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REVIEW

The impact of social and environmental factors on cancer biology in Black Americans

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Abstract

Low socioeconomic status (SES) is associated with early onset of chronic diseases and reduced life expectancy. The involvement of neighborhood-level factors in defining cancer risk and outcomes for marginalized communities has been an active area of research for decades. Yet, the biological processes that underlie the impact of SES on chronic health conditions, such as cancer, remain poorly understood. To date, limited studies have shown that chronic life stress is more prevalent in low SES communities and can affect important molecular processes implicated in tumor biology such as DNA methylation, inflammation, and immune response. Further efforts to elucidate how neighborhood-level factors function physiologically to worsen cancer outcomes for disadvantaged communities are underway. This review provides an overview of the current literature on how socioenvironmental factors within neighborhoods contribute to more aggressive tumor biology, specifically in Black U.S. women and men, including the impact of environmental pollutants, neighborhood deprivation, social isolation, structural racism, and discrimination. We also summarize commonly used methods to measure deprivation, discrimination, and structural racism at the neighborhood-level in cancer health disparities research. Finally, we offer recommendations to adopt a multi-faceted intersectional approach to reduce cancer health disparities and develop effective interventions to promote health equity.

Keywords Neighborhood factors · Cancer disparities · Tumor biology · Neighborhood environment · Social environment

Introduction

Despite a decreasing cancer mortality for Black individuals living in the U.S. over the past 20 years, including African Americans and individuals of African descent living in the U.S., Black women and men continue to experience significantly higher overall cancer mortality rates than other U.S. population groups [1]. Targeted therapies and interventions to help ameliorate health disparities and promote health equity in the Black community are increasing. Still, are our current efforts sufficiently comprehensive to achieve health justice for this community? Black Americans have endured unimaginable hardships over centuries, from the atrocities of slavery to racist Jim Crow laws and discriminatory housing and financial practices, making upward mobility all but impossible for Black Americans. All these historical factors led to deprived and segregated living conditions of many Black communities, in which neighborhoods lack clean air, healthy food options, or access to adequate healthcare [2–5].

Research has shown that where we live has a profound impact on our health [6–8]. One long-term follow-up study found that neighborhood-level factors, including socioeconomic disadvantage and lack of healthcare access, translated to a 50% increase in all-cause mortality for those living in the most deprived neighborhoods [9]. This mortality rate increased to 90% when considering the mortality specific to prostate cancer. This trend has also been observed in other cancer types, including breast cancer, where Black women in low socioeconomic neighborhoods have greater odds of a late-stage diagnosis, higher grade tumors, more aggressive breast cancer subtypes, and increased mortality [10–13]. In the context of cancer, the influence of neighborhood factors on mortality literally translates to life or death.

How the neighborhood environment impacts cancer risk and mortality is complex and includes a myriad of social,

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environmental, economic, and structural factors that combine to produce more aggressive tumors and poorer cancer outcomes for these historically marginalized populations. For example, neighborhood-level redlining, defined as the historic and systematic denial of mortgage lending in certain neighborhoods with a high proportion of Black, foreign-born, or low-income residents [14], correlates with late-stage cancer diagnosis and an elevated risk of experiencing lethal breast, lung, cervical, and colorectal cancer [14–17]. Additionally, structural racism, or the way in which society perpetuates racist beliefs and practices through mutually reinforcing systems [18], has also been implicated as a causal factor in cancer disparities, accounting for a proportion of the differences in cancer survival outcomes between racial groups for certain cancer types [19, 20]. These studies and others reveal how structural racism, coupled with economic disinvestment in neighborhoods, can have a direct impact on the health of its residents.

A growing body of evidence points to ancestry-related, stress-dependent, and environmentally induced mechanisms that alter tumor biology and cancer survival outcomes, thereby contributing to cancer disparities. Many of the stress-dependent and environmental risks have their origin in both the physical and social environments. This narrative review primarily discusses literature published in the last 5 years. Our aims were to (1) summarize these findings as they relate to aggressive tumor biology; (2) review tools for measuring neighborhood-level factors as they relate to health disparities; and (3) discuss recommendations to mitigate the impact of these neighborhood-level factors on cancer biology and outcomes.

