PART 2.

MECHANISMS AND CONTEXT UNDERLYING SOCIAL INEQUALITIES IN CANCER

CHAPTER 8.

Theoretical frameworks and cancer inequities

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Introduction

Cancer inequities: this short phrase encompasses a plethora of ideas. It requires us to think about social injustice, populations, biology, the risk of disease and its treatment, survival, and death. To understand what makes population distributions of cancer inequitable, within and across populations and the places and time periods they inhabit, it is imperative to use theory, specifically, theories of disease distribution (Krieger, 2011). Such theory is critical to defining, analysing, and remedying health inequities, that is, social group differences in health that are unfair, unnecessary, and, in principle, preventable (Whitehead, 1991; Braveman and Gruskin, 2003). This is because in the case of science, it is theory that structures understanding of causal processes (Ziman, 2002; Krieger, 2011). Without theory, observation, explanation, and interventions are compromised and critical evaluation of the strengths and limitations of extant empirical evidence is undermined.

Although the centrality of theory to scientific observation and causal inference has been recognized for centuries (Ziman, 2002), until recently population health research on cancer and other outcomes has rarely been forthright about the theories of disease distribution informing study hypotheses, the interpretation of findings, and recommendations for action (Krieger, 1994, 2005, 2011, 2014; Wemrell et al., 2016). The central argument of this chapter is that theoretical blindfolds can lead to needless suffering and preventable deaths, and to the neglect or worsening of cancer and other health inequities. In this chapter, I deliberately refer to health inequities as opposed to health inequalities to underscore that theorizing is concerned with causal processes, agency, and accountability, and not solely empirical observation of differences.

The problematic dominant disregard for explicit theories of disease distribution and conceptualizing the societal causes of health inequities

For the past century the dominant approach to research and teaching in epidemiology, including cancer

epidemiology, has been to treat the discipline as a theory-free set of methods applied to health data (Krieger, 1994, 2011). The sources of the hypotheses being tested were seen as a matter of either common sense or inspiration, motivated by the available facts at hand.

What went without comment, and perhaps without recognition, was the pervasive theoretical orientation structuring the available facts and ways of thinking about them: that of the biomedical model (Table 8.1). Prioritizing the micro over the macro, both ideologically and technically, the biomedical model simultaneously (i) focuses on the physical, chemical, and biological causes of disease, and (ii) renders invisible how the societal context simultaneously shapes disease rates and the way their causes are conceptualized and analysed, and by whom (Tesh, 1988; Krieger, 1994, 2011; Greene and Loscalzo, 2017). If any social variables appear, they do so as individual risk factors and behavioural choices, framed by the complementary and equally individualistic lifestyle theory (Table 8.1) (Tesh, 1988; Krieger, 1994, 2011; Wemrell et al., 2016). Health inequities receive scant attention. Instead, observed physiological or other biological differences between social groups are largely recast as a matter of intrinsic (also known as genetic) difference, especially for race or ethnicity (Krieger, 1994, 2011).

Biomedical research fosters an aura of being more objective, precise, and potentially actionable, not to mention more scientific and prestigious, compared with the presumptively messier and more subjective research that addresses macro social phenomena that scientists by themselves cannot directly manipulate (even as scientists can contribute to and evaluate policy-relevant evidence) (Ziman, 2002; Krieger, 2011). There is an undeniable allure to use new tools of –omics, systems, and network biology to peer into cells, identify biomarkers of exposure and disease, and elucidate mechanisms involving biological development and pathological processes. New and exciting opportunities exist to study DNA expression and its regulation, the life-cycle of cells, and the functioning of and interactions between tissues (Gilbert and Epel, 2015; Greene and Loscalzo, 2017), and also to collect and analyse terabytes of health-relevant sensor, cell phone, Internet, and electronic medical record data (Mooney and Pejaver, 2018).

