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Social Capital–Neighborhood Stress Pathways in Aging: A Conceptual Framework for Prostate Cancer Vulnerability in Middle- and Old-age Black Men in the USA

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Middle- and old-age Black men in the United States continue to experience disproportionately high prostate cancer incidence, aggressive disease, and mortality despite decades of research on biological, behavioral, and healthcare determinants. Existing health disparities frameworks clearly identify relevant social and structural factors but offer only a limited explanation of the mechanisms by which neighborhood disadvantage becomes biologically embedded in this population. This conceptual paper proposes a multilevel mechanistic framework linking neighborhood structural stressors to prostate cancer vulnerability among middle- and old-age Black men. Drawing on life course theory, fundamental cause theory, the stress process model, social capital theory, and allostatic load theory, the framework outlines a cascading pathway in which neighborhood disadvantage influences bonding, bridging, and linking social capital, which, in turn, affects sleep health as a proximal recovery process. Impaired sleep is proposed to increase allostatic load, accelerate biological aging, and ultimately elevate prostate cancer risk and adverse outcomes. The framework advances prostate cancer disparities research by integrating structural, relational, and biological processes into a single testable model, distinguishing the unique roles of different forms of social capital, and positioning sleep as a key mechanistic pathway linking social environments to physiological dysregulation. This paper generates empirically testable hypotheses and identifies modifiable intervention targets at neighborhood, community, clinical, and policy levels to advance precision prevention strategies and reduce persistent prostate cancer disparities among middle- and old-age Black men.

Keywords: *Prostate cancer disparities; Black men; Neighborhood stress; Social capital; Sleep Health*

Introduction

The Persistent Challenge of Prostate Cancer Disparities

In 2025, there were 313,780 new cases of prostate cancer (PCa), a 15% proportion of all new cancer cases in the United States (National Cancer Institute 2026). This cancer also led to an estimated 35,770 (5% of all cancer deaths) deaths in the same year. The latest population data also reveal that over 3.5 million US men were living with PCa in 2022. A closer look at age-adjusted incidence rates reveals that Non-

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Hispanic Black men (Black men) continue to suffer from the disease at a disproportionately higher rate than all other racial groups, with an incidence rate of 194.8 per 100,000 (based on 2018 – 2022 data). Black men are 67% more likely to be diagnosed with prostate cancer and are more than twice as likely to die from it compared to Non-Hispanic White men. The disparity persists in older age as PCa rates remain highest in Black men 50 years and older (9.3 per 10,000, based on 2022 data) than other racial groups (SEER, 2026). Recent increases in disease incidence rates are partly driven by annual surges of 2.6% in men younger than 55 years, 6.0% in men aged 55–69, and 6.2% in men aged 70 and older (Kratzer et al. 2025). Despite the clear documentation of these PCa disparities, efforts to prevent or reduce the severity of the disease among the disproportionately affected middle- and old-age Black men have had little impact. This signals a need for a mechanistic understanding of the pathways through which PCa predictors operate to cause adverse health outcomes in this population.

At a minimum, we know that the highlighted disparities persist even after accounting for individual risk factors, such as age and socioeconomic status, signaling the significance of structural and social determinants of PCa incidence and mortality in Black men. The adverse PCa health effects of neighborhood disadvantage, structural racism, barriers to quality care, and the cumulative disadvantage caused by chronic stress are well documented (Boyle et al. 2024). These factors intersect with individual factors, making it hard to eliminate disparities with a single strategy (Lowder et al. 2022). Indeed, theories and frameworks for understanding health disparities have called for multilayered approaches to conceptualizing and operationalizing key constructs. Despite the well-documented structural determinants of prostate cancer disparities, the field lacks a parsimonious and testable framework linking these distal factors to biological risk. Existing models identify relevant domains, but do not specify how structural conditions become physiologically embedded. This paper addresses this gap by proposing a conceptual framework in which neighborhood disadvantage shapes social capital (a critical pathway in the low-resource areas), which, in turn, influences sleep, a central recovery mechanism driving biological vulnerability.

Conceptual Innovation

The proposed framework contributes to existing research on prostate cancer disparities research in three ways. First, it integrates neighborhood structural stressors, social capital, and sleep health into a single multilevel framework. Second, it distinguishes bonding, bridging, and linking social capital as distinct intermediary pathways. Third, it positions sleep as a proximal biobehavioral recovery mechanism linking social exposures to physiological dysregulation and prostate cancer vulnerability.

Limitations of Existing Disparity Models

This paper proposes a mechanistic framework to supplement existing health disparities models, which have significant limitations. First, individual-level models explain genetic predisposition, health behaviors, and clinical risk factors, but they inherently do not account for environmental influences that explain the persistence

of racial disparities in prostate cancer outcomes after statistical adjustment. Empirical studies consistently show that adjusting for individual-level factors, such as socioeconomic status, health behaviors, and clinical risk factors, does not eradicate racial disparities in prostate cancer outcomes between Black and White men (Albain et al. 2009, Robbins et al. 2016). This suggests salient contextual and structural determinants play a central role in shaping prostate cancer risk and progression. Interventions targeting behaviors without addressing structural barriers have limited long-term effectiveness in reducing disparities (Thornton et al. 2016).

At the multilevel, predominant frameworks for understanding health disparities, such as the National Institute on Minority Health and Health Disparities (NIMHD) Research Framework (NIMHD 2025), identify key health domains, including biological, behavioral, environmental, sociocultural, and health care system factors. Still, they do not specify the causal relationships and mediating pathways linking these domains. As a result, these frameworks are often applied descriptively, leading to fragmented analyses that identify relevant factors but lack a clear explanation of how they interact in a predictive sequence. This challenge leads to fragmented analyses and poor identification of critical intervention points in health disparities research (Brown et al. 2019). Hence, the key limitation is not the identification of determinants, but rather the absence of clear pathways through which structural conditions become biologically embedded to produce prostate cancer vulnerability.

