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Review article

Health risk behaviours and allostatic load: A systematic review

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ABSTRACT

Health risk behaviours (HRB) across the lifespan have been associated with higher cumulative physiological burden as measured by allostatic load (AL). This study examines the contribution of HRB and their effects on multisystem biological risk associated with morbidity and early mortality. We systematically reviewed the literature to assess the links between HRB and AL. Twenty-six eligible human studies were included in our assessment of the current literature investigating the association of different HRB that included overeating/obesity, alcohol, smoking, drug use, physical inactivity and sleep impairments in relation to AL. We found that 50 % of obesity and substance abuse, 75 % of sleep and 62.5 % of combined HRB studies showed a significant association with AL. Lifestyle coping behaviours therefore have a significant contribution to AL. This study is among the first to explore multiple domains of HRB in relation to AL. Further research should focus on evaluating lifestyle factors that adapt HRB as a strategy to cope with chronic stress to help decrease AL and resulting long-term negative health consequences.

1. Introduction

Chronic, non-communicable disorders that are linked to health risk behaviours and chronic stress are becoming increasingly prevalent worldwide (World Health Organization, 2018a). Most health risk behaviours co-occur with chronic stress either as risk taking or as coping behaviours and contribute to various health problems such as cardiovascular, metabolic and inflammatory conditions or can delay recovery from some ongoing disease (McEwen, 1998b). Lifetime strain is a fundamental problem associated with daily life in today's societies (Shields and Slavich, 2017). Across the board, chronic stress is a risk factor for premature biological aging and early death (Epel et al., 2004). The knowledge and awareness that stressors exert cumulative physiological 'wear and tear' throughout lifespan development has encouraged researchers to evaluate specifically how chronic stress can cause such widespread detrimental effects on health.

1.1. Allostasis and allostatic load

The concept of allostasis was first developed to understand and recognise the physiological basis for disparate patterns of unexplained morbidity and mortality, for example, by socio-economic status (SES) and lifestyle factors that includes health behaviours (Sterling and Eyer, 1988). McEwen and Stellar (1993) proposed the construct of *allostatic load* (AL) to convey the cumulative impact of progressive physiological 'wear and tear' on the brain and the body that could predispose individuals to disease by causing deterioration of physical and mental health (McEwen, 1998a). Any form of long-term stress exerts negative effects on health via continued activation of the hypothalamic pituitary adrenal axis (HPA) and the autonomic nervous system (ANS). Our body adjusts to maintain homeostatic stability via allostatic mechanisms; however, when the exposure to stress becomes exaggerated and prolonged it results in the failure or dysregulations of these systems (McEwen and Stellar, 1993). The sympathetic adrenal medullary (SAM) axis and the HPA axis release primary stress mediators such as cortisol, epinephrine and norepinephrine activate adaptive effects resulting in altering the function and structure of specific cells and tissues (McEwen, 2000).

The cumulative outcome of primary effects in response to primary mediators leads to secondary outcomes whereby metabolic (e.g.,

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insulin, glucose, total cholesterol, high density lipoprotein cholesterol, triglycerides, visceral fat depositing), cardiovascular (e.g., systolic and diastolic blood pressure), and immune (e.g., fibrinogen, c-reactive protein (CRP)) parameters reach sub-clinical levels. The final stage of AL progression is allostatic overload, whereby physiological dysregulations leads to disordered, diseased, and deceased endpoints referred to as tertiary outcomes (Juster et al., 2010). The important feature of allostasis-adaptation is that these physiological mediators are interconnected with nonlinear outcomes. However, it is unclear how long it will take to develop secondary and tertiary outcomes once the primary stress mediators are activated (Juster et al., 2010).

There are different methods in computing sources of health-related effects of stress (Kopp et al., 2010). The most frequent approach used in measuring an AL index that encompasses primary mediators and secondary outcomes to predict tertiary outcomes was developed by Seeman et al. (1997). The original AL index included 10 biomarkers from neuroendocrine (cortisol, adrenalin, noradrenalin, and dehydroepiandrosterone-sulphate [DHEAS]), metabolic (total cholesterol to high density lipoprotein [HDL] cholesterol, HDL cholesterol, glycosylated haemoglobin [HbA1c], and waist to hip ratio), and cardiovascular (systolic and diastolic blood pressure) systems (Seeman et al., 1997). These indicators contribute in the evaluation of the cumulative effects of psychosocial factors on health and wellbeing due to the damaged systems (Seeman et al., 1997).

The AL model proposes that by measuring various biomarkers, biomedical advances can be made in the early detection of tertiary outcomes in high risk individuals (McEwen, 2000; McEwen and Seeman, 1999). Physicians incorporate many of these biomarkers; however, less attention is given to identifying pre-clinical values along with other measures if feasible, e.g., psychosocial, genotypes, and phenotypes (Juster et al., 2010). This will help in the early prediction of some of the co-morbid diseases such as cardiovascular, metabolic and inflammatory because mediators leading to AL and disease susceptibilities interact in a non-linear manner whereby fluctuations in values induce compensatory remediation over time and delineating time-courses of dysregulation is rather difficult (McEwen, 2008).

1.2. Health behaviours

Risk behaviour has been defined as "behaviours that increase the likelihood of adverse physical, social, or psychological consequences" (as cited by M D Resnick, Professor of Pediatrics, School of Medicine, and Professor of Public Health, University of Minnesota, personal communication) (Carr-Gregg et al., 2003). Healthy risk behaviours such as alcohol and substance abuse, as well as consumption of unhealthy foods have bidirectional relationships with chronic stress and therefore with AL (McEwen, 1998b). Besides, chronic stress has an adverse effect on physical activity resulting in obesity (Scott et al., 2012). These relationships are interchangeable resulting in impacts on biophysical and psychological system, which later exacerbate AL.

There has been growing consensus that further research is needed to delineate the relationship between health behaviours and mental illness which leads to the use of certain coping strategies such as alcohol, use of nicotine and other recreational drugs to relieve negative emotions and other mental illnesses (Lazarus and Folkman, 1984; Wills and Shiffman, 1985). This results in maladaptation of physiological systems further deteriorating existing diseases.

Health risk behaviours (HRB) have been found to correlate with increased morbidity and mortality. These are 1) consuming diet that is high in fat, sugar, sodium but low in important nutrients intake of trans–fatty acids and foods with a high glycaemic index (Danaei et al., 2009; Mente et al., 2009), 2) living a sedentary life with very little physical activity (Fogelholm, 2010; Qin et al., 2010) 3), smoking (Lloyd-Jones Donald et al., 2010), and 4) substance abuse, such as with alcohol (Carlsson et al., 2005; Costanzo et al., 2010) and illicit drugs (Coughlin and Mavor, 2006); as well as 5) sleep abnormalities (McEwen, 2006).

Research shows that health compromising behaviours are more common among people living with stress, depression, and anxiety (Ameringer and Leventhal, 2010; Conner et al., 2009; Ng and Jeffery, 2003; Steptoe et al., 1996, 1998). This relationship has been found for a variety of health behaviours, including substance use (tobacco, alcohol, and illegal drugs) (Ameringer and Leventhal, 2010; Conner et al., 2009; Hassan and Ali, 2011; Jané-Llopis and Matytsina, 2006); sedentary behaviour and high body mass index (Leas and McCabe, 2007) and sleep quality (Kim and Dimsdale, 2007).

Smoking, drinking, disturbed sleep, and physical inactivity have the potential to promote and exacerbate pathophysiology by dysregulating key biological processes, such as inflammation (Friedman et al., 2005; Parrott, 1999; Salmon, 2001). Physical training has antidepressant and anxiolytic effects and protects against harmful consequences of stress on both physical and mental health (Salmon, 2001). There is existing literature disclosing an association between health risk behaviour and low SES and health risk behaviour and mental illness (Walsh et al., 2013). Nevertheless, very few studies have investigated the literature for the links between risk behaviour-mental health relationship in association with AL. There is an emerging interest in exploring health enhancing lifestyle behaviours and their potential beneficial effects on reducing AL and thus subsequent disease burden (Fig. 1). This study examines the contribution of HRB and their effects on increasing multisystem biological risk associated with morbidity and mortality. The aim of this systematic review was to identify and assess whether unhealthy behaviours mediates the relationship between allostatic load and the risk of developing multisystem physiological dysregulation.

2. Methods

2.1. Study selection

Original research articles, investigating HRB and AL in individuals were systematically identified by searching the databases MEDLINE OVID, Scopus, PsycINFO Quest and PubMed. Publications eligible for review were those published from 1988 to August 2018. The search

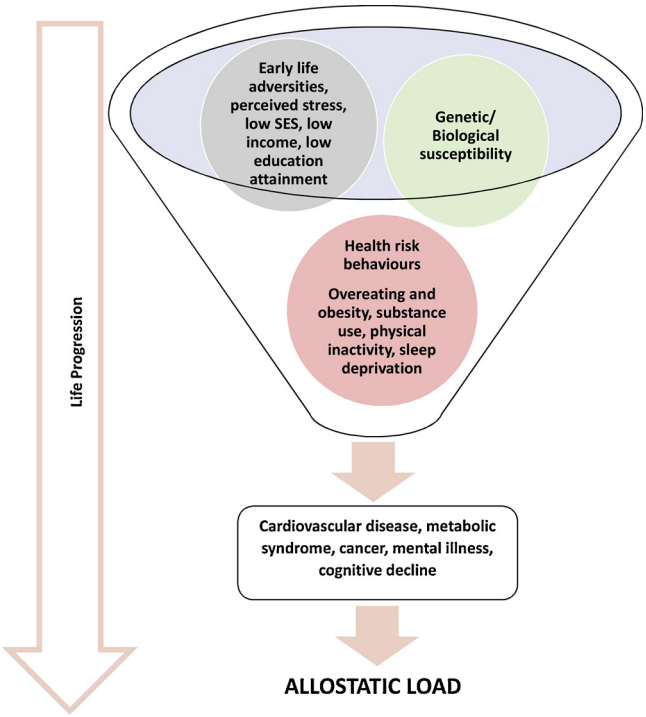

Fig. 1. Pathways linking health and allostatic oad.

strategy included the search terms “health behaviours”, “unhealthy behaviours”, “HRB “sleep disturbances”, “fast foods”, “processed foods”, “obesity”, “over-eating, “AL”, “allostasis”, “chronic stress”, “smoking”, “alcohol”, “substance abuse”, “drug addiction”, “sedentary lifestyle”, “physical inactivity”, “alcohol + AL”, “smoking + AL”, “substance of abuse + AL”, “drug addiction + AL”, “physical inactivity + AL”, “over eating + AL”, “obesity + AL”, “HRB + AL”, “alcohol + stress”, “smoking + stress”. The methods of the analysis were as specified following the Preferred Reporting Items for Systematic Reviews and Meta-Analysis statement or PRISMA.

