Bridging the Divide: A Structured Narrative Review of Health Disparities through Immunology and Medical Sociology, with Implications for Nigeria and Sub-Saharan Africa

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ABSTRACT

Background: *Health disparities reflect social conditions, yet immunological research often treats social exposures as background variables, and medical-sociological theories are rarely linked to immune mechanisms.* ***Aim:*** *To synthesise evidence on how socially patterned exposures may influence immune-related outcomes, and to identify implications and evidence gaps for Nigeria and sub-Saharan Africa.* ***Methods:*** *A structured narrative review, guided by SANRA, of PubMed, Europe PMC and African Index Medicus from 1990 to 14 October 2026; of 983 records screened, 137 met eligibility criteria, with thematic synthesis and evidence-type labelling.* ***Results:*** *Synthesis was organised around four pre-specified pathways: chronic psychosocial stress, environmental contaminant exposure, social isolation, and socially patterned health behaviours, with intersectionality as an overarching framework. These pathways converge on neuroendocrine, inflammatory, oxidative and epigenetic processes. The evidence is largely observational and from high-income settings; direct Nigerian and sub-Saharan African evidence is concentrated in environmental and occupational exposures, with mixed findings.* ***Implications:*** *Social risk should be measured directly, because C-reactive protein and interleukin-6 are not screening tests for social adversity. Longitudinal, locally grounded and intersectional studies are needed.* ***Conclusion:*** *Linking immune pathways to sociological accounts of exposure strengthens explanations of health disparities, but the evidence supports biological plausibility rather than established causation.*



Keywords: *Health disparities; immunology; medical sociology; social determinants of health; psychoneuroimmunology; intersectionality; Nigeria; sub-Saharan Africa; biological embedding*

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INTRODUCTION

Health disparities refer to systematic differences in health outcomes between socially defined population groups. The related term health inequity is used when these differences are considered avoidable, unfair, and produced by unequal social arrangements. Such differences are neither random nor adequately explained by individual biology alone. They reflect unequal exposure to hazards, unequal access to protective resources, and unequal capacity to prevent or recover from disease (Link & Phelan, 1995; Marmot, 2005). Racial and socioeconomic gradients have been documented across multiple causes of morbidity and mortality, although the evidence base, magnitude, and mechanisms vary by population, disease, and setting (Williams & Collins, 2001; Williams et al., 2019). The problem remains current. In a multicohort analysis of nearly 780,000 participants in the United Kingdom and Finland, low education and low adult socioeconomic position were associated with higher risk of age-related disease (Kivimäki et al., 2025). The hazard ratios for any such disease ranged from 1.21 to 1.31. Protein analyses in British and American cohorts in the same study linked social disadvantage to plasma proteins involved in inflammatory and cellular stress responses, which mediated up to 39% of the associations with specific diseases. We identified no comparable data from sub-Saharan Africa. In this review, social determinants of health denote the social, economic and environmental conditions under which people live and work across the life course. Social position denotes a person's location in hierarchies of resources and power, indexed by socioeconomic position, ethnicity, gender, and related axes. Social adversity denotes the exposures, such as deprivation and discrimination, that disadvantaged positions make more likely. These terms are used consistently throughout and are not treated as interchangeable; socioeconomic status is retained where a cited study uses that term.

Immunological and psychoneuroimmunological research has identified plausible molecular and cellular pathways linking psychosocial and environmental exposures with variation in disease susceptibility and progression. These pathways include neuroendocrine signalling, altered leukocyte trafficking and function, inflammatory regulation oxidative stress, and epigenetic modification (Alotiby, 2024; Dhabhar, 2014; Segerstrom & Miller, 2004; Suzuki et al., 2020). Medical sociology, in turn, offers frameworks for examining how social position structures exposure to deprivation, discrimination, occupational hazards, social exclusion, and constrained health-related behaviours (Cockerham, 2005; Link & Phelan, 1995).

The two disciplines are not wholly separate, since psychoneuroimmunology, social epidemiology, ecosocial theory, biological embedding, and environmental justice scholarship already cross disciplinary boundaries. Nevertheless, immune mechanisms and sociological explanations are often presented in different literatures, which can obscure how structural conditions may be reflected in biological processes. The central problem addressed by this review is therefore that the social conditions which generate health disparities and the immune mechanisms through which those conditions may register biologically are rarely examined within a single analytic framework. They are examined even more rarely with reference to Nigerian or sub-Saharan African evidence. This review therefore brings together four interacting domains: chronic psychosocial stress; environmental contaminant exposure; social isolation, social networks, and social support; and socially patterned health behaviours. These four were selected because each possesses both an established sociological literature describing its unequal social distribution and a separate immunological literature describing plausible biological pathways, so each provides a point at which the two disciplines can be joined. Taken together they address the principal routes by which social position may be translated into immune-relevant exposure, namely neuroendocrine activation, toxicant burden, relational resources, and constrained behaviour. Intersectionality is treated as an overarching framework for understanding how these pathways are configured by multiple dimensions of social position. Particular attention is given throughout to Nigeria and other sub-Saharan African settings, where evidence linking socially patterned exposure to measured immune endpoints remains sparse.

Recent reviews have begun to connect social determinants with inflammatory pathways. Vijendra et al. (2025) mapped the five Healthy People 2030 domains onto cytokine dysregulation, Toll-like receptor activation, hypothalamic-pituitary-adrenal axis change, and gut microbiome disruption. That review drew on a social-ecological model and the weathering hypothesis, with evidence largely from the United States and other high-income settings. The present review differs in three respects. First, it grounds each pathway in an explicit medical-sociological account of how exposure is distributed, namely fundamental cause theory, health lifestyle theory, environmental justice scholarship, and the weathering hypothesis. Second, it treats intersectionality as an overarching analytic framework rather than as an additional domain. Third, it inventories the available Nigerian and sub-Saharan African evidence and separates it from extrapolation (Table 2).

The review addressed four questions. (RQ1) Through which socially patterned pathways may social position shape immune-relevant exposure? (RQ2) What biological mechanisms and immune endpoints link those exposures to immune-related outcomes, and what types of evidence support them? (RQ3) What direct evidence exists from Nigeria and other sub-Saharan African settings? (RQ4) What research, clinical, and policy implications follow, and what evidence gaps remain?

Review Approach

A structured narrative search was undertaken in PubMed Europe PMC and African Index Medicus. Scopus and Web of Science, used in earlier unrecorded searching, were not accessible for the documented search, and the African Journals Online article search was unavailable on the search date because of a server error. Europe PMC was added to extend coverage of preprints and additional life-science journals, and African Index Medicus to improve retrieval of African evidence. Search concepts included health disparities, health inequities, social determinants of health, immune function, inflammation, psychoneuroimmunology, chronic stress, environmental immunotoxicology, environmental justice, social isolation, loneliness, social support, health behaviour, biological embedding, and intersectionality. Database-specific Boolean strings combining controlled vocabulary with free-text synonyms are reported in Supplementary File S1. The review prioritised peer-reviewed empirical studies, systematic and scoping reviews, meta-analyses, and influential theoretical publications published from 1990 to 14 October 2026. The 1990 lower limit was chosen because the psychoneuroimmunology, fundamental cause, and environmental justice literatures that frame this review were consolidated during that decade. Earlier landmark sources were retained when they established concepts central to the synthesis. The search reported here was documented in full. Each database was searched on 14 October 2026, and the exact strings, filters, dates, and record counts were archived (Supplementary File S1). The review remains a structured narrative review and does not claim the exhaustiveness of a systematic review.

Sources were considered when they directly addressed at least one of four areas. These were socially patterned exposure; measurable immune, inflammatory, neuroendocrine, or immune-sensitive clinical outcomes; sociological mechanisms relevant to exposure or resource distribution; or methodological approaches for integrating social and biological data. Eligibility was operationalised as follows. For empirical studies, inclusion required both a socially patterned exposure and a measurable immune, inflammatory, neuroendocrine or immune-sensitive clinical outcome; records lacking either were excluded (codes E1 and E2 in Supplementary File S1). Environmental and occupational exposures were treated as socially patterned where they arise from residence, occupation or household fuel use, which are distributed by social position, although some included environmental studies did not measure social patterning directly. Inclusion also required (i) human studies of any design, theoretical or conceptual publications, systematic, scoping, or narrative reviews, meta-analyses, or animal and in vitro studies cited for mechanism only; (ii) English-language publication; and (iii) publication from 1990 onward, except landmark theoretical sources. No geographical restriction was applied, but a supplementary search with a Nigerian and sub-Saharan African filter was run to maximise retrieval of regional evidence (Supplementary File S1). Conference abstracts without full text and commentaries without original evidence or argument were excluded. The authors used an AI assistant (Claude, Anthropic) to carry out title-and-abstract screening and full-text assessment on 15 October 2026. It applied these criteria to every record and recorded each decision with a reason code in a screening workbook, and the authors take responsibility for the decisions. Full texts were sought from openly available sources (Europe PMC, PubMed Central, open-access links indexed by Unpaywall, OpenAlex and Semantic Scholar, and publisher and repository pages); where only an accepted manuscript or preprint was available, that version was assessed and this is noted in Supplementary File S5. Records whose full text could not be obtained were excluded and listed. Screening was not duplicated by an independent human reviewer, and no sample of the AI decisions was re-screened by the authors. The search yield and reasons for exclusion are summarised in Supplementary File S1, and the included records and those not retrieved are listed in Supplementary File S5. Evidence was organised into the four thematic domains specified in the Introduction, and the resulting synthesis is summarised in (Table 1). Within each domain, the narrative synthesis reported the study designs and immune endpoints predominating in the cited literature and identified the principal limitations of that evidence in the text. Data from the empirical studies identified outside the documented search were extracted into a structured matrix recording author and year, country, design, sample, social exposure, immune endpoint, principal finding, adjustment for confounding, and principal limitation (Supplementary File S2). For the sub-Saharan African studies from the documented search, setting, design, population, exposure, endpoint, principal finding and key limitation are recorded in (Table 2), and the principal finding of every included record is recorded in Supplementary File S5. Because the purpose was interpretive integration rather than exhaustive effect estimation, no meta-analysis, formal risk-of-bias assessment, or certainty grading was undertaken. Instead, sources supporting substantive claims were labelled by evidence type: theoretical (T), animal or in vitro mechanistic (M), human experimental or randomised (E), observational (O), and review or meta-analysis (R). These labels, used in (Tables 1 and 2), indicate the kind of evidence on which a claim rests, not its quality. Formal risk-of-bias tools were not applied because they are design-specific and the included evidence spans theoretical, mechanistic, experimental, observational, and review sources that no single tool can appraise on a common scale. SANRA itself assesses the quality of the review article rather than that of the studies it includes

Table 1. Summary of social-to-immune pathways across the four domains reviewed.

Domain	Sociological mechanism	Biological pathway	Representative immune endpoint	Evidence type	Principal limitation	Nigerian / SSA evidence identified
Chronic psychosocial stress	Insecurity, discrimination and deprivation distributed by social position; flexible resources govern avoidance (fundamental cause theory; weathering)	HPA and sympathetic activation; glucocorticoid receptor resistance; allostatic load	Inflammatory regulation; cellular immunity; vaccine response; latent-virus control; tissue repair	R, O (including prospective studies with experimental viral challenge); T	Largely cross-sectional; endpoint-specific; allostatic load is not an immune measure	Winful et al. (2024), one cross-sectional Nigerian study; Roba et al. (2025), Aljohani et al. (2026), Christensen et al. (2024), Kohler et al. (2013) and Kelly et al. (2021) elsewhere in SSA
Environmental contaminant exposure	Siting, segregation, housing markets and unequal political capacity (environmental justice)	Oxidative stress; AhR signalling; endocrine disruption; epigenetic regulation	Innate and adaptive signalling; inflammation; host defence; allergic and respiratory outcomes	R, M, O; trials in Nigeria and Ghana (E)	Contaminant-specific; mixtures rarely studied; justice evidence mainly from the United States	Oluwole et al. (2013); Yaqub et al. (2014); Tirima et al. (2016); Olopade et al. (2017); 21 further SSA studies and one review (Table 2), with mixed findings
Social isolation, networks and support	Unequal access to participation, networks, support and trust	Neuroendocrine regulation; inflammatory signalling; behavioural mediation	Systemic inflammatory markers; antiviral and vaccine responses	R (incl. meta-analysis), O	Constructs not interchangeable; reverse causation; few longitudinal designs	None included
Socially patterned health behaviours	Class-linked life chances constrain health lifestyles (health lifestyle theory); access to resources governs risk avoidance (fundamental cause theory)	Micronutrient status; activity-related inflammation; sleep-immune regulation; tobacco effects	Inflammatory and immune profiles; host defence	R, O, E (WASH trial), T	Direction-specific effects; supplementation benefit clearest where baseline status is low	Umuhoza & Ataguba (2018); Myburgh et al. (2020); DeCaro et al. (2016); Gough et al. (2020); Liu et al. (2022)

Note. Intersectionality is treated as an overarching analytic framework operating across all four domains rather than as a fifth pathway. Entries recording no Nigerian or sub-Saharan African evidence refer to studies identified in this review and do not establish that none exists. Evidence type: T, theoretical; M, animal or in vitro mechanistic; E, human experimental or randomised; O, observational; R, review or meta-analysis. AhR, aryl hydrocarbon receptor; HPA, hypothalamic-pituitary-adrenal; RCT, randomised controlled trial; SSA, sub-Saharan Africa.

(Baethge et al., 2019). Synthesis proceeded in four steps. Included sources were assigned to one or more of the four domains, or to cross-cutting socioeconomic, racial or methodological themes, and findings within each domain were grouped by immune endpoint and evidence type. Agreement, disagreement, and gaps were then identified, and the domain findings were interpreted through the sociological theory relevant to that domain. ThisThe narrative design limits claims of comprehensiveness and precludes formal ranking of the evidence across domains. The review was developed with reference to SANRA, particularly its criteria concerning justification, aims, literature searching, referencing, scientific reasoning, and presentation of relevant and appropriate endpoint data (Baethge et al., 2019). A SANRA self-assessment is provided in Supplementary File S3.

Evidence Synthesis

The documented search identified 983 records after removal of duplicates. Of these, 293 were sought in full text; 115 could not be obtained, 178 were assessed, and 137 met the eligibility criteria (Supplementary Files S1 and S5). All 137 are summarised, with their principal findings, in Supplementary File S5. The narrative synthesis below cites all 32 sub-Saharan African records and the syntheses and primary studies most directly relevant to each domain; the remaining records are reported in

Table 2. Nigerian and sub-Saharan African evidence identified in this review, including studies retrieved by the documented search.

Study	Setting	Design (type)	Population and comparison	Exposure	Immune or biological endpoint	Principal finding	Key limitation
Oluwole et al. (2013)	Rural south-west Nigeria	Cross-sectional (O)	59 mother-child pairs cooking almost exclusively with biomass fuel; no unexposed group	Household biomass smoke; indoor fine particulates	C-reactive protein; lipid peroxidation; lung function	Particulates >60 times WHO guideline; CRP correlated with lipid peroxidation in children and inversely with lung function in mothers	No comparison group
Yaqub et al. (2014)	Nigeria (site not stated in abstract)	Comparative cross-sectional (O)	30 pesticide applicators and 30 other farmers vs 30 unexposed controls	Occupational organophosphate pesticides; serum acetylcholinesterase	Leukocyte subsets; immunoglobulins; skin prick tests	Lower white cell and neutrophil counts; higher lymphocytes and eosinophils; IgM lower in applicators vs other farmers	Small sample; cross-sectional
Tirima et al. (2016)	Zamfara State, Nigeria	Remediation programme report (O)	Children aged ≤5 years in affected villages	Lead from artisanal gold-ore processing	Blood lead (no immune endpoint)	Geometric mean blood lead 149 µg/dL before chelation; 15 µg/dL after the 4-year programme	Exposure and clinical data only
Olopade et al. (2017)	Ibadan, Nigeria	Randomised controlled trial (E)	324 pregnant women randomised (271 analysed); bioethanol stove vs continued firewood or kerosene	Household air pollution	TNF-α, IL-6, IL-8, malondialdehyde, retinol-binding protein (first vs third trimester)	No between-arm difference overall; TNF-α reduced only in subgroup of prior firewood users (n = 99)	Single city; positive finding confined to a subgroup
Umuhoza & Ataguba (2018)	Six SADC countries	Cross-sectional survey (O)	World Health Survey respondents	Socioeconomic position	None (behavioural and environmental risk factors; self-assessed health)	Smoking, poor diet, biomass fuel and unimproved water and sanitation concentrated among poorer households	No biological measures
Winful et al. (2024)	Lagos, Nigeria	Cross-sectional (O)	138 adults at a hospital dental clinic	Psychological stress (GHQ-12); cortisol	Salivary CRP	Stress score did not predict salivary CRP; BMI, age, sex, ethnic group and cortisol did	Small sample; single measurement
Myburgh et al. (2020)	South Africa	Cross-sectional (O)	1,569 Black South African adults	Education and other SES factors; smoking; CRP gene variants	CRP	≥12 years of education associated with ~19% lower CRP (adjusted); smoking interacted with a CRP variant	Cross-sectional; SES factors not associated individually
DeCaro et al. (2016)	Mwanza, Tanzania	Cross-sectional (O)	88 mother-infant pairs	Household food insecurity; wealth; subjective status; maternal mental health	Infant high-sensitivity CRP	Severe food insecurity and high maternal depressive symptoms each associated with above-median infant CRP; mediation not supported	Cross-sectional; single site
Kohler et al. (2013)	Rural Malawi	Population-based cross-sectional (O)	About 1,500 adults sampled (MLSFH)	Schooling; wealth; marital status; religion	Wide-range CRP; lipids; renal markers	Schooling and wealth not associated with CRP; being married weakly associated with lower CRP	Cross-sectional; weak associations overall

