Population Research and Policy Review
https://doi.org/10.1007/s11113-019-09534-1

RESEARCH BRIEFS

Parental Status and Biological Functioning: Findings from the Nashville Stress and Health Study

Reed T. DeAngelis[1] · **John Taylor**[2]

Received: 3 April 2019 / Accepted: 5 June 2019

Abstract

Does childrearing affect the biological functioning of parents? To address this question, we analyze cross-sectional survey and biomarker data from Vanderbilt University's Nashville Stress and Health Study, a probability sample of non-Hispanic White and Black working-age adults from Davidson County, Tennessee (2011–2014; $n = 1252$). Multivariable regression analyses reveal a linear dose–response relationship between the number of children living in a respondent's home and (a) increased allostatic load, and (b) decreased leukocyte telomere length. We found no differences in biological functioning between childless respondents and empty-nest parents. These findings also withstood controls for a battery of socioeconomic factors. The implications of these findings and suggestions for future research are discussed.

Keywords Childrearing · Parental health · Biological functioning · Allostatic load · Telomere length

Introduction

Research on the relationship between parenting and the physical health of parents is limited in scale and scope. To date, most research into the health burdens of parenting have focused on mental health outcomes or on the caregiving demands of parenting children with special needs. There are at least two reasons why the physical health outcomes of parenting have been neglected by population researchers. First, active parenting usually occurs at a stage in the life course that precedes the onset of easily detectable chronic illnesses. Second, until recently population researchers have lacked access to biological data subtle enough to detect presymptomatic

✉ John Taylor
jrtaylor@fsu.edu

[1] University of North Carolina at Chapel Hill, Chapel Hill, USA

[2] Department of Sociology, Florida State University, 526 Bellamy Building, Tallahassee, FL 32306-2270, USA

Published online: 12 June 2019

stages of health disorders. Thus, the question of whether and to what extent parenting increases risk for physical health problems has largely gone unanswered in the population health literature.

The present study addresses this gap. Specifically, we analyze data from Vanderbilt University's Nashville Stress and Health Study to assess the relationship between parental status and two dimensions of biological functioning: allostatic load (AL) and leukocyte telomere length (LTL). Allostatic load represents an index of biomarkers that has been shown to respond to the physiological wear and tear resulting from exposure to environmental stressors (Justin et al. 2010). Telomeres are considered biological indicators of cellular aging that reduce in length over the life course as a natural function of recurring cell division. However, studies have shown that chronic stress exposure can prematurely shorten the length of telomeres and lead to early senescence (Epel et al. 2004). We therefore test the hypothesis that the burdens of childrearing will translate into higher levels of AL and diminished LTL.

Methods

Data

We analyzed survey and biomarker data from Vanderbilt University's Nashville Stress and Health Study (NSAHS). The NSAHS is a probability sample of non-Hispanic White and Black working-age adults from Davidson County, Tennessee. Survey Sampling International produced a random sample of 199 block groups in Davidson County. To gather an adequate sample of Black households, block groups were stratified by the percentage of Black residents according to 2010 Census data. The final sampling frame consisted of 2400 randomly sampled households of which 2065 were contacted to participate in the study. Nearly 61% of contacted households eventually agreed to participate, resulting in a sample of 1252 adult residents. Interviews were computer-assisted and conducted either in the respondent's home or on Vanderbilt campus. Trained interviewers conducted the interviews and were matched with respondents based on race. The average interview lasted approximately 3 h. All respondents received $50 for their participation in the survey phase of the interview.

During the survey interview, respondents also received instructions and materials for collecting biomarkers. The morning following the survey interview, a trained clinician visited the respondent before breakfast to collect (1) a 12-hour urine sample, (2) a venous blood sample, (3) three blood pressure readings (spaced by 2-min intervals), and (4) anthropometric measurements of hip, waist, height, and weight. Respondents received an additional $50 for providing biomarkers. Due to the complex design of the NSAHS, data collection lasted from April 2011 to January 2014 or roughly 3 years.

Measures

Allostatic Load (AL)

Our AL index included the following biomarkers: (1) epinephrine and norepinephrine (sympathetic stress response); (2) dehydroepiandrosterone sulfate or DHEA-S and cortisol (parasympathetic stress response); (3) the average of three readings for systolic and diastolic blood pressure (cardiovascular); and (4) waist-to-hip ratio, total glycosylated hemoglobin, total cholesterol, and high-density lipoprotein (metabolic). Guided by prior research (Seeman et al. 1997), we calculated allostatic load scores by dummy-coding each biomarker into "high risk" quartiles and then summing each dummy-coded biomarker into a count index of allostatic load. High risk scores for DHEA-S and high-density lipoprotein were calculated by dummy-coding the bottom quartile (=1) versus top three quartiles (=0), since their depletion (rather than accretion) reflects greater allostatic load. All other biomarkers were dummy-coded into top (=1) versus bottom three quartiles (=0). Higher scores on this index indicate a greater count of high-risk biomarkers. Cutoff points and descriptive statistics for each biomarker are listed in "Appendix 1".