2.2. Inclusion and exclusion criteria

Eligibility criteria included 1) calculation of an AL index or similar algorithm assessing physiological function with several biomarkers, 2) studies reporting HRB; such as alcohol, drugs of abuse, smoking, sleep, physical inactivity, over-eating, fast foods and obesity in relation to AL; 3) human studies; 4) full text articles; 5) articles written in English; and 6) no limitations on participant demographics (age, sex, ethnicity, and SES).

Exclusion criteria were: 1) animal studies; 2) studies that analysed only cortisol concentrations; 3) review articles; 4) editorials or commentaries; and 5) book chapters. In the event of an abstract that was unavailable, the full text was screened to determine the eligibility of the article. Two reviewers (BS & ZS) screened the full text of the articles for suitability.

2.3. Data extraction

The following elements were abstracted: authors, year of publications, number and type of physiological biomarkers (Seeman et al., 1997) following previously used classifications (Juster et al., 2010), number of participants, age, sex, study sample (mean, range and standard deviation), country and study findings. In line with the AL concept, primary mediators comprise of neuroendocrine factors (e.g., HPA axis and sympathetic-adrenal-medullary [SAM] axis), neurophysiological factors (e.g., vagal activity), and anti-inflammatory markers (e.g., cytokines). More downstream of primary mediators, secondary outcomes were categorized into four categories: metabolic factors, inflammatory markers, cardiovascular factors, and organ functions.

2.4. Assessment of study quality

Three authors (BS, AS and RP) conducted a quality assessment using the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Checklist (Von Elm et al., 2007). No modifications to this checklist were made and it consisted of a total of 36 items, which evaluated the reporting of each study’s title, abstract, introduction, methodology, results, and discussion. Each item was evaluated as ‘reported’, ‘not reported’, or ‘non applicable’. The number and percentage of the studies meeting each category, was reported. The completeness of reporting (COR) score was calculated for each study using the formula: COR (%) = (yes/(yes + no)) x 100). Studies were given an overall COR score of 'moderate’ (if 50–74% of items were met) or ‘high’ (if ≥75 % of items were met).

3. Results and discussion

3.1. Search results

The search strategy identified 1335 studies from four databases. In addition, 12 papers were included from cross-referencing, book chapters and dissertations resulting in a total of 1347 studies (Fig. 2). After the removal of duplicates, 484 articles were screened for inclusion. We excluded 349 studies during the screening process based on their title and abstract and whether they were review articles, animal studies or not accessible in the form of full text. We were able to retrieve full texts for 135 articles and they were all screened again for eligibility. One hundred and nine studies were excluded on the basis of study design (they did not specifically measure AL biomarkers or HRB or the association between these variables). In summary, 26 studies fulfilling our inclusion criteria were included in the review (Fig. 2).

3.2. Biomarker selection and measurement

A variety of different biomarkers, ranging between 2 and 25, were used across the studies to calculate AL indices. Of the 26 studies analysed, 17 studies used 6–10 biomarkers (9.12 ± 1.41 [Mean ± SD]), 5 studies used 11–20 biomarkers (12.6 ± 1.81) and only 4 studies used 21+ biomarkers (23.25 ± 0.96). Across the 26 studies in the review no study used the same biomarkers in calculating the AL index (Fig. 3) as in the original MacArthur study (Seeman et al., 1997). The commonly shared biomarkers are blood pressure (systolic and diastolic), glycated haemoglobin, high density lipoproteins, total cholesterol, C-reactive protein/high sensitivity CRP, body mass index, waist to hip ratio, triglycerides appearing in most of the studies. The other biomarkers used hardly overlapped and have not been used much until recently. The other biomarkers were used in less than 23 % of studies and have only been cited in the last 4 years. (Fig. 4).

3.3. Study characteristics

Of the 26 full text articles identified through our literature search and screening procedure, six studies focused on eating habits/obesity, two studied physical activity, four studied sleeping habits, six studied substance abuse and smoking and eight studied multiple HRB and AL in general. The latter also included studies that influence HRB based on racial/ethnic disparities. The reviewed articles included mostly cardiovascular and metabolic biomarkers. Only some studies measured the stress hormone cortisol in conjunction with these biomarkers. Overall, these studies used either cross-sectional or longitudinal designs. The participants (both male and female) within these studies were from different race/ethnicities, cultures and socioeconomic backgrounds.

3.4. Quality assessment

A STROBE Checklist quality assessment was performed on 26 studies as shown in Table 1. The most reported items were the description of abstract (item 1b, 100 %), background/rationale (item 2, 100 %), objectives (item 3, 100 %), study design (item 4, 100 %), settings (item 5, 100 %), participants eligibility criteria (item 6a, 100 %), variables (item 7, 100 %), data sources/management (item 8, 100 %), study size (item 10, 100 %), quantitative variables (item 11, 100 %), other analyses (item 17, 100 %) and key results (item 18, 100 %). When applicable, the least reported items included matching bias (Item 9, 11.5 %) and the reasons for non-participation (Item 13b, 23.1 %). Partial reporting was often associated with low scores. Of the 26 studies, four studies had COR score of 50 %–74 % whilst the other 22 studies scored greater than or equal to 75 %. The mean COR score was 77.8 ± 1.14 %. Therefore, on average, the reviewed studies met the criteria for just over 75 %, applicable to their study, outlined in the STROBE Checklist (Table 1).

3.5. Association between allostatic load (AL) and health risk behaviours

There was considerable heterogeneity in the measurement of HRB whether it be eating habits/obesity, physical inactivity, substance abuse (smoking, alcohol, drugs of abuse) and sleep. In our systematic review we focused on the association between HRB and AL. Table 2 details key findings from 26 studies incorporating HRBs (eating habits/obesity, physical inactivity, substance abuse ([smoking, alcohol, drugs of abuse], sleep and combined HRB) and allostatic load. Of the 4 sleep

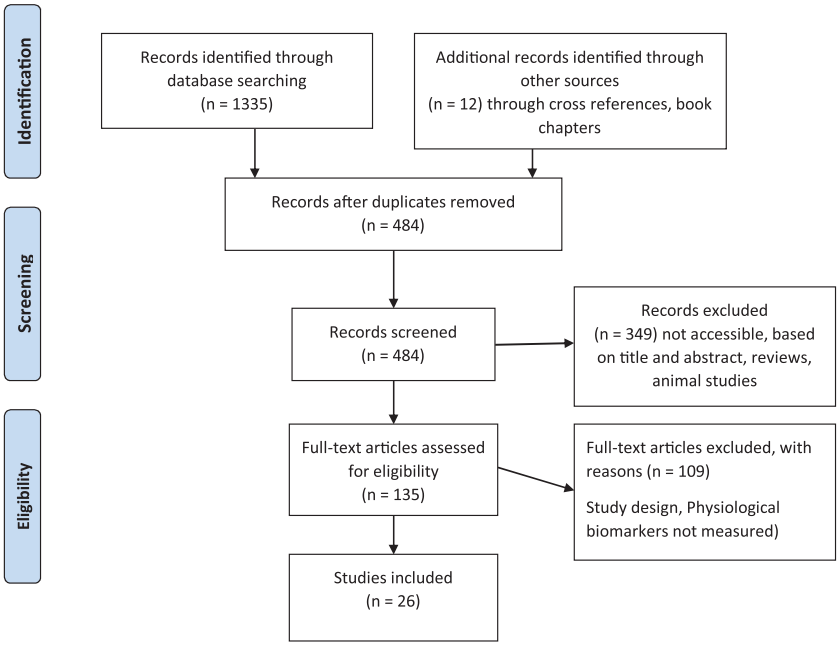

Fig. 2. Flow diagram of literature search and study inclusion.

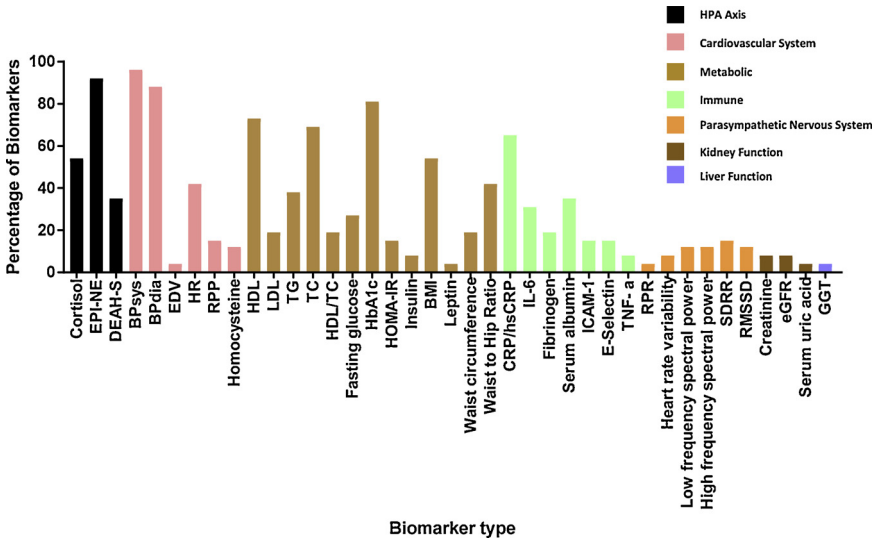

Fig. 3. Percentage of biomarkers used in studies.

studies analysed, impaired sleep quality was associated with higher AL in 3 of them. In addition, we identified six studies focusing on obesity and AL. All of them reported that obesity was associated with an increased AL. Furthermore, of the 6 substance abuse (smoking and alcohol/drugs) studies, a significant association was found in between substance abuse and elevated AL in three of the studies. Moreover, decreased physical activity (or inactivity) correlated with higher AL in both studies specifically investigating this relationship. Finally, of the 8 combined HRB studies, 5 studies demonstrated that a combination of different HRBs also predict an elevated AL.

