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Early life disadvantage, phenotypic programming, and health disparities

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Much research finds that early life socioeconomic disadvantage predicts poorer health later in life, even among those whose conditions improve in adulthood. Although there are numerous factors that contribute to this association, recent research suggests that growing up in adverse socioecological environments may promote developmental patterns that facilitate pre-reproductive survival in harsh environments, but can also come at the cost of reduced longevity. Here, we review recent research demonstrating that early life exposure to low socioeconomic status can become embedded in the mechanisms that regulate (a) bodily inflammatory activity and (b) energy regulation in ways that contribute to poor health. This research offers new insights into ways that early life environments can get under one's skin to impact health and longevity.

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Over the last three decades, a growing body of research has demonstrated that growing up in conditions characterized by low socioeconomic status (SES) and psychosocial stress are associated with the development of physiological and behavioral patterns that can have undesirable consequences for health. For example, research finds that growing up in low SES environments increases one's risk of metabolic disorders [1], cardiovascular disease [2], teenage pregnancy [3], mental health problems [4], and infectious illnesses [5,6]. These risks emerge largely independent of one's adult economic circumstances [7,8,9*], suggesting that growing up in difficult socioecological contexts may have lasting negative impacts on health that are not easily reversed.

There are myriad factors that contribute to the association between early life disadvantage and negative health outcomes in adulthood. For example, research finds that — when compared to children from middle-class and upper-class homes — children growing up in conditions of low SES have greater exposure to stress [10] and harmful chemicals [11], while at the same time having reduced access to healthcare [12], healthful food options [13], and safe places to exercise and play [14,15]. Each of these variables — particularly when occurring together and over long periods of time — plays a role in contributing to the disparities in health that are observed between those from poorer versus wealthier childhood environments.

One's childhood SES plays an important role in determining one's exposure to features of the environment that can promote or detract from health. However, research in the evolutionary sciences suggests that the greater burden of poor health shouldered by those who grow up in disadvantaged environments may also emerge as a byproduct of developmental tradeoffs that have been favored by natural selection to promote survival in stressful ecologies. According to this perspective — which is grounded in insights from life history theory — one's socioecological conditions are an important source of environmental input that is used to regulate development in ways that help match an individual's adult phenotype to their expected ecology [16–18]. Because socioeconomically disadvantaged environments are generally more dangerous, unpredictable, and resource deprived than advantaged environments, they are reasoned to encourage developmental tradeoffs that promote survival and reproduction in such conditions, even if they have a negative impact on long-term health and longevity [see e.g. 19,20]. Here, we review recent research that has employed this perspective to understand the developmental conditions that favor developmental tradeoffs that can contribute to health disparities in diseases related to inflammation and obesity.

Early life socioeconomic disadvantage and inflammation

Growing up poor, for much of human history, has meant having to manage some relatively perilous circumstances. Inherent in these environments were a host of recurrent challenges that would have posed threats to survival and reproductive success, such as predation, conflict with other humans, greater risk of infection and injury, and limited access to nutrients and other resources. Accordingly, researchers have hypothesized that growing up in resource scarce environments should encourage the

development of phenotypic adjustments that promote survival and reproduction in these perilous environments — that is, the development of a ‘defensive phenotype’ [21,22]. A defensive phenotype is characterized by developmental modifications to neuroendocrine pathways through which the body regulates stress [23–25] and sexual development/behavior [26,27], as well as the immune system [28–30]. These modifications are reasoned to allow the body to be better able to defend itself, make use of available resources, and reproduce sooner rather than later, minimizing one’s risk of perishing before first having had the chance to reproduce.

One of the many systems proposed to be influenced by this developmental programming is the body’s immune system, which is the primary means through which the body protects and defends itself from injury and infection. When the immune system is stimulated, it releases signaling proteins, such as proinflammatory cytokines, which promote bodily inflammatory activity, prevent/clear infections, and heal injuries. Given the greater risk of danger and exposure to contaminants present in low SES environments, researchers have proposed that growing up in the context of socioeconomic disadvantage should cause monocytes (cells of the innate immune system) to develop response tendencies that would help promote bodily repair and recovery in relatively perilous ecologies, but can also promote chronic inflammation [30]. Because childhood represents a sensitive period when immune function is highly plastic, it is reasoned that these tendencies are long-lasting and difficult to reverse, even for those who find themselves in more benign ecological conditions in adulthood.