Methods to measure deprivation, discrimination, and structural racism at the neighborhood-level

With an increasing interest in health disparities research over the past decade, it is imperative that researchers avoid health equity tourism, or the pivot of inexperienced researchers into health equity work without the necessary expertise or sound methodologies to produce high-quality health disparities studies [21, 22]. We, as health disparities researchers, must not only ensure our study populations are well-represented in all relevant demographics to ensure statistically meaningful results, but also keep a health equity lens when designing these studies through careful consideration of the methods used to measure the factors that influence the health of historically marginalized populations. We will outline and discuss commonly used tools for measuring neighborhood-level factors, as these have been shown to impact cancer patients on many levels, including their cancer risk, care, and outcomes [23–26].

Neighborhood deprivation

The effects of neighborhood-level factors on health are an important consideration in health disparities research. Biological differences in cancer risk factors between population groups often originate from the neighborhood environment [27]. Researchers have developed several publicly shared deprivation indices intended to measure social and economic factors that determine a neighborhood's deprivation level. Many of these are based on tracts from the U.S. Census determined via participant residential zip code or address, while others use county level indicators as the unit of analysis. One of the most popular indices used to measure area socioeconomic deprivation is the Neighborhood Deprivation Index (NDI), introduced by Messer et al. [28]. The NDI uses Census-tracts as the unit of analysis as these are generally small, relatively permanent boundaries for counties, and designed to be homogenous with respect to social and economic factors [29]. Overall, the NDI uses data reduction methods to empirically summarize five key sociodemographic domains previously shown to be associated with health outcomes, including education, income/poverty, employment, housing, and occupation [28]. Similarly, the Area Deprivation Index (ADI) is another commonly used comprehensive index composed of 17 indicators within similar domains as the NDI [30, 31]. As a point of comparison, the NDI has been used longer and has been extensively implemented in the public health literature. In contrast, the ADI is newer, but is a database that includes all U.S. neighborhoods and is regularly maintained and updated. While there are other deprivation indices available [32–37], the NDI and ADI are two of the most comprehensive metrics of U.S.-based neighborhood deprivation currently used. For further discussion of area-based socioeconomic indices, several reviews, including a recent scoping review by Trinidad et al., compares these indices in greater detail [38, 39].

Racism and discrimination

The development of sound techniques for measuring both individual- and structural-level racism and discrimination are imperative to achieving health equity. This includes both quantitative and qualitative methods that are reproducible and able to be validated in many different contexts, including health disparities research [40]. On an individual level, racism and discrimination have been measured using various scales, each attempting to capture different facets of an individual's experience with perceived racism and discrimination. These include the Major and Everyday Discrimination Scales [41], the Experiences of Discrimination measure [42, 43], the Racial Microaggressions Scale [44], and the Race-related Events Scale [45], among others. Several

context-dependent adaptions of the Everyday Discrimination Scale are also routinely used, such as the Discrimination in Medical Settings scale [46–48], with the aim of capturing patient experience with perceived racial discrimination when receiving health care and services.

In a much broader context, measuring structural racism is a more arduous task, but it is crucial to breaking down the mutually reinforcing systems that perpetuate racist ideals and beliefs within societies, communities, and neighborhoods, which ultimately have an impact on health [40, 49, 50]. Several studies and commentaries have summarized prominent factors to consider when measuring structural racism, including residential segregation and housing discrimination, perceived racism in social institutions, SES, criminal justice, civil rights laws and legal racial discrimination, and workplace environment [51, 52]. While these components of structural racism have historically been modeled separately, the intersectionality of these measures is also an important consideration in determining the collective and interactive effects of structural racism on the health and cancer outcomes in historically marginalized populations [53–55]. To this end, a recent paper by Dougherty et al. created a multi-indicator scale to measure structural racism at the county-level in the U.S. using publicly available data [56], providing significant advancement in the development of methodological approaches to measure structural racism.

