Journal of Psychosomatic Research 184 (2024) 111831

Contents lists available at ScienceDirect

Journal of Psychosomatic Research

journal homepage: www.elsevier.com/locate/jpsychores

Review article

Social determinants of inflammatory markers linking depression and type 2 diabetes among women: A scoping review

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ARTICLE INFO

Keywords:
Early life stress
Gender
Health disparities
Inflammation

ABSTRACT

Objective: Inflammation is implicated in the pathophysiology of depression and type 2 diabetes (T2D) and is linked to social determinants of health (SDoH) associated with socioeconomic disadvantage. The objective of this review is to identify and map the range of SDoHs associated with inflammation in depression, T2D, or their co-occurrence among women.

Methods: PubMed, CINAHL, PsychINFO, and Web of Science were searched March–July 2023 to identify studies where 1) an SDoH was a predictor or independent variable, 2) depression or T2D was a clinical focus, 3) inflammatory markers were collected, and 4) analysis was specific to women. We used the National Institute on Minority Health and Health Disparities research framework to guide searching SDoHs, organize findings, and identify gaps.

Results: Of the 1135 studies retrieved, 46 met criteria. Within the reviewed studies, the most used inflammatory measures were C-reactive protein, interleukin-6, and tumor necrosis factor-α, and the most studied SDoHs were early life stress and socioeconomic status. Individual and interpersonal-level variables comprised the bulk of SDoHs in the included studies, while few to no studies examined built environment ($n = 6$) or health system level ($n = 0$) factors. Disadvantageous SDoHs were associated with higher levels of inflammation across the included studies.

Conclusion: The scope and intersection of depression and T2D represent a syndemic that contributes to and results from socioeconomic inequities and disproportionately affects women. Simultaneous inclusion of social and inflammatory measures, particularly understudied SDoHs, is needed to clarify potent targets aimed at advancing health and equity.

Depression and type 2 diabetes (T2D) represent two substantial public health concerns, each with mounting prevalence and significant consequences. In the U.S., T2D is the 7th leading cause of morbidity, mortality, and health costs including end-stage kidney disease, cardiovascular disease, stroke, lower-extremity amputation, and blindness [1]. Depression has a lifetime prevalence of 20%, is the primary cause of disability, and is projected to be *the* leading source of global public health burden by 2030 [2–5]. Even subclinical depressive presentations (i.e., criteria for major depressive disorder [MDD] are not met) have negative impacts on health, lifespan, and quality of life [6–8].

Depression and T2D co-occur twice as often than what would be expected by chance alone [9]. The substantial overlap between these two conditions has been observed since the 17th century [10], and relationships between the two have been described as bidirectional, as each condition increases the risk for the other [11]. In some cases, the emotional and economic burden of T2D combined with demanding treatment regimens can lead to depression development or exacerbation [10,12–14]. However, depression's role may be more predictive than

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https://doi.org/10.1016/j.jpsychores.2024.111831
Received 14 March 2024; Received in revised form 3 June 2024; Accepted 10 June 2024
Available online 15 June 2024

consequential of T2D [9,12]. Depressive symptoms—including decreased energy, concentration, motivation, self-worth, and sleep—can impair self-care activities (e.g., diet, physical activity) critical to maintaining metabolic health [10,15]. Co-occurrence may also be coincidental, a product of shared etiological factors such as poor diet, smoking, and physical inactivity [10,12], resulting in compounded negative effects on functioning, quality of life, health costs, and mortality [7,16–18].

Of the hypothesized shared pathways between depression and T2D, many have centered around individual-level psychological and behavioral factors, while the social environment in which behaviors are contextualized has been less explored. Burgeoning literature suggests that social determinants of health (SDoHs) play profound roles in mental and metabolic health [19,20]. The World Health Organization defines SDoHs as non-medical factors that influence health outcomes or the conditions in which people are born, grow, work, live, and age and the wider forces and systems shaping these conditions [19,20]. SDoHs drive health inequities by influencing health in positive and negative ways. Examples include early childhood development, education, exposure to violence, material needs insecurity (e.g., food, housing), neighborhood safety, social inclusion (i.e., non-discrimination), access to healthcare, and economic resources, the last of which is arguably foundational to many others [19,20]. Accordingly, depression and T2D disproportionately affect low-income populations, historically minoritized racial and ethnic groups, and women [14,21,22]. Indeed, the scope and intersection of poverty, depression, and T2D, have been described as a syndemic, i.e., mutually reinforcing epidemics that contribute to and result from persistent social and economic inequities [23]. Women in particular experience the greatest syndemic risk, as they comprise the majority of people who live in poverty, experience depression at twice the rate as men, and experience a greater risk of fatal T2D complications than men [24]. The complexity of this problem warrants research grounded in a comprehensive framework to elucidate modifiable molecular and macro-level targets and open the door for novel prevention, risk mitigation, and therapeutic interventions to be developed. However, the specific mechanisms by which SDoHs biologically manifest in the course of depression, T2D, and their co-occurrence are not clear.

Inflammation is a pathway implicated in depression and T2D with a growing literature about its relationship to the social environment [25]. Traditionally, mechanistic understandings of the links between SDoHs and illness have centered on the hypothalamic-pituitary-adrenal (HPA) axis, an important pathway linking stressors to depression and T2D [9,26,27]. Links between inflammation and stress response are well-known [27–29], but the impact of the stressors themselves on inflammation and their timing across the life course may vary. The knowledge of whether financial strain, racial discrimination, or neighborhood safety, for example, influence inflammation in this context is critical to elucidating mechanisms needed to design potent interventions, and identify early warning indicators or responses to treatment. A comprehensive inquiry that includes SDoHs experienced at individual, interpersonal, and community levels is needed to identify missing relevant factors contributing to inflammation, due to its attendant mental and physical health risks. Our review aims to identify SDoHs associated with biological dysregulation beyond the well-studied HPA axis by focusing on inflammation in the context of depression, T2D, and their co-occurrence. Sex-related differences in the immune and inflammatory process exist (e.g., sex hormone receptors on immune cells) as do differences in social expectations of women that give rise to distinctions in SDoH exposure by gender [25,30,31]. These considerations in combination with the prevalence of depression and T2D among women warrant a discrete discussion centered on women.

The objective of this review is to identify and map the range of SDoHs associated with inflammatory markers in women with or at risk for depression, type 2 diabetes (T2D), or their co-occurrence.

Our research questions incorporate key elements of the population, concept, and context outlined by Joanna Briggs Institute (JBI) guidelines as follows:

1. What specific SDoHs have been investigated and reported to be associated with inflammation in women with or at risk for depression and/or T2D?
2. What are the differences in SDOHs associated with inflammation for women at risk for depression compared to those at risk for T2D?

1. Methods

A scoping review was selected due to the breadth of our research question and our overall aims to map current evidence and identify gaps, rather than evaluate the strength of relationships or inform clinical practice guidelines. This review was conducted with consideration of the JBI (2015) guidelines and the Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) Extension for Scoping Reviews [32].

The search was conducted from March through April 2023 and updated in July 2023. A comprehensive search strategy was developed with the consultation of a health science librarian. The National Institute on Minority Health and Health Disparities (NIMHD) research framework guided the search and contextualized findings. This framework (Fig. 1) is a matrix of SDoHs across several domains (rows) and levels of influence (columns) [33,34]. The search included SDoHs terms across multiple domains (behavioral, physical/built environment, sociocultural environment, healthcare systems) and levels of influence (Table 1). We selected PubMed, PsychINFO, Web of Science, and CINAHL to provide the most comprehensive coverage for this transdisciplinary research question. In addition to Medical Subject Health (MeSH) terms, keywords were used to find unindexed articles. We did not restrict based on publication year.

Resulting abstracts were independently screened by two reviewers (NP, NH, or FW) according to the following criteria: 1) an SDoH was identified as a predictor or independent variable, 2) depression and/or T2D was a clinical focus, 3) inflammatory markers were collected or analyzed, and 4) the analysis focused on either exclusively on women or was stratified by sex. Inflammatory markers were defined broadly as acute phase plasma proteins (e.g., C-reactive protein [CRP]), cytokines (e.g., interleukin [IL]-6), and differential methylation or expression of genes that encode for these markers (e.g., hypomethylation of *IL6*) Per our review objective, a broad interpretation of the clinical foci criteria was applied such that articles related to the risk continuum of depression or T2D qualified (e.g., subclinical depressive symptoms, metabolic syndrome) and were not limited to studies with samples diagnosed with MDD or T2D. Reviews, study protocols, qualitative research, clinical trials (experimental) research, and articles not in English were excluded. Further, studies concerning pre-clinical models, pediatric populations, or inflammatory, immune, or infectious diseases were excluded. Finally, peripartum populations were excluded because pregnancy and postpartum periods have known increased mood, metabolic, and inflammatory risks, meriting a separate discussion [35]. Any discrepancies between the two reviewers regarding article screening inclusion were discussed among all three reviewers to reach a consensus. For articles in which these criteria could not be determined from the abstract, we conducted a full-text review. Each full-text screening was completed by two reviewers. Reviewers discussed any conflicts and reached a consensus before making final inclusion decisions.

