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BRIEF REPORT

Psychosocial Adversity and Allostatic Load Burden in Midlife and Older Ages

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Objective: To investigate the individual and cumulative impact of childhood and adulthood adversity on allostatic load (AL) burden. ***Method:*** Retrospective cross-sectional study design involving 4,165 participants from the first wave of The Irish Longitudinal study on Ageing (TILDA). AL was operationalized using 12 biomarkers across four physiological systems (cardiovascular, metabolic, renal, and immune). Measures of psychosocial adversity included poverty, abuse, loss, and illness. Negative binomial regression models estimated the relationship of individual adversities and a cumulative count of adversities with AL burden, controlling for age and sex. Multivariable models adjusted additionally for a range of other sociodemographic and lifestyle factors. ***Results:*** Childhood poverty, childhood physical abuse, and having a spouse/partner/child experience a life-threatening illness/accident were associated with 10% (95% CI [1.04, 1,16]), 10% (95% CI [1.01, 1.18]), and 6% (95% CI [1.01, 1.11]) greater AL burden, respectively. Cumulative adversity was associated with 3% (95% CI [1.01, 1.04]) higher AL burden. Adjusting for sociodemographic and lifestyle covariates rendered the association of childhood poverty (IRR= 1.04, 95% CI [.98, 1.09]; p = .190) and childhood physical abuse (IRR= 1.07, 95% CI [.99, 1.15]; p = .081) with AL burden nonsignificant, while the association of having an ill spouse/partner/child on AL persisted (IRR= 1.06, 95% CI [1.01, 1.11]; p = .021). ***Conclusions:*** This study provided limited support for the idea that psychosocial stress leads to higher AL, with just three out of 11 adversities associated with AL.

Keywords: adversity, allostatic load, stress, physiological dysregulation, life course

Supplemental materials: https://doi.org/10.1037/hea0001098.supp

Trauma, maltreatment, and socioeconomic adversity are all recognized as having a profound impact on risk of chronic disease (Cohen et al., 2007). Although the mechanisms underlying this relationship remain to be fully elucidated, the allostatic load (AL) framework represents a plausible biological intermediary between psychosocial stress exposure and health. While stress is known to protect under acute circumstances, repeated or chronic activation of the stress response may lead to physiological "wear and tear," predisposing an individual to disease (McEwen & Seeman, 1999).

Nevertheless, research to date has not been conclusive in establishing that major repeated or prolonged exposure to adverse events leads to higher burden of AL in later life. While AL is conceptualized within a life course framework, studies linking adverse experiences with dysregulation have mainly focused on isolated life stages (Friedman et al., 2015; Glei et al., 2007; Weinstein et al., 2003), or specific life events (Dich et al., 2015; Gallo et al., 2011). Furthermore, early life may represent a critical period for the development of stress-response systems (Barboza Solís et al., 2015) but many studies exploring psychosocial adversity and AL do not include data on childhood adversities. Further scrutiny of the relative impact of individual adversities on AL burden, in lieu of the commonly employed crude counts of adversities, is warranted. This is suggested by the heterogeneity of findings across studies examining the association of isolated adverse events and

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Sinéad McLoughlin served as a lead for conceptualization, data curation, formal analysis, methodology, and writing–original draft. Rose Anne Kenny served as supporting role for resources and writing–review & editing. Cathal McCrory serves as lead for funding acquisition and Supervision, contributed equally for writing–review & editing, and served in a supporting role for conceptualization.

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AL. For example, work stress, financial strain, relationship stress, and caregiving stress were positively associated with AL in a sample of Mexican American women, yet no association was reported between health, housing, or relationship stress and AL (Gallo et al., 2011). The purpose of this study was to examine the relationship of childhood and adulthood adversities with AL. We hypothesize that the experience of these stressors will be positively correlated with AL at midlife and older ages.

