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Social support and telomere length: a meta-analysis

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Abstract

Previous studies have shown that lower social support is associated with higher all-cause mortality (Holt-Lunstad et al. in *PLoS ONE Medicine* 7:e1000316, 2010). While social support has been associated with system-specific biological measures (e.g., cardiovascular), there is the need to elucidate more general biological mechanisms linking social support to health risk across a number of diseases. In this meta-analytic review, the link between social support and telomere length (Cawthon et al. in *Lancet* 361:393–395, 2003) was conducted based on the updated PRISMA guidelines (Page et al., 2021). Across 17 studies, higher social support was not significantly related to longer telomere length ($Zr = 0.010$, 95% CI $[-0.028, 0.047]$, $p > 0.05$). The confidence interval indicated that the bulk of plausible values were small to null associations. Little evidence for bias was found as shown by funnel plots and Kendall's Tau. Moderator analyses focusing on the measure of support, health of sample, age, type of assay specimen, and gender were not significant. In conclusion, this review showed no significant relationship between social support and telomere length and highlights important future directions.

Keywords

Social support; Telomeres; Cellular aging; Meta-analysis

Introduction

Social support refers to the perceived or actual functions served by our social support networks (e.g., emotional/informational support, Holt-Lunstad et al., 2010) and has been inversely related to the prevalence and causes of several diseases and all-cause mortality (Berkman, 1995; Cohen, 2004; Tucker et al., 1996; Uchino et al., 2001). Holt-Lunstad et al. (2010), found that across 148 independent studies, an individual's relationships predicted mortality. More specifically, individuals who had sufficient quantity and quality of social

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Conflict of interest The author declares that they have no conflict of interest.

Human and animal rights and Informed consent All procedures followed were in accordance with ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1975, as revised in 2000. Informed consent was obtained from all patients for being included in the study.

relationships had a 50% greater likelihood of survival compared to those with poor and inadequate social relationships. These findings remained consistent across factors such as age, sex, initial health status, cause of death, and follow-up period. In fact, they found that the association between social support and health were comparable with well-known risk factors such as smoking, blood pressure, and physical activity (also see Berkman and Glass, 2000; House et al., 1988; Uchino, 2004).

Consistent with these epidemiological findings, there is also research on the biological mechanisms potentially linking social support to disease morbidity and mortality. Previous studies have shown that lower social support has been associated with an increased cardiovascular response during acute stress and lower cortisol levels (Gerin et al., 1992; Rosal et al., 2004). Importantly, both measures have been linked to future cardiovascular risk (Chida and Steptoe, 2010; Pickering et al., 2006). However, with the exception of inflammation, there is a lack of knowledge on more general biological mechanisms linking social support to health risk across several diseases. Thus, the main aim of this review was to conduct a meta-analysis of the association between social support and telomere length which is an index of cellular aging linked to a number of disease processes (Cawthon et al., 2003).

Telomeres and health

Human telomeres act as protectors against cell degradation that can happen during cell division (Blackburn, 1991). This shortening of the telomere sequence can, in some cases, increase susceptibility to age-related diseases such as cancer, cardiovascular disease, as well as increase the likelihood of hypertension, diabetes, and obesity (Zalli et al., 2014). In a study done on Utah residents aged 60–97 years old, Cawthon and colleagues (2003), found that telomere length was a significant predictor of mortality, specifically for people aged 60–74 years of age. Individuals from the bottom quartile of the telomere length ratio had a heart disease mortality rate of over three times compared to their counterparts in the upper quartile. This study also demonstrated that participants who were in the bottom quartile of telomere length, had mortality rates from infectious disease that were eight times higher than participants who were in the upper quartile. These findings were some of the first showing a correlation between aging, disease risk, and telomere length. Across many subsequent studies, shorter leukocyte telomere length has been shown to increase the risk for cardiovascular, autoimmune, neurodegenerative disorders, and cancer. (Cawthon et al., 2003; Vaiserman & Krasneinkov, 2021; Willeit et al., 2010a, b). Shorter telomeres have also been associated with worse clinical outcomes (Fordyce et al., 2005), including poor outcomes in patients with coronary heart disease (Krauss et al., 2011), and infectious disease (Spyridopoulos et al., 2009).