Data from selected studies were charted, collated, and summarized numerically (e.g. frequency counts) [36]. Two reviewers completed data extraction for each article, and a data matrix using Covidence was created to extract the following from each article: study aims and design, sample characteristics, geographic setting, SDoHs according to NIMHD domain, inflammatory markers, clinical foci, and main findings. Trends across the body of literature (e.g., SDoHs and populations studied, hypothesis confirming versus null results) were discussed among all

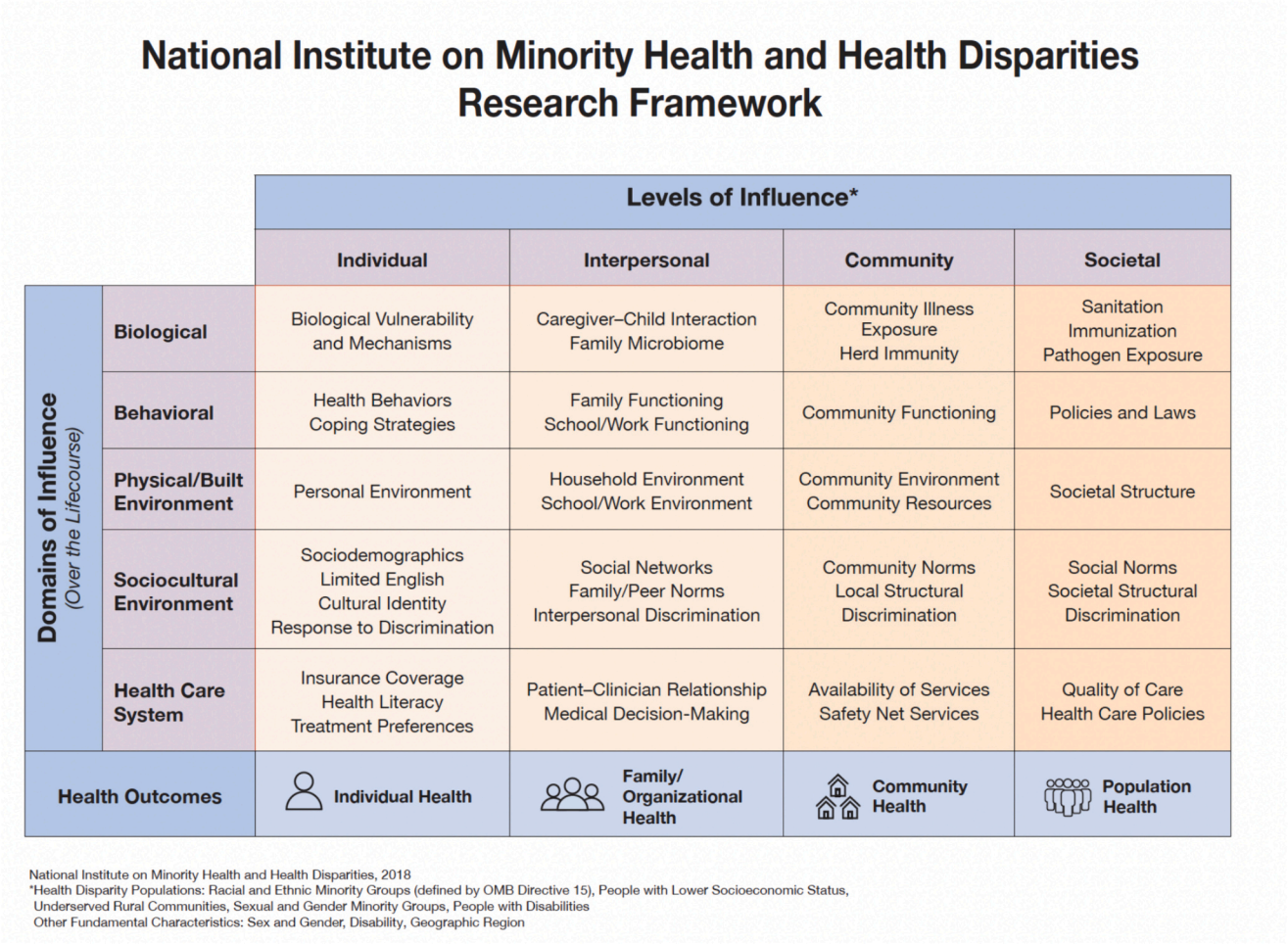

Fig. 1. NIMHD Research Framework.
Note: Available at https://www.nimhd.nih.gov/about/overview/research-framework/nimhd-framework.html. Accessed on February 13, 2024.

reviewers (NP, NH, FW). Relevant commonalities and differences in findings across studies of varied sample populations, clinical foci, geographic regions, and inflammatory markers were examined.

2. Results

Our initial literature search identified a total of 1135 studies (Fig. 2). The 46 included studies were published from 2006 to 2022. All studies were observational (16 cohort and 30 cross-sectional) in design. Twenty-seven studies were conducted in the U.S. Participants' ages ranged from 23 to 92 years, but most studies reported means in middle age. Three studies centered on African American women [37–39], while 46% included diverse samples comprising Hispanic, non-Hispanic white, non-Hispanic Black, and Asian populations. Information on race or ethnicity was not reported in 37% of studies. Refer to Table 2 for study-level findings.

2.1. SDoHs across NIMHD research framework domains

Depression and T2D were investigated within the context of several SDoHs, across multiple NIMHD Framework domains and levels of influence (Table 3). The most commonly investigated areas were within the behavioral domain at the interpersonal level and included concepts related to stress, trauma, and abuse—studied in 58% (n = 27) of the literature. Measurement tools varied as did whether these events were cumulative or occurred during childhood. Early life stress (ELS) was a focus in 17 of these studies, and many authors conceptualized it as childhood adversity, trauma, abuse, or stress. The most used measures were the Childhood Trauma Questionnaire [40] followed by the Adverse Childhood Experiences (ACE) study questionnaire developed by [41]. The remaining ten studies examined adversity, stress, and trauma across the life course, eight of which examined sexual abuse and interpersonal violence with measures including the Life Stressor Checklist-Revises (LSC-R) [42], the Sexual Experiences Survey [43], and the Partner Violence Screen [44].

Studies of physical/built environment domain variables were limited to 13% (n = 6) of the surveyed literature and included particulate matter air pollution [45,46], neighborhood deprivation indices [47,48], neighborhood socioeconomic disadvantage composites [49], and perceived neighborhood safety [37]. Early-life built environment, number of children, and joint family status were discussed in two studies [50,51].

Sociocultural factors such as socioeconomic status (SES), partnership status, financial strain, work stress, and discrimination, were a focus of 58% (n = 27) of the reviewed studies, and most were concentrated within the individual level (n = 20). SES was conceptualized and measured heterogeneously via proxies such as educational attainment, household income, and household amenities. Work-related factors included employment, work stress, job demands, and job control at the individual level and organizational justice at the community level [55]. Race or ethnicity were examined in six studies [47,50,70,71,84,89]. Discrimination was examined at community and interpersonal levels in five studies [31,45,80,87,88] and measured by the Detroit Area Study Discrimination Scale and Experiences of Discrimination (EOD) index

Table 1
Search Strategy.

Database	Search Terms*				Articles Retrieved
	Social Determinant	*Inflammation*	*Clinical Focus*	*Women*	
PubMed	"Social Determinants of Health"[Mesh] "Socioeconomic Factors"[Mesh] "social determinant*" poverty [TW] OR income [TW] OR socioeconomic[TW] OR racism [TW] OR discrimination[TW] OR inequality[TW] OR "community resource*"[TW] OR "Employment"[Mesh] OR "food secur*"[TW] OR "neighborhood safe*"[TW] OR violen*[TW] OR "Educational Status"[Mesh] OR "adverse childhood experience*"[TW] OR "early life stress"[TW] OR "Medically Uninsured"[Mesh] OR "Health Services Accessibility"[Mesh	Inflammation"[Mesh] OR "Cytokines"[Mesh] OR inflammat*[TW] OR chemokine*[TW] OR interferon* [TW] OR interleukin*[TW] OR cytokine*[TW] OR "inflammatory gene"	Diabetes Mellitus, Type 2"[Mesh] OR "type 2 diabetes"[TW] OR "Metabolic Syndrome"[Mesh] OR "Insulin Resistance"[Mesh]) OR ("Depression"[Mesh] OR "Depressive Disorder"[Mesh] OR depress*[TW]))	woman*[TW] OR female* [TW]	797
PyschINFO	exp Socioeconomic Factors/ or exp. Socioeconomic Status or "social determinant" or poverty or low-income or racism or discrimination or ("community resource" or violence or employment or "food security") or ("neighborhood safety" or "adverse childhood experiences" or "early life stress") or health insurance/ or "underinsured (health insurance)"/ or "uninsured (health insurance or exp. Health Care Access or housing/ or built environment	exp Inflammation or exp. Interleukins or exp. Cytokines or exp. Interferons or "Inflammatory gene" or "chemokine"	exp Diabetes/ or exp. Type 2 Diabetes/ or diabetes or exp. Metabolic Syndrome or "metabolic syndrome" or "insulin resistance" or exp. Major Depression/ or depression.mp. or exp. Endogenous Depression/ or exp. or exp. Recurrent Depression/ or exp. "Depression (Emotion)"/ or exp. Reactive Depression/ or exp. Late Life Depression/ or exp. Atypical Depression/ or exp. Treatment Resistant Depression/ or exp. "Long-term Depression (Neuronal)" exp. Symptoms/ or depressive	Women or exp. Human Females	43
Web of Science	TS = ("social determinant" OR poverty OR racism OR discrimination OR low-income OR "community resource" OR "food security" OR "adverse childhood experiences" OR "early life stress" OR violence OR uninsured OR "access to care" OR "socioeconomic")	TS = (Inflammation OR cytokine OR chemokine OR interferon OR interleukin OR "inflammatory gene")	TS = ("type 2 diabetes" OR "diabetes mellitus" OR "Metabolic Syndrome" OR Depression OR Depressive)	TS = (women OR female OR sex)	468
CINAHL	"social determinant" OR "social Class" OR "poverty" OR MM "Socioeconomic Factors" OR "low-income" OR "racism" OR "discrimination" OR "community resource" OR "food security" OR "nutritional security" OR "employment" or "neighborhood safe" OR MM Violence or MM "Educational Status" OR MM "Adverse Childhood Experiences" OR "early life stress" OR "uninsured" OR MM "Medically Uninsured" OR "access to care" OR "housing" OR MM "Health Services Accessibility" OR MM "Built Environment"	MM "Inflammation" OR "cytokine" OR "chemokine" OR "interleukin" OR "interferon" OR "inflammatory gene"	MM "Diabetes" OR "metabolic syndrome" OR "insulin resistance" OR MM "Depression" OR "depressive"	MM "Women's Health" OR "women" OR MM "Sex Factors"	34

Note: All search terms within topic headings were combined with OR, and search between topic headings combined with AND.