Leukocyte Telomere Length (LTL)

LTL was measured as the ratio of telomeric DNA (T) to a single-copy sequence (T/S ratio) via monochrome multiplex quantitative polymerase chain reaction (MMQ-PCR) method with albumin as the single-copy reference sequence. Scores were coded such that higher values indicated longer telomeres (i.e., less cellular aging). For detailed information on the collection and measurement of telomere data in the NSAHS, see Hill et al. (2016).

Parental Status

We operationalized parental status in two ways. First, we measured the number of children currently living in the respondent's home. Second, we created a categorical variable with the following groups: childless (reference), empty-nest parent, one child in the home, and multiple children in the home. Including this second operationalization allowed us to relax assumptions of linearity in the relationship between children and parental health, as well as to determine whether the biological health of empty-nest parents differed from respondents who were childless or currently rearing children.

Control Variables

Models controlled for age (in years), race–ethnicity (1=Black, 0=White), gender (1=female, 0=male), marital status (1=married/cohabiting, 0=single), and educational attainment (1=college or greater, 0=less than college). We also controlled for two additional dimensions of socioeconomic status with known implications for health and well-being. First, we controlled for a mean index of financial resources

comprised of ordinal measures of household income, value of home, and liquid assets ($\alpha = .80$). We standardized each measure before creating the index.

Second, we controlled for a mean index of occupational self-direction or autonomy (Kohn and Schooler 1983). Respondents were asked how often at work (1) they made decisions on their own, (2) they controlled the speed at which they worked, (3) their supervisor decided what they did and how they did it (reverse-scored), (4) they had a lot of freedom to decide how to do their work, and (5) they did the same thing over and over again (reverse-scored). Response categories ranged from "never" (= 1) to "almost always" (= 4). We averaged responses to each item ($\alpha = .60$) before collapsing index scores into the following categories: low occupational autonomy (i.e., average score of 1–3.2), high occupational autonomy (i.e., average score of 3.4–4), and unemployed. Low occupational autonomy serves as the reference category in our multivariable analyses.

Analytic Strategies

All statistical analyses were conducted in Stata 14. We treated the AL index as an over-dispersed count variable (mean = 2.29, variance = 2.89) and estimated scores with negative binomial regression techniques. LTL was estimated with ordinary least squares (OLS) regression techniques. In ancillary analyses, we also estimated AL scores with Poisson and OLS regression techniques. Findings were comparable regardless of the technique used. All of our regression estimates adjusted for post-stratification weighting and clustering at the census block group level.

Finally, the following variables had missing data: AL ($n = 30$), LTL ($n = 144$), educational attainment ($n = 1$), and financial resources ($n = 44$). We followed the advice of Johnson and Young (2011) and replaced missing values for education and financial resources with 25 iterations of multiple imputation by chained equation. Missing data on AL and LTL were handled with listwise deletion. Main findings were substantively identical before and after imputation.

Results

Table 1 presents weighted descriptive statistics of study variables. The average respondent was 44 years old, had approximately one child in the home, and exhibited roughly two high-risk biomarkers at the time of the interview. Twenty-four percent of respondents were empty-nest parents and thirty percent were childless. Additional descriptive statistics are reported in Table 1.

Table 2 reports incidence rate ratios (IRR) predicting the number of high-risk biomarkers in two sets of estimates. The first set of estimates ("Model 1") tests the association between parental status and AL while holding control variables constant. The second set of estimates ("Model 2") tests the association between the number of children in the home and AL while holding control variables constant. An IRR > 1 indicates a positive association while an IRR < 1 indicates a negative association. Model 1 shows that parents with multiple children in the home had a 21% greater

Table 1 Weighted descriptive statistics: Nashville Stress and Health Study ($n = 1252$)

