Why Are Childhood Family Factors Associated With Timing of Maturation? A Role for Internal Prediction

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Abstract

Children, particularly girls, who experience early familial adversity tend to go on to reach sexual maturity relatively early. This feature of adolescent development is believed to be an evolved strategy that arose because individuals with genes that caused them to mature relatively early under certain conditions left behind more descendants than those who did not. However, although much has been done to uncover the psychological and physiological mechanisms underlying this process, less attention has been paid to the evolutionary reasons behind why it might be advantageous. It has previously been suggested that this strategy evolved because early familial adversity accurately indicated later environmental adversity, under which conditions early reproduction would likely maximize evolutionary fitness. In this paper we contrast this 'external prediction' model with an alternative explanation, which builds upon the existing explanation and is mutually compatible with it, but which is distinct from it. We argue that accelerated development is advantageous because early adversity detrimentally affects the individual's body, increasing later morbidity and mortality; individuals may adapt to this internal setback by accelerating their development. Unlike the external prediction model, this 'internal prediction' relies not upon temporal environmental continuity, but on long-term effects of early circumstances on the body.

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Many studies have found associations between aspects of the family environment experienced in early life and the onset of reproductive maturity. Most well-known among these findings, in social contexts where nuclear families predominate, menarche occurs at a younger age among girls with 'absent' fathers (B. Jones, Leeton, McLeod, & Wood, 1972; Moffitt, Caspi, Belsky, & Silva, 1992; Tither & Ellis, 2008). Studies that investigate the apparent effects of family circumstances in detail have revealed that early menarche occurs in girls with less affectionate and cohesive parent-child relationships (Chisholm, Quinlivan, Petersen, & Coall, 2005; Graber, Brooks-Gunn, & Warren, 1995; Steinberg, 1988), those who experience greater parent-child conflict (Graber et al., 1995; Kim & Smith, 1998; Mezzich et al., 1997), or who are exposed to greater parent-parent conflict (Chisholm et al., 2005; Ellis & Garber, 2000; Ellis, McFadyen-Ketchum, Dodge, Pettit, & Bates, 1999), and those who experienced physical or sexual abuse (Costello, Worthman, & Erkanli, 2007; Turner & Runtz, 1999; Vigil, Geary, & Byrd-Craven, 2005). Studies measuring age at first sexual activity or first pregnancy reveal patterns similar to those examining onset of menarche (Barglow, Bornstein, Exum, Wright, & Visotsky, 1968; Dorius, Heaton, & Steffen, 1993; Nettle, Coall, & Dickins, 2011). The association between familial adversity in childhood and early maturation is often referred to as "psychosocial acceleration" (Ellis, 2004)

A crucial question is whether associations between family environment and maturational timing result from a causal role of the family environment, or result from other processes, such as genetic correlations between parents and offspring. Studies

controlling for maternal age at menarche, taken in sum, suggest that both genetic correlations and effects of the family environment play a role (Belsky, Steinberg, Houts, Halpern-Felsher, NICHD Early Child Care Research Network, 2010; Mendle et al., 2006) as do studies that have controlled for genetic effects through twin or sibling designs (D'Onofrio et al., 2006; Mendle et al., 2006; Rice et al., 2010; Tither & Ellis, 2008). Recent structural equation modeling aimed at identifying causal pathways supports effects of family relationships on age at pubertal development (Belsky et al., 2010; James, Ellis, Schlomer, & Garber, 2012; Neberich, Penke, Lehnart, & Asendorpf, 2010), and so does the 'natural experiment' provided by a study showing that wartime separation Finnish children from their parents was associated with earlier menarche (Pesonen et al., 2008). Studies of other mammals have shown similar relationships between rearing environments and sexual development (Cameron, 2011; Cameron et al., 2008; Maestripieri, 2005) most convincingly by experimental cross-fostering of rat pups between mothers bred for different levels of maternal grooming (Cameron et al., 2008).

'External Prediction' as an Explanation of Psychosocial Acceleration

Within and between populations, age at maturity varies greatly, and a large part of this variation appears to be non-genetic in origin (Belsky, Steinberg, & Draper, 1991; Walker et al., 2006). Age at maturity thus provides an example of phenotypic plasticity: the ability of a genotype to produce different phenotypes, depending on environmental conditions. Because age at maturation is closely linked to reproductive lifespan, and thus in most contexts also to the number of offspring an individual is likely to produce (their evolutionary 'fitness'), we can expect that evolutionary forces will play an important part in explaining plasticity in age at maturity. Over

evolutionary history, natural selection ensures that individuals are generally efficient at extracting resources from the environment, and converting those resources into descendants. Selection for this efficiency leads to trade-offs between different activities that can ultimately serve that purpose. When a female becomes sexually mature, she is potentially diverting resources away from her own growth towards reproduction, disadvantaging herself and her offspring in some respects (e.g., producing smaller neonates with lower survival prospects (Rickard et al., 2012)), but gaining an advantage of earlier reproduction (e.g., offsetting the risk of dying herself before becoming reproductively successful). The optimal age for a female to become sexually mature changes as the costs and benefits of starting to reproduce early relative to delaying alter. Shifts in this optimum may be partly responsible for variation in age at sexual maturation. Variation in the age at which girls become sexually mature may therefore be an example of adaptive phenotypic plasticity, whereby individuals respond to changing environments in ways that maximize reproductive success in those environments (Stearns & Koella, 1986)