The impact of environmental factors on tumor biology

How the built environment impacts health and cancer outcomes has been widely studied for decades, especially considering that we spend most of our time either at home or at work. Living conditions, street layout, accessible green spaces, light at night, walkability, and other infrastructures affect food access and dietary choices, physical activity levels, environmental exposures, and behavioral and lifestyle habits [57–60]; which all directly impact our health, including our cancer risk. Little data exists, however, directly connecting these factors to tumor biology and more work is needed in this area. Here, we will describe how environmental factors, including pollution and neighborhood socioeconomic deprivation, impacts biological outcomes that either directly or indirectly lead to an altered tumor biology and adverse cancer outcomes. We will assess how environmental factors, including environmental pollution and neighborhood socioeconomic deprivation, impact biological processes that either directly or indirectly influence tumor biology and cancer outcomes. For the purpose of this review, we will concentrate on air pollution as an environmental risk factor, as there exists a more established mechanistic link between it and tumor biology compared to other environmental exposures, for which much of the available data is focused on cancer risk.

Environmental racism and air pollution

Residential segregation created and perpetuated by discriminatory housing policies in the U.S. has led to racial and ethnic minoritized groups to be concentrated in neighborhoods that have often been economically and politically disempowered [61]. A central side effect of this community disinvestment is lower commercial and residential property values, making land in these areas easier and cheaper to acquire for industrial interests. Proximity of predominantly Black communities to these industrial sites means greater exposure to the environmental hazards known to adversely impact human health, e.g., waste disposal, toxic dumping, oil and gas extraction, and close presence of power plants, petrochemical facilities, and Superfund sites. 70% of Superfund sites, which are areas declared to be severely contaminated by the U.S. Environmental Protection Agency (EPA), are located within one mile of federally-funded housing, in which a disproportionate number of low-income and Black Americans reside [62]. Further, intentional placement of highways, bus depots, landfills, and incinerators in historically segregated neighborhoods has resulted in significantly higher concentrations of air pollutants [63, 64]. Ultimately, the concentrated presence of these hazardous and undesirable factors lowers property values and thus perpetuates a vicious cycle of poverty and disproportionate environmental harm on Black communities in the U.S., now termed environmental racism. The insidious effects of environmental racism can adversely impact cancer risk, biology, and survival in several ways.

Exposure to outdoor air pollution poses an increasingly urgent public health challenge to all people living in the U.S. and worldwide; however, exposure to air pollutants and their detrimental impact on health is disproportionately high in certain communities. It is well-documented that air pollution is segregated by race and SES [65]. Large-scale reviews funded by the NIEHS have found unequal distribution of air pollution in the U.S., with greater exposure to air pollution in poorer communities [66, 67]. The findings identified air pollution as a contributor to health disparities [68]. Epidemiologic studies have reported an elevated risk of cancer driven by specific air pollutants, such as fine particulate matter that measure less than 2.5 microns (PM2.5) and coarse fraction particles that measure between 2.5 and 10 microns (PM10) [69]. Other commonly studied pollutants in cancer incidence and mortality include carbon monoxide (CO) [70], gaseous ozone (O3) [71], nitrogen dioxide (NO2) [72], pesticides, and a variety of others, all of which have been linked in various ways to increased cancer risk.

Representing a heterogeneous mixture of organic, inorganic, and biologic compounds [69, 73], PM2.5 is a one of the most used metrics for air quality and is actively studied in relation to cancer. The origin of these fine particles outdoors can be traced to automobiles, construction exhausts, and even power plants, all of which are elevated in polluted areas and thus, on average, more concentrated in communities with predominantly Black residents. While larger particulates, such as PM10, are generally caught in the nose and throat and not the lung [69], PM2.5 can be inhaled and deposited deeply into lung tissue, thus making it a major focus of lung cancer research. However, studies have shown PM2.5 may also increase the risk of developing multiple other cancers, including colon [74], prostate [75], and bladder, independent of cigarette smoking [76–78]. PM2.5 levels have also been positively associated with breast cancer incidence [79, 80], though epidemiologic evidence for this relationship has been somewhat less consistent and has been weakened by several null studies [81, 82]. Accordingly, PM2.5 has been designated an IARC Group 1 carcinogen. In addition to risk, it is well-established that air pollution levels have been shown to lead to poorer survival in cancer patients [79, 83, 84] A number of recent reviews provide an excellent, in-depth compilation of evidence linking both outdoor and indoor air pollution to inflammation [85], cancer [69, 86], and health [87].