We identified substantial heterogeneity in terms of the use of biomarkers used to quantify AL. Currently, there is no standard set of biomarkers to calculate AL indices explaining the wide range of biomarkers identified in our review article. Environmental factors including smoking, alcohol, lack of physical activity and diet choice are all major contributors to AL where an individual likely engages in these unhealthy behaviours to cope with a stressful challenge (McEwen, 1998b).

3.5.1. *Eating habits/obesity and AL*

Unhealthy eating habits and obesity have been linked to high AL. Studies on eating habits and AL have revealed that high intake of meat, sweets and French fries was significantly associated with elevated AL, while the traditional Puerto Rican diet of rice, beans and oil was not (Mattei et al., 2011). However, a significant association between increased sweets consumption and heightened AL disappeared after restricting analysis to participants without diabetes and a significant difference was found in diabetics, however no data was shown (Mattei et al., 2011). Similarly, high AL was associated with consumption of fast foods, sugar-sweetened beverages and artificially sweetened beverages (van Draanen et al., 2018).

A diet high in vegetables, fresh fruits, whole grains, and legumes, as well as in lean, low-fat protein sources such as American Heart Association diet (AHA), a diet rich in fruits and vegetables, decreased AL resulting in an improvement of the metabolic syndrome (Kusano et al., 2016; Mattei et al., 2013; Petrovic et al., 2016). High sodium intake was related to an increased AL in both women and men (Kusano

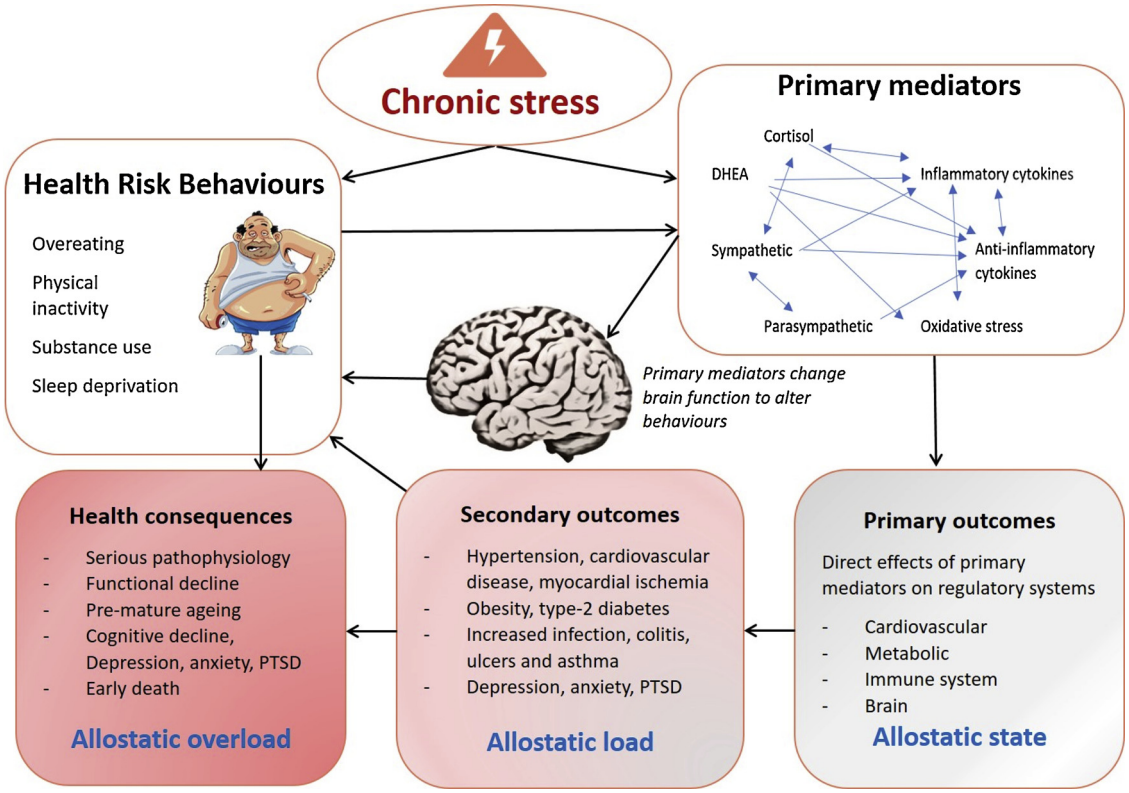

Fig. 4. Biological Mechanism of Allostatic Load.

et al., 2016; Petrovic et al., 2016).

In addition, the associations between consumption of certain types of food and AL, there is a significant relationship between food insecurity and primary mediators of AL. Food insecurity is the lack of persistent access to healthy, sufficient and safe food. The findings indicated that food insecurity was associated with primary system (neuroendocrine, inflammatory, HPA axis, SNS) dysregulation and this correlation is much stronger for those who experience food insecurity and not taking part in Supplemental Nutrition Assistance Program (SNAP) (McClain et al., 2018).

Individuals experiencing discrimination based on a person's weight (weight discrimination) had twice the risk of high AL compared to those who did not experience weight discrimination despite being obese. This raises the possibility that elevated AL can develop through both physiological and psychological mechanisms in individuals with obesity (Vadiveloo and Mattei, 2017).

High AL is associated with alterations in the grey matter structure of areas in the brain that regulate behaviour, sensory reward processing and cognition, particularly in obese individuals (Ottino-González et al., 2017). Regions of the prefrontal cortex that are affected in overweight as well as stressed individuals include orbitofrontal, dorsolateral (middle and superior frontal gyrus), ventrolateral portions (inferior frontal gyrus) and medial portions of the prefrontal cortex (medial superior frontal gyrus) (Ottino-González et al., 2017). The possible functional implications of such alterations are important with regards to health risk behaviours. The orbitofrontal region plays a role in reward processing whilst the dorsolateral and ventrolateral regions play a role in regulating behaviour. Lastly, the medial portion of the prefrontal cortex plays a role in motivation and decision making (García-García et al., 2013; Marqués-Iturria et al., 2015). The abnormal functioning of these circuits may contribute in the emergence and maintenance of HRBs. Cortical thinning was observed in overweight individuals, whereas in lean individuals, cortical thickening was found in relation to increased AL (Ottino-González et al., 2017). This interaction occurred in several bilateral anterior (frontal and prefrontal cortex) and posterior (temporal and parietal cortex) cortical regions. Some of these areas are part of networks involved in monitoring behaviour, including eating behaviour, sensory-reward processing and support basic cognitive abilities, such as memory and attention (Ottino-González et al., 2017).

Obesity is a serious, relapsing metabolic disorder influenced by early life environmental determinants (Brantley et al., 2005; Luppino et al., 2010; Nyberg et al., 2018; Jaacks et al., 2019). The above findings on the relationship between overweight/obesity and increased AL emphasise the importance of an individual's food choices and behaviours as they can lead to widespread multi-system dysregulation manifested as increased AL.

According to the World Health Organization in 2016, more than 1.9 billion adults (18+) were classified as overweight, while over 650 million adults were classified as obese. Alarmingly, the prevalence of obesity has tripled between 1975 and 2016 (World Health Organization, 2018c). Our systematic review identified a correlation between diet and AL; that is, unhealthy eating habits and obesity are clearly associated with higher AL (van Draanen et al., 2018; Mattei et al., 2011, 2013; McClain et al., 2018; Ottino-González et al., 2017).

Unhealthy eating habits such as high intake of meats, salt, sweets, fast foods and soft drinks have been linked to obesity (Noel et al., 2009; Paradis et al., 2009) and are risk factors for most major chronic, non-communicable diseases, such as cardiovascular disease, metabolic syndrome and cancer (Heidemann et al., 2008). Chronic stress brings about changes in the HPA axis which has been proposed as one of the main biological mediators of the interaction between stress and poor health. During stress, high circulating cortisol increases appetite by changing food preferences to comfort foods, such as fatty and sugary foods (Pasquali et al., 2010). It has been previously shown that individuals consuming high saturated fat, sweets, junk/fried/fast foods will exhibit elevated cortisol levels (Duong et al., 2012; Laugero et al., 2011). The link between diet and cortisol appears to be intricate and bidirectional, although the role of diet on HPA axis dysregulation is well supported (Laugero et al., 2011).

The association between fast-food and AL leads to increased risk of

Table 1
Number of items met by each study (N = 26) using the STROBE Checklist and their respective Completeness of Reporting Score (COR).

