International Journal of Current Research and Review
DOI: http://dx.doi.org/10.31782/IJCRR.2021.SP122

Scopus

Original Research

IJCRR
Section: Healthcare
ISI Impact Factor (2019-20): 1.628
IC Value (2019): 90.81
SJIF (2020) = 7.893

Lifetime Trauma, posttraumatic stress disorder Symptoms and Early Adolescence Risk Factors for Poor Physical Health Outcome Among Malaysian Adolescents

Chen YY, Ghazali SRB

Department of Psychological Medicine, Faculty of Medicine and Health Sciences, University Malaysia Sarawak, Malaysia.

ABSTRACT

Introduction: Individuals who experienced traumatic events were more susceptible to non-communicable diseases and adoption of health-harming behaviours. Clearer understanding of the relationship between trauma and PTSD on the risk trajectories of CVD, especially among adolescents are missing in the literature.

Objective: This study investigated the associations of posttraumatic stress disorder (PTSD) manifestation and early risk factors for cardiovascular physiological measures, i.e., high blood pressure and heart rate among adolescence.

Methods: A total number of 606 adolescents aged 14-19 years old (M=16.9, SD = 1.28) to participate in the present study. Their blood pressure, heart rate and body mass index (BMI) were measured. They were asked to rate their possible trauma experiences and PTSD symptoms via questionnaires.

Results: Results showed that PTSD symptoms had a significant effect on blood pressure and heart rate reading. Total PTSD scores and only specific trauma types were significant predictors for the blood pressure and heart rate changes among adolescence.

Conclusion: It is concluded that risk factors related to reported associations between trauma, PTSD symptoms, and physiological outcomes among adults might be able to identify in adolescence suggesting early detection and intervention to reduce adverse physical health outcomes are required.

Key Words: Adolescence, Blood-pressure, Heart-rate, PTSD, Trauma

INTRODUCTION

An adverse traumatic experience may lead to Posttraumatic Stress Disorder (PTSD) diagnosis and may affect physical and mental health which persisted into adulthood.[1,2] Individuals with PTSD tend to have difficulties in their later married life; especially problems with social life.[3,4] This may due to the durable changes in human metabolic and homeostasis as well as imbalanced allostatic systems in which lead to progressive physiological damage.[5,6] Thus, individuals who experienced traumatic events were more susceptible to non-communicable diseases and adoption of health-harming behaviours. However, only for the last few decades, additional concern focused on the rising risk for adverse physical health outcomes due to potentially traumatic experiences, and the development and manifestation of PTSD.[7]

The increasing number of cardiovascular diseases (CVD) cases has become a leading health issue globally, including in Malaysia. Trauma exposure and PTSD were hypothesized as one of the risk factors for the CVD development from two different aspects - biological or behavioural alteration or pathway.[8-10] The biological alteration or pathway in explaining the relationship of traumatic exposure, PTSD and CVD involved the elevation of blood pressure and heart rate as well as other biomarkers of inflammation.[11] The behavioural alteration or pathway, on the other hand, involved individuals' health-related behaviours such as alcohol drinking, smoking, physical inactivity, being overweight or obese in which were also known as the traditional risk factors of CVD. However, there was a need for a clearer understanding of the relationship between trauma and PTSD on the risk trajectories of CVD, especially among adolescents. This would

Corresponding Author:
Chen YY, Department of Psychological Medicine, Faculty of Medicine and Health Sciences, University Malaysia Sarawak, Malaysia.
Email: yychen@gmail.com

ISSN: 2231-2196 (Print) **ISSN:** 0975-5241 (Online)

Received: 14.10.2020 **Revised:** 10.11.2020 **Accepted:** 12.12.2020 **Published:** 23.02.2021

have great benefit in understanding the aetiology and pathology of the disease for prevention and healing.