The precise biological mechanisms that mediate the effects of various air pollutants in carcinogenesis and tumor biology are still being investigated and large gaps in knowledge exist. Most of the literature has historically focused on effects of various air pollutants along the respiratory tract. Several studies have found that air pollution, which contains groups of mutagenic and carcinogenic compounds, increases the formation of DNA adducts [88–90]. Further, several studies have identified inactivating somatic mutations and epigenetic silencing in *TP53* and other tumor suppressor genes that are linked to environmental exposures [91–94]. A recent study in 2020 by Letellier et al. uncovered associations between PM2.5 and NO_2 exposures that occurred 5- to 10-years prior to cancer diagnosis with somatic mutations in the *TP53* gene in non-small cell lung cancer patients [95]. Zhenzhen Wang et al. proposed an interesting mechanism based on their recent animal study, whereby fine particulate matter in air pollutants promoted lung cancer progression through thickening of the tissue matrix, which restricted infiltration by immune cells with antitumor activity. They identified a mediator of collagen IV crosslinking called peroxidasin (PXDN) as the enzyme responsible for this overly dense matrix [96].

Overall, air pollution has been linked to increased low grade inflammation [97] and oxidative stress [98], both of which have been found to initiate or exacerbate cancer across multiple cancer types. However, fewer studies have endeavored to examine air pollution beyond the respiratory tract. A 2019 study by Reyes-Caballero et al. demonstrated substantial metabolic dysregulation in glucose and lipid metabolism in the liver after exposure of mice to PM2.5 [99]. While this investigation was conducted in the context of insulin resistance and type II diabetes, its biological implication may extend to cancer where energetic dysregulation plays an important role in tumor biology.

Neighborhood deprivation

While several studies have investigated the link between neighborhood disadvantage and cancer risk and outcomes [100–105], there is a paucity of studies directly investigating the biological impact of neighborhood socioeconomic deprivation on the molecular underpinnings of tumors. This growing area of research is important, as knowledge of how the neighborhood influences biological pathways will broadly inform the cancer research community, beyond health disparity research, linking the environment to individual cancer risk, tumor characteristics, disease aggressiveness, and survival. This research will also be instrumental to establish both proof of causality and socioenvironmental influences as *bona fide* cancer risk factors.

One potential underlying pathway in which neighborhood deprivation influences cancer biology is through accelerated biological aging. Powell-Wiley et al. investigated the relationship between neighborhood deprivation and leukocyte telomere length using samples from the National Health and Nutrition Examination Surveys (NHANES). The length of telomeres, which are the protective caps that prevent the ends of chromosomes from deteriorating, are markers of biological aging and are associated with genomic instability and cancer risk [106]. This study found that individuals living in both medium and high deprivation neighborhoods had significantly shorter telomere length, pointing to accelerated aging as a potential biological mechanism for the negative impacts of socioeconomic disadvantage on health [107]. This hypothesis was tested in a cancer context in a more recent follow-up study, in which Shen et al. investigated the impact of neighborhood disadvantage on several markers of biological aging including allostatic load, telomere length, and DNA methylation in a cohort of recently diagnosed breast cancer patients [11]. They reported that individuals from a neighborhood with high deprivation were 20% more likely to experience an increased allostatic load, in addition to a large decrease in global methylation, when compared to individuals from a deprived neighborhood. They did not, however, find an association between neighborhood deprivation and telomere length in their study cohort [108, 109].

Other studies on the impact of neighborhood deprivation on telomere length observed that with each unit of improvement in neighborhood SES, there was a proportional

incremental decrease in telomere length attrition [110, 111]. Yet, a meta-analysis on this topic did not support the existence of a robust relationship between neighborhood disadvantage and premature aging defined by telomere length attrition [112]. Thus, the interrelationship between neighborhood deprivation and biological aging markers and cancer is likely complex. More studies are needed to determine the biological mechanisms mediating the negative effects of neighborhood deprivation on accelerated aging and its impact on cancer.

