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COMMENT

Match Fitness: Development, Evolution, and Behavior: Comment on Frankenhuis and Del Giudice (2012)

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The application of evolutionary thinking to human physical and psychological medicine suggests several pathways through which evolutionary processes affect risk of disease. Among these is the concept of mismatch between an individual and its environment, either because the environment has changed for the whole species (*evolutionary novelty*) or because the environment has changed for an individual during its lifetime (*developmental mismatch*). Here we set a discussion of maladaptation and mismatch as a cause of psychopathology (Frankenhuis & Del Giudice, 2012) in the broader framework of developmental plasticity and life history trade-offs.

Keywords: evolutionary medicine, evolutionary novelty, developmental plasticity, mismatch, trade-off

The concept of mismatch is now well established in evolutionary medicine as one of the major pathways that can affect health and disease risk (Gluckman, Low, Buklijas, Hanson, & Beedle, 2011). The concept has been applied extensively to the risk of diseases such as obesity, diabetes, and myopia and to life-course events such as puberty. For example, with regard to obesity, the model simply posits that humans evolved to be adapted to a particular range of nutritional density and workload, whereas the modern environment now frequently lies outside this range, leading to metabolic capacity being overloaded and consequent obesity and metabolic disease.

This concept is central to a review by Frankenhuis and Del Giudice (2012), who have summarized psychological adaptation and maladaptation within an evolutionary and developmental framework. Their review is useful in no small part because it brings attention to the problems of language and metaphor. Adaptation and maladaptation clearly have very different meanings to the evolutionary biologist and to the practicing psychologist. Beyond this, their review serves to underscore an important tenet of evolutionary medicine and psychology—physiological and psychological mechanisms have both evolved to optimize fitness in the Darwinian sense, and not necessarily health, wellbeing, or longevity. Importantly, Frankenhuis and Del Giudice—particularly in their discussion of risky adaptive strategies—emphasize the need to put the context of evolutionary perspectives on individual variation rather than on group variation. Indeed, it could be argued that one of the great insights of Darwin was to focus on individual variation rather than "type," as was the norm in 19th-century biology, and it remains too easy to fall back into typology rather than assessing the context—biological, developmental, environmental, and social—of the individual.

But the concept of mismatch extends beyond the simple *evolutionary novelty* form of mismatch to that occurring within a life course—*developmental mismatch* (Gluckman et al., 2011). As recognized by Frankenhuis and Del Giudice (2012), the most extensive use of the concept of *developmental mismatch* has been in studies that fall under the rubric of *developmental origins of health and disease*, whereby factors in early life predispose adults to have a greater risk of obesity, heart disease, and diabetes. It has been suggested that the normative processes of developmental plasticity can become maladaptive if there is a mismatch between the circumstances that induce changes in developmental trajectory and the actual environment into which the organism matures (Bateson et al., 2004; Gluckman, Hanson, Spencer, & Bateson, 2005).

There is not a one-to-one link between genotype and phenotype. In virtually all multicellular taxa, a range of phenotypes can develop from a single genotype in response to environmental cues acting in early development. These mechanisms of developmental plasticity are strongly conserved, and there is a large body of literature arguing for their adaptive (in the evolutionary sense of increasing reproductive fitness) role (West-Eberhard, 2003). Developmental plasticity and robustness are often portrayed as polar opposites, but, as has been recently pointed out, there are multiple mechanisms leading to both consistency of phenotype (robustness) and plasticity, and these interact during development (Bateson & Gluckman, 2011). Multiple levels of organization from the molecular to the whole organism are involved. Although much plasticity is limited by critical windows during development, learning can be considered as a set of plastic mechanisms with a much broader temporal capacity.

