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Theory and empiricism: A comment on “Interrogating the environmental affordances model” by Pamplin and colleagues

Briana Mezuk^{a,*}, Juan Del Toro^b, Margaret Gough Courtney^c, Keri F. Kirk^d, Xing Zhang^e, Erica C. Spears^f, Tiffany Green^g, Hedwig Lee^h, Darrell Hudsonⁱ

^aCenter for Social Epidemiology and Population Health, Department of Epidemiology, University of Michigan School of Public Health, USA

^bLearning Research and Development Center, University of Pittsburgh, USA

^cDepartment of Sociology and Anthropology, University of La Verne, USA

^dDepartment of Family Medicine, Georgetown School of Medicine, Medstar Georgetown University Hospital, USA

^eCollege of Health Solutions, Arizona State University, USA

^fSchool of Public Health, University of North Texas Health Science Center, USA

^gDepartments of Populations Health Sciences and Obstetrics and Gynecology, University of Wisconsin-Madison, USA

^hCenter for the Study of Race, Ethnicity and Equity, Department of Sociology, Washington University in St. Louis, USA

ⁱBrown School at Washington University in St. Louis, USA

Abstract

We strongly support efforts to generate, rigorously test, and falsify hypotheses derived from the Environmental Affordances (EA) Model of Health Disparities, as originated by the late Dr. James S. Jackson (1940–2020). Such efforts are critical to establishing robust, theoretically grounded scientific frameworks that explain the fundamental causes of racial disparities in health and wellbeing. Pamplin et al. (2021) fundamentally misrepresents the EA Model as a framework that (falsely) reifies the role of race as a determinant of health behaviors and health outcomes. Further, both their study design and analytic approach are inappropriate for testing predictions of this framework. We address these issues with the goal of recentring the scholarly conversation about how stress contributes to health, and disparities in health, over the life course.

Keywords

Conceptual models; Disparities; Mental health; Theory development; Scientific rigor

*Corresponding author. Department of Epidemiology, University of Michigan School of Public Health, Suite 2649B, 1415 Washington Heights, Ann Arbor, MI, 48109, USA. bmezuk@umich.edu (B. Mezuk).

1. Overview

We strongly support efforts to generate, rigorously test, and falsify hypotheses derived from the Environmental Affordances (EA) Model of Health Disparities, as originated by the late Dr. James S. Jackson (1940–2020). Such efforts are critical to establishing robust, theoretically grounded scientific frameworks that explain the fundamental causes of racial disparities in health and wellbeing. A central premise of the EA Model is that social (dis)advantage shapes contextual determinants of stress and health behaviors throughout the life course, and that social (dis)advantage is allocated differentially across racial groups due to structures like racism and discrimination. Importantly, as we emphasize below, the EA Model does not argue there is anything intrinsic about *race* as it pertains to stress, health behaviors, or coping with stress. Indeed, as we reiterate below, it argues the complete opposite.

Pamplin et al. (2021) (Pamplin et al., 2021) fundamentally misrepresents the EA Model as a framework that (falsely) reifies the role of race as a determinant of health behaviors and health outcomes. Further, both their study design and analytic approach are inappropriate for testing predictions of this framework. We address these issues with the goal of recentring the scholarly conversation about how stress contributes to health, and disparities in health, over the life course.

Claim 1: “Race” is a central analytic variable of the EA Model.

Pamplin states: “The EA model hypothesizes that Black people’s coping with stressor exposure through engaging in unhealthy behaviors (UHBs) such as smoking, drinking alcohol, and consuming calorie-dense foods, simultaneously increase risk of physical illness, and protects against the negative psychological consequences of stressor exposure (i. e., depression) (Jackson et al., 2010).”

In this quote, and throughout the paper, Pamplin fundamentally misrepresents the role of race in the EA Model. The question is not “Are black people more likely to engage in UHBs in response to a stressor” (Figure 1b in their paper). Rather, the EA Model asks: “*Is the relationship between stress, health behaviors, depression the same across groups with different environmental affordances and opportunities?*”

An appropriate empirical test of the latter question is to: (a) assess whether the relationship between stress and risk of depression within each group, and (b) determine whether the inclusion of an interaction term between health behaviors and stress provides additional utility in modeling of risk of depression within each group; Jackson et al. (2010) (Jackson et al., 2010) and Mezuk et al. (2010) (Mezuk et al., 2010) do so and find empirical support for these conjectures. In contrast, Pamplin and colleagues conflate tests of variation *within* groups (i.e., stratifying analyses by race) with tests of variation *between* groups (i.e., stratifying analyses on the basis of stressors) (Schwartz and Meyer, 2010).