With strong consistency between trauma and/or PTSD-related structural brain research, stress served as the mediator with greater adverse health outcomes, especially cardiovascular health.[12,13] According to allostatic load theory, higher allostatic responses referred to the aggregate physiological detriment resulting from adaptation to high levels of stress. This would increase inflammation, heart rate and blood pressure, prolonged activation of hypothalamic-pituitary-adrenal (HPA) and the sympathetic nervous system (SNS), which eventually may lead to adverse physical health outcomes.[5,14] Individual's heart rate was regulated due to the SNS or the parasympathetic nervous system (PNS), and the Systolic Blood Pressure (SBP) and Diastolic Blood Pressure (DBP) as the key indicator for the cardiac physiological disturbance.[11]

Trauma exposure and/or PTSD were also associated with increased blood pressure, basal catecholamine and decreased cortisol level which ultimately leads to atherosclerosis and cardiovascular system damage.[15,16] A higher heart rate was found in women with PTSD than those without.[17] Using cardiovascular ambulatory monitoring research design, Beckham et al.[18] were able to investigate cardiovascular functioning and its relationship with PTSD among 193 women for 24 hours. Women with PTSD has a significant association with a higher ambulatory heart rate and DBP than those without PTSD, resulting in harm cardiovascular functioning. However, Beckham et al.[18] study limited the interpretation of the causal effect of PTSD and blood pressure and did not test the cardiovascular ambulatory monitoring on the male population.

The association between traumatic exposure and/or PTSD with heart rate[11] and blood pressure was found even in adolescence.[16] Blood pressure increases were due to the vasoconstriction of vessels in the blood reservoir due to the secretion of renin, angiotensin I and II which may have affected by consistent high level of stress.[16] One hundred and fifteen adolescents with a history of lifetime violence exposure showed elevated blood pressure and heart rate at both baselines and in response to laboratory stressor.[16] Another National Longitudinal Study of Adolescent Health comprised of 3,555 males and 4,416 females (aged 11 to 17 years) found the association between exposure and hypertension, and subsequently adult cardiovascular risk.[19]

In a nationally representative study of 9,699 adolescents and young adults who suffered from severe victimization and perpetration to physical intimate partner violence reported an increase in blood pressure and incidence of hypertension.[20] Suglia et al.[21] showed a significant elevation of blood pressure, which resulted in adverse cardiovascular outcomes in adulthood. One hundred and eighty-five military students participated in a highly realistic mock-captivity scenario and their blood pressure was measured before (as the baseline), directly after and 24 hours and finally, after the captivity scenario.[22] The result showed a significant elevation in SBP even 24 hours after the stressful mock training, especially for female participants. One criticism of this study was that the elevation of SBP after a stressful event in 24 hours was a normal physiological reaction of the body, thus was weak to draw a strong conclusion.

A cardiovascular physiological reactivity change (i.e., the elevation of blood pressure) occurring in traumatized individuals was found even before they developed PTSD.[15] This may provide hints for clinicians in detecting blood pressure among traumatized individuals and clinicians may treat this action as an early detection procedure of CVD. However, the statistical power of the result obtained in Gandubert et al.[15] study was weak due to its small sample of middle-aged adults. Drawing any conclusion regarding the course of the pathology processes for adverse physical health outcome was further hindered since most individuals experienced traumatic events at the age of 16 to 20.[23] Thus, the present study aimed to examine the relationship of traumatic exposure on blood pressure among adolescents (age ranged from 14 to 18) to further investigate for the relationship.

Trauma exposure and/or PTSD concerning CVD might also be explained by sympathetic over activation.[24] Overactivation of sympathetic responses resulted in an elevated heart rate which was closely associated with being overweight as well as obesity.[24-26] Being overweight and obese served as mediators of increased sodium retention in blood vessels which lead to an increase of blood volume and pressure; this increase of volume and pressure, therefore, hurts cardiovascular health.[24,25] Apart from the relationship between trauma exposure and/or PTSD with excessive weight and obesity, researchers also associated trauma exposure and/or PTSD with other unhealthy behaviours such as alcohol drinking, smoking, and sleeping less that have a significant relationship with adult cardiovascular disease.[27,28] Most of the cross-sectional and retrospective study reported that individual with trauma exposure has poorer sleep.[27]

This study collected data from adolescents across different districts of Malaysia to investigate the unresolved issues. This is not only reduced the potential recall bias that resulted in an over-or under-estimation of trauma exposure. The present study included physiological measures such as measurement of blood pressure and BMI which may provide richer data set in explaining the relationship of trauma exposure and/or PTSD in health-related behaviours.

MATERIALS AND METHODS

Participants and procedure

The study sample comprised 606 adolescents who were recruited from different states, villages and schools to participate in the

present study based on multistage sampling. Their age ranged between 14 to 19 years (M = 16.9, SD = 1.28) with 63.2% female and 36.8% male. Most of them were Malays (77.4%), followed by Iban (10.4%), Chinese (4.1%), others bumiputra (6.1%), Bidayuh (1.2%) and Indian (0.8%).