Socioecological models are also great for organizing cross-level determinants, from individual to structural contexts (Bronfenbrenner 1978, McLeroy et al. 1988). However, these models function as organizing descriptive models rather than mechanistic frameworks with clear conceptual links between levels. While they highlight the significance of cross-level associations, they do not explicitly outline the intermediate processes linking distal structural factors to proximal biological outcomes (Golden & Earp 2012). The lack of mechanistic pathways across the levels hinders the development of interventions with clear conceptual directions using socioecological models (Golden & Earp 2012). For example, on the topic of Black men and prostate cancer, these models may identify neighborhood disadvantage at the community level and health behavior at the individual level, but fail to specify the intervening mechanisms, such as stress, sleep disruptions, and social capital, that often explain how neighborhood-level factors manifest in health behaviors and outcomes.

Taken together, these frameworks identify relevant domains of health determinants but do not often provide a parsimonious and testable guide on how structural disadvantage translates into prostate cancer vulnerability. This gap limits both empirical testing and intervention development, underscoring the need for mechanistic frameworks that specify the sequential pathways linking structural conditions, social processes, and biological outcomes.

Study Aim

Given these limitations, this paper aims to develop a multilevel conceptual pathway framework that addresses the mechanistic gaps in existing prostate cancer dipartites models. This mechanistic framework explicitly specifies how distal structural determinants translate into prostate cancer vulnerability through intermediate social

capital and recovery processes. The core innovation of this framework lies in its comprehensive distal → intermediate → , and proximal cascade. Neighborhood structural factors (distal) impact the availability and quality of social capital (intermediate), which, in turn, impairs bodily recovery processes (proximal), ultimately affecting physiological aging and increasing PCa risk. We center sleep as a primary and measurable recovery mechanism because of its strong link to quality of oncological health across age groups, especially among middle- and old-age individuals (Han et al., 2025). Positioning sleep as a key proximal pathway through which neighborhood adversity and social capital are embodied (McEwen, 1988; Williams et al., 2019) provides a novel, testable framework for understanding how structural disparities and social relationships jointly shape cancer disparities in aging Black men.

As such, we offer a mechanistic causal model that adds to the existing descriptive and multi-level frameworks. Specifically, the framework proposes that neighborhood structural disadvantage shapes the composition and use of social capital, which influences sleep as a recovery mechanism, leading to biological dysregulation and increased prostate cancer vulnerability. In the framework, accelerated aging and allostatic load represent biological vulnerability, while prostate cancer incidence and progression are clinical outcomes. This framework is designed to support empirical testing through multilevel mediation models that link neighborhood conditions, social capital, sleep, and biological markers.

Roadmap of the Paper

This paper proceeds as follows: (1) The *literature review* synthesizes evidence on prostate cancer disparities in the US, neighborhood stressors, social capital, racism, and sleep as a behavioral mechanism, to shape the rationale for a mechanistic framework. (2) The *methodology for framework development* section outlines the theoretical grounding, construct selection, and integration approach. (3) The *results* section describes the cascading multilevel conceptual framework detailing the distal (neighborhood stressors), intermediate (social capital), and proximal (sleep) pathways, and presents explicit testable hypotheses. The discussion shows the contributions of the framework relative to existing ones, as well as its implications and limitations. (4) The *conclusion* section summarizes the manuscript and offers directions for empirical testing.

Literature Review

Prostate Cancer Disparities in Black Men: Beyond Individual Risk

Black men in the United States face a disproportionately higher burden of prostate cancer (PCa) compared to all other racial or ethnic groups. Recent SEER (CDC 2014) data show that this group has the highest age-adjusted incidence rate of 170–180 per 100,000, compared with 100–110 per 100,000 for their White counterparts. The disparities in incidence rates persist across different age groups and all stages, including early-onset and advanced disease (Krazter et al. 2025). Black men also have a PCa mortality rate more than twice that of White men (approximately 40 per 100,000

compared to 18 per 100,000), with the disparity remaining even after accounting for stage at diagnosis (CDC 2014, Chai et al. 2025). This signals factors beyond clinically presenting factors (e.g., treatment access). Another indicator of the gap is that Black men are more likely to be diagnosed with high-grade and advanced state PCa (Gleason score ≥ 8), and they are more likely to experience the disease at earlier ages (Lillard et al. 2022, Wiginton 2024). This shows a possible acceleration of disease progression related to structurally induced cumulative stress exposures. Individual-level analyses have also shown similar gaps. For example, studies have shown that adjusting for individual-level socioeconomic variables does not eliminate PCa burden across Black and White racial or ethnic groups (Albain et al. 2009, Robbins et al. 2016). This result indicates the significance of unmeasured factors such as neighborhood exposures, discrimination, and chronic distress.

Geronimus (1992) explained that cumulative exposure to adverse conditions, such as racism, leads to premature biological aging and health deterioration among Black Americans – weathering. This view argues that health declines earlier and more rapidly in marginalized populations due to chronic stress. Therefore, accumulated stress leads to premature aging and overall disease vulnerability, conditions that then lead to early onset and intense severity of the age-related PCa disease (Geronimus 1992). Empirical studies highlight that Black men report higher allostatic load than White counterparts, and these higher scores are associated with worse cancer outcomes (Geronimus et al. 2006, Li et al. 2024). The weathering perspective may explain why Black men are more likely to report early onset and more advanced PCa versus White men. The weathering hypothesis provides a critical framework that links neighborhood factors to biological risk factors for PCa. Still, it does not offer mediating explanations from structural adversity to biological wear-and-tear.