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A fundamental feature of developmental processes is that of trade-offs, which are often formulated within life-history theory, although essentially any potentially adaptive response to an environmental cue or trait involves some form of trade-off. A favored classification of developmental plasticity uses the temporal dimension of any trade-off as its basis, considering plastic responses as either immediately adaptive or predictively adaptive (Gluckman & Hanson, 2004; Gluckman, Hanson, Spencer, & Bateson, 2005). For example, fetal growth retardation is a case of immediate adaptation in which the organism has reduced its growth rate because of maternal or placental signals of undernutrition so as to survive the fetal phase, but with a reduced chance of postnatal survival being the trade-off of being born smaller. In contrast, other responses do not induce an early phenotypic change or a phenotypic change with concurrent survival advantage. A predictive adaptive response may be a phenotypic change induced early in life for advantage later in the life course, or it may be a shift in developmental trajectory that leads to a phenotype of advantage in the anticipated later environment. To be adaptive, that advantage must be reflected in increased fitness—the most obvious way is in increased survival until and through peak reproduction. Some inductive cues may be reliable, such as in the case of day length inducing differences in coat thickness in the offspring of multivoltine rodents, or they may be probabilistic, as in the case of predicting future nutritional or stressor environments from those experienced in early life. However, shifts in phenotypic development may become disadvantageous later in life if the correlation between the inductive cue and the environmental conditions actually encountered is poor. This general model is well categorized (Gluckman, Hanson, & Beedle, 2007; Gluckman, Hanson, & Spencer, 2005) and has been extensively used to explain the developmental origins of health and disease (Gluckman, Hanson, Cooper, & Thornburg, 2008).

The application of evolutionary arguments to behavior has been fraught with controversy, in no small part because of the deterministic approach taken by the school of thought led by Cosmides and Tooby (Duchaine, Cosmides, & Tooby, 2001). Similarly, the widely held dichotomy of explaining behaviors as either learned (nurture) or innate (itself a word fraught with difficulty; Mameli & Bateson, 2011; but generally assumed to reflect genetic determinants) has been equally limiting. The growing understanding of development both at a systems level and at a molecular level, where there is increasing evidence for the role of epigenetic mechanisms, provides a paradigmatic shift in our understandings of how phenotypes emerge and persist.

Equally, we have a problem of translating concepts relating to behavior in a current generation to those of our evolutionary past. Nowhere is this more obvious than in the concept of fitness itself. *Fitness* has a precise meaning in evolutionary biology and relates to the probability that a trait of the organism will be transmitted to future generations. In practice this is generally interpreted as the number of grand-offspring or fertile offspring carrying that trait. Inclusive fitness extends this concept to allow for kin selection. But formal estimates of fitness in humans can take no account of evolutionarily novel cultural practices, such as contraceptive use, that disconnect behavior from fertility. This limits the way in which hypothesis testing relating to biological, as opposed to cultural, evolution is conducted in modern evolutionary medicine and psychology. A recent review by Nesse (2011) is commended for discussing how to create and test hypotheses in this domain. Equally, it is important to recognize that mismatch is but one set of pathways by which evolutionary mechanisms generate risk for an individual (Gluckman et al., 2011). For example, a number of emotional symptoms and states (anxiety, depression, and phobias) can be interpreted as exaggerated defense mechanisms (Nesse & Ellsworth, 2009).

In their discussion of psychological maladaptation, Frankenhuis and Del Giudice (2012) outline the maladaptive consequences of developmental mismatch, distinguishing two potential mechanisms: (a) a change in environment after initiation of a developmental program based on correct perception of an early life cue and (b) failure to correctly perceive an early life cue even if the lifetime environment remains constant. Here we are beginning to see tantalizing opportunities for intervention by manipulation of early life cues—an approach already demonstrated for the physiological consequences of developmental mismatch (Vickers et al., 2005)—but much research remains to be done, particularly around the identification of critical periods during which intervention is possible before the developmental program "locks in."

Within this evolutionary framework, it is interesting to consider another aspect of risk-taking behavior reflecting both developmental and evolutionary mismatch, namely that associated with adolescence. Indeed, adolescence itself might be considered a consequence of changing social and physical environments leading to a loss of temporal mismatch between different maturing systems—in this case the reproductive system and frontothalamic pathways controlling executive function (Steinberg, 2005). Gluckman and Hanson have argued that adolescence has in recent decades become a much more prominent component of the life course (Gluckman & Hanson, 2006a, 2006b). The age of puberty and therefore the age of onset of adolescence have fallen dramatically since the early 19th century. This appears to be related to improvements in maternal and child nutrition and health. Although speculative, Gluckman and Hanson suggest that this might reflect the removal of constraints on maturation associated with urbanization and agriculture that historically arose from malnutrition and infection. They further speculate that the earlier age of modern puberty might reflect a return to a putative age of maturation in a Paleolithic past. Against this background, other influences such as poor prenatal nutrition or perinatal stress may further modulate life history patterns to accelerate timing to maturation in an environment of risk. Such an explanation is given further support by the observation of accelerated maturation in modern hunter–gatherer groups with high extrinsic juvenile mortality (Walker et al., 2006). But on the other hand, Gluckman and Hanson point out that in modern Western societies the end of adolescence, in other words the age of acceptance as an adult within the community, has been further delayed. The consequence is that the duration of "adolescence" has stretched from perhaps 2 to 4 years at the beginning of the 19th century to more than a decade at present—within 200 years. This is not without consequences—both boys and girls who undergo earlier puberty exhibit higher rates of conduct disorder, depression, and other symptomatology than do their peers who undergo puberty at a normal age (e.g., Michaud, Suris, & Deppen, 2006). In that study, boys in particular showed adverse outcomes, with a nearly fivefold greater risk of attempted suicide.