Belsky et al. (1991) proposed a hypothesis in which the family environment experienced during childhood provides the individual with information about "the availability and predictability of resources (broadly defined) in the environment, of the trustworthiness of others, and of the enduringness of close interpersonal relationships" (Belsky et al., 1991, p.650). Having sampled this information, the child might infer that it was relevant to not only the current state of the environment (e.g., as being unsupportive or dangerous versus supportive or benign), but also to the likely future environment (Figure 1, top panel). She would then use this information as a 'cue' to guide appropriately her reproductive development (e.g., by accelerating it). Such a response need not rely on conscious calculation of the optimal response, or

even explicit recognition of the environmental risk. Rather, effects on physiological development may be mediated by any number of candidate autonomic, neuroendocrine, metabolic, and immune mechanisms (Belsky et al., 1991; Del Giudice, Ellis, & Shirtcliff, 2011; Ellis, 2004).

The model of Belsky et al. (1991) is of particular importance because the relationship between familial environment and pubertal timing was in fact a novel prediction derived from their paper. Subsequent empirical support for this 'uncanny' prediction has led to their model being highly influential, with several elaborations being put forward (Belsky, Schlomer, & Ellis, 2012; Boyce & Ellis, 2005; Chisholm, 1993; Del Giudice, 2009; Del Giudice et al., 2011; Ellis, 2004; Ellis, Figueredo, Brumbach, & Schlomer, 2009; Frankenhuis, Gergely, & Watson, 2013a). The idea that individual humans tailor their pace of sexual maturation to their expected future external environment has become the dominant evolutionary explanation for relationships between childhood environment and sexual maturation. For the purpose of our argument, we collectively refer to such explanations as 'external prediction' models, which we will later contrast with a different possibility, termed 'internal prediction'.

Deriving Information From the Environment to Predict the Future

The benefit of adaptive phenotypic plasticity is that it increases the adaptive fit of individuals to their circumstances (West-Eberhard, 2003), that is to say, it molds their phenotype in such a way that it increases their likely reproductive success under a particular set of conditions. However, just as with the evolution of any trait, in order for plastic traits (such as a developmental 'switch' leading to psychosocial acceleration), to evolve, the benefits of the ability to respond to the environment in

that way must outweigh its costs. Developmental plasticity often requires a degree of commitment to a particular phenotype (Frankenhuis & Panchanathan, 2011a): decisions that occur during the construction of an adult body cannot always be easily undone, a fact that has long been appreciated by those studying psychosocial acceleration (Belsky, 2000; Ellis, 2004; Frankenhuis & Del Giudice, 2012). Where individuals permanently calibrate aspects of their phenotype based on early-life experience, they are effectively predicting the future based on imperfect information available in the present. In considering the plausibility of external prediction models, we must therefore be sure to weigh the potential advantages of plasticity by the probability of a prediction being true, and the disadvantages by the probability of a prediction being wrong (Rickard & Lummaa, 2007; Stephens, 1991). In particular in long-lived animals like humans, the environment that is used for guiding development might well change before maturity is reached, in which case prediction on the basis of childhood experience would not be useful.

There are some examples in nature of plastic responses that appear to involve the use of external information available in early life to make long-term predictions, although examples of such plasticity being adaptive may be rare (Uller, Nakagawa, & English, 2013). In particular, in longer-lived animals including humans, evidence showing that early experiences improve performance in adulthood in similar conditions has not been forthcoming (Hayward & Lummaa, 2013; Hayward, Rickard, & Lummaa, 2013; Nussey, Kruuk, Morris, & Clutton-Brock, 2007). This absence of evidence might be because researchers have not gathered the relevant data, or it could be because there is less scope for natural selection to favor such strategies in animals that have longer lifespans. The extent to which such strategies could have been favored by natural selection depends on the historical degree of temporal continuity in

fitness-determining aspects of environment. The degree to which—across evolutionary time—environmental dimensions were stable within human lifespans is an open and important question, which warrants more empirical attention than it currently receives. Likely, the level of temporal environmental continuity will vary between dimensions: e.g., climate, disease, predation, violence, social organization, position within the social hierarchy, etc. Some of these dimensions might have had a relatively high degree of continuity, others a lesser degree.

