Social Science & Medicine 145 (2015) 132–137

Contents lists available at ScienceDirect

Social Science & Medicine

journal homepage: www.elsevier.com/locate/socscimed

Evolution and social epidemiology

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ARTICLE INFO

Article history:
Received 26 June 2015
Accepted 11 August 2015
Available online 14 August 2015

Keywords:
Social epidemiology
Evolutionary biology
Cultural evolution
Intervention planning

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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http://dx.doi.org/10.1016/j.socscimed.2015.08.015
0277-9536/

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.

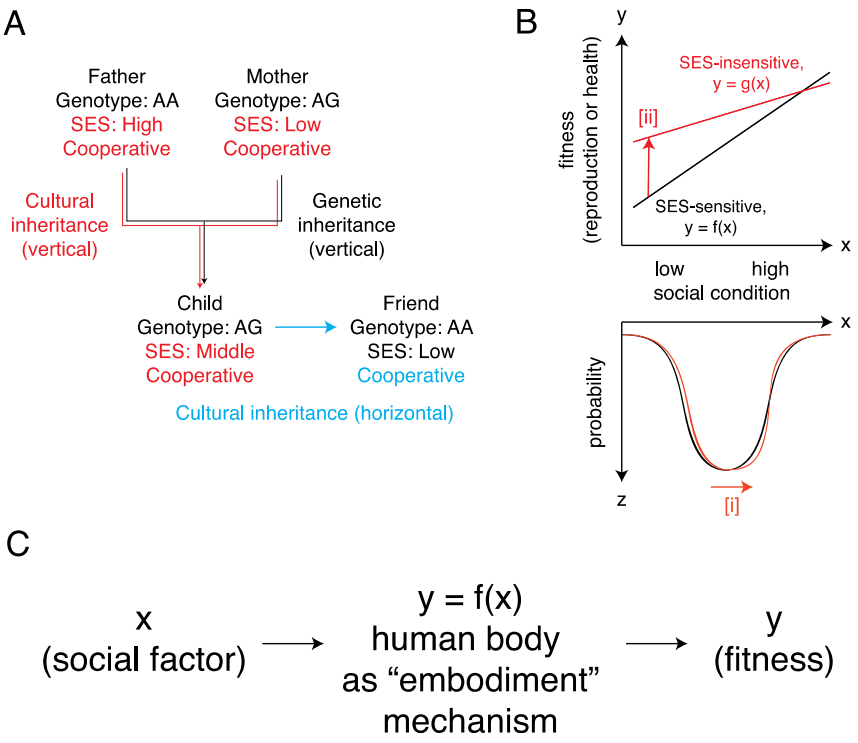

Fig. 1. Fitness gradient in reproduction or health by socioeconomic gradient can relate to cultural evolution. (A) Genetic inheritance represents the half of parental genotypes in a locus (e.g. AA and GG) is transmitted to child genotypes (e.g. AG). "Vertical" cultural inheritance represents the parental cultural traits (e.g. socioeconomic status [SES]) is reflected into child cultural traits, while "horizontal" cultural inheritance represents the individual's cultural traits (e.g. cooperative personality) has an influence on cultural traits of friends or other neighbors in social networks. (B) A social condition is distributed (bottom), and the different levels of the social condition results in the different levels of fitness (above) following $y = f(x)$. Therefore, reducing a lower social condition (e.g. free education provision, cash transfer or progressive tax) [the 1st approach, i], or addressing the embodiment mechanism to make human biology less sensitive to a lower social condition toward $y = g(x)$ [the 2nd approach, ii] can prevent worse people's health due to a lower social condition. (C) A human body is an embodiment function getting inputs from social factors and extracting a disease as an outcome ($f(\cdot)$ and $g(\cdot)$ are the functions with different slopes as seen in Panel B).

3. Example 1: addressing an ongoing embodiment mechanism

A conventional interventional approach in social epidemiology is improving social determinants of health. From the fitness gradient perspective, this interventional approach aims to shift the people at a lower level to a higher level (the 1st approach in Fig. 1B [i]), in a situation where people with a higher level of a social condition can achieve higher fitness than those with a lower level of the social condition. Such a fitness gradient is also used when discussing the difference of advantageous allele and disadvantageous allele at a locus in genetic evolution (Nowak, 2006a).