Method

Data

This analysis uses data from the first wave (2009–2011) of The Irish Longitudinal study on Ageing (TILDA), a nationally representative prospective study of over 8,175 persons aged 50+ and their spouses living in the Republic of Ireland. Data was collected through computer assisted personal interview (CAPI) carried out by a trained interviewer, a leave behind self-completion questionnaire (SCQ), and a comprehensive clinic-based health assessment (Whelan & Savva, 2013). If respondents were unable to attend the health assessment center, a trained nurse administered a subset of cognitive and physical tests in the respondent's home. Ethical approval for the study was provided by the Faculty of Health Science Research Ethics board in Trinity College Dublin, while informed consent was obtained from all respondents during data collection.

Measures

Allostatic Load

Twelve biomarkers were used to create the summary AL index encompassing four physiological systems. This includes *the cardiovascular system*: systolic blood pressure, diastolic blood pressure, resting heart rate, pulse wave velocity; *the metabolic system*: waist-hip ratio, body mass index, glycosylated hemoglobin, high-density lipoprotein, total cholesterol; *the renal system*: creatinine, Cystatin-C; and the *immune* system: C-reactive protein. Log transformations were applied to C-reactive protein and pulse wave velocity to account for right-skewed distributions, and high-density lipoprotein was reverse coded such that higher values represented increased risk. Empirically defined high-risk thresholds were distinguished based on the distribution of that biomarker in the sample, separately for men and women. Biomarkers were dichotomized at the 75th percentile of risk and summed to create an index (range = 0–12) with higher scores reflecting higher AL. This method has been shown to work as well as alternative scoring algorithms (McLoughlin et al., 2020).

Psychosocial Stressors

Items adapted from the lifetime trauma list and administered as part of a self-completion questionnaire (SCQ; Krause et al., 2004) were used to retrospectively measure adversities experienced in childhood (i.e., parental alcohol/drug issues, physical abuse, and sexual abuse) and adulthood (i.e., experience of a fire/flood or natural disaster, spouse/child drug addiction, serious assault, personal life threatening illness, spouse/partner/child life threatening illness, and death of a child). These variables were coded "no/yes." In addition, whether a participant experienced the death of a parent in childhood was derived from dates provided during the CAPI, and a subjectively measured item on the economic circumstances of the household prior to the age of 14 captured childhood poverty (not poor/poor). Details on the measurement of these variables is available in the online supplemental materials. Cumulative counts were calculated by summing the number of adversities reported: childhood events (0–5), adulthood events (0–6), and overall exposure (0–11).

Analytical Approach

To reduce any potential bias arising from item nonresponse, we imputed for individuals missing on any of the individual adversity components and for whom we had an AL score. As the outcome variable (AL) was zero inflated and over dispersed (M = 2.9, variance = 4.3), we modeled the association of each adversity type with AL using negative binomial regression. Unstandardized coefficients were exponentiated and reported as incidence rate ratios (IRR), which can be interpreted as the percentage change in AL associated with the experience of each adversity type. In the base models we adjust for age and sex (Model 1) as the experience of adversity may differ between age cohorts and sexes. In secondary models we adjust for sociodemographic and lifestyle factors using hierarchical entry of covariates. Even though it is not possible to unequivocally establish relative timing for most of these variables as they were measured contemporaneously, it might still be informative to examine the degree to which their common correlation attenuates the association of adversity with AL in an exploratory way to help inform future hypothesis generation. These covariates are described in detail in the online supplemental materials. Model 2 added educational attainment (primary, secondary, tertiary); Model 3 added smoking pack years, problematic alcohol consumption (four-item CAGE), and physical activity (eight-item International Physical Activity Questionnaire; low, medium, high); Model 4 added log total household income (after tax); and Model 5 added social connectedness (four-item Berkman Syme Social Network Index [SNI]). All analyses were conducted in Stata v.15 (StataCorp, 2017), using TILDA dataset v.1.8.0.

Missing Cases

The initial case base for the analysis consisted of 6,914 respondents (84.6%) who returned the SCQ. Further exclusions included 2,248 respondents who did not take part in the nurse led health assessment, and 501 missing on one or more of the AL biomarkers. Full biomarker data was available for 4,165 participants, forming the analytical sample (Supplementary Figure 1). Standard errors of estimates were adjusted to account for the clustered design effect and stratification, and data were weighted using survey weights to account for the fact that respondents who attended the health center were younger, better educated, and in better health. Multiple imputation by chained equations was conducted to estimate missing values based on observed information, assuming all data were missing at random (Little's test: $\chi^2(6{,}812)$, N = 4,165, 2974.3, p = 1.00). The percentage of cases for which we imputed on any individual adversity ranged from .1% to 5.1% (Supplementary Table 2). The imputation model included all variables used in the final analysis, including sociodemographic and lifestyle variables. 20 data sets were imputed with random seed to facilitate replication.

