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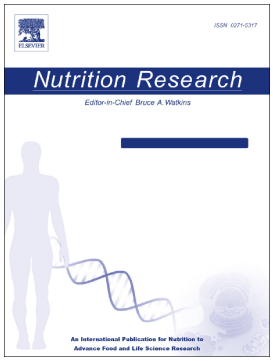

Conflicting relationship between dietary intake and metabolic health in PTSD: A systematic review

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TITLE: Conflicting relationship between dietary intake and metabolic health in PTSD: A systematic review

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Abbreviations

aHEI; Alternate Health Eating Index

BMI; Body mass index

BP; Blood pressure

BTQ; Brief Trauma Questionnaire

CIDI; Composite International Diagnostic Interview

CVD; Cardiovascular disease

DASH; Dietary Approaches to Stop Hypertension

DSM-IV; Diagnostic and Statistical Manual of Mental Disorders 4th Edition

FFQ; Food frequency questionnaire

HDL; High-density lipoprotein

HTN; Hypertension

LDL; Low-density lipoprotein

MetS; Metabolic Syndrome

NHMRC; National Health and Medical Research Council

NHSII; Nurses' Health Study II

PDSQ; Psychiatric Diagnostic Screening Questionnaire

PTSD; Posttraumatic stress disorder

SSQ-IV PTSD; Short Screening Scale for DSM-IV PTSD

T2DM; Type 2 diabetes mellitus

UCLA; University of California Los Angeles

WC; Waist Circumference

WHR; Waist to hip ratio

Abstract

Posttraumatic stress disorder (PTSD) is a disabling psychological disorder associated with significant physical comorbidities. There has been growing evidence to support the relationship between PTSD and cardiometabolic disease. Disordered eating behaviors often seen in people with PTSD symptoms may explain increased cardiometabolic risk. This systematic review aimed to assess the quality of evidence surrounding dietary intake of individuals with symptoms or a diagnosis of PTSD and their associated risk with cardiometabolic health outcomes. Online databases Scopus, ProQuest (Health), Embase, Medline, PsycINFO, and CINAHL with Full Text were searched for peer-reviewed English articles prior to December 2017 that examined dietary intake and cardiometabolic health outcomes in adults with PTSD symptoms or diagnosis. The quality of each study was graded based on the design and methodology using adapted quality assessment tools. Seven studies with five unique participant samples were included in the review. Study methods, design, populations, and outcomes were inconsistent across studies. Dietary intake was considerably varied and limited associations were demonstrated between dietary intake and cardiometabolic risk factors in the PTSD cohorts. Due to the variability of measures and study outcomes, there was insufficient evidence to determine the relationship between dietary intake and PTSD-related cardiometabolic health outcomes. Future studies are needed to examine these associations in individuals with PTSD: specifically higher quality descriptive studies are necessary to confirm a link between diet and cardiometabolic disease in PTSD.

Keywords

Dietary intake; Diet quality; Cardiometabolic disease; Posttraumatic stress disorder; Systematic review

1. Introduction

Posttraumatic stress disorder (PTSD) is a disabling psychological condition that may develop following exposure to one or more traumatic events. Re-experiencing trauma, intrusive memories, avoidance, and increased arousal are hallmark symptoms of PTSD which can disrupt social, occupational, and interpersonal activities [1]. In addition to psychological burden, PTSD is associated with significant physical comorbidities. Over the last decade, a large body of evidence has emerged supporting a positive relationship between PTSD and cardiometabolic disease. In particular, individuals with PTSD are at increased risk of developing metabolic syndrome (MetS), cardiovascular disease (CVD), and type 2 diabetes mellitus (T2DM) [2-5]. Furthermore, other cardiometabolic disease risk factors including obesity, hypertension, and dyslipidaemia are significantly associated with PTSD symptoms and diagnosis [4, 6, 7].

The relationship between PTSD and cardiometabolic disease is complex and involves both biological and behavioral mediating factors [3]. Lifestyle factors such as smoking [8], increased alcohol intake [9, 10], and limited physical activity [11, 12], and disordered eating behaviors [13] are often coexisting with cardiometabolic dysfunction in individuals with PTSD. Specifically, diet may play an important role in mediating both PTSD and PTSD-related cardiometabolic disease (Figure 1).

The impact of food behaviors and diet quality is of emerging interest across broad clinical fields of mental health [14]. PTSD has been associated with stress-related eating disorders [15-17], emotional eating [18, 19], and poor diet quality [18, 20]. In particular, poor dietary intake such as increased sugar intake and low fruit and vegetable intakes have been

associated with increased PTSD symptomology [18, 20]. Similarly, poor diet has been identified as a modifiable risk factor for other mental health conditions such as depression anxiety, and schizophrenia [21-24].

In the general population, high caloric intake and specific food or nutrient intake including low fruit and vegetable intakes, and high added sugars, saturated or trans-fats have been directly linked to cardiometabolic risk [25, 26] including overweight and obesity [27]. However the mediating role of dietary intake in PTSD-related cardiometabolic disease remains unknown.

In order to better understand how PTSD increases the risk of cardiometabolic disease, the mediating role of dietary intake must be evaluated. Therefore the present systematic review aimed to provide a comprehensive overview of the literature and to assess the quality of current evidence surrounding dietary intake of individuals with symptoms or a diagnosis of PTSD and their associated risk for cardiometabolic health outcomes. Using six online databases, Scopus, ProQuest (Health), Embase, Medline, PsycINFO, and CINAHL with Full Text, a literature search was conducted from time of database conception to December 2017 to retrieve articles assessing dietary intake and cardiometabolic health outcomes in adults with PTSD.

2. Approach

2.1 Search Strategy

This review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [28]. Two reviewers conducted a broad search between time of database inception to December 2017 using six online databases, Scopus, ProQuest

(Health), Embase, Medline, PsycINFO, and CINAHL with Full Text. The search terms included: posttraumatic stress disorder (post-traumatic stress disorder, PTSD), dietary intake (diet*, nutrit*, food, eating, feeding, alcohol, beverage, behavio?r, pattern, habit, consum*), and cardiometabolic risk (metabolic, cardio*, CVD, type 2 diabetes, T2DM, noninsulin dependent diabetes mellitus, diabetes mellitus, DM, health, disease, syndrome). These key words were applied across the titles, abstracts and full-text articles. The search was limited to English articles, abstract available, and academic journals and dissertations. Subsequently, the reference lists of review papers were hand-searched for peer-reviewed original research articles [3, 29, 30].