Section	Item	Recommendation	Criteria Met N (%)		
			Yes	No	N/A
Title and abstract	1a	Indicate the study's design with a commonly used term in the title or the abstract	11 (42)	15 (58)	0 (0)
	1b	Provide in the abstract an informative and balanced summary of what was done and what was found	26 (100)	0 (0)	0 (0)
Introduction					
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported	26 (100)	0 (0)	0 (0)
Objectives	3	State specific objectives, including any prespecified hypotheses	26 (100)	0 (0)	0 (0)
Methods					
Study design	4	Present key elements of study design early in the paper	26 (100)	0 (0)	0 (0)
Setting	5	Describe the setting and locations.	26 (100)	0 (0)	0 (0)
Participants	6a	Cohort study—Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up Case-control study—Give the eligibility criteria, and the sources and methods of case ascertainment and control selection. Give the rationale for the choice of cases and controls Cross-sectional study—Give the eligibility criteria, and the sources and methods of selection of participants	26 (100)	0 (0)	0 (0)
	6b	Cohort study—For matched studies, give matching criteria and number of exposed and unexposed Case-control study—For matched studies, give matching criteria and the number of controls per case	6 (23)	2 (7.7)	18 (69.2)
Variables	7a	Clearly define all outcomes, exposures, and predictors.	26 (100)	0 (0)	0 (0)
Data sources/ measurement	8	For each variable of interest, give sources of data and details of methods of assessment (measurement). Describe comparability of assessment methods if there is more than one group	26 (100)	0 (0)	0 (0)
Bias	9	Describe any efforts to address potential sources of bias	3 (11.5)	23 (88.5)	0 (0)
Study size	10	Explain how the study size was arrived at	26 (100)	0 (0)	0 (0)
Quantitative variables	11	Explain how quantitative variables were handled in the analyses. If applicable, describe which groupings were chosen and why	26 (100)	0 (0)	0 (0)
Statistical methods	12a	Describe all statistical methods, including those used to control for confounding	24 (92.3)	2 (7.7)	0 (0)
	12b	Describe any methods used to examine subgroups and interactions	24 (92.3)	2 (7.7)	0 (0)
	12c	Explain how missing data were addressed	16 (61.5)	10 (38.5)	0 (0)
	12d	Cohort study—If applicable, explain how loss to follow-up was addressed Case-control study—If applicable, explain how matching of cases and controls was addressed Cross-sectional study—If applicable, describe analytical methods taking account of sampling strategy	21 (80.8)	5 (19.2)	0 (0)
	12e	Describe any sensitivity analyses	15 (57.7)	11 (42.3)	0 (0)
Results					
Participants	13a	Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed	24 (92.3)	2 (7.7)	0 (0)
	13b	Give reasons for non-participation at each stage	6 (23.1)	20 (76.9)	0 (0)
	13c	Consider use of a flow diagram	3 (11.5)	23 (88.5)	0 (0)
Descriptive data	14a	Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders	25 (96.2)	1 (3.8)	0 (0)
	14b	Indicate number of participants with missing data for each variable of interest	4 (15.4)	22 (84.6)	0 (0)
	14c	Cohort study—Summarise follow-up time (eg, average and total amount)	2 (7.7)	5 (19.2)	19 (73.1)
Outcome data	15a	Cohort study—Report numbers of outcome events or summary measures over time	6 (23.1)	1 (3.8)	19 (73.1)
	15b	Case-control study—Report numbers in each exposure category, or summary measures of exposure	N/A	N/A	N/A
	15c	Cross-sectional study—Report numbers of outcome events or summary measures	19 (73.1)	0 (0)	7 (26.9)
Main results	16a	Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95 % confidence interval). Make clear which confounders were adjusted for and why they were included	20 (76.9)	6 (23.1)	0 (0)
	16b	Report category boundaries when continuous variables were categorized	22 (84.6)	4 (15.4)	0 (0)
	16c	If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period	0 (0)	0 (0)	0 (0)
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses	26 (100)	0 (0)	0 (0)
Discussion					
Key results	18	Summarise key results with reference to study objectives	26 (100)	0 (0)	0 (0)
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision. Discuss both direction and magnitude of any potential bias	25 (96.2)	1 (3.8)	0 (0)
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence	25 (96.2)	1 (3.8)	0 (0)
Generalisability	21	Discuss the generalisability (external validity) of the study results	23 (88.5)	3 (11.5)	0 (0)
Other information					
Funding	22	Give the source of funding and the role of the funders for the present study and, if applicable, for the original study on which the present article is based	24 (92.3)	2 (7.7)	0 (0)
Completeness of Reporting Mean of 26 Studies (%)			**77.8** ± **1.**14 %		

Table 2
Review of studies incorporating health risk behaviours and allostatic load.

Authors, years	Sample (nationality: type)	N	Age (mean ± SD; range) > > time	Sex (M/F)	Ethnicity	Total no AL Biomarkers	Covariates/Adjustments	↑AL main findings
Eating habits and Obesity								
van Draanen et al. (2018)	National Longitudinal Study of Adolescent to Adult Health, US	11,562	24-34	M & F	White 69.7 Black 15.0 Hispanic 12.0 Asian 3.3	10 SBP, PR, PP, waist circumference, BMI, Hb1Ac, TC, HDL, hsCRP, EBV capsid antigen IgG	Adjusting for demographics, and medication usage	Fast foods, sugar-sweetened & artificially sweetened beverages→ ↑AL
McClain et al. (2018)	Longitudinal Boston Puerto Rican Health study	733	45-75	M & F	Puerto Ricans	11 Serum DHEA-S, urinary (cortisol, epinephrine & norepinephrine), CRP, waist circumference, Hb1Ac, BP (SBP, DBP), plasma lipids, HDL, TC	Baseline AL, sociodemographic, cultural, behavioural	Food insecurity exhibits strong association → ↑ AL biomarkers
Ottino-González et al. (2017)	Spain	63	21-40	M & F	Spanish	15 SBP, DBP, HbA1c, glucose, creatinine, TC, HDL, LDL, TG, CRP, IL-6, insulin, cortisol, fibrinogen, leptin	Age, years of education, gender and waist circumference	Changes in the grey matter → ↑AL (Cortical thinning in brain in obese and cortical thickening in lean individuals)
Vadiveloo and Mattei (2017)	MIDUS	986	25-75	M & F	White 93.1 % Black 2.6 % Other 4.4 %	24 Resting (SBP, HR, PP), BMI, WHR, TG, HDL, LDL, HbA1c, FBG, HOMA -IR, CRP, IL-6, Fibrinogen, E-selectin, sICAM, (Urine Epinephrine, Norepinephrine, cortisol), blood DHEA-S, SDRR, RMSSD, low frequency spectral power, high frequency spectral power	Age, race, household income, education level, smoking, physical activity	Perceived weight discrimination →↑AL
Mattei et al. (2013)	Boston Puerto Rican Heart study	1318	45-75	M & F	Puerto Ricans	10 SBP, DBP, serum DHEA-S, urinary (norepinephrine, epinephrine, cortisol), HDL, TC, Hb1Ac, waist circumference	Age, sex, smoking behaviour, educational level, household income, medication use, PSS, physical activity, MI	AHA diet associated with ↓ AL and MetS variables (diverse results by sex)
Mattei et al. (2011)	Boston Puerto Rican Health study	1,117	45-75 Mean age = 57.5 ± 7.5	M & F	Puerto Ricans	10 SBP, DBP, serum DHEA-S, urinary (cortisol, epinephrine, norepinephrine), HDL, TC, Hb1Ac, waist circumference	Age, sex, alcohol intake, smoking, medications, energy intake, BMI or physical activity	Meat, processed meat, French fries → ↑AL
Physical inactivity								
Gay et al. (2015)	Cameron county Hispanic cohort, Mexican American adults	330	≥18yrs	M&F	Mexican American	10 SBP, DBP, HR, TC, HDL, BMI HbA1c, CRP, TNFα IL6	Age, sex, and education	↑Physical activity → ↓AL & ↓Inflammatory markers
Upchurch et al., 2015	NHANES -White, Black, and Mexican American midlife women.	1680	40- 59	F	Non-Hispanic White, Non-Hispanic Black, and Mexican American	10 SBP, DBP, PR, Homocystéine, BMI, HbA1c, HDL, TC, Serum Alb, CRP	Age, ethnicity/race, physical activity	↑Physical activity → ↓AL
Substance use and Smoking								
Chen et al. (2015)	SHAPE (Strong African American Families Healthy Adult Panel)	452	≥18yrs	M & F	African American	6 Overnight cortisol, epinephrine, norepinephrine; resting SBP & DBP; BMI	Gender, neighbourhood ethnicity, cumulative family Socioeconomic status risk, factors related to participants college experiences	Youth attending college and ↓ substance use (high poverty) → ↑AL
Robertson et al. (2015)	West of Scotland	999	35-55yrs	M&F	South Asians (Punjabi with origins in the Indian subcontinent) & Irish Catholic descent	9 SBP, DBP, PR, Hb, TC, HDL WHR, CRP Serum alb	Sex adjustment	↑Socioeconomic position (SEP) and ↓AL Smoking ↑ in low SEP ↑ Smoking = ↑ AL

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Table 2 (*continued*)

Authors, years	Sample (nationality: type)	N	Age (mean ± SD; range) > > time	Sex (M/F)	Ethnicity	Total no AL Biomarkers	Covariates/Adjustments	↑AL main findings
Doan et al. (2014)	New York Co-Operative Extension and Public-school districts	162	9-17 Mean age = 9.18	M	White	6 Overnight Cortisol, epinephrine, norepinephrine, DBP, BMI	Age, race and gender	Smoking ↑link b/w Socioeconomic status & AL→↑AL
Gallo et al. (2011)	Mexican American San Diego (California-Mexican border)	301	40-65	F	Mexican American	12 SBP, DBP, waist circumference, TC, HDL, TC/HDL, Hb1Ac, urinary (cortisol, epinephrine, norepinephrine), CRP, IL-6, TNF-α	Age, menopausal status, SES, employment, marital status, health insurance status, language assessment	Alcohol ↓ AL
Hu et al., 2007	SEBAS 2000 +Survey of Health and Living Status of the Near Elderly and Elderly in Taiwan	1023	Mean age = 67.9 (SD 8.5Y; Median 68Y)	M & F	Taiwanese	10 SBP, DBP, TC, HDL, Hba1c, WHR, serum DHEA-S, urinary (cortisol, adrenaline, noradrenaline)	Education, income	Alcohol ↓ AL
Crimmins et al., 2009	NHANES III (1988 – 1994) & NHANES (1999 – 2004)	12,000	≥ 20	F	White and others- 80.55 % in NHANES III & 75.93 % in NHANES (1994–2004), Black- 10.62 % in NHANES III & 10.37 % NHANES (1994–2004), Hispanic - 8.83 % in NHANES III & 13.69 % in NHANES (1994–2004)	9 SBP, DBP, PR, TC, HDL, BMI, Hb1Ac, CRP, alb	Current smoking, heavy drinking, no exercise	Physiological biomarkers for high risk disease and mortality increases with age amongst poor and have low income individuals
Sleep habits								
Bei et al. (2017)	MIDUS-2 study	463	Mage ± SD = 54.1 ± 11.7, 60.3 % F	M&F	White: 305 (70.1 %) Africa American: 122 (28.0 %) Other: 8 (1.8 %)	23 Resting PP, SBP, HOMA-IR, FBG,HbA1c,Cortisol, blood serum DHEA-sulfate,CRP,IL6,Fibrinogen, soluble E-Selectin, Soluble intra cellular adhesion molecule 1, TG, HDL, LDL, WHR, resting PR and measures of HR variability, SDRR, RMSSD, low and high freq spectral power, 12 hr overnight Urinary epinephrine, norepinephrine,	Sex (women/men), age, race, education, employment status, bed partner, presence of depression or generalized anxiety disorder, alcohol, smoking, physical activity, perceived stress, chronic major medical conditions, cortisol medications, waist-to-hip ratio. Age, sex, W:H ratio not included -as it was included in AL system	Sleep patterns → blunted diurnal cortisol trajectories. Mean bedtime associated with→ ↑ AL Association b/w sleep intraindividual variability & AL appeared weak after accounting for covariates
Carroll et al. (2015)	MIDUS	1023	25-74 Mean age 54.5, SD = 11.8	M & F	White: 77.6 % Non-White: 22.4 %	22 SBP, DBP, PP, HR, TG, HDL, LDL, BMI, WHR, FBG, HbA1c, HOMA-IR, CRP, IL-6, e-Selectin, ICAM-1, fibrinogen, (urinary-epinephrine, norepinephrine), HRV, SD of R-R intervals, low frequency (LF) and high frequency (HF) spectral power), (urinary cortisol, serum DHEA-S)	Age, gender, race, SES education, income/poverty ratio, BMI	Poor sleep quality→ ↑ AL
Chen et al. (2014)	NHANES – US African American or Hispanic Americans	3330	≥18yrs	M&F	Whites-1,593, African Americans-712, Hispanic Americans-635, and other racial/ethnic group-390	9 SBP, DBP, HR, TC, HDL BMI, HbA1c, CRP, alb	Sociodemographic and life style factors	Sleep apnoea, insomnia, Short sleep duration and sleep disorder associated with → ↑AL
		5226	49-63yrs m-54yrs		Danish			

