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Embodiment and Ecosocial Theory

Nancy Krieger interviewed by Kerstin Palm, Sigrid Schmitz and Marion Mangelsdorf

Nancy Krieger is a Professor of Social Epidemiology at the Harvard School of Public Health in Boston, Massachusetts, where she has been researching the relationship between group-specific disease rates and social inequality. One focus of her areas of specialty is gender-specific aspects of health, in particular on the relationship between biological and social factors in health research (sex-linked biology and gender relations), including in relation to social class, racism, and other societal determinants of health. The relevance of the central concepts in her work, especially "embodiment" and "ecosocial theory," reaches far beyond the boundaries of health research and could be used to develop a basis for research in all life sciences, as well as interdisciplinary projects on bodies in context.

The following interview highlights some central theoretical and methodical aspects of her approach which can offer interesting possibilities for an integrative ecosocial perspective, drawing on social and biological insights regarding the development of bodily features.

1. To overcome the relative poverty of theory in the field of epidemiology and develop an integrative perspective linking social and biological sciences, you developed the concept of ecosocial theory. Could you explain to us what ecosocial theory is? What are its most important characteristics?

Developed to address the question "who and what drives social inequalities in health," a central focus of ecosocial theory is on how we literally biologically embody exposures arising from our societal and ecological context, thereby producing population rates and distributions of health. At issue are socially patterned exposure-induced pathogenic pathways, mediated by physiology, behavior, and gene expression, that affect the development, growth, regulation, and death of our body's biological systems, organs, and cells, culminating in disease, disability, and death. The contrast is to frameworks that treat causes of disease – and of group differences in biological characteristics and disease rates – as primarily innate, e.g., as long argued for racial/ethnic health inequities.

Indeed, integral to ecosocial theory – and part of its concerns with agency and accountability – is a painful awareness of the contested history of scientific ideas and practice, whereby eminent scientists, including in the health sciences, have been just as or more likely to develop and use scientific frameworks that justify, rather than question, discrimination and social inequality. Well-documented examples about which reams have been written include eugenics broadly and scientific racism, sexism, and heterosexism in particular.

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Ecosocial theory accordingly requires explicit consideration of pathways of embodiment in relation to types and levels of exposure, the period and spatial expanse involved (i.e., spatiotemporal scale), and historical context, along with phenomena that affect susceptibility and resistance to exposure, ranging from micro (e.g., role of the gut microbiome in innate immunity) to macro (e.g., social organizing to challenge health inequities). Also core are issues of accountability (causal responsibility for) and agency (the power and ability to act) at every level, because they pertain to not only the magnitude of health inequities but also how they are monitored, analyzed, and addressed. Moreover, as emphasized by ecosocial theory's simultaneous focus on exposure, susceptibility, and resistance – including how people resist injustice and its health-harming effects, individually and collectively, and the resilience that enables them to do so – also must be examined. Historical context in turn determines which pathways matter and are operative, at what level and at what point in the life course, such that the embodied manifestations are necessarily historically contingent, both the health outcomes themselves and the magnitude of their health inequities.

A critical knowledge of history is thus essential: the history of the exposures and outcomes under consideration, and the history of contending ways in which scientists have, in the context of their times, debated possible causal links. As with any scientific theory, the point is to frame and guide analysis of the phenomena of interest – in this case, population distributions of health, disease, disability, and well-being – and, as with any reflexive science, to generate knowledge relevant to altering the phenomena under study, in this case, the existence of health inequities.

The point is not that every study can or should attempt to measure every specified pathway at every level and at all relevant spatiotemporal scales – which obviously is impossible to do. Rather, the value of a theoretical framework is that it can help concretize systematic substantive thinking about potential causal pathways, the constructs and entities employed and how they are operationalized and measured, the types of statistical analyses should be conducted, potential threats to validity, and the complexities involved in interpreting study findings.