Kelly et al. (2021)	Blantyre, Malawi	Cross-sectional analysis of cohort baseline (O)	279 adults with advanced HIV starting ART	Education; income; household assets; water source	T-cell and monocyte activation; endothelial markers	Higher education and income with more activated CD8 T cells; lower education with more intermediate monocytes; water-kiosk use associated with lower activation	HIV-specific population; direction differs by pathway
Roba et al. (2025)	Northern Kenya	Cross-sectional during drought (O)	128 Daasanach household heads	Chronic stress (fingernail cortisol); household water insecurity	Dried-blood-spot CRP	Higher fingernail cortisol and severe water insecurity each associated with higher CRP	Cross-sectional; one community; all severely food insecure
Aljohani et al. (2026)	Uganda	96-week prospective cohort (O)	49 youth with perinatal HIV and 51 without, aged 10 to 18	Adverse childhood experiences; socioeconomic factors	Monocyte and T-cell activation; arterial stiffness	Higher-ACE cluster had greater monocyte and T-cell activation; neglect associated with arterial-stiffness progression in youth with HIV	Small sample; ACE clusters overlap with HIV status
Christensen et al. (2024)	Drakenstein, South Africa	Birth cohort (O)	225 mother-infant pairs	Prenatal indoor air pollutants; SES; maternal depressive symptoms	Infant IL-1β, IL-6 and TNF-α at 6 weeks	Indoor pollutants (notably benzene) associated with higher markers; high-pollution, low-SES, high-depression profile associated with higher inflammation	Single cohort; one infant time point
Rylance et al. (2015)	Blantyre, Malawi	Bronchoscopy study with in vitro challenge (O; M)	Healthy non-smoking adults exposed to cooking smoke	Household biomass smoke (macrophage particulate loading)	Alveolar macrophage oxidative burst, phagocytosis and CXCL8	Higher particulate loading associated with reduced oxidative burst and dampened CXCL8 responses	Small sample; exposure proxied by macrophage carbon
Kaali et al. (2021)	Kintampo, Ghana	Cohort within cookstove trial (E; O)	Mother-infant pairs (CO measured in 97; PM2.5 in 60)	Prenatal household air pollution; LPG or improved stove vs open fire	Cord-blood mononuclear cell telomere length	Higher PM2.5 associated with shorter telomeres; LPG arm had longer telomeres	Small exposure subsample; telomere length nonspecific
Abdo et al. (2021)	Northern Ghana	Randomised cookstove trial (E)	200 households; adult cooks and children	Improved biomass cookstoves	Serum amyloid A and CRP (dried blood spots)	Some evidence of lower serum amyloid A and CRP among cooks in intervention arms; no other detectable effects	Continued use of traditional stoves
Misra et al. (2018)	Rural South Africa	Cross-sectional within cohort (O)	415 women	Household cooking fuel (wood vs electricity)	Plasma interleukins, TNF-α and CRP	Little evidence of association between fuel use and inflammatory markers	Fuel type as exposure proxy
Olsson et al. (2024)	Butajira and Addis Ababa, Ethiopia	Cross-sectional (O)	60 mother-child pairs	Solid-fuel use; pneumococcal density	Nasopharyngeal CXCL8 and other mediators	Solid-fuel use not associated with nasopharyngeal inflammation; higher CXCL8 in rural mothers	Small sample
Lin et al. (2021)	Rural Senegal	Cross-sectional field study (O)	441 women exposed to household air pollution	Household air pollution; secondhand smoke	Dried-blood-spot malondialdehyde and CRP	Malondialdehyde higher with secondhand smoke and associated with cardiopulmonary symptoms	No unexposed group; self-reported symptoms

Di Lenardo et al. (2020)	Limpopo, South Africa	Birth cohort (O)	425 fully vaccinated children	Blood lead at age 1 (median 1.9 µg/dL)	Vaccine-specific IgG at 3.5 years	Higher lead associated with greater risk of tetanus IgG below protective level (RR 1.88 per 10-fold increase); not measles	Low exposure range
Prahl et al. (2021)	Tororo, Uganda	Cohort nested in an antimalarial trial (O)	Pregnant women and their infants	In utero bendiocarb after indoor residual spraying	Fetal immune-cell phenotype and function; infant vaccine titres	Fewer regulatory CD4 T cells, more cytokine production, reduced antigen-driven proliferation; higher measles titres at one year	Single pesticide; observational exposure
Kataba et al. (2023)	Kabwe, Zambia	Cross-sectional (O)	Adults grouped by blood lead (<10 vs >10 µg/dL) and sex	Environmental lead from mining	Plasma TNF-α and IL-8	No overall association; higher lead associated with lower TNF-α in women	Small groups; two cytokines
Hernandez-Vargas et al. (2015)	West Kiang, The Gambia	Cohort (O)	115 mother-infant pairs	Maternal dietary aflatoxin	Infant white-blood-cell DNA methylation	Aflatoxin associated with methylation at 71 CpG sites	Functional immune effects not measured
Ghantous et al. (2021)	West Kiang, The Gambia	Cohort (O)	244 children	Early-life dietary aflatoxin	White-blood-cell DNA methylation at 24 months	Aflatoxin associated with differentially methylated positions and regions	Functional immune effects not measured
Ennin et al. (2022)	Kumasi, Ghana	Cross-sectional (O)	86 hardwood workers vs 89 non-woodworkers	Occupational wood dust	White cell count; serum cytokines	Higher Th1 and Th2 cytokines in woodworkers	Exposure not measured individually
Tiekwe et al. (2021)	Douala and Dschang, Cameroon	Cross-sectional (O)	126 motorbike drivers in Douala vs 65 in Dschang	Urban air pollution (occupational)	Leukocyte differential; platelets; CRP	Monocytes, lymphocytes and CRP higher among Douala drivers	Exposure inferred from city
Tiekwe et al. (2024)	Douala and Dschang, Cameroon	Cross-sectional (O)	191 motorbike drivers	Fine and ultrafine particles (occupational)	Superoxide dismutase; malondialdehyde; leukocyte subsets	Oxidative markers higher in Douala and correlated with monocyte mobilisation	Exposure inferred from city
Mamuya et al. (2018)	Kilimanjaro, Tanzania	Cross-sectional (O)	70 exposed and 65 unexposed workers	Occupational volcanic-rock dust	Fractional exhaled nitric oxide	Stone cutters had higher FeNO than unexposed workers	Cross-sectional
Tungu et al. (2013)	Tanzania	Cross-sectional (O)	127 cement workers vs 28 controls	Occupational cement dust	Fractional exhaled nitric oxide	FeNO did not differ between exposed workers and controls	Possible healthy-worker effect
Nigatu et al. (2015)	Ethiopia	Cross-sectional (O)	248 female flower-farm workers (FeNO in 114)	Occupational endotoxin	Fractional exhaled nitric oxide; symptoms	Endotoxin exposure low; few with raised FeNO; more nasal symptoms in greenhouse workers	Short employment duration
Buthelezi et al. (2024)	Highveld, South Africa	Cross-sectional with lagged exposure (O)	706 schoolchildren aged 9 to 14	Ambient PM2.5	Fractional exhaled nitric oxide; lung function	Lagged PM2.5 associated with raised FeNO and lower lung function	Station-level exposure
Makamure et al. (2016)	Durban, South Africa	Panel study (O)	129 children aged 9 to 11	Ambient SO2, NO2, NO and PM10; TNF-α −308 genotype	Plasma TNF-α; lung function	Genotype not associated with TNF-α; modified pollutant-related lung-function decrements	Small sample
Liu et al. (2022)	Mangochi, Malawi	Secondary analysis of trial cohort (O)	620 children aged 18 months	Household environmental exposures	Stool markers of intestinal inflammation, repair and permeability	No associations with the exposures measured	Secondary analysis
Morakinyo & Mokgobu (2022)	South Africa	Review (R)	16 studies; 16,920 participants	Indoor household exposures	Asthma; airway inflammation; early-life immune function	Indoor exposures associated with asthma, airway inflammation and poor early-life immune function	Heterogeneous primary studies
Olude et al. (2022)	Africa	Narrative review (R)	Not applicable	Poverty, pollution, violence and infection	Glial neuroinflammation	Proposes these stressors induce neuroinflammation; calls for African data	Largely extrapolated from other settings

Note. The first eight rows are studies identified outside the documented search (earlier unrecorded searching or targeted supplementary searches; Supplementary File S4); the remainder were retrieved by the documented search. Seven Nigerian studies retrieved by the search could not be assessed in full text and are listed in Supplementary File S5. ACE, adverse childhood experience; ART, antiretroviral therapy; BMI, body mass index; CO, carbon monoxide; CRP, C-reactive protein; FeNO, fractional exhaled nitric oxide; GHQ-12, 12-item General Health Questionnaire; IgG, immunoglobulin G; IgM, immunoglobulin M; IL, interleukin; IMT, intima-media thickness; LPG, liquefied petroleum gas; MLSFH, Malawi Longitudinal Study of Families and Health; NO, nitric oxide; NO2, nitrogen dioxide; PM10, particulate matter 10 µm or smaller; PM2.5, fine particulate matter; RR, risk ratio; SADC, Southern African Development Community; SES, socioeconomic status; SO2, sulphur dioxide; TNF-α, tumour necrosis factor-alpha; WHO, World Health Organization. Evidence-type codes as in Table 1.

Supplementary File S5 only. These were discussed together with 44 sources identified outside the documented search: from earlier unrecorded searching and citation tracking, from targeted supplementary searches, and from the first author's published work (Supplementary File S4). Sources identified before the documented search, through searching whose histories were not retained and through citation tracking, were retained where they met the eligibility criteria; how each cited source was identified is recorded in Supplementary File S4. Findings are presented below by domain (RQ1 and RQ2), with the Nigerian and sub-Saharan African evidence identified for each domain (RQ3); implications (RQ4) follow the Discussion.

Chronic Psychosocial Stress and Immune Regulation

Stress responses are context dependent. Acute, time-

limited stress can transiently redistribute leukocytes and enhance selected defensive functions, whereas prolonged or recurrent psychosocial stress is more consistently associated with immune dysregulation (Alotiby, 2024; Dhabhar, 2014). Two major signalling systems are involved: the hypothalamic-pituitary-adrenal (HPA) axis, which regulates glucocorticoid release, and sympathetic pathways, which release catecholamines that act on adrenergic receptors expressed by multiple immune-cell populations (Alotiby, 2024; Dhabhar, 2014). The effects depend on the duration, intensity, predictability, and controllability of the stressor, as well as age, health status, and the immune endpoint measured (Dhabhar, 2014; Segerstrom & Miller, 2004).

Chronic stress has been associated with changes in cellular immunity, inflammatory regulation, antibody responses to some vaccines, control of latent herpesviruses, and tissue repair (Cohen et al., 2012; Kiecolt-Glaser et al., 2002; Segerstrom & Miller, 2004). Poorer cellular control of latent herpesviruses has been inferred from raised antibody (Kiecolt-Glaser et al., 2002); such titres are an indirect marker and are not equivalent to direct measurement of viral reactivation. The direction and magnitude of these associations vary by stressor and immune endpoint, and together they describe a pattern of dysregulation rather than a single state of immune suppression. Elevated inflammation can coexist with impaired antiviral, cellular, or vaccine responses, so higher concentrations of inflammatory markers do not indicate stronger immune competence.

One proposed mechanism is reduced cellular sensitivity to glucocorticoid signalling. Cohen et al. (2012) reported that chronic stress was associated with glucocorticoid receptor resistance and poorer regulation of inflammatory responses in experimental viral-challenge studies. The mechanism is heterogeneous and is not explained by cortisol exposure alone. Receptor expression, intracellular signalling, immune-cell composition, infection, metabolic status, medication use, and behavioural factors may all influence observed associations.

Medical sociology provides frameworks for explaining the unequal distribution of chronic adversity. Material deprivation, insecure employment, overcrowded housing, neighbourhood disorder, and interpersonal or institutional discrimination are patterned by socioeconomic and racialised social structures (Williams et al., 2019). Sociologically, chronic stress usefully understood as a property of social position than as an individual disposition. Fundamental cause theory holds that socioeconomic position governs access to money, knowledge, power, prestige, and beneficial social connections, and that these flexible resources help people avoid risks and limit the consequences of disease once it occurs (Link & Phelan, 1995). Applied to chronic stress, the theory implies that the same resources shape who can avoid a stressor altogether and who must absorb it repeatedly. Stress exposure is on this account socially produced, through the distribution of insecurity, subordination, and demands that exceed the capacity to control them. Health lifestyle theory makes a parallel argument about responses to adversity, holding that the options realistically available to a person are shaped by class-linked life chances and not by individual choice alone (Cockerham, 2005). Racial residential segregation and the socioeconomic gradient both describe arrangements that are durable rather than episodic, so the adversity they generate recurs and is difficult to avoid (Marmot, 2005; Williams & Collins, 2001). The inference drawn in this review, rather than a finding of those studies, is that this persistence, more than the intensity of any single event, is what would connect social position to sustained neuroendocrine and inflammatory activation. Allostatic load provides a composite measure of the cumulative multisystem dysregulation associated with such prolonged adaptation, although it indexes multisystem physiological burden rather than immune senescence specifically (Beese et al., 2022).

The weathering hypothesis proposes accelerated physiological deterioration among populations exposed to sustained social and economic adversity (Forde et al., 2019). Empirical work has documented earlier and higher multisystem allostatic burden among Black Americans, with important variation by age and sex (Geronimus et al., 2006). Studies of leukocyte telomere length provide additional, although nonspecific, evidence that racial discrimination may be associated with accelerated biological ageing (Chae et al., 2020). The social factors implicated in accelerated biological ageing in this literature are cumulative socioeconomic disadvantage, racial discrimination, and residential segregation with its associated neighbourhood deprivation (Forde et al., 2019; Geronimus et al., 2006; Williams et al., 2019). Telomere length indexes these influences only indirectly, since it also reflects inherited factors, leukocyte composition, disease, behaviour, and assay method. More recent multicohort evidence extends this pattern to the plasma proteome: socioeconomic disadvantage was associated with proteins that regulate inflammatory and cellular stress responses. Participants whose circumstances improved over the life course had more favourable protein profiles than those whose circumstances did not (Kivimäki et al., 2025). These findings are observational and come from high-income cohorts, so they support biological embedding as a plausible pathway without establishing it as causal or transferable.

Evidence from the documented search

Syntheses retrieved by the documented search support a modest but consistent association between lower socioeconomic position and systemic inflammation. Lower socioeconomic status was associated with higher C-reactive protein and interleukin-6, whether measured by income or by education (Muscatell et al., 2020). Childhood disadvantage was associated with about 25% higher adult

C-reactive protein, an association attenuated by adult body mass index (Liu et al., 2017). Lower parental socioeconomic position was associated with higher adult C-reactive protein and allostatic load, although the pooled interleukin-6 estimate was imprecise (Rivillas-García et al., 2026). Evidence for neuroendocrine and cellular-ageing markers is weaker. Socioeconomic status was not consistently related to cortisol (Dowd et al., 2009), and its association with telomere length was weak and not robust (Robertson et al., 2013). An early systematic review of 11 allostatic load studies found higher allostatic load at lower socioeconomic status, but interleukin-6 and C-reactive protein were each included in only two of the indices (Szanton et al., 2005). Allostatic load indices have been constructed so inconsistently that studies are difficult to compare (Johnson et al., 2017). Everyday discrimination was associated with C-reactive protein but not with cortisol, interleukin-6 or telomere length (Lawrence et al., 2022). A systematic review of 32 studies linked discrimination with telomere shortening and with pro-inflammatory and antiviral gene-expression patterns, mostly in cross-sectional designs (Cuevas et al., 2026). An earlier review of 12 studies, all from the United States, found only weak evidence of a main association between discrimination and telomere length (Coimbra et al., 2020). Taken together, the telomere evidence is inconsistent and mostly cross-sectional, and adds little beyond the inflammatory-marker evidence. Among children and young people, a meta-analysis found minimal to moderate positive associations of experienced racism with C-reactive protein, interleukin-6 and salivary cortisol (Priest et al., 2024). Early adversity involving threat was more strongly associated with accelerated cellular ageing than deprivation or low socioeconomic status (Colich et al., 2020), whereas evidence linking early adversity to circulating inflammation in children remains limited and heterogeneous (Kuhlman et al., 2020). The translational implication is not that social adversity can be diagnosed through a cytokine panel. The evidence assembled here is largely cross-sectional, so temporal sequence, mediation, and reverse causation remain difficult to separate, and the biological pathways described operate downstream of the social conditions that generate them.

