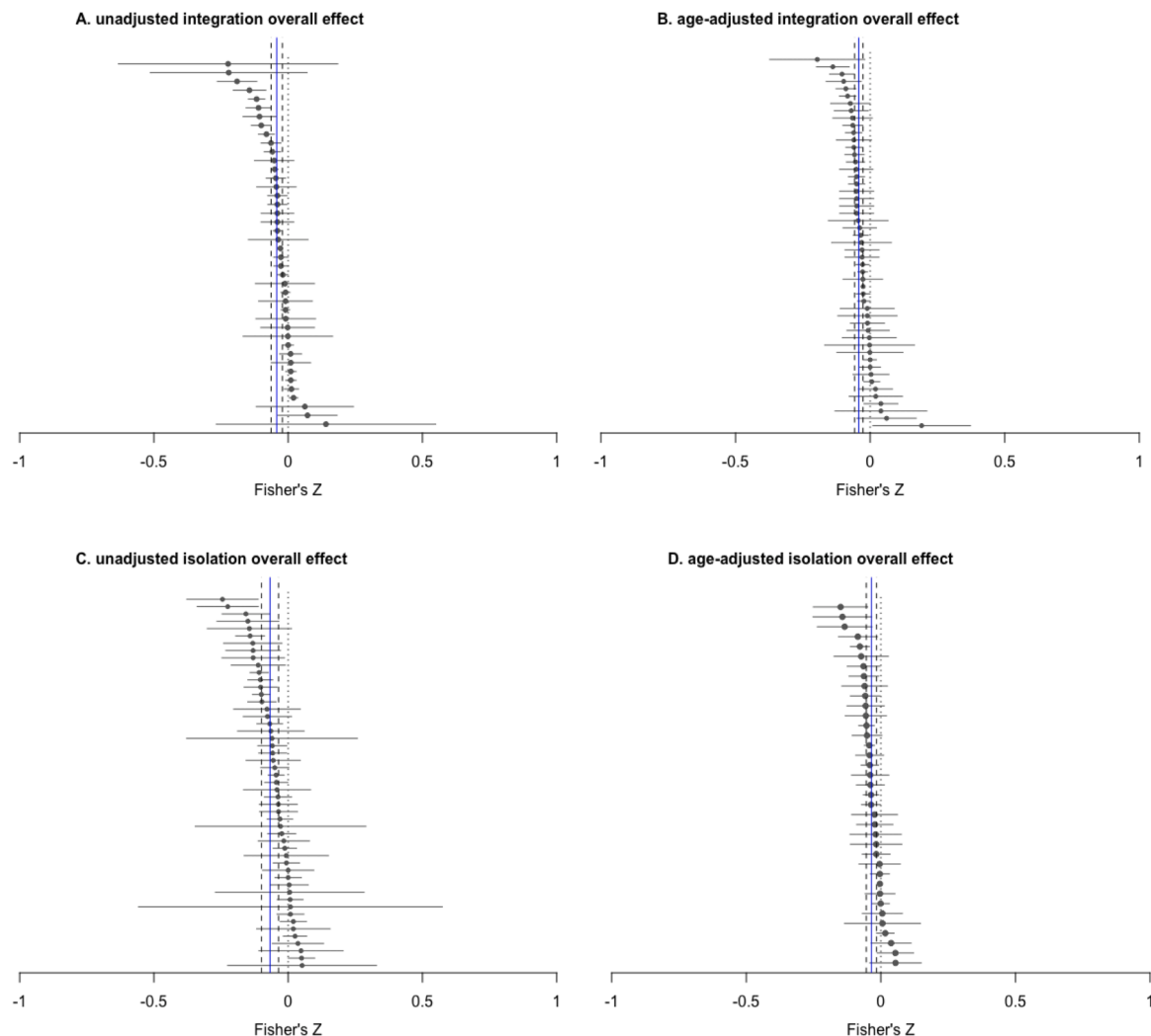


Fig. 2. Caterpillar Plots. Caterpillar plots represent the individual Fisher's Z effect sizes for each study. The blue line represents the synthesized Fisher's Z estimate, and the dashed lines represent confidence intervals.

conditions as covariates and moderators of the link between social role participation and inflammation. Because these variables were not available for all studies, the sample size varied across analyses, summarized in Table 1. Across all analyses, the synthesized effect of social participation on inflammation remained significant after controlling for SES, mental, and physical health (p 's < .037), and the magnitude of the effect size did not vary substantively ($+/- .02$).