As shown in the quoted texts below, the EA Model conceptualizes variables like “race” as social factors which are structurally tied - historically, intergenerationally, and across the life course - to economic, geographic, and political factors. These macro-level factors, in turn,

shape environmental affordances and opportunities - including the quality and quantity of exposure to stress, and opportunities to cope with stress. These texts from the Jackson et al. (2010) and Mezuk et al. (2013) (Mezuk et al., 2013) paper to demonstrates this point (emphasis added):

Jackson (2010, pg. 938) (Jackson et al., 2010): “*This process is not inherently linked to racial group membership.* On the contrary, a large part of the negative outcomes observed among the Black population is attributable to the disproportionate distribution among Blacks—a distribution that is quantitatively higher and qualitatively worse than among Whites—of chronic, negative environmental, social, and psychological stressors, as well as the greater availability of environmental sources of unhealthy behaviors. *We believe that White Americans who lived under similar situations and facilitating structures would demonstrate the same processes and outcomes that we are suggesting occur among the Black American population.*”

Mezuk (2013, pg. 83) states (Mezuk et al., 2013): “The EA Model embodies Williams and Earl’s (2007) perspective that “race is an imprecise variable that captures differential exposure to the resources and rewards in society” (Williams and Earl, 2007:759)... Similar to racial discrimination, *it is the accumulation of differential exposures and experiences associated with these categorizations that produces racial disparities in health over the life course. Thus, we argue that race is not best thought of as an immutable category; instead, racial membership is a “destination” that individuals through their experiences arrive at over time.*”

Claim 2: The EA Model is inconsistent in its articulation of whether UHBs should be treated as mediators vs. moderators of the relationship between stress and depression.

Pamplin et al. state (emphasis added): “Inconsistencies between articulated theory and empirical tests are also evident within studies. For example, in Jackson et al. (2010), the authors hypothesized that UHBs would *mediate the relationship between stressor exposure and depression*, muting the negative psychological effects of stressor exposure. However, their analytic approach and results correspond more with examining UHBs as *effect modifiers of the relationship between stressors and depression* (p. 935) (Jackson et al., 2010).”

In no articulation, whether in text or diagram, of the EA Model is it posited that UHBs “mediate” the stress-depression relationship. The word “mediator,” “mediates,” etc. *does not even appear* in Jackson et al. (2010) except on pg. 934 when describing neurophysiologic mediation of nicotine’s impact on the brain through the meso-limbic system (Jackson et al., 2010). The only mention of “mediation” in Mezuk et al. (2010) is the notion that exposure to *stress mediates the relationship between “social disadvantage”* (notably, not “race” for the reasons cited above) *and depression*, and that *UHBs mediate the relationship between social disadvantage and physical morbidity* (Mezuk et al., 2010).

There is no text in either Jackson (2010) or Mezuk (2010) that could be interpreted to mean that the authors “hypothesize that UHBs would mediate the relationship between

stressor exposure and depression.” Instead, these papers state directly that health behaviors are conceptualized as moderators of the stress-depression relationship (emphasis added):

Jackson (2010, pg. 935 (Jackson et al., 2010)): “Specifically, the analyses examined the direct and *moderating* effects of stressors and unhealthy behaviors on meeting DSM-III criteria for depression, and on being in the higher of a 2-category measure of chronic conditions experienced within the prior 12 months.”

Mezuk (2010, pg. 1240 (Mezuk et al., 2010)): “An interaction term was generated between the count of PHB and median-centered life stress *to examine whether PHB moderated* the influence of stress on risk of depression.”

Claim 3: The EA Model argues that Black people are more likely to engage in UHB in response to stress relative to white people.

Pamplin states (emphasis added): “Notably, these studies do not account for the causal pathways implied by the mediation formulation, namely that UHB engagement results from stressor exposure and *that Black people are more likely to engage in UHBs in response to stressor exposure.*”

There is no “mediation formulation” of the EA Model, as described above. Moreover, Mezuk (2013) states (emphasis added) (Mezuk et al., 2013): “*We do not posit* that the patterns of health behaviors, mental health, and physical health differ by race (i.e., for both blacks and whites, major depression is associated with medical morbidity; *persons with depression, regardless of race, are more likely to engage in poor health behaviors than those without.*”

Finally, in Jackson (2010) (Jackson et al., 2010), Table 1 shows that non-Hispanic whites reported more UHBs than Black individuals (1.34 vs. 1.27), which, if Pamplin’s claim were true, would suggest that Jackson presented data contrary to his hypothesis as evidence of his theory.