Transferability

Only one Nigerian study linking measured psychosocial stress with an immune endpoint was identified in this review. Among 138 adults attending a dental clinic in Lagos, stress measured with the 12-item General Health Questionnaire did not predict salivary C-reactive protein, whereas body mass index, age, sex, ethnic group, and cortisol did (Winful et al., 2024). The study is small and cross-sectional, but it shows that self-reported stress and inflammatory markers need not align. Elsewhere in sub-Saharan Africa, three studies identified by the documented search linked social or psychosocial adversity with immune measures. During the 2022 drought in northern Kenya, fingernail cortisol and severe household water insecurity were each associated with higher C-reactive protein among 128 Daasanach pastoralists (Roba et al., 2025). Among Ugandan adolescents, a cluster with greater exposure to adverse childhood experiences, which included more youth with perinatally acquired HIV, showed greater monocyte and T-cell activation (Aljohani et al., 2026). In a South African birth cohort, a prenatal profile combining high indoor air pollution, low socioeconomic status and maternal depressive symptoms was associated with higher infant inflammatory markers (Christensen et al., 2024). Socioeconomic position itself showed weak or inconsistent associations. In rural Malawi, schooling and wealth were not associated with C-reactive protein (Kohler et al., 2013). Among Malawian adults starting antiretroviral therapy, higher education and income were associated with more activated CD8 T cells, whereas lower education was associated with more intermediate monocytes (Kelly et al., 2021). The direction of socioeconomic gradients in immune markers may therefore differ where infection, HIV and undernutrition shape both exposure and immune activation. The sociological mechanisms are plausible but largely untested in these settings, and the locally salient stressors, such as economic insecurity, insecurity of tenure, and displacement, and their distribution across ethnic, religious, and regional lines remain to be characterised empirically.

Environmental Exposure, Social Stratification, and Immunological Vulnerability

Pollution is a major public-health burden (Landrigan et al., 2018), but immunological effects require contaminant-specific evidence. Selected environmental contaminants have been associated with immune effects involving oxidative stress, aryl hydrocarbon receptor signalling, endocrine disruption, and epigenetic regulation. Evidence concerns particulate matter, polycyclic aromatic hydrocarbons, selected metals, and endocrine-disrupting chemicals (Suzuki et al., 2020), and, in occupational settings, organophosphate pesticides (Yaqub et al., 2014). Effects vary by contaminant and depend partly on host sensor systems, such as the aryl hydrocarbon receptor and KEAP1-NRF2 pathways, that regulate detoxification, antioxidant, and anti-inflammatory responses (Suzuki et al., 2020). Reported outcomes include altered innate and adaptive signalling, inflammatory activation, changes in host defence, and associations with allergic or respiratory (Akpoka et al., 2026; Landrigan et al., 2018; Suzuki et al., 2020). These outcomes are contaminant-specific and do not constitute a single uniform immunotoxic phenotype.
Populations are exposed to chemical mixtures rather than isolated agents, but epidemiological methods for evaluating combined exposures are still developing. A National Institute of Environmental Health Sciences workshop concluded that no single statistical approach

clearly outperformed the others (Taylor et al., 2016). Such designs remain uncommon, with the result that most available evidence characterises single agents while most real-world exposure is combined. This is a gap in the evidence rather than a demonstration of mixture toxicity, and attributing a specified immune outcome to a particular mixture requires direct exposure measurement, suitable comparison groups, temporality, dose-response assessment, and control of relevant co-exposures.

Environmental justice scholarship has documented that hazardous facilities and pollution burdens are not distributed randomly in several studied contexts. In the United States, industrial facilities, waste sites, and traffic-related pollution have frequently been concentrated in racialised and socioeconomically disadvantaged communities (Mohai et al., 2009; Morello-Frosch et al., 2002). These patterns may reflect siting decisions, residential segregation, housing markets, and unequal political capacity to resist hazardous land uses. This evidence is drawn overwhelmingly from the United States. The specific patterns it describes cannot be transposed directly to Nigeria. There, the salient axes of environmental inequality include oil-producing areas of the Niger Delta, artisanal mining districts, and peri-urban industrial and waste sites rather than racialised residential segregation. The Zamfara lead episode illustrates the difference, in that exposure followed the location of artisanal ore processing and the households that carried it out rather than a racial geography (Tirima et al., 2016). What transfers is the analytic principle: social and political arrangements influence who is exposed, at what dose, and with what capacity for protection or remediation.

Epigenetic change is one plausible route through which environmental exposure may produce persistent changes in gene regulation (Jirtle & Skinner, 2007). The mechanism matters for disparities research because it offers a candidate pathway by which exposures concentrated in disadvantaged settings could produce effects that outlast the exposure itself. Evidence for effects extending across generations, however, rests largely on animal models. In human populations, apparent intergenerational patterning is difficult to separate from direct fetal exposure, shared environments, social transmission of exposure-related conditions, or simple continuity of exposure across successive generations. Persistence within an exposed individual therefore rests on firmer ground than inherited transmission.

The exposures most likely to matter in Nigeria and comparable low- and middle-income settings differ from those that dominate the North American literature. They include mining-related metal exposure, agricultural pesticide use, ambient and household air pollution, contaminated water (Orebanjo et al., 2026), and inadequately regulated industrial discharge, such as untreated abattoir effluent (Solanke et al., 2026). Low- and middle-income countries bear a disproportionate share of the global pollution burden (Landrigan et al., 2018). The Nigerian studies identified in this review that link measured environmental exposures with immune or inflammatory endpoints were few and are summarised in (Table 2). Among 59 mother-child pairs in south-west Nigeria who cooked almost exclusively with biomass fuel, median indoor fine particulate concentrations were more than sixty times the World Health Organization guideline value. C-reactive protein was correlated with a marker of lipid peroxidation in children and was inversely associated with lung function in mothers. The study included no unexposed comparison group, so it establishes co-occurrence rather than effect (Oluwole et al., 2013). In 60 Nigerian farmworkers (30 pesticide applicators and 30 other farmers) occupationally exposed to organophosphate pesticides, total white cell and neutrophil counts were lower and lymphocyte and eosinophil counts higher than in 30 unexposed controls; s. Serum immunoglobulin M was reduced in pesticide applicators relative to other farmers rather than relative to controls (Yaqub et al., 2014). Artisanal gold-ore processing in Zamfara State produced an unprecedented epidemic of childhood lead poisoning. Among children aged five years or younger, geometric mean pre-chelation blood lead fell from 149 micrograms per decilitre before remediation to 15 micrograms per decilitre after the four-year programme (Tirima et al., 2016). Together these studies document substantial exposures and selected biological or clinical associations. They are small, mostly cross-sectional, and concentrated in a few states, and they do not characterise the immune burden at population level. They also differ in the kind of evidence they provide. The Zamfara reports document exposure and clinical poisoning without immune endpoints, and the farmworker study documents immune differences without establishing causation. The Ibadan stove trial was the only Nigerian study to test an intervention against inflammatory endpoints, and it found no overall difference between arms (Olopade et al., 2017). The documented search identified a larger body of evidence from other sub-Saharan African countries (Table 2), and its findings are mixed. Household air pollution was associated with impaired alveolar macrophage oxidative burst and dampened inflammatory responses in Malawian non-smokers (Rylance et al., 2015) and with shorter cord-blood telomeres in a Ghanaian cookstove trial (Kaali et al., 2021). By contrast, solid-fuel use was not associated with inflammatory biomarkers among rural South African women (Misra et al., 2018) or with nasopharyngeal inflammation in Ethiopian mothers and children (Olsson et al., 2024). A second Ghanaian cookstove trial found only some evidence of lower serum amyloid A and C-reactive protein among cooks in the intervention arms, and traditional stoves remained in use (Abdo et al., 2021). For metals and pesticides, low-level blood lead was associated with a higher risk of tetanus antibody titres below the protective level in South African children (Di Lenardo et al., 2020). In utero exposure to household-sprayed bendiocarb was associated with altered fetal

immune-cell homeostasis in Uganda (Prahl et al., 2021), whereas blood lead showed no overall association with inflammatory cytokines in Kabwe, Zambia (Kataba et al., 2023). Occupational dust studies were similarly divided. Hardwood workers in Kumasi had higher serum cytokines (Ennin et al., 2022), and volcanic-rock stone cutters in Tanzania had higher exhaled nitric oxide (Mamuya et al., 2018), but Tanzanian cement workers did not (Tungu et al., 2013). Six further Nigerian studies of lead, heavy metals, municipal solid waste or wood dust with immune or inflammatory endpoints were identified, as was one of respiratory syncytial virus antibodies in children. Their full texts could not be obtained, so they could not be assessed. They are listed in Supplementary File S5 as priorities for retrieval. The infrastructure that would close that gap, comprising routine environmental monitoring, biomonitoring, geocoded exposure mapping, and linked biobank resources, remains uneven or absent.

Social Isolation, Social Support, and Immune-Inflammatory Outcomes

Social isolation, loneliness, social-network structure, perceived support, social integration, and social capital are related but distinct constructs. Social isolation refers primarily to the objective scarcity of social contacts, whereas loneliness is the subjective perception that social relationships are inadequate (Smith et al., 2020). Social support concerns the perceived or received availability of emotional, informational, or instrumental assistance (Uchino, 2006). Social capital is a broader relational and community construct involving resources generated through social participation, networks, trust, and reciprocity (Ehsan et al., 2019). Because these constructs are captured by different instruments and are not interchangeable, evidence generated for one is not evidence for another, and the distinctions are preserved throughout the section that follows.

Observational and experimental studies have associated poorer social relationships with differences in neuroendocrine regulation, inflammation, and selected immune outcomes (Kiecolt-Glaser et al., 2002; Uchino, 2006). A meta-analysis concluded that loneliness and social isolation may be linked with systemic inflammation, but also emphasised methodological limitations and the need for more robust research (Smith et al., 2020). In that review, social isolation was associated with C-reactive protein and fibrinogen in less-adjusted analyses, and only the fibrinogen association persisted after fuller adjustment. Loneliness was not associated with C-reactive protein or fibrinogen, but was associated with interleukin-6 in most-adjusted analyses. A later scoping review found that studies of loneliness and inflammatory biomarkers were heterogeneous in populations, loneliness measures, and biomarkers. It reported that all 12 included studies identified an association with at least one inflammatory marker, most often C-reactive protein or interleukin 6 (Kim et al., 2024). Because an association with any one of the markers a study measured was counted, and the studies differed in which markers they measured, the evidence is better described as scattered associations than as uniformly positive. These findings support biological plausibility but do not establish a universal, clinically large, or causal effect.

Behavioural factors may contribute to, or partly account for, associations between social disconnection and immune outcomes. Loneliness and isolation can co-occur with sleep disturbance, reduced physical activity, tobacco use, alcohol use, and reduced engagement with healthcare (Kiecolt-Glaser et al., 2002; Uchino, 2006). A systematic review of 23 mostly cross-sectional studies linked loneliness with inflammatory markers, although findings for C-reactive protein were inconsistent (Granieri et al., 2026). Other syntheses retrieved by the documented search add little support for effects beyond inflammation. Social support and integration were associated with higher antibody responses to vaccination, but the pooled effect was small and its confidence interval included zero (Uchino et al., 2020). Social support was not associated with telomere length across 17 studies (Montoya & Uchino, 2023). In women receiving chemotherapy for breast cancer, social support buffered the association between stress and infection, although the immune markers measured did not explain that association (Roy et al., 2021).

Two methodological problems limit what the more recent evidence adds to the meta-analysis by Smith et al. (2020). First, measurement is heterogeneous: studies use different loneliness and isolation instruments and different inflammatory biomarkers, so findings cannot be pooled or compared directly (Kim et al., 2024). Second, reverse causation is plausible because illness and its inflammatory accompaniments can themselves reduce social participation, so cross-sectional associations cannot establish direction. Repeated measurement of both the social construct and the biomarker would be needed to separate these possibilities. The available evidence does not establish that social isolation causes immune dysregulation. The reviews by Smith et al. (2020) and Kim et al. (2024) both reported marked methodological heterogeneity, and Smith et al. (2020) concluded that more robust methodology is needed to confirm the associations and to clarify their mechanisms. Social connection has been associated with protective patterns in some studies, but effect estimates vary and are open to influence by baseline illness, reverse causation, socioeconomic conditions, and measurement choices.

Transferability

No Nigerian or sub-Saharan African study of social isolation, loneliness, or social support with immune endpoints was included in this review. One Kenyan study of refugees that examined physiological profiles by

perceived social support was identified by the search but could not be assessed in full text. Extended-family and community structures in these settings may alter both the distribution and the meaning of isolation, so measures validated elsewhere should not be assumed to transfer.

Socially Patterned Health Behaviours and Immune Outcomes

Diet, physical activity, sleep, tobacco exposure, and alcohol use influence immune and inflammatory processes. Deficiencies of selected micronutrients, including vitamins A and D and zinc, have been associated with impairment of particular epithelial, innate, or adaptive immune functions (Calder et al., 2020). Calder et al. (2020) argued for supplementation, including intakes of vitamins C and D above recommended dietary allowances. However, several of the benefits they describe, for example in trials of vitamins C and D, were most marked in people with low baseline intake or blood concentrations. Whether supplementation improves immune outcomes in people who are already replete is less certain. Regular moderate physical activity is generally associated with favourable immune and inflammatory profiles, whereas the acute immune response to exercise depends on its intensity and duration (Nieman & Wentz, 2019). Heavy exertion has been associated with transient immune dysfunction, raised inflammatory biomarkers, and a higher risk of upper respiratory tract infection (Nieman & Wentz, 2019). Sleep disruption has been linked with increased inflammatory activity and impairment of selected immune responses, including responses to vaccination (Irwin, 2015). Tobacco smoke promotes airway inflammation and alterscan intensify some immune responses while suppressing others, altering multiple components of innate and adaptive immune responsiveness (Qiu et al., 2017). These effects are heterogeneous and direction-specific, differing by exposure, tissue compartment, and immune endpoint rather than amounting to uniform enhancement or suppression of the immune system.

Medical sociology challenges explanations that treat health behaviour as solely a matter of unconstrained individual choice. Socioeconomic position governs access to resources that help people avoid health risks (Link & Phelan, 1995), such as food affordability, housing stability, occupational predictable working schedules, access to safe physical activity environments, and healthcare. Residential environments also shape access to healthy food and opportunities for physical activity (Diez Roux, 2003). This patterning is not confined to high-income settings. Across six southern African countries, smoking and inadequate fruit and vegetable consumption were concentrated among poorer households, as were reliance on biomass cooking fuel and use of unimproved water and sanitation. Poor self-assessed health was also more common among poorer adults, although significantly so only in South Africa (Umuhoza & Ataguba, 2018). Some harmful behaviours may function as short-term coping strategies in settings where safer or more sustainable alternatives are inaccessible. This does not make them biologically harmless or inevitable, but it changes the appropriate intervention target. Interventions aimed only at individual behaviour leave intact the material conditions that constrain it, and those conditions bear most heavily on people for whom the healthier option is least accessible.

Transferability

Beyond survey evidence on socioeconomic gradients in behavioural risk factors (Umuhoza & Ataguba, 2018), two sub-Saharan African studies with inflammatory endpoints were identified. Among 1,569 Black South Africans, no socioeconomic factor was associated with C-reactive protein when examined individually. In adjusted analyses, however, twelve or more years of formal education was associated with about 19% lower C-reactive protein concentration (Myburgh et al., 2020). In the same study, active smokers carrying a particular C-reactive protein gene variant had a higher risk of elevated concentrations. Among 88 mother-infant pairs in Mwanza, Tanzania, severe household food insecurity and high maternal depressive symptoms were each associated with above-median infant C-reactive protein, although maternal mental health did not mediate the association with household conditions (DeCaro et al., 2016). Both studies are cross-sectional, and no comparable Nigerian study was identified. Food insecurity, one of the exposures DeCaro et al. (2016) measured, has been linked more broadly with inflammatory and hormonal dysregulation and with higher allostatic load (Blank et al., 2026). In rural Zimbabwe and Malawi, improvements in water, sanitation and hygiene, and the household environmental exposures measured, had no effect on, or were not associated with, biomarkers of intestinal and systemic inflammation in infants (Gough et al., 2020; Liu et al., 2022).

Intersectionality as an Overarching Framework

Intersectionality originated as a framework for examining how systems of power and social categories operate together rather than independently (Crenshaw, 1989). Its relevance to immune-health research lies in the possibility that exposure, resources, vulnerability, and healthcare access are configured differently at the intersections of race or ethnicity, gender, socioeconomic position, age, disability, migration status, occupation, and geography. Intersectionality is therefore not merely the addition of several demographic covariates. For example, the weathering literature illustrates how racialised and gendered disadvantage may jointly influence cumulative physiological burden among Black women in the United States (Geronimus et al., 2006). In other settings, an intersectional research model might examine how migration status, hazardous employment, economic

deprivation, social isolation, and exclusion from healthcare jointly shape immune-related outcomes. These examples are analytic propositions unless directly supported by studies measuring both intersecting social conditions and immune endpoints. In Nigeria and other sub-Saharan African settings, the axes likely to be salient include ethnicity, religion, gender, socioeconomic position, disability, displacement, occupation, and rural, urban, or regional marginalisation. Which of these carries the greatest weight varies by locality, and the ordering cannot be settled in advance of empirical work, since an axis that structures exposure in one region may be immaterial in another. Statistical interaction analysis contributes to such work but does not exhaust it. Interaction terms describe the joint distribution of categories without accounting for the institutions, historical conditions, and power relations that produce those categories, or for the processes through which they acquire social consequences.

Mixed-methods designs address this limitation directly, pairing biomarker and exposure measurement with interviews, ethnography, or participatory inquiry. The quantitative component locates the pattern; the qualitative component helps elucidate how the exposure arises, how it is experienced, and where it is contested. Without the second, an observed difference between ethnic or gender categories invites the inference that the category is itself biologically explanatory. The more defensible reading is that the category marks a position in a social structure that distributes exposure unequally. Recent methodological work extends intersectional analysis. Simkus et al. (2024) propose using intersectionality to identify covariates across biological, social, environmental, and economic domains in health-equity evaluations rather than relying on conventional demographic measures alone. Bowleg (2026) sets out a Framework for Applied Intersectionality Research (FAIR) that treats intersectionality as a tool for equity action as well as empirical description. Analytically, multilevel analysis of individual heterogeneity and discriminatory accuracy (MAIHDA) nests individuals within intersectional strata. It estimates the variation in an outcome attributable to those strata without the proliferation of interaction terms that conventional regression requires (Evans et al., 2018). In a Nigerian study of an immune endpoint, this would mean defining strata from locally salient axes, for example gender by occupation by geopolitical zone. It would also mean sampling deliberately so that each stratum is adequately populated, and pairing the stratum estimates with qualitative work on how exposure arises within each stratum.