Across both integration and isolation studies, the proportion of the sample with socioeconomic disadvantage moderated unadjusted effect sizes ($b = .13$, 95% CI [.049, .219], $p = .005$). As depicted in Figure S3, the link between social role participation and lower inflammation was weaker for samples with greater SES disadvantage ($r = -.034$, $Z = -.034$, $CI_z[-.056, -.012]$) than in samples with less SES disadvantage ($r = -.074$, $Z = -.074$, $CI_z[-.098, -.050]$). This moderation remained when restricting the analysis to only studies (26 of 34 studies) that uniformly assessed SES with education (i.e., proportion of participants without college or high school degree or sample's mean education levels; $b = .18$, 95% CI [.058, .31], $p = .008$).

This moderation by SES was not significant when tested among only integration studies (p 's > .05), isolation studies (p 's > .294), or in age-

adjusted effect sizes (p 's > .346). Moreover, the proportions of sample with or at risk for mental or physical health³ did not moderate unadjusted or age-adjusted effect sizes among integration studies (p 's > .05), among isolation studies (p 's > .488), or when pooling integration and isolation studies (p 's > .373).

Within the isolation studies, we also tested whether the isolation threshold moderated the relationship between social isolation and inflammation. There was no significant moderation in the adjusted or unadjusted isolation samples (p 's > .294).

5. Discussion

Over the past several decades, studies have investigated whether holding more social roles is linked to lower systemic inflammation, a biological pathway proposed to link social role participation to health outcomes. The current meta-analysis synthesized evidence from 38 unique samples (65 unadjusted and 77 age-adjusted effect sizes), including studies that assessed social role participation continuously (degree of integration) and categorically (social isolation vs. not), spanning diverse demographic characteristics, measurement approaches, and inflammatory biomarkers.

³ When testing physical health as a moderator, sensitivity analyses included sample mean age as an additional covariate and the results remained consistent.

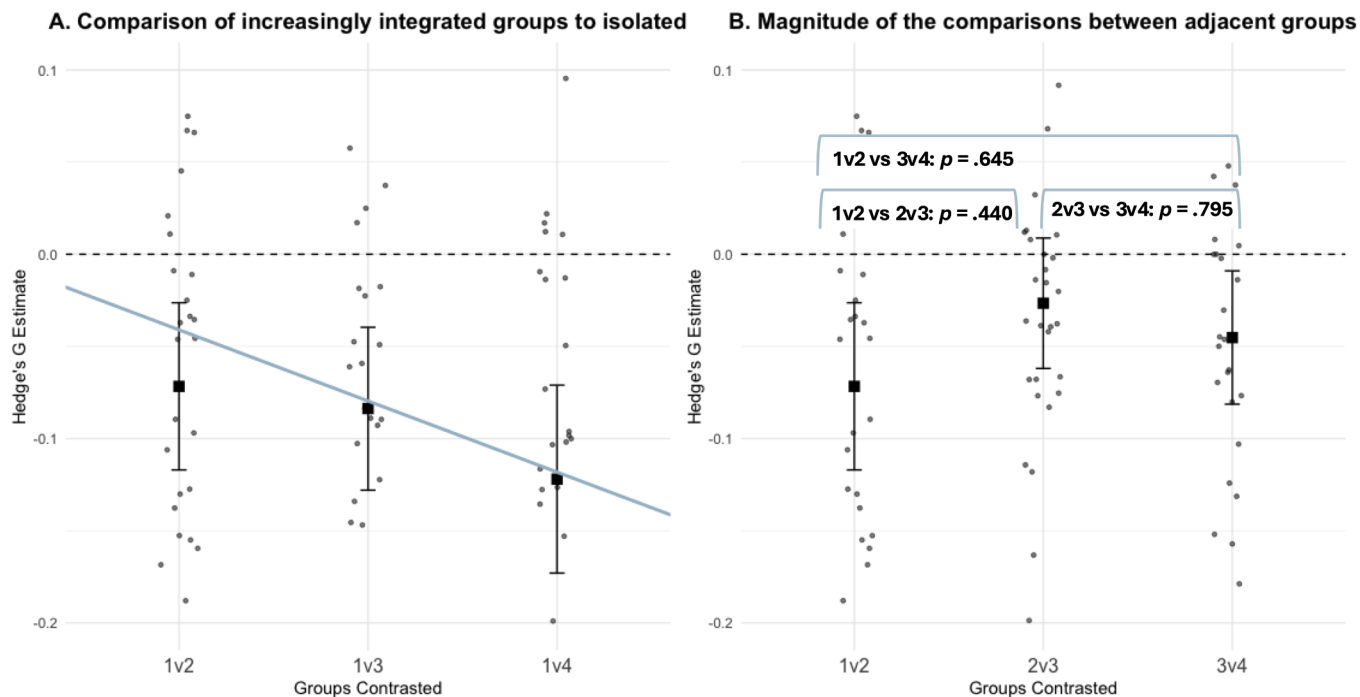


Fig. 3. Mean Comparisons Between Each Level of Social Integration. The square shaped points represent the synthesized Hedge's g estimates for each increasingly integrated group (i.e., the second lowest integrated, intermediate integrated, and most integrated) compared to the most isolated group and the CIs are extracted from the model estimate. In Panel A the blue line represents the linear decrease of the Hedge's g estimate across each comparison to the isolated group ($b = -.04$, 95% CI $[-.070, -.009]$, $p = .017$). Panel B demonstrates there are no significant differences in the magnitude of the comparisons between adjacent groups. Panel A provides support for a graded effect and Panel B demonstrates there are no significant threshold points.