Neighborhood Environment as a Structural Determinant of Health

There is a deep history of research indicating that neighborhood disadvantage increases risks to morbidity and mortality among underserved Black populations (Holder et al. 2025). Cancer studies conceptualize neighborhood environments based on socioeconomic (e.g., employment rates), social (e.g., crime rates), and physical (e.g., public transportation network) characteristics of the areas where people live (Gomez et al. 2015). Disadvantaged neighborhoods experience overcrowding, reduced social cohesion, inadequate access to services, and violence (Namin et al. 2021, Urban Institute 2017), all of which are risk factors for poorer PCa health. Additionally, such neighborhoods have insufficient oncological health facilities for PCa screening, treatment, and care, and the existing facilities are overwhelmed because of limited resources (Gardner et al. 2025, President's Cancer Panel, 2022). Numerous studies have reported associations between residence in a disadvantaged neighborhood and various physiological consequences, including higher PCa risks, prevalence, and mortality (Ziegler-Johnson 2011). The higher PCa risk and burden among Black men do not decrease even after accounting for race or ethnicity (Mahal et al. 2022).

Neighborhoods are also significant predictors of cellular aging. Epidemiological studies that account for neighborhood factors such as zip codes have shown that people living in neighborhoods with scarce socioeconomic resources, such as

quality housing, proper food, green spaces, and jobs, are likely to have high cellular aging (Gomez et al. 2015, Rodrigues et al. 2026). For example, a study of 1,125 US adults found that living in low-opportunity neighborhoods was associated with significantly higher CDKN2A RNA levels, a marker of biological aging (Rodrigues et al. 2026). It also showed that associations were strongest for social and economic opportunities, depicting a socially structured pattern of aging. Accelerated biological aging, as depicted by such studies, is strongly linked to increased risks of developing prostate cancer (PCa), typically called early-onset occurrence (Yin et al. 2024). Therefore, to lower PCa risk, we must address neighborhood stressors that cause early-onset and aggressive cases in Black men.

Social Capital: Conceptualizations and Links to Health

Social capital refers to the resources embedded in social networks that facilitate collective benefits and individual well-being through trust and norms of reciprocity (Putnam 2000). Putnam (2000) further distinguished two types of social capital: ***bonding*** (strong homogeneous ties that provide emotional and instrumental support for individuals to get by) and ***bridging*** (weaker cross-group connections that provide opportunities and informational support to get ahead). ***Linking*** capital (vertical links of respect and institutional support between individuals and those in positions of power) was later introduced by Szreter and Woolcock (2004). In the context of aging and health disparities, the clear theme across these three forms of capital is the health-impacting resources that individuals derive from the unique levels of capital (Poortinga 2012). These resources may either buffer or exacerbate the effects of social disadvantage across the lifespan, especially late-life health outcomes.

Empirical evidence indicates that social capital is generally protective against adverse cancer outcomes in structurally disadvantaged neighborhoods, but its effects are context dependent (Fuemmeler et al. 2023). Higher levels of social capital, especially bonding and bridging types, are associated with greater cancer screening uptake, better treatment adherence, and reduced mortality risk among older adults, even after accounting for individual sociodemographic factors (Dean et al. 2015, Leader & Michael 2013). In the PCa literature, higher neighborhood social capital (e.g., neighborhood participation) is associated with a greater likelihood of PSA screening among high-risk Black men (Dean et al. 2015). In contrast, lower neighborhood linking capital is associated with lower prostate cancer incidence but higher PCa mortality (Hamano et al. 2021). Evidence linking bonding capital to health reveals a conflicting theme: while dense close ties offer support for good health, they may also produce stress, negative affect, and maladaptive behaviors that are detrimental to health in resource-limited neighborhoods (Niu et al. 2025, Poortinga 2012). Linking capital (vertical connections to health providers and institutional trust) is protective against adverse health outcomes because it enables older men to navigate health systems to reduce structural barriers (Hamano et al. 2021, Szreter & Woolcock 2004). Collectively, these studies emphasize social capital as a key consideration in PCa health and invite researchers to account for bonding, bridging, and linking forms of social capital as mediating mechanisms that influence PCa outcomes at the physiological level.

Recovery Processes: Sleep as a Proximal Biological Mechanism

At the physiological level, sleep serves as a critical restorative process that supports healing and is strongly linked to health outcomes. Studies underscore that chronic stress and circadian disruption accumulate physiological wear and tear, problems regulated through nightly restoration of immune and metabolic balance (MacEwen & Karatsoreos 2022, McEwen 2015). Black individuals report sleep health disparities, such as poorer sleep quality, higher insomnia rates, and shorter sleep duration, even after accounting for socioeconomic factors (Jackson et al. 2020, Slopen et al. 2016). These adverse reports are caused by strong evidence linking neighborhood disadvantages to sleep risk factors: noise, perceived insecurity, and hyperarousal caused by heightened vigilance (Hicken et al. 2013, Troxel et al. 2018). Further evidence suggests that sleep is a proximal driver of inflammation, metabolic dysregulation, and accelerated cancer progression. Specifically, insomnia and poor actigraphy-measured sleep patterns are associated with greater PCa disease burden, including higher PCa occurrence and increased mortality risk among Black men in the US (Anukam et al. 2026).

