the hoped for synthesis will ultimately occur outside of the discipline. Considering the power of biocultural interactions, it is not surprising that colleagues in other disciplines see the value in synthetic perspectives that we, as anthropologists, are all too willing to trivialize or neglect. The October 2013 special issue of the *American Journal of Public Health* focusing on the integration of behavioral, social science, and genetics research (Spittel, Spotts, and Deeds 2013) is a case in point. Few of the contributing authors were anthropologists, yet the collection represents some of the most insightful biocultural discussions to date. Our intradisciplinary failures to sustain consistent levels of collaboration required to advance the biocultural synthesis may be the signal that anthropologists wishing to pursue research on developmental systems and inequality need to broaden their collegial horizons while focusing the research more locally.

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Nature-Nurture Interaction Is Ubiquitous, Essential, but Elusive

Hicks and Leonard are correct in focusing on the challenge of incorporating political, economic, and historical impacts in studies of human biology. There is one fundamental reason that political economy is, and, indeed, must be, compatible with evolutionary approaches: phenomena in both fields run on the same fuel, namely, that of actions of selfish agents maximizing their individual success. The structural similarity of both fields led to the unifying development of evolutionary theory of cooperation (Axelrod and Hamilton 1981), stemming from the theory of games (developed originally for economics).

Hicks and Leonard argue that "cultural norms are actively constructed." However, behaviors that may be perceived as expressing cultural norms to "reinforce or magnify existing inequalities" may, in fact, have nothing to do with ideology but instead be outcomes of complicated and often counterintuitive dynamics of network-based social phenomena (Watts 2003). For example, the process of competition for limited resources occurring among agents who exhibit natural variation in their abilities inevitably leads to a skewed distribution of success (Łomnicki 1988). There is no political Machiavellianism here, only mathematical inevitabilities, with profound implications for many ecological processes. Having said that, we do not mean to sound socially insensitive and complacent, and we agree with Hicks and Leonard that critical evaluation of "cultural constructions that naturalize poverty" is essential.

To make matters more complicated, however, genes themselves may become vehicles that help propagate these "cultural constructions" and maintain, for example, multigenerational health disparities observed between racial categories (whether they be defined culturally or biologically; Jasienska 2013). The latest discoveries in epigenetics bring to the forefront the notion of striking cultural influences that may reverberate across generations through not only cultural transmission but also genetic effects. Trauma due to partner violence experienced by women during pregnancy may affect the methylation status of genes of their adolescent children, with potential lifelong effects on psychosocial health (Radtke et al. 2011).

But the transgenerational effects could be more profound than that. Dias and Ressler (2014) showed that F_1 and even F_2 progeny of male mice exposed to both a scent and an electric shock showed increased sensitivity to that scent and increased expression of genes responsible for scent detection. It means that epigenetic programming of genes may be inherited paternally, independently of maternal effects. Psychosocial and nutritional stresses may therefore affect not only the health of people who experienced deprivation but also the health of their distant descendants. Low birth weight (Jasienska 2009) and high risk of cardiovascular diseases (Kuzawa and Sweet 2009) in contemporary African Americans have been suggested to result from conditions experienced by their ancestors during slavery.

An important methodological issue is how to implement a research paradigm in which biological and cultural factors are explicitly taken into account. This is more difficult than to liberally advocate the inclusion of a multitude of factors, sometimes for ideological rather than scientific reasons, and often with a lot of hand waving and lip service paid to "the complex interactions between these inputs." The crucial word here is "explicitly," that is, building research approaches where it is clearly stated what the dimensionality of the problem is (Lewontin 1969) and what types of interactive effects between causal factors are being postulated. Are the interactions multiplicative or do they have more complex, nonlinear forms that cannot be easily simplified with appropriate transformations? Lewontin (1974) argued that numerically the same analyses of variance models may hide qualitatively different types of interaction between nature and nurture.

Next, what empirical design should be applied (Jasienski 1996)? When a true experimental approach is impossible, we are left with a daunting task of untangling the Gordian knot of nature-nurture effects through cross-sectional or longitudinal studies. For example, level of paternal education is an important variable influencing the direction of the relationship between the number of a father's children and his testosterone concentration (Jasienska, Jasienski, and Ellison 2012); also, high average level of education of women in a village influences fertility decisions even of women who are not themselves highly educated (Colleran et al. 2014).