Social support and its potential association to telomere length

Perceived support refers to the potential an individual has to access support in their social networks. Studies have found an association between perceived support and lower mortality rates even after demographic and physical health status were controlled for (Berkman et al., 1992; Blazer, 1982; Brummett et al., 2001). These results are often above and beyond that of the extent of one's social ties (e.g., Blazer, 1982) suggesting the quality of one's relationships are of importance. In fact, the perception that an individual has of their social

support availability (i.e., perceived support) has been shown to consistently buffers the effects of stress on psychological distress, depression, and anxiety (reviewed by Cohen et al., 1985; Kawachi and Berkman, 2001). This is important because stressful life events are associated with negative biological processes that can adversely influence telomere length (Aschbacher, et al., 2013; Mathur et al., 2016).

On the other hand, an individual's interaction with others (i.e., received support) refers to the direct receipt of support. It is also thought to aid in emotional regulation, thus increasing positive affect and limiting the intensity and duration of negative life moments if responsive (Cohen, 2004). Additionally, these positive cognitions and emotions help to suppresses neuroendocrine responses and enhance immune function (Cohen, 2004; Uchino et al., 1996). However, the link between received support and health has been less consistent and dependent on other contextual factors (e.g., responsiveness of the received support, Uchino, 2009).

Importantly, social support has been shown to predict biological processes that influence telomere length. Social support has been associated with lower levels of blood pressure, cardiovascular reactivity (Steptoe et al., 2000), lower plasma and urinary catecholamine levels (Grewen et al., 2005), lower overall cortisol levels (Rosal et al., 2004), and better immune function (Yang et al., 2014). In addition, Uchino et al. (2018) found, in their meta-analysis of 41 studies, that social support and social integration were significant predictors of lower inflammation with little to no evidence for bias. Because increased inflammation due to stress has been associated with shorter telomeres (Yen and Lung, 2013), and social support has been seen as a buffer to the harmful stress reactions by decreasing negative cardiovascular and neuroendocrine reactivity during stressful situations, researchers have previously hypothesized a relationship between telomere length and such psychosocial processes (Epel et al., 2004; Lin et al., 2012; Puterman et al., 2015; Shalev et al., 2013). These studies have also shown that low social support and negative social interactions with family members and racial discrimination (Chae et al., 2020) are correlated with shorter telomere length.

In one of the only meta-analyses that included relationship measures, Pepper et al. (2018) examined 138 studies using telomeres as an outcome in relation to physical disease, environmental hazard, nutrition (poorer), psychiatric illness, smoking, alcohol, sleep, physical activity (less), psychosocial, parental care, and socioeconomic status. These factors were shown to have associations with telomere length, but due to inconsistency in the methods and the smaller sample size in psychosocial studies, their results could not be determined conclusively (Pepper et al., 2018). In addition, exploratory analyses focusing on social networks found no evidence for an association. However, this meta-analysis mostly examined simple measures of social networks which are weaker predictors of health compared to perceived social support (Holt-Lunstad et al., 2010).

Since the review by Pepper and colleagues (2018), there have been a number of studies that have examined perceived support and telomere length. For instance, Stein and colleagues (2018b) utilized a sample of war veterans and found perceived support to be significantly related to longer telomeres. However, a large sample from the Health and Retirement Study

(HRS) showed no significant link between perceived support and telomere length (Lincoln et al., 2019). In fact, many studies in the area appear to show only small effect sizes (often not significant) linking social support to telomeres (e.g., Cabeza de Baca et al., 2020; Hill et al., 2016). Thus, the main aim of this review is to examine the meta-analytic association between social support and telomeres given the apparent inconsistencies in the literature.

Potential moderators

A secondary aim was to test several potential moderators of the link between social support and telomere length based on theory. A potential moderator is the different operationalizations of social support (Cohen et al., 1985) as perceived support reflects an individual's perception of the access to support whereas received support is the actual support received during a certain time frame (Wills et al., 2000). The distinction is significant because the perception of support has been consistently related to better health outcomes (Uchino, 2009). On the other hand, received support has variable links to health as it can be viewed as intrusive and threaten one's sense of independence (Bolger & Amarel, 2007). Thus, perceived support should be more strongly related to telomeres compared to received support. A second potential moderator is the health status of the sample which can inform what stage of the disease process social support might be operating. Does social support influence the development of chronic conditions or does it mostly play a protective role once one has a chronic condition and the need for support is high (Uchino, 2009)? For instance, perceived support has been observed to be the more stable measure of support over time (even when social circumstances change) and has been linked to reports of parental support and warmth (Sarason et al., 1986; Shaw et al., 2004). Because of this stability, perceived support could influence the development of chronic conditions that take decades to become clinically significant and hence should also be beneficial in healthy samples compared to received support that might be unhelpful during certain contexts.