Results

Supplementary Table 1 reports the sample characteristics; mean age of participants was 63.3 years (*SD* = 9.3), and 48.2% were male. Average AL was 3.1 (*SD* = 2.1). Supplementary Table 2 reports the number of cases and survey weighted prevalence for each adversity type. Figure 1A displays the age and sex adjusted independent association of each adversity type with AL, while Figure 1B reports the full multivariable adjusted estimates. Controlling for age and sex (Supplementary Table 3, Model 1), participants who reported childhood poverty had significantly higher AL burden (IRR= 1.10, 95% CI [1.04, 1.16]; p = .001), as did those who reported childhood physical abuse (IRR= 1.10, 95% CI [1.01, 1.18]; p = .021). Participants who reported having an ill spouse or child had a 6% higher AL burden (IRR= 1.06, 95% CI [1.01, 1.11]; p = .026). No other individual adversity was significantly associated with AL in the base models. The cumulative count of psychosocial adversities was associated with a 3% increase in AL burden (IRR= 1.03, 95% CI [1.01, 1.04]; p = .003).

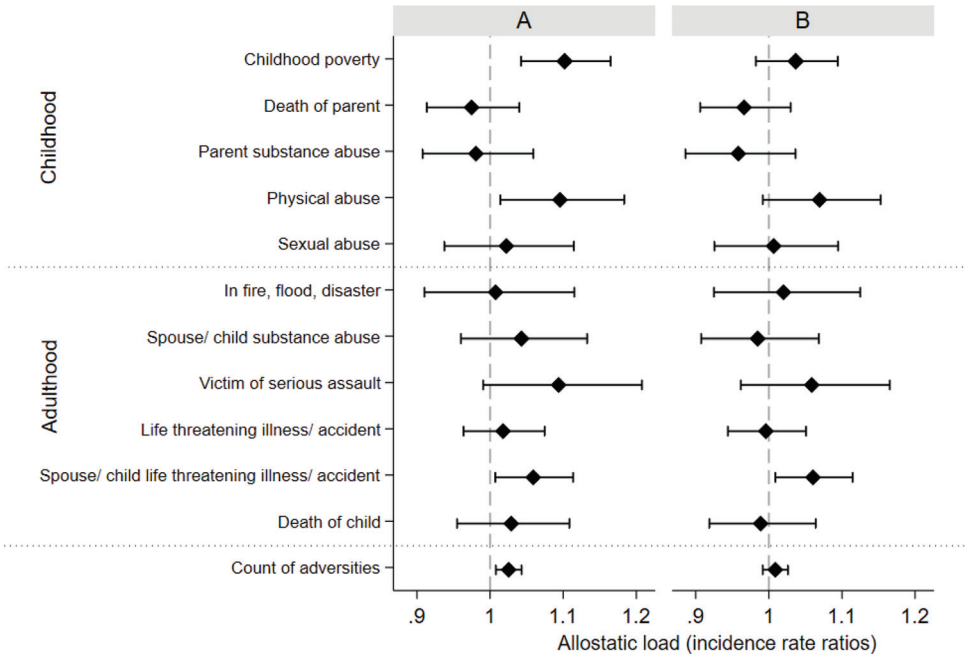
Supplementary Table 4 displays the relationships of the exposures with the covariates, and the relationships of the covariates with the outcome. The inclusion of all covariates rendered the association of childhood poverty (IRR= 1.04, 95% CI [.98, 1.09]; p = .190) and childhood physical abuse (IRR= 1.07, 95% CI [.99, 1.15]; p = .081) with AL burden nonsignificant, while the association of having an ill spouse/partner/child with AL persisted (IRR= 1.06, 95% CI [1.01, 1.11]; p = .021; Supplementary Table 3, Model 5). The count of adversities was not significantly associated with AL burden after multivariable adjustment (IRR: 1.01, 95% CI [.99, 1.03]; p = .313). As a sensitivity check, we repeated the analyses using clinical cut-points (Supplementary Table 5) and on nonimputed data (Supplementary Table 6) but the broad pattern of results were not appreciably different. However, when we included measures of parasympathetic functioning which were available for a subset of participants (n = 3,672), being a victim of serious assault was now associated with higher AL burden in the base, but not the multivariable adjusted models, while the relationship of spouse/partner/child life threatening illness with AL burden was nonsignificant, possibly due to lower statistical power in this reduced sample (Supplementary Table 7).