The existing external prediction explanations for psychosocial acceleration depend on family factors being reliably associated—across evolutionary time—with prevailing environmental conditions. They also rely on the temporal continuity of ancestral environments being sufficiently high so that fitness-relevant environmental features remained stable for periods of at least one or several decades. Yet at the same time, environments must have been variable enough for natural selection to maintain plasticity: this requirement is because in environments that are highly stable *across generations*, mechanisms supporting plasticity become superfluous and may be disfavored because they are costly to produce. We have recently developed a model, tailored to the human life history (where many years pass between birth and maturity), examining the conditions necessary for plasticity in human reproductive strategy to be adaptive. Results show that extremely high levels of temporal environmental continuity are required (Nettle, Frankenhuis, & Rickard, 2013), echoing results from an evolutionary model of developmental plasticity in metabolic phenotype (Baig, Belsare, Watve, & Jog, 2011).

An Alternative Account: The Role of Internal State

Accounts of biological processes argued to have come about through natural selection can be said to require both proximate (mechanistic) and distal (functional) explanations. Proximate (mechanistic) explanations account for the physiological and/or psychological processes involved, whereas ultimate (functional) explanations are concerned with how these processes influence fitness.

Drawing primarily upon behavioral ecology, but also from similar ideas that have been put forward in the context of human metabolic plasticity (J. Jones, 2005; Wells, 2012), we here propose an alternative model of *why* psychosocial acceleration might have been favored by natural selection under conditions of social adversity. This model invokes some of the same proximate phenomena as the existing 'external prediction' models, but relies on a subtly different ultimate argument. Importantly, the processes described in this model are not mutually exclusive with the processes described by 'external prediction' models, but could exist alongside and be complementary to them. Despite the fact that internal and external prediction processes are not mutually exclusive, the internal prediction argument we put forward does raise the empirical question of which of the two processes has been more important in the evolution of human plasticity—and as we outline at the end of the paper, the answer to this question has practical implications. At the ultimate level of explanation, 'external prediction' models of psychosocial acceleration focus on the environment to be adapted to as external to the individual (outside the bodily envelope). However, as proponents of such models acknowledge, optimal development and behavior depend not only on the external environment, but also on internal 'somatic' (i.e., of the body) factors that vary between individuals (such as body size, energetic reserves, immune functioning, quality of cell-repair mechanisms, and other aspects of condition (Frankenhuis, Panchanathan, & Clark Barrett, 2013b;

Mangel & Clark, 1988; McNamara & Houston, 1999). These factors are usefully described by the concept of internal 'state' (McNamara & Houston, 1999), which shapes individual fitness just as does the external environment, but with effects that are specific to each individual, resulting from his or her own particular history of genetic and environmental influences. We now discuss the potential of internal state to play a key role in adaptive developmental plasticity.

'Silver Spoon' Effects On State

Some aspects of state, such as hunger, will be of a transient nature; others may be remarkably stable and persist for substantial portions of an individual's life. Adult state can be profoundly and permanently influenced by environmental conditions that an individual experiences during development. Consider, for instance, an individual who suffers from physical damage in early embryogenesis (the developmental stage where the major organs are being formed); such damage will endure a lifetime. This example is just one of a phenomenon that is widespread in biology, whereby shortfalls of resources or other adversities during development affect the individual's adult phenotype in ways that are detrimental to their fitness potential. In ecology these are termed 'silver spoon' effects, after the apparently enduring effects of long-term affluence in early life with which humans are only too familiar (Grafen, 1988). The reason why early-life experiences have a big impact is that it is during this life stage that an individual is putting in permanent place the building blocks of his or her body: an individual will always live with the body he or she developed in fetal life, infancy and childhood, and never another one (Gavrilov & Gavrilova, 2004). Early-life adversity can thus have profound consequences for individual evolutionary fitness. This principle is supported by a body of evidence from the ecological

{Rickard:2010dt, Uller:2013dy}, laboratory (Bertram & Hanson, 2001) and epidemiological (Gillman, 2005) literatures that shows how adversity in early life has effects on individual phenotype that will on average lead to reduced fitness, and that even if individuals with compromised early starts in life get the opportunity to 'catch up' in terms of growth, they still pay fitness costs in the long-term (reviewed in Metcalfe & Monaghan, 2001).

Individuals Adapt To Their State

Just as different external environments favor different responses, so too do different internal states. For instance, an individual who is currently nutrient-deprived might invest its available energy and time in food acquisition, whereas a sated individual might more profitably invest in other traits or activities (e.g., seek mates). Thus the tendency of a particular behavior to increase fitness varies with individual state, and evolution should favor strategies that appropriately adjust an individual's behavior in accordance with their state. In behavioral ecology the concept of 'status-dependent alternatives' (Gross, 1996) describes the idea that aspects of an individual's state, such as body condition or social status, determine the optimal behavior that it should adopt in order to likely maximize its evolutionary fitness.