[52]. Two studies examined acculturative stress [45,78].

2.2. Inflammatory markers

Inflammatory measures included CRP, IL-6, Tumor Necrosis Factor-alpha (TNF-α), fibrinogen, and other cytokines. The most studied inflammatory measures were CRP, followed by IL-6 and TNF-α. Other inflammatory markers included an IL-1β, IL-1, IL-1RA, IL-4, IL-8, IL-10, sIL-6R, and TNF receptor 2 (sTNFR2). Two studies included DNA or RNA gene expression, specifically a macrophage-associated M1/M2 gene expression (RNA) phenotype and mRNA expression of glucocorticoid and mineralocorticoid receptors [53,54]. Almost all studies examined these markers from blood specimens ($n = 44$) versus saliva ($n = 2$).

2.3. Clinical foci

The studies were evenly split, with 45% ($n = 21$) of articles focused on T2D or metabolic risk, and 35% ($n = 16$) focused on depression or depressive symptoms. In 20% ($n = 9$) of studies, both depression and metabolic health were examined. Although most studies used validated tools, conceptualization and measurement of depression varied considerably across studies. Self-reported measures including the Center for Epidemiological Studies for Depression (CES—D), Beck Depression Inventory (BDI), and the Patient Health Questionnaire (PHQ-9) were the most prevalent [55–57]; however, treatment of depression as a continuous or categorical construct varied. Clinician-administered tools, including the Hamilton Rating Scale Depression (HAM—D) [58], the Structured Clinical Interview for DSM Diagnosis (SCID-I) [59], and DSM [15] criteria were also used. Measurement varied for the T2D-centered articles. Of these, three included populations with self-reported or clinically evaluated diagnoses of T2D, with one using the standard International Diabetes Federation definition [60]. Other metabolic outcome measures included anthropometrics such as body mass index (BMI), blood pressure, and waist circumference; metabolic syndrome

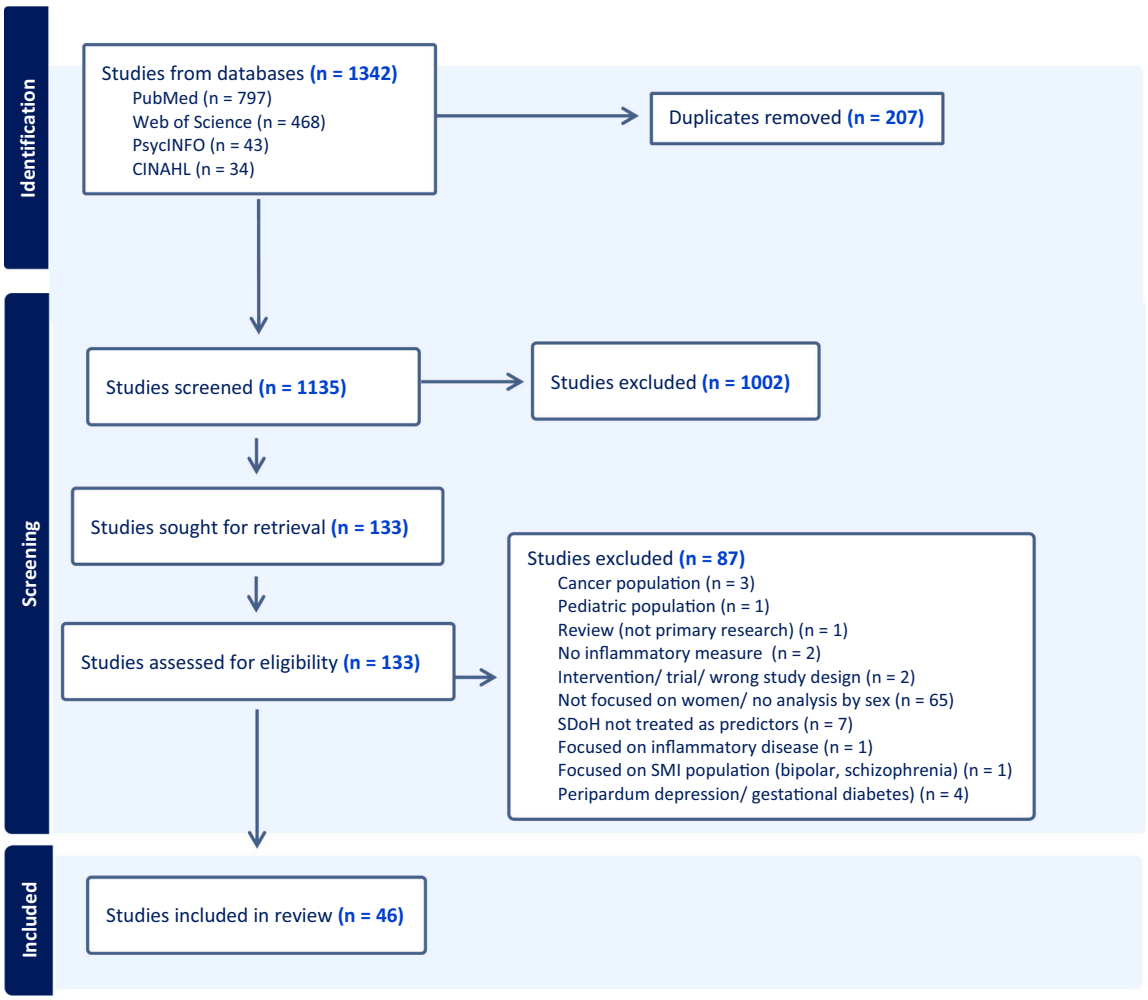

Fig. 2. PRISMA Diagram.

scores; and clinical labs (e.g., hemoglobin A1C [HbA1c]).

2.4. Trends in SDoH-inflammation links

2.4.1. Depression

The depression-focused studies included in this review suggest several adverse SDOHs are associated with increased levels of inflammation. Still, the literature in this area was limited to the behavioral/ interpersonal and sociocultural/individual matrix cells of the NIMHD research framework. ELS was significantly associated with inflammatory markers in adulthood in nine of the twelve studies (see Table 3) that investigated ELS and depression. Namely, these included higher levels of CRP, TNF-α, IL-1β, IL-6, lower levels of 1 L-2 and IL-4, and increased inflammatory gene expression. In one study, the relationship between ELS and depressive symptoms was mediated by inflammation [61]. Stress and adversity in adulthood and cumulatively also demonstrated associations with increased CRP and IL-6, in four of the depression-focused studies [45,62–64]. Sexual abuse or assault consistently associated with increased inflammatory markers (CRP, IL-6, TNF-α) in these studies [65–68]. In two studies, exposure to interpersonal/ intimate partner violence (IPV) was associated with inflammatory gene expression and CRP [53,69].

Socioeconomic disadvantage was also linked to inflammation throughout the depression-focused studies ($n = 7$). Specifically, six of these seven studies found that lower levels of income, education, and employment were associated with higher levels of CRP, 1 L-6, and fibrinogen [64,70–74].

2.4.2. Type 2 Diabetes

T2D and metabolic risks were the focus of several studies reporting associations between SDoHs across behavioral, physical/ built environment, and sociocultural environmental domains and individual, interpersonal, and community levels. Here again, ELS was associated with higher levels of CPR and IL-6 [75–77], but no significant association was found between inflammation and lifetime stress and adversity [78]. Few T2D-focused studies examined the association between sexual assault/IPV and inflammation, but significant links with CRP and IL-6 were reported among those that did [53,68,75]. Mediation effects of inflammation on the relationships between SDoHs and T2D were also identified. Specifically, IL-6 demonstrated mediation effects between social support and TD2, and sIL-6R mediated the relationship between chronic financial strain and HbA1c [79,80].

T2D-focused studies ($n = 6$) examined factors at the built environment level. Among hypothesis-confirming findings, particular material air pollution was associated with higher CRP and complement factor C3 (C3C) [45,46], which was also associated with higher rates of metabolic syndrome and/or T2D. Associations between inflammation, neighborhood disadvantage, and neighborhood safety were mixed [37,47].

Similar to findings of the depression-focused studies, lower SES education or income was associated with inflammatory markers in five out of six of the T2D-focused studies [50,80–83], except in the study in which a composite SES measure was used [84]. Other occupational and relational factors were associated with increased CRP and IL-6 including lower job control, higher work stress, marital stress, and divorce [60,85,86].

Table 2
Summary of Included Studies.