Chronic inflammation due to a deprived neighborhood environment has also been shown to have an impact on tumor biology. The studies that address this topic directly point to chronic stress as a leading driver of neighborhood deprivation-based inflammation, resulting in a more aggressive tumor biology [27, 63, 113, 114]. In addition to the negative behavioral habits that chronic stress imposes upon an individual that may increase cancer risk (e.g., tobacco use, alcohol consumption, poor diet), prolonged stress stimuli increase the allostatic load, a composite index commonly used to estimate stress-induced biological risk over the life course [115]. This triggers the development of stress-related diseases, such as cancer, through suppression of immunological responses via increased levels of circulating stress hormones, like catecholamines and glucocorticoids [27, 116, 117]. Additionally, individuals living in more deprived neighborhoods have been shown to experience increased levels of proinflammatory biomarkers, such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor receptor-2 [118–120]. Recent meta-analyses summarized the impact of SES on inflammatory markers, specifically CRP and IL-6, and found that lower SES associates with higher levels of these markers and systemic inflammation, defining inflammation as a candidate mechanism for the adverse impact of SES on overall health [121, 122]. Additional emphasis needs to be placed on the role of socioenvironmental determinants in influencing these inflammatory biomarker levels and stress response stimuli that can, in turn, exacerbate tumor aggressiveness and negatively influence cancer outcomes.

Effects of the social environment on tumor biology

Much like the physical environment, our social experiences can greatly impact our health including our stress levels, mental health, social relationships, and overall well-being. This is especially important in cancer, as several studies have confirmed that inadequate social support can lead to a marked increase in cancer mortality [123–125]. It has been postulated that this association stems from socially isolated individuals having decreased instrumental support regarding their cancer treatment and care. Additionally, socially supported individuals have the benefit of "social control", the concept that people with strong networks are healthier because negative health behaviors are discouraged [126, 127]. There has been evidence, however, that social experiences can also impact basic biological functioning which, in turn, could impact tumor biology. Here, we discuss how social factors, including social support networks and racial discrimination, impact biological function including cancer biology and outcomes.

Social environment

A person's social environment bears a great impact on health and wellness, both mental and physical. This extends to individuals living with cancer, where an adverse social environment, including social isolation, has been repeatedly associated with reduced survival across multiple cancer types [123, 124]. Social isolation, defined as a lack of social interaction, can be measured using criteria that is both objective (e.g., social network size, number/frequency of interactions with other individuals) and subjective (e.g., perceived social isolation or loneliness at the individual level) [127]. At the neighborhood level, social cohesion—which is defined as the network of relationships, shared values, and norms [128]—has been identified as a primary contributor to individual health [129]. Social cohesion is usually determined through subjective measures based on perceptions of trust, helpfulness, and other positive metrics among neighbors [130]. Traditional theories explaining the positive effects of both social cohesion and support on health outcomes have rested heavily on positive social influence to deter negative health behaviors using observational and intervention studies [126]. However, now that researchers have recapitulated these effects in animal models [131–133], the field has more recently embraced alternate explanations based in the physiological response to a positive social environment. While no causal mechanisms have been firmly established to date, research examining how cancer initiation and progression is influenced by the classical stress response through activation of the sympathetic nervous system and hypothalamic–pituitary–adrenal axis is an active area of study [134–137].

Social isolation has been documented as a chronic stressor and is associated with elevated stress-induced hormones [138]. Beta-adrenergic signaling has been shown to increase cancer invasion, survival, and angiogenesis, as well as modulate tumor-immune interactions [139]. This includes release of glucocorticoids, which have been associated with experiencing loneliness and the activation of downstream cellular processes [140]. Oxytocin, which is released during social and physical contact [126], has also been implicated as a mediator between stress and cancer, though direct evidence is still lacking. The effects of stress on cellular aging

have also been explored in this context, which is particularly relevant to Black individuals, as accelerated aging has been shown to be associated with racial discrimination [141, 142]. A study by Brody et al. found supportive family environments modify the effects between racial discrimination and epigenetic aging [143]. In a recent longitudinal study conducted in 2022 by Hailu et al. within the Multi-Ethnic Study of Atherosclerosis, neighborhood social cohesion modified the effect of discrimination on telomere length attrition in leukocytes [144]. While causal mechanisms remain to be established, some of the biological ramifications of the neighborhood social environment have been elucidated and appear to have a tangible impact on cancer progression and survival.

