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Evolution and social epidemiology

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ABSTRACT

Evolutionary biology, which aims to explain the dynamic process of shaping the diversity of life, has not yet significantly affected thinking in social epidemiology. Current challenges in social epidemiology include understanding how social exposures can affect our biology, explaining the dynamics of society and health, and designing better interventions that are mindful of the impact of exposures during critical periods. I review how evolutionary concepts and tools, such as fitness gradient in cultural evolution, evolutionary game theory, and contemporary evolution in cancer, can provide helpful insights regarding social epidemiology.

1. Challenges in social epidemiology

An urgent concern, repeatedly emphasized by the World Health Organization, is the negative impact of poor social conditions on health (Commission on Social Determinants of Health (2008)), which is the research focus of social epidemiology. Social epidemiologists have explored social conditions closely linked to reproduction, health-related behaviors, diseases, and mortality, and have been especially interested in the impact of socioeconomic status (SES), neighborhood residence, income inequality, and social support (Berkman et al., 2014; Braveman et al., 2005; Commission on Social Determinants of Health (2008); Cwikel, 2006; Galea, 2007; Krieger, 2011; Marmot, 2005; O'Campo and Dunn, 2012). For example, evidence suggests that people with fewer years of education or with poorer social support often experience poorer health conditions (obesity, coronary heart disease, cancer, mortality, etc) in later life (Braveman et al., 2005; Uchino, 2006). Social epidemiologists assume that the human body gets inputs from "societal arrangements of power, property, and patterns of production, consumption, and reproduction" (Krieger, 2011), processes them biologically, and experiences the consequences for health and diseases. They refer to this mechanism as "embodiment" (or "biological embedding") (Krieger, 2011; Kuh et al., 2003; Kuzawa and Sweet, 2009; Roux, 2012).

Social epidemiologists and others have also aimed to prevent the negative health consequences of social factors, and have performed several randomized controlled trials (RCTs). A randomized housing mobility experiment in the United States (Moving to Opportunity, MTO) shows that having the chance to move from *high-poverty neighborhoods* to lower-poverty neighborhoods improves physical health (obesity and type 2 diabetes), mental health, and subjective wellbeing (Ludwig et al., 2011, 2012). The Enhancing Recovery for Coronary Heart Disease Patients (ENRICHD) trial aimed to improve *social support* to reduce re-infarction and all-cause mortality among post-myocardial infarction patients, which find no difference in outcome as a result of the intervention (Berkman et al., 2003). The High/Scope Perry Preschool Program (PPP) aimed to improve *pre-kindergarten education* for the potential health benefits, which resulted in improvement in several health-related behaviors, but a null effect on physical health outcomes after a 37-year follow-up (Muennig et al., 2009). Overall, such RCTs have not been as successful as one might hope (Baicker et al., 2013; Berkman, 2009; Berkman et al., 2003; Ludwig et al., 2012, 2011; Muennig et al., 2009). Reflecting on these difficulties (Berkman, 2009; Canning and Bowser, 2010; Krieger, 2001; Nishi et al., 2015a; Roux, 2012), social epidemiologists have explored novel directions.

First, one promising avenue is individual heterogeneity in the health consequences of social exposures (*uniqueness argument*) (El-Sayed et al., 2013; Ogino et al., 2013). Indeed, human genomes are unique to each individual (genetic diversity), and their experiences in their life and environment are also unique to each individual (lifecourse diversity). Such diversities can produce vast heterogeneity in health consequences (Nishi et al., 2015a; Ogino et al., 2013).

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For example, it is known that a genotypic variation in nicotinic acetylcholine receptor (*CHRNA6*) is an important effect modifier on the effect of tobacco taxation on tobacco use (Fletcher, 2012), suggesting the importance of better use of genomic data in a public health context (El-Sayed et al., 2013).

Second, the effect of health and diseases on social conditions needs to be jointly considered (*dynamicity argument*) (Canning and Bowser, 2010). For example, a previous study reported young women living with hearing loss in Japan are more likely to be unmarried, smoking, and psychologically distressed (Kobayashi et al., 2015), and such social conditions and behaviors may induce other health consequences in their later life. It is also reported that, in the U.S., sicker individuals are more likely to lose their jobs, while job loss also has a negative health impact (Strully, 2009). Although causal inference in social determinants of health aims to control for such "reverse causality," considering the innate dynamic interplay of social factors and health (even over multiple generations), all of the health effects of social factors, the social effects of health factors, the health effects of other health factors, and social effects of other social factors – would, ideally, be simultaneously understood.

Third, social epidemiologists need to evaluate potential means by which interventions could arrest the health consequences of social determinants of health when exposures happened in the past or are no longer modifiable in a focal population (*temporality argument*) (Gilman, 2012; Nishi et al., 2015a). For example, it is known that nutritional deficits in early life, including prenatal famine exposure, can cause multiple health issues including obesity, glucose tolerance, and mental illnesses (Hayward et al., 2013; Lumey et al., 2011); however, such under-nutrition happened in the past and is not modifiable, so improving the nutritional status of such people after the fact is not a plausible intervention strategy.