2.2 Selection Criteria

Selection criteria for inclusion include: (1) published as peer-reviewed academic journal or dissertations with full-text available, (2) adults (mean aged ≥18 years) with PTSD symptoms or diagnosis determined by a validated instrument, (3) measured either qualitative or quantitative dietary intake with an analysis of at least one cardiometabolic health outcome. Cardiometabolic health outcomes included CVD, MetS, T2DM, measurements of adiposity, blood lipids, blood glucose, and blood pressure (BP). All study designs were eligible for inclusion. Studies were excluded for the following reasons: (1) non-human studies, (2) children or adolescents (<18 years), (3) adults with no clinical diagnosis or PTSD symptoms stated, (4) the study included only one of the measures of interest, (5) the study was not original research. All articles yielded from search results were reviewed by one reviewer initially based on title and abstract. All articles were independently reviewed by two reviewers. Any disagreements were resolved following discussion between reviewers or following consultation with the third author.

2.3 Data Extraction

The following information was extracted from selected articles: (1) year of publication; (2) study design and country of origin; (3) sample size and study population demographics; (4) measures used to screen and/or assess PTSD severity and dietary intake; and (5) main findings relating to PTSD symptoms, dietary intake and cardiometabolic health outcomes. For this review, all forms of qualitative or quantitative dietary intake and cardiometabolic outcomes were reported.

2.4 Quality Assessments

The included studies were graded for methodological quality using the National Health and Medical Research Council (NHMRC) Evidence Hierarchy based on study design [31]. Overall study quality was further evaluated based on an amended protocol from Fletcher *et al.* [32]. Quality was rated based on study design, setting, participants, exposure and outcome variables, data collection and statistical methods. Please see Table 1 for the full description of variables and scoring criteria. A maximum of 15 points could be allocated and a percentage was calculated. Additionally, dietary assessment methods and reporting quality were evaluated using the method from Burrows *et al [33]*. Total scores were based on six components including methodology validation, data collection quality, and reporting of scoring with a maximum score of seven. Studies were rated as: poor (≤2), acceptable (2.5-<3.5), good (3.5-<5) or excellent (≥5). Two reviewers independently scored each study and any discrepancies were resolved by discussion.

3. Findings

3.1 Search Results

A total of 269 records were identified following database and reference list searches (See Figure 2). After removing duplicates and screening articles based on titles and abstracts, 18 full-text articles were assessed for eligibility. Eleven articles were excluded based on the

following reasons: did not involve adults with PTSD (*n*=1); did not report dietary intake (*n*=5); overlap in data (*n*=1); and non-original research (*n*=4). The seven remaining articles were included in this systematic review.

3.2 Study Characteristics

The study characteristics are summarized in Table 2. Five unique participant samples were reported in the seven articles. Sumner *et al*. 2015, 2016, and Roberts *et al*. reported separate findings from the same cohort, the Nurses' Health Study II (NHSII) [34-36]. Studies originated in the United States (*n*=6) and the Netherlands (*n*=1) and consisted of cross-sectional studies (*n*=4) and longitudinal cohort studies (*n*=3). Participant populations varied between studies and included nurses [34-36], young women attending reproductive clinics [37], community members with [38] and without [39, 40] metabolic disease, and veterans [39]. The number of participants ranged from 80 to 49978 with mean ages ranging from 20.8 to 46 years. Four of the seven articles included females only [34-37], with the remaining three including both males and females [38-40]. Participants with reported PTSD symptoms or diagnosis of PTSD ranged from 18.7 to 52%.

3.3 Study Measures

All studies included PTSD and non-PTSD groups; however the measure used to diagnose or assess PTSD severity differed. The majority of studies used validated or partially validated screening tools to assess PTSD symptom severity [34-38]. Remaining studies utilized validated diagnostic tools to diagnose presence of PTSD [39, 40]. Depending on the measure, PTSD groups were categorized based on diagnosis, the number of symptoms reported, or by severity score.

All studies reported at least one metabolic risk factor or cardiometabolic disease outcome. Body mass index (BMI) was reported in every study and was calculated from measured [37-40] or self-reported [34-36] height and weight. Three studies also assessed other anthropometric outcomes, including body composition using bioelectrical impedance analysis and waist circumference (WC) or waist-to-hip ratio (WHR) [38-40]. Fasting plasma lipids including low-density lipoprotein (LDL) and high-density lipoprotein (HDL) were determined by laboratory analysis [40]. Longitudinal incidence of self-reported hypertension [35], T2DM [34], and CVD including myocardial infarction, angina, or stroke [36], were reported from the NHSII cohort. Additionally, another study included BP measurements [39]. Self-reported BMI, T2DM, CVD, and hypertension were validated in the NHSII by in-person weighing, medical record review or in-person BP measurement in a random subset of participants [34-36].

Several different self-report assessment tools were used to evaluate dietary intake. Two studies utilized a non-validated 5-item questionnaire which included fast food and soda consumption [37, 39] along with fruit and vegetable intake [37, 39]. Another study utilized a 24-hour food recall method which included food types, amounts consumed, and preparation methods [40]. The remaining studies utilised a Food Frequency Questionnaire (FFQ) [34-36, 38]. Diet quality from the FFQ was assessed using the Alternative Healthy Eating Index (aHEI), the aHEI-2010 or the Dietary Approaches to Stop Hypertension (DASH). Scores are calculated based on different food components including fruits, vegetables, whole grains, and sweetened beverages. High scores from both the aHEI/aHEI-2010 and DASH have been previously associated with reduced risk of T2DM and CVD [41].

3.4 Methodological Quality Assessment

Dietary reporting and study quality assessments are reported in Table 2. Overall study quality was considered high for cross-sectional and longitudinal study design based on the amended protocol from Fletcher *et al. [32]*. Dietary assessment and reporting quality ranged from poor (*n*=3) to good (*n*=4) with reduced quality ratings mostly due to use of non-validated scales or insufficient methodology reporting. In particular, studies lacked information on the qualifications of the person administering or scoring the tool. The NHSII cohort and the study by Gavrieli *et al*. utilized FFQs previously validated in relevant adult American populations [42, 43].

3.5 Main Findings

The associations between dietary intake and cardiometabolic health outcomes in individuals with PTSD are reported in Table 2.