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Table 2 (*continued*)

Authors, years	Sample (nationality: type)	N	Age (mean ± SD; range) > > time	Sex (M/F)	Ethnicity	Total no AL Biomarkers	Covariates/Adjustments	↑AL main findings
Clark et al. (2014)	Danish Copenhagen Aging and Midlife Biobank			M & F		9 SBP, DBP, hsCRP, TG, HbA1c, WHR, HDL/TC, IL6	Adjusting for age, sex education, income, race/ethnicity, BMI comorbid chronic condition	Poor sleep quality, short sleep(< 5 h) and long sleep (> 8.5 h) → ↑AL
Combined HRBs								
Kim et al. (2018)	Korean Metabolic Syndrome Mortality Study Korea	70, 713	40-79	M & F	Koreans	7 SBP, DBP, PR, Hb1Ac, HDL, TC, alb	Age, sex, marital status, MI	High Socioeconomic position → ↓AL (health behaviours are the mediators link between socioeconomic status and mortality)
Rodriquez et al. (2018)	NHANES	12,272	40-79	M & F	African American or Black, Latino or Hispanic, and White.	10 SBP, DBP, BMI, HbA1c, TC, HDL, Total/HDL cholesterol ratio, CRP, alb, creatinine clearance	Age, gender, education levels, mon-missing data for all variables	Unhealthy behaviours→ did not alter AL Engaging in unhealthy behaviours-associated with depressive disorders
Bingham et al., 2016	African Immigrants in US	238	age 40 ± 10 (mean ± SD), range 21–64 years, 27.8 ± 4.4)	M&F	African Americans	10 SBP, DBP, TC, TG, homocysteine, BMI,HbA1C, alb, eGFR, hsCRP	Age	↑Age → ↑ AL (Immigration after adulthood → ↑AL)
Petrovic et al. (2016)	Swiss study	803	Mean age 48 ± 16	M & F	Caucasian origin	14 (Mean -SBP, DBP, HR), Blood glucose, blood insulin, BMI, WHR, urinary cortisol, HDL, TC, TG, serum uric acid, GGT (oxidative stress), CRP	AntiHTN drugs, lipid lowering drugs, anti-diabetes drugs	↑ Salt intake, heavy drinkers (males) and abstinence alcohol (women)→ ↑AL ↑ Physical activity → ↓AL
Robinette et al. (2016)	MIDUS	999	34-84 Mean age = 55; SD = 12	M & F	Americans & African Americans	24 SBP, DBP, HR, cortisol, DHEA-S, CRP, IL-6, ICAM-1, fibrinogen, E-Selectin, Hb1Ac, FG, HOMA-IR, HDL, LDL, TG, BMI, WHR, low frequency spectral power, high frequency spectral power, SD of IBIs, RMSSD, epinephrine, nor epinephrine	Sociodemographic	Low Socioeconomic status and accounted by anxious arousal & health behaviours → ↑AL
Kusano et al. (2016)	Nagasaki Prefecture, Japan	96	Average age = 67.9 years (range = 55–89; SD = 8.65	M & F	Japanese	10 SBP, DBP, W/H, Hbg, TC, HDL, DHEA-S, cortisol, adrenaline, noradrenaline	Age, sex, education	Consumption of green/yellow veg & non-red meat in women → ↓AL, but positive association to AL In men, consumption of alcohol (spirits) ↑ AL
Duru et al. (2012)	National Health and Nutrition Examination Survey (1988–1994)	4515	35-64	M & F	non-Hispanic black or non-Hispanic white	10 WHR, HbA1c, SB, DBP, TC, TG, homocysteine, alb, CRP, eGFR	Sociodemographic factors and health insurance	Blacks (independent of SES and Health behaviours) → ↑AL
Hampson et al. (2009)	Hawaii Personality and Health cohort Honolulu	470	40-50 men: (M = 50.3, SD = 2.0 years) women: (M = 50.0, SD = 2.0 years)	M & F	42 % Japanese Americans; 17 % Native Hawaiians; 12 % European Americans Others- Chinese, Filipino, Okinawan, Latino, Korean & other Pacific Islanders	11 SBP, DBP, TC, TC/HDL, TG, FBG, BMI, WHR, urine protein, cholesterol meds, BP meds	–	Smoking & unhealthy food consistent in M&F → ↑ AL Males with more physical

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Table 2 (*continued*)

Authors, years	Sample (nationality: type)	N	Age (mean ± SD; range) > time	Sex (M/F)	Ethnicity	Total no AL Biomarkers	Covariates/Adjustments	↑AL main findings
								activity, ate more fibre, drank alcohol → ↓ AL

Allostatic load (AL), Blood pressure (BP), Systolic blood pressure(SBP), Diastolic blood pressure (DBP), Pulse rate (PR), Pulse pressure (PP), Heart rate (HR), Waist height ratio (WHR), Body mass index (BMI), Haemoglobin (Hbg), glycated haemoglobin (HbA1c), Fasting glucose (FG), Fasting blood glucose (FBG), Homeostatic assessment model for Insulin resistance (HOMA-IR), Total cholesterol (TC), High density lipoproteins -cholesterol (HDL-C), Low density cholesterol (LDL), Triglycerides (TG), serum albumin (Alb), C-reactive protein (CRP), High sensitivity C-reactive protein (hsCRP), Interleukin-6 (IL-6), Tumor necrosis factor-alpha (TNF-α), Soluble intercellular adhesion molecule-1 (sICAM), Epstein Barr viral (EBV) capsid antigen IgG, Serum dehydroepiandrosterone sulfate, Sympatho-adrenal medullary (SAM), Hypothalamic-pituitary-adrenal axis (HPA), Parasympathetic nervous system (PNS), Standard deviation of heart beat to heart beat intervals (SDRR), Root mean square of successive heart beat differences (RMSSD), Gamma glutamyltransferase (GGT), Estimated glomerular filtration rate (eGFR).

obesity, insulin resistance and systemic inflammation (Duffey et al., 2010; Kant et al., 2015) but was not observed in van Draanen's study (2018). This inconsistency may be due to the specificity of food items recorded or the quantity, calories, sodium intake or fat in the food consumed. Artificial sweeteners may increase cravings and consumption of sugar-sweetened, energy dense foods and beverages and decrease the ability to estimate an accurate nutrient intake (Swithers and Davidson, 2008) and thus increase BMI, adiposity and type 2 diabetes (Nettleton et al., 2009). It was proposed by McEwen two decades ago that role of dietary pattern and behaviours are major contributors to AL (McEwen, 1998b). Stress, elevated AL and it's co-morbidity are multifactorial and complex thereby reflecting HPA axis dysregulation and secondary responses such as metabolic syndrome which is a risk factor for type 2 diabetes and cardiovascular disease (McEwen, 2008).

High sugar consumption has been associated with poor health, therefore, non-nutritive sweeteners (NNSs) are increasing to replace free sugars due to their reduced costs (Wang et al., 2018; Suez et al., 2014) and low caloric intake (Ruiz-Ojeda et al., 2019). However, there is growing evidence that NNSs have unforeseen effects on human health that potentially result in metabolic dysfunction affecting body weight (Fowler, 2016) and glucose tolerance (Suez et al., 2014). NNSs have direct bacteriostatic effects and can change intestinal microbiota (Wang et al., 2018). The gut microbiota consists of numerous bacterial species that are involved in multiple physiological functions such as metabolism, immunity, growth and fermentation of undigested carbohydrates (Ruiz-Ojeda et al., 2019). The dysfunction of gut microbiota is associated with obesity and insulin resistance (Nettleton et al., 2016). These gut microbes communicate with the immune system, giving signals leading to maturation of immune cells that results in numerous pathophysiologic conditions such as autoimmune and allergic diseases, obesity, inflammatory bowel disease (IBD) and diabetes (Clemente et al., 2012). The structure and function of the microbiome are modulated and can be altered by diet and due to this, the importance of studying the gut microbiome is being addressed currently (Dominguez-Bello et al., 2019; Suez et al., 2015).

The gut microbiota may provide a direct link between diet-related HRBs, hormonal and immune activation and systemic and brain disorders. Increased intestinal wall permeability ("leaky gut") that is altered by dietary factors such as high-fat, high carbohydrate "Western" diet may promote the interaction between the immune system and the gut microbiota (Obrenovich, 2018; Sherwin et al., 2019; Cryan et al., 2019a,b). The elevated pro-inflammatory cytokines then in turn activate the HPA axis through hypothalamic CRH-mediated mechanism resulting in an elevated circulating cortisol level (Foster et al., 2017). Furthermore, changes induced by unhealthy diet in the gut microbiota can lead to glucose and lipid metabolic abnormalities that in the longer term contribute to obesity and metabolic syndrome (Cryan et al., 2019a,b). Altogether, the elevated HPA axis activity, the chronic-low-grade inflammatory state maintained by the increased pro-inflammatory cytokines and the metabolic abnormalities can contribute to the increased AL caused by unhealthy eating. Therefore, it is plausible to hypothesise that the gut microbiome may play a central role in linking unhealthy diet with elevated AL and resulting health consequences (Cryan et al., 2019a,b; Adan et al., 2019).