Table 2
Moderator Meta-regression table.

Age-adjusted Integration	Estimate	SE	t-value	dfs	P	CI lower	CI upper
Intercept	-0.067***	0.012	-5.396	7.14	0.001	-0.097	-0.038
Inflammatory marker (ref= the grand mean)							
CRP	0.005	0.014	0.360	6.72	0.730	-0.029	0.040
Fibrinogen	-0.003	0.014	-0.221	7.24	0.831	-0.035	0.029
IL-6	0.010	0.012	0.829	6.88	0.435	-0.018	0.038
Other inflammatory marker	-0.012	0.028	-0.425	2.58	0.704	-0.110	0.086
Proportion male	-0.081	0.042	-1.929	4.13	0.124	-0.197	0.034
Study quality	-0.005	0.006	-0.951	10.24	0.363	-0.018	0.007
Conversion confidence	-0.001	0.012	-0.093	7.92	0.928	-0.030	0.027
Mean age	-0.002**	0.001	-2.591	7.17	0.035	-0.003	-0.000
Age range	0.000	0.000	1.056	10.43	0.315	-0.001	0.002
Prospective design (ref= cross-sectional)	0.043**	0.015	2.868	5.73	0.030	0.006	0.080
Year of social measure	0.000	0.001	0.499	9.96	0.629	-0.002	0.002
Social measure Berkman-Syme (ref= Berkman)	0.021	0.015	1.431	13.37	0.175	-0.011	0.053

Note: $p < .001$ ***, $p < .05$ ***, $p < .1$

Accordingly, this review quantified the overall effect size between social role participation and inflammation and leveraged methodological heterogeneity to test theory-driven moderators. Across studies, we observed a small, but significant, association between greater social role participation and lower systemic inflammation. Notably, this association appeared graded, rather than threshold, with inflammation decreasing at each higher level of social integration. Lastly, in some models, there was also suggestive evidence that the association between social integration and inflammation may be stronger at older ages.

5.1. Overall effect size

The overall effect size for the link between social role participation and inflammation was modest ($r = -.05$ in unadjusted models; $r = -.04$ in models adjusted for age). These estimates are small by conventional standards and align with a prior meta-analysis of social integration ($r = -.08$; Uchino et al., 2018), but are smaller than those reported in an isolation-focused synthesis (CRP: $r = -.18$; fibrinogen: $r = -.10$; K. J. Smith et al., 2020). One factor that may account for this difference is the small number of studies included in the prior meta-analysis. Our analyses were based on 38 unique samples spanning multiple inflammatory markers, whereas Smith and colleagues (2020) synthesized seven studies for CRP and six for fibrinogen, rendering the synthesized estimates potentially more sensitive to individual findings. Indeed, in the CRP synthesis reported in Smith et al. (2020), most studies reported small effect sizes, whereas one study reported a much larger value ($r = -.82$); excluding this study would reduce the synthesized effect size in Smith et al. (2020) from $-.18$ to $-.06$, which is comparable to our current estimate.

Nevertheless, the modest synthesized effect size observed in the current meta-analysis invites the question of what the clinical and public health relevance of examining social integration may be. The World Health