Participants' written consent for participation was obtained in which permission from a parent or legal guardian was obtained. All participants were informed about their rights, the possible risks of the study, issues of confidentiality and the procedure of data collection. All participants completed the demographic questionnaire themselves. Other information including potential traumatic events, mental and physical health history, substance use habits (i.e. alcohol and cigarette), and physical activities were gathered through a face-to-face individual interview and anthropometric measures. This study was approved by the Ethics Committees of University Malaysia Sarawak (reference letter number: UNIMAS/NC-21.02/03-02(51) and the Malaysian Ministry of Education (reference letter number: JPS(W)/SK2P/(Lat)153/08/02/05/Jld.51(41)), followed by the local District Office and Education Departments. This study was approved by the Malaysian Ministry of Health and the Malaysian National Institute of Health (reference number: NMRR-15-721-25741 (IIR).

Possible Trauma Events

Life Event Checklist for DSM-5 (LEC-5[29]) with seventeen potential traumatic or negative life events with additional three items such as near-drowning, persecution/humiliation and childhood neglect was used to investigate participant's direct or indirect exposure to the potentially traumatic events. The Cronbach's alpha for the full sample in the present study was 0.73.

Posttraumatic Stress Disorder (PTSD)

The Posttraumatic Stress Disorder Checklist for DSM-5 (PCL-5[30]) was used to screen for PTSD symptoms. Participants were asked to rate 20 items self-administered questionnaire that corresponding to the DSM-5 PTSD symptoms on a 5 Likert scale, ranging from 0 points (Not at all) to 4 (extremely). A total score was obtained and a cut-off of 33 was used to determine a participant with or without PTSD[31,32]). PCL-5 showed good internal consistency (α = .91) and test-retest reliability (r = .61).

Health-related behaviours and physiological measures

Participants were asked about their health-related behaviours using an adapted US National Health Interview Survey Checklist.[33] The questions include habit and the quantities of cigarettes smoking, alcohol drinking, sleeping hours, and a variety of physical health complaints such as back pain, hypertension, and chronic headaches. Participants who reported never had smoking habits (rated as 0), smoked one to ten cigarettes per day rated as 1, eleven to twenty cigarettes rated as 2, above 20 cigarettes as 3, and a former smoker as 4. Heavy alcohol drinker drank 5 or more drinks (more than 3000 ml for 2-5% alcohol percentage alcohol beverage) in the past 30 days. For the full sample of participants, the Cronbach's alpha of adapted US National Health Interview Survey Checklist in the present study was 0.72.

Field-testing protocol and measures

Anthropometric measurements were taken for each participant before they were asked to rest in a quiet place. They were asked to remove their shoes before testing. Their height and body mass were measured using a portable stadiometer and electronic medical scale. Blood pressure and heart rate were measured at different time points using an automatic oscillometric blood pressure unit. The participants were asked to rest in a quiet location in the school for 15 minutes with arm resting on the table. The automatic blood pressure unit took three measures: once before the participants answered any questions and have rested for 15 minutes, another measurement was taken when they were answering the trauma and PTSD questionnaires and the last one was taken before they left the room. The measurement of blood pressure and heart rate at different time points was important in examining the direct effects of traumatic events on physical reactions. Blood pressure and heart rate were only measured by using the non-dominant hand to avoid the effect of movement on blood pressure reading. All the measurements were done by a nutritionist.

Data Analysis

The IBM SPSS statistics- version 22 was used for data analysis after data checking and data cleaning. A descriptive analysis of frequency and percentage including the prevalence of trauma exposure means of socio-demographic characteristics, and BMI scores were reported. Unadjusted odds ratios (OR) with 95% confidence intervals (95% CI) were used to estimate the risk of the physical health condition, PTSD and other adult psychopathology as they appear naturally in the population. Significant associations or relationships or predictors were claimed as obtained a p-value was less than 0.05.[34] A stepwise regression was used in predicting the risk of cardiovascular disease.

