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Wiley, Joshua F., Bei, Bei, Bower, Julianne E. and Stanton, Annette L.

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Relationship of Psychosocial Resources with Allostatic Load: A Systematic Review

Joshua F. Wiley, Ph.D.^{1,2}, Bei Bei, DPsych (Clinical), Ph.D.^{3,4}, Julienne E. Bower, Ph.D.^{1,5,6}, and Annette L. Stanton, Ph.D.^{1,5,6}

¹Department of Psychology, University of California Los Angeles, USA

²Centre for Primary Care and Prevention, Mary MacKillop Institute for Health Research, Australian Catholic University, Victoria, Australia

³School of Psychological Sciences and Monash Institute of Cognitive and Clinical Neurosciences, Faculty of Biomedical and Psychological Sciences, Monash University, Victoria, Australia

⁴Centre for Women's Mental Health, Royal Women's Hospital, Department of Psychiatry, University of Melbourne, Victoria, Australia

⁵Department of Psychiatry and Biobehavioral Sciences, University of California Los Angeles, USA

⁶Jonsson Comprehensive Cancer Center, University of California Los Angeles, USA

Abstract

Objective—Allostatic load (AL) represents cumulative wear-and-tear on the body, and is operationalized as a multi-system index of biomarkers. AL is associated with morbidities and mortality, leading to a growing body of literature that uses AL as an outcome on its own right. Psychosocial resources (PSRs), such as mastery and social support, may influence health outcomes in part via AL, and the current review seeks to characterize the relations between PSRs and AL.

Methods—A systematic review was conducted by searching PubMed, CINAHL Plus, PsycINFO, Scopus, and Embase for studies examining the relation between PSR(s) and AL in humans. From 1,417 abstracts screened, 60 full-text articles were reviewed, and 24 studies met inclusion criteria.

Results—Mixed evidence exists for a relationship between PSRs and AL. Most (14/24) studies used a cross-sectional design and only one study investigated whether a PSR predicted change in AL. Compared to cross-sectional studies, longitudinal studies were more likely to report a significant relationship (8/14 versus 8/10, respectively). Studies with statistically significant main or moderated effects had larger sample sizes than those reporting null effects. Whether a study reported a significant main or moderated relationship did not differ by whether psychological (8/11) or social (10/16) resources were assessed.

Address for Correspondence: Joshua F. Wiley, 18 Innovation Walk, Clayton Campus, Clayton VIC 3800, Australia, Phone: +61 3 990 59598, joshua.wiley@monash.edu.

Conflicts of Interest

The authors declare no conflicts of interest.

Conclusions—Evidence for a relationship between PSRs and AL is equivocal, and obtained significant relationships are generally small in magnitude. Gaps in the current literature and directions for future research are discussed. Longitudinal studies are needed that repeatedly assess PSRs and AL.

Keywords

allostatic load; psychosocial resources; social relationships; systematic review; health

Allostatic load (AL) theory posits that repeated adaptation to challenge or maintaining stability through change—termed allostasis (1)—leads to impaired ability to respond to stress, such as a failure to properly downregulate stress response systems, and cumulative wear-and-tear on the body. Since its introduction and the classic papers by McEwen and colleagues (2–4), research on AL and allostasis has grown steadily, with the yearly number of published studies in PubMed increasing from 4 in 1998 to 130 in 2015. Research to identify contributors to AL demonstrates its robust link with stress and adversity. In particular, low socioeconomic status (SES) as well as measures of chronic stress in the domains of work, finances, and caregiving are associated with higher AL (5). Researchers have also examined relations of potentially protective, psychosocial factors and AL. However, to our knowledge, no synthesis exists of this emerging literature. The primary goal of this systematic review is to characterize the relations of AL with psychological and social resources.

Psychosocial Resources

Psychosocial resources (PSRs) represent personal and relational factors that are intrinsically valuable, or facilitate access to something intrinsically valuable (6) in promoting mental and physical health (7). PSRs are distinguished from other resources such as SES in their focus on either intraindividual psychological resources (e.g., optimism) or interindividual social resources (e.g., supportive social relationships). In the field of psychosomatic medicine, the demonstrated relations between PSRs and physical health are of particular interest. For example, social support predicts physical health outcomes (8). Both functional (e.g., received and perceived support) and structural (e.g., social integration) support are protective factors for mortality on par in magnitude to commonly known risk factors such as smoking (for reviews, see 9, 10). Another PSR, optimism demonstrates modest relations with both psychological and physical health outcomes (for a review, see 11) and a meta-analysis of 43 studies yielded a mean effect size of $d = 0.11$ for objective physical health outcomes (12). Therefore, in examining PSRs as potential contributors to physical health, it is important to study outcomes or surrogate outcomes that are sensitive to small differences, such as biomarkers or AL.