However, technological advances notwithstanding, in both biomedical and lifestyle research the individual remains entrenched as the unit of analysis (Krieger, 2011, 2014). Selection bias remains a potent problem; studies often lack sufficient social and economic diversity to encompass the etiologically relevant range of exposures and outcomes (O'Neil, 2016). Causal agents identified using older methods continue to wreak havoc on population health and health inequities, as exemplified by smoking-related diseases such as lung, oesophageal, and cervical cancer (Proctor, 2011). These persistent problems have spurred vigorous debate about the limits of biomedical and lifestyle theories, and have brought new prominence to theorizing about the societal determination of health and health inequities (Krieger, 1994, 2011, 2014; Berkman and Kawachi, 2000; Solar and Irwin, 2010; Wemrell et al., 2016). A central insight is that all science, whether at the micro or macro level, is conducted by people and incorporates people's value-laden (and often simplifying) assumptions about the world; it is explicit use of theory that enables these assumptions to become visible to and testable by independent investigators (Tesh, 1988; Ziman, 2002; Krieger, 2011).

Epidemiological theories of disease distribution for analysing health inequities

Table 8.1 lists key conceptual features of the three major sets of complementary theories of disease distribution in use in contemporary social epidemiology: sociopolitical, psychosocial, and ecosocial (Krieger, 1994, 2011, 2014; Solar and Irwin, 2010; Wemrell et al., 2016). All of these theories are concerned with the causal processes that give rise to health inequities. All reject the individualistic and decontextualized premises of the dominant biomedical and lifestyle theories, and all seek to promote health equity. Nevertheless, their emphases differ.

Sociopolitical theories

The common thread of the six sociopolitical theories listed in Table 8.1 is that they focus on analysing patterns of disease distribution in relation to power, politics, economics, and rights, and pay less (or no) attention to the biology involved in embodying social inequality. Among these theories, the three most explicit in terms of the political and economic drivers of health inequities are: social production of disease or political economy of health, Latin American social medicine or collective health, and critical epidemiology (also from Latin America) (Breilh, 2008; Krieger, 2011 [pp. 167–180, 187–190], 2014; Birn et al., 2017). By providing a frank analysis of who gains from and who

Table 8.1. Epidemiological theories of disease distribution: explicit theoretical focus

Theory	Disease mechanism	Health behaviour	Political economy and social production or reproduction of society	Politics, polities, and social movements	Social determinants (as factors)	Psychosocial exposures	Biological pathways of embodiment	Life-course	Levels	Place	Spatio-temporal scale	History and historical contingency	Ecosystem	Reflexivity: of theory and theorists
Dominant: decontextualized														
Biomedical	X	X												
Lifestyle		X												
Social epidemiology alternatives: contextualized														
Sociopolitical														
Social production of disease or political economy of health		X	X	X	X				X	X				X
Latin American social medicine or collective health		X	X	X	X	X			X	X		X		X
Critical epidemiology		X	X	X	X	X		X	X	X		X	X	X
Health and human rights		X		X	X				X					X
Population health or social determinants of health	X	X			X	X	X	X	X	X				
Fundamental cause		X			X	X			X					
Psychosocial	X	X			X	X	X	X	X					
Ecosocial and other ecologically oriented theories														
Eco-epidemiology	X	X			X	X	X	X	X	X			X	
Ecosocial	X	X	X	X	X	X	X	X	X	X	X	X	X	X

Source: reprinted from Krieger (2014) by permission from Springer Nature and adapted from Krieger (2011) by permission of Oxford University Press, USA.

is harmed by inequities involving power, wealth, and material resources, all three theories are rooted in European critiques of 19th and 20th century capitalism and imperialism and their imperative to maximize private profit. Intended to be applicable to any type of political economy, they also engage with "how political-economic systems and priorities that value social justice can produce health equity" (Krieger, 2011 [p. 167]). Forged under conditions of middle–late 20th century military dictatorships, the two Latin American theories have more similarities than differences. However, they focus more on the role of collectivities and popular movements in promoting health equity, as opposed to theories from the Global North that focus more on analysing and promoting state-led public health policies and actions (of a type not feasible under military dictatorship).