RESULTS

There were 90.6% of the participants reporting at least one exposure to trauma (n = 549). Approximately 41.9% (n = 254) of the participants were retraumatized. Of 606 participants, 13.7% of them reported PTSD symptoms. The most prevalent forms of trauma among participants who reported PTSD symptoms were a natural disaster (26.5%),

transportation accident (22.9 %), near drowning (8.4%), other forms of trauma (8.4%), and physical assault (7.2%). Average SBP in the sample was 112 (± 12) mmHg, average DBP was 70 (± 9) mmHg and mean HR was 84 (± 12) beats per minutes. On average, adolescents have a BMI of 21.8 (± 5) kg/m.[2]

Numbers of trauma was significantly associated with SBP changes ($F_{(22, 556)} = 2.44$, $p < 0.001$) and heart rate changes ($F_{(22, 556)} = 1.644$, $p = 0.033$) among the adolescents but not DBP changes ($F_{(22, 556)} = 0.968$, $p = 0.503$). Both SBP and HR changes have linear relationship with the numbers of trauma experienced and reached its significant level, $p = 0.003$ and $p = 0.046$ respectively.

Risk Factors of Blood Pressure and Heart Rate Change

With all variables included in the analysis, a significant model has explained 21.2% of the variance, $F_{(3, 560)} = 37.74$, $p < 0.001$. Gender ($t = 2.08$, $p = 0.038$), ethnicity ($t = 2.21$, $p = 0.028$), total score of PCL-5 ($t = 11.64$, $p < 0.001$) were the significant predictors of SBP change. In models adjusted for socio-demographic factors and PTSD, the model reached it significance level, $F_{(6, 572)} = 7.82$, $p < 0.001$, in which nature disaster ($t = 2.64$, $p = 0.009$), near drowning ($t = 2.32$, $p = 0.021$) captivity ($t = 2.46$, $p = 0.014$), humiliation ($t = 2.55$, $p = 0.011$), serious injured that caused by participant ($t = 2.33$, $p = 0.020$) and absence of parent ($t = 2.07$, $p = 0.039$) were the significant predictors for SBP change.

With all variables included in the analysis, a significant model has explained 3.1% of the variance, $F_{(3, 562)} = 5.94$, $p = 0.001$. Ethnicity ($t = 2.506$, $p = 0.012$), age ($t = 2.049$, $p = 0.041$) and total score of PCL-5 ($t = 3.15$, $p = 0.002$) were the significant predictors for DBP change. In models adjusted for socio-demographic factors and PTSD, near drowning ($t = 2.19$, $p = 0.029$) was the significant predictors for DBP change and the model reached it significance level, $F_{(1, 577)} = 4.81$, $p = 0.029$.

With all variables included in the analysis, a significant model has explained 6.1% of the variance, $F_{(2, 562)} = 15.32$, $p < 0.001$). Gender ($t = 2.55$, $p = 0.011$), and total score of PCL-5 ($t = 5.29$, $p < 0.001$) were the significant predictors for HR change. In models adjusted for socio-demographic factors and PTSD, serious accident ($t = 2.24$, $p = 0.025$) and parental divorce ($t = 2.86$, $p = 0.004$) was the significant predictors for SBP change and the model reached it significance level, $F_{(2, 576)} = 6.19$, $p = 0.002$.

With all variables included in the analysis, a significant model has explained 3.6% of the variance, $F_{(3, 580)} = 7.30$, $p < 0.001$). Age ($t = 3.53$, $p < 0.011$), ethnicity ($t = 2.38$, $p = 0.017$) and hours of sleep ($t = 2.06$, $p = 0.040$) were the significant predictors for adolescents' BMI.

DISCUSSION

Understanding the aetiology of PTSD would help in explaining the mechanism of the PTSD and physical health outcome association among adolescence with local data. Inconsistent with the work of Pretty et al.[35] who found no significant traumatic effect on children (aged 11 to 14 years old) blood pressure, the adolescent sample in the present study demonstrated trauma event was significantly associated with SBP and HR reading. The result also showed that PTSD served as a significant predictor for the SBP, DBP and HR changes. This suggested that that as early as late adolescence the effect of trauma and PTSD was shown in their blood pressure and heart rate measure. A similar suggestion was made by Stein et al.[37] a data from ten countries (N = 18, 630), showing that individuals with 2 or more childhood trauma have their onset of hypertension at the age of 21 years old. Previous researchers commented that CVD as one of the chronic diseases which generally have its onset during mid to late adulthood should be viewed as a cumulative result of developmental disorder that begin in the early of life.[36,37] They claimed that the incident of CVD could be reduced if early detection and treatment were given to alleviate the stress-related symptoms among the children and adolescents.[21,37] Thus, the present finding might give a hint for the early detection of adverse physical health outcomes through monitoring blood pressure and heart rate among this age group.

