

Social Determinants of Health Beyond Risk Factors: Revisiting the Mechanisms Connecting Social Conditions, Behavior, and Health Outcomes

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Abstract

Social determinants of health are frequently represented as sets of exposures associated with disease, healthcare use, or mortality. Although useful for surveillance and risk stratification, this representation can obscure the processes through which social conditions become consequential for health. This Critical Review examines social determinants as components of interconnected material, psychosocial, relational, behavioral, healthcare, institutional, and temporal mechanisms rather than as independent risk factors. Evidence is interpreted with explicit separation between observed association, mechanism-compatible findings, causal inference, and the synthesis proposed by this review. Material resources alter feasible opportunities as well as perceived scarcity; chronic threat may become biologically embedded but does not operate through a uniform biomarker pathway; social relationships can buffer adversity while coexisting with structural disadvantage; institutions distribute resources, constraints, and power; and behavior can function simultaneously as mediator, adaptation, and outcome. Healthcare systems further modify how social conditions translate into access, treatment trajectories, and clinical consequences. The review argues that meaningful explanation requires attention to scale, timing, feedback, competing mechanisms, and unequal capacity to exercise agency. This mechanistic reframing provides a basis for more discriminating intervention design, measurement, and causal research without treating the proposed framework as a validated universal model.

Keywords: Social determinants of health, Health inequalities, Causal mechanisms, Structural determinants, Health behavior, Health equity

Introduction

Social determinants of health have become central to explanations of patterned differences in morbidity, mortality, healthcare access, and patient experience. Yet their increasing visibility has also encouraged a simplifying habit: housing, income, education, discrimination, social support, neighborhood context,

and healthcare access are often entered into analyses as parallel variables and interpreted as if each represented an independent “risk factor.” Such approaches can identify where unequal outcomes cluster, but they are less successful at explaining how a social condition changes what people are exposed to, what options are realistically available, which psychological or behavioral responses become adaptive, or how institutions amplify or attenuate those consequences. Mechanism-oriented scholarship has therefore argued that SDOH research must move beyond domain inventories toward processes linking conditions to outcomes [1].

A second limitation is that structural conditions can disappear analytically when they are translated into individual-level variables. Structural determinants are

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not simply more distal versions of personal social characteristics. They concern rules, institutional arrangements, political decisions, and distributions of power that shape who receives resources, protection, exposure, or constraint [2]. Treating these conditions only as characteristics possessed by individuals risks locating explanation downstream of the processes that produced the unequal distribution in the first place.

This review therefore asks a different question from conventional risk-factor cataloging: through what mechanisms do social conditions become behaviorally, psychologically, biologically, clinically, and institutionally consequential? The aim is not to replace one list of determinants with another. It is to distinguish exposure conditions from operating mechanisms, mechanisms from correlates, behavior from unconstrained choice, healthcare access from treatment response, and structural constraint from deterministic accounts that erase agency. The resulting synthesis is explicitly proposed as an organizing framework. Its components draw on empirical and conceptual evidence, but the complete architecture has not itself been prospectively validated.

Review scope and critical approach

This Critical Review examines evidence relevant to the pathways connecting social conditions, behavior, and health outcomes across individual, relational, community, healthcare, institutional, and policy levels. Evidence was selected for its capacity to clarify mechanisms, causal assumptions, contradictions, boundary conditions, or intervention implications rather than to produce an exhaustive inventory of all studies labeled as SDOH research. The approach therefore does not claim systematic-review comprehensiveness or pooled-effect estimation.

A mechanistic review also requires restraint about causal language. Describing health differences by income, neighborhood, discrimination exposure, or other social variables already raises causal questions, but descriptive adjustment alone does not identify the effect of changing those conditions. Confounding, selection processes, exposure definition, temporal ordering, positivity, and the specification of a meaningful intervention or comparator all affect interpretation. Translating substantive theories into explicit causal structures can clarify what must be observed and what assumptions are required before an association is interpreted as intervention-relevant evidence [3-5].