3.5.1 Dietary Intake and PTSD

The relationship between PTSD and dietary intake varied among studies. In the study by Hirth *et al*., PTSD symptoms were positively associated with an increased likelihood of eating fast food more frequently and consuming more than one soda a day [37]. In the study by Godfrey et al., although no differences in fast food or soda consumption between PTSD and controls were identified, participants with PTSD reported significantly reduced fruit intake compared to controls [39]. Gavrieli *et al*. reported an association between PTSD severity and reduced DASH and aHEI-2010 scores (an indication of poor diet quality) when adjusted for age and gender. However, when adjusted for education, PTSD severity was no longer associated with DASH or aHEI scores [38]. Adjusting for additional confounders

including income, BMI, and total energy intake further attenuated the relationship between PTSD and diet quality. Similarly, in the study by de Vries *et al*. there were no differences in dietary intake including total energy, proteins, fats, and carbohydrates between the PTSD group and controls [40]. In the NHSII cohort higher aHEI scores (an indication of good diet quality) were associated with an increased number of PTSD symptoms [34-36]. Furthermore, increased PTSD symptoms were positively associated with reduced sugar-sweetened and artificially sweetened beverages [35].

3.5.2 Associations between dietary intake and cardiometabolic health in PTSD

The majority of studies reported a positive association between PTSD and increased cardiometabolic dysfunction (Table 2). While most studies also tested for direct associations between dietary intake and cardiometabolic risk factors in the PTSD cohorts [34-36, 38, 40], limited independent associations were demonstrated. Controlling for dietary intake did not reveal additional differences in lipoprotein levels; however it did reverse the direction of effect for LDL [40]. Additionally, dietary intake did not significantly attenuate the relationships between PTSD and cardiometabolic outcomes and it was not used as an independent variable when assessing the relationships between symptoms of PTSD and T2DM and hypertension [34-36].

4. Discussion

This systematic review identified and summarized all studies published prior to December 2017 that investigated dietary intake and cardiometabolic health outcomes in adults with PTSD. In summary, only seven studies met inclusion criteria. Poor dietary intake is well-defined as a modifiable risk factor for cardiometabolic dysfunction and disease [44, 45]. However, this review indicates dietary intake remains mostly unexplored in PTSD with a lack

of dietary data reporting and/or inconsistencies in dietary data collection methods and conflicting findings relating to associations between diet and metabolic risk. This identifies a significant gap in our understanding of modifiable contributors to metabolic risk in PTSD and a need to assess dietary habits in a robust fashion.

Two major findings of this review are: dietary intake is variable across studies and accounting for dietary intake as a mediator in the relationship between PTSD and cardiometabolic dysfunction is essential. After adjusting for confounders, the relationship between diet and PTSD varied across studies with positive, neutral, and negative associations observed. Poor eating habits [37-39] observed in studies of people with PTSD may be explained as stress-related overeating [46], use of food to self-medicate [47] or as a side effect of psychotropic medications [48]. However, interestingly, the NHSII cohort reported an association between higher diet quality and increased PTSD symptom severity [34-36]. The variability in dietary intake between studies may be a result in the differences in dietary assessment tools, or in population characteristics such as symptom severity, age, gender, education, psychotropic medication use, ethnicity, or marital status. Specifically, in this review, higher education levels in the NHSII cohort may have been associated with higher diet quality [49]. Additionally, when controlling for education, the relationship between PTSD and poor diet quality was no longer significant [38]. This emphasizes the need to identify potential factors like education that may mediate lifetime dietary intake. Correspondingly, the association between physical activity and PTSD is also quite variable [29], and may be related to differences in hyperarousal symptomology [50].

While intuitively, improvements in diet quality (independent of, or alongside increases in physical activity) may reduce cardiometabolic risk in people with PTSD; this review identified limited available data that addresses this hypothesis. Emerging evidence for the broad benefits of diet and exercise programs for other chronic mental health conditions [51, 52] indicates the importance of high quality dietary research in PTSD populations to inform the development of clinical practice in this field. Controlling for dietary intake in combination with other lifestyle factors (physical activity, alcohol intake, and smoking) minimally, although not significantly, mediated the relationship between cardiometabolic outcomes and PTSD in the NHSII [34-36]. Additionally controlling for diet reversed the direction of effect for LDL [40]. This highlights the importance of controlling for dietary intake in the PTSD-cardiometabolic health relationship.

4.1 Limitations and Gaps in the Literature

With only seven studies meeting inclusion criteria, this review identifies a significant gap in the literature regarding dietary intake and PTSD-related cardiometabolic dysfunction. Several key limitations of the current literature were identified.

Firstly, studies included in this review were significantly limited by dietary assessment tools and cross-sectional study design. Although overall quality of studies was high, almost half of the studies either did not use validated dietary assessment tools [37, 39] or did not provide sufficient information to determine tool validity [40].The use of the aHEI or DASH [34-36, 38] to assess diet quality may result in a loss of information as overall high quality scores may mask individual dietary deficiencies. Similarly, the use of brief non-validated questionnaires [37, 39] provided very limited information on the overall diet of participants.

Additionally due to the cross-sectional nature of most studies, the inference regarding causality of the effects of diet and PTSD-related cardiometabolic health indicators is restricted. Better quality observational data, to lead to more randomized controlled studies and cohort studies, are necessary to provide stronger evidence in order to make conclusions or recommendations regarding dietary intake.

Secondly, in addition to dietary intake assessments, variable measures were used across studies to assess cardiometabolic dysfunction and PTSD, thus preventing inter-study comparisons. More studies are required to determine how diet may mediate individual outcomes. Additionally, as each study utilized different screening tools to measure PTSD symptom severity or to diagnose PTSD, the prevalence of participants with PTSD among the selected studies was not directly comparable. Similarly, the use of a PTSD symptom severity measure does not provide an actual diagnosis of PTSD and may result in overestimation of the clinical population.

Thirdly, most studies included predominantly female participants of Caucasian background. Although females have been found to have increased risk of developing PTSD following trauma exposure [53, 54], males represent a significant proportion of adults with PTSD [55]. Furthermore, gender differences in dietary intake and food behaviors are well accepted and may impact on the generalizability of the emerging data in this review. A more proportionate distribution of sex and ethnicity would better represent the general population of adults with PTSD. International applicability of the results may also be limited as most studies included in the review originated in the United States.