Consistent with this hypothesis, the link between low childhood SES and heightened inflammatory activity has now been demonstrated in several studies [for recent meta-analyses, see Refs. 31*,32*]. This pattern appears early in life [33] and persists into adulthood — even after statistically controlling for numerous potential confounds (including smoking, body weight, and adult SES) [32*]. For example, in one landmark study, Kittleson *et al.* [8] investigated the impact of low childhood SES on the development of coronary heart disease (CHD) in over 1000 adult male medical students who had achieved high adult SES. The researchers found that low childhood SES led to over a twofold increase in risk of developing CHD at or before 50 years of age, even while controlling for body mass index, cholesterol level, amount of exercise, depression, coffee drinking, smoking, hypertension, diabetes mellitus, and parental CHD history. Others find complementary patterns of results when looking at patterns of DNA methylation in inflammatory genes [34] and circulating levels of inflammation in plasma [35].

Although inflammation is critical for survival, excessive or prolonged inflammation is known to promote or exacerbate diseases of the cardiovascular, metabolic, musculoskeletal,

nervous, and immune systems [36] and contributes to accelerated aging and reduced longevity [37,38]. For example, chronic inflammation is now theorized to be the common pathway of many stress-related diseases, such as cardiovascular disease (CVD) [39], which the World Health Organization has identified as the leading cause of death globally [40]. Indeed, the burden of CVD and other inflammation-mediated diseases are disproportionately borne by the poor [41], suggesting that development of a proinflammatory phenotype in response to early life adversity may have a lasting negative impact on health.

In addition to the negative impact that unchecked bodily inflammatory activity itself can have on health and longevity, recent research implicates inflammation in impulsivity and the desire for immediate versus delayed rewards [42**,43*]. The desire for immediately available resources in the context of inflammation can promote survival by increasing access to the resources needed to aid in bodily repair and recovery without the body needing to reallocate these resources from other functions. However, when inflammation is prolonged or protracted — as often occurs among those from low childhood SES — such decision patterns can exacerbate poor health (see Figure 1; [42**]). For example, impulsivity is known to predict a number of health-harming behaviors, such as smoking, excess alcohol consumption, binge-eating, and substance use [see e.g. 44]. These behaviors, which themselves promote inflammatory activity, can create a health-harming feedforward cycle of inflammation and health harming behaviors that can be difficult to escape (see Figure 1; [42**]), creating even greater health disparities over time [45]. Indeed, research finds that low childhood SES — in addition to its impact on one’s tendency toward inflammation — is also predictive of a number of behavioral tendencies that, themselves, could exacerbate inflammation, including tobacco use [46,47], alcohol dependence [48], substance use [49], risky sexual behavior [50], and general risk taking [51].

Early life socioeconomic disadvantage and eating in the absence of hunger

In addition to having an impact on the development of the immune system, growing up in disadvantaged environments can also contribute to health disparities through its impact on developmental tradeoffs that help maximize available energy in environments where nutrients and resources are scarce. Because conditions in early life provide a blueprint for the types of environments one is likely to encounter in adulthood, life history theory predicts that developmental exposure to conditions typical of low SES — which tend to be relatively resource scarce — will promote the development of an adult phenotype that is well-adapted to survive in such conditions [52]. Consistent with this hypothesis, research finds that exposure to resource scarcity in utero and in early childhood plays an important role calibrating physiological and psychological development, encouraging the