Discussion

This study examined the association of a number of childhood and adulthood adversities with AL in a large, community-based sample of older Irish adults. The experience of adversity is common throughout life, with 57.9% experiencing at least one adversity; however, only three out of 11 adversities (childhood poverty, childhood physical abuse, spouse/partner/child life-threatening illness or accident) were associated with higher AL, albeit merely a select few from the spectrum of adversities that could be experienced during a lifetime. These findings elicit the question as to why we observe linkages between some adversity types and higher AL burden but not others. Although the majority of associations were in the positive direction, it could be conceded that these null findings with the majority of adversities and AL may result from crude measurement of the exposure (never vs. ever), which fails to capture variation in the timing, duration or severity of the event.

Figure 1

Independent Association of Each Diversity Type With Allostatic Load in the Minimally (Figure 1A) and Full Multivariable (Figure 1B) Adjusted Models



Note. Model A adjusts for age and sex. Model B = Model A + education, lifestyle factors (alcohol abuse, smoking pack years, and physical activity), income, social network index.

Unfortunately, this information is not available in TILDA, but it would be expected to moderate the impact of the stressor on AL burden. Moreover, the lack of association between many of the adulthood adversities and AL may arise because these exposures occur "more proximal" in time to the measurement of AL, and the biological consequences of exposure may only appear with a lag. In accordance with the simplistic "multiples matter" paradigm (Spratt, 2012); cumulative adversity was positivity associated with greater physiological dysregulation. However, the addition of a number of sociodemographic and behavioral factors attenuated the relationship of childhood poverty, childhood physical abuse and cumulative adversity with AL burden, implicating potential pathways whereby exposure to psychosocial adversity becomes biologically embedded. Strengths include the large nationally representative sample of the community-dwelling older population of Ireland, and the range of different measures of adversity in childhood and adulthood. Limitations include the fact that the adverse events were retrospectively reported and subject to recall bias. The AL measure may be less sensitive for capturing the direct effects of HPA-axis and SAM axis dysregulation as it does not include measures of the "primary mediators" (e.g., cortisol, epinephrine, norepinephrine; McEwen & Seeman, 1999).

Conclusion

Overall, this study provides rather limited support for the idea that psychosocial stress causes physiological dysregulation, at least as indexed using the measure of AL as defined in this study. Future work should seek to broaden the variety and type of adverse events examined in the context of AL and go beyond simple dichotomies of ever versus never exposed to develop a more nuanced understanding of how timing, duration and severity impact upon one's biological health in the long run.

References

Barboza Solís, C., Kelly-Irving, M., Fantin, R., Darnaudéry, M., Torrisani, J., Lang, T., & Delpierre, C. (2015). Adverse childhood experiences and physiological wear-and-tear in midlife: Findings from the 1958 British birth cohort. *Proceedings of the National Academy of Sciences of the United States of America*, *112*(7), E738–E746. https://doi.org/10.1073/pnas.1417325112

Berkman, L. F., & Syme, S. L. (1979). Social networks, host resistance, and mortality: A nine-year follow-up study of Alameda County residents. *American Journal of Epidemiology*, *109*(2), 186–204. https://doi.org/10.1093/oxfordjournals.aje.a112674

Bernadt, M. W., Mumford, J., Taylor, C., Smith, B., & Murray, R. M. (1982). Comparison of questionnaire and laboratory tests in the detection of excessive drinking and alcoholism. *Lancet*, *1*(8267), 325–328. https://doi.org/10.1016/S0140-6736(82)91579-3