Allostatic Load

AL is conceptualized as the cumulative wear-and-tear on physiological systems from repeated allostasis. Specifically, stress and allostasis are thought to affect “primary” mediators (13), such as the hypothalamic-pituitary-adrenal (HPA) system or the sympathetic or parasympathetic nervous systems, and then more systemic “secondary” mediators such as

the immune or cardio-metabolic systems (13). AL is often operationalized as a composite index of biomarkers from physiological systems capturing primary (e.g., cortisol, epinephrine, norepinephrine) and secondary mediators (e.g., blood pressure, glucose, cholesterol, c-reactive protein). The original operationalization was based on the markers available in the MacArthur study (13), and comprised: overnight urinary cortisol, epinephrine and norepinephrine, serum dehydroepiandrosterone sulfate (DHEA-S), systolic and diastolic blood pressure, waist-to-hip ratio (WHR), high density lipoprotein (HDL) and total cholesterol, and glycosylated hemoglobin (HbA_{1c}). Not all biomarkers have existing clinical risk thresholds, and existing thresholds are not relevant to all populations (e.g., healthy populations). AL is often used as a relative measure with high risk defined as the upper (or lower) quartile. Biomarkers are commonly averaged within systems and then summed, though more nuanced measurement models for AL exist (14).

Research using multisystem indices of AL demonstrates robust relations between AL and health. Higher AL is associated with declines in physical functioning (e.g., balance, lower extremity strength, dexterity) and memory and cognition (15), higher incidence of cardiovascular disease (16), and mortality (17). A reduction in AL is associated with lower all-cause mortality (18). Despite limitations examining biomarkers as health outcomes (19), AL's demonstrated relations with morbidity and mortality render the study of its determinants important. AL is an attractive surrogate outcome for pre-clinical studies, where poor health has not progressed to clinical thresholds. AL also may be a mechanism linking PSRs to health.

Potential Mechanisms Linking PSRs and AL

Figure 1 shows an overview of potential mechanisms relating PSRs and AL, and supporting literature is briefly reviewed here. First, PSRs are hypothesized to prevent stressors from occurring, such as through proactive coping (20). Second, PSRs may buffer the effects of stress (21, 22) or the stress response, such as diminishing physiologic reactivity to stress and/or by buffering stress effects on health behaviors. Third, PSRs may directly promote health behaviors, such as an individual high in self-efficacy believing she or he can exercise and then doing so. It is also possible that AL affects PSRs. Higher AL may act as a stressor directly or indirectly through its relation with incidence and prevalence of chronic disease and thus undermine resources (e.g., diagnosis with hypertension may result in a loss of self-esteem or perceived control). To the extent that AL is driven by stress and adversity such as low SES, models such as the reserve capacity model would suggest an increase in AL may be related to a decline of PSRs (22). Plausible physiological mechanisms also exist, such as inflammation, a component of AL, causing feelings of social disconnection (23). These mechanisms have focused on higher AL driving lower PSRs. However, an alternate perspective comes from cognitive adaptation theory (24) that argues that in the process of adapting to stress or threatening events (such as disease), individuals often seek to regain mastery or perceived control over their lives and engage in self-enhancement to boost self-esteem or bolster their resources (24). Thus it is also possible that higher AL is related to higher PSRs.

The purpose of this systematic review is to examine the empirical literature on the relations between PSRs and AL. Given theories suggesting effects from PSRs to AL and vice versa, studies including AL or PSRs as predictors or outcomes and correlational studies were included. Both main and moderated relations were examined. Studies on all ages were included as research has demonstrated that indices of AL are sensitive enough to differ based on age, race, and SES even from a young age, with SES differences shown in 10–14 years olds (25) and racial and age differences shown in a representative sample of 18–64 year olds (26).

Methods

This systematic review was conducted with a standard protocol in accordance with the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guidelines (27).