Accordingly, the review uses four evidential distinctions. Association indicates an observed statistical relationship. Mechanism-compatible evidence indicates findings consistent with a proposed process but insufficient to identify it causally. Causal evidence is reserved for designs and assumptions capable of supporting the relevant causal contrast. Proposed synthesis refers to the framework developed here by integrating evidence across levels. These categories are deliberately non-equivalent: a variable that improves prediction need not explain why an outcome occurs, and a plausible biological or behavioral mediator cannot be assumed simply because an exposure and outcome are associated.

The limitations of risk-factor thinking

Risk-factor thinking becomes problematic when analytically different social conditions are treated as interchangeable predictors. Social determinants are commonly correlated with one another because they arise from shared institutions, cumulative histories, or linked opportunity structures. Data-informed pathway analysis across US census tracts, for example, identified extensive dependency among economic, educational, healthcare, and technological conditions rather than a set of isolated exposures [6]. Such analyses cannot establish individual causal effects, but they illustrate why adding social variables independently to a regression model can misrepresent the systems that generate them.

Four failures follow from excessive reliance on the independent-risk-factor model. The first is atomization: income, housing, neighborhood conditions, discrimination, and healthcare access are separated even when one changes the probability or meaning of another. The second is level collapse, in which structural or community conditions are treated as if they were individual attributes. The third is temporal flattening: current exposure is used to represent trajectories that may differ according to onset, persistence, cumulative duration, or developmental timing. The fourth is prediction–explanation substitution, whereby improved risk discrimination is interpreted as evidence that the included social variable identifies the mechanism producing the outcome.

This review proposes that risk factors remain useful descriptors when the scientific question is prediction or surveillance, but mechanism-oriented research requires an additional layer: what process connects the condition to a health-relevant change, under which contextual conditions, and with what plausible competing

explanation? The same social variable may participate in several mechanisms simultaneously and may have different implications across populations or historical settings.

From social domains to causal mechanisms

Moving from domains to mechanisms requires decomposing broad labels into the processes they make possible. Financial circumstances illustrate the distinction. Perceived financial scarcity predicts poorer health beyond objective income and may operate partly through reduced perceived mastery, while adverse events may also feed back into later scarcity [7]. “Economic stability” therefore cannot be represented solely as household income; material availability, perceived insufficiency, control, exposure to shocks, and behavioral adaptation are analytically distinct.

Place-based evidence makes the same point. Longitudinal neighborhood histories suggest that accumulated exposure to economic inequality is associated with mortality in ways that differ from a single contemporaneous neighborhood measure [8]. Likewise, decomposition of neighborhood health inequalities in

northern Sweden indicated that adult differences reflected socioeconomic and psychosocial conditions developing from adolescence through later life [9]. A neighborhood variable can therefore signify opportunity, stress exposure, social composition, environmental constraint, or accumulated trajectory depending on the causal question.

The proposed mechanism-discrimination approach used here separates six questions: what condition is being experienced; what process could transmit its effect; what contextual feature modifies that process; what competing explanation remains plausible; where the pathway may fail or reverse; and what consequence follows for measurement or intervention. This structure does not claim that each pathway is established. Its purpose is to prevent social categories from being mistaken for mechanisms.

Because these distinctions are difficult to recover when domains are discussed sequentially in prose, **Table 1** separates recurring evidence patterns according to the mechanism, modifier, competing interpretation, and inference boundary that must remain visible.