Figure 1

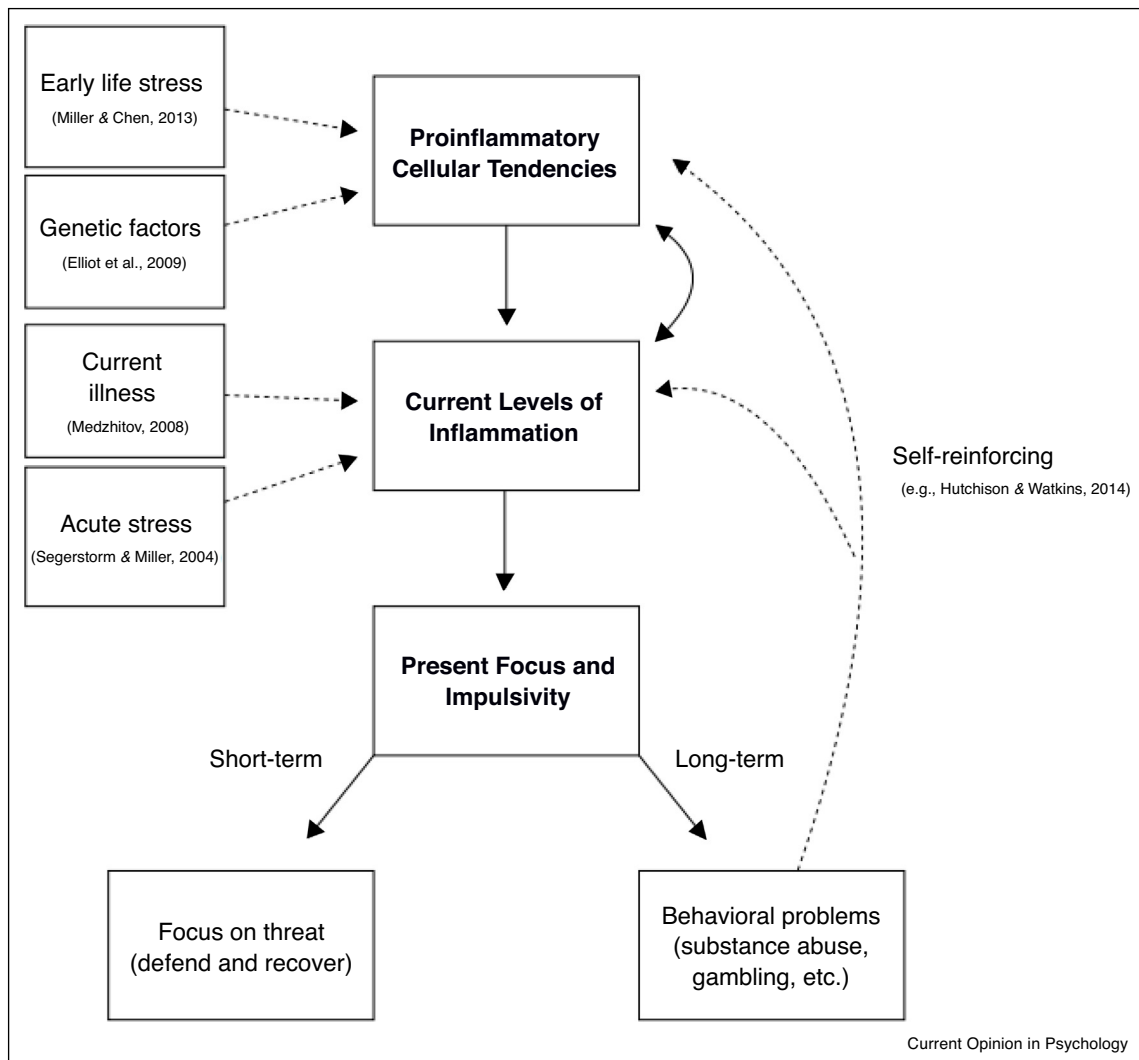


Figure taken from Gassen *et al.* [42**]. Inflammation tends to be self-reinforcing, making the impact of early life disadvantage on health difficult to escape.

development of a *thrifty phenotype*, which is characterized by a smaller body size, slower metabolism, and a reduced level of behavioral activity [53].

More recently, researchers have built off these ideas to examine whether growing up poor might also impact the mechanisms that govern food intake in ways that would promote survivability in resource scarce environments. Mechanisms of homeostatic energy regulation typically develop such that current energy needs play an important role in regulating food intake, such that people typically eat more when hungry than when full. However, in low SES environments — where access to food may be unpredictable or scarce — such an eating strategy doesn't optimize energy availability. Instead, because excess calories can be stored on the body in the form of adipose tissue, it makes better survival sense to eat when food is available, even if

current need is low. Although eating in the absence of bodily need is associated with obesity in contemporary food-rich environments [54,55], it would help promote survivability in those that are resource-scarce.