Another issue was whether trauma exposure or PTSD itself played a more important role in the blood pressure and heart rate changes. This study suggested that PTSD played a more important role in such changes. The present findings showed that PTSD was persistent predictor throughout the models for blood pressure and heart rate changes among the adolescents even after adjusting the cofounders.[38] The current finding was consistent with other studies where authors used different methodologies to investigate the role of PTSD in predicting adverse physical health outcomes such as Goldberg et al.[38] and Paulus et al.[39] Sumner et al.[40] found that only 14% of participants with traumatic experience but with no PTSD symptoms were associated with adverse physical outcomes, whereas 47% of the participants who experienced trauma and exhibited PTSD symptoms were associated with CVD. This again showed that traumatized individuals with PTSD symptoms were at a higher risk of CVD than those with only traumatic experience but no PTSD symptoms.

Despite evidence indicating that PTSD was a risk factor for physiological measure change, resulting in elevated blood pressure and heart rate, other studies presented inconsistent results. For example, Clark, Thatcher and Martin[41] and Gooding et al.[42] found no association between history of childhood abuse and blood pressure elevation in adolescents and young adults aged between 12 to 32-years-old. This raised interest in the effect of numbers of traumatic experiences in explaining

this inconsistent finding. Most of these findings focused on a single specific trauma exposure such as violence, or sexual abuse, or 9/11 attack; however, it was important to take lifetime trauma into account as multiple trauma exposures were common.[23] The present study found that numbers of trauma were the significant linear association with SBP and HR changes, suggesting that with increased numbers of traumatic experience, individuals were at higher risk of having elevated blood pressure and heart rate as young as late adolescence. This would give a new perspective and focus on the paediatric practice, parents, and teachers that served as the first-line guardians of a healthy child and adolescent development through increase mental health literacy especially topic related to trauma exposure and PTSD.

This was further supported by a recent imaging study, showing that emotional stressors significantly associated with amygdala activity in which increased the arterial inflammation that can lead to cardiovascular disease in human beings.[43] This further supported the allostatic load theory that explained the association between trauma exposure, PTSD and cardiovascular-related outcomes. Human physiological changes that associated with stress are due to the activation of the amygdale.[24,40] Kraynak et al.[24] and Tawakol et al.[40] study not only focused on the physical manifestations of stress, but the biochemical consequences of the stress perceived. The result of these studies was consistent with the allostatic load theory that has been discussed above, discussing that stress might induce cardiovascular events. As illustrated above, PTSD and traumatic event have a high level of chronic stress.[44]

Limitation and Recommendation

One of the limitations of the present study was the retrospective self-reports that may argue to have recall bias in reporting their history. However, Scott, McLaughlin, Smith and Ellis[45] commented that such bias was insufficient to invalidate the study. They also commented that the retrospective study would be able to explore the underlying cognitive mechanisms of traumatized individuals and their medical outcomes after the events. The present study did not include the biochemical effect of trauma exposure and PTSD on cardiovascular disease because this was beyond the scope of the study.

PTSD might play its mediator role that exaggerated the physiological responses and increased the risk of adverse physical health outcome among this group of adolescents. However, it was possible that other variables such as dyslipidemia, age, diabetes mellitus which might affect the development of CVD, thereby contributing to our finding that PTSD was not a significant mediator. A bigger sample size that involved clinical sample may be needed in a future study to draw any conclusion.

Cardiovascular reactivity was demonstrated to associate with the experience of negative valence emotions, for example, shame.[26] These negative emotions were always associated with typical behaviour patterns like avoidance as coping mechanisms.[46] Alcohol drinking and smoking were common examples of avoidance coping style especially after experiencing the traumatic event.[47] Negative feelings of shame, guilt, and anger as variables in the future study might able to serve as moderators and to provide a more comprehensive explanation of the PTSD – adverse physical health outcome association.