Method

The review protocol used in this meta-analysis is detailed below and followed the updated PRISMA guidelines (Page et al., 2021). A literature search was first conducted using the major databases of PsycInfo, Medline, and the Psychology and Behavioral Sciences Collection by crossing the exact keywords of "social support and telomere", "perceived support and telomere", "social relationships and telomere", "social support and cellular aging", and "social relationships and cellular aging." This search was run up to September of 2021 and identified 79 records, with 69 remaining after duplicates were removed (see Fig. 1). Thirty-nine records were excluded after abstract review as they were reviews, they had no support measure documented, or were nonhuman studies. Of the remaining 30 records, 12 were excluded for various reasons, such as no direct measure of support, or the studies did not measure telomeres. In addition, the first authors of 3 paper that appeared to have the data to test the main effect of social support on telomeres were contacted. One author replied and provided the requested information (Beach et al., 2014) which resulted in a final sample of 18 published studies in the meta-analysis. However, only 17 effect sizes were entered as 2 publications were averaged as they used the same sample but different measures of support (Stein et al., 2018a, b).

Analysis plan and data extraction

Major details regarding studies (e.g., sample, population, outcomes) were first characterized and examined in tabular form. Both authors verified the accuracy of the details listed in the table that was subsequently also used in the moderator analyses. The subsequent meta-analysis was performed using a commercially available software package (MetaWin V2.1, Rosenberg et al., 2000). These analyses were based on a random effects model that assumes effect sizes vary across studies so that broader inferences can be made (Borenstein et al., 2009). To reduce the problem of nonindependence in omnibus analyses, when multiple assessments were reported (e.g., separate analyses for subgroups) they were first transformed to a common metric (i.e., z-scores), averaged, then entered into the meta-analysis. Correlation coefficients (r) were used as the common metric for data entry. When correlations were not presented, measures of effect size were converted to r values. Standardized regression weights were converted using the formula $r = \beta + 0.05 \lambda$, (Peterson & Brown, 2005). When p -values were the only source of data, they were transformed using the equation $r = \frac{z}{\sqrt{N}}$ based on the one-tailed z-score. Results reported as nonsignificant utilized a significance level of 0.50 (Borenstein et al., 2009). Publication bias for the omnibus meta-analysis was examined at the outcome level by calculating a funnel plot, Kendall's Tau, and the fail-safe n . Meta-regressions were performed to examine the type support measure and sample as moderators. Finally, certainty assessments were based on GRADE (Guyatt et al., 2008) and rated by both authors. As recommended for observational studies, the rating started at "low quality" and was adjusted based on the GRADE criteria.

Results

Overview of studies

The main characteristics of these studies are shown in Table 1. In total 11,616 participants were included in the meta-analysis. Almost half of these studies included a mixed sample of clinical and non-clinical participants (47%). Seven studies were non-clinical samples (41%), while only 2 studies were strictly clinical samples (12%). In addition, fourteen of the studies measured perceived support while two studies combined both perceived and received support into an index. One study (Chmelar et al., 2017) was excluded in the moderator analysis as it was the only study that exclusively measured received support.

Across all 17 studies, higher social support was not significantly related to longer telomere length ($Zr = 0.010$, 95% CI $[-0.028, 0.047]$, $p > 0.05$). The confidence interval indicated that the bulk of plausible values were small to null associations. Effect sizes ranged from $Zr = -0.18$ to $Zr = 0.38$ and the test of heterogeneity was marginally significant ($Q_{(16)} = 25.3$, $p = 0.065$). To examine the possibility of bias, a funnel plot of effect sizes as a function of sample size was plotted. It should show a roughly symmetric funnel plot that varies with increasing samples sizes (Rosenberg et al., 2000). As shown in Fig. 2, this pattern was roughly obtained suggesting no bias in the results. A direct test of the association between effect sizes and sample sizes revealed a non-significant link ($Kendall's\ Tau = -0.265$, $p =$

0.14) consistent with little bias. Thus, the evidence for an association between social support and telomere length appears weak.