Table 1. Distinguishing social conditions from the mechanisms through which they may become health-relevant

Mechanism requiring separate identification	Contextual modifier that can change the pathway	Competing explanation or uncertainty	Boundary or exception	Consequence for interpretation
Social-condition pattern — Financial insufficiency or insecurity				
Reduced material feasibility, perceived scarcity, diminished control, or adaptation to resource volatility	Household obligations, life stage, local costs, available services	Poor health may itself reduce income or increase expenditure	Equal income does not imply equal disposable resources or security	Measure resources, perceived scarcity, timing, and feedback separately
Social-condition pattern — Neighborhood disadvantage or inequality				
Differential opportunity, environmental exposure, institutional access, chronic threat, or social comparison	Residential history, mobility, segregation, age, policy environment	Selection into neighborhoods and unmeasured contextual factors	A current address may poorly represent accumulated exposure	Use longitudinal place histories where the hypothesized mechanism is cumulative
Social-condition pattern — Housing difficulty				
Financial strain, environmental exposure, instability, crowding, or restricted recovery conditions	Tenure, housing market, household composition, age	Housing problems may proxy broader deprivation	Homeownership alone does not establish housing quality or security	Match the housing measure to the mechanism being claimed
Social-condition pattern — Discrimination or social threat				
Psychological threat, anticipatory vigilance, behavioral adaptation,	Frequency, chronicity, coping resources, social support, racialized context	Biomarker associations may be absent or exposure measurement incomplete	Group membership cannot substitute for measured discrimination	Measure the experienced process rather than infer it from demographic status

**relational disruption, or
biological stress processes**

Social-condition pattern — Social cohesion or connectedness				
Support, information exchange, normative influence, trust, collective efficacy, or buffering	Type of relationship, rurality, cultural context, outcome	Cohesion may coexist with structural disadvantage or restrictive norms	High cohesion does not guarantee healthcare access or healthy behavior	Disaggregate relational dimensions and evaluate what they permit materially
Social-condition pattern — Structural policy condition				
Redistribution of resources, rights, exposures, institutional practices, or agentic burden	Jurisdiction, implementation, political power, historical moment	Policy adoption may differ from implementation and population reach	“Upstream” location does not guarantee beneficial or equitable effects	Specify the institutional mechanism and who must act for benefit to occur

Note: The classifications in this table are a proposed synthesis derived from the reviewed evidence. They are not a validated causal taxonomy.

Material resources and opportunity structures

Material determinants operate by changing what actions are feasible, sustainable, or costly. This is broader than socioeconomic ranking. Housing analyses among older US adults, for example, indicate that housing problems and financial strain can remain associated with cardiometabolic risk beyond conventional socioeconomic indicators [10]. The relevant mechanism may involve direct environmental exposure, financial pressure, residential instability, restricted capacity to recover from illness, or combinations of these processes. A broad category such as “low socioeconomic status” cannot discriminate among them.

The concept of opportunity structures is therefore useful if it is defined carefully. In this review, it refers to the material and institutional configuration of actions that are realistically available—not merely the choices an individual reports preferring. Transportation, paid leave, stable housing, food availability, childcare, insurance, safe environments, digital connectivity, and service distribution can determine whether a recommended behavior is practicable. Such conditions should not be treated as background covariates when the proposed mechanism depends on them.

This framing also avoids a common interpretive error: equating the existence of an option with equal capacity to use it. Two patients offered the same clinical recommendation may face radically different time costs, financial exposure, caregiving obligations, or neighborhood resources. Material disadvantage can therefore influence health directly and also alter the effort required to translate knowledge or motivation into behavior.

Stress, threat, and biological embedding

Psychosocial and structural adversity may influence health partly through sustained threat responses, but “biological embedding” should not be treated as a universal bridge that converts every social exposure into disease. US studies examining socioeconomic position, racial discrimination, and inflammatory markers demonstrate both supporting and limiting evidence. Some analyses identify contributions of educational disadvantage or discrimination to racialized inflammatory differences, while others show biomarker-specific, attenuated, or inconsistent relationships [11-13]. The relevant inference is therefore conditional: chronic social threat can be biologically consequential, but neither exposure nor group membership establishes one fixed inflammatory pathway.

Recent molecular work reinforces that caution. Associations between psychosocial stress, poverty, circulating mitochondrial DNA, extracellular-vesicle mitochondrial DNA, and inflammatory proteins varied across demographic and contextual combinations [14]. Such findings are compatible with complex stress biology but do not justify a single deterministic sequence from social adversity to molecular change to disease.