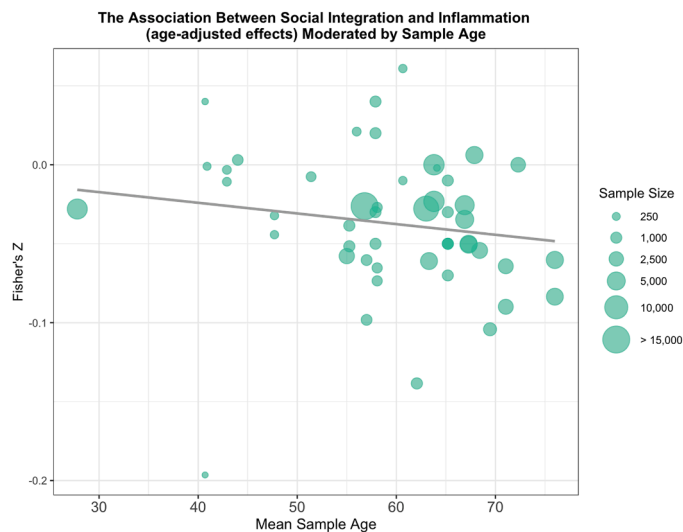


Fig. 4. Moderation by Sample Age. The figure depicts the association between sample mean age and the Fisher's Z effect sizes for each sample in the age-adjusted integration analyses. The plot depicts a trend where, as the sample mean age increases, the Fisher's Z estimates get stronger (i.e., more negative association between social integration and inflammation). The size of each point represents the size of the samples. In simultaneous moderator analyses, when all moderators were entered into the model (i.e., sex, inflammatory marker, study design, type of social role participation measure, and year of social measure), age was a significant moderator of integration effect sizes ($b = -.002$, $p = .035$).

Organization estimates that somewhere between 25% and 33% of adults experience social isolation (World Health Organization, 2025). Therefore, even though the effect size is small, when considering single individuals, the issue of social isolation affects so many people that even small effects may accumulate across populations, resulting in tangible consequences for public health (Funder and Ozer, 2019). Further, the current effect size roughly translates to 10%⁴ decreased odds of systemic inflammation for each 1 standard deviation unit increase in social integration. This decrease could be clinically relevant and determine whether or not individuals meet the cardiovascular high-risk CRP cut-off of 3 mg/L. Still, the effect size is modest, and future work is needed to clarify whether its magnitude is consistent across diverse populations, identify the most sensitive inflammatory biomarkers, and evaluate potential moderating factors that could amplify or attenuate the association.

5.2. Graded vs. threshold patterns

This meta-analysis simultaneously synthesized studies that operationalized social role participation continuously (integration) and dichotomously (isolation vs. not). Findings demonstrated that both more integration and less isolation were significantly associated with lower levels of inflammation. While showing that both forms are significantly associated with systemic inflammation supports robustness, it did not clarify whether the underlying relationship is linear such that increasing degree of social integration continues to be associated with lower levels of inflammation, or whether inflammatory risk emerges only beyond a certain low threshold of social role participation. To answer this question, we meta-analytically compared inflammation levels across increasing levels of social integration. Results supported a graded pattern, such that each higher level of integration was associated with incrementally lower inflammation; furthermore, the magnitude of these decreases was comparable, providing no evidence of a threshold. To our knowledge, this is

among the first empirical evaluations of the shape of the link between social role participation and systemic inflammation; only one prior empirical study has tested this question directly, finding that the dichotomous categorization of the Social Network Index resulted in better model fit than the continuous variable (Yang et al., 2013). However, that study aimed to oversample older adults and minority groups, which may distinguish it from the broader evidence synthesized in our analyses. Future research should continue to investigate the shape of the association between social role participation and health.

That incremental increases in social roles were associated with incremental reduction in systemic inflammation in the present meta-analysis may help shed light on underlying mechanisms. Rather than merely helping individuals avoid the negative effects of social isolation, holding a greater number of roles may actively confer additional positive psychological, social, and behavioral resources. First, as described by the role accumulation hypothesis (Thoits, 2012), investing in a variety of voluntary roles provides individuals with a broader set of identities. Because each identity helps communicate, and reinforce, who one is and why one matters, collectively, these identities should foster positive self-appraisals, stronger sense of purpose, and meaning in life. Indeed, empirically, individuals who held more social roles reported greater sense of purpose and meaning in life (Ahrens and Ryff, 2006; Lee and Martin, 2023; Pinquart, 2002). Furthermore, increasing role participation may also increase opportunities to engage in meaningful group activities, such as volunteering or religious affiliations, which have unique associations with enhancing subjective well-being (Morrow-Howell et al., 2003; Wilmoth et al., 2014). Importantly, these psychological benefits can contribute to reduced activation of biological stress pathways. For example, greater sense of purpose and meaning in life has been linked to better emotional (Schaefer et al., 2013) and physiological recovery (Fogelman and Canli, 2015) following exposure to stress, which in turn may promote healthier neuroendocrine functioning over time, including less prolonged activation of the HPA axis and sympathetic nervous systems. These stress response pathways are thought to modulate the inflammatory arm of the immune system. Specifically, glucocorticoids released by the HPA axis and catecholamines released by the sympathetic nervous system bind to receptors on immune cells resulting in either down- or up-regulation of inflammatory signaling, respectively. Prolonged and/or repeated activation of the HPA axis is thought to result in decreased sensitivity of immune cells to the anti-inflammatory properties of cortisol contributing to sustained elevation of systemic inflammatory markers (Marsland et al., 2017; Miller et al., 2002). The psychological benefits of social role participation may buffer activation of these pathways and thus contribute to lower levels of inflammation. Second, participation in a wider variety of roles may also increase the chances that individuals receive multiple types of social support, including emotional and instrumental support (Lin et al., 1999; Wellman and Wortley, 1990), which may help buffer the physiological stress response, reducing downstream inflammatory activity, and long term effects on health (Cohen and Wills, 1985). Such increased access may also maximize the likelihood of receiving matching support – support that aligns with the specific needs of the receiver – which is considered a crucial factor for support to exert its buffering effects (Cohen and Wills, 1985). Third, holding multiple social roles may also promote healthier behaviors through social regulation and structure (Thoits, 2012). Roles often promote maintaining social expectations and routines, such as maintaining regular schedules, avoiding risky behaviors, and staying physically capable to meet obligations, that can accompany anti-inflammatory health benefits.