2. You introduce one of your papers on embodiment with the statement "our bodies tell stories about our lives." Could you explain what you understand by the term "embodiment" and how this concept can help us to better understand the interaction or relationship between sex-linked biology and gender relations? How do you see the current acceptance of this (or a similar) concept in the field of public health?

At the most general level, embodiment refers to how we, like any living organism, literally incorporate, biologically, the world in which we live, including our societal and ecological circumstances. In the case of population health, for both people and other species, it recognizes that individuals are: (1) simultaneously ever-developing social beings and biological organisms, and (2) inherently mem-

bers of populations and are shaped by and also shape both their membership in these populations and the ecosystems in which they are engaged.

Population characteristics expressed by people's bodies (such as health and disease status) are thus not simply the sum of a priori individual traits, even as population rates are tallied up based on counts of individuals' bodies (who constitute the numerators and the denominators for these rates). They instead emerge from the dynamic development of individuals belonging to social groups that are influenced by and affect their societal and ecological contexts. One important causal implication accordingly involves the well-known population science insight that what explains differences between populations often is not the same as what explains variation among individuals within a given population.

Consider the classic example of lung cancer: as Geoffrey Rose famously argued in 1985, in a population in which everyone smoked, exposure to smoking would not explain risk of disease (because there is no variation in the exposure), even as it was the reason for the high rates of lung cancer in this population as compared to a population in which no one smoked. Additionally, for lung cancer, as for any health issue, it is important to ask whether gender relations, sex-linked biology, both, or neither are relevant as potentially independent or synergistic determinants of population rates. Ample research, for example, demonstrates that gender relations clearly matter for creating differences in risk of lung cancer among women and men, with gendered differentials in rates and histories of smoking (individually and across generations) shaped in part by gendered marketing practices of tobacco companies (who have notoriously promoted smoking as a way to demonstrate one's masculinity, for men, and one's independence, for women), as well as gendered differentials in exposure to other exogenous agents implicated in causing lung cancer (e.g., occupational exposures to asbestos, nickel, and chromium, occurring in industries which, due to occupational gender segregation, predominantly employ men). As for sex-linked biology, although some investigators argue women may be intrinsically more biologically susceptible to risk of lung cancer at low levels of smoking exposure compared to men (usually invoking hormonal hypotheses to explain why), large epidemiologic studies do not uphold this hypothesis and also raise important questions as to the accuracy of measuring life-time history of exposure. At issue is not only individuals' self-report of an increasingly socially unacceptable health behavior, but also exposure to second-hand smoke, which may be more common for women, if they are part of households in which men – e.g., father, partner, son(s), or others – are smoking and they are not. Differentials in lung cancer rates among women and men can thus primarily be understood, via the construct of embodiment, as a biological expression of gender – thereby keeping attention focused on who and what drives the gendered patterns of exposure, for whose benefit and at whose cost. Such knowledge is in turn critical for formulating effective population-level strategies to reduce smoking, as required for challenging the highly gender-conscious tobacco industry, for whom gender has been a key motif of advertising since the early 20th century CE: to keep rates high among men and get rates among women to rise.

Whether used literally or figuratively, embodiment thus insists on bodies as active and engaged entities. It consequently reconceptualizes the individual and population phenomena of health and disease (both somatic and mental) as emergent embodied phenotype, one that is contingent on population context, and hence embodied history. The contrast is to dominant gene-centric paradigms which give primary causal agency to an organism's inherited genotype and emphasize a decontextualized and dehistoricized biology.

Additionally, in the case of people, the construct of embodiment recognizes that bodies tell stories about our lives, whether or not these are ever consciously expressed. For example, an infant is not conscious of its birthweight or gestational age, even as both may be relevant to infant and adult health. Considerable research further indicates that persons who are abused may not necessarily identify their treatment in this way, instead considering it to be "deserved" – even as their health behaviors and health status nevertheless manifest the impact of such abuse. And, in the case of the lung cancer example above, people can be exposed to carcinogens whether or not they are conscious of this exposure. For these reasons and more, bodies can tell stories – and reveal histories – above and beyond what our words can express.