Cohen, S., Janicki-Deverts, D., & Miller, G. E. (2007). Psychological stress and disease. *Journal of the American Medical Association*, *298*(14), 1685–1687. https://doi.org/10.1001/jama.298.14.1685

Craig, C. L., Marshall, A. L., Sjostrom, M., Bauman, A. E., Booth, M. L., Ainsworth, B. E., Pratt, M., Ekelund, U., Yngve, A., Sallis, J. F., & Oja, P. (2003). International physical activity questionnaire: 12-country reliability and validity. *Medicine and Science in Sports and Exercise*, *35*(8), 1381–1395. https://doi.org/10.1249/01.Mss.0000078924.61453.Fb

Dich, N., Hansen, Å. M., Avlund, K., Lund, R., Mortensen, E. L., Bruunsgaard, H., & Rod, N. H. (2015). Early life adversity potentiates the effects of later life stress on cumulative physiological dysregulation. *Anxiety, Stress, and Coping*, *28*(4), 372–390. https://doi.org/10.1080/10615806.2014.969720

Ewing, J. A. (1984). Detecting alcoholism. The CAGE questionnaire. *Journal of the American Medical Association*, *252*(14), 1905–1907. https://doi.org/10.1001/jama.1984.03350140051025

Friedman, E. M., Karlamangla, A. S., Gruenewald, T. L., Koretz, B., & Seeman, T. E. (2015). Early life adversity and adult biological risk profiles. *Psychosomatic Medicine*, *77*(2), 176–185. https://doi.org/10.1097/PSY.0000000000000147

Gallo, L. C., Jiménez, J. A., Shivpuri, S., Espinosa de los Monteros, K., & Mills, P. J. (2011). Domains of chronic stress, lifestyle factors, and allostatic load in middle-aged Mexican-American women. *Annals of Behavioral Medicine: A Publication of the Society of Behavioral Medicine*, *41*(1), 21–31. https://doi.org/10.1007/s12160-010-9233-1

Glei, D. A., Goldman, N., Chuang, Y. L., & Weinstein, M. (2007). Do chronic stressors lead to physiological dysregulation? Testing the theory of allostatic load. *Psychosomatic Medicine*, *69*(8), 769–776. https://doi.org/10.1097/PSY.0b013e318157cba6

Krause, N., Shaw, B. A., & Cairney, J. (2004). A descriptive epidemiology of lifetime trauma and the physical health status of older adults. *Psychology and Aging*, *19*(4), 637–648. https://doi.org/10.1037/0882-7974.19.4.637

Mayfield, D., McLeod, G., & Hall, P. (1974). The CAGE questionnaire: Validation of a new alcoholism screening instrument. *The American Journal of Psychiatry*, *131*(10), 1121–1123. https://doi.org/10.1176/ajp.131.10.1121

McEwen, B. S., & Seeman, T. (1999). Protective and damaging effects of mediators of stress. Elaborating and testing the concepts of allostasis and allostatic load. *Proceedings of the National Academy of Sciences of the United States of America*, *896*(1), 30–47. https://doi.org/10.1111/j.1749-6632.1999.tb08103.x

McLoughlin, S., Kenny, R. A., & McCrory, C. (2020). Does the choice of Allostatic Load scoring algorithm matter for predicting age-related health outcomes? *Psychoneuroendocrinology*, *120*, 104789. https://doi.org/10.1016/j.psyneuen.2020.104789

Spratt, T. (2012). Why multiples matter: Reconceptualising the population referred to child and family social workers. *British Journal of Social Work*, *42*(8), 1574–1591. https://doi.org/10.1093/bjsw/bcr165

StataCorp. (2017). *Stata statistical software: Release 15*. StataCorp LLC.

Weinstein, M., Goldman, N., Hedley, A., Yu-Hsuan, L., & Seeman, T. (2003). Social linkages to biological markers of health among the elderly. *Journal of Biosocial Science*, *35*(3), 433–453. https://doi.org/10.1017/S0021932003004334

Whelan, B. J., & Savva, G. M. (2013). Design and methodology of the Irish Longitudinal Study on Ageing. *Journal of the American Geriatrics Society*, *61*(Suppl. 2), S265–S268. https://doi.org/10.1111/jgs.12199

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