Figure 1 Conceptual framework linking social structure and social position, through four socially patterned pathways and biological embedding, to immune-related outcomes and health disparities. Intersectionality operates across all components. The dashed feedback arrow denotes reverse causation, and the lower panel lists measurement and confounding considerations.

The theory associated with each pathway is shown in parentheses. Read across their rows, Tables 1 and 2 show three cross-domain patterns. First, every domain combines a well-developed sociological account of unequal distribution with a biological literature that is largely observational and drawn from high-income settings. Second, the immune endpoints differ by domain, ranging from glucocorticoid sensitivity and vaccine response in the stress literature to contaminant-specific signalling in the environmental literature, so no common immune readout allows the domains to be compared directly. Third, the Nigerian and sub-Saharan African evidence is concentrated in the environmental and occupational domain, where exposures are measurable and exposed populations are identifiable, but findings there are mixed and several studies were null. The stress and health behaviour domains are each represented by a few small studies, and no regional study of social isolation was included.

DISCUSSION

This review set out to examine how social position may shape immune-relevant exposure (RQ1), through which mechanisms (RQ2), with what Nigerian and sub-Saharan African evidence (RQ3), and with what implications (RQ4). Across the four domains, social position appears to shape exposure through distinct routes, namely sustained insecurity and discrimination, contaminant burden, relational resources, and constrained behaviour. These routes nonetheless converge on a limited set of biological processes: neuroendocrine signalling, inflammatory regulation, oxidative stress, and epigenetic regulation (Figure 1). This convergence is the principal reason for treating the domains as potentially interacting rather than independent. Co-exposure is plausible because disadvantaged positions concentrate several exposures at once, but joint effects were rarely tested; in the Drakenstein birth cohort, a profile combining high indoor pollution, low socioeconomic status and maternal depressive symptoms was associated with higher infant inflammation (Christensen et al., 2024).

The evidence also contains apparent contradictions that are better read as endpoint specificity. Chronic stress is associated with higher inflammatory markers but with poorer antiviral and vaccine responses, whereas for social disconnection the associations with inflammatory markers are inconsistent and the pooled association with vaccine response is imprecise. Environmental contaminants have been associated with contaminant-specific rather than uniform immune effects. Inflammatory biomarkers therefore index one dimension of immune function, not immune competence as a whole. The strength of the evidence also differs by domain. The stress literature includes prospective studies with experimental viral challenge, although stress itself was measured observationally (Cohen et al., 2012). The environmental

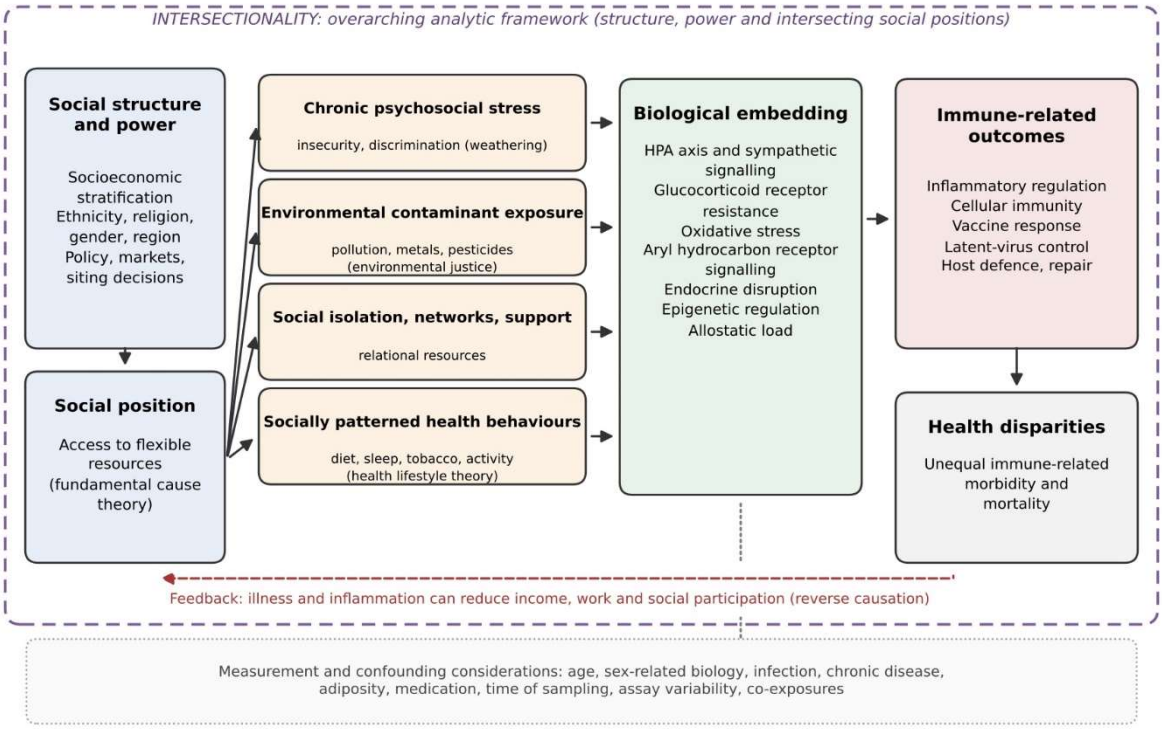

Figure 1: Integrates the four pathways and the sociological theories that account for their unequal distribution.

literature combines mechanistic and observational evidence and, in Nigeria, one randomised trial with a null overall result (Olopade et al., 2017). The social isolation and health behaviour literatures in this context are predominantly observational.

Causal inference remains the central limitation. Most human evidence is cross-sectional, reverse causation is plausible in every domain, and the same social conditions that generate exposure also generate confounders such as infection, adiposity, and limited healthcare access. The defensible conclusion is that the pathways are biologically plausible and consistent with the available associations, not that their magnitude or causal structure has been established. Transferability is a further constraint. The sociological principles are plausible in these settings but largely untested. However, the specific exposures, the axes along which they are distributed, and the immune consequences in Nigerian and sub-Saharan African populations have been measured in a modest, mixed and mostly cross-sectional body of studies. Much of that body comes from outside Nigeria.

Table 3 compares the present review with recent related reviews and frameworks. What distinguishes it from them is that each biological pathway is matched to a sociological explanation of who is exposed, together with an assessment of how much of that link has been studied in Nigerian and other sub-Saharan African populations.

Implications for Research, Practice, and Policy

The implications below differ in the strength of evidence on which they rest. Immediately actionable are assessing social risk with locally validated instruments where it can guide care, and not using C-reactive protein or interleukin-6 as screening tests for social adversity. These follow from the nonspecificity of the markers rather than from new causal evidence. Interventions such as clean cooking are plausible on exposure-reduction grounds but have not yet been shown to change immune or inflammatory endpoints. A trial design against inflammatory endpoints already exists in Nigeria, although that trial found no overall effect (Olopade et al., 2017). Research priorities, for which the evidence identifies a need rather than an effect, are longitudinal cohorts, biomonitoring infrastructure, and intersectional study designs. Immunological studies should incorporate validated social measures when those measures are relevant to the causal question. These may include multidimensional socioeconomic position, discrimination, neighbourhood conditions, occupational exposure, food insecurity, housing insecurity, and social support. Investigators should specify whether social factors are treated as exposures, confounders, mediators, moderators, or consequences. Longitudinal and repeated-measures designs are particularly useful for examining cumulative exposure and within-person change. In

Table 3. Comparison of the present review with recent related reviews and frameworks.

Source	Design and scope	Agreement with present review	What the present review adds
Smith et al. (2020)	Systematic review and meta-analysis: loneliness, social isolation and inflammation	Associations with systemic inflammation are possible but methodologically limited	Places social connection alongside three other socially patterned pathways and a sociological account of distribution
Kim et al. (2024)	Scoping review: loneliness and inflammatory biomarkers	Heterogeneous measures and biomarkers; scattered rather than uniform associations	Distinguishes isolation, loneliness, support and social capital; addresses sub-Saharan African evidence gap
Simkus et al. (2024)	Methodological framework: intersectionality for covariate selection in health-equity research	Intersectionality is more than adding demographic covariates	Applies intersectionality to immune endpoints and to Nigerian axes of social position
Vijendra et al. (2025)	Narrative review: five Healthy People 2030 domains and inflammatory pathways; mainly high-income evidence	Social determinants plausibly linked to inflammation via HPA axis, cytokine and innate pathways	Grounds each pathway in medical-sociological theory; treats intersectionality as overarching; inventories Nigerian and SSA evidence; separates evidence types

Note. HPA, hypothalamic-pituitary-adrenal; SSA, sub-Saharan Africa.

Nigerian and other sub-Saharan African settings, the immediate priority is the infrastructure that makes such studies possible, comprising environmental monitoring and biomonitoring, geocoded exposure mapping linked to community characteristics, and cohort or biobank resources capable of supporting longitudinal analysis.

Immune outcomes must also be selected and interpreted carefully. Cytokines, C-reactive protein, leukocyte subsets, antibody responses, latent-virus titres, and clinical infection outcomes represent different biological processes. No single marker provides a global measure of immune competence. Studies should account for acute infection, chronic disease, obesity, medication use, smoking, sleep, age, sex-related biology, time of sampling, and assay variability where relevant.

In clinical and public health practice, social risks should be assessed when the information can guide care, referral, safeguarding, occupational advice, or access to services. Inflammatory biomarkers such as C-reactive protein or interleukin-6 should not be used as routine screening tests for social adversity. C-reactive protein is nonspecific, interleukin-6 is not a standard population-screening test, and no evidence reviewed here establishes that socially targeted cytokine screening improves outcomes. Biomarkers should be ordered according to established clinical indications.

Community health-worker programmes, peer support, social prescribing, and participatory community initiatives are reasonable responses to loneliness and unmet practical need. Whether they produce sustained change in inflammatory biomarkers or immune-sensitive clinical outcomes is, however, an open question that requires direct intervention studies. Such studies are feasible in the settings of interest. The Ibadan stove trial described above evaluated an exposure-reduction intervention against inflammatory endpoints, and its null overall result shows why interventions should be evaluated rather than assumed to work (Olopade et al., 2017). Community-based participatory research may improve contextual validity, exposure definition, trust, and the relevance of proposed interventions, although these benefits should be evaluated within the setting in which the approach is used. Policies governing housing, employment, income, urban planning, environmental regulation, education, and food systems may plausibly shape immune-related health through exposure, stress, behaviour, and access to protective resources. A structurally informed approach to health behaviour combines individual support with attention to food environments, housing, working conditions, tobacco control, safe public spaces, and access to preventive services. A Health in All Policies orientation provides one framework for considering these cross-sector effects (Baum et al., 2014; Rudolph et al., 2013). The environmental justice movement has also provided an influential civil-society framework for challenging unequal environmental burdens. Policy claims should nevertheless be linked to measurable exposure reduction and health outcomes rather than assumed from intent alone.

Limitations

This review has several limitations. First, it is a structured narrative review rather than a systematic review. Although a structured search was used, no preregistered protocol, exhaustive screening process, or Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram was employed. The search reported here was documented in full, but earlier searching that identified some of the cited sources was not documented, and the documented search may not have retrieved every eligible study, so selection bias is possible. Second, screening and full-text assessment were carried out by an AI assistant applying the stated criteria, without

independent duplicate screening by a human reviewer, so systematic misclassification is possible, and interpretation rests with the two authors. In addition, 115 of the 293 records sought in full text could not be obtained, including seven Nigerian studies with immune or inflammatory endpoints, so the synthesis may be affected by availability bias. Third, the methodological quality and risk of bias of individual studies were not formally assessed, and the evidence was not graded for certainty; the evidence-type labels describe study design, not quality.

Fourth, the included literature used heterogeneous definitions of social exposure and diverse immune endpoints. Loneliness, social isolation, social support, and social capital are not interchangeable, while inflammation, immune activation, and impaired host defence represent different biological states. Fifth, much of the human evidence is observational and may be affected by residual confounding, reverse causation, baseline illness, medication use, adiposity, infection, and health behaviour. Mechanistic findings from animal or experimental studies cannot be transferred uncritically to population-level human disparities.

Sixth, much of the empirical literature was produced in the United States and Western Europe. Direct transferability to Nigeria and other sub-Saharan African settings is limited by differences in social stratification, environmental exposure, demography, health systems, and disease burden. The Nigerian and other sub-Saharan African studies cited here partly offset this, but they are few, mostly cross-sectional, and unevenly distributed across exposures and regions. Regional examples should be treated as research priorities except where direct local immune evidence is available. Although African Index Medicus was searched, the African Journals Online search was unavailable, Scopus and Web of Science were not accessible, and grey literature and non-English sources were not searched, so the Nigerian and sub-Saharan African inventory may be incomplete. One practitioner guide is cited, for policy framing only. The search design may also have shaped the regional findings. The regional environmental search (Search C) did not require a social-exposure term, whereas the regional social-exposure search (Search B) required social terms in the title. The concentration of regional evidence in environmental and occupational exposures may therefore partly reflect the search rather than the literature. Finally, the search extended to 14 October 2026, but rapid developments across social immunology, environmental health, and intersectional methods mean that later evidence may alter some interpretations.

Conclusion

In answer to the review questions, social position appears to shape immune-relevant exposure through four pre-specified and potentially interacting pathways (RQ1). These converge on neuroendocrine, inflammatory, oxidative, and epigenetic processes, supported mainly by observational and mechanistic evidence with limited experimental confirmation (RQ2). Direct Nigerian and sub-Saharan African evidence is concentrated in environmental and occupational exposures, where findings are mixed, with few studies in other domains, although this concentration may partly reflect the search design (RQ3). The principal implications are to measure social exposure directly, to avoid using inflammatory biomarkers as proxies for adversity, and to build the monitoring and cohort infrastructure that local evidence requires (RQ4). The principal contribution of this review is to pair each immune pathway with an explicit sociological account of how exposure is distributed, and to show how little of that pairing has yet been tested in Nigeria and sub-Saharan Africa.

The immune system is embedded within social and environmental contexts. Chronic adversity, environmental contaminants, social disconnection, and socially patterned health behaviours may contribute to immune-related health differences through interacting neuroendocrine, inflammatory, behavioural, and exposure pathways. These relationships are biologically plausible and consistent with a substantial but uneven literature, and their magnitude and causal structure vary by population, measurement, and outcome.

Of the four domains examined, chronic psychosocial stress has the most extensive literature linking social exposure with neuroendocrine and immune-inflammatory outcomes, although this reflects the interpretive judgement of the authors rather than a formal ranking of the evidence. Environmental immunotoxicology in low- and middle-income settings, although the best represented regional domain, remains small, mixed and largely cross-sectional, and the immune consequences of intersecting social positions outside Western contexts remain an important gap. Future work should combine rigorous exposure assessment, clearly defined immune endpoints, longitudinal designs, and sociological analysis of institutions and power.

Bridging immunology and medical sociology strengthens explanations of health disparities by organising the evidence on how social structures may shape exposure and how that exposure may register in biological and clinical processes. The objective is not to medicalise social disadvantage through nonspecific biomarkers. It is to produce evidence that connects biological mechanisms with modifiable social and environmental conditions and can inform more equitable research, clinical practice, and public policy.

Declarations

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Ethics statement: This structured narrative review did not involve primary data collection from human participants. Ethics approval was therefore not required.

Data availability statement: No new primary data were generated. The screening workbook recording every screening decision is available from the authors on request. All evidence discussed was derived from the sources cited in the manuscript. The search strategy and yield, evidence-extraction matrix, SANRA self-assessment, provenance of cited sources, and the records included from and not retrieved by the documented search are provided as Supplementary Files S1 to S5. The de-duplicated search records (n = 983), with every screening decision and reason, are available from the corresponding author on reasonable request.

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Supplementary File S1. Search strategy and yield

The search uses three targeted searches rather than one broad search, because a single combination of all social-exposure and immune terms returned over 90,000 PubMed records, which a structured narrative review cannot screen. Search A identifies reviews and meta-analyses that link social exposures with immune outcomes; Searches B and C identify primary studies from Nigeria and sub-Saharan Africa. Limits: English language; published from 1 January 1990; human studies.