Gaps and Need for an Integrated Framework

Together, the evidence discussed above positions sleep as a central biobehavioral pathway through which structural disadvantage and social capital factors become biologically embedded. It depicts a cascading relationship that explicitly includes sleep (proximal) and social capital (intermediate) as critical links between neighborhood adversity and prostate cancer vulnerability in middle- and old-age Black men in the US. To our knowledge, no existing framework combines distal neighborhood factors, intermediate social capital, and proximal sleep into a testable research tool for studying and addressing PCa health disparities in Black men. The proposed framework fills this gap by specifying pathways from neighborhood structural adversity social capital → sleep → prostate cancer/accelerated aging.

Methodology for Framework Development

Approach and Study Design

This paper utilizes a conceptual synthesis methodology proposed by Jaakkola (2020) for developing a multilevel model that examines conceptual pathways from neighborhood structural stressors to prostate cancer vulnerability in middle- and old-age Black men. As per the model paper template (Jaakkola 2020), this paper proposes a focal phenomenon, the cascading pattern from distal adversity to accelerated aging and PCa vulnerability, by identifying key constructs (neighborhood stressors, social capital, sleep, and physiological vulnerability) and proposing novel theory- and evidence-backed relationships between them. The data used for the proposed model are drawn from existing theories and empirical findings in social epidemiology, gerontology, and the biopsychosocial literature.

The study design follows two recommendations outlined by Jaakola (2020). First, the choice of theories or concepts is justified. The framework integrates life-course perspectives, fundamental cause theory, stress process model, social capital theory (bonding, bridging, and linking), and allostatic load theory. These are well-suited for this framework because they collectively explain the sequential influence of structural conditions on accelerated aging and PCa vulnerability, through social capital and biological mechanisms – an area insufficiently addressed by existing descriptive frameworks in cancer health disparities literature. Second, the choice of domain and method theories. Domain theories consist of literature that outlines critical elements of the phenomenon under study, prostate cancer disparities in aging Black men.

In contrast, method theories provide conceptual frameworks for explaining how distal stressors translate into proximal biological outcomes (Lukka & Vinnari 2014). Empirical testing is beyond the scope of this work, but the framework presents testable, literature-based hypotheses. Based on literature-informed claims, evidence, and theories, the design will ensure the resulting framework is both logically coherent and positioned for future empirical testing.

Theoretical Grounding and Integration

Five interconnected theories inform the proposed framework. Each addresses a unique component of the cascading effects of neighborhood structural adversity on prostate cancer (PCa) vulnerability**.** First, the *lifecourse perspective* (Elder 1998) provides a temporal lens and posits that old-age health is predicted by the accumulation of exposures and key transitions across the lifespan, not by a single event. This lens informs our conceptualization of PCa vulnerability among middle- and old-age Black men, showing most visibility in this age group. The greater risk and vulnerability to adverse PCa health indicate accumulated stress on the body (Geronimus 1992). Relatedly, *fundamental cause theory* (Link & Phelan 1995) adds that structural conditions (e.g., neighborhood disadvantage, segregation, and violence) are key drivers of individual health that must be addressed to reduce or eliminate disparities. Third, the *stress process model* (Pearlin et al. 1981) emphasizes that the effects of distal stressors (e.g., neighborhood adversity) on individual health are transmitted through intermediate social resources (e.g., social capital) and manifest as biopsychological dysregulation. Fourth, *social capital theory* (Bourdieu 1986, Szreter & Woolcock 2004) proposes a three-tiered model of bonding (strong, homogeneous ties), bridging (weaker, cross-group connections), and linking (vertical ties with powerful individuals and institutions). The three tiers are unique and have divergent effects on health, especially for individuals in under-resourced settings (Szreter & Woolcock 2004). Finally, *allostatic load theory* (McEwen 1998) justifies the biological outcome pathway, explaining how chronic psychological stress is embedded in adverse PCa risks and accelerated aging.

The framework proposed here integrates these theories into a unified conceptual cascade. Particularly, it expands the stress model theory into three novel ways: (1) it unpacks the mediator (social capital) into bonding, bridging, and linking, allowing for the modelling of varied non-linear mediating effects; (2) it includes linking capital (trust in institutions and power brokers) which has been under-represented

in oncology research despite its clear relevance to healthcare use, navigation and quality for older Black men (Wood & Patel 2024); and (3) it identifies sleep as the proximal outcome (or chief recovery process) through which stress process becomes biologically embedded. This positions sleep as a vital pathway linking social capital to allostatic load, a mechanism absent from prior PCa disparities frameworks. Together, these theories are the conceptual foundation for a model that includes temporal (life course), structural (fundamental cause), causal (stress process), relational (social capital, and biological (allostatic load) elements.

Construct Selection and Organization

This paper uses an explicit three-step criterion to construct the proposed framework. First, all constructs must have **empirical grounding** in peer-reviewed literature that links them to prostate cancer risk, allostatic load, and racial health disparities. Second, constructs should be **modifiable** to enhance the framework's translational utility. That is, they can be influenced through policy, community, or individual-level interventions. Third, each construct is **relevant to the population** (middle- and old-age Black men in the United States) and the **outcome** (prostate cancer vulnerability - including incidence, progression, and mortality) of interest.

Applying this criterion identified concentrated poverty, residential segregation, environmental disorder, and community violence as distal constructs of neighborhood structural distress; three intermediate constructs of social capital (bonding, bridging, and linking capitals); and two proximal constructs (sleep health and allostatic load). Table 1 below provides the levels and measurement approaches for each construct, drawn from the literature or standardized instruments. These details move the framework from abstract conceptualization to a tool that can be empirically tested.