4.2 Future Research

Despite growing evidence of the relationship between PTSD and cardiometabolic disease, limited studies have investigated dietary intake as a mediator. This review highlights the need for more research in this field; specifically, higher quality descriptive studies to confirm a link between diet and cardiometabolic disease in PTSD and ascertain key mediators for nutritional targets. The standardization and validation of dietary intake assessment tools [33] and a commitment to strengthen the reporting of dietary studies by adhering to standardized criteria (e.g. STROBE-NUT guidelines) [56] will contribute to the development of evidence based guidelines for integrated care for PTSD.

4.3 Conclusion

In conclusion, this systematic review provides insufficient evidence to elucidate the relationship between dietary intake and cardiometabolic health outcomes in individuals with PTSD. However, the importance of controlling for dietary intake when examining the relationship between PTSD and cardiometabolic health was established. Although diet is a modifiable lifestyle feature that can have a negative impact on overall health, the extent to which dietary intake may mediate PTSD-related cardiometabolic disease is not established. Targeted studies are needed to elucidate the role of dietary influences on cardiometabolic health outcomes in individuals with PTSD.

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References

[1] American Psychiatric Association, D. S. M. Task Force. Diagnostic and statistical manual of mental disorders: DSM-5. Arlington, VA: American Psychiatric Association; 2013.
[2] Edmondson D, Kronish IM, Shaffer JA, Falzon L, Burg MM. Posttraumatic stress disorder and risk for coronary heart disease: a meta-analytic review. Am Heart J. 2013;166:806-14.
[3] Koenen KC, Sumner JA, Gilsanz P, Glymour MM, Ratanatharathorn A, Rimm EB, et al. Post-traumatic stress disorder and cardiometabolic disease: improving causal inference to inform practice. Psychol Med. 2017;47:209-25.
[4] Rosenbaum S, Stubbs B, Ward PB, Steel Z, Lederman O, Vancampfort D. The prevalence and risk of metabolic syndrome and its components among people with posttraumatic stress disorder: a systematic review and meta-analysis. Metabolism. 2015;64:926-33.
[5] Vancampfort D, Rosenbaum S, Ward PB, Steel Z, Lederman O, Lamwaka AV, et al. Type 2 Diabetes Among People With Posttraumatic Stress Disorder: Systematic Review and Meta-Analysis. Psychosom Med. 2016;78:465-73.
[6] Levine AB, Levine LM, Levine TB. Posttraumatic stress disorder and cardiometabolic disease. Cardiology. 2014;127:1-19.
[7] Maguen S, Madden E, Cohen B, Bertenthal D, Neylan T, Talbot L, et al. The relationship between body mass index and mental health among Iraq and Afghanistan veterans. J Gen Intern Med. 2013;28 Suppl 2:S563-70.
[8] Farr OM, Ko BJ, Joung KE, Zaichenko L, Usher N, Tsoukas M, et al. Posttraumatic stress disorder, alone or additively with early life adversity, is associated with obesity and cardiometabolic risk. Nutr Metab Cardiovasc Dis. 2015;25:479-88.
[9] Vaccarino V, Goldberg J, Magruder KM, Forsberg CW, Friedman MJ, Litz BT, et al. Posttraumatic stress disorder and incidence of type-2 diabetes: a prospective twin study. J Psychiatr Res. 2014;56:158-64.
[10] Vaccarino V, Goldberg J, Rooks C, Shah AJ, Veledar E, Faber TL, et al. Post-traumatic stress disorder and incidence of coronary heart disease: a twin study. J Am Coll Cardiol. 2013;62:970-8.
[11] Rosenbaum S, Vancampfort D, Steel Z, Newby J, Ward PB, Stubbs B. Physical activity in the treatment of Post-traumatic stress disorder: A systematic review and meta-analysis. Psychiatry Res. 2015;230:130-6.
[12] Rosenbaum S, Tiedemann A, Berle D, Ward PB, Zachary S. Exercise as a novel treatment option to address cardiometabolic dysfunction associated with PTSD. Metabolism. 2015;64:e5-6.
[13] Slane JD, Levine MD, Borrero S, Mattocks KM, Ozier AD, Silliker N, et al. Eating Behaviors: Prevalence, Psychiatric Comorbidity, and Associations With Body Mass Index Among Male and Female Iraq and Afghanistan Veterans. Mil Med. 2016;181:e1650-e6.
[14] Hjorth P, Espensen CH, Madsen NJ, Viuff AG, Munk-Jorgensen P. Reducing the Risk of Type 2 Diabetes in Nonselected Outpatients With Schizophrenia: A 30-Month Program. J Psychiatr Pract. 2018;24:21-31.
[15] Mason SM, Flint AJ, Roberts AL, Agnew-Blais J, Koenen KC, Rich-Edwards JW. Posttraumatic stress disorder symptoms and food addiction in women by timing and type of trauma exposure. JAMA Psychiatry. 2014;71:1271-8.
[16] Mason SM, Frazier PA, Austin SB, Harlow BL, Jackson B, Raymond NC, et al. Posttraumatic Stress Disorder Symptoms and Problematic Overeating Behaviors in Young Men and Women. Ann Behav Med. 2017;51:822-32.
[17] Strickler HL. The Interaction between Post-Traumatic Stress Disorders and Eating Disorders: A Review of Relevant Literature. J Trauma Treat. 2013;3:183.