PubMed (run 2026-10-14)

#1 Social exposure (title words): ("social determinant*"[ti] OR "health disparit*"[ti] OR "health inequ*"[ti] OR socioeconomic[ti] OR "socio-economic"[ti] OR "social position"[ti] OR "social status"[ti] OR poverty[ti] OR deprivation[ti] OR discrimination[ti] OR racism[ti] OR "chronic stress"[ti] OR "psychosocial stress"[ti] OR adversity[ti] OR "social isolation"[ti] OR loneliness[ti] OR "social support"[ti] OR "social network*"[ti] OR intersectional*[ti] OR "environmental justice"[ti] OR "food insecurity"[ti])
#2 Immune outcome: ("C-Reactive Protein"[Mesh] OR "Interleukin-6"[Mesh] OR "Psychoneuroimmunology"[Mesh] OR "Allostasis"[Mesh] OR "Inflammation"[Majr] OR inflammat*[tiab] OR "C-reactive protein"[tiab] OR interleukin*[tiab] OR cytokine*[tiab] OR "immune function"[tiab] OR "immune response*"[tiab] OR "allostatic load"[tiab] OR "biological embedding"[tiab] OR "glucocorticoid resistance"[tiab] OR "telomere length"[tiab])
#3 Region (country names are used instead of a truncated Africa* term, which also retrieved studies of African Americans): ("Africa South of the Sahara"[Mesh] OR "Nigeria"[Mesh] OR Nigeria*[tiab] OR "sub-Saharan"[tiab] OR "Africa"[tiab] OR Ghana*[tiab] OR Kenya*[tiab] OR Uganda*[tiab] OR Tanzania*[tiab] OR Malawi*[tiab] OR Ethiopia*[tiab] OR "South Africa"[tiab] OR Cameroon*[tiab] OR Zambia*[tiab] OR Zimbabwe*[tiab])
#4 Environmental or occupational exposure: ("Environmental Exposure"[Mesh] OR "Occupational Exposure"[Mesh] OR pollut*[tiab] OR pesticide*[tiab] OR lead[ti] OR "heavy metal*"[tiab] OR biomass[tiab] OR "household air"[tiab])
#5 Review designs: (review[pt] OR "systematic review"[pt] OR "meta-analysis"[pt])
#6 Limits: english[la] AND ("1990/01/01"[dp] : "3000"[dp]) AND humans[mh] NOT (animals[mh] NOT humans[mh])
Search A: #1 AND #2 AND #5 AND #6 (460 records). Search B: #1 AND #2 AND #3 AND #6 (26 records). Search C: #4 AND #2 AND #3 AND #6 (141 records).

Europe PMC (run 2026-10-14)

The PubMed searches were translated to Europe PMC field syntax (TITLE: and ABSTRACT:), with the same structure, English language and publication years 1990 to 2026. Europe PMC has no human-studies filter. Search A: 465 records; Search B: 20; Search C: 120. Of 603 unique records, 431 were already retrieved by PubMed, leaving 172 additional records.
Search A: (TITLE:"social determinant*" OR TITLE:"health disparit*" OR TITLE:"health inequ*" OR TITLE:socioeconomic OR TITLE:"socio-economic" OR TITLE:"social position" OR TITLE:"social status" OR TITLE:poverty OR TITLE:deprivation OR TITLE:discrimination OR TITLE:racism OR TITLE:"chronic stress" OR TITLE:"psychosocial stress" OR TITLE:adversity OR TITLE:"social isolation" OR TITLE:loneliness OR TITLE:"social support" OR TITLE:"social network*" OR TITLE:intersectional* OR TITLE:"environmental justice" OR TITLE:"food insecurity") AND (TITLE:inflammat* OR ABSTRACT:inflammat* OR TITLE:"C-reactive protein" OR ABSTRACT:"C-reactive protein" OR TITLE:interleukin* OR ABSTRACT:interleukin* OR TITLE:cytokine* OR ABSTRACT:cytokine* OR TITLE:"immune function" OR ABSTRACT:"immune function" OR TITLE:"immune response*" OR ABSTRACT:"immune response*" OR TITLE:"allostatic load" OR ABSTRACT:"allostatic load" OR TITLE:"biological embedding" OR ABSTRACT:"biological embedding" OR TITLE:"glucocorticoid resistance" OR ABSTRACT:"glucocorticoid resistance" OR TITLE:"telomere length" OR ABSTRACT:"telomere length" OR TITLE:psychoneuroimmunolog* OR ABSTRACT:psychoneuroimmunolog*) AND (PUB_TYPE:"review" OR PUB_TYPE:"systematic-review" OR PUB_TYPE:"meta-analysis") AND LANG:"eng" AND PUB_YEAR:[1990 TO 2026]
Search B: (TITLE:"social determinant*" OR TITLE:"health disparit*" OR TITLE:"health inequ*" OR TITLE:socioeconomic OR TITLE:"socio-economic" OR TITLE:"social position" OR TITLE:"social status" OR TITLE:poverty OR TITLE:deprivation OR TITLE:discrimination OR TITLE:racism OR TITLE:"chronic stress" OR TITLE:"psychosocial stress" OR TITLE:adversity OR TITLE:"social isolation" OR TITLE:loneliness OR TITLE:"social support" OR TITLE:"social network*" OR TITLE:intersectional* OR TITLE:"environmental justice" OR TITLE:"food insecurity") AND (TITLE:inflammat* OR ABSTRACT:inflammat* OR TITLE:"C-reactive protein" OR ABSTRACT:"C-reactive protein" OR TITLE:interleukin* OR ABSTRACT:interleukin* OR TITLE:cytokine* OR ABSTRACT:cytokine* OR TITLE:"immune function" OR ABSTRACT:"immune function" OR TITLE:"immune response*" OR ABSTRACT:"immune response*" OR TITLE:"allostatic

load" OR ABSTRACT:"allostatic load" OR TITLE:"biological embedding" OR ABSTRACT:"biological embedding" OR TITLE:"glucocorticoid resistance" OR ABSTRACT:"glucocorticoid resistance" OR TITLE:"telomere length" OR ABSTRACT:"telomere length" OR TITLE:psychoneuroimmunolog* OR ABSTRACT:psychoneuroimmunolog*) AND (TITLE:Nigeria* OR ABSTRACT:Nigeria* OR TITLE:"sub-Saharan" OR ABSTRACT:"sub-Saharan" OR TITLE:"Africa" OR ABSTRACT:"Africa" OR TITLE:Ghana* OR ABSTRACT:Ghana* OR TITLE:Kenya* OR ABSTRACT:Kenya* OR TITLE:Uganda* OR ABSTRACT:Uganda* OR TITLE:Tanzania* OR ABSTRACT:Tanzania* OR TITLE:Malawi* OR ABSTRACT:Malawi* OR TITLE:Ethiopia* OR ABSTRACT:Ethiopia* OR TITLE:"South Africa" OR ABSTRACT:"South Africa" OR TITLE:Cameroon* OR ABSTRACT:Cameroon* OR TITLE:Zambia* OR ABSTRACT:Zambia* OR TITLE:Zimbabwe* OR ABSTRACT:Zimbabwe*) AND LANG:"eng" AND PUB_YEAR:[1990 TO 2026]
Search C: (TITLE:pollut* OR ABSTRACT:pollut* OR TITLE:pesticide* OR ABSTRACT:pesticide* OR TITLE:"heavy metal*" OR ABSTRACT:"heavy metal*" OR TITLE:biomass OR ABSTRACT:biomass OR TITLE:"household air" OR ABSTRACT:"household air" OR TITLE:"occupational exposure" OR ABSTRACT:"occupational exposure" OR TITLE:"environmental exposure" OR ABSTRACT:"environmental exposure") AND (TITLE:inflammat* OR ABSTRACT:inflammat* OR TITLE:"C-reactive protein" OR ABSTRACT:"C-reactive protein" OR TITLE:interleukin* OR ABSTRACT:interleukin* OR TITLE:cytokine* OR ABSTRACT:cytokine* OR TITLE:"immune function" OR ABSTRACT:"immune function" OR TITLE:"immune response*" OR ABSTRACT:"immune response*" OR TITLE:"allostatic load" OR ABSTRACT:"allostatic load" OR TITLE:"biological embedding" OR ABSTRACT:"biological embedding" OR TITLE:"glucocorticoid resistance" OR ABSTRACT:"glucocorticoid resistance" OR TITLE:"telomere length" OR ABSTRACT:"telomere length" OR TITLE:psychoneuroimmunolog* OR ABSTRACT:psychoneuroimmunolog*) AND (TITLE:Nigeria* OR ABSTRACT:Nigeria* OR TITLE:"sub-Saharan" OR ABSTRACT:"sub-Saharan" OR TITLE:"Africa" OR ABSTRACT:"Africa" OR TITLE:Ghana* OR ABSTRACT:Ghana* OR TITLE:Kenya* OR ABSTRACT:Kenya* OR TITLE:Uganda* OR ABSTRACT:Uganda* OR TITLE:Tanzania* OR ABSTRACT:Tanzania* OR TITLE:Malawi* OR ABSTRACT:Malawi* OR TITLE:Ethiopia* OR ABSTRACT:Ethiopia* OR TITLE:"South Africa" OR ABSTRACT:"South Africa" OR TITLE:Cameroon* OR ABSTRACT:Cameroon* OR TITLE:Zambia* OR ABSTRACT:Zambia* OR TITLE:Zimbabwe* OR ABSTRACT:Zimbabwe*) AND LANG:"eng" AND PUB_YEAR:[1990 TO 2026]
Scopus and Web of Science were not searched, because the authors did not have subscription access at the time of the search.

African Index Medicus (run 2026-10-14)

Searched through the WHO Global Index Medicus interface, restricted to the African Index Medicus database. Simple keyword combinations were used because the platform does not support the full Boolean strings: (inflammation OR immune OR cytokine OR "C-reactive protein") combined in turn with each of ten terms. Results: stress: 31; poverty: 47; socioeconomic: 70; pollution: 2; pesticide: 0; lead: 26; biomass: 1; smoking: 5; food insecurity: 4; social support: 28; total 214, 187 unique records. No year or language limits were applied at search; eligibility criteria were applied at screening.

African Journals Online

The same ten keyword combinations were attempted in the African Journals Online article search on 14 October 2026. The search function returned a server error (HTTP 500) for every query, including single-word test queries, so no records could be retrieved.

Database	Date searched	Records retrieved (unique new records)	Full text assessed	Included
PubMed (A + B + C)	2026-10-14	627 (624)	142	115
Europe PMC (A + B + C)	2026-10-14	605 (172 not already in PubMed)	36	22
African Journals Online	2026-10-14	Search unavailable (server error)	–	–
African Index Medicus	2026-10-14	214 (187)	0 (2 sought, not retrieved)	0
After de-duplication		983	178	137
Theoretical, landmark or previously identified sources added				44

Flow of records: 983 screened on title and abstract; 690 excluded; 293 sought in full text; 115 not retrieved; 178 assessed in full text; 41 excluded; 137 included. Screening and full-text assessment were carried out by an AI assistant (Claude, Anthropic) applying the eligibility criteria in the Methods, and each decision was recorded with a reason code. Records are counted under the first database that retrieved them.
Reasons for exclusion at title and abstract: E1, no socially patterned exposure (237); E2, no immune, inflammatory, neuroendocrine or immune-sensitive clinical outcome (312); E3, animal or in vitro only, not used for mechanism (105); E4,

not English (11); E5, published before 1990 (0); E6, conference abstract, commentary or editorial without original evidence (17); E7, duplicate or superseded report (8).
Reasons for exclusion at full text: E8, full text not available (115); E1, no socially patterned exposure (20); E3, animal or in vitro only (9); E2, no immune, inflammatory, neuroendocrine or immune-sensitive outcome (9); E9, not peer-reviewed (thesis) (2); E6, conference abstract without full text (1).

Supplementary File S2. Evidence-extraction matrix (empirical studies)

Reviews and theoretical sources are not extracted here; their evidence type is recorded in Table 1.

Study	Country	Design (type)	Sample	Social exposure	Immune endpoint	Principal finding	Adjustment	Limitation
Cohen et al. (2012)	USA	Two prospective studies with rhinovirus challenge (O; experimental viral exposure)	Study 1: 276 healthy adults; Study 2: 79 adults	Chronic stress	Glucocorticoid receptor resistance; inflammatory response	Recent chronic threatening stress associated with GCR resistance; GCR resistance predicted colds (Study 1) and greater nasal pro-inflammatory cytokine production when infected (Study 2)	Age, sex, race, BMI, education, season, prechallenge antibody; virus type (Study 1)	Healthy volunteers; experimental infection model
Geronimus et al. (2006)	USA	Cross-sectional, NHANES (O)	Adults aged 18–64	Race; age; sex; poverty	Allostatic load	Higher allostatic load among Black adults at all ages, largest at 35–64; highest among Black women; not explained by poverty	Poverty status examined	Allostatic load not immune-specific
Chae et al. (2020)	USA	Longitudinal cohort, CARDIA (O)	391 African American adults; ~10-year follow-up	Racial discrimination across life domains	Leukocyte telomere length	Greater and increasing discrimination associated with shorter LTL and faster shortening	Baseline discrimination and LTL; other covariates in full text	Telomere length nonspecific

Supplementary File S3. SANRA self-assessment

The checklist below shows where the manuscript addresses each of the six SANRA items (Baethge et al., 2019), with the authors' self-assessed score for each item (0, low; 1, moderate; 2, high). The total self-assessed score is 11 of 12. This is the authors' own judgement and not an independent rating.

SANRA item	Where addressed in the manuscript	Score (0 to 2)	Basis for score
1. Explanation of the review's importance	Introduction: central problem statement; contemporary evidence; Nigeria and sub-Saharan African gap	2	Problem and gap stated with current evidence
2. Statement of the aims	Abstract aim; Introduction RQ1 to RQ4; answered in Discussion and Conclusion	2	Aim and four review questions stated explicitly
3. Description of the literature search	Review Approach; Supplementary File S1 (strings, dates, yield)	2	Databases, strings, dates and yield reported
4. Referencing	Key statements referenced; evidence-type labels in Tables 1 and 2	2	Key statements referenced; all references verified
5. Scientific reasoning	Evidence types distinguished; association distinguished from causation; transferability discussed for Nigeria and sub-Saharan Africa	1	Evidence types labelled, but no formal appraisal of individual studies
6. Presentation of relevant and appropriate endpoint data	Tables 1 to 3; Figure 1; Supplementary File S2	2	Endpoint data tabulated in Tables 1 and 2 and Supplementary Files S2 and S5

Kivimäki et al. (2025)	UK, Finland, USA	Multicohort (O)	778,732 for disease outcomes (UK Biobank 492,257; Finnish Public Sector study 286,475); proteomics in Whitehall II (n = 6,235) and ARIC (n = 11,798 midlife)	Early-life and adult socioeconomic disadvantage	Plasma proteome incl. inflammatory proteins; age-related disease	Disadvantage associated with 14 age-related proteins (NF-κB/IL-8 pathway enriched) and risk of 66 diseases; proteins mediated up to 39% of associations	Age, sex and ethnicity	High-income cohorts; observational
Oluwole et al. (2013)	Nigeria	Cross-sectional (O)	59 mother-child pairs	Biomass smoke	CRP; lipid peroxidation	See Table 2	Lung-function models adjusted for age, height, tobacco smoke	No comparison group
Yaqub et al. (2014)	Nigeria	Comparative (O)	30 applicators; 30 farmers; 30 controls	Organophosphates (serum acetylcholinesterase)	Leukocyte subsets; immunoglobulins	See Table 2	None; controls age- and sex-matched; unadjusted t-tests	Small sample
Tirima et al. (2016)	Nigeria	Programme report (O)	Children aged ≤5 y	Lead	Blood lead only	GM blood lead 149 µg/dL before, 15 µg/dL after programme	Not applicable (descriptive before-after)	No immune endpoint
Olopade et al. (2017)	Nigeria	RCT (E)	324 randomised; 271 analysed	Household air pollution	TNF-α; IL-6; IL-8; MDA; RBP	No overall between-arm difference; TNF-α reduction in prior firewood users only	Randomised	Single city; subgroup-only effect
Umuhoza & Ataguba (2018)	Six SADC countries	World Health Survey 2002/04 (O)	National samples, six countries	Household expenditure	None	See Table 2	Standardised concentration indices	No biological measures
Winful et al. (2024)	Nigeria	Cross-sectional (O)	138 adults	GHQ-12 stress; cortisol	Salivary CRP	See Table 2	Multivariable regression	Small; single measure
Myburgh et al. (2020)	South Africa	Cross-sectional (O)	1,569 adults	Education; smoking	CRP	See Table 2	Adjusted models (covariates in full text)	Cross-sectional
DeCaro et al. (2016)	Tanzania	Cross-sectional (O)	88 mother-infant pairs	Food insecurity; maternal mental health	Infant hs-CRP	See Table 2	Mediation analysis	Cross-sectional

Note. ARIC, Atherosclerosis Risk in Communities; BMI, body mass index; CARDIA, Coronary Artery Risk Development in Young Adults; Cd, cadmium; CRP, C-reactive protein; GCR, glucocorticoid receptor; hs-CRP, high-sensitivity C-reactive protein; IL, interleukin; LTL, leukocyte telomere length; MDA, malondialdehyde; NHANES, National Health and Nutrition Examination Survey; NF-κB, nuclear factor kappa B; Pb, lead; RBP, retinol-binding protein; RCT, randomised controlled trial; SADC, Southern African Development Community; TNF-α, tumour necrosis factor-alpha. Evidence-type codes as in Table 1.

Supplementary File S4. Provenance of cited sources

For each cited source, the table records how it entered the review and whether the documented PubMed search (Supplementary File S1) retrieves it. Sources added from the documented search are those cited in the main text; all records included from that search are listed in Supplementary File S5. Sources identified outside the documented search were found through earlier searching whose histories were not retained and through citation tracking; they are reported here rather than as part of the documented search. Theoretical and landmark works are not expected to be retrieved by searches restricted to reviews and regional primary studies.