Table 1. *Operationalization of Key Constructs*

Construct	Level	Proposed Measure(s)
Neighborhood structural stressors	Distal	Percentage of population below poverty line, percentage of Black residents, violent crime rate, physical disorder index
Bonding social capital	Intermediate	Family network size, frequency of contact, perceived emotional support
Bridging social capital	Intermediate	Collective efficacy scale, neighborhood cohesion scale
Linking social capital	Intermediate	Trust in police, healthcare system, and local government

Sleep health	Proximal	Actigraphy (sleep efficiency, duration), Pittsburgh Sleep Quality Index, insomnia symptoms
Allostatic load	Proximal / Outcome	Composite index: C-reactive protein, interleukin-6, systolic and diastolic blood pressure, hemoglobin A1c, high-density lipoprotein, waist-hip ratio, urinary cortisol
Prostate cancer outcomes	Outcome	Registry-linked or self-reported diagnosis, Gleason grade, stage at diagnosis, mortality

Results: The Multilevel Conceptual Framework

Overview of the Framework

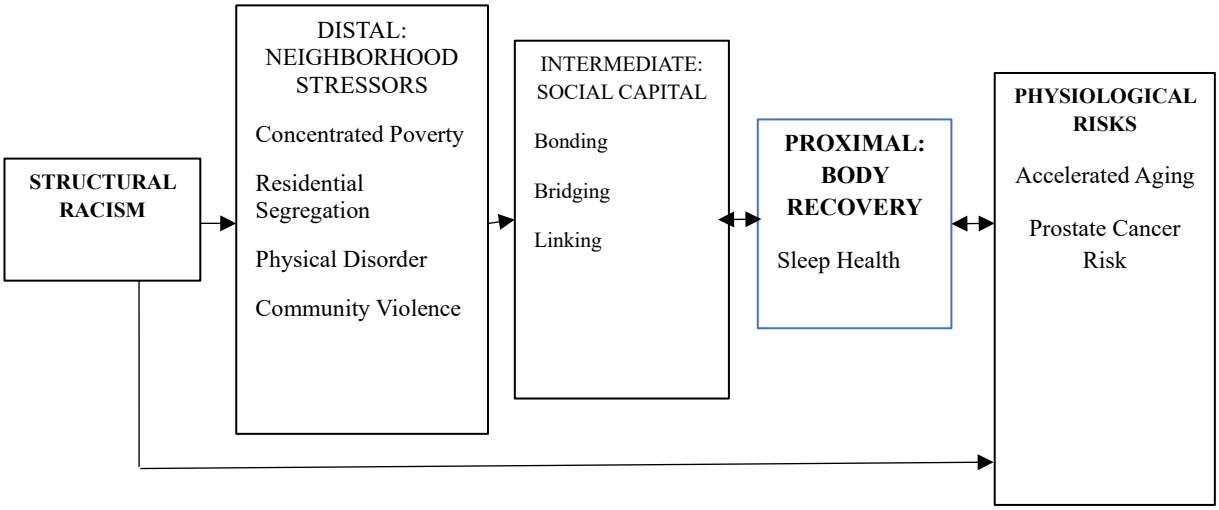
Figure 1 below shows the proposed conceptual framework. It outlines a cascading pathway from distal structural determinants to prostate cancer vulnerability among middle- and old-age Black men in the US. The conceptual sequence involves three interconnected levels: distal (neighborhood structural stressors), intermediate (social capital), and proximal (body recovery through sleep). The sequence culminated in physiological vulnerability operationalized as accelerated aging and prostate cancer risk. The main argument is that neighborhood structural stressors dictate the availability and quality of social capital, which in turn affects sleep recovery processes, ultimately affecting allostatic load and prostate cancer outcomes. Theoretically, the model also indicates bidirectional relationships, including the potential reduction of social capital due to poor health outcomes and social withdrawal due to sleeping problems. Level-by-level descriptions are outlined below.

Level 1: Distal – Neighborhood Structural Stressors

Fundamental health theory (Link & Phelan 1995) posits that social conditions are key drivers of health disparities because they provide flexible resources that buffer health risks. Chronic exposure to neighborhood stressors is the foundation for understanding psychological strain and biological dysregulations that lead to PCa disparities across the life course of Black men in the US. Critical race theorists have argued that systemic racism is the bedrock that produced unjust sociopolitical indicators of health, which deny equitable access to education, health care, employment, housing, infrastructure, and political institutions (Bailey et al. 2021). Elder (1998) adds that these hardship conditions accumulate, with consequences visible in middle and old age. Inequitable access to resources disenfranchises Black

neighborhoods, causing chronic neighborhood stress. Following DeRouen and colleagues' (2025) lead, we operationalize neighborhood-level structural stressors using four interconnected domains: concentrated poverty, residential segregation, physical disorder, and community violence.

Proposition 1 (Neighborhood → Social Capital): *Chronic exposure to neighborhood structural stressors degrades all forms of social capital, leading to poor sleep health*. The result is a maladaptive social capital profile: low bridging/linking and high (but often low-quality) bonding. Existing evidence outlines the relationships among factors in the proposed cascade, but few frameworks have tested the proposed mediation path linking neighborhood stressors and sleep via social capital. Indeed, Dean and colleagues (2015) found that neighborhood social capital varies across contexts and is associated with health behaviors among Black men. Barber and colleagues (2016) used data from the Jackson Heart Study to illustrate that higher neighborhood adversity was significantly associated with lower neighborhood social cohesion. Additionally, living in neighborhoods with limited resources is directly associated with shorter and lower-quality sleep (Troxel et al. 2018). On the one hand, neighborhood stressors increase reliance on bonding ties, which may, in turn, increase (rather than reduce) stress in contexts of high disadvantage (Poortinga 2012). On the other hand, chronic neighborhood stress exposure erodes linking and bonding capital (Poortinga 2012). Collectively, these studies confirm that neighborhood factors determine levels of social capital, which, in turn, impair sleep health. The full mediation path remains scantily defined and untested on the topic of prostate cancer among middle- and old-age Black men.