Once a human body is viewed as a biological function that gets input from social factors and then produce outputs of diseases in later life, we can write a function of embodiment: $y = f(x)$, where x is a vector of social factors (which may or may not be independent from each other), y is a vector of *fitness* (reproduction, health, diseases, etc), and $f(\cdot)$ represents how a human body processes the social factor in a life span (Fig. 1C). For example, people get inputs of fewer years of education and poorer social support, and output obesity, coronary heart disease, and cancer in later life. Here, I assume that the $f(\cdot)$ of multiple individuals in a population have substantial communality with some variation among them, and $f(\cdot)$ evolves over time and over generations. While the currently dominant interventional approach in social epidemiology is the 1st approach (Fig. 1B [i]), the evolutionary perspectives tell us that we have another approach, which have been less well understood. The approach aims to block the embodiment mechanism, resulting in the decrease in the effect of social factors on health (i.e., changing $f(\cdot)$ to $g(\cdot)$) among a group of individuals with a poorer social condition (the 2nd approach in Fig. 1B [ii]). This should be especially appropriate and effective when the exposure to social factors cannot be changed or happened in the past (Nishi et al., 2015a).

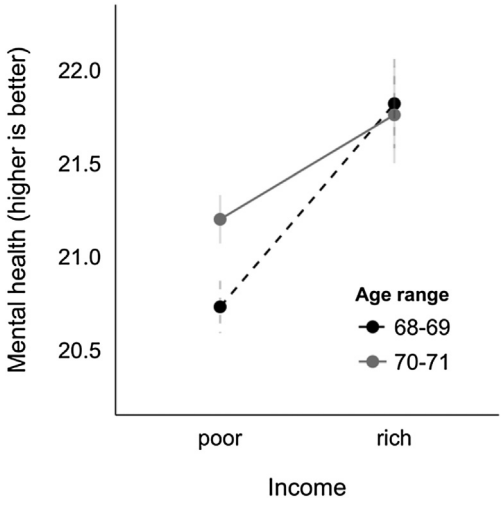

Fig. 2. Fitness gradient among older people in Japan under the universal coverage. At 68 and 69 years of age, patient cost sharing is 30% both for rich and poor individuals, and the fitness gradient is steeper. At 70 and 71 years of age, patient cost sharing is 10% for poor individuals and 30% for rich individuals, and the fitness gradient is shallower. There is an income threshold for the reduced patient cost sharing in Japan, and I denote here the individuals below the threshold as poor and those above it as rich. Data were obtained from Nishi et al., 2012, and the figure shown here was newly created. Error bars, mean ± s.e.m.

Then, how can we modify f(•) to g(•)? An illustrative study comes from health policy research (Fig. 2) (McWilliams, 2007; McWilliams et al., 2009; Nishi et al., 2012). For example, in Japan (with an universal health care), individuals whose income is below a threshold need to pay only 10% copayment when they turn 70 years of age, while all individuals before 70 years of age, and rich individuals after 70 years of age, need to pay 30%. A previous paper (Nishi et al., 2012) examines the effect of the reduced cost sharing on mental health (reversed K6 score, 0–24; higher is better), and showed that, just before 70 years of age, mental health of poor individuals is 20.73, that of rich individuals is 21.82. Just after 70 years of age, that of poor individuals is 21.20, that of rich individuals is 21.76. If we introduce a dichotomous variable to represent rich individuals, the embodiment function for mental health before 70 years of age: $y = f(x) = 1.09x + 20.73$ (steeper fitness gradient); that after 70 years of age: $y = 0.56x + 21.20$ (shallower fitness gradient), where $x = 1$ for rich individuals and $x = 0$ for poor individuals. Therefore, this policy approach to reduce cost sharing for poorer individuals can reduce the fitness gradient on mental health among older individuals by about 50% without improving the poorer social condition itself. Such an effect of patient cost sharing was also reported in the U.S. (McWilliams, 2007; McWilliams et al., 2009).