Source	How identified	Retrieved by documented PubMed search
Abdo (2021)	Documented search, PubMed, search C	Yes (Search C)
Akpoka (2026)	The first author's published review; not retrieved by the documented search	No
Aljohani (2026)	Documented search, Europe PMC, search B	No (retrieved by Europe PMC search B)
Alotiby (2024)	Earlier unrecorded searching and citation tracking	No
Baethge (2019)	Earlier unrecorded searching and citation tracking	No
Baum (2014)	Earlier unrecorded searching and citation tracking	No
Beese (2022)	Earlier unrecorded searching and citation tracking	No
Blank (2026)	Documented search, Europe PMC, search A	No (retrieved by Europe PMC search A)
Bowleg (2026)	Targeted supplementary search, outside the documented search	No
Buthelezi (2024)	Documented search, Europe PMC, search C	No (retrieved by Europe PMC search C)
Calder (2020)	Earlier unrecorded searching and citation tracking	No
Chae (2020)	Earlier unrecorded searching and citation tracking	No
Christensen (2024)	Documented search, PubMed, search C	Yes (Search C)
Cockerham (2005)	Earlier unrecorded searching and citation tracking	No
Cohen (2012)	Earlier unrecorded searching and citation tracking	No
Coimbra (2020)	Documented search, PubMed, search A	Yes (Search A)
Colich (2020)	Documented search, PubMed, search A	Yes (Search A)
Crenshaw (1989)	Earlier unrecorded searching and citation tracking	No
Cuevas (2026)	Documented search, PubMed, search A	Yes (Search A)
DeCaro (2016)	Targeted supplementary search, outside the documented search	No
Dhabhar (2014)	Earlier unrecorded searching and citation tracking	No
Di Lenardo (2020)	Documented search, PubMed, search C	Yes (Search C)
Diez Roux (2003)	Earlier unrecorded searching and citation tracking	No
Dowd (2009)	Documented search, PubMed, search A	Yes (Search A)
Ehsan (2019)	Earlier unrecorded searching and citation tracking	No
Ennin (2022)	Documented search, PubMed, search C	Yes (Search C)
Evans (2018)	Targeted supplementary search, outside the documented search	No
Forde (2019)	Earlier unrecorded searching and citation tracking	No
Geronimus (2006)	Earlier unrecorded searching and citation tracking	No
Ghantous (2021)	Documented search, PubMed, search C	Yes (Search C)
Gough (2020)	Documented search, PubMed, search C	Yes (Search C)
Granieri (2026)	Documented search, PubMed, search A	Yes (Search A)
Hernandez-Vargas (2015)	Documented search, PubMed, search C	Yes (Search C)
Irwin (2015)	Earlier unrecorded searching and citation tracking	No
Jirtle (2007)	Earlier unrecorded searching and citation tracking	No
Johnson (2017)	Documented search, PubMed, search A	Yes (Search A)
Kaali (2021)	Documented search, Europe PMC, search C	No (retrieved by Europe PMC search C)
Kataba (2023)	Documented search, PubMed, search C	Yes (Search C)
Kelly (2021)	Documented search, PubMed, search B	Yes (Search B)
Kiecolt-Glaser (2002)	Earlier unrecorded searching and citation tracking	No
Kim (2024)	Earlier unrecorded searching and citation tracking	Yes (Search A)
Kivimäki (2025)	Targeted supplementary search, outside the documented search	No
Kohler (2013)	Documented search, Europe PMC, search B	No (retrieved by Europe PMC search B)
Kuhlman (2020)	Documented search, PubMed, search A	Yes (Search A)
Landrigan (2018)	Earlier unrecorded searching and citation tracking	No
Lawrence (2022)	Documented search, PubMed, search A	Yes (Search A)
Lin (2021)	Documented search, Europe PMC, search C	No (retrieved by Europe PMC search C)
Link (1995)	Earlier unrecorded searching and citation tracking	No

Liu (2017)	Documented search, PubMed, search A	Yes (Search A)
Liu (2022)	Documented search, PubMed, search C	Yes (Search C)
Lunjani (2024)	Documented search, PubMed, search C	Yes (Search C)
Makamure (2016)	Documented search, PubMed, search C	Yes (Search C)
Mamuya (2018)	Documented search, PubMed, search C	Yes (Search C)
Marmot (2005)	Earlier unrecorded searching and citation tracking	No
Misra (2018)	Documented search, PubMed, search C	Yes (Search C)
Mohai (2009)	Earlier unrecorded searching and citation tracking	No
Montoya (2023)	Documented search, PubMed, search A	Yes (Search A)
Morakinyo (2022)	Documented search, PubMed, search C	Yes (Search C)
Morello-Frosch (2002)	Earlier unrecorded searching and citation tracking	No
Muscatell (2020)	Documented search, PubMed, search A	Yes (Search A)
Myburgh (2020)	Targeted supplementary search, outside the documented search	No
Nieman (2019)	Earlier unrecorded searching and citation tracking	No
Nigatu (2015)	Documented search, Europe PMC, search C	No (retrieved by Europe PMC search C)
Olopade (2017)	Earlier unrecorded searching and citation tracking	Yes (Search C)
Olsson (2024)	Documented search, PubMed, search C	Yes (Search C)
Olude (2022)	Documented search, PubMed, search C	Yes (Search C)
Oluwole (2013)	Earlier unrecorded searching and citation tracking	Yes (Search C)
Orebanjo (2026)	Co-authored primary study by the first author, cited for the occurrence of the exposure in Nigeria; not retrieved by the documented search	No
Prahl (2021)	Documented search, PubMed, search C	Yes (Search C)
Priest (2024)	Documented search, PubMed, search A	Yes (Search A)
Qiu (2017)	Earlier unrecorded searching and citation tracking	No
Rivillas-García (2026)	Documented search, PubMed, search A	Yes (Search A)
Roba (2025)	Documented search, PubMed, search B	Yes (Search B)
Robertson (2013)	Documented search, PubMed, search A	Yes (Search A)
Roy (2021)	Documented search, Europe PMC, search A	No (retrieved by Europe PMC search A)
Rudolph (2013)	Earlier unrecorded searching and citation tracking	No
Rylance (2015)	Documented search, PubMed, search C	Yes (Search C)
Segerstrom (2004)	Earlier unrecorded searching and citation tracking	No
Simkus (2024)	Targeted supplementary search, outside the documented search	No
Smith (2020)	Earlier unrecorded searching and citation tracking	Yes (Search A)
Solanke (2026)	Co-authored primary study by the first author, cited for the occurrence of the exposure in Nigeria; not retrieved by the documented search	No
Suzuki (2020)	Earlier unrecorded searching and citation tracking	No
Szanton (2005)	Documented search, PubMed, search A	Yes (Search A)
Taylor (2016)	Targeted supplementary search, outside the documented search	No
Tiekwe (2021)	Documented search, PubMed, search C	Yes (Search C)
Tiekwe (2024)	Documented search, Europe PMC, search C	No (retrieved by Europe PMC search C)
Tirima (2016)	Earlier unrecorded searching and citation tracking	No
Toe (2022)	Documented search, PubMed, search C	Yes (Search C)
Tungu (2013)	Documented search, PubMed, search C	Yes (Search C)
Uchino (2006)	Earlier unrecorded searching and citation tracking	Yes (Search A)
Uchino (2020)	Documented search, PubMed, search A	Yes (Search A)
Umuhoza (2018)	Earlier unrecorded searching and citation tracking	No
Vijendra (2025)	Targeted supplementary search, outside the documented search	No
Williams (2001)	Earlier unrecorded searching and citation tracking	No
Williams (2019)	Earlier unrecorded searching and citation tracking	No
Winful (2024)	Targeted supplementary search, outside the documented search	No
Yaqub (2014)	Earlier unrecorded searching and citation tracking	No

Supplementary File S5. Records from the documented search: included, and sought but not retrieved

Part A lists the 137 records included after full-text assessment, with the domain to which each was assigned and its principal finding as extracted. Part B lists the 115 records included at title and abstract whose full text could not be obtained from openly available sources on 15 October 2026; they were therefore excluded (code E8). Part B is ordered by priority for retrieval: (1) Nigerian primary studies, (2) other sub-Saharan African studies, (3) systematic or scoping reviews and meta-analyses, and (4) narrative reviews. Screening and full-text assessment were carried out by an AI assistant applying the eligibility criteria in the Methods; every decision and reason is recorded in the screening workbook.

A. Records included (n = 137)

Study	Domain	SSA	Principal finding
Abdo et al. (2021)	Environment (household air pollution)	Yes	Northern Ghana cookstove RCT (200 households): some evidence of lower serum amyloid A and CRP among cooks in intervention arms; no substantial overall health improvement, with continued traditional stove use
Aiello & Kaplan (2009)	Socioeconomic position; Methods	No	Review: SEP and inflammatory and immune CVD biomarkers; measurement and feasibility issues for population surveys
Aliberti et al. (2026)	Socioeconomic position; Health behaviour; Environment	No	Meta-analysis: epigenetic age acceleration associated with behavioural, environmental and social determinants; strongest for metabolic-inflammatory markers and environmental exposures
Aljohani et al. (2026)	Stress (ACEs)	Yes	Uganda cohort (n=100 youth, half with perinatal HIV): higher-ACE cluster had greater monocyte and T-cell activation and arterial-stiffness progression; neglect associated with stiffness progression in youth with HIV
Andersen (2022)	Stress (early adversity)	No	Review: abuse history associated with raised inflammatory markers; preclinical evidence on neuroinflammation and brain maturation
Angelo et al. (2026)	Socioeconomic position (community)	No	Scoping review (63 studies): community-level social determinants associated with sepsis incidence and mortality; heterogeneous methods
Antoni & Dhabhar (2019)	Stress	No	Review: in cancer, psychosocial stress associated with lower NK-cell cytotoxicity and inflammation; stress-management interventions improve some immune measures
Arregi et al. (2024)	Environment (traffic noise)	No	Review: road traffic noise linked to cortisol, inflammation (IL-12, hsCRP), oxidative stress and DNA methylation in human cohorts and animals
Asghar et al. (2022)	Socioeconomic position; Race and racism	No	Narrative review: MIS-C after COVID-19 disproportionately affected Black, Latino and lower-SES children; higher mortality in low-resource settings
Audet et al. (2014)	Stress; Social isolation	No	Review: social stressors are accompanied by raised circulating cytokines in humans and animals; social support may limit cytokine elevations; links to mood disorders
Baumer et al. (2023)	Socioeconomic position; Stress; Environment	No	Review: social determinants affect cardiometabolic risk via glucocorticoid resistance, inflammatory cytokines, immune-cell distribution and epigenetic regulation
Berens et al. (2017)	Stress (childhood adversity)	No	Review: biological embedding of childhood adversity via neuroendocrine, immune and epigenetic mechanisms; prospective cohort evidence
Beurel & Nemeroff (2024)	Stress (early adversity)	No	Review: early adversity primes inflammatory responses partly via microbiome maturation
Bladon et al. (2024)	Socioeconomic position; Race and racism	No	Rapid systematic review (50 studies): sepsis risk and outcomes associated with sociodemographic, race and community inequalities; mostly US
Blair & Raver (2012)	Stress (poverty-related adversity)	No	Review: poverty-related adversity shapes child stress physiology (cortisol reactivity); longitudinal low-income cohort evidence
Blank et al. (2026)	Health behaviour (food insecurity)	No	Narrative review (SANRA-guided): food insecurity linked to inflammatory and hormonal dysregulation and allostatic load
Burns et al. (2003)	Stress	No	Review: psychosocial stress negatively associated with antibody response to vaccination, most apparent for thymus-dependent vaccines and at longer follow-up
Buthelezi et al. (2024)	Environment (ambient PM2.5)	Yes	South African Highveld schoolchildren (n=706): lagged PM2.5 associated with raised FeNO (airway inflammation) and lower lung function
Cacioppo et al. (2011)	Social isolation	No	Review: perceived isolation associated with HPA activation and decreased inflammatory control and immunity (incl. glucocorticoid gene expression)
Chen et al. (2021)	Stress (childhood adversity)	No	Review: childhood adversity associated with raised inflammatory markers, latent herpesvirus reactivation, telomere shortening and epigenetic change
Christensen et al. (2024)	Environment; Stress; Socioeconomic position	Yes	Drakenstein birth cohort, South Africa (n=225): indoor pollutants (notably benzene) positively associated with infant inflammatory markers; profile of high indoor pollution, low SES and maternal depressive symptoms associated with higher inflammation
Christian et al. (2025)	Stress (structural racism); Health behaviour (sleep)	No	Review: structural racism, sleep deficiency and physiological stress (incl. stimulated cytokine production) in maternal health inequities among Black Americans
Coimbra et al. (2020)	Stress (discrimination)	No	Systematic review of 12 studies (all US): mixed results, weak evidence of a main association between discrimination and telomere length; possible interactions with depressive symptoms, acculturation and socioeconomic status
Colich et al. (2020)	Stress (threat, deprivation); Socioeconomic position	No	Meta-analysis (54 studies, n=116,010): threat-related adversity associated with accelerated cellular ageing (telomeres, DNA methylation age) and earlier puberty; deprivation and SES less so
Cooper et al. (2012)	Socioeconomic position; Environment	No	Review: in Latin America, non-atopic childhood asthma linked to poverty-related factors (hygiene, diet, obesity, psychosocial stress); early infections reduce atopy but not asthma
Cuevas & Cole (2025)	Stress (discrimination)	No	Review: discrimination linked to inflammation as pathway to psychiatric disparities; evidence mostly cross-sectional
Cuevas et al. (2020)	Stress (discrimination)	No	Scoping review: discrimination associated with higher systemic inflammation in several but not all studies; mainly cross-sectional US evidence
Cuevas et al. (2026)	Stress (discrimination)	No	Systematic review (32 studies): discrimination associated with telomere shortening, CTRA-type pro-inflammatory/antiviral transcription and immune-ageing cell shifts; mostly cross-sectional
DeCaro & Helfrecht (2022)	Methods	No	Review: field-friendly chronic stress biomarkers (cortisol, CRP, EBV antibodies, allostatic load) for complex ecological, including low-resource, contexts
Di Lenardo et al. (2020)	Environment (lead)	Yes	Venda, South Africa: low-level blood lead associated with higher risk of tetanus IgG below protective level (RR 1.88 per 10-fold increase), not measles
Dowd et al. (2009)	Socioeconomic position	No	Review: SES not consistently related to cortisol; lower SES more consistently related to flatter diurnal cortisol and higher allostatic load
Ennin et al. (2022)	Environment (wood dust, occupational)	Yes	Kumasi, Ghana: hardwood workers (n=86) had higher Th1 and Th2 cytokines than non-woodworkers (n=89)