Author (Year) Country	Purpose	SDoHs	Sample size (% women)	Population	Mean Age (SD) Race**	Inflammatory Markers	Main Findings
1. Aschbacher et al., (2021) United States	Examine whether reports of adversity during childhood or pregnancy associate with inflammation ß	Early life adversity Total life adversity Adversity in pregnancy Income Interpersonal violence	53 (100%)	Child-Parent Psychotherapy Health study (CPP-HEALTH)	32.1(NR) 72% Hispanic/ Latina 13% NHW 10% Asian American 4% African American	CRP, IL-6, TNF-a, IL-1B, IL-1, IL-1RA, IL-4, IL-10, M1/M2 gene expression	Higher adversity in early life associated with higher M1/M2 gene expression. Higher lifetime adversity associated with lower immunosuppressive gene expression. Adversity during pregnancy was associated with M1/M2 imbalance among low-income women with interpersonal violence exposure.
2. Beatty et al., (2014) United States	Determine whether CRP was independently predicted by everyday discrimination or whether race or BMI modified this association over 7-years†	Everyday discrimination	2490 (100%)	Study of Women's Health Across the Nation (SWAN) Cohort	46.3(2.7) 49% White 24.6% Black 9.8% Japanese 8.8% Chinese 7.1% Hispanic	CRP	Black & Hispanic women had higher levels of CRP than White, Japanese, and Chinese women. Everyday discrimination predicts CRP over time in non-obese women regardless of race.
3. Bertone-Johnson et al., (2012) United States	Evaluate associations between early-life physical and sexual abuse and inflammation†	Childhood physical abuse Childhood Sexual abuse Interpersonal violence	702 (100%)	Premenopausal women from the Nurses' Health Study	43.8(NR) 90% White 10% NR	CRP; IL-6; TNFR2	CRP and IL-6 were associated with unwanted sexual touching and forced sex. CRP and sTNFR2 were not associated with childhood or adolescent abuse. Higher levels of CRP and IL- 6 in adulthood were associated with sexual abuse during adolescence.
4. Brummett et al., (2013) United States	Examine the association between early life SES & CRP and mediation of the association by BMI, smoking and alcohol, sex and race†	Early life-built environment Parent and participant education Annual household income	11,371 (53.8%)	National Longitudinal Study of Adolescent Health (Add Health) Cohort Wave IV	29(range 24–32) Among women, 70.26% White 29.56% Black	CRP	Increased SES, education, and built environment were directly associated with decreased CRP in White women. There was a significant direct negative relationship between SES and CRP in Black women. BMI mediated the relationship between all measures of SES and CRP for White women. No mediation in Black participants.
5. Clark et al., (2013) United States	Examined associations between neighborhood socioeconomic disadvantage, perceived neighborhood safety and cardiometabolic risk factors†	Neighborhood disadvantage Perceived neighborhood safety	3909(63%)	Jackson Heart Study	53(median 44) 100% African American	CRP	Neighborhood socioeconomic disadvantage was associated with MetS. Lack of perceived safety was associated with elevated glucose and waist circumference. Neighborhood socioeconomic disadvantage was not associated with either CRP or insulin resistance.
6. Cunningham et al., (2012) United States	Examine the influence of race, ethnicity, and gender on the association between	Experiences of discrimination	3336 (44.3%)	Coronary Artery Risk Development in Young Adults Study (CARDIA)	31.64(3.84) Black 32.63(3.37) White 28.7%	CRP	Black women reporting 1 or 2 experiences of discrimination had higher levels of CRP compared to Black

(*continued on next page*)

Table 2 (*continued*)

Author (Year) Country	Purpose	SDoHs	Sample size (% women)	Population	Mean Age (SD) Race**	Inflammatory Markers	Main Findings
	discrimination and inflammation†				White 27.0% Black		women reporting no or ≥ 3 experiences of discrimination. White women reporting ≥3 experiences of discrimination had higher levels of CRP compared to White women reporting no experiences of discrimination independent of modifiable risks.
7. Cutrona et al., (2015) United States	Test a mediation model in which chronic financial strain predicts peripheral blood stream inflammation which leads to increased HbA1c and increased T2D risk†	Financial strain	312 (100%)	Community Health Study(FACHS)	46.9(7) 100% African American	CRP, IL-6R	Chronic financial strain predicted sIL-6R. Relationship between chronic financial strain and HbA1c was mediated by sIL-6R. The association between financial strain and HbA1c was mediated by inflammation (sIL-6R).
8. D'Elia et al., (2022) Brazil	Measure differences in inflammatory markers and HPA hormone levels between women with PTSD due to sexual violence and controls at baseline and after 1-year follow-up*	Childhood trauma Sexual assault	99 (100%)	Women from a sexual assault clinic and healthy volunteers	26.3(7.2) Race NR	CRP, IL-6, TNF-a, ACTH, IL-1B	Women with childhood trauma had higher levels ACTH, IL-1B, CRP, TNF-a, and cortisol. History of sexual violence increased inflammatory markers even with depression improvement.
9. Daniels et al., (2022) United States	Determine if women with a history of chronic depression and repeated sexual violence exposure will have immune alterations that increased risk of HIV*	Sexual abuse	77 (100%)	Women's Interagency HIV Study(WIHS)	35.2 (Median 47.9) 64.9% Black 22.0% White 11.7% Other	IL-6; TNF-a; Other; IL-1a, IL-1B, IL-8, MIP-3a, IP-10	In HIV-uninfected population, compared to other groups, History of Abuse + Depression group demonstrated higher levels MIP-3a and IP-10 and lower levels of IL-1B. In HIV-infected groups, Abuse and Abuse + Depression groups had higher levels IL-6 and lower levels of MIP-3a and IP-10.
10. Dittrich et al., (2021) Germany	Determine the effects of (rMDD) and early life maltreatment (ELM) on CRP and IL-6. And determine how rMDD and ELM types affect CRP and IL-6*	ELM of any type	126 (100%)	Multicenter clinical population study Understanding and Breaking the Intergenerational Cycle of Abuse (UBICA)	39.22(5.78) German Nationality Race NR	CRP; IL-6	Sum severity of ELM was correlated with CRP. Higher neglect and physical abuse scores were correlated with CRP. Physical abuse and overall severity of ELM were positively correlated with CRP.
11. Elovainio et al.,2010) United Kingdom	Evaluate if organizational justice contributes to inflammation over a 14-year follow-up independent of age, sex, occupational grade, BMI smoking, depression, and alcohol β	Organizational justice	4408 (27%)	Whitehall II study, (British civil servants)	43.9(5.9) Race NR	CRP; IL-6	No relationship between organizational justice and CRP or IL-6.
12. Fang et al., (2014) United States	Examine associations between acculturative stress and inflammatory markers among Chinese American women†	Negative life events Acculturation stress	407 (100%)	Community sample	43.8(4.54) 100% Foreign-born Chinese American	CRP; TNF receptor 2 (sTNFR2)	Higher levels of acculturative stress were positively associated with CRP and sTNFR2. Stressful life events were not associated with CRP or sTNFR2.
13. Fuller et al., (2019)	To estimate the association ambient particle number	Negative life events PNC	1499 (70%)	Boston Puerto Rican Health Study (BPRHS)	57.3(7.5) 100%	CRP	Women had higher CRP levels than men. Stress and negative life

(*continued on next page*)

Table 2 (*continued*)

Author (Year) Country	Purpose	SDoHs	Sample size (% women)	Population	Mean Age (SD) Race**	Inflammatory Markers	Main Findings
United States	concentration (PNC) on CRP and modification by depression, acculturation, stress, discrimination, negative life events ß				Puerto Rican		events increased the impact of air pollution on CRP.
14. Gardhouse et al., (2021) Canada	Investigate whether neuroendoimmune activity mediates the relationship between perceived stress and depressive symptoms*	Early childhood stress Lifetime stress experiences	59 (100%)	medically healthy adult females	28.7(9.1) 53.9% White 12.6% >1 race 27.0% Asian 3.2% Black 3.2% Other	CRP; IL-6; TNF-a; cortisol	Cumulative life stress severity was associated with increased free cortisol. Increased IL6 and decreased free cortisol mediate the effect of early childhood stress on depressive symptoms.
15. Ghosh et al., (2018) United States	Examine immuno-biological mechanisms associated with sexual abuse, depressive symptoms, and HIV*	Chronic sexual abuse	77 (100%)	WIHS	35.2 (Median 47.9) 64.9% Black 22.0% White 11.7% Other	IL-6; TNF-aIIL-1a, IL-1B, IL-8, MIP-3a, IP-10	In HIV- women, chronic sexual abuse was associated with increased IL-1a, MIP-3a, and IP-10. In HIV+ women, chronic sexual abuse was associated with decreased TNFa, IL6, IL1a, and increased MIP3a.
16. Gruenewald et al., (2009) United States	Examine SES gradients in levels of CRP and IL-6 among Black and White men and women β	Income Education	3549 (50.7%)	CARDIA	Black females 44.7 (3.9); White females 45.7 (3.4) 50.0% White 49.9% Black	CRP; IL-6	CRP and IL-6 levels were inversely associated with education and income levels in White and Black females. CRP and IL-6 levels were inversely associated with education and income levels in both White males and females. Inflammation biomarker levels inversely associated with SES variables in Black females, except for CRP by income level.
17. Halpern et al., (2016) United States	Describe the association among intimate partner violence (IPV) exposure, facial injury, and adverse health outcomes*	IPV	78 (100%)	females, aged 18–64 years from the Meharry Medical College Oral Surgery Clinic	Age NR 47.44% African American 52.56% other ethnicities	CRP; Cortisol; Cortisol/DHEA; and CRP from saliva	Stress, PTSD, and depression were significantly greater in IPV+ African American females. There were no significant differences seen in Cortisol, Cortisol/DHEA ratios, and CRP between IPV+ and IPV- groups. Salivary DHEA was statistically significant in IPV+. No differences were seen in Cortisol, Cortisol/DHEA ratios, and CRP between IPV+ and IPV- groups.
18. Heath et al., (2013) United States	Investigate the relationships between IPV, psychological distress, and CRP*	IPV	139 (100%)	GYN patients with no major illnesses	28.46(7.76) 83.5% Black 5.8% White 6.4% Other 4.3% Hispanic/ Latina	CRP	Exposure to IPV predicted the presence of depression and PTSD diagnoses. Individuals who experienced PTSD exhibited higher CRP levels, even after adjusting for depression.
19. Hellmann-Regen et al., (2019) Germany	Disentangle the effects of ACEs on endocrine and immune alterations in MDD*	ACEs Childhood Trauma	87 (100%)	women with depression and ACEs	32(11) MDD- / ACE– 37(10) MDD+ / ACE- 33(11)	mRNA expression of glucocorticoid (GR) and mineralocorticoid receptor (MR) ex vivo in PBMC	Increased proliferation rate of PBMCs in women with ACE detected, regardless of MDD. Neither childhood trauma nor depression had an effect on ex vivo