Null findings are especially important. In South Asian adults in the MASALA Study, discrimination was associated with poorer self-rated health, yet it was not associated with the inflammatory markers examined; coping style modified some relationships [15]. A psychologically consequential exposure can therefore relate to health without operating through the biomarker selected by the investigator. Biological embedding should be understood as a family of potentially interacting pathways whose relevance depends on exposure timing, physiology, coping, social context, measurement, and outcome. This distinction protects

mechanistic research from converting plausible biology into assumed mediation.

Social relations, norms, and network processes

Relationships can transmit resources, information, norms, expectations, recognition, and practical support. They may also amplify surveillance, exclusion, obligation, or exposure to unhealthy norms. A systematic review of racism, discrimination, social connectedness, and health found that connectedness often buffered or mediated adverse associations, while also emphasizing heterogeneity in constructs, designs, and measurement [16]. “Social support,” “cohesion,” and “connectedness” should consequently not be treated as interchangeable protective variables.

Context can even produce apparently paradoxical patterns. Among older adults in the mid-Atlantic United States, rural respondents reported greater neighborhood social cohesion yet poorer healthcare access and health status than urban respondents; cohesion was associated with selected outcomes but did not remove rural structural disadvantages [17]. Similarly, dimensions of neighborhood cohesion and trust showed different relationships with post-pandemic vaccination intentions [18]. Relational conditions matter partly because of what relationships enable, normalize, or constrain—not because cohesion has a uniform health effect.

Other evidence shows clustering among loneliness, stress, discrimination, support, and diabetes-related risk among young adults, while leaving temporal direction unresolved [19]. Prospective research among older adults also suggests that neighborhood cohesion predicts some preventive services, such as influenza vaccination and cholesterol testing, but not all preventive behaviors [20]. These differences caution against assigning one pathway to “social connection.” The same relational environment may influence information flow, emotional regulation, help-seeking, normative behavior, or healthcare navigation differently across outcomes.

Institutions, power, and structural constraint

A mechanistic account of social determinants becomes incomplete if institutions are treated as distant contextual scenery. Institutions determine eligibility, rights, resource allocation, labor conditions, taxation, healthcare financing, neighborhood investment, education, and exposure to sanctions or protections. Critical work on structural determinants therefore argues that agents and power relations must be made visible rather than

absorbed into depoliticized social-domain frameworks [21].

Policy processes are also reciprocal rather than mechanically upstream. Analysis of Child Tax Credit policymaking illustrates how actors, institutional authority, competing interests, temporal context, and structural arrangements influence one another during policy formation [22]. Structural determinants are consequently produced and reproduced through decisions; they are not static external exposures.

This distinction matters for behavior-oriented health programs. Social and behavior change initiatives can inadvertently locate problems within individuals when the behavior being targeted is constrained by resource distribution, institutional rules, or political conditions [23]. Conversely, labeling an intervention “structural” does not establish that it improves equity. A systematic review of structural interventions affecting racial inequities reported heterogeneous outcomes, with some socioeconomic and opportunity-related policies improving population health while restrictive policies could worsen inequity [24]. The proposed framework therefore represents structural constraint as a process that changes the distribution of exposure, resources, feasible actions, and institutional responsiveness. Its health consequences remain contingent on policy content, implementation, population reach, and power.

Behavior as mediator, adaptation, and outcome

Health behavior is often positioned downstream of social conditions, but that placement can imply more autonomy than the evidence warrants. Behavior may be a mediator when a social exposure alters smoking, diet, activity, care-seeking, medication use, or another health-relevant action. It may be an adaptation when an action is a reasonable response to scarcity, threat, unstable employment, unsafe environments, or competing obligations. It may also be an outcome when participation, avoidance, disclosure, or service use is itself the phenomenon requiring explanation.