In many species, individuals develop profoundly different behavioral repertoires or subsequent physical characteristics depending on aspects of state (e.g., size) at a critical point in development (Emlen & Nijhout, 1999; Gross, 1985; Smallegange, 2011). For example, in the bulb mite *Rhizoglyphus robini*, males develop one of two life-history strategies: 'fighters', which sport a specialized third pair of legs with which they can kill other mites, or 'scramblers', which have unmodified legs and are defenseless. Fighter adults are more likely to develop from

larger juveniles, and scrambler adults from smaller juveniles. Why should this be? The advantages of adopting a 'fighter' strategy are dependent on the individual's competitive ability, which depends on physical size (Smallegange, 2011). If the developing individual is unlikely to be competitive, it may pay, in fitness terms, to adopt an alternative (scrambler) tactic, rather than play a high-stakes (fighting) game in which it runs a high risk of losing.

Consideration of the above two phenomena in combination illuminates how silver spoon effects on individual state can induce adaptive variation that may be of general importance in understanding developmental plasticity in humans and other animals. In the bulb mite, experimental administration of a rich diet increases the size of individuals at the end of their juvenile life stages, and in turn increases the likelihood that they will develop into 'fighters' (Smallegange, 2011). This example is based upon two extreme morphs whose divergence far outstrips anything seen in any mammal, let alone humans. However, it nonetheless usefully illustrates a principle that may apply to continuously varying aspect of state, and corresponding adaptive strategies in many species, including humans. We here call this principle 'internal prediction' in order to distinguish it from prediction of the external environment; the individual uses its current internal state to determine the best behavioral strategy for it to adopt in later stages of its life.

The utility of internal prediction does not depend on continuity of external environments between early and adult life, but instead on internal state in early life affecting internal state in later life. The stronger this effect is, the greater will be the strength of selection for developmental mechanisms that tailor the development of

behavioral strategies to internal state, as the lower will be the risk of an individual developing a maladaptive phenotype by doing so (Nettle et al., 2013).

Internal Prediction and Psychosocial Acceleration

Following on from the general argument above, we now make the case for internal prediction as a potential explanation for the phenomenon of psychosocial acceleration. The result is a subtle, but significant, recasting of the hypothesis outlined by Belsky et al. (1991). It takes inspiration from their original 'external prediction' formulation, and concords with it in viewing the relationship between early adversity and age at sexual maturity as being due to an evolutionarily adaptive plastic response to the long-term consequences of that adversity. However, whereas the model of Belsky et al. involves individuals adapting to the external environment, the 'internal prediction' model involves them (alternatively or additionally; discussed below) adapting to their internal, somatic state. Thus, although some of the same *proximate* mechanisms may be (partially) involved in mediating adaptive developmental effects in both processes, the internal prediction and external prediction models argue for different *ultimate* evolutionary functions.

We begin by discussing, in broad strokes, the similarities and differences between external and internal prediction models. Both models can be conceptualized as involving three components: (A) exposure to psychosocial stress, (B) biological embodiment of the effects of stress, and (C) adaptive development of a reproductive strategy (Figure 1). The respective details of these three components differ in internal vs. external prediction models. External prediction models propose that the function of B is to regulate C in order to match A, whereas in the internal prediction model, A

influences B, and then C is regulated to match B, but there is no implication that A and C go together or are coordinated in a functional manner[1].

We now elaborate what the internal prediction model of psychosocial acceleration claims happens at each of these stages. First, individuals experience psychosocial stress (A). Second, psychosocial stress has negative long-term cellular and molecular effects on the body that increase morbidity and mortality risk, not only immediately but also enduringly (B). The consequence of these effects is to shorten likely healthy reproductive lifespan. Third, the body assesses its likely healthy reproductive lifespan as being relatively shortened, and accelerates reproductive maturation as an adaptive response to its own internal state (C).

Consequences of psychosocial stress

It may seem paradoxical to claim both that the stress system is an evolved, adaptive mechanism, and that stress damages the body; however, both of these statements are likely to be true. The resolution of the apparent paradox is that the function of the stress system (primarily mediated in humans, including human children, by the hormone cortisol) is to divert bodily resources to the short-term ability to respond to dynamic, demanding or threatening situations, and away from other functions whose importance is only felt in the much longer term (McEwen & Wingfield, 2003). These functions include growth, development, self-maintenance and tissue repair. Self-repair of bodily tissues is constantly required, as metabolism continuously produces oxidative stress. Oxidative stress refers to the net effects of many reactive oxygen species (ROS) that arise through normal metabolic activity and which damage DNA, protein and lipids, hence cumulatively causing a decline in

[1] With thanks to the anonymous reviewer who suggested this useful mode of explanation.

function (Monaghan, Metcalfe, & Torres, 2009). Oxidative stress also damages telomeres, the protective 'caps' on the end of chromosomes (Zglinicki, 2002). When telomeres become critically short, cells become unable to replicate accurately, with negative consequences for tissue function. Telomere length has been found to be a good predictor of an individual's future health and longevity in humans (Bakaysa et al., 2007; Kimura et al., 2008; Njajou et al., 2009).