Finally, my sense is that the idea of embodiment as articulated in ecosocial theory is beginning to gain a footing in public health and related fields. Recent relevant examples of books and articles that explicitly draw on the ecosocial construct of "embodiment" include:

Gravlee, Clarence C. (2009): How race becomes biology: embodiment of social inequality. American Journal of Physical Anthropoly 139, 1, pp. 47-57.

Roberts, Dorothy (2011): Fatal Invention: How Science, Politics, and Big Business Re-Create Race in the Twenty-First Century. New York: The New Press.

Walters, Karina L./Mohammed, Selina A./Evans-Campbell Teresa/Beltrán, Ramona, E./Chae, David H./Duran, Bonnie (2011): Bodies don't just tell stories, they tell histories: embodiment of historical trauma among American Indians and Alaska Natives. In: Du Bois Review 8, 1, pp. 179-189.

Connell, Raewyn (2012): Gender, health and theory: conceptualizing the issue, in local and world perspective. In: Social Science Medicine 74, 11, pp. 1675-1683.

Coburn, Jason (2013): Healthy City Planning: From Neighborhood to National Health Equity. New York: Routledge.

3. Until recently, (mainstream) epidemiological research tended to explain a higher group-specific prevalence of disease (for example, high rates of depression among women or cardiovascular disease among African Americans) by referencing the interaction of race- and sex-specific biological predisposition with exposures. You criticize this interpretation and suggest other explanations, which include social conditions and contexts of racism and sexism. Could you summarize your critique and illustrate it using a short example?

One example involving embodiment, biological expressions of racism and sexism, and racialized and gendered expresssions of biology, concerns that of the breast

cancer estrogen receptor. As shown by any quick perusal of the breast cancer epidemiology literature (predominantly US, but also increasingly global), one routinely repeated "fact" is that black women, as such, are less likely than white women, as such, to have estrogen receptor positive (ER+) tumors, for reasons explicitly stated to involve genetic predisposition (i.e., ostensibly innate biology). The ER status of a breast tumor is important, because it is directly linked to breast cancer survival, given current treatment options. Briefly stated, ER+ tumors can be treated with anti-estrogenic chemotherapeutic drugs, such as tamoxifen and raloxifene, whereas ER- tumors cannot – and, because these are the main drugs used to treat breast cancer, this treatment difference in part contributes to the poorer survival of women with ER- compared to ER+ tumors. Thus, the conventional causal logic is that black women, by virtue of being black, are less likely to have ER+ tumors and this is a key reason for why, in the US, their breast cancer survival is worse than that of white women (above and beyond black women being less likely to be able to have adequate access to health care).

Consider, however, the recent research demonstrating that use of hormone therapy is more likely to increase not only risk of breast cancer, but specifically ER+ breast cancer. As critically analyzed by a considerable body of critical work on gender, sexism, and biomedicine, in the mid-1960s the framing of menopause as a "hormonal deficiency disease" that could be treated by "hormone replacement therapy" (HRT) gained ascendance, and sales of HRT rocketed, with only a brief lull in the 1970s when evidence indicated the kind of formulation then used (estrogen only) increased risk of uterine cancer. Newly formulated to include progestin as well as estrogen (a combination that did not increase risk of uterine cancer), sales resumed, with uptake in the US much higher among affluent women who could afford to pay for prescriptions for HRT and who did not suffer from conditions which precluded their being prescribed these drugs – such that, not surprisingly, HRT users were predominantly white, healthy, affluent women. Starting in the 1980s, popularity of HRT increased given rising claims that it was a "preventive" medicine that could ward off cardiovascular disease, such that prescribing HRT became the standard of care, notwithstanding epidemiologic research indicating it could increase risk of breast cancer.