Policy-oriented resilience research illustrates how resources and services, reductions in stress exposure, and adaptive psychological or interpersonal processes may connect policy conditions to subsequent behavior and well-being [25]. Yet intervention research rarely tests such contextual moderation adequately. A review of health-behavior intervention syntheses found that social determinants were incompletely and often atheoretically examined as moderators, with many tested effects null or

heterogeneous [26]. It is therefore unsafe to assume that identical motivational or educational interventions operate similarly across materially different contexts. Cultural and psychological variation reinforces the point. Evidence from Hispanic/Latino cardiovascular-health research indicates broadly adverse associations for several stress and psychological conditions but more variable findings for interpersonal and cultural factors [27]. Behavior should thus not be interpreted as a transparent measure of motivation, compliance, or preference. The same observable action may arise from different combinations of beliefs, opportunity, threat, competing demands, norms, and healthcare design. The mechanism depends on why the behavior occurs, not merely that it was observed.

Healthcare access and treatment response

Healthcare is frequently represented as one SDOH domain alongside housing, education, or economic stability. Mechanistically, however, healthcare also functions as a transmission and modification layer through which other social conditions acquire clinical consequences. Insurance, transportation, continuity, communication, appointment flexibility, institutional trust, caregiving support, digital access, and treatment affordability can shape entry into care, persistence in care, and the capacity to act on clinical recommendations. Prediction studies demonstrate why this layer must be interpreted carefully. In US adults, cumulative SDOH burden modified relationships between PREVENT cardiovascular-risk estimates and mortality and added modest mortality discrimination beyond the clinical model [28]. That finding establishes prognostic relevance; it does not identify which social mechanism produced mortality differences or imply that adding a social-risk score corrects the underlying disadvantage. Prediction and explanation remain separate tasks.

Transitions of care provide a clinically concrete example. Insurance, income, social support, and neighborhood disadvantage can identify patients at increased risk during transitions between settings, when medication access, follow-up, transport, caregiving, and information continuity become especially important [29]. Similar processes extend into specialty care: surgical disparities can arise across access, treatment, recovery, and post-discharge pathways rather than appearing only as preoperative “social risk” [30].

For this review, healthcare access is therefore distinguished from utilization, experience, treatment

response, and clinical outcome. A patient may obtain an appointment yet lack continuity, understand a recommendation yet be unable to implement it, initiate treatment yet face unstable follow-up, or receive technically equivalent care within markedly unequal social circumstances. Healthcare can attenuate social disadvantage when services reduce practical barriers or adapt to unequal needs; it can also reproduce disadvantage when successful participation requires resources that are unevenly distributed. Mechanistic analysis must specify which part of the care pathway is affected and whether the hypothesized process lies in access, interaction, treatment feasibility, continuity, or downstream response.

Life-course accumulation and feedback

Social conditions acquire meaning through duration, timing, and sequence. Long-running ARIC evidence illustrates how individual and neighborhood circumstances measured across the life course relate to cardiovascular, renal, cognitive, and mortality outcomes [31]. A contemporaneous measure can therefore underestimate pathways generated by earlier exposures or persistent disadvantage.

Persistence also matters. In a prospective Black American cohort, prolonged poverty predicted mortality differently from transient early poverty, while education and social integration did not confer identical protection across earlier socioeconomic trajectories [32]. Longitudinal pain research similarly indicates that childhood adversity, discrimination, poverty, and later social circumstances can combine rather than operate as isolated exposures [33].

The review therefore proposes that life-course mechanism models distinguish timing, persistence, accumulation, and feedback. Feedback is particularly important because illness can subsequently alter employment, income, social participation, caregiving requirements, and healthcare dependence, thereby changing the social conditions initially treated as exposures.

Reconciling structure and agency

Structural explanation should not imply that individuals lack agency, but neither should observed behavior be interpreted as evidence of equally exercisable choice. Agency depends on whether an action is materially feasible, institutionally permitted, socially supported, and sustainable under competing demands.