Positive associations between meat, processed meat, French fries and AL score was found with increasing levels of DHEA-S and high HbA1c but not with the traditional diet (Mattei et al., 2011). Mattei et al. (2013) proposed that this could be dependent on the source and the type of the meat consumed however, the type of meat proposed was not mentioned. Legumes may be protective as they are rich in fibre, protein, folate, zinc and other nutrients (Messina, 1999). Papanikolaou and Fulgoni (2008) also found that individuals who consumed beans showed a 22 % lower risk of obesity and a 23 % lower risk of developing central obesity as measured by waist circumference when compared to non-bean consumers. The increase in high Hb1Ac could be related to the consumption of rice, a source of carbohydrate that may

prevent release of epinephrine, which is normally secreted during low blood sugar (Mattei et al., 2011). Furthermore, DHEA-S has been implicated as the activation of adrenal or liver androgens, enzymatic activity in the liver that might result in an altered androgen metabolism, and in the production of inflammatory markers (Mattei et al., 2013).

In a study that evaluated if dietary components recommended by the American Heart Association (AHA) were related with cardiometabolic risk factors, MetS, and AL in Puerto Rican men and women found that there was a substantial inverse relationship between the AHA dietary score and MetS in men alone supporting previous studies (Bernabe-Ortiz et al., 2012; Bhupathiraju et al., 2011; Mattei et al., 2010). In the majority of this cohort, a significant association in women only was observed between serum insulin and waist circumference and a weak relationship was observed between AHA score and HDL-C in women, although this was significant in men. However, no relationship was found between diet and CRP (Mattei et al., 2013). Additionally, AHA diet score was inversely associated with norepinephrine level in men (Mattei et al., 2013).

This was supported by other studies, with low levels of norepinephrine and high energy carbohydrate diet consumption along with omega-3 supplementation especially during mental stress (Delarue et al., 2003; Young et al., 1992). They found that individuals on omega-3 supplementation blunted mental stress that almost abolished both epinephrine and cortisol secretion. This effect is likely exerted at the central nervous system (CNS) but also has a direct action suggesting that stimulation of adrenal gland was inhibited (Delarue et al., 2003). The probable mechanism behind this is that intake of healthy diet, particularly with high amount of fibre and whole grains, regulates blood glucose levels by blunting the catecholaminergic response. Also, omega-3 fatty acids consumption prevents sympathetic stimulation provoked by mental illness possibly through effects exerted at the level of CNS (Delarue et al., 2003).

High salt ingestion was also associated with increased AL (Kusano et al., 2016; Petrovic et al., 2016). Previous research demonstrated that high salt intake causes an increase in the glomerular filtration rate (GFR) in the kidneys and therefore contributes to high AL (Berge-Landry and James, 2004). This suggests that the allostatic adaptation to increased sodium in the diet causes an increase in the GFR, which restores water balance. It was postulated following the concept of AL (McEwen, 1998b; Seeman et al., 2001) that an individual with a high dietary sodium will also maintain a high GFR, which eventually results in the inability of the kidney to regulate filtration dynamics leading to renal failure. Chronic stress activates the SAM axis which stimulates rennin release leading to increases in angiotensin II and aldosterone secretion resulting in increased sodium and water retention. This over time develops in high blood pressure and consequently into renal failure (Berge-Landry and James, 2004).

Ottino-González et al. (2017) found that overweight to obese participants showed cortical thinning in left and right superior frontal gyrus as supported in the findings of Marqués-Iturria et al. (2013). Furthermore, overweight and obese individuals exhibited higher AL index than lean controls who had cortical thickening. However, AL index did not show any correlation with cortical thickness when the entire sample of overweight to obese and lean subjects was analysed together. During ongoing inflammation, in initial stages of depression (Qiu et al., 2014) and schizophrenia (van Haren et al., 2011) there is cortical thickening. Cortical thinning may be suggestive of dendritic reaction (Qiu et al., 2014) and thickening might be related to glial overactivity due to presence of inflammatory mediators (Kempuraj et al., 2017).

The release of pro-inflammatory cytokines stops neuronal degeneration by increasing neurotrophic factor release (e.g., brain-derived neurotrophic factor, or BDNF) as observed in lean individuals (Ottino-González et al., 2017). However, in obese subjects, if this response fails to revert and keeps escalating, it could result in dendritic retraction and neuronal degeneration. Triglycerides, LDL cholesterol and leptin are associated with adiposity that have an impact on the integrity of blood vessels causing atherosclerosis and hypertension. These are risk factors for cerebrovascular disease, impeding glucose and oxygen supply in the brain that further damages the structure and function of the neurons (Kemeny, 2003). The role of reward processing is affected in obesity (García-García et al., 2013; Marqués-Iturria et al., 2015) and stress (Porcelli et al., 2012) and is mediated by the orbitofrontal regions, also called as secondary gustatory cortex.

Weight discrimination is the fourth most prevalent form of discrimination among adults (Puhl et al., 2008). Discrimination can have an impact on individuals contributing to adverse physiologic changes (Berger and Sarnyai, 2015; Tomiyama, 2014; Tomiyama et al., 2014). It contributes to obesity by discouraging people from seeking help from the health care system, decreasing social networking and making unhealthy choices of food, and less physical activity (Phelan et al., 2015; Puhl and Brownell, 2012; Puhl et al., 2008, 2012; Sikorski et al., 2014; Sutin et al., 2014, 2016; Tomiyama, 2014). Weight stigma has been linked with high glycaemic parameters (Tsenkova et al., 2011) and CRP levels as observed in some longitudinal studies (Sutin et al., 2014).

Vadiveloo and Mattei (2017) observed that perceived baseline and chronic weight discrimination were associated with more than twice the risk of developing AL and suggested that prevention efforts should be made in relation to reduce weight-related stigma. Similar negative behavioural adaptations were observed in other studies supporting the associations between weight discrimination and AL (Potter et al., 2015; Tsenkova et al., 2011). Biomarkers of the HPA axis, the sympathetic and parasympathetic nervous system and CVD dysregulation that are linked to AL were not found be influenced by weight discrimination. This indicates the possibility of obesity occurring before the dysregulation of the HPA axis (Vadiveloo and Mattei, 2017).

Obesity would result in long standing stress-induced cortisol elevations promoting further accumulation of adiposity and hence weight gain (Incollingo Rodriguez et al., 2015). However, in one of the studies conducted on chronic stress participants rather than in individuals with obesity, they showed more abdominal fat and their HPA axis profiles suggested hypo-activity (Tomiyama et al., 2011). The authors raised the possibility that long-standing stress, along with excessive comfort food consumption, increases abdominal fat, which, in turn, facilitates the opioidergic inhibition on the HPA axis. Irregularities in adipocyte cortisol metabolism were also observed, along with the hypo-activity of HPA axis and continuous cortisol levels (Tomiyama et al., 2011). This signifies a strong link between abdominal obesity and over expression of 11 β- hydroxysteroid dehydrogenase type 1 (11 β-HSD1) in adipose tissue and under expression in hepatic tissue (Tomiyama et al., 2011). 11 β-HSD1 is an NADPH dependent enzyme that reduces cortisone to cortisol and is highly expressed in metabolic tissues including liver and adipose tissue (Morton and Seckl, 2008).

Chronic stress associated with unhealthy behaviours including smoking, excessive/binge drinking, physical inactivity and poor diet were most likely to be associated with depressive symptoms in the long term (Rodriquez et al., 2017). In a recent study, Rodriquez and colleagues (2018) observed that African Americans and Latinos engaged in unhealthy behaviour and high AL were at a risk of developing a depressive disorder when compared to Whites. However, there were no significant associations observed between AL and being at risk for depressive symptoms in any racial/ethnic groups, which was supported by other studies (Keyes et al., 2011; Walsh et al., 2013). Chronic stress was measured as a physiological biomarker rather than a self-report in this study, which might explain some conflicting results from other studies as subjective experience of chronic stress affects depression differently than the physiological impact also found in two other studies (Jackson et al., 2010; Tennant, 2002). A study conducted by Berger et al. (2019) in Aboriginal and Torres Strait Islander adolescents and adults in three communities in North Queensland, Australia, did not demonstrate an overall association of physiological stress biomarkers and multisystem dysregulation with depressive symptoms. However, anhedonia

(inability to feel pleasure in normally pleasurable activities) and insomnia with AL was established and significant in one of the study communities. Some studies suggest that unhealthy behaviours may conceal the effects of chronic stress in developing depressive illness (Dallman et al., 2003; Koob et al., 1998; Piazza and Le Moal, 1998). The assessment of the relationship between AL and volume/activity of certain brain structure with regards to mental illness has received considerable interest lately (Chiappelli et al., 2017; Savransky et al., 2017).

3.5.2. Physical inactivity and AL

Like diet, physical activity also contributes to an individual's AL. Five studies identified that higher physical activity is associated with a decrease in AL (Gay et al., 2015; Hampson et al., 2009; Hu et al., 2007; Kusano et al., 2016; Upchurch et al., 2015). Furthermore, an important finding was that high AL associated with physical inactivity in a female sample was mediated by race. This association was detected when White, Black and Mexican American women exhibited a high AL were compared to each other. White women had the lowest AL, followed by Mexican American women and highest in Black women (Upchurch et al., 2015). Overall, there is considerable empirical data to support the protective effects of physical activity on overall health (Gay et al., 2015; Upchurch et al., 2015). It was also noted that high level of physical activity was associated with low dysregulation of cardiovascular, metabolic, lipid axes and tended to be linked with low oxidative stress (Petrovic et al., 2016).

According to the Center for Disease Control (CDC), about 1 in 5 (21 %) adults meet the 2008 Physical Activity Guidelines. Around 23 % non-Hispanic white adult, 18 % of non-Hispanic black adults and 16 % of Hispanic adults meet the 2008 Physical Activity Guidelines for aerobic and muscle-strengthening activity. Furthermore, while 54 % of men meet the physical guidelines for aerobic activity, much fewer women (46 %) meet this same criteria (Centers for Disease Control and Prevention, 2018b).