Based on these data, the two authors provided a rating of the certainty of evidence based on the GRADE criteria (Guyatt et al., 2008). The meta-analysis was based on observational studies so the rating started on low quality and was adjusted based on the available evidence (e.g., bias, precision, magnitude of effect). Due to the small effect size, inconsistency of the effect sizes across studies, and little evidence of bias, both authors agreed that the final rating was low quality which means that the true effect size might be markedly different from the estimated effect (Guyatt et al., 2008). Thus, the evidence for an association between social support and telomere length is weak.

Moderator analyses

The above analyses suggest uncertainty in the association between telomere length and social support. Moderator analyses were conducted to examine if effect sizes varied as a function of the a priori moderators. It is possible that effect sizes for perceived support might be larger given the prior epidemiological literature (Holt-Lunstad et al., 2010). For studies examining only perceived support the effect size was not significant ($Zr = 0.003$, 95% CI [- 0.041, 0.047]) whereas for studies examining a combined index of perceived and received support the effect was also not significant ($Zr = 0.074$, 95% CI [- 0.588, 0.735]). The test of the moderator effect was not significant ($Q_{(1)} = 1.58$, $p = 0.21$). Of course, these analyses are limited by the lack of studies examining only measures of received support. It is also possible that effect sizes might differ based on whether the sample was clinical, nonclinical, or mixed (i.e., both clinical/nonclinical). Seven studies were determined to be non-clinical samples. The effect size for this group was $Zr = 0.02$ (95% CI [- 0.065, 0.105]), while the effect size for mixed samples was similar at $Zr = 0.005$ (95% CI [- 0.061, 0.071]). The effect size for the strictly clinical sample was also small at $Zr = 0.024$ with a wide confidence interval as only 2 studies were in this category (95% CI [- 0.710, 0.758]). The difference between these effect sizes was not significant ($Q_{(2)} = 0.15$, $p = 0.93$).

Finally, we examined several post hoc methodological moderators that might influence the link between social support and telomeres given the lack of an association. The first was the age of the sample as telomeres shorten with age (Blackburn, 1991). There was a clear separation in ages so we coded articles as using a sample with an average age less than 30 (4 studies) in comparison to studies using an average age over 30 (13 studies, analyses treating age as a continuous variable produced similar results). Although not significantly different ($p = 0.48$), studies with younger participants showed a negative association between social support and telomere length, $Zr = - 0.025$ (95% CI [- 0.19, 0.14]), whereas studies with relatively older participants showed a positive association between social support and telomere length, $Zr = 0.014$ (95% CI [- 0.03, 0.06]). The second methodological moderator was the specimen sample used to extract telomeres given differences in measurement (Lin et al., 2019). Most of the studies examined telomeres from blood but 3 studies examined salivary specimens. Effect sizes between social support and telomere length were comparable ($p = 0.76$) for both blood, $Zr = 0.014$ (95% CI [- 0.03, 0.06]), and saliva, $Zr = 0.001$ (95% CI [- 0.16, 0.17]). Finally, we examined if the gender

composition of the sample could explain variability in the effect sizes given that women might have longer telomeres compared to men (Gardner et al., 2014). For these analyses we calculated the percentage of the sample that were women and entered it as a continuous predictor. The gender composition of the sample did not moderate the link between social support and telomere length ($p = 0.16$).

Discussion

The main aim of this review was to examine if there was evidence for a meta-analytic link between social support and telomere length that could explain associations to broad-based health outcomes. A secondary aim was to examine if the type of support measure or health of the sample moderated any associations given its theoretical importance. The review showed virtually no correlation between social support and telomere length based off the 17 studies included, and analyses revealed no evidence for moderation (theoretical or methodological) although some categories had a relatively small number of studies. There was also little evidence for bias in the review. These results suggest that currently there is an undetermined and possibly null association between social support and telomere length although more studies might be needed to more accurately characterize the associated effect size between social support and telomeres.