Although social epidemiologists have been affected by several other disciplines – including genetics and epigenetics, lifecourse epidemiology, sociology, neuroscience, psychology, and behavioral economics – in order to address these topics, the perspective of *evolution* might beneficially be introduced into social epidemiology research and practice (Galea et al., 2010; Krieger, 2011). Therefore, I aim to review the role of evolution in relation to the foregoing challenges.

2. Core concepts of evolution

Evolutionary biology aims to explain how history, selection, and random processes have shaped adaptation, diversity, and complexity (Stearns and Hoekstra, 2005). Current evolutionary thought is a synthesis of Darwin's ideas and Mendelian genetics supplemented with concepts from development and epigenetics (Futuyma, 2013; Muehlenbein, 2010; Nowak, 2006a). Evolution has two parts: macroevolution (evolutionary process above the species level) and microevolution (the evolutionary process within a single species). The present paper mostly relates to microevolution.

Microevolution occurs when a trait (e.g., height, disease susceptibility, or personality) varies among individuals (1st condition), and when at least some of the variation in the trait is based on the variation in genes (units of heredity) (2nd condition). This is one of the sources of individual heterogeneity. One type of microevolution is adaptive evolution, in which a 3rd condition is necessary: the variation in the trait is correlated with reproductive success (fitness) (Nowak, 2006a). The reproduction of successful individuals alters the frequency of heritable traits and genes over generations, and a stronger correlation of the trait with fitness causes a more rapid change in the frequencies of the genes that influence the trait. Natural selection can thus be defined as "nonrandom differences in the rate of survival or reproduction among classes of entities that differ in inheritable characteristics" (Muehlenbein, 2010). Notably, humans in modern societies continue to experience natural selection (Byars et al., 2010; Courtiol et al., 2012).

The other type of microevolution is neutral evolution. Neutral evolution focuses on changes in heritable traits and genes by random processes, both of which can happen without an association with fitness (3rd condition). Genetic drift falls into this category, and represents a random change in the allele frequency of genotypes in a population. Both types of microevolution are reflected in changes in gene frequencies. Since microevolution can occur as long as these conditions hold, the entities in an evolving population can be humans, other animals, or cells (see the section of Example 3).

The concepts of biological evolution can also be applied to cultural evolution (a.k.a. social evolution) (Fig. 1A) (Laland et al., 1999; Richerson et al., 2010). In cultural evolution, the heritable components are not genes, but social factors such as ideas, habits, and assets, which also have individual heterogeneity. Any forms of non-genetic heritable components may cause cultural evolution; often they produce differences in fitness between haves and have-nots. An idea or habit may be transmitted either horizontally to non-kin (i.e. social learning or diffusion of innovation) or vertically to children and grandchildren (Rogers, 2003; van Schaik and Burkart, 2011). For example, obesity-related behaviors are contagious over human social networks (Christakis and Fowler, 2007; Hill et al., 2010; Shoham et al., 2012), which can reflect social learning of obesity-producing behavior from friends, spouses, parents, and family members. Recent advances in understanding cultural evolution have been made using evolutionary game theory, which investigates the evolution of social behaviors such as cooperation, punishment, homophily, and overconfidence (Buss, 2012; Fehr and Gachter, 2002; Fowler and Christakis, 2010; Gintis, 2009; D. D. Johnson and Fowler, 2011; Nowak, 2006b). In sum, genes and cultures are both important drivers of human evolution; this is the premise of "gene-culture coevolution" (Laland et al., 1999; Richerson et al., 2010).

Not all phenomena in modern humans need be consequences of long-term evolution. "The mismatch between biological bodies and modern lifestyles" also helps to explain current society-health interrelations (Gluckman and Hanson, 2008). Biology cannot evolve as rapidly as culture, and, in particular, the agricultural and industrial revolutions have led to mismatches that produce chronic illnesses including type 2 diabetes, obesity, cardiovascular disease, neurodegenerative disease, and some of the age-related cancers (Gluckman and Hanson, 2008; Krieger, 2011; Neel, 1962). This is one of the major research topics in evolutionary medicine (Gluckman et al., 2009; Stearns, 2008). While mismatch in lifestyle and nutrients can be the target of health interventions (Eaton et al., 2002), how to intervene has been controversial (Hayward et al., 2013).

The perspective of evolution has been applied in many disciplines: anthropology (Bowles and Gintis, 2002; E. A. Smith et al., 2010), economics (Veblen, 1899), medicine (Gluckman et al., 2009; Stearns, 2008), and psychology (Buss, 2012; Mitchell, 1999; Tooby and Cosmides, 1989), leading, for example, to *evolutionary psychology*, which posits that a behavior is an output of a psychological mechanism with informational input, and that the mechanisms originate from evolutionary processes at some level (Buss, 2012). *How can we apply these evolutionary perspectives in social epidemiology?* I introduce three examples, using different aspects of evolutionary perspectives.

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