Another three of the sociopolitical theories – social determinants of health, population health, and fundamental cause – are also concerned with how social conditions shape population health profiles, but with little or no attention paid to the political economy of who gains from the status quo and at whose expense (Solar and Irwin, 2010; Krieger, 2011 [pp. 180–184], 2014; Birn et al., 2017). All three theories focus on finely calibrated social gradients in health, on social and status hierarchies, and on institutional policies and practices that affect the social and physical quality of where people live and work; none, however, explicitly name who benefits from injustice. For example, although the theories are concerned with the adverse impact of low income, they do not specify whose interests are served by low wages, reduced benefits, and austerity budgets. Of the three, theories on social determinants of health and on population health pay the most attention to biology, primarily in relation to the type and timing of exposures across the life-course from conception onwards. Fundamental cause, by contrast, treats specific exposures as superficial causes; its focus is the flexible resources people can use, such as knowledge, power, prestige, and interpersonal networks, to minimize health-related risks (Link and Phelan, 1995).

Also a sociopolitical theory, the health and human rights framework engages with how both promotion and violation of human rights by governments (and, increasingly, non-state actors) can affect individual and population health (Gruskin et al., 2007; Krieger, 2011 [pp. 190–191]). Based on the Universal Declaration of Human Rights issued by the United Nations in 1948 and aspects subsequently codified in international human rights laws, this theory analyses health inequities in relation to a range of social, political, civic, economic, and cultural rights held to be universal, interrelated, and indivisible. Accordingly, it offers unique tools to analyse the health impacts of government policies and hold governments accountable, including legally, for those impacts.

Psychosocial theories

The central focus of psychosocial theories is, as their name suggests, the health consequences of people's psychological perceptions of – and emotional and behavioural responses to – their social status, social interactions, and social conditions (Krieger, 2011 [pp. 191–201], 2014; Kubzansky et al., 2014). Building on a century of research on the biological responses of organisms to fear and other psychological stimuli, a major emphasis has been on the brain-mediated biology of stress and its physiological consequences across the life-course (and also intergenerationally, across the placenta). Attention is also given to stress-related health behaviours (e.g. eating, smoking, alcohol consumption, and use of other psychoactive substances). More recently, the scope of theorizing has expanded beyond the biology of stress to consider intersections between psychology, behavioural economics, and neuroscience, albeit without tackling political economy. The aim is to promote policies and institutional practices that can increase the likelihood that all people, not just those with resources (e.g. education and income), can engage in and maintain healthy behaviours (Kawachi, 2014).

Ecosocial theory

The ecosocial theory of disease distribution, first proposed in 1994 and elaborated upon since (Krieger, 1994, 2011, 2014), is an integrative social epidemiological theory that explicitly pays heed to: societal and ecological context; life-course and historical generation; spatiotemporal scales and levels of analysis; pathogenesis; and diverse forms of inequitable relationships within and between countries, including in relation to political economy, racism, class, sex, and sexuality. As illustrated in Fig. 8.1, a central focus is embodiment, referring to how we literally embody, biologically, our lived experience in a societal and ecological context, thereby creating population patterns of health and disease.

Another focus is accountability and agency, both for social inequalities in health and for ways they are (or are not) monitored, analysed,