Search Strategy

Systematic searches of literature were carried out across five databases: PubMed, CINAHL Plus, PsycInfo, Scopus, and Embase. The search duration was between the inception of each database and December 2015. No language restriction was applied; records written in foreign languages were translated for review. Search strategy and terms were decided a priori. All records with either “allostatic load” or “allostasis” occurring anywhere in the title, abstract, or keywords of articles were included. The online supplement provides further details on the search strategy and exact terms for PubMed.

Selection Criteria

Reference management software (PaperPile) was used to remove duplicates automatically, and then two authors (JFW and BB) independently screened titles and abstracts for eligibility on the following criteria: *1) included empirical data and results, 2) published in peer-reviewed journals* (e.g., unpublished dissertations and conference presentations were not included), *3) conducted on humans, 4) includes measures of at least one primary and one secondary system (defined below) with a composite index of AL, and 5) included at least one PSR*. Disagreements were resolved via discussion. Full texts of the remaining articles were retrieved and screened independently (JFW and BB) for eligibility on all above criteria, plus: *6) tested or reported the relation between PSRs and AL*.

PSRs were defined and operationalized as individual differences and social relationship factors that are intrinsically valuable or facilitate access to something intrinsically valuable (6, 7). *Psychological resources* included: optimism, hope, self-esteem, self-acceptance, self-efficacy, self-regulation, control, mastery, sense of coherence, positive affect, emotion regulation, and coping. *Social resources* included any form of perceived social support, measures of structural support such as measures of social network size, frequency of contact with family/friends, social integration or social isolation, loneliness. Composite measures that included a PSR were also included. Studies in which the only PSR was marital status were excluded, because 1) marital status alone is not a comprehensive measure of structural support, 2) many studies do not focus on the relationship of marital status and AL, but rather

include it among other covariates, and 3) a meta-analysis by Holt-Lunstad, Smith (10) of various measures of social network revealed that marital status alone had one of the weaker relations to mortality.

Due to the limited research on PSRs and AL and to the difficulty in distinguishing opposite ends of a unidimensional construct (e.g., a small social network could be termed socially isolated or lacking social integration), psychosocial variables where either high or low levels are protective were included. Constructs where neither high nor low levels were necessarily protective were not included (e.g., low depressive symptoms do not indicate high levels of positive affect or social connection). In this paper, the terms “social resources” and “psychological resources” are used generally for these variables regardless of whether it is advantageous to be high (e.g., social integration) or low (e.g., social isolation).

There is no standard way to operationalize AL. Based on AL theory that there is a cascade from the primary mediators (including the HPA system and sympathetic adrenal medullary systems) to secondary mediators (involving systems such as metabolic and the immune system), we defined AL as requiring at least one biomarker from the primary and secondary mediator systems. This definition was selected to provide a minimum quality criterion, although many studies assessed more biomarkers and systems, and as the emphasis on primary mediators is a differentiating feature of AL compared to related constructs such as the metabolic syndrome (28, 29).

Data Extraction and Quality Assessment

A data extraction sheet was developed based on the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement, a guideline for reporting observational studies. In addition to study and participant characteristics, data on the following variables were extracted: any PSR variable, AL, and any moderator of the relationship between PSRs and AL. There is not a standard quality assessment tool for systematic reviews of observational studies without group comparisons. We assessed three criteria related to quality of information on the association between PSRs and AL that were not already included in the study characteristics (Table 1): 1) whether the study had a clearly described, directional *a priori* hypothesis, 2) whether the sample size was justified, and 3) whether the measures of PSRs were validated and had adequate internal consistency reliability. These criteria were coded in relation to PSRs and AL. For example, a study with *a priori*, directional hypotheses, but not hypotheses related to PSRs and AL would be coded as low quality.

Main and moderated effects of PSRs with AL are reported separately in tables. Moderated effects were grouped by the moderating factor into themes: 1) stress or adversity (i.e., testing a stress buffering hypothesis), 2) age, and 3) sex, as studies have reported sex differences in AL and specific resources such as social support. Meta-analysis was not undertaken due to the limited number of studies and variability in the measures and design of studies. However, we extracted or calculated standardized effects (e.g., using the formula $\beta = b * SD_X / SD_Y$) and note if neither were possible.