	Mean	SD	Range
Allostatic load	2.29	1.70	0–8
Leukocyte telomere length (T/S ratio)	0.71	0.21	0.25–2.10
Parental status			
Childless (reference)	0.30	0.46	0–1
Empty-nest parent	0.24	0.43	0–1
One child in home	0.23	0.42	0–1
Multiple children in home	0.22	0.42	0–1
Number of children in home	0.79	1.08	0–8
Age (in years)	44.32	11.72	22–69
Black (vs. White)	0.28	0.45	0–1
Female (vs. male)	0.52	0.50	0–1
Married (vs. single)	0.57	0.49	0–1
College (vs. less than college)	0.42	0.49	0–1
Financial resources	0.17	0.84	−1.87 to 2.22
Occupational status			
Low occupational autonomy (reference)	0.58	0.49	0–1
High occupational autonomy	0.19	0.39	0–1
Unemployed	0.24	0.43	0–1

Table 2 Negative binomial regression estimates of the number of high-risk biomarkers ($n = 1222$)

	Model 1			Model 2		
	IRR	95% CI	*p* value	IRR	95% CI	*p* value
Parental status (ref. = childless)						
Empty-nest parent	1.031	[0.901–1.179]	0.661		↓	
One child in home	0.985	[0.860–1.127]	0.823		↓	
Multiple children in home	1.206	[1.057–1.376]	0.006		↓	
Number of children in home		↓		1.068	[1.025–1.111]	0.002
Age	1.016	[1.011–1.021]	0.000	1.017	[1.012–1.021]	0.000
Black	1.251	[1.144–1.368]	0.000	1.253	[1.144–1.374]	0.000
Female	0.688	[0.620–0.764]	0.000	0.688	[0.620–0.763]	0.000
Married	1.012	[0.927–1.104]	0.795	1.008	[0.922–1.103]	0.856
College	0.805	[0.722–0.897]	0.000	0.805	[0.723–0.896]	0.000
Financial resources	0.912	[0.840–0.990]	0.028	0.911	[0.840–0.989]	0.027
Occupational status (ref. = low occupational autonomy)						
High occupational autonomy	0.987	[0.866–1.126]	0.850	0.983	[0.861–1.121]	0.794
Unemployed	0.950	[0.844–1.070]	0.398	0.947	[0.841–1.067]	0.371
Constant	1.311	[1.027–1.674]	0.030	1.269	[0.995–1.617]	0.055

Incidence rate ratios (IRR) reported with 95% confidence intervals and two-tailed *p* values. Estimates are adjusted for probability weighting and clustering at the census block group level

rate of high-risk biomarkers relative to their childless peers, net of control variables (IRR = 1.206; $p < .01$). Model 2 further shows that each additional child in the home was associated with an average increase of 7% in the rate of high-risk biomarkers, net of controls (IRR = 1.068; $p < .01$).

Table 3 reports unstandardized coefficients from OLS regression estimates of LTL (T/S ratio). Model 1 shows that respondents with multiple children in the home had an average T/S ratio that was 0.062 units lower than their childless counterparts, holding control variables constant ($b = -0.062$; $p < .01$). Model 2 demonstrates that each additional child in the home was associated with an average decrease in T/S ratio of 0.020, holding control variables constant ($b = -0.020$; $p < .01$).

Figures 1 and 2 visually depict the associations of parental status with AL and LTL, respectively. As Fig. 1 illustrates, the average count of high-risk biomarkers for respondents with multiple children in the home was 2.63, whereas the same count was 2.18 for childless respondents. Similarly, Fig. 2 demonstrates that respondents with multiple children in the home had the shortest telomeres (T/S ratio = 0.67) relative to childless respondents.

Conclusion

To our knowledge, this is the first study to examine whether childrearing associates with AL and LTL, and one of only a handful of studies that has assessed the relationship between parental status and biological functioning within a population-based

Table 3 Ordinary least squares (OLS) regression estimates of leukocyte telomere length ($n = 1108$)

	Model 1			Model 2		
	b	s.e.	*p* value	*b*	s.e.	*p* value
Parental status (ref. = childless)						
Empty-nest parent	0.001	(0.020)	0.962		↓	
One child in home	−0.031	(0.023)	0.176		↓	
Multiple children in home	−0.062	(0.019)	0.001		↓	
Number of children in home				−0.020	(0.007)	0.005
Age	−0.006	(0.001)	0.000	−0.006	(0.001)	0.000
Black	0.050	(0.015)	0.001	0.049	(0.015)	0.001
Female	0.025	(0.015)	0.105	0.025	(0.015)	0.105
Married	0.012	(0.017)	0.479	0.010	(0.017)	0.572
College	0.040	(0.018)	0.027	0.040	(0.017)	0.021
Financial resources	0.007	(0.011)	0.507	0.006	(0.011)	0.582
Occupational status (ref. = low occupational autonomy)						
High occupational autonomy	−0.008	(0.020)	0.673	−0.007	(0.020)	0.714
Unemployed	0.019	(0.027)	0.484	0.020	(0.027)	0.461
Constant	0.943	(0.038)	0.000	0.934	(0.037)	0.000