Racism and discrimination

Methods to measure racism and discrimination accurately and cohesively, including structural racism, are steadily emerging, which will bring forth new studies on how the health of marginalized communities are impacted by these discriminatory practices. Past studies have related racism and discrimination directly to cancer outcomes, with the overall consensus that living in areas with high levels of structural and institutional racism, both past and present, are associated with greater odds of more aggressive cancers and poorer survival outcomes for Black individuals [4, 20, 145]. The effects of racism and discrimination on cancer outcomes can be attributed not only to the impact that racism has on cancer care for Black individuals, but also the body's physical and mental response to the stress of perceived racism and discrimination. Recent studies have shown racial disparities in not only end-of-life care for individuals with metastatic cancer [146], but also the presence of high levels of distrust and negative attitudes among Black cancer patients toward physician practices, especially when the patient-physician relationship is racially-discordant or there are past patient experiences of medical racism [147–149]. Interventions to reduce medical bias, as well as improve patient beliefs/attitudes toward cancer care, will be imperative in improving cancer outcomes for marginalized populations.

From a biological perspective, there is a gap in the literature directly linking quantitative or qualitative measures of racism and discrimination to more aggressive tumor biology, specifically as it relates to Black individuals. Studies that have investigated the biological impact of racial discrimination on health implicated stress, depression, inflammation, immune response, and accelerated cellular aging as mediators in this relationship. A 2015 prospective cohort study on perceived racial discrimination on diurnal cortisol levels showed that the effects of perceived racial discrimination on cortisol levels were more pervasive for Black individuals, especially during adolescence when developmental periods are more sensitive [150]. Stress and depressive symptoms have also been shown to mediate the relationships between discrimination and smoking and discrimination and obesity, with smoking and obesity being on their own major risk factors for cancer [151, 152]. Additionally, the effects of racial discrimination on accelerated aging have mostly been performed in the context of telomere length, and much like the effects of neighborhood deprivation on health, several studies have shown an inverse relationship between increased early life racial discrimination and shorter telomere length, specifically among Black adolescent males [141, 142, 153, 154].

Coming back to inflammation, multiple studies have linked gender- and race-related discrimination to inflammatory markers (such as CRP and IL-6), concluding that racism and discrimination appear to have a direct impact on these markers after adjusting for relevant covariates and upregulate them [155–157]. Further studies have linked adverse cardiovascular outcomes to racism and discrimination in Black individuals, concluding that this population experiences an excessive risk of hypertension due to these discriminatory practices [158, 159]. Moreover, Black individuals may also experience an overall decline in health through continued exposure to social and economic disadvantage (also known as the "weathering hypothesis") [160–163]. Additional research is needed to better understand the biologic effects of racism and discrimination, especially structural racism, on cancer outcomes so that effective interventions can be created.

A path forward

Integrating exposures from both the social and environmental factors into studies that seek to understand the aggressive tumor biology in Black individuals from the U.S. is essential to reduce the excessive cancer mortality experienced by this population (see Fig. 1). This avenue of research can no longer be performed in silo. We will outline three recommendations for cancer disparities researchers to help eliminate health disparities and move toward health equity.