Results

Study Design and Characteristics

Figure 1 shows a study flow diagram to the 24 eligible studies and inter-rater reliability for inclusion/exclusion reasons. Quality assessments are in Table 2. Most ($k = 14/24$) studies had an a priori directional hypothesis and used validated measures of PSRs or reported adequate internal consistency reliability ($k = 12/24$). No study justified the sample size a priori (e.g., specifying expected effect size and conducting power analysis).

Studies came from 13 unique samples (see Table S1, Supplemental Digital Content 1). Studies were geographically diverse, conducted in North America ($k = 21$), Asia ($k = 3$), and Europe ($k = 1$) with most ($k = 15$) using data from large community or epidemiologic studies, often on subsamples of participants for whom biomarkers were available. Average age ranged from 9.2 years to 74.5 years with approximately equal numbers of females and males. Studies were observational and employed cross-sectional ($k = 14$) or longitudinal ($k = 10$) designs. Study characteristics are in Table S2, Supplemental Digital Content 1.

There were no apparent differences in whether significant main or moderated results were found by whether psychological ($k = 8/11$, 72.7%) or social resources ($k = 10/16$, 62.5%) were assessed. The median sample size was larger in studies reporting at least one versus no statistically significant effect for main ($N=871$ vs $N=324$) and moderated ($N=420$ vs $N=282.5$) effects. Compared to cross-sectional studies, more longitudinal studies reported a significant main or moderated effect ($k = 8/14$, 57.1% versus $k = 8/10$, 80.0%). Studies with an a priori, directional hypothesis were more likely to test for moderation ($k = 12/14$, 85.7% vs $k = 3/10$, 30.0%), but there was no difference in whether a significant effect was found ($k = 9/12$, 75.0% for a priori directional hypotheses and $k = 2/3$, 66.7% for no directional hypotheses). The quality of PSR measures included did not appear to influence whether studies reported a statistically significant effect.

Measures of Allostatic Load

The most common method of creating an AL index was by dichotomizing biomarkers into “at risk” or “not at risk” based on quartiles and then summing ($k = 21$), with the remaining studies standardizing and summing biomarker scores ($k = 2$), or calculating risk based on the sum of biomarkers outside the 10th/90th percentile ($k = 1$). Scores then were either directly summed or averaged within a biological system and then summed. The number of biomarkers ranged from 6 to 24 (Median = 10). The most common biomarkers included blood pressure ($k = 24$) and catecholamines ($k = 22$). Table S3 (Supplemental Digital Content 1) shows the biomarkers used in each study. Figure S1 (Supplemental Digital Content 1) shows the count of studies using each biomarker.

Primary Findings

The 24 included studies are presented under themes of psychological ($k = 11$) and social ($k = 16$) resources. Studies that examined both psychological and social resources ($k = 3$) are included in both. Detailed results are in Table S4 (Supplemental Digital Content 1) for

psychological or psychological and social resources and in Table S5 for only social resources.

Psychological Resources (k = 11)

Control, Mastery, and Self-regulation (k = 8): In adults, two epidemiologic studies reported mastery associated with lower AL with standardized β between $-.07$ and $-.09$ (30, 31), whereas two other studies reported null effects (32, 33). In children/adolescents, control was not associated with AL (34–37).

Regarding moderated effects, in adults, mastery exacerbated the effects of caring for spouses with Alzheimer's disease on AL (32). In children/adolescents, the picture is complex. Higher self-control/regulation is associated with higher AL for those with high SES risk in one study (34) but was not moderated by poverty in another (37). A third study showed that self-regulation moderated the effects of negative emotionality on prospective change in AL such that negative emotionality was only harmful for children low in self-regulation (35).

Other Psychological Resources (k = 6): In adults, two studies assessed positive affect (33, 38) and found no main effects, although one found that positive affect buffered the deleterious effect of parenting a child with a developmental disorder (38) on AL. Two studies reported no effects for optimism (30, 39). Seven dispositional coping styles had no significant cross-sectional associations with AL (39). One study reported that for adults with low SES, the combination of “shift” (positive reappraisal, control, and emotion regulation) and persistence was associated with lower AL (40). In children, one study reported no relation between self-worth or persistence with AL (36).