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Table 2 (*continued*)

Author (Year) Country	Purpose	SDoHs	Sample size (% women)	Population	Mean Age (SD) Race**	Inflammatory Markers	Main Findings
					MDD+ / ACE+ 35(12) MDD- / ACE+ Race NR		GR sensitivity. No main effects of group on GR and MR expression as assessed by mRNA expression. Women with ACE showed an increased immune response after mitogen stimulation independent of MDD.
20. Hostinar et al., (2015) United States	Examine childhood and recent stressors in inflammation at mid-life and behavioral modifiers of the associations*	ACEs Recent life events (RLEs)	1180 (56%)	middle-aged and older adults from the Midlife in the United States (MIDUS) Biomarker Project	57.3(11.5) 74.9% NHW 17.9% African American 4% Other 3.2% Hispanic	CRP; IL-6; fibrinogen, Molecule-1 (ICAM-1)	ACEs and RLEs were independently associated with higher levels of inflammation. ACEs and RLEs were independently associated with higher levels of inflammation.
21. Hughes et al., (2015) United Kingdom	Examine associations of unemployment with inflammatory markers*	Unemployment	23,025 (52.7%)	men and women participants from the Health Survey for England(HSE) and Scottish Health Survey(SHeS) between 16 and 64 years of age	43.1(12.4) Race NR	CRP; fibrinogen	CRP, fibrinogen and odds of CRP >3 mg/L were significantly raised for unemployed participants, even following adjustment for several social and clinical covariates. Employment predicted CRP, fibrinogen, and odds of CRP >3 mg/L among English participants. In Scotland, associations with all three measures of inflammation were robust. Within England, there were regional interactions for CRP/ fibrinogen.
22. Keita et al., (2014) United States	Examine the association of neighborhood socioeconomic deprivation and metabolic syndrome with inflammation†	Neighborhood area deprivation	19,079 baseline Quintile 1, 2309(62%); Quintile 2, 2191(58%); Quintile 3, 2036(54%); Quintile 4, 1961(52%); Quintile 5, 1838(49%).	Black and White participants from the Reasons for Geographic and Racial Differences in Stroke Study who were age > 45 years	64.5(9.5) 61% White 39% Black	CRP	Among Black adults, residing in the most deprived neighborhoods was associated with increased odds of elevated fasting glucose, inflammation, and CRP-MetS. Among White adults, neighborhood deprivation was associated with higher glucose, inflammation, and CRP-MetS.
23. Kim et al., (2019) United States	Examine the link between three types ACEs and biomarkers of neuroplasticity (BDNF]), CRP and difference by sex*	ACEs	85 (71.8%)	Undergraduate students at a state university	20.68(1.76) 33% NHW 54% Hispanic 13% Other	CRP	Childhood adversity and particularly family dysfunction had a significant positive effect on BDNF. The link between family dysfunction and CRP was stronger in female students. Family dysfunction was a significant predictor of CRP and BDNF.
24. Krämer et al.,2010) Germany	Examine the association between traffic-related air pollution and incident T2D†	Air pollution (PM)	1775 (100%)	nondiabetic middle-aged women from the Study on the Influence of Air Pollution on Lung, Inflammation and Aging (SALIA)	54.4(0.70) Race NR	Complement factor 3 cleavage product C3c	Hazards for T2D were increased by 15–42% per interquartile range of PM or traffic-related exposure; C3c was associated with PM pollution at baseline and was a strong independent predictor of T2D.

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Table 2 (*continued*)

Author (Year) Country	Purpose	SDoHs	Sample size (% women)	Population	Mean Age (SD) Race**	Inflammatory Markers	Main Findings
25. Li et al., (2022) China	Attempt to link stressful life events (SLEs) and depression through multiple analysis and to explore depression susceptibility*	SLEs Income	185 (49.73%)	healthy community participants with stressful life events	47.51(NR) Race NR	IL-6	The path analysis model confirmed the significant partial effect of SLEs-IL-6-white matter (WM) network differences-depression. Females, individuals with lower exercise frequency or income were more likely to have higher IL-6 level after SLEs.
26. Lopes et al., (2012) Brazil	Investigate the effects of childhood maltreatment (CMT) and PTSD symptoms on neuroendocrine/ immunological components of women with recurrent MDD*	ELS CMT	53 (100%)	38 females with MDD patients and 15 HCs	39.73 (10.12) MDD 41.06(6.46) CMT 37.078 (5.64) HC Race NR	IL-6; TNF-a; IL-2, IL-4, IL-6, IL-10, TNF-a, and IFN- y.	MDD participants +/− previous trauma had similarly lower cortisol and DHEAS and blunted T-cell proliferation. Their PBMCs were significantly less sensitive to dexamethasone or epinephrine than the HCs. The MDD and CMT groups had significantly lower IL-2 and IL-4 levels compared to HCs.
27. MagnussonHanson et al., (2019) United Kingdom	Investigate whether the circulating inflammatory markers mediate the relationship between work characteristics and diabetes†	Social support at work Job strain Job demands Job control	4638 (28%)	Whitehall II cohort	49.6(6.0) Race NR	CRP; IL-6	Low social support at work, but neither high job demands nor low job control was associated with diabetes and higher IL-6. Inflammatory markers and diabetes were bidirectionally associated. IL-6 partially mediated the association between social support and diabetes, job control associated with IL-6 in women.
28. Magnusson Hanson et al., (2017) France	Quantify differences in adverse adiposity, metabolic, respiratory, and inflammatory biomarker levels between individuals with and without work stress†	Work stress	43,593 (52%)	working adults from a French population-based sample aged 18–72 years (the CONSTANCES cohort)	43(10.8) Race NR	White blood cell count, triglycerides, LDL, HDL, total cholesterol	Work stress was associated altered metabolic profile, increased systemic inflammation. Work stress was associated with higher WBC and altered metabolic profile associated with increased cardiometabolic risk.
29. Morris et al., (2011) United States	Test whether the association between depression and inflammation differs by race and sex*	Race	512 (60.5%)	Morehouse and Emory Team up to Eliminate Health Disparities (META-Health) Study	52(9) White 50(9) African American 53.5% White 46.5% Black	CRP	No difference in depression scores between African American and White women. White women with mild to severe depression had higher CRP. CRP and depression were only associated in white women.
30. Nakamuraet al., (2022) United States	Identify modifiable, social factors that moderate the relationship between ELS and depressive symptoms and inflammation*	Early life stress	3416 (58.25%)	adults in the 2006 wave of the Health and Retirement Study, a nationally representative sample of older adults in the United States	68.41 (10.24) Race NR	CRP	A small, positive association between ELS and depressive symptoms, moderated by social contact and perceived support was detected. ELS was only associated with elevated depressive symptoms for participants with limited social contact

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Author (Year) Country	Purpose	SDoHs	Sample size (% women)	Population	Mean Age (SD) Race**	Inflammatory Markers	Main Findings
							and low perceived support.
31. Newton et al., (2011) United States	Examine associations between IPV and CRP and IL-6 in midlife women with past IPV ß	IPV Adversity before age of 12	68 (100%)	Apparently healthy, midlife women with divorce histories recruited from the community	54.68(3.23) 85.29% European American or White 14.71% African American or Black	CRP; IL-6; also in vitro IL-6 production by PBMCs	A history of being stalked was positively correlated with CRP levels, association was attenuated adjusting for BMI. Physical assault history was negatively correlated IL-6. IPV was associated with increased CRP and in vitro stimulated IL-6 but not associated with depression. Childhood adversity was not associated with biomarkers.
32. Niles et al., (2018) United States	Examine longitudinal associations (over 4 years) between depression and anxiety symptoms and inflammation*	Education	13,775 (59%)	Health and Retirement Study	66.7(10.8) 2006 Cohort 68.3(10.5) 2008 Cohort 80–84% White 16–20% African American or Other	CRP	Inflammation predicted worsening depression for women, but not men. Somatic symptoms predicted increasing inflammation for men and were predicted by inflammation for women. Inflammation predicted worsening dysphoric symptoms of depression. Lack of positive affect predicted increasing inflammation. Higher CRP was associated with older age and lower education.
33. Osborn et al., (2020) United States	Determine whether childhood maltreatment is associated with higher CRP later in life and whether individuals with official and retrospective self-reports of maltreatment and men and women show similar increases in risk†	Childhood physical/sexual abuse and neglect	443 (48.5%)	offspring of parents from a longitudinal study about consequences of childhood maltreatment	23.4(5.23) 59.1% White 40.9% Black	CRP	Individuals with official reports of child maltreatment and, specifically, physical abuse, had significantly higher levels of CRP than non-maltreated individuals. Maltreated females showed elevated CRP, independent of control variables, whereas no significant association was observed in males. Retrospective self-report measures of child maltreatment did not predict elevated CRP. Official reports of childhood maltreatment were predictive of elevated CRP in women.
34. Phillips et al., (2009) United States	Examine the association of both parental and individual educational attainment with CRP†	Parental education attainment Individual educational attainment	811 (51%)	mid-life community sample participants from the University of Pittsburgh Adult Health and Behavior (AHAB) project	44.8 (6.7) 84% European-American (women) 16% African-American (women)	CRP	Both parental education and individual education was associated inversely with CRP in women, but not in men. This persisted after adjustment for both lifestyle risk factors and individual SES. Women whose parents had fewer years of education showed higher plasma concentrations of CRP than those with higher parental educational