Esch et al. (2018)	Stress	No	Review: psychological stress associated with shorter telomeres and higher oxidative stress; mind-body interventions discussed
Fanfan et al. (2024)	Socioeconomic position; migration	No	Scoping review: social determinants may affect immigrant health via microbiome shifts linked to immune and inflammatory disorders; limited evidence
Freilich (2023)	Social isolation	No	Scoping review: loneliness may become embedded via HPA disruption, gene-expression and methylation changes; evidence gaps noted
Galiatsatos et al. (2019)	Race and racism	No	Systematic review and meta-analysis (21 studies; 6 pooled): Black race not significantly associated with sepsis-related mortality versus White (OR 1.20, 95% CI 0.81 to 1.77); race alone cannot fully explain sepsis disparities
Ghantous et al. (2021)	Environment (dietary aflatoxin)	Yes	Gambia (n=244): early-life aflatoxin exposure associated with differential white-blood-cell DNA methylation at 24 months
Gough et al. (2020)	Environment (WASH)	Yes	Zimbabwe SHINE trial: WASH and feeding interventions had no meaningful effect on enteric dysfunction or systemic inflammation biomarkers (null)
Gouin et al. (2008)	Stress (caregiving)	No	Review: older caregivers show poorer vaccine responses, impaired latent virus control, raised inflammatory mediators and accelerated cellular ageing
Granieri et al. (2026)	Social isolation and support	No	Systematic review of 23 studies, mostly cross-sectional: loneliness associated with inflammatory markers and, through inflammation and health behaviours, with cardiovascular risk; findings for CRP inconsistent, with some null associations
Gupta et al. (2024)	Stress (childhood adversity)	No	Review: childhood adversity, inflammation and pubertal development of anhedonia; mostly small cross-sectional studies
Hailu et al. (2022)	Stress (discrimination)	No	MESA cohort: examined whether neighbourhood social cohesion moderated longitudinal association of discrimination with 10-year telomere change
Hawkley & Capitanio (2015)	Social isolation	No	Review: perceived isolation linked to impaired immunity, altered HPA activity and pro-inflammatory gene expression across the lifespan
Heikkilä et al. (2024)	Socioeconomic position	No	Genome-wide association study with Mendelian randomisation (925,649 individuals; FinnGen, Estonian Biobank, UK Biobank): genetic correlation of psoriasis with lower education; bidirectional causal relationship between psoriasis and lower education level; assessed from the medRxiv preprint version
Hernandez-Vargas et al. (2015)	Environment (dietary aflatoxin)	Yes	Gambia: maternal aflatoxin exposure in early pregnancy associated with infant white-blood-cell DNA methylation at 71 CpG sites
Holuka et al. (2020)	Socioeconomic position; Stress (early adversity)	No	Review: early adversity and low SES alter immune phenotype (e.g., CD8 T-cell senescence), plausibly affecting COVID-19 severity
Hong et al. (2021)	Stress (maternal); Race and racism	No	Review: maternal stress and social determinants of Black-White preterm birth disparity; epigenetic and gene-environment evidence
Igboanugo & Mielke (2023)	Stress (occupational)	No	Framework review: work-related psychosocial stress and allostatic load in firefighters; proposes AL as multisystem indicator
Johnson (2022)	Stress; Socioeconomic position	No	Perspective: stress from social and economic contexts linked via HPA and inflammatory pathways to health disparities; calls for multilevel interventions
Johnson et al. (2017)	Socioeconomic position; Methods	No	Systematic review (26 studies): allostatic load-SEP studies used inconsistent biomarker sets and scoring; most included an immune marker, fewer an HPA marker
Kaali et al. (2021)	Environment (household air pollution)	Yes	Ghana cookstove trial cohort: higher prenatal PM2.5 associated with shorter cord-blood telomere length; LPG arm had longer telomeres
Kataba et al. (2023)	Environment (lead)	Yes	Kabwe, Zambia: no overall association of blood lead with IL-8 or TNF-alpha; in women, higher lead associated with lower TNF-alpha
Kelly et al. (2021)	Socioeconomic position	Yes	Malawi PLWH starting ART (n=279): higher education and income associated with more activated CD8 T cells, lower SES with more intermediate monocytes; water-kiosk use associated with lower activation
Khalid et al. (2024)	Socioeconomic position; Stress (early adversity)	No	Review: early-life social adversity and low SES linked to epigenetic changes and oral disease
Kiely et al. (2022)	Race and racism; Socioeconomic position; Environment	No	Review: inflammation and immune-response differences among population groups (linked to environmental and chronic inflammatory exposures) contribute to cancer disparities
Kim et al. (2024)	Social isolation	No	Cited in the main text; see the relevant domain section
Kohler et al. (2013)	Socioeconomic position	Yes	Malawi MLSFH (about 1,500 adults): only weak associations of SES with biomarkers; schooling and wealth not associated with CRP; marriage weakly with lower CRP
Koss & Gunnar (2018)	Stress (early adversity; Socioeconomic position)	No	Review: early adversity and HPA axis; lower SES associated with flatter diurnal cortisol slopes; poverty linked to early elevation then blunting
Kuhlman et al. (2017)	Stress (childhood adversity)	No	Review: developmental PNE and PNI pathways from childhood adversity to elevated glucocorticoids and inflammation
Kuhlman et al. (2020)	Stress (early adversity)	No	Systematic review and meta-analysis: limited and heterogeneous evidence linking early adversity to circulating inflammation in children and adolescents
Latendresse (2009)	Stress (poverty, racism)	No	Review: chronic stressors including poverty and racism linked to preterm birth via HPA/CRH and inflammatory pathways
Lawrence et al. (2022)	Stress (discrimination)	No	Meta-analysis (25 articles): everyday discrimination associated with higher CRP (r=0.11, k=10) but not cortisol, IL-6 or telomere length
Lemus-Roldan et al. (2025)	Stress (early adversity)	No	Narrative review: early adversity associated with HPA activation, oxidative stress and inflammatory biomarkers predicting later brain health; positive childhood experiences may buffer
Li & Xia (2026)	Social isolation	No	Narrative review: social isolation and loneliness linked to subclinical atherosclerosis via HPA activation, inflammation and oxidative stress; mainly cross-sectional
Liang et al. (2025)	Stress; Social isolation; Socioeconomic position	No	Scoping review: social adversity (loneliness, low SES) linked to cognition via HPA, inflammatory and epigenetic mediators
Lin et al. (2021)	Environment (household air pollution)	Yes	Rural Senegal women (n=441) exposed to biomass smoke: dried-blood-spot MDA higher with secondhand smoke and associated with cardiopulmonary symptoms; CRP also measured
Liu et al. (2017)	Socioeconomic position (childhood)	No	Meta-analysis (15 studies, n=43,629): least advantaged childhood SES had 25% higher adult CRP; attenuated by adult BMI
Liu et al. (2022)	Environment (WASH)	Yes	Rural Malawi (iLiNS-DYAD): no associations between environmental exposures and enteric dysfunction biomarkers at 18 months (null)
Logue & Nemeroff (2025)	Stress (early adversity)	No	Review: early adversity associated with chronic inflammation implicated in later cognitive impairment; possible sex differences
Lunjani et al. (2024)	Environment (rural-urban exposures)	Yes	AmaXhosa children, South Africa: early-life exposures (animals, outdoor time) dominated immune-cell transcriptome over atopic dermatitis status

Makamure et al. (2016)	Environment (ambient SO2, NO)	Yes	Durban schoolchildren (n=129): TNF-alpha -308 A allele modified pollutant-associated lung function decrements; genotype not associated with cytokine levels
Mamuya et al. (2018)	Environment (occupational dust)	Yes	Kilimanjaro volcanic-rock miners: stone cutters had higher FeNO than unexposed; more cough and eye symptoms
Martinez et al. (2021)	Stress (structural racism); Environment	No	Review: structural racism drives asthma and atopic dermatitis disparities via segregation, SEP, environmental exposures and stress-related immune pathways
Martínez et al. (2022)	Stress (discrimination)	No	Review: case for biobehavioural research on physiological (HPA, inflammatory) effects of discrimination in Latinx populations
Mathur et al. (2022)	Stress (childhood adversity)	No	Conceptual review: early adversity linked to adult inflammation; emotion regulation proposed as pathway
Mattos dos Santos (2020)	Social isolation; Socioeconomic position	No	Mini-review: isolation, social stress and low SES may shift immune function towards pro-inflammatory, reduced antiviral responses (CTRA), relevant to COVID-19
Mavrych et al. (2025)	Social isolation	No	Review: social isolation and loneliness associated with cortisol elevation, glucocorticoid resistance and pro-inflammatory, reduced antiviral gene expression
McDermott et al. (2024)	Socioeconomic position; Race and racism	No	Review: chronic stress and allostatic load proposed to contribute to racial and socioeconomic glaucoma disparities via neuroinflammation
Miljevic et al. (2023)	Stress (childhood adversity)	No	Review: childhood abuse linked to cytokine alterations (e.g., raised CRP) in schizophrenia
Miller et al. (2021)	Stress (discrimination)	No	Integrative review (11 studies): 9 found discrimination positively associated with allostatic load; heterogeneous measures
Misra et al. (2018)	Environment (household fuel)	Yes	Rural South African women (n=415): little evidence that wood or other fuel use was associated with inflammatory biomarkers (null); imprecise higher odds of wheeze
Montoya & Uchino (2023)	Social support	No	Meta-analysis: social support not significantly related to telomere length (Zr=0.010)
Morakinyo & Mokgobu (2022)	Environment (indoor exposures)	Yes	Review (16 studies, n=16,920): indoor household exposures in South Africa associated with asthma, airway inflammation and poor early-life immune function
Morello-Frosch & Shenassa (2006)	Environment; Stress; Socioeconomic position	No	Review: place-based psychosocial stressors may compromise host resistance and amplify effects of environmental hazards, explaining maternal and child health disparities
Morris et al. (2019)	Socioeconomic position; Stress (ACEs)	No	Review: severe socioeconomic deprivation and ACEs associated with adult inflammation, HPA dysfunction, glucocorticoid-receptor epigenetics and oxidative stress
Muscatell et al. (2020)	Socioeconomic position	No	Meta-analysis: lower SES associated with higher CRP (Z=0.12) and IL-6 (Z=0.15); held for income and education; effect sizes varied by sample and analysis
Nazmi & Victora (2007)	Socioeconomic position; Race and racism	No	Systematic review (32 studies): 19 of 20 analyses found inverse SEP-CRP associations; most found higher CRP in Black, Hispanic and South Asian than White groups; adjustment attenuated
Nelson et al. (2022)	Race and racism; Socioeconomic position	No	Review: social determinants plausibly fuel prostate inflammation; Black-White outcome differences disappear with equal treatment
Nettis et al. (2019)	Stress (early adversity)	No	Review: early-life adversity associated with raised CRP, IL-6 and TNF-alpha in adulthood, linked to psychiatric-physical comorbidity
Nigatu et al. (2015)	Environment (occupational)	Yes	Ethiopia flower-farm workers (n=248): endotoxin exposure low; few workers had raised FeNO; greenhouse workers reported more nasal symptoms
Noren Hooten et al. (2022)	Race and racism; Socioeconomic position; Environment	No	Review: race and social determinants associated with accelerated ageing phenotype, oxidative stress and inflammation biomarkers
Noushad et al. (2021)	Methods	No	Systematic review of physiological biomarkers of chronic stress (cortisol, inflammatory and oxidative markers); measurement guidance
Nusslock & Miller (2016)	Stress (early adversity)	No	Review: neuroimmune network hypothesis; early adversity leads to chronic low-grade inflammation interacting with threat and reward circuitry
Olopade et al. (2017)	Environment (household air pollution)	Yes	Cited in the main text; see the relevant domain section
Olsson et al. (2024)	Environment (household air pollution)	Yes	Ethiopia mothers and children: solid fuel use not associated with nasopharyngeal inflammation (null); rural mothers had higher CXCL8
Olude et al. (2022)	Stress	Yes	Review: stressors common in Africa (poverty, conflict, infection) and glial neuroinflammation; African perspective on stress-related mental health
Oluwole et al. (2013)	Environment (household air pollution)	Yes	Cited in the main text; see the relevant domain section
Olvera Alvarez et al. (2018)	Stress; Environment	No	Integrative review: early-life stress and air pollution jointly linked to inflammation; social isolation in childhood predicted adult CRP; model of social-environmental adversity
Pipelias et al. (2026)	Stress (early adversity)	No	Narrative review: early life stress associated with HPA, immune and epigenetic alterations
Pourriyahi et al. (2021)	Social isolation	No	Review: loneliness associated with immunologic (inflammatory markers) and metabolic alterations; proposed as immunometabolic syndrome
Prahl et al. (2021)	Environment (pesticide spraying)	Yes	Uganda: in utero bendiocarb exposure after household spraying associated with fewer regulatory CD4 T cells, increased cytokine production, reduced antigen-driven proliferation and higher measles titres at one year
Priest et al. (2024)	Stress (racism)	No	Systematic review and meta-analysis of racism and health in children and youth (42 studies with physical or biomarker outcomes): minimal to moderate positive associations of racism with C-reactive protein, interleukin-6 and salivary cortisol; imprecise estimates for TNF-alpha; assessed from the medRxiv preprint version
Qaisiya et al. (2025)	Social isolation	No	Review: social isolation and loneliness linked to oxidative stress, inflammation and metabolic dysregulation
Rajaprakash et al. (2023)	Methods; Socioeconomic position	No	Review: DNA methylation signatures as biomarkers of socioeconomic position; EWAS and candidate-gene evidence
Rausch et al. (2026)	Socioeconomic position	No	Integrative review: low SES linked to stress, inflammation and an elevated leptin-to-adiponectin ratio
Reeder-Hayes et al. (2023)	Race and racism (structural racism)	No	Review: cyclical social and biological drivers of breast cancer disparities; structural racism linked to tumour biology via obesity, inflammation and methylation
Reid & Danese (2020)	Stress (early adversity)	No	Review: methodological challenges in linking early adversity to a pro-inflammatory phenotype and psychopathology
Ribeiro et al. (2018)	Socioeconomic position (neighbourhood)	No	Scoping review: most studies (12) found neighbourhood deprivation associated with higher allostatic load; mainly US cross-sectional
Ridout et al. (2018)	Stress (early adversity)	No	Meta-analysis: early adversity associated with shorter telomeres; effect varied by adversity type, timing and design

Rivillas-García et al. (2026)	Socioeconomic position (parental)	No	Meta-analysis (38 studies; 12 pooled, n=388,674): lower parental SEP associated with higher adult CRP and allostatic load; IL-6 estimate imprecise (CI included zero)
Roba et al. (2025)	Stress; Environment (water insecurity)	Yes	Daasanach pastoralists, northern Kenya, 2022 drought (n=128): fingernail cortisol and severe water insecurity each associated with higher CRP and greater odds of low-grade inflammation
Robertson et al. (2013)	Socioeconomic position	No	Meta-analysis: weak association of lower education with shorter telomeres (SMD 0.06), not robust to sensitivity analyses; no association with contemporaneous or childhood SES
Rook (2024)	Socioeconomic position	No	Review: low SES linked to reduced exposure to immunoregulatory microbiota and higher chronic inflammation
Rook et al. (2014)	Socioeconomic position; Environment	No	Review: low SES reduces exposure to immunoregulatory 'old friends' microbiota and green space, plausibly raising background inflammation
Ross et al. (2024)	Social isolation	No	Narrative review (18 studies, 15 cross-sectional): loneliness associated with greater IBD disease activity and symptoms
Roy et al. (2021)	Social support	No	Breast cancer chemotherapy cohort (n=50): social support buffered the association between stress and infection, but the immune markers measured did not explain it
Rylance et al. (2015)	Environment (household air pollution)	Yes	Malawi non-smokers (bronchoscopy): higher alveolar macrophage particulate loading associated with reduced oxidative burst and dampened CXCL8 responses
Salomon et al. (2020)	Methods	No	Scoping review: minimally invasive methods for HPA (cortisol, n=173 studies) and immune/inflammatory markers (n=118) in chronic stress research; timing and handling concerns
Saygideger et al. (2022)	Stress (childhood abuse)	No	Systematic review (36 studies): childhood abuse associated with epigenetic modification of asthma- and inflammation-related genes
Scatà et al. (2023)	Social isolation	No	Narrative review: social isolation (space missions, lockdown) linked to autonomic activation and inflammatory change
Smith et al. (2020)	Social isolation	No	Cited in the main text; see the relevant domain section
Szanton et al. (2005)	Stress and allostatic load (SES)	No	Systematic review of 11 allostatic load studies: allostatic load higher at lower socioeconomic status; IL-6 and CRP each included in only two of the 11 indices; justification for chosen markers varied
Tam et al. (2024)	Social isolation; Stress	No	Review: model in which social stress and social support shape biological ageing in marginalised populations through inflammation
Thomas et al. (2023)	Stress (threat, deprivation)	No	Systematic review of 74 articles on childhood deprivation and threat in the psychosis spectrum: findings inconsistent; threat associated with higher C-reactive protein and with both higher and lower cortisol measures; associations less evident in controls; assessed from the accepted manuscript
Thorpe et al. (2025)	Stress (racism); Socioeconomic position	No	Review: racism-driven inequities in social determinants manifest as neurohormonal, immune, inflammatory, oxidative and epigenetic responses
Tiekwe et al. (2021)	Environment (urban air pollution, occupational)	Yes	Douala and Dschang, Cameroon (1,182 active people recruited; haematology compared in 126 motorbike drivers in Douala and 65 in less polluted Dschang): monocytes, lymphocytes and CRP higher among Douala motorbike drivers
Tiekwe et al. (2024)	Environment (traffic particles, occupational)	Yes	Cameroon motorbike drivers (n=191): higher SOD and MDA in more polluted Douala, correlated with monocyte mobilisation
Toe et al. (2022)	Environment (ambient PM2.5)	Yes	Ugandan adolescents (n=119; 69 with HIV): PM2.5 correlated with intestinal permeability, monocyte activation and carotid IMT in those with HIV only
Tungu et al. (2013)	Environment (occupational dust)	Yes	Tanzanian cement workers: FeNO did not differ between dust-exposed workers and controls (null)
Uchino (2006)	Social support	No	Cited in the main text; see the relevant domain section
Uchino et al. (2020)	Social support	No	Meta-analysis: social support/integration related to higher vaccine antibody levels, but small effect with confidence interval including zero (Zr=0.06)
Valencia et al. (2022)	Race and racism; Stress	No	Review: calls for integrating biological pathways (inflammation, epigenetics, allostatic load) of social experience into cancer disparities research
Vargovic et al. (2026)	Stress (early adversity)	No	Review: early adversity alters HPA function, immune responses and gut microbiome
Webb et al. (2024)	Stress (racism)	No	Narrative review: racism-related stress associated with neuroendocrine and inflammatory markers and allostatic load in Black Americans
Yu et al. (2026)	Social isolation	No	Systematic review: loneliness and isolation linked to cognitive decline via inflammation, HPA function and gene expression (mixed) and psychobehavioural pathways
Zannas (2019)	Stress	No	Review: psychosocial stress accelerates epigenetic ageing; caregiver stress linked to telomere shortening
Zhou & Ryan (2023)	Stress (early adversity)	No	Review and scoping review: early adversity embedded via DNA methylation; human evidence for intergenerational epigenetic transmission limited

Note. ACE, adverse childhood experience; AL, allostatic load; ART, antiretroviral therapy; BMI, body mass index; CI, confidence interval; COVID-19, coronavirus disease 2019; CRP, C-reactive protein; CRH, corticotropin-releasing hormone; CTRA, conserved transcriptional response to adversity; CVD, cardiovascular disease; EBV, Epstein-Barr virus; EWAS, epigenome-wide association study; FeNO, fractional exhaled nitric oxide; HPA, hypothalamic-pituitary-adrenal; IBD, inflammatory bowel disease; IL, interleukin; iLiNS-DYAD, International Lipid-Based Nutrient Supplements DYAD trial; IMT, intima-media thickness; LPG, liquefied petroleum gas; MDA, malondialdehyde; MESA, Multi-Ethnic Study of Atherosclerosis; MIS-C, multisystem inflammatory syndrome in children; MLSFH, Malawi Longitudinal Study of Families and Health; NK, natural killer; NO, nitric oxide; OR, odds ratio; PLWH, people living with HIV; PM2.5, fine particulate matter; PNE, psychoneuroendocrine; PNI, psychoneuroimmune; PUFA, polyunsaturated fatty acids; RR, risk ratio; RCT, randomised controlled trial; SEP, socioeconomic position; SES, socioeconomic status; SHINE, Sanitation Hygiene Infant Nutrition Efficacy trial; SMD, standardised mean difference; SO2, sulphur dioxide; SOD, superoxide dismutase; SSA, sub-Saharan Africa; US, United States; WASH, water, sanitation and hygiene.