The negative effects of oxidative stress can be counteracted to some extent by investment in antioxidant activity and repair mechanisms, and telomeres can be maintained by production of the enzyme telomerase (Blackburn, 1991). These self-repair processes, along with immune activity (Segerstrom & Miller, 2004) and bone formation (Chyun, Kream, & Raisz, 1984) are the kinds of long-term investments in the body that are turned down by the cortisol-mediated stress response as it diverts energy and optimizes physiological state in the pursuit of more immediate survival priorities (Gidron, Russ, Tissarchondou, & Warner, 2006; Joergensen et al., 2011; Zafir & Banu, 2009). In life-history terms, these investments are subject to being 'traded-off' when immediate need for investment in urgent other fitness-related activity is high.

In view of the processes described above, it is not surprising that psychosocial stress has negative, and extremely well documented, effects on long-term bodily function. This includes psychosocial stress experienced during childhood. Empirical evidence support can be found in epidemiological studies (Miller, Chen, & Parker, 2011). For example, parental divorce during childhood is associated with poorer self-rated health in young adulthood (Roustit et al., 2011) and with reduced life expectancy (Schwartz et al., 1995), and being physically abused as a child is associated with an increased risk for a wide range of health problems in adulthood

(Wegman & Stetler, 2009). Telomere erosion rates are also higher in individuals who have experienced social adversity during early life (Entringer et al., 2011; Epel et al., 2004; Kananen et al., 2010), and telomere erosion, as discussed above, predicts subsequent health and lifespan. Although studies of the health impacts of early stress in humans control for obvious confounds such as the continuing effect of the social environment later in life, they are necessarily correlational in design. However, clean demonstrations exist in rats, where experimentally elevating glucocorticoid levels in pups has long-term fitness-negative effects on aspects of neurological development (Neal, Weidemann, Kabbaj, & Vázquez, 2004), renal function (Liu et al., 2008), hypertension (Tonolo, Fraser, Connell, & Kenyon, 1988) and survival (Liu et al., 2008).

Thus, overall, the chronic or repeated activation of stress mechanisms by psychosocial conditions during childhood will plausibly lead to an adult body that is less physically robust, and has accumulated more oxidative damage and telomere loss, than it would have done if that stress had not been experienced. Such a body will likely experience a shortened expected period of healthy reproductive life before it succumbs to mortality or morbidity (Geronimus, 2013).

Accelerating Maturation In Response to Internal State

The final component to our argument states that it is adaptive for an individual to respond to an increased morbidity-mortality risk by accelerating maturation. This is indeed optimal when local rates of ‘extrinsic’ mortality and morbidity are high, because the benefits of delaying reproduction are offset at a younger age by the risk of failure to reproduce, or reduced reproduction resulting from early death (Chisholm, 1993; Nettle et al., 2011; Stearns & Koella, 1986). ‘Extrinsic’ in this context merely

means that the individual can do nothing to alter these factors. However, *extrinsic* does not have to mean *external*. If the individual's somatic condition is irreversibly damaged by what occurred during childhood, such that her subsequent health and survival is poorer, then that individual faces a higher personal extrinsic rate of mortality and morbidity than other individuals experiencing the same external environment but who did not experience the same damage. Thus, just as accelerated development might be adaptive when the externally imposed risk of extrinsic morbidity-mortality is high, it may also be adaptive when the risk of morbidity-mortality is increased due to internal causes. Exactly how the body is able to sense its own state is not clear, but there is no principled reason that cues from the internal milieu – levels of ROS, or damaged cells, for example – should not be available to hormonal and neural systems that control behavior and sexual development.

Predictions of the Internal Prediction Model

The internal prediction model of why psychosocial acceleration is adaptive states that individuals experiencing childhood psychosocial stress should accelerate their maturation because early-life social adversity 'damages' their internal state, increasing their levels of morbidity-mortality and shortening their expected (reproductive) lifespan. As in external prediction models, in our alternative view individuals are responding adaptively to their likely future, shifting towards a faster reproductive strategy when future prospects are poor. However, according to the model we propose, this prediction does not rely upon a forecast of parameters of the external environment, but rather upon effects of the early environment on the long-term health state of the individual's body. Although the internal prediction model involves cues of the external environment being assimilated into the individual's

soma and thereafter embodied in it, the process we propose here involves the individual's internal state *itself* determining reproductive lifespan, albeit probabilistically, irrespective of future external environment.

Our alternative account is compatible with findings used to support Belsky et al.'s (1991) evolutionary account of psychosocial acceleration (e.g., early adversity is associated with accelerated maturation). Yet the two accounts are different in some respects. The internal prediction model makes several predictions that allow the assessment of the degree to which it is empirically valid. Before listing these, we emphasize again that the internal prediction model is not mutually exclusive with the models proposed by Belsky and colleagues (and extensions of these models). Mechanisms determining timing of maturation might integrate cues about both internal and external state (Fawcett & Johnstone, 2003; Frankenhuis et al., 2013b), and the evolutionary relationships assumed in the two models could co-exist, with their respective importance for the evolution of human plasticity to be determined empirically (Nettle et al., 2013). However, the predictions we discuss below follow more directly from the ultimate role of internal prediction in guiding psychosocial acceleration than they do from models based purely on external prediction.