[18] Carmassi C, Antonio Bertelloni C, Massimetti G, Miniati M, Stratta P, Rossi A, et al. Impact of DSM-5 PTSD and gender on impaired eating behaviors in 512 Italian earthquake survivors. Psychiatry Res. 2015;225:64-9.
[19] Talbot LS, Maguen S, Epel ES, Metzler TJ, Neylan TC. Posttraumatic stress disorder is associated with emotional eating. J Trauma Stress. 2013;26:521-5.
[20] Smith-Marek EN, Durtschi J, Brown C, Dharnidharka P. Exercise and Diet as Potential Moderators Between Trauma, Posttraumatic Stress, Depression, and Relationship Quality Among Emerging Adults. Am J Fam Ther. 2016;44:53-66.
[21] Peet M. Diet, diabetes and schizophrenia: review and hypothesis. Br J Psychiatry Suppl. 2004;47:S102-5.
[22] Marx W, Moseley G, Berk M, Jacka F. Nutritional psychiatry: the present state of the evidence. Proc Nutr Soc. 2017;76:427-36.
[23] Jacka FN, Cherbuin N, Anstey KJ, Butterworth P. Dietary patterns and depressive symptoms over time: examining the relationships with socioeconomic position, health behaviours and cardiovascular risk. PLOS One. 2014;9:e87657.
[24] Lai JS, Hiles S, Bisquera A, Hure AJ, McEvoy M, Attia J. A systematic review and meta-analysis of dietary patterns and depression in community-dwelling adults. Am J Clin Nutr. 2014;99:181-97.
[25] de Souza RJ, Mente A, Maroleanu A, Cozma AI, Ha V, Kishibe T, et al. Intake of saturated and trans unsaturated fatty acids and risk of all cause mortality, cardiovascular disease, and type 2 diabetes: systematic review and meta-analysis of observational studies. BMJ. 2015;351:h3978.
[26] Jayalath VH, de Souza RJ, Ha V, Mirrahimi A, Blanco-Mejia S, Di Buono M, et al. Sugar-sweetened beverage consumption and incident hypertension: a systematic review and meta-analysis of prospective cohorts. Am J Clin Nutr. 2015;102:914-21.
[27] Knight JA. Diseases and disorders associated with excess body weight. Ann Clin Lab Sci. 2011;41:107-21.
[28] Liberati A, Altman DG, Tetzlaff J, Mulrow C, Gøtzsche PC, Ioannidis JPA, et al. The PRISMA Statement for Reporting Systematic Reviews and Meta-Analyses of Studies That Evaluate Health Care Interventions: Explanation and Elaboration. PLOS Med. 2009;6:e1000100.
[29] Hall KS, Hoerster KD, Yancy WS, Jr. Post-traumatic stress disorder, physical activity, and eating behaviors. Epidemiol Rev. 2015;37:103-15.
[30] Sagud M, Jaksic N, Vuksan-Cusa B, Loncar M, Loncar I, Peles AM, et al. Cardiovascular Disease Risk Factors in Patients with Posttraumatic Stress Disorder (PTSD): A Narrative Review. Psychiatr Danub. 2017;29:421-30.
[31] Council NHaMR. NHMRC levels of evidence and grades for recommendations for developers of guidelines. In: Council NHaMR, editor.2009.
[32] Fletcher E, Leech R, McNaughton SA, Dunstan DW, Lacy KE, Salmon J. Is the relationship between sedentary behaviour and cardiometabolic health in adolescents independent of dietary intake? A systematic review. Obes Rev. 2015;16:795-805.
[33] Burrows T, Golley RK, Khambalia A, McNaughton SA, Magarey A, Rosenkranz RR, et al. The quality of dietary intake methodology and reporting in child and adolescent obesity intervention trials: a systematic review. Obes Rev. 2012;13:1125-38.
[34] Roberts AL, Agnew-Blais JC, Spiegelman D, Kubzansky LD, Mason SM, Galea S, et al. Posttraumatic stress disorder and incidence of type 2 diabetes mellitus in a sample of women: a 22-year longitudinal study. JAMA Psychiatry. 2015;72:203-10.
[35] Sumner JA, Kubzansky LD, Roberts AL, Gilsanz P, Chen Q, Winning A, et al. Post-traumatic stress disorder symptoms and risk of hypertension over 22 years in a large cohort of younger and middle-aged women. Psychol Med. 2016;46:3105-16.

[36] Sumner JA, Kubzansky LD, Elkind MS, Roberts AL, Agnew-Blais J, Chen Q, et al. Trauma Exposure and Posttraumatic Stress Disorder Symptoms Predict Onset of Cardiovascular Events in Women. Circulation. 2015;132:251-9.
[37] Hirth JM, Rahman M, Berenson AB. The association of posttraumatic stress disorder with fast food and soda consumption and unhealthy weight loss behaviors among young women. J Womens Health (Larchmt). 2011;20:1141-9.
[38] 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;64:1597-610.
[39] Godfrey KM, Lindamer LA, Mostoufi S, Afari N. Posttraumatic stress disorder and health: a preliminary study of group differences in health and health behaviors. Ann Gen Psychiatry. 2013;12:30.
[40] de Vries GJ, Mocking R, Assies J, Schene A, Olff M. Plasma lipoproteins in posttraumatic stress disorder patients compared to healthy controls and their associations with the HPA- and HPT-axis. Psychoneuroendocrinology. 2017;86:209-17.
[41] Schwingshackl L, Hoffmann G. Diet Quality as Assessed by the Healthy Eating Index, the Alternate Healthy Eating Index, the Dietary Approaches to Stop Hypertension Score, and Health Outcomes: A Systematic Review and Meta-Analysis of Cohort Studies. J Acad Nutr Diet. 2015;115:780-800.e5.
[42] Block G, Hartman AM, Dresser CM, Carroll MD, Gannon J, Gardner L. A data-based approach to diet questionnaire design and testing. Am J Epidemiol. 1986;124:453-69.
[43] Willett WC, Sampson L, Stampfer MJ, Rosner B, Bain C, Witschi J, et al. Reproducibility and validity of a semiquantitative food frequency questionnaire. Am J Epidemiol. 1985;122:51-65.
[44] Bell LK, Edwards S, Grieger JA. The Relationship between Dietary Patterns and Metabolic Health in a Representative Sample of Adult Australians. Nutrients. 2015;7:6491-505.
[45] Suliga E, Kozieł D, Cieśla E, Głuszek S. Association between dietary patterns and metabolic syndrome in individuals with normal weight: a cross-sectional study. Nutr J. 2015;14:55.
[46] Geiker NRW, Astrup A, Hjorth MF, Sjödin A, Pijls L, Markus CR. Does stress influence sleep patterns, food intake, weight gain, abdominal obesity and weight loss interventions and vice versa? Obes Rev. 2018;19:81-97.
[47] Brewerton TD. Food addiction as a proxy for eating disorder and obesity severity, trauma history, PTSD symptoms, and comorbidity. Eat Weight Disord. 2017;22:241-7.
[48] Jensen-Otsu E, Austin GL. Antidepressant Use is Associated with Increased Energy Intake and Similar Levels of Physical Activity. Nutrients. 2015;7:9662-71.
[49] Pampel FC, Krueger PM, Denney JT. Socioeconomic Disparities in Health Behaviors. Annu Rev Sociol. 2010;36:349-70.
[50] Vancampfort D, Richards J, Stubbs B, Akello G, Gbiri CA, Ward PB, et al. Physical Activity in People With Posttraumatic Stress Disorder: A Systematic Review of Correlates. J Phys Act Health. 2016;13:910-8.
[51] Teasdale SB, Latimer G, Byron A, Schuldt V, Pizzinga J, Plain J, et al. Expanding collaborative care: integrating the role of dietitians and nutrition interventions in services for people with mental illness. Australas Psychiatry. 2018;26:47-9.
[52] Teasdale SB, Ward PB, Rosenbaum S, Watkins A, Curtis J, Kalucy M, et al. A nutrition intervention is effective in improving dietary components linked to cardiometabolic risk in youth with first-episode psychosis. Br J Nutr. 2016;115:1987-93.

