International Journal of Advance and Applied Research (IJAAR)
Peer Reviewed Bi-Monthly

ISSN – 2347-7075
Impact Factor – 7.065
Vol.7 No.5 May–June 2020

THE RELATIVE CONTRIBUTIONS OF BEHAVIOURAL, BIOLOGICAL, AND PSYCHOLOGICAL RISK FACTORS IN THE ASSOCIATION BETWEEN PSYCHOSOCIAL STRESS AND ALL-CAUSE MORTALITY AMONG MIDDLE- AND OLDER-AGED ADULTS

Dr. Anurag Upadhayay

Assistant Professor,

P.G Department of Psychology, K.B.P. G College Mirzapur,

Mahatma Gandhi Kashi Vidyapith .Varanasi.

ABSTRACT:

Evidence of an association between psychosocial stress and mortality continues to accumulate. However, despite repeated calls in the literature for further examination into the physiological and behavioural pathways though which stress affects health and mortality, research on this topic remains limited. This study addresses this gap by employing a counterfactual based mediation analysis of eight behavioural, biological, and psychological pathways often hypothesized to play a role in the association between stress and health. First, we calculated the survival rate of all-cause mortality associated with cumulative psychosocial stress (high vs. low/moderate) using random effects accelerated failure time models among a sample of 710 adults from the Midlife in the United States panel study. Then, we conducted a multiple mediator mediation analysis utilizing a counterfactual regression framework to determine the relative contributions of each mediator and all mediators combined in the association between stress and mortality. Exposure to high psychosocial stress was associated with a 0.76 times reduced survival rate overthe follow-up period 2015-17, while adjusting for age, sex, race, income, education, baseline health, and study design effects. The mediators accounted for 49% of this association. In particular, smoking, sedentary behavior, obesity/BMI, and cardiovascular disease displayed significant indirect effects and accounted for the largest reductions in the total effect of stress on mortality, with natural indirect effects of 14%, 12%, 11%, and 4%, respectively. In conclusion, traditional behavioral and biological risk factors play a significant role in the association between psychosocial stress and mortality among middle and older adults in the India context.

Keywords: Adults, Psychosocial, Stress, Mortality.

INTRODUCTION:

Psychosocial stress, a familiar phenomenon to most, may lead to adverse states of health if exposure becomes excessive and/or chronic in nature. An expansive literature has been generated over the past century demonstrating associations between various measures of psychosocial stress and a multitude of mental and physical health outcomes, including mortality (1–16). However less is known about the relative contributions of the hypothesized causal pathways connecting stress to health, especially with regard to mortality (8–12, 17–20). Understanding these causal pathways, as well as their relative contributions, is important for both theory and practice. From a theoretical standpoint, investigating causal pathways aids in testing and revising existing theoretical frameworks, constructing novel theoretical frameworks, and establishing new avenues of research. From a public health perspective, a more precise understanding of causal mechanisms, especially the relative importance of individual pathways, facilitates the design of targeted interventions and efficient use of resources.

Hypothesized causal pathways the hypothesized causal pathways between psychosocial stress and physical health outcomes, including mortality, can be summarized by three main pathways.

1. Biological, (2) psychological, and (3) behavioural. The first two are often referred to as “direct” pathways, whereas the third is generally considered an “indirect” pathway. Each may operate independently or in conjunction with one another. While the physiological response to acute stress a process known as all stasiscan be beneficial in the near-term (e.g., by providing additional energy and mental acuity for effectively managing a given stressor), it can become detrimental over time through chronic exposure . Prolonged or repeated exposure to stress and the corresponding activation of the body’s stress response systems (i.e., the hypothalamic pituitary adrenal (HPA) axis and the sympathetic adrenal medullary (SAM) system) may result in biological “wear and tear “orallosteric load . As allosteric load accumulates within the body over time, stress response systems may become dysregulated, perpetuating inflammatory processes and the development of various diseases including cardiovascular disease, diabetes, inflammatory diseases, influenza, and some cancers. Exposure to chronic stress may also increase one’s risk of developing negative mental health outcomes.