1. Non-social And Social Adversity Similarly Affect Health And Rate Of Maturation

Although we have in this paper addressed the specific example of the effects of psychosocial stress, the application of the internal prediction model to sexual maturation rates is not restricted to this kind of stress. In fact, the model predicts that any adversity likely to cause damage to somatic state should be associated with accelerated reproductive development. For instance, Waynforth (2012) recently

showed that British girls who experienced chronic disease in childhood developed accelerated reproductive strategies in adulthood, even though the incidence of chronic disease was uncorrelated with other measures of ecological stress (e.g., socio-economic status, father absence). Childhood disease is not amongst the social adversities usually studied in the context of psychosocial acceleration, but it is likely to be associated with later morbidity-mortality, and so its association with reproductive acceleration is consistent with the internal prediction model.

2. Internal State Mediates The Link Between Early Adversity And Rate Of Maturation

There are well-established links between childhood social adversity and mortality-morbidity in later life (Roustit et al., 2011; Schwartz et al., 1995; Wegman & Stetler, 2009). For the internal prediction model, such links are expected and indeed their existence is the reason that psychosocial acceleration is adaptive. Purely external prediction accounts have to explain them more indirectly; for example, early social adversity is embodied via neural or endocrine mechanisms that, as a side effect of their main function of calibrating the individual to her external environment, have an impact on later health. Alternatively, they may arise as a consequence of individuals favoring reproductive effort over somatic effort (Del Giudice et al., 2011). Thus the internal prediction model gives a more central significance to effects of early environment on measures of general health over time. Possible markers of general health that could be studied in this regard would be levels of oxidative stress or telomere length (see above), or developmental instability (Hope et al., 2013; Penke et al., 2009).

3. Childhood Adversity Precedes Somatic Damage, Which Precedes Accelerated Maturation

The correlation between poor individual health and psychosocial acceleration may be accommodated with the external prediction model by it being a consequence of individuals favoring reproductive effort over somatic effort (Del Giudice et al., 2011). This view leads to the expectation that some somatic damage follows the adoption of an accelerated reproductive strategy. However, in the internal prediction model, the order of events is reversed: damage precedes an accelerated reproductive strategy. Therefore, the two models make different predictions about the sequence of changes to the individual's soma and life-history strategy, the internal prediction model explicitly proposing that early damage precedes adjustment of reproductive strategy, and the external prediction model emphasizing that some damage to state will follow it. Informative in this respect will be the extent to which measurable damage to the soma (e.g., in terms of changes in telomere length in early vs. late childhood) precedes vs. follows the developmental stages at which pubertal timing is determined.

4. Early Adversity Negatively Influences Fitness, Even When Early-life And Adult Conditions Match

External prediction models predict that an individual's evolutionary fitness in our ancestral environment would have been the product of the extent to which information upon which they based their developmental decisions was reliable, i.e., the early environment was predictive of the adult environment. If the purpose of plasticity is to allow individuals to better 'match' to their future environment, there must be disadvantages—on average—to making the 'wrong' decision, i.e., to

experiencing a mismatch (Belsky, 2000; Frankenhuis & Del Giudice, 2012; Rickard & Lummaa, 2007; Stephens, 1991). If external prediction processes have been important, then there should be reduced evolutionary fitness in individuals for whom the early-life and adult environments are discordant (e.g., benign-harsh relative to harsh-harsh). On the other hand, if internal state processes are relatively more important, then there should always be a fitness advantage to having had a benign early environment, regardless of what the adult environment is like. Such empirical tests of adaptive developmental plasticity have recently been carried out in other contexts and have not found strong evidence for prediction of the external environment (Hayward & Lummaa, 2013; Hayward et al., 2013). For a detailed discussion of the predictions of the effects of environment on fitness under different kinds of plasticity, see Uller et al. (2013).

Implications for Health and Disease

We have written this paper from a basic science perspective. However, understanding the determinants of environmental variation in rates of maturation is of interest from a medical perspective, because the onset of physical and physiological adulthood is defined by an array of changes that have profound implications for many aspects of the body's ability to function. Beyond that, the relationships between health and age at menarche (Cho et al., 2012; Webb, Marshall, & Abel, 2011; Widen et al., 2012) suggest that understanding why individuals differ in the rate at which they enter puberty may yield insights into the causes of inter-individual variation in health and propensity to disease.

We may go further: What we have considered in this article is the extent to which internal vs. external prediction models are empirically valid as functional

explanations for a well-described developmental phenomenon. This question has implications for our understanding of precisely how early family environment, sexual maturation rates, and health, are interrelated. These details have real-world implications for our understanding of the etymology of health differentials. We now consider what clinical importance our predictions might have.