Since the mental health disparity still remains, a careful interpretation on policy effectiveness is necessary; however, more importantly, by changing the environment or policies, which shapes the magnitude of fitness gradient, social epidemiologists may address an ongoing embodiment mechanism by removing the excess fitness disadvantages among poorer individuals, where income is the non-genetic heritable component in cultural evolution. Therefore, the idea of fitness gradient in cultural evolution can provide a tool to reexamine existing evidence to address the *temporality argument*.

4. Example 2: Avoiding unintended consequences when intervening in income inequality

It is known that people living in a society with greater income inequality have, on average, worse health status than those living in a society with less inequality (Kondo et al., 2009). It is known that the pathway from income inequality to worse health status is partially mediated by the decrease in social capital quality (Kawachi et al., 1997), and the quality of the social network (social support, cohesion, or capital) is associated with health status (K. P. Smith and Christakis, 2008; Uchino, 2006). Therefore, it seems interventions to reduce income inequality (e.g., progressive taxation, conditional cash transfers) might improve health status. *Are there any potential unintended negative consequences of planned interventions?* This question relates to the dynamic interplay of multiple social factors, all of which have a health impact in later life.

Evolutionary game theory studies which behaviors are stable in the given environment from an evolutionary perspective; consists of mathematical theories, simulations, and empirical studies in laboratory and field experiments; and models the dynamic interplay of multiple factors (Gintis, 2009; Nowak, 2006a). Simulation studies provide interesting evidence of the dynamic interplay of inequality (resource heterogeneity) and social support (level of cooperation). For example, it is known that people can choose to cooperate more frequently under certain settings of initial resource *heterogeneity* (i.e. inequality) (Abou Chakra and Traulsen, 2014; T. Johnson and Smirnov, 2012; Kun and Dieckmann, 2013; Li et al., 2012; Nishi et al., 2015b; Vasconcelos et al., 2014; Wang et al., 2010). Although results from experiments with human subjects do not always support the theoretical predictions from simulation studies (Buckley and Croson, 2006; Chan et al., 1999; Cherry et al., 2005; Levati et al., 2007; Milinski et al., 2008; Reuben and Riedl, 2013; Tavoni et al., 2011), it is known that several tricks are useful to avoid the decay of cooperation: communication between subjects, chance to convince subjects, punishment of free-riders, and leadership (where rich people take an initiative on the construction of cooperative networks) (Levati et al., 2007; Milinski et al., 2008; Reuben and Riedl, 2013; Tavoni et al., 2011). Moreover, economic literature shows that higher inequality gives people incentives to work harder (Cingano, 2014), and thus the reduction of inequality may also undermine economic growth in certain settings. In sum, a lesson here is that a simple intervention to modify the current income inequality may result in unintended negative consequences in a society (Paina and Peters, 2012). Examining intervention plans with simulations or experiments using online subjects in advance, for example, may make unintended negative consequences avoidable in a real target population (Buhrmester et al., 2011; Hayward et al., 2013; Nishi et al., 2015b; Shirado et al., 2013). Therefore, the interdisciplinary approach with evolutionary game theory (Gintis, 2009) and related fields such as mathematical sociology (Yamaguchi, 2005), experimental economics (Fréchette and Schotter, 2015), and evolutionary economics (Dopfer and Potts, 2008; Veblen, 1899) can provide a tool to see a potential negative influences of planned interventions, and to address the *dynamicity argument*.

5. Example 3: elucidating the roles of social exposures in cancer evolution

Social factors play an important role in the epidemiology of cancer. Consider breast cancer. Risks associated with age, race/ethnicity, reproductive history and menopause, and lifestyle (e.g. smoking and obesity) are widely recognized for breast cancer (Krieger, 2013; Polyak, 2007; Wallace et al., 2011).