In this systematic review we identified a negative relationship between physical activity and AL. Specifically, lower physical activity was associated with higher AL (Gay et al., 2015; Hampson et al., 2009; Petrovic et al., 2016; Upchurch et al., 2015). There is strong evidence for a role of physical activity in the primary prevention of cardiovascular disease, diabetes, and some cancers. Earlier research viewed an advantageous effect of physical activity on various physiological systems (Warburton et al., 2006). This notion was supported by Petrovic et al. (2016), who found that men and women performing high physical activity had lower AL and lower dysregulation of physiological system such as metabolism, cardiovascular axis and oxidative stress. A handful of studies (Hampson et al., 2009; Gallo et al., 2011; Gay et al., 2015) explored the correlation between physical activity and AL displaying mixed outcomes. This inconsistency may be due to the use of different measures of physical activity or type of sample used (Upchurch et al., 2015). The extent and the intensity of physical activity must be recorded in an objective and unbiased fashion in order to understand the health benefits. Additionally, studies have reported a link between physical activity with inflammatory markers, that compared cytokine levels in active versus sedentary adults (Pitsavos et al., 2005) and controlled exercise training trials (Mathur and Pedersen, 2008). Uncertainties about the causal relationship between physical activity and AL (Upchurch et al., 2015) could be addressed by using key biomarkers such as cortisol, epinephrine and norepinephrine to identify any associations.

Hu et al. (2007) found in a cross-sectional study of older people in Taiwan that higher AL was associated with poorer self-rated health and several physical activity difficulties, however, associations with activities of daily living and instrumental activities of daily living limitations were not significant. Chronic stress and depression are closely associated with increased risk of cardiovascular disease and lack of exercise may be crucial for understanding biobehavioral mechanism (Stults-Kolehmainen and Sinha, 2014). Physical activity was found to have antidepressant effects and additionally it was found that active individuals appear to be more biologically resilient to psychosocial stressors (Hamer, 2012).

3.5.3. Substance abuse and AL

For the purpose of this review, substance abuse includes alcohol consumption, smoking, and drug use. Alcohol consumption has been linked to high AL (Kusano et al., 2016; Petrovic et al., 2016). However, in other studies alcohol consumption was identified as a significant predictor for decreased AL, with greater alcohol consumption predicting less AL scores (Gallo et al., 2011; Hampson et al., 2009; Hu et al., 2007); whereas, smoking was not related to AL (Gallo et al., 2011). In a recent, longitudinal study, smoking but not alcohol or drug use, was an important mediator between early life adversities and increased AL (Doan et al., 2014). In a rural African American sample, it was found that youth coming from high poverty neighbourhoods who made it to college and showed lower rates of substance use (smoking, drinking alcohol, marijuana) exhibited higher AL compared to those who did not attend college (Chen et al., 2015). These findings are somewhat paradoxical as one would expect academic success and lower substance use to be associated with lower, rather than higher, allostatic load. However, it is plausible that overcoming socioeconomic disadvantage may exert significant toll on the stress system. Alternatively, one can argue that overcoming disadvantage (coming from high poverty African American neighbourhood) results in the need of being successful in a majority setting (college) as an under-represented minority. This may exacerbate the experience of discrimination, which is perceived as a chronic stress, leading to heightened AL (Berger and Sarnyai, 2015; Chen et al., 2015).

Brody et al. (2013) found that rural African American pre-adolescents that demonstrated high levels of self-control when under high SES-related risk exhibited low levels of adjustment problems despite exhibiting high AL at age 19. These findings were consistent with the "John Henry effect", which posits that many rural African Americans will emulate the legendary folk hero, John Henry, by being hardworking, goal orientated, focused on success and avoiding unconventional behaviour, despite growing up in a high risk context and manifesting biomarkers of physiological stress.

Additionally, Berger et al. (2017) reported that Indigenous Australian university students show impaired cortisol awakening response, which correlated with perceived discrimination. Williams (2005) found that Blacks had a 30 % higher overall death rate compared to Whites, with other minority groups (American Indians, Native Hawaiians, Pacific Islanders and Hispanics) having a lower death rate than Whites. It was also found that Blacks suffer more of and have a higher/elevated health risks for stress-related outcomes such as hypertension and suicidal tendencies (Williams, 2005). They also found that AL scores were inversely correlated with SES (high AL-low SES) in high functioning elderly, which suggests that SES is one of strongest determinants of variations in health (Williams, 2005).

Income status, home ownership and smoking play a vital role in understanding the socioeconomic discrepancies in AL and low SES (Robertson et al., 2015). People of low SES pose high biological risk and tend to age early (Crimmins et al., 2009). This suggests that lifestyle factors such as alcohol consumption, smoking may play a part in the relationship between chronic stressors and AL.

Here we identified a relationship between substance use and AL; showing that an increased consumption of substances is associated with higher AL (Doan et al., 2014; Robertson et al., 2015). However, we found an inverse relationship between alcohol consumption and low AL (Gallo et al., 2011; Hu et al., 2007). There is a causal association between alcohol consumption, smoking and mental and behavioural disorders (McEwen, 2008). In the US, alcohol consumption leads to about 80,000 deaths and 2.5 million years of potential life lost every year (Centers for Disease Control and Prevention, 2018a). There are 3

million deaths occurring due to alcohol use every year representing 5.3 % of all deaths (World Health Organization, 2018b). Percentage of US adults aged 18 years or older who were current cigarette smokers in 2016, were 15.5 % of all adults (37.8 million people): 17.5 % of males, 13.5 % of females (Jamal et al., 2018). Tobacco smoking causes serious health conditions such as heart disease, diabetes, stroke, cancer, renal disease, eye disease and respiratory conditions such as asthma, emphysema and bronchitis.

The association between alcohol ingestion and mortality is usually U or J shaped with abstainers and people who drink in large amounts have higher mortality rate in comparison to moderate drinkers (Rehm and Sempos, 1995). Moderate alcohol consumption showed beneficial effects in lowering AL in both men and women, which could be due to beneficial effects on increasing HDL cholesterol and insulin sensitivity (Di Castelnuovo et al., 2006; Gallo et al., 2011; O'Keefe et al., 2007), when compared to the harmful effects of high alcohol abuse on the cardiovascular system (Mukamal et al., 2010).

A strong association was observed between SES, lifestyle factors, such as alcohol consumption and smoking, as well as educational and occupational position (Gallo et al., 2011; Juster et al., 2013; Petrovic et al., 2016). Working longer hours and having a significant amount of responsibility could result in the ability to endure stress, especially in males, as seen by their low AL when compared to men with non-manual intermediate occupations not involving any physical strain. Among women with household responsibilities, low job status and less income, a negative effect on health was found that contributed to both physical and mental strain (Bonjour and Gerfin, 2001; Artazcoz et al., 2004).

Using AL score as a measure of stress-induced physiological dysregulation, Bingham et al. (2016) found that there was no significant association between stress and unhealthy behaviours, specifically, smoking, alcohol consumption, and sedentary lifestyle in adult African Americans, who migrated to the United States (US) recently. This could be because stress can be minimised by reuniting families, as social contact has a positive influence on the psychological and physiological functioning (Kikusui et al., 2006). In contrast, the adult immigrants who lived in the US for long time (> 10 years) displayed high AL (Bingham et al., 2016). Potential reasons include lifestyle transition from a black majority country to one with a black minority population (Doamekpor and Dinwiddie, 2015). Furthermore, concern for family members not living with them in US, constant worry about their homeland conditions and anxiety about a potential permanent transition to the US (Bingham et al., 2016).

A number of studies support the link between smoking and high AL (Crimmins et al., 2009; Robertson et al., 2015; Moffatt, 1988; Omvik, 1996; Tonstad and Cowan, 2009; Will et al., 2001), showing that smoking is associated directly and/or indirectly with various physiological systems of the individuals, that increases the risk of developing AL, with a strong relationship with lower SES (Hiscock et al., 2012). The findings from Doan et al. (2014) supports the theory (McEwen, 1998b; McEwen and Gianaros, 2010; Szanton et al., 2005) that health behaviours may be one of the pathways from socioeconomic position (SEP) to AL. In contrast, Petrovic et al. (2016) found that smoking displayed a weak and insignificant association with high AL. The inconsistency in the findings of various studies indicate that poverty-related risks influencing AL are not the same across the lifespan (Doan et al., 2014). This is an important step in better understanding the pathways and mechanisms linking SEP, physiology and health.

Chen et al. (2015) found that African American youth coming from impoverished neighbourhood who did not attend college had a higher rate of substance use than those who attended college. Chen et al. (2015) suggested that college students who resisted temptation to use substances was attributed to their high levels of self-control, determination and tremendous hard work. This also supported the results obtained by Watt (2008) who stated that African American youth using substances experience more negative health effects during the transition into adulthood. This results in drug-related incidents in adulthood, including alcohol dependence and drug arrests (Galea and Rudenstine, 2005; Mitchell and Caudy, 2015). Therefore, understanding the factors that decrease youth from taking risky behaviour as they enter into adulthood may be important (Chen et al., 2015).

People who engaged in physical activity, smoked less and ate less fast food had significantly lower AL (Robinette et al., 2016). These findings support both theoretical (McEwen, 1998b) and empirical (Friedman et al., 2015) reports of an association between HRB and allostatic load.

Low socioeconomic position (SEP) has been a strong predictor of negative health outcomes and there is a growing research establishing the conceptual theory behind low SES and high AL (McEwen and Stellar, 1993). Studies have shown that poorer neighbourhood residents (Friedman et al., 2015) engage in HRB such as smoking, fast food consumption and low physical activity that are associated mainly with cardiovascular diseases. These behaviours correlate with AL and partially accountable for the relationships between lower individual SES and higher AL (Gruenewald et al., 2012). A low SEP was associated with high AL increasing the risk for mortality (Kim et al., 2018) which was consistent with other studies showing significant relationship of AL with SEP (Gruenewald et al., 2012; Szanton et al., 2005) and mortality (Karlamangla et al., 2006; Seeman et al., 2001). SEP has a modest, but significant, indirect effect on mortality through physical exercise and smoking status (Kim et al., 2018). However, the findings cannot be compared to previous studies due to the differences in the socioeconomic and behavioural measures used (Khang and Kim, 2005).