Though our data showed no association, there were several limitations that need discussion. The first limitation would be that the number of studies wasn't large and hence a less reliable estimate of the relationship between telomeres and social support. In addition, while lifestyle choices and environment factors during adult life can alter the length of telomeres, Benetos et al. (2013) found that those effects are modest compared to the effects of telomere regeneration during growth and development. This suggests that any observed effect sizes are likely to be very small which would require a larger number of studies to detect. It is worth noting that the largest studies in this literature had effect sizes that clustered around zero (see Fig. 2).

It is also important to note that the enzyme telomerase plays an important role in telomere maintenance and regeneration (Effros, 2011). Only one study in this meta-analysis measured both telomeres and telomerase (Zalli et al., 2014). They found that the short telomere group with high telomerase activity had the lowest levels of social support (Zalli et al., 2014). Although the results are complicated, future work should also examine telomerase given its well-established role in telomere regeneration and work showing that positive psychosocial factors might increase telomerase activity (e.g., mindfulness, Schutte & Malouff, 2014).

Our post hoc moderation analyses addressed several salient methodological issues that might have obscured an association between social support and telomeres. That is, age, specimen type, and gender did not appear to moderate the findings, albeit the small number of studies in some categories notwithstanding. However, there are a number of other methodological issues that are difficult to separate in the current set of studies including protocol variations in measurement related to quantitative PCR (qPCR, Lin et al., 2019). Future research will be needed to determine the impact of such methodological differences in evaluating links between psychosocial processes and telomere length.

Social support having an impact on health is widely accepted among social scientists (also see Berkman, 1995; Berkman and Glass, 2000; Cohen, 2004; Volgi et al., 2007; House et al., 1988; Uchino, 2004; Uchino et al., 2001). These results currently argue against telomeres as a viable biological pathway given the current state of the literature. Social support has been linked in meta-analyses to other biological pathways including inflammation and cardiovascular reactivity (Teoh & Hilmert, 2018; Uchino et al., 2018). However, there are some additional considerations based on the support literature that might guide future work in the area. One important issue is the source of the support, as well as the quality of that relationship which could be differentially related to telomere length (Barger and Cribbet, 2016; Uchino et al., 2012a; Lincoln et al., 2019; Mitchell et al., 2018). For instance, family members might be more important as sources of support compared to other relationships (e.g., friends) especially in more collective cultures (Shor et al., 2013). Besides general perceptions of support, future research should focus on specific relationships as well as measures of received support.

It is also important to discuss that not all relationships are uniformly positive. Negativity in relationships is also important to examine. Positivity in relationships broadly refers to the interpersonal process that take place in successful relationships (Uchino, 2004). In contrast, relationship negativity refers to the events in one's social network that may involve conflict, apathy, and interference that can be detrimental to relationships (Brooks & Dunkel Schetter, 2011). In addition, ambivalence in relationships is conceptualized as a network member who is a source of both positivity and negativity (Uchino et al., 2012). Illustrating the value of more complex assessments, Uchino et al. (2012a) found that ambivalence toward parents predicted shorter telomere length in a community sample. Ambivalent ties have also been linked to higher inflammation and blood pressure which can impact telomere length (Birmingham et al., 2009; Holt-Lunstad et al., 2003; Uchino et al., 2013). Future studies examining more nuanced operationalizations of relationship and social support could prove to be helpful.

In summary our results showed no significant relationship between social support and telomere length. Future research will be needed to address the limitations of this review including the relatively small number of studies, additional moderators, and different sources/operationalizations of relationships. Until then, the search for general biological mechanisms linking social support to broad-based mortality will continue, with the strongest evidence so far being its links to inflammatory processes.

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Availability of data and materials

The authors have full control of all primary data and agree to allow the journal to review the data if requested.