In 2002, however, publication of the results of the US Women's Health Initiative completely turned the tables on HRT use, with results showing not only that it not only did not prevent, but may have elevated, risk of cardiovascular disease, as well as reconfirmed earlier findings that it increased risk of breast cancer. Sales of HRT plummeted and its name also became shorter: the treatment was reconceptualized and renamed "hormone therapy" (HT) – and became a treatment that was supposed to be reserved for women experiencing severe effects of menopausal transition (e.g., hot flashes leading to severe sleep deprivation), as opposed to being a treatment for all women to "replace" the lower estrogen levels that were part and parcel of becoming and being menopausal. Major debates continue to play out in the literature regarding which women, if any, should be prescribed HT, with some researchers still advocating their more general use, and with pharmaceutical companies continuing to fund research to

answer this question (which is not surprising, given what an important source of revenue HRT provided).

One biological as well as logical implication of these changed trends in HT use would be that breast cancer incidence would fall – which it did, with this fall not surprisingly occurring not among "women" in general, but rather among those women who used HT. In the US, this translated to a decline in breast cancer incidence following publication of the WHI chiefly among white affluent women age 50 and older, as my research team and I have shown. A less well appreciated consequence of the changing pattern of HT use, however, was its impact on the white versus black risk for being ER+ among women diagnosed with breast cancer. As we have also demonstrated, far from the white versus black risk being a stable and invariant, this risk changed over time: it increased from 1992 to 2002, and then declined, especially among women ages 45 to 54. Our results, the first to test rigorously changing estimates over time for the white versus black risk for being ER+, thus simultaneously challenge notions of innate racial difference while also providing evidence of how gender relations – as per the mid-1960s call for women to be "forever feminine" via promotion of use of HT – affected the risk and biological expression of breast cancer among women.

4. You view the experience of discrimination as having a central influence on health. In order to access this aspect in the framework of an empirical-quantitative approach you successfully developed and implemented various instruments for collecting data such as the EOD (Experience of Discrimination) and the IAT (Implicit Association Test). Could you please describe how these instruments work and what outcomes can be produced using them?

As clarified by ecosocial theory, there is not just one way that racism and other forms of discrimination can harm health: there are many. In the case of racism and health, relevant pathways include: (1) economic and social deprivation; (2) excess exposure to toxins, hazards, and pathogens; (3) social trauma; (4) health-harming responses to discrimination; (5) targeted marketing of harmful commodities; (6) inadequate medical care; and (7) especially (but not only) for Indigenous peoples, ecosystem degradation and alienation from the land. The implication is that diverse kinds of research are needed to study how inequitable race relations, i.e., structural racism, at multiple levels and over historical generations, can adversely affect health, as expressed in both people's exposure and responses to institutional discrimination and its translations to inter-individual and internalized racism.

As revealed by a 2013 literature search I have just completed for an update to an article I published in 1999 that was the first epidemiologic review article on discrimination and health, the vast majority of current research on discrimination and health focuses on psychosocial exposures at the individual level. More research on structural racism is clearly required, to gain a better understanding of how racism affects population health. One example is a study my team and I have just completed that is now in press and which will be published soon, that

demonstrates, using new methods and extended data sets, the beneficial impact of the abolition of Jim Crow (legal racial discrimination in the US, abolished by the 1964 US Civil Rights Act) on black infant mortality (noting that to date only 5 prior empirical studies have examined the health consequences of the abolition of Jim Crow, despite this being an epoch-changing event with enormous implications for US population health). That said, insofar as research continues to be done with individual study participants, whether in quantitative or qualitative studies, it is important to be aware of and address the many complexities of obtaining valid data on people's exposure to discrimination.