Intervention evidence helps expose this distinction. An umbrella review of equity-oriented policies found that interventions demanding less individual agency produced the most consistently favorable equity patterns, whereas higher-agentic interventions could widen inequalities unless adapted to unequal circumstances [34]. This does not establish a universal hierarchy of intervention effectiveness. It indicates that the burden placed on individuals is itself a mechanistic feature of intervention design.

This review therefore proposes exercisable agency as the capacity to translate intention into action under actual

social conditions. Structure changes that capacity by distributing resources, constraints, exposure, and institutional responsiveness. Agency, in turn, can modify trajectories through adaptation, resistance, help-seeking, social coordination, or disengagement. The relationship is reciprocal but asymmetrical: individuals act within conditions they did not equally choose and cannot equally alter.

The practical value of this distinction is clearer when patient or community states are compared according to what action is realistically possible rather than according to motivation alone (**Table 2**).

Table 2. Locating exercisable agency within unequal opportunity and intervention demands

Constraint shaping feasible action	Form of agency still possible	Agentic burden created by intervention	Interpretation boundary	Implementation consequence
Patient or care-context state — Knowledge present, resources restricted				
Cost, time, transport, housing or caregiving constraints	Prioritization, substitution, delayed action	High when benefit requires repeated self-management	Non-action does not establish low motivation	Reduce material and logistical requirements
Patient or care-context state — Motivation present, institutional access unstable				
Eligibility, scheduling, continuity or insurance barriers	Persistence, navigation, advocacy	High when patients must repeatedly overcome system failures	Engagement cannot compensate indefinitely for unavailable services	Redesign access and continuity
Patient or care-context state — Strong social support, weak structural opportunity				
Supportive relationships but limited local services or economic resources	Collective problem-solving and assistance	Variable	Cohesion does not eliminate structural disadvantage	Couple relational support with resource expansion
Patient or care-context state — High autonomy, high intervention complexity				
Multiple choices but substantial cognitive or administrative workload	Preference expression and active management	Potentially high	More choice does not necessarily mean more usable control	Simplify decision and administrative load
Patient or care-context state — Low-resource context with low-agentic structural intervention				
Limited personal capacity to act	Benefit can occur without repeated individual action	Relatively low	Structural location alone does not ensure equitable implementation	Evaluate reach, implementation and unintended effects
Patient or care-context state — Proposed synthesis: constrained but adaptive agency				
Multiple interacting limitations	Adaptation, resistance, delegation or selective engagement	Depends on intervention architecture	Adaptive behavior should not be misclassified as irrationality	Match intervention demands to realistic capacity

Note: “Exercisable agency” and “constrained but adaptive agency” are proposed constructs in this review rather than validated clinical categories.

An integrated mechanistic framework

The evidence reviewed here supports neither a single pathway nor a flat collection of social risks. The proposed synthesis instead treats health production as a nested system in which structural and policy arrangements influence material opportunities and institutional practices; community and relational environments alter exposure, support, and norms; psychosocial and behavioral processes affect adaptation and participation;

healthcare can amplify or attenuate these pathways; and biological or clinical consequences emerge over time.

Crucially, scale transfer is not automatic. Neighborhood inequality is not an individual psychological state, discrimination is not implied by demographic identity, prediction is not mechanism, and biomarker change is not required for a social exposure to matter. Each transition between levels therefore represents an inference boundary requiring appropriate measurement and design.

Figure 1 compresses these distinctions into a nested architecture in which social conditions can operate across levels without implying deterministic control by the outermost level.