First, basic scientists must collaborate and communicate with experts in social and environmental epidemiology when addressing questions related to cancer disparities in marginalized populations. As this review has summarized, the worsened outcomes and more aggressive tumor biology in Black individuals with cancer is likely mediated by underlying biological processes stimulated by the effects of socioenvironmental factors (e.g., neighborhood deprivation, environmental hazards, racial discrimination). Therefore, we must encourage cross-training of cancer disparities researchers to be able to approach this complex problem

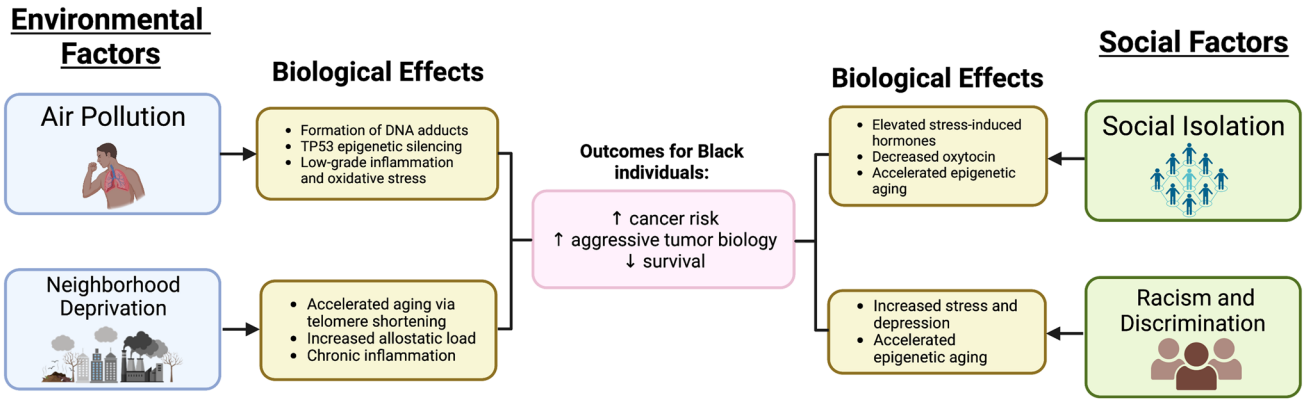

Fig. 1 Biological effects of the neighborhood environments that impact cancer outcomes for Black individuals. Created by Biorender.com

from multiple perspectives, including designing studies that assess both the social and biological contributors of more aggressive disease and worse cancer outcomes among Black Americans.

Second, we must embrace the fact that no one single exposure, social or biological, causes cancer disparities. These factors work in concert to inflict a milieu of biological effects on its host that makes tumors more aggressive and worsens outcomes for individuals from disadvantaged backgrounds. As such, we must now focus on the interaction between these exposures. Much like polygenic risk scores encompass all known relevant genetic factors to determine genetic risk for a specific disease state, polysocial risk scores must now be established, validated, and then integrated as a method to determine an individual's social risk for disease [164–166]. These scores should be updated over time, as the underlying risk profiles can fluctuate [167]. Moreover, continued and expanded research on exposomes, which describe the totality of exposures (both internal and external) impacting an organism's health over its life course, will be necessary to better understand the interactive and additive effects that social, psychosocial, and environmental components have on biological health outcomes, especially for individuals from marginalized communities [162, 168–170].

Third, the impact of these social and environmental factors on tumor biology will eventually lead to the identification of novel therapeutic targets that could hold promise for new treatments. However, it is critical to ensure equitable access to such treatments through promoting and encouraging the participation of systematically excluded populations in research studies and clinical trials, including as scientists and/or as study participants. This involves (1) increasing research funding and support for Black investigators at all levels, (2) carefully designing cancer disparities studies to be inclusive in both language and study population, and (3) actively engaging stakeholders, collaborators, and community advocates from minoritized populations as leaders on these studies.

The field of racial disparities in cancer has come a long way and has advanced in its scientific rigor with the use of more integrative approaches, but the work is far from over. Additional well-executed studies are needed to decipher the relationship between neighborhood-level factors and tumor biology as it relates to cancer outcomes. We must embrace the complexities and myriad of factors that define cancer risk, development, and progression, both socially and biologically, in diverse populations to design appropriate interventions and strategies to mitigate the impact of this disease on the most vulnerable communities in the U.S.

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Data availability All studies cited in this review are publicly available.

Declarations

Competing Interests The authors have no relevant financial or non-financial interests to disclose.

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Consent to participate Not applicable.

Consent for publication Not applicable.

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