[53] Crum-Cianflone NF, Jacobson I. Gender differences of postdeployment post-traumatic stress disorder among service members and veterans of the Iraq and Afghanistan conflicts. Epidemiol Rev. 2014;36:5-18.
[54] Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson CB. Posttraumatic stress disorder in the National Comorbidity Survey. Arch Gen Psychiatry. 1995;52:1048-60.
[55] Hourani L, Williams J, Bray RM, Wilk JE, Hoge CW. Gender Differences in Posttraumatic Stress Disorder and Help Seeking in the U.S. Army. J Womens Health (Larchmt). 2016;25:22-31.
[56] Lachat C, Hawwash D, Ocké MC, Berg C, Forsum E, Hörnell A, et al. Strengthening the Reporting of Observational Studies in Epidemiology—Nutritional Epidemiology (STROBE-nut): An Extension of the STROBE Statement. PLOS Med. 2016;13:e1002036.

Figure Captions

Figure 1. An illustration of known risk factors or mediators for the relationship between PTSD and increased cardiometabolic (CM) risk. There are established associations between diet and PTSD with potential mechanisms related to inflammatory, immune and gut-brain axis pathways; however the effect of diet as a modifiable risk factor for CM risk in PTSD remains unexplored.

Figure 2. PRISMA Flowchart for article selection process

Table 1: Study quality assessment criteria based on methodology by Fletcher *et al.* [35]

Criteria (rating: 0.5 or 1 for yes, 0 for no, unclear)	**Score (max 15)**
Study design	
1. Is the study design presented?	**0.5**
AND is the study design longitudinal and not cross sectional?	**0.5**
Target population	
2. Do the authors describe the target population they wanted to research?	**1**
Sample	
3. Was a random sample of the target population taken/described?	**0.5**
AND/OR was the response rate 60% or more?	**0.5**
4. Is the participant selection described or referred to?	**1**
5. Is the participant recruitment described or referred to?	**1**
6. Are the inclusion and/or exclusion (eligibility) criteria stated, or referred to?	**1**
7. Is the study sample described? (min. size, gender, age, BMI)	**1**
8. Are the numbers of participants at each stage of the study reported? (reported at least the no. of eligible, recruited, or those with complete data)	**1**
Variables	
9. Are the measures of PTSD, dietary intake and metabolic health outcome sufficiently described in detail?	**1**
Data sources and collection	

10. Do authors describe the source of their data? (registry, health survey)	**0.5**
AND did authors describe how the data were collected? (e.g. by mail, by survey)	**0.5**
Measurements	
11. Was the validity of PTSD assessment tool mentioned or referred to?	**1**
12. Was the validity of dietary intake assessment tool mentioned or referred to?	**1**
13. Was the metabolic health outcome measured objectively and not by self-report?	**1**
Statistical methods	
14. Were appropriate statistical methods used and adequately described (including taking into account number of participants and clustering effects)?	**0.5**
AND/OR did the statistical methods address confounders?	**0.5**
15. Were the numbers/% of participants with missing data indicated?	**0.5**
AND if more than 20% of data in the primary analyses were missing, were methods used to address missing data?	**0.5**
TOTAL	15

Table 2: Description of included studies

Study	Study Design[1] +Setting	*n*	Age Sex Ethnicity[2]	PTSD Measure Current PTSD	Study Quality[3]	Dietary Measure	Dietary Reporting Quality[4]	Diet + PTSD	Diet + PTSD + Cardiometabolic Outcomes
de Vries *et al.* (2017) [42]	IV NL	94 PTSD group: *n*=49 Control group: *n*=45	46 ±10 years 70% Female 70% Dutch 12% Turkish 18% Other	SCID-I 52% with PTSD	80%	24-hour dietary recall	Poor	No differences in dietary intake (fats, protein + carbohydrates) between PTSD patients and controls p>0.05	LDL: PTSD>Controls, p<0.05 LDL:HDL PTSD<Controls, p<0.05 Controlled for dietary intake LDL: PTSD<Controls, p<0.05 LDL:HDL PTSD<Cont

									rols, p<0.05
									No difference in BMI or WC, p>0.05
Gavrieli *et al*. (2015) [40]	IV US	151	35–55 years 45.6±3.5 years 50% Female 50% African America n 50% European America ns	UCLA PTSD Scale 0 score: 51% 1-21 score: 24.5% 22-57 score: 24.5%	83%	FFQ aHEI-2010 DASH	Good	↑ PTSD score = ↓diet quality p<0.05 When diet controlled for education + other variables association was no longer significant	↑ PTSD score = ↑BMI p<0.05 ↑ PTSD score = ↑% body fat mass p<0.05 No difference in WC p>0.05 Did not control for

									diet
Godfrey *et al.* (2013) [41]	IV US	80 PTSD group: *n*=25 Control group: *n*=55	39.9±13.5 years 45% Female 21% Hispanic 19% Black 60% White	CIDI 31% with PTSD	77%	5-item questionnaire	Poor	PTSD diagnosis = ↓fruit intake p<0.05 No differences in vegetables, soda, caffeine, and fast food intake p>0.05	BMI: PTSD>Controls p<0.05 No difference in WHR or BP, p>0.05 Controlled for alcohol intake and physical activity; Did not control for diet
Hirth *et al.* (2011) [39]	IV	3154	16-24 years 20.8±2.5 years	PDSQ altered subscale	77%	5-item questionnaire	Poor	↑ PTSD symptoms = ↑fast	No differences in BMI