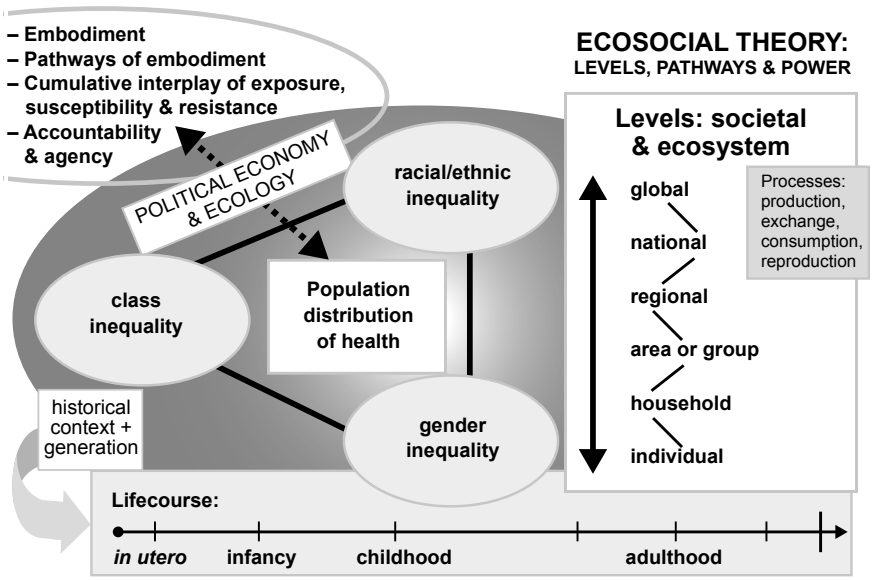
and addressed. Ecosocial theory shares with other social epidemiological theories of disease distribution a theoretical focus on political economy and the structural determination of material, social, and psychological exposures (both beneficial and adverse). In its ecological orientation, ecosocial theory expands beyond ecoepidemiology (which mainly theorizes about levels) (Susser and Susser, 1996; Lau et al., 2018) by explicitly including concepts and insights from fields such as political ecology, ecological evolutionary developmental biology, Indigenous traditional knowledge, and the history and philosophy of science. The point is not a theory of everything but rather a coherent set of conceptual principles and questions about causal processes to guide research. A starting point is recognition that all biological phenomena – including development, health, and evolution (Gilbert and Epel, 2015) – are necessarily expressions of biological embodiment in historical, societal, and ecological context.

Cancer inequities: why theories of disease distribution matter

Why do theories of disease distribution matter? They should spark their users to step back from the current roster of so-called facts and instead promote critical and creative causal thinking, to see who and what is missing (Krieger, 2011).

Selected examples are provided here to illustrate why theory, and choice of theory, matters for cancer inequities.

Fig. 8.1. Ecosocial theory and embodying inequality: core constructs, referring to processes conditional upon extant political economy and political ecology. Source: Krieger (2018).



1. ***Embodiment***, referring to how we literally incorporate, biologically, in societal and ecological context, the material and social world in which we live.

2. ***Pathways of embodiment***, via diverse, concurrent, and interacting pathways, involving: adverse exposure to social and economic deprivation; exogenous hazards (e.g. toxic substances, pathogens, and hazardous conditions); social trauma (e.g. discrimination and other forms of mental, physical, and sexual trauma); targeted marketing of harmful commodities (e.g. tobacco, alcohol, other licit and illicit drugs); inadequate or degrading health care; and degradation of ecosystems, including alienation of Indigenous populations from their lands.

3. ***Cumulative interplay of exposure, susceptibility, and resistance across the life-course***, referring to the importance of timing and accumulation of, plus responses to, embodied exposures, involving gene expression and not simply gene frequency.

4. ***Accountability and agency***, both for social disparities in health and research to explain these inequities.

Table 8.2. Cancer inequities: conceptual grid (Krieger, 2005 [p. 11]) for systematically reviewing evidence availability and gaps, using example of cervical cancer (Newmann and Garner, 2005 [p. 64]). The literature search identified only 45 articles with relevant data; the numbers in the table cells refers to the number of studies with relevant data for each cell (note that one study might have data relevant to more than one cell), and blank cells indicate that the literature review yielded no studies with relevant data.