Social Resources (k = 16)

Social Support/Quality of Relationships (k = 15): In adults, eight studies reported non-significant (30, 33, 39, 41–45), and five studies reported significant main effects of social support (46–50). However, not all effects were in the same direction. For example, one study found that spouse and friend support were associated with significantly lower ($\beta = -.08$) and higher ($\beta = .07$) AL, respectively (46), but other studies have shown protective effects of friend and overall support, with comparable or slightly larger effect sizes, Cohen's $d = .26$ (47) and $\beta = -.11$ (50). Some of the studies reporting null effects had comparable effect sizes, but smaller samples (e.g., $r = -.08$ for social support; 39) suggesting one reason for apparent mixed findings may be a lack of statistical power. In children/adolescents, two studies on the same dataset reported no main effect of emotional support (51, 52).

In adults, six studies examined moderated effects for social support (42, 44–47, 49). Considering moderated effects of potentially stressful circumstances, social support significantly moderated the effect of household income (49), but two studies tested and found no effects for occupational status or job demands (42, 44). Other significant moderators include age (46) and sex (45), though other studies did not find moderation by age (42) or sex (46, 47). Support for stress buffering was more consistent in children/adolescents where emotional supported buffered the effect of having high and stable perceived discrimination (51) and persistent neighborhood poverty (52).

Structural Social Resources (k = 9): In adults, nine studies assessed structural measures of support such as number of social ties or connections or frequency of social activities or interactions (30, 33, 39, 43, 45–48, 53). Four studies reported no main effects of structural support (30, 39, 43, 53). In the five studies reporting significant effects for structural support, four found higher structural support was associated with lower AL (33, 45, 47, 48) and only one reported opposite results (46). No studies in children/adolescents assessed structural social resources.

In adults, only four studies assessed moderated effects of structural social resources (30, 45–47). One study found that structural support as part of a broader composite moderated the effects of stress on AL such that higher resources buffered the effects of stress on AL (30). One study found that sex moderated the effects of structural measures (45), but two other studies found no sex differences (46, 47), though these two studies has simpler measures of structural support. Finally, one study reported no moderation of structural support by age (46).

Discussion

Summary of Findings

This systematic review characterized empirical findings on the relations between PSRs and AL. Indices of AL were well constructed, with 15 of 24 studies using at least 10 biomarkers. Half the studies (50%) used a validated PSR measure or reported acceptable psychometric properties. Most studies were cross-sectional (k = 14/24), and the longitudinal studies used earlier PSRs to predict later AL assessed at a single time point, with only one using PSRs to predict change in AL over two time points (35).

In general (k = 16), when PSRs were significantly associated with AL, higher resources were associated with lower AL either as a main effect or for a subgroup, such as those under economic strain. However, four studies reported that higher PSRs (k = 2 control/mastery and k = 2 social support/contact) were significantly associated with worse AL, or that PSRs exacerbated the effects of stress and adverse conditions.

An important finding from this review is that many studies (k = 15/24) tested for moderation, and non-significant main effects often (k = 7/15) were qualified by significant interactions. Significant moderators include perceived discrimination, neighborhood poverty, SES-related risk, household income, parental education (construed as childhood SES), age, caregiver status (presumably a chronic stressor), and sex. Thus, effects of PSRs varied as a function of other environmental and individual contexts. It is noteworthy that of the 14 studies with both a priori directional hypotheses and adequate PSR measures, 12 tested for moderation. However, not all moderated effects were in the expected direction. For example high levels of mastery were associated with higher AL among adult caregivers, but not controls (32).

Effect Sizes

To facilitate comparison, though in different metrics, a Cohen's d of 0.20, a log odds ratio of 0.36, and correlation (or standardized beta in the case of simple linear regression) of 0.10 are

approximately equivalent in magnitude (54). Effect sizes for the associations between PSRs and AL were typically small, even for statistically significant results. For example, statistically significant results for psychological resources were typically in the range of $\beta = .07$ to $.15$. Non-significant results were comparable, but often from studies with smaller sample sizes. For example, one study had a $\beta = .06$ (37) and another reported (absolute) correlations of $.08$ and $.09$ (39). Examining social resources, effect sizes were comparable, with β s from $.07$ to $.14$ for statistically significant results, as well as one study reporting adjusted Cohen's d of $.22$ and $.26$ for social ties and support (31). Again, studies without statistically significant effects sometimes reported comparable effect sizes, with one reporting absolute correlations of $.15$ and $.09$ (53), but in a smaller sample.