The manifestation of PTSD may also promote and exaggerate the unhealthy behaviours such as drinking alcohol, smoking, binge eating which played an important role in CVD development. It is, therefore, giving treatment or psycho-intervention to patients with PTSD not only reducing the mediation of the unhealthy behaviours, physiological measures, but would increase physical activities,[47,48] and sleeping hours. The treatment or psycho-intervention could also introduce skills or techniques such as deep breathing exercises, alternate nostril breathing[49] to manage their overwhelmed emotions. With these changes, it might able to re-stabilize and balanced the stress-response system, subsequently reduced the risk of developing CVD. As the participants recruited in the present study was at a young age without any physical health diagnosis, psychoeducation was suggested to be suitable to imply. However, further research was needed to investigate the effectiveness of psychoeducation among individuals with trauma exposure and/or PTSD in the prevention of cardiovascular health.

CONCLUSIONS

The present study has several clinical implications; most importantly that PTSD could serve as one of the potential risk factors for elevated blood pressure and heart rate. Exposure to a traumatic event is likely to influence fundamental biological processes leading to adverse health outcomes in adulthood. It is yet to draw any conclusion regarding the PTSD role in any adverse physical health outcomes, the study showed the association of the consequences of trauma on physical health at a young age which might predispose to the outcomes. Given that the association of physiological changes among traumatized young adults suggested more studies should be conducted especially trauma interventions and prevention programmes that might have an impact on the development of adult adverse physical health outcomes.

ACKNOWLEDGMENTS

We acknowledge the immense help received from the scholars whose articles are cited and included in references to this manuscript. We also thank the Research Innovation and Enterprise Centre (RIEC) of Universiti Malaysia Sarawak for supporting this study. Thank you to all participants who had given us their full cooperation during the data collection.

Conflict of Interest

There is no conflict of interest.

Financial Support

This research was funded by The Ministry of Higher Education Malaysia, RACE/e (1)/888/2012 (06).

Author's Contribution

The authors confirm contribution to the paper as follows:

Chen Yoke Yong, Siti Raudzah Ghazali: study proposal, conception, and design;

Chen Yoke Yong: data collection;

Chen Yoke Yong, Siti Raudzah Ghazali: analysis, interpretation of results and draft manuscript preparation;

All authors reviewed the results and approved the final version of the manuscript.