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Author (Year) Country	Purpose	SDoHs	Sample size (% women)	Population	Mean Age (SD) Race**	Inflammatory Markers	Main Findings
							attainment, an effect that was independent of women's own educational attainment, age, and race.
35. Pikhartova et al., (2014) United Kingdom	Examine the role of childhood social position and later inflammatory markers and health behaviors in developing T2D at older ages using a pathway analytic approach†	Childhood social position	7015 (57%)	men and women from the English Longitudinal Study of Aging cohort	68.9(NR) Race NR	CRP	Disadvantaged social position in childhood, measured as a father's manual occupation, was associated with an increased risk of T2D in adulthood. This relationship was mediated by inflammation. Childhood SES was associated with increased CRP and T2D later in life.
36. Ploubidis et al., (2015) United Kingdom	Examine the association between trajectories of partnership status over the life course and health indicators in midlife†	Partnership status	18,558 (52%)	data from the British National Child Development Study, birth cohort study	44–46 (NR) Race NR	CRP	A longitudinal typology of partnership status spanning 21 years was associated with inflammatory and hemostatic markers and health outcomes. Midlife women who divorced in their 30s had increased midlife CRP compared to women who married and stayed married in their 20s.
37. Powers et al., (2016) United States	Examine the differential associations between trauma exposure, emotion dysregulation, current MDD, current PTSD, and CRP in a traumatized urban, sample of women with T2D ß	Childhood abuse Lifetime trauma	40 (100%)	African-American women with T2DM recruited from an urban hospital	51.88(7.57) 100% African American	CRP	Emotional dysregulation was significantly positively correlated with CRP concentrations. The association between child abuse severity and CRP trended toward significance. However, overall trauma exposure (excluding child abuse) was not significantly correlated with CRP. Child abuse was not significantly related to CRP among AA women with T2D.
38. Prescott et al., (2007) Denmark	Explore the association between socio-economic factors and MetS and whether associations are explained by psychosocial and behavioral factors†	Education	6038 (57%)	random sample of the general population from the Copenhagen City Heart Study	46–66* mean varied slightly by quintile Race not reported	CRP	Higher educational was associated with a better metabolic profile (waist/hip ratio), dyslipidemia (HDL and triglycerides), SBP, glucose intolerance, and CRP. MetS was strongly associated with educational level in both men and women. Psychosocial/ behavioral factors did not explain association between educational level and MetS components. Education level was inversely correlated with CRP and all other measures of MetS.
39. Rathmann et al., (2006)	Evaluate whether CRP impacts the associations of low SES	Education Occupational	1476 (48.06%)	data from The KORA (Cooperative Health Research in the	65.1(4.7)* (low SES) 63.1(5.0)	CRP	Low SES was not related to prediabetes. CRP was significantly associated

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Author (Year) Country	Purpose	SDoHs	Sample size (% women)	Population	Mean Age (SD) Race**	Inflammatory Markers	Main Findings
Germany	with T2D or diabetes precursors (impaired glucose tolerance, impaired fasting glucose) in the KORA Survey participants†	status Income		Region of Augsburg) Survey 2000 (a population-based study in Southern Germany)	(mid SES) 63.5 (6.0) (high SES) Race NR		with diabetes precursors. Inflammation appeared not to play a major role linking low SES and T2D in older adults. In women but not in men, continuously decreasing CRP with increasing SES were found. After stratifying for glucose tolerance status, the inverse relation remained in women with NGT and prediabetes but in T2D subjects' trend was not significant.
40. Saban et al., (2018) United States	Examine the influence of perceived discrimination on the inflammatory response to a laboratory acute stress paradigm in women at risk for CVD†	Perceived discrimination	99 (100%)	postmenopausal women with 2 or more CVD risk factors	60.2(6.7) 50.5% African American 49.50% NHW	CRP; IL-6	Perceived discrimination was significantly associated with the IL-6 response to the TSST, and CRP.
41. Shen et al., (2010) Sweden	Examine the relationships among marital stress, waist circumference, and CRP; Test whether marital stress was associated with CRP and whether this association was moderated by WC†	Marital stress	201 (100%)	Healthy married or partnered women from the Stockholm Female Coronary Risk Study	56.13(6.99) Race NR	CRP	Marital stress was not directly associated with CRP. There was a significant interaction between marital stress and WC such that marital stress was positively associated with CRP among women with a larger waist.
42. Stacey et al., (2019) Australia	Compare general and biomedical health status of informal carers with non-carers with an emphasis on gender differences ß	Family caregiving	1788 (52.07%)	members of the Northwest Adelaide Health Study 40 years and older	Age 40 and over Race NR	CRP; IL-6; TNF-a	Among women, there were no significant differences with regard to the other inflammatory biomarkers IL-6, and TNFa. Association between caregiving and CRP, IL6, TNFa; hsCRP was positively associated with caregiving only when men and women analyzed together.
43. Stepanikova et al., (2017) United States	Investigate the associations BMI and WC with markers of systemic inflammation in midlife by race and gender†	Race Discrimination Income Education	1075 (56.47%)	non-institutionalized English speaking US residents over 35 years from the Survey of Midlife in the United States (MIDUS II)	54.79 (11.55) White women 51.50 (10.95) Black women 84% White 16% Black	CRP; IL-6; fibrinogen; E-selectin	For most race-gender groups, WC showed more consistent associations with inflammation markers than did BMI, although the observed relationships varied by inflammation measure and population group. For White men and women, WC related to all four biomarkers of inflammation. For Black women, WC was a significant contributor to fibrinogen, CRP, and IL-6. Higher education was related to lower fibrinogen among White women. Biomarkers that increased with income included fibrinogen and CRP for White women, and IL-6 for Black

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Author (Year) Country	Purpose	SDoHs	Sample size (% women)	Population	Mean Age (SD) Race**	Inflammatory Markers	Main Findings
							women. E-selectin concentrations decreased with higher income among Black women. Perceived lifetime discrimination was associated with higher fibrinogen, CRP, and IL-6 concentrations among White women, and higher fibrinogen among Black women.
44. Waisberg et al., (2011) South Africa	Determine ethnic differences in body fat distribution, adipokine levels, and SES that may explain population differences in the prevalence of these metabolic disorders†	SES Education Ethnicity	100 (100%)	African and Indian women residing in the Greater Johannesburg area	45.4(1.1) Indian 42.2(0.9) African 50% African 50% Asian-Indian	CRP; IL-6; TNF-a	TNFa levels were significantly higher in the African group, whilst visceral fat levels were significantly lower. The CRP and IL-6 levels were not different between groups. Ethnic differences in CRP, IL-6 or leptin receptor were not noted in lean or obese subjects. No correlations were observed between TNFa, IL-6 and anthropometric parameters, CT-scan analysis and SES or education in either ethnic group.
45. Weaver et al., (2015) India	Develop and test a series of hypotheses about the relationships that might exist between diabetes, psychological distress, social role fulfillment, and biological markers measuring blood sugar control, generalized inflammation, and immune stress ß	Symptoms of "tension" Stressful life events Number of children Joint family status SES	280 (100%)	women with and without T2D from approached in clinic waiting rooms	54 (diabetes) 47.3(no diabetes) Range: 27.0–78.0 Race NR	CRP	CRP not associated with SES or life events in women with or without T2D. Results suggest that influences from North Indian women's with T2D social lives become embodied in their physical and mental health.
46. Zainal et al., (2021) United States	Test if elevated inflammatory activity predicts future within-person 9-year change in MDD diagnosis*	Childhood trauma Income	945 (52.78%)	community-dwelling adults	54.33 (11.06) 95.37% White 4.63% African American, Asian, Pacific Islander, or other ethnicities	CRP; IL-6, fibrinogen	Increased CRP, fibrinogen, and IL-6 levels predicted 9-year MDD diagnostic status change more strongly in younger than older adults, women but not men, those with low (vs. high) income, as well as persons with high (vs. low) childhood trauma frequency and number of chronic illnesses. CRP, IL6 predicted depression most strongly in women with low incomes, high childhood trauma.