These effects may be small partly because the total effect of psychosocial factors on AL is small and partly because the effects at any snapshot in time are likely limited but may cumulate over time. Another potential reason for small effects is that PSRs were incompletely assessed, with studies typically assessing the effects of specific resources rather than the total effect of multiple PSRs. Finally, many studies adjusted for covariates including health behaviors such as physical activity and smoking, which may represent mechanisms through which PSRs are linked to AL. Controlling for mechanisms linking PSRs and AL would diminish the apparent effect size of PSRs. Further, although these effects are small, they are consistent with the larger literature. A systematic review and meta-analysis of 148 studies, found a log odds ratio of 0.41 for the effect of social relations on mortality (10). Similarly, these effects are close to the magnitude of established risk factors. For instance, the log odds ratio for the effect of smoking cessation on mortality in individuals with coronary heart disease is about 0.50 (10). Nevertheless, a practical implication of small effect sizes is that studies on PSRs and AL will need large samples to be adequately powered—power of $.80$ to detect a Cohen's d of 0.20 requires a sample size of 788 , and for a simple linear regression to detect a $\beta = .10$ requires a sample size of 779 .

Gaps and Opportunities

Conceptual and Theoretical Issues—Current biopsychosocial theories provide a framework for understanding how PSRs may be related to AL, including via prevention of stress, buffering of stress responses, and health behaviors (7). For example, individuals high in PSRs may be less likely to smoke cigarettes or more likely to engage in physical activity, and PSRs may prevent or buffer the deleterious effects of stress and physiological stress reactivity on health (e.g., 21). However, current studies did not examine potential mechanisms linking PSRs to AL, although many studies adjusted for covariates some of which included affect and health behaviors, which may in fact represent mechanisms rather than confounds. Future studies are needed to examine pathways that account for the association between PSRs and AL. The mechanisms linking PSRs to physiology are also important to consider when determining which covariates to include in studies of PSRs and health, as they may represent the mechanisms through which PSRs influence physiology. For example, if people who are high in PSRs are more adherent to medical treatment and have a better diet, including adherence and diet as covariates, without examining how PSRs relate to each, could mask the salutary effects of PSRs on health through health behaviors.

The current review revealed a number of moderated effects, and distinctive effects from different types of PSRs, on AL. For example, certain resources (e.g., perceived mastery and control) exacerbated the effects of stressors on AL (SES-related risk and caring for a spouse with Alzheimer's disease), whereas others (positive affect) buffered the effect of caring for a child with a developmental disorder. Likewise, the effects of social resources were related to lower AL among women or among men in different studies. Theories are needed to delineate which PSRs, for whom (e.g., age, sex), and under what conditions (e.g., caregiving, high or low socioeconomic status), are related to AL.

The literatures on AL as a predictor and outcome are distinct. Few studies have merged these two approaches, and none of the studies included in this systematic review included a comprehensive model, as suggested by Friedman and Kern (19). For example, no study examined a model that included PSRs, AL, and health outcomes, with PSRs predicting change in AL, and change in AL predicting health outcomes. If AL is to be used as a surrogate endpoint in place of health outcomes, it is important to demonstrate that AL is both related to the health outcomes and accounts for the relationship between PSRs and health outcomes. Only once the causal ordering of PSRs, AL, and health outcomes has been established, can we draw strong inference about health outcomes or risk for health outcomes from studies of PSRs and AL alone.

Methodological Considerations—PSRs are inconsistently operationalized, and studies tended to examine only a few PSRs, including ad-hoc measures (e.g., in a longitudinal study, count of assessment waves with weekly contact with non-resident children was used as an index of social network or integration). There was also a diversity of PSRs assessed, which makes it challenging to compare results across studies as differences may be due to sample characteristics, study time frame, or measuring different resources. This is particularly true for pediatric versus adult literatures, where there was little overlap in the resources assessed.

The use of ad-hoc measures is a particular challenge for social integration or structural support measures. Although these measures have face validity, there is inconsistent use of developed and validated assessment tools or scales. Diverse and thorough measures of social networks or integration as well as rich measures of psychological resources are needed to provide more conclusive evidence for the presence or absence of an association between PSRs and AL. This may be particularly important for social integration, in light of a meta-analysis (10) showing stronger effects for complex versus simple measures of social integration on mortality. It may also be helpful to examine the relations of composite measures of PSRs capturing overall resource levels, which have been shown to predict biomarkers such as cortisol (55). Where ad-hoc measures are used, studies should report the psychometric properties of constructed measures and any other validity data that can be mustered.