REFERENCES

1. Bellis MA, Lowey H, Leckenby N, Hughes K, Harrison D. Adverse childhood experiences: retrospective study to determine their impact on adult health behaviours and health outcomes in a UK population. J Pub Heal 2014;36(1):81-91.
2. Hughes K, Bellis MA, Hardcastle KA, Sethi D, Butchart A, Mikton C, Jones L, Dunne MP. The effect of multiple adverse childhood experiences on health: a systematic review and meta-analysis. Lan Pub Health 2017;2(8):e356-66.
3. Olofsson N, Lindqvist K, Shaw BA, Danielsson I. Long-term health consequences of violence exposure in adolescence: A 26–year prospective study. Bri Med Comm Publ Health 2012;12(1):411.
4. Westphal M, Olfson M, Gameroff MJ, Wickramaratne P, Pilowsky DJ, Neugebauer R, Lantigua R, Shea S, Neria Y. Functional impairment in adults with past posttraumatic stress disorder: findings from primary care. Depr Anx 2011;28(8):686-695.
5. Danese A, McEwen BS. Adverse childhood experiences, allostasis, allostatic load, and age-related disease. Phys Behav 2012;106(1):29-39.
6. Tulloch H, Greenman PS, Tassé V. Post-traumatic stress disorder among cardiac patients: prevalence, risk factors, and considerations for assessment and treatment. Behav Sci 2015;5(1):27-40.
7. Coughlin SS. Post-traumatic stress disorder and cardiovascular disease. Open Cardi Med J 2011;5:164.
8. Dedert EA, Calhoun PS, Watkins LL, Sherwood A, Beckham JC. Posttraumatic stress disorder, cardiovascular, and metabolic disease: a review of the evidence. Ann Behav Med 2010;39(1):61-78.
9. Kibler JL. Posttraumatic stress and cardiovascular disease risk. J Trau Disor 2009 Apr 3;10(2):135-150.
10. Sherin JE, Nemeroff CB. Post-traumatic stress disorder: the neurobiological impact of psychological trauma. Dialog Clin Neur 2011;13(3):263.
11. Ganzel BL, Morris PA, Wethington E. Allostasis and the human brain: Integrating models of stress from the social and life sciences. Psych Rev 2010;117(1):134.
12. Eckart C, Kaufmann J, Kanowski M, Tempelmann C, Hinrichs H, Elbert T, Heinze HJ, Kolassa IT. Magnetic resonance volumetry and spectroscopy of hippocampus and insula concerning severe exposure of traumatic stress. Psychoph 2012;49(2):261-270.
13. Seo D, Tsou KA, Ansell EB, Potenza MN, Sinha R. Cumulative adversity sensitizes neural response to acute stress: association with health symptoms. Neuropsyhology 2014 Feb;39(3):670-680.
14. Berger M, Juster RP, Sarnyai Z. Mental health consequences of stress and trauma: allostatic load markers for practice and policy with a focus on Indigenous health. Austr Psychi 2015;23(6):644-649.
15. Gandubert C, Scali J, Ancelin ML, Carriere I, Dupuy AM, Bagnolini G, Ritchie K, Sebanne M, Martrille L, Baccino E, Hermes A. Biological and psychological predictors of posttraumatic stress disorder onset and chronicity. A one-year prospective study. Neurob Stress. 2016;3:61-67.
16. Murali R, Chen E. Exposure to violence and cardiovascular and neuroendocrine measures in adolescents. Ann Behav Med 2005;30(2):155-163.
17. Ulmer CS, Calhoun PS, Bosworth HB, Dennis MF, Beckham JC. Nocturnal blood pressure non-dipping, posttraumatic stress disorder, and sleep quality in women. Behav Med 2013;39(4):111-121.
18. Beckham JC, Flood AM, Dennis MF, Calhoun PS. Ambulatory cardiovascular activity and hostility ratings in women with chronic posttraumatic stress disorder. Biol Psych. 2009;65(3):268-272.
19. Ford JL, Browning CR. Effects of exposure to violence with a weapon during adolescence on adult hypertension. Ann Epidemiol 2014;24(3):193-198.
20. Clark CJ, Everson-Rose SA, Alonso A, Spencer RA, Brady SS, Resnick MD, Borowsky IW, Connett JE, Krueger RF, Suglia SF. Effect of partner violence in adolescence and young adulthood on blood pressure and incident hypertension. PLoS One 2014;9(3):e922-924.
21. Suglia SF, Sapra KJ, Koenen KC. Violence and cardiovascular health: a systematic review. Am J Prev Med 2015;48(2):205-212.
22. Taylor MK, Larson GE, Hiller Lauby MD, Padilla GA, Wilson IE, Schmied EA, Highfill-McRoy RM, Morgan CA. Sex differences in cardiovascular and subjective stress reactions: prospective evidence in a realistic military setting. Stress 2014;17(1):70-78.
23. Kessler RC, Petukhova M, Sampson NA, Zaslavsky AM, Wittchen HU. Twelve-month and lifetime prevalence and lifetime morbid risk of anxiety and mood disorders in the United States. Int J Meth Psych 2012;21(3):169-84.
24. Parati G, Esler M. The human sympathetic nervous system: its relevance in hypertension and heart failure. Eur Heart J 2012;33(9):1058-1066.
25. Da Silva AA, Do Carmo J, Dubinion J, Hall JE. The role of the sympathetic nervous system in obesity-related hypertension. Curr Hypert Rep 2009;11(3):206-208.
26. Kraynak TE, Marsland AL, Gianaros PJ. Neural mechanisms linking emotion with cardiovascular disease. Curr Cardiol Rep 2018;20(12):128-130.
27. Gavrieli A, Farr OM, Davis CR, Crowell JA, Mantzoros CS. Early life adversity and/or posttraumatic stress disorder severity are associated with poor diet quality, including consumption of trans fatty acids, and fewer hours of resting or sleeping in a US middle-aged population: A cross-sectional and prospective study. Metabolism 2015;64(11):1597-1610.
28. May-Ling JL, Loxton D, McLaughlin D. Trauma exposure and the subsequent risk of coronary heart disease among mid-aged women. J Behav Med 2015;38(1):57-65.