Note: Clinical foci of the studies are denoted as follows: * = depression, † = type 2diabetes/ metabolic risk, ß = depression + type 2 diabetes/ metabolic risk; ** racial composition given for women only when relevant and available in the studies; ACEs = adverse childhood experiences; ACTH = Adrenocorticotropic hormone; BMI = body mass index; CRP = C-reactive protein; CVD = Cardiovascular disease; DHEAS = dehydroepiandrosterone sulfate; GR = glucocorticoid; HbA1c = hemoglobin A1C; HPA = hypothalamic-pituitary-adrenal; IL = interleukin; IP-10 = protein-10; IPV = intimate partner violence; MDD = major depressive disorder; MetS = metabolic syndrome; MIP-3α = macrophage inflammatory protein 3-alpha; M1/M2 ratio = Immunogenomic phenotypes (M1/M2 ratio); MR = mineralocorticoid receptor; NGT = normal glucose tolerance; NHW = Non-Hispanic White; NR = not reported; PBMC = Peripheral blood-derived mononuclear cells; PTSD = post-traumatic stress disorder; rMDD = major depressive disorder in remission; SDoH = social determinant of health; SE = socioeconomic; SES = socioeconomic status; sTNFR2 = the soluble fraction of tumor necrosis factor alpha receptor 2; TNF = tumor necrosis factor; TSST = Trier Social Stress Test; T2D = Type 2 Diabetes; WC = Waist Circumference.

Table 3
Social Determinants of Health Explored by Included Studies Across NIMHD Research Framework.

		Levels of Influence			
		Individual (22/46 = 47%)	Interpersonal (36/46 = 78%)	Community (8/26 = 17%)	Societal
Domains of Influence (Over the Life course)	Biological Behavioral (27/46 = 58%)		Childhood adversity, trauma, abuse, stress[8,10,14,19,20,23,26,30,46*; 3,4,33,34,35†; 1,31,37ß] Sexual abuse or assault[8,9,15*; 3†; 31ß] Interpersonal/ intimate partner violence[17,18*; 3†; 1ß] Lifetime stress and adversity [14,20,25*; 12†; 13,37,4] Family caregiving[42ß] Marital stress[41†]		
	Physical/ Built Environment (6/46 = 13%)		Early-life built environment[4†] Number of children and joint family status[45ß]	Air pollution[24†; 13ß] Neighborhood deprivation[22†] Neighborhood disadvantage [5†] Perceived neighborhood safety[5†]	
	Sociocultural Environment (27/46 = 58%)	Acculturation stress [12†; 13ß] Race or Ethnicity[29*; 4,22,43,44†; 16ß] Income [25,46*; 4,39,43†; 1,16ß] Education[29,32*; 4,34,38,39,43,44†; 16ß] SES composite[44†; 45ß] Employment Status[21*; 39†] Financial strain[7†] Work stress[28†]	Partnership or marital status[36†] Social support at work[27†] Job demands and control[27†] Everyday discrimination [2,6,40,43†; 13ß]	Community discrimination [2,6,40†] Organizational justice[11ß]	
	Health System				

Note: Table 3 displays SDoHs explored in the studies according to clinical focus of depression/depressive symptoms(*), T2D/metabolic risk(†), or both (ß). Studies with null findings are indicated in grey. The percentages of studies that assessed SDoHs across 1) domains and 2) levels of influence are also indicated. Occasionally, SDoHs of interest spanned more than one domain or level of influence, and therefore domain and level percentage totals may exceed 100%. [1]Aschbacher et al., (2021); [2]Beatty et al., (2014); [3]Bertone-Johnson et al., (2012); [4]Brummett et al., (2013); [5]Clark et al., (2013); [6]Cunningham et al., (2012); [7]Cutrona et al., (2015); [8]D'Elia et al., (2022); [9]Daniels et al., (2022); [10]Dittrich et al., (2021); [11]Elovainio et al.,(2010); [12]Fang et al., (2014); [13]Fuller et al., (2019); [14]Gardhouse et al., (2021); [15]Ghosh et al., (2018); [16]Gruenewald et al., (2009); [17]Halpern et al., (2016); [18]Heath et al., (2013); [19]Hellmann-Regen et al., (2019); [20]Hostinar et al., (2015); [21]Hughes et al., (2015); [22]Keita et al., (2014); [23]Kim et al., (2019); [24]Krämer et al.,2010); [25]Li et al., (2022); [26]Lopes et al., (2012); [27]Magnusson Hanson et al., (2019); [28]Magnusson Hanson et al., (2017); [29]Morris et al., (2011); [30]Nakamuraet al., (2022); [31]Newton et al., (2011); [32]Niles et al., (2018); [33]Osborn et al., (2020); [34]Phillips et al., (2009); [35]Pikhartova et al., (2014); [36]Ploubidis et al., (2015); [37]Powers et al., (2016); [38]Prescott et al., (2007); [39]Rathmann et al., (2006); [40]Saban et al., (2018); [41]Shen et al., (2010); [42]Stacey et al., (2019); [43]Stepanikova et al., (2017); [44]Waisberg et al., (2011); [45]Weaver et al., (2015); [46]Zainal et al., (2021).

The T2D-focused studies examined factors particularly relevant to minoritized populations. These factors included racial discrimination, and acculturative stress, which were found to be associated with higher CRP and IL-6 [31,78,87–89]. Several studies also found that associations between various SDoHs and inflammation varied by race or ethnicity [50,71,84,89].

2.4.3. Co-occuring Depression and Type 2 Diabetes

Four of the nine papers that had a dual focus on depression and T2D, found significant associations between SDoHs and measures of inflammation. Across the four studies, adverse SDoHs were associated with higher levels of CRP, IL-6, and M1/M2 inflammatory gene expression. These SDoHs included early life adversity, lower SES such as low-income and lower education, and interpersonal violence [71,90,91]. One study found that stress and negative life events increased the impact of air pollution on CRP [92]. Table 3 displays the SDoHs studied including null results comparing clinical foci across the NIMHD framework.

3. Discussion

3.1. Summary of key findings

In this scoping review, we found mounting evidence of inflammatory links with two key areas across the SDoH landscape among women with or at risk for depression and T2D. First, a wealth of evidence implicates stress, adversity, and trauma across the life course as important predictors of inflammation within the context of depression and T2D. Second, components of socioeconomic disadvantage were associated with a variety of inflammatory markers. Our findings are aligned with and expand a recent review of articles linking SDoHs to psychological symptoms through epigenetic mechanisms, wherein authors identified studies pointing to differential methylation of genes related to inflammatory processes [93]. Building on these findings, we reviewed literature that focused on not only inflammatory genes but the markers themselves. Further, nearly 70% of the identified literature in that review focused on depressive symptoms. Similar to our findings, stress and trauma, particularly in childhood, were frequently studied, and studies of built/ environment or healthcare system effects were rarely identified. The distribution of accumulated knowledge related to socioeconomic and early life stress (abundant), discrimination and neighborhood environment (limited), and healthcare system (virtually non-existent) links to inflammation in our review is similar to general population studies [94–97]. This context suggests that SDoHs which are understudied in this review may not be unique to women with or at risk for depression and T2D. To the best of our knowledge, ours is the first review expressly aimed at identifying inflammation-linked SDoHs in the context of depression and T2D in women—a population disproportionately affected by these conditions.

3.2. Early life stress

Findings from this review support a large body of literature linking early life adversity with chronic disease [98,99]. The foundational work in this area was conducted in the late 1990s by Felitti and colleagues, who investigated ten adverse childhood experiences, now referred to as "ACEs," including abuse, neglect, and household dysfunction (e.g.,

witnessing intimate partner violence). These investigators found that ACEs were associated with a myriad of health problems and health-risk behaviors in adulthood, including cardiovascular disease, cancer, obesity, depression, smoking, and alcoholism [41]. Since this time, a burgeoning field of research has further confirmed the link between early life adversity and health [100,101]. The concept of childhood adversity has been expanded to include other potentially traumatic early life experiences, including experiences of racial discrimination, bullying from peers, and food or housing insecurity [102,103].

While the links between childhood adversity and adulthood disease are multifactorial and complex, stress and inflammation have been implicated as major pathways through which early adversity "gets under the skin" [104,105]. Shonkoff and colleagues coined the term "toxic stress," which describes the physiologic process through which exposure to chronic stressors contributes to poor health [106]. In a toxic stress response, persistent activation of the HPA axis and sympathetic nervous system lead to a release of primary mediators of the stress response, including glucocorticoids, catecholamines, and inflammatory cytokines. Over time, these mediators disrupt brain development and "wear and tear" on the cardiovascular, respiratory, metabolic, and immune systems, and ultimately contribute to chronic disease and susceptibility to illness [107,108].

3.3. Adulthood interpersonal violence and abuse

Our findings concerning the deleterious effects of interpersonal violence are consistent with a previous review of chronic inflammation and violent incidents globally [109]. IPV, sexual abuse, and sexual assault during adulthood have a discernible impact on inflammation through pathways involving PTSD, depression, or other forms of psychological distress. Heath et al. (2013) noted mediation effects of depression and PTSD on the relationship between IPV and inflammation [69]. Intercorrelations between stress (such as violence), depression, and inflammation have also been proposed [110,111]. However, there is a noticeable gap in the consideration of socioeconomic context on women's health. A recent review found that the highest correlations of inflammatory diseases with violence exist among lower socioeconomic populations [109], suggesting that certain advantageous SDoHs such as a robust social network and improved financial circumstances, may act as inflammation-mitigating factors against the adverse effects of abuse.