Unstandardized coefficients (b) reported with standard errors (s.e.) and two-tailed *p* values. Estimates are adjusted for probability weighting and clustering at the census block group level

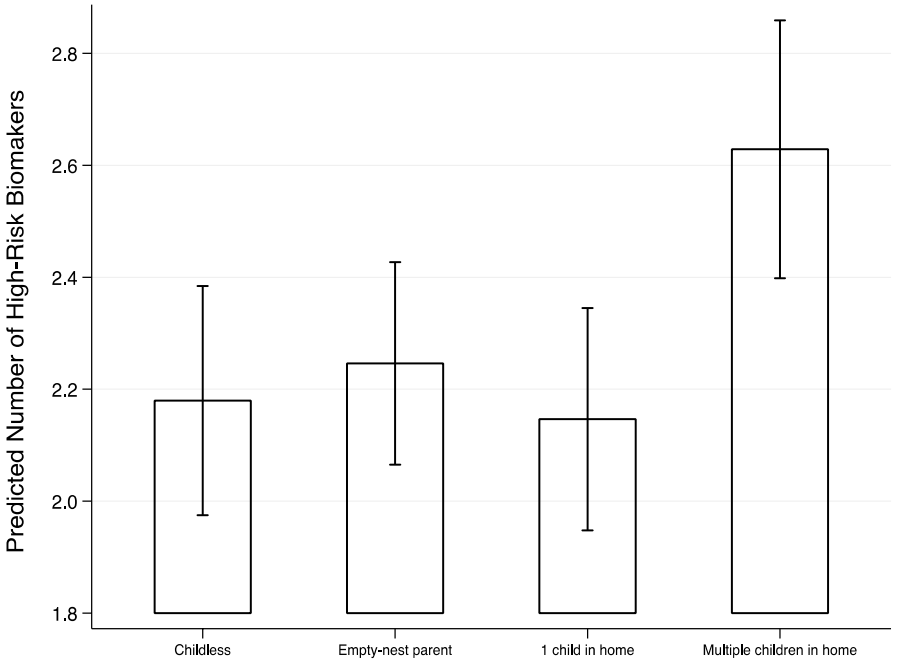

Fig. 1 Predicted number of high-risk biomarkers by parental status ($n = 1222$)

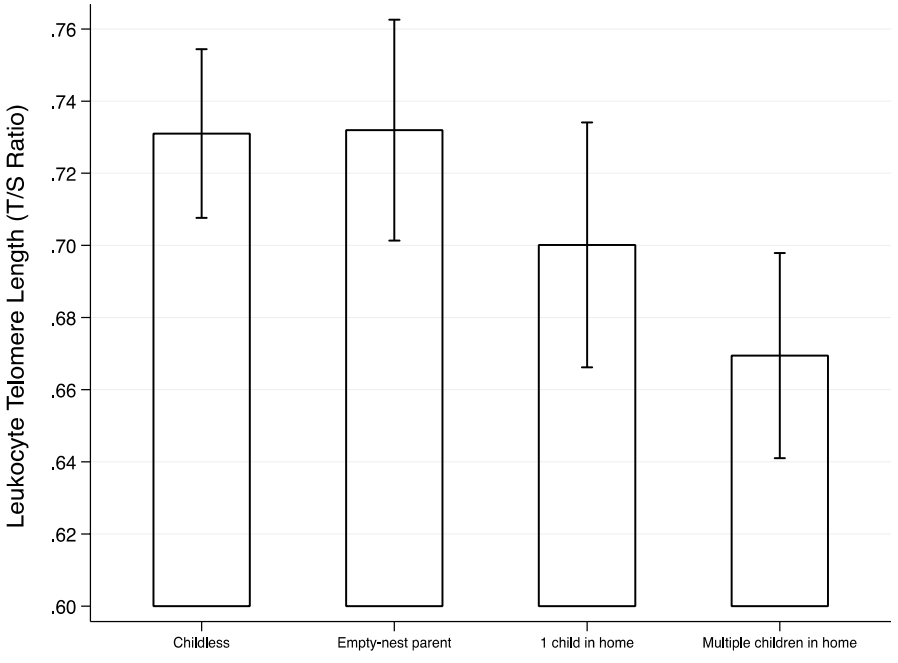

Fig. 2 Mean leukocyte telomere length by parental status ($n = 1108$)

sample. The findings presented here support the hypothesis that parental status is associated with biological dysregulation. Moreover, our multivariable regression analyses suggest that this relationship operates as a threshold effect for AL when multiple children in the home and as a linear dose response for telomere length. Importantly, we were able to identify biological dysregulation of parents in a study population that is generally younger than those who experience the onset of physical symptomatology (i.e., working-age adults).