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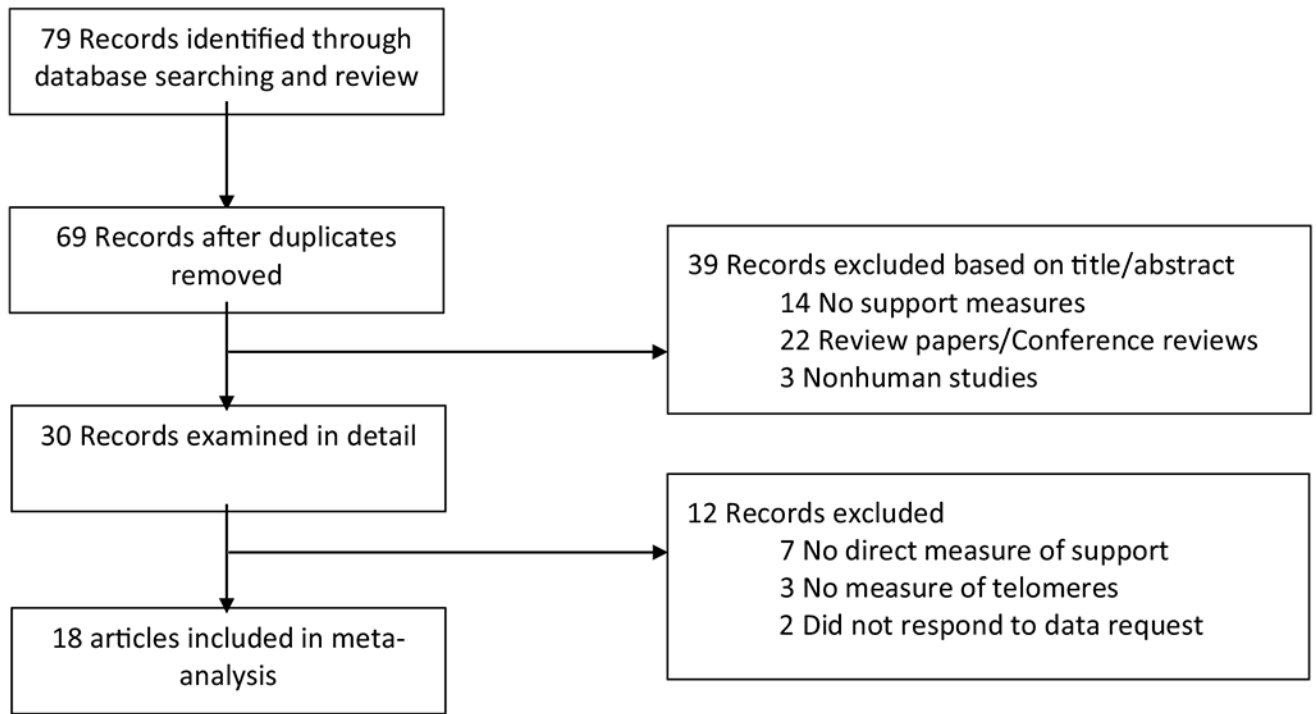


Fig. 1.
Meta-analytic flow chart describing selection of studies

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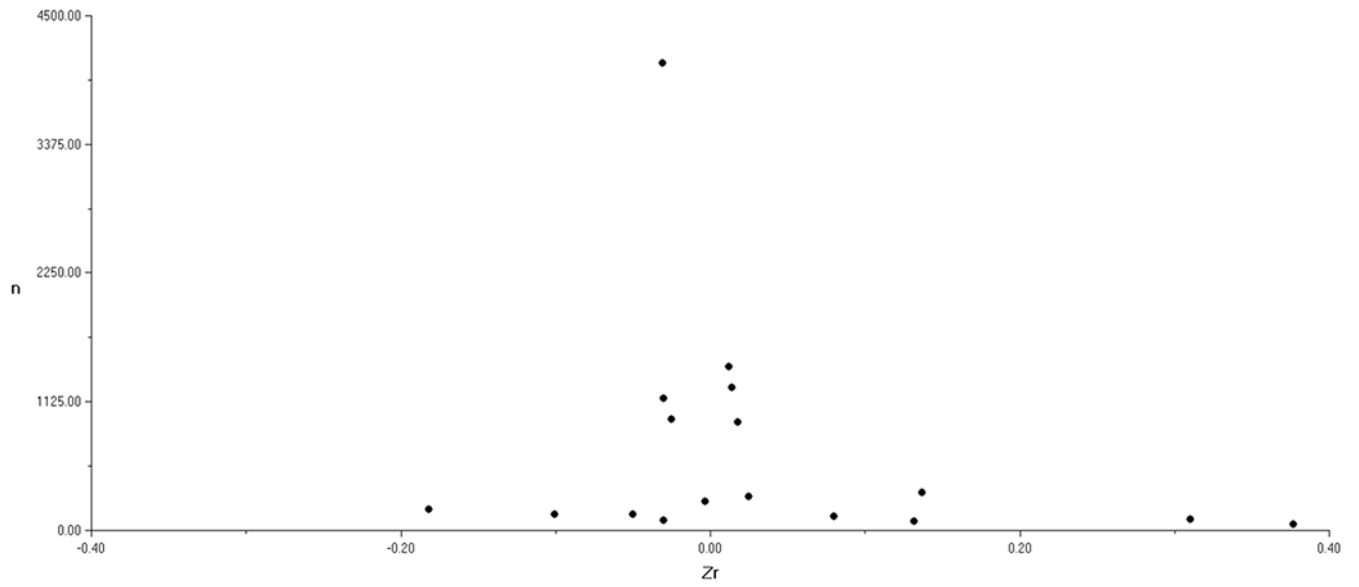