Level 2: Intermediate – Social Capital as a Mediator

The stress process model outlines that distal stressors impact individual-level health through intermediate social factors, such as social resources. Social capital theory (Bourdieu 1986, Szreter & Woolcock 2004) further breaks social resources

into bonding (strong, homogeneous relationships), bridging (weaker, cross-group ties), and linking (even weaker relationships with institutions). The proposed framework, grounded in these theories, posits that the three forms of capital have distinct mediating effects on the relationship between neighborhood structural stressors and sleep health.

Proposition 2a (Bonding ties → Sleep): *Bonding ties exhibit a non-linear association with sleep health, where increased bonds promote restorative sleep through emotional support, but very high levels of these homogenous ties disrupt sleep through compassion stress, role obligations, and indirect trauma*. A recent study using nationally representative data from the National Survey of American Life Study showed that receiving emotional support (bonding capital) from family was associated with reduced likelihood of restless sleep (Nguyen et al. 2024). Williams and colleagues (2015) found gender and racial differences in the positive effects of close ties on sleep health. They discovered that emotional support was associated with 38 minutes longer sleep time among Black men only, not among women or other racial groups. However, Mitchel and LaGory (2002) confirmed that despite the known protective behavioral health outcomes of bonding ties, they show diminishing health returns in high-poverty, racially segregated, inner-city neighborhoods. The detrimental effects of high bonding in high-stress neighborhoods on sleep remain undertested in middle- and old-age Black men in the US.

Proposition 2b (Bridging connections → Sleep): *Bridging capital (including collective efficacy, neighborhood cohesion, and social participation) shows a linear, protective association with sleep health*. A recent cross-sectional study of 252 Black adults in Connecticut found that participants reporting poor sleep quality were significantly more likely to live in neighborhoods with low social cohesion (Nam et al. 2018). In terms of specific dimensions of sleep health, Echverria et al. (2008) reported positive associations between neighborhood social cohesion and sleep duration in a multi-ethnic study of Atherosclerosis, including Black men. Similar patterns have been documented in studies that collect nationally representative, real-time sleep health data in this population using actigraphy. For example, Chen and colleagues (2016) found that greater social participation (religious attendance and volunteering) was associated with fewer wake episodes and less sleep interruption. These studies provide clear evidence that strong bridging ties are associated with better sleep health.

Proposition 2c (Linking connections → Sleep): *Linking capital (i.e., trust in institutions, such as the police, healthcare systems, and local government) is positively associated with sleep health, mediated by heightened vigilance and hyperarousal.* Studies testing the association between linking capital and sleep are scarce. Most existing studies focus on the psychological consequences of structural discrimination and racism on sleep health (Hickens et al. 2013, Johnson et al. 2022). For example, racism-induced vigilance was associated with greater sleep difficulty (including trouble falling asleep, staying asleep, and early morning awakening) among Black Americans (Hickens et al. 2013). Relatedly, Richardson and team (2021) showed that violent crime was associated with greater actigraphy-measured wakefulness after sleep onset, but perceived police presence was not a significant mediator. This finding is especially interesting, as it underscores the need to implement institutionally driven

changes to address neighborhood stressors rather than increase the presence of powerful authorities, such as the police.

Level 3: Proximal – From Sleep to Allostatic Load and Prostate Cancer

Allostatic load theory (Ballesio et al. 2026) outlines how chronic stress is biologically embedded through dysregulation of immune, metabolic, and neuroendocrine systems. Sleep is the primary restorative process that regulates these systems (McEwen & Karatsoreos 2015). Chronic poor sleep (irregular, brief, and low efficiency) elevates allostatic load, which in turn accelerates biological aging and increases prostate cancer risk.

Preposition 3 (Sleep → Allostatic load). *Sleep health (duration, efficiency, and regularity) is inversely associated with allostatic load. S*leep deprivation increases levels of circulating inflammatory markers, such as interleukin-6, impairs glucose regulation, elevates blood pressure, and alters cortisol rhythms; all dimensions of allostatic load (Ballesio et al. 2026, McEwen & Karatsoreos 2015). Precisely, experimental sleep deprivation for ≤ 4.5 hours per night over multiple nights significantly increased circulating IL-6 and CRP (Ballesio et al. 2026). Circadian irregularity is strongly linked to prostate cancer through mechanisms such as immune suppression and metabolic dysregulation (Clemente-Suarez et al. 2025). Unfortunately, middle- and old-age Black individuals face poorer sleep health even after adjusting for socioeconomic and behavioral factors (Chen et al. 2015). And the circadian irregularity (depicted through poor sleep) is strongly linked to prostate cancer through mechanisms such as immune suppression and metabolic dysregulation (Clemente-Suarez et al. 2025). Collectively, these studies confirm that poor sleep contributes to physiological wear and tear, increasing vulnerability to poor prostate cancer health.