Domains of social inequality	Prevention	Etiology	Screening	Diagnosis	Access to clinical trials	Treatment	Survival	Morbidity	Mortality
Race or ethnicity and racism	3		5[a]	4		2	4[a]	1	2
Socioeconomic position	1		4	3			1		1
Sex			3						
Sexuality		1	1						
Age				3		2			
Language			1						
Literacy	1								
Disability			2	1					
Immigrant status	4		9	1					
Insurance status			1	4			1		
Geography							1	1	1
Housing status			1						

[a] Contradictory evidence.
Source: reprinted from Newmann and Garner (2014) by permission from Springer Nature and adapted from Krieger (2005) by permission from Springer Nature.

Theory makes the invisible visible

To see theory in action, consider the conceptual grid (applied to cervical cancer) shown in Table 8.2. This grid was developed for a Dana Farber/Harvard Cancer Center interdisciplinary working group on cancer disparities (Krieger, 2005). Analytically informed by the ecosocial theory of disease distribution, the intent of the grid was to identify gaps in knowledge about cancer inequities across the cancer continuum by systematically addressing a specified set of "Domains of Social Inequality: singly & combined, involving adverse conditions & discrimination at multiple levels (person, place, institutional, societal), across the lifecourse" (Krieger, 2005). Used in relation to breast, prostate, colorectal, and cervical cancers (Bigby and Holmes, 2005; Gilligan, 2005; Newmann and Garner, 2005; Palmer and Schneider, 2005), the grid systematically reveals where evidence exists and where it is sparse, thereby helping to guide the next generation of research on cancer inequities (Koh, 2009).

Using social epidemiological theories to see data gaps is nothing new. In the early 1970s, such theories enabled researchers from Howard University, a historically Black university in Washington, DC, to shock the United States cancer establishment by reporting that cancer mortality among Black Americans since 1954 had grown by 32% compared with only 3% among White Americans (Fontaine et al., 1972; Henschke et al., 1973), a fact obscured by the then-routine reporting of solely non-White versus White data. The fallout galvanized the newly formed United States Surveillance, Epidemiology, and End Results Program for cancer statistics, launched in 1972, to ensure that its catchment sites could include and report data for "diverse ethnic subgroups" (Wailoo, 2011 [pp. 120–145]).

Theory guides choices of metrics for monitoring

Theory can also be useful for identifying the kinds of variables needed to monitor health inequities. For example, theories that explicitly address structural racism, such as ecosocial theory and political economy of health, point to the utility of monitoring economic and racial or ethnic cancer inequities using not only conventional individual- and

household-level socioeconomic measures but also measures of economic and racial or ethnic polarization at the neighbourhood, city, or town, and regional levels; these latter measures keep in view the privileged who benefit from inequitable relations, and not just those harmed by these inequities.

One such metric is the Index of Concentration at the Extremes (ICE), which quantifies the extent to which an area's residents are concentrated into groups of extreme levels of high versus low economic or social privilege (Massey, 2001; Krieger et al., 2016). For example, the recently developed ICE for racialized economic segregation quantifies the extent to which an area's residents are concentrated into the extremes of affluent racially privileged groups versus impoverished racially oppressed groups; it can also be used to quantify solely economic or racial polarization (Krieger et al., 2016). Notably, these ICE measures can be meaningfully used at multiple geographical sociopolitical levels, from residential neighbourhood to city or town to region to state. This is in contrast to the more widely used Gini index for income inequality and the Index of Dissimilarity for racial segregation, which are uninformative for small areas precisely because of how segregation reduces inequality within such areas by increasing spatial social polarization (Massey, 2001; Krieger et al., 2016). In an era of growing economic, social, and spatial polarization within and between countries, measures that keep in focus the full range of privilege and deprivation will be crucial to global monitoring and analysis of cancer and other health inequities (Galster and Sharkey, 2017; Krieger, 2017).