Our study should not be interpreted as suggesting the solution to the health risks of parenting is to avoid having children altogether. Rather, we believe the current findings signal a need for an increased attentiveness to the health risks of childrearing, particularly for parents with multiple children in the home. We hope the information provided here can inform parents and their healthcare providers of the potential health risks associated with parenting. Still, we hasten to add that more scholarly work is needed to determine (a) the social and biological mechanisms by which childrearing affects parental biological functioning, and (b) whether or to what extent parents can leverage certain resources to buffer the stressors of raising children.

Indeed, although we believe the present paper expands our understanding of parenting and health, there remain several important unanswered questions that we could not address in this brief report. For example, we did not assess whether marital status, SES, race, gender, or other psychosocial resources condition the relationship between parenting and biological functioning. The NSAHS data also lacked biomarkers of immune functioning, such as c-reactive protein and interleukin 6, which are important components of the biological stress response. Future research is needed to identify how such factors inform the present research.

Two additional limitations merit discussion. First, we were unable to establish the temporal ordering between parenting and biological functioning given the cross-sectional study design of the NSAHS. Also, the sample was limited to black and white residents in Nashville. Consequently, the findings presented here may not generalize to other racial groups or metropolitan areas. We encourage additional research into the health effects of childrearing that employs longitudinal data collection with multiethnic samples.

Acknowledgements This work is supported by Grant R01AG034067 from the Office of Behavioral and Social Science Research and the National Institute on Aging. No direct support was received from Grant R01 AG034067 for this analysis.

Appendix 1

See Table 4.

Table 4 Weighted descriptive statistics of allostatic load biomarkers

Biomarker	Cutoff	Mean	SD	Range	N
Epinephrine (μg/ML)[a]	≥ .003 μg/mL	0.003	0.005	0.0001–0.1523	1159
Norepinephrine (μg/ML)[a]	≥ .033 μg/mL	0.025	0.033	0.0014–1.0823	1206
DHEA-S (μg/dL)[b]	≤ 68 μg/dL	149.181	105.474	15–868	1140
Cortisol (μg/L)[a]	≥ 13.8 μg/L	10.913	11.414	1–194	1159
Systolic BP	≥ 131	121.507	13.884	80–198	1192
Diastolic BP	≥ 83	77.599	9.243	48–119	1192
Waist-to-hip ratio	≥ 0.95	0.896	0.088	0.471–1.327	1203
HbA1C (%)[b]	≥ 5.9%	5.542	0.913	3.3–17.4	1172
Total cholesterol (mg/dL)[b]	≥ 208 mg/dL	188.170	41.045	81–395	1179
High-density lipoprotein (mg/dL)[b]	≤ 37 mg/dL	47.767	14.867	18–141	1179

Biomarkers collected by [a]urine and [b]venous blood samples

References

Epel, E. S., Blackburn, E. H., Lin, J., Dhabhar, F. S., Adler, N. E., Morrow, J. D., et al. (2004). Accelerated telomere shortening in response to life stress. *Proceedings of the National Academy of Sciences, 101*(49), 17312–17315.

Hill, T. D., Ellison, C. G., Burdette, A. M., Taylor, J., & Friedman, K. L. (2016). Dimensions of religious involvement and leukocyte telomere length. *Social Science and Medicine, 163,* 168–175.

Johnson, D. R., & Young, R. (2011). Toward best practices in analyzing datasets with missing data: Comparisons and recommendations. *Journal of Marriage and Family, 73*(5), 926–945.

Justin, R.-P., McEwen, B. S., & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience and Biobehavioral Reviews, 35,* 2–16.

Kohn, M., & Schooler, C. (1983). *Work and personality: An inquiry into the impact of social stratification*. Norwood, NJ: Ablex Publishing.

Seeman, T. E., Singer, B. H., Rowe, J. W., Horwitz, R. I., & McEwen, B. S. (1997). Price of adaptation—Allostatic load and its health consequences: MacArthur studies of successful aging. *Archives of Internal Medicine, 157*(19), 2259–2268.