PRACTICE IMPLICATIONS:

Depression, anxiety, and chronic sleep problems. Mental health conditions are in turn associated with increased risk of adverse physical health conditions and mortality. Physical health outcomes, including mortality, may also be influenced indirectly through the adoption of unhealthy coping behaviour's, such as alcohol consumption, cigarette smoking, substance use, unhealthy diet, and sedentary activity (14, 22, 40–53). Existing evidence on hypothesized causal pathways Biological pathway Stress has been linked to biological, psychological, and behavioural measures through a number of experimental and observational studies (1–4, 20, 27–30, 54–56). Experimental studies have been especially important for studying the complex sequence of biological changes that occur during and immediately following stress exposure. For example, a review of experimental stress test studies found that exposure to an acute stressor was associated with activation of the HPA axis (as measured by cortisol, adrenocorticotropic hormone, vasopressin, and dehydroepiandrosterone), activation of the sympathetic-adrenal-medullary system (measured by adrenaline and noradrenaline), changes in immune activity (observed through increased release and concentration of pro-inflammatory markers) (including IL-6, lymphocytes, monocytes, neutrophils, basophils, granulocytes, T cells, T helper cells, and natural killer cells), cardiovascular stimulation (heart rate peak and variability), changes in mood (reduced calmness, increased anxiety), and cognitive perturbations (memory, task-switching, dual task performance, cognitive flexibility, creativity, choice between present and future rewards, answering questions requiring estimation, strategizing, making decisions involving risk, and assessing risk-taking behaviour. However, it is also worth noting that there may be considerable variation in reactivity to such experimental stress tests, with studies observing heterogeneities by age, gender, personality, health, genetics, culture, and other factors (55–60). Psychological pathway Experimental and observational studies have also demonstrated associations between stress and mental health. For example, massive meta-analyses of published and unpublished European cohort studies with over 100,000 participants found that job strain, job insecurity, and unemployment were each associated with a 20–30% increased risk of depressive

Behavioural pathway lastly, a number of studies have also shown correlations between stress and various health-related behaviour's (14, 20–22, 40–51, 66–69). Psychosocial stress may influence health-related behaviour's through a number of possible mechanisms. For example, some behaviour's, such

as licit or illicit substance use (including alcohol and tobacco) or consuming unhealthy foods (e.g., calorie-rich "comfort foods"), may act as coping mechanisms that aim to placate the pain and discomfort associated with experiences of stress. Exposure to stress may also affect behaviour through alterations of the biological systems described above. For example, studies have shown that chronic stress-induced dysregulation of the HPA axis can increase appetite-related hormones (e.g., leptin and ghrelin), promote acquisition of food reward, escalate intake of high fat diets, stimulate compulsive food seeking of palatable foods, and promote reward-dependent habits—ultimately leading to increased consumption of energy-dense calories and substance use including alcohol and tobacco (67–73). Increased consumption of drugs or high fat diets may then lead to an increased sensitization of reward pathways through alterations of corticotrophin-releasing factor, glucocorticoids, and noradrenergic activity, thereby perpetuating a feedback loop marked by craving and consumption of addictive substances and high fat foods.

METHOD:

Outcome measure: all-cause mortality All-cause mortality data for this study was obtained as part of MIDUS 2 and MIDUS 3 projects, who originally collected mortality data from National Death Index reports and household proxy reports. Year of death covered the entire study period from 1995 to 2015. Survival time was calculated as the difference between the month of death or last completed survey and the month in which the baseline survey was completed (1995– 1996). Participants who were still alive at the end of wave 3 (censored observations) had survival times equal to the length of follow-up (a maximum of 240 months). Explanatory variable: cumulative psychosocial stress In order to construct a robust, multi-domain measure of psychosocial stress, we constructed an eight-domain cumulative stress measure based on the scale used by Slopen et al. . The cumulative psychosocial stress score was calculated as the standardized sum of eight standardized stress domains, each composed of a sum of a varying number of stress-related subscales, which were themselves the sum of a varying number of individual survey questions. The eight subdomains of stress included relationship stress, financial stress, work stress, work- family spill over, perceived inequality, neighbourhood stress, discrimination, and past-year family problems. Relationship stress consisted of four measures: family strain, friend strain, marital risk, and spouse/ partner strain. Financial stress was composed of four individual survey items: current financial situation

Hypothesized mediators Smoking was inferred from a single, self-report question: “How many cigarettes do you smoke per day”, where non-smokers and ex-smokers received values of 0. Cigarettes per day were then standardized to facilitate comparison with other mediators. Alcohol consumption Participants were asked about their alcohol consumption behaviour on the self-administration portion of the baseline survey via the following questions: “During the year you drank most, about how many drinks would you usually have on the days that you drank?” and “Think about the one year in your life when you drank most. During that year, how often did you TYPICALLY had [sic] at least one drink?” The first question yielded continuous responses, while the second question prompted respondents to select one of the following options: “every day”, “5 or 6 days a week”, “3 or 4 days a week”, “1 or 2 days a week”, “less than one day a week”, or “never drink”. These responses were re-coded to 7, 5.5, 3.5, 1.5, 0.5, and 0, respectively. The re-coded alcohol frequency question was then multiplied by the original alcohol amount/day question and again by 52 to yield the total number of drinks per year. This variable was then standardized in order to enable comparison with other mediators.