Finally, we must use appropriate quantitative methods of analysis to protect us from mathematical or statistical artefacts

(Jasienski and Bazzaz 1999). Scientists tend to rely on indexes that are meant to capture the essence of the processes, but unfortunately, such indexes are very often ratios of several measurable variables. This gives us an illusion of simplicity by reducing the number of variables but makes statistical analyses less powerful and, more importantly, sweeps under the carpet the actual form of interaction between the variables comprising the ratio index. Alas, disentangling nature from nurture allows no conceptual or methodological shortcuts.

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Why Evolutionary Biology Is Crucial for Effective Public Health Policy

Having finished Hicks and Leonard's excellent review, many readers will be convinced that newer approaches within evolutionary biology avoid the essentialism that has marred some applications of evolutionary theory within anthropology. But even if there are nonreductionistic examples of evolutionary theory to draw inspiration from, you may well ask, what do evolutionary principles really provide us in a practical sense? Is not the obvious fact of health inequality and its alignment with social constructs such as class and race proof enough that societal changes are needed? As someone who works in the area reviewed by Hicks and Leonard, I suggest that evolutionary principles are needed if we hope to inform policy, which requires going beyond observing patterns of health inequality to making a case for what causes what, specifically.

Hicks and Leonard discuss recent research showing that fetal undernutrition or stress alter multiple biological systems that negatively affect adult health and can even influence health in offspring (Benyshek, Martin, and Johnston 2001; Kuzawa and Sweet 2009). Evidence for such examples of developmental plasticity raises important questions about their underlying mechanistic basis. If their long-term health effects simply reflected damage imposed by early stress, we would not need evolutionary biology to help us advocate for effective interventions (Schell and Magnus 2007). For example, the finding that early micronutrient deficiency impairs cognitive development provides a strong rationale for policies that help ensure adequate nutrition in young children (Engle et al. 2007).

But other examples are not so straightforward. Take fat deposition—a key link between early nutrition and cardiovascular disease (CVD). Lower birth weight (LBW) individuals are more likely to develop CVD, but this is not because they become obese. In fact, they end up lighter, but they deposit fat preferentially in the visceral depot, which is innervated by sympathetic nerve fibers originating in the brain. When the body experiences stress, these nerves secrete adrenaline in visceral fat, which releases this energy to help overcome the stressor. Not only do LBW individuals deposit more fat in this depot but also their fat cells also mobilize more fats during a stressor (Boiko et al. 2005). There is nothing about these biological changes that indicates damage. Rather, they suggest that the body learns to prioritize depositing fat in a rapidly usable depot in response to the experience of early undernutrition with downstream effects on risk for diabetes and CVD (Kuzawa 2010).

Given evidence that fetal undernutrition resets fat metabolism in LBW individuals, one might reasonably expect that supplementing the diet of pregnant women would lead to higher birth weights and lower CVD risk. Contrary to this hope, pregnancy nutritional interventions often have negligible effects on offspring birth weight (Kramer and Kakuma 2003). We are thus faced with a paradox and also a policy dilemma: LBW—indicating reduced fetal nutrition—predicts future health, but supplementing women's diets during pregnancy seems not to appreciably change birth weight. What might account for this apparent disconnect between what the mother consumes and the nutrients that the fetus receives?

Evolutionary theory and the principles of human adaptability give us clues. It would not make sense for the fetus of a long-lived species to set metabolic priorities for life based on ecological conditions during a few months of gestation, which are subject to the vagaries of seasonal and other short-term variability. Instead, the quantity of nutrients that the mother's body transfers to the fetus is buffered against such short-term variability, but it is correlated with, and thus communicates information about, her average nutritional experiences as embodied biologically through her lifetime of experiences (Kuzawa 2005). Considering this longer timescale of adaptation potentially helps explain why traditional pregnancy supplementations often yield so little: if the mother's body filters out short-term "noise" to help the fetus track stable ecological trends, fetal development will ignore unreliable detours of the sort that dietary supplements represent. An evolutionary perspective thus points to the need for an intergenerational and sustained approach to interventions: improving the developmental nutrition of future mothers should help send the signal of long-term nutritional change, elevating offspring birth weights and reversing future adult health disparities (Kuzawa and Thayer 2011).

If the long-term health effects of early environments simply reflected damage, evolutionary biology would buy us little. But some examples of developmental plasticity are more complex than simple vulnerability, and we need evolutionary principles to help make sense of them. If the nutrients transferred to the fetus do indeed communicate information about the past, designing effective interventions will require understanding the nature of these cues and what they respond to during the mother's (and grandparents'; Pembrey 2010) development. The recent developments discussed by Hicks and Leonard not only make evolutionary biology less threatening to