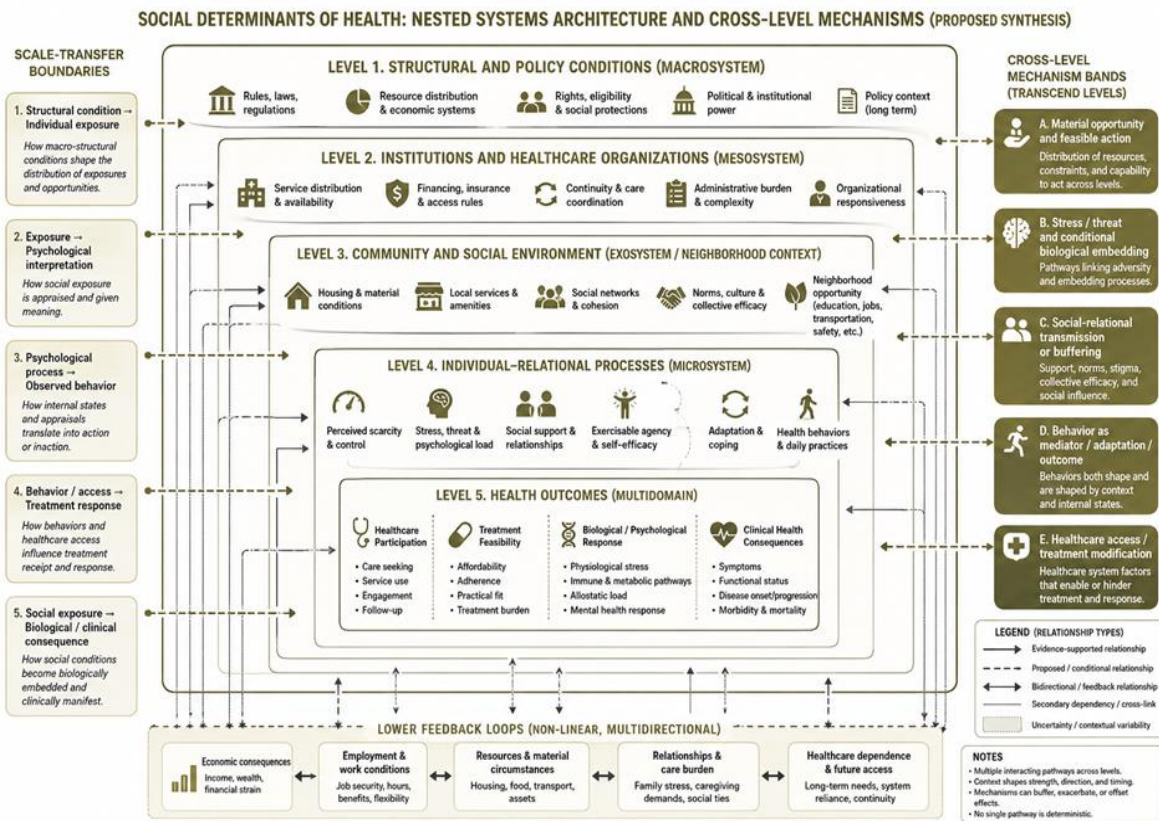


Figure 1. Scale-transfer boundaries in the mechanistic translation of social conditions into health

Implications for intervention design

Mechanism-oriented intervention design asks not only which population has elevated risk, but which process produces the disadvantage and what the intervention requires people or institutions to do. Resource scarcity requires a different response from informational uncertainty; administrative exclusion differs from low trust; unsafe environments differ from limited motivation. Structural interventions may reduce individual burden, but their effects remain dependent on implementation, reach, and political context [34]. A useful proposed criterion is mechanism–burden fit: the intervention should target the process plausibly generating disadvantage without requiring those most constrained by that process to carry disproportionate responsibility for overcoming it. This principle does not imply that individual-level interventions are inappropriate. Rather, educational, behavioral, clinical, organizational, and policy approaches should be judged

partly by whether their required agency is realistic under the conditions they intend to change.

Implications for measurement and causal research

Mechanistic research requires alignment among construct, level, timing, and outcome. A measure of neighborhood disadvantage cannot substitute automatically for experienced scarcity; demographic identity cannot substitute for discrimination exposure; healthcare utilization cannot substitute for access; and a biomarker cannot validate a psychosocial mechanism merely by being biologically plausible.