In sum, we found evidence that the link between social role participation and systemic inflammation may follow graded pattern and did not find evidence for a threshold. This may be due to the additive accumulation of psychological, social, and behavioral resources that together may contribute to lower systemic inflammation over time. These findings begin to elucidate the functional form of the social integration-inflammation link and invite questions for future research. For example, would the graded effect observed here be expected across all populations, or might its shape

⁴ Odds Ratio was estimated based on the conventional formula: $r^*(\pi/\sqrt{3})$ (Chinn, 2000)

Table 3
Overall Effects and Subgroup Analyses.

	Integration								Isolation							
	k, es	unadjusted			k, es	age-adjusted			k, es	unadjusted			k, es	age-adjusted		
Overall Synthesized Effect Size*	k = 24 es = 42	-0.043***	-0.043	0.01	k = 26, es = 51	-0.042***	-0.042	0.01	k = 27, es = 51	-0.068***	-0.068	0.01	k = 17, es = 37	-0.036*	-0.036***	0.01
Inflammatory marker type																
CRP	k = 21, es = 27	-0.039***	-0.039	0.011	k = 23, es = 27	-0.043***	-0.043	0.009	k = 23, es = 25	-0.060***	-0.060	0.016	k = 17, es = 18	-0.032***	-0.032	0.01
Fibrinogen	/	/	/	/	/	/	/	/	/	/	/	/	/	/	/	/
IL-6	/	/	/	/	k = 10, es = 12	-0.043*	-0.043	0.018	/	/	/	/	/	/	/	/
Other	/	/	/	/	/	/	/	/	/	/	/	/	/	/	/	/
Study design																
Cross-sectional	k = 22, es = 38	-0.043***	-0.043	0.01	k = 23, es = 34	-0.047***	-0.047	0.010	k = 24, es = 47	-0.062***	-0.062	0.01	k = 16, es = 33	-0.034***	-0.034	0.01
Prospective	/	/	/	/	/	/	/	/	/	/	/	/	/	/	/	/
Sex																
Male (> median=.47 proportion of men to women)	k = 11, es = 20 ^a	-0.050***	-0.050	0.007	k = 14, es = 26	-0.054***	-0.054	0.011	k = 15, es = 29	-0.064***	-0.064	0.010	k = 8, es = 19	-0.048***	-0.048	0.011
Female	k = 13, es = 22	-0.037*	-0.037	0.016	/	/	/	/	k = 12, es = 22	-0.073	-0.073	0.038	k = 10, es = 18	-0.029**	-0.029	0.010
Age																
Older (> median= 62 years)	/	/	/	/	/	/	/	/	k = 12, es = 22	-0.061**	-0.061	0.015	/	/	/	/
Younger	k = 15, es = 25 ^a	-0.037**	-0.037	0.013	k = 14, es = 27	-0.029**	-0.029	0.009	k = 17, es = 29	-0.060**	-0.060	0.021	/	/	/	/
Year of social measure																
More recent (> median= year 2004)	k = 16, es = 28 ^a	-0.054***	-0.054	0.013	k = 18, es = 29	-0.039***	-0.039	0.010	k = 14, es = 20	-0.117***	-0.117	0.021	/	/	/	/
Less recent	k = 8, es = 12	-0.018	-0.018	0.012	/	/	/	/	k = 13, es = 31	-0.063**	-0.063	0.018	k = 13, es = 28	-0.043***	-0.043	0.010
Study Quality																
Higher quality (>median= 11.61)	k = 16, es = 30 ^a	-0.036***	-0.036	0.012	k = 16, es = 35	-0.042***	-0.042	0.008	/	/	/	/	/	/	/	/
Lower quality	k = 8, es = 12	-0.063**	-0.063	0.020	k = 10, es = 16	-0.038**	-0.038	0.013	k = 14, es = 20	-0.074***	-0.074	0.022	k = 10, es = 22	-0.044**	-0.044	0.013
Social Measure Type																
Berkman-Syme	k = 9, es = 19	-0.054***	-0.054	0.007	k = 12, es = 29	-0.053***	-0.053	0.009	k = 11, es = 30	-0.072***	-0.072	0.009	k = 14, es = 30	-0.034***	-0.034	0.010
Other	k = 16, es = 23 ^a	-0.039**	-0.039	0.014	k = 16, es = 22	-0.037**	-0.037	0.015	k = 16, es = 21 ^a	-0.064**	-0.064	0.025	/	/	/	/
Mental Health																
More conditions (> median =.17)	k = 10, es = 20 ^a	-0.053**	-0.053	0.016	k = 10, es = 15	-0.059***	-0.059	0.011	/	/	/	/	/	/	/	/
Fewer conditions	/	/	/	/	/	/	/	/	/	/	/	/	/	/	/	/
Physical Health																
More conditions (> median =.28)	k = 12, es = 22	-0.041*	-0.041	0.017	/	/	/	/	/	/	/	/	/	/	/	/
Fewer conditions	/	/	/	/	k = 8, es = 15	-0.038**	-0.038	0.013	k = 12, es = 26	-0.064	-0.064	0.033	/	/	/	/
Low SES																
Greater proportion low SES (> median =.28)	k = 9, es = 17 ^a	-0.022*	-0.022	0.008	k = 13, es = 25	-0.037**	-0.037	0.009	/	/	/	/	/	/	/	/
Lower proportion low SES	k = 12, es = 21	-0.062***	-0.062	0.017	k = 11, es = 23	-0.036**	-0.036	0.013	/	/	/	/	/	/	/	/