Depressive symptoms Participants were asked the following eight, binary questions to ascertain whether they were currently experiencing depressive symptoms disorders: “During the past 12 months, was there ever a time when you felt sad, blue, or depressed for two weeks or more in a row?”, “During two weeks in past 12 months when you felt sad, blue, or depressed, did you lose interest in most things?”, “During two weeks in past 12 months when you felt sad, blue, or depressed.

Table 1 Distribution of study variables from the midlife in the united states study for the total sample and by cumulative psychosocial stress

	Total (*N*=7108)	Low stress (*N*=5352)	High stress (*N*=1756)	
	N (%) or mean (SD)	*N* (%) or mean (SD)	*N* (%) or mean (SD)	*P* value
Death	1237(17%)	951 (18%)	286 (16%)	0.1660
Age (years)	46.41(13)	47.5 (13.12)	43.13 (12.06)	<0.001
Sex				
Men	3439 (48%)	2662 (50%)	777 (44%)	<0.001
Women	3669 (52%)	2669 (50%)	1000 (56%)	
Race				
White	6358 (89%)	4911 (92%)	1447 (81%)	<0.001
Black	421 (6%)	230 (4%)	191 (11%)	
Other	329 (5%)	190 (4%)	139 (8%)	
Individual income (dollars)				
<$15,000	2848 (40%)	2048 (38%)	800 (45%)	<0.001
$15,001–$30,000	1220 (17%)	865 (16%)	355 (20%)	
$30,001–$50,000	1428 (20%)	1061 (20%)	367 (21%)	
>$50,000	1612 (23%)	1358 (26%)	254 (14%)	
Participant's highest education				<0.001
<HS	681 (10%)	464 (9%)	217 (12%)	
HS or GED	2060 (29%)	1547 (29%)	518 (30%)	
Some college	2173 (31%)	1592 (30%)	588 (34%)	
≥College graduate	2181 (31%)	1749 (33%)	433 (25%)	
Self-reported health				<0.001
Poor	73 (1%)	48 (1%)	25 (1%)	
Fair	169 (2%)	107 (2%)	62 (4%)	
Good	782 (11%)	532 (10%)	252 (14%)	
Very good	1951 (28%)	1421 (27%)	534 (30%)	
Excellent	4126 (58%)	3244 (61%)	883 (50%)	
Cigarette smoking				

RESULTS DESCRIPTIVE STATISTICS:

The analytic sample used in this study consisted of 7108 participants who, at baseline, were an average of 46 years old (range: 20–75), 52% female, 89% white, and predominantly of middle-class backgrounds with a median individual income of between $20,000 and 25,000 per year in 1995 dollars, and 90% having a high school degree or more education (Table 1). One thousand seven hundred seventy-one (25%) participants were classified as high stress, while 5331 (75%) were low to moderate stress. Those in the higher stress group tended to be younger, were more likely to be women, were less likely to be white, had lower individual incomes, and were more likely to report worse physical health at baseline. Furthermore, those who reported greater stress were more likely to engage in unhealthy behaviour's and experience adverse states of mental and physical health. There also tended to be greater variance in measures of health behaviour's and adverse states of physical and mental health in the high-stress group compared to the low/moderate stress group above (Table 1).

Table 2 Associations between standardized behavioral, biological, and psychological risk factors and all-cause mortality ($n = 7108$)[a]

Risk factors (standard deviations)	Survival rate (95%CI)
Cigarette smoking	0.78 (0.74, 0.82)***
Alcohol consumption	0.95 (0.90, 0.99)**
Physical inactivity	0.87 (0.85, 0.89)***
Substance use	0.94 (0.89, 0.99)*
Cardiovascular symptoms scale	0.90 (0.86, 0.95)***
WHR-adjusted BMI	0.88 (0.81, 0.94)***
Depressive symptoms scale	0.94 (0.89, 1.00)†
Sleep problems	0.95 (0.90, 0.99)*

[a] Survival rates calculated from separate accelerated failure time models adjusting for age, sex, race, income, education, self-reported health, sampling weights, and sibling clustering. † $p < 0.10$, * < 0.05, ** < 0.01, *** < 0.001

Table 3 Associations between cumulative psychosocial stress and standardized behavioral, biological, and psychological risk factors ($n = 7108$)[a]