Preposition 4 (Allostatic load → Prostate cancer). *Higher allostatic load predicts prostate cancer incidence and adverse prostate cancer outcomes (Gleason grade ≥8 and advanced stage, cancer-specific mortality).* A nationally representative cohort of almost 21000 men showed that Black men with high allostatic load had more than 4-fold increased risk of cancer death compared with Black men with lower loads (Li et al. 2024). Another study of prostate cancer patients showed that higher allostatic load was associated with increased odds of regional disease and worse overall cancer survival (Santaliz-Casiano et al. 2025). Zoomed-in analysis of results in the study revealed higher epinephrine levels (stress biomarker) and regional disease among Black men, and the higher epinephrine levels were significantly linked to cancer-specific mortality (Santaliz-Casiano et al. 2025). These representative studies show that allostatic load is a significant predictor of prostate cancer health and that stress biomarkers are particularly crucial factors for Black men.

Proposition 5 (Full serial cascade). *The full pathway from neighborhood disadvantage→ degraded social capital → impaired sleep → elevated allostatic load → increased prostate cancer risk is the central theoretical contribution of this framework.* There are no empirical studies to support the full cascade, but each relationship in the sequence is informed by recent evidence. For example, neighborhood factors such as murder rate, median income, and car theft index were among the strongest predictors of pre-diagnostic allostatic load among 4.258 prostate cancer

patients, including 886 Black men (Stabellini et al. 2024). This study also showed that Black men faced 63% of the 19 social determinants of health assessed. Neighborhood adversity and systemic biases increase cancer risk among Black people (Nwakasi et al. 2024). Additionally, a large cohort study found that neighborhood deprivation was directly associated with 23% higher risk of short sleep (≤7 hours), a 38% higher risk of circadian disruption, poor sleep quality, and 8% higher risk of long sleep (≥ 9 hours; Barber et al. 2025). The intermediate role of social capital is supported by evidence showing that poor sleep quality among Black adults is significantly associated with lower neighborhood social cohesion (Nam et al. 2018), and higher social participation is associated with fewer wake bouts and less sleep disruption (Chen et al. 2016). Ballesio and colleagues (2026) show that less than 4.5 hours of sleep raises circulating IL-6 and CRP (key components of allostatic load), while Petrov et al. (2024) note that Black people experience poorer sleep outcomes even after adjusting for socioeconomic factors.

Discussion

Summary of Key Findings and Theoretical Contributions

This conceptual paper has developed a multilevel mechanistic framework linking neighborhood structural stressors to prostate cancer vulnerability in middle- and old-age Black men in the US. The proposed framework depicts a cascading relationship from neighborhood disadvantage to social capital (bonding, bridging, and linking) to sleep to allostatic load to prostate cancer risk. It also leaves room for a feedback loop across the factors. Overall, the framework proposes three distinct theoretical contributions.

First, it breaks down social capital into bonding, bridging, and linking types. While not theoretically innovative, this breakdown allows for nonlinear (bonding) and linear (bridging and linking) mediating pathways, a novel specificality previously absent from previous cancer disparities frameworks. *Second*, the framework proposes that linking capital is non-linear and may operate differently across institutional domains. This is an important nuance because, beyond non-linearity, it highlights that institutional influences are not monolithic. Instead, they are diverse and contingent on the history and context of the institution type, which may provide resources in one context while acting as a primary source of structural stress in another. *Third*, it positions sleep as a key proximal biological mechanism linking social exposures to allostatic load, shifting sleep from a general health behavior to a mechanistic pathway to prostate cancer vulnerability. This decision is supported by experimental evidence that sleep deprivation increases interleukin-6 and C-reactive protein (Ballesio et al. 2026), and data showing significant racial disparities in actigraphy-measured sleep, indicating poorer sleep health in Black people (Petrov et al. 2020).

Methodological and Research Implications

The primary goal of this implications section is to provide a general and replicable roadmap for empirical testing. Precise analytic decisions will be determined by the researchers seeking to test the proposed pathways in the proposed framework. The hypotheses proposed in the paper can be tested using existing nationally representative datasets in the US. Such data include the Jackson Heart Study, the National Social Life, Health, and Aging Project, the Health and Retirement Study, the National Health and Nutrition Examination Survey, the Southern Community Cohort Study, and the Prostate, Lung, Colorectal, and Ovarian Cancer Screening Trial. However, these tests must be conducted under the following methodological conditions.

Multilevel analyses with random intercepts must be used to test the hypotheses representing the relationships between social capital and sleep (H2a – H2c). This is a necessary approach because individuals are nested within community-level factors. Failure to account for nested relationships will lead to errors, including type I inflation (Finch 2022). The hypothesized non-linear influence of bonding ties (H2a) must be analyzed using procedures that account for both the quadratic effect of bonding capital and its cross-level interaction with neighborhood disadvantage. The quadratic consideration accounts for the assumed inverted U-shape, testing whether the turning point of the relationship shifts under high stress. These considerations acknowledge context-specific associations where bonding relationships may induce negative affect in high-stress environments (Umberson, Donnelly & Pollitt 2022).

For the mediation hypotheses (H3–H5), robust approaches, such as structural equation modeling (SEM), are appropriate for the serial mediation model (H5), which requires three or more waves of data to capture temporal precedence. SEM and comparable approaches are particularly suited for testing complex relationships, addressing complex sampling strategies, and testing theoretical assumptions (Kline 2023). Bootstrap estimates should also be considered to account for possible non-parametric features of large datasets (Yuan & Hayes 2021). Analytic assessments of the hypotheses must account for possible variability by age (e.g., middle vs. old age), neighborhood disadvantage, and socioeconomic position. Additionally, all constructs can be operationalized using validated measures and biomarkers described in Table 1. In terms of data types, H1 – H4 can be assessed using cross-sectional data to establish associations, but longitudinal data with at least 3 waves is necessary for H5. To handle missing data and minimize bias, modern techniques, such as Full Information Maximum Likelihood in SEM, or traditional approaches, like multiple imputation, can be employed (Lee & Shi 2021).