	US		100% Female 46% Hispanic 26% Black 28% White	0 Symptoms: 58% 1-3 Symptoms: 20% +4 Symptoms 18%				food and soda consumption p<0.05	Did not control for diet
Roberts *et al*. (2015) [36]	III-2 US	49739	25-42 years 100% Female 94% Caucasian	SSS-IV PTSD BTQ No trauma: 30% 0 symptoms: 50.9% 1-3 symptoms: 9.6% 4-5 symptoms:	87%	FFQ aHEI	Good	↑ PTSD symptoms = ↑diet quality p<0.05	↑ PTSD symptoms = ↑ BMI, p<0.05 ↑ PTSD symptoms = ↑ incidence of T2DM[5] p<0.05 Diet[6] did not significantly attenuate the

				5.8% 6-7 symptoms: 3.8%					relationship between PTSD and T2DM
Sumner *et al.* 2015 [38]	III-2 US	49978	25-42 years 100% Female 94% Caucasian	SSS-IV PTSD BTQ No trauma: 30% 0 symptoms: 51% 1-3 symptoms: 9.5% ≥4 symptoms: 9.5%	90%	FFQ aHEI	Good	↑ PTSD symptoms = ↑diet quality p<0.05	↑ PTSD symptoms = ↑ incidence of CVD[7] p<0.05 Diet[6] did not significantly attenuate the relationship between PTSD and CVD
Sumner *et al.* 2016 [37]	III-2 US	47514	25-42 years 100% Female 94%	SSS-IV PTSD BTQ No trauma:	83%	FFQ aHEI	Good	↑ PTSD symptoms = ↑diet quality	↑ PTSD symptoms = ↑ incidence of HTN[5] p<0.05

Caucasian	31% 0 symptoms: 50.2% 1-3 symptoms: 9.4% 4-5 symptoms: 5.6% 6-7 symptoms: 3.7%		p<0.05 ↑ PTSD symptoms = ↓sugar + artificially sweetened beverage consumption p<0.05	Diet[6] did not significantly attenuate the relationship between PTSD and HTN

Values are means ± standard deviation;[1]Grading based on National Health and Medical Research Council (NHMRC) Evidence Hierarchy; [2]Ethnicity as reported from source; [3]Study quality based on adapted method by Fletcher *et al* [34]; [4]Based on method by Burrows *et al* [35]; [5]Attenuated by body mass index and antidepressant usage; [6]Dietary intake plus other lifestyle variables; [7]Attenuated by hypertension, diabetes and antidepressant usage; PTSD = posttraumatic stress disorder; IV = Cross-sectional design; NL; the Netherlands; III-2 = Longitudinal cohort study; SCID-I = the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders 4th Edition; UCLA = University of California Los Angeles; CIDI = Composite International Diagnostic Interview; PDSQ = Psychiatric Diagnostic Screening Questionnaire; DSM-IV = Diagnostic and Statistical Manual of Mental Disorders 4th Edition; SSQ-IV PTSD = Short Screening Scale for DSM-IV PTSD; BTQ = Brief Trauma Questionnaire; FFQ = Food frequency questionnaire; aHEI = Alternate Health Eating Index; DASH = Dietary Approaches to Stop Hypertension; BMI = Body Mass Index; LDL = Low-density lipoprotein; HDL = High-density lipoprotein; WC = Waist circumference; WHR = Waist to hip ratio; BP = Blood pressure; T2DM = Type 2 Diabetes Mellitus; CVD = Cardiovascular disease; HTN = Hypertension

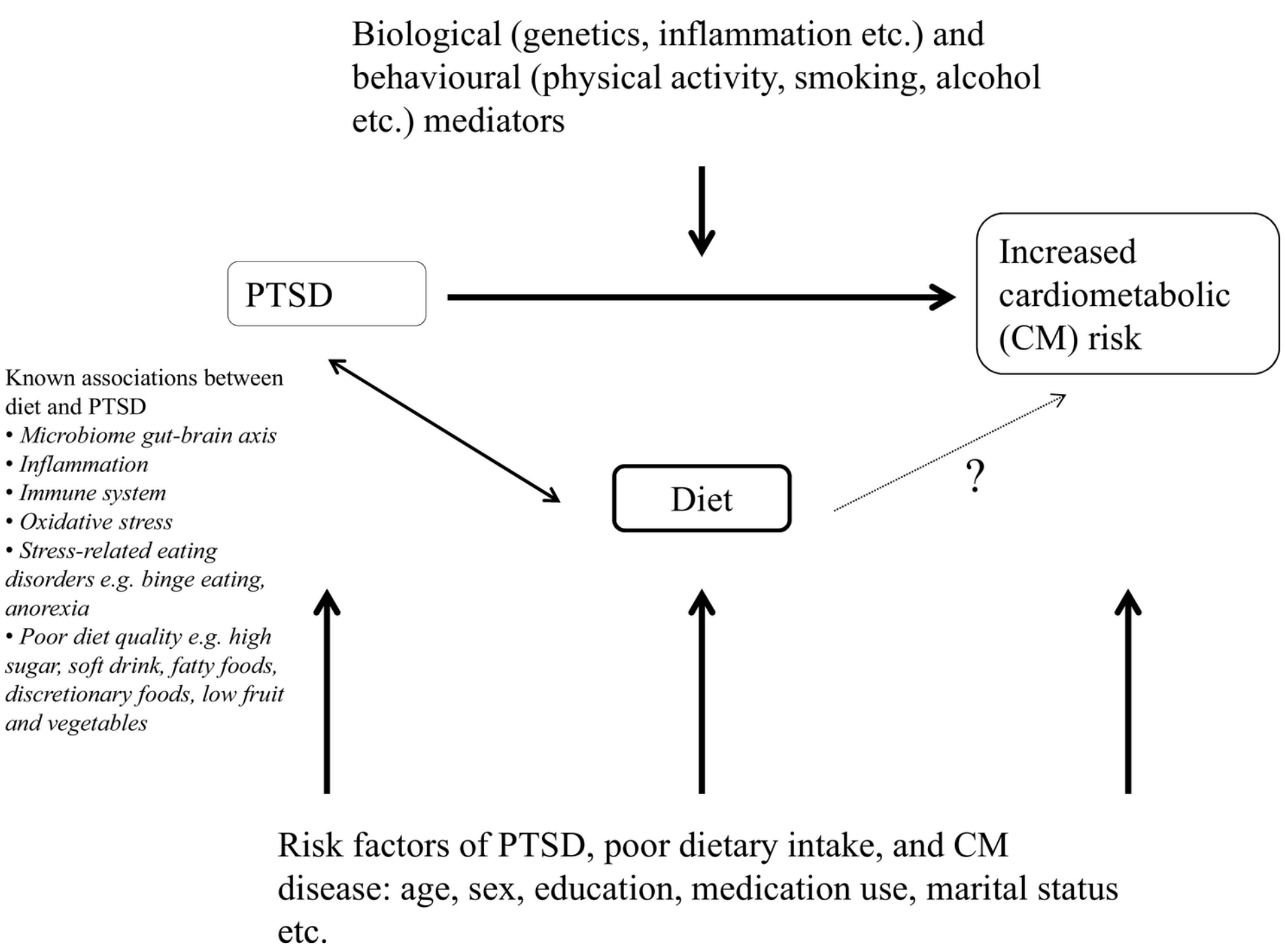
Biological (genetics, inflammation etc.) and behavioural (physical activity, smoking, alcohol etc.) mediators
PTSD
Increased cardiometabolic (CM) risk
Known associations between diet and PTSD
• Microbiome gut-brain axis
• Inflammation
• Immune system
• Oxidative stress
• Stress-related eating disorders e.g. binge eating, anorexia
• Poor diet quality e.g. high sugar, soft drink, fatty foods, discretionary foods, low fruit and vegetables
Diet
?
Risk factors of PTSD, poor dietary intake, and CM disease: age, sex, education, medication use, marital status etc.