2. Limitations of the Pamplin (2021) analysis

Here, we highlight five key limitations of the empirical analysis presented in Pamplin (2021) that hinder its ability to appropriately test the claims of the EA Model.

Use of cross-sectional data.

First, the authors critiqued causal claims within the EA Model, yet puzzlingly conducted a cross-sectional analysis, despite using data from a longitudinal cohort. Prospective data are a necessary component of causal inference, specifically the relationships between stress and mental and physical health as articulated in the EA model. Further, Jackson and Knight (2006) (Jackson et al., 2006) argued that the relationships between stress and health behavior vary over the life course. As Pamplin and colleagues could not even examine age as a covariate (given the lack of variation in their sample), their data were inappropriate for testing a model that explicitly focuses on biopsychosocial processes over the life course.

Insufficient statistical power.

Second, the authors attribute their non-significant findings to the perceived limitations of the EA Model. However, they observed only 63 *lifetime* depressive episodes, as compared to 139 in Mezuk et al. (2010) and 350 in Jackson et al. (2010) at their respective follow-up assessments. Therefore, Pamplin may have been substantially under-powered to detect the associations of interest.

Lack of attention to socioeconomic status.

Third, the authors failed to include any indicators of socioeconomic status or structural context in their analyses. Omitting such a critical set of variables from an analysis that attempts to test the EA Model, a theory informed by Fundamental Cause Theory (Link and Phelan, 1995), is antithetical to empirical tests of this framework. The authors do not even mention this omission as a limitation of their analysis, despite the myriad ways that structural racism impacts the economic opportunities and environmental contexts of Black Americans (Hicken et al., 2018; Williams and Collins, 2001).

Need to adequately address missing data.

Fourth, there was insufficient treatment of missing data and the oversampling of Black participants (i.e., no application of sampling or selection weights). Nearly one-third of data were imputed, although the specific missing variables were never stated, nor was it noted if these data were differentially missing by race. There is no description of any sensitivity analyses to assess the impact of this substantial amount of imputation.

Lack of variation in insurance coverage.

Finally, the sample consisted solely of people whose mothers were privately insured through the Kaiser Permanente Health Plan in the early 1960s. How factors like structural racism relate to access to healthcare goods, like insurance, and therefore selection into this cohort should be considered in any effort to use these data to examine the EA Model (Williams and Rucker, 2000).

3. Conclusion

Despite the claims of Pamplin (2021), the EA Model is not “about” the 1/0 variable “black v. white”, nor is it “about” depression. The EA Model is about centering the widely-replicated “inconvenient patterning” *vis-a-vis* dominant social stress theories of mental disorders and physical morbidity as a function of social status that has been downplayed, or outright ignored, by dominant social science theories used within public health. It is about demonstrating that mental and physical health are not interchangeable outcomes regarding how social status relates to health. It is about recognizing insights from neuroscience which show that health behaviors like tobacco, diet, alcohol, etc. are reinforced (e.g., rewarding) (Koob and Volkow, 2016), are influenced by stress exposure (Koob, 2008), and are established contributors to physical morbidity and mortality. Its fundamental premise is that “race” is not a risk factor for health, and that health disparities researchers must instead focus on social structures, like *racism*, as factors that shape environmental affordances and opportunities across the lifespan and across generations (VanderWeele and Robinson, 2014).

Notwithstanding the inappropriateness of their study design for supporting such an analysis, the authors' claim that "an analytic test of the full [EA] model was never warranted" suggests a fundamental misunderstanding of how conceptual models relate to the scientific enterprise. There is no single analysis that will ever test the "full" EA Model, just like there is no single analysis that can ever test the "full" fundamental cause framework, life course framework, stress-process model, etc. Rather, these theories, like the EA Model, inform the generation of research questions and the formation of testable hypotheses, determine choice of study design, inclusion/exclusion criteria, measurement strategies, and analytic tools, and guide the interpretation of results (Mezuk et al., 2017).

Box (1979) said: "All models are wrong but some are useful" (Box, 1979). The EA Model is useful insofar as it supports well-conducted empirical research on the fundamental biopsychosocial mechanisms of health and health disparities (Engel, 1977). It is useful as an example of a truly interdisciplinary framework, drawing insights from sociology, psychology, epidemiology, critical race theory and neuroscience, to understand causes of racial health disparities. We feel confident saying that Dr. Jackson would welcome any rigorous interrogations of the theoretical model he proposed as they further the scientific conversation about the sources, and potential solutions, to long-standing racial disparities in health. We hope this commentary will help inform such future efforts.

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