Support for this hypothesis has now been found across several studies [56**,57*]. For example, in one study, researchers had men and women come into the lab after an overnight fast. Participants were then randomly assigned to drink a 12-ounce Sprite (which decreased energy need) or a 12-ounce lemon-lime mineral water (which kept energy need high). Participants were then given a bowl of snacks from which they were free to eat as much or as little as they desired. Results are shown in Figure 2 [56**]. Although those who grew up in higher SES environments ate less when energy needs were lower than they did when energy need was high, those from

Figure 2

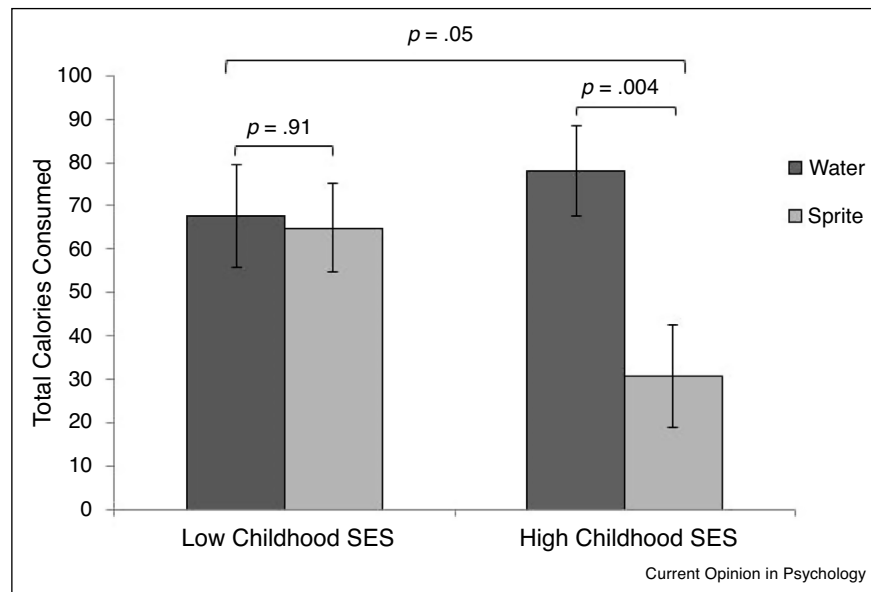


Figure taken from Hill *et al.* (Study 2) [56**]. Growing up in conditions of low SES decreases the role that one's current energy needs play in guiding eating behavior. Participants from higher SES childhood environments ate differing numbers of calories, depending on their energy need. Participants from lower SES childhood environments ate a comparable number of calories across levels of energy need.

lower SES environments eat comparably high numbers of calories regardless of their current energy needs. Further, consistent with the hypothesis that these patterns emerge from differences in one's developmental conditions (rather than one's adult SES, which often covaries with the conditions present in childhood), these results were found to be specific to one's childhood and not adult SES.

Additionally, follow-up research has found that these patterns are driven by lowered body awareness, or knowledge about one's internal states, such as hunger or temperature, among those from lower SES environments [57*] and are already manifest in children as young as three (Proffitt Leyva *et al.*, unpublished). Growing up in low SES conditions and all that this entails — greater unpredictability, less safety, and more harsh and inconsistent parenting — contribute to eating in the absence of hunger [56**,57*, Proffitt Leyva *et al.*, unpublished]. Although these patterns would have helped facilitate survival in the types of unpredictable and resource scarce ecologies regularly confronted by our ancestors, they may contribute to the greater obesity risk found among those who grow up in the context of contemporary, Western poverty [see e.g. 58*]. Calories, rather than being scarce, as is the case in historical resource scarce environments, are abundant, but low quality. This has created a paradox in which obesity is often comorbid with hunger and undernutrition in the context of contemporary, Western poverty [59].

Although more research is needed in order to understand the etiology and mechanisms that govern the divergent

patterns of eating behavior observed in those from low-SES versus high-SES childhood environments, what is known suggests that the impact of childhood SES on eating behavior is long-lasting and not easily reversed. Indeed, much research has now found evidence of a powerful link between low childhood SES and obesity and its related co-morbidities [see e.g. 60,61,58*].

Conclusions

Growing up in conditions characterized by low socioeconomic status may contribute to patterns of development and behavior that can have negative consequences for health. Although there is a tendency to pathologize the developmental patterns that are favored in the context in socioecological disadvantage, they may reflect adaptive developmental fine-tuning to better match one's adult phenotype to the unique demands of disadvantaged environments. This is not to argue that such developmental patterns — particularly when they are disadvantageous to health — are desirable or should be encouraged. Instead, by identifying the characteristics of the environments that promote the development of patterns that contribute to health disparities, this perspective highlights what are likely to be the most effective targets of intervention. Changing the negative health and behavioral outcomes of those growing up in difficult socioecological circumstances will likely require changing these environments in meaningful ways. It is our hope that the results of the reviewed research suggest promising targets for such intervention.

Conflict of interest statement

Nothing declared.

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