Note. $p < .1$ * $p < .05$ ** $p < .01$ ***; ^a Models that did not have sufficient variation in conversion confidence so only study quality was covaried; Estimates with Satterthwaite $df < 4$ are considered unreliable and indicated with /.

vary across broader sociocultural contexts? In the United States, women are more likely than men to assume more emotionally demanding responsibilities in their social roles, such as caregiving or bridging roles in the family, positions between other network members who are not connected (Gerstel and Gallagher, 1993; Navaie-Waliser et al., 2002). Bridging family members can accompany greater risk of experiencing social network strain or conflict (Goldman, 2016). These expectations are often rooted in gendered social norms that assign women primary responsibility for maintaining relational and familial cohesion. As a result, while avoiding isolation may be beneficial for women, holding additional roles may yield more modest biological returns, or even become taxing, when increased social role participation is paired with increased time demands and caregiving burden (Goldman, 2016; Roth et al., 2020). Although not empirically tested, a narrative review that visually inspected cardiovascular risk across levels of social integration found that the most common pattern among men was graded, whereas women more often showed a combination of graded and threshold effects (Chin and Cohen, 2020), supporting these postulations. Moreover, developmental stage may be another important context to consider for future research. Individuals in midlife often fulfill many obligatory social roles in the workplace, as parents to children, and as caregivers to their own parents (Mehta, 2024). Therefore, in midlife, holding additional social roles captured by social network indexes (e.g., employment, group membership) may not provide any additional benefits. Because the current meta-analysis had relatively few studies with sufficient data to examine the shape of the association between social role participation and inflammation, it was not possible to determine whether the shape varied across the adult lifespan. To better understand the shape of this association and whether or not it is context dependent, an integrative data analysis approach could compare whether the pattern of effects varies based on contextual factors including life stage, gender, or culture.

5.3. Adulthood development

This study also extended previous meta-analyses on social integration (Uchino et al., 2018) and social isolation (Smith et al., 2020) and inflammation by testing age as a moderator. This question is theoretically relevant: On the one hand, older adulthood is marked by smaller social networks (Wrzus et al., 2013) as well as greater biological vulnerability, including elevated systemic inflammation (Furman et al., 2019), suggesting that social role participation may be more protective later in life by compensating for smaller networks and having increased potential impact as biological vulnerability is higher. On the other hand, the association can be stronger in earlier adulthood, a period when individuals are establishing their social identities, making them more vulnerable to experiences of social isolation and rejection (Laursen and Hartl, 2013).

Findings, however, were inconclusive: We observed a significant moderation by sample mean age such that social integration was more strongly linked to lower inflammation among samples with older mean ages; however, this moderation only emerged when controlling for other study characteristics (i.e., social measure type, year at which social role participation was assessed, inflammatory marker type, study design, and sex composition). In addition, no significant moderation emerged among unadjusted effect sizes. Together, there is only modest meta-analytic evidence that the link between social role participation and inflammation varied by age.