Figure 1

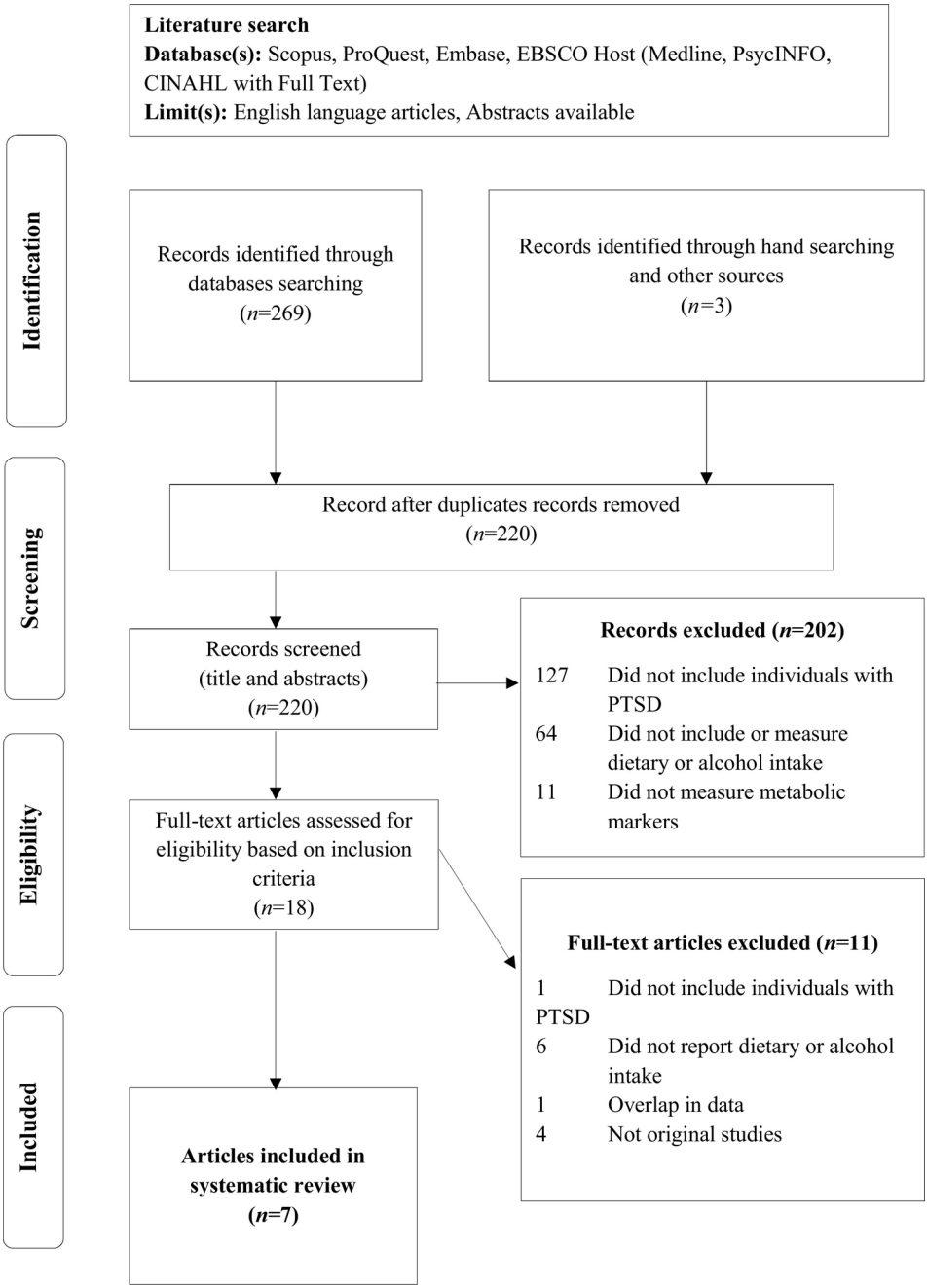
Literature search
Database(s): Scopus, ProQuest, Embase, EBSCO Host (Medline, PsycINFO, CINAHL with Full Text)
Limit(s): English language articles, Abstracts available
Identification
Records identified through databases searching (n=269)
Records identified through hand searching and other sources (n=3)
Screening
Record after duplicates records removed (n=220)
Records screened (title and abstracts) (n=220)
Records excluded (n=202)
127 Did not include individuals with PTSD
64 Did not include or measure dietary or alcohol intake
11 Did not measure metabolic markers
Eligibility
Full-text articles assessed for eligibility based on inclusion criteria (n=18)
Full-text articles excluded (n=11)
1 Did not include individuals with PTSD
6 Did not report dietary or alcohol intake
1 Overlap in data
4 Not original studies
Included
Articles included in systematic review (n=7)

Figure 2