All studies included in this review were observational and many were cross-sectional. Studies that were longitudinal used PSRs to predict AL, but not *change* in AL (with one exception). Studies are needed that move beyond cross-sectional associations to assess change in AL and PSRs over time in order to determine the magnitude and direction of association. If stronger observational data provide evidence that PSRs predict change in AL,

then experimental or intervention studies designed to manipulate PSRs may be considered to provide stronger causal evidence.

Finally, findings suggest that relationships of PSRs with AL may be qualified by significant interactions. Future research on PSRs and AL should carefully consider potential interactions (e.g., with SES, caring for an ill loved one, gender), in order to avoid calling a missed moderated relationship a null relationship.

Limitations

Because the field of research on PSRs and AL is small, we opted for an inclusive definition of PSRs, although researchers may not consider some included variables (e.g., social isolation) as PSRs. Indeed, some factors such as social isolation may be considered a stressor and others such as loneliness or (low) self-worth/self-esteem are included in common measures of depression symptoms. Due to the ambiguity in separating these constructs, the findings reported may partially overlap with relations of constructs including stress and negative psychological states that also relate to AL.

It is possible that some studies included a PSR only as a covariate and did not mention it in titles, abstracts, or keywords. We mitigated the risk of missing relevant studies by using a broad search strategy to retrieve all studies including “allostatic load” or “allostasis” and using two independent raters. Several of the included studies assessed PSRs only as covariates or potential confounds, suggesting that our search process identified even studies that did not focus specifically on PSRs. Because we only searched for “allostatic load” or “allostasis” eligible studies including composite measures of biomarkers, but that did not refer to or conceptualize them as AL could have been missed. The current results may also be influenced by publication bias; however, many studies reporting multiple PSRs included null as well as significant results, and there were studies published with solely null results. Meta-analysis was not possible due to substantial heterogeneity in measures of PSRs, study design, and whether moderation was examined.

Conclusion

Across populations diverse in geographic location, age, race, and sex, mixed evidence exists for both a direct relationship between PSRs and AL, and a role of PSRs moderating the relations of stressors such as discrimination, SES, and caregiving status with AL. Findings are tempered by null and often cross-sectional results. Theoretical models that explain the relations among PSRs, moderators, and AL are much needed to guide future research. Rigorous, longitudinal studies that predict change in AL from comprehensive assessments of PSRs will provide more conclusive evidence on the association between PSRs and AL.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Acronyms

AL	allostatic load
PSR	psychosocial resources
SES	socioeconomic status

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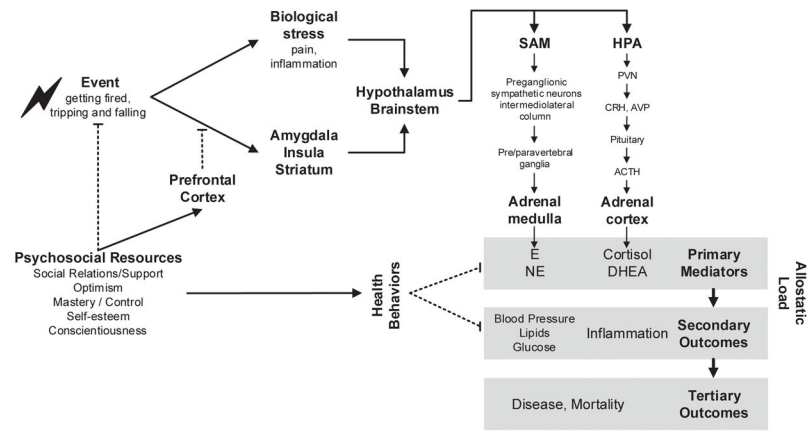


Figure 1. Conceptual overview of potential mechanisms for the relations between psychosocial resources and allostatic load. Solid and dashed lines indicate positive and negative effects. SAM = sympathetic adrenal medullary system; HPA = hypothalamic pituitary adrenal axis; E = epinephrine; NE = norepinephrine; DHEA-S = serum dehydroepiandrosterone sulfate.