3.5.4. Sleep and AL

We found four studies that established sleep impairment and its association to increased AL (Bei et al., 2017; Carroll et al., 2015; Chen et al., 2014; Clark et al., 2014). The finding by Clark and colleagues of long sleep duration being related to increased AL is in concordance with Carroll and colleagues, suggesting that both inadequate sleep and excessive sleep are related to heightened AL based on biomarkers that included elevated immune markers and hypothalamic-pituitary-adrenal-axis products (Carroll et al., 2015; Clark et al., 2014). Sleep variability (rise time and bedtime) were associated with flat diurnal cortisol slopes. Only later mean bedtime (those who sleep late with short sleep time), but not sleep variability, was significantly associated to AL (Bei et al., 2017). It is important to point out that circadian cortisol rhythm and HPA stress response are related. For example, a flat cortisol rhythm prevents a robust HPA stress response in which turning on and off the activation supports successful adaptation and therefore mitigates AL (Akana et al., 1988; Jacobson et al., 1988). Therefore, irregular patterns and/or poor sleep quality is associated with physiological dysregulation across several individual's regulatory systems resulting in an elevated multisystem biological risk.

We also identified a negative correlation between sleep impairments and AL, indicating that poor sleep quality is associated with higher AL (Bei et al., 2017; Carroll et al., 2015; Chen et al., 2014; Clark et al., 2014). Sleep disorders cause a substantial individual and societal burden and form a major health concern (Hillman and Lack, 2013). The annual costs of insomnia in US is between $92.5 billion and $107.5 billion (Reeder et al., 2007) affecting 30–45 % of the adult population (Wade et al., 2008). About 41,000 people suffer injuries every year due to sleep-related accidents and more than 800 people die because of sleep-related accidents (U.S. Department of Transportation NHTSA, 2018). Lack of sleep has negative health risks as well as increases the risk of accidents (Knutsson, 2003). Adults sleeping less than 7 h per day are reported to be less physically active, obese and smoke. They are at a high risk for developing CVD, respiratory disease, cancer, mental illness and metabolic disease compared to people sleeping more than 7 h per day (Centers for Disease Control and Prevention, 2017). Limited studies have been conducted on possible associations between sleep impairments and AL. It has been suggested that poor quality of sleep might act as a physiological stressor that could impair neurobiological regulation

of circadian rhythm (McEwen, 2006). Furthermore, it could also impair the neurophysiological functions leading to AL by elevating evening cortisol, insulin insensitivity, proinflammatory cytokines and oxidative stress (McEwen, 2006; Tremblay and Chaput, 2012), establishing an important link between impaired sleep, AL and later morbidity and mortality.

Sleep deprivation, short- or long-term, is associated with a number of biological mechanisms such as hormonal changes in regulation of energy balance and appetite, activation of inflammatory markers, glucose intolerance and insulin resistance. It also has some molecular effects in adipocytes, alterations in gene expression and vascular calcifications that leads to cardio-metabolic disease (Cappuccio and Miller, 2017). A future longitudinal study will be useful in understanding the bidirectional and causal relationships between sleep pattern and AL. An association between sleep impairment, AL and depression was suggested by previous studies (Juster et al., 2010; McEwen and Stellar, 1993) raising the possibility that AL may be due to inadequate recovery after the sleep disturbance (Van Cauter and Spiegel, 2006; Van Cauter et al., 2008). Nevertheless, sub-analyses in Clark's study showed that disturbed sleep might be an index of obesity-related disorders, e.g. sleep apnoea or chronic physical disorders, in line with other studies establishing a link between AL and chronic disorders such as cardiovascular disease and diabetes (Clark et al., 2014; Juster et al., 2010).

The findings in the study by Carroll et al. (2015) strengthens previous results documenting similar relationships of short sleep duration with high risk for cardiovascular system, immune and metabolic system (Chen et al., 2014; Clark et al., 2014). Importantly, people sleeping for long duration are also more likely to develop impaired functioning, which may serve as a risk for negative health outcomes. This could be due to sleep fragmentation, fatigue, changes in the immune system (inter- & intra -cellular cytokines), photoperiodic irregularities, lack of challenge due to stressors, a pre-disposing condition e.g., sleep apnoea, hypertension, heart disease and depression (Grandner and Drummond, 2007).

Sleep and circadian disruption have been conceptualised as key drivers of AL (McEwen and Karatsoreos, 2015). Good sleep gives the body time to rest and repair damages, thereby restores wear and tear that would lead to AL (McEwen and Seeman, 1999; Seeman et al., 1997). Short sleep duration may be a predictive indicator of mortality in patients with type 2 diabetes and may increase cardiovascular stress (Hamasaki et al., 2017). Long-term, inadequate sleep contributes to gradual changes in the regulatory set points that governs blood pressure, cholesterol, blood sugar and inflammatory activity leading to disease pathophysiology (Chung et al., 2009; Dokken, 2008; Franceschi and Campisi, 2014; Libby and Theroux, 2005; Miller et al., 2011; Kondapally et al., 2011). Sleep variability or chronic sleep restriction are associated with circadian misalignment which appear to influence SNS, PNS, HPA and immune system resulting in an elevated multisystem biological risk (McEwen, 2006). Longitudinal studies will be important to delineate the bidirectional effects of sleep impairments and AL changes among populations that cope with chronic stress. Therefore, future research is recommended to using various AL antecedents not only as covariates but as mediators and/or moderators.

3.6. Limitations

Despite these findings, limitations are observed across the 26 articles and these must be taken into consideration. The use of a cross-sectional study design limits the attribution to causal association between HRB and AL. In addition, there were studies that included particular age groups (Gallo et al., 2011; McClain et al., 2018; Ottino-González et al., 2017; Robertson et al., 2015) and it is unclear if the results can be generalised to other age group. McClain et al. (2018) did not consider early life food insecurity experiences that might influence later life factors and disease development. Similarly, another study (Ottino-González et al., 2017) looked at age group of 20–40 years examining the cardiometabolic changes through brain imaging. The inclusion of age groups above 40 would have been beneficial as the metabolic syndrome is most likely to occur late in life. Few studies used females which suggest a gender bias (Crimmins et al., 2009; Gallo et al., 2011; Upchurch et al., 2015) which may have influenced the health-related outcomes.

Detailed dietary information was not provided in studies that evaluated overeating and obesity as it might have been an important mediator in associating with AL (Vadiveloo and Mattei, 2017; van Draanen et al., 2018). A temporal relationship between diet history and physiological biomarkers was difficult to establish in van Draanen et al. (2018) study as the cumulative measure of physiological dysregulation based on the consumption of food and beverages was observed for a short period of time. A number of studies focused on particular ethnic groups which makes it difficult to generalise the results across different ethnicities (Bingham et al., 2016; Doan et al., 2014; Gay et al., 2015; Kusano et al., 2016; Petrovic et al., 2016; Upchurch et al., 2015). Studies examining the cardiovascular and metabolic system failed to use important biomarkers such as cortisol or epinephrine or an inflammatory marker such as fibrinogen and therefore it is challenging to interpret elevated AL (Gay et al., 2015; Petrovic et al., 2016; Robertson et al., 2015; Upchurch et al., 2015). Studies that observed sleep patterns and variability (Bei et al., 2017; Carroll et al., 2015; Clark et al., 2014) and impaired sleep and their associations with AL failed to include the study of sleep apnoea, which is a significant predictor of health issues like cardiovascular, metabolic and stroke.

Most studies used self-report assessment for some of the unhealthy behavioural variables leading to possible under- or over-reporting of HRB and also could have been subject to measurement error and or report bias (Doan et al., 2014; Robertson et al., 2015; Rodriquez et al., 2018). Studies examining neighbourhood poverty or SES associating biological risk looked at mainly education attainment and income status (Crimmins et al., 2009; Duru et al., 2012; Robinette et al., 2016). However, observations such as crime rate, social cohesion and inadequate local resources (such as public transportation, recreational facilities and health centres) would have been beneficial. Moreover, in a few studies, no follow up has occurred (Chen et al., 2015; Doan et al., 2014; Hu et al., 2007). A longitudinal study design would prove to be beneficial when assessing causality with a periodic follow-up. This would help to determine temporal associations among physiological biomarkers with other co-morbidities. The complexity of the relationships between chronic stress, HRB and mental health among diverse racial/ethnic groups. Only one study (Rodriquez et al., 2018) considered exploring engaging in HRB and its association with AL and developing mental illness. In addition to this, only one study mentioned about genetics and the role it plays in developing high AL (Petrovic et al., 2016).

Finally, throughout the 26 studies, there has been considerable heterogeneity in the biomarkers selected to assess AL and in the cut-off points for defining high AL. A consensus should be reached on how to best measure AL and one could question whether the use of consistent biomarkers would have improved the results obtained by the 26 studies.

4. Conclusion

To the best of our knowledge, this is the first systematic review to comprehensively assess the relationship between health risk behaviours (HRB) and Allostatic Load (AL). We reviewed 26 articles exploring different HRB and their association with AL, with only 2 studies reporting a low AL on moderate consumption of alcohol. We found that individuals who consumed high intake of fruits, vegetables, whole grains, fish, poultry and who practiced physical activities had lower AL in comparison to those who did not. Therefore, eating healthy foods combined with physical activity, can help in maintaining a healthy weight and reduce the risk of chronic diseases (like heart disease,

metabolic syndrome), and promote overall good health. Additionally, people with low socioeconomic status are at a higher risk of developing multiple HRB. Finally, it was found that those with sleep apnoea, or who slept for too long or too short and had disturbed/poor sleep quality were associated with higher AL. Thus, there is a prevailing supposition that sleep can be a pre-clinical indicator of disease risk, premature death, high-risk levels of anthropometric, and metabolic and immune risk markers. Individuals with cardiovascular disease, diabetes, sleep disturbance, cognitive dysfunction (e.g., Parkinson's disease, dementia), and hyperactivity of inflammatory pathways are also more likely to experience depressive symptoms.

The Allostatic Load concept holds promise for studying inter-individual differences in exposure to long term stressful events and health outcomes. With increased focus on specificity and refinement of measurement, the AL model may be a useful tool in early detection of illnesses, allowing for the assessment and correlation of health risk behaviours to early or late life depressive illness. Further research is required to understand the cumulative effect of stress and associated coping behaviours on biological system and health outcomes.

Declaration of Competing Interest

The authors report no biomedical financial interests or potential conflicts of interest.

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