3.4. Physical/built environment

Physical/Built Environment factors were minimally represented in this review. This finding is striking, given the major impact of environmental factors (e.g., water sanitation, air quality, safety, housing) on health and well-being [112]. The built environment was defined differently across the included studies, making it difficult to ascertain specific environmental factors that impact inflammatory markers in women. Operationalizing the European Union [113] or the United Nations Sustainability Development Goals [114] as frameworks could support a comprehensive understanding of the impact of the environment on health to drive public projects and policies to improve health outcomes globally. Understanding inflammation as a potential pathway linking built environment exposure and health outcomes may improve risk stratification and precision risk mitigation efforts which may have potential for use before the downstream emergence of disease.

The links between the built environment, SES, and racial discrimination are difficult to disentangle. For example, lower SES is associated with a neighborhood food environment that negatively impacts BMI and contributes to the overall risk of poor cardiometabolic health [115,116]. Built environment factors may also be attributable to racial discrimination that contributes to lower SES in historically minoritized populations (i.e., structural racism), and synergistic effects of built environment and discrimination are likely at play. Using objective measures of neighborhoods that include racial heterogeneity, urbanicity, and residential stability as done in one reviewed study [48] has the potential to disentangle the impact of the built environment, individual SES, and racial discrimination on inflammatory markers.

Material needs insecurity was largely unexamined in the included studies, representing an important gap. Food insecurity, housing insecurity, and healthcare insecurity are known risk factors for depression and other mental health concerns as well as impediments to self-management that is essential to those with or at risk for T2D [14,117,118]. Food insecurity is particularly illustrative, as it is related to other risk factors for depression and T2D such as poor diet [119]. For women in particular—who are often socialized to put others' needs ahead of their own—food insecurity has been identified as an important component of disease syndemic. For example, refusing to allow family members—particularly children—to go hungry, can further situate women in high-risk cyclical contexts (e.g., transactional sex, sexual violence, and drug use) [120]. Further, links between food insecurity and inflammation have been demonstrated in the general population and among women with HIV [121,122]. Given the effect of physical environment and nutrition on the gut microbiome and the growing recognition of the gut microbiome's role in inflammation, depression, and T2D [123–125], further examination of material needs insecurity in conjunction with biological markers is warranted.

3.5. Socioeconomic status

Socioeconomic status was consistently associated with increases in inflammatory markers despite measurement and geographic variations. A strong body of evidence implicates lower SES as a stronger risk factor for early mortality than many lifestyle behaviors [126–128]. Whether SES alone or the implicit bias of SES proxy measures (i.e., educational attainment, occupational prestige) contributes to its role as an SDoH requires further study. Utilizing common data elements such as the individual and structural SDoH PhenX Toolkit measures can enhance study comparability and reduce implicit social order biases. Occupational and organizational factors of this domain are underexplored yet show significant inflammatory effects when job-related characteristics are suboptimal.

3.6. Discrimination and acculturative stress

Thus far, our discussion has centered on the SDoHs that relate to experiences and access to material resources—in other words, what you have. However, a handful of studies in our review focused on SDoHs related to membership within a historically marginalized, minoritized, or non-dominant group—in other words, who you are. These factors include experiences of discrimination and acculturative stress. Although discrimination was not explored in the depression-focused studies in our review, it is a well-known risk factor for depression, especially for Black women in the U.S. [129–132]. Further, there is evidence that experiences of discrimination imparts changes to the epigenome [133,134]. Several studies focused on T2D in our review showed associations between discrimination and inflammatory markers, in line with other studies [29]. What remains less explored across the broader literature are the specific reasons for discrimination (e.g., gender, race, class), which may stem from multiple intersecting identities. Only two studies explored acculturative stress, which may have resulted from our restriction to English-language articles. Acculturative stress can involve significant cultural instability, separation from family, language barriers, and social isolation, and it has been linked to a host of poor health outcomes and the upregulation of genes involved in the inflammatory process [78]. More research is needed to better understand how strategies to promote equity, inclusion, and belonging may mitigate deleterious effects of these factors on health which are each fundamentally related to being alienated and devalued.

3.7. Limitations and strengths

Our review is not without limitations. Firstly, it is possible that relevant articles were not captured by our search strategy, as looking comprehensively at just one specific SDoH requires many terms. However, our objective in this scoping review was to cast a broad net, as a starting point for future in-depth systematic reviews on underexplored SDoHs. Our consultation with a health science librarian helped to mitigate the risk of missed articles as did our decision to include both MeSH terms and keywords so that our search would not be limited only to indexed literature. Secondly, although the selected studies spanned six continents, an overwhelming majority of the articles were focused on populations from North America and Europe, limiting our ability to generalize to a global population of women. It is difficult to know if this lack of representation was an effect of our exclusion criteria or truly reflective of the available science. The findings of these studies need to be interpreted with caution and contextual consideration as SDoHs may differ significantly by country and/or regionality. Thirdly, we did not search for factors that could contribute to buffering disparities (e.g., community cohesion, social support, resilience), as this was outside the scope of our questions. Psychological factors such as cognitive appraisal of stressors were also not included, and it is important to note that appraisal is often necessary for external *stressors* to become internalized *stress* [135]. Interindividual variation of these qualities may have more influence on inflammation than intra-individual differences in stressor exposure. However, this dichotomy is likely false, as a role for both processes has been suggested in multiple frameworks [34,136]. A strength inherent to our focus on external factors (SDoH) is that it may guide actionable steps to developing strategies for healthy communities rather than reliance on individual-level behavioral interventions (e.g., strategies to improve coping) delivered individually within strained healthcare systems.

3.8. Future directions

Our findings underscore the importance of assessing SDoHs when examining inflammation associations with depression and T2D. As most of the articles in this review focused on the inflammation effect of one or two SDoHs at a time, our current understanding of how exposure to multiple disadvantageous SDoHs is limited. Future studies should prioritize prospective longitudinal designs and consider how the SDoHs may work synergistically (e.g., discrimination, unemployment, food insecurity) to promote health deterioration and better explain disparities using structural equation modeling guided by appropriate conceptual frameworks [137,138]. To that end, investigators could consider including the NIMHD framework and depending on the nature of the research questions (e.g., hypothesis-generating versus testing), integrate other frameworks such as those described by Bronfenbrenner, Williams, and Guilamo-Ramos, which are more explicitly directional, to examine relationships in more detail [34,137,139]. Future studies should include potential moderating factors such as social support, community resources, known protective factors of toxic stress, and relationships with healthcare providers. These factors may prove to buffer the detrimental effects of several SDoHs and guide future novel interventions aimed at mitigating their detrimental effects, whilst policy efforts to reduce exposure continue [14,140–142].

Most studies in this review examined peripheral circulating inflammatory markers, and notably, many of these are not strictly pro- or anti-inflammatory but context-dependent. Cytokines, for example, are subject to diurnal variability, changing significantly in concentration over 24 h [143], a detail unaddressed in the reviewed studies. Expression or methylation of inflammatory genes is a more stable measure for future research to understand the inflammatory effects of chronic (rather than acute) stressors, especially for large epidemiological studies as these methods are becoming increasingly scalable and affordable.

For future research it is imperative to delve into the underlying mechanisms through which exposure to socioeconomic disadvantage, trauma, abuse, and, violence during childhood and adulthood shapes inflammation, considering the influence of diverse sociocultural and built environment factors. Tandem inclusion and repeated measures of inflammatory markers and HPA biomarkers (e.g., corticotrophin-releasing hormone, adrenocorticotropic hormone (ACTH), and cortisol) are warranted to understand more complete exposure-specific paths upon which to monitor risk and identify early warning indicators and targets for intervention and prevention [144].

We found in our analysis that not all SDoH domains and levels of influence across the NIMHD research matrix were studied equally. This review produced no studies that fell under the health system domain or society level of influence, and few studies examined built/ environment factors, exposure to racism, discrimination, and acculturative stress, signaling areas where more investigation is needed. Future research should prioritize the participation of historically minoritized groups and reporting of such groups when describing sample characteristics.

4. Implications and conclusions

In this review, we note meaningful trends in inflammation and mental and metabolic health among women exposed to several disadvantageous SDoHs. Recommendations for clinical practice are generally beyond the bounds of scoping reviews. However, our findings underscore several social factors of which clinicians should be mindful, as they may have significant effects not only on patient outcomes but also on inflammation as an early warning indicator. Clinicians should be supported at the system level to hold spaces for patients to explore their specific social context, potential barriers, and facilitators of reaching their health goals. Continued efforts to bolster economic security and interpersonal safety (e.g., abuse and violence prevention) across social groups that are disproportionately impacted by depression and T2D remain a priority. Similarly, action to reduce and buffer the effects of these social factors on children is an urgent imperative. Together, these research, practice, and policy efforts, may reduce the burden of depression and T2D that remains highly prevalent and costly globally.

Funding

This scoping review was conducted during Nicole Perez's post-doctoral appointment funded by the National Institutes of Nursing Research (P20 NR018075).

CRediT authorship contribution statement

Nicole Perez: Writing – review & editing, Writing – original draft, Supervision, Project administration, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Ning He:** Writing – review & editing, Writing – original draft, Formal analysis. **Fay Wright:** Writing – review & editing, Writing – original draft, Methodology, Formal analysis, Data curation. **Eileen Condon:** Writing – review & editing, Writing – original draft. **Sheri Weiser:** Writing – review & editing, Conceptualization. **Brad Aouizerat:** Writing – review & editing, Conceptualization.

Declaration of competing interest

All authors have completed the Unified Competing Interest form at https://www.icmje.org/coi_disclosure.pdf and declare that authors have no competing interests to report.

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