29. Weathers FW, Litz BT, Keane TM, Palmieri PA, Marx BP, Schnurr PP. The PTSD checklist for DSM-5 (PCL-5)–standard [measurement instrument]. Retrieved from http://www. ptsd. va. gov. 2013.
30. Weathers FW, Litz BT, Keane TM, Palmieri PA, Marx BP, Schnurr PP. The PTSD Checklist for DSM-5 (PCL-5) Scale. Retrieved from the National Center for PTSD website: www. ptsd. va. gov. 2013.
31. Ghazali SR, Chen YY. Reliability, concurrent validity, and a cutoff score of PTSD Checklist (PCL-5) for the Diagnostic and Statistical Manual of Mental Disorders, among Malaysian adolescents. Traumatology 2018;24(4):280-288.
32. Wortmann JH, Jordan AH, Weathers FW, Resick PA, Dondanville KA, Hall-Clark B, et al. Psychometric Analysis of the PTSD Checklist-5 (PCL-5) Among Treatment-Seeking Military Service Members. Psychol Ass 2016;28(11):1392-1403.
33. National Center for Health Statistics. National Health Interview Survey Questionnaire. Hyattsville, MD: US Department of Health and Human Services, Centers for Disease Control and Prevention; 2000.
34. Field A. Discovering statistics using IBM SPSS statistics. Sage 2013 Feb 20.
35. Pretty C, D O'Leary D, Cairney J, Wade TJ. Adverse childhood experiences and the cardiovascular health of children: a cross-sectional study. BMC Pediatrics 2013;13(1):208.
36. Stein DJ, Scott K, Abad JM, Aguilar-Gaxiola S, Alonso J, Angermeyer M, et al. Early childhood adversity and later hypertension: data from the World Mental Health Survey. Ann Clin Psych 2010;22(1):19.
37. Shonkoff JP, Garner AS, Siegel BS, Dobbins MI, Earls MF, McGuinn L, et al. The lifelong effects of early childhood adversity and toxic stress. Pediatrics 2012;129(1):e232-246.
38. Goldberg J, Magruder KM, Forsberg CW, Kazis LE, Üstün TB, Friedman MJ, et al. The association of PTSD with physical and mental health functioning and disability (VA Cooperative Study# 569: the course and consequences of posttraumatic stress disorder in Vietnam-era veteran twins). Qual Lif Res. 2014 Jun 1;23(5):1579-1591.
39. Paulus EJ, Argo TR, Egge JA. The impact of posttraumatic stress disorder on blood pressure and heart rate in a veteran population. J Trauma Stress 2013;26(1):169-172.
40. Sumner JA, Kubzansky LD, Elkind MS, Roberts AL, Agnew-Blais J, Chen Q, et al. Trauma exposure and posttraumatic stress disorder symptoms predict the onset of cardiovascular events in women. Circulation 2015;132(4):251-259.
41. Clark DB, Thatcher DL, Martin CS. Child abuse and other traumatic experiences, alcohol use disorders, and health problems in adolescence and young adulthood. J Pediatr Psyc 2010;35(5):499-510.
42. Gooding HC, Milliren C, McLaughlin KA, Richmond TK, Katz-Wise SL, Rich-Edwards J, et al. Child maltreatment and blood pressure in young adulthood. Chi Ab Negl 2014;38(11):1747-1754.
43. Tawakol A, Ishai A, Takx RA, Figueroa AL, Ali A, Kaiser Y, et al. The relation between resting amygdalar activity and cardiovascular events: a longitudinal and cohort study. Lancet 2017;389(10071):834-845.
44. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders-IV Text Revision, APA. Washington, DC. 2000.
45. Scott KM, McLaughlin KA, Smith DA, Ellis PM. Childhood maltreatment and DSM-IV adult mental disorders: comparison of prospective and retrospective findings. Br J Psych 2012;200(6):469-475.
46. Perren S, Gutzwiller-Helfenfinger E, Malti T, Hymel S. Moral reasoning and emotion attributions of adolescent bullies, victims, and bully-victims. Brit J Dev Psychol 2012;30(4):511-530.
47. Held P, Owens GP, Anderson SE. The interrelationships among trauma-related guilt and shame, disengagement coping, and PTSD in a sample of treatment-seeking substance users. Traumatics 2015;21(4):285-289.
48. Desai B, Desai D. To Compare the Effect of Specific Yoga and Aerobic Exercise Program on Vital Parameters in Young Adult Females. Int J Cur Res Rev 2020;12(02):1-7.
49. Patel P. Influence of alternate nostril breathing on cardiorespiratory functions among hypertensive subjects. Int J Curr Res Rev 2012;4(10):96-100.