Measurement instruments can improve construct discrimination. Development of the Social Determinants of Health Questionnaire, for example, supported a two-factor structural/intermediary organization in its sampled population, while also requiring further validation beyond that context [35]. Psychometric fit therefore addresses measurement performance, not causal validity or universal transferability.

Future causal research should measure exposure trajectories, candidate mechanisms, contextual modifiers, competing pathways, and outcomes on timescales appropriate to the proposed process. Multilevel and longitudinal designs are especially important when conditions cross neighborhood, institutional, relational, and individual levels.

Critical gaps and theoretical priorities

The largest gap is not the absence of additional determinant lists but insufficient identification of the links connecting levels. Much evidence remains observational, cross-sectional, geographically concentrated, or dependent on broad constructs whose mechanisms are underspecified. Null biological findings, heterogeneous social-cohesion effects, uneven

intervention moderation, and context-dependent policy effects should therefore be treated as theoretically informative rather than inconvenient exceptions.

Priority tests include repeated-measurement studies separating exposure from mediator and outcome; cross-context tests of whether the same social condition operates through different mechanisms; stronger quasi-experimental and causal designs for policy and institutional change; explicit modeling of power and implementation; and measurement studies that test invariance across populations rather than assuming transferability.

The remaining uncertainties can be organized more clearly by distinguishing what existing evidence can support from what must still be tested before mechanism-level interpretation is justified (**Table 3**).

Table 3. Evidence–interpretation boundaries that define the next tests of social-determinant mechanisms

Mechanism–context problem	What current evidence can support	Interpretation that remains premature	Critical boundary	Priority test
Social exposure associated with health outcome	Patterned association and hypothesis generation	Specific causal pathway	Confounding, selection, temporality	Longitudinal or quasi-experimental causal design
Discrimination associated with self-reported health but not selected biomarkers	Psychosocial relevance without confirmed tested biological mediation	Universal inflammatory embedding	Biomarker choice and timing	Repeated multimarker and alternative-pathway testing
Cohesion related differently to access or preventive behaviors	Relational context matters in outcome-specific ways	Uniform protective effect of “social cohesion”	Construct dimensionality and structural opportunity	Harmonized multidimensional measures across settings
Social-risk variables improve clinical prediction	Incremental prognostic information	Explanation of why outcomes differ	Prediction–mechanism boundary	Mediation/causal analyses tied to actionable exposures
Structural intervention changes population conditions	Policy-level exposure alteration	Automatic equity improvement	Implementation, reach, power and agentic demand	Comparative policy evaluation with distributional outcomes
Valid SDOH questionnaire in one population	Construct measurement within tested context	Universal construct equivalence	Cultural/population measurement invariance	External validation and invariance testing
Proposed cross-level framework	Coherent hypotheses linking levels	Validated predictive or intervention model	Scale transfer and link-specific evidence strength	Test individual pathways before validating the whole architecture

Note: The final row identifies this review’s proposed synthesis and should not be interpreted as an empirically validated model.

Conclusion

Social determinants become scientifically informative when they are treated as conditions that shape mechanisms rather than as an expanding inventory of independent risks. Material resources alter feasible opportunity; stress and threat can become biologically

consequential without following one universal pathway; relationships transmit both support and constraint; institutions distribute resources and power; behavior reflects both agency and adaptation; healthcare modifies the translation of social conditions into clinical consequences; and life-course feedback makes exposure and outcome mutually consequential over time.

The framework proposed here does not replace empirical causal identification. Its purpose is to make the questions requiring identification more explicit. Moving beyond risk-factor thinking therefore means specifying the level at which a condition exists, the process through which it is hypothesized to matter, the context that modifies that process, the alternative explanations that remain plausible, and the intervention burden required to change it. Such distinctions can support more precise causal research and more equitable intervention design while preserving uncertainty where the evidence remains incomplete.

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