B. Records sought but not retrieved in full text (n = 115)

Priority	Study	Title	DOI
1	Author not recorded (2014)	Evaluation of IgG antibodies against respiratory syncytial virus (RSV), and associated risk factors for severe respiratory tract infections in pre-school children in North-central, Nigeria	None recorded
1	Anetor (1998)	Decreased immune status in Nigerian workers occupationally exposed to lead	None recorded
1	Folorunso (2023)	Heavy metals and inflammatory, oxidative/antioxidant and DNA damage biomarkers among people living with HIV/AIDS (PLWHA) in Niger Delta, Nigeria	10.1080/10934529.2023.2185004
1	Nsonwu-Anyanwu (2023)	Perturbations in indices of oxidative stress, oxidative DNA damage and lung function in chronic exposure to wood dust in Southern Nigeria	10.1080/08958378.2023.2224388
1	Nwanaforo (2026)	The toxic footprint of lead exposure: oxido-inflammatory biomarkers in urban populations of Niger Delta, Nigeria	10.1007/s10534-026-00864-0
1	Odewabi (2013)	Effect of exposure to solid wastes in relation to employment duration on some important markers of health and disease in waste management workers of Ogun State in southwest Nigeria	10.1177/0960327113488612
1	Odewabi (2013)	The levels of inflammatory markers and oxidative stress in individuals occupationally exposed to municipal solid waste in Ogun State, South West Nigeria	10.1177/0748233712445043
2	Author not recorded (2012)	Social Grants for People Living with HIV and on Antiretroviral Therapy in KwaZulu-Natal; South Africa: a Longitudinal Study	None recorded
2	Anderson (2018)	HIV induced nitric oxide and lipid peroxidation, influences neonatal birthweight in a South African population	10.1016/j.envint.2018.08.042
2	Everson (2020)	Personal exposure to NO2 and benzene in the Cape Town region of South Africa is associated with shorter leukocyte telomere length in women	10.1016/j.envres.2019.108993
2	Fusi Suh (2025)	Use of agrochemicals resulted in anemia and inflammation among farmers in Fako Division, Cameroon	10.1080/01480545.2024.2429629
2	Gettler (2024)	Refugee health and physiological profiles in transitional settlements in Serbia and Kenya: Comparative evidence for effects of gender and social support	10.1016/j.psyneuen.2024.107024
2	Nansook (2018)	IL-17A[G197G]-Association between NOx and gestational age in a South African birth cohort	10.1111/iji.12358
2	Nansook (2018)	IL-1β haplotype influences the effect of NOx exposure on gestational age in the South African MACE birth cohort	10.1177/0960327117728386
2	Ndlovu (2014)	Asthma associated with pesticide exposure among women in rural Western Cape of South Africa	10.1002/ajim.22384
2	Needham (2021)	Family socioeconomic status and child telomere length among the Samburu of Kenya	10.1016/j.socscimed.2021.114182
2	Olaniyan (2019)	Asthma-related outcomes associated with indoor air pollutants among schoolchildren from four informal settlements in two municipalities in the Western Cape Province of South Africa	10.1111/ina.12511
3	Ahlberg (2023)	Linking Sepsis with chronic arterial hypertension, diabetes mellitus, and socioeconomic factors in the United States: A scoping review	10.1016/j.jcrc.2023.154324
3	Almoosawy (2026)	The Association Between Socioeconomic Position and Mortality in Patients With Sepsis and Septic Shock-A Systematic Review and Meta-Analysis	10.1097/CCM.0000000000007053
3	Barthel (2025)	Habituation of the biological response to repeated psychosocial stress: a systematic review and meta-analysis	10.1016/j.neubiorev.2024.105996
3	Beckie (2012)	A systematic review of allostatic load, health, and health disparities	10.1177/1099800412455688
3	Brown (2018)	Loneliness and acute stress reactivity: A systematic review of psychophysiological studies	10.1111/psyp.13031
3	Cardenas (2025)	Is Discrimination Related to Markers of Systemic Inflammation? A Systematic Review and Meta-analysis	10.1097/PSY.0000000000001429
3	Castro-Quintas (2023)	Salivary secretory immunoglobulin A as a potential biomarker of psychosocial stress response during the first stages of life: A systematic review	10.1016/j.yfrne.2023.101083
3	Desjardins (2022)	Sexual minority health and allostatic load in the National Health and Nutrition Examination Survey: A systematic scoping review with intersectional implications	10.1016/j.psyneuen.2022.105916
3	Gamage (2025)	The early origins of socioeconomic inequalities in inflammation: a scoping review and recommendations for life course and longitudinal studies	10.1332/17579597Y2025D000000039
3	Green (2025)	Exploring intersectionality in psychoneuroendocrinology research: A systematic review	10.1016/j.psyneuen.2025.107536
3	Harb (2026)	Discrimination and allostatic load in black middle-aged and older adults: A systematic review and meta-analysis	10.1016/j.psyneuen.2025.107714
3	Jesuthasan (2025)	Childhood adversity as a risk factor for autoimmune disease: A systematic review and meta-analysis with implications for psychiatry	10.1016/j.bbi.2025.04.036
3	Johnson (2013)	Systematic review of the evidence of a relationship between chronic psychosocial stress and C-reactive protein	10.1007/s40291-013-0026-7

3	Maayan (2024)	Inflammatory mediation of the relationship between early adversity and major depressive disorder: A systematic review	10.1016/j.jpsychires.2023.11.025
3	Magan (2026)	Understanding allostatic load and the physiological impact of chronic stress in family caregivers: a scoping review	10.1080/13607863.2025.2570824
3	Milaniak (2019)	Childhood socioeconomic status and inflammation: A systematic review and meta-analysis	10.1016/j.bbi.2019.01.018
3	Misiak (2022)	Adverse childhood experiences and low socioeconomic status with respect to allostatic load in adulthood: A systematic review	10.1016/j.psyneuen.2021.105602
3	Murkey (2022)	Disparities in allostatic load, telomere length and chronic stress burden among African American adults: A systematic review	10.1016/j.psyneuen.2022.105730
3	Perez (2024)	Social determinants of inflammatory markers linking depression and type 2 diabetes among women: A scoping review	10.1016/j.jpsychores.2024.111831
3	Scalise (2025)	Biomarkers and Social Determinants in Atherosclerotic Arterial Diseases: A Scoping Review	10.1016/j.avsg.2024.12.076
3	Slopen (2012)	Childhood adversity and immune and inflammatory biomarkers associated with cardiovascular risk in youth: a systematic review	10.1016/j.bbi.2011.11.003
3	Thacker (2024)	Antibiotics, passive smoking, high socioeconomic status and sweetened foods contribute to the risk of paediatric inflammatory bowel disease: A systematic review with meta-analysis	10.1002/jpn3.12303
3	Uchino (2018)	Social support, social integration, and inflammatory cytokines: A meta-analysis	10.1037/hea0000594
3	Yuan (2024)	Associations of Socioeconomic Factors and Unhealthy Lifestyles with Allostatic Load: A Meta-analysis	10.1007/s12529-023-10235-5
4	Adams (2007)	No association between socio-economic status and white blood cell telomere length	10.1111/j.1474-9726.2006.00258.x
4	Anyane-Yeboa (2022)	The Impact of the Social Determinants of Health on Disparities in Inflammatory Bowel Disease	10.1016/j.cgh.2022.03.011
4	Aubert (2008)	Psychosocial stress, emotions and cytokine-related disorders	10.2174/187221308784543647
4	Avitsur (2015)	Early adversity, immunity and infectious disease	10.3109/10253890.2015.1017464
4	Berger (2015)	"More than skin deep": stress neurobiology and mental health consequences of racial discrimination	10.3109/10253890.2014.989204
4	Blanco (2025)	Loneliness and social isolation in people with HIV	10.1097/COH.0000000000000912
4	Briones (2007)	Psychoneuroimmunology and related mechanisms in understanding health disparities in vulnerable populations	None recorded
4	C (2022)	Neighborhoods, Racism, Stress, and Preterm Birth Among African American Women: A Review	10.1177/01939459211041165
4	Carlson (2005)	Allostatic load and health disparities: a theoretical orientation	10.1002/nur.20084
4	Carter (2022)	Approaching Mental Health Equity in Neuroscience for Black Women Across the Lifespan: Biological Embedding of Racism From Black Feminist Conceptual Frameworks	10.1016/j.bpsc.2022.08.007
4	Cavigelli (2012)	Social status, glucocorticoids, immune function, and health: can animal studies help us understand human socioeconomic-status-related health disparities?	10.1016/j.yhbeh.2012.07.006
4	Chiang (2015)	Early adversity, neural development, and inflammation	10.1002/dev.21329
4	Costa (2026)	Bridging Biology and Society: Molecular Insights into Social Determinants of Vascular Disease	10.1016/j.avsg.2025.08.006
4	Destin (2019)	Socioeconomic mobility, identity, and health: Experiences that influence immunology and implications for intervention	10.1037/amp0000297
4	Diamond (2021)	Systemic inflammation as a driver of health disparities among sexually-diverse and gender-diverse individuals	10.1016/j.psyneuen.2021.105215
4	Doan (2021)	Allostatic load: Developmental and conceptual considerations in a multi-system physiological indicator of chronic stress exposure	10.1002/dev.22107
4	Duah (2026)	Metals exposures and telomere length as a marker of DNA damage among electronic waste workers and a reference group	10.1007/s11356-026-37582-6
4	Ehlert (2013)	Enduring psychobiological effects of childhood adversity	10.1016/j.psyneuen.2013.06.007
4	Elwenspoek (2017)	The effects of early life adversity on the immune system	10.1016/j.psyneuen.2017.05.012
4	Emeny (2021)	Health disparities: Intracellular consequences of social determinants of health	10.1016/j.taap.2021.115444
4	Goldmeier (2008)	Does chronic stress lead to increased rates of recurrences of genital herpes--a review of the psychoneuroimmunological evidence?	10.1258/ijsa.2007.007304
4	Gomaa (2019)	The Biology of Social Adversity Applied to Oral Health	10.1177/0022034519876559
4	Gupta (2024)	Inflammation as a shared mechanism of chronic stress related disorders with potential novel therapeutic targets	10.1007/s00210-024-03205-5
4	Helget (2024)	Health disparities in gout	10.1097/BOR.0000000000000996
4	Hlongwa (2026)	The physiology of poverty: gut microbiome and pathways to child stunting	10.1098/rstb.2025.0034
4	Hogan (2002)	Social support interventions: do they work?	10.1016/s0272-7358(01)00102-7
4	Hunter (2013)	Does psychosocial stress play a role in the exacerbation of psoriasis?	10.1111/bjd.12478

4	Hänsel (2010)	Inflammation as a psychophysiological biomarker in chronic psychosocial stress	10.1016/j.neubiorev.2009.12.012
4	Jain (2025)	Food Insecurity in Hidradenitis Suppurativa: A Cross-Sectional Analysis from the Population-Based All of Us Research Program	10.1159/000546500
4	Jassal (2015)	Pediatric asthma and ambient pollutant levels in industrializing nations	10.1093/inthealth/ihu081
4	Jensen (2017)	Effects of poverty on interacting biological systems underlying child development	10.1016/s2352-4642(17)30024-x
4	Jia (2025)	Chronic stress, gut microbiota, and immunity: interconnections and implications for health	10.1007/s11010-025-05376-y
4	Juster (2010)	Allostatic load biomarkers of chronic stress and impact on health and cognition	10.1016/j.neubiorev.2009.10.002
4	Jylhä (2026)	Socio-economic influences from egg to exit: Emerging biology	10.1016/j.arr.2026.103104
4	Kam (2023)	The impact of climate change on atopic dermatitis and mental health comorbidities: a review of the literature and examination of intersectionality	10.1111/ijd.16557
4	Katz (2012)	The cost of chronic stress in childhood: understanding and applying the concept of allostatic load	10.1521/pdps.2012.40.3.469
4	Kort (1994)	The effect of chronic stress on the immune response	10.1016/s0960-5428(06)80186-5
4	Kraft (2021)	Explaining socioeconomic disparities in health behaviours: A review of biopsychological pathways involving stress and inflammation	10.1016/j.neubiorev.2021.05.019
4	Krause (2020)	Epigenetic mechanisms activated by childhood adversity	10.2217/epi-2020-0042
4	Kulkarni (2006)	Non-invasive assessment of pollutant particles in the lower airway of children and adults	None recorded
4	Langer-Gould (2025)	Embodiment of structural racism and multiple sclerosis risk and outcomes in the USA	10.1038/s41582-025-01096-5
4	Leonel Javeres (2020)	Analysis of PON1 gene polymorphisms (rs662 and rs854560) and inflammatory markers in organophosphate pesticides exposed cohorts from two distinct populations	10.1016/j.envres.2020.110210
4	Lisco (2024)	Chronic Stress as a Risk Factor for Type 2 Diabetes: Endocrine, Metabolic, and Immune Implications	10.2174/1871530323666230803095118
4	Mak (2026)	Influence of Socioeconomic Development on Incidence and Disease Phenotype in Inflammatory Bowel Disease: A Population-Based Incident Study Across 16 Regions	10.1016/j.cgh.2026.08.031
4	Marwaha (2025)	Exploring the complex relationship between psychosocial stress and the gut microbiome: implications for inflammation and immune modulation	10.1152/japplphysiol.00652.2024
4	Mengelkoch (2020)	Early life disadvantage, phenotypic programming, and health disparities	10.1016/j.copsyc.2019.06.014
4	Minejima (2021)	Impact of Socioeconomic Status and Race on Sepsis Epidemiology and Outcomes	10.1093/jalm/jfaa151
4	Moen (2012)	Respiratory inflammation among workers exposed to airborne dust with endotoxins in a coffee curing factory	10.1097/JOM.0b013e318250ca24
4	Naidoo (2019)	Maternal miRNA-146a G/C rs2910164 variation, HIV/AIDS and nitrogen oxide pollution exposure collectively affects foetal growth	10.1177/0960327118781902
4	Nelson (2017)	Hazards to Early Development: The Biological Embedding of Early Life Adversity	10.1016/j.neuron.2017.09.027
4	Olaniyan (2020)	The association between ambient NO2 and PM2.5 with the respiratory health of school children residing in informal settlements: A prospective cohort study	10.1016/j.envres.2020.109606
4	Palumbo (2020)	Chronic Stress and Glucocorticoid Receptor Resistance in Asthma	10.1016/j.clinthera.2020.03.002
4	Powell (2013)	Psychosocial stress and inflammation in cancer	10.1016/j.bbi.2012.06.015
4	Quadt (2020)	Brain-body interactions underlying the association of loneliness with mental and physical health	10.1016/j.neubiorev.2020.06.015
4	Rajasekera (2021)	Maternal Exposure to Adversity: Impact on the Microbiota-Gut-Brain Axis, Inflammation and Offspring Psychiatric Outcomes	10.1159/000510415
4	Rakers (2017)	Transfer of maternal psychosocial stress to the fetus	10.1016/j.neubiorev.2017.02.019
4	Rohleder (2012)	Acute and chronic stress induced changes in sensitivity of peripheral inflammatory pathways to the signals of multiple stress systems --2011 Curt Richter Award Winner	10.1016/j.psyneuen.2011.12.015
4	Rohleder (2014)	Stimulation of systemic low-grade inflammation by psychosocial stress	10.1097/PSY.0000000000000049
4	Rohleder (2019)	Stress and inflammation - The need to address the gap in the transition between acute and chronic stress effects	10.1016/j.psyneuen.2019.02.021
4	Sarkar (2018)	Atopic dermatitis in Indian children: The influence of lower socioeconomic status	10.1016/j.clindermatol.2018.05.009
4	Seeman (2010)	Socio-economic differentials in peripheral biology: cumulative allostatic load	10.1111/j.1749-6632.2009.05341.x
4	Silverberg (2024)	Atopic Dermatitis: The Role of the Social Determinants of Health on Severity and Access to Care	10.1016/j.det.2024.06.003
4	Sun (2021)	Psychosocial Stress and Mechanisms of Skin Health: A Comprehensive Update	10.36849/JDD.5608
4	Swaby (2023)	Lifestyle and host determinants of antitumor immunity and cancer health disparities	10.1016/j.trecan.2023.08.007

4	Tippairote (2026)	Chronic stress and the mitochondria-telomere axis: human evidence for a bioenergetic-debt model of early aging	10.1007/s10522-025-10377-x
4	Underhill (2026)	Personal household air pollution exposure and SARS-CoV-2 antibody responses among women and infants: an analysis within the HAPIN trial	10.1016/j.envint.2026.110180
4	Vargason (2016)	Socioeconomic Disparities in the Presentation and Treatment of Graves' Disease and Thyroid Eye Disease	10.1080/08820538.2016.1185322
4	Voth-Gaeddert (2020)	Assessment of environmental exposure factors on child diarrhea and systemic inflammation in the Eastern Cape	10.1016/j.watres.2019.115244
4	Waisberg (2011)	Adipokine serum concentrations, anthropometric measurements and socio-economic status in two ethnic groups with different prevalence levels for cardiovascular diseases and type 2 diabetes	10.1055/s-0031-1283139
4	Warren (2025)	Loneliness as a driver of allostatic load: mechanisms linking social disconnection to physiological dysregulation and health disparities	10.1080/10253890.2025.2594067
4	Wilkinson (2011)	Childhood adversity and allostatic overload of the hypothalamic-pituitary-adrenal axis: a vulnerability model for depressive disorders	10.1017/S0954579411000472
4	Wise (2016)	Child Poverty and the Promise of Human Capacity: Childhood as a Foundation for Healthy Aging	10.1016/j.acap.2016.01.014
4	Zhang (2025)	Social Determinants of Health in Uveitis: A Literature Review	10.1080/09273948.2025.2465774
4	Zhang (2026)	Chronic stress at the crossroads: Decoding the HPA-SAM-immune-gut axis in inflammatory bowel disease pathogenesis and therapeutics	10.1016/j.smim.2026.102038