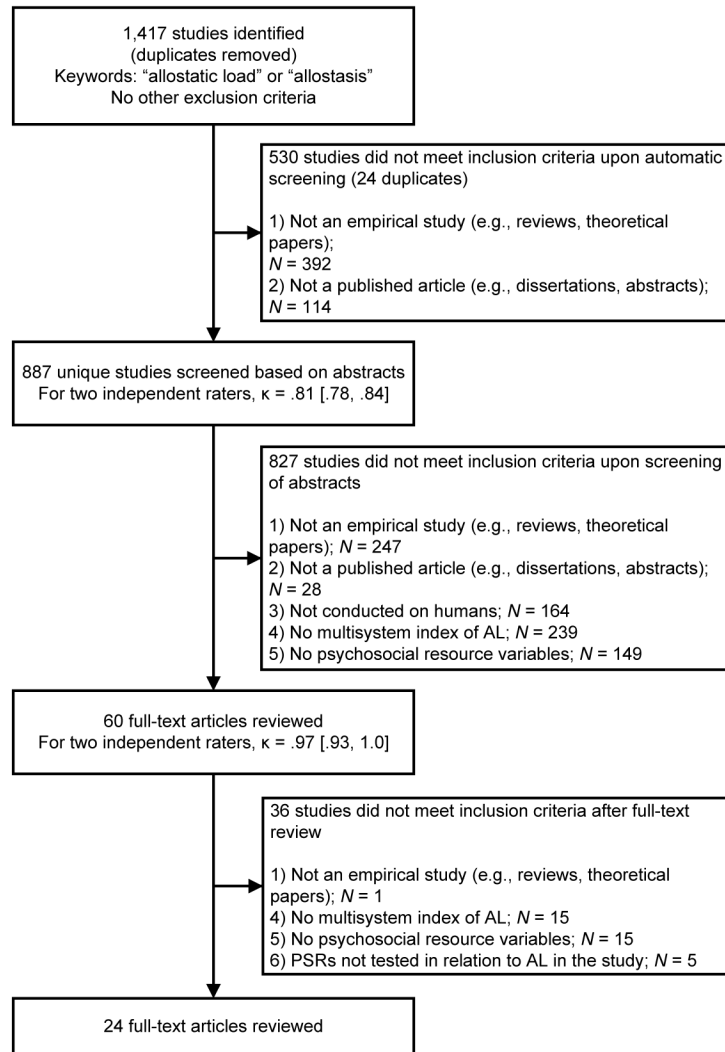


Figure 2. Flow chart of study retrieval, review, and inclusion.

Table 1

Quality Assessment Scoring

	Low (-)	Medium (O)	High (+)
A Priori Hypothesis	No hypothesis reported	Hypothesis reported, but non-directional	Directional hypothesis reported
Justification for Sample Size	No information on sample size determination reported	Sample size unjustified or discussed post hoc	Sample size justified to be adequate (e.g., power analysis)
Quality of PSR Measure	Unvalidated and no psychometric properties reported	Psychometrics reported but inadequate (Cronbach's $\alpha < .70$)	Validated scale, objective outcome, or good psychometric properties reported

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Table 2

Study Quality Assessment

	A Priori Hypothesis	Justification for Sample Size	Quality of PSR Measure(s)
Brody 2014a (51)	+	-	+
Brody 2014b (52)	+	-	+
Brody 2013 (34)	+	○	+
Brooks 2014 (46)	+	○	+
Chen 2012 (40)	+	○	+
Dich 2015 (35)	+	-	+
Evans 2003 (36)	-	-	○
Evans 2013 (37)	+	-	+
Friedman 2015 (41)	○	-	-
Glei 2007 (30)	○	-	○
Gruenewald 2012 (33)	-	-	○
Hawkey 2011 (39)	○	-	+
Juster 2013 (42)	+	○	+
Maselko 2007 (43)	-	-	-
Roepke 2010 (32)	+	-	+
Schnorpfeil 2003 (44)	+	-	○
Seeman, M. 2014 (31)	+	○	○
Seeman, T. 2004 (45)	+	-	-
Seeman, T. 2014 (47)	○	-	○
Seeman, T. 2002 (48)	○	-	-
Singer 2000 (49)	○	○	+
Song 2014 (38)	+	○	+
Sotos-Prieto 2015 (50)	○	-	-
Weinstein 2003 (53)	+	○	-

Note. Category description are in Table 1.

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