In the internal prediction model, general health is of pivotal importance. Long-term variation in morbidity-mortality is emphasized as a major reason why variation in pubertal timing, as indexed by age at menarche, exists. The internal prediction model thus suggests that early age of menarche is likely to be a reliable marker of poor long-term health (at least within populations). Although the external prediction model is not incompatible with age at menarche being related to later health, it is nonetheless useful to recognize the special significance that is attached to the maturation-health relationship by the internal state model. In this case accelerated reproduction is claimed to reflect an increase in bodily damage that has been instigated many years in the past and will be difficult to reverse, whereas external prediction models (to varying degrees) are more likely to consider such damage as being caused by the ongoing reproductive strategy, which may thus be reversible. The internal prediction model's emphasis on the environment shaping individual health supports the assertion that removing children from abusive, stressful or otherwise harmful environments as early as possible would be of *paramount* importance for improving future health prospects. If internal prediction has been a powerful force in influencing human developmental plasticity, the lesser the potential there is to reverse effects of early adversity and the earlier such effects leave their mark on phenotype.

The issues of timing and relative irreversibility in the internal prediction model have broader implications for understanding the extent to which early

environment shapes health. If stress-inducing effects of the early environment on individual state are indeed significant enough to have shaped the evolution of plastic reproductive strategy, then this is a strong indication that such effects have constituted a *profound* selection pressure on our phenotypes over the course of our evolutionary history. This fact would place emphasis on the importance of understanding how effects of the early social environment become embodied in influencing health disparities. Furthermore, it would lead to the prediction that plasticity in other life-history traits (e.g., future discounting) may have been shaped adaptively by the effects of the early social environment on internal state.

Conclusions

We have highlighted differences between our internal prediction model and prevailing external prediction models of why psychosocial acceleration exists. However, we conclude by reiterating similarities. We agree it is likely to be adaptive to accelerate maturation when childhood family conditions are harsh, because future prospects tend to be poor where childhood conditions are harsh. Our model makes the single modification to the argument that one functional *reason* future prospects are poor where childhood environment is harsh may be the detrimental effects of harshness on the developing body. The process we propose involves individuals adapting not, or not only, to their future external environment but rather to their own bodies (or internal state). We thus uphold some of the contentions of Belsky et al. (1991) and others but provide an alternative, or additional, reason for why those contentions may hold. In particular, we have built upon the theoretical successes of external prediction models in explaining patterns of variation less in terms of pathology or systemic dysregulation (McEwen & Wingfield, 2003), and more in

terms of coordinated adaptive adjustments (Belsky et al., 1991; Del Giudice et al., 2011; Ellis, Del Giudice, & Shirtcliff, 2013), which are likely to be, in some form, the result of natural selection acting on individuals experiencing variable environments. The internal prediction model recognizes the existence, and importance, of pathology, but advances the idea that interactions between pathology and adaptive adjustment have been important over the course of human evolutionary history.

We have not addressed the fact that levels of plasticity itself may vary across individuals ("differential susceptibility"); that is, some individuals are more affected than others by the same kinds of social experiences (e.g., maltreatment, social support), not only in terms of immediate impact, but also long-term developmental response (Belsky & Pluess, 2009; Frankenhuis & Panchanathan, 2011b). There are at least two interesting implications of internal prediction for theorizing about differential susceptibility. First, individuals might be differentially susceptible in the extent to which their somas are detrimentally impacted by early-life stressors (e.g., due to prior differences in condition). Our model predicts that individuals whose somas are more susceptible to damage or repair will be more prone than less susceptible individuals to adjust their long-term development in response to damaging or healing experiences. Second, even if all individuals use both internal and external cues to predict their lifespan, in principle, individuals could differ in their sensitivity to each type of information: that is, the development of some individuals may be shaped more by their internal somatic states (which predict the weathering of their bodies over time), whereas the development of others may be shaped more by their predictions of their external environment later in life. An alternative possibility is that some individuals are more susceptible to both types of information—internal and external—and relying less, for instance, on evolved prior expectations about

probabilities of environmental states. These are open and interesting questions that we leave for a future investigation.

Our motivation in writing this paper was not to contest current evolutionary accounts of psychosocial acceleration, but rather to enrich these accounts by freeing them from (exclusive) reliance on assumptions about environmental continuity. Our internal approach can account for empirically successful predictions of the model of Belsky et al. (1991), whilst also, as we have shown, generating novel and unique predictions. By relying less on temporal environmental continuity, the internal prediction process we propose extends the range of evolutionary conditions under which the mechanisms proposed by Belsky et al. might operate (Nettle et al., 2013). Our hope is that the predictions discussed here will be tested using longitudinal data, in order to determine to what extent each process (internal and/or external prediction) accounts for patterns in existing data; and, of course, we hope that our model will facilitate discovery of new data patterns as well. In general, we hope our article will stimulate studies of psychosocial acceleration, so that this fascinating and important phenomenon—with many implications for health and disease—will be better understood.

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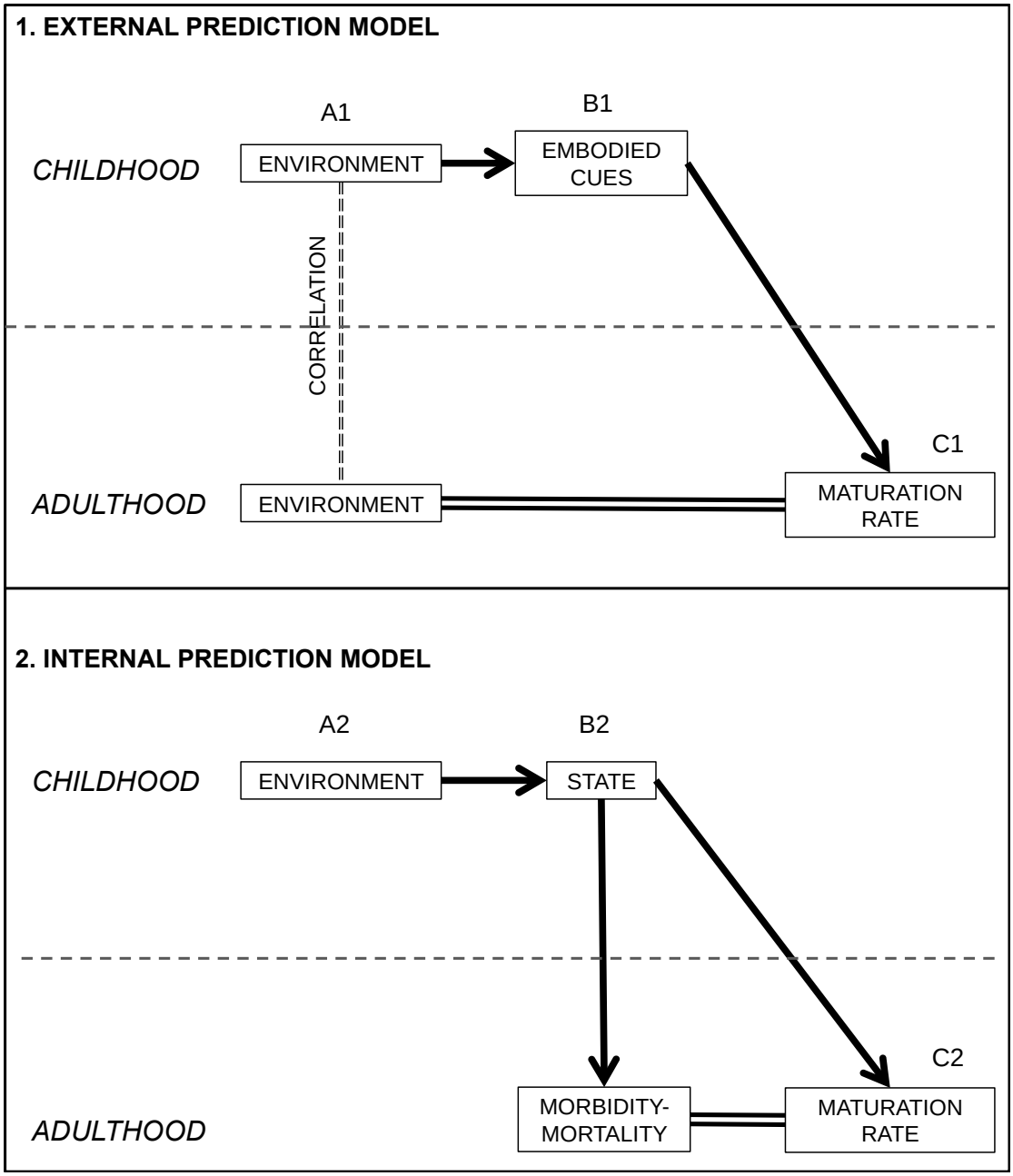
Figure 1. Schematic of (1) external and (2) internal prediction models showing conceptual similarities and differences between the three components A-C in each case. Arrows show causal pathways; the double lines show the adaptive relationship between maturation rate and either (1) external environment or (2) internal morbidity-mortality risk; the dashed line shows the correlation between environment in childhood and adulthood. In both cases the individual experiences the negative effects of psychosocial stress (A1 and A2). In the external prediction model, these effects become embodied cues (B1) that guide the individual's maturation rate (C1), so that it is adapted to external environment. In the internal prediction model, effects of early social stress are embodied not as cues, but as negative influences on 'state' (B2), which increases the individual's morbidity-mortality in adulthood, to which the maturation rate is adapted (C2).


1. EXTERNAL PREDICTION MODEL
A1
B1
CHILDHOOD
ENVIRONMENT
EMBODIED CUES
CORRELATION
C1
ADULTHOOD
ENVIRONMENT
MATURATION RATE
2. INTERNAL PREDICTION MODEL
A2
B2
CHILDHOOD
ENVIRONMENT
STATE
C2
ADULTHOOD
MORBIDITY-MORTALITY
MATURATION RATE

633

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