One explanation may be that social role participation confers similar benefits across adulthood. That is, younger adults benefit from greater social role participation because it may increase their sense of identity (Lodi-Smith and Roberts, 2010), whereas older adults may benefit from enhanced positive emotions (Story et al., 2007). Future research should examine the mechanisms linking social role participation to lower inflammation (e.g., sense of identity and positive affect), and test whether developmental stage shapes *how*, rather than *how much*, social roles relate to reduced inflammation. It is also possible that the strength

of the link between social role participation and inflammation depends less on developmental stage, but more on the specific features of the role engagement, such as its quality reciprocity (e.g., mutually supportive relationships), or emotional demands of social roles (e.g., caregiving roles).

Finally, there may also be methodological explanations for the current findings. Although a meta-analytic approach provides a feasible way to examine patterns across adulthood development, age could only be analyzed at the study-level mean, which obscures within-sample variation and introduces noise due to differing age distributions across studies. Moreover, moderation estimates were derived based on relatively few studies per age decade (average: 6 studies per decade, range: 1–14), limiting precision in effect size estimate across adulthood development. Future research would again benefit from integrative data approaches that pool individual-level data across cohorts or leverage large, longitudinal studies that cover young adulthood to late adulthood (e.g., Add Health, MIDUS, HRS) to examine how social role participation and inflammation vary across adulthood development.

5.4. Other moderators

We tested additional moderators that were selected *a priori* based on their potential to influence the association between social roles and inflammation, including inflammatory marker, study design, sex, year of social measure, and social measure type. Additionally, during the revision process, we included supplemental moderator analyses to test SES, physical health, and mental health as covariates and moderators. Within the isolation samples we also tested whether the threshold used to determine the isolation cut-off moderated the link between categorical measures of social isolation and inflammation.

For the primary moderator analyses, across all models, only study design emerged as a significant moderator, and only in the age-adjusted integration analyses. Within this subset, cross-sectional studies showed stronger effect sizes than prospective studies. This pattern suggests that the effects of social role participation on inflammation may be strongest when measured concurrently and could weaken over time. On average, the prospective studies included in our analysis collected inflammatory data 3 years after the baseline social role participation measure. Stronger cross-sectional findings may also be consistent with reverse causality and reflect a reciprocal relationship between social role participation and inflammation; more social roles may contribute to lower inflammation, and heightened inflammation may accompany health conditions that constrain an individual's ability to participate in more social roles (Eisenberger et al., 2010). Most studies to date are cross-sectional and cannot be used to determine directionality. One longitudinal study has explicitly tested for bi-directional associations and provided initial support for a reciprocal association between social participation and CRP (Gao et al., 2024). To indirectly test the reciprocal hypothesis, that physical health influences social role participation, we tested sample level physical health as a moderator. Larger effects of social role participation on inflammation in the samples with a greater proportion of individuals with chronic illness would be consistent with the reciprocal hypothesis, but we did not find that physical health was a moderator in any analysis. Study design also did not moderate associations in the overall analyses, isolation analyses, or unadjusted integration analyses. It is worth noting that across the analytic subsamples the average number of prospective effects ($es = 7$) was much smaller than the number of cross-sectional samples ($es = 38$). Meta-analytically, we cannot provide conclusive evidence regarding directionality; additional prospective research in this area is needed to better understand the direction of these associations.

In supplemental analyses, controlling for SES, physical, and mental health did not change the significance or substantially change the magnitude of the overall effect of social role participation on inflammation. Additionally, SES, but not physical or mental health, moderated the link between social role participation and inflammation, and only in unadjusted overall analyses. Samples that had a greater proportion of

individuals with socioeconomic disadvantage observed a weaker association between social role participation and inflammation. This finding contradicts the idea that social resources may have more potential to buffer stress and attenuate inflammation for individuals with lower SES, who tend to have higher levels of chronic stress and inflammation (Muscattell et al., 2020). Our moderation findings are more consistent with the diminished returns literature, which posits that social disadvantages shape not only differential risk of exposure, but also the extent to which individuals may benefit from protective resources (Assari, 2018). Empirical findings show that socially disadvantaged groups derive fewer health benefits from participating in more social roles, such as smaller gains in life expectancy (Assari, 2017). One explanation is that structural challenges, including chronic adversity and reduced access to opportunities, constrain the extent to which social resources can translate to improved health outcomes (Assari and Zare, 2024). Nonetheless, this finding should be interpreted cautiously. First, the moderation effect was not evident in the age-adjusted effect sizes. Second, these analyses were based on a reduced subset of studies because some primary studies did not report sufficient SES information. Moreover, SES was operationalized using a range of indicators (e.g., education, income, composite measures) across studies, introducing additional heterogeneity despite our efforts to harmonize these measures. Finally, a meta-analysis is not the optimal context in which to evaluate socioeconomic moderation, due to the lack of individual-level data. This question is better addressed in primary cohort studies with individual-level data, such as the Health and Retirement Study (HRS) or the Midlife in the United States (MIDUS) study, which permit direct tests of the social role participation by socioeconomic status interaction. Therefore, the present findings should be considered tentative and hypothesis-generating for future primary studies to examine whether SES shapes the association between social role participation and inflammation.