I initially developed what is now called the "Experiences of Discrimination" (EOD) measure in the late 1980s, prompted by a lack of research instruments measuring self-reported experiences of racial discrimination that could be feasibly used in population health studies that obtain data on hundreds if not thousands of individuals. Its emphasis was and continues to be on identifying domains in which individuals have experienced discrimination, both because specification of domains is important for cognitively grounding the questions and also because of key ecosocial concerns regarding accountability and agency: the occurrence of discrimination in diverse domains, such as discrimination at work, in housing, in education, and in health care, is legally actionable, and knowing where discrimination occurs, as opposed to treating it only as a free-floating psychosocial stressor, is relevant to ending it. The 9 domains included for racial discrimination thus are: (1) at school, (2) getting hired or getting a job, (3) at work, (4) getting housing, (5) getting medical care, (6) getting service in a store or restaurant, (7) getting credit, bank loans, or a mortgage, (8) on the street or in a public setting, and (9) from the police or in the courts. Additional questions pertain to frequency of occurrence, how someone typically responds (accept it as a fact of life versus take action; keep it to oneself versus talk to others), and also worries about racial discrimination as a child and as an adult, for oneself and for one's racial/ethnic group. To date, higher exposure to racial discrimination as measured by the EOD has been associated with increased risk of elevated blood pressure, hypertension, preterm delivery, cigarette smoking, illicit drug use, self-reported health, and psychological distress.

Answers to the EOD cannot, however, be taken simply at face-value, even as they are important. At issue are two phenomena: (1) among persons who belong to groups that have historically been and/or currently are subjected to discrimination, individuals may not be willing or able to self-report their experiences of discrimination, and (2) among persons who belong to groups that have historically been and/or currently are the perpetrators of discrimination, individuals may claim that societal remedies, such as affirmative action, constitute forms of "reverse discrimination" and thus reframe themselves as the target of discrimination.

As one partial check on these threats to validity, it is thus always essential to pair explicit self-report questions on experiences of discrimination with measures of social desirability, referring to how likely people are to give an answer they believe is "socially acceptable" as opposed to what they truly believe. The reason for doing so is that considerable research shows that people with less

power not surprisingly rank higher on "social desirability" scales, since those with more power feel most able and entitled to say what they truly think. In a recent study on racial discrimination and health that employed a social desirability scale, for example, we found that the social desirability score was highest among black women and men, with little difference by socioeconomic position, with the only white group to have similarly high scores being white women with few socioeconomic resources, and the lowest scores occurring among the white men with the most socioeconomic resources.

One of the newer approaches in the discrimination and health literature that seeks to minimize well-known cognitive problems affecting self-report data is the Implicit Association Test (IAT). Initially developed to measure people's racial preferences (e.g., black versus white), my team and I have adapted it to measure people's sense of themselves and their group as a target versus perpetrator of racial discrimination.

In brief, the IAT is a computer-based reaction-time methodology designed to capture phenomena that lie outside of the reaches of introspective access. The test contrasts the time it takes to make associations between two sets of items, e.g., "flowers" with the word "good," and "bugs" with the word "bad" – and then compares what happens when participants alternatively are asked to pair "flower" with "bad" and "bugs" with "good." A difference in average matching speed for opposite pairings determines the IAT score. Participants are typically aware that they are making these connections but are unable to control them given the rapid response times and structure of the test. More than 500 studies have employed numerous versions of the IAT and have found the results to be robust, especially for phenomena that are subject to social desirability.

For our IAT, which we have employed in studies in involving US black and white participants, we used two sets of targets. First, for discrimination against oneself, the measure used the pronouns "me," "my," "mine," "them," "their," and "theirs." Second, for discrimination against one's group, we used photos of black and white persons. For both measures, the attribute categorization words were: "abuser," "racist," "bigot," "target," "victim," and "oppressed." Using these measures, we could ascertain the differences in strength of association for being a perpetrator versus target of discrimination. To date, we not only have found, as expected, a low correlation between the explicit (EOD) and implicit (IAT) measures of exposure to racial discrimination, but we have also shown the two measures are independently associated with risk of hypertension among black but not white Americans. Additionally, controlling for the EOD and IAT eliminated the excess risk of hypertension among black compared to white participants observed in models that controlled for age, gender, socioeconomic position, body mass index, social desirability, and response to unfair treatment. These preliminary results thus point to the likely utility of health research on discrimination supplementing self-report data with IAT data.