	Smoking	Alcohol	Phys. Inactivity	Substance use	CVD [b]	BMI [b]	Depression	Sleep
Stress	β (95% CI)	β (95% CI)	β (95% CI)	β (95% CI)	β (95% CI)	β (95% CI)	β (95% CI)	β (95% CI)
High	0.15 (0.10, 0.20)	0.20 (0.14, 0.25)	0.29 (0.24, 0.34)	0.31 (0.25, 0.37)	0.18 (0.12, 0.24)	0.28 (0.23, 0.34)	0.38 (0.33, 0.44)	0.45 (0.39, 0.50)
Low	Ref.	Ref.	Ref.	Ref.	Ref.	Ref.	Ref.	Ref.
P value	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001	<0.001

[a] Parameter estimates refer to the difference in standard deviations for each risk factor comparing high vs. low stress participants, adjusting for age, sex, race, income, education, pre-existing health, sampling weights, and sibling clustering

[b] *CVD* cardiovascular disease, *BMI* body mass index

Table 4 Role of behavioral, biological, and psychological mediators in explaining the association between psychosocial stress and all-cause mortality ($n = 7108$) [a]

	Total Effect	NDE	NIE	Proportion mediated [b]
	SR (95%CI)	SR (95%CI)	SR (95%CI)	% (95%CI)
	0.78 (0.68, 0.90)***	0.87 (0.73, 1.03)		
All mediators combined				70%
Cigarette smoking			0.97 (0.96, 0.98)***	17% (10%, 23%)
WHR-adjusted BMI			0.97 (0.95, 0.99)**	17% (6%, 26%)
Physical inactivity			0.97 (0.95, 0.99)**	16% (5%, 24%)
Cardiovascular symptoms			0.99 (0.98, 1.99)*	7% (1%, 13%)
Depressive symptoms			0.99 (0.98, 1.03)	5% (−32%, 12%)
Sleep problems			0.99 (0.98, 1.04)	4% (−32%, 14%)
Substance use			0.99 (0.97, 1.02)	3% (−17%, 17%)
Alcohol consumption			1.00 (0.99, 1.01)	1% (−8%, 8%)

[a] Estimates for indirect effects derived from a single outcome model simultaneously accounting for all other mediators and eight individual mediator models. All models adjusted for age, sex, race, income, education, self-reported health, sampling weights, and sibling clustering

[b] Proportion mediated calculated from the equation ($SR_{NDE} * (SR_{NIE} - 1)/(SR_{NDE} * SR_{NIE} - 1)$). † $p < 0.10$, * < 0.05, ** < 0.01, *** < 0.001

SR survival rate, *NDE* natural direct effect, *NIE* natural indirect effect, *WHR* waist hip ratio, *BMI* body mass index

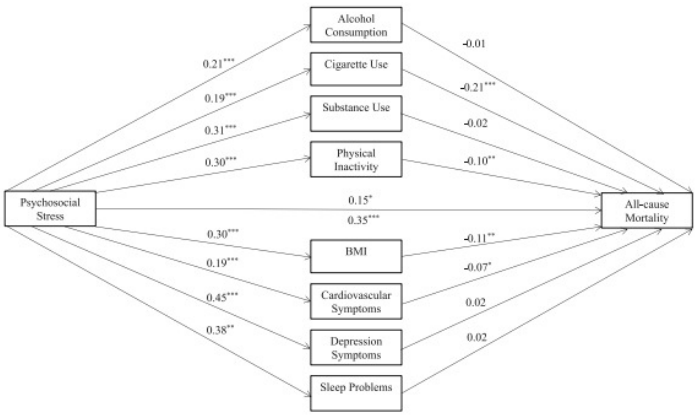

Fig. 1 Diagrammatic representation of the total, direct, and indirect effects of cumulative psychosocial stress on all-cause mortality. Standardized path coefficients estimated from regression models adjusting for age, sex, race, income, education, and self-reported health. $^{\dagger}p < 0.10$, $^{*} < 0.05$, $^{**} < 0.01$, $^{***} < 0.001$

DISCUSSION:

BROADER IMPLICATIONS FOR PUBLIC HEALTH:

The results presented in this study provide preliminary evidence on the relative magnitudes of multiple behavioural, biological, and psychological pathways connecting exposure to psychosocial stress and all-cause mortality within the US. Taken together with existing stress literature, these findings can help guide public health interventions to modulate the adverse effects of stress on population health. For example, short of eliminating stress altogether, public health practitioners may utilize results from this and similar studies to target important stress pathways (e.g., unhealthy coping behaviour's, biological processes, and psychological intermediaries) in order to limit the adverse health consequences of stress. Thus, stress prevention interventions—targeting both environmental and individual modifications—should be implemented as part of a broader public health effort including attention to the social, material, behavioural, and biological determinants of disease and mortality.

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