5.5. Strengths and limitations

The current study is the first meta-analysis to synthesize the association between social role participation and inflammation across *both* social integration and isolation studies. Leveraging recent meta-analytic models that allow inclusion of multiple effect sizes within each study, this work is also the first to systematically and quantitatively evaluate the *shape* of the link between social role participation and inflammation, a finding that has theoretical relevance for the underlying mechanisms. Finally, we also included unadjusted and age-adjusted effect sizes, providing separate synthesized estimates that may help future researchers compute statistical power more accurately.

There are limitations to our work. First, the current meta-analysis is based on a relatively small number of studies, reducing power for identifying moderators and computing synthesized effect size stratified by sample or methodological characteristics. Additionally, many of our analyses were conducted using subsamples of the total studies extracted (i.e., integration vs. isolation and unadjusted vs. adjusted); doing so reduced unwanted heterogeneity in the effects but also further reduced the sample sizes. Our moderation findings did not consistently replicate across these smaller subsamples and should be interpreted as exploratory. Second, this synthesis is based on peer-reviewed publications. Although this may improve the quality of the studies incorporated in our synthesis, it could also introduce bias if null findings were less likely to be published, thus inflating our overall estimate of the effect of social role participation on inflammation. However, we analytically evaluated for publication bias and did not find evidence that it influenced our effect sizes. Last, not all measures of social participation were used consistently across the lifespan, making it unclear whether the observed variation in effect sizes across the adult lifespan is entirely due to developmental periods as opposed to variation in the types of measures used. A future integrative analysis that pools raw data across a wide age range of cohorts and computes age-adjusted harmonized social integration scores could be well suited to identify any developmental stages where social roles have the strongest associations with inflammation.

5.6. Future directions

This synthesis focused specifically on objective measures of social role participation. This targeted scope reduces heterogeneity in the types of social measures that were used and improves the interpretability of our results. However, subjective aspects of social relationships are also relevant for physical health (Heinrichs et al., 2003; Kamarck et al., 1990). Prior meta-analyses that have synthesized both objective and subjective measures of social relationships from separate studies have demonstrated slightly larger effects of objective measures on inflammatory outcomes (Uchino et al., 2018). Among the few empirical studies to directly compare objective and subjective measures, most show an independent association between objective measures and inflammation that is above and beyond the subjective (Busch et al., 2018; Garvin et al., 2009; Gleib and Goldman, 2012; Kamiya et al., 2010; Shankar et al., 2011), but see also support for an independent association of subjective (Busch et al., 2018; Koyama et al., 2021). Further research is needed to tease apart the unique contributions of objective and subjective social factors, by entering both types of measures into the same models simultaneously. In the current meta-analysis, too few studies ($k = 6$) tested this analytically to make it possible to synthesize the effects.

The current synthesis highlights various pathways, including biological (i.e., HPA axis and SNS) and behavioral, through which social role participation may plausibly be linked with inflammation. However, meta-analysis is not suitable for testing mediators, and further longitudinal research examining the intermediate pathways is needed. Our analyses attempt to control for physical health, to indirectly test the reciprocal hypothesis that physical health may account for the observed association between social participation and inflammation, but this is not the most rigorous approach. Additional longitudinal work may also elucidate reciprocal associations between social role participation and inflammation (Gao et al., 2024).

6. Conclusions

The current meta-analysis provides robust support that holding more social roles is linked to lower levels of inflammation. Our preliminary age moderation findings also suggest that social integration may be increasingly important for health as we get older. Social relationships may buffer some of the negative correlates of aging and lower the risk of age-related decline across various health outcomes related to inflammation, including mobility, frailty, and cognition (Biddle et al., 2019; Ding et al., 2017; Yeom et al., 2008). Initial work in England has demonstrated that older adults are receptive to referrals that encourage social engagement (Fancourt and Steptoe, 2024). This initiative, known as “Social Prescribing”, may foster connections and be especially beneficial for the aging population. Last, we extend prior work by identifying this as a graded association where each additional social role progressively decreases inflammation. Thus, avoiding social isolation alone may not maximize the potential benefits of social relationships; rather, participation in a diverse set of social roles (e.g., volunteering, adult learning, exercise groups, arts/crafts/music groups) may continue to provide additional health benefits.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at [doi:10.1016/j.neubiorev.2026.106962](https://doi.org/10.1016/j.neubiorev.2026.106962).

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