5. In some of your papers you have argued that the lack of an ecosocial perspective in the way (mainstream) epidemiology conducts research not only leads to fewer results, (in particular to a reduced number of plausible explanations

for rates of disease prevalence), but also propagates and legitimizes increasing social inequality. Could you please explain more clearly why that is?

One germane example concerns claims arising in the mid-2000s that, in a context of on-average overall health improving, growing health inequities were not a big deal, but instead just an inevitable result of health getting better for everyone, albeit more quickly for better-off people. Trickle-down health, in other words. Guided by ecosocial theory, I suspected this claim was short-sighted, due in part to reliance on only recent data that necessarily reflected the post-1980 ascendance of neoliberal policies. My team and I thus went about testing this claim by examining trends in US premature mortality (i.e., death before age 65), using US national mortality data for the period 1960 to 2006. What we found was that, contrary to the claims of the "trickle-down health" hypothesis, in fact racial/ethnic and income inequities in premature mortality shrank between 1965 and 1980, when mortality rates were also declining for everyone, and only thereafter stagnated then widened. Likely contributing to these trends was the enactment of the US Civil Rights Act in 1964, which overturned Jim Crow, i.e., legal racial discrimination, also the 1965 US Voting Rights Act, and other progressive legislation involving the "War on Poverty," along with creation of Medicare, Medicaid, the Environmental Protection Agency (EPA) and the Occupational Safety and Health Administration (OSHA), with these progressive changes then subjected to challenge during a long-period of backlash, starting under the Carter presidency in the latter 1970s and taken to new levels during the Reagan administration (commencing in 1980) and continued thereafter.

Despite this suggestive evidence that political priorities play a large role in driving both trends in on-average population health and health inequities, nevertheless mainstream accounts tend to focus on smoking and access to appropriate medical care as being the key drivers, as per the conclusions of a recent National Academy of Sciences report. I accordingly decided it would be useful to examine trends in *non-smoking* mortality (i.e., all causes of death NOT included among the 24 causes of death listed in the 2008 U.S. CDC report on smoking related causes of death). Here it is critical to flag the hugely important success of anti-smoking campaigns, whereby between 1960 and 2006, the proportion of deaths in the US not due to causes of death linked to smoking rose from approximately 40% to 60%; of note, it not only rose in all income quintiles, but the proportions throughout were highest among US populations of color, reflecting their higher exposure to many adverse living and working conditions independent of cigarette smoking. What we found was that in the total US population and also among both the white population and populations of color, the magnitude of income inequities in mortality for deaths *not* due to smoking has *grown*. Smoking thus cannot be the full explanation for observed trends in US health inequities.

Next, consider the patterns we observed for US mortality rates for what is deemed medically preventable mortality, i.e., deaths that should not happen were someone to receive accepted standards of medical care (and so: prevention of death, given illness, not primary prevention). For these analyses, also using

data from 1960-2008, we used the same 14 causes of medically preventable deaths that were considered preventable by 1960s standard of medical care. Two findings stand out. First, rates of mortality due to these causes *did* decline over time, in all income quintiles in both the US white population and populations of color. That said, the temporal pattern was one of steep declines between 1965 and 1980, followed by stagnation until about 1997, after which followed a new period of less steep decline. Such results are explicable only by societal factors, e.g., access to appropriate medical care, not by technology (which improved throughout the period).

As these examples and the others I have provided make clear, challenging claims that health inequities are natural and normal, let alone bound to rise, requires attention not simply to the political ideologies that might inform such views, but to the theoretical and methodological assumptions built into the research. Theory is needed not only to see and critique these assumptions but also to offer alternative causal explanations and to generate and employ the methods by which these alternatives can be tested. As explained in my response to question 1, the ecosocial theory of disease distribution, with its emphasis on embodiment, offers one such useful framework for advancing work on analyzing who and what causes health inequities and generating insights useful for promoting health equity.