Fig. 2.
Funnel plot of effect sizes as a function of sample size

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Table 1

Study details used in meta-analytic review

Study	N*	Sample	Support measure	Effect size Zr	Var(Zr)
Uchino et al. (2012a)	83 men, 53 women 93% White	B: Healthy, chronic conditions (excluding cancer and other immunologic conditions)	General perceived support	- 0.1003	0.0075
Yen and Lung (2013)	298 Taiwanese (59.4% male)	B: Healthy, cardiovascular disease, and diabetes	Neighborhood perceived support	0.0250	0.0034
Carroll et al. (2013)	948 (47.5% men) 176 (18.6%) White, 502 (53%) Hispanic, and 270 (28.5%) African American	B: Healthy, diabetic, cardiovascular disease and obesity	General perceived support	0.0180	0.0011
Beach et al. (2014)	183 African Americans	NC	Perceived family support separated from family conflict scores	- 0.1820	0.0056
Zalli et al. (2014)	165 men, 168 women 100% White	NC	General perceived support	0.1368	0.0030
Hill et al. (2016)	1252 Black and White adults from Nashville Stress and Health Study	NC	Perceived family, friend, religious support	0.0140	0.0008
Barger and Cribbet (2016)	619 women, 811 men from the National Health and Nutrition Examination Survey 86% White	B: Healthy, chronic conditions	Received spousal support Number of received support sources Perceived financial support	0.0120	0.0007
Koenig et al. (2016)	251 women 112 White, 93 Black, 21 Hispanic, 18 Asian, and 7 were Other	NC	Number of supports Perceived support Religious support	- 0.0030	0.0040
Chmelar et al. (2017)	112 women, 29 men	B: Healthy, cardiac risk, diabetes	Received work support	- 0.0500	0.0072
Mitchell et al. (2018)	81 pregnant women 42 White, 39 Black/African American	NC	Perceived support from family, friends, significant other	0.1318	0.0128
Al Ahwal et al. (2018)	21 Saudi women, 29 Saudi men	C: Cancer patients	General perceived support	0.3769	0.0213
Stein et al. (2018a, b)	99 male Israeli veterans	B: Healthy, diabetes, kidney disease, cancer, cardiovascular conditions	Perceived emotional and instrumental support/Received support	0.3106	0.0104
Manczak et al. (2020)	122 girls White: 64%, African American: 2%, Latina 4%, Asian: 7%, mixed or other: 23%	NC	Perceived peer support	0.0802	0.0084
Lincoln et al. (2019)	4080 men and women (86% White, 14% African American)	C	Perceived support	- 0.0310	0.0002
Sosnowski et al. (2019)	90 White women	NC	Perceived support from friends and relatives	- 0.0300	0.0115
Hailu et al. (2020)	618 women, 535 men 26.8% White, 30.3% Black/African American, 42.9% Hispanic and Latino	B: Healthy, hypertension, diabetes, cancer	General perceived support	- 0.0300	0.0009

Study	N*	Sample	Support measure	Effect size Zr	Var(Zr)
Cabeza de Baca et al. (2020)	633 women and 336 men from CARDIA study 59.55% White, 40.45% Black	B: Healthy and chronic conditions (based on other publications from CARDIA)	Perceived emotional support from family	- 0.0250	0.0010

* Race/ethnicity is not tabled when not reported

NC = nonclinical; C = clinical; B = both clinical, nonclinical; Zr = Fisher's transformation of r; var(Zr) = variance of Zr