Theory illuminates spatiotemporal scale and level in a historical context

Theory can also spark research to improve understanding of the historical, place-based, and sociopolitical dimensions of current cancer inequities in biomarkers and molecular phenotypes. For example, the ecosocial theory of disease distribution prompts the following four questions (source: Krieger, 2013 [p. 23]).

- ***"Question 1: Societal history.*** What data exist on historical trends in the average population rates of—and health inequities in—the embodied biomarker or outcome? (For example, between and within countries and regions, defined geopolitically and in relation to societal divisions involving property, power, resources, and discrimination, including socioeconomic position, race/ethnicity, Indigenous status, gender, sexuality, disability, nativity, and immigrant status.)
- ***"Question 2: Individual (life course) history.*** What is the "natural"—and "unnatural"—history of the embodied biomarker or outcome across a person's life course? Does its expression change over time for a given course of illness, or across repeat bouts of an illness? Does its expression vary by the societal groups considered in Question 1 (i.e., display health inequities)?
- ***"Question 3: Pathological/cellular history.*** What is the "natural"—and "unnatural"—history of the embodied biomarker analyzed at the level of the tissue(s) involved? Does its expression change over the course of the disease? Or vary by the societal groups considered in Question 1 (i.e., display health inequities)?
- ***"Question 4: Evolutionary history.*** What is known—and debated—about the evolutionary history of the embodied biomarker or outcome under analysis? What insight does this history provide regarding the likely dynamics of expression, within and across individuals, historical generations, and societal groups?"

In the case of the estrogen receptor (ER), which plays an important role in breast cancer, research motivated by such questions readily reveals the fallacies of prevalent biomedical assumptions about alleged innate racial differences underlying observed Black (or African) versus White (or European or Euro-American) differences (Iqbal et al., 2015; Newman, 2015). The scant data on population distributions of this biomarker in a handful of African countries show wildly divergent prevalences of ER-positive and -negative tumours (Eng et al., 2014). Within the USA, Black versus White patterns of breast cancer ER status (and their pace of change, by biological generation) have been shown to vary by historical period, place of birth (states with vs without legal racial discrimination ["Jim Crow"]), socioeconomic position, and both access to and quality of medical care (Krieger et al., 2011, 2018, 2017; Krieger, 2013; Kohler et al., 2015; Rauscher et al., 2016). The evolutionary history of ER further suggests that its expression would be highly sensitive to extracellular signals, for example, hormonal medications, or exposure to periods of famine and great destitution (Krieger, 2013; Krieger et al., 2017). Theory makes the distinction between seeing a difference as fixed and an inequity that can be modified.

Theory pinpoints accountability and agency

Finally, in a period of mounting conservative and corporate-led attacks on public health, on environmental regulations that limit exposure to carcinogens and other adverse substances, and on the science of global climate change (Freudenberg, 2014; Birn et al., 2017), social epidemiological theories that identify the culprits and their motives are crucial for building alliances to protect the health of the people and that of this planet (Birn et al., 2017; Klein, 2017).

Conclusions

In conclusion, the rationale for explicit use of social epidemiological theories of disease distribution for the analysis of cancer inequities is not a faddish concern with conducting politically correct science; it is, instead, a concern to conduct correct science (Krieger, 2011). The ultimate test of the knowledge produced is whether it aids the collective tasks of (i) imagining a world free of health inequities; (ii) identifying the obstacles to health equity; and (iii) equitably engaging all who must work together to bring about a kinder, healthier, more equitable, and more sustainable human world, informed by deep recognition of our interconnection with, and dependence on, our wondrous and threatened planet.

Key points

- Explicit use of social epidemiological theories of disease distribution is critical for defining, analysing, and remedying health inequities, that is, social group differences in health that are unfair, unnecessary, and, in principle, preventable.
- The three major sets of complementary theories of disease distribution used in contemporary social epidemiology are: sociopolitical, psychosocial, and ecosocial.
- Rigorous use of social epidemiological theories that identify the obstacles to health equity are crucial for building alliances to protect the health of people and that of this planet.

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