Selected bibliography

Krieger, Nancy (1994): Epidemiology and the web of causation: has anyone seen the spider? In: Social Science & Medicine 39, 7, pp. 887-903.

Krieger, Nancy (2001): Theories for social epidemiology in the 21st century: an ecosocial perspective. In: International Journal of Epidemiology 30, 4, pp. 668-677.

Krieger Nancy (2003): Genders, sexes, and health: what are the connections – and why does it matter? In: International Journal of Epidemiology 32, 4, pp. 652-657.

Krieger, Nancy (2005): Embodiment: a conceptual glossary for epidemiology. Journal of Epidemiology & Community Health 59, 5, pp. 350-355.

Krieger, Nancy/Löwy Ilana and the "Women, Hormones, and Cancer" group (Aronowitz, R./ Bigby, J./Dickersin, K./Garner, E./Gaudillière, J-P./ Hinestrosa, C./Hubbard, R./Johnson, P.A./Missmer, S.A./Norsigian, J./Pearson, C./Rosenberg, C.E./Rosenberg, L./Rosenkrantz, B.G./Seaman, B./Sonnenschein, C./Soto, A.M./Thorton, J./ Weisz, G.) (2005): Hormone replacement therapy, cancer, controversies & women's health: historical, epidemiological, biological, clinical and advocacy perspectives. In: Journal of Epidemiology & Community Health 59, 9, pp. 740-748.

Krieger, Nancy/Smith, Kevin/Naishadham, Deepa/Hartman, Cathy/Barbeau, Elizabeth M. (2005): Experiences of discrimination: validity and reliability of a self-report measure for population health research on racism and health. In: Social Science and Medicine 61, 7, pp. 1576-1596.

Krieger, Nancy/Rehkopf, David H./Chen, Jarvis T./Waterman, Pamela D./Marcelli, Enrico/Kennedy, Malinda (2008): The fall and rise of US inequities in premature mortality: 1960-2002. PLOS Medicine 5, 2. <http://www.plosmedicine.org/article/info%3Adoi%2F10.1371%2Fjournal.pmed.0050046>.

Krieger, Nancy (2011): Epidemiology and The People's Health: Theory and Context. New York: Oxford University Press.

Krieger Nancy/Chen, Jarvis T./Waterman, Pamela D. (2011): Temporal trends in the black/white breast cancer case ratio for estrogen receptor status: disparities are historically contingent, not innate. In: Cancer Causes & Control 22, 3, pp. 511-514.

Krieger, Nancy/Waterman, Pamela D./Kosheleva, Anna/Chen, Jarvis T./Carney, Dana R./ Smith, Kevin W./Bennett, Gary G./Williams David R./Freeman, Elmer/ Russell, Beverly/Thornhill, Gisele/Mikolowsky, Kristin/ Rifkin, Rachel/Samuel, Latrice (2011): Exposing racial discrimination: implicit & explicit measures – the My Body, My Story study of 1005 US-born black & white community health center members. PLOS ONE 6, 11. <http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0027636>.

Krieger, Nancy (2012): Who and what are 'populations?' – historical debates, current controversies, and implications for understanding 'population health' and rectifying health inequities. In: Milbank Quarterly 90, pp. 634-681.

Krieger, Nancy (2012): Methods for the scientific study of discrimination and health: from societal injustice to embodied inequality – an ecosocial approach. In: American Journal of Public Health 102, 5, pp. 936-945.

Krieger, Nancy/Chen, Jarvis T./Kosheleva, Anna/Waterman, Pamela D. (2012): Not just smoking and high-tech medicine: socioeconomic inequities in US mortality rates, 1960-2006. International Journal of Health Services 42, 2, pp. 293-322.

Krieger, Nancy (2013): History, biology, and health inequities: emergent embodied phenotypes & the illustrative case of the breast cancer estrogen receptor. In: American Journal of Public Health 103, 1, pp. 22-27.