Evidence suggests breast cancer is not a single entity, but a collective term of tumor with heterogeneity occurring in a breast region (Ogino et al., 2013; Polyak, 2007). Recent studies also show that there is a key molecular factor in breast cancer: the estrogen receptor 1 (ER-alpha) (Krieger, 2013; Polyak, 2007; Wallace et al., 2011). ER-alpha is expressed in some breast cancer cells; these cells (ER-positive) need estrogen to grow. Breast cancer cells in which ER is not expressed (ER-negative) do not need estrogen to grow. Thus, the path to carcinogenesis in ER-positive cancers differs from that in ER-negative cancers, which are typically more aggressive with poorer prognosis (Wallace et al., 2011). Moreover and interestingly, several studies show that women with lower SES are more likely to develop ER-negative breast cancer than those with higher SES in observational studies (reviewed in (Vona-Davis and Rose, 2009)), suggesting the process of cancer development in those with lower SES is diverted from that in those with high SES at a certain timing. Although an interdisciplinary approach with life-course epidemiology can partly contribute to identifying critical periods of social exposures for molecular-specific cancer development (Nishi et al., 2015a; Ogino et al., 2013), its biological understanding remains a challenge.

Evolutionary biology in cancer has sought a conceptual and mathematical understanding of how exactly social factors alter which part of the evolutionary process of carcinogenesis. Cancer can be understood as a process of clonal selection that is initiated when mutations occur in dividing cells (Burrell et al., 2013; Michor et al., 2004; Nowak, 2006a). Mutations in genes that monitor DNA damage, carry out DNA repair, or elicit cell death if DNA cannot be repaired, are particularly likely to lead to abnormal cell division events. In carcinogenesis, the dynamics are complex under selection pressure, which permits a certain type of mutant subclones to become dominant, and other types to become extinct or to be dormant (Greaves and Maley, 2012). Clonal diversity and branched

evolution (distinct subclones evolve in parallel) have been demonstrated in breast cancer (Nik-Zainal et al., 2012; Shah et al., 2009, 2012). To understand these evolutionary processes, mathematical modeling and simulations have been used as well as biological studies (Diaz et al., 2012; Michor et al., 2004). Therefore, cancer control can be seen as "managing contemporary evolution" (Carroll et al., 2014), in which destructive evolution occurs in rapidly reproducing cells with larger population size. Please note that such "contemporary evolution," which happens at the cellular level, is much faster than genetic evolution happening over multiple generations of humans.

Therefore, regarding the "embodiment" function at the human level, how the input of harmful social factors is processed inside the body and leads to the molecular evolution of cancer needs to be understood in the context of contemporary evolution. Such biological and evolutionary understanding of the embodiment mechanism can help social epidemiologists address the *uniqueness argument*. If we can develop a way to stop ongoing cancer evolution in human cells influenced by the input of harmful social factors, this will be an example of the 2nd approach, which addresses the ongoing embodiment mechanism (Fig. 1B [ii]).

6. Conclusions and future directions

In conclusion, determining the nature of embodiment can be a shared focus of evolutionary biology and social epidemiology. The broad range of topics in social epidemiology can be worth rethinking from an evolutionary perspective. Although I have focused on socioeconomic status, which is one of the most important topics of social epidemiology here, the applications of evolutionary biology into other research topics of social epidemiology such as gender, race and ethnicity, working environment, and neighborhood quality are also important directions of future research. The integrated approach could be named *evolutionary social epidemiology*, a framework in which evolutionary biologists have the opportunity to address urgent global issues in humans and to help social epidemiologists. This approach would then become part of applied evolutionary biology (Carroll et al., 2014). Although differences in primary foci will remain (evolutionary reproduction for evolutionary biologists and health and disease for social epidemiologists), a unified approach to model both of them at the same time and a shared interest in fitness can allow evolutionary biologists and social epidemiologists jointly to understand the dynamic interplay of society, reproduction, and health.

Author contributions

AN was involved in the design and the analysis for the present article.

Funding statement

AN was awarded by the Japan Society for the Promotion of Science (JSPS) for his research abroad. Support for this research was provided by a grant from the Konosuke Matsushita Memorial Foundation (12-079).

Ethical approval

N/A.

Conflict of interest

I declare that I have no conflict of interest.

Acknowledgments

I thank Nicholas A Christakis and Stephen C Stearns for helpful comments at the earlier version of the manuscript.

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