Practice and Intervention Implications

The framework proposes modifiable targets at each level of the cascade, outlining a roadmap for multi-level interventions. At the neighborhood level, context-based interventions can reduce violent crime, improve housing quality, increase neighborhood capital, and address economic struggles. For example, Richardson et al. (2021) showed that violent crime was associated with actigraphy-measured wakefulness after sleep onset in predominantly Black neighborhoods, supporting crime reduction as a sleep-

promoting intervention. Evidence-based programs such as the Rapid Employment and Development Initiative (READI) Chicago, which used cognitive-behavioral interventions in combination with workforce employment, transitional jobs, and stipends, have shown effectiveness in reducing neighborhood gun violence in Black neighborhoods (Bhatt et al. 2023). Future studies must consider objective sleep health measures.

The proposed framework endorses interventions that develop bridging capital and linking capital at the community level. Community programs that connect middle- and old-age Black men to healthcare resources can compensate for eroded linking capital by increasing trust in medical institutions. However, trust-building initiatives to build linking capital must address medical mistrust by intentionally including community representatives on advisory boards and through culturally tailored outreach. As for bonding capital, men's peer support groups that provide structured support are effective for better behavioral and chronic health outcomes among Black men (Ewen et al. 2024, Lee et al. 2018). However, such approaches to improve sleep and prostate cancer vulnerability remain largely untested in this population. Combined approaches that build trust in health institutions while leveraging structured peer-support practices may be more effective in this area.

In terms of practice, culturally tailored sleep health interventions for Black men are a priority. Beyond the proposed social capital promotion strategies, mental health practitioners and sleep specialists can use evidence-based interventions (EBI) to treat sleep health in this population. However, EBI approaches such as the cognitive-behavioral therapy for insomnia (CBT-I) have demonstrated efficacy across diverse populations (Savin et al. 2023), but implementation in this population will require cultural and geographic adaptations to address the group-specific challenges identified here, e.g., hyperarousal (Zhou et al. 2022). Additionally, community-based sleep and cancer health education strategies can be delivered through community-trusted settings such as churches, local gyms, and barbershops. Using these settings has shown remarkable success for reducing the adverse effects and risk factors for other highly prevalent non-communicable diseases in Black men because of group buy-in and trust (Ewen et al. 2024, Lee et al. 2018).

Policy-level interventions can also use the proposed model to understand the connections among neighborhood capital, the effective influence of community organizations, and the need to implement targeted interventions to address PCa and other health burdens among Black men. For example, studies have shown that police presence in Black neighborhoods alone is not enough for better health outcomes, and improving the Black community's trust and perception of the police force through policy and programming is more effective for better health outcomes among Black men, e.g., reduced hypervigilance and improved perceptions of safety (Theall et al. 2022). Policies created by institutions such as the police should leverage the influence and effectiveness of community organizations to improve health outcomes by building trust (Pollack Potter et al. 2018). Ultimately, the most effective policy approach to reducing PCa vulnerability in middle and old-age Black men is multilayered and interconnected, as portrayed in the proposed model. Few interventions simultaneously address neighborhood conditions, social capital, and sleep as poignant pathways for reducing prostate cancer outcomes. The proposed

framework offers a rationale for such trials, including device-measured sleep, allostatic load biomarkers, and prostate cancer outcomes.

Limitations

The pathways in the proposed framework should be interpreted with the following limitations in mind. First, the discussion overemphasizes a unidirectional cascade from neighborhood conditions to social capital to sleep to prostate cancer vulnerability. It must be noted that reverse paths are plausible. For example, poor sleep health can lead to social withdrawal, eroding social capital, and a cancer diagnosis can reduce social networking and disrupt sleep. The framework includes bidirectional arrows and a feedback loop to acknowledge bidirectionality. Longitudinal data with at least 3 time points must be used to establish temporal precedence between the constructs and to test causality. Second, the framework does not explicitly identify potential confounders, such as healthcare access or individual health behaviors, like diet and exercise. These factors are known to impact most of the discussed constructs, and scholars testing this framework must include covariates when data is available. Third, the framework focuses on PCa health in Black middle- and old-age individuals.

Hence, its applicability to other types of cancer and to other populations is unaddressed. While the core logic is portable, the interactions among the factors and the net influence of each pathway may differ across groups. Hence, researchers using this framework for other cancers and populations should account for potential context-specific variations. Finally, the literature contains a few contradicting results for some pathways. While this is expected, it offers windows for contrary perspectives on the proposed paths. The proposed framework does not claim empirical confirmation; its value lies in providing falsifiable hypotheses to explain PCa outcomes and in including overlooked but crucial mediating pathways through sleep and social capital.

Conclusions

Prostate cancer disparities affecting Black men in the United States persist despite decades of cross-sectoral interventions. Disparities, ecological, and behavioral health frameworks offer guidance for studying and addressing this problem, but they remain descriptive, individualistic, and incomprehensive in terms of pathway specifications. The framework presented here is a theoretically grounded and mechanistically sound addition that offers testable pathways from neighborhood structural stressors to prostate cancer vulnerability in middle- and old-age Black men. The framework's core novel propositions are that: (1) social capital (bonding, bridging, and linking) mediated the neighborhood stress → sleep relationship; (2) sleep is a key proximal biological process linking social and neighborhood exposures to biological health outcomes; and (3) the path from neighborhood structural stress to PCa vulnerability is serially mediated. Addressing the persistent PCa health disparities in middle- and old-age Black men must confront the structural, relational, and biological realities of their lives. This framework is a guide towards that effort.

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