Neurophysiological studies are casting considerable light on this temporal mismatch and its psychological consequences. Func-

tional studies have shown that aspects of judgment do not mature until into the third decade of life (Cauffman & Steinberg, 2000). Other studies have suggested that risk-taking behaviors and their underlying neural pathways may be particularly exaggerated during adolescence (Somerville & Casey, 2010). But the most intriguing observations are the multiple imaging studies that suggest that frontothalamic pathways are not fully mature until well into the third decade of life (Gogtay et al., 2004; Lebel, Walker, Leemans, Phillips, & Beaulieu, 2008). This raises three evolutionary hypotheses that are testable and very relevant to the issues of concern in respect of adolescent behavior.

First, is it simply that executive function, emotional control, and self-control have always been underpinned by a very slowly maturing system, which is reflected in the well-established respect all cultures give to older people for their wisdom and judgment? But what has happened is that those skills were not needed in the less complex societies of the pre-electronic age where people essentially operated in small networks, even if they lived in urban conurbations (one is reminded of Dunbar's (2003) calculation that humans evolved to live in groups of no more than 100 to 150 people). But now with the combination of greater physical and certainly greater communicative ability, these later components of neural maturation are needed to cope optimally in society. In this model, adolescent behaviors can be seen to reflect the evolutionary novelty of the more complex world, and they are given further emphasis by the shifting pattern of biological maturation induced by environmental change.

Second, it could be argued that the changed social milieu means that there are simply more skills to learn, and it takes a longer time to get to the level of social skill necessary in the typical modern environment. There is little or no evidence to support this hypothesis.

But a third hypothesis is one associated with developmental plasticity and cue perception. Could it be that the big changes in the way modern children are reared have changed the patterns of frontothalamic maturation? It is well established in neurobiology, for example in the visual system, that early experience can change the pattern of development of neural pathways. Further, there is compelling evidence that noncognitive capacities expressed in later life are dependent on the pattern of early child rearing (Knudsen, Heckman, Cameron, & Shonkoff, 2006). In the last three decades there has been a dramatic shift in patterns of child rearing, with adolescence becoming less rigid, particularly because of more affluence and more fiscal freedom alongside the freedom created by the Internet and cell phones. On the other hand, the rearing of infants and prepubertal children has become more controlled from very young ages for a variety of reasons. These include the impact of women entering the workforce, requiring greater supervisory control of children for longer each day, media-fueled concerns over child safety, and greater attention being given to formal education from a younger age at preschool. Are we seeing here an example of developmental mismatch in which the maladaptive consequences might benefit from more thoughtful attention to early childhood experience?

In the example of adolescent behavior, we see the conflation of the neurophysiological with the behavioral. Increasingly we must see these as two sides of the same coin. For too long the language of one has been ignored by the other. Indeed, Frankenhuis and Del Giudice (2012) are to be congratulated for emphasizing the need for an integrated approach. Importantly, they realize that child and adolescent development is not tightly canalized; rather, it is a process in which the inherited genotype and perhaps inherited epigenotype give rise to an organism that is subsequently molded by experiential processes, inducing epigenetic and neurobiological changes (learning) that have an evolutionary and adaptive underpinning. Such plastic responses arise from evolved mechanisms that allow an organism to adapt to circumstances that change in a time frame intermediate between that which is coped with by homeostasis on one hand and selection on the other, but they do not necessarily create behaviors that modern society judges to be optimal